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REPORT

Parasite establishment in host communities

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Abstract

Many pathogens and parasites attack multiple host species, so their ability to invade a host community can depend on host community composition. We present a graphical isocline framework for studying disease establishment in systems with two host species, based on treating host species as resources. The isocline approach provides a natural generalization to multi-host systems of two related concepts in disease ecology – the basic reproductive rate of a parasite, and threshold host density. Qualitative isocline shape characterizes the threshold community configurations that permit parasite establishment. In general, isocline shape reflects the relative forces of inter- and intraspecific transmission of shared parasites. We discuss the qualitative implications of parasite isocline shape for issues of mounting concern in conservation ecology.

Keywords

Host–parasite interaction, indirect effects, infectious disease, isocline analysis, multiple host species, parasite invasion.

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INTRODUCTION

Recent years have seen an enormous increase in our understanding of the role of parasites and pathogens in regulating host populations (Grenfell & Dobson 1995; Dobson & Foufopoulos 2001) and structuring ecological communities (Anderson & May 1986; Dobson & Hudson 1986; Hudson & Greenman 1998). Yet, the theoretical framework developed to explore the population dynamics of host–parasite interactions has to date dealt mostly with one-host, one-parasite interactions (Anderson & May 1991). This is a sensible starting point, but single parasite species often cross-infect multiple host species (Woolhouse *et al.* 2001; Power & Flecker 2003). For instance, Williams & Jones (1994) reviewed the specificity of fish parasites and found that more than 50% utilize greater than one host species at the definitive or intermediate life cycle stages; some parasite species use a very large number of intermediate host species. Understanding the dynamics of multi-host–parasite systems is important in conservation, given the significant impact generalist pathogens can have on endangered species, the increasing transport of infectious agents via human activities, and the effects of ecological community structure on the risk to humans of vector-borne and zoonotic diseases (Ostfeld & Keesing 2000; Schmidt & Ostfeld 2001).

Several authors (e.g. Holt & Pickering 1985; Dobson 1990; Hochberg & Holt 1990; Bowers & Begon 1991; Begon *et al.* 1992; Begon & Bowers 1993; Yan 1996; Greenman & Hudson 1997, 1999, 2000) have begun to examine the dynamics of multispecies host–parasite assemblages. For the most part, these studies have closely analysed particular models of host–parasite dynamics. In such studies, general insights can at times become obscured in a welter of algebraic detail. Our goal here is to begin to develop a broad conceptual framework for analysing multiple-species host–parasite systems. We make a first step towards this goal by generalizing two central concepts in the population biology of infectious diseases – namely, the basic reproductive rate, R_0, and threshold host population size, N_T (Anderson 1981) – to systems with two host species. Our own prior work on multi-host

systems (e.g. Holt & Pickering 1985; Dobson 1990; Begon *et al.* 1992) has focused on indirect competitive interactions among hosts that arise from shared parasitism. But the existence of indirect interactions via shared parasitism requires that parasite establishment be successful in the first place. We present a simple graphical approach to analysing parasite establishment in host communities, complementing the recent development of matrix transmission models to describe pathogen establishment in multi-host systems (Dobson & Foufopoulos 2001).

Two core concepts in parasite population biology

(i) Basic reproductive rate

The quantity R_0 measures reproductive success for a parasite in a given host population. For microparasites (viruses, bacteria, and protozoa), the basic reproductive rate is the expected number of secondary cases of an infection produced by an infectious individual, introduced into a population with a defined density of susceptibles (Anderson 1981). For macroparasites, R_0 is the average number of mature female offspring produced during the lifetime of a mature female parasite (Anderson 1981). The general approach we present is broadly applicable to any host–parasite system.

Consider the following simple model for the spread of an infectious disease (Anderson & May 1981). Let S and I, respectively, be the density of susceptible and infected hosts. Assume the rate of production of new infections is given by the mass action term, βSI, where β is the transmission coefficient, and that loss of infectives is ΓI, where Γ measures the rate of depletion of the pool of infected individuals through death or recovery. The net rate of change of infectives is

$$dI/dt = \beta SI - \Gamma I \qquad (1)$$

Equation 1 would normally be embedded in a set of equations tracking changes in the numbers of susceptibles (and other classes) because of infection, mortality, etc. For the moment, we assume host density is fixed and focus on parasite establishment.

The instantaneous per capita rate of increase of the infection is $r = \beta S - \Gamma$; if S is fixed over an interval T, the finite rate of increase of the infection during T is $\exp(rT) = \exp[(\beta S - \Gamma)T]$. Because the expected length of time an individual remains infected is $1/\Gamma$, we substitute for T to find the finite reproductive rate, $R_0 = \exp[(\beta S/\Gamma) - 1]$. Establishment of infection requires $R_0 > 1$, or equivalently $r > 0$.

(ii) Threshold host population size

In model (1), the per capita rate of increase of the infection increases linearly with S, implying a threshold host density. Let $N = S + I$ be total host population, and N_T the host density required for successful parasite invasion when all hosts are susceptible. Parasite invasion occurs only if $r > 0$. In the initial stages of invasion I is small, so $N \sim S$ and the host density at which the infection neither increases nor decreases is $N_T = \Gamma/\beta$. As discussed in Anderson & May (1991) and elsewhere, the deterministic criterion that $N > N_T$ is a necessary criterion for long-term persistence of the parasite in the host population.

A graphical model of conditions for parasite invasion

The above criterion for parasite invasion generalizes to a wide range of host–pathogen models as follows: (i) the instantaneous growth rate of the infection when that infection is rare, $r(S)$, is an increasing function of susceptible host density ($dr/dS > 0$); (ii) the infection dies out without susceptible hosts [$r(0) < 0$]; (iii) for some $S = N_T$, $r(N_T) = 0$. With these assumptions, parasite establishment fails if $S < N_T$. If K denotes long-term average host abundance without the parasite (i.e. host carrying capacity), the invasion condition of a specialist parasite is just $K > N_T$.

This characterization of host threshold densities generalizes naturally to multi-host systems. Rather than a threshold host density, we now search for threshold community configurations. For simplicity, consider just two host species. Let S_i denote the density of susceptibles in host species i. Assume that a bivariate function $r(S_1, S_2)$ describes the rate of growth of the infection in an environment with fixed host densities. In general, we expect that $r < 0$ when both hosts are scarce, and $r > 0$ when at least one host is abundant. Let N_{iT} be the threshold abundance for host species i, when alone [i.e. $r(N_{1T}, 0) = 0$, and $r(0, N_{2T}) = 0$].

With both hosts, there should be a set of combined host species densities separating community states permitting parasite invasion, from those precluding invasion. These threshold configurations are given by $r(S_1,S_2) = 0$, describing a curve in a phase space with axes corresponding to the densities of each host. This curve is the zero net-growth isocline (ZNGI) for a parasite. Isoclines are familiar conceptual tools in community ecology, particularly in theoretical treatments of resource–consumer interactions (e.g. Rosenzweig & MacArthur 1963; MacArthur 1972; Holt 1977; Tilman 1982; Chase & Leibold 2003). Hosts can be viewed as resources exploited by a particular kind of consumer, namely parasites. At a broad, qualitative level, different isocline shapes for invading parasites should reflect different epidemiological scenarios. These parallel Tilman's (1982) well-known classification of resource–consumer isoclines (Fig. 1).

Case 1. Non-interactive hosts (Fig. 1a). With no cross-host infections the parasite's zero growth isocline is rectangular. Parasite invasion in this limiting case requires

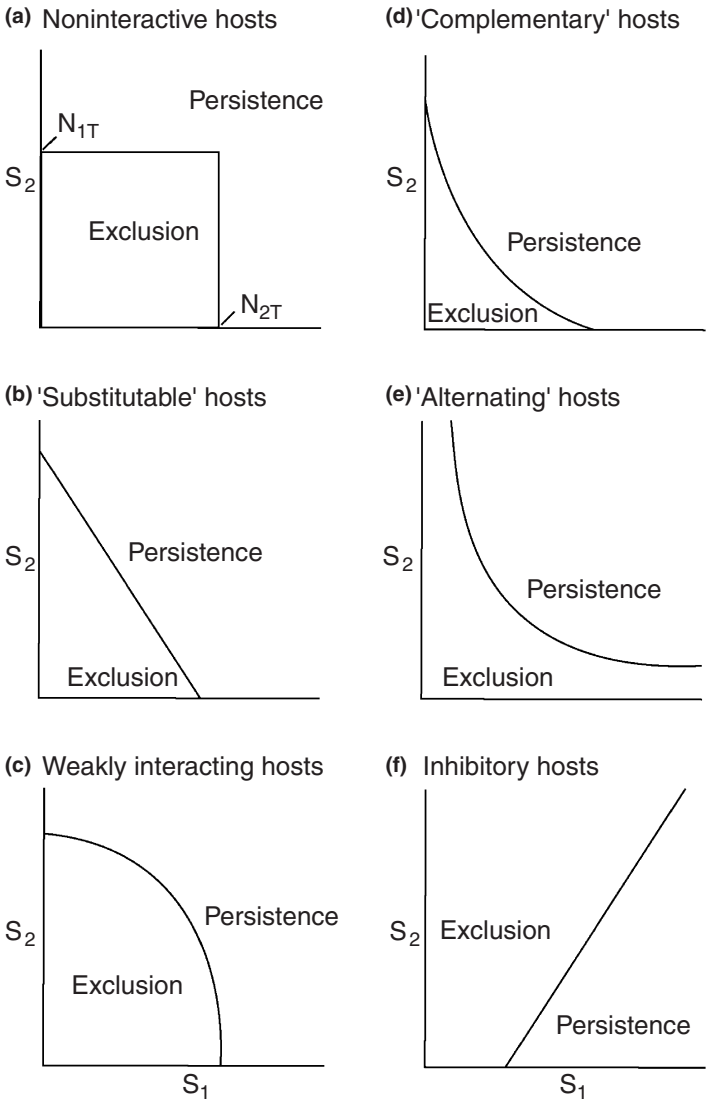

Figure 1 Potential isocline shapes for parasite establishment on two host species (see text for details). (a) Non-interactive hosts, (b) substitutable hosts, (c) weakly interacting hosts, (d) complementary hosts, (e) alternate hosts, (f) inhibitory hosts.

that the carrying capacity of at least one host exceed that host's threshold density. Thus, the existence of alternative hosts is irrelevant for parasite establishment. Figure 1a matches the 'switching' consumer isoclines in Tilman's (1982) classificatory scheme, but the mechanistic interpretation is completely different.

Case 2. Substitutable hosts (Fig. 1b). In some cases, the parasite population may respond to a weighted sum of the two hosts, so that one can write $r(S_1, S_2) = r(aS_1 + bS_2)$, where a and b are constants >0: in a sense, there is a fixed equivalency of the two hosts from the perspective of parasite invasion (Dobson 1990, briefly considers this case). The parasite can invade all combinations of host densities outside a straight line of negative slope. In contrast to case (1), a parasite may invade a combination of host species, when neither alone permits invasion. These parasite zero-growth isoclines correspond to Tilman's 'substitutable' resources.

Case 3. Partially decoupled host–parasite systems (Fig. 1c). If infections occur readily within each host species, but there is a trickle of cross-species infection, the isocline should lie within the box of case (1), but bow outward compared with case (2), so that parasite establishment is modestly enhanced by the presence of multiple host species. This scenario matches Tilman's 'antagonistic' resources case, but again the underlying mechanism is quite different.

Case 4. Strong cross-species infection (Fig. 1d). If infection occurs much more readily between than within host species, the isocline bows towards the origin. This might arise if there are strong mechanisms for spacing within-species (e.g. territoriality), leading to more potential

contacts across than within species. In this case, a mixture of host species more readily sustains the parasite than does either host alone. This matches the 'complementary' resource case of Tilman (1982).

Two final cases correspond to situations where there is, for one or both host species, no threshold density permitting parasite invasion:

Case 5. Alternate hosts (Fig. 1e). If the parasite requires passage through each of two host species to complete its life cycle, no host species alone can sustain the parasite. But a combination of hosts can, provided each host's carrying capacity is sufficiently great. It is useful to separate two subcases: (i) the isocline asymptotically approaches each axis; (ii) the isocline approaches asymptotes, one (or both) of which are displaced away from the axes. In the former, for any given fixed density of one host, there is a some density of the alternate host above which the parasite can invade; in the latter, there is a minimum host density required for parasite persistence, regardless of the abundance of the alternate host. This case resembles the 'essential' resource case of Tilman.

Case 6. Inhibitory host (Fig. 1f). A final possibility is that one host cannot sustain the parasite on its own, at any density, and moreover diminishes the rate at which the other host becomes infected. This implies an isocline with positive slope. This may at first glance seem improbable, but some of the models mentioned below generates this scenario quite naturally, because one host species provides a 'sink' for infective propagules produced by the alternative host (see also Schmidt & Ostfeld 2001). This case was not part of Tilman's original classificatory scheme, but has since been recognized as feasible in some predator–prey systems (e.g. Holt 1983); for instance, if a predator cannot discriminate between toxic and non-toxic prey until after consumption, the predator's zero-growth isocline will have positive slope.

This graphical approach can be applied to any specific multi-host pathogen model. For example, consider the model for two host species sharing an infectious disease with direct transmission explored by Holt & Pickering (1985) and expanded by Begon *et al.* (1992) to incorporate host self-regulation. Without the pathogen, the hosts have logistic growth. With the pathogen, hosts are either infected, or healthy but susceptible. Transmission dynamics are described by mass action terms comparable to that in model (1), albeit with different rates of transmission within (β_{ii}) and between species (β_{ij}, the rate of transmission from species *j* to species *i*). For this model, if the pathogen is rare and susceptible densities are fixed, one can derive an expression for the growth rate of the pathogen by solving for the eigenvalues of the pair of differential equations describing the infection. This expression can be expressed as a function of the density of each host, and so can be displayed as an isocline. The qualitative form of the pathogen isocline turns out to depend upon the relative rates of within- vs. between host species transmisson, and in particular the value of the ratio $\beta_{21}\beta_{12}/\beta_{11}\beta_{22}$. If this quantity is unity, the zero-growth isocline is linear; if greater than unity, the isocline is concave inward; if less than unity, the isocline bends outward (in the limit of zero cross-species transmission for either species, non-interactive isoclines emerge).

Thus, most isocline shapes sketched earlier emerge as special cases of this model. Qualitative isocline shape (e.g. linear vs. concave inward) is governed entirely by the relative magnitude of within vs. between species transmission. Other epidemiological parameters (e.g. parasite-induced mortality) affect the intercepts but not the qualitative shape of the pathogen's zero growth isocline. However, models with direct transmission do not lead to inhibitory hosts (Fig. 1f). One way to generate inhibitory hosts is to assume that infection occurs from free-living pathogen spores, as in the models developed by Bowers & Begon (1991) and Begon & Bowers (1994, 1995). Isoclines with positive slopes arise if hosts, in effect, absorb more spores than they release before death. These hosts deplete the pool of infectious stages, so reduce the force of infection experienced by the alternative host, and hence comprise a demographic 'sink' for the parasite. Comparable effects arise in systems where transmission is via a free-living vector population (the 'dilution' effect of Norman *et al.* 1999; Ostfeld & Keesing 2000; Schmidt & Ostfeld 2001).

DISCUSSION

We have described a graphical approach to the problem of disease establishment in a two-host context. This approach, analogous to consumer–resource isoclines familiar to ecologists, provides an easily interpretable framework for considering how community structure affects disease establishment. Many of our qualitative insights (e.g. the notion of substitutable vs. complementary hosts) extend readily to systems with greater than two host species. Isocline shape is governed by the relative rates of intra- and interspecific transmission. Estimating transmission rates is difficult (Becker & Yip 1989; Anderson & May 1991), and may be further complicated by the hidden presence of multiple hosts. Estimates of transmission parameters from age-prevalence data and other indirect indices that ignore the presence of other host species will overestimate the rate of direct transmission in a focal host. Moreover, transmission rates between species will be determined in part by the resource utilization patterns and spatial distributions of the hosts. Species that compete for resources may also have a higher potential for interspecific disease transmission, unless competition leads to habitat segregation. This can greatly complicate attempts to disentangle effects of resource

competition from those of shared natural enemies (Holt 1977; Holt & Lawton 1994). Leaving that problem aside, these observations suggest that one may be able to ascertain some qualitative features of requirements for parasite persistence in multiple host systems using rather broad considerations of parasite life cycles (e.g. monoxenic vs. heteroxenic) and host and vector ecology.

Elucidating how parasite persistence depends on host community structure pertains to the conservation of parasites as components of species diversity, and is also relevant to the mitigation of disease risk for humans, domestic animals, crops, and wild species of conservation or economic concern. Parasites are a major component of species diversity (Hugot *et al.* 2001) and play important ecological roles in population dynamics, species coexistence, and trophic interactions (Price *et al.* 1986; Poulin 1999). Parasite loss may explain the success of invaders at the expense of native species (Elton 1958; Dobson 1988; Keane & Crawley 2002; Torchin *et al.* 2002), so parasite persistence has important repercussions for community structure. Parasites with complex life cycles (with 'essential resource' isoclines) may be particularly vulnerable to extinction in depauperate communities. The persistence of such parasites may provide an indicator of ecosystem integrity (Lafferty 1997).

Host community composition can fundamentally influence the establishment and prevalence of diseases that affect humans and other organisms. Anthropogenic changes to host community composition can indirectly affect disease impacts. Our framework provides insight into cases where a rich host community is likely to enhance or inhibit disease establishment, and cases where introducing a novel host may potentially endanger native species. In general, additional host species are likely to inhibit the establishment of infectious diseases if they produce isoclines with positive slopes (the 'inhibitory resource' case). This case does not typically arise in models with directly transmitted parasites, but can easily emerge from models with free-living infectious stages or vectors (species that are poor hosts for a pathogen may reduce its prevalence by diverting vectors away from more competent reservoirs, Ostfeld & Keesing 2000).

Species may be threatened by invaders via shared parasites. The graphical framework we have developed can be applied to the question of how shared parasites impact host coexistence. For example, if a pathogen is highly virulent to host species i, the intercept of the isocline on the S_i axis will likely exceed K_i (the carrying capacity of host i) and the pathogen will not persist in a population of host i alone. If host species j is more tolerant of infection, then the isocline will intersect the S_j axis near the origin and the pathogen may persist in host j alone. Consequently, if host j is introduced and reaches a sufficiently high density, the pathogen can persist and the vulnerable host i will be exposed. Given sufficient interspecific transmission, host i will not persist in the presence of both host j and the pathogen (this case of shared parasitism is examined formally in Holt & Pickering 1985; Begon *et al.* 1992; and Greenman & Hudson 1997).

The graphical theory presented in this paper provides a flexible framework for studying disease establishment in multi-host systems and for linking host–parasite ecology to a familiar paradigm in resource-consumer ecology. Understanding how parasite establishment relates to community composition is relevant to the conservation of parasites as components of biodiversity and also to the conservation of species subject to apparent competition with species introduced or made abundant by human activities. There is an urgent need for more empirical data on the relative forces of inter- and intraspecific transmission, as well as on the host–specific pathogenicity of shared parasites.

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