VOL. 154, NO. 2 THE AMERICAN NATURALIST AUGUST 1999

The Evolutionary Advantages of Dying Young: Epidemiological Implications of Longevity in Metapopulations

J. W. Kirchner[1,*] and B. A. Roy[2,†]

1. Department of Geology and Geophysics, University of California, Berkeley, California 94720-4767;
2. Geobotanical Institute, Swiss Federal Institute of Technology (ETH), Zürichbergstrasse 38, CH-8044, Zürich, Switzerland

Submitted October 13, 1998; Accepted March 25, 1999

ABSTRACT: Here we show that pathogen-mediated selection can influence the evolution of host longevity. Greater longevity can impair the fitness of host organisms subject to pathogen attack, by reducing the mortality rate of infected hosts and thus creating a larger and more persistent reservoir of disease, from which infection can spread to the healthy population. Where longer-lived and shorter-lived hosts can infect one another (and thus all share the same risk of infection), selection will favor longer-lived individuals, to the detriment of the host population as a whole. But in metapopulations, selection can favor shorter-lived hosts that are otherwise identical to their longer-lived competitors, because the populations in which they occur will have lower incidence of disease. Under some conditions, shorter-lived hosts can even invade metapopulations of longer-lived hosts, displacing them and driving them to extinction. Our results support three general propositions. First, an organism's life-history traits, and not just its resistance genes, can affect its risk of pathogen attack. Second, pathogen-mediated selection may therefore influence the evolution of host life-history traits that are unrelated to resistance, per se. Third, the magnitude—and even the direction—of selection on host longevity can depend on the structure of the host population.

Keywords: life span, senescence, life-history evolution, founder effects, patchy environments, group selection.

Host life-history traits can affect host vulnerability to pathogen attack. Thus, one can expect disease to act as a selective force shaping host life-history traits (Sheldon and Verhulst 1996; Shykoff 1997; Clay and van der Putten, in press). Host life-history traits can also affect the fitness of neighboring individuals by altering the local incidence of disease and thus altering the risk of infection for nearby hosts. Here we examine whether this mechanism can create population-level feedbacks that affect the evolution of host life-history traits. We show that under certain conditions, population-level feedbacks can shape the evolution of host longevity, such that early senescence is favored by selection.

Senescence limits an organism's reproductive potential and thus presents an evolutionary paradox (Williams 1957; Keller and Genoud 1997). Senescence has been viewed as an unstoppable process of cellular degeneration (Finch 1990; Rose 1991), as an evolutionary by-product of maximizing early reproductive success (Williams 1957; Hamilton 1966; Stearns 1992), or as the result of weaker selection against any mutations that become harmful after individuals have already reproduced, passing their genetic liabilities on to their offspring (Haldane 1941; Medawar 1946; Partridge and Barton 1993). But could earlier senescence and death actually be advantageous, and thus be directly favored by natural selection? Here, using simple models of host-pathogen interactions, we show that under certain conditions intrinsically short life spans can confer an evolutionary advantage by suppressing the spread of disease.

Senescence is paradoxical because under most circumstances increased longevity implies increased fitness. Except in monocarpic species, longer-lived individuals have longer reproductive life spans and therefore should have a selective advantage over shorter-lived individuals. Thus, genes conferring increased longevity will normally become fixed, over time, in populations into which they are introduced.

In some circumstances, however, the costs of increased longevity can outweigh its benefits. For example, if increased longevity is associated with a more-than-proportional decrease in fertility, or decreased offspring survival, it will not be favored by selection (Stearns 1992). In this article, we show that the longevity of host organisms indirectly affects their fitness by altering the incidence of diseases afflicting the host population. We explore whether

* E-mail: kirchner@geomorph.berkeley.edu.

† To whom correspondence should be addressed; e-mail: roy@geobot.umnw.ethz.ch.

Am. Nat. 1999. Vol. 154, pp. 140–159.

longevity can affect disease incidence sufficiently for selection to favor shorter, rather than longer, host life spans.

Host Longevity and Disease Incidence

We begin by describing how host life span affects the incidence of disease, and thus host fitness. By host life span (or host longevity), we mean the intrinsic life span of the host organism—that is, its genetically determined life span in the absence of disease and other stress factors. This distinction is important because disease may itself affect how long an organism lives, but this extrinsic control on longevity is not our primary concern. Thus, except where we explicitly state otherwise, we use the terms longevity and life span to refer to hosts' intrinsic life spans, rather than the length of time that they happen to live.

Factors controlling the incidence of disease can be important for host evolution when the fitness consequences of infection are substantial. For example, sterilizing infections have obvious consequences for host fitness, and are common in both animals (Baudoin 1975) and plants (Clay 1991; Roy 1993; Roy and Bierzychudek 1993). In this section, we primarily explore how host longevity affects the incidence of a completely sterilizing disease and thus affects host fitness. We focus on sterilizing infections in order to simplify the mathematical analysis; in the appendix, we show how this approach can be broadened to include nonsterilizing infections. Qualitatively similar results are obtained for both sterilizing and nonsterilizing infections, as long as the fitness consequences of infection (through reductions in fertility or increases in mortality) are substantial.

Figure 1 shows that, for organisms subject to pathogen attack, intrinsic life span affects fitness by two different pathways. One pathway is shown by links (1) and (2): all else equal, greater longevity reduces the mortality rate of healthy hosts, leading to a larger host population. But as links (3)–(6) in figure 1 show, greater longevity can also impair host fitness by reducing the mortality rate of infected hosts, thus creating a larger and more persistent reservoir of infected hosts, from which infection can spread to the healthy population. By this indirect pathway, longer life spans can diminish the healthy (and thus reproductively viable) host population. If this second pathway is more influential than the first, longer life spans may diminish host fitness.

At least in theory, then, shorter host life spans may confer fitness advantages by suppressing the spread of infection to the uninfected (and reproductively viable) host population. But is this fitness advantage quantitatively significant? Consider a simple model of a single, genetically uniform host population infected by a single pathogen, which we have borrowed, with modifications, from May and Anderson (1983). The model's structure and its key expressions are shown in figure 1. We denote the uninfected and infected host populations by X and Y, respectively, each expressed as fractions of the carrying capacity. The pathogens cannot survive without hosts, so they need not be modeled explicitly; instead, their dynamics are represented by the infected host population. We assume that the pathogen is transmitted only horizontally, so that all hosts are born uninfected. We further assume that reproduction, infection, and death are controlled by simple Lotka-Volterra expressions. Uninfected hosts reproduce at a rate $a(1 - N)X$, where a is the potential per capita reproduction rate in the absence of carrying capacity constraints, $1 - N = 1 - (X + Y)$ is the fraction of carrying capacity that is unoccupied (and thus available for new individuals to become established), and X is the total uninfected (and thus reproductively viable) population. Uninfected hosts die at a rate X/τ, where τ is the mean life

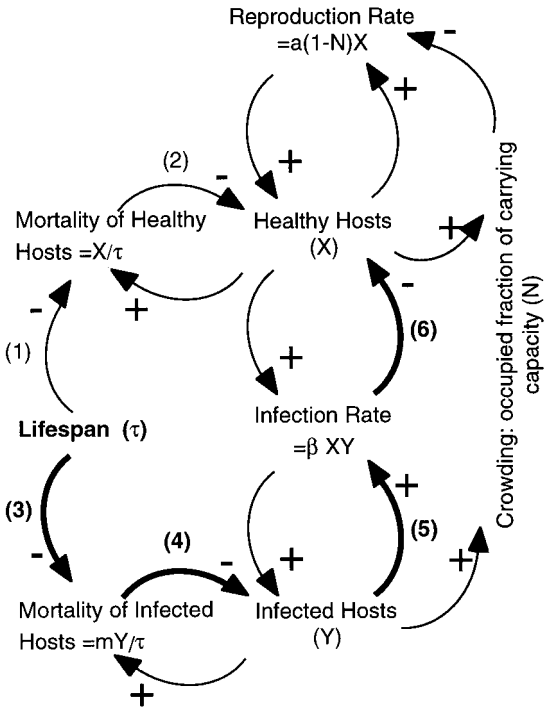

Figure 1: Causal loop diagram of the simple host-pathogen model given in equations (1) and (2). Arrows indicate causal linkages, not material flows. Signs (*plus* or *minus*) of causal arrows indicate whether cause and effect move in the same (*plus*) or opposite (*minus*) directions. Causal arrows (*1*) and (*2*) show how greater host longevity may increase the population of healthy hosts by reducing host mortality. Causal arrows (*3*)–(*6*), shown in bold, show how greater host longevity may decrease the population of healthy hosts, by reducing the mortality of infected individuals and thus increasing the rate at which healthy hosts are lost to infection.

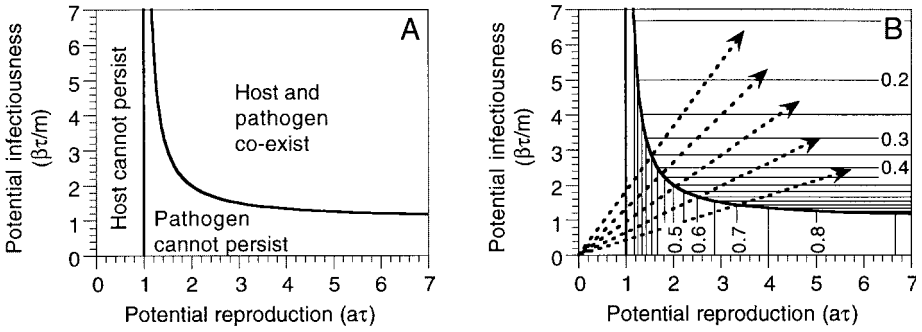

Figure 2: Equilibrium behavior of the simple host-pathogen model given in equations (1)–(5). Domains of feasibility (*A*) and equilibrium host population (*B*) are shown as functions of potential host reproduction ($a\tau$) and potential pathogen infectiousness ($\beta\tau/m$). Panel *B* shows the same feasibility domains as panel *A*, with contours (*thin lines*) showing the equilibrium population of healthy hosts. These contours run vertically through the domain where pathogens cannot persist (eq. [4]) and pivot to run horizontally through the domain where hosts and pathogens can coexist (eq. [5]). The population of healthy hosts increases from top to bottom in the domain where hosts and pathogens can coexist, and increases from left to right in the domain where only hosts can persist. Arrows show paths of increasing host life span (τ), for constant values of a, β, and m. Note that as host life span increases, the equilibrium population of healthy hosts increases if infection is absent (below the curved line), but decreases if infection is present (above the curved line). Thus, for all combinations of a, β, and m (i.e., for all possible rays outward from the origin), the population of healthy hosts is maximized at the boundary along which infection vanishes.

span in the absence of infection. Infected hosts die at a rate mY/τ, where m is the ratio ($m \geq 1$) by which infection shortens life span (and thus accelerates mortality). Hosts become infected at a rate βXY, where β reflects pathogen infectiousness and host susceptibility, X is the fraction of the carrying capacity occupied by susceptible uninfected hosts, and Y is the population of infected (and thus infectious) hosts. The uninfected host population will change at a rate determined by the balance between the rates of reproduction, infection, and death:

$$\frac{dX}{dt} = a(1 - N)X - \beta XY - \frac{X}{\tau}. \tag{1}$$

Similarly, the infected host population will change at a rate determined by the balance between infection and mortality:

$$\frac{dY}{dt} = \beta XY - \frac{mY}{\tau}. \tag{2}$$

Leaving aside for the moment the time-dependent evolution of equations (1) and (2), let us consider their equilibrium behavior ($dX/dt = 0$, $dY/dt = 0$) as an indicator of the model system's general tendencies. The equilibrium solutions are governed by two dimensionless parameters, potential host reproduction ($a\tau$) and potential pathogen infectiousness ($\beta\tau/m$). Potential host reproduction expresses the number of offspring each healthy host could produce in its lifetime in the absence of carrying capacity constraints. Potential pathogen infectiousness expresses the number of healthy hosts each infected host could hypothetically infect before dying, if the carrying capacity were filled with susceptible individuals. It can easily be shown that equations (1) and (2) yield three classes of stable equilibria, depending on the life span τ, as shown in figure 2. If the lifetime reproductive potential of uninfected hosts, $a\tau$, is <1, then hosts cannot reproduce rapidly enough to offset their own mortality, and any host population will eventually decay away to the trivial equilibrium of:

$$X = 0 \quad \text{and} \quad Y = 0 \quad \text{if} \quad 1 \leq \frac{1}{a\tau}. \tag{3}$$

This situation can arise if either the per capita reproduction rate, a, is too low or host longevity, τ, is too short to allow hosts to replace themselves before dying. These conditions correspond to the domain along the left edge of figure 2. Alternatively, host longevity may be sufficient to maintain a population of uninfected hosts, but at a density that is too low ($X < m/\beta\tau$) to permit infection to sustain itself (Anderson and May 1991). If each infected host cannot spread infection to at least one other host before dying (or, equivalently, if the potential pathogen infectiousness is less than $1/[1 - 1/a\tau]$), only uninfected hosts will exist at equilibrium:

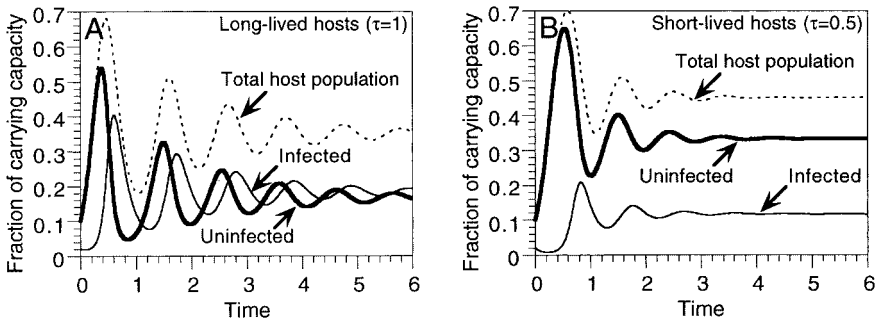

Figure 3: Effect of host life span on host-pathogen population dynamics. Time-dependent behavior of the host-pathogen system described by equations (1) and (2), for a long-lived host ($\tau = 1$; *A*) and a shorter-lived host ($\tau = 0.5$; *B*); initial conditions and all other parameter values are identical in both panels ($a = 10$, $\beta = 30$, $m = 5$). When hosts are shorter lived, infected individuals die more rapidly and are thus less able to spread infection to new hosts. As a result, the infected fraction of the host population is smaller, and both the uninfected and the total host populations are larger.

$$\begin{aligned} X &= 1 - \frac{1}{a\tau} \\ \text{and} \quad Y &= 0 \\ \text{if} \quad \frac{1}{a\tau} &\le 1 \le \frac{1}{a\tau} + \frac{1}{\beta\tau/m}. \end{aligned} \tag{4}$$

These conditions correspond to the domain along the bottom edge of figure 2. In this case, increased longevity implies a larger equilibrium population of uninfected hosts, as one would intuitively expect. If the uninfected hosts are sufficiently numerous that infection can spread rapidly enough to keep pace with the mortality of infected individuals, both infected and uninfected hosts will coexist at equilibrium:

$$\begin{aligned} X &= \frac{1}{\beta\tau/m} \\ \text{and} \quad Y &= \frac{a}{a+\beta}\left(1 - \frac{1}{\beta\tau/m} - \frac{1}{a\tau}\right) \\ \text{if} \quad \frac{1}{a\tau} &+ \frac{1}{\beta\tau/m} \le 1. \end{aligned} \tag{5}$$

Note that in this case, greater longevity reduces the equilibrium population of uninfected hosts and increases the equilibrium population of infected hosts (fig. 2*B*). This counterintuitive result arises because greater host longevity prolongs the survival of infected hosts and thus increases the infected host population, accelerating the spread of infection to uninfected hosts. Increased host longevity slows the loss of uninfected hosts caused by mortality, but not as much as it accelerates their loss because of infection. Thus, increased host longevity diminishes host fitness.

This result holds for all parameter values (a, β, τ, and m) that permit infected hosts to persist at equilibrium. As figure 2 shows, increasing host longevity diminishes the healthy (and thus reproductively viable) host population throughout the entire domain where hosts and pathogens coexist. It has been clear for some time that pathogens can impair their own fitness if they are too quick to kill the hosts on which they live (May and Anderson 1983). The analysis above demonstrates the counterpart of this principle from the host's perspective: excessive host longevity benefits pathogens and thus diminishes host fitness.

Figure 3 illustrates how differences in host life span affect the incidence of infection, and thus the uninfected host population, for the model in equations (1) and (2) under one particular set of parameter values. One can see that in this case (as in every case for which hosts and pathogens can coexist), shorter host life spans help to suppress infection and thus benefit the host population. Shorter life spans are also beneficial to host populations subject to nonsterilizing infections, depending on the degree to which disease accelerates mortality (see the appendix).

From equations (4) and (5), one can readily determine the optimal life span, taken here to mean the life span that supports the largest uninfected (and thus reproductively viable) host population. Equation (4) shows that in a self-sustaining host population, the equilibrium population of uninfected hosts (X) increases with increasing longevity, up to the limit of $\tau = (1/a) + (m/\beta)$. Equation (5) shows that for all longer life spans, the equilibrium uninfected population decreases systematically. Thus, for any combination of a, β, and m, the optimal longevity is $\tau = (1/a) + (m/\beta)$, which is the longest life span that is still short enough to prevent infection from being self-sus-

taining. If host longevity were at this optimum, disease would be rare. Therefore, the ubiquity of disease in natural populations suggests that host longevity is typically longer than would be optimal for controlling infection. Competitive mechanisms can readily lengthen host longevity beyond this optimum, as we explain in the next section.

Evolution of Longevity in Populations

The results shown above suggest that if longevity can be controlled, shorter life spans may benefit host organisms. Should we therefore expect natural selection to favor life spans near the optimum shown in figure 2? It is easy to show that this will not generally be the case. Although reducing the average longevity of a host population would lessen the burden of disease borne by the population as a whole, these population-level benefits are shared by the long-lived and short-lived individuals alike. Thus, within a population, long-lived individuals will have a reproductive advantage over their short-lived neighbors, and selection will favor increased longevity, to the detriment of the host population as a whole.

This behavior can be illustrated with a simple invasion analysis. Consider a single host population consisting of two strains, a short-lived strain with longevity τ_S and a longer-lived strain with longevity $\tau_L > \tau_S$. We will assume that both strains share the same pathogens and that each host strain can spread infection to the other. We will also assume, for the sake of simplicity, that the host species is haploid and that longevity is controlled by a single locus (diploid genetics would complicate the analysis but would alter the results only in minor detail [May and Anderson 1983]). Under these assumptions, the simple model given in equations (1) and (2) can be extended to a two-strain host population as follows:

$$\frac{dX_L}{dt} = a(1 - N)X_L - \beta X_L(Y_L + Y_S) - \frac{X_L}{\tau_L}, \quad (6)$$

$$\frac{dX_S}{dt} = a(1 - N)X_S - \beta X_S(Y_L + Y_S) - \frac{X_S}{\tau_S}, \quad (7)$$

$$\frac{dY_L}{dt} = \beta X_L(Y_L + Y_S) - \frac{mY_L}{\tau_L}, \quad (8)$$

$$\frac{dY_S}{dt} = \beta X_S(Y_L + Y_S) - \frac{mY_S}{\tau_S}, \quad (9)$$

where the subscripts L and S refer to the long-lived and short-lived host strains, respectively; X and Y are the uninfected and infected host populations (expressed as fractions of the carrying capacity), respectively; a is the per capita rate of reproduction in the absence of carrying capacity constraints; $1 - N = 1 - (X_L + Y_L + X_S + Y_S)$ is the fraction of carrying capacity that is unoccupied (and thus available for new individuals to become established); and β controls the rate of pathogen transmission from infected to uninfected hosts. It is clear from inspection that if $\tau_L \neq \tau_S$, there is no equilibrium solution to equations (6)–(9) unless one host strain becomes extinct. Because the two strains differ only in longevity and are otherwise identical (and share the same environment, including the same rates of pathogen attack), competitive exclusion will eliminate the shorter-lived strain. This competitive exclusion is illustrated in figure 4, which shows the invasion of a short-lived host population by a strain that lives twice as long.

As figure 4 shows, a longer-lived host strain can rapidly grow to dominate the host population in just a few generations. The longer-lived strain becomes dominant even though this entails an overall decrease in the uninfected population (fig. 4*A*), an overall increase in the infected population (fig. 4*B*), a decrease in the total host population (fig. 4*C*), and an overall doubling in the incidence of disease (fig. 4*D*). By any of these measures, the invasion of the long-lived host strain leaves the host population worse off.

Given the large contrast in longevity between the two host strains in figure 4 ($\tau_L = 2\tau_S$), it is perhaps surprising that it takes roughly 10 generations (or 20 generations of the short-lived host) for the longer-lived host strain to dominate the population. This occurs because, although the long-lived strain's longevity is twice that of the short-lived strain, the long-lived strain's reproductive life span is only about 20% greater. Hosts are lost from the reproductively viable (i.e., uninfected) population by both mortality and infection; as a result, the average reproductive life span is the average time before a healthy host either dies or becomes infected. This reproductive life span is not the intrinsic life span τ_i (where i is a placeholder for either the long-lived or short-lived strain) but is instead $\tau_i/[1 + \beta\tau_i(Y_L + Y_S)]$. In the simulation shown in figure 4, healthy hosts are more rapidly lost to infection than to mortality (i.e., $\beta\tau_i[Y_L + Y_S] \gg 1$), so extending host longevity does not proportionally extend the reproductive life span, nor does it proportionally increase fitness. Putting the same point somewhat differently, although both hosts can infect one another, and thus both are subject to the same rate of pathogen attack, the longer-lived hosts have a higher incidence of infection because they live longer and therefore have more chances to become infected. The higher incidence of infection among the longer-lived hosts eliminates part of the fitness advantage that they would otherwise enjoy.

So far our analysis has demonstrated two main points. First, in genetically uniform host populations subject to

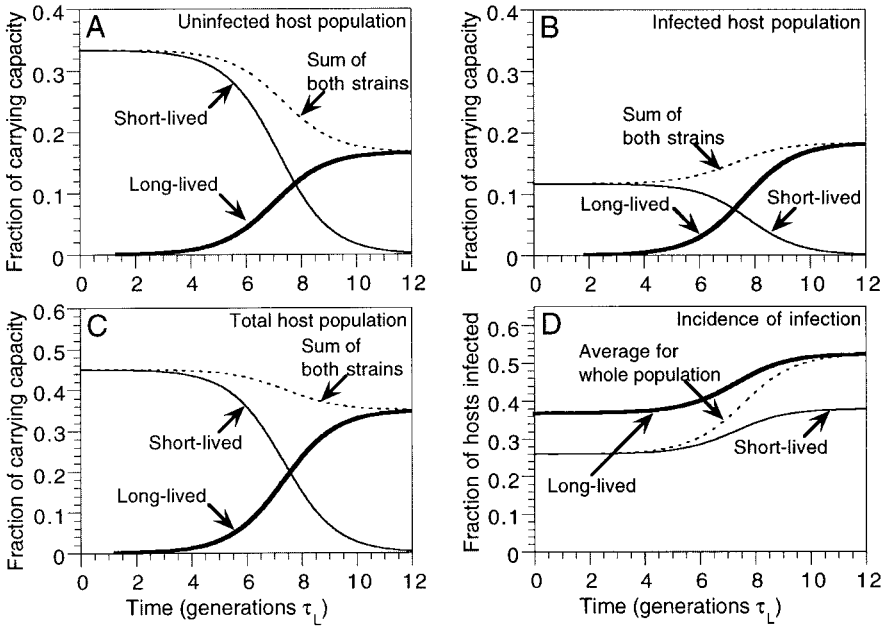

Figure 4: Invasion analysis showing competitive exclusion (and eventual extinction) of a short-lived host strain by a longer-lived host strain, where infected hosts of either strain can spread infection to the other. Model is equations (6)–(9), with parameter values the same as in figure 3 ($a = 10$, $\beta = 30$, $m = 5$, $\tau_S = 0.5$, and $\tau_L = 1$); short-lived hosts (X_S and Y_S) are initialized in equilibrium at time = 0, and long-lived hosts (X_L and Y_L) are initially very small. Because both host strains can infect each other, they share the same per capita rate of infection, $\beta(Y_L + Y_S)$, and the disease-suppression benefits of the short-lived hosts (or, conversely, the disease-enhancement costs of the longer-lived hosts) affect both host strains equally. Thus, the longer-lived strain has a reproductive advantage over the shorter-lived strain and rapidly displaces it, even though this entails an overall decrease in the uninfected population (*A*), an overall increase in the infected population (*B*), a net decrease in the total population (*C*), and a doubling in the incidence of infection (*D*).

pathogen attack, long life spans can be disadvantageous because they create a more persistent reservoir of disease from which new hosts can become infected. However, when short-lived and long-lived individuals share each other's pathogens, the disease-suppression benefits conferred by the short-lived strain are shared by both strains. Because the two host strains share the same level of pathogen attack, long-lived individuals will have a competitive advantage over short-lived individuals and will be favored by selection, to the detriment of the host population as a whole. This illustrates the widely recognized principle that evolution will generally favor traits that confer individual selective advantage, regardless of whether those traits are advantageous at some higher level (group, population, species, etc.; Williams 1966; Maynard-Smith 1976).

Nonetheless, are there circumstances in which selection by disease can favor shorter life spans? Our first analysis presented above (fig. 2; eqq. [3]–[5]) implies that selection can favor decreased longevity if the disease-suppression benefits of short life spans accrue to the short-lived hosts alone. However, our invasion analysis shows that selection will not favor decreased longevity if the disease suppression benefits of short life spans are shared between short-lived and long-lived hosts alike. Taken together, these results suggest that selection can favor shorter life spans when the levels of pathogen attack in the short-lived and long-lived host populations are sufficiently isolated from one another. This can occur in host metapopulations, in which many isolated patches are connected by infrequent host dispersal. Although each population of short-lived hosts is unstable against invasion by long-lived hosts, we show that short-lived hosts will persist if the rate of dispersal is sufficiently low compared with the rate of disturbance events (which create vacant patches for colonization).

Evolution of Longevity in Metapopulations

Model Structure

Here we model the evolution of host longevity in a fragmented habitat, consisting of many small patches that are largely isolated from one another. Our purpose is to explore whether, in such fragmented habitats, short-lived hosts can thrive despite their vulnerability to invasion by long-lived hosts. In keeping with the spirit of our invasion analysis above, we consider a metapopulation of host or-

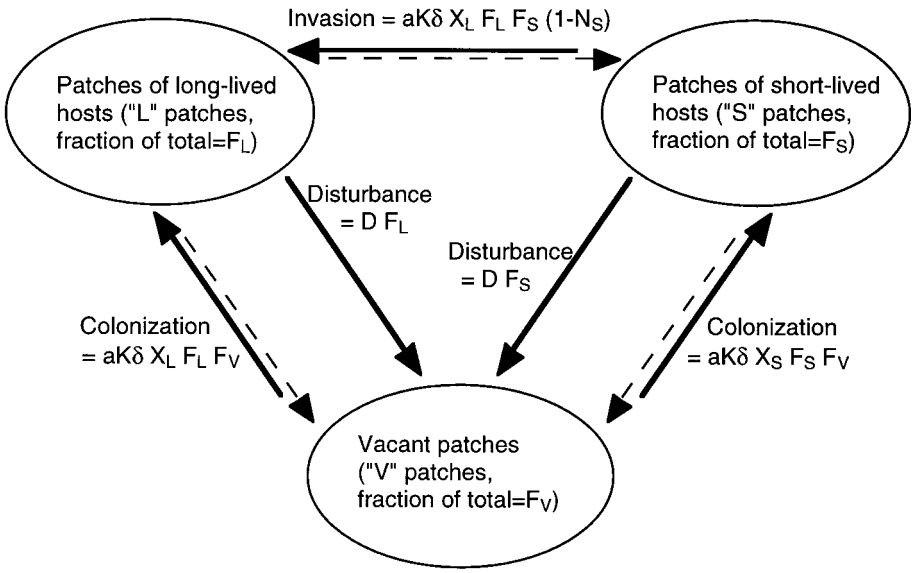

Figure 5: Transformations of habitat patches in metapopulation model. Heavy arrows indicate transformation of patches from one state to another. Thin dashed arrows indicate movement of seeds or offspring between patches (thus facilitating colonization of vacant patches, or invasion of short-lived patches by long-lived host organisms).

ganisms that belong to one of two strains: long-lived hosts with longevity τ_L and short-lived hosts with longevity τ_S.

Our metapopulation model is structurally similar to that of Nee and May (1992), consisting of three types of habitat fragments: vacant patches, patches dominated by short-lived hosts, and patches dominated by long-lived hosts (termed V patches, S patches, and L patches, respectively). Similar to our analysis above, in which we expressed the host populations as fractions of the total carrying capacity, here we express the abundance of V, S, and L patches as fractions of the total number of patches, and we denote these fractions as F_V, F_S, and F_L, respectively. We emphasize that our S and L patches are distinguished by the longevity of the host organisms that inhabit them, and not necessarily by the longevity of the patches themselves. That is, our S patches are inhabited by shorter-lived hosts, but the S patches themselves may not be proportionally shorter lived. Here our focus is not on the longevity of the patches themselves, but instead on how longevity traits of hosts evolve in patchy habitats subject to pathogen attack.

Our analysis assumes that disease inocula are ubiquitous (as is often true of wind-borne pathogens), but that the incidence of disease in each individual patch depends only on the dynamics of infection and mortality in that patch, not on the incidence of disease in neighboring patches. In other words, we assume that although disease can be transmitted from one patch to another, each host's exposure to pathogen attack is determined by the infected population of hosts within its own patch, not in other patches. This assumption is equivalent to assuming that disease dispersal can occur over long distances (and thus can occur between patches), but that the likelihood of transmission decreases with distance between individual hosts, and that the distance between patches is much greater than the size of each patch (so each host's risk of infection is determined by disease incidence within its own patch rather than in other patches). Note that, although we assume that disease inocula are ubiquitous, infection itself may not be, depending on whether host longevity is sufficient to sustain infection in any individual patch.

Our metapopulation analysis models three types of interactions between patches: colonization of vacant patches by either long-lived or short-lived hosts, invasion of patches of short-lived hosts by long-lived hosts, and creation of new vacant patches by disturbance (see fig. 5). Our analysis does not explicitly model the epidemiological or evolutionary dynamics within each patch. Instead, we assume that within-patch processes are in approximate equilibrium over the timescales appropriate for modeling the interactions between patches.

This separation of timescales simplifies the analysis considerably, and is achieved through two approximations. First, we assume that whenever a long-lived host becomes established in a patch of short-lived hosts, its genes for

longevity immediately spread throughout the patch and the whole population of the patch becomes long-lived. We showed in figure 4 that patches of short-lived hosts are unstable against invasion by long-lived hosts. The approximation we invoke here is to assume that the spread of long-lived individuals within the patch is instantaneous, rather than requiring a span of time (as in fig. 4). Our second approximation is that we assume that infection spreads fast enough within each patch (as described by eqq. [1] and [2]) that, to first approximation, the host populations in each individual patch can be assumed to be at equilibrium, as described by equations (3)–(5).

Our analysis assumes that the number of patches is large enough that, although patches are transformed by discrete events (invasion, disturbance, and colonization) the rates of these processes can be approximated by continua. This continuum assumption implies that the metapopulation is adequately described by the fraction (not the number) of patches that are V, S, and L.

Although we model the transitions between different types of patches, here we ignore the complexities of spatially explicit patch dynamics. This analysis could be easily extended to treat spatial dynamics, using cellular automaton techniques. However, here we seek to illuminate longevity's evolutionary consequences in the presence of pathogens (rather than focusing on the spread of infection from patch to patch); for the present purpose, our more straightforward approach is appropriate.

Interactions among Patches

Let us now describe the mechanisms and mathematical expressions that control the intra-patch processes of disturbance, colonization, and invasion shown in figure 5. We assume that all occupied patches have the same risk of becoming vacant because of habitat disturbance, and we denote the disturbance risk per unit time by E. Thus, the rate at which patches will be rendered vacant by disturbance is E times the number of patches. Expressed as a fraction of the total number of patches (of all types), this rate is EF_L or EF_S for patches occupied by long-lived and short-lived hosts, respectively.

The rate that host organisms can colonize vacant patches will depend on the rate that they produce viable offspring, the fraction of those offspring that are dispersed to other patches, and the fraction of the total number of patches that are vacant (and thus available for colonization). By "viable offspring," we mean offspring that will survive and grow to maturity if there is sufficient unexploited carrying capacity to support them. The rate that these are produced, per reproductively viable host, is the per capita potential reproduction rate, a, in equation (1). Some of these will grow in locations where the local resources are already exploited by other individuals, and thus they will not survive; this is the origin of the crowding factor, $1-N$, in equation (1). Here we seek to estimate the number of viable offspring produced per patch and the number of these that will be dispersed to colonize other patches.

If we denote the carrying capacity of an individual patch by K, and assume that only uninfected hosts are reproductively viable, then the rate that an individual patch produces viable offspring is aKX, where, as in equation (1), X is the fraction of carrying capacity that is occupied by uninfected hosts. As mentioned above, we assume that X is determined by the equilibrium conditions described in equations (3)–(5). We use δ to denote the fraction of these offspring that are dispersed to other patches, and we assume that $\delta \ll 1$, so that the number of offspring that are dispersed to other patches does not significantly alter the number remaining within their patch of origin and thus does not alter the reproduction term $a(1 - N)X$ in equation (1). This mathematical sleight of hand, in which we double-count a few offspring as being dispersed and also remaining behind, simplifies the analysis considerably but does not materially change the dynamics if δ is small. Thus, the rate at which individual patches will disperse viable offspring to other patches is $aK\delta X$. Just as the parameter a is the potential rate of reproduction for an individual host, the product $aK\delta$ is the potential rate of dispersal from an individual patch (if its entire carrying capacity were filled with reproductively viable hosts, i.e., if $X = 1$). The dimensionless ratio $E/aK\delta$ (i.e., the ratio between the rate of disturbance and the potential rate of dispersal) exerts primary control over the interactions between patches, as is usually the case in models of this kind.

If individual patches disperse viable offspring to other patches at a rate of $aK\delta X$, then they will disperse viable offspring to vacant patches, and thus colonize them, at a rate of $aK\delta XF_V$, where F_V is the fraction of all patches that are vacant. Expressed as a fraction of the total number of patches, the rate that hosts disperse from S patches and colonize vacant patches is thus $aK\delta X_S F_S F_V$; the comparable rate of colonization by L patches is $aK\delta X_L F_L F_V$.

In addition to colonizing vacant patches, long-lived hosts can also invade S patches, since short-lived hosts cannot compete successfully against long-lived hosts when they share the same burden of pathogens (this also prevents reciprocal invasion of L patches by short-lived hosts). The process of invasion is similar to the process of colonization, with one important difference. Colonization requires only that viable offspring are dispersed to vacant patches, because no locations in a vacant patch are currently exploited and all are available for offspring establishment. By contrast, invasion requires both that viable long-lived offspring are dispersed to S patches, and that they become established in the unexploited fraction of

those patches. The rate that an individual L patch will disperse viable offspring to short-lived patches is $aK\delta X_L F_L F_S$, by analogy with the colonization rate derived above. But only a fraction $(1 - N_S)$ of these offspring will be dispersed to points within the patch that are not already exploited, and thus will be able to become established. Thus, the rate of successful invasions of S patches by long-lived individuals, as a fraction of the total number of patches, will be $aK\delta X_L F_L F_S(1 - N_S)$.

Colonization converts V patches to S patches or L patches, disturbance converts occupied patches to vacant patches, and invasion converts S patches to L patches. Combining the rate expressions for these three processes, as derived above, we can write the net rate of change in the fraction F_L of all patches that are occupied by long-lived hosts as

$$\frac{dF_L}{dt} = aK\delta X_L F_L F_V + aK\delta X_L F_L F_S(1 - N_S) - EF_L, \quad (10)$$

where the first term on the right-hand side of the equation represents colonization, the second term represents invasion, and the third term represents distubance. The corresponding equation for the net rate of change in F_S, the fraction of all patches that are occupied by short-lived hosts, is

$$\frac{dF_S}{dt} = aK\delta X_S F_S F_V - aK\delta X_L F_L F_S(1 - N_S) - EF_S, \quad (11)$$

where the first term on the right-hand side of the equation represents colonization, the second term represents invasion, and the third term represents distubance. Finally, the rate of change in the fraction of vacant patches is

$$\frac{dF_V}{dt} = -\left(\frac{dF_L}{dt} + \frac{dF_S}{dt}\right) = E(F_L + F_S) - aK\delta F_V(X_L F_L + X_S F_S), \quad (12)$$

where the first term in the second line of the equation represents disturbance, and the second term represents colonization. As we explained above, we assume that the within-patch dynamics reach equilibrium on much shorter timescales than the between-patch dynamics, so that X_S, X_L, and N_S will be determined by the equilibrium solutions of equations (1) and (2) above, or

$$X_i = 0 \quad \text{if} \quad \tau_i < \frac{1}{a},$$
$$X_i = 1 - \frac{1}{a\tau_i} \quad \text{if} \quad \frac{1}{a} \le \tau_i < \frac{1}{a} + \frac{m}{\beta}, \quad (13)$$
$$X_i = \frac{m}{\beta\tau_i} \quad \text{if} \quad \tau_i \ge \frac{1}{a} + \frac{m}{\beta},$$

and

$$N_S = X_S \quad \text{if} \quad \tau_S < \frac{1}{a} + \frac{m}{\beta},$$
$$N_S = \frac{1}{1 + \beta/a}\left(1 + \frac{m - 1}{a\tau_S}\right) \quad \text{if} \quad \tau_S \ge \frac{1}{a} + \frac{m}{\beta}, \quad (14)$$

where the subscript i is a placeholder for S and L, representing the short-lived and long-lived strains, respectively. Equations (13) and (14) assume that only uninfected hosts can reproduce (i.e., that infection is completely sterilizing). If infection is not completely sterilizing but instead reduces host reproduction by a fraction η (ranging between 0 and 1), then X_L and X_S in equations (10)–(12) should be replaced by the equivalent reproductive population, $X_{eff} = X + (1 - \eta)Y$, in which each uninfected host counts fully, and each infected host counts according to the fraction $(1 - \eta)$ of its reproductive potential that remains after infection. In this case, the relevant values of X, Y, and N for long-lived and short-lived hosts are estimated from equations (A3)–(A5) in the appendix.

Metapopulation Dynamics

Figure 6 shows the evolution of our metapopulation (eqq. [10]–[12]) through time under three different levels of disturbance, beginning from an initial condition in which S, L, and V patches each make up one-third of the habitat. As figure 6*A* shows, when rates of disturbance are low compared with rates of dispersal, the number of vacant patches is relatively small. As a result, S patches cannot colonize vacant patches rapidly enough to offset invasion from L patches, and the short-lived strain is driven to extinction. At intermediate rates of disturbance, short-lived and long-lived hosts can coexist in equilibrium, as figure 6*B* shows. At higher rates of disturbance relative to dispersal, S patches can have a definite advantage because their lower burden of disease permits a higher rate of offspring production per patch and, therefore, more efficient colonization of vacant patches. Thus, even though they are vulnerable to invasion by long-lived hosts, they can persist and thrive while long-lived hosts go extinct (fig. 6*D*).

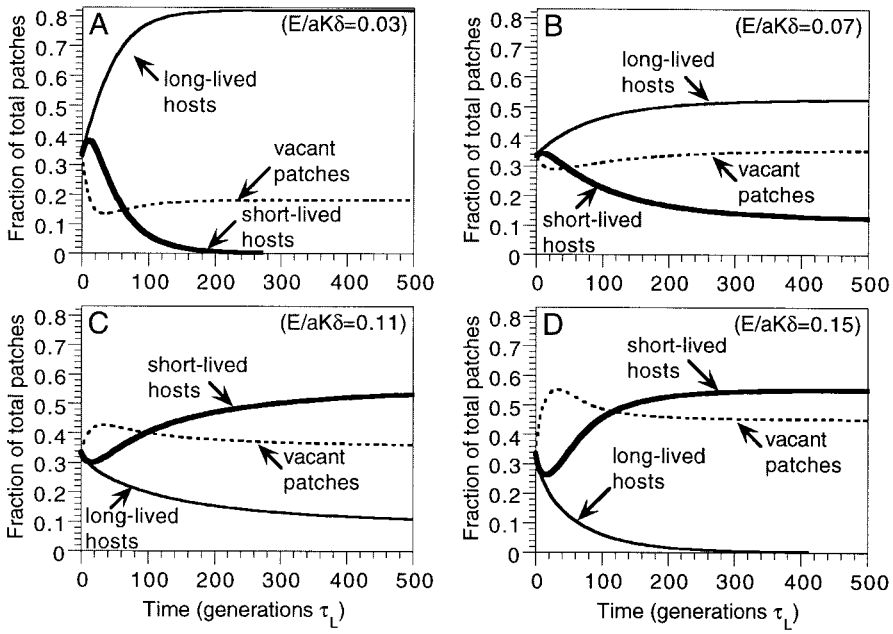

Figure 6: Numbers of patches occupied by long-lived hosts ($\tau_L = 1$) and short-lived hosts ($\tau_S = 0.5$) in metapopulation model (eqq. [10]–[14]) for four different levels of disturbance ($E = 0.015$, 0.035, 0.055, and 0.075 in *A*, *B*, *C*, and *D*, respectively, corresponding to disturbance risks of 1.5%, 3.5%, 5.5%, and 7.5% per long-lived generation). In all four panels, $aK\delta = 0.5$, implying a potential dispersal rate of 50% per long-lived generation. *A*, The risk of disturbance is low compared with rate of dispersal, and short-lived hosts are driven to extinction by invasion by long-lived hosts. *B*, The rate of disturbance is somewhat higher, and short-lived and long-lived hosts can coexist. *C*, The rate of disturbance is higher still, with the result that the short-lived hosts outnumber the long-lived hosts. *D*, The rate of disturbance is high enough that only the short-lived hosts can survive; the patches of long-lived hosts, owing to their higher burden of disease, cannot disperse quickly enough to offset losses to disturbance. Other model parameters are held constant at $a = 10$, $\beta = 30$, $m = 5$, and $K\delta = 0.05$ in all four panels.

Longer-lived hosts will out-compete shorter-lived hosts whenever they share a common environment and common pathogens. However, populations dominated by shorter-lived hosts will have higher overall offspring production rates because a larger fraction of their hosts will be uninfected (and thus reproductively viable). Populations of shorter-lived hosts will therefore have an advantage in colonizing new sites, even though they will be vulnerable to invasion by longer-lived hosts. Thus, shorter-lived hosts can persist, even though they cannot successfully compete against longer-lived hosts, as long as they can colonize new sites rapidly enough to offset their inevitable loss to invasion by longer-lived hosts. This requires that new sites are created rapidly enough by disturbance, compared with the rate of dispersal (which controls the invasion of S patches by long-lived hosts). All else being equal, higher rates of dispersal shorten the average period that S patches can persist before longer-lived individuals invade them.

Note that the intrinsic reproduction rates of the short-lived and long-lived hosts are exactly the same, but populations of short-lived hosts reproduce (and disperse) more rapidly overall because fewer of their members are sterilized by infection. We reiterate that in our analysis, short life span is not genetically linked to a higher reproduction rate, or indeed to any other trait. Short-lived hosts differ from long-lived hosts solely by having a shorter intrinsic life span. Short life span is not, in our model, a genetic trade-off resulting from higher reproduction rates. Instead, higher average rates of reproduction are achieved at the population (not individual) level, as an ecological (not genetic) consequence of disease suppression through prompt host mortality.

Their greater capacity for dispersal means that short-lived hosts can invade metapopulations of long-lived hosts, and that they can also (under somewhat more restrictive conditions) invade and become fixed, driving the long-lived hosts to extinction. Figure 7 shows two invasion scenarios, under different rates of disturbance. Both scenarios are initialized in a single-strain equilibrium. At

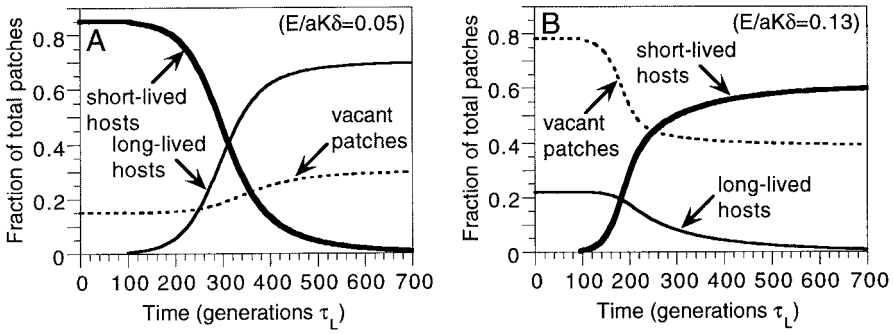

Figure 7: Invasion by long-lived hosts leading to extinction of short-lived hosts (*A*) and invasion by short-lived hosts leading to extinction of long-lived hosts (*B*). Single-strain populations are in stable equilibria before time = 100, when a very small population of the other host strain is introduced. $E = 0.025$ and 0.065 in *A* and *B*, respectively, corresponding to disturbance risks of 2.5% and 6.5% per long-lived generation; all other parameters are the same as in figure 6.

time = 100, a small population ($F = 0.005$ of the total metapopulation of patches) of the other strain is introduced. At low rates of disturbance (fig. 7*A*), S patches can persist in equilibrium if long-lived hosts are absent, but long-lived hosts can invade and drive the short-lived hosts to extinction. This result is perhaps unsurprising. However, as figure 7*B* shows, under some conditions the converse is true. That is, short-lived hosts can invade and drive long-lived hosts to extinction, even in conditions where long-lived hosts could persist in equilibrium if the short-lived hosts were absent. This can occur through an unusual kind of competitive exclusion. Short-lived and long-lived hosts are competing for the same "resource," namely, vacant patches for colonization. Although long-lived hosts can colonize both vacant and S patches, they colonize S patches less efficiently since a smaller fraction of their carrying capacity is unexploited, and thus available for long-lived offspring to become established (this is the origin of the term $1 - N_S$ in eqq. [10] and [11], as discussed above). Thus, as short-lived hosts colonize vacant patches, they diminish the overall capacity of long-lived hosts to colonize new patches. When rates of disturbance are high enough, the conversion of vacant patches to S patches can thus make long-lived hosts unable to colonize new sites rapidly enough to survive.

Metapopulation Equilibria

Under what range of conditions can short-lived hosts persist and thrive in the presence of longer-lived strains? Inspection of equations (10)–(14) shows that the metapopulation model depends on no less than eight parameters—a, K, δ, E, β, m, τ_L, and τ_S (plus η, if one wants to look at different degrees of sterilization by infection). Comprehensively exploring a such a large parameter space is difficult, but the number of dimensions can be reduced somewhat. We will consider only the equilibria of the metapopulation system, rather than its time-dependent dynamics, which has the effect of reducing the parameter space by a single parameter that incorporates a timescale (a, E, β, τ_L, or τ_S). This means we will not know how fast the system changes through time but only where it comes to rest. In the equilibrium solutions to equations (10)–(12), the parameters K, δ, and E always appear together in the dimensionless disturbance/dispersal ratio $E/aK\delta$, further reducing the parameter space by two dimensions. We will explore several slices through the resulting five-dimensional parameter space.

The equilibrium solution to equations (10)–(12) depends on whether one or both host strains are present. If either short-lived or long-lived hosts are absent, the solution is

$$F_S = F_S^* = 1 - \frac{E}{aK\delta}\frac{1}{X_S} \quad \text{if} \quad F_L = 0,$$

$$F_L = F_L^* = 1 - \frac{E}{aK\delta}\frac{1}{X_L} \quad \text{if} \quad F_S = 0, \qquad (15)$$

where F_L^* and F_S^* are the equilibrium frequencies of patches of long- and short-lived hosts in the absence of competition from the other host strain. If both host strains are present, the equilibrium solution is a weighted sum of F_L^* and F_S^*,

$$F_L = \frac{F_L^* - F_S^* N_S}{1 - N_S[1 + (X_L/X_S)(1 - N_S)]}$$
$$\text{if} \quad F_S > 0,$$
$$F_S = \frac{F_S^* - F_L^*[1 + (X_L/X_S)(1 - N_S)]}{1 - N_S[1 + (X_L/X_S)(1 - N_S)]} \tag{16}$$
$$\text{if} \quad F_L > 0.$$

In all cases, there is an implicit lower bound of 0 for F_L, F_S, F_L^*, and F_S^*. As in equations (10)–(12), the values of X_L, X_S, and N_S are determined by equations (13) and (14) for completely sterilizing infections. For infections that do not completely sterilize the host, X_L and X_S are replaced by the equivalent reproductive populations for the two host strains, which (along with N_S) are then calculated via equations (A3)–(A5) in the appendix.

Figure 8 shows how the relative longevity of long-lived and short-lived hosts affects their relative abundance under four different disturbance/dispersal ($E/aK\delta$) regimes. The difference between the thin and thick lines shows how short-lived (*solid lines*) and long-lived (*dashed lines*) hosts are affected by competition from the other host strain. Competition from long-lived hosts narrows the range of life spans over which short-lived hosts can thrive, particularly when the disturbance/dispersal ratio is small. Short-lived hosts cannot persist when their life span is close to that of their long-lived competitors, because this diminishes the disease-suppression advantage of the short-lived hosts while their vulnerability to invasion remains unchanged. However, over a range of life spans, shorter-lived hosts have a net advantage over longer-lived hosts, in some cases even driving them to extinction. This occurs over wider life span ranges at higher rates of disturbance relative to dispersal. That is, competition from short-lived hosts affects long-lived hosts more severely at higher rates of disturbance. Conversely, short-lived hosts are hurt more by long-lived competitors when rates of disturbance are relatively low. These results show that shorter-lived hosts can coexist with longer-lived hosts over wide ranges of relative life span (τ_S/τ_L) and disturbance/dispersal ratio ($E/aK\delta$), and can dominate over their longer-lived competitors (even to the point of competitively excluding them) over a significant range of conditions.

One useful way to summarize the behavior of the metapopulation model is to delimit the conditions under which each host strain can exist at equilibrium. The possibility of competitive exclusion implies that whether each host strain can exist will depend, in part, on whether the other strain is present. Several domains are potentially interesting: domains where neither host strain can exist, domains where only one host strain can exist, domains where one host strain can exist only if the other is absent (i.e., domains of competitive exclusion), and domains where both host strains can coexist. These domains can be mapped out from equations (15) and (16). Long-lived hosts can persist if the rate of disturbance, relative to dispersal, is low enough that they can colonize new patches before being wiped out by disturbance. The presence of short-lived hosts lowers the critical level of disturbance somewhat because their patches are more difficult to colonize than vacant patches are (as discussed above). In quantitative terms,

$$F_L > 0 \quad \text{when} \quad \frac{E}{aK\delta} < X_L \quad \text{if} \quad F_S = 0,$$
$$F_L > 0 \quad \text{when} \quad \frac{E}{aK\delta} < X_L \frac{1 - N_S}{1 - N_S X_L/X_S} \tag{17}$$
$$\text{if} \quad F_S > 0.$$

For short-lived hosts the situation is slightly more complex. Like long-lived hosts, short-lived hosts require that the rate of disturbance, relative to dispersal, permits them to colonize new patches before they are wiped out by disturbance. When long-lived hosts are present, however, short-lived hosts also require a minimum rate of disturbance (relative to dispersal) to survive; they need new vacant patches to be created rapidly enough so that they can colonize new sites fast enough to offset their inevitable invasion by the long-lived strain. In quantitative terms,

$$F_S > 0 \quad \text{when} \quad \frac{E}{aK\delta} < X_S \quad \text{if} \quad F_L = 0,$$
$$F_S > 0 \quad \text{when} \quad X_S > \frac{E}{aK\delta} > \frac{X_L^2}{X_S} \frac{1 - N_S}{1 - N_S X_L/X_S} \tag{18}$$
$$\text{if} \quad F_L > 0.$$

Using equations (17) and (18), we can delimit the conditions under which each host strain can survive, with and without the other host strain (fig. 9).

Figure 9 shows the domains of feasibility for metapopulations of short-lived and long-lived hosts under attack by diseases with widely varying characteristics. Each panel of figure 9 represents a disease with a different combination of consequences for host fertility and survival. The three columns of panels correspond to different degrees of sterilization by infection (increasing left to right), and the four rows correspond to different degrees of path-

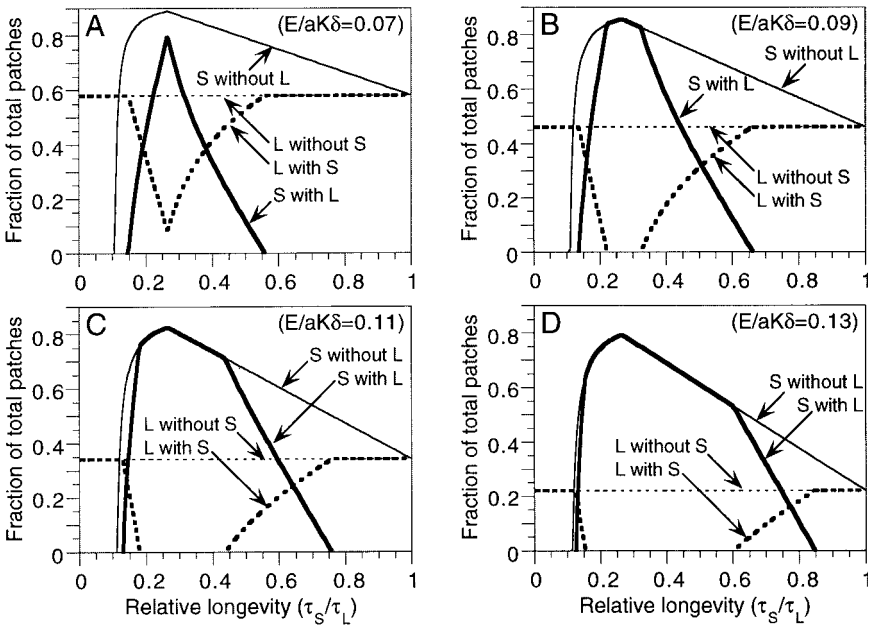

Figure 8: Equilibrium frequencies of long-lived and short-lived patches in metapopulation model (eqq. [10]–[16]), for four different rates of disturbance relative to potential dispersal ($E/aK\delta$). Lines show the equilibrium frequencies of short-lived patches (*solid lines*) and long-lived patches (*dotted lines*) when the other host strain is absent (*thin lines*) and present (*thick lines*). As the level of disturbance relative to dispersal increases, short-lived hosts with wider ranges of relative longevity (τ_S/τ_L) can exist, despite competition from longer-lived hosts. As the disturbance/dispersal ratio increases, short-lived hosts are affected less and less by the presence of long-lived hosts (i.e., the thin and thick solid lines converge over a wider range of τ_S/τ_L), and, conversely, long-lived hosts cannot persist in the presence of short-lived hosts over a wider range of τ_S/τ_L. Other parameters are held constant in all four panels: $a = 10$, $\beta = 30$, $\tau_L = 1$, $m = 5$, $\eta = 1$.

ogen lethality (increasing top to bottom). The axes are the same on all panels of figure 9 except *C* and *F*, which are expanded to show more detail. As one might intuitively expect, greater longevity is always advantageous if infection has a relatively small impact on both life span and fertility (i.e., panels toward the upper left of fig. 9). When the fitness consequences of infection are small, shorter-lived hosts can only survive over more restricted ranges of disturbance/dispersal conditions than longer-lived hosts, and only if the longer-lived hosts are themselves absent (the domains marked "L excludes S" in fig. 9*A*, *B*, *D*). By contrast, when infection has severe fitness effects (i.e., panels toward the right or bottom of fig. 9), the disease-suppression benefits of short life spans are more consequential, enabling shorter-lived hosts to persist under disturbance/dispersal conditions in which longer-lived hosts cannot survive (the domains marked "S only" in fig. 9*C*, *F*, *H*–*L*). Particularly when infection greatly shortens life span, there are substantial ranges of disturbance/dispersal conditions under which shorter-lived hosts can displace longer-lived hosts and drive them from the metapopulation (domains marked "S excludes L"). Short-lived and long-lived hosts can coexist in the domains marked "S&L" in figure 9, which span wide ranges of relative life spans (τ_S/τ_L) but surprisingly narrow ranges of disturbance/dispersal conditions ($E/aK\delta$). As a result, only narrow ranges of disturbance/dispersal rates separate the conditions that lead to competitive exclusion of the longer-lived host ("S excludes L") from the conditions that lead to competitive exclusion of the shorter-lived host ("L excludes S").

Implications

The model results presented above support three general propositions. First, an organism's life-history traits, and not just its resistance genes, can affect its susceptibility to pathogen attack. Second, for this reason, pathogen-mediated selection may influence the evolution of host life-history traits. Third, the magnitude—and even the direction—of selection on host life-history traits can depend on the structure of the host metapopulation.

In our metapopulation model, selection for or against

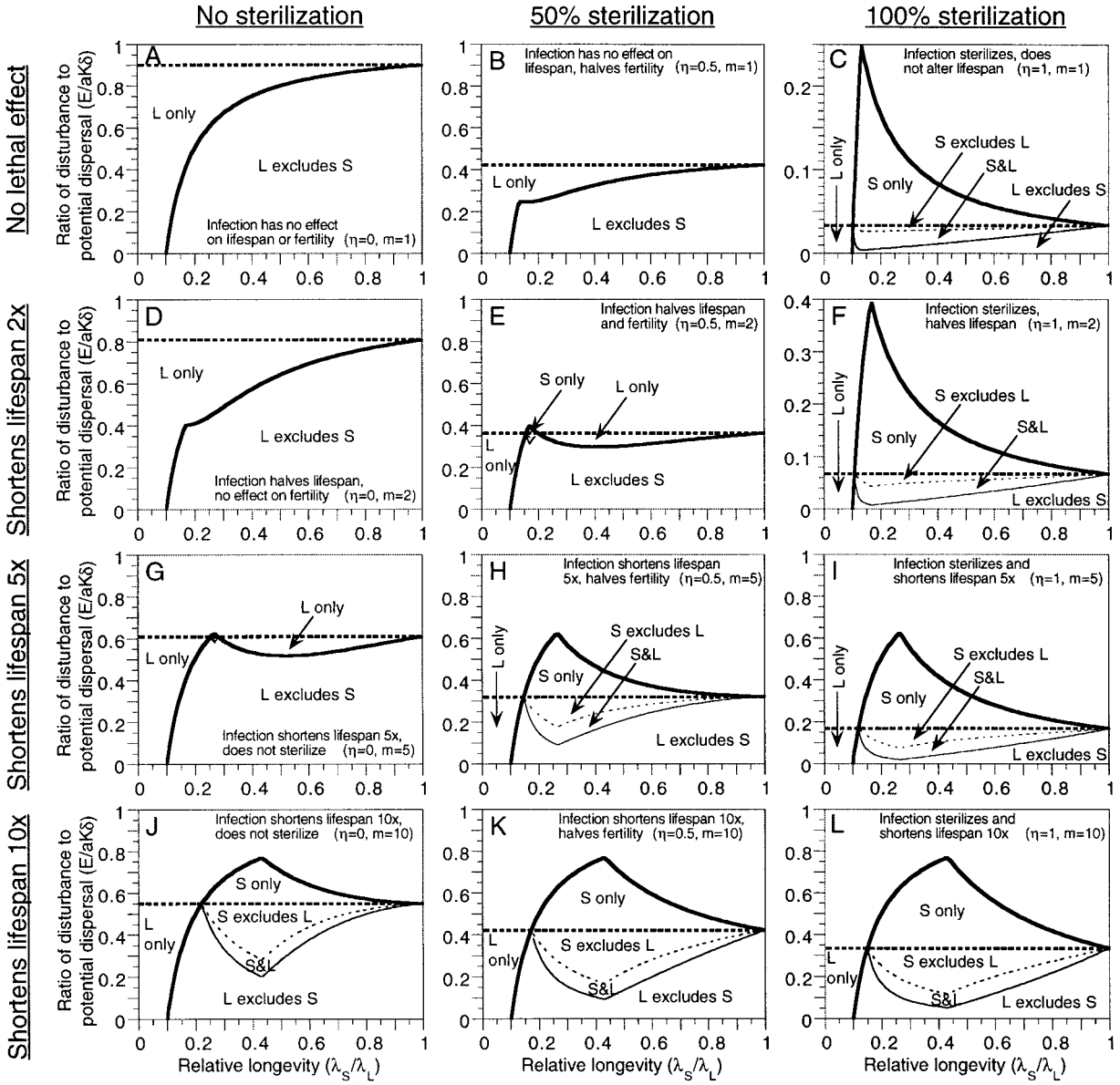

Figure 9: Feasibility domains for metapopulation model, showing disturbance/dispersal conditions under which long-lived (L) and short-lived (S) host organisms can persist. Domains marked "L only" (or "S only") delimit conditions under which only long-lived (or short-lived) hosts can exist. Domains marked "L excludes S" delimit conditions under which short-lived hosts can exist if long-lived hosts are absent, but under which they cannot survive in competition with long-lived hosts. In the absence of long-lived hosts, short-lived hosts could survive in all conditions lying below the heavy solid line. If long-lived hosts are present, short-lived hosts can survive only in the domain lying between the heavy solid line and the light solid line. Domains marked "S excludes L" delimit conditions under which long-lived hosts can exist if short-lived hosts are absent, but under which they cannot survive in competition with short-lived hosts. In the absence of short-lived hosts, long-lived hosts could survive in all conditions lying below the heavy dotted line. If short-lived hosts are present, long-lived hosts can survive only in the domain lying below both the heavy dotted line and the light dotted line. Domains marked "S&L" indicate conditions in which short- and long-lived hosts can coexist stably. Panels are organized in three columns, corresponding to nonsterilizing infections ($\eta = 0$; *left column*), sterilizing infections ($\eta = 1$; *right column*), and infections that reduce fertility by 50% ($\eta = 0.5$; *center column*). The four rows of columns correspond to infections with four different degrees of lethality: those that do not accelerate mortality ($m = 1$; *top row*), and those that shorten life span by twofold, fivefold, and 10-fold ($m = 2$, 5, and 10, next three rows). Other parameters are held constant in all panels: $a = 10, \beta = 30, \tau_L = 1$. When infection affects fitness severely, by either diminishing fertility or accelerating mortality, there are sizeable domains ("S only" and "S excludes L") in which short-lived hosts have an advantage over long-lived hosts.

greater host longevity depends critically on the rate of patch disturbance compared with the rate of host dispersal between patches. Small differences in the disturbance/dispersal ratio separate conditions where longer-lived hosts are driven to extinction from conditions where shorter-lived hosts are driven to extinction (figs. 6, 7, 9).

Inspection of figure 9 reveals a common pattern in the way that the disturbance/dispersal ratio affects selection on longevity of hosts subject to pathogen attack. For all relative life spans (τ_S/τ_L) for which short-lived hosts can potentially survive, they are competitively excluded by longer-lived hosts when rates of disturbance (relative to dispersal) are low. At somewhat higher rates of disturbance (or lower rates of dispersal), shorter-lived and longer-lived host strains can coexist at equilibrium. At still higher disturbance/dispersal ratios, shorter-lived hosts competitively exclude longer-lived hosts. Finally, at even higher rates of disturbance (relative to dispersal), only shorter-lived hosts can persist; longer-lived hosts cannot survive, even without competition from shorter-lived hosts. Figure 9 shows that this pattern holds for many different kinds of pathogens, including those that completely sterilize the host and those that have no effects on fertility but greatly accelerate host mortality. Because this pattern appears to be general, it may be testable in laboratory experiments or in field studies, such as the *Silene* metapopulation systems (Antonovics et al. 1994; Giles and Goudet 1997; Thrall and Burdon 1997). In the real world, of course, rates of disturbance and dispersal will vary both spatially and temporally. Thus, their effects on selection are not likely to be as clear in the real world as they are in the model. Nonetheless, as long as pathogen infection has severe fitness consequences, we expect to see a general tendency for selection to favor shorter life spans at higher rates of disturbance relative to dispersal, and to favor longer life spans at lower disturbance/dispersal ratios.

It has long been understood that, under certain conditions, two species can coexist in a metapopulation even if one has a competitive advantage within each subpopulation, as long as the inferior competitor can disperse more rapidly (Hutchinson 1951; Skellam 1951; Slatkin 1974; Hanski 1983; Hanski and Ranta 1983; Nee and May 1992). Our system is unusual in that a single trait—diminished longevity—is solely responsible both for making the short-lived strain competitively inferior, and also for enabling it to disperse more rapidly (because of its lower burden of disease). Our system is also striking because, under some conditions, the shorter-lived strain can invade a stable metapopulation of longer-lived hosts and drive them to extinction, even though the shorter-lived strain is competitively inferior in every patch within the metapopulation.

The ecological dynamics that permit short-lived hosts to persist and thrive in our model superficially resemble those that permit ruderal "invasive" species to flourish in disturbed habitats, even though they are poor competitors in stable environments. But whereas ruderal species thrive in disturbed environments because they have a higher intrinsic reproduction rate (usually achieved at the cost of a shorter life span), our short-lived host strain has exactly the same intrinsic reproduction rate (a) as the long-lived strain. Our short-lived host strain can disperse more rapidly than the long-lived strain simply because its shorter life span means that it bears a smaller burden of disease. This point bears emphasis: in our analysis, short-lived hosts differ from long-lived hosts solely in having a shorter intrinsic life span; in all other respects, their traits are exactly the same. Short-lived individuals do not "compensate" for their diminished longevity by reproducing more rapidly. They can disperse more rapidly from a single-strain population than long-lived hosts can, not because they reproduce more rapidly but because a greater fraction remain uninfected and thus reproductively viable. This is not an individual trait but instead a population-level phenomenon, and one that depends on the ecological context—the degree of habitat fragmentation, the presence or absence of pathogens, and so forth (Wade and Kalisz 1990). In our analysis, short-lived hosts have no advantage that compensates for the disadvantage posed by their diminished longevity. Instead, it is their shorter life spans themselves that are either advantageous or disadvantageous, depending on the ecological context.

Dispersal is another example of a single trait that is advantageous in metapopulations because it permits colonization of vacant patches, even though it is disadvantageous within occupied patches (Van Valen 1971; Olivieri et al. 1995). High dispersal simultaneously makes individuals superior colonizers and inferior within-patch competitors (because fewer of their offspring remain in the home patch, compared with the offspring of low dispersers). But whereas high dispersers are intrinsically better colonizers regardless of their surroundings, short-lived hosts are better colonists than longer-lived hosts only when they are surrounded by other short-lived individuals. Host fitness depends on the incidence of infection, which is not a trait exhibited by individuals but rather a global (or "emergent") property of groups; this creates selection gradients between groups that are different from the selection gradients within them (Heisler and Damuth 1987).

In our analysis, selection acts on individuals, not on groups. However, our analysis illustrates how the mechanisms and consequences of selection depend critically on the ecological context in which it occurs, including the characteristics of other individuals in the population (Goodnight and Stevens 1997). When individuals are surrounded by others sharing similar longevity traits (as one

would expect in isolated populations with strong founder effects), short life spans are advantageous because they suppress disease (figs. 2, 3). In contrast, when populations combine individuals with different life spans, short life spans are disadvantageous; although short-lived hosts spread disease less readily, this benefits the population as a whole but provides no competitive advantage to the short-lived hosts. Thus, whether selection favors shorter or longer life spans will depend on the degree of isolation between populations, the degree of homogeneity within populations, and the extent to which pathogen infection affects fitness.

We do not know whether real-world conditions often favor selection for shorter host life spans. However, given that infectious diseases often have severe fitness consequences (Baudoin 1975; Price 1980; Agrios 1988; Clay 1991), and given that host populations are often highly fragmented (Grenfell and Harwood 1997; Hanski and Gilpin 1997), it is important to consider the possibility that selection may not always favor greater host longevity. It would obviously be helpful if hosts promptly died on acquiring permanent sterilizing infections, thus preventing them from spreading their diseases to uninfected kin. What our analysis demonstrates is less obvious and more general, namely, that natural selection can favor shorter life spans even when they are imposed on infected and uninfected individuals alike.

Besides illuminating patterns of selection in nature, our observations may be relevant to agricultural pest control. They suggest that if one can limit the longevity of host organisms, and thus limit the reservoir of infection, one can reduce the spread of disease to healthy hosts. We are not aware of any crop breeding programs that have manipulated longevity to control disease. However, crops are commonly turned under or burned after harvest, specifically to control disease by removing the supply of host organisms, and thus inoculum. We point out that these practices control the spread of disease by effectively limiting host longevity to the minimum required for crop production.

We note that pathogen-mediated selection for shorter longevity can arise through mechanisms other than the metapopulation processes modeled here. Any mechanism that sufficiently isolates different host strains can enable a short-lived strain to enjoy the population-level disease-suppression benefits of its lesser longevity. For example, this kind of isolation can arise through purely genetic mechanisms, such as pleiotropy or linkage, in which individuals with different longevity traits are susceptible to different pathogen strains. This "privatizes" the epidemiological consequences of longevity to each host strain because each is infected by a different pathogen strain. The degree of epidemiological isolation between host strains, and thus the fitness consequences of longevity, will be controlled by the degree of host-pathogen genotype specificity (J. W. Kirchner and B. A. Roy, unpublished manuscript), just as it is controlled by the disturbance/dispersal ratio in metapopulations.

Summary

Senescence has previously been explained as a process that evolution cannot protect against (Finch 1990; Partridge and Barton 1993) or an unavoidable evolutionary by-product of selection on other traits (Williams 1957; Stearns 1992). By contrast, our work shows that shorter life spans can be selectively favored by evolution, in their own right, for the fitness advantages they convey.

When pathogen infection carries fitness consequences, host characteristics that affect rates of infection will be subject to pathogen-mediated selection. A simple model of host-pathogen population dynamics (fig. 1; eqq. [1]–[5]), shows that under a wide range of conditions, greater host longevity leads to a larger and more persistent reservoir of infection and thus diminishes host fitness (figs. 2, 3). However, when longer-lived and shorter-lived hosts can infect each other, the consequences of host longevity for rates of infection are shared among longer-lived and shorter-lived hosts alike. In this case, selection will favor greater host longevity to the detriment of the population as a whole (fig. 4; eqq. [6]–[9]). However, in highly fragmented populations with strong founder effects, individuals are likely to be surrounded by others that share their characteristics. In these circumstances, the disease-suppression benefits of short life spans are likely to be shared among individuals who are all short lived, and thus short life spans can be evolutionarily advantageous. Thus, whether selection favors shorter or longer life spans will depend on the degree to which long-lived and short-lived populations are isolated from one another.

We explored these concepts using a simple metapopulation model (fig. 5; eqq. [10]–[14]) in which vacant patches are created by disturbance and colonized by dispersal of both long-lived and short-lived hosts. The success of the long-lived and short-lived strains in our metapopulation model depends on the relative rates of disturbance and dispersal (figs. 6–9), which control the relative importance of founder effects (which favor shorter-lived hosts) and intra-patch competition (which favors longer-lived hosts). Under surprisingly wide ranges of conditions, shorter-lived hosts can persist in the metapopulation model, even though they have a clear disadvantage in intra-patch competition with longer-lived hosts (figs. 8, 9; eqq. [17], [18]). Under somewhat more restrictive conditions, shorter-lived hosts can invade a stable metapopulation of

longer-lived hosts, displacing them and driving them to extinction (figs. 7–9). These results demonstrate the potential for pathogen-mediated selection to influence the evolution of host life-history traits, including traits not normally considered to be connected to disease resistance. The magnitude and direction of selection will depend on the ecological context in which host traits are expressed, which in turn may depend on the structure of the host metapopulation.

Acknowledgments

We thank S. M. Eppley, C. M. Lively, M. L. Stanton, and S. C. Stearns for their comments on an earlier version of the manuscript and D. McCauley, I. Olivieri, and P. H. Thrall for their comments on the draft of the present article. This work was supported by National Science Foundation grant EAR-9357931 to J.W.K. and Swiss National Fund grant 3100-046865.96 to B.A.R.

APPENDIX

Epidemiology with Incomplete Host Sterilization

The simple model of disease epidemiology presented in equations (1)–(5) and figures 1–3 assumes that infection completely sterilizes the host, as is common for many plant (Clay 1991) and animal (Baudoin 1975) diseases. What are the consequences of relaxing this assumption? Here we summarize results from a slightly modified model, in which infection is assumed to reduce host fertility by a fraction η, which can take on values between 0 and 1. As before, we assume that the potential reproduction rate of uninfected hosts is a (i.e., in the absence of carrying capacity constraints each healthy host would reproduce at a rate of a). But rather than assuming that infected hosts cannot reproduce, here we assume that they reproduce at a rate $a(1 - \eta)$, where $\eta = 1$ implies that infection completely sterilizes the host, and $\eta = 0$ implies that infection has no effect on host fertility. Under this assumption, the model equations become

$$\frac{dX}{dt} = a(1 - N)[X + (1 - \eta)Y] - \beta XY - \frac{X}{\tau} \tag{A1}$$

and

$$\frac{dY}{dt} = \beta XY - \frac{mY}{\tau}. \tag{A2}$$

As before, we will look at the equilibrium behavior of these equations as an indicator of the model system's general tendencies. If the host life span is too short for infection to sustain itself, then the equilibrium behavior is unchanged from that of equations (3) and (4):

$$X = \max(0, 1 - \frac{1}{a\tau}) \quad \text{and} \quad Y = 0 \quad \text{if} \quad \tau \leq \frac{1}{a} + \frac{m}{\beta}. \tag{A3}$$

If host life span is sufficient for infection to sustain itself, then the equilibrium solution of equations (A1) and (A2) is:

$$X = \frac{1}{\beta\tau/m} \quad \text{and} \quad Y = \sqrt{Q_1^2 + Q_2} - Q_1 \quad \text{if} \quad \tau \geq \frac{1}{a} + \frac{m}{\beta}, \tag{A4}$$

where

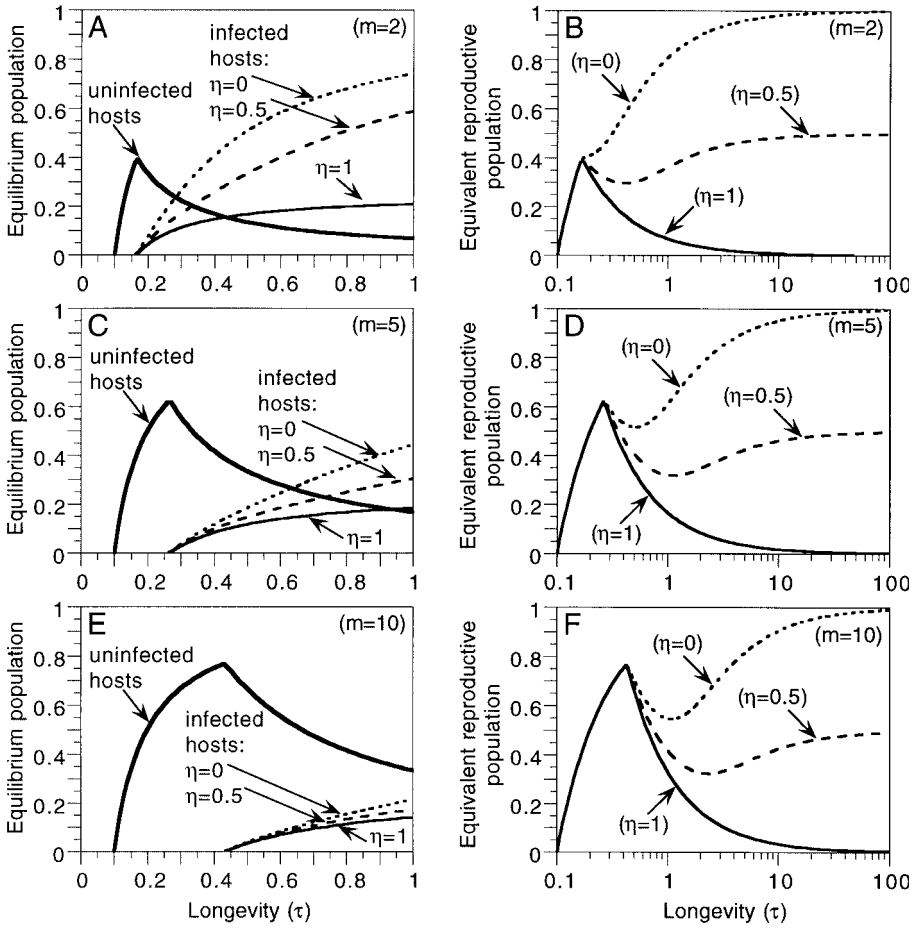

Figure A1: Equilibrium populations of uninfected and infected hosts implied by equations (A1) and (A2) for infections that alter host fertility and mortality by different degrees. Each panel shows equilibria for three different levels of sterilization by infection: complete sterilization ($\eta = 1$), partial sterilization ($\eta = 0.5$), and no sterilization ($\eta = 0$). Left-hand panels show the equilibrium populations of infected and uninfected hosts as fractions of carrying capacity. Right-hand panels show the equivalent reproductive population, defined as $X + (1 - \eta)Y$. The top row of panels shows equilibria for an infection that halves host life span ($m = 2$); in the middle row, infection diminishes life span by fivefold ($m = 5$), and in the bottom row, infection diminishes life span by 10-fold ($m = 10$). In all panels, $a = 10$ and $\beta = 30$.

$$Q_1 = \frac{1}{2}\frac{1}{\beta\tau/m}\left[1 + \frac{1 + \beta/a}{1 - \eta}\right] - \frac{1}{2},$$

$$Q_2 = \frac{1}{\beta\tau/m}\left\{\frac{1 - [1/(\beta\tau/m)] - (1/a\tau)}{1 - \eta}\right\}. \tag{A5}$$

Note that the equilibrium infected population (Y) depends on the degree of sterilization (η), but the uninfected population (X) does not.

The equilibrium behavior of equations (A1) and (A2) is shown in figure A1. When infection completely sterilizes the host, the optimal longevity is, as before, that which maximizes the population of uninfected (and thus reproductively viable) hosts. If infection partially sterilizes the host, however, the optimal longevity will be that which maximizes the

total reproductive potential of the host population, including the (partially impaired) reproductive potential of the infected hosts. Here we define this total reproductive potential in terms of an "equivalent reproductive population," $X_{\text{eff}} = X + (1 - \eta)Y$, in which each uninfected host counts fully, and each infected host counts according to the fraction $(1 - \eta)$ of its reproductive potential that remains after infection.

From equations (A3)–(A5), one can show that the equivalent reproductive population will be maximized either at $\tau = (1/a) + (m/\beta)$ (the point at which the infected population vanishes) or in the limit as τ rises toward infinity (and thus the infected population dominates the system). Whether there is one local optimum or two, and which is the global optimum, will depend on the value of η compared with a, β, and m. If $\eta = 1$, then the only optimum is $\tau = (1/a) + (m/\beta)$. Alternatively, if $\eta < (2 - m)/[2 + (am/\beta)]$, the only optimum is $\tau \to \infty$. If $(2 - m)/[2 + (am/\beta)] < \eta < 1$, then both of these values of τ are local optima. When there are two local optima, $\tau = (1/a) + (m/\beta)$ will be the global optimum if $\eta > 1/[1 + (am/\beta)]$, and $\tau \to \infty$ will be the global optimum if $\eta < 1/[1 + (am/\beta)]$.

Literature Cited

Agrios, G. N. 1988. Plant pathology. Academic Press, San Diego, Calif.

Anderson, R. M., and R. M. May. 1991. Infectious diseases of humans: dynamics and control. Oxford University Press, Oxford.

Antonovics, J., P. H. Thrall, A. M. Jarosz, and D. Statton. 1994. Ecological genetics of metapopulations: the *Silene-Ustilago* plant-pathogen system. Pages 146–170 *in* L. Real, ed. Ecological genetics. Princeton University Press, Princeton, N.J.

Baudoin, M. 1975. Host castration as a parasitic strategy. Evolution 29:335–352.

Clay, K. 1991. Parasitic castration of plants by fungi. Trends in Ecology & Evolution 6:162–166.

Clay, K., and W. van der Putten. In press. Pathogens and plant life histories. *In* T. Vuorisalo and P. Mutikainen, eds. Life history evolution in plants. Kluwer, Dordrecht.

Finch, C. E. 1990. Longevity, senescence and the genome. University of Chicago Press, Chicago.

Giles, B. E., and J. Goudet. 1997. Genetic differentiation in *Silene dioica* metapopulations: estimation of spatiotemporal effects in a successional plant species. American Naturalist 149:507–526.

Goodnight, C. J., and L. Stevens. 1997. Experimental studies of group selection: what do they tell us about group selection in nature? American Naturalist 150(suppl.): S59–S79.

Grenfell, B., and J. Harwood. 1997. (Meta)population dynamics of infectious diseases. Trends in Ecology & Evolution 12:395–399.

Haldane, J. B. S. 1941. New paths in genetics. Allen & Unwin, London.

Hamilton, W. D. 1966. The molding of senescence by natural selection. Journal of Theoretical Biology 12:12–45.

Hanski, I. 1983. Coexistence of competitors in patchy environment. Ecology 63:493–500.

Hanski, I., and M. E. Gilpin. 1997. Metapopulation biology: ecology, genetics, and evolution. Academic Press, San Diego, Calif.

Hanski, I., and E. Ranta. 1983. Coexistence in a patchy environment: three species of *Daphnia* in rock pools. Journal of Animal Ecology 52:263–269.

Heisler, I. L., and J. Damuth. 1987. A method for analyzing selection in hierarchically structured populations. American Naturalist 130:582–602.

Hutchinson, G. E. 1951. Copepodology for the ornithologist. Ecology 32:571–577.

Keller, L., and M. Genoud. 1997. Extraordinary lifespans in ants: a test of evolutionary theories of ageing. Nature (London) 389:958–960.

May, R. M., and R. M. Anderson. 1983. Epidemiology and genetics in the coevolution of parasites and hosts. Proceedings of the Royal Society of London B, Biological Sciences 219:281–313.

Maynard-Smith, J. 1976. Group selection. Quarterly Review of Biology 51:277–283.

Medawar, P. B. 1946. Old age and natural death. Modern Quarterly 1:30–56.

Nee, S., and R. M. May. 1992. Dynamics of metapopulations: habitat destruction and competitive coexistence. Journal of Animal Ecology 61:37–40.

Olivieri, I., Y. Michalakis, and P.-H. Gouyon. 1995. Metapopulation genetics and the evolution of dispersal. American Naturalist 146:202–228.

Partridge, L., and N. N. Barton. 1993. Optimality, mutation and the evolution of ageing. Nature (London) 362: 305–311.

Price, P. W. 1980. Evolutionary biology of parasites. Monographs in Population Biology. Princeton University Press, Princeton, N.J.

Rose, M. R. 1991. The evolutionary biology of aging. Oxford University Press, Oxford.

Roy, B. A. 1993. Floral mimicry by a plant pathogen. Nature (London) 362:56–58.

Roy, B. A., and P. Bierzychudek. 1993. The potential for rust infection to cause natural selection in apomictic

Arabis holboellii (Brassicaceae). Oecologia (Berlin) 95: 533–541.

Sheldon, B.C., and S. Verhulst. 1996. Ecological immunology: costly parasite defenses and trade-offs in evolutionary ecology. Trends in Ecology & Evolution 11: 317–321.

Shykoff, J. A. 1997. Effects of the anther smut fungus *Microbotryum violaceum* on host life-history patterns in *Silene latifolia* (Caryophyllaceae). International Journal of Plant Sciences 158:164–171.

Skellam, J. G. 1951. Random dispersal in theoretical populations. Biometrika 38:196–218.

Slatkin, M. 1974. Competition and regional coexistence. Ecology 55:128–134.

Stearns, S.C. 1992. The evolution of life histories. Oxford University Press, Oxford.

Thrall, P. H., and J. J. Burdon. 1997. Host-pathogen dynamics in a metapopulation context: the ecological and evolutionary consequences of being spatial. Journal of Ecology 85:743–753.

Van Valen, L. 1971. Group selection and the evolution of dispersal. Evolution 25:591–598.

Wade, M. J., and S. J. Kalisz. 1990. The causes of natural selection. Evolution 44:1947–1955.

Williams, G. C. 1957. Pleiotropy, natural selection, and the evolution of senescence. Evolution 11:398–441.

———. 1966. Adaptation and natural selection. Princeton University Press, Princeton, N.J.

Associate Editor: David E. McCauley