OIKOS 61: 297–306. Copenhagen 1991

Population dynamic consequences of the interplay between parasitism and intraspecific competition for host-parasite systems

Michael E. Hochberg

Hochberg, M. E. 1991. Population dynamic consequences of the interplay between parasitism and intraspecific competition for host-parasite systems. – Oikos 61: 297–306.

Parasites have the potential to influence *intra*specific competition within the population of a single host species in a manner analogous to a predator or a parasite influencing the outcome of *inter*specific competition between two competing species. I show how the competitive interactions of healthy and diseased hosts may influence the population dynamics of the host and, more particularly, the ability of susceptible and infected sub-populations of the host to persist. The dynamic character of the system can resemble interspecific competition between two species, predator-prey interactions, or, in some cases, a combination of the two. I suggest that in real systems it may be difficult to discriminate the relative contributions of parasitism and intraspecific competition to the limitation of host populations.

M. E. Hochberg, Centre for Population Biology, Imperial College, Silwood Park, Ascot, Berks SL5 7PY, England (present address: Laboratoire d'Ecologie – URA 258, Ecole Normale Supérieure – CNRS, 46 Rue d'Ulm, F-75230 Paris, France).

Both experimental and theoretical studies indicate that parasites can be instrumental in mediating interspecific competition between speices of host (Park 1948, Holt and Pickering 1985, Price et al. 1986, 1988). In such instances, the parasite influences competitive outcomes by having a larger negative impact on the population of one host species than on the other. For example, Holt and Pickering (1985) showed that two host species which otherwise did not compete could do so via an indirect link with a shared parasite. Some of the predictions of their model are supported by empirical studies (Price et al. 1988).

Parasites may also influence predator-prey interactions through induced changes in host behaviour (Anderson 1979a, Dobson 1988). Often, this involves the parasite rendering its intermediate host more vulnerable to predation by the definitive host. For example, fresh water amphipods (*Gammarus lacustris*) infected with an acanthocephalan parasite (*Polymorphus minutus*) have a tendency to attach themselves to floating objects. Mallard ducks forage the environs of floating objects extensively, and thus encounter and consume infected gammarids much more frequently than healthy ones (Denny 1969, Holmes and Bethel 1972). Recently, Dobson (1988) developed mathematical models for parasite induced changes in (1) the behaviour of insect vectors, (2) the susceptibility to predation of intermediate hosts, and (3) intermediate host fecundity. He argued that the transmission biology of the parasite (e.g. use of vectors or intermediate hosts) combined with habitat heterogeneity should ultimately determine how parasites influence host behaviour, and by extension, the evolution of complex life-cycles of the parasite and population dynamics.

The argument developed in the present study is that, on a more basic level than the mediation of interspecific competition or predator-prey interactions, the harbouring of a parasite by individual hosts explicitly divides the host population into competing sub-populations of healthy and infected hosts. In principle, the competitive relationships between these sub-populations can have many of the characteristics of interspecific competition

Accepted 12 April 1991

Table 1. Biological interpretations of the rate constants from Eqs (1) and (2) in the main text.

Parameter	Interpretation
a	Birth rate of healthy offspring from healthy mother
c	Birth rate of healthy offspring from diseased mother
v	Birth rate of infected offspring from diseased mother
b	Death rate due to causes independent of density and disease
μ	Death rate due to disease which is independent of densities
γ	Recovery rate of infected hosts to the healthy state
β	Transmission rate of the parasite
α_{ij}	Loss rate[1] of population i, dependent on the density of population j

[1] Loss rate includes reductions in reproduction and survival.

between two different species. Although it is reasonable to expect that harbouring a parasite will usually result in little change or a decrease in competitivity, this need not be the case. For example, parasitisation of juvenile sand shrimps in the genus *Crangon* by a microsporidian, *Pleistophora crangoni*, results in substantial increases in shrimp weight as compared to uninfected shrimp (Breed and Olsen 1977). What impact these larger shrimp have on the competitive interactions, however, is not known.

The aim of this study is to elucidate how potentially lethal, directly transmitted, microparasites (such as viruses, fungi, bacteria, and protozoa) can influence the population dynamics of their invertebrate host via the competitive relationships of healthy and infected hosts. First, I develop a simple mathematical model of Lotka-Volterra form where both parasitism and intraspecific competition may act to limit the population of the host. Second, I present the persistence conditions for each of the populations of healthy and diseased hosts. Third, I show how the dynamics of the system formally resemble either interspecific competition between two species or predator-prey interactions, depending on the intrinsic growth rate of the parasitised sub-population of the host and the impact of the parasite on the competitive ability of diseased hosts. Finally, I discuss these results with respect to the interacting roles of parasitism and competition in the dynamics of infectious disease.

The mathematical model

The model considered here extends previous theoretical developments on intraspecific competition in host-parasite systems (Anderson 1979a, Anderson and May 1981, Bernstein 1986) to situations in which the competitive relationships involving healthy and diseased hosts are not necessarily the same. The purpose of this analysis is not to give an exhaustive account of how parasites may affect their host populations, but to highlight some of the interesting biological relationships between parasites and their hosts which give rise to population patterns. Biological interpretations of the rate constants of the model are given in Table 1.

The model is compartmental in nature and considers populations of healthy invertebrate hosts and those infected with a directly transmitted microparasite, such as a virus or bacterium. The actual distribution of parasites among individual hosts is not explicitly modelled as is often the case for macroparasitic infections (e.g. helminths and arthropods, Anderson 1979a). The model is kept intentionally simple so as to highlight the potential importance of some simple demographic processes to real systems. More realistic models could include, for instance, resistance to parasitic infection (Anderson 1986) or long-lived external stages of the parasite (Anderson and May 1981, Hochberg 1989).

In the model system, the healthy (= susceptible) class of hosts of density S reproduce and die at per capita rates of a and b, respectively. The parasite is transmitted horizontally at a rate β when a member of the diseased (= infected and infectious) class of host (of population density I) comes into direct contact with a susceptible host. The term for overall transmission rate, βIS, assumes that the healthy and diseased populations of the host mix homogeneously and that there are no long-lived external stages of the parasite, a reasonable first approximation for directly transmitted microparasites (Anderson and May 1981). Once infected, a host may either give birth to susceptible offspring at a per capita rate c, vertically transmit the parasite to its offspring (v), recover to the susceptible state (γ), or die from natural or disease-induced causes (b and μ, respectively). The total per capita rate of reproduction of hosts infected with the parasite is thus $c + v$. The ability to recover from infection and/or reproduce whilst infected is common among vertebrate hosts (for examples, see Anderson and May 1979, Dobson 1988). Though less sophisticated than for vertebrates, invertebrate hosts are also known to possess immune responses to parasitism (Harvell 1990).

Furthermore, the competitive effects (resulting in population losses due to mortality or reductions in reproduction) on class i by class j, or α_{ij}, are characterized by the constants α_{IS}, α_{SS}, α_{SI}, and α_{II}, for susceptibles on infecteds, susceptibles on susceptibles, infecteds on susceptibles, and infecteds on infecteds, respectively (Table 1). These constants do not have the same interpretations as conventional competition coefficients; rather they encapsulate the negative impact of one host class on its own survival and reproduction, or on the survival and reproduction of the other host class. Though probably a reasonable first approximation to competitive relationships, more realistic modifications should be made in future studies.

When all of the competition constants are equal (i.e.

parasites have no impact on competitive relationships) we recover the density dependent model considered by Anderson and May (1981). The analysis presented here extends their results to cases in which competitive interactions are modified by the parasite. Various biological mechanisms may give rise to differences in the competition constants. Three different mechanisms, which need not act independently of one another, are described below.

First, the disease caused by the parasite can alter the impact of competition on infected hosts via constants α_{II} and α_{IS}. As the health status of infected hosts is reduced, there is a corresponding increase in the per capita negative effect of all other competitors (healthy or infected) on the population growth rate of the infecteds. This makes intuitive sense if we think of competition as increasing the nutritional stress experienced by diseased hosts (Steinhaus 1958, Schultz and Baldwin 1982). However, we could also envisage situations in which the parasite reduces the impact of resource limitation on diseased hosts, as in cases where the organism is a beneficial symbiont.

Second, harbouring the parasite can change the competitive impact of diseased hosts on other diseased hosts and on healthy hosts as reflected by the constants α_{SI} and α_{II}. This may involve changes in the infected host's feeding rate (exploitation) and/or feeding behaviour (interference). In general, parasitism can be expected to lower the competitive impact of diseased hosts. Situations in which the parasite induces the host to consume more resource, or become more aggressive so as to prevent other hosts from obtaining resources, are also possible, though probably more rare in nature. This is the case for some macroparasites, such as insect parasitoids (e.g. Moore 1989). For example, Moore (1989) has recently shown that the braconid wasp, *Cotesia koebelei*, can induce an extra feeding larval instar in the butterfly, *Euphydryas editha*.

Third, spatial segregation of infected and healthy hosts should result in reductions in the intensity of interclass competition (via reductions in constants α_{SI} and α_{IS}). Although spatial segregation between healthy and diseased hosts may be a widespread feature of animal populations (e.g. Breed and Olsen 1977), little is known about the effects of segregation on the population growth rates of host-parasite systems.

The mathematical model is composed of two differential equations, one each for the densities of susceptible (S) and infected (I) hosts:

$$\frac{dS}{dt} = rS + eI - \beta IS - (\alpha_{SI}I + \alpha_{SS}S)S \qquad (1)$$

$$\frac{dI}{dt} = \beta IS + (v - d)I - (\alpha_{IS}S + \alpha_{II}I)I \qquad (2)$$

Here, for notational convenience, r is the net intrinsic rate of increase of susceptible hosts (i.e. $r = a + b$), e is the contribution of infected hosts to the growth of the susceptible population (i.e. $e = \gamma + c$), and d is the net loss rate from the infected population (i.e. $d = b + \mu + \gamma$). d is commonly referred to as a measure of the pathogenicity of the parasite, even though the evolution of d could also reflect changes in the host's ability to recover from infection (Anderson and May 1981). For the special case of $e = 0$ (the infection is invariably lethal and infected hosts do not give birth to healthy offspring) and $v - d > 0$ (i.e. vertical transmission exceeds losses due to parasitism) we recover the mathematical form of the classical Lotka-Volterra competition equations. Unless the parasite induces infected hosts into reproducing at a faster rate than for healthy hosts, then the condition $r \geq v - d + e$ (or substituting and rearranging parameters, $a \geq c + v$) must hold.

Persistence conditions of the host and its disease

Except for one special case (see below), the persistence of the host and the disease can be determined directly from their respective basic reproductive rates (Anderson and May 1979, 1981). When the basic reproductive rate of a given population exceeds unity, it can be said to persist; whereas if inferior to unity it eventually goes (locally) extinct. A more detailed mathematical treatment of the invasion and persistence of the host and parasite is presented elsewhere (Hochberg; unpubl.).

Persistence of healthy hosts

The basic reproductive rate of the healthy host, R_{0S}, is the average number of surviving progeny produced by a single host individual (i.e. as the density of the healthy host approaches zero). It takes the general form (from Eq. (1))

$$R_{0S} = \frac{a + eI/S}{b + \beta I + \alpha_{SS}S + \alpha_{SI}I} \qquad (3a)$$

Clearly, as $S \to 0$ the healthy host always persists so long as $e > 0$.

If $e = 0$ (the disease is invariably lethal and diseased hosts cannot give birth to healthy hosts), then persistence of the host is no longer assured. In this case, we can simplify (3a) to give

$$R_{0S} = \frac{a}{b + (\beta + \alpha_{SI})I} \qquad (3b)$$

One of two further situations arise depending on the

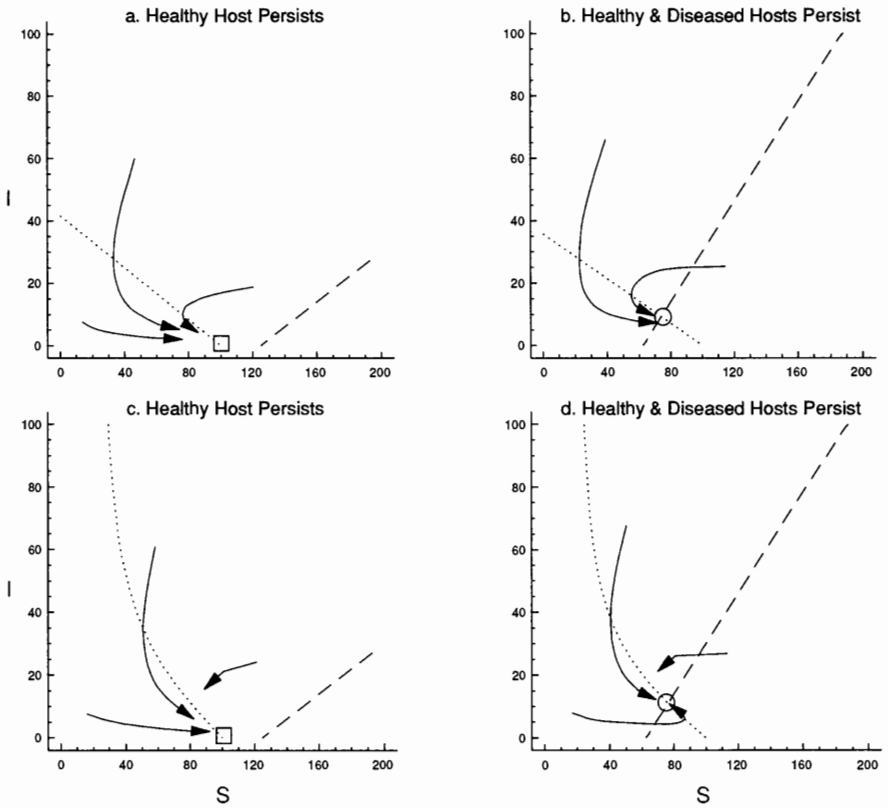

Fig. 1. Phase diagrams of dynamic outcomes when the parasite is principally transmitted horizontally. a. Infected hosts cannot persist ($\beta = 0.014$, $e = 0$); b. Healthy and infected hosts persist at stable equilibrium ($\beta = 0.018$, $e = 0$); c. Infected hosts cannot persist ($\beta = 0.014$), $e = 0.5$); d. Healthy and infected hosts persist at stable equilibrium ($\beta = 0.018$, $e = 0.5$). Isoclines for healthy hosts (dotted line) and infected hosts (dashed line) and typical trajectories (given by arrows) are based on Eqs (1) and (2). Symbols: square = stable equilibrium of healthy host alone; triangle = stable equilibrium of infected host alone; circle = stable equilibrium of healthy and infected hosts. Parameter values, unless otherwise specified, $r = 1$, $v - d = -0.5$, and for all i and j, $\alpha_{ij} = 0.01$.

sign of the intrinsic growth rate of infected hosts ($v - d$).

First, if $v < d$ then the infected cannot persist in the absence of the healthy host (i.e. prior to the invasion of the latter) and introduction of the healthy host will result in its own persistence if its intrinsic growth rate is positive, or $a > b$.

Second, when $v > d$, persistence is determined by evaluating (3a) at the equilibrium of the infected host alone ($v - d/\alpha_{II}$), resulting in the condition

$$R_{0S} = \frac{a}{b + (\beta + \alpha_{SI})\,(v - d/\alpha_{II})} > 1 \qquad (3c)$$

or, that the net intrinsic rate of increase of susceptible hosts is sufficiently high and the parasite sufficiently pathogenic. This requirement simply says that by shortening the life-span of infected hosts, pathogenic parasites reduce losses in the healthy population through reduced transmission and competition over the lifetime of the infected host.

Persistence of infected hosts

The infected host persists in the system if its basic reproductive rate (the average number of new infections caused by one primary case), R_{0I}, is greater than unity. Assuming that the intrinsic rate of increase of healthy hosts is positive, we can directly cast R_{0I} in a similar form to Eq. (3c). Thus, if

$$R_{0I} = \frac{(\beta r/\alpha_{SS}) + v}{d + r\alpha_{IS}/\alpha_{SS}} > 1 \qquad (4)$$

then the parasite persists. As will be discussed below, this is a sufficient, but not a necessary condition for the persistence of the disease when $e > 0$.

Inequality (4) can be rearranged to show that the parasite can only invade the system if it is either sufficiently vertically ($v > d$) or horizontally transmitted ($\beta > \alpha_{IS}$). Note that increasing the competitivity of the system (via α_{SS}) can either increase or decrease the invasibility of the disease, depending on the rate of horizontal transmission. If $\beta < \alpha_{IS}$, then increased competitivity results in increased invasibility, whereas when this condition is reversed the opposite effect occurs. In circumstances where the general competitivity of the environment increases (i.e. proportional increases in α_{IS} and α_{SS}), we should expect decreased invasibility of the diseased population.

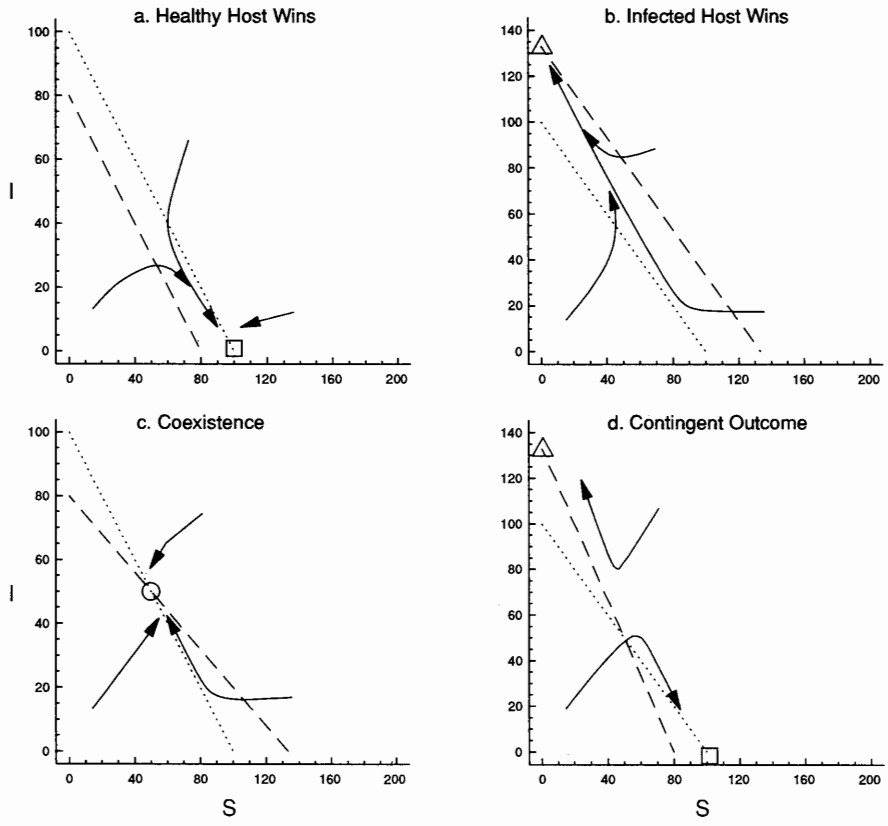

Fig. 2. Phase diagrams of dynamic outcomes when the parasite is principally transmitted vertically. a. Healthy hosts competitively eliminate infected hosts; b. Infected hosts competitively eliminate healthy hosts ($\alpha_{IS} = \alpha_{II} = 0.006$); c. Healthy and infected hosts persist at stable equilibrium ($\alpha_{IS} = 0.006$); Competitive elimination of either healthy or infected hosts, depending on initial densities ($\alpha_{II} = 0.006$). Parameter values, unless otherwise specified, $r = 1$, $v - d = 0.8$, $\beta = 0$, $e = 0$, and for all i and j, $\alpha_{ij} = 0.01$. Isoclines and symbols as for Fig. 1.

The influences of transmission strategy and competitive interactions on the persistence of the host and its disease

Here I employ graphical techniques to describe some interesting qualitative dynamic outcomes of the system. This involves the plotting of zero-growth population isoclines for each of the two host classes, and examining typical trajectories of the populations in the different areas of the phase-space (Rosenzweig and MacAurthur 1963). The mathematical formalism behind this analysis can be found elsewhere (Hochberg, unpubl.)

As is evident from the mathematical form of Eqs (1) and (2), classical elements of predation (i.e. β), competition (i.e. α's), and parasitism (i.e. d and e) are each a part of the host-parasite system. Indeed, the basic character of the system (whether it is predatory or competitive) depends on the signs of only two growth rate terms: the intrinsic growth ($v - d$) and the density dependent growth ($\beta - \alpha_{IS}$) of the infected class of host. A second important character of the system (especially in the competitive case described below) is whether it is 'parasitic' or not. This is primarily determined by the extent of the recovery of infected hosts and the birth of healthy offspring from infected mothers (e).

This section is divided into three parts. In the first, I consider how horizontally transmitted parasites give rise to population dynamics which are similar to conventional predator-prey systems. In the second part, I show how the population dynamics of vertically transmitted parasites can be formally similar to, or even the same as, classical competition between two different species. In the third part, extreme cases are described for parasites which are sufficiently transmitted both vertically and horizontally, and for parasites with insufficient transmission rates.

(1) Predation ($v < d$ and $\beta > \alpha_{IS}$)

The impact of infectious disease on host population dynamics assumes a predatory character when the parasite relies on horizontal rather than vertical transmission for persistence. According to the conditions outlined in Eqs (3a-c), the healthy host (i.e. prey) always persists in the presence of the disease. The dynamic character of the system, depends to some extent on whether there is feedback from the infected to susceptible population class.

If $e = 0$, then one of two outcomes can occur. In the first, the parasite cannot persist in the system because

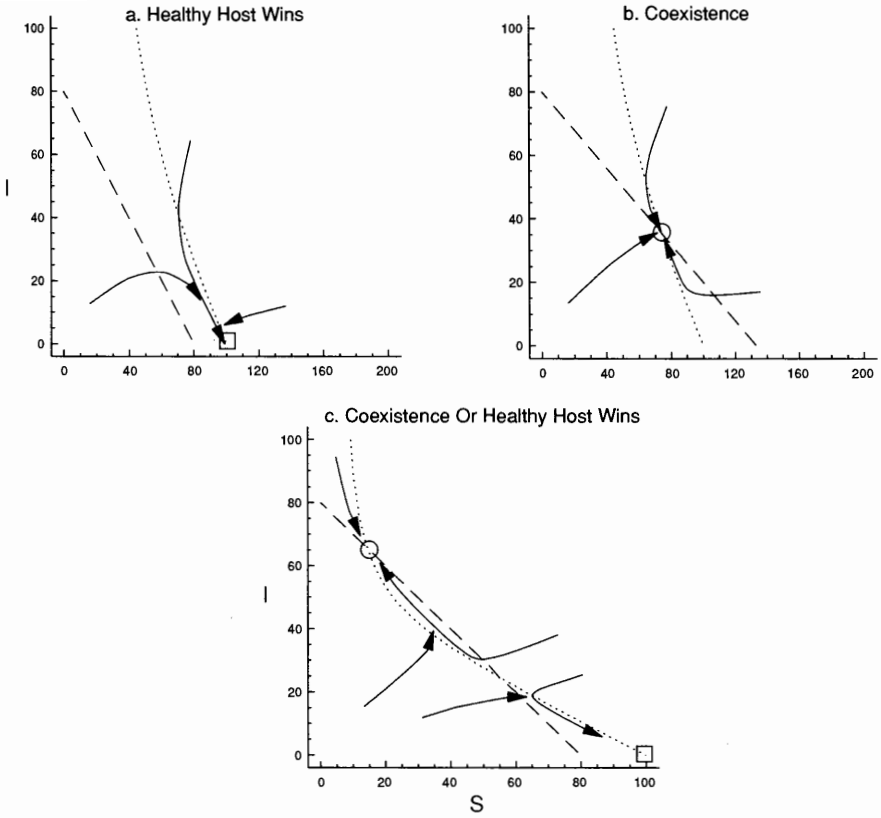

Fig. 3. Phase diagrams of dynamic outcomes when the parasite is principally transmitted vertically and infected hosts contribute to the population growth of the healthy host. a. Healthy hosts competitively eliminate infected hosts; b. Healthy and infected hosts persist at stable equilibrium ($\alpha_{IS} = \alpha_{II} = 0.006$); c. Either healthy and infected hosts coexist at a stable equilibrium or the infected host is competitively eliminated, depending on initial densities ($e = 0.1$, $\alpha_{SI} = 0.02$). Parameter values, unless otherwise specified, $r = 1$, $v - d = 0.8$, $\beta = 0$, $e = 0.2$, and for all i and j, $\alpha_{ij} = 0.01$. Isoclines and symbols as for Fig. 1. Note the difference in scale of the horizontal axis of Fig. 3c.

its transmission rate is not sufficiently larger than the competitive effect of healthy on infected hosts (condition (4), Fig. 1a). According to inequality (4), parasite persistence is also reduced as the system becomes more competitive (via α_{SS}), and pathogenicity is increased (via d). Standard techniques can be used to show that the system equilibrates to constant populations of the healthy host (May 1974). In the second outcome, if β is sufficiently greater than α_{IS} (as determined by $R_{0I} > 1$), then healthy and diseased hosts coexist at a stable equilibrium point (Fig. 1b). Similar to the classical Lotka-Volterra model of predation, the system has a tendency to exhibit population cycles. These cycles are dampened to a stable equilibrium point due to the presence of the self-limitation terms α_{SS} and α_{II}.

If $e > 0$, then the same two persistence outcomes hold as for the case of $e = 0$ (Fig. 1c, d), but with the difference that the parasitic nature of the system tends to dampen oscillations (compare cases of Fig. 1a, b with Fig. 1c,d). Biologically, we would expect that increases in e should be accompanied by decreases in $v - d$, since they share some of the same parameters. Hence, the added resilience of the system afforded by increases in e should be accompanied by a reduced propensity for the disease to persist in the system.

One example of a parasite which acts much as a 'predator' is the baculovirus of the palm rhinoceros beetle, *Oryctes rhinoceros*. The virus is efficiently transmitted at beetle densities too low for intraspecific competition between beetles to be of much importance (i.e. $\beta > \alpha_{IS}$) (Zelazny 1976, Hochberg and Waage 1991). This, in combination with the ability of infected adult beetles to lay eggs for a short period following infection (i.e. $e > 0$), are thought to be important in the ability of the virus to regulate populations of the beetle to low and constant densities (Hochberg and Waage 1991).

(2) Interclass competition ($v > d$ and $\beta < \alpha_{IS}$)

This situation transpires when the parasite is principally transmitted vertically, rather than horizontally. The dynamics of this case is more complicated than the predation scenario.

If $e = 0$, then the model formally resembles the classical Lotka-Volterra model of competition (i.e. the sub-populations of healthy and infected hosts compete as if they were two separate species), and persistence of the healthy host is not assured. One of four 'competitive' outcomes can occur depending on the relative density independent and density dependent growth rates of each population. These outcomes are: (1) healthy hosts competitively eliminate infected hosts (condition (3c) holds and condition (4) is violated; Fig.

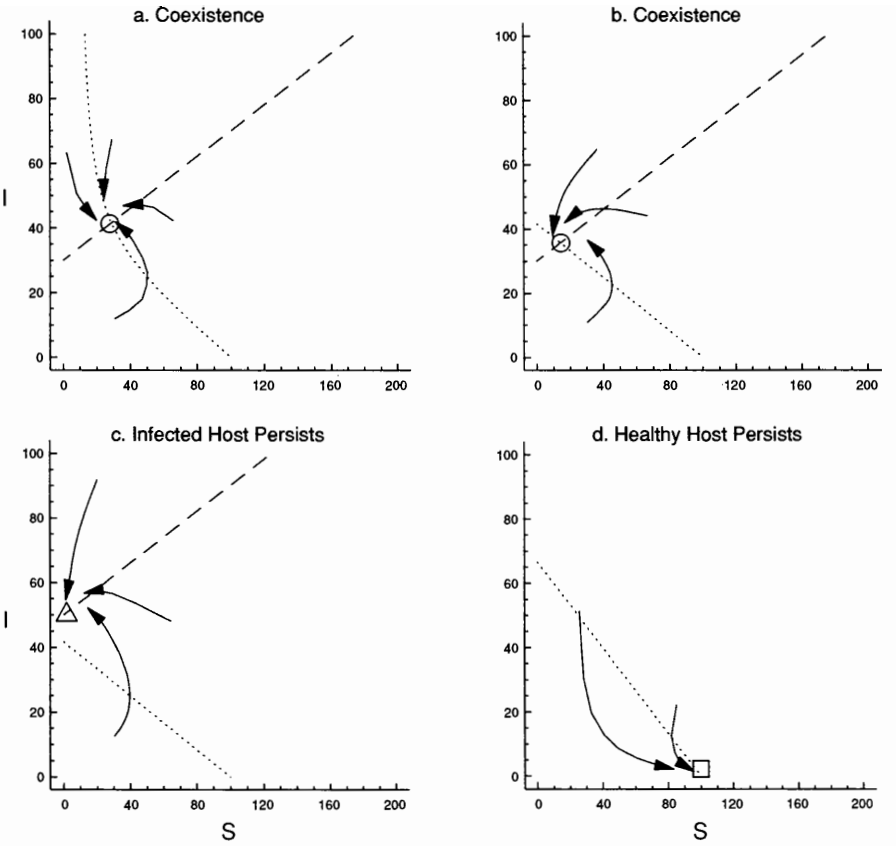

Fig. 4. Phase diagrams of dynamic outcomes when parasite is transmitted at high (a-c) or low (d) rates both horizontally and vertically. a. Coexistence at a stable equilibrium point when infected hosts contribute to the growth of the healthy population ($v - d = 0.3$, $e = 0.2$); b. Coexistence at a stable equilibrium point when infected hosts do not contribute to the growth of the healthy population ($v - d = 0.3$, $e = 0$); c. Infected hosts eliminate the population of healthy hosts ($v - d = 0.5$, $e = 0$); d. Infected hosts cannot persist ($v - d = -0.5$, $\beta = 0.005$, $e = 0$). Parameter values, unless otherwise specified, $r = 1$, $\beta = 0.014$, and for all i and j, $\alpha_{ij} = 0.01$. Isoclines and symbols as for Fig. 1.

2a), (2) infected hosts competitively eliminate healthy hosts ((3c) is violated and (4) holds; Fig. 2b), (3) healthy and infected hosts coexist at a stable equilibrium point (both (3c) and (4) hold; Fig. 2c), and (4) either healthy or infected hosts persist, but not both, depending on their initial densities (both (3c) and (4) are violated; Fig. 2d).

The healthy host tends to persist in the system as the competitive effect of infected on susceptible hosts diminishes with respect to infecteds upon themselves, whereas persistence of the infected host is enhanced by a reduced competitive effect of susceptibles on infecteds with respect to susceptibles on themselves. One possible way in which both host classes could persist at a stable equilibrium point would be if competition were reduced between host classes as compared to within host classes (e.g. spatial segregation of healthy and diseased hosts). This is the same condition required for competitive coexistence in the classical Lotka-Volterra model.

Allowing $e > 0$ qualitatively changes the competitive landscape, and condition (4) is no longer necessary (but is sufficient) for parasite persistence. The other condition is whether intraclass density dependence ($\alpha_{II}\alpha_{SS}$) exceeds interclass density dependence ($(\alpha_{SI} + \beta)(\alpha_{IS} - \beta)$). Three different phase-planes are possible: (1) the healthy host competitively eliminates the infected host (sufficient conditions are that (4) is violated and intraclass exceed interclass interactions; Fig. 3a), (2) both host classes coexist at a stable equilibrium point (condition (4) holds; Fig. 3b), or (3) either the healthy host eliminates the infected host, or the two classes persist at a locally stable equilibrium point (necessary conditions are that (4) is violated and interclass exceed intraclass interactions; Fig. 3c), depending on initial densities.

The final case is qualitatively different from the outcomes of the Lotka-Volterra competition model. If the density of healthy hosts is large compared to infecteds, then the infecteds are competitively eliminated. This equilibrium point resembles the unstable one for the case $e = 0$. But, if infecteds are numerous as compared with healthy hosts, then the two sub-populations may coexist at a stable node. Although this equilibrium resembles the stable case for pure competition ($e = 0$), it is not produced by the same mechanism. The purely 'competitive' equilibrium (i.e. case 2, Fig. 3b) is stabilised by self-limitation exceeding inter-class limitation. The stable 'parasitic' equilibrium described here is a result of the same mechanism which lends stability to the predatory scenario, that is births and recoveries to the susceptible class by infected hosts. Limits on the size of e are to be expected, since (unless infected hosts

produce more total offspring than healthy hosts) $r \geq e + v - d$.

Vertical transmission of microparasites such as viruses and microsporidia are well documented in the insects (Steinhaus 1963, Evans and Entwistle 1987), but there is very little information about how purely vertically transmitted parasites may affect the populations of their invertebrate hosts. An example where healthy and infected hosts appear to coexist at a competitive equilibrium is that of the spruce budworm, *Choristoneura fumiferana*, and its microsporidian parasite, *Nosema fumiferanae* (Wilson 1977). Thomson (1958a) has shown that the parasite can be transmitted either horizontally or vertically (ca. 100% efficiency). Although the infection can be lethal to larval budworms, sublethal infections (e.g. resulting in reductions in pupal weight and fecundity) appear to be more common (Thomson 1958b). Thus, two of the important conditions for the coexistence of healthy and diseased hosts appear to be met: sufficient vertical transmission and intraclass interactions exceeding interclass interactions (since some horizontal transmission occurs and α_{SI} should be substantially reduced as compared with other α's). However, since the magnitude of the competition constants are not known, it is possible that $\beta > \alpha_{IS}$ (near the disease equilibrium point) and, as such, the scenario given below and illustrated in Fig. 4b applies.

(3) Parasites which are either sufficiently or insufficiently transmitted both vertically and horizontally ($v > d$ and $\beta > \alpha_{IS}$, or $v < d$ and $\beta < \alpha_{IS}$)

In the first case, the parasite assumes a facultative strategy of being transmitted at relatively high rates both vertically and horizontally. It always persists in the system. Healthy hosts persist along with diseased ones at a locally stable equilibrium point if there is some inefficiency in vertical transmission (i.e. $e > 0$) (Fig. 4a). If, on the other hand, vertical transmission is 100% efficient and if recovery from disease is not possible, then healthy hosts can only invade (and coexist with infecteds) if the horizontal and vertical transmission rates are not too high and infected hosts have relatively little competitive impact on healthy hosts (Fig. 4b). Failure of these equilibrium requirements to be satisfied will mean than the infection persists at a prevalence of 100% in the host population (Fig. 4c).

In the second case, the parasite is insufficiently transmitted both vertically and horizontally and cannot persist in the system (Fig. 4d). Although I know of no real examples of this, it could potentially occur in cases where the competitiveness of the local environment increases so as to nullify the condition $\beta > \alpha_{IS}$.

Discussion

An important feature of the population biology of host-parasite associations is the creation of a distinct subpopulation of the host which can compete with healthy members of the host population for limiting resources, much in the same way as would two separate species. Specifically, the population dynamics of host-parasite systems may formally resemble interspecific competition between two species, a predator-prey interaction, or, in some cases, a combination of the two. Which of the two dominates the dynamics of the system depends importantly on the levels of horizontal and vertical transmission and the competitive impact of susceptible hosts on infecteds. As horizontal transmission dominates, the system takes on the character of a predator-prey interaction. But as the parasite relies increasingly on vertical transmission as a means for persistence, the association shifts more to a competitive system between susceptible and infected hosts. In highly competitive environments (i.e. when $\beta < \alpha_{IS}$), even if the parasite can be readily transmitted horizontally, it can only persist if sufficiently transmitted vertically. Persistence of host-parasite associations therefore should depend not only on the transmission and pathogenicity of the parasite, but on competitive relations involving healthy and infected hosts.

The considerable importance of the transmissibility (β) and pathogenicity (incorporated in parameters d and e) of parasites to the dynamics of arthropod host-parasite interactions is well recognised (e.g. Anderson and May 1981, May and Anderson 1983). The present study extends these results to cases where the parasite may alter the competitive balance of the host population. Thus, for instance, a healthy host population can, in a sense, 'rid itself' of a parasite by competitively eliminating infected hosts from the system. Conversely, parasites may persist via their induced competitive impact of infected hosts on susceptible ones (although sufficient horizontal and/or vertical transmission rates are also necessary). In cases where infected hosts do not contribute to the positive growth rate of the healthy host (i.e. $e = 0$), parasitism can even lead to the 'competitive elimination' of healthy hosts from the system.

In cases where the disease caused by the parasite does not affect the competitive relationships in the host population (i.e. α_{ij}'s equal for all i and j) some interesting dynamic outcomes may still result. For instance, if the intrinsic growth rate of the diseased host is sufficiently positive, then even small amounts of horizontal transmission will increase the propensity of the competitors to stably coexist (since intraclass interactions will always exceed interclass ones). Because when $\beta > 0$ interclass interactions must always be less than or equal to intraclass interactions, the two-equilibrium case cannot transpire (e.g. Fig. 3c).

But in order to obtain the full range of outcomes outlined in the competitive scenario (i.e. for vertically

transmitted parasites), parasites must alter competitive relationships involving healthy and infected hosts. Such modifications in competitive interactions not only have important implications for the persistence of infectious disease, but also for the coevolution of host-parasite associations (May and Anderson 1983). To some extent, coevolution will be driven by changes in the transmissibility of the parasite and associated pathogenicity of the disease (May and Anderson 1983). The present study shows that inter-class competition effects also enter into the basic reproductive rates of healthy and infected hosts (Eqs (3c) and (4)), and thus their evolution as either direct or indirect targets of natural selection should be taken into account.

In the model outlined here, regulation of the host population in the absence of the parasite occurred via intraspecific competition for a limiting resource. Even when the parasite is present in the system, directly imposed decreases in the life-span of hosts are not necessary for parasites to contribute to the regulation of their host (Anderson 1979a). Further, other types of population regulation could also be mediated through the action of parasites, such as vulnerability to predation (Anderson 1979a, Dobson 1988), parasitism (e.g. Levin 1983, Hochberg and Holt 1990, Holt and Hochberg, unpubl.), and interspecific competition (Holt and Pickering 1985). For example, Hochberg and Holt (1990) have recently shown that the regulatory ability of a primary parasite may be either enhanced or reduced by the invasion of a facultative hyperparasite. Although the details differ, the same appears to hold for the invasion of an obligate hyperparasite into an established host-parasite system (Holt and Hochberg, unpubl.).

In addition, incorporating more realistic functional forms for transmission and competition than those employed here should further diversify the dynamic landscape of the system. For instance, in cases where the parasite produces relatively short-lived external transmission stages, Anderson (1979b) has shown that transmission can be modelled using the functional form $\beta SI/(S_0 + S)$ (where S_0 is a saturation constant). At sufficiently low densities of susceptible hosts, the transmission rate will be larger than the competitive effect of susceptibles on infecteds and the parasite will act like a 'predator'; whereas when the population of susceptibles exceeds the threshold $(\beta/\alpha_{IS}) - S_0$, the interaction is dominated by competition. In brief, the densities of healthy and infected hosts themselves may determine the qualitative character of the system's dynamics.

Finally, host-parasite associations present an instructive model of how difficulties may arise appropriating specific sources of mortality (or changes in reproductive rates) to changes in the growth rate of populations. Thus, we may underestimate the contribution of resource limitation to changes in population growth by apportioning mortalities (and/or reductions in reproduction) solely to parasitism, based on the pathology of disease. On the other hand, in cases of asymptomatic infections for example, death or reduced reproduction of the host leading to population decline may be overattributed to resource limitation. The results of this study suggest that accurate estimation of how parasitism and resource limitation interact is essential to the understanding of the persistence of host-parasite associations.

Acknowledgements – I would like to thank R. D. Holt, J. H. Lawton and J. K. Waage for helpful discussions during the course of this study. This work forms part of the programme of the Natural Environmental Research Council's Centre for Population Biology at Silwood Park.

References

Anderson, R. M. 1979a. The influence of parasitic infection on the dynamics of host population growth. – In: Anderson, R. M., Turner, B. D. and Taylor, L. R. (eds), Population dynamics. 20th Symposium of the British Ecological Society, London 5–7 April, 1978. Blackwell, Oxford, pp. 245–281.

– 1979b. The persistence of direct life cycle infectious diseases within populations of hosts. – In: Levin, S. A. (ed.), Lectures on mathematics in the life sciences. Vol. 12, Am. Math. Soc., Providence, RI, pp. 1–67.

– 1986. Genetic variability in resistence to parasitic invasion: population implications for invertebrate host species. – Symp. Zool. Soc. Lond. 56: 239–274.

– and May, R. M. 1979. Population biology of infectious diseases: Part I. – Nature, Lond. 280: 361–367.

– and May, R. M. 1981. The population dynamics of microparasites and their invertebrate hosts. – Phil. Trans. R. Soc. Lond. B 291: 451–524.

Bernstein, C. 1986. Density dependence and the stability of host-parasitoid systems. – Oikos 47: 176–180.

Breed, G. M. and Olsen, R. E. 1977. Biology of the microsporidian parasite *Pleistophora crangoni* n. sp. in three species of Crangonid sand shrimps. – J. Invertebr. Pathol. 30: 387–405.

Denny, M. 1969. Life cycles of helminth parasites using *Gammarus lacustris* and an intermediate host in a Canadian lake. – Parasitology 59: 795–827.

Dobson, A. P. 1988. The population biology of parasite-induced changes in host behavior. – Q. Rev. Biol. 63: 139–165.

Evans, H. F. and Entwistle, P. F. 1987. Viral diseases. – In: Fuxa, J. R. and Tanada, Y. (eds), Epizootiology of insect diseases. Wiley, New York, pp. 257–322.

Harvell, C. D. 1990. The ecology and evolution of inducible defences. – Q. Rev. Biol. 65: 323–340.

Hochberg, M. E. 1989. The potential role of pathogens in biological control. – Nature, Lond. 337: 262–265.

– and Holt, R. D. 1990. The coexistence of competing parasites. I. The role of cross-species infections. – Am. Nat. 136: 517–541.

– and Waage, J. K. 1991. A model for the biological control of *Oryctes rhinoceros* (L.) (Coleoptera: Scarabaeidae) by means of pathogens. – J. Appl. Ecol. 28: (in press).

Holmes, J. C. and Bethel, W. M. 1972. Modification of intermediate host behaviour by parasites. – In: Canning, E. U. and Wright, C. A. (eds), Behavioural aspects of parasite transmission. Academic Press, London, pp. 123–149.

Holt, R. D. and Pickering, J. 1985. Infectious disease and species coexistence: a model of Lotka-Volterra form. – Am. Nat. 126: 196–211.

Levin, S. A. 1983. Some approaches to the modelling of coevolutionary interactions. – In: Nitecki, M. (ed.), Coevolution. University of Chicago Press, Chicago.

May R. M. 1974. Stability and complexity in model ecosystems. Second Ed. – Princeton University Press, Princeton, NJ.
– and Anderson, R. M. 1983. Epidemiology and genetics in the coevolution of parasites and hosts. – Proc. R. Soc. Lond. B 219: 281–313.
Moore, S. D. 1989. Regulation of host diapause by an insect parasitoid. – Ecol. Ent. 14: 93–98.
Park, T. 1948. Experimental studies of competition. I. Competition between populations of the flour beetles, *Tribolium confusum* Duval and *Tribolium castaneum* Herbst. – Ecol. Monogr. 18: 265–308.
Price, P. W., Westoby, M., Rice, B., Atsatt, P. R., Fritz, R. S., Thompson, J. N. and Mobley, K. 1986. Parasite mediation in ecological interactions. – Ann. Rev. Ecol. Syst. 17: 487–505.
– , Westoby, M. and Rice, B. 1988. Parasite-mediatated competition: some predictions and tests. – Am. Nat. 131: 544–555.
Rosenzweig, M. L. and MacArthur, R. H. 1963. Graphical representation and stability conditions of predator-prey interaction. – Am. Nat. 97: 209–223.
Schultz, J. C. and Baldwin, I. T. 1982. Oak leaf quality declines in response to defoliation by gypsy moth larvae. – Science 217: 149–150.
Steinhaus, E. A. 1958. Crowding as a possible stress factor in insect disease. – Ecology 39: 503–514.
– 1963. (Ed.) Insect pathology. – Academic Press, New York.
Thomson, H. M. 1958a. Some aspects of the epidemiology of a microsporidian parasite of the spruce budworm, *Choristoneura fumiferana*. – Can. J. Zool. 36: 309–316.
– 1958b. The effects of a microsporidian parasite on the development, reproduction and mortality of the spruce budworm, *Choristoneura fumiferana*. – Can. J. Zool. 36: 499–511.
Wilson, G. G. 1977. Observations on the incidence rates of *Nosema fumiferanae* in a spruce budworm, *Choristoneura fumiferana* population. – Proc. Ent. Soc. Ont. 108: 144–145.
Zelazny, B. 1976. Transmission of a baculovirus in populations of *Oryctes rhinoceros*. – J. Invertebr. Pathol. 27: 221–227.