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The effect of predation on the prevalence and aggregation of pathogens in prey

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ABSTRACT

Although pathogens and predators have been widely used as bio-control agents against problematic prey species, little has been done to examine the prevalence and aggregation of pathogens in spatially structured eco-epidemiological systems. Here, we present a spatial model of a predator–prey/host–parasite system based on pair approximation and spatially stochastic simulations, with the predation pressure indicated by predator abundance and predation rates. Susceptible prey can not only be infected by contacting adjacent infected individuals but also by the global transmission of pathogens. The disease prevalence was found to follow a hump-shaped function in response to predation pressure. Moreover, predation pressure was not always negatively correlated with pathogen aggregation as proposed from empirical studies, but depending on the level of predation pressure. Highly connected site network facilitated the parasites infection, especially under high predation pressure. However, the connectivity of site network had no effect on the prevalence and aggregation of pathogens that can infect health prey through global transmission. It is thus possible to better design biological control strategies for target species by manipulating predation pressure and the range of pathogen transmission.

1. Introduction

Pathogen infection and predation are distinctive but analogous inter-specific interactions, with each an important field of research in its own right (Bairagi et al., 2007; Raffel et al., 2008; Mata-Machuca et al., 2010; Kihara et al., 2011). Eco-epidemiology that considers both ecological and epidemiological dynamics ties these two fields together and has attracted increasing attention (Chattopadhyay and Bariagi, 2001; Webb et al., 2007a; Bairagi et al., 2007; Su et al., 2009a; Greenman and Hoyle, 2010). Studies in eco-epidemiology have provided increasing insights to the complex dynamics in the system and their applications in conservation management, such as the biological control of problematic species using their natural enemies through the interplay of disease transmission and predation (Holt and Roy, 2007; Greenman and Hoyle, 2010). For instance, although predation has been viewed as an important force to prevent successful invasion of pathogens into prey (Packer et al., 2003; Bairagi et al., 2007), the prevalence of pathogenic diseases can, nonetheless, enhance the predation risk (Hethcote et al., 2004; Hatcher et al., 2006; Williams, 2008). As such, understanding such complex processes and dynamics in eco-epidemiological systems and elucidating the impact of predation on the control of epidemics are necessary and have important implications in wildlife conservation and management.

Theoretical studies have provided certain propositions regarding the impacts of predation on the pathogen loads in natural predator–prey/host–parasitoid systems (Packer et al., 2003; Bairagi et al., 2007; Roy and Holt, 2008; Williams, 2008; Greenman and Hoyle, 2010). Evidently, because predators prefer infected prey as easy targets, they can potentially alter the prevalence of disease in prey population (Hudson et al., 1992; Packer et al., 2003; Ostfeld and Holt, 2004; Hall et al., 2005; Roy and Holt, 2008). Packer et al. (2003) thus suggest that the removal of predators can be indirectly detrimental to prey and facilitate pathogen invasion and transmission (also see Bairagi et al., 2007; Williams, 2008). However, recent work has questioned the generality of Packer et al.'s proposition by demonstrating results that depend on prey's mechanisms of population regulation (Holt and Roy, 2007; Roy and Holt, 2008). For instance, if considering the acquired immunity in prey, the overall relationship between pathogen prevalence and predator abundance could be hump-shaped (Holt and Roy, 2007). Enhanced predation pressure (either by manipulating predator density or enhancing predation efficiency) could also facilitate the transmission of a pathogen under certain circumstances (Greenman and Hoyle, 2010).

Preference in predation can also have a bearing on pathogen transmission. Although studies often consider that predators prefer infected (less active) prey (Chattopadhyay and Bariagi, 2001; Hethcote et al., 2004; Bairagi et al., 2007), it has also been suggested that such preferential predation could also depend on the type of pathogens and the life-history characteristics of predators (Dawkins, 1982; Bhattacharyya and Mukhopadhyay, 2010).

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The effect of predation preference on pathogen transmission and prevalence is worthy of additional investigation.

Another important factor in eco-epidemiology is the spatial structure of prey which affects and is also affected by pathogen transmission (Bonsall and Hassell, 2000; Su et al., 2009a). For a parasitic infection that transmits to nearby hosts via direct contact, traditional analyses based on the mean-field assumption that individuals are well mixed and have equal probability to encounter one another could be inappropriate. In this regard, using pair approximation to consider the spatially explicit dynamics has allowed for better understanding the role of spatial structure (autocorrelation) in a wide range of ecological and epidemiological questions (e.g. Satō et al., 1994; Tilman and Kareiva, 1997; Hui and McGeoch, 2007; Webb et al., 2007a,b; Okuyama, 2008). Invasion and persistence of pathogens have been shown to become more difficult under realistic spatial structure as opposed to under the mean-field assumption (Boots and Sasaki, 1999; Bauch, 2005; Webb et al., 2007a). Various degrees of spatial autocorrelation have been observed in healthy and infected prey (Boots and Sasaki, 1999), and it is therefore necessary to examine the effect of a full range of spatial autocorrelation on the eco-epidemiological dynamics.

One important finding when considering the spatial structure in eco-epidemiology is the role of aggregated distribution on the persistence of prey and pathogens (Shaw et al., 1998; Joly and Messier, 2004). Recent works further suggest that the aggregation of pathogen distribution can be sensitive to predation (Rousset, 1996; Joly and Messier, 2004). For instance, Joly and Messier (2004) show that the aggregation of parasites (*Echinococcus granulosus*) in the moose hosts could be reduced by the parasite-induced vulnerability to predation by wolfs. Clearly, it is impossible to quantify the aggregation of parasites and pathogens using the mean-field approximation (Su et al., 2009b). Pair approximation enables such spatially explicit analysis of species aggregated distribution (Satō et al., 1994; Su et al., 2009b) and is thus suitable for examining the potential effect of the aggregation in eco-epidemiological systems.

We here explore the effect of predation (pressure and preference) on the epidemic transmission in spatially structured prey populations. Using a combination of approximation techniques and spatially stochastic simulations, we examine the effect of adding spatial components on the pathogen transmission and aggregation in an eco-epidemiological system. Specifically, we examine how local reproduction and a mixture of local and global pathogen transmission on the prevalence and aggregation of diseases in prey. We provide a threshold for pathogen invasion based on the analysis of invasion matrix (Greenman and Hoyle, 2010). Given the conceptual similarity between the join-count statistics and pair approximation (Hui et al., 2006, 2010; Su et al., 2009b), we further explore the aggregation degree of infected prey in response to different predation pressure and preference, as well as different transmission types, which has not been previously studied for eco-epidemiological systems. These analyses enable us to address the following questions: (1) how does the mean-field dynamics change after considering the spatial structure of prey and diseases? (2) How do the predation pressure and preference of predators affect the invasion, prevalence and aggregation of pathogens? (3) How does the spatial structure of infected prey change in response to different levels of predation pressure and transmission rates? Answers to these questions can shed light on the entangled dynamics and processes in eco-epidemiological systems and provide clues for more efficient biodiversity conservation and management.

2. Model

Pair approximation is a method for analyzing the first-order join-count spatial autocorrelation (Fortin and Dale, 2005; Hui et al., 2010) and for tracing the global and local densities in a dynamical system (Keeling and Rand, 1996; Hiebeler, 2005; Su et al., 2009b). We designed a pair approximation model for a predator–prey system, where the prey is also the host of a transmissible pathogen in an epidemiological system and can either be susceptible to, infected by, or recovered from the pathogenic disease. Following Packer et al. (2003) and Roy and Holt (2008), we only considered the predator abundance (P_C) as a surrogate for predation pressure. The model was run on a network of sites, with each representing either empty (0) or occupied by a susceptible prey (S), an infected one (I) or a recovered individual (R). Each susceptible or recovered individual reproduced at a rate of r and deposited its offspring into adjacent empty sites (Fig. 1a). Reproduction from infected individuals was also included but at a lower rate (rf, where $f \in [0, 1]$). Susceptible prey can be infected by the pathogen either through global transmission from any other infected prey (at a rate of βL_I, where β indicates the transmission rate of infection and L_I ($0 \leq L_I \leq 1$) denotes the proportion of global transmission) or through direct contact with surrounding infected individuals (at a rate of $\beta(1 - L_I)$, Fig. 1b). The recovery from infection (at a rate of ϖ) can lead to immunity from further infection (Fig. 1a). The mortality of hosts (prey) consisted of three components: intrinsic death rate ($m_{\sigma 0}$, $\sigma \in \{S, I, R\}$), pathogen-induced death rate (μ and predation-induced death rate $\alpha_\sigma P_C$, where α_σ is the predation rate). The enhanced mortality due to predation can be represented as $m_\sigma = m_{\sigma 0} + \alpha_\sigma P_C$, where we assumed $m_{S0} = m_{R0} = m$ for susceptible and recovered prey and $m_{I0} = m + \mu$ for infected prey. The definition of parameters and estimated values are summarized in Table 1.

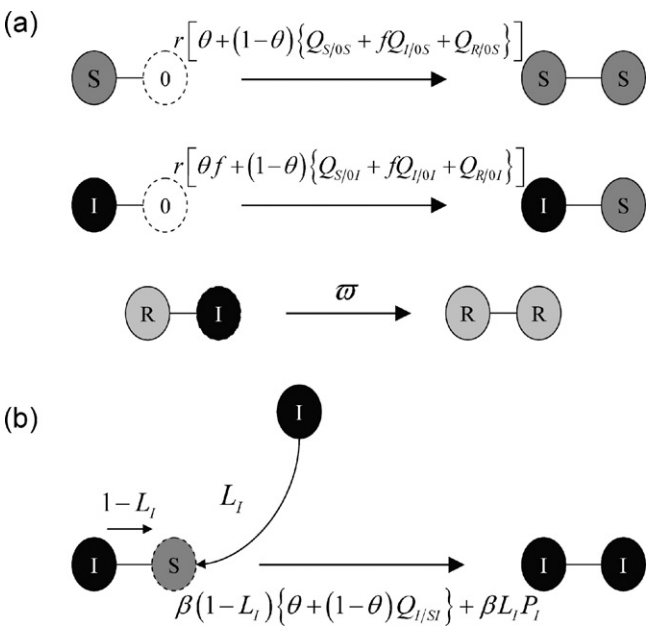

Fig. 1. (a) The reproductive and recover process with possible pair states and their transition probability. (b) The infection process of a susceptible individual who can be infected from a nearest-neighbor site at probability $1 - L_I$ and globally from a distant site at probability L_I.

Let the global density $P_{\sigma'}(t)$ ($\sigma' \in \{S, I, R, 0\}$) denote the probability that a randomly chosen site is in state σ' at time t. Doublet density $P_{\sigma'\sigma''}$ represents the probability that a randomly chosen pair of two adjacent sites is in state $\sigma'\sigma''$. Local density $Q_{\sigma'/\sigma''}$ represents the conditional probability that a randomly chosen neighbor of a site in state σ'' is in state σ', and $Q_{\sigma'/\sigma''\sigma'''}$ is the conditional probability that a randomly chosen neighbor of the σ'' site in a $\sigma''\sigma'''$ pair is in state σ' (σ'', $\sigma''' \in \{S, I, R, 0\}$). By comparing the global and local densities of prey, we can classify the spatial distribution of individuals into aggregated, segregated and random (Fortin and Dale, 2005; Hui et al., 2006, 2010). A spatially aggregated