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REVIEW AND SYNTHESIS

The community ecology of pathogens: coinfection, coexistence and community composition

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Abstract

Disease and community ecology share conceptual and theoretical lineages, and there has been a resurgence of interest in strengthening links between these fields. Building on recent syntheses focused on the effects of host community composition on single pathogen systems, we examine pathogen (microparasite) communities using a stochastic metacommunity model as a starting point to bridge community and disease ecology perspectives. Such models incorporate the effects of core community processes, such as ecological drift, selection and dispersal, but have not been extended to incorporate host–pathogen interactions, such as immunosuppression or synergistic mortality, that are central to disease ecology. We use a two-pathogen susceptible-infected (SI) model to fill these gaps in the metacommunity approach; however, SI models can be intractable for examining species-diverse, spatially structured systems. By placing disease into a framework developed for community ecology, our synthesis highlights areas ripe for progress, including a theoretical framework that incorporates host dynamics, spatial structuring and evolutionary processes, as well as the data needed to test the predictions of such a model. Our synthesis points the way for this framework and demonstrates that a deeper understanding of pathogen community dynamics will emerge from approaches working at the interface of disease and community ecology.

Keywords

Coinfection, community ecology, disease ecology, dispersal, drift, metacommunity, metapopulation, pathogen, selection, speciation.

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INTRODUCTION

Only a fraction of parasite and pathogen biodiversity has been discovered (Poulin & Morand 2004; Dobson *et al*. 2008), and each free-living species serves as a host for multiple pathogen species (Dobson *et al*. 2008; Seabloom *et al*. 2009; Borer *et al*. 2013). The assemblage of coinfecting pathogens ('infracommunity' Poulin & Morand 2004) can include a broad array of facilitative, neutral and antagonistic interactions (Al-Naimi *et al*. 2005; Seabloom *et al*. 2009; Poulin 2010; Telfer *et al*. 2010; Borer *et al*. 2013). Pathogen interactions can enhance or inhibit transmission (Al-Naimi *et al*. 2005; Koskella *et al*. 2006; Pedersen & Fenton 2007; Jolles *et al*. 2008; Borer *et al*. 2013) and virulence (Murall *et al*. 2012; Alizon *et al*. 2013), and ultimately mediate disease emergence (Earn *et al*. 2002; Pedersen & Fenton 2007; Telfer *et al*. 2010). The interactions among pathogens also can have major ramifications for both host and pathogen populations through their impacts on host fitness, behaviour, immunity and population demographics (Rohani *et al*. 1998; Hood 2003; Park *et al*. 2009; Hersh *et al*. 2012; Borer *et al*. 2013).

The importance of pathogen interactions to pathogen–host dynamics has motivated recent exploration of the drivers and consequences of pathogen community composition, enhancing links between disease and community ecology (Dove & Cribb 2006; Pedersen & Fenton 2007; Poulin 2010; Seabloom *et al*. 2010; Telfer *et al*. 2010; Mihaljevic 2012). We suggest that continuing to strengthen the interplay of disease and community ecology can guide more general explorations of pathogen communities while also extending our understanding of community ecology, which has focused primarily on free-living organisms (Pedersen & Fenton 2007; Prosser *et al*. 2007; Poulin 2010; Vellend 2010; Costello *et al*. 2012; Mihaljevic 2012). Community ecology theory can clarify complex dynamics arising from models of even the simplest pathogen communities (Rohani *et al*. 1998; Keeling & Rohani 2008; Seabloom *et al*. 2009). In turn, rapid dynamics of host–pathogen systems provide ideal models for investigating the interface of evolutionary and ecological processes (Mihaljevic 2012).

Community and disease ecology are linked through the use of analogous patch-occupancy models (May & Nowak 1994). Patch-occupancy models in community ecology (e.g. metapopulation or metacommunity models) examine the dynamics of the proportion of patches in a landscape occupied by species (Levins 1969; Slatkin 1974), while in disease ecology patch models (e.g. epidemic or susceptible-infected 'SI' models) track

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the density or proportion of hosts in a population that are currently uninfected (i.e. susceptible) or infected. If we conceive of hosts as patches of habitat for pathogens, basic SI and metapopulation models can be mathematically identical, leading to analogies between coexistence of pathogens and the meta-community dynamics of free-living species in spatially structured landscapes (May & Nowak 1994). For example, given a strict competitive hierarchy in which no two pathogen species can coinfect the same host (habitat patch), coexistence depends on a 'limit to similarity' in the virulence (i.e. fitness effects on the host) or transmission rates (colonisation rates) of competing species (May & Nowak 1994; Tilman 1994). Similarly, the elimination of the most virulent pathogens in response to vaccination is mathematically identical to the extinction of the most competitive species in a community in response to habitat destruction (May & Nowak 1994; Tilman *et al*. 1994).

Community theory and disease ecology theory have diverged as they have developed beyond these first patch-occupancy models (e.g. May & Nowak 1994; Tilman *et al*. 1994). For example, a defining characteristic of models in disease ecology has been a focus on the linkages between pathogen and host (i.e. patch) dynamics (Box 1; Anderson & May 1991), whereas community ecology models typically do not incorporate patch dynamics or have a constant patch loss rate that is independent of occupancy (i.e. infection status for host–pathogen models) (Scenarios 1–4 and 6 in Table 2; Levins 1969; Slatkin 1974; Hastings 1980; Tilman 1994; Klausmeier 2001) (but see Mena-Lorca *et al*. 2006). However, the dynamics of even the simplest pathogen communities (e.g. non-spatial models of two pathogens sharing a single host; Box 2) rapidly become complex as the number of pathogen species in the model increases (Rohani *et al*. 1998; Keeling & Rohani 2008; Seabloom *et al*. 2009). For this reason, these models typically do not address dynamics of more diverse pathogen communities, despite a large empirical literature examining the underlying structure of pathogen communities and host–pathogen networks that is especially well developed for macroparasites (Vazquez *et al*. 2007; Fenton *et al*. 2010; Poulin 2010; Reperant 2010). In contrast to the archetypal host–pathogen models, recent advances in metacommunity

Box 1 A stochastic metacommunity model for pathogens

Here, we describe the basic elements of a stochastic metacommunity model that captures the processes of drift, dispersal, selection and speciation (Vellend 2010). The model draws on the extensive body of work on neutral community dynamics (Caswell 1976; Hubbell 2001; Noble *et al*. 2011). Instead of assuming demographic equivalence and neutral dynamics, we use a model that allows for fitness differences among species (Noble *et al*. 2011). Like many metacommunity models, Noble *et al*. (2011) envision many local communities embedded within a broader metacommunity (Figs 1 and 2). Here, we interpret the local community to be the community of pathogens residing on or in a single host individual and the metacommunity to be the collection of pathogens circulating among a population of hosts (Fig. 2).

Noble *et al*. (2011) fix the number of individuals (pathogens) in each local community (host) as a constant (J_L) that is equal across local communities. The total number of pathogen species in the metacommunity (S) and their relative abundances are also fixed. As a result, regional persistence of all species is guaranteed, although species can become extinct within local populations (i.e. within individual hosts). Although not explicit in Noble *et al*. (2011), we can assume (as most neutral models do) that the metacommunity species richness S will depend positively on the rate of pathogen *speciation*.

Within each host, pathogen individuals perish at a constant per capita rate. When a pathogen individual dies, it is replaced either by the immigration of a new pathogen individual from the metacommunity (i.e. transmission from another host), or by the reproduction of another pathogen individual already present within the host (Fig. 2). Immigration occurs with probability m, which may differ among pathogen species, and reproduction occurs with probability $1 - m$. If immigration occurs, then the species that successfully immigrates is determined by each species' relative abundance in the metacommunity (x) multiplied by its ecological fitness (w). If reproduction occurs instead of immigration, the species that successfully reproduces is determined by the species' local abundance in the host (n) multiplied by its fitness (w). In this way, the within-host dynamics are determined by migration from the metacommunity to the individual community (*dispersal, m*), fitness differences among pathogen species (*selection, w*) and stochastic fluctuations (*drift*) that generate variation among host communities even though all host communities are governed by the same basic processes.

For any choice of parameters, this model yields equilibrium probability distributions for the abundance of each pathogen species that integrate the impacts of dispersal, selection, speciation and drift on the diversity and composition of within-host–pathogen communities (Fig. 3). In contrast to classical SIR models (e.g. Box 2; Fig. 4), this type of metacommunity approach scales readily to diverse communities, incorporates both within- and among-host dynamics, and predicts within host abundances. However, we are not aware of models in the metacommunity lineage that incorporate feedbacks between community composition and dispersal or host (i.e. patch) dynamics, feedbacks that can alter pathogen community dynamics (Box 2; Fig. 5).

Furthermore, this model only allows local extinctions, as it makes the strong assumption that abundances in the metacommunity are fixed and independent of local abundances. For this reason, this model is only informative for understanding local (i.e. within-host) abundance of pathogens, and is not informative about regional coexistence in its current form. Relaxing the assumption of a fixed regional species pool and allowing regional extinctions would be a natural first step in modifying this model.

Table 1 Overview of a general model of multipathogen communities within host populations

Rate equations

$$\dot{S} = \alpha(S, A, B, X) - \nu_S S - \lambda_A S - \lambda_B S + \gamma_A A + \gamma_B B$$
$$\dot{A} = \lambda_A S - \nu_A A - \gamma_A A + \gamma_B X - \psi\lambda_B A$$
$$\dot{B} = \lambda_B S - \nu_B B - \gamma_B B + \gamma_A X - \psi\lambda_A B$$
$$\dot{X} = \psi\lambda_B A + \psi\lambda_A B - (\gamma_A + \gamma_B + \nu_X) X$$

State variables	
S	Density of uninfected hosts
A	Density of hosts infected only with pathogen A
B	Density of hosts infected only with pathogen B
X	Density of coinfected hosts
N	Total host population density (= S + A + B + X)
Options for host recruitment	
Constant	$\alpha(S,\ A,\ B,\ X) = \alpha_0$
Density independent	$\alpha(S,\ A,\ B,\ X) = \alpha_0(S + k_A A + k_B B + k_X X)$
Density dependent	$\alpha(S, A, B, X) = \alpha_0(S + k_A A + k_B B + k_X X)(1 - N/K)$
Options for transmission	
Density dependent	$\lambda_A = \beta_A(A + qX)$
Frequency dependent	$\lambda_A = \beta_A(A + qX)/N$ $\lambda_B = \beta_B(B + qX)/N$
Parameters	
α_0	Baseline host recruitment
k_A, k_B, k_X	Reductions (or increases) in host fecundity from infection
K	Host carrying capacity
β_A, β_B	Transmission rates (equal to a contact rate times a probability of transmission given contact)
γ_A, γ_B	Clearance rates of pathogens
ν_S, ν_A, ν_B, ν_X	Host mortality rates
q	Infectivity of coinfected hosts, relative to infectivity of singly infected hosts
ψ	susceptibility of singly infected hosts to a second infection

models specifically address the stochastic dynamics of spatially structured, diverse communities, albeit with the simplifying assumption that habitat patches are static or have dynamics unrelated to patch occupancy (Klausmeier 2001; Noble *et al*. 2011) (but see Mena-Lorca *et al*. 2006).

Here, we build on the conceptual and mathematical linkages between disease and community ecology to deepen our understanding of multipathogen (microparasite) dynamics. To do this, we use a stochastic metacommunity model (Box 1; Noble *et al*. 2011) to explore the dominant processes in community ecology and their implications for understanding pathogen communities. We then address unique aspects of host–pathogen systems that are not fully incorporated in community ecology theory using a single-host, two-pathogen SI model (Box 2). As a case study, we also illustrate how the processes highlighted in these models structure the dynamics of a group of viral plant pathogens (Box 3). Finally, we use this framework to clarify some of the areas in pathogen community ecology that stand out as fruitful avenues for future research.

Although we focus our discussion on pathogen communities, hosts carry many microbial symbionts that may have negative, neutral or positive effects on host fitness (Borer *et al*. 2013). For example, the assemblage of microbes within a single human contains about 100 times more genes than the human host (Nelson *et al*. 2010) and comprises about 50% of an individual human's total carbon (Shively *et al*. 2001). Our current focus on pathogen communities reflects the vastly more developed theory for host–pathogen dynamics; however, most of the concepts and opportunities discussed in this review apply to symbiont communities, generally (e.g. Prosser *et al*. 2007; Costello *et al*. 2012; Mihaljevic 2012). For example, the metacommunity framework presented in Box 1 is particularly well suited as a starting point for modelling diverse assemblages of asymptomatic symbionts, as it is more scalable to larger numbers of species and does not include host dynamics.

DOMINANT PROCESSES IN COMMUNITY ECOLOGY

In a recent synthesis of community ecology, Vellend (2010) suggests that four fundamental processes govern community dynamics: ecological drift, selection, dispersal and speciation. These are analogous to the dominant forces in population genetics: drift, selection, gene flow and mutation. He further argues that community ecology theory can be synthesised into a general theory of community dynamics, in which species are added to communities through speciation and dispersal and their relative abundances are altered by drift and selection. In this context, selection is the outcome of the interactions among species with different fitnesses and niches (Adler *et al*. 2007; Vellend 2010).

This simple and general framework provides a theoretical link with processes that are likely to control the composition of pathogen communities (Fig. 1). We explore these links using a stochastic metapopulation model which allows us to examine the effects of ecological drift, ecological selection and dispersal among hosts on the distribution of pathogen

Table 2 Relationship among seven different disease ecology scenarios, as represented in the general multipathogen model (Table 1, Fig. 4) assumptions regarding host dynamics, host–pathogen interactions and among-pathogen interactions, and analogous models from community ecology including metapopulation, metacommunity, competition-colonisation trade-off and neutral models

Disease scenario	Host dynamics	Host–pathogen interactions	Among pathogen interactions	Community ecology analogue
1. Non-interacting pathogens	No host dynamics: $\alpha_0 = 0$, $v_S = \nu_A = \nu_B = \nu_X = 0$	No interactions	No interactions: $\psi = 1$, $q = 1$	Two coincident metapopulations (Levins 1969)
2. Multiple interacting pathogens	No host dynamics: $\alpha_0 = 0$, $\nu_S = \nu_A = \nu_B = \nu_X = 0$	No interactions	Pathogens interact through altered susceptibility to secondary infection: $\psi < 1$ (competition) or $\psi > 1$ (facilitation), $q = 1$	Metacommunity (Slatkin 1974)
3. Cross-protection – transmission trade-off	No host dynamics: $\alpha_0 = 0$, $\nu_S = \nu_A = \nu_B = \nu_X = 0$	No interactions	Pathogen A competitively displaces pathogen B from infected hosts: $\psi = 0$ for species B transmitting to a host infected with species A γ_B of coinfected hosts $\rightarrow \infty$ $\beta_B > \beta_A$ gives pathogen B a transmission advantage over A	Competition-colonisation trade-off (Hastings 1980; May & Nowak 1994, Tilman 1994)
4. Pathogen independent host dynamics	Host populations are dynamic: $\alpha_0 > 0$, $\nu_S = \nu_A = \nu_B = \nu_X = \nu > 0$	No interactions	No interactions: $\psi = 1$, $q = 1$	Habitat destruction models (Klausmeier 2001; but $\alpha_0 = 0$; Tilman *et al.* 1994; assumes a competitive hierarchy)
5. Pathogens have detrimental effects on their hosts	Host populations are dynamic: $\alpha_0 > 0$, $\nu_A = \nu_B = \nu_X > \nu_S > 0$	Pathogens affect host survival or recruitment: ν_A, ν_B, $\nu_X > \nu_S$or k_A, k_B, $k_X < 1$if host recruitment is not constant	No interactions: $\psi = 1$, $q = 1$	Dynamic landscapes (Mena-Lorca *et al.* 2006; assumes a competitive hierarchy)
6. Pathogen competition or interference	No host dynamics: $\alpha_0 = 0$, $\nu_S = \nu_A = \nu_B = \nu_X = 0$	No interactions	Pathogens compete through reduced transmission to infected hosts, reduced transmission from coinfected hosts, and increased clearance of coinfections (assuming simultaneous clearance is possible): $\psi < 1$, $q < 1$, $\gamma_X > \gamma_A$, γ_B	Patch occupancy models of competition (Klausmeier & Tilman 2002)
7. Pathogens are ecologically neutral	Host populations are dynamic: $\alpha_0 > 0$, $\nu_A = \nu_B = \nu_X > \nu_S > 0$	Pathogens affect host survival: ν_A, ν_B, $\nu_X > \nu_S$	Pathogens compete through reduced transmission from coinfected hosts (assuming simultaneous clearance, γ_x, is possible): $\gamma_A = \gamma_B = \gamma_X$, $q = 1/2$, $\psi \leq 1$	Neutral model (Hubbell 2001; Lipsitch *et al.* 2009)

abundances among hosts (Box 1; Fig. 2; Noble *et al.* 2011). Although we discuss diverse examples throughout this review, we also provide a unified example of the role of these processes for a group of well-studied viral pathogens of grasses, barley and cereal yellow dwarf viruses (B/CYDVs; Box 3).

Ecological drift

In the absence of any differentiation among species, the stochastic nature of births and deaths will cause a community composed of a finite number of individuals to drift stochastically until all but one species has gone extinct (Hubbell 2001; Vellend 2010; Rosindell *et al.* 2012). As a result of ecological drift, local community composition will vary even in a metacommunity of species having identical fitness and homogenous patches (Fig. 3). The rate at which communities drift to the single species equilibrium is slower in communities supporting large numbers of individuals and when there is strong negative density dependence (Adler *et al.* 2007). Ultimately, the random walk to a single species community can be counterbalanced by dispersal and selection, as discussed below. While the recognition of ecological drift as an important process in community ecology emerged at least as early as the theory of island biogeography (MacArthur & Wilson 1967), there has been a resurgence of interest and controversy in ecological drift in the past decade since the introduction of the Unified

Box 2 A deterministic two-pathogen SI model

To illustrate the links between community and disease ecology theory, we present a basic two-pathogen susceptible-infected (SI) model (Fig. 4; Table 1) based on the classic compartmental modelling framework for microparasite diseases (Anderson & May 1991) and other more recent multipathogen models (Vasco *et al*. 2007; Jolles *et al*. 2008; Keeling & Rohani 2008; Martcheva *et al*. 2008; Seabloom *et al*. 2009). We simplified this model by not allowing for simultaneous infection of an uninfected host with both pathogens (an $S \rightarrow X$ transition) or for simultaneous clearance of both pathogens from a coinfected host (an $X \rightarrow S$ transition; but see Scenario 7 in Table 2). More complex versions of this model have been applied to a variety of pathogen systems (Vasco *et al*. 2007; Jolles *et al*. 2008; Keeling & Rohani 2008; Martcheva *et al*. 2008; Seabloom *et al*. 2009).

When pathogens have no impact on each other or host dynamics (Scenario 1; Table 2), the prevalence of coinfected individuals is the product of the separate prevalences of each pathogen. In this case, processes decreasing separate pathogen prevalences, such as decreased transmission or increased clearance by hosts, will decrease the prevalence of coinfected individuals. Most metacommunity models assume that habitat patches are not dynamic or have dynamics that are unrelated to patch occupancy (Scenarios 1–4 and 6 in Table 2; Levins 1969; Slatkin 1974; Hastings 1980; Tilman 1994; Klausmeier 2001; but see Mena-Lorca *et al*. 2006). This assumption can be appropriate for pathogens whose dynamics are much faster than host lifespan and that do not induce mortality or long-term immunity. However, when infection influences host dynamics (e.g. Scenarios 4, 5, 7 in Table 2; $k_A, k_B, k_X < 1$, $v_A, v_B, v_X > v_S$), it is no longer true that the abundance of coinfected hosts will be equal to the product of the marginal prevalences of the individual pathogens.

Non-interactive pathogens can coexist as long as they do not negatively affect each other's fitness (Scenarios 1, 4 in Table 2); however, pathogen coexistence becomes more difficult when pathogens interact negatively with one another. In this case, two pathogens can coexist if they both have positive growth rates when the other pathogen is present at its equilibrium prevalence (Fig. 5). In this model, pathogen species can interact through (1) reduced transmission from coinfected hosts relative to singly infected hosts (Scenarios 6 and 7 in Table 2; $q < 1$), (2) reduced susceptibility of infected hosts to a second infection (Scenarios 2, 3, 6 and 7 in Table 2; $\psi < 1$; including immunological interference) (Rohani *et al*. 2003; Keeling & Rohani 2008; Seabloom *et al*. 2009) and (3) host demographic consequences of infection (Scenarios 5, 7 in Table 2; $v_A, v_B, v_X > v_S$; $k_A, k_B, k_X < 1$ Scenarios 2, 3, 6 and 7 in Table 2; $\psi < 1$; including ecological interference sensu) (Rohani *et al*. 2003; Keeling & Rohani 2008) and synergistic mortality of coinfected hosts (e.g. $v_A, v_B < v_X$; Fig. 5) (Vasco *et al*. 2007; Seabloom *et al*. 2009). As these competitive effects become stronger, the opportunities for pathogen coexistence and coinfection decrease (Fig. 5). Coinfection also can result in immunosuppression, which may promote pathogen coexistence (Fig. 5).

In the absence of niche and fitness differences, purely neutral dynamics may arise in which pathogen species are functionally indistinguishable (approximately Scenario 7 in Table 2 except that a neutral model would require introducing an $X \rightarrow S$ transition so that coinfected hosts can clear both pathogens simultaneously; Lipsitch *et al*. 2009). In this case, fluctuations in the abundance of competing pathogens will be determined entirely by stochastic drift, in the same way that drift drives community composition in the neutral theory of community biodiversity (Caswell 1976; Hubbell 2001). A stochastic version of the model given in Table 1 is required to determine whether stochastic fluctuations in pathogen abundance caused by drift are relevant on ecological and epidemiological timescales.

As in any community of competing species, the outcome of competition depends on the relative competitive abilities of each species, as well as on their niche differences (e.g. ability to infect already-infected hosts, $\psi > 0$, for example, by infecting different host tissues). In community ecology, the competition-colonisation trade-off (Hastings 1980; May & Nowak 1994; Tilman 1994) provides a familiar example of how habitat competitors can coexist through life-history trade-offs. A similar result holds in the two-pathogen disease model here if we assume a strict competitive asymmetry (Scenario 3 in Table 2) (May & Nowak 1994).

Neutral Theory of Biodiversity in 2001 (i.e. neutral theory) (Caswell 1976; Hubbell 2001; Noble *et al*. 2011; Rosindell *et al*. 2012). Ecological drift and neutral processes have received less attention in pathogen communities than in communities of free-living organisms (but see Dove & Cribb 2006; Lipsitch *et al*. 2009; Seabloom *et al*. 2010), although the role of stochastic processes in temporal dynamics has been studied in depth (e.g. Bolker & Grenfell 1995; Keeling & Rohani 2008; Lipsitch *et al*. 2009; Cobey & Lipsitch 2012). Nevertheless, as we argue below, simple community models driven only by ecological drift are relevant for disease ecology.

Ecological drift has the potential to structure pathogen assemblages (Fig. 3; Table 2; Scenario 7), either within host individuals or within host populations, especially among pathogens with similar fitness, overlapping niches (Chesson 2000; Adler *et al*. 2007) and small populations within a host (Vellend 2010). The link between population size and drift suggests that pathogens that persist at low abundance (e.g. titre) or in a limited portion of host tissue or cells will be more strongly influenced by drift. Strong suppression of pathogen populations by the host immune system, medication or by other microbes also may increase the importance of drift for the suppressed pathogens by lowering population size. Drift also may be more important at the beginning of an infection, as the pathogen begins to invade the host. For example, infections by vector-transmitted plant viruses begin with only a few viral particles (Moury *et al*. 2007; Ali 2008; Tromas *et al*. 2014), and movement between cells or organs

Box 3 The community ecology of barley and cereal yellow dwarf viruses (B/CYDVs)

Here, we present a brief case study of the how drift, ecological selection and dispersal act to structure a community of aphid-vectored, RNA viruses that infect many grass species in natural and agricultural ecosystems worldwide (D'Arcy 1995; Miller & Rasochova 1997). B/CYDVs are among the most widespread of viral pathogens and cause one of the most severe diseases of grasses (D'Arcy 1995; Miller & Rasochova 1997; Seabloom *et al.* 2009). B/CYDVs also have been implicated in promoting one of the most dramatic plant invasions known, the invasion of 40 million ha of California (USA) grasslands by annual grasses from the European Mediterranean region (Borer *et al.* 2007). This viral community is ideal for studying pathogen community dynamics, because the viruses co-occur within hosts and host populations (Seabloom *et al.* 2009, 2010), and the composition of the viral community can alter important host and pathogen vital rates (Miller & Rasochova 1997; Seabloom *et al.* 2009; Lacroix *et al.* 2014). For example, coinfection can lower viral transmission rates through cross-protection, increase transmission through expanded vector range, and increase host mortality through synergistic mortality (Miller & Rasochova 1997; Seabloom *et al.* 2009; Lacroix *et al.* 2014).

Recent empirical and theoretical studies have shown how *Ecological Drift, Ecological Selection* and *Dispersal* interact to structure the B/CYDV community. *Ecological drift* is expected to create variability in pathogen community composition among identical hosts in a single population (Box 1; Fig. 3a,b) (Dove & Cribb 2006; Seabloom *et al.* 2010; Noble *et al.* 2011). In an observational study of five B/CYDVs in 26 host populations distributed along the Pacific Coast of North America, Seabloom *et al.* (2010) found that the within-host viral community was more variable (i.e. higher β diversity) in southern populations suggesting a stronger role of ecological drift and dispersal limitation. A potential complementary approach to detecting ecological drift would be to experimentally inoculate identical hosts with multiple pathogens, and track community divergence through time. *Ecological Selection* also has been shown to play an important role in the structuring of these viral communities, and host identity and nutrient supply rates often play a strong role in mediating these interactions (Seabloom *et al.* 2013; Lacroix *et al.* 2014). For example, Lacroix *et al.* (2014) used experimental, laboratory inoculations to demonstrate deterministic selection, such that BYDV-PAV suppressed the infection success of its competitor CYDV-RPV at low nutrient supply rates and that increased host nitrogen supply weakened this antagonistic effect. Selection ultimately can play out at the landscape scale. Power (1996) documented the competitive displacement of BYDV-MAV by BYDV-PAV over a period of two decades in New York state using long-term observational data, likely due to BYDV-PAV's higher dispersal (i.e. transmission) rate, because it is a vector generalist.

Dispersal is predicted to increase community diversity and to ameliorate the effects of fitness differences (Fig. 3c,d) (Noble *et al.* 2011), and theoretical models of the B/CYDV system show that increased transmission (i.e. dispersal) leads to increased coexistence and coinfection rates (Seabloom *et al.* 2009). However, dispersal effects are complex in these aphid-vectored pathogens, because different species of aphids carry distinct subsets of the viral community, and viruses that share vectors also have higher coinfection rates in field populations than viruses that do not share vectors (Seabloom *et al.* 2009, 2010, 2013). Vector behaviour also is critical in this system, and the distribution of these viruses across different grass hosts in natural grasslands is predicted by the foraging preferences of the aphid vectors (Seabloom *et al.* 2013).

While core community ecology processes such as ecological drift, selection and dispersal provide insight into the structure of the B/CYDV communities; the composition of these viral communities also reflects the interactions with host resistance and host vital rates. For example, some pairs of B/CYDV species induce cross-protection in their hosts (Miller & Rasochova 1997), which is expected to reduce the frequency of coinfection of these species (Fig. 5) (Seabloom *et al.* 2009). Coinfection by some pairs of B/CYDV species can also increase host mortality rates (Miller & Rasochova 1997), which is also expected to lower the coinfection frequency of these viruses based on theoretical models of the system (Fig. 5) (Seabloom *et al.* 2009). The complex interactions between pathogen–pathogen interactions (cross-protection, immune suppression or synergistic mortality) and host–pathogen dynamics open new avenues for research that will inform both disease ecology and community ecology.

can impose strong bottlenecks on population size (French & Stenger 2003; Sacristan *et al.* 2003; Tromas *et al.* 2014) both of which can reduce genetic variation.

Ecological drift also may influence the relative abundance of pathogens at the host population scale. Ecological drift is likely to be of greater importance in small host populations. This situation can lead to random fluctuations in the prevalence of a pathogen due to stochasticity in the demographic rates that determine prevalence, chiefly pathogen transmission and host recovery or mortality rates. Much previous research has examined how this can lead to the extinction ('fadeout') of a pathogen from a local host population (Bolker & Grenfell 1995; Keeling & Rohani 2008). However, when the host population supports multiple pathogen species, stochasticity in pathogen species' prevalences will amount to ecological drift in the structure of the pathogen assemblage. As a result, processes that reduce host population densities may increase the importance of drift in pathogen communities.

Drift will also play a stronger relative role when host populations are highly isolated and dispersal of pathogens between host populations is rare, as shown by the greater variance in abundances when migration is low in Figure 3. As a result, drift-induced variability in pathogen abundance will decline with increased dispersal among hosts (Fig. 3).

While drift is clearly important, it is challenging to quantify the effects of drift empirically. Drift could be detected as

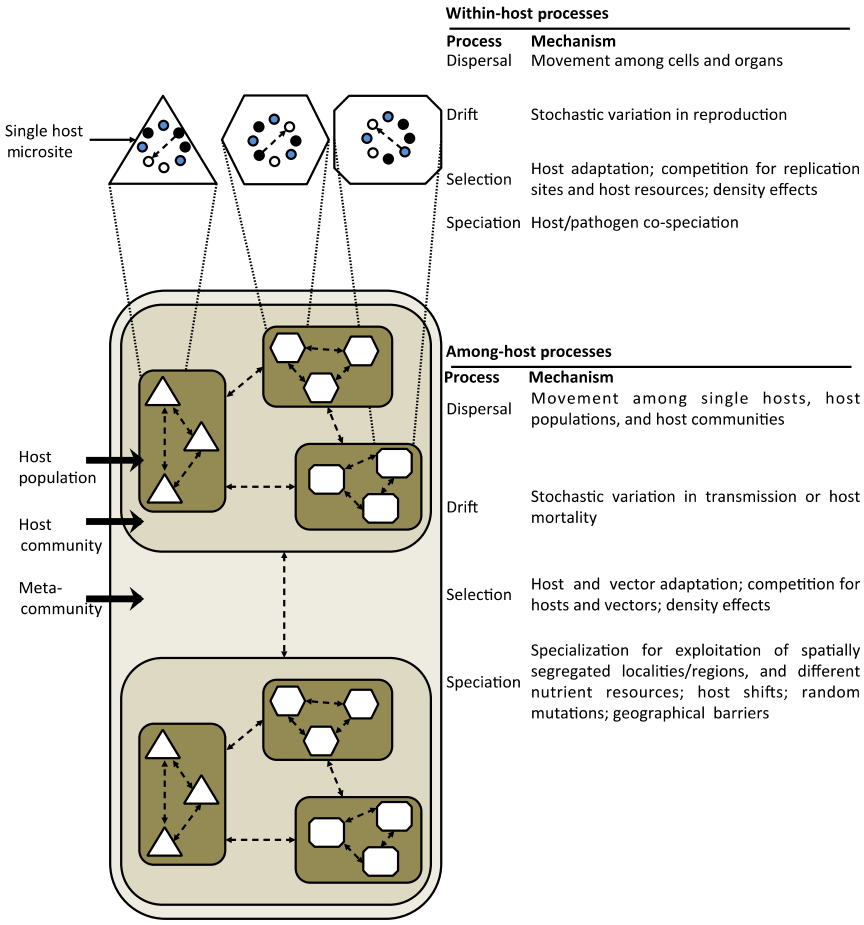

Figure 1 Multiscale interactions among pathogens. An assemblage of pathogens (as illustrated with black, white and blue circles) coinfecting an individual host or vector is an 'infra-community'. At larger scales, pathogen communities can also be defined as the suite of pathogens found within a population of a single host species or a community of host species (represented by the white shapes) at single sites or across a metapopulation or metacommunity of hosts occupying multiple sites. At each scale, ecological processes (dispersal, selection, drift and speciation) and environmental conditions (e.g. homogeneity/heterogeneity in hosts and resources within and among patches, and landscape structure) interact to drive patterns of pathogen species diversity and distribution.

variation in pathogen species composition and relative abundance (β diversity) among identical (e.g. experimental) host populations (Fig. 3), or among host populations after controlling for relevant variation among the host populations (e.g. in density, age, isolation and environment) (Dove & Cribb 2006; Seabloom *et al*. 2010). However, compositional differences may also arise from unmeasured factors such as environmental gradients. Similarly, while exponential decay in the similarity of pathogen communities with spatial distance can be a signature of ecological drift in empirical data (Poulin 2003), such decay in similarity may reflect localised dispersal or environmental gradients.

Ecological selection

Despite the spirited debates about the relative importance of neutral (drift) and niche (selection) processes, even the staunchest proponents of neutral theory recognise that species fitnesses are not identical and strong deterministic mechanisms can govern the outcome of species interactions (Rosindell *et al*. 2012). Ecological selection can act on constant fitness differences among species as well as frequency-dependent population growth, which may result from niche differences (Chesson 2000; Adler *et al*. 2007; Vellend 2010; Noble *et al*. 2011).

Frequency-independent (constant) selection occurs when fitness differences among species are consistent regardless of their relative abundances (Vellend 2010; Noble *et al*. 2011). Under constant selection and in the absence of frequency dependence, the species with the highest fitness will exclude all others (Chesson 2000; Adler *et al*. 2007; Vellend 2010), although stochastic processes may alter the rate of this random walk to extinction and may create cases where a species with higher fitness is excluded (Fig. 3c,d) (Vellend 2010; Noble *et al*. 2011). Equalising mechanisms reduce fitness differences between species and may mitigate competitive exclusion (Table 2 Scenario 3) (Chesson 2000; Adler *et al*. 2007). Stabilising processes increase the strength of intraspecific density dependence relative to interspecific density dependence, causing a species' population growth to decline with increasing relative abundance (i.e. frequency) (Chesson 2000; Adler *et al*. 2007). Stabilising processes that can lead to coexistence include differences in resource use or host-specific natural

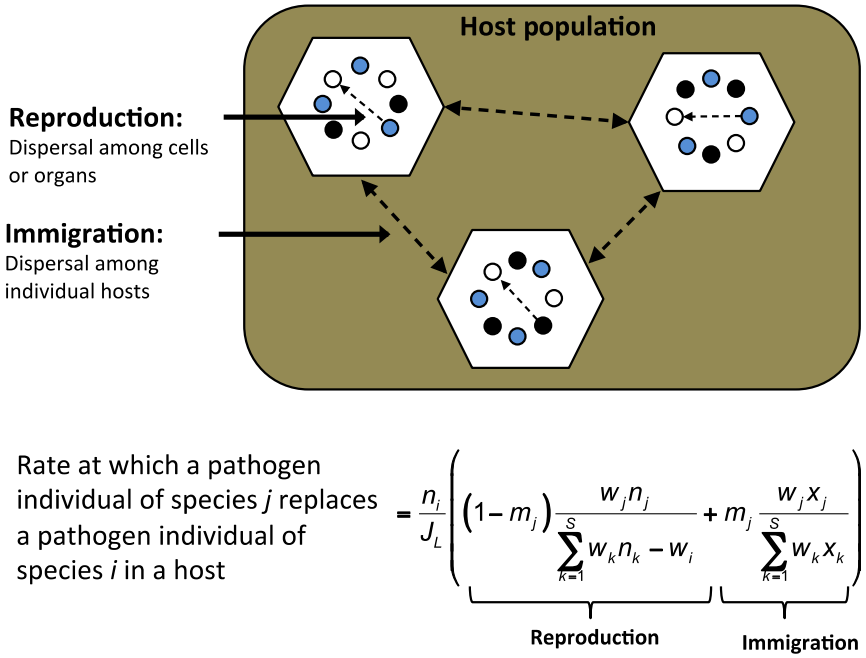

Figure 2 Stochastic metacommunity model incorporating drift, fitness difference (ecological selection) and dispersal within and among hosts after Noble *et al.* (2011), and adapted to pathogen community dynamics. Host individuals (hexagons) support local pathogen communities (circles; colours represent individual pathogen species), and are embedded in a larger metacommunity composed of host populations. New pathogen individuals appear in a host either as the result of immigration from the metacommunity or within-host reproduction; each of these processes can be influenced by the fitness of individual pathogens. Pathogen speciation governs pathogen species richness in the metacommunity. Parameters are described in Box 1.

enemies (Box 3), and may act in conjunction with spatial or temporal variation in the environment (e.g. spatial or temporal storage effects) (Chesson 2000; Adler *et al.* 2007; Vellend 2010).

Constant selection acting on fitness differences among pathogens may be responsible for reducing the number of pathogen types within a host (Eswarappa *et al.* 2012). In our example drawing on Noble *et al.* (2011) (Box 1), pathogens with lower fitness are absent from some hosts (i.e. they have a non-zero y-intercept in Fig. 3c). Selective competitive forces within a host can result from at least three general mechanisms (Mideo 2009): exploitation competition for shared host resources, apparent competition with the immune system acting as the predator (e.g. Scenario 2 in Table 2; $\psi < 1$) and interference competition, in which the pathogens chemically or mechanically inhibit each other's growth. While the particular form of competition among pathogen species within hosts remains often unclear (Ishii *et al.* 2002; Lello *et al.* 2004), recent studies have attempted to provide further evidence for exploitation competition among pathogen species by manipulating the host's resource supplies (Lacroix *et al.* 2014; Lange *et al.* 2014) (Box 3). In addition, experiments investigating immune-mediated interactions have demonstrated both facilitation and competition among coinfecting pathogen species (Lysenko *et al.* 2005; Murphy *et al.* 2013). Exclusion of one pathogen by another can occur within hosts (Hood 2003; Al-Naimi *et al.* 2005) and also within vectors (Gildow & Rochow 1980; Cohen *et al.* 1989; Ng & Chen 2011).

Competitive exclusion may be slowed by ecological drift (Noble *et al.* 2011) or prevented by equalising mechanisms (Adler *et al.* 2007). For example, bacteriophages demonstrate a trade-off between reproduction rate and survival, in which faster reproduction leads to less stable virions and similar life-history trade-offs have been hypothesised for human viruses (De Paepe & Taddei 2006; Murall *et al.* 2012). However, long-term stable coexistence also requires negative frequency-dependent, stabilising selection (Chesson 2000; Adler *et al.* 2007). Cobey and Lipsitch (2012) identified both stabilising and equalising mechanisms for serotypes of *Streptococcus pneumoniae* coexisting among hosts, with acquired serotype-specific immunity stabilising pathogen diversity. This mechanism is analogous to specialist predators that create density-dependent feedback loops in their prey populations, known as the Janzen–Connell hypothesis (Connell 1970; Janzen 1970).

Within-host coexistence mechanisms are often assumed in coinfection models (Lipsitch *et al.* 2009). However, there is evidence for within-host niche differences, such as pathogens that occupy different regions or tissue types in a host (e.g. root and foliar fungal pathogens), so that pathogens sharing a host compete more strongly with conspecific individuals than with heterospecific individuals (Fig. 1). To date, most research on within-host selection and microparasite coexistence has been directed towards the evolution of virulence, evaluating the competitive ability of strains or serotypes of the same species both within and among hosts (Alizon *et al.* 2013).

Dispersal

Dispersal is an important determinant of coexistence, particularly in neutral communities composed of identical species

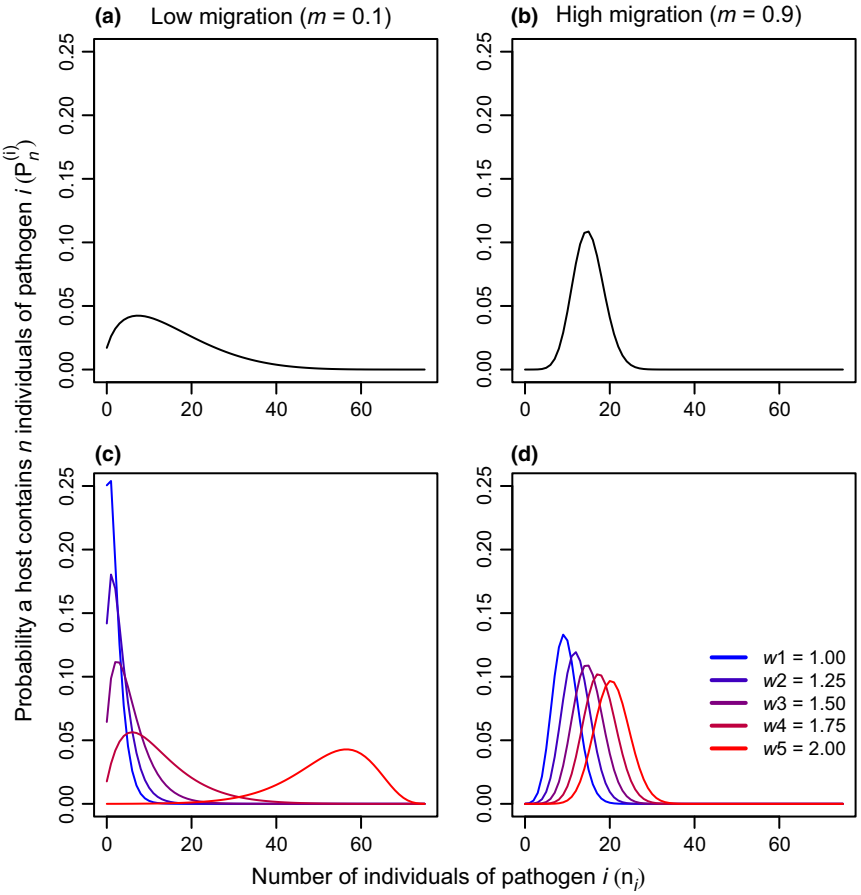

Figure 3 Equilibrium abundance probability distributions for a community of five pathogens with high and low migration among hosts (e.g. among-host transmission). (a, b) Distribution for a neutral community with identical species fitness. Distributions for all species are identical in (a) and (b). (c, d) Distribution of abundances for a non-neutral community in which pathogen fitness (w_i) varies among pathogens.

(MacArthur & Wilson 1967; Hubbell 2001; Noble *et al.* 2011; Rosindell *et al.* 2012). Dispersal is also important in its interaction with selection. For example, dispersal limitation of a superior competitor can allow a competitively inferior species to persist indefinitely, via a trade-off between competitive ability (or virulence in the case of pathogens) and dispersal ability (Hastings 1980; May & Nowak 1994; Tilman 1994; Chesson 2000).

Metacommunity theory integrates dispersal and selection and was developed explicitly to address the mechanism governing the distribution of multiple species across a spatially structured landscape (Leibold *et al.* 2004). It provides a particularly promising framework for understanding the diversity and composition of pathogen communities (Mihaljevic 2012). The parallels between spatially discrete patches in island biogeography and metacommunity theory, and disease ecology and epidemiology, in which hosts or host populations can be considered as discrete patches that are susceptible or infected by one or more pathogen strains, have long been appreciated (Kuris *et al.* 1980; May & Nowak 1994; Grenfell & Harwood 1997). By making the small conceptual jump from physical distances among patches to connectivity via host 'patch' behaviour, interaction rates or vector dynamics and quantifying the rates of host colonisation and extinction by parasites, metacommunity theory and island biogeography theory provide a fertile theoretical framework for understanding parasite species richness within hosts (MacArthur & Wilson 1967; Kuris *et al.* 1980; Grenfell & Harwood 1997; Reperant 2010).

In pathogen systems, host colonisation and pathogen extinction rates, regional pathogen species pool and host size have been used to predict pathogen density, an observation that builds on components of the original island biogeography theory (MacArthur & Wilson 1967; Kuris *et al.* 1980; Grenfell & Harwood 1997; George-Nascimento *et al.* 2004; Hall *et al.* 2007; Reperant 2010). For example, local pathogen richness can be limited primarily by the richness of the species pool present at large spatial scales (Dove & Cribb 2006; Seabloom *et al.* 2009, 2010). In addition to host size, many pathogens and parasites infect hosts that vary widely in their competence (LoGiudice *et al.* 2003; Keesing *et al.* 2006; Johnson *et al.* 2013), thus creating a heterogeneous transmission landscape that would be well-informed by metacommunity theory as it involves the interaction between dispersal and ecological selection. However, few empirical studies have used an explicit metacommunity framework to interpret the outcome of host heterogeneity for the dynamics of pathogens.

In Box 1, we illustrate how a stochastic metacommunity model (Noble *et al.* 2011) can be applied to pathogen

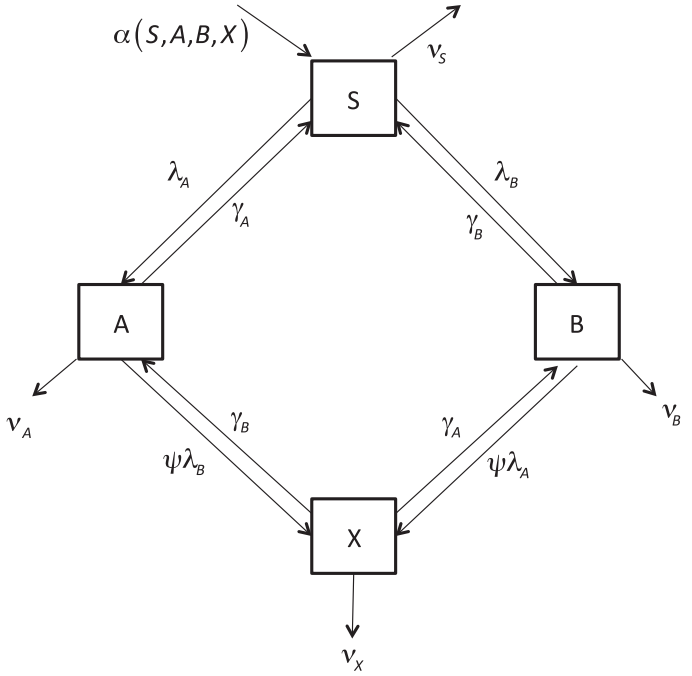

Figure 4 Diagram of a simple one-host, two-pathogen compartmental model. State variables (boxes) are uninfected [(S)usceptible) hosts, hosts infected by either pathogen A, pathogen B or by both pathogens (X). Transitions among states are depicted by arrows, and the rates of those transitions are denoted by the corresponding parameter. Transitions originating from a compartment (all but α) are given on a per capita basis. Parameters and model scenarios are described in Tables 1 and 2.

communities to provide insight into the interactions among drift, selection and dispersal in pathogen communities. For example, dispersal from a regional metacommunity can reduce the likelihood of local (within host) extinctions resulting from selection acting on fitness differences (Fig. 3c,d). Thus, we would expect that any processes leading to increased transmission would lead to increased levels of coinfection and pathogen diversity (Seabloom *et al.* 2009). Similarly, stochasticity in demography can create cases where a species with lower fitness excludes a competitor with higher fitness (Vellend 2010; Noble *et al.* 2011).

Speciation

Just as mutation is an important source of diversification of genes and alleles, speciation helps maintain diversity in communities that would otherwise become monospecific (Hubbell 2001; Vellend 2010). While dispersal between communities may increase local species diversity, it will also cause convergence in species composition among localities in the absence of speciation (MacArthur & Wilson 1967; Vellend 2010). In our example model, we see this effect in the lower variance in the metacommunities with high migration rates (Fig. 3b,d). Speciation can diversify local and regional species pools. While speciation is clearly important in pathogen communities, delimiting species is challenging for fast-evolving microbes, as we state below.

For pathogens, the transmission process itself can result in population bottlenecks and founder effects that lead to drift and promote speciation (Ali & Roossinck 2008). Vector transmission of viruses also can result in significant reductions in genetic diversity of within-host virus populations. Experiments with cucumber mosaic virus suggest that bottlenecks are most severe during vector inoculation of the host (Ali *et al.* 2006), but bottlenecks also occur during vector acquisition of virus from infected hosts or cell-to-cell movement within a host (Tromas *et al.* 2014). Recent studies have demonstrated that plant virus infections can be initiated with as few as 1-3 virions (Moury *et al.* 2007; Betancourt *et al.* 2008; Tromas *et al.* 2014). Vertically transmitted pathogens (those transmitted intergenerationally by gametes) also may experience particularly severe bottlenecks, since the barriers to embryo infection mean that very few pathogen genotypes can successfully infect this tissue (Escarmis *et al.* 2006; Simmons *et al.* 2013).

Such bottlenecks additionally create spatial structure in pathogen diversity within hosts and populations, with the potential to lead to pathogen speciation. Virus phylogenetic studies have indicated that such population bottlenecks have led to speciation in several lineages (e.g. Wang *et al.* 2006). In host–parasite systems, speciation of one interacting lineage (e.g. the pathogen) can be driven by the speciation of the other (e.g. the host). For example, strong congruence of host and parasite phylogenies has revealed some patterns of cospeciation in plant–fungus, plant–virus, and primate–virus systems (Giraud *et al.* 2008; Stobbe *et al.* 2012).

When divergence between populations has arisen, reduced dispersal and gene flow is essential to maintain reproductive isolation leading to speciation, especially for communities evolving in sympatry. In host–vector–pathogen systems, reproductive isolation and divergence of different microbial populations can arise by vector specialisation on different hosts, even if host species co-occur in communities (van Putten *et al.* 2007; Giraud *et al.* 2008). In addition, a large body of evidence supports the process of speciation via specialisation for different ecological niches (MacLean 2005; Schluter 2009).

Species delimitation can be challenging especially for rapidly evolving microorganisms, because the different mechanisms of speciation are not mutually exclusive and can occur at different time scales and chronological order. Many pathogens are characterised by short generation times, high potential of genetic polymorphism and a capacity for producing large populations that enables evolutionary and ecological dynamics to occur on the same time scale. In addition, many microbial pathogens reproduce asexually (e.g. bacteria and some fungi) or are nonliving infectious entities (viruses), making it difficult to apply the traditional criteria of reproductive isolation used to define unique eukaryotic species. A more relevant general definition for microbial species may be as an independent phylogenetic lineage that includes individuals that share some morphological properties and occupy a particular ecological niche (van Regenmortel & Mahy 2004; Konstantinidis *et al.* 2006; Giraud *et al.* 2008). This definition highlights the important contribution of genomic and meta-genomic information to delimit clusters and species and the inadequacy of relying on a single discriminating criterion (e.g. host susceptibility and percentage of nucleotide similarity) for microbial species demarcation.

GAPS IN THE APPLICATION OF COMMUNITY THEORY TO PATHOGEN SYSTEMS

While a system of host patches linked by transmission lends itself to a metacommunity-modelling framework (May & Nowak 1994), there are unresolved challenges in the application of metacommunity models to pathogen systems (Mihaljevic 2012). Perhaps the most defining challenges arise from the links between pathogen dynamics and the population dynamics, behaviour and immune responses of the hosts. In discussing these challenges, we focus primarily on the SI model in Box 2 that dynamically couples pathogen and host dynamics.

Host movement and demography create complex spatial and temporal patch dynamics that are not generally considered in metapopulation models (Leibold *et al.* 2004; Mihaljevic 2012). Most notably, patch dynamics (i.e. births and deaths of host individuals) are often coupled with pathogen community composition (e.g. Scenarios 4, 5 and 7 in Table 2; Mihaljevic 2012), whereas most metacommunity models assume either that patches comprise a static landscape (Box 1) (e.g. Scenarios 1–3 in Table 2; α_0, v_S, v_A, v_B, $v_X = 0$; Leibold *et al.* 2004; Pillai *et al.* 2011) or that patches are lost and gained independent of their resident communities (e.g. Scenario 4 in Table 2). In addition, the same community composition can differ in its effects on the dynamics of different host species. For example, the degree of synergistic mortality resulting from coinfection can vary widely among host species (e.g. Scenarios 5, 7 in Table 2; v_A, v_B, $v_X > v_S$; kA, k_B, $k_X < 1$; Hersh *et al.* 2012). Synergistic host mortality occurs in many plant and animal multipathogen infections and generally decreases the region of coexistence of pathogens, lowering coinfection rates (Box 3; Fig. 5) (Vasco *et al.* 2007; Seabloom *et al.* 2009). Thus, the coupling between patch (host) dynamics and the communities colonising the patches represents a novel, dynamically important feature of pathogen metacommunities.

The coupling of pathogen and host dynamics creates a situation in which pathogens compete for susceptible hosts, which would be analogous to species competing for entire habitat patches in a metacommunity rather than microsites within a patch. The classical SIR model predicts that, in the absence of stabilising mechanisms, the pathogen species that can persist with the lowest number of susceptible hosts will be the dominant competitor in the pathogen community (Dwyer *et al.* 1990). Pathogens that elicit similar immune responses can cause cross-protection in hosts (e.g. Scenario 2 in Table 2; Box 3), thereby decreasing the susceptibility of a host to infection by a second pathogen (Anderson & May 1991; Ferguson *et al.* 1999). These immune-system-mediated priority effects can lower prevalence of both individual pathogen species and coinfections as a result of pathogens pre-empting individual hosts (Seabloom *et al.* 2009) (Fig. 5). More dissimilar pathogens also can compete with each other through ecological interference, which occurs when infection-induced mortality (Scenarios 5, 7 in Table 2; v_A, v_B, $v_X > v_S$), quarantine or behaviorial changes caused by one pathogen removes hosts from a competitor's susceptible host pool (Rohani *et al.* 1998).

The interactions between infection and host dynamics create an opportunity for competitive interactions to occur at the whole-host scale. Equalising and stabilising mechanisms at the among-host scale can maintain diversity in spite of competition for susceptible hosts. For example, variation in serotype-specific immunity among hosts, a stabilising mechanism, promotes a high diversity of pneumococcal serotypes within human populations (Cobey & Lipsitch 2012). However, this stabilising mechanism must be paired with non-specific immunity, an equalising mechanism that disproportionately impedes better competitors, for models to predict observed levels of diversity (Cobey & Lipsitch 2012). More generally, if cross-protection is stronger within pathogen species than between them, stable coexistence among hosts is possible (Colijn *et al.* 2010).

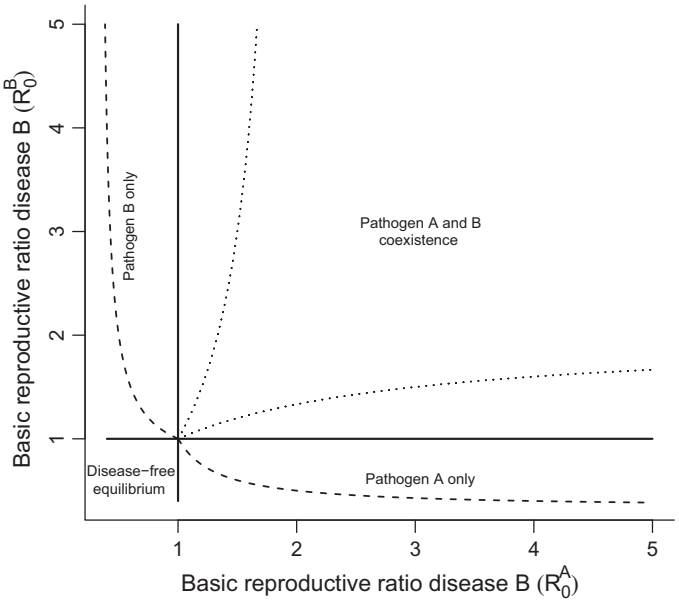

Figure 5 R_0 parameter space determining two-pathogen coexistence (a modified version of Fig. 1, Vasco *et al.* 2007). The invasibility criteria used to produce this figure are derived from the two-pathogen SI model with density-independent host recruitment and frequency-dependent transmission (Box 2). The solid lines indicate that in the absence of interactions between pathogens, coexistence is possible given each species' R_0 is greater than 1 ($\alpha_0 = v_S = v_A = v_B = v_X = 0.025$, $k_A = k_B = k_X = 1$, $q = 1$, $\psi = 1$). With immunosuppression, the coexistence region increases (dashed line, $\psi = 3$). Both cross-protection and ecological interference decrease the coexistence region (dotted line, $\psi = 0.5$ or $v_A = v_B = v_X = 0.05$, respectively).

Interactions with the host immune system also create unique opportunities for positive feedbacks and facilitation between pathogens within host patches. For example, there are a variety of mechanisms by which coinfection can lead to increased rates of reproduction and transmission. Viral pathogens can have higher reproductive rates in coinfected hosts, as is the case for human immunodeficiency viruses (Tirado & Yoon 2003). Pathogens also may lower the efficiency of the host immune system, and the resulting immunosuppression may increase the probability a host will acquire a secondary infection, thereby increasing coinfection rates (e.g. Scenario 2 in Table 2; Fig. 5) (Vasco *et al.* 2007; Seabloom *et al.* 2009).

SYNTHESIS AND FUTURE DIRECTIONS

The synthesis of community and disease ecology has significantly advanced our understanding of the impacts of host diversity and community composition on single pathogen systems (Keesing *et al*. 2006), and we suggest that there is a need for a similar synthesis focused on understanding the dynamics of spatially structured, multipathogen communities. For example, in spite of several well-studied multi-host disease models (Dobson 2004; Keesing *et al*. 2006; Begon 2008), it remains unknown how often spatial aggregation of multiple host species exacerbates or dampens the prevalence of vector-transmitted pathogens and coinfection, or how localised pathogen transmission may feed back onto and potentially alter competitive interactions among multiple host species. These spatial interactions will be further complicated when applied to mobile host or vector species in which transmission networks change through time. Using theory to guide a deeper empirical understanding of diverse pathogen communities in spatially structured, multihost systems remains a fruitful area for future research.

This synthesis further suggests that the spatial scale of ecological patterns in evolutionary models is an important avenue for future research. Maintenance of species diversity in local communities may rely on the spatial scale at which dispersal and inter-specific interactions occur and on the richness of the species pool at large spatial scales (Dove & Cribb 2006; Seabloom *et al*. 2010). Urban & Skelly (2006) link ecological and evolutionary processes within the evolving metacommunity concept, and this more synthetic perspective may clarify how local adaptation, gene storage and rescue effect, can influence species interactions, coexistence and speciation in spatially structured landscapes. This approach will be particularly relevant for investigation of microbial metacommunities because of the rapid rate of microbial evolution.

Empirical data to test theoretical predictions are lacking in several areas. In particular, ecological drift has received surprisingly little attention as a process underlying spatial patterns in pathogen communities. Similarly, although stabilising mechanisms are essential for maintaining coexistence among multiple species competing for shared resources (Chesson 2000; Adler *et al*. 2007), little work to date has focused on identifying within-host stabilising mechanisms for pathogens (but see Cobey & Lipsitch 2012; Colijn *et al*. 2010 for among-host coexistence). Overall, the extensive research on competition and coexistence among free-living organisms provides insights into how we expect pathogens to interact, but ecologically motivated studies on pathogens are necessary to bridge these fields. In particular, there is a need for interaction experiments among multiple pathogens (e.g. Lacroix *et al*. 2014) that allow the quantitative parameterisation and testing of multipathogen metacommunity and SIR models (e.g. Boxes 1 and 2).

Metacommunity models are well suited to examine the relative importance of these fundamental processes and will enhance our ability to predict the outcome of multipathogen-host interactions in diverse communities. However, many pathogen interactions are mediated through interactions with host demography and immune systems, and these dynamical linkages have not yet been incorporated into the metacommunity approach. For this reason, existing metacommunity models will be most relevant for diverse communities of weakly virulent pathogens and for dynamics that occur at faster rates than host dynamics (e.g. non-lethal childhood diseases). Coupled host–pathogen dynamics are a defining characteristic of SIR models in disease ecology; however, these models become intractable for diverse, stochastic and spatially structured communities. Thus, neither of these two modelling frameworks, SIR models from disease ecology and the stochastic metacommunity models from community ecology, is sufficient for understanding the full dynamics of pathogen communities. However, these approaches are complementary, and each has advantages depending on the problem at hand. While it is unlikely a single modelling approach would ever be sufficient to understand the full complexity of diverse natural pathogen communities whose dynamics play out across a stochastic mosaic of abiotic and biotic forces, this review demonstrates that a more unified conceptual modelling framework that draws on the strengths of each approach will lead to a deeper understanding of the complexity of pathogen community dynamics.

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AUTHORSHIP

EWS wrote the first draft of the manuscript, and all authors contributed substantially to revisions.

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