Article

Modeling at the interface of ecology and epidemiology

Min Su, **Hong Wang**
School of Mathematics, Hefei University of Technology, Hefei 230009, China
E-mail: sum04@163.com

Received 25 July 2015; Accepted 3 August 2015; *Published online 1 December 2015*
(cc) BY

Abstract
We briefly present a synthesis of theoretical models in eco-epidemiology which merges the fields of ecology and epidemics. In particular we discuss the role of parasites/pathogens in community assembly formation and species coexistence, as well as the potential of biological control. Recent works have revealed that the complexity in parasite-mediated interactions can alter the dynamic behavior of eco-epidemiological systems, exhibiting oscillations, switching stability regimes. Both community structure and interaction strength also can affect the role of parasites in the host-parasite dynamics. The emerging research area focuses on the spatial structure and distribution pattern in eco-epidemiology. Compared with the well mixed system, spatial structure in eco-epidemiology can lead to different dynamic behavior. We therefore highlight the need to address the impact of parasites/pathogens on real community structures and combine the evolutionary potential to predict the complex dynamics during the biological control in eco-epidemiological systems.

Keywords eco-epidemiology; community structure; spatial structure; predation pressure; species coexistence.

Computational Ecology and Software
ISSN 2220-721X
URL: http://www.iaees.org/publications/journals/ces/online-version.asp
RSS: http://www.iaees.org/publications/journals/ces/rss.xml
E-mail: ces@iaees.org
Editor-in-Chief: WenJun Zhang
Publisher: International Academy of Ecology and Environmental Sciences

1 Introduction

Understanding the community context of infectious diseases is critically important in the field of ecology and epidemiology. Such understanding can increase awareness of how and to what extent parasites affect interaction strength, community structure, species distribution and dynamic complexity (Anderson, 1991; Prenter et al., 2004; Wood, 2006; Hatcher et al., 2006, 2008). Early theoretical framework developed to explore the population dynamics of host-parasite interactions mainly focuses on the host-parasite interaction between a single pair of species (Anderson and May, 1986; Anderson, 1991). However, hosts species are usually not isolated, but embedded in a complex web of ecological interactions. All host and parasite species depend on the ecological networks in which they live. Community interactions can impart complex feedbacks on host-parasite dynamics through direct or indirect effects (Raffel, 2010; Hatcher et al., 2014). Moreover, the diverse community of host offers a wealth of opportunities for an infective pathogen to jump from one species to another, then the parasite species could cross-infect multiple host species (Hatcher et al., 2008, 2014). For

these two reasons, when we analyze the role of infectious disease in host, it is generally convenient to account explicitly for the population dynamics of the potential hosts and of other species that can interact directly and indirectly with them, as unexpected connections and feedbacks among the different species of the community may well occur (Hatcher et al., 2006).

Eco-epidemiology is a new branch in mathematical biology which considers both ecological and epidemiological issues simultaneously and potentially connects these two fields together (Chattopadhyay and Bariagi, 2001; Bairagi et al., 2007; Su et al., 2009a; Greenman and Hoyle, 2010; Su et al., 2015). Anderson and May (1986) were the first who merged two fields and formulated a predator-prey model where prey species was infected by some diseases. From this starting point, most subsequent eco-epidemiological studies have merged different ecological models involving parasites (Hatcher et al., 2006; Bairagi et al., 2007), which predicted various complex dynamic behaviors (e.g. limit cycle and chaos).

The framework of eco-epidemiology has been developed and mainly focuses on two issues. One research issue is how parasites affect community structures, complex dynamics and species coexistence (Chattopadhyay and Arino, 1999; Chattopadhyay and Bariagi, 2001; Bairagi et al., 2007; Hatcher et al., 2006; Lafferty et al., 2008). Most studies have showed that parasite play key roles in structuring communities, often via indirect interactions with non-host species. These effects can be density-mediated (through mortality) or trait-mediated (behavioral, physiological and developmental), and may be crucial to population interactions, including biological invasions (Hatcher et al., 2006, 2014). Current theories have demonstrated that parasites can alter species trait which result in changing or reversing the outcome of competition, inducing complex dynamics in predator-prey systems, affecting the species' distribution structure, and even causing trophic cascades in different modules of food webs (Lafferty et al., 2006; Lafferty et al., 2008; Hatcher et al., 2008, 2014).

However, community structure also in turn affects the successful invasion of infectious disease and host-pathogen dynamics, which is another important issue in the field of eco-epidemiology (Holt and Roy, 2007; Roy and Holt, 2008; Su and Hui, 2011). Because the importance of community structure in host-pathogen systems, there is an increasing realization of the need to examine the influence of interspecific interactions on infectious disease processes (Holt and Dobson, 2006). We know the particularly important class of trophic interactions is predation. Generalist predators can dramatically shape community structure and ecosystem properties. Recent researches have showed that predator also plays a key roles in host-parasite systems because predation itself can strongly alter population dynamics of hosts and parasites. Predators can alter disease incidence by attacking infected prey (Hudson et al., 1992; Packer et al., 2003; Hall et al., 2005) and may even prevent the successful invasion of parasites into host population (Dunn et al., 2012). Most theoretical studies of eco-epidemiology with host-parasite-predation interactions have simplified the predator behavior to be isolated in an ecosystem (Packer et al., 2003; Holt and Roy, 2007). The potentially harmful consequence of predator removal could be increasing disease incidence and enhancing "spillover" infection to novel host species, including humans (Packer et al., 2003; Holt and Roy, 2007; Holt and Dobson, 2006).

Various mathematical methods have been employed in order to depict the complex web of interactions and capture the dynamics in the eco-epidemiological systems. The classical approach to predict the complex dynamics of eco-epidemiological systems was based on ordinary differential equation models (Chattopadhyay and Arino, 1999; Chattopadhyay and Bairagi, 2001; Bairagi et al., 2007; Su et al., 2008). These models are generally following homogenous assumption and consider deterministic dynamics without spatial attributes (Hethcote et al., 2004; Haque and Venturino, 2006; Bairagi et al., 2007). However, when spatially explicit interactions and environment heterogeneity are considered, the complexity of model output will be increased. Thus, spatially structured eco-epidemiological models have been developed to explore a host-parasite and predator-prey dynamics (Okuyama, 2007; Webb et al., 2007a, b; Su et al., 2009a, b; Su and Hui, 2011). The

dynamics of eco-epidemiological system can be captured by spatially explicit stochastic simulation (Hui and McGeoch, 2007; Webb et al., 2007a, b; Su et al., 2008) and moment-closure approximations (e.g. pair approximation) introduced by Matsuda et al. (1992; see also Iwasa, 2000). These spatial methods have been successfully applied in a wide range of ecological, epidemiological, and evolutionary systems (Satō et al., 1994; Keeling and Rand, 1996; Boot and Sasaki, 1999; Ovaskavinen et al., 2002; Hiebeler, 2005; Hui and McGeoch, 2007; Okuyama, 2007). Pair approximation is a spatial method through 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).

In this review, we attempt to review the interface of ecology and epidemiology. Firstly, how parasites indirectly influence the interactions between other species. The interactions considered here normally involve three (or more) species. Another issue is how community structure affects the host-parasite system and how such interplay can be applied in conservation management, such as the biological control using their natural enemies (Holt and Roy, 2007; Greenman and Hoyle, 2010).

2 Eco-epidemiological System with No Spatial Structure

Epidemic transmission is one of the critical density-dependent mechanisms that affect species viability and stability of ecological systems. The presence of complex community structures result in parasite transmission cross different host species, often with unexpected repercussion at the population and community level. Eco-epidemiological theories demonstrate that interplay of parasites with ecological systems may depend crucially on many different factors, including community structure (e.g., competition interactions, predator-prey interactions and intraguild predation), host life history (Allee effects, host immune or host activity) and trophic levels of hosts (Chattopadhyay and Arino, 1999; Chattopadhyay and Bairagi, 2001; Han and Ma, 2001; Chattopadhyay et al., 2002; Hethcote et al., 2004; Haque and Venturino, 2006; Lafferty et al., 2006; Bairagi et al., 2007; Su et al., 2008).

2.1 Effects of parasite on eco-epidemiology dynamic behaviors

Infectious diseases coupled with ecological community to produce a complex combined effect as regulators of host abundance and dynamics. The complexity of modeled eco-epidemiological systems has been increasing. Several theoretical studies have indicated that the emergent stability of ecological communities might be altered once parasites are incorporated in species (Chattopadhyay and Arino, 1999; Chattopadhyay and Bairagi, 2001; Hethcote et al., 2004). A classic controlled study of the effects of parasites on predator-prey system was conducted by Anderson and May (1986), who assumed two extreme cases (parasite of prey only and parasite of predator only) and obtained various different conclusions. As is the case for parasites of prey interacting with predators, previously stable predator-prey dynamics may become oscillatory when a parasite of the predator is introduced (Anderson and May, 1986). However, the situation is more complicated because predator-prey model influenced by other factors simultaneously.

2.1.1Trophic level of host

Most of current studies focused mainly on parasite infection with predation interactions, but parasite in prey only (Hadeler and Freedman, 1989; Venturino, 1995; Chattopadhyay and Arino, 1999; Venturino, 2002; Hethcote et al., 2004). In real systems, parasites may impact predatory interactions in different ways, depending on whether they infect the prey, the predator, or both the predator and prey (Anderson and May, 1986; Venturino, 1994; Venturino, 2002; Su et al., 2008; pada Das et al., 2011). Hadeler and Freedman (1989) have previously studied a predator-prey model with parasite infection where the disease is allowed to cross the species barrier. Moreover, they obtained a threshold condition above which an endemic equilibrium or an endemic periodic solution may appear in the case where there is coexistence of the predator with the

uninfected prey. Kooi et al. (2001) demonstrated that the predator suffering from an infectious disease can cause chaos when the infected predator individuals are ecologically not functioning (not feeding and no offspring). The oscillatory coexistence of the species which is very common in nature is observed for predator disease free system. Venturino (2002) studied the dynamics of two competing species when one of them is subject to a disease, but the disease cannot cross the species barrier. However, the recent outbreaks of severe acute respiratory syndrome (SARS) and animal-to-human transmission of avian influenza (H5N1) demonstrated the possibility of infectious disease caused by a microorganism crossing the species barrier between different species by enlarging its host range (Lewis, 2006), including that of between prey and predator populations.

2.1.2 Role of predator preference

Empirical evidences have showed that parasite alter the trait of hosts activity and made the infected hosts are more vulnerable to predation to susceptible ones (Chattopadhyay and Bairagi, 2001; Su and Hui, 2011). Then, one chief prediction is that increased predation can enhance the number of susceptible host, reduce the number of infected ones and disease prevalence (i.e. the proportion of the host population that is infected). Chattopadhyay and Bairagi (2001) proposed an eco-epidemiological model (SI type) of Salton Sea with the assumption that predator consumes infected prey only. They observed that the eco-epidemiological system is stable or non-stable depends on the predator preference.

2.1.3 Effect of predator's functional response

Fenton and Rands (2006) studied two eco-epidemiological models and showed that prey manipulation by parasites can greatly alter the quantitative dynamics of the community, potentially resulting in high amplitude oscillations in abundance. However, the precise outcome of the interaction depends crucially on both the form of manipulation and the nature of the predator's functional response. Bairagi et al. (2007) also proposed an infected predator-prey system with different predators' functional response. They revealed that the interplay between two key parameters (the infection rate and attack rate of predators) yields a diverse array of biologically relevant behavior, including switching of stability, extinction and oscillations.

2.1.4 Role of Allee effect

Parasite in the ecological system may alter the impacts of some life-history strategy on the stability of predation systems. Su et al. (2008) have explored an eco-epidemiological system with disease on predator and demonstrated that whether the Allee effect is a stabilizing or destabilizing force in the system could be determined by the intensity of Allee effect. However, previous studies of local predator-prey system with no parasite showed that Allee effect is considered as a destabilizing factor in the population dynamics of competition and predation (Zhou et al., 2005). And Allee effects can amazingly regulate dynamics and improve the persistence of metapopulation. Dynamic complexity in metapopulation with no parasite will decrease with the increasing of Allee effect intensity (Hui and Li, 2003; Hui and Li, 2004).

2.1.5 Parasite in species coexistence

The potential effect of parasites on community structure, in particular their role in promoting species coexistence, has been discussed in recent literature (Hatcher et al., 2008; Soufbaf et al., 2012). Hatcher et al. (2008) demonstrated that parasitism can increase the range of conditions leading to coexistence when the parasite exerts a greater deleterious effect on the "stronger" species in terms of the combined effects of competition and predation. Such a parasite can enable an inferior competitor that is also the less predatory to persist, and may actually lead to numerical dominance of the species. Hethcote et al. (2004) proposed a predator-prey model including a susceptible-infective-susceptible (SIS) infection with standard incidence in prey population and also assumed that the infected prey is more vulnerable to predation. They discovered several interesting cases where the disease infection in prey could promote coexistence. Theoretical studies

have further shown that pathogen-induced modification of competitive and foraging abilities could affect the coexistence of multiple predator species in the intraguild predation system (Hatcher et al., 2008, 2014).

2.2 Effect of community structure on eco-epidemiology

Parasitism can play a keystone in species interactions between competitors, between predators and prey, and also intraguild predation (Hatcher et al., 2006). Roles of parasite in eco-epidemiology can be further affected by community structure and the strength of species interactions (Hudson et al., 1992; Packer et al., 2003; Hatcher et al., 2008; Roy and Holt, 2008; Su and Hui, 2011). For instance, intraguild predation in species interactions has been viewed as an important force to affect the role of pathogens in host populations (Hatcher et al., 2008, 2014). Hatcher et al. (2008) showed that the equilibrium population sizes in relation to parasite virulence can be altered by the existence of intraguild predation.

Strength of species interactions also affects the role of parasite in regulating the host populations. Hudson et al. (1992) parameterized a mathematical model of the grouse-nematode system to examine the effects of predation. Predators were represented as a parameter (rather than having explicit equations to represent population numbers), so this model is more appropriate for long-lived or generalist predators whose population dynamics are unlikely to be coupled to that of a single host species (Packer et al., 2003; Roy and Holt, 2008). If predators were non-selective or preferred infected individuals, increased predation will reduce the mean parasite burden in grouse populations, and then result in a reduction in the amplitude of oscillations in grouse numbers. They argued that because selective predation removes a disproportionate fraction of parasites, and then the regulatory role of parasites is reduced (in this case, reducing parasite-driven oscillations caused by delayed density-dependent effects on host fecundity).

Evidently, because predators prefer infected prey as easy targets, they can potentially alter the disease prevalence in prey population (Hudson et al., 1992; Packer et al., 2003; Hall et al., 2005; Ostfeld 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; Su and Hui, 2011). Enhanced predation pressure (either by manipulating predator density or enhancing predation efficiency) could also facilitate the transmission of pathogen under certain circumstances (Greenman and Hoyle, 2010). Studies the importance of community trait in eco-epidemiology have provided increasing insights to the complex dynamics in the system and also 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).

3 Spatial Eco-epidemiological Systems

Not only species interactions but also spatial structure of populations like random or local contact interaction as well as the variability of the environment may affect the spatiotemporal dynamics of populations and disease transmission (Boots and Sasaki, 1999; Bonsall and Hassell, 2000; Bauch, 2005; Ostfeld et al., 2005; Su et al., 2009a, b; Webb et al., 2007a, b). Spatial structure can cause variety of spatiotemporal population structures and induce a more comprehensive framework for the dynamics of human and agricultural host-parasite interactions (Anderson, 1991; Hudson et al., 1998; Keeling et al., 2001; Webb et al., 2007a, b). Lloyd and May (1996) showed that spatial structure play an important role in the persistence and dynamics of epidemics of children diseases. Compared to the non-spatial model, spatial structure induced chaotic solutions

are observed for weaker seasonal forcing and these solutions have a more realistic minimum number of infected. Several ecological processes (contact neighbor structure, pathogen dispersal or spatial heterogeneity) can also result in strong spatial patterns of such risk or incidence (Malchow et al., 2005; Su et al., 2008; Su et al., 2009a, b). It has already become clear that differently structured networks lead to different types of epidemiology (Keeling, 1999). For example, a sparsely connected host population is more difficult to invade than a densely connected host population.

3.1 Approaches of spatial eco-epidemiological model

Although ODEs can capture the quantitative information about the process and depict biological phenomenon closely, they do not account for spatial attributed of the systems. Spatial models have been introduced into epidemiology to resolve vividly the spatial transmission dynamics of the epidemic. In studying the spatial aspects of epidemic dynamics, there are various levels of realism in the description of space, which have three broad categories (Tilman and Kareiva, 1997). One is based on the analytical reaction-diffusion models, which can be either discrete or continuous in time and space (Cruickshank et al., 1999). The approach has been well developed in the system of biological invasions (Hall, 2011). A second framework is based on the cellular automaton or lattice model, and contact processes by assuming that organisms divide a habitat into a series of equal-sized, identical patches that occur in a grid (Rhodes and Anderson, 1997; Hui et al., 2006; Zhang et al., 2006; Ramanantoanina et al., 2011). Each patch in one of a finite number of states, time is often discrete and patch states are updated according to probabilistic rules at each time step, which depending on the current state of the patch and its surrounding neighborhoods. Spatially explicit modelling (e.g., distinguishing local and global interactions) has generated a number of new hypotheses (Webb et al., 2007a, b). For example, parasite-driven behaviour heavily relies on the spatial structure of species (Webb et al., 2007b). Couple Map Lattice is also typically used in eco-epidemiological models (Grenfell et al., 1995; Keeling et al., 1997; Park et al., 2001; Li et al., 2005). Here the populations is divided into a number of subpopulations or patches, where each patch contains ecological populations and the eco-epidemiological dynamics are usually described by a set of homogeneous mixing population equations. These patches are linked to allow for transmission from one patch to another with the connections defining the spatial structure of the host populations (Park et al., 2001).

More recently, moment-closure approximations (e.g. pair approximation) introduced by Matsuda et al. (1992; see also Iwasa, 2000), which has been successfully applied in eco-epidemiological systems (Satō et al., 1994; Keeling and Rand, 1996; Hiebeler, 2005; Su et al., 2008). Pair approximation is a mathematical technique to analyze spatially extended systems (Matsuda et al., 1992; Satō et al., 1994; Van Baalen, 2000; Iwasa, 2000). For example, a correlation dynamics model accurately predicted temporal patterns observed in childhood diseases in terms of contact structures (Keeling et al., 1997; Keeling, 1999). 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. 2008).

3.2 Spatial Structure in eco-epidemiological systems

In spatially structured populations, it is easier for coexistence, diversity and altruism to develop, and epidemics have more realistic time series and critical community sizes. In a mean-field (non-spatial) approximation, the population is assumed to be well-mixed, such that each individual is equally likely to contact any other individuals in the population. Hence, the success or failure of an invasion depends only upon non-spatial parameters such as transmission rates or recovery rates. By comparison, in a spatially structured population, spatial structure also plays a role in determining the success or failure of the invasion. But to what extent will the contact structure of host population affect the parasite dynamics, in particular of their distribution pattern? Can we control the disease transmission by modifying these contact structures? Webb et al. (2007a, b) and Su et al. (2009a) carried out computer simulations of infectious disease in spatially structured host populations

and concluded that neighbor contact effects have less persistence of disease, and a greater possibility of parasite driven extinction and limit cycles with respect to well-mixed systems. Previous studies have demonstrated that the choice of uniform interaction within a finite neighborhood is particularly prone to exhibiting spatial patterns that are not robust with respect to the shape of the interaction neighborhood (Su et al., 2008). However, parasites require close contact between the infected host and new susceptible hosts for successful transmission and then epidemic forms a spreading wave under each neighboring structure (Shaw et al., 1998; Hoogendoorn and Heimpel, 2002).

In fact, the spatially observable resultants of the eco-epidemiological system depend on environmental heterogeneity, demographic stochasticity and behavioural characteristics as well as the coupling effects of these factors. Su et al. (2008) have demonstrated that the demographic stochasticity dramatically influences the spatial distribution of eco-epidemiological system (Fig. 1). The spatial distribution changes from oil-bubble-like (due to local interaction) to aggregated spatially scattered points (due to local interaction and demographic stochasticity). Malchow et al. (2005) explored an eco-epidemiological model with infectious disease in phytoplankton-zooplankton system. They revealed the interesting spiral waves behaviors using stochastic reaction-diffusion equations approaches. Evidences of real systems also revealed the importance of spatial structure (Dray et al., 2006; Roux et al., 2011). Roux et al. (2011) explored a multi-scale spatial modelling of the occurrence of Chagas disease insect vectors and revealed the spatial patterns of the eco-epidemiological system. The local scale of “near-to-near” dispersal and surrounding heterogeneous environment produce a higher insect density at the village periphery.

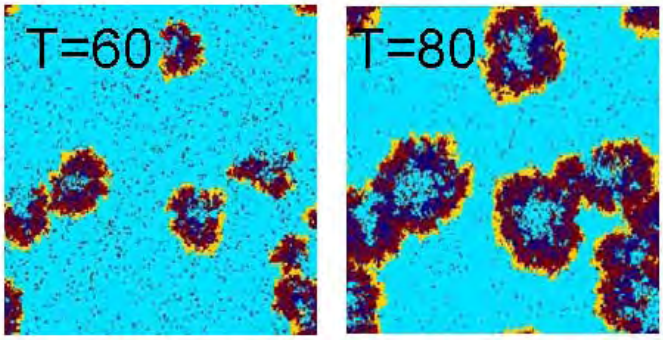

Fig. 1 Spatial distribution pattern of the eco-epidemiological dynamics with the demographic stochasticity.

Disease invasion and transimission dynamics are also influenced by the spatial landscape heterogeneity. Developing a general understanding of the relationship between disease persistence and landscape heterogeneity is therefore a key goal in spatial eco-epidemiology (Hiebeler, 2005; Su et al., 2008; Su et al., 2009a, b). Landscape heterogeneity will often restrict species movement and dispersal, likely increasing contact rates among individuals and ultimately the spread of disease (Zhao et al., 2014). The effects of landscape composition (types of elements) and configuration (spatial positions of those elements) on the disease dynamics, which suggest that a true integration of landscape ecology with eco-epidemiology will be fruitful and have been developing. Ostfeld et al. (2005) have incorporated explicit landscape elements into spatial epidemiology and suggested that greater incorporation of ecological landscape approaches would improve our understanding and prediction of disease risk. Su et al. (2009a, b) have explored the effect of landscape heterogeneity (designed by amount of habitat loss and clump degree of lost patches) on parasite invasion through both pair approximation and stochastic spatially explicit simulation. Results showed that more fragmented landscape was shown to be detrimental to the parasitic disease invasion and transmission.

Although the complex dynamics indued by interactions of parasite and predators have been widely in the eco-epidemilogy, little has been done to examine the prevalence and aggregation of pathogens in spatially structured eco-epidemiological systems (Su and Hui, 2011). Simulation studies of Su and Hui (2011) using pair approximation and spatially stochastic simulations demonstrated that highly connected site network facilitated the parasites infection, especially under high predation pressure. And pathogen aggregation was not always negatively correlated with predation pressure as proposed from empirical studies, but depending on the pressure level. It is thus possible to better design biological control strategies for target species by manipulating predation pressure and the range of pathogen transmission.

4 Biological Invasion in Eco-epidemiological System

Biological invasions are global threats to biodiversity and parasites might play a key role in determining invasion outcomes. They can facilitate or limit invasions, and positively or negatively impact the native species (Dunn et al., 2012). Invasions can interact with eco-epidemiological systems through both the introduction of exotic species with parasites and the effects of invading hosts on native hosts dynamics with parasites (Prenter et al., 2004; Telfer and Bown, 2012). Many empirical examples have showed that parasites and pathogens can alter the outcome of biological invasion by mediating a range of competitive and predatory interactions among native and invading species (e.g. MacNeil et al. 2003; Tompkins et al. 2003; Prenter et al. 2004; Dunn and Perkins, 2012). Transmission of parasites from invading to native species can occur, aiding the invasion process, whilst the 'release' of invaders from parasites can also facilitate invasions. Parasites may determine the speed of invasion, as seen in the case of red squirrel replacement by greys (Tompkins et al., 2003, Hall, 2011). Tompkins et al. (2003) have demonstrated that parapoxvirus is likely to have played a crucial role in the native red squirrel decline through comparing the different rates of the ecological replacement with both invading-native species competition and parasite, and only competition.

In the eco-epidemiological systems, natives and invaders may also share predators and parasite infections affect the success of biological invasions through their impact on the predation interactions (Fagan et al., 2002; Prenter et al., 2004; Dunn et al., 2012). Invasive and native hosts differ in susceptibility to parasitism and/or in their responses to particular parasites, which this parasite-mediated predation might render them differentially vulnerable to shared predators (MacNeil et al., 2003). Recent studies suggest that parasites can change the vulnerability of native and invading amphipods to fish predation, thereby influencing the invasion process (MacNeil et al., 2003). Native/invader interactions for many species are governed by intraguild predation which influenced by parasite infection. Hatcher et al. (2014) have showed that parasites can alter intraguild predation (IGP) between native and invasive crustaceans, and then reverse the invasion outcomes.

5 Discussion

In this article, we have briefly reviewed the development of theoretical works on eco-epidemiology. One of the biggest challenges to further develop eco-epidemiology is to examine the interplay between disease infection and species interactions in ecological communities (Hatcher et al., 2006; Lafferty, et al., 2008). Some behaviors of an eco-epidemiological system under different biological conditions have become known, but, to our knowledge there is no general theoretical treatment. More work is also need further explored in real complex community, spatial structure of populations and evolutionary dynamics. Current studies of eco-epidemiological theories are mainly based on predator-prey community and competition structures, but few theoretical treatments of more complex community structures, e.g., ecological networks of host-parasite and prey-predator. Some evidences have shown that parasite can modify the species trait and strength of species interaction through both direct and indirect effects, which affect the stability of entire ecological network

structure through trophic cascade (Hatcher et al., 2006; Ings et al., 2009). Moreover, the combination of theoretical and empirical approaches is further required to examine how parasite interactions within and between modules scale up to community-level processes.

The evolution of virulent pathogens is becoming a cause of great concern in the protection of threatened wildlife communities and ecosystems (O'Keefe, 2005). But few theoretical studies reveal the evolutionary aspects in an eco-epidemiological system. As analyzing the virulence evolution in wildlife, it is generally convenient to account explicitly the population dynamics of the potential hosts and other species that can interact directly and indirectly with them, as unexpected connections and feedbacks among the different species of the community may well occur (Price et al., 1988). So the pertaining question is how does virulence evolution or the coevolution of pathogen-hosts affected by prey-predator interactions and transmission process? Can evolution of eco-epidemic changed by the spatial structure and landscape heterogeneity? Hence, if we are to determine the effects of parasites within the ecological communities, the evolution of eco-epidemiology need be further investigated.

In addition, diseae controls that are varied in the parameters of the eco-epidemiological system can also be an important part of management's weaponry (Smith et al., 2009). Further exploration on the dynamic control of disease in the eco-epidemiological system is still needed because the complicated realities in these systems (e.g. considering age structured and environmental heterogeneity). Considering the complexities in future studies can be important to derive more optimal strategies for biological control and provide better conservation plans (Chattopadhyay et al., 2002; Delgado et al., 2006; Williams, 2008; Zhao et al., 2015).

Acknowledgements

This work was supported by the National Natural Science Foundation of China (31000192), China Postdoctoral Science Foundation (20100481159) and the Fundamental Research Funds for the Central Universities (2014HGXJ0065).

References

Anderson RM, May RM. 1986. The invasion, persistence, and spread of infectious diseases within animal and plant communities. Philosophical Transactions of the Royal Society of London Series B-Biological Sciences, 314: 533-570

Anderson RM. 1991. Populations and infectious-diseases: ecology or epidemiology- the 8th Tansley lecture. Journal of Animal Ecology, 60: 1-50

Bairagi N, Roy PK, Chattopadhyay J. 2007. Role of infection on the stability of a predator-prey system with several response functions – A comparative study. Journal of Theoretical Biology, 248: 10-25

Bauer ON, Pugachev ON, Voronin VN. 2002. Study of parasites and diseases of sturgeons in Russia: a review. Journal of Applied Ichthyology, 18: 420-429

Bauch CT. 2005. The spread of infectious diseases in spatially structured populations: An invasory pair approximation. Mathematical Biosciences, 198: 217-237

Bonsall MB, Hassell MP. 2000. The effects of metapopulation structure on indirect interactions in host-parasitoid assemblages. Proceedings of the Royal Society of London Series B-Biological Sciences, 267: 2207-2212

Boots M, Sasaki A. 1999. 'Small worlds' and the evolution of virulence: infection occurs locally and at a distance. Proceedings of the Royal Society of London Series B-Biological Sciences, 266: 1933-1938

Chattopadhyay J, Arino O. 1999. A predator-prey model with disease in the prey. Nonlinear Analysis, 36: 747-766

Chattopadhyay J, Bariagi N. 2001. Pelicans at risk in Salton Sea–an eco-epidemiological study. Ecological Modelling, 136: 103-112

Chattopadhyay J, Sarkar RR, Ghosal G. 2002. Removal of infected prey prevent limit cycle oscillations in an infected prey-predator system-a mathematical study. Ecological Modelling, 156: 113-121

Cruickshank I, Gurney WSC, Veitch AR. 1999. The characteristics of epidemics and invasions with thresholds. Theoretical Population Biology, 56: 279-292

Delgado M, Molina-Becerra M, Suárez A. 2006. Analysis of an age-structured predator–prey model with disease in the prey. Nonlinear Analysis, 7: 853-871

Dray S, Legendre P, Peres-Neto PR. 2006. Spatial modelling: a comprehensive framework for principal coordinate analysis of neighbour matrices (PCNM). Ecological Modelling, 196: 483-493

Dunn AM, Perkins SE. 2012. Invasions and infections. Functional Ecology, 26: 1234-1237

Dunn AM, Torchin ME, Hatcher MJ, Kotanen PM, Blumenthal DM, et al. 2012. Indirect effects of parasites in invasions. Functional Ecology, 26: 1262-1274

Fagan W F, Lewis MA, Neubert MG, van den Driessche P. 2002. Invasion theory and biological control. Ecology Letters, 5: 148-157

Fenton A, Rands SA. 2006. The impact of parasite manipulation and predator foraging behavior on predator-prey communities. Ecology, 87: 2832-2841

Greenman J, Hoyle A. 2010. Pathogen Exclusion from Eco-Epidemiological Systems. American Naturalist, 176: 149-158

Grenfell B T, Bolker BM, Kleezkowski A. 1995. Seasonality and extinction in chaotic metapopulations. Proceedings of the Royal Society of London Series B-Biological Sciences, 259: 97-103

Hadeler KP, Freedman HI. 1989. Predator-prey populations with parasitic infection. Journal of Mathematical Biology, 27: 609-631

Hall SR, Duffy MA, Cáceres CE. 2005. Selective predation and productivity jointly drive complex behavior in host-parasite systems. American Naturalist, 165: 70-81

Hall R J. 2011. Eating the competition speeds up invasions. Biology Letters, 7: 307-311

Han L, Ma Z. 2001. Four-predator prey models with infectious diseases. Mathematical and Computer Modelling 34: 849-858

Haque M, Venturino E. 2006. The role of transmissible diseases in Holling-Tanner predator-prey model. Theoretical Population Biology, 70: 273-288

Hatcher MJ, Dick JTA, Dunn AM. 2006. How parasites affect interactions between competitors and predators. Ecology Letters, 9: 1253-1271

Hatcher MJ, Dick JTA, Dunn AM. 2008. A keystone effect for parasites in intraguild predation? Biology Letters, 4: 534-537

Hatcher MJ, Dick JTA, Dunn AM. 2014. Parasites that change predator or prey behaviour can have keystone effects on community composition. Biology Letters, 10: 1-5

Hethcote HW, Wang W, Han L, Ma Z. 2004. A predator–prey model with infected prey. Theoretical Population Biology, 66: 259-268

Hiebeler DE. 2005. A cellular automaton SIS epidemiological model with spatially clustered recoveries. Lecture Notes in Computer Science 3515: 360-367

Holt RD, Dobson AP. 2006. Extending the principles of community ecology to address the epidemiology of host-pathogen systems. In: Disease Ecology: Community Structure and Pathogen Dynamics (Collinge SK,

Ray C, eds). 6-27, Oxford University Press, Oxford, UK

Holt RD, Roy M. 2007. Predation can increase the prevalence of infectious disease. American Naturalist, 169: 690-699

Hoogendoorn M, Heimpel E. 2002. Indirect interactions between an introduced and a native ladybird beetle species mediated by a shared parasitoid. Biological Control, 25: 224-230

Hudson PJ, Dobson AP, Newborn D. 1992. Do parasites make prey vulnerable to predation? Red grouse and parasites. Journal of Animal Ecology, 61: 681-692

Hudson P J, Dobson AP, Newborn D. 1998. Prevention of Population Cycles by Parasite Removal. Science, 282: 2256-2258

Hui C, Li ZZ. 2003. Dynamical complexity and metapopulation persistence. Ecological Modelling, 164: 201-209

Hui C, Li ZZ. 2004. Distribution patterns of metapopulation determined by Allee effects. Population Ecology, 46: 55-63

Hui C, MeGeoch MA, Warren M. 2006. A spatially explicit approach to estimating species occupancy and spatial correlation. Journal of Animal Ecology, 75: 140-147

Hui C, MeGeoch MA. 2007. Spatial patterns of Prisoner's Dilemma Game in Metapopulations. Bulletin of Mathematical Biology, 69: 659-676

Ings TC, Montoya JM, Bascompte J, Blüthgen N, Brown L, et al. 2009. Ecological networks – beyond food webs. Journal of Animal Ecology, 78: 253-269

Iwasa Y. 2000. Lattice models and pair approximation in ecology. In: The Geometry of Ecological Interactions: Simplifying Spatial Complexity (Dieckmann U, Law R, Metz J, eds). Cambridge University Press, Cambridge

Keeling MJ, Rand DA. 1996. Spatial correlations and local fluctuations in host-parasite ecologies. In: From Finite to Infinite Dimensional Systems (Glendinning P, ed). Kluwer, Amsterdam, Netherlands

Keeling MJ, Rand DA, Morris AJ. 1997. Correlation models for childhood epidemics. Proceedings of the Royal Society of London Series B-Biological Sciences, 264: 1149-1156

Keeling MJ. 1999. Correlation equations for endemic diseases: externally imposed and internally generated heterogeneity. Proceedings of the Royal Society of London Series B-Biological Sciences, 266: 953-961

Keeling MJ, Woolhouse ME, Shaw DJ, Matthews L, Chase-Topping M, Haydon DT, Cornell SJ, Kappey J, Wilesmith J, Grenfell BT. 2001. Dynamics of the 2001 UK foot and mouth epidemic: stochastic dispersal in a heterogeneous landscape. Science, 294: 813-817

Kooi BW, van Voorn GAK, pada Das K. 2011. Stabilization and complex dynamics in a predator-prey model with predator suffering from an infectious disease. Ecological Complexity, 8:113-122

Lafferty KD, Dobson AP, Kuris AM. 2006. Parasites dominate food web links. Proceedings of the National Academy of Sciences of the United States of America, 103: 11211-11216

Lafferty KD, Allesina S, Arim M, Briggs CJ, et al. 2008. Parasites in food webs: the ultimate missing links. Ecology Letters, 11: 533-546

Lewis D B. 2006. Avian flu to human influenza. Annual Review of Medicine, 57: 139-154

Li ZZ, Gao M, Hui C, Han XZ, Shi H. 2005. Impact of predator pursuit and prey evasion on synchrony and spatial patterns in metapopulation. Ecological Modelling, 185: 245-254

Lloyd AL, May RM. 1996. Spatial heterogeneity in epidemic models. Journal of Theoretical Biology, 179: 1-11

Matsuda H, Ogita A, Sasaki A, Satō K. 1992. Statistical mechanics of population: the lattice Lotka-Volterra model. Progress of Theoretical Physics Supplement, 88: 1035-1049

MacNeil C, Dick JTA, Hatcher MJ, Terry RS, Smith JE, Dunn AM. 2003. Parasite-mediated predation between native and invasive amphipods. Proceedings of the Royal Society of London Series B-Biological Sciences, 270: 1309-1314

Malchow H, Hiker FM, Sarkar RR, Brauer K. 2005. Spatiotemporal Patterns in an Excitable Plankton System with Lysogenic Viral Infection. Mathematical and Computer Modelling, 42: 1035-1048

O'Keefe KJ. 2005. The evolution of virulence in pathogens with frequency-dependent transmission. Journal of Theoretical Biology, 233: 55-64

Okuyama T. 2008. Intraguild predation with spatially structured interactions. Basic and Applied Ecology, 9: 135–144

Ostfeld RS, Glass GE, Keesing F. 2005. Spatial epidemiology: an emerging (or re-emerging) discipline. Trends in Ecology and Evolution, 20: 328-336

Ovaskainen O, Satō K, Bascompte J, Hanski I. 2002. Metapopulation models for extinction threshold in spatially correlated landscapes. Journal of Theoretical Biology, 215: 95-108

Packer C, Holt RD, Hudson PJ, Lafferty KD, Dobson AP. 2003. Keeping the herds healthy and alert: implications of predator control for infectious disease. Ecology Letters, 6: 797-802

Pada Das K, Kundu K, Chattopadhyay J. 2011. A predator–prey mathematical model with both the populations affected by diseases. Ecological Complexity, 8: 68-80

Park AW, Gubbins S, Gilligan CA. 2001. Invasion and persistence of plant parasites in a spatially structured host population. Oikos, 94: 162-174

Prenter J, MacNeil1 C, Dick JTA, Dunn AM. 2004. Roles of parasites in animal invasions. Trends in Ecology and Evolution, 19: 385-390

Price PW, Westoby M, Rice B. 1988. Parasite-mediated competition: Some predictions and tests. American Naturalist, 131: 544-555

Raffel TR. 2010. Parasitism in a community context: trait-mediated interactions with competition and predation. Ecology, 91: 1900-1907

Ramanantoanina A, Hui C, Ouhinou A. 2011. Effects of density-dependent dispersal behaviours on the speed and spatial patterns of range expansion in predator-prey metapopulations. Ecological Modelling, 222: 3524-3530

Rhodes C J, Anderson RM. 1997. Epidemic thresholds and Vaccination in a lattice model of disease spread. Theoretical Population Biology, 52: 101-118

Roy M, Holt RD. 2008. Effects of predation on host–pathogen dynamics in SIR models. Theoretical Population Biology, 73: 319-331

Roux E, de Fátima Venâncio A, Girres JF, Romaña CA. 2011. Spatial patterns and eco-epidemiological systems – part I: multi-scale spatial modelling of the occurrence of Chagas disease insect vectors. Geospatial Health, 6: 41-51

Satō K, Matsuda H, Sasaki A. 1994. Pathogen invasion and host extinction in lattice structured populations. Journal of Mathematical Biology, 32: 251-268

Soufbaf M, Fathipour Y, Hui C, Karimzadeh J. 2012. Effects of plant availability and habitat size on the coexistence of two competing parasitoids in a tri-trophic food web of canola, diamondback moth and parasitic wasps. Ecological Modelling, 244: 49-56

Shaw DJ, Grenfell BT, Dobson AP. 1998. Patterns of macroparasite aggregation in wildlife host populations. Parasitology, 117: 597-610

Smith KF, Acevedo-Whitehouse K, Pedersen AB. 2009. The role of infectious diseases in biological conservation. Animal Conservation, 12: 1-12

Su M, Hui C, Zhang Y, Li Z. 2008. Spatiotemporal dynamics of the epidemic transmission in a predator-prey system. Bulletin of Mathematical Biology, 70: 2195-2210

Su M, Hui C, Zhang Y Y, Li ZZ. 2009a. How does the spatial structure of habitat loss affect the eco-epidemic dynamics? Ecological Modelling, 220: 51-59

Su M., Li W L, Li ZZ, Zhang F P, Hui C. 2009b. The effect of landscape heterogeneity on host-parasite dynamics. Ecological Research, 24: 889-896

Su M, Hui C. 2011. The effect of predation on the prevalence and aggregation of pathogens in prey. BioSystems, 105: 300-306

Su M, Hui C, Lin Z. 2015. Effects of the transmissibility and virulence of pathogens on intraguild predation in fragmented landscapes. BioSystems, 129: 44-49

Telfer S, Bown K. 2012.The effects of invasion on parasite dynamics and communities. Functional Ecology, 26: 1288–1299

Tompkins DM, White AR, Boots M. 2003. Ecological replacement of native red squirrels by invasive greys driven by disease. Ecology Letters, 6: 189-196

Tilman D, Kareiva P. 1997. Spatial Ecology: The Role of Space in Population Dynamics and Interspecific Interactions. Princeton University Press, Princeton, USA

Webb SD, Keeling MJ, Boots M. 2007a. Host-parasite interactions between the local and the mean-field: How and when does spatial population structure matter? Journal of Theoretical Biology, 249: 140-152

Webb SD, Keeling MJ, Boots M. 2007b. Spatially extended host-parasite interactions: The role of recovery and immunity. Theoretical Population Biology, 71: 251-266

Williams PD. 2008. Unhealthy herds: Some epidemiological consequences of host heterogeneity in predator-host-parasite systems. Journal of Theoretical Biology, 253: 500-507

Wood MJ. 2006. Parasites entangled in food webs. Trends in Parasitology, 23: 8-10

Van Baalen, M. 2000. Pair Approximation for Different Spatial Geometries. In: The Geometry of Ecological Interactions: Simplifying Spatial Complexity (Dieckmann U, Law R, Metz J, eds). Cambridge University Press, Cambridge, USA

Venturino E. 1994. The influence of disease on Lotka-Volterra systems. Rocky Mountain Journal of Mathematics, 24: 381-402

Venturino E. 1995. Epidemics in predator-prey models: disease in the prey. In: Mathematical Population Dynamics: Analysis of Heterogeneity (Arino O, Axelrod D, Kimmel M, Langlais M, eds), 1: 381-393

Venturino E. 2002. Epidemics in predator-prey models: disease in the predators. IMA Journal of Mathematics Applied in Medicine and Biology, 19: 185-205

Zhang F, Li ZZ, Hui C. 2006. Spatiotemporal dynamics and distribution patterns of cyclic competition in metapopulation. Ecological Modelling, 193: 721-735

Zhao ZH, Hui C, Hardev S, Ouyang F, Dong Z, Ge F. 2014. Responses of cereal aphids and their parasitic wasps to landscape complexity. Journal of Economic Entomology, 107: 630-637

Zhao ZH, Hui C, He DH, Li BL. 2015. Effects of agricultural intensification on ability of natural enemies to control aphids. Scientific Reports, 5: 8024

Zhou SR., Liu YF, Wang G. 2005. The stability of predator-prey systems subject to the Allee effects. Theoretical Population Biology, 67: 23-31