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The Allee effect in hosts can weaken the dilution effect of host diversity on parasitoid infections

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ABSTRACT

Although numerous studies have shown disease prevalence to be influenced by changes in biodiversity, few studies have explored which factors affect the relationship between biodiversity and disease. When species richness increases, the abundance of host species decreases due to the limited available resources. Low population density may yield a low population growth rate and a high chance of extinction caused by a variety of mechanisms such as mate limitation and inefficient cooperative defense/breeding, i.e. the Allee effect. However, few studies investigating the diversity-disease relationship have taken the Allee effect into account. Here, using a multi-host-parasitoid model, we found that the Allee effect can weaken the host dilution effect, to a degree that depended on the strength of the Allee effect and the aggregation of parasitoid attack. The Allee effect within hosts can protect them from being parasitized and increase the risk of host-parasitoid system collapse. These results may provide an alternative explanation for non-linear relationships between biodiversity and parasitic disease prevalence, and, therefore, can contribute to reconciliation of current disputes over dilution effects.

1. Introduction

Accumulated evidences have suggested that the emergence and prevalence of an infectious disease may relate to the level of host biodiversity (i.e., biodiversity-disease relationship) (Keesing et al., 2006; Cardinale et al., 2012; Johnson et al., 2013). Some studies have proposed that a high species diversity may buffer the emergence and transmission of infectious diseases via a hypothesized 'dilution effect' (i.e., high host species diversity can reduce disease risk) (Huang et al., 2015; Johnson et al., 2013; Keesing et al., 2010; Ostfeld and Keesing, 2012). The dilution effect, which merges public health and conservation interests (Cardinale et al., 2012; Schmidt and Ostfeld, 2001), has attracted much attention to the identification of ecosystem services and functions. The dilution effect can operate through several mechanisms: 1) Susceptible host regulation, where the low-quality host species in a diverse community can reduce the abundance of competent hosts, and, therefore, reduce the disease risk; 2) Encounter reduction, where the low-quality host species can reduce the encounter rates between ideal hosts or between competent hosts and vectors; 3) Transmission interference, where the addition of a new host species can reduce the transmission rate among competent hosts (Keesing et al., 2006).

Evidence supporting the dilution effect mechanism has been provided through studies on a wide range of diseases, including both plant and animal disease systems, such as West Nile virus (Allan et al., 2009), Lyme disease (LoGiudice et al., 2008), trematode parasites (Johnson et al., 2013), and four barley and cereal yellow dwarf viruses (Lacroix et al., 2014). However, some researchers argue that the dilution effect is likely an atypical response, which only applies under very specific circumstances (Randolph and Dobson, 2012; Salkeld et al., 2013; Wood et al., 2014). It is also suggested, from some recent studies, that diversity can sometimes amplify a disease (Chen and Zhou, 2015; Lafferty and Kuris, 1999) and, in other circumstances, diveristy can have no influence on the outcome of a disease (Salkeld et al., 2013). For instance, Wood et al. (2014) argued that biodiversity has little effect on most infectious diseases and, when it does influence diseases, biodiversity seems most likely to facilitate the transmission of an infectious disease. Given the ambiguous nature of the biodiversity-disease relationship, this debate may be partially due to failure to consider some important ecological factors that can affect the dynamics of the host community and disease transmission.

The Allee effect, described as a decrease in the per capita growth rate at low densities, can be caused by a number of mechanisms, such as mating limitations, inbreeding depression, anti-predator defenses, and dispersal costs (Davis et al., 2004; Lamont et al., 1993). The Allee effect

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has been detected in diverse animal taxa, Mollusca, Arthropoda and Chordata (Kramer et al., 2009). On one hand, with increases in species richness, the abundance of each species usually decreases because of the fixed total resources available within the habitat, the phenomenon that is termed as density compensation after MacArthur et al. (1972). Thus, the number of rare species may increase with species richness. The Allee effect, which is termed as the decrease in the per capita growth rate at low densities, may cause the extinction of these rare species. In this sense, the Allee effect may have a negative impact on diversity (Roques et al., 2012). On the other hand, theoretical and field studies have shown the Allee effect to affect disease transmission (Deredec and Courchamp, 2006; Liu et al., 2009). For instance, the presence of a strong Allee effect within hosts can, even in simple epidemic models, lead to surprisingly diverse dynamics including sustained oscillations, multiple stable states, and catastrophic collapse of an endemic equilibrium (Hilker et al., 2009). However, almost all the studies along this line are aimed at dynamical complexity, the stability of the system and the prevalence of the parasitism (Bompard et al., 2013; Briggs and Hoopes, 2004; Hilker et al., 2009; Jang and Diamond, 2007). To sum up, the Allee effect may not only affect the diversity of host communities, but also disease prevalence, therefore, it may potentially modify the diversity-disease relationship. Up to now, however, there has been no study exploring the roles of the Allee effect on the diversity-disease relationship.

Most experimental studies have found a dilution effect of host diversity on parasitism in host-parasite systems. For example, trematodes can parasitize the body surface, body cavities, and various organs of amphibians, causing mortality and severe malformations (Johnson et al., 2012). A field study in 345 wetlands showed a high diversity of amphibians can inhibit the prevalence of malformations and infections within the amphibian populations (Johnson et al., 2013). Additionally, heterogeneity of parasitoid attack can also affect parasitic disease dynamics (Liu et al., 2009; May, 1978; Rohani et al., 1994). In the real natural communities, parasitism is often found to be aggregated in patches with high host density (May, 1978), with many hosts sharing a low parasite load, and a few hosts bearing a very high burden of parasitism (Begon et al., 1996). For instance, Shaw et al. (1998) showed that 45 out of 49 published wildlife host-macroparasite systems exhibited some degree of aggregation of parasite attack and 43 systems supported a high intensity of aggregation (aggregation index, $K < 1$). Here, by incorporating the Allee effect and parasitism aggregation in hosts into a multi-host-parasitoid model, we examined the influences of a host Allee effect on the diversity-disease relationship at different levels of parasitism aggregation. We first investigated the life history trade-off between investment into growth and defense against parasitism existed for the host species (i.e. fast-lived species generally invest more energy in growth and simultaneously less in defense against diseases), then relaxed to simulate the random case (i.e. no trade-off between investment into growth and defense against parasitism) to get a more general conclusion.

2. Materials and methods

2.1. Multi-host-parasitoid model

A modelling tradition, initiated mainly by entomologists with insect hosts and their parasitoids in mind, assumes that populations have discrete and synchronized generations (May et al., 1981). The usual framework for discrete-generation host–parasitoid models is given by (Hassell, 2000):

$$H_{t+1} = \lambda H_t f(H_t, P_t)$$
$$P_{t+1} = \beta H_t [1 - f(H_t, P_t)]'$$

This model is widely used to describe the dynamics of hosts and parasitoids (Jang, 2006; May et al., 1981). We extended the above usual framework to describe the dynamics of multiple host species:

$$H_{i,t+1} = \lambda_i(t) H_{i,t} f(H_{i,t}, P_{i,t})$$
$$P_{i,t+1} = \beta H_{i,t} [1 - f(H_{i,t}, P_{i,t})] \quad (1)$$

where $H_{i,t}$ is the population size of the susceptible host species i at time t, and $P_{i,t}$ denotes the number of parasitoid individuals on host species i at time t. The parameter $\lambda_i(t)$ is the net growth rate of host i at time t in the absence of parasites. β is the average number of parasitoid progeny per attacked host (Hassell, 2000). The function $f(H_{i,t}, P_{i,t})$ defines the fractional survival of hosts from being parasitized, and therefore 1- $f(H_{i,t}, P_{i,t})$ gives the probability of host species i being parasitized.

2.2. Host species growth under the Allee effect

We assumed the growth rate of the host species to follow an over-compensatory density dependence (due to intra- and inter-specific competition) pattern:

$$\lambda_i(t) = e^{r_i(1-\sum_j H_{j,t}/c)\cdot H_{i,t}/(H_{i,t}+m)} \quad (2)$$

where c is the carrying capacity for host in the absence of parasitoid and r_i is the intrinsic growth rate for species i (Liu et al., 2009). We incorporated the Allee effect into host population growth by adding the term: $H_{i,t} / (H_{i,t} + m)$ (Berec et al., 2007), where the parameter m is the Allee effect constant, and a large value for m reflects a strong Allee effect regardless of parasitism.

2.3. Parasitoid attack aggregation

In the classic host-parasitoid model, it assumes the parasitoids search independently and randomly (Nicholson and Bailey, 1935), thence the proportion of hosts escaping from parasitism f can be calculated by the zero term of a Poisson distribution, i.e. $f = e^{-aP_t}$, where aP_t are the mean encounters per host. However, May (1978) pointed out that random parasitism is less likely to occur in the real world where host individuals are bound to vary in their spatial location, phenotype, and stage of development. Extensive series of experiments showed that the distribution of attacks per host is well described by a negative binomial distribution, as is the number of eggs laid in hosts (Griffiths and Holling, 1969; KFIR et al., 1976). The probability of escaping parasitism is the zero term of negative binomial, that is $f = (1 + {}^{aP_t}/_{K})^{-K}$, where K is the clumping parameter, which defines the degree to which parasitism among hosts is aggregated. The smaller K is, the stronger the aggregated parasitism will be ($K \to \infty$ when parasitoid-host interactions are random (i.e., Poisson distribution) and $K \to 0$ for extreme aggregation of parasitism (Liu et al., 2009; May, 1978; Rohani et al., 1994). In our multi-host-parasitoid model, f can be rewirtten as:

$$f(H_{i,t}, P_{i,t}) = \left(1 + \frac{E_i}{K} - K\right), \quad (3)$$

E_i measures the mean encounter rate of the parasitoids with the host species i :

$$E_i = \frac{H_{i,t}}{\sum_l H_{i,t}} \cdot \sum_j \alpha_{ij} P_{j,t} \quad (4)$$

Here $H_{i,t}/\sum_l H_{i,t}$ is the proportion of host species i in the total community, α_{ij} is the searching efficiency of parasitoid on host species j for host species i, i.e. the interspecific searching efficiency. The Eq. (4) implies that the parasitoid species dedicates a lot for foraging time or egg supply to an alternative host species or ignores rare host species in favor of more abundant hosts. We first applied a methodologically simple method that α_{ij} was the mean of the intraspecific searching efficiency modified by a constant scaling factor (i.e. $\alpha_{ij} = c_{ij}(\alpha_{ii} + \alpha_{jj})/2$). Then we performed a sensitivity analysis by randomly drawing α_{ij} from a normal distribution irrespective of the identity of the two host species (i and j) involved. Thus, Eqs. (1)–(4) forms the whole simulation models in this