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Host competence, interspecific competition and vector preference interact to determine the vector-borne infection ecology

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Understanding how ecological interactions affect vector-borne disease dynamics is crucial in the context of rapid biodiversity loss and increased emerging vector-borne diseases. Although there have been many studies on the impact of interspecific competition and host competence on disease dynamics, few of them have addressed the case of a vector-borne disease. Using a simple compartment model with two competing host species and one vector, we investigated the combined effects of vector preference, host competence, and interspecific competition on disease risk in a vector-borne system. Our research demonstrated that disease transmission dynamics in multi-host communities are more complex than anticipated. Vector preference and differences in host competence shifted the direction of the effect of competition on community disease risk, yet interspecific competition quantitatively but not qualitatively changed the effect of vector preference on disease risk. Our work also identified the conditions of the dilution effect and amplification effect in frequency-dependent transmission mode, and we discovered that adding vector preference and interspecific competition into a simple two-host-one-vector model altered the outcomes of how increasing species richness affects disease risk. Our work explains some of the variation in outcomes in previous empirical and theoretical studies on the dilution effect.

KEYWORDS

biodiversity-disease relationship, dilution effect, amplification effect, compartment model, contact heterogeneity

Introduction

Vector-borne diseases refer to the infectious diseases that are transmitted by blood-sucking arthropods such as mosquitoes, fleas, lice, ticks, etc. Most vector-borne diseases are zoonosis, they pose a great threat to human health and wildlife conservation. According to estimates, more than 1.5 million people die from vector-borne diseases

worldwide each year, which accounts for 3/4 of the newly emerging infectious diseases in recent years (World Health Organization [WHO], 2004). The effective prevention and management of vector-borne diseases remains one of the main challenges of current scientific research.

There is an increased likelihood that host diversity and total disease risk are inversely related, i.e., the dilution effect hypothesis (Keesing et al., 2006, 2010; Ostfeld and Keesing, 2012). Ecologists have recently confirmed that the dilution effect does exist in some systems through the study of Lyme disease (Ostfeld and Keesing, 2000; LoGiudice et al., 2003), West Nile virus (Kilpatrick et al., 2006), amphibian trematodes (Johnson et al., 2013), and leaf fungal diseases (Mitchell et al., 2002; Liu et al., 2016). They have also come to the following conclusion about the underlying mechanism of the dilution effect: the order of community (dis)assembly is non-random, the most competent host species (the hosts' ability to obtain and transmit pathogens) have the lowest probability of extinction (Halliday et al., 2019, 2020). As host diversity increases, a large number of low-competent or non-competent hosts (a host can become infected but is unable to transmit pathogens) are added to the community, which decreases the contact rate between highly competent hosts and vectors, a dilution effect occurs (Johnson et al., 2013). If this result holds, then the disease risk is highest when the community contains only the most competent host. However, some studies have questioned the universality of the dilution effect by demonstrating that the highest disease risk occurs when the community consists of several host species (Ostfeld and LoGiudice, 2003; Simpson et al., 2012; Costa et al., 2021). Additionally, several researchers have suggested that amplification and no effects also exist in the natural world (Wood et al., 2014; Halliday et al., 2017; Rohr et al., 2019; Vadell et al., 2019). These debates imply that a variety of ecological factors may affect disease risk, and different factors may play different or even perhaps opposite roles in different systems. Exploring how interspecific interactions (resource competition, transmission between species, and vector-host contact rate) affect vector-borne infection is critical not only for theoretical analysis, but also for vector-borne disease prevention and management.

Most vector-borne diseases are transmitted among a variety of hosts, but each host has its own ability to attract vectors, a phenomenon known as the vector preference (Rivera et al., 2020). Several empirical studies supported the existence of vector feeding preferences. For example, *Anopheles gambiae*, the vector of malaria, prefers to bite humans over cattle (Tirados et al., 2006). *Triatoma* bugs that transmit Chagas disease have a strong feeding preference for dogs over chickens and cats (Gürtler et al., 2009). Vector preference is an important ecological factor contributing to the heterogeneity in contact rates between hosts and vectors (Simpson et al., 2012). Both theoretical and experimental studies have found that vector preference not only affects the likelihood of disease outbreak and prevalence, but also interacts with other factors to influence disease dynamics (Kilpatrick et al., 2006; Simpson et al., 2012; Zeilinger and Daugherty, 2014).

Interspecific competition can alter the host's behavior, abundance, living habits, fitness, etc., which can directly or indirectly increase disease risk. For instance, while increased rodent richness facilitates disease transmission between individuals, it can also result in increased interspecific competition, allowing for a decrease in host density, leading to an overall decrease in the prevalence of Sin Nombre Hantavirus (Cortez and Duffy, 2021). Importantly, accounting for interspecific competition may qualitatively alter predictions of diversity-disease relationships. For example, using a mathematical model of infection dynamics and a high-resolution multisite dataset, Luis et al. (2018) found that the prevalence of hantavirus decreased as small mammal diversity increases. However, competition effects, which cause hosts (deer mice) to congregate in refuges far from their main competitors, leading to increased host-to-host contact and thus increased incidence. Therefore, species diversity concurrently dilutes and amplifies disease transmission through competing mechanisms. Similarly, Strauss et al. (2015) used a general trait-based model and found that varying the relationship between the host's ability to compete and its potential to transmit disease could produced three different outcomes: a dilution effect, an amplification effect, and no significant effect. These findings highlight the need for a better understanding of how interspecific competition affects disease ecology in multi-host communities. Although there have been several studies on the effect of host competition on disease transmission, to the best of our knowledge, few research has examined the situation of a vector-borne system (but see Marini et al., 2017).

The purpose of this paper is twofold. On the one hand, to investigate the combined effects of vector feeding preference and host interspecific competition on vector-borne infection ecology. On the other hand, to explore the exact conditions under which diversity amplification or dilution occurs. To this end, we developed a simple compartment model with two competing host species and one vector, based on the SIS framework, to describe the transmission dynamics of a vector-borne system. The basic reproduction number R_0 is used to quantify disease risk, which has been used to identify the conditions under which disease risk would increase or decrease in a multi-host community (Roberts and Heesterbeek, 2013; O'Regan et al., 2015). We found that the conversion of a disease from being extinct to becoming endemic can be made possible by changing the intensity of competition, and that the direction of the effect of competition on disease risk (increasing or decreasing) can be shifted by a combination of vector preference and host competence. Importantly, incorporating vector preference and interspecific competition into a simple model changes the outcomes of how increasing species richness affects disease risk. These findings underscore the importance

of linking vector preference, interspecific competition, and host competence in describing vector-borne infection ecology.

Materials and methods

Model description

Mathematical models have been widely used to study complicated ecological processes. Many previous studies have used SIS or SIR compartmental models as a theoretical framework to analyze transmission dynamics in multi-host systems (Dobson, 2004; O'Regan et al., 2015). Similar to the models proposed by Lord et al. (1996) and Simpson et al. (2012), we constructed a simple compartment model with two hosts and one vector, both of which were classified according to whether they are susceptible S or infected I. We refer to host species 1 as the focal host that is a permanent community resident and host species 2 as the introduced host. Assuming that host species 1 and species 2 can compete with each other for resources (Kambatuku et al., 2010), and pathogens can only be transmitted indirectly through the vector, not directly through host-to-host transmission. Vectors feed on both host species, but with different feeding preferences. To simplify the model formulation and analysis, we assumed that infected hosts could die from disease or recover as susceptible individuals, but the vector could not recover from infection.

To model interspecific competition between two host species, as in O'Regan et al. (2015) and Marini et al. (2017), we assumed that the growth of both host species follows Lotka–Volterra dynamics. The following set of differential equations can be used to illustrate the model:

$$\begin{aligned}
\frac{dN_1}{dt} &= r_1 N_1\left(1-\frac{N_1+c_{12}N_2}{K_1}\right)-\mu_1 I_1\\
\frac{dN_2}{dt} &= r_2 N_2\left(1-\frac{N_2+c_{21}N_1}{K_2}\right)-\mu_2 I_2\\
\frac{dN_v}{dt} &= r_v N_v-\mu_v I_v\\
\frac{dI_1}{dt} &= \frac{P_{v1}b_1 I_v S_1}{N_1}-d_1 I_1-\delta_1 I_1-\mu_1 I_1\\
\frac{dI_2}{dt} &= \frac{P_{v2}b_2 I_v S_2}{N_2}-d_2 I_2-\delta_2 I_2-\mu_2 I_2\\
\frac{dI_v}{dt} &= \frac{P_{1v}b_1 I_1}{N_1}S_v+\frac{P_{2v}b_2 I_2}{N_2}S_v-d_v I_v-\mu_v I_v
\end{aligned}. \quad (1)$$

Where S_i and I_i represent the number of susceptible and infected individuals of species i, respectively, and $N_i$represents the total population size of species i,$N_i = S_i + I_i$ (i = 1, 2, v). c_{ij} represents the competition coefficient of species j on species i. K_1 and K_2 denote the carrying capacities of hosts 1 and 2, respectively. Previous studies have suggested that the transmission of pathogens in vector-borne systems follows a frequency-dependent mode. That is, the total number of bites per unit time by a single vector is independent of the host density (Dobson, 2004; Cortez and Duffy, 2021), therefore the incidence $\frac{P_{vi}b_i I_v S_i}{N_i}$ and $\frac{P_{iv}b_i I_i S_v}{N_i}$ denote the number of infected individuals per unit time for host species i and vector, respectively. To simplify notation, we let $\Gamma_i = d_i + \delta_i + \mu_i$ and $\Gamma_v = d_v + \mu_v$, which denote the per capita removal rate from the I_i class and I_v class, respectively. All parameters and definitions are listed in **Table 1**.

Model analysis

As mentioned above, we used the basic reproduction number R_0 as a measure of disease risk. It identifies the number of secondary infections induced by an infected host individual during the disease duration and the lifespan of the vector (Roberts and Heesterbeek, 2013). If $R_0 < 1$, the disease eventually disappears, otherwise, the disease will break out and become endemic. The basic reproduction number R_0 of system (1) is calculated by using the next-generation matrix approach, which involves linearizing the system at the disease-free equilibrium (N_1^*, N_2^*, N_v^*) and decomposing the resulting Jacobian matrix into two matrices, F and V, which describe the disease transmission and transitions out of the infection state, respectively. The dominant eigenvalue of the next-generation matrix is the basic reproduction number R_0 (Diekmann et al., 2009; Roberts and Heesterbeek, 2013).

The disease-free non-trivial equilibrium point can be found by setting the first three equations of system (1) to 0:

$$N_1^* = \frac{K_1 - c_{12}K_2}{1 - c_{12}c_{21}}, N_2^* = \frac{K_2 - c_{21}K_1}{1 - c_{12}c_{21}}, N_v^* = K_v.$$

Specifically, if there is no host interspecific competition, i.e., $c_{12} = c_{21} = 0$, then $N_1^* = K_1, N_2^* = K_2, N_v^* = K_v$.

Here the epidemiological transmission matrix F at the disease-free equilibrium point (N_1^*, N_2^*, N_v^*) is,

$$F = \begin{pmatrix} 0 & 0 & P_{v1}b_1 \\ 0 & 0 & P_{v2}b_2 \\ \frac{P_{1v}b_1 N_v^*}{N_1^*} & \frac{P_{2v}b_2 N_v^*}{N_2^*} & 0 \end{pmatrix},$$

and the epidemiological transition matrix V is,

$$V = \begin{pmatrix} d_1+\delta_1+\mu_1 & 0 & 0 \\ 0 & d_2+\delta_2+\mu_2 & 0 \\ 0 & 0 & d_v+\mu_v \end{pmatrix} = \begin{pmatrix} \Gamma_1 & 0 & 0 \\ 0 & \Gamma_2 & 0 \\ 0 & 0 & \Gamma_v \end{pmatrix}$$

Therefore, the next-generation matrix M is,

$$M = FV^{-1} = \begin{pmatrix} 0 & 0 & \frac{P_{v1}b_1}{\Gamma_v} \\ 0 & 0 & \frac{P_{v2}b_2}{\Gamma_v} \\ \frac{P_{1v}b_1 N_v^*}{N_1^*\Gamma_1} & \frac{P_{2v}b_2 N_v^*}{N_2^*\Gamma_2} & 0 \end{pmatrix}.$$

The dominant eigenvalue of M is the basic reproduction number R_0 of system (1),

$$R_0 = \sqrt{\frac{P_{v1}P_{1v}b_1^2 N_v^*}{N_1^*\Gamma_1\Gamma_v} + \frac{P_{v2}P_{2v}b_2^2 N_v^*}{N_2^*\Gamma_2\Gamma_v}}. \quad (2)$$

TABLE 1 Parameters and definitions.

Parameter	Definition
S_i	Number of susceptible individuals of species i (i = 1, 2, v, the same below)
I_i	Number of infected individuals of species i
N_i	Total population size of species i,$N_i = S_i + I_i$
r_i	The growth rate of species i
d_i	Per capita natural death rate of species i
δ_i	Per capita recovery rate of species i (i = 1, 2)
μ_i	Per capita disease-induced death rate of species i
c_{ij}	The effect of competition of species j on species i ($i, j \in \{1, 2\}$, $i \neq j$)
p_{vi}	The efficiency that an infected vector would infect a susceptible individual of host species i during one feeding event
p_{iv}	The efficiency that an infected individual of host species i would infect a susceptible vector during one feeding event
b_i	Biting rate between the vector and host species i
$b_{\max}$	The daily biting rate of the vector to the entire community, $b_{\max} = b_1 + b_2$
K_i	Carrying capacity of species i
Γ_i	Per capita removal rate from the I_i class, $\Gamma_i = d_i + \delta_i + \mu_i$ (i = 1, 2)
Γ_v	Per capita removal rate from the I_v class, $\Gamma_v = d_v + \mu_v$
α	The vector's feeding preference to host species 1 compared to that of species 2
g_i	The transmission ability of the host species i
γ	The transmission ratio of host species 1–2, i.e., $\gamma = g_1/g_2$

Let $g_i = \frac{p_{vi}p_{iv}}{\Gamma_i}$ (i = 1, 2), according to Table 1, this parameter characterizes the transmission efficiency that an infected host species i successfully infects a conspecific individual ($p_{vi}p_{iv}$) *via* the vector during its disease duration ($1/\Gamma_i$), thus representing the competence of host species i. The higher the g_i, the greater the ability of host species i to transmit disease. Substitute g_i into Eq. 2, it can be simplified to

$$R_0 = \sqrt{\frac{g_1 b_1^2 N_v^*}{N_1^* \Gamma_v} + \frac{g_2 b_2^2 N_v^*}{N_2^* \Gamma_v}}. \tag{3}$$

As shown in Eq. 3, one of the key factors to determining disease transmission potential and how R_0 varies with species richness is how the bites are divided between the two host species, b_1 and b_2, representing the biting rates of the vector to species 1 and 2, respectively. Assuming that host density is high enough not to limit the biting rate of the vector so that the vector has a fixed daily biting rate $b_{\max}$ (Rogers, 1988). If a vector has no preference for any hosts, then the biting rate of the vector for a specific host is determined by the density of the host (Marini et al., 2017), therefore $b_i = b_{\max} \cdot \frac{N_i}{N_1+N_2}$, $i = 1, 2$. However, if host species i is preferred, i.e., $\frac{b_i}{b_{\max}} > \frac{N_i}{N_1+N_2}$, as in Simpson et al. (2012) and Miller and Huppert (2013), we introduced a preference parameter α to represent the feeding preference of the vector, which represents the feeding preference of the vector for host species 1 relative to species 2. From this, the biting rate becomes

$$b_1 = b_{\max} \cdot \frac{\alpha N_1}{\alpha N_1 + N_2}, b_2 = b_{\max} \cdot \frac{N_2}{\alpha N_1 + N_2}.$$

Note that when $\alpha = 1$, the vector has no preference for any host and when $\alpha > 1$, the vector prefers host 1, and vice versa.

Substituted b_1, b_2 into Eq. 3, it becomes

$$R_0 = \frac{b_{\max}}{\alpha^2 N_1^* + N_2^*} \sqrt{\frac{(\alpha^2 g_1 N_1^* + g_2 N_2^*) N_v^*}{\Gamma_v}}. \tag{4}$$

By analyzing and numerically simulating Eq. 4, we can figure out how vector preference and host interspecific competition affect disease risk in vector-borne systems.

Another major purpose of this study is to explore the exact conditions under which diversity amplification or dilution occurs. Analytical results of the effect of host species richness on disease risk were obtained *via* a single- and two-host species community comparison. To this end, we calculated the basic reproduction number R_0^1 of a community composed of a single host species (focal host) and compared it to the community composed of focal and alternative host species. The calculation of R_0^1 is similar to the process of calculating R_0 (see **Supplementary material**), and we obtained

$$R_0^1 = \sqrt{\frac{g_1 b_{\max}^2 N_v^*}{N_1^* \Gamma_v}}.$$

To explore the conditions under which the dilution effect occurs, we need to find the conditions in which disease risk is reduced in a community consisting of a focal and introduced host species compared to a community with only the focal host species, i.e., $R_0 < R_0^1$. If this inequality holds, it is equivalent to

$$\frac{g_1 b_1^2 N_v^*}{N_1^* \Gamma_v} + \frac{g_2 b_2^2 N_v^*}{N_2^* \Gamma_v} < \frac{g_1 b_{\max}^2 N_v^*}{N_1^* \Gamma_v} \tag{5}$$

When Eq. 5 holds, the dilution effect will occur. Otherwise, there will be an amplification effect.

To simplify Eq. 5, a dimensionless parameter $\gamma = g_1/g_2$ is introduced to measure the host competence of species 1 relative to 2, which we define as the transmission ratio. When $\gamma = 1$, the two hosts were comparable in their ability to transmit disease, and when $\gamma > 1$ or $0 < \gamma < 1$, the more competent host is species 1 or 2, respectively.

Next, we will find out the conditions that satisfy Eq. 5 under each of the four combinations with or without vector preference and with or without host interspecific competition. (i) When the vector has no preference for any host ($\alpha = 1$) and there is no interspecific competition between hosts ($c_{12} = c_{21} = 0$), the non-trivial disease-free equilibrium point is $N_1^* = K_1, N_2^* = K_2, N_v^* = K_v,$, and the biting rate $b_i = b_{\max} \cdot \frac{K_i}{K_1+K_2}$, substitute these parameters into Eq. 5, it is equivalent to $g_2 < g_1(2 + \frac{K_2}{K_1})$. Since $\gamma = g_1/g_2$, the expression becomes $\gamma > \frac{K_1}{2K_1+K_2}$. That is, when there is no vector preference and no interspecific competition, as long as $\gamma > \frac{K_1}{2K_1+K_2}$ holds, the dilution effect can occur. (ii) When there is both vector preference and host interspecific competition (i.e., $\alpha \neq 1, c_{12}, c_{21} > 0$), then

$$N_1^* = \frac{K_1 - c_{12}K_2}{1 - c_{12}c_{21}}, N_2^* = \frac{K_2 - c_{21}K_1}{1 - c_{12}c_{21}}, N_v^* = K_v,$$

and $b_1 = b_{\max} \cdot \frac{\alpha N_1}{\alpha N_1+N_2}, b_2 = b_{\max} \cdot \frac{N_2}{\alpha N_1+N_2}$.

In the same way, by substituting these parameters into Eq. 5, it is possible to derive the following criteria for the occurrence of the dilution effect:

$$\gamma > \frac{K_1 - c_{12}K_2}{2\alpha(K_1 - c_{12}K_2) + K_2 - c_{21}K_1}.$$

Note that in this scenario, if interspecific competition is symmetric ($c_{12} = c_{21}$) and both hosts have the same carrying capacity ($K_1 = K_2$), the above inequality is simplified to $\gamma > \frac{1}{2\alpha+1}$, i.e., whether dilution or amplification occurs in this case is only related to vector preference α and transmission ratio γ. The calculation of the conditions for the dilution effect in the other two scenarios (with preference, without competition, or without preference, with competition) is similar to the above. **Table 2** provides a summary of the analytical conditions for the model parameters for which Eq. 5 is valid.

TABLE 2 The analytical conditions for the criterion of $R_0 < R_0^1$ in four different cases.

	Without vector preference	Vector preference
Without host interspecific competition	$\gamma > \frac{K_1}{2K_1+K_2}$	$\gamma > \frac{K_1}{2\alpha K_1+K_2}$
Host interspecific competition	$\gamma > \frac{K_1-c_{12}K_2}{K_1(2-c_{21})+K_2(1-2c_{12})}$	$\gamma > \frac{K_1-c_{12}K_2}{2\alpha(K_1-c_{12}K_2)+K_2-c_{21}K_1}$

Results

The effects of host interspecific competition on R_0

To exclude the impact of the presence of other factors on the outcomes, focusing on how interspecific competition affect R_0, we first investigated the situation where there is no vector preference and both hosts have the same ability to transmit disease. We assumed that the vector bites hosts based on their density, with a daily biting rate $b_{\max} = 0.3$ and the disease transmission ratio $\gamma = 1$. As seen in **Figure 1A** (left panel), the maximum R_0 occurs when both the competition coefficient c_{12} and c_{21} are high (top right), whereas the minimum R_0 occurs when both c_{12} and c_{21} are very low (bottom left). For a fixed value of c_{ij}, an increase in c_{ji} will increase R_0. Moreover, we also found that even when all other parameters are held constant, a change in c_{12} and c_{21} will make R_0 changes from less than 1 to larger than 1 (R_0 ranges from 0.85 to 1.18). That is, by changing the intensity of competition, the disease may change from extinction to endemic. Secondly, we relaxed the restrictions to consider the scenarios where there is vector preference and differences in host competence. To do this, we simulated the following parameter combinations of $\gamma, \alpha \in \{0.5, 1, 2\} \times \{0.5, 1, 2\}$ (**Supplementary Figure 1**). In each subplot of **Supplementary Figure 1**, when the competition coefficients c_{12} and c_{21} are large, R_0 is large, and vice versa. For a fixed value of c_{ij}, R_0 increases with the increase of $c_{ji}(i, j = 1, 2)$, these findings were consistent with the results in **Figure 1**. In addition, we found that for the constant values of $c_{12}$and c_{21}, the larger γ is, the larger R_0 is, especially when the preferred host is the highly competent one (see **Supplementary Figure 1**).

In **Figure 1B** (right panel), we showed how the effect of interspecific competition on R_0 is influenced by the biting rate, which we set $b_{\max} = 0.1, 0.2, 0.3$, respectively. We found that for a fixed interspecific competition coefficient, the larger $b_{\max}$ is, the larger R_0 is. At each value of $b_{\max}$, R_0 increases linearly with $c_{12} = c_{21}$. Since there is no significant difference in the results for any of the three values of $b_{\max}$, we only consider the case of $b_{\max} = 0.3$ in the following study.

Next, we investigated how interspecific competition coefficient c_{12} affects R_0 in the presence of vector preference and differences in host competence (i.e.,$\alpha \neq 1, \gamma \neq 1$) (**Figure 2**). The horizontal axis represents the competition effect of host species 2 on species 1 (i.e., c_{12}), and the vertical axis represents R_0. As can be seen from **Figure 2**, for a fixed c_{12}, the larger the transmission ratio γ, the larger the R_0, especially when the preferred host is a highly competent one. As c_{12}increases, R_0 shows a non-linear trend, increasing or decreasing depending on the combined effect of transmission ratio γ and vector preference α, suggesting that vector feeding preference and differences in host competence may shift the direction of the effect of interspecific competition on R_0 .

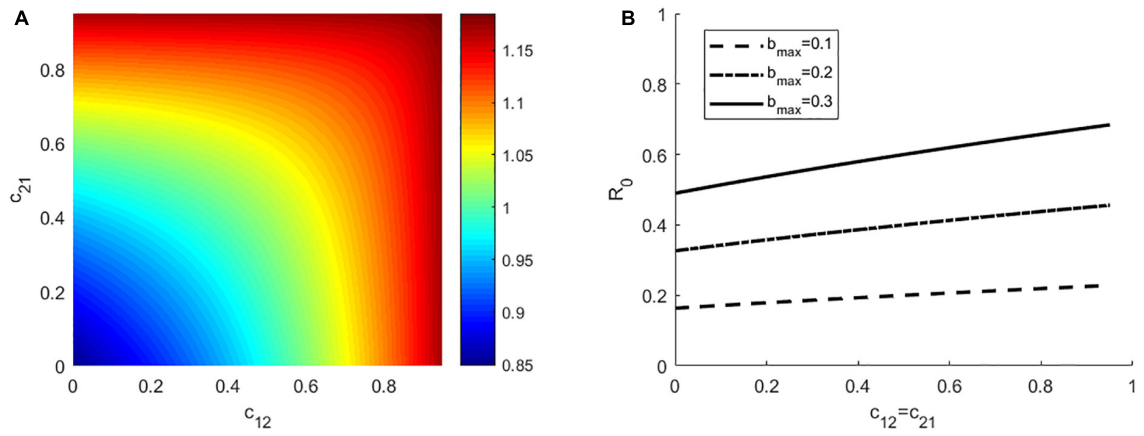

FIGURE 1
(A) The effect of interspecific competition on R_0. **(B)** The effect of interspecific competition (only for $c_{12} = c_{21}$) on R_0at three different values of b_{max} = 0.1, 0.2, 0.3, respectively. In panel **(A)**, b_{max} = 0.3, the other parameters in the two panels have the same values as: $\alpha = 1$, $\gamma = 1$, $K_1 = K_2 = 1000$, $K_v = 4000$, $\Gamma_v = 0.1$, $g_1 = g_2 = 0.4$.

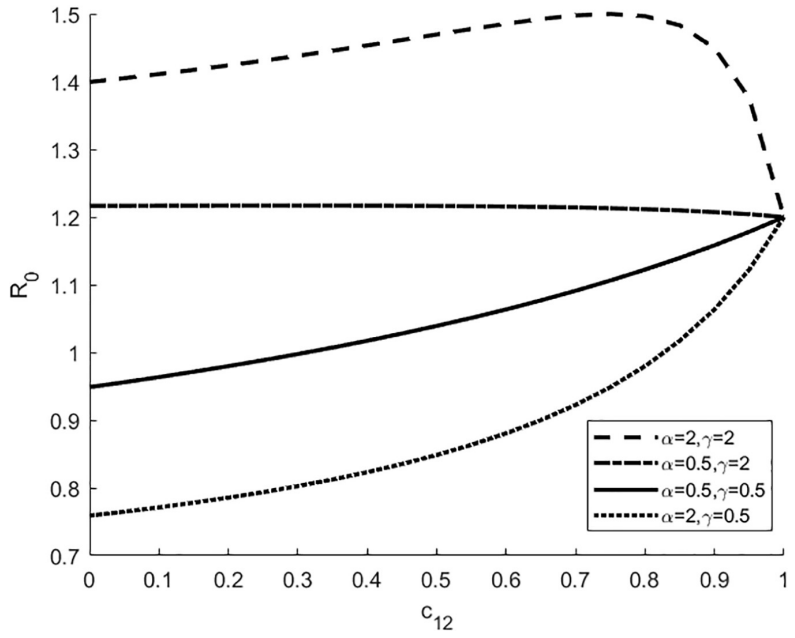

FIGURE 2
R_0 according to interspecific competition coefficient c_{12} in the presence of vector preference and differences in host competence. The four lines represent four different combinations of vector preferences α and transmission ratios γ. Here $c_{21} = 0.5$, b_{max} = 0.3, $K_1 = K_2 = 1000$, $K_v = 4000$, $\Gamma_v = 0.1$, $g_2 = 0.4$.

The effects of vector preference on R_0

Vector feeding preferences can lead to heterogeneity in host-vector contact, which may affect disease dynamics. To understand the effect of vector preference on disease transmission potential, we first assumed that there was no interspecific competition between hosts. In Figure 3, the horizontal axis represents the feeding preference index α, which ranges from 0 to 2, and the vertical axis represents R_0. It can be seen from Figure 1 that as α increases, R_0may increase, decrease, or vary slightly, depending on the host transmission ratio γ. If species 1 is the more competent host at this time ($\gamma > 1$), then R_0increases with the increase of α. On the contrary, if species 1 is the lower competent host, then R_0decreases as αincreases. When the two hosts are comparable in their competence ($\gamma = 1$), R_0varies within a small range as α increases. In fact, in this

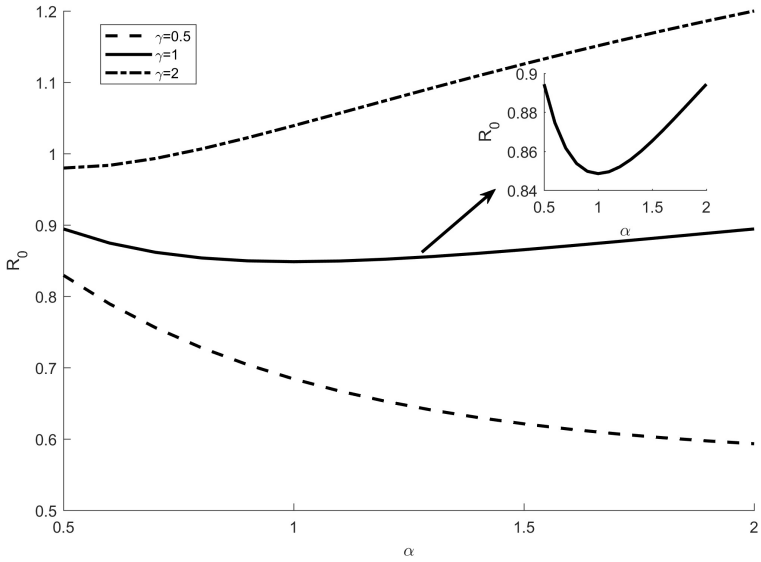

FIGURE 3
The effect of vector preference α on R_0 without host interspecific competition. Different lines represent the results of different values of the transmission ratio γ. The inset of figure shows the case of $\gamma = 1$ when narrowing the vertical axis. The other parameters are: $c_{12} = c_{21} = 0$, $b_{max} = 0.3$, $K_1 = K_2 = 1000$, $K_v = 4000$, $\Gamma_v = 0.1$, $g_2 = 0.4$.

case, when we narrow the range of the vertical axis (the inset graph of Figure 3), we find that R_0 first decreases and then increases as α increases, and when the vector has no preference for any hosts ($\alpha = 1$), R_0 takes the minimum value.

Secondly, we considered the effect of vector preference α on R_0 in the presence of interspecific competition by simulating different competition coefficient combinations (Supplementary Figure 2). Different panels of Figure 2 refer to different values of (c_{12}, c_{21})that assume the values of 0.1, 0.5, 0.9. It was found that there is no qualitative difference with the results in Figure 3, except that when species 1 is a less competent host or the two hosts are comparable in their competence. As α increases, the magnitude of the decrease of R_0 is lower than that in the absence of interspecific competition (Figure 3). All these results indicate that the presence or absence of interspecific competition quantitatively but not qualitatively changes the effect of vector preference on R_0 .

Dilution effect versus amplification effect

Table 2 summarizes the analytical conditions that are satisfied $R_0 < R_0^1$ under each of the four combinations with or without vector preference and with or without host interspecific competition (see section "Materials and methods"). In this section, we focused on how interspecific competition, vector preference, and host competence affect the occurrence of dilution effects. According to Table 2, when both interspecific competition and vector preference exist, a dilution effect can occur when

$$\gamma > \frac{K_1 - c_{12}K_2}{2\alpha(K_1 - c_{12}K_2) + K_2 - c_{21}K_1}.$$

Conditions for $\gamma = \frac{K_1 - c_{12}K_2}{2\alpha(K_1 - c_{12}K_2) + K_2 - c_{21}K_1}$ (i.e., $R_0 = R_0^1$) were represented by a linear configuration of vector preference α and the transmission ratio γ, the line divided α-γ parameter space into disjoint regions for which dilution and amplification effects were exhibited (Figure 4). Different lines represent different competition intensities of c_{12}, which are taken as 0.1, 0.5, and 0.9, respectively. On the upper right side of each line, it shows the parameter area where the dilution effect happens (symbol DE). On the lower left side of each line, it shows the parameter area where the amplification effect happens (symbol AE).

As shown in Figure 4, adding new species to a community can lead to both a dilution effect and an amplification effect, depending on the values of α and γ. When the focal species (species 1) is a highly competent host and the vector prefers the focal species, the addition of the introduced species (species 2) dilutes the proportion of the focal species and reduces the effective contact between the focal species and the vector, resulting in a dilution effect ($R_0 < R_0^1$) (the upper right area of Figure 4). Conversely, when the introduced species is a highly competent host and the vector prefers the introduced species, the addition of the introduced species results in a higher disease risk compared to the community containing only the focal host, and thus an amplification effect occurs ($R_0 > R_0^1$) (the lower left

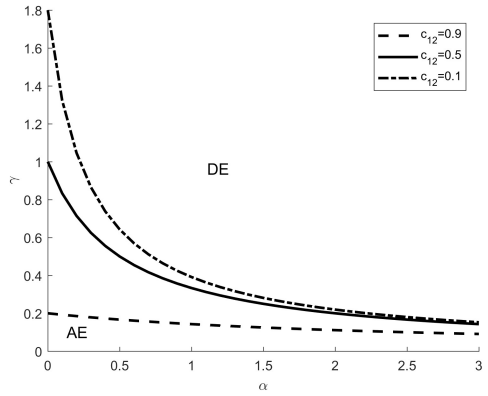

FIGURE 4
Analytical relationships for combinations of interspecific competition, vector preference and transmission ratio. The dotted and solid lines represent parameter sets (α,γ) for which $R_0 = R_0^1$ under different competition intensity c_{12}. These lines divided $\alpha - -\gamma$ parameter space into disjoint regions where dilution and amplification effects were exhibited. The upper right of each line represents parameter pairs for which a dilution effect occurs (symbol DE), whereas the lower left of each line represents parameter pairs for which an amplification effect occurs (symbol AE). The other parameters are: $K_1 = K_2 = 1000$, $c_{21} = 0.5$.

part of **Figure 4**). In addition, for a fixed value of c_{21}, the larger the c_{12}, the larger the parameter area where the dilution effect can occur, and vice versa.

Discussion

Understanding how ecological interactions and significant ecological factors affect vector-borne disease dynamics is crucial given the rapid loss of biodiversity and the rise in newly emerging vector-borne diseases (Rivera et al., 2020). In this study, we constructed a simple compartment model with two competing host species and one vector, in which the vector has different preferences for the hosts, to study the combined impact of vector feeding preferences and host interspecific competition on vector-borne infection ecology. Furthermore, we investigated the relationship between host species richness and disease risk by comparing disease risk R_0 in single- and two-host communities. We demonstrated that disease transmission dynamics in multi-host communities are more complex than anticipated, highlighting the significance of linking vector preference, interspecific competition, and host competence in describing vector-borne infection ecology. More specifically, we found that vector preference and differences in host competence shifted the direction of the effect of competition on R_0, yet interspecific competition quantitatively but not qualitatively changed the effect of vector preference on R_0. Furthermore, this study quantified the conditions of dilution effect and amplification effect, and clarified that incorporating vector preference and interspecific competition into a simple two-host-one-vector model changes the outcomes of how increasing species richness affects disease risk R_0.

For vector-borne diseases, the dynamics of pathogen transmission depend on the ability of host species to maintain and transmit disease and on ecological factors such as interspecific competition and contact rate between hosts and vectors (Simpson et al., 2012). Interspecific competition can either increase R_0 by increasing the vector/host ratio or decrease R_0 by decreasing the density of the host population (Marini et al., 2017), the general pattern of the effect of competition on R_0 is hard to predict, and whether competition has a positive or negative effect on R_0 depends largely on host preferences and host competence. We also found that R_0 is strongly influenced by vector feeding preference. R_0 increases with the increase of vector preference, as long as the preferred host is a highly competent host. The reason for this phenomenon may be that vector preference allows a large number of bites to be concentrated on the preferred host, increasing the effective contact between the vector and highly competent hosts, thereby increasing the efficiency of disease transmission. In fact, these findings have been confirmed in some field experiments and theoretical studies. For example, if all individuals in the community have the same ability to transmit disease, vector-host contact heterogeneity owing to vector preference can increase the risk of disease outbreak (Woolhouse et al., 1997; Miller and Huppert, 2013). These facts tell us that it is important to be scientific and rational in developing disease control strategies, and that if the control measures are not appropriate, they may be counterproductive. For example, for a population with identical individuals, selective use of insect repellent will result in a higher concentration of vectors on the unprotected individuals, which is equivalent to the vector feeding preference, the disease risk R_0 may rise rather than fall (Miller and Huppert, 2013).

Whether increased host species richness results in greater or lower disease risk has been controversial in the literature, leading to calls for theoretical studies on what conditions promote amplification versus dilution (Buhnerkempe et al., 2015; Halsey, 2018). Many previous studies on the biodiversity-diseases risk relationship have demonstrated that when diseases are transmitted in a frequency-dependent mode, an increase in species richness decreases the community R_0 (Dobson, 2004; Rudolf and Antonovics, 2005; Rohr et al., 2019). However, an interesting finding of this study is that, even in frequency-dependent transmission mode, the introduction of new species to a community may increase or decrease disease risk if interspecific competition and contact heterogeneity due to vector preference are taken into account, which is in contrast to the results of previous studies. The reason for this phenomenon can be understood from the mechanism of the dilution effect. Keesing et al. (2006) constructed a general mathematical model framework and proposed five mechanisms by which dilution

effects occur. Among them, encounter reduction and susceptible host regulation have been confirmed in many empirical studies (Allan et al., 2009; Johnson and Thieltges, 2010). In a vector-borne disease system, encounter reduction refers to the presence of additional species that affects host behavior, reduces the probability of contact between vector and host, or influences vector behavior, decreasing the likelihood that susceptible individuals will become infected individuals (Clay et al., 2009). Susceptible host regulation refers to the fact that the addition of new host species to a community regulates susceptible host numbers through interspecific interactions such as competition or predation (Keesing et al., 2006). If the above assumptions are satisfied, an increase in species richness may lead to a dilution effect. Conversely, if the introduced host is a highly competent host or one that the vector prefers to feed on, or if the introduced host has strong interspecific competition ability, which reduces the frequency of low competent hosts through interspecific competition, then an increase in species richness will increase the communityR_0, whereby an amplification effect occurs. This explains, to some extent, why an amplification effect can occur in the frequency-dependent transmission mode. Our study emphasizes the need to focus not only on the transmission mode of disease, but also on interspecific interactions (interspecific competition and vector-host contact rate) and host competence (Cortez and Duffy, 2021; Su et al., 2022).

Although mathematical models have been widely used to study complex ecological phenomena, explaining and validating many empirical studies, there are still several limitations (O'Regan et al., 2015). In fact, the two-host-one-vector Lotka–Volterra competition model used in this study is very simplistic compared to the complexity of real community ecology. For example, we only compared disease risk R_0 when pathogen was transmitted in single- and two-host communities, despite the fact that natural communities can have dozens of host species. We focused only on the effect of interspecific competition on disease risk, ignoring the possible effect of intraspecific competition. Moreover, we used the basic reproduction number R_0 as a measure of community disease risk, which is challenging to estimate in field and empirical studies and does not allow for comparisons between studies (Roberts and Heesterbeek, 2018; Cortez and Duffy, 2021). In addition, we ignored some ecological factors that could affect the potential of outbreaks, including the seasonal variation in vector feeding preference (Burkett-Cadena et al., 2012; Marini et al., 2017) and demographic stochasticity (Dizney and Ruedas, 2009). However, we believe that this study of a simplified scenario provides a theoretical framework for incorporating interspecific competition, vector preference and host competence into vector-borne systems. Our work emphasizes the significance of ecological interactions in determining infection dynamics in a multi-host vector system, and contributes to explaining some of the variation in outcomes in previous empirical and theoretical studies on the dilution effect.

Data availability statement

The original contributions presented in the study are included in the article/**Supplementary material**, further inquiries can be directed to the corresponding author.

Author contributions

LC and SC designed the study and analyzed the model. LC and PK performed the software. LC and LZ drafted the manuscript. All authors have read and approved the manuscript.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Publisher's note

Supplementary material

The Supplementary Material for this article can be found online at: https://www.frontiersin.org/articles/10.3389/fevo.2022.993844/full#supplementary-material

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