An Eco-epidemiological System with Infected Predator

Min Su*
School of Mathematics
Hefei University of Technology
Hefei 230009, China
Email: sum04@163.com

Cang Hui
Department of Botany and Zoology
University of Stellenbosch
Private Bag X1, Matieland 7602, South Africa

***Abstract*—Epidemic is considered as one of the primary causes of species extinction, especially it also can induce populations suffering from Allee effect. Using ordinary differential equation and lattice model, we here explored the epidemic transmission in a predator-prey system. Numerical simulation indicated that the Allee effect can both destabilize and stabilize the system: a moderate Allee effect will destabilize the dynamics, but it is not true for the extreme Allee effect (weak or strong). Moreover, we have pointed out that the infection rate was key to determine the temporal transmission of disease. Through the join-count statistics method, results showed that the spatial structure of disease is influenced dramatically by the Allee effect and ecological neighborhood. These results are new to eco-epidemiology and imply a possibility of restricting disease diffusion range to control the spread of epidemics in the ecological system.**

Keywords-eco-epidemiology; lattice model; Allee effect; local stability; Join-count statistics

I. INTRODUCTION

Eco-epidemiology is a new branch in mathematical biology which considers both the ecological and epidemiological issues simultaneously [1]. Since [2] modeled firstly a disease spreading among interacting populations, scientists are paying increasing interests to this new field due to its theoretical and empirical importance [1-7]. As a result, the study of diseases in a predator-prey system has also become a very popular topic in eco-epidemiology and made a significant progress in understanding different scenarios for disease transmission [1-6]. Among these studies, most considered the transmission of disease in prey populations. However, epidemic diseases can attack predators through various means, such as food, mating and parasites, then infectious diseases in predator species has need to be explored.

Among the most widely used ecological process in theoretical ecology, the Allee effect plays a special role in view of the interesting dynamics it possesses [5, 7-9]. Allee effect is considered as a destabilizing factor in the dynamics of predator-prey systems [9] but can play two roles (stabilizing and destabilizing force) in the dynamics of an eco-epidemiological system with standard incidence [5]. However, in eco-epidemiological models, horizontal incidence (the infection rate of susceptible individuals through contact with infected ones) is usually of the type βSI (mass action incidence) or $\beta SI/(S+I)$ (standard incidence), and which may result different dynamics [10]. Thus, it is necessary to understand the role of this epidemic induced Allee effect in an eco-epidemiological context with mass action incidence.

In general, the classical approach to study the dynamic of eco-epidemiological systems is based on the ordinary differential equations, which indicate that the population is homogeneously mixed [1, 2]. However, there is likely to be some degree of local interaction whether spatially or socially in all host species [11]. As such, there is now a well developed theory that examines the role of spatial structure in a number of classical ecological scenarios [11, 12]. The most popular method to elucidate the role of the spatial structure of individual hosts within populations is by using lattice/cellular automaton models, where local interactions are particularly strong [5, 11].

Here, we will set up two eco-epidemiological models: the ordinary differential equation model and lattice model to study the above issues. First, through mathematical analysis of the non-spatial model, we will present the dynamic behavior corresponding to different intensity of the Allee effect and infection rate. Second, using the lattice model, we will firstly discuss the local transmission factor (neighborhood structure) and Allee effect that influences the spatial spread of an epidemic. Furthermore, to express the spatial structure of the disease, we will adopt the spatial autocorrelation index, join-count statistics (JCS) [12, 13].

II. THE MODEL

A. Ordinary differential equation model

According to the susceptible-infected model [2] and the classical predator-prey system [14], we can make the following assumptions for incorporating a transmissible disease in the predator species: (A1) In absence of predator, the prey population grows logistically; (A2) The predators are divided into two categories, i.e., those susceptible to the disease, S, and those infected by the disease, I; (A3) Disease is transmitted through the contact between the susceptible and infected individuals. The transmission rate follows the law of

mass action. Once infected population have recovered (at rate δ), they rejoin the susceptible class; (A4) Infected individuals are not active enough to forage; (A5) Susceptible predators are inefficient in hunting when their abundance is low, i.e., an Allee effect.

An ordinary differential equation (ODE) model that obeys the above assumptions could be rewritten as:

$$\frac{dN}{dt} = rN\left(1-\frac{N}{K}\right) - \varepsilon NS$$

$$\frac{dS}{dt} = \eta NS\frac{S}{S+a} - d_1 S - \beta SI + \delta I \qquad (1)$$

$$\frac{dI}{dt} = \beta SI - d_2 I - \delta I$$

where ε, η ($\varepsilon \geq \eta > 0$) are, respectively, the maximum and effective predation rate; d_1, d_2 ($d_2 \geq d_1 > 0$) are, respectively, the death rate of susceptible and infected predator; β is the infection rate which represents the force of infection; δ is the removal rate from infected class to susceptible class. The term $S/(S+a)$ is for the Allee effect, and a can be defined as the Allee effect constant [9]. Higher value of a implies a lower per capita growth rate of the predator, especially when S is small. When the predators do not suffer from the Allee effect, the positive equilibrium of (1) can be solved easily: $N^* = K(1-S^*\varepsilon/r)$, $S^* = (d_2+\delta)/\beta$, $I^* = S^*(\eta N^* - d_1)/d_2$ and this equilibrium is asymptotically stable if it exists, i.e., $0 < \varepsilon/r < \beta(1-d_1/\eta K)/(d_2+\delta)$ (Proof is omitted).

B. Lattice model

To examine how the local interactions affect the eco-epidemic dynamics, a prey-predator with disease model was constructed in a lattice landscape. The local dynamics and interactions of an infected predator-prey system were then incorporated into a patch-occupant lattice (individual-based) model. The synchronous updating and Periodic boundaries were adopted [5, 13].

TABLE I. PROBABILITIES OF STATE TRANSITION IN LATTICE MODEL

$p_t(i,j) \to p_{t+1}(i,j)$	**Probability**
$N \to S$	$\left(1-(1-\varepsilon)^{N_S}\right) N_S/(N_S+a)$
$S \to I$	$1-(1-\beta)^{N_I}$
$S \to E$	d_1
$I \to S$	δ
$I \to 0$	d_2
$E \to N$	$1-(1-r)^{N_N}$

Table footnote: N_N, N_S, N_I are, respectively, the sum of patches occupied by prey, susceptible predator and infected ones in the neighborhood. $N_S/(N_S+a)$ represents the Allee effect.

Considering a disease that infects the predator populations, we thus have four possible states for each patch $p_t(i,j)$: empty (E), occupied by a prey (N), occupied by an susceptible predator (S), or occupied by a infected one (I). Detailed rules governing the dynamics of this eco-epidemiological model are listed in Table I. To reveal the effect of neighboring structures on the epidemic spreading, we chose three kinds of neighborhood: von Neumann neighborhood ($Z=4$), Moore neighborhood ($Z=8$) and 24-neighborhood ($Z=24$). Simulations were all run on a two-dimensional lattice network of 50×50 patches.

To describe the spatial structure of the simulation results from the above model, we selected a simple spatial-autocorrelation index, namely the join-count statistics (JCS) [12, 13]. Through counting the occupancy and spatial correlation (i.e. the clumping degree), we used JCS to classify the distributions as spatially aggregated, segregated or random [12, 13]. Occupancy P_σ is the proportion of patches being occupied by population σ ($\sigma = N, S, I$). The local density $Q_{\sigma/\sigma}$ is the conditional probability that a randomly chosen nearest neighbor of a patch in state σ is found in state σ. Therefore, spatial explicit aggregation distribution can be described by the JCS as $C_\sigma = Q_{\sigma/\sigma} - P_\sigma > 0$, which implying the positive first-order spatial correlation between two adjacent occupied patches. The spatial random distribution has $C_\sigma = 0$ and indicates the independence of two adjacent, occupied patches. The spatial segregated distribution has $C_\sigma < 0$ and indicates a negative spatial correlation between two adjacent patches [13].

III. RESULTS

Using ODE model, we can study the stability of (1) impacted by Allee effect. Through Numerical simulation, a more intriguing behavior of the eco-epidemiological system with change of the Allee effect constant was revealed. First, we found two kinds of attractor depending on the Allee effect constant: the point attractor and the limit cycle (Fig. 1). Second, a weak Allee effect (e.g., $a=0$, 1) and a strong Allee effect ($a>28$) can stabilize the dynamics of the eco-epidemiological system (Fig. 1). The Allee effect at a moderate intensity ($1<a<28$) will make the steady state undergo Hopf bifurcation and induce a periodic oscillation.

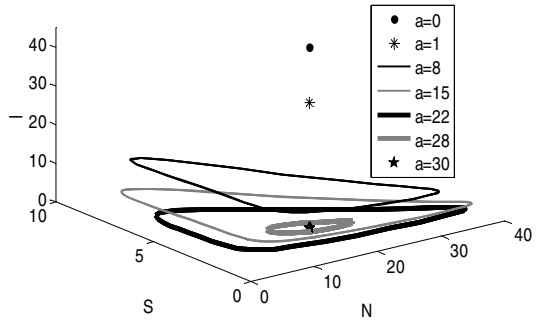

Figure 1. The attractors of (1) under various intensity of Allee effect. $r=0.8$, $\varepsilon=0.23$, $\eta=0.18$, $K=60$, $\beta=0.08$, $d_1=0.1$, $d_2=0.15$, $\delta=0.05$.

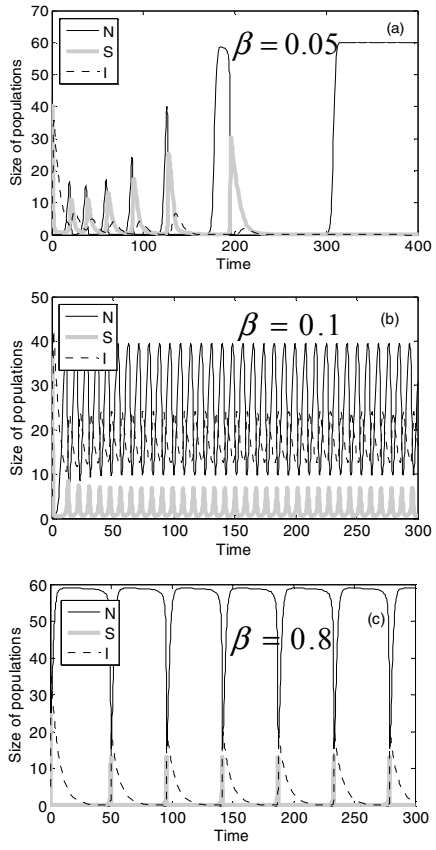

Figure 2. Dynamical behaviors of (1) under different infection rate. Other parameter values are the same as Figure 1.

Next, we will discuss how the infection rate, β, affect the dynamics of eco-epidemiological system with the Allee effect (Fig. 2). Results showed that there exist three distinct and typical dynamics of epidemic, namely, outbreak but last disappearance ($\beta = 0.05$, Fig. 2a), endemic and periodic oscillation ($\beta = 0.1$, Fig. 2b), and burst-like ($\beta = 0.8$, Fig. 2c). Comparing Fig. 2b and c, we can find that variation in the time period of oscillation with increasing infection rate. It is clear that the oscillating period with low infection rate is smaller than the one with high infection rate. This can be explained by the rapid depletion of the susceptible predators with too high infection rate, which effectively reduces the transmission of the disease. Seeing from Fig. 2c, we can also find that the population of the infected predator peaks almost immediately after the susceptible predator peaks. And the prey decreases very rapidly with the growth of the predator species inducing a sharp decline in population of the preys.

In order to explore the effects of Allee effect and local transmission process on the spreading dynamic of the epidemic disease, we compared the difference with three different intensities of Allee effect ($a = 0, 4, 8$) and neighboring structures (Von Neumann $Z = 4$; Moore neighbourhood $Z = 8$; twenty-four neighbourhood $Z = 24$) under lattice model. First, the occupancy of infected predator populations decreases dramatically as the Allee effect increases (Fig. 3a) and neighborhood size decreases (Fig. 3b). Second, The JCS of the disease (infected predator) was increasing with the intensity of Allee effect (Fig. 3c). Moreover, with the increasing of neighborhood, the spatial correlation of infected predator declined obviously, which except at $Z = 4$ induced by the extinction of infected predators (Fig. 3d). The above results indicate a high clustering degree of infected predators will be under strong Allee effect and low neighborhood.

IV. DISCUSSION

The ordinary differential equation revealed two key results. Firstly, the Allee effect is a stabilizing or a destabilizing force in eco-ecological system could be determined by its intensity. This result is agreement with our previous eco-ecological model following the standard incidence [5], which confirms that the impacts of Allee effect on eco-ecological systems are robust. Secondly, the infection rate plays a key role in the dynamics of eco-epidemiological system. Very low infection rate may induce predator being extinct. The biological reasons could be due to as follows: very low infection rate makes the infected predators rarely, and then the susceptible ones have a higher chance to increase, which induces the prey population at very low level. As a consequence, the resource shortage and the Allee effect may cause the predators to decline dramatically and be extinct lastly. Too high infection rate induces the susceptible predators and infected ones both show large amplitude "burst" type of oscillatory dynamics. Thus, the disease is not only increases the oscillatory period in the populations, it also can increase the maximum population size of both the susceptible and infected predators.

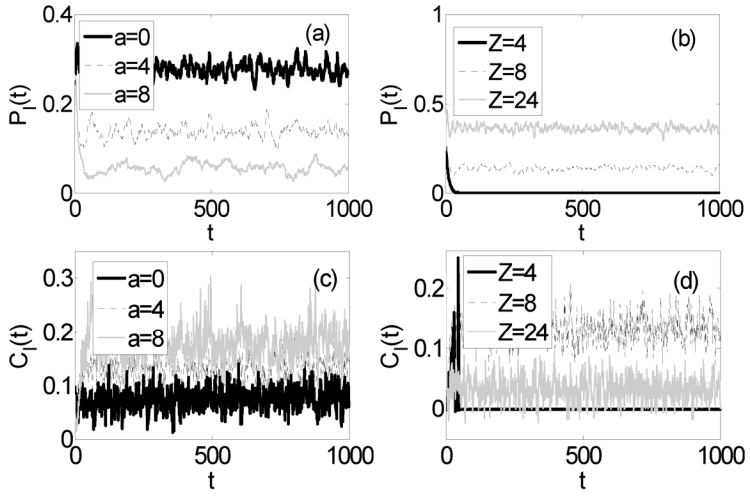

Figure 3. The temporal dynamics of infected predator response to different Allee effect and number of neighborhood patches under lattice model. Parameter values: $Z = 8$ (a, c); $a = 4$ (b, d). Other parameter are: $r = 0.15, \varepsilon = 0.1, \beta = 0.15, d_1 = 0.05, d_2 = 0.1, \delta = 0.1.$

The decline in the number of neighboring patches indicates the drop of patch connectivity, which markedly reduces the abilities for colonization, predation and infection. So the decrease of the number of neighbors will have the same effect as habitat loss [12, 13]. As obtained from our results, the decreasing number of neighbors can contribute to the control of

the epidemic. The biological reason for this phenomenon may be that fewer neighbors make the local susceptible predator deplete rapidly due to Allee effect, which further reduces the transmission of the disease. Moreover, result from JCS showed that the spatial clumping degrees of the epidemic will increase with Allee effect and decline in response to neighborhood. The results confirm that a highly aggregated distribution of species is a common behavioral strategy when dealing with the environmental stresses.

The aim of mathematical epidemiology is possibly to obtain the dynamics of disease transmission and devise reasonable vaccination policies according to the spreading trend. Consulting the temporal dynamics of the infected predator, we know that the disease control could be implemented by restricting the range of disease diffusion and (or) increase the intrinsic biological mechanism (e.g., Allee effect). As such, all the results presented in this paper are only a starting point for understanding the spreading of eco-epidemiology, which surly requires further exploitations.

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