

Weak sinks could cradle mutualistic symbioses – strong sources should harbour parasitic symbioses

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Abstract

Using a population model of selection on an obligate symbiont and its host, we evaluate how demographic differences across geographical landscapes can produce selection mosaics in interacting species. The model assumes that the host populations vary geographically from demographic sources to sinks in the absence of effects by the symbionts, and that a virulent and a relatively avirulent form of the symbiont compete with one another across all habitats. Our results indicate that productivity gradients can create selection mosaics across habitats, resulting in complex fitness landscapes over which evolution occurs. We find that relatively virulent symbionts only persist if they have an advantage over avirulent strains or species in terms of interference (i.e. competition, and/or cross-transmission) interactions. When such a trade-off exists, we predict that the more virulent symbiont is most likely to be found in habitats where host population growth is highest, whereas the more avirulent symbiont should tend to persist in more marginal habitats or even habitat sinks for symbiont-free hosts. Demographic sinks may be the habitats most likely to favour the origin of new mutualisms. Very productive mutualisms can be exploited by hyperparasites or cheaters. We discuss our findings in terms of geographical scenarios for the emergence of mutualisms, and the long-standing debate about geographical patterns in the maintenance of sex.

Introduction

How mutualisms arise is one of the outstanding questions in population biology and coevolution. Mutualistic interactions are ubiquitous, occurring in all biological communities, and involving all eukaryotes. These associations have been fundamental in the diversification of life (e.g. the historical origins of mitochondria and chloroplasts; Margulis, 1993), and they shape the basic trophic structure on which communities may develop. Nutrient accumulation in deep sea vents, the development of coral reefs, and terrestrial plant succession all rely upon mutualistic interactions. Nevertheless, many potentially mutualistic associations involve elements of

antagonism. Different environmental conditions can tip the balance back and forth between mutualism and antagonism (Palumbi, 1985; Thompson, 1988; Cushman & Beattie, 1991; Bronstein, 1994; Saikkonen *et al.*, 1998; Herre *et al.*, 1999). The result may be a geographical mosaic of outcomes reflecting antagonistic selection in some communities, and mutualistic selection in others. This geographical template of variable outcomes establishes the conditions for a particularly striking form of geographically structured coevolution (Thompson, 1994, 1999), in which the directionality of interspecific interactions varies across space (Nuismer *et al.*, 1999). Analyses of the conditions favouring the local dominance of antagonism or mutualism across geographical landscapes provide a way of understanding the origin and ongoing evolutionary dynamics of mutualistic interactions.

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Mutualisms are generally thought to arise from antagonistic interactions. Several authors have used models to explore the conditions that allow mutualisms to develop from ancestral free-living (e.g. Roughgarden, 1975; Frank, 1995; Law & Dieckmann, 1998) or symbiotic antagonistic (e.g. Yamamura, 1996) relationships. These models focus on the direction of selection in a local interaction between two species.

Recent models have expanded upon the results of these studies by exploring how the development of mutualisms may be shaped by spatial contexts (Doebeli & Knowlton, 1998; Nuismer *et al.*, 1999; Parker, 1999). For example, in a genetic model of coevolving interactions that differ in outcome among communities, Nuismer *et al.* (1999) have shown that a local mutualism may be either maintained or lost, depending upon the relative strengths of antagonistic selection in surrounding communities and the degree of gene flow into the focal community. In a model analysing geographically structured mutualisms, Parker (1999) has shown that mutualistic interactions may diverge geographically along different trajectories, depending upon initial conditions. Both models show that local mutualisms can sometimes be protected from invasion by alternative genotypes. In Nuismer *et al.*'s model, when local mutualistic selection is stronger than antagonistic selection in a neighbouring community, a local mutualism may remain stable even in the presence of a moderate level of gene flow from the antagonistic populations. In Parker's model, an established, local mutualism can be stable against invasion of alternative mutualistic traits from other populations.

Neither of these latter analyses included geographical differences in demography as a factor contributing to geographical patterns in mutualism or reduced levels of antagonism. A recent model of predator–prey coevolution over geographical gradients, however, suggested that evolution of lower rates of investment in costly antagonistic traits may sometimes be driven by the local demography of the interacting species (Hochberg & van Baalen, 1998). In particular, predators evolve to low attack rates and prey to low defence capabilities in less suitable habitats for the prey. The reason is that the prey is constrained to low levels of defence via a trade-off with per capita birth rate, and this reduction in prey defence induces the predator to invest less in costly offensive strategies.

In the present study we evaluate how geographical variation in the demographics of interacting symbionts and hosts can result in a gradient of outcomes ranging from antagonism to mutualism. Although the focus of our study is the evolution of symbiotic mutualism, we are more generally interested in the geographical evolution of virulence in host–parasite interactions. The current study considers how habitat suitability for the host and interference competition between symbionts (strains or species) contribute toward determining the symbiont's effect on its host.

Model

The types of associations we consider are those in which individuals of one species (which we will call 'symbionts') depend completely on individuals of a second species (called the 'host') for growth and reproduction. Typical examples are nutritional mutualisms between endosymbionts and their hosts (Smith & Read, 1997; Douglas, 1998). Hosts are assumed always to be infected by one of two symbiont types: 'virulents' of density V , and 'avirulents' of density A . We assume no introgression between symbiont types (as in a clonal selection model). The total population density of hosts is therefore $N = V + A$. A model in which hosts can recover from infection and infected hosts can give birth to uninfected offspring is currently under analysis.

When the symbiont has no effect on host biology, the per capita birth rate is a_U and natural death rate b . Both subpopulations may compete, their densities being limited jointly and uniformly according to a logistic-type model with a rate constant q (see also Hernandez, 1998). The symbiont can have pathogenic effects on the host, changing the background death rate (b) by rates α_V and α_A , for virulents and avirulents, respectively.

A mixture of horizontal and vertical transmission is a common syndrome in a range of endosymbiotic interactions (e.g. Douglas, 1994, 1995). We assume that each symbiont is potentially transmitted to new hosts via both routes.

A key ingredient in host–parasite models is the confrontation between symbiont strains within single host individuals following horizontal transmission (e.g. Levin & Pimentel, 1981; Levin, 1983; Hochberg & Holt, 1990; Mosquera & Adler, 1998). There appears to be no broad empirical pattern linking a symbiont's effect on its host with its degree of within-host competitive dominance (Douglas, 1995). For simplicity, we assume that only one symbiont strain can be associated for any substantial time period with a given host, such that when a virulent (or avirulent) is transmitted to a host already associated with an avirulent (or virulent), the latter is successfully displaced at a rate c_V (or c_A). Usually, c will be bounded between 0 and 1, but in cases where one species specializes in displacing the other – a 'hyper-symbiont' – $c > 1$ (Holt & Hochberg, 1998). Models of superinfection, where all symbiont strains or species are explicitly taken into account, have been recently reviewed by Mosquera & Adler (1998) (see also Levin, 1983), but are not considered further here. Thus in our model, hosts harbouring the competing symbiont become infected with virulent and avirulent symbionts via horizontal transmission at per capita rates $c_V V$ and $c_A A$, respectively. These rates follow the 'mass-action rule', often employed in models of predation and parasitism (e.g. Anderson & May, 1981).

Furthermore, we assume that vertical transmission occurs with 100% efficiency at rates $a_V + a_U$ and $a_A + a_U$

for hosts harbouring virulent and avirulent symbionts, respectively. The parameters a_V and a_A represent changes (either positive or negative) in reproduction experienced by hosts infected with virulent or avirulent symbionts, respectively. Models in which infected hosts lose symbionts and survive or reproduce without transmitting the symbiont are difficult to analyse (see Hochberg & Holt, 1990; Hochberg, 1991; Lipsitch *et al.*, 1996; for host-parasite interactions), and we leave such analyses for future study.

The differential equations describing the system are as follows:

$$dV/dt = [(a_U + a_V) - b - \alpha_V + (c_V - c_A)A - q(V + A)]V, \quad (1a)$$

$$dA/dt = [(a_U + a_A) - b - \alpha_A + (c_A - c_V)V - q(V + A)]A. \quad (1b)$$

Equations 1a–b can be represented more concisely as per capita changes, or

$$dV/Vdt = r_U + r_V + \delta A - qN \quad (1a')$$

$$dA/Adt = r_U + r_A - \delta V - qN, \quad (1b')$$

where $r_U = a_U - b$, $r_V = a_V - \alpha_V$ and $r_A = a_A - \alpha_A$. The quantity r_U is the per capita rate of increase of hosts independent of the effect of symbionts, and the quantities r_V and r_A are the *changes* in host growth rate, given associations with virulent and avirulent symbionts, respectively. The parameter $\delta = c_V - c_A$ characterizes the *net* rate of cross-strain or cross-species infection of symbionts (Levin, 1983; Hochberg & Holt, 1990). When $\delta > 0$, the virulent strain is the net winner in cross-infection, whereas when $\delta < 0$ the avirulent symbiont tends to prevail. A net flow from one symbiont population to another requires that the net benefactor be superior at either horizontal transmission or within-host interference.

We define the ‘virulence’ of a generic symbiont (call it ‘S’) as the net quantity $\alpha_S - a_S$ and not simply α_S as is often done (see Hochberg, 1998; Ebert, 1998 for recent discussions). Mathematically, therefore, avirulents and virulents are distinguished by the following condition:

$$r_A > r_V. \quad (2)$$

Because the symbiont is dependent upon the host for its survival, the host automatically has a positive effect on the symbiont population. Below, we refer to a symbiont as a ‘mutualist’ when it has a net positive impact on host growth rate such that $r_A > 0$ or $a_A - \alpha_A > 0$. This definition implies, for example, that the net growth rate of a host–symbiont couple may be positive without the interaction being mutualistic, i.e. $r_U + r_A > 0 > r_A$. It may also happen that the mutualism is not demographically viable, or $r_A > 0 > r_U + r_A$. Finally, it is possible that a mutualistic symbiont inflicts a greater mortality rate on the host than does a virulent parasite, that is $\alpha_A > \alpha_V$ but $a_A \gg a_V$.

Habitat productivity

We are interested here in how habitat productivity affects the persistence of different symbiont types. By ‘habitat productivity’ we mean the growth rate of the host population (i.e. the habitat for the symbionts) in the absence of symbionts. Our use of ‘productivity’ should be differentiated from notions of individual host ‘condition’ (e.g. stress), although the two may be interrelated.

In our model, habitat productivity is encapsulated in two parameters $-r_U$ and q – each of which may vary across the host’s geographical range. Productivity increases with r_U and decreases with q , either tendency leading to higher host abundances in the absence of symbionts. Note that the parameter q could be factored out of model eqn 1 without loss of mathematical generality; however, given the importance of its intuitive meaning (limitation of host abundance due to generalist predators or resource competition) we prefer not to make this simplification.

Results

We identify dynamic outcomes of the interaction through a combination of phase-space and invasion analyses. Zero growth clines of hosts harbouring virulent or avirulent symbionts are constructed by setting the left-hand sides of eqns 1a’ and 1b’ equal to zero, and tracing the relationships between A and V . Application of the Routh–Hurwitz conditions to each equilibrium point indicated that all were locally stable to small perturbations in equilibrium densities (May, 1974).

We found that the fate of the system depended on: (i) habitat productivity (parameters r_U and q); (ii) symbiont interference (δ); and (iii) symbiont effect on the host (r_V and r_A).

Consider first what happens when the virulent symbiont is at equilibrium (V'). If the avirulent is introduced at low densities into the system (i.e. appearance of a mutant symbiont, or via immigration from another habitat) then invasion only succeeds if eqn 1b’ is positive, or

$$r_A - r_V > \delta V'. \quad (3a)$$

If the virulent symbiont is so virulent that it cannot persist on its own (i.e. $r_U + r_V < 0$ and $V' = 0$) then invasion of the avirulent is assured (see eqn 2). On the other hand, if $V' = (r_U + r_V)/q > 0$ then the avirulent symbiont is increasingly likely to take hold as it becomes increasingly beneficial to the host (large r_A), and as it dominates the virulent at cross-transmission (i.e. lower δ).

Assuming that the virulent dominates at direct competition (i.e. $\delta > 0$), the intrinsic growth of the avirulent has to be at least $\delta V'$ more than the virulent for the avirulent to invade. The discrepancy in virulences necessary for invasion increases with both the absolute rate of cross-infection and the quality of the environment (as measured by q). Therefore, only if the virulent symbiont

dominates at cross-infection (i.e. $\delta > 0$) and can persist on its own ($V' > 0$) is avirulence impeded from invasion, and this impediment should be most intense in environments of high quality for the host (high r_U and/or low q). In contrast, if the avirulent dominates in direct competition ($\delta < 0$), then the avirulent always invades the system, regardless of environmental quality.

Finally, consider what happens if the interaction occurs in a marginal habitat for the host (i.e. $r_U \approx 0$), or even a sink ($r_U < 0$). Clearly nothing changes if the virulent cannot persist on its own (i.e. eqn 3a is satisfied). However, so that $V' > 0$, the so-called 'virulent' form must actually be beneficial to its host ($r_V > 0$), and in such cases if the avirulent symbiont dominates cross-infection ($\delta < 0$), then lowering habitat productivity impedes the invasibility of the avirulent. On the other hand, if the virulent dominates ($\delta > 0$), then habitat degradation may actually *enhance* the invasion of the avirulent symbiont.

Now take the avirulent symbiont to be alone at equilibrium, or $A' = (r_A + r_U)/q$, and assume the virulent is introduced at low densities ($V_0 \approx 0$). The invasion criterion is similar to eqn 3a:

$$r_A - r_V < \delta A'. \tag{3b}$$

Recall that by definition $r_A > r_V$ and thus it follows that if the avirulent is the better cross-transmitter (i.e. $\delta < 0$), then invasion is impossible. When the virulent is superior ($\delta > 0$), invasion only succeeds if the habitat is of sufficient quality (high r_U and/or low q).

In contrast to the invasion of the avirulent into a virulent equilibrium (eqn 3a), the reverse scenario is affected positively by increasing productivity if the virulent dominates at interference competition, whereas invasion is impeded if the avirulent dominates.

Combining these invasion scenarios allows us to deduce the final system configuration. There are four possibilities.

Contingent competition

It can be shown that at least one of the two inequalities (eqns 3a, 3b) must hold, and therefore that outcomes contingent on initial population densities are not possible in our system (i.e. an unstable two-population equilibrium). Such contingent outcomes could be feasible if the symbionts were to have differing effects on the

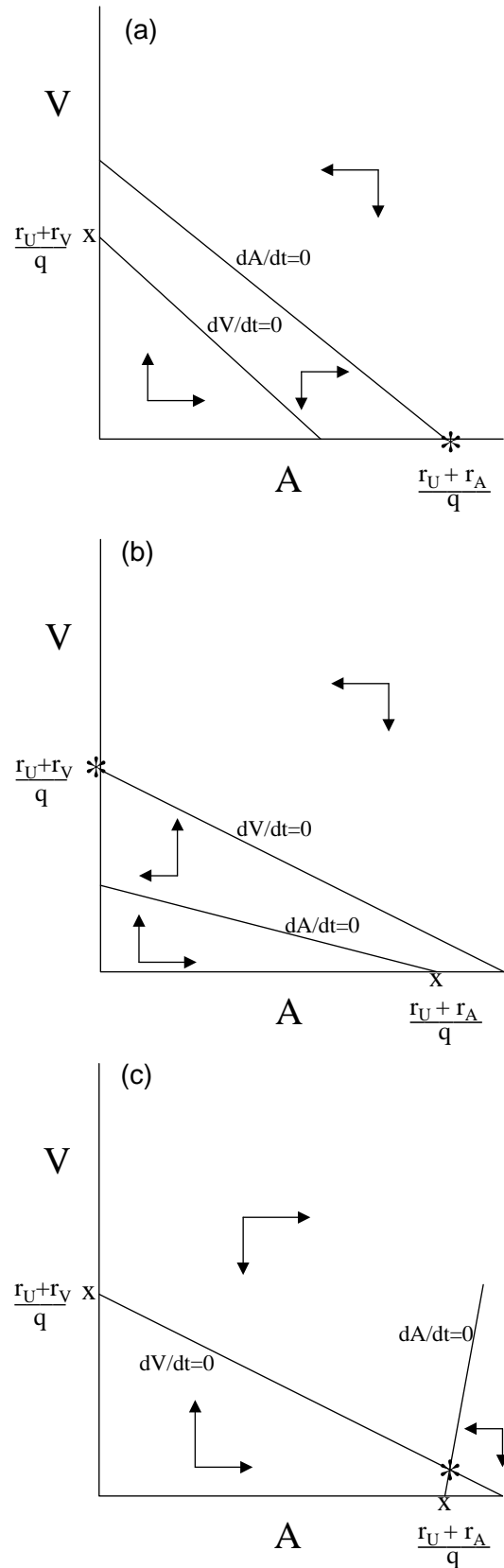


Fig. 1 Typical phase planes of the three qualitatively different outcomes of our model. The phase-space isoclines are formally the same as the Lotka–Volterra competition model when $|\delta| < q$; that is, host–host competition exceeds symbiont–symbiont competition. (a) The avirulent symbiont excludes the more virulent symbiont (see conditions, eqn 4a), (b) the virulent excludes the avirulent (conditions in eqn 4b) and (c) the symbionts coexist (conditions, eqn 5). For any given nonstationary area, the vertical and horizontal vectors indicate the movement directions of virulents and avirulents, respectively.

competitive ability of hosts (i.e. q would be expanded into within- and between-population components for both symbionts) (Hochberg, 1991).

The avirulent excludes the virulent (Fig. 1a)

If eqn 3a holds but eqn 3b does not, or

$$\delta V' < r_A - r_V > \delta A' \quad (4a)$$

then the avirulent symbiont excludes the more virulent one. This automatically holds if the avirulent is superior at cross-infection, or $\delta < 0$. More generally the avirulent wins if $\delta/(r_A - r_V) < 1/A'$. This indicates that avirulence prevails in unproductive environments (low r_U and high q) and as the effects of the two symbionts on the host differ from one another (i.e. as $r_A \gg r_V$). Indeed when the effect of the more virulent symbiont is sufficiently negative, or $r_V < r_A - \delta A'$, the virulent never persists when confronted with the avirulent.

The virulent excludes the avirulent (Fig. 1b)

If eqn 3b holds but eqn 3a does not, or

$$\delta V' > r_A - r_V < \delta A' \quad (4b)$$

then the virulent excludes the avirulent. This clearly cannot be true if the avirulent is the better cross-infecter ($\delta < 0$). Because the left-hand inequality ensures the satisfaction of the right-hand one, the virulent wins when $\delta/(r_A - r_V) > 1/V'$. Thus, high productivity favours this more virulent symbiont, as does similarity to the more avirulent symbiont in its effect on the host ($r_A \rightarrow r_V$). Note that although the virulent symbiont's effect on the host can be negative, it should not be so negative that $r_V < (qr_A - \delta r_U)/(q + \delta)$, otherwise it cannot exclude the avirulent. In contrast, if the environment is so marginal such that $r_A/\delta > r_U/q$, then a relatively virulent symbiont can only oust its competitor if it itself is beneficial to the host, or $r_V > 0$.

The symbionts coexist (Fig. 1c)

The two symbionts coexist if both eqns 3a and 3b hold, or

$$\delta V' < r_A - r_V < \delta A'. \quad (5)$$

Clearly, $\delta > 0$ for both inequalities to hold (unless $r_U + r_V < 0$ and $V' = 0$, and then only the right-hand inequality need be true). Thus, for coexistence, the advantage the avirulent has at intrinsic growth (i.e. indirect competition) must be offset sufficiently by the superiority of the virulent at direct competition (Hochberg & Holt, 1990). Superiority at each of these two forms of competition (exploitative = intrinsic growth, interference = cross-transmission) must be intermediate in magnitude for coexistence. Moreover, coexistence is generally promoted at intermediate levels of the habitat productivity parameters, q and r_U (e.g. Levin & Pimentel, 1981; Hochberg & Holt, 1990). It is noteworthy that coexistence between two moderately beneficial mutualists

or two moderately virulent parasites are both possible outcomes.

The two population equilibrium can easily be found from eqn 1':

$$V^* = (q(r_V - r_A) + \delta(r_A + r_U))/\delta^2, \quad (6a)$$

$$A^* = (q(r_A - r_V) - \delta(r_V + r_U))/\delta^2, \quad (6b)$$

$$N^* = V^* + A^* = (r_A - r_V)\delta. \quad (6c)$$

Note that conditions (eqn 5) can usefully be re-written as

$$V' < V^* + A^* < A', \quad (7)$$

which means that coexistence results in total host populations levels which are intermediate to either alternative single population equilibrium.

Figure 2 illustrates how densities of each symbiont type vary over a simple gradient in habitat productivity (r_U) when the virulent has an advantage at interference ($\delta > 0$) (recall, when the avirulent dominates at interference the virulent cannot persist regardless of productivity).

In general, the virulent tends to occupy habitats of higher productivity than the avirulent (Fig. 2). Proceeding from high- to low-productivity habitats, we observe that the virulent persists alone, followed by coexistence at intermediate productivities, and finally avirulents alone at low productivities. Because sufficiently low productivities eventually drive densities to zero, somewhat counter-intuitively, virulent symbiotic associations are able to attain higher population densities than avirulent ones.

The level of symbiont interference can have important effects on density patterns over different habitat productivities (Fig. 2a). As expected, increasing interference superiority for the virulent increases the range of habitat types it occupies and decreases the range occupied by avirulents. Moreover, the range of habitat productivities yielding coexisting systems is shifted to ever decreasing levels as virulent interference superiority increases (see also inequalities, eqn 5). Assuming that the avirulent can persist on its own with the host, when $\delta > (r_A - r_U)/A'$ the virulent is an obligate parasite of the avirulent, because the former can persist in environments that would not otherwise be possible if the avirulent were absent. This condition tends to be satisfied in habitats of sufficiently (but not too) low productivity.

As illustrated in Fig. 2(b),(c), symbiont effect has important consequences for productivity patterns. Whereas increasing the negative effects of the more virulent symbiont decreases the range of habitat productivities where it persists, such increases actually *increase* the range of habitat productivities yielding coexisting systems (Fig. 2b). This is because the competitive effect of the virulent on the avirulent (mediated through qV) decreases with lower r_V ; lower direct

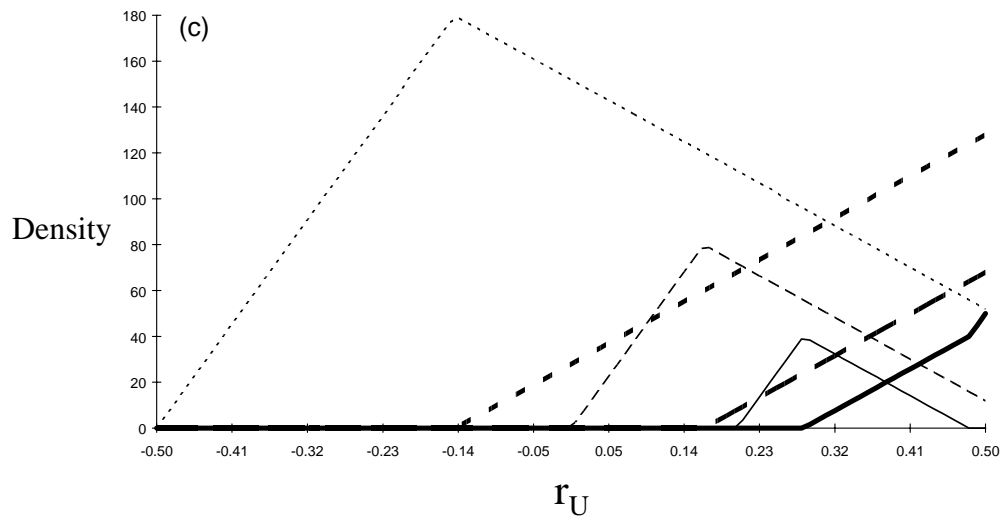
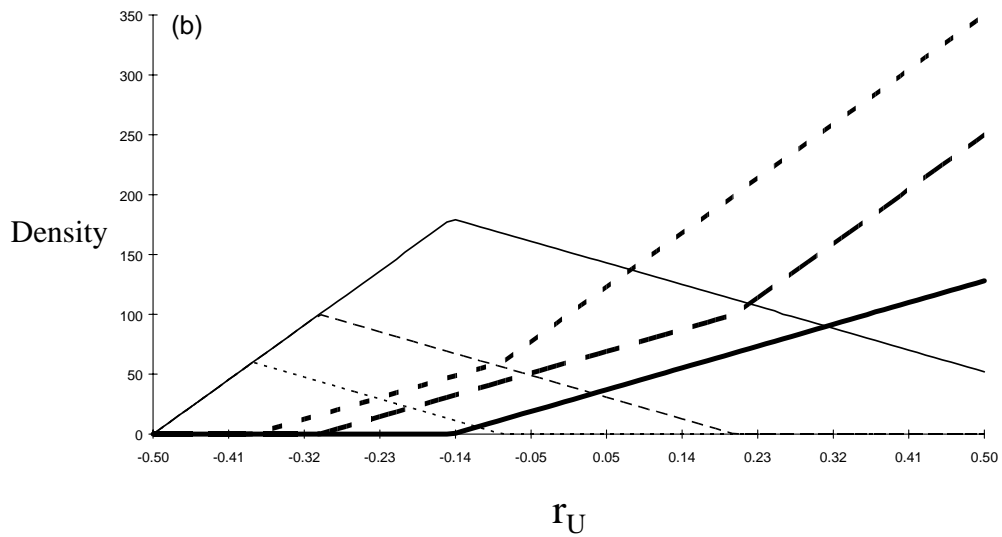
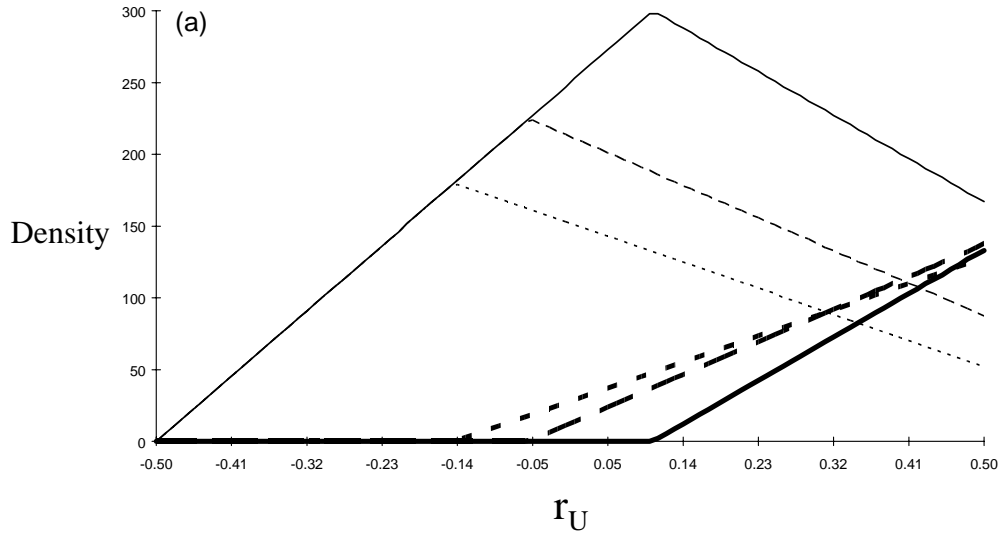


Fig. 2 Densities of virulent and avirulent symbionts as a function of habitat productivity (r_U) and (a) the competitive advantage of virulent symbionts ($\delta = 0.003, 0.004, 0.005$ for solid, dashed and dotted lines, respectively), or (b) the effect of virulent symbionts ($r_V = -0.5, 0, 0.2$ for solid, dashed and dotted lines, respectively), or (c) the effect of avirulent symbionts ($r_A = -0.2, 0, 0.5$ for solid, dashed and dotted lines, respectively). Thickened lines refer to virulent densities whereas fine lines are avirulent densities. Baseline parameter values: $\delta = 0.005, r_V = -0.2, r_A = 0.5, q = 0.002$. Densities of single symbiont systems are shown only when symbiont coexistence is not feasible.

competition means a greater propensity for coexistence. As expected, increasing the positive effect of the avirulent symbiont tends to increase the ranges of productivities yielding both host persistence and symbiont coexistence (Fig. 2c).

Discussion

Although there are documented examples of interactions that vary in outcome across environments, we have not been able to find any studies that have specifically examined how natural selection may have shaped obligate symbioses across productivity gradients. Various interactions within natural communities have been demonstrated to have the potential to vary in outcome, ranging from antagonism to commensalism or mutualism (Palumbi, 1985). Differences may occur among individuals within populations, depending upon the age, size, physiological state, or genetic composition of individuals (Thompson, 1988; Douglas, 1998). In addition, the mean outcome may vary from mutualism to antagonism among populations, depending upon differences in the physical environment or the community context in which the interaction takes place. These environment-dependent 'interaction norms' (*sensu* Thompson, 1988) are the species-level equivalent of reaction norms in evolutionary genetics and are the kinds of ecological situations envisioned in our model. Examples include endophytic fungal relationship with plants (Saikkonen *et al.*, 1998), mycorrhizal relationships (Francis & Read, 1995; Stetälä *et al.*, 1997), pollinating floral parasites (Thompson & Pellmyr, 1992), ant-fed plants (Janzen, 1979; Thompson, 1981), and other interactions in which the effects of symbionts on host survival and reproduction is environment-dependent. Reviews of mutualism have increasingly emphasized the conditional outcomes of many mutualistic relationships (Cushman & Beattie, 1991; Bronstein, 1994; Douglas, 1998).

We found that a relatively avirulent symbiont always displaces its more virulent competitor unless there exists a trade-off, whereby the virulent symbiont is either more apt to be horizontally transmitted to hosts and/or is better at ousting the competing avirulent strain within hosts. Our results point to habitat productivity for the symbiont

(parameters q and r_U) as an important mediator of competition between avirulent and virulent symbionts. In particular, relatively avirulent strains tend to dominate virulent ones in less productive environments; mutualistic symbionts should tend to be found in marginal and sink environments of the host. Coexistence between mutualists and obligate hyperparasites (or 'cheaters') is possible under certain habitat conditions. We know of no demonstration of the patterns we predict, probably because biologists have not been alerted to the potential for their occurrence.

Marginal and sink habitats selecting for avirulence or even beneficial effects could be associated with low host reproduction rates, high host mortality rates, low resource densities resulting in high levels of competition between hosts, or high densities of generalist predation on hosts. Sink habitats are only one component of the geographical structure of species, but differences in productivity among communities are a common part of the biology of all species – it is possible that fine-grained productivity mosaics result in subtle spatial structures in parasite virulence and mutualistic interactions. Our results support the general conclusion of Hochberg & van Baalen (1998) that productivity gradients can create selection mosaics, favouring different outcomes for interspecific interactions in different habitats.

The patterns in symbiont effect we observe are created by habitat to habitat differences in resource exploitation (i.e. the functional response or the 'force of infection') by symbionts, and interference between symbionts. Symbionts not exhibiting functional or numerical responses to their hosts (Loreau & de Mazencourt, 1999), and hosts not subject to intraspecific competition may not produce the types of patterns we observe. Moreover, the patterns could be altered if we were to incorporate constraints on the ability of the symbiont to contribute to host population growth, symbiont evolution that influences density dependence in host reproduction rates (Loreau & de Mazencourt, 1999; Hochberg & van Baalen, manuscript in preparation), or norms of reaction in parameters which tend to favour virulent symbionts in more marginal environments for the host. An example of the latter could transpire if individual hosts tended to be more stressed in marginal as opposed to productive environments, and if the horizontal and cross-transmission of more virulent symbionts were differentially enhanced by individual host stress.

Our findings have direct parallels with theoretical explanations of the foundations for the evolution of parasite virulence. Theory predicts that as the expectation of within-host parasite diversity increases, so too does selection favour the most virulent parasite types (e.g. Frank, 1994a, 1995; van Baalen & Sabelis, 1995). In our model we assumed a superinfection process (*sensu* Mosquera & Adler, 1998) such that one symbiont completely dominates the other in coinfecting host individuals when $\delta \neq 0$. Specifically, all else being equal,

the more virulent symbiont can only persist if it tends to out-compete the less virulent symbiont in coinfecting hosts (i.e. $\delta > 0$). This interference condition, although very simple, is implicitly similar to the effect of multiple infections on the evolution of virulence (Mosquera & Adler, 1998).

Limitations and future directions

Our analysis examined the confrontation of two symbiont types. This could reflect a single evolutionary step if the symbionts are variants of a single species, or competition between two similar symbiont species (see also Bever, 1999). Hence, our results constitute a precondition for geographically structured coevolution, but do not explore the ultimate coevolutionary process that ensues. Geographical differences in host traits that permit formation of symbiotic mutualisms or resist attack by pathogens are now known empirically for a wide range of taxa (e.g. Burdon & Thrall, 1999; Parker, 1999). Coevolution is likely to continually reshape the evolutionary optima of species (e.g. evolutionary stable strategies) as well as the pattern of evolutionary dynamics (i.e. ecological and evolutionary transients) across habitats of differing quality for the host.

We have not explicitly considered the extent to which the migration of genes and individuals affects the geographical diversity of symbiotic interactions (Nuismer *et al.*, 1999). Low rates of gene flow tend to increase geographical diversity, whereas high rates destroy it (Frank, 1991; Hochberg & van Baalen, 1998). If migration tends to homogenize symbiont genotypes across geographical landscapes, then we would expect overall selection across a host species' range for increased virulence (see also Hochberg & van Baalen, 1998) if (i) migrating virulent types in productive environments numerically dominate avirulents in nonproductive environments (i.e. a swamping effect), and (ii) there is increased within-host competition between strains or species (as would be expected under panmixis). A future challenge in evolutionary biology will be to understand how the density and configuration of habitat suitabilities over geographical ranges and the flow of genes and individuals over this template combine to mould biogeographical patterns in adaptation and coadaptation.

How patterns in virulence and mutualism are shaped will also depend upon the evolutionary genetics of the interacting species. Our analysis does not specify the genotypic and phenotypic architectures of the populations. 'Architecture' includes (i) the recognition systems that host and symbiont use to distinguish self from nonself (e.g. Frank, 1994b; Burdon, 1998), (ii) the nature of their reactions when nonself is recognized (e.g. Harvell, 1990; Karban & Baldwin, 1998), and (iii) any costs, constraints or trade-offs which may intervene to limit the evolution of recognition and reaction (e.g. Mitchell-Olds & Bradley, 1996). It is our view that the most salient studies for the

future will integrate all three of these, since *a priori* all three are likely to be present in some form in endosymbiosis.

Speculation

Parasites have been often suggested as important components in the evolution of sex in their hosts (e.g. see Ebert & Hamilton, 1996 for recent review). According to our findings, this drive towards asexuality should tend to occur in marginal or sink habitats for the host. Intriguingly, this expectation matches the empirical pattern found in many plant and animal species (e.g. Ghiselin, 1974; Glesener & Tilman, 1978; Bell, 1982; Ladle, 1992). We suggest that our sink-cradle model provides one environmental axis on which natural selection may act to produce geographical patterns in the evolution of sex. To the degree that obligate symbionts affect the evolution of sexual reproduction, populations of a given species should tend to be sexual in productive environments where antagonistic natural enemies reign, whereas this same species should tend toward asexual reproduction in more marginal environments where avirulent or mutualistic symbionts dominate (see also Law & Lewis, 1983). Other aspects of symbioses, however, could complicate such simple patterns. For instance, if productivity is heightened by a mutualist, such that a parasite is now able to persist locally, then sexual reproduction in the mutualist may once again be favoured. In addition, in some long-lived taxa with clonal reproduction, the boundaries between mutualism, parasitism, and sex become clouded. For example, some endophytic fungi prevent sexual reproduction in their plant hosts but increase long-term survival and vegetative propagation of the host genotypes they attack. Clay & Klover (1996) have called this the 'Red Queen Captured Hypothesis'; the existence of this phenomenon clouds the question of what constitutes antagonism and mutualism in obligate symbioses.

Conclusion

Previous theoretical studies have highlighted the importance of low host survival or viability to the evolution of mutualistic symbioses (e.g. Roughgarden, 1975; Law & Dieckmann, 1998). The present study extends these former ones by emphasizing how species demography may contribute importantly to selection mosaics found in interspecific interactions. We suggest that the nastiest parasites should be found in the most productive environments for the host. Moreover, our results indicate that marginal habitats may be crucially important geographical cradles for the origin of new symbiotic mutualisms and the maintenance of genetic diversity in geographically diverse obligate symbioses. Accordingly, our results highlight the importance for conservation of habitats that are often dismissed as unproductive within species' geographical range.

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