Ecosystems and Parasitism: The Spatial Dimension

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Introduction

Most species of pathogens and their hosts occur in multiple, spatially distributed sites, dispersed over much larger distances than the spatial arena that circumscribes the lives of individuals and interactions between pathogens and hosts. Yet, some pathogens have tiny spores and can move great distances via aerial dispersal, which potentially leads to coupling of host-parasite systems at continental and even global spatial scales (Brown and Hovmoller 2002). The combination of local interactions and dispersal at various scales leads to a range of important implications of spatial dynamics for the ecology and evolution of host-pathogen interactions, with consequences for the ecosystem impacts of parasitism (Hochberg and Holt 2002).

There is a vast literature on the dynamics of infectious disease in well-mixed populations (Anderson and May 1991). For many years, many authors have recognized the importance of space for the ecological and evolutionary dynamics of host-pathogen systems (e.g., Cliff et al. 1981, Bolker and Grenfell 1995). Mollison and Levin (1995) provide a useful review of earlier work. We will not attempt to synthesize this far-flung literature (which would take a volume all by itself), but instead draw out some highlights that seem most pertinent to ecosystem issues.

Ecosystem implications of parasitism: some potential major effects

From an ecosystem perspective, parasites have several distinct consequences; spatial dynamics can matter for each of these.

Population limitation and regulation. Understanding how populations are limited in abundance, and bounded in their fluctuations, is essential to understanding how ecosystems as a whole are governed. Although not sufficient for understanding ecosystem processes, the standard concerns of population and community ecology – the focus on processes of positive and negative feedbacks arising from density-dependence, resource dependencies, and interspecific interactions – are necessary for understanding patterns of energy and nutrient flows in ecosystems, and the responses of ecosystems to disturbance and secular environmental change. Demographic effects of parasites leading to population limitation and regulation, when quantitatively strong, can affect many aspects of ecosystem dynamics. For instance, one hypothesis for the rich diversity of trees in tropical rain forest is that species experience strong, intraspecific density-dependence due to host-specific pathogens acting on seedling plants (the Janzen-Connell hypothesis). Given that diversity is maintained (for whatever reason), there are likely to be numerous other differences present among species. Such differences could be important at buffering the system from environmental change (the 'insurance hypothesis', Loreau et al. 2003)

Spatial dynamics can influence the ability of parasites to limit and regulate their hosts, over both ecological and evolutionary time-scales; theoretical reasons why this is to be expected are given below. It is thus likely that the ecosystem roles of parasites have important spatial dimensions.

Energy and nutrient flows. A core concern of ecosystem ecology is to understand the fluxes of energy and material through a given population that is potentially available to the rest of the ecosystem. Flux rates are closely related to the death rate of the population, which governs the provisioning of biomass either for consumption by higher trophic levels, or for decomposition. Pathogens that directly increase the death rate of their hosts will thereby facilitate entry of nutrients into the decomposer food web. Pathogens that make their hosts more vulnerable to predation will alter the strength of trophic interactions, and thus the channeling of energy and nutrients and food webs. Parasites which lead to morbidity in their hosts may make those hosts less capable as consumers, making these species less significant factors in the dynamics of their resources (both biotic and abiotic). Spatial heterogeneity and dynamics which influences the average parasite 'load' of a host population can thus have profound ecosystem consequences.

As an example of how to place host-pathogen interactions into a canonical ecosystem context, consider a simple host-pathogen interaction with classic SI dynamics (e.g., Anderson and May 1981), in which the host is regulated entirely by the pathogen

$$\frac{dS}{dt} = (b-d)S - \beta SI + \delta I$$

$$\frac{dI}{dt} = \beta SI - (d'+\delta)I$$

Here, S is the density of healthy hosts, and I the density of infected hosts. Alternatively, if hosts have a given biomass, these equations could describe changes in biomass. Healthy hosts give birth at a per capita rate b, and die at a rate d. Infected hosts die at a rate d', do not give birth, and recover at a rate Ω. There is no permanent immunity, or lingering demographic consequence of having once been infected, following recovery. The disease transmission process is the usual mass action term, with transmission scaled by β. As long as the recovery rate is non-zero, the population will reach an equilibrial abundance. The equilibrial densities of healthy and infected hosts are respectively $S^* = (d'+\Omega)/\beta$, and $I^*=(b-d)(d'+\Omega)/\beta d'$.

A principal concern of ecosystem ecology is characterizing and interpreting the causes of flux rates among compartments. The total rate of production of biomass by the host population at equilibrium must equal the rate at which biomass enters other compartments in the ecosystem (e.g., the food web). Adding dS/dt and dI/dt, at equilibrium we have total deaths equal total production, or

$$dS^*+d'I^* = bS^* = b(d'+\Omega)/\beta.$$

Note that this measure of production does not depend upon the basic death rate of the host, when healthy, but instead depends upon the death rate of infected hosts. Given that the pathogen regulates host numbers, one ecosystem 'function' performed by that host (namely, its production) appears to be governed by the death rate of infected hosts. However, note that an alternative parameterization of the model is to write the death rate of infected hosts as the basic death rate of healthy hosts, plus a difference term, i.e. $d' = d + q$, where q measures the mortality effect of the pathogen. So the basic death rate of the host is not necessarily irrelevant, but environmental factors which may affect the death rates of healthy hosts but not the death rate of infected hosts will not alter the productivity of the population, and thus not change the flux of materials and energy it provides via deaths to other ecosystem compartments. This is not an implausible scenario. For instance, if deaths arise due to aggressive contest competition, and infected individuals avoid such aggressive encounters, spatial variation in the intensity of competition will not influence the death rate of infected individuals and so would not be expressed in ecosystem fluxes through the population.

This model of course does not directly consider space, but it does suggest some hypotheses regarding spatial affects that could be assessed in more complex models. For instance, if spatial dynamics tends to produce systems with overall lower transmission rates because of the spatial localization of interactions (see below), this should increase host population size, and thus enhance the particular ecosystem process of production.

If spatial dynamics leads to shifts in virulence (as measured by d'), then this will likewise alter the contribution of this host species to ecosystem productivity. Some models discussed below suggest that spatial dynamics can characteristically produce systems with lower levels of virulence (viz., lower d'). If so, then the total production of the host population in the ecosystem context will be *reduced*. This may seem counterintuitive. The reason for this is that with lower virulence, the host population will

equilibrate with fewer healthy hosts, and more infected hosts. In other words, with lower virulence the host carries a heavier load of parasites. We have assumed that infected individuals do not reproduce, and that the parasite is the sole factor regulating host numbers; hence, this decrease in virulence can shift individuals from productive to non-productive states, and so depress host population productivity.

Also, somewhat counter-intuitively, an increase in the recovery rate for individuals at the level of the population translates into an increase in total death rate (for all individuals). Any ecosystem factors that might influence recovery rates (e.g., the presence of bioaccumulated toxins) could thus indirectly alter population productivity and flux rates to other ecosystem compartments.

Finally (and to return to the spatial theme of this chapter), assume that the above model applies in each of a number of distinct habitats, which each reach their own respective demographic equilibria. The habitats differ in one or more parameters in a fixed manner (e.g., due to topographic, edaphic, or climatic factors, or because they are at different stages of plant succession). An expression for the average productivity per habitat is given by taking the expectation of the above expression, or $E[b(d'+\Omega)/\delta]$. If the parameters vary across space independently, then by using Jensen's inequality it is immediately apparent that the only parameter for which spatial variation affects the mean is the transmission rate. A host with a spatially varying transmission rate has a higher production, averaged over a landscape, than does a similar host with the same average transmission rate, but one which is spatially invariant.

For most of the remainder of this chapter, we will not directly consider ecosystem processes, but rather focus on the population and evolutionary dynamics of the host-pathogen system, and how this is influenced by space. Throughout, however, there is assumed to be an implicit link to ecosystem function, via impacts of pathogens on host abundance and stability. Moreover, even if this link is not of direct interest, ecosystem context (e.g., habitat productivity, patterns of spatial connectivity) can be of great importance in determining the population and evolutionary dynamics of host-parasite systems.

Spatial variability in empirical patterns of parasite distribution within ecosystems

Before reviewing models of spatial dynamics in host-parasite systems and their implications for the understanding of epidemiology and evolution, we present some empirical patterns that stress the role of space at various levels within ecosystems and highlight a series of factors that have been considered or need to be considered in theoretical studies.

Geographical distribution of parasites species: availability of hosts and opportunity for transmission.

Parasites need their host(s) to complete their life cycle, either as an important source of nutrients, e.g., for many ectoparasites like fleas, mosquitoes or ticks

parasitizing vertebrates, or also as a habitat to live and reproduce, e.g., for some helminthes and microparasites such as bacteria and viruses. The distribution of hosts in the environment will thus condition the distribution of their parasites. This constraint is especially strong as most parasites are specialised to a limited number of hosts and also some parasite life cycles are complex and involve series of hosts, with some playing the role of vectors or of intermediate hosts (Combes 2001). Before considering the factors affecting the spatial variability in the distribution of parasites within a given host population, a first step is thus to see how heterogeneity in the spatial distribution of parasite species within ecosystems relates to the spatial distribution of their hosts.

As most species are parasitized by several parasites, most of which are specialized to a given host species, the diversity of parasite fauna is spatially constrained, within and among ecosystems, by the diversity and ecology of their component species. The spatial distribution of parasites is also affected by the opportunities for completing their cycle which can be prevented by abiotic conditions outside their hosts. An example of this involves arctic ecosystems where an important component of the parasite fauna of seabirds are the flukes (Digena) and where a detailed study of such parasites compared their distribution between two intermediate host species s and among spatial locations (Galatkionov & Bustnes 1999). Different species of digeneans have life cycles which may consist of one intermediate host and no free-living larval stages, two intermediate hosts and one free-living stage, or two intermediate hosts and two free-living larval stages. The study examined the distribution of such parasites in the intertidal zones of the southern coast of the Barents Sea (northwestern Russia and northern Norway) by investigating two species of periwinkles (*Littorina saxatilis* and *L. obtusata*) which are intermediate hosts of many species of digeneans. A total of 26020 snails from 134 sampling stations were collected. The study area was divided into 5 regions, and the number of species, frequency of occurrence and prevalence of different digenean species and groups of species (depending on life cycle complexity) were compared among these regions, statistically controlling for environmental exposure. The authors found 14 species of digeneans, of which 13 have marine birds as final hosts. The number of species per sampling station increased westwards, and was higher on the Norwegian coast than on the Russian coast. The frequency of occurrence of digeneans with more than one intermediate host increased westwards, making up a larger proportion of the digeneans among infected snails. The prevalence of different species showed the same pattern, and significantly more snails of both species were infected with digeneans with complicated life cycles in the western regions. The authors concluded that the causes of changing species composition between regions are probably (1) the harsh climate in the eastern part of the study area reducing the probability of successful transmission of digeneans with complicated life cycles, and (2) the distribution of different final hosts (Galatkionov & Bustnes 1999).

The combined effect of spatial variability in host availability and abiotic conditions on levels of parasite infestation has also been addressed for other ecosystems. Ecosystems at tropical latitudes are well known for harbouring much higher number of animal and plant species than at higher latitudes (e.g., Rosenzweig 1995), and these areas are thus expected to harbour more parasite species. The picture is not that simple though. For instance, despite some evidence of higher parasite richness in marine fish

ectoparasites (Rohde & Heap 1998), field studies conducted on communities of endoparasites of freshwater fish as a function of latitude have reported lower richness in host species living in tropical areas than under higher latitudes (Choudhury & Dick 2000). This result holds even after controlling for potential confounding effects such as sampling effort, host body size and phylogenic relationships among host species (Poulin 2001). Geographical differences in the diet of related host species is likely to affect the richness of the endoparasite fauna whereas, latitudinal effects in ectoparasite richness may be more related to the abiotic characteristics of the environment in which the host species live (Rohde & Heap 1998). Other factors such as spatial variability in the seasonality in host reproduction and the biogeographic history and diversity of actual or potential host species also have to be considered.

The geographic range of a host and its specialist parasites may thus differ, with the geographic range of the host being usually larger than the one of the parasite (though interesting exceptions to this generalization may occur if parasites have widely ranging transmission stages in their life histories). Some parasite species with complex life histories and intermediate host species may actually show a much broader apparent geographic distribution that any one of their host species. In such cases the parasite geographic distribution may nevertheless be strongly constrained by one of the host species, as for instance when the transmission among final hosts need to be done through a species playing the role of a vector. Vertebrate species affected by blood parasites such as *Plasmodium* spp may carry the parasite all over the world in their body (e.g., during seasonal migration or business travel) but the transmission of the parasite to another final host is nevertheless constrained by the need for a local host species that will play the role of a competent vector (Kiple 1993). The type of life cycle will thus affect greatly the geographic distribution of a parasite and its ecological meaning: some microparasites which will be directly transmitted among host will be found everywhere the host is found in sufficient density, but some parasites with extensive free living stages or complex life cycles involving different host species may to the contrary have geographic distributions that do not match tightly the one of their host(s).

A low richness of parasite and host communities in some areas, e.g., at very high latitudes, does not mean that parasites are of negligible importance in these systems. For instance, an extremely high prevalence of infestation of the sibling vole *Microtus rossiaemeridionalis* by the taeniid tapeworm *Echinococcus multilocularis* has recently been reported in a geographically isolated and very small population of that small mammal in the Svalbard archipelago (Henttonen et al. 2001). The life cycle of the parasite involves the arctic fox *Alopex lagopus* as final host; long distance movements by foxes between Siberia and Svalbard, together with the human-mediated introduction of the vole to Svalbard, are likely responsible for the presence of the parasite in such a remote population of the intermediate host (Henttonen et al. 2001). *E. multilocularis* is the agent of a life threatening zoonosis. Thus, this example highlights the different roles that humans can play in a spatial context, sometime being inadvertently efficient at changing the spatial availability of hosts and facilitating the completion of life cycles. A better understanding of the role of space in the dynamics of host-parasite interactions can be gained by considering the processes responsible for the distribution of parasites among hosts at different scales.

Aggregation of parasites among hosts and spatial distribution of hosts and parasites

A striking and taxonomically widespread pattern is that the distribution of parasites among hosts within populations is typically aggregated, i.e. most host individuals have no parasites but a few hosts are infested by many parasites (Shaw & Dobson 1995). Another often reported pattern [when the spatial locations where the host individuals were sampled are known] is that the proportion (prevalence) of parasitized hosts varies among areas (Wilson et al. 2002). These observations are key to understanding the importance of spatial variability in host-parasite interactions within ecosystems. Analyses of spatial aggregation have mostly been for macroparasites (Hudson & Dobson 1995). Little explicit attention has been given to this in microparasites , the abundance of which are usually not quantified within individual hosts, and for which the reporting of prevalence is often linked with information on their rate of spread in the host population (see below). The existence of latent periods in infection, and asymptomatic infected host individuals, is however consistent with heterogeneity among host individuals in the abundance of pathogens within them. A concern with overdispersion and aggregation as defining attributes of the distribution of parasites among hosts is important, as this form of spatial heterogeneity has been identified as a key factor for the stability of the dynamics of host and parasite populations (Anderson & May 1978; Jaenike 1996).

Many factors can contribute to generate an aggregated distributionof parasites among hosts, including hosts with different histories of exposure of hosts to parasites, and differential susceptibility of host individuals to parasites. These factors may be structured in space at different scales, and this structuring may contribute to the aggregated distributions of parasites among hosts. Indeed, pooling of individuals from locations with different level of infestation into single combined analyses can generate an overall aggregated distribution. Classically, explicit information on the relative spatial location of the host individuals has seldom been considered in the analysis of aggregative distribution patterns. It is nevertheless interesting to measure aggregation at different spatial scales to attempt to identify the spatial scale at which the aggregative process is occurring (Boulinier et al. 1996). The tick *Ixodes uriae* has for instance been found aggregated among nestlings of their seabird host, the black-legged kittiwake (*Rissa tridactyla*), when all samples were pooled, but when the level of aggregation was quantified both within-nest among nestlings and among nests within an area, the ticks were found aggregated among nests but not among nestlings within nests (Boulinier et al. 1996). This pattern is not surprising, given the specific features of the system considered; the ectoparasite, which has limited mobility, infests the nesting substrate of the breeding colonies of its host and nestlings within each nest share traits in common likely to affect their level of tick infestation.

In this species, aggregation is evident among nests. This pattern may be due to a combined effect of the correlated age of the nestlings within each nest, some genetic basis for susceptibility to the ticks (Boulinier et al. 1997) and spatial heterogeneity in local exposure to the ticks which overwinter in the nesting substratum. Within a breeding cliff, spatial autocorrelation in the level of infestation of nestlings has for instance been reported (McCoy et al. 1999). A comparable approach to partitioning aggregation at different scales has been conducted with other systems (e.g., Elston et al. 1999;

Haukisalmi & Henttonen 1999; Poulin & Rate 2001; Latham & Poulin 2003**)** and often a minimal spatial scale is identified at which little aggregation is found (Jaenike 1994). In such cases, the identification of the spatial scale at which aggregation occurs helps to identify which processes are potentially important in the transmission of the parasites and the maintenance of infestation. It can also facilitate the identificaiton of environmental variables possibly responsible for the variable levels of local infestation, as is done in the field of landscape epidemiology. In landscape epidemiology, geographic information systems (GIS) combining field data with remotely collected data on climate and landscape attributes (Hess et al. 2002), and geostatistic modelling (e.g., Kitron et al. 1996; Diggle et al. 2002; Srividya et al. 2002) are being increasingly applied. When the spatial distributions studied are those of vectors for parasites, host aggregation is especially important to consider; the reason for this is that aggregative responses to vectors to hosts can define foci of transmission (e.g., Perkins et al. 2003).

It should further be noted that spatial structure in infestation levels is not static, because it arises from the interplay of the local population dynamics of hosts and parasites, and dispersal by each species, potentially at different scales. This can be especially evident when studies are conducted at different time intervals, permitting the temporal dynamics of the spatial distribution of infestation to beapprehended. The role of space in this context is highlighted in the study of the spatial dynamics of epidemics in ecological landscapes (see below).

Determinants of dispersal and hosts-parasites interactions

Dispersal is now recognised to be a major factor affecting the spatial dynamics of populations. This is especially likely for parasites, as hosts and groups of hosts can be considered as islands among which parasites must disperse, to persist. Dispersal can enable individual parasites (either within or outside of host bodies) to reach groups of host that are susceptible and/or uninfected. It will also lead to gene flow.

Dispersal can be linked with transmission, but dispersal and transmission can occur also at times occur independently. Dispersal is usually modelled as a rate that can vary with habitat, but factors directly related to the host-parasite interaction can be involved as well. For instance, dispersal of hosts could be affected by the local level of parasite infestation. In colonial birds, increased natal dispersal of cliff swallows (*Hirundo pyrrhonota*) (Brown & Brown 1992) and breeding dispersal in black-legged kittiwakes have been associated with higher level of ectoparasite infestation of nesting areas (Boulinier et al. 2001). Nevertheless, few studies have addressed the question of how dispersal rates and transmission rates and patterns are interrelated.

If parasite infestation can lead to host dispersal, movement of hosts can also be responsible for the dispersal of parasites. And indeed, the few studies that looked at the population genetic structure of parasites as a function of their host ecology have shown patterns suggesting that host movement is responsible for parasite dispersal (in large herbivores [Blouin et al. 1995], seabirds [McCoy et al. 2003] and salmons [Criscione & Blouin 2004]). Movement of hosts leading to parasite dispersal may not imply host dispersal. Making such a distinction is important as it will affect differently the spread of parasites, and thus epidemiology, but also the relative gene flow of host and parasites,

and thus the dynamics of host-parasite coevolution (see Holt and Hocberg 2002 and below).

Keeping in mind the potential need to incorporate these complex spatial processes when considering some specific host-parasite systems, simple modelling approaches can nevertheless capture the main properties of the dynamics of host-parasite interactions in a spatial context.

Host-parasite interactions in coupled, heterogeneous patches

One straightforward way to incorporate space into host-pathogen systems is to imagine that the environment is comprised a number of distinct habitats. In each of these, there is a well-mixed population of hosts and pathogens, described by standard epidemiological models. The habitats are then coupled by dispersal of hosts, pathogens, or both. Examples of authors who have explored such models include Post et al. (1983), Rodrigues and Torres-Sorando (2001), Hethcote and Ark (1987), Dieckmann et al. (1990), and Sattenspiel and Dietz (1995; Sattenspiel and Simon 1988). In general, the conditions for establishment of the parasite, and its equilibrial incidence can be strongly influenced by heterogeneity among habitats. Sattenspiel and Dietz (1995) provide an expression for establishment of an infectious disease in a spatially heterogeneous population, where total numbers are fixed by factors other than the disease (as appropriate for many infections of humans, for example). Rodriguez and Torres-Sorando (2001) develop comparable models for malarial infections of humans, and Diekmann et al. (1990) discuss the general issue of calculating the basic reproductive ratio R_0 in heterogeneous populations.

In principle, there are no conceptual complexities in this, but in practice it can be difficult to wade through the algebraic tangles which arise when analyzing models with multiple patches and non-uniform mixing. As a relatively simple example, we consider the problem of initial establishment of an infectious disease in a landscape consisting of just two distinct habitats (one with area A_1, the other with area A_2 ,coupled by host movement). In the absence of the disease, we assume that the host in each habitat equilibrates at a carrying capacity, that the disease has direct transmission, and that both healthy and infected hosts can move between habitats at constant per capita rates (though possibly at different rates in the two habitats). A model for this scenario which describes the initial stage of infection is given by duplicating the above SI model, with two pools of infected individuals (recall, for the moment we are assuming that the host population is initially all healthy hosts, with fixed densities). The model describing the initial stages of infection is

$$\frac{dI_1}{dt} = \beta_1 S_1 I_1 - (d_1' + \delta_1) I_1 - m_{12} I_1 + m_{21} I_2 (A_2 / A_1)$$

$$\frac{dI_2}{dt} = \beta_2 S_2 I_2 - (d_2' + \delta_2) I_2 - m_{21} I_2 + m_{12} I_1 (A_1 / A_2)$$

All the parameters in the above earlier *SI* model have now been made habitat-specific. In addition, we have assumed that infected individuals can move between habitats, at rates that are also potentially habitat-specific. (Healthy hosts may also be moving, but if so, they are assumed to do so in a manner that does not alter the pattern of abundances between habitats.) Because the variables are cast in terms of density, the fluxes between habitats have to be readjusted to account for the fact that the absolute number of individuals moving from habitat *i* to habitat *j* equals the density in habitat *i*, times the area of habitat *i*; whereas the impact this influx of individuals has on density in habitat *j*, has to be scaled against the area of habitat *j*.

The above model is a pair coupled linear differential equations, so it can be fully analyzed. In particular, the dominant eigenvalue of the characteristic matrix defines the growth rate of the infection over both habitats, after an initial transient phase. For simplicity, we combine infection, death, and recovery into a habitat-specific intrinsic growth rate for the infection, when rare:

$$r_i = \beta_i S_i - (d_i' + \delta_i)$$

A habitat may foster a high initial growth rate for the parasite simply because there is a high density of hosts there, or instead because of individual impacts of the infection upon hosts (e.g., locally low death rates of infected individuals). We assume that habitat 1 has the higher growth rate. With this notation, the growth rate of the infection can be shown to be

$$\frac{1}{2}\left[-m12 - m21 + r1 + r2 + \sqrt{(m12 + m21 - r1 - r2)^4 + 4\,m21\,r1 - m12\,r2 + r1\,r2} \right]$$

(see Holt 1985 for an analogous treatment of population increase in a two-habitat environment, albeit with symmetrical movement).

This expression can be manipulated to make some general statements about how habitat heterogeneity influences the establishment of a disease.

i. Note that the relative habitat areas drop out. The slightly counterintuitive result is that a combination of intrinsic habitat qualities influences invasion rates, but not relative habitat areas. The reason for this is basically that with reciprocal movement, the descendents of any given individual cycle through both habitats. Overall, the asymptotic growth rate reflects a nonlinear averaging of the growth rates of the two habitats.

ii. If we let both movement rates be equal, and then take the limit as they get very large in the above expression, the growth rate simply becomes the arithmetic average over the two habitats, $(r_1+r_2)/2$. In this limit, the landscape is actually just one habitat with internal heterogeneity. Given rapid movement of infected individuals, landscapes with

the same average growth rate (averaged among habitats) but different degrees of internal heterogeneity, nonetheless should have the same growth rate for the infection.

iii. If both intrinsic growth rates are positive, so is the overall growth rate; conversely, if both growth rates are negative, the overall growth is also negative. Parasite invasion requires that there be some habitats in the landscape which are intrinsically favorable, and could potentially sustain an invasion on their own (were infected individuals not to move).

iv. If we let m_{21} approach zero, while keeping the other movement rate fixed, all movements will be from habitat 1 into habitat 2. In this case, the growth rate overall converges on the growth rate of the better habitat (which we have assumed to be habitat 1). Thus, an infectious disease may grow in habitats where inherently it has an intrinsic growth rate less than zero, provided it is maintained in habitats where it has a positive growth rate. All else being equal, such 'spillover' modes of invasion by an infection should be most noticeable in habitats with lower than average host densities (this result emerges from inspecting the eigenvector describing the distribution of individuals between the two habitats, when the invasion has settled into its equilibrial rate of change).

The above bit of theory provides insight into how spatial heterogeneity can influence parasite establishment. We would be the first to admit that this provides just a first pass through this problem. A full analysis of this issue would require one to analyze more complex landscapes, alternative transmission dynamics, additional classes (e.g., hosts with acquired immunity) and so on. Moreover, we have not paid attention to feedbacks via depression of healthy host numbers, or to transient dynamics.

Some insights into the consequences of habitat heterogeneity for disease dynamics can be also gleaned from parallel studies of the classical Lotka-Volterra predator-prey model, in which prey grow exponentially in the absence of predation, predators die at a constant rate, and the two are coupled by a mass action term describing predator attacks. This familiar model emerges as a limiting case of the standard SI epidemiological model (Holt and Pickering 1985), when infected individuals have very low recovery and birth rates. Holt (1984) analyzed two habitat patches with Lotka-Volterra interactions occurring in each, and predator movement. With some simple reinterpretation of parameter definitions, this is also the SI host-parasite model for two habitats, with movement of infected individuals. (A number of authors have found general Lotka-Volterra models to provide useful limiting cases for explorations of host-parasite ecology and evolutionary dynamics, e.g. Frank 1997). Spatial heterogeneity is broadly stabilizing, because it permits source-sink populations to develop, in which some host species are more heavily exploited than expected from just local conditions alone.

When healthy hosts are also allowed to move (Nisbet et al. 1993), more complex scenarios are feasible in heterogeneous landscapes. The basic point is that in some circumstances spatial heterogeneity coupled with dispersal can lead to stability, and in others, it sometimes leads to instability (Holt 2002). Despite this potential for a diversity

of effects of spatial heterogeneity, our sense is that more often than not, spatial heterogeneity is broadly stabilizing (Hoopes et al., in review).

The above remarks pertain to heterogeneity that arises from variance in local conditions (e.g., productivity, or rates of infection). In other systems, heterogeneity in rates of movement or mixing can itself also promote stability (Holt 1984 notes this case for the Lotka-Volterra model). Gubbins and Gilligan (1997) carried out an experiment and demonstrated that heterogeneity in parasite establishment (due to incomplete mixing) promoted the persistence of the mycoparasite *Sporidesmium sclerotivorum*, a biological control agent on *Sclerotina minor* (a fungus on lettuce).

In heterogeneous landscape, flows among habitats can permit greater parasite loads to be maintained in some habitats, than would be expected just from local dynamics. Hochberg and Ives (1999) show that if there is substantial spatial variation in host productivity, flows of natural enemies (e.g., pathogen transmission stages among habitats) can lead to restriction of species from particular habitats, and even define the edges of geographical ranges for hosts. The model of Holt (1984; see also Holt 2002) mentioned above shows that 'spillover' limitation of a host in a low productivity habitat can readily arise, if this habitat is juxtaposed with a high productivity habitat. This can be viewed as a kind of apparent competition, linking the dynamics of host populations that live in different habitats. Hosts that occupy low productivity habitats are vulnerable to the impact of parasites maintained in more productive habitats. This is true, regardless of whether the populations are the same biological species of host, or different host species. In the latter case, one may observe indirect exclusion of one host by another via shared parasitism (Holt and Pickering 1987), even though the two hosts never cooccur in the same habitat patch (a specific metapopulation model of this effect is in Holt 1997). All of these landscape effects depend on the movement of parasites across space, either because infected hosts themselves move, or because the parasites have free-living, mobile life history stages (e.g. aerial spores), or because movement is provided by the behavior of vectors.

Epidemics in a spatial context

One broad class of examples of spatial host-parasite interactions, to which we will not attempt to do real justice, is the study of epidemic waves across space (e.g., of dengue fever emanating from foci in Thailand, Cummings et al. 2004). The theoretical models which have most often been used in this context are partial differential equations to study invasive waves of epidemic disease. Just to mention one interesting example, Murray et al. (2003) explored a spatially distributed mass mortality event in an Australian pilchard (*Sardinops sagax*) population. Their model tracked susceptible, infected and latent, infected and infectious, and finally removed (dead or recovered) individuals. As in most such models, the wave velocity is sensitive to diffusion coefficients, viral transmission rates (which enter into local intrinsic growth rates, see above), and latency period. Large-scale spatial hetereogeneity in these parameters can help explain differences among regions in the time course, spatial development, and intensity of the epidemic.

The broad ecosystem consequence of these epidemic waves is that they act as major disturbances in the ecosystem, with potential ripple effects on many other species. An example of such a perturbation is provided by the chestnut blight fungus (*Cryphonetria parasitica*), which decimated the American chestnut (*Castanea dentata*) throughout the eastern deciduous biome of the U.S. in the early decades of the 20th century. This species was once the dominant tree in this biome, but the fungus destroyed approximately 3.5 billion trees (Taylor 2002). Other species of tree have filled in the gaps left by the demise of this species. Its wood is resistant to decay, and there are still many places in the southern Appalachians where chestnut logs are prominent features of the understory (personal observation). Although the ecosystem consequences of this epidemic (say on soil properties) are not well-documented, they are doubtless profound and long-lasting in these systems.

Effects of space in homogeneous environments

Spatial dynamics can influence stability and persistence of host-parasite systems, even in the absence of heterogeneity. For instance, Jansen and de Roos (2000) analyzed the Lotka-Volterra model for two coupled habitats, with uniform predator movement, and no parameter differences between the two habitats. Ultimately, these systems settle into spatially uniform, neutrally stable oscillations. In a single patch, these oscillations can be arbitrarily large. But the transient dynamics (which may be very long) in the two patch model can be very different. In particular, Jansen and de Roos show that in the long run, fluctuations of large amplitude will not be observed, for nearly all inhomogeneous starting conditions.

Such effects become greatly amplified when one pays due attention to the discreteness of individuals. Because interactions are spatially localized, and occur between individuals who experience the chance vicissitudes of birth, death, and movements, even in a homogeneous environment stochastic variation alone can lead to a shifting pattern of heterogeneity in host-pathogen interactions. This basic insight underlies a vast array of recent studies of space in ecological systems (Tilman and Kareiva 1997). Space becomes particularly important when infection occurs only over short distances among individuals who themselves do not move (e.g., plant populations). Keeling et al. (2000) provide a general argument on how limited movement in natural-enemy victim systems generically leads to spatial structure, which in effect provides refuges for the host/prey, and generates exploitative competition among parasites/predators.

Introducing demographic stochasticity creates many challenging mathematical problems, but also a consideration of demographic stochasticity points to some important potential implications of spatially localized infection processes. Rand et al. (1995) and Keeling (1999, 2000) considered a system in which a virulent disease is spreading through a slowly growing, sessile population (e.g., a fungal pathogen on a plant population). The model is a probabilistic cellular automata model for a host-parasite system, which attempts to capture in a simple way the consequences of localized infection and host renewal processes. The system consists of a lattice of sites, each of

which can be empty; or, occupied by a healthy host; or, occupied by a parasitized host. What happens at each site depends upon its current state and that of its immediate neighbors. Healthy hosts send offspring into empty adjacent sites (e.g., a plant sending out seeds over a short distance); if healthy hosts are next to infected hosts, they can be infected, with a fixed probability. For simplicity, Keeling assumes that infection is lethal.

This model suggests a number of important messages that appear to characterize a much broader range of models.

First, there is a range of transmissibilities, within which the pathogen persists, and outside of which it cannot. If transmission rates are too low, then the 'birth' rate of new infections will not exceed the rate at which infections are lost to mortality. This of course describes nonspatial infection dynamics, too. More interestingly, if transmission rates are too high, then persistence may also be unlikely. The reason for this is that the pathogen in effect overexploits its hosts in localized arenas, and then itself is vulnerable to extinction. The interaction between the host and pathogen leads to a fracturing of both populations into small isolated patches; the pathogen can then easily disappear locally due to demographic stochasticity.

Second, given that the interaction persists, it can do so at very low levels of overall prevalence, compared to expectations drawn from homogeneous, mean-field models, in effect because the localization of interactions and dispersal permits the emergence of ephemeral transient refuges. In a sense, the localization of interactions can be viewed as a reduction in the overall rate of infection, per host, so that the impact of the parasite upon the population dynamics of its host is reduced.

Third, because the system is probabilistic and tracks integer numbers of individuals, the local environment is often found in a state which is very far from the global average, and there are dramatic fluctuations in infection at a local level.

Fourth, there is an emergent spatial structure, in which parasites spread as wavefronts through the susceptible hosts, with patches empty of hosts left behind in the wake of the wave.

Many of these results appear in a wide range of models. Haraguchi and Sasaki (2000) examined how spatial structure influenced the evolution of virulence and transmission rate in a parasite, interacting with a host in a lattice. The host was assumed to not be evolving. Constraining transmission so that it only occurs through local contact leads to evolutionariliy stable traits of parasites that are completely different than expected with complete mixing. Viscosity tends to select for an intermediate ESS rate of transmission, even without the classical tradeoff between transmission and virulence. They found an interaction between the host growth rate, and parasite evolution; at low host growth rates, the parasite had difficulty persisting near its ESS, whereas at high host growth rates, the parasite could over-exploit its host, with both risking extinction. This is an evolutionarily-driven analogue of the classical 'paradox of enrichment'. Analysis of a similar system by Rauch et al. (2003) using techniques which tracked genetic

phylogenies revealed some interesting features. Mutant strains continually arise with higher transmission and virulence. However, these strains, after a period of growth, deplete hosts within regions, and then themselves go extinct. This leaves behind pathogens with intermediate virulence. Thus, the evolution of the whole system reflects a self-organized spatial structure (which we might note amounts to a kind of group selection).

If these results prove to be general, they obviously have important consequences for ecosystems. For instance, the spatial localization of interactions may mean that some pathogens may be less important factors regulating population size (and thus exert a relatively weak influence on biomass production, nutrient pool fluxes, and so on) than expected judging from the direct impact of the pathogen upon individual hosts. Such ecosystem effects, moreover, may be highly heterogeneous across space, and through time.

Properties of the ecosystem may in turn feed back on the host-pathogen interactions observed. One general finding in lattice models (which is usually treated as an inconvenience by theoreticians) is that the size of the lattice can influence the probability of persistence of the infection. There are two distinct reasons for this. First, there is often a characteristic length scale describing the correlation among nearby cells, reflecting the emergence of spatial asynchrony in dynamics. A lattice that is smaller than this characteristic scale will not contain sufficient spatial heterogeneity among patches in the phase of the host-parasite interaction to persist. Second, in models which are stochastic (e.g., individual-based models), the probability of randomly fluctuating to extinction over a given time frame goes up rapidly as the maximal number of individuals declines (a result which in general ecology goes back at least to MacArthur and Wilson 1967). This is closely related to the concept of a 'critical community size' in epidemiology, which is defined as the minimum size of a population required for a disease to persist (Bartlett 1957). Wilson et al. (1998) explored a tritrophic host-parasitoid-hyperparasitoid interaction. Within each cell in a lattice, interactions tended to be unstable, and dispersal occurred among adjacent cells. There was a very strong lattice size effect on the persistence of the system, and the full tritrophic interaction required a much larger lattice to persist, than did the host-parasitoid interaction along. It is often difficult to gauge the critical size theoretically (Dye et al. 1995), but it is clear that ecosystem size is an important ecosystem factor which can influence the character of the host-parasite interactions one might observe in natural systems. We suspect that characterizing the effects of ecosystem size on host-parasite systems is a topic that will receive much more attention in the future (for related thoughts on how ecosystem size governs food web attributes, see Holt et al. 1999, Post 2000, and Holt and Hoopes, ms.).

The generalization that the spatial localization of interactions may quite broadly facilitate the persistence of parasites in ecosystems emerges in many situations. For instance, Grenfell et al. (1995, see also Keeling 1997) modeled the dynamics of measles. Here, the patches are cities or large towns, and spatial coupling reflects traffic among towns. With 10 such identical cities, a very weak amount of coupling (0.1% individuals moving) was shown to increase the persistence of the disease overall. The reduction in

extinction rate provided by spatial localization of interactions in large measure reflects the rescue effect (Brown and Astrid-Brown 1977). The decorrelation of dynamics among different sites permits some populations to be large, even when others are quite rare; the former can then provide immigrants which boost numbers in the latter, preventing local extinctions. This effect tends to increase when spatial localization is assumed (rather than weak global mixing among patches), as well as when the model explicitly considers birth, death, and movement at the level of individuals (demographic stochasticity). Both factors tend to decrease the correlation among sites in population dynamics.

In the real world, spatial localization of interactions may make it more difficult for many pathogens to persist. Phocine distemper in the harbour seal (*Phoca vitulina*) in the North Sea provides a potential example. This species is distributed in well-defined local populations, separated by unsuitable habitat. Colonies can go extinct and then become re-established. From the point of view of the virus, each group of seals is a patch in a metapopulation. Swinton et al. (1998) parameterized a model, so as to analyze conditions for persistence. They concluded that a very large population, indeed one larger than the entire population of seals in the North Sea, was required to maintain the disease. The reason is that within each local population, there is a rapid fadeout of the disease, followed by a slow entry of new susceptibles via birth. This suggests that even larger spatial scales must be considered if one is to understand the origin and maintenance of this disease.

A consideration of extinctions and patchy populations leads naturally to the theme of metapopulation perspectives on host-parasite dynamics. Often, local disease dynamics seem to imply that extinction is expected (e.g., due to 'fade-out'), but persistence actually occurs. Analyses of plant-pathogen interactions at landscape scales can reveal considerable stochasticity, suggesting the importance of recurrent colonization and extinction events (Burdon and Thrall 2001). Often, host-pathogen systems exist in ecosystems where there are other drivers that determine local extinctions (e.g., episodic disturbances). In this case, metapopulatoin perspectives should be particularly useful.

This observation suggests that the pattern of connectivity may be critical in governing the importance of parasites in ecosytems. In conservation biology, for many years there has been a concern with how fragmentation reduces connectivity and thus may foster the erosion of biodiversity (ranging from the loss of genetic diversity within species, to extinctions of entire clades of extinction-prone species). This in turn has led to considerable attention given to the potential value of corridors linking habitat patches. Hess (1994, 1996a,b) pointed out that there was a dark dimension to corridors, namely that they might promote the spread of infectious diseases, which could in turn reduce the conservation value of the habitat patches themselves. He developed metapopulation models to explore this idea. These models suggest that if a disease is highly contagious, and moderately severe (in terms of enhanced local extinction risks), a disease could become widespread in strongly connected landscapes, thus increasing the probability that the host would go extinct.

One metapopulation model considered by Hess (1996a) has the following simple form:

$dS/dt = mS(1-I-S)-eS -mpIS$, $dI/dt = mI(1-I-S)-e'I+mpIS$.

Here, S is the fraction of patches occupied by disease-free populations, and I is the fraction containing the disease. The fraction of patches that are empty is $1-I-S$. This model assumes that empty patches are equally likely to be colonized from either healthy or infected patches, and that cross-infection (scaled by p) can occur leading to the conversion of healthy into infected patches.

In the absence of the disease, the model reduces to the familiar Levins formulation, with an equilibrial occupancy of S* = 1-e/m. It should be noted that formally, this model is an example of 'intraguild predation' (Holt and Polis 1997). Healthy and infected patches both compete for empty patches, and in addition healthy patches can be exploited by infected patches. There is a general tendency for the top predator to exclude the intermediate predator in intraguild predation; in this model, the infection can dominate the population if the extinction rate of infected patches is not elevated too much.

A simpler version of the model is to assume that infected patches can only infect healthy patches. In this case, the model becomes identical to a special case of a predator-prey metapopulation model considered in Holt (1997), where a model is sketched for two host species occupying distinct habitats, but sharing a parasite that both increases local extinction rates, and can colonize across habitats. Holt (1997) demonstrates that indirect competitive exclusion can occur in this system. The species which occupies the rarer habitat is particularly vulnerable to exclusion.

Spatial dimensions of host-parasite evolution

There is a rich and rapidly growing literature on the implications of space for genetic and evolutionary aspects of host-pathogen interactions (e.g., Hochberg and Holt 2002, and references cited therein). Although of considerable intrinsic interest, it should be cautioned that many evolutionary studies may not actually directly bear on ecosystem processes. If all pathogens do is alter relative fitnesses of individuals within host species, without any overall impact upon population size, turnover, or stability, it is not clear that these studies have direct implications for ecosystem processes. For instance, in theories of sexual selection in which mate choice is based upon parasite load, an assumption is often made of 'soft selection', in which parasites and hosts reciprocally determine fitnesses in each other, but parasites do not directly affect host population size. The most useful class of models for the purposes of ecosystem ecology are those which simultaneously examine population dynamics and evolutionary genetic processes, and in particular those which elucidate the interplay of ecological and evolutionary phenomena. In the next few paragraphs, we discuss some major themes in host-parasite coevolutionary dynamics. Essentially none of the literature we consider is directly concerned with the ecosystem implication of these dynamics.

There is considerable evidence for spatial variability in adaptation in host-parasite systems (a very useful review is by Dybdahl and Storfer 2003). Local adaptation may be defined as occurring when the mean fitness of a population when measured in sympatry is greater than in allopatry (Gandon and Michalakis 2002); operationally, one can carry out a series of cross-population infectivity studies, and assess relative performance of parasites and hosts, when paired with the population they normally encounter, compared with 'foreign' populations. There is a remarkable variety of patterns that have been reported in empirical studies of host-parasite coadaptation.

One popular theory of host-parasite coevolution leads to the expectation that parasites should be locally adapted to their hosts (with greater replication rates and prevalence in sympatric hosts, to which they have evolved) than to allopatric hosts (to which they have not evolved). This could arise for instance because parasites often have large effective population sizes and short generation lengths, relative to their hosts, and so should be able to track slow shifts in the genetic composition of local host populations (Dybdahl and Lively 1998, Lively 1999). There are some excellent examples of local adaptation by parasites to their hosts (e.g., Morand et al. 1996). Thrall and Burdon (2003) for instance show that virulent pathogens dominate in host populations with resistant hosts, whereas avirulent pathogens characterize host populations with more susceptible hosts.

But in other systems, exactly the opposite patterns are found. For instance, Altizer (2001) examined variation among geographical races of the Monarch butterfly and its protozoan parasite *Ophryocystis elektroscirrha*. The prevalence of this parasite varies dramatically among populations, as does host resistance and parasite virulence. The migratory populations tend to have higher resistance and experience lower virulence. The parasite is not in this case more infectious to their native hosts, and indeed may be more maladapted. Altizer proposes that this pattern is due to selection being strong in the migratory population (where small parasite loads could translate to large fitness disadvantages, due to the energetic requirements of migration), and to correlated shifts in the relative importance of horizontal and vertical transmission routes. Another potential cause is that migratory populations may experience more effective gene flow, and so tend to have the genetic variation needed to mount strong adaptive responses to parasitism.

Theoretical studies of host-parasite coevoluton suggest that which patterns are observed depends on a number of factors, and in particular on the relative rates of dispersal of the interacting species, and the presence of spatial differences in patch quality (Gandon et al. 1996, Gandon 2002, Nuismer et al. 1999, Gomulkiewicz et al. 2000, Nuismer et al. 2000). Lively (1999) also notes that the pattern one observes is likely to shift with time; because the systems are expected to be dynamic at both a local and global level, local adaptation at any given site, for either species, is likely to wax and wane with time.

As with analyses of spatial effects on host-parasite ecology, it is useful to distinguish scenarios in which space solely matters because interactions and dispersal are

localized, and those in which there exists spatial heterogeneity in extrinsic environmental factors. Biotic and abiotic factors are rarely uniform across a species' range, and such variation has implications for the strength and even direction of selection (Thompson 1994, 1999). Thompson (1994) refers to sites where each of a pair of species has strong, reciprocal effects upon each others' fitnesses as coevolutionary 'hotspots'. Typically, for a variety of reasons such hotspots will be embedded in landscapes with many coevolutionary 'coldspots', where just one species responds to the other, or evolution is locally decoupled (Thompson 1994, 1999). Environmental gradients in climate or resource availability may for instance account for spatial variance in the virulence of parasitoids of *Drosophila melanogaster* (Kraiijeveld and van Alphen 1995). Patterns of local adaptation can be strongly influenced by the spatial mixture of hot and cold spots (Gomulkiewicz et al. 2000).

The primary ecosystem driver of environmental productivity in particular has been identified as a factor that indirectly governs the strength of coevolution between hosts and pathogens (Hochberg and van Baalen 1998, Hochberg and Holt 2002), leading to the prediction that virulence should decline to lower levels when productivity is lower. The mechanistic reason for this is that in host-pathogen systems (as is true for resource-consumer interactions in general), an increase in local productivity indirectly increases the abundance of the pathogen. This automatically increases the strength of selection on the host via selection to resist attack rates. In the simple *SI* model discussed above, note that as host intrinsic growth rate increases (*b*-*d*), which should be facilitated by increased productivity, the relative number of hosts that are infected, versus healthy, will increase. This in turn implies that the strength of selection on withstanding the infection (e.g., by recovering) will increase, as gauged against potential costs of such defense for reproduction by healthy hosts. In systems with top-down regulation of hosts by parasites, increases in productivity also tends to reduce the strength of density-dependence. This makes the relative advantage of defense against parasitism increase, and so indirectly also increases the likelihood of an evolutionary response.

Depending upon the details, these effects on the host can in turn alter evolution in the parasite. In some (though not all) situations, selection on the parasite to overcome host resistance will increase. Moreover, if the increase in host productivity translates into greater parasite numbers, more genetic variation should become available via mutation, upon which selection can then act. Thus, high productivity sites should be coevolutionary 'hotspots'.

Patterns of dispersal should also include coevolution between hosts and parasites. Complex patterns of local maladaptation and adaptation may arise, because of the mixing of traits among populations that are pulled in different evolutionary directions (Thompson et al. 2002). The idea that gene flow can hamper local selection is a familiar, old idea in evolutionary biology. A countervailing factor is that gene flow permits an infusion of genetic variation, providing the raw material for evolution by natural selection (Gomulkiewicz et al. 1999, Holt et al. 2003). The degree of local adaptation should strongly depend on the magnitude of dispersal relative to the strength of selection. It is an

open question whether mis-matching, or matching, of coevolved traits is the typical condition of natural systems.

Dispersal should interact with productivity. If there is weak dispersal, then in effect sites with different conditions provide distinct, largely closed arenas for adaptive evolution. Moreover the only sites where the species will be present will be those where they can sustain viable populations. Small amounts of dispersal in this case may mainly matter as providing avenues for the infusion of useful genetic variation; in any case, dispersal is not likely to create local maladaptation, if it is sufficiently weak. Indeed, in this case immigration tends to corrode local adaptation. However, if the population is unable to persist without immigration (i.e., the habitat is a sink), dispersal will tend to enhance the initial stages of adaptation to the environment (Gomulkiewicz et al. 1999, Holt et al. 2003). Whether or not it does so may depend upon the quantitative magnitude of mutational effects upon fitness (Kawecki and Holt 2002), and the impact of dispersal upon fitness (given intraspecific density dependence, Gomulkiewicz et al. 1999). In severe sinks, it may be difficult for evolutionary responses to occur at all (Holt and Hochberg 2002). In heterogeneous environments, dispersal is likely to be asymmetric between environments varying in productivity. Environments with low productivity and population sizes are likely to be net recipients, rather than sources, of immigrants. All else being equal, these are also potentially sites where a host, or pathogen, can be maladapted (Holt and Hochberg 2002; Nuismer et al. 2003).

Ecosystem drivers of host-parasite interactions: from parasitism to mutualism

Because parasites live intimately in the bodies of their hosts, there is the potential for selection to favor avirulence, or even the transformation of a parasitic association into a mutualism. Different environmental conditions can shift the balance between mutualisms and antagonistic interactions (e.g., Herre et al. 1999). Hochberg et al. (2000) explored how this transition from negative to positive interactions might be modulated by demographic differences among locations. They found that virulence tended to emerge most often in habitats where host population growth was highest. The reason for this was two-fold. First, total host numbers could be higher there, so if virulence was associated with transmission, it could be selected. Moreover, there was greater opportunity for cross-infection among strains, so that virulent strains could replace avirulent strains within hosts. Conversely, when host populations were unproductive, as in marginal habitats, avirulence and mutualisms could be favored.

This relationship between levels of productivity and the shift between parasitism and mutualism has potentially significant implications for ecosystem processes. Many mutualisms are associated with resource acquisition (e.g., nitrogen fixation in plants). If parasitic associations are labile, and tend to evolve towards mutualism in unproductive environments, this provides a kind of buffering in terms of ecosystem productivity.

On the topic of maladaptation

One important implication of spatial flows is that moderate (and at times severe) degrees of maladaptation are to be expected. Thompson et al. (2002) discuss in particular how geographical structuring in coevolutionary systems can lead to a substantial incidence of maladaptation.

There are several basic reasons to expect maladaptation in geographically structured coevolving systems.

1. Change leads to evolutionary lags. When the selective environment changes, there will usually be an evolutionary lag before a focal species settles into a new adaptive equilibrium. The length of time of the lag depends upon a variety of factors, such as the degree to which adaptation depends upon standing variation available at the time of the environmental change, or instead upon novel variation generated by mutation. Such lags are expected even to changes in the physical environment. Host-parasite interactions can exhibit dynamical instability in the selective environments faced by one or both species, either because of fluctuations in population size, or because evolution in one species in effect amounts to a deterioration in the environment for the other species.

2. Gene flow perturbs local adaptation. Another is that if there are fixed differences among populations, such differences could lead to differences in fixed local outcomes (Hochberg and Holt 2002). In this case, movement among populations can displace species from their local adaptive optima, because of the interplay of gene flow and selection.

3. Gene flow provides adaptive genetic variation. Adaptation by natural selection depends upon genetic variation, the migrational input of variation can permit one species to evolve more rapidly or effectively than another. Mathematical models of coevolution with migration at different rates for the interacting species reveal an interesting pattern. Gandon et al. (1996) predicted that if parasites migrate much more than do hosts, the parasites should be locally adapted. Conversely, if hosts migrate more than do parasites, hosts may be better adapted. The latter can arise for instance if the dispersing stage of the life history is different than the one harboring the parasite. Oppliger et al. (1999) tested this prediction with a lacertid lizard (Gallotia gallota) from the Canaries, and a haemogregarine blood parasite. Juveniles appear to be parasite free, and are the life stage when dispersal occurs. Cross-infection experiments revealed that parasites performed better on hosts from allopatric populations, revealing that the parasites are maladapted to the hosts with which they live.

4. Hosts and parasites have different spatial ranges. Another explanation has to do with the different geographical arenas for hosts and parasites. Nuismer et al. (2003) have recently explored implications of the general pattern that hosts and parasites do not typically have completely congruent geographical ranges. Their model suggests that spatial zones of maladaptation in one or both species are to be expected in spatially distributed host-parasite systems.

As we noted earlier, at the ecosystem level, these emergent patterns of adaptation and maladaptation will be reflected in overall rates of death for hosts, which in turn should alter the ecosystem fluxes associated with particular host species. Spatial dynamics could have an important and underappreciated impact on ecosystem processes, mediated through the realized death rates of hosts across spatially heterogeneous landscapes.

Global change, ecosystems and parasitism: the spatial dimension

Most components of global change involve a spatial dimension and this could result in parasitism playing an especially important role via these spatial dimensions. Increased fragmentation due to human activities can affect dispersal of host and parasites, and thus influence the persistence of their interactions at different scales, as well as the emergence of new diseases. Climate change should lead to geographic change in species ranges and habitat productivity which, as seen above, are critical for the dynamics of host-parasite interactions. Finally, increasing rates of introduction of species to foreign ecosystems can also result in dramatic shifts in the role of parasitism, either directly via the introduction of parasitic species, but also more indirectly by the introduction of potential hosts for parasites. The spatial dimension of the role of parasitism in ecosystem should thus become even more relevant in future attempt to understand and predict the ecological effects of global change.

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