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ORIGINAL ARTICLE

Min Su · Wenlong Li · Zizhen Li · Fengpan Zhang
Cang Hui

The effect of landscape heterogeneity on host–parasite dynamics

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Abstract Environmental heterogeneity has been shown to have a profound effect on population dynamics and biological invasions, yet the effect of its spatial structure on the dynamics of disease invasion in a spatial host–parasite system has received little attention. Here we explore the effect of environment heterogeneity using the pair approximation and the stochastic spatially explicit simulation in which the lost patches are clustered in a fragmented landscape. The intensity of fragmentation is defined by the amount and spatial autocorrelation of the lost habitat. More fragmented landscape (high amount of habitat loss, low clustering of lost patches) was shown to be detrimental to the parasitic disease invasion and transmission, which implies that the potential of using artificial disturbances as a disease-control agency in biological conservation and management. Two components of the spatial heterogeneity (the amount and spatial autocorrelation of the lost habitat) formed a trade-off in determining the host–parasite dynamics. An extremely high degree of habitat loss was, counter-intuitively, harmful to the host. These results enrich our understanding of eco-epidemiological, host–parasite systems, and suggest the possibility of using the spatial arrangement of habitat patches as a conservation tool for guarding focal species against parasitic infection and transmission.

Keywords Host–parasite interactions · Spatial heterogeneity · Spatial correlation · Pair approximation · Individual-based simulation

M. Su (✉) · Z. Li (✉)
School of Mathematics and Statistics,
Lanzhou University, Lanzhou 730000, China
E-mail: sum04@lzu.cn
E-mail: zizhenlee@lzu.edu.cn
Tel.: +86-931-8913370
Fax: +86-931-8912823

W. Li
School of Pastoral Agriculture Science and Technology,
Lanzhou University, Lanzhou 730000, China

F. Zhang
School of Mathematics and Information Sciences,
Henan University, Kaifeng 475001, China

C. Hui
Centre for Invasion Biology,
Department of Botany and Zoology,
University of Stellenbosch, Private Bag X1,
Matieland 7602, South Africa

Introduction

The effect of landscape spatial heterogeneity on population dynamics has been thoroughly studied in recent decades (Cantrell and Cosner 1991; Andren 1994; Hiebeler 2000; Ovaskainen et al. 2002; Hiebeler and Morin 2007; Hirzel et al. 2007). In these studies, it has been found that the habitat loss induced environmental heterogeneity is mostly due to unprecedented anthropogenic disturbances. It is also obvious that the real landscape is heterogeneous, where the quality of habitat varies across space. Suitable habitat patches, are thus interspersed among the matrix of unsuitable habitat patches. Consequently, the number of studies on the effect of the spatial configuration of the landscape on population dynamics has largely increased, both theoretically and empirically (Hiebeler 2000; North and Ovaskainen 2007; Hirzel et al. 2007). The spatial heterogeneity of the landscape has been shown to strongly affect the population dynamics, as well as the epidemiological behavior. Specifically, spatial clustering of suitable or unsuitable habitat patches can increase the population equilibrium in single species models (Hiebeler 2005; Hiebeler and Morin 2007; Hirzel et al. 2007). We intend to explore how this spatial heterogeneity in landscape affects the spatial dynamics of a host–parasite system.

Infection disease has been shown to be a crucial factor affecting species viability and dynamics (Rand et al. 1995; Keeling et al. 2001; Packer et al. 2003;

Bairagi et al. 2007; Su et al. 2008a). Scientists are increasingly interested in eco-epidemiological systems (Chattopadhyay and Bariagi 2001; Keeling et al. 2001; Greenman et al. 2004; Webb et al. 2007a; Garrett et al. 2007). Since Anderson and May's (1979) theoretical demonstration of the regulation strength of parasites towards their hosts, much attention has been paid to the impact of these parasites on host populations in natural systems (Boots and Sasaki 2001; Deredec and Courchamp 2006; Webb et al. 2007a, 2007b). As a result, an epidemiological theory of host–parasite systems has been developed (Anderson 1991; Satō et al. 1994; Rand et al. 1995; Webb et al. 2007a, 2007b), based largely on the classical mean-field SIR model (e.g. Bairagi et al. 2007). However, the habitat in these studies is either homogeneous or made up of identical patches (e.g., Satō et al. 1994), with very few works really investigating the spatial and environmental heterogeneity in the landscape. The field is now beginning to recognize the importance and complexity of spatial structure of the eco-epidemiological system, particularly through the use of computer simulations. Empirical studies have revealed that the environmental fragmentation and heterogeneity are crucial to the disease transmission (Murray 1993; Garrett et al. 2007). It is, thus, necessary to explore the effect of fragmented landscape with spatially structured heterogeneity on the host–parasite dynamics.

Many methods have been used to study the spatial heterogeneity in ecological systems (Li et al. 2005; Okuyama 2007; Webb et al. 2007a, 2007b; Bolker and Pacala 1997; North and Ovaskainen 2007), among which the spatially stochastic simulation is often used (Hui and MeGeoch 2007; Webb et al. 2007b; Okuyama 2007; Su et al. 2008b). In particular, the role of spatially structured heterogeneity in the eco-epidemiological system has been examined successfully using this method (Hiebeler 2005; North and Ovaskainen 2007; Melbourne et al. 2007). A potential problem with such a method is that the analysis is often restricted to direct computer simulations, making mathematical analysis impossible. This dilemma can be resolved by using the method of moment-closure approximations (e.g., pair approximation) introduced by Matsuda et al. (1992); see also Iwasa 2000), which has been successfully applied in a wide range of ecological, epidemiological, and evolutionary systems (Satō et al. 1994; Keeling and Rand 1996; Ovaskavinen et al. 2002; Hiebeler 2005; Hui and MeGeoch 2007; Okuyama 2007; Su et al. 2008b). Pair approximation is a method for constructing a system of ordinary differential equations for global and local densities, and dealing with them as separate state variables that change over time (Iwasa 2000). Furthermore, it is also conceptually and mathematically similar to the join-count statistics that considers the correlations between and within focal species (Hui et al. 2006; Su et al. 2008b).

In this paper, we use the pair approximation and stochastic simulation to examine the effect of a heterogeneous landscape on classical host–parasite systems. We not only test the effect of the amount of habitat loss but also the spatial autocorrelation of lost patches. Previous eco-epidemiological theory using spatially random habitat loss is compared under different spatial structures. Specifically, the following three questions are to be addressed: (1) How does the spatially structured heterogeneity affect the invasion and transmission of the parasitic disease? (2) How do the occupancy and the spatial correlation of different populations respond to the environmental heterogeneity? (3) Do these two measures of spatial heterogeneity (i.e., the amount and the spatial autocorrelation of lost patches) impact on each other?

Model

To examine the effect of spatially structured heterogeneity on the host–parasite dynamics, a simple host–parasite lattice model of the micro-parasite SI systems was designed. The heterogeneous lattice environment contains two habitat types: suitable and unsuitable, where the unsuitable patches can not be occupied (Hiebeler 2000; Hiebeler and Morin 2007). The proportion of unsuitable patches in the landscape is described as p_u and indicates the amount of habitat loss. The clustering parameter $q_{u/u}$ represents the spatial autocorrelation of lost patches, and is the probability that a randomly chosen neighbor of an unsuitable patch is also found unsuitable. Therefore, the proportion of suitable patches is $p_s = 1 - p_u$, and the clustering degree of suitable patches can also be calculated as, $q_{s/s} = 1 - \frac{p_u}{1-p_u}\left(1 - q_{u/u}\right)$. Such a framework allows us to depict habitats that are not randomly distributed but rather clustered together by specific degrees and, thus, facilitate a systematic investigation of the impact of landscape structure on spatially explicit ecological processes and dynamics.

A classical lattice dynamical model of local host–parasite interactions is then placed on the above heterogeneous landscape. There are four possible states for each patch: unsuitable (1), suitable but empty (0), occupied by a susceptible individual (S), or by an infected one (I). A susceptible host can only reproduce if it is adjacent to a suitable empty patch. Parasitic infection happens through the contact of infected and susceptible individuals from neighboring patches. The parasitic disease transmits both horizontally (to neighbors) and vertically (to progenies) (Deredec and Courchamp 2006). An infected individual can, thus, reproduce a progeny in a neighboring suitable, empty patch, and also transmit the parasite to a susceptible neighbor through contact. The infected individual has a higher death rate than the susceptible individual due to the parasitic infection. A suitable, occupied patch becomes empty after the individual within it dies and can be reoccupied in the next time step by the progenies from individuals in the neighboring patches.

Pair approximation

Based on the rules described above, a pair approximation model can be constructed. Let the global density $P_\sigma(t)$ ($\sigma \in \{S, I, 0, 1\}$) denote the probability that a randomly chosen patch is in the state σ at time t. Doublet density $P_{\sigma\sigma'}$ is the probability that a randomly chosen pair of two neighboring patches is in state $\sigma\sigma'$. Local density $Q_{\sigma/\sigma'}$ is the conditional probability that a randomly chosen neighbor of a patch in state σ' is in state σ, and $Q_{\sigma/\sigma'\sigma''}$ is the conditional probability that a randomly chosen neighbor of the σ' patch in a $\sigma'\sigma''$ pair is in state σ ($\sigma'\sigma'' \in \{S, I, 0, 1\}$). The pair approximation model is as follows,

$$P'_S = rP_SQ_{0/S} - dP_S - \beta P_SQ_{I/S} \tag{1}$$

$$P'_I = \beta P_IQ_{S/I} + rP_IQ_{0/I} - (d+\alpha)P_I \tag{2}$$

$$P'_0 = dP_S + (d+\alpha)P_I - rP_0Q_{S/0} - rP_0Q_{I/0} \tag{3}$$

$$P'_{SS} = 2r\{\theta + (1-\theta)Q_{S/0S}\}P_{S0} - 2dP_{SS} - 2\beta(1-\theta)P_{SS}Q_{I/SS} \tag{4}$$

$$P'_{II} = 2\beta\{\theta + (1-\theta)Q_{I/SI}\}P_{SI} + 2r\{\theta + (1-\theta)Q_{I/0I}\}P_{I0} - 2(d+\alpha)P_{II} \tag{5}$$

$$P'_{00} = 2(d+\alpha)P_{I0} + 2dP_{S0} - 2r(1-\theta)P_{00}(Q_{S/00} + Q_{I/00}) \tag{6}$$

$$P'_{S0} = r(1-\theta)P_{00}Q_{S/00} - r\{\theta + (1-\theta)(Q_{S/0S} + Q_{I/0S})\}P_{S0} - dP_{S0} + dP_{SS} + (d+\alpha)P_{SI} - \beta(1-\theta)P_{S0}Q_{I/S0} \tag{7}$$

$$P'_{I0} = r(1-\theta)P_{00}Q_{I/00} + \beta(1-\theta)P_{S0}Q_{I/S0} + dP_{SI} + (d+\alpha)P_{II} - (d+\alpha)P_{I0} - r\{\theta + (1-\theta)(Q_{S/0I} + Q_{I/0I})\}P_{I0} \tag{8}$$

$$P'_{10} = dP_{S1} + (d+\alpha)P_{I1} - r(1-\theta)P_{10}(Q_{S/01} + Q_{I/01}) \tag{9}$$

$$P'_{1S} = -dP_{1S} - \beta(1-\theta)P_{1S}Q_{I/S1} + r(1-\theta)P_{10}Q_{S/01} \tag{10}$$

$$P'_{1I} = -(d+\alpha)P_{1I} + r(1-\theta)P_{10}Q_{I/01} + \beta(1-\theta)P_{1S}Q_{I/S1} \tag{11}$$

$$P'_{SI} = r(1-\theta)P_{I0}Q_{S/0I} + r(1-\theta)P_{S0}Q_{I/0S} + \beta(1-\theta)P_{SS}Q_{I/SS} - (2d+\alpha)P_{SI} - \beta P_{SI}\{\theta + (1-\theta)Q_{I/SI}\} \tag{12}$$

where r represents the reproduction rate, β the transmission rate of disease; $\theta = 1/z$ (z is the number of nearest neighboring patches); d the mortality rate of a host individual. An infected individual has an additional mortality (α) due to the infection. Furthermore, we have $P_{\sigma\sigma'} = P_\sigma Q_{\sigma'/\sigma} = P_{\sigma'\sigma} = P_{\sigma'}Q_{\sigma/\sigma'}$, (particularly, $P_{11} = p_u q_{u/u}$), $P_\sigma = \sum_{\sigma'} P_{\sigma\sigma'}$, $\sum_{\sigma'} Q_{\sigma'/\sigma} = 1$, $p_u = P_1$ and $p_s = P_0 + P_S + P_I$. According to the pair approximation, in which triplet densities are substituted by pair densities (i.e., $Q_{\sigma/\sigma'\sigma''} \approx Q_{\sigma/\sigma'}$) (Iwasa 2000; Su et al. 2008b), there are only seven independent variables namely P_S, P_I, P_{SS}, P_{II}, P_{0S}, P_{0I}, P_{SI}. All the other singlet and doublet probabilities can be calculated from these seven variables.

Spatial analysis

The analytical results from the pair approximation are too complicated to examine here. We, therefore, adopt two other methods for analyzing the host–parasite dynamics. First, we analyzed the spatial dynamics and equilibriums of the model using numerical solutions (Hui and MeGeoch 2007). The value of P_σ and $Q_{\sigma/\sigma'}$ at time step $t = 3000$ were assumed to be the equilibriums of the model. Second, the method of invisibility analysis was used to examine the disease invasion dynamics (Satō et al. 1994; Iwasa 2000; full detail is given in the Appendix).

To describe the spatial pattern of population distribution, we adopt a commonly used spatial correlation index, join-count statistics (Su et al. 2008b). Through counting the occupancies and spatial correlations, we can classify population distributions into spatially aggregated, segregated, and random (Hui et al. 2006), which is also consistent with the definition of the spatial structure of landscape. Spatially explicit aggregation can be described as $C_\sigma = Q_{\sigma/\sigma} - P_\sigma > 0$, implying the positive first-order spatial correlation between two adjacent occupied patches. The spatially random distribution has $C_\sigma = 0$ and indicates the independence of two adjacent, occupied patches. The spatially segregated distribution can be depicted by $C_\sigma < 0$, i.e., a negative spatial correlation between two adjacent patches (Hui et al. 2006; Hui and MeGeoch 2007).

Mean-field approximation

We also presented the results from the mean-field approximation, for purposes of comparison. This is equivalent to assuming $Q_{\sigma/\sigma'} = P_\sigma$, indicating the probability that an adjacent patch occupied by σ is the same as the global density of population σ. Therefore, we have the following mean-field approximation model,

$$P'_S = rP_S(1 - p_u - P_S - P_I) - dP_S - \beta P_SP_I \tag{13}$$

$$P'_I = \beta P_SP_I - (d+\alpha)P_I + rP_I(1 - p_u - P_S - P_I) \tag{14}$$

Individual-based model

Spatially stochastic simulations were also designed to check the validity and robustness of the pair approximation. All simulations were run on a two-dimensional lattice network of 100×100 patches. The landscapes with spatially structured heterogeneity were generated using Hiebeler's (2000) algorithm. Synchronized updating and the von Neumann neighborhood were also adopted (Hui and MeGeoch 2007). Periodic boundaries

were used (Su et al. 2008b). Initially, host individuals (susceptible and infected) were located randomly in the suitable patches because equilibrium results in such models are not sensitive to initial conditions (Hiebeler and Morin 2007; Webb et al. 2007a). For each combination of model parameters, five repeats were run and the results were presented as the mean and standard deviation for comparison. A snapshot of the typical spatial patterns from simulations in landscapes with different degrees of spatial heterogeneity was given in Fig. 1 ($t = 3000$).

Results

A comparison of the results from various amounts of habitat loss p_u predicted by the pair approximation and the mean-field approximation are shown in Fig. 2a. There are four areas in the r–β phase diagram from the pair approximation, which was similar to the result from the mean-field approximation (Fig. 2a). These four areas represent: host population extinct due to the excess mortality, disease-free, endemic but partial infected, and entirely infected. The areas of "excess mortality" and "disease-free" expanded with the increase of p_u, but the "entirely infected" area shrank (Fig. 2a). Infected hosts were more sensitive to habitat loss than susceptible hosts because the former were at a higher artificial trophic level than the latter. At the highest values of p_u, the "entirely infected" area disappeared in the pair approximation model, but not in the mean-field approximation model (Fig. 2a). The difference indicates that the non-spatially-structured model can overestimate the density of infected hosts. Figure 2a also showed that for a given reproduction rate, the thresholds of disease invasion and entire infection both increased monotonically with the increase of p_u. Results from the thresholds of disease invasion, measured as the minimum amount of habitat loss p_u for disease persistence, were consistent with the above results (Figs. 2a and 3a). The individual-based model was found to return a higher value than pair approximation. Furthermore, the "entirely infected" area expanded with the increase of $q_{u/u}$ while areas of "excess mortality" and "disease-free" both shrank (Fig. 2b). The invasion thresholds of disease under different $q_{u/u}$ were also illustrated in Fig. 3b. The decline in invasion threshold and the increase in total infection area with the increase of the spatial autocorrelation $q_{u/u}$ suggest that a high clustering degree of spatial heterogeneity favors the invasion and transmission of disease.

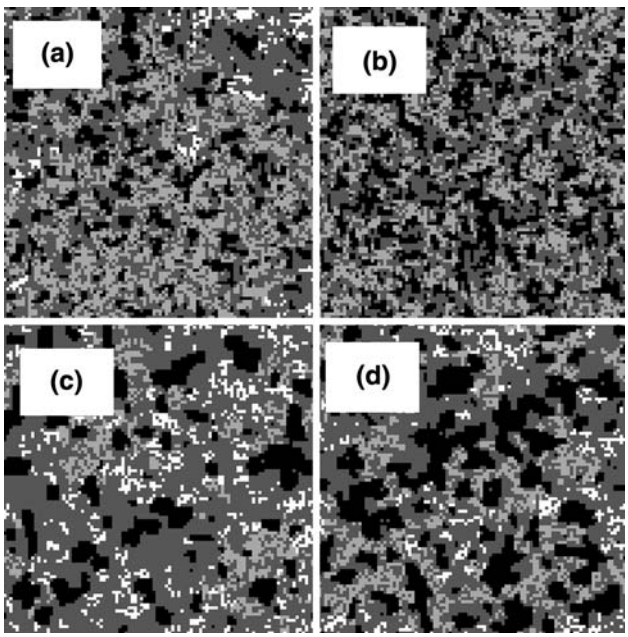

Fig. 1 Spatial distributions of population under different spatial heterogeneity in a 100×100 lattice system. The snapshots were taken at the 3,000th time step. The *white squares* represent infected hosts (parasite) and the *light gray squares* indicate patch occupied by susceptible hosts. The *dark gray squares* represent the empty and suitable patches, and *black squares* denote unsuitable patches. Parameters: $d = 0.1$, $\alpha = 0.05$, $r = 0.25$, $\beta = 0.5$. Other parameters: **a** $p_u = 0.2$, $q_{u/u} = 0.6$; **b** $p_u = 0.35$, $q_{u/u} = 0.6$; **c** $p_u = 0.2$, $q_{u/u} = 0.8$; **d** $p_u = 0.35$, $q_{u/u} = 0.8$

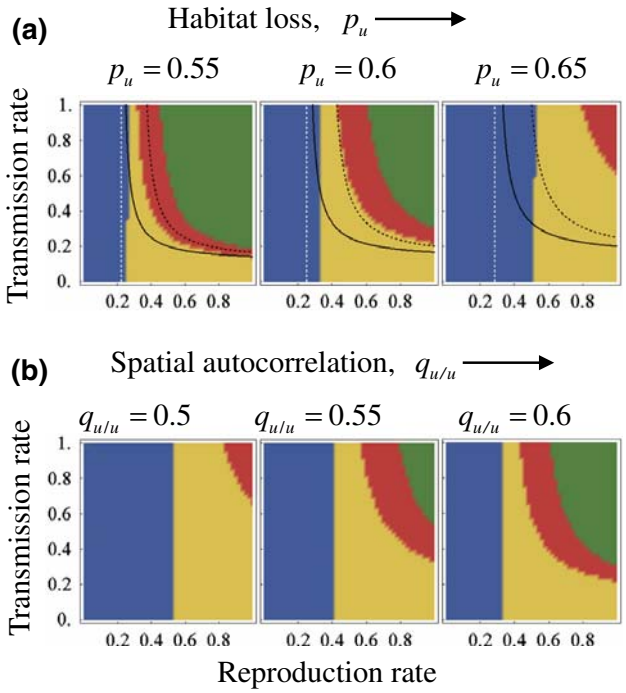

Fig. 2 The influence of the amount (p_u) and spatial clustering ($q_{u/u}$) of habitat loss on a spatial host–parasite system. **a** A comparison between results from pair approximation and mean-field approximation under various p_u with $q_{u/u} = 0.6$. The *white vertical dashed lines* in phase diagrams of the mean-field approximation model denote the minimum host reproduction rate for survival, $r > d/(1 - p_u)$; the *solid curves* indicate the invasion threshold, $\beta > \alpha/(1 - p_u - d/r)$; the *black dashed curves* represent the entire infection, $\beta > \alpha/(1 - p_u - (d + \alpha)/r)$. **b** The bifurcation regions of pair approximation under different values of $q_{u/u}$ with $p_u = 0.6$. The parameter space can be divided into four areas corresponding to host extinction (*blue*), disease-free (*yellow*), partial infection (*red*) and entire infection (*green*). Parameters: $d = 0.1$, $\alpha = 0.05$, $z = 4$

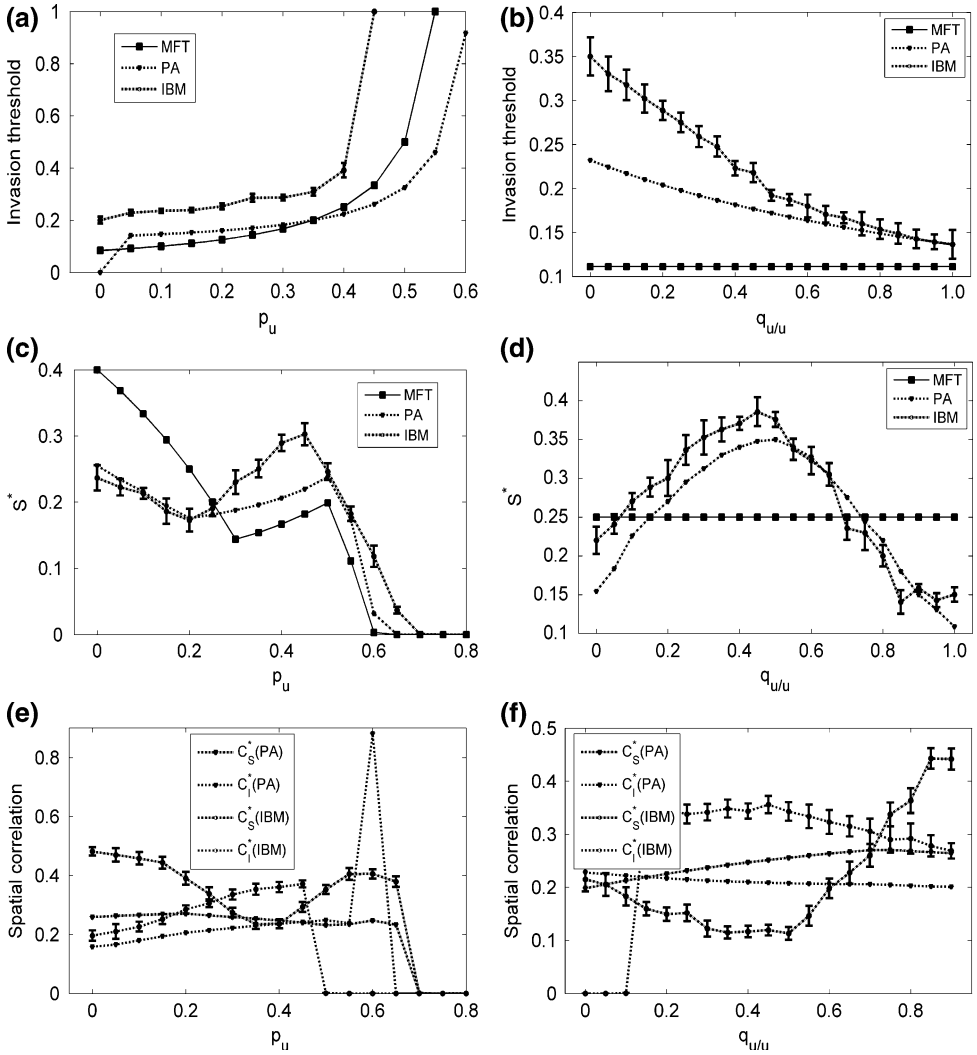

Fig. 3 The invasion thresholds, relative occupancy of the total hosts (S^*), and the spatial correlations of susceptible and infected hosts (C_S^*, C_I^*) at equilibrium as a function of the amount (p_u) and the spatial clustering of lost patches ($q_{u/u}$) under three different models, respectively. 'MFT', 'PA', and 'IBM' represent the mean-field approximation, pair approximation, and individual-based simulation models. The *error bars* represent the standard deviation from five independent individual-based simulations. Parameters: **a** $q_{u/u} = 0.75$; **b** $p_u = 0.15$; **c**, **e** $\beta = 0.5$, $q_{u/u} = 0.7$; **d**, **f** $\beta = 0.5$, $p_u = 0.2$. Other parameters: $d = 0.1$, $\alpha = 0.05$, $r = 0.25$, $z = 4$

To further examine the effect of habitat loss, we presented the relative occupancy of total host populations ($S^* = (P_s^* + P_I^*)/p_s$) and the spatial correlations of susceptible hosts (C_S^*) and infected ones (C_I^*) (Fig. 3c, e). First, all three models showed that S^* decreased at first but eventually increased with the increasing amount of habitat loss, but declines again with increase of p_u (Fig. 3c). As noted, the interim increase because the infected hosts decayed at very fast rate, just before becoming extinct. The shape of S^* changed again after the infected hosts had been driven extinct, which was due to the fact that the host density per patch was not sufficiently high at very serious habitat fragmentation. Second, the spatial correlation of susceptible hosts C_S^*, from the simulation, declined monotonously at first and then increased gradually with p_u, whereas no clear decline was observed from the pair approximation (Fig. 3e). With the increasing of p_u, the spatial correlation of infected hosts C_I^* increased under both models (Fig. 3e), indicating a high degree of clustering of infected hosts under severe habitat loss. The value of C_I^* became zero after the extinction of infected hosts.

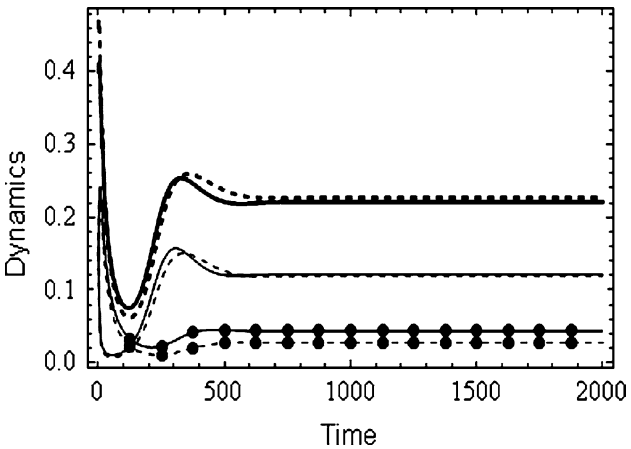

Fig. 4 The temporal dynamics of global densities of susceptible hosts (P_s, *light lines*), infected ones (P_I, *thick lines with dots*) and the relative occupancy of the total hosts (S, *thick lines*) based on the pair approximation model under the two different heterogeneity scenarios: $p_u = 0.25$, $q_{u/u} = 0.25$ (*solid lines*); $p_u = 0.35$, $q_{u/u} = 0.5$ (*dashed lines*). Parameters: $d = 0.1$, $\alpha = 0.05$, $r = 0.25$, $\beta = 0.5$, $z = 4$

Figure 3d and f display the equilibrium of the relative occupancy of total host populations and the spatial correlations of susceptible and infected hosts as $q_{u/u}$ varied. The mean-field approximation, failed to perceive the spatial structure of the landscape. Both individual-based simulations and the pair approximation showed that S^* increased monotonously at first and then declined with the increase of $q_{u/u}$ (Fig. 3d) because infected host increased at high values of $q_{u/u}$ and eventually infected the susceptible hosts at an even higher rate. Figure 3f illustrates the influence of spatial clustering of the landscape on the spatial correlations of susceptible and infected hosts. Spatial correlation of susceptible hosts C_S^* increased with the clustering level of lost patches, while this increasing rate based on the individual-based model was higher than from pair approximation (Fig. 3f). Intriguingly, when $q_{u/u} < 0.55$, the spatial correlation of susceptible hosts obtained from pair approximation was higher than from the individual-based simulation. The spatial correlation of infected hosts C_I^* declined slightly with the increasing of $q_{u/u}$. These results suggest that increasing the clustering degree of lost patches has the same effect as reducing the amount of habitat loss. Since the amount of habitat loss and spatial correlation of lost patches had the opposite effects, then increasing p_u and $q_{u/u}$ at the same time may initiate a trade-off, resulting in a similar equilibriums not only for global densities (P_σ) but also the relative occupancy ($S = (P_S + P_I)/p_S$) under two different scenarios of spatial heterogeneity ($p_u = 0.25$, $q_{u/u} = 0.25$ and $p_u = 0.35$, $q_{u/u} = 0.5$) (Fig. 4).

Discussion

Spatial heterogeneity in landscape is a ubiquitous phenomenon in the natural world that has challenged many assumptions in theoretical work. Because the amount and the spatial structure of habitat loss together determine the fragmentation level of the landscape, studies of such spatial heterogeneity are justified (North and Ovaskainen 2007). Since Satō et al. (1994) firstly showed that spatial structure can dramatically affect the parasite (e.g., the emerging of parasite-driven extinction), the spatial host–parasite interaction has been of interest in population ecology (Webb et al. 2007a, 2007b). Following Hiebeler (2000), the effect of fragmented landscapes on the host–parasite system has been properly examined here. This work follows a prominent research direction (Hirzel et al. 2007), and thus provides an extension to the field of eco-epidemiology.

Due to both the horizontal and vertical disease transmission, four possible outcomes emerged (Fig. 2), similar to results predicted from Deredec and Courchamp (2006). Regarding the amount of habitat loss p_u, habitat loss can impede the invasion and transmission of parasitic disease, and is consistent with empirical studies (Garrett et al. 2007). Severe habitat loss will increase the chance of dispersing the progeny of infected hosts into unsuitable patches. Furthermore, habitat loss can decrease the habitat connectivity and form a boundary between suitable and unsuitable patches (North and Ovaskainen 2007), which can favor the susceptible host in escaping from infection. Interestingly, the infected host is more vulnerable to habitat loss than the susceptible host, even though they are originally in the same trophic level. This is because the host–parasite interaction pushes the infected host to a higher trophic level than the susceptible one. Meanwhile, results also identified that there exists a trade-off between two opposite effects of habitat loss for susceptible hosts (Fig. 3c): decrease of the host fitness (negative) and inhibition of disease transmission (positive). Within a certain range of habitat loss, susceptible hosts can counterbalance the positive and negative effects. This result is consistent with previous studies of the effect of habitat loss in the predator–prey systems (Bascompte and Solé, 1998) and in altruistic games (Zhang et al. 2005).

Spatial clustering of habitat loss has the opposite effect on disease transmission, and therefore the amount of habitat loss and its spatial configuration can interplay and initiate a trade-off (Fig. 4). When increasing the clustering degree of lost patches, the clustering degree of suitable patches also increases. Consequently, more patches are bordered by those with identical habitat types (Hirzel et al. 2007). Therefore, the spatial clustering of lost patches can favor the invasion and transmission of disease. For a community with competition interactions, the environmental heterogeneity may improve the invasion of exotic species (Melbourne et al. 2007). This is because much higher heterogeneity (fragmentation) could reduce the interspecific conflict, forming refuge areas for less competitive species. Our work indicates that whether or not spatial heterogeneity can affect the success of an invasion is dependent on the particular ecological process that is under consideration.

Spatial interactions and heterogeneity have always been under the spotlight in ecology, and there are a number of ways to explore their implications (Hiebeler 2000; Okuyama 2007). The individual-based stochastic model and moment-closure approximations have been two successful methods (Keeling and Rand 1996; Hiebeler 2005; Webb et al. 2007a, 2007b). Pair approximation and stochastic simulations can capture the characteristics of spatially structured heterogeneity, where the mean-field method fails. Results indicated that pair approximation can maintain the qualitative behaviors of host–parasite model based on the stochastic simulation, although some differences still exist. For example, spatially stochastic simulation predicted a higher value for the invasion threshold of disease than predicted using pair approximation (Fig. 3a, b). The demographic stochasticity in the spatial stochastic simulation model is more realistic, and makes the invasion of disease more difficult than predicted by the pair

approximation. Read and Keeling (2007) have shown that the stochasticity can induce an evolutionary instability in infectious diseases. This issue of stochasticity in epidemiological systems deserves further study.

Environmental heterogeneity can usually be classified into three major types: temporal heterogeneity, fixed spatial heterogeneity, and invader-driven heterogeneity (Melbourne et al. 2007). Recent progress in the theory of invasion has also indicated that environmental heterogeneity plays a defining role in the outcome of invasions and the rate of spread (Melbourne et al. 2007). We only discussed the effects of fixed spatial heterogeneity on the host–parasite system. Further explorations about the roles of other types of environmental heterogeneity are needed. Moreover, a study of the case with only horizontal transmission rather than both vertical and horizontal transmission is another interesting issue, and then would be explored in the future. An improved pair approximation method that can minimize the deviation between its predictions and results from stochastic simulations is also worth pursuing in solving relevant questions in applied ecology (Satō et al. 1994).

Conclusions

Spatial heterogeneity is definitely a key factor affecting population dynamics and should be considered in all cases.

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Appendix: invasion criteria

We calculate the critical transmissibility of disease in detail. Let v denote the vector (P_I, P_{IS}, P_{II}, P_{I0}, P_{I1}), the equations involving I can be written as $\frac{dv}{dt} = M \cdot v$, where

$$M = \begin{pmatrix} -d-\alpha & \beta & 0 & r & 0 \\ 0 & \beta(1-\theta)Q_{S/S} - (2d+\alpha) - \beta\theta & 0 & 2r(1-\theta)Q_{S/0} & 0 \\ 0 & 2\beta\theta & -2(d+\alpha) \cdots & 2r\theta & 0 \\ 0 & d+\beta(1-\theta)Q_{0/S} & d+\alpha & -(d+\alpha) - r\big(\theta+(1-\theta)Q_{S/0}\big) + r(1-\theta)Q_{0/0} & 0 \\ 0 & \beta(1-\theta)Q_{1/S} & 0 & r(1-\theta)Q_{1/0} & -d-\alpha \end{pmatrix}$$

.

Now, we derive the global and local densities at their stationary values for the disease-free equilibrium. From $P_S + P_1 + P_0 = 1$, $P_1 = p_u$, we can get

$$P_0 = 1 - p_u - P_S \tag{15}$$

Eq. (1) yields

$$Q_{0/S} = \frac{d}{r} \tag{16}$$

and after substituting it into $P_S Q_{0/S} = P_0 Q_{S/0}$ can lead to

$$Q_{S/0} = \frac{d}{r}\frac{P_S}{1-p_u-P_S}. \tag{17}$$

From Eq. (6), we get

$$Q_{0/0} = \frac{d}{r(1-\theta)}. \tag{18}$$

Eq. (7) gives $Q_{S/S} = \theta + (1-\theta)Q_{S/0}$, after replacing (17) then yields

$$Q_{S/S} = \theta + (1-\theta)\frac{d}{r}\frac{P_S}{1-p_u-P_S}. \tag{19}$$

Substituting $Q_{0/1} = 1 - Q_{1/1} - Q_{S/1}$, $Q_{1/1} = q_{u/u}$ and (17) into Eq. (10), we can obtain

$$Q_{S/1} = \big(1-q_{u/u}\big)\frac{(1-\theta)P_S}{1-p_u-\theta P_S}. \tag{20}$$

Using $P_1 Q_{S/1} = P_S Q_{1/S}$, then

$$Q_{1/S} = p_u\big(1-q_{u/u}\big)(1-\theta)\big/(1-p_u-\theta P_S). \tag{21}$$

According to $P_1 Q_{0/1} = P_0 Q_{1/0}$, we can get

$$Q_{1/0} = \frac{p_u\big(1-q_{u/u}\big)}{1-p_u-\theta P_S} \tag{22}$$

Substituting (16), (19) and (21) into $Q_{0/S} + Q_{1/S} + Q_{S/S} = 1$, we then get the equation involving the value P_s, and solve it to give all other global and local densities at the disease-free equilibrium.

Substituting these stationary values for the disease-free equilibrium into M and evaluating $|M - \lambda I|$ gives a characteristic polynomial $(\lambda + d + \alpha)^2 H(\lambda)$, where

$$H(\lambda) = \lambda^3 + c_2\lambda^2 + c_1\lambda + c_0. \tag{23}$$

The expressions for c_i are rather lengthy and we omit them for brevity. Clearly the polynomial has two negative eigenvalues ($\lambda_1 = \lambda_2 = -d - \alpha$). According to Routh–Hurwitz conditions, the zeros of $H(\lambda)$ have $\mathrm{Re}(\lambda) < 0$ must satisfy:

$$c_2 > 0, \quad c_1 c_2 - c_0 > 0, \quad c_0 > 0 \tag{24}$$

Therefore, we can get the critical transmissibility of disease through analyzing Eq. (24) numerically for fixed parameters.

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