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Effects of the transmissibility and virulence of pathogens on intraguild predation in fragmented landscapes

Min Su [a,b,*], Cang Hui [c,d], Zhenshan Lin [a]

[a] School of Geography Science, Nanjing Normal University, Nanjing 210046, China
[b] School of Mathematics, Hefei University of Technology, Hefei 230009, China
[c] Centre for Invasion Biology, Department of Mathematical Sciences, Stellenbosch University, Matieland 7602, South Africa
[d] Mathematical and Physical Biosciences, African Institute for Mathematical Sciences, Muizenberg 7945, South Africa

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ABSTRACT

It is well known that pathogenic infection can have a profound effect on the outcome of competition and predation, however the role of pathogenic infection in systems where predators and prey also compete for other resources is yet to be explored (i.e. in systems of intraguild predation). Using a cellular automaton model, we here explore the effect of pathogenic infection on the spatial dynamics of species that also engage in intraguild predation (IGP) in a fragmented landscape. First, the shared pathogen by the predator and prey can enhance species coexistence in the IGP system, consistent with results for non-spatial IGP systems. Second, equilibrium population sizes of the predator and prey depend crucially on the pathogen virulence to the predator but are insensitive to the change in the virulence to the prey. This asymmetric response to virulence change is due to the fact that the predator species has to juggle between predation, resource competition and pathogenic infection. Finally, the response of the pathogen to habitat fragmentation is largely determined by its life-history strategy (transmissibility and virulence) and the trophic level of its host. These results enrich our understanding on the role of pathogens in the ecosystem functioning of eco-epidemiological systems.

1. Introduction

The interaction between pathogens and their hosts is among the most intimate biotic interactions. Understanding how and to what extent pathogens affect interaction strength, community structure and species distribution is central to theoretical ecology (Anderson and May, 1991; Prenter et al., 2004; Wood, 2006; Hatcher et al., 2006, 2008). To this end, eco-epidemiology as a new branch in mathematical biology focuses especially on integrating epidemiological processes into systems of community ecology (Chattopadhyay and Arino, 1999; Holt and Dobson, 2006; Sieber and Hilker, 2011). Studies in eco-epidemiology have provided increasing insight into understanding the dynamics of complex systems and enhancing the efficacy of conservation management (Chattopadhyay and Arino, 1999; Byers, 2009; Su and Hui, 2011). Current theories have demonstrated that a pathogen can alter or even reverse the outcome of competition, induce complex dynamics in predator–prey systems, affect the structure of species distribution, and cause trophic cascades in food webs (Lafferty et al., 2006, 2008). Although some eco-epidemiological systems have been examined in depth, knowledge gaps remain, in particular for communities comprising complex biotic interactions. The presence of complex community structures offers a wealth of opportunity for a pathogen to jump from one host species to another, often with unexpected repercussion at the population and community level.

Intraguild predation (IGP) is a common community interaction module and depicts the competition for other resources between predators and their prey (Polis et al., 1989; Arim and Marquet, 2004; Amarasekare, 2006). Although IGP plays an important role in community structure and stability, how pathogens affect the IGP dynamics remains an emerging research focus (Hatcher et al., 2006). The coexistence of predators and prey in an IGP system is possible only if the prey is a superior resource competitor compared to the predator due to the obvious trophic advantage of the predator to its prey (Polis et al., 1989; Holt and Polis, 1997). However, some evidence has shown that pathogens can modify the strength of IGP interaction through both direct and indirect effects. Pathogen-induced change in competitive and foraging abilities can affect the coexistence of multiple predator species in an IGP system (MacNeil et al., 2004; Hatcher et al., 2006; Sieber and Hilker, 2011).

* Corresponding author at: School of Mathematics, Hefei University of Technology, Hefei 230009, China. Tel.: +86 55162902592.
E-mail address: sum04@163.com (M. Su).

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Specifically, Hatcher et al., (2008) have demonstrated that parasites can broaden the condition of coexistence when the pathogen exerts a greater deleterious effect on the predator.

It is well-known that the success of a transmissible pathogen depends on its life-history traits, including the mode of transmission, the virulence (pathogen-induced mortality of the host), and the immunity of the host (Morand and Gonzalez, 1997; Hatcher et al., 2008; Horns and Hood, 2012; Webb et al., 2013). In a multi-host system, high virulence and transmissibility can result in the exclusion of the host that is a superior competitor (Schmitz and Nudds, 1994). An extremely high transmissibility can even lead counter-intuitively, to the disappearance of the pathogen for a long period (Sun et al., 2010). To this end, it is reasonable to speculate that these life-history traits will also affect the spatial distribution of pathogens and their hosts, especially when pathogenic infection occurs through direct contacts of infected and susceptible individuals (Rand et al., 1995; Keeling, 1999). The spatial structure in this system could even lead to pathogen-driven extinction (Webb et al., 2007) and consequently, the spatial structure has been explicitly taken into account when examining the transmission dynamics and the evolution of virulence (Boots and Sasaki, 1999; Haraguchi and Sasaki, 2000; Boots et al., 2004; Webb et al., 2007, 2013; Su et al., 2008a, 2009a).

An important process that can affect the species distribution and survival is habitat destruction (Tilman, 1994; Su et al., 2009b). Habitat fragmentation per se is a landscape-level phenomenon in which species that survive in habitat remnants are confronted with a modified environment of reduced area, increased isolation and novel ecological boundaries (Ewers and Didham, 2006; North and Ovaskainen, 2007). Empirical and theoretical studies have revealed that habitat fragmentation (one component of habitat destruction) can indeed change the behaviour of host-pathogen dynamics (Ewers and Didham, 2006; Su et al., 2009b). Heterogeneous habitats offer a variety of refuge niches and thus can promote survival and coexistence of species (Bonsall and Hassell, 2000). As such, fragmented landscapes could become detrimental to the invasion and transmission of pathogens (Su et al., 2009a). Interestingly, the effect of habitat destruction is also mediated by the life-history traits of the pathogen (Froeschke et al., 2013). To this end, it is important to examine (1) how the transmission dynamics of pathogens is influenced by the spatial structure of habitat, and (2) how the coupling of habitat structures and pathogen life-history traits affects the dynamics of an IGP system.

Here, we examine the role of habitat destruction in the spatiotemporal dynamics of pathogen transmission in a multi-trophic eco-epidemiological IGP system. Habitat destruction has normally been analysed by making a stipulated fraction of habitat patches unavailable to a focal species (With, 1997), often in clusters. We develop a cellular automaton (CA) to examine the spatial pattern formation in the IGP system under different levels of habitat destruction and different sets of pathogen life-history traits. CA is the simplest description for nearest neighbour interactions and is preferred in studies when stochasticity and individual interactions are essential (Boots and Sasaki, 1999; Haraguchi and Sasaki, 2000).

2. Model

We examined the spatial dynamics of the IGP system in a lattice landscape, with each cell being either suitable or unsuitable to the predators and prey. A fractal landscape was generated using the mid-point displacement algorithm (With, 1997), and a binary landscape was created by assigning a fraction (h) of cells with the lowest values in the fractal landscape as unsuitable. Both the degree of spatial autocorrelation of the fractal landscape (measured by the roughness constant, H) and the proportion of unsuitable habitat (h) were varied to create an array of complex landscape structures (see Fig. 1 for illustrations). Suitable habitat patches, are thus interspersed among the matrix of unsuitable habitat patches (Fig. 1).

We considered a transmissive pathogen that affects an IGP system in the above fractal landscape, where the two species in the IGP system (the intraguild predator and prey) compete for resources in the suitable habitat (Okuyama, 2008; Su et al., 2008b). We, thus, have five possible states of each cell: unsuitable (U), suitable but empty (E), occupied by a susceptible prey (S_N) or an infected prey (I_N), occupied by a susceptible predator (S_P) or an infected predator (I_P). We use the Moore neighbourhood method which considers the eight nearest neighbours engaging in a chess-kings-move in the two-dimensional circular-bounded space. Synchronous updating of all cell states was used in the CA (Hui and McGeoch, 2007; Su et al., 2009a). The transition rules from step t to $t+1$ are set as follows:

(i) An empty but suitable cell can be colonized by one susceptible prey or one susceptible predator at a probability of r_n and r_p, respectively. The successful colonizer is randomly chosen from the offspring of neighbouring individuals, with a probability of $1-(1-r_n)^{N_{S_N}}$ being a prey and a probability of $1-(1-r_p)^{N_{S_P}}$ being a predator (Rhodes and Anderson, 1997), where N_{S_N} and N_{S_P} are the total number of neighbouring cells with susceptible prey and susceptible predators, respectively.

(ii) Horizontal transmission describes the movement of a pathogen from one individual to the next through direct or indirect contact, which is consistent with the CA model of local interactions. Thus, pathogenic transmission here only occurs horizontally (through contacts between individuals), not vertically (to progenies), so that the offspring of infected mothers are healthy and susceptible at birth (Holt and Pickering 1985; Su et al., 2009a). The pathogen can be transmitted from individual i to j through direct contact at a probability of β_{ij}; for simplicity, we assume $\beta_{ij}=\beta$. Consequently, a susceptible prey can be infected at a probability of

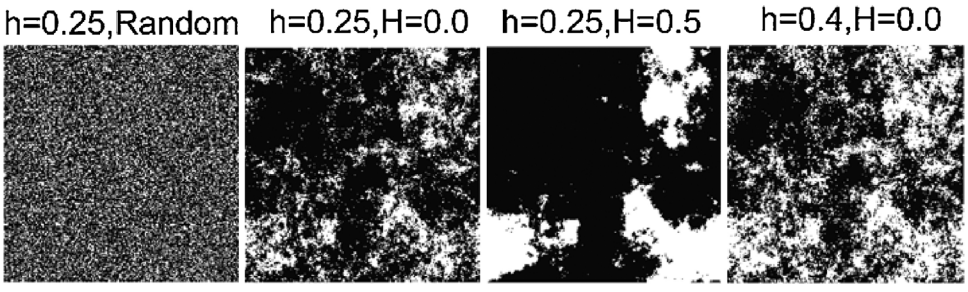

Fig. 1. Three spatial landscape structures in 256 × 256 lattices, each of which has 25% of habitat loss (white) and a gradient of fragmentation (random is the most fragmented; H refers to the degree of spatial autocorrelation of the fractal landscape patterns).