The diversity–disease relationship: evidence for and criticisms of the dilution effect

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SUMMARY

The dilution effect, that high host species diversity can reduce disease risk, has attracted much attention in the context of global biodiversity decline and increasing disease emergence. Recent studies have criticized the generality of the dilution effect and argued that it only occurs under certain circumstances. Nevertheless, evidence for the existence of a dilution effect was reported in about 80% of the studies that addressed the diversity–disease relationship, and a recent meta-analysis found that the dilution effect is widespread. We here review supporting and critical studies, point out the causes underlying the current disputes. The dilution is expected to be strong when the competent host species tend to remain when species diversity declines, characterized as a negative relationship between species' reservoir competence and local extinction risk. We here conclude that most studies support a negative competence–extinction relationship. We then synthesize the current knowledge on how the diversity–disease relationship can be modified by particular species in community, by the scales of analyses, and by the disease risk measures. We also highlight the complex role of habitat fragmentation in the diversity–disease relationship from epidemiological, evolutionary and ecological perspectives, and construct a synthetic framework integrating these three perspectives. We suggest that future studies should test the diversity–disease relationship across different scales and consider the multiple effects of landscape fragmentation.

Key words: competence–extinction relationship, identity effect, infection prevalence, abundance, amplification effect, habitat fragmentation.

THE DILUTION EFFECT: A HYPOTHESIS UNDER DEBATE

Host–pathogen interactions can be strongly affected by species diversity and community composition (Keesing *et al*. 2006; Ostfeld and Keesing, 2012). Understanding how species diversity influences pathogen transmission has long been a central question in disease ecology (Keesing *et al*. 2006). In particular, assessing the role of species diversity in pathogen transmission is critical for predicting disease dynamics in the context of global biodiversity decline, and may provide valuable insights into disease interventions and control measures.

Several studies have developed the 'dilution effect hypothesis' that high host species diversity can reduce the risk of pathogen transmission (Norman *et al*. 1999; Ostfeld and Keesing, 2000; Schmidt and Ostfeld, 2001; Allan *et al*. 2003; Johnson and Thieltges, 2010). This negative diversity–disease relationship, representing an exciting convergence of conservation and public health interests (Randolph and Dobson, 2012; Young *et al*. 2013; Wood *et al*. 2014), has been reported in various parasite systems, such as hantavirus (Clay *et al*. 2009*b*; Suzán *et al*. 2009), Lyme disease (Ostfeld and Keesing, 2000; LoGiudice *et al*. 2003; Turney *et al*. 2014; Werden *et al*. 2014), West Nile virus (Swaddle and Calos, 2008; Allan *et al*. 2009), schistosomiasis (Johnson *et al*. 2009), *Ribeiroia ondatrae* (Johnson *et al*. 2013*b*), *Phytophthora ramorum* (Haas *et al*. 2011) and so on. According to recent reviews (Cardinale *et al*. 2012; Ostfeld and Keesing, 2012), the negative diversity–disease relationship occurs in about 80% of the studied cases (85% in studies on plant diseases and 67% in studies on animal diseases). A recent meta-analysis also provided broad evidence that host diversity inhibits parasite abundance in various functional groups of parasites (Civitello *et al*. 2015*a*). In fact, disease dilution has even been indicated as a general ecosystem service of biodiversity in several studies (Ostfeld and Keesing, 2012). However, despite this overwhelming support, some studies still criticize the generality of the dilution effect and argue that a publication bias in favour of the dilution effect may influence the magnitude of species diversity effects on disease risk (Randolph and

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Dobson, 2012; Civitello *et al*. 2015*b*; Salkeld *et al*. 2015). They consider the dilution effect to be idiosyncratic and only occur under certain conditions (Begon *et al*. 2008; Ogden and Tsao, 2009; Randolph and Dobson, 2012; Salkeld *et al*. 2013; Wood and Lafferty, 2013; Mihaljevic *et al*. 2014; Wood *et al*. 2014). This diversity–disease relationship debate has attracted much attention, partly due to current interest in identifying utilitarian functions of biodiversity (Bonds *et al*. 2012; Mace *et al*. 2012; Randolph and Dobson, 2012; Ostfeld, 2013; Wood *et al*. 2014). In addition, only a few studies have discussed the role of habitat fragmentation in determining the diversity–disease relationship; this knowledge gap is surprising, since habitat fragmentation has been considered a main driver of species loss (Fahrig, 2003). Considering the current ongoing habitat loss and fragmentation (Mitchell *et al*. 2015), it is imperative to get a better understanding of the effects of habitat fragmentation on host–pathogen interactions.

Here, we review previous studies on the dilution effect and point out what the basis is of the current disputes. By investigating the proposed three prerequisites for the dilution effect, we explore both the evidence and critiques, trying to reconcile them and indicate the gaps in our knowledge. We then explore the role of habitat fragmentation in the diversity–disease relationship, extending the discussion to fragmented landscapes. We illustrate that the complex role of habitat fragmentation may lead to a complicated diversity–disease relationship and suggest that future studies should take into account the role of landscape fragmentation.

THREE PREREQUISITES FOR THE DILUTION EFFECT

Recent studies investigating the dilution effect of species diversity have proposed that the generality of this hypothesis relies on the three following prerequisites (Keesing *et al*. 2006; Ostfeld and Keesing, 2012; Young *et al*. 2013; Johnson *et al*. 2015):

P1: Host species differ in their reservoir competence (i.e. the potential of a species to support and transmit pathogens, see Glossary Box. 1).

P2: Low-competence hosts in the community can reduce, through different kinds of mechanisms, pathogen transmission.

P3: Competent hosts are relatively resilient to local species loss, whereas low-competence hosts are relatively vulnerable and occur mainly in more diverse communities.

It seems that the validity of the first prerequisite has been accepted by consensus. Interspecific variation in reservoir competence is primarily attributed to differences in contact rates and susceptibility (the

Box 1. Glossaries.

Amplification effect: A phenomenon that high species diversity can increase disease risk.

Basic reproductive number (R_0): the expected number of secondary cases caused by the first infectious individual in a wholly susceptible population.

Complementarity effect: A non-additive mechanism underlying the influence of biodiversity on ecosystem function. It occurs when biodiversity improves ecosystem performance through positive interspecific interactions or niche partitioning (Loreau and Hector, 2001; Hooper *et al*. 2005).

Connectivity: The degree to which a landscape facilitates the movement of organisms and matter (Mitchell *et al*. 2015).

Density-dependent transmission: Transmission in which the number of new infections per unit time is proportional to the product of the density of infected hosts and the density of susceptible hosts (McCallum *et al*. 2001).

Encounter reduction: A mechanism for the dilution effect. The addition of low-competence host species can reduce the encounter rates between competent hosts or between competent hosts and vectors, and thus, reduce disease risk (Keesing *et al*. 2006).

Frequency-dependent transmission: Transmission in which the number of new infections per unit time is proportional to the product of the proportion (or frequency) of infected hosts and the density of susceptible hosts (McCallum *et al*. 2001).

Habitat fragmentation: The process that leads to discontinuities in the habitat of a species. Habitat fragmentation often results from human activities, such as land conversion (Fahrig, 2003).

Identity effect: The presence of a species in a community with a particularly higher or lower than average contribution to ecosystem functioning. The identity effect can be caused by selection effect or by sampling effect. Positive or negative selection occurs when a particular species increases or decreases, respectively, its relative abundance in diverse communities, due to its higher or lower than average performance in a monoculture. A sampling effect results from the increased probability that a particular species will be present in a species-rich community (Loreau and Hector, 2001).

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Local extinction or **extirpation:** The absence of a species (or a taxon) in a geographic area of study; however, it continues to exist elsewhere.

Metapopulation: A group of spatially separated populations of the same species that interact at a higher spatial level. The concept has been widely applied to species in fragmented habitats.

Prevalence: The proportion of a population found to have a condition (typically a disease here).

Reservoir competence: The potential of a species to support and transmit pathogens (Huang *et al*. 2013*a*). Reservoir competence can be a function of several epidemiological parameters, namely host susceptibility (probability of a host becoming infected by infected vectors), host infectivity (probability of a vector becoming infected, when feeding on an infected host) and duration of infectiousness (how long that a host remains infectious).

Susceptible host regulation: A mechanism for the dilution effect, where the addition of low-competence host species can reduce the abundance of competent hosts, and thus, reduce disease risk (Keesing *et al*. 2006).

competence of becoming infected) or infectiousness (the competence of infecting other hosts) (Kilpatrick *et al*. 2006; Cronin *et al*. 2010; Huang *et al*. 2013*a*). However, the other two prerequisites (P2, P3) have been criticized by several studies arguing that they are often not fulfilled (Randolph and Dobson, 2012; Joseph *et al*. 2013; Salkeld *et al*. 2013; Wood and Lafferty, 2013; Young *et al*. 2013; Wood *et al*. 2014).

DILUTION OR AMPLIFICATION: INDICATOR OF DISEASE RISK MIGHT MATTERS

An important prerequisite (P2, Fig. 1) for the generality of the dilution effect is that the low-competence host species can reduce disease risk. This could happen through different mechanisms, such as encounter reduction and susceptible host regulation (Keesing *et al*. 2006). Scientists usually use the prevalence and/or abundance of infection (either infected hosts or infected vectors) to measure disease risk and describe the dynamics of pathogen transmission in empirical studies. It has been demonstrated that these two different indicators of disease risk may respond differently to changes in risk factors, including species diversity (Table 1). Thus, the first criticism of the dilution effect (C1, Fig. 1) is that low-competence hosts may have opposite effects on infection prevalence and abundance (Roche *et al*. 2012; Huang *et al*. 2015).

When testing the dilution effect of species diversity, many studies used infection prevalence to measure disease risk (Schmidt and Ostfeld, 2001; LoGiudice *et al*. 2003; Ezenwa *et al*. 2006; Suzán *et al*. 2009). For example, the prevalence of Sin Nombre virus infections in deer mice was reported to negatively correlate to rodent species diversity in the Great Basin Desert of America (Clay *et al*. 2009*a*). However, in considering public health, a more suitable indicator for disease risk in humans might be the abundance of infected hosts/vectors (Roche *et al*. 2012; Wood and Lafferty, 2013), because wildlife–human contacts are typically density-dependent (Roche *et al*. 2012). The abundance of infection, as the product of infection prevalence and the host/vector population size, may increase with increasing host/vector population even if the infection prevalence decreases (Ogden and Tsao, 2009; Wood *et al*. 2014). For example, a theoretical study incorporating empirical laws of community assemblage found that high species richness could reduce the infection prevalence of directly transmitted pathogens, while the number of total infected hosts in the community increases due to the increasing host abundance (Roche *et al*. 2012). In a previous modelling study, we tested the effect of habitat fragmentation on disease risk in metapopulations, and demonstrated that two disease risk indicators (prevalence and number of infected hosts) changed differently as connectivity increased (Huang *et al*. 2015). We found that the risk indicator most likely to be negatively correlated to connectivity and species richness depended on the mechanism underlying the dilution effect (susceptible host regulation or encounter reduction) and its strength (Huang *et al*. 2015).

The importance of distinguishing the abundance and prevalence of infection (C1, Fig. 1) is most relevant for vector-borne diseases, because low-competence host species present in highly diverse communities may also influence disease risk by affecting (either amplifying or regulating) vector populations (Randolph and Dobson, 2012; Wood *et al*. 2014). High species diversity may increase the total host abundance, or increases the main vector-food-source species, providing more blood meals for invertebrate vectors and increasing vector abundance. Thus, the abundance of infected vectors, an important indicator for human disease risk, could increase with increasing species diversity, even when infection prevalence in vectors decreases (Schmidt and Ostfeld, 2001; Randolph and Dobson, 2012; Wood and Lafferty, 2013; Wood *et al*. 2014). When investigating the effect of community composition on the transmission dynamics of Lyme disease in different habitats of Northeastern Connecticut, for example, States *et al*. found that the infection prevalence in tick nymphs was similar between the islands (with lower species diversity) and the mainland (with higher species diversity), but the mainland had a higher density of infected nymphs, due to the higher

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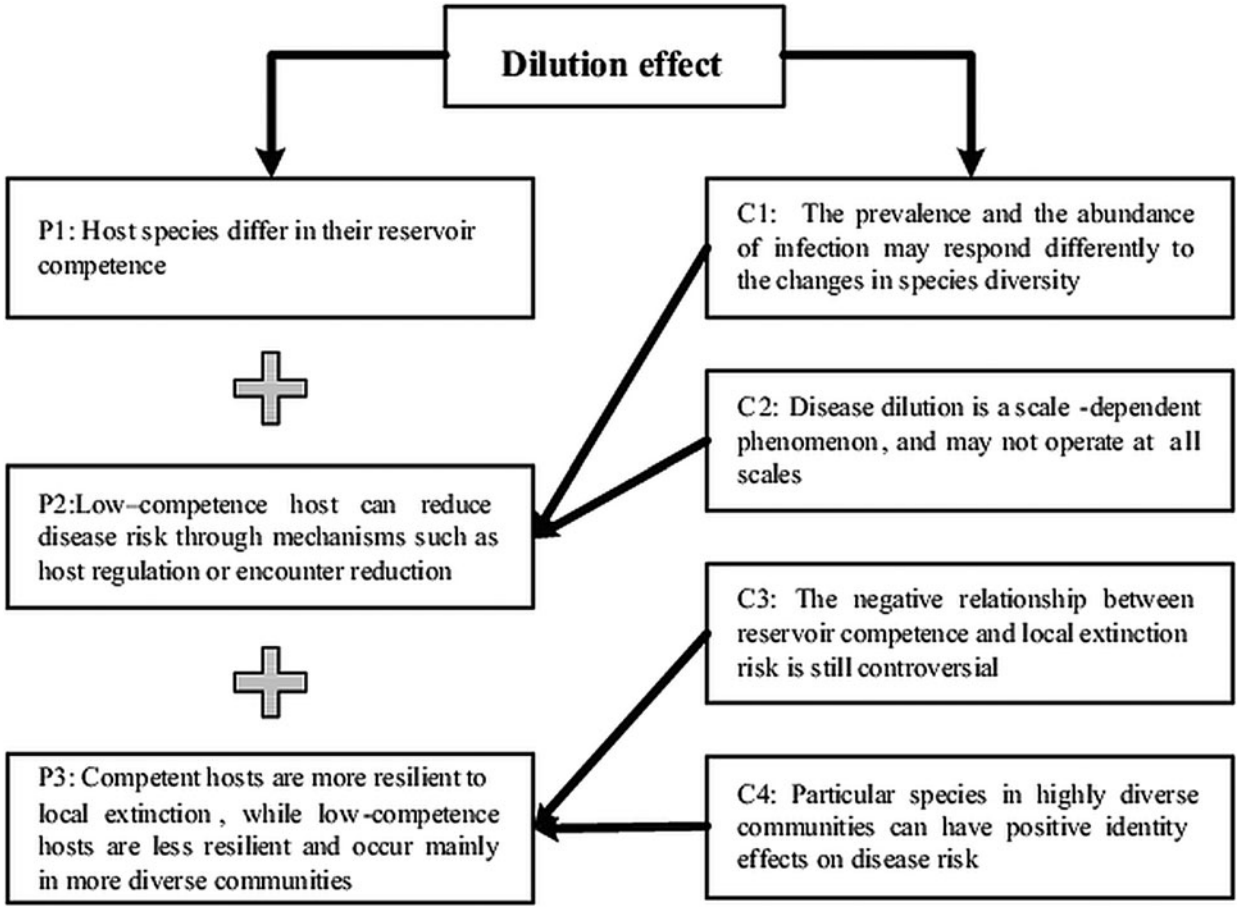

Fig. 1. Illustration of the prerequisites (P1–P3) for generality of the dilution effect and their criticisms (C1–C4). Arrows indicate corresponding prerequisites and criticisms.

Table 1. Summary of studies demonstrating that species diversity had a different relationship with the prevalence than with the abundance of infection (host or vector).

Parasite	Transmission type	Study design	Prevalence	Abundance	References
Ribeiroia ondatrae	Direct	Experiment	Host, no relationship	Host, negative	(Johnson *et al*. 2012*a*)
Andes virus	Direct	Field	Host, no relationship	Host, negative	(Piudo *et al*. 2011)
A general parasite	Direct	Theoretical	Host, negative	Host, positive	(Roche *et al*. 2012)
Bartonella spp.	Flea-borne	Field	Host, no relationship	Host, negative	(Young *et al*. 2014)
Borrelia burgdorferi	Tick-borne	Field	Tick, no relationship	Tick, positive	(States *et al*. 2014)
Borrelia burgdorferi	Tick-borne	Theoretical	Tick, non-linear	Tick, non-linear	(Ogden and Tsao, 2009)

abundance of ticks fed by more hosts on the mainland (States *et al*. 2014). On the other hand, some low-competence host species may regulate vector abundance [e.g. opossums can largely reduce tick abundance by grooming; (Keesing *et al*. 2009)], making the abundance of infected vectors decrease even when the infection prevalence in vectors increases. Therefore, for vector-borne diseases, infection prevalence and abundance are likely to show different relationships with species diversity, given that low-competence hosts may have different effects on vector abundance.

In most empirical studies, host infection prevalence and abundance were determined at a focal host which was usually referred to a most competent host species. However, disease risk can also be measured at the whole community level. For example, many theoretical studies (Dobson, 2004; Chen and Zhou, 2015) used the community basic reproductive number

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(R_0) which measures the invasion risk of a pathogen into the community. Contrasting results might also arise through using the risk measures from different levels (local host or the community level). We suggest that future studies investigating the diversity–disease relationship integrate the risk measures from empirical and theoretical approaches, as well as from the focal host and the whole community.

SCALE-DEPENDENCY OF THE DILUTION EFFECT

Recent studies have argued that low-competence host species do not always reduce the transmission risk, because the diversity–disease relationship may be scale-dependent (Moore and Borer, 2012; Wood and Lafferty, 2013). Host–pathogen interactions can be directly or indirectly driven by animal movements and species distributions, community composition, landscape structure, topography, climate, etc. (Estrada-Peña *et al*. 2014). These factors operate over a broad range of spatial scales and different ecological processes might be involved at these scales. Consequently, the impact of low-competence hosts is expected to be modified by different ecological processes across different scales and therefore scale-dependent (Moore and Borer, 2012). The critiques arise when the negative impacts of low-competence host on pathogen transmission are not observed over all scales (C2, Fig. 1).

Previous theoretical studies demonstrated that high host diversity was more likely to increase disease risk of directly transmitted pathogens (usually with a density-dependent transmission where the number of new infections per unit time is proportional to the product of the density of infected hosts and the density of susceptible hosts) if the presence of low-competence hosts cannot regulate the densities of competent hosts (Dobson, 2004; Rudolf and Antonovics, 2005; Joseph *et al*. 2013). When considering transmission at larger spatial scale (e.g. population level or landscape level), contact rates between populations (or animal herds) may remain stable due to spatial constraints and be independent of population size (or herd size), thus transmission between population or herds will be frequency-dependent (Roy and Pascual, 2006). In this way, although species diversity may increase disease risk at local scale, the dilution effect may operate at larger scales through the encounter reduction mechanism. For instance, additional host species generally amplified bovine tuberculosis risk of cattle at local scales (Hardstaff *et al*. 2013). However, a recent study found that high mammalian species richness was negatively associated with bovine tuberculosis risk in cattle at the regional scale in Africa, and proposed that low-competence mammalian species may reduce the contact rates between cattle herds by acting as physical barriers to herd movements (Huang *et al*. 2014).

For vector-borne pathogens, vector dispersal and distribution play important roles in host–pathogen interactions (Moore and Borer, 2012). Host community configurations over large spatial scales can affect vector dispersal and distribution, and thus mediate the influence of species diversity on host–pathogen interactions at local scale (Plantegenest *et al*. 2007). Upon reviewing the effect of biodiversity on Lyme disease risk, Wood and Lafferty (2013) argued that the dilution effect might operate at fine levels (e.g. communities in forests) under some circumstances; but typically, biodiversity was positively correlated with disease risk at the landscape or larger scale (urban to suburban to rural). They considered that habitats with relatively high species diversity, particularly when deer are present, can provide more blood meals for ticks, and thus increase the density of infected questing ticks. However, a recent study found that both host species richness and forest cover were negatively associated with Lyme disease incidence at the regional scale across America (Turney *et al*. 2014), which is not in agreement with the predictions of Wood and Lafferty (2013). This scale-dependency may also apply to some mosquito-borne diseases, such as malaria. For example, several studies have demonstrated the efficacy of zooprophylaxis, a phenomenon related to the dilution effect, where domestic animals are used near a house to attract mosquitoes away from humans and reduce the risk of malaria in humans (Nah *et al*. 2010). At the landscape scale, however, elevated mosquito densities, supported by the zooprophylactic animals, may increase the average malaria risk in the region (Dobson *et al*. 2006).

A recent review (Johnson *et al*. 2015) concluded that the dilution effect generally operates at local community scale, while at regional scale high species richness is usually positively correlated with parasite richness or disease diversity due to the 'diversity begets diversity' hypothesis (Hechinger and Lafferty, 2005). However, as parasite richness is different to the risk of a specific disease, and their relationship can be either positive (Read and Taylor, 2001) or negative (Johnson and Hoverman, 2012; Johnson *et al*. 2013*a*), more efforts are needed to test the effect of host diversity on the risk of a specific disease at regional scale. To conclude, the diversity–disease relationship can be scale-dependent and future studies should take into account the spatial scale of analysis to avoid misinterpretations.

THE CONTROVERSIAL COMPETENCE–EXTINCTION RELATIONSHIP

Another central prerequisite (P3 in Fig. 1) for the generality of the dilution effect is a negative competence–extinction relationship (Ostfeld and Keesing, 2012; Randolph and Dobson, 2012; Joseph *et al*. 2013; Young *et al*. 2013). That is, low-competence host species (with low reservoir competence) are

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most vulnerable to local extinction with increasing species loss; while competent species tend to survive in the community (Ostfeld and Keesing, 2012; Joseph *et al*. 2013). The importance of the competence–extinction relationship in determining the direction of the diversity–disease relationship was analysed over a decade ago when the dilution effect hypothesis was first proposed (Schmidt and Ostfeld, 2001; LoGiudice *et al*. 2003). Nevertheless, only recent studies have focused on testing, directly or indirectly, this competence–extinction relationship (Rubio *et al*. 2014), and the results of those studies have triggered a critical debate over the generality of the dilution effect (C3 in Fig. 1).

It is hypothesized that the negative competence–extinction relationship arises from two alternative (but non-exclusive) mechanisms: the life-history theory and the parasite local adaptation theory (Joseph *et al*. 2013). According to the life-history theory, short-lived species tend to invest less in adaptive immunity, making them less resistant and more susceptible for pathogens. On the other hand, they usually have higher fecundity and population densities, and thus are more resilient to local extinction in disturbed communities (Blackburn *et al*. 1993; Cardillo, 2003). Previous empirical studies have found that life span has a negative relationship with host's susceptibility in the amphibian parasite, *R. ondatrae* (Johnson *et al*. 2012*b*), *Trypanosoma cruzi* parasite (Gottdenker *et al*. 2012), Barley yellow dwarf virus (Cronin *et al*. 2014) and Cucumber mosaic virus (Hily *et al*. 2014). Besides susceptibility, infectivity and duration of infectiousness also contribute to hosts' reservoir competence (Logiudice *et al*. 2008; Cronin *et al*. 2010; Huang *et al*. 2013*a*). Recent critical studies argued the short-lived species with high susceptibility may lead to a short duration of infectiousness due to pathogen-induced morbidity and mortality, limiting their potentials as reservoirs for pathogen transmission (Randolph and Dobson, 2012; Cronin *et al*. 2014; Hily *et al*. 2014). However, a negative relationship between life span and host's reservoir competence has also been found in Lyme disease (Previtali *et al*. 2012), Eastern equine encephalitis virus (Huang *et al*. 2013*a*) and West Nile virus (Huang *et al*. 2013*a*), providing evidence for this central prerequisite.

Over a decade ago, Ostfeld and Keesing proposed that a negative competence–extinction correlation could also be explained by the parasite local adaptation theory (Ostfeld and Keesing, 2000). This theory suggests that parasites may constantly evolve to exploit the most common host in response to selective pressure of losing hosts during community disassembly (Lajeunesse and Forbes, 2002). To maximize their reproductive rates in hosts, parasites may show a balance between transmissibility and pathogenicity, leading to a high reservoir competence of common hosts. However, this mechanism has only received limited attention in the diversity–disease debate (Joseph *et al*. 2013). A recent study tested the parasite adaptation theory in three tick-borne diseases, and found that host population density, taken as a proxy for encounter rates between hosts and pathogens, was positively correlated with reservoir competence (Ostfeld *et al*. 2014). However, since host population density is also highly correlated to life-history traits, it remains difficult to disentangle the importance of parasite adaptation from that of host species traits in explaining the interspecific variation in reservoir competence.

In addition to the studies on life-history theory and parasite adaptation theory, several studies directly addressed the competence–extinction relationship. For instance, when exploring amphibian community disassembly, Johnson *et al*. showed that the most competent host for the trematode *R. ondatrae* is also the most common amphibian in California ponds (Johnson *et al*. 2013*b*). These patterns in community assemblage structures were similar for four generalist aphid-vectored pathogens (Lacroix *et al*. 2014). However, using parasite richness as the proxy for reservoir competence, a meta-analysis showed no relationship between parasite richness and host resilience to human disturbances (Young *et al*. 2013). Long-lived species (Morand and Harvey, 2000) and increased host diversity (Kamiya *et al*. 2014) are usually positively correlated with parasite richness, which has been one of the most frequently used criticisms of the dilution effect (Lafferty, 2012; Young *et al*. 2013). However, some studies argued that parasite richness is not an appropriate index for measuring the host's reservoir competence and may not be positively correlated to the risk of a specific disease (Johnson and Hoverman, 2012; Johnson *et al*. 2013*a*; Johnson *et al*. 2015). For example, higher plant diversity promoted the diversity of fungal pathogens, while it reduced pathogen infection per plant in a long-term, large-scale, biodiversity experiment (Rottstock *et al*. 2014). Parasites can interact with one another both directly and indirectly. With increasing parasite diversity, the risk of a specific disease can either increase due to the suppression on host immunity (Read and Taylor, 2001) or decrease because of the competition between co-infecting parasites (Johnson and Hoverman, 2012; Johnson *et al*. 2013*a*).

Despite these direct or indirect empirical supports, some studies pointed out that many stochastic and demographic factors could influence community assemblage structure (Hooper *et al*. 2005) or obscure local adaptation patterns (Kaltz and Shykoff, 1998), and these confounding factors may weaken the negative competence–extinction relationship. Variation around the negative competence–extinction correlation may cause host species diversity to have inconsistent effects on disease risk, making the dilution

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effect disappear, or lead to a positive correlation between species diversity and disease transmission (Schmidt and Ostfeld, 2001; LoGiudice *et al*. 2003; Joseph *et al*. 2013). However, the broad evidence for the dilution effect provided in a current rigorous meta-analysis study (Civitello *et al*. 2015*a*) indicates that the negative competence–extinction relationship might occur widely.

IDENTITY EFFECT FROM PARTICULAR SPECIES

Studies criticizing the dilution effect also argued that pathogen transmission might increase in high-diversity communities due to the increased chance of including a particular species that has a positive effect on pathogen transmission (Randolph and Dobson, 2012; Wood and Lafferty, 2013). This positive effect on pathogen transmission, recently termed as the identity effect (Hantsch *et al*. 2013; Huang *et al*. 2014), may mask the dilution effect and even lead to a positive diversity–disease relationship. The identity effect, together with the complementarity effect, was first proposed to explain how biodiversity influences ecosystem functioning (BEF) (Loreau and Hector, 2001). A complementarity effect occurs when high levels of biodiversity improve ecosystem performance (e.g. productivity) through positive interspecific interactions or niche partitioning (Loreau and Hector, 2001; Hooper *et al*. 2005). The identity effect was defined as the presence of a key species in high-diversity communities with a particular higher or lower than average contribution to ecosystem functioning (Hooper *et al*. 2005). The identity effect can be caused by either a sampling effect, where increasing species diversity results in an increased probability of the presence of a key species, or a selection effect where on average, species that perform higher or lower than average in the monoculture increase their relative abundance in diverse communities (Loreau and Hector, 2001; Hantsch *et al*. 2013).

Studies have demonstrated that the identity effect of a particular species indeed plays an important role in determining the diversity–disease relationship (Hantsch *et al*. 2013; Becker *et al*. 2014; Huang *et al*. 2014). For example, when investigating the effect of tropical amphibian species diversity on the infection load of *Batrachochytrium dendrobatidis*, Becker *et al*. (2014) detected an identity effect (resulted from a selection effect) that one particular terrestrial species showed reduced infection loads in diverse assemblages at the expense of neighbouring aquatic hosts becoming heavily infected. Generally, the identity effect on pathogen transmission can be observed in two different situations. One is that a key species with particular high or low reservoir competence may present in communities when species diversity increases. This can occur if the negative competence–extinction relationship is violated by stochastic, demographic or phylogenetic confounding factors. For example, a recent study on bovine tuberculosis detected a negative effect of mammal species richness on disease risk (Huang *et al*. 2013*b*, 2014). However, the occurrence of African buffalo (*Syncerus caffer*), which was positively correlated with mammal species richness, increased disease risk due to its high transmission competence (Huang *et al*. 2013*b*, 2014). The other situation is where a species can affect vector abundance (either positive or negative) or the life cycles of free-living parasites (e.g. helminth). The identity effect on pathogen transmission can be either positive (Hantsch *et al*. 2013; Huang *et al*. 2014) or negative (Becker *et al*. 2014). For example, the presence of white-tailed deer (*Odocoileus virginianus*), an important blood meal host for adult ticks, can largely increase tick abundance and thus amplify the risk of tick-borne diseases, such as Lyme disease (Bouchard *et al*. 2013). On the contrary, since opossum can largely reduce tick abundance by grooming (Keesing *et al*. 2009), the presence of opossum in diverse communities may create a negative identity effect. When the identity effect is negative, it may enhance the strength of the negative diversity–disease relationship; when the identity effect is positive, it may weaken the negative diversity–disease relationship and lead to critique (C4 in Fig. 1) with regard to the existence of a dilution effect.

If disease dilution is considered an ecosystem service of biodiversity as several previous studies indicated, then we can extend the concepts about the identity effect in BEF to pathogen transmission. In this way, the strength of disease dilution can be determined by two effects, the identity effect and the effect of species diversity *per se*. Here, the effect of species diversity *per se* is equivalent to the complementarity effect in previous BEF studies, as it also operates through interspecific interactions (such as the abovementioned susceptible host regulation mechanism) or niche partitioning (such as encounter reduction where shared habitat use and pathogen transmission are reduced). In fact, recent studies have shown that the identity effect and the effect of species diversity *per se* can operate simultaneously (Hantsch *et al*. 2013; Huang *et al*. 2014; Venesky *et al*. 2014), which was also consistent with previous BEF studies regarding the effect of species diversity on ecosystem productivity (Loreau and Hector, 2001) and nutrient retention (Reich *et al*. 2001). For example, Hantsch *et al*. (2013) examined the effects of tree species richness on the risk of foliar fungal pathogens in Germany, and found that tree species richness was negatively correlated with the pathogen load of common mildew species, but the presence of *Quercus*, a particularly disease-prone species, was correlated with a high pathogen load at plot level. Here, we extended the previous

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concepts derived from studies on BEF to pathogen transmission. This theoretical framework will be beneficial to reconcile the disputes with regard to the existence of the dilution effect, because the dilution effect then will be restricted to the effect of species diversity *per se*, and the identity effect, as another important component in the diversity–disease relationship, can be disentangled from the dilution effect.

MISSING LINK BETWEEN HABITAT FRAGMENTATION AND DIVERSITY–DISEASE RELATIONSHIP

Habitat fragmentation driven by anthropogenic factors has been considered as one of the primary drivers of species loss (Fahrig, 2003). Habitat fragmentation also considerably alters host–pathogen interactions by modifying host movements, host/vector distribution, micro-environments, and certainly, species diversity and interactions (Estrada-Peña *et al*. 2014). Therefore, habitat fragmentation can potentially influence the diversity–disease relationship. However, only a few studies investigated the role of habitat fragmentation in the diversity–disease relationship (Suzán *et al*. 2012, 2015), and these studies showed contrasting results, even in the same disease system (Salkeld *et al*. 2013). For example, both the prevalence and abundance of infected ticks for Lyme disease were positively correlated with habitat fragmentation in New York State (Allan *et al*. 2003), and this relationship was also found for infection prevalence of ticks in Connecticut (Brownstein *et al*. 2005). However, when the study area was extended across New York, New Jersey and Connecticut, the positive relationship was inconsistent [only detected in one of two study periods (Logiudice *et al*. 2008)]. These conflicting results indicated that habitat fragmentation may play a complex role in pathogen transmission.

Habitat fragmentation has been demonstrated to affect host–pathogen interactions from ecological, epidemiological and evolutionary perspectives (Fig. 2). From an epidemiological perspective, increasing connectivity (increased host movements and contact rates among host populations) typically increases the incidence and severity of a disease (Hess, 1996). However, sometimes it reduces the probability of pathogen persistence by generating large epidemics that deplete susceptible individuals (Keeling, 2000). From an evolutionary perspective, studies have shown that increased connectivity can generate higher genetic diversity within a host population (Jousimo *et al*. 2014). This mechanism provides higher variation in host resistance to be selected for (Carlsson-Granér and Thrall, 2002; Jousimo *et al*. 2014), which decreases disease risk. Host populations with a single genotype usually exhibit high disease risk (Zhu *et al*. 2000; Mundt, 2002; Altermatt and Ebert, 2008). Finally, from an ecological perspective, connectivity in a human-driven fragmented landscape typically modifies host diversity and composition (Loreau and Mouquet, 1999), and thus can influence disease risk via the effect of species diversity *per se* or the identity effect (Allan *et al*. 2003; Keesing *et al*. 2006). For vector-borne diseases, habitat fragmentation also modifies disease risk through an effect on vector abundance (Estrada-Peña *et al*. 2014). Increased host movements in more connected habitats may increase vector abundance, and thus, promote disease risk (Estrada-Peña, 2003). In addition, higher genetic and species diversity in the host are expected to increase the genetic and species diversity of the parasite by enhancing colonization opportunities (Hechinger and Lafferty, 2005). Species diversity and genetic diversity of the parasite can affect disease risk, either positively, by suppression on host immunity (Read and Taylor, 2001), or negatively, via intra-host competition among co-infecting parasites (Johnson and Hoverman, 2012; Johnson *et al*. 2013*a*).

To predict the impact of fragmentation on disease dynamics, it is critical to understand the role of habitat fragmentation in pathogen transmission and the diversity–disease relationship. A few recent studies have recognized the necessity of integrating the abovementioned three perspectives to explore the diversity–disease relationship in fragmented landscapes (Jousimo *et al*. 2014; Huang *et al*. 2015). Based on previous empirical and theoretical studies, we constructed a synthetic framework (Fig. 2) to integrate these different perspectives (epidemiological, evolutionary and ecological). The three perspectives are not equally important in a given disease system. Moreover, these three perspectives can generate many paths through which habitat fragmentation influences pathogen transmission (Fig. 2). These paths construct a complex causality web; and the loading effects vary in their directions and strengths, making it difficult to predict the net effect of fragmentation. The net effect of fragmentation on disease risk therefore depends on the relative importance of these paths. For example, a recent theoretical study exploring the effect of habitat connectivity on disease risk in metapopulations found that disease risk was non-linearly related to connectivity, and the net impact of connectivity depended on the relative strength of the dilution effect (via increasing species diversity in ecological perspective) *vs* the facilitation effect (via increasing host movements among patches in epidemiological perspective (Huang *et al*. 2015). When mapping the risk of Lyme disease in northern Spain, Estrada-Peña demonstrated that the density of questing ticks was higher in sites with higher spatial connectivity, presumably due to increased host movements through these patches (Estrada-Peña, 2003). This vector amplification effect can counteract the potential

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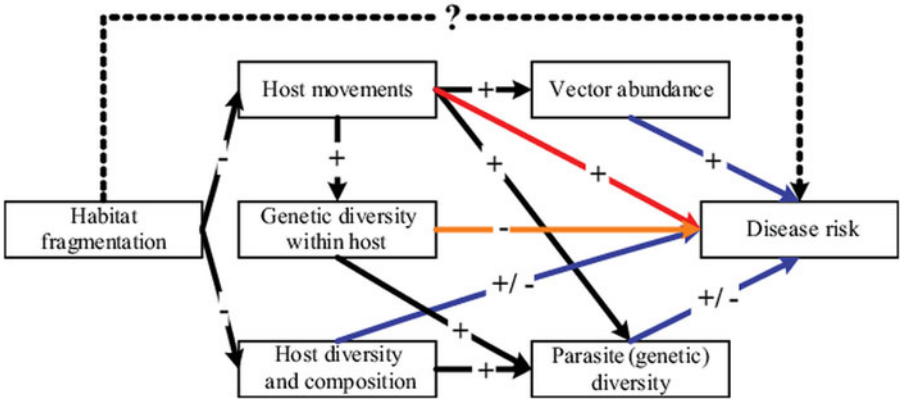

Fig. 2. Path diagram for how habitat fragmentation influences disease risk. Factors can directly influence disease risk via ecological processes (blue arrows), epidemiological processes (red arrow) and evolutionary processes (orange arrow); or factors indirectly affect disease risk through other factors (black arrows). For each path, the sign (from empirical studies) indicates whether the relationship between two factors is positive, negative or either.

dilution effect in habitats with high connectivity (Estrada-Peña, 2009). These studies highlight the importance of integrating the different perspectives when testing the diversity–disease relationship in fragmented landscapes.

CONCLUDING REMARKS AND FUTURE DIRECTIONS

This review summarized and discussed the evidence for and criticisms of the prerequisites for the dilution effect. We concluded that most empirical studies support a negative competence–extinction relationship. However, variations in this relationship may reduce the generality of the dilution effect. The extremes might be the result of an identity effect (either positive or negative) from particular species. We suggested to extending previous concepts derived from the effect of biodiversity on ecosystem functioning to pathogen transmission and using the identity effect as an additional mechanism to better understand the effect of species diversity on disease risk. In addition, previous empirical studies supported the notion that the shape and direction of the diversity–disease relationship can depend on the scale of analyses and on the indicator used to measure disease risk (e.g. the prevalence and/or abundance of infection). We argued that many of the current disputes over the dilution effect could be settled by taking into account the identity effect of particular species, the scale of analyses, and the risk indicator used. In addition, we believed that fruitful research will come from assessing the diversity–disease relationship and determining the key factors across different spatial scales. Those studies would not only increase our understanding about the generality of the dilution effect, but also be relevant for developing intervention and control measures at different scales (Glavanakov *et al*. 2001). Both field surveys at different spatial scales and meta-analyses of published information should be fruitfully informative to this point.

We highlighted the complex role of habitat structure on host-pathogen interactions, and constructed a synthetic framework that integrated ecological, epidemiological and evolutionary perspectives. The paths generated from these three perspectives construct a complex causality web (Fig. 2). The correlations or causalities from one perspective can be confounded by factors from other perspectives. Consequently, the diversity–disease relationship might be obscured or misinterpreted in studies that only focus on one perspective. Therefore, the diversity–disease relationship in fragmented landscapes must be investigated within a multi-perspective context, with an integrating approach [e.g. by applying structural equation models; (Bonds *et al*. 2012)]. Also, analyses at the metacommunity scale will be advantageous (Suzán *et al*. 2015), because they will include processes that occur at both the local scale (demographic change, interspecific interactions, etc.) and the landscape scale (dispersal, anthropogenic changes, etc.).

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