Ecological Modelling 277 (2014) 97–107

Contents lists available at ScienceDirect

Ecological Modelling

journal homepage: www.elsevier.com/locate/ecolmodel

Can we use disease to control biological invasion?—A theoretical research

Yinghui Yang[a], Weide Li[b,*], Gang Wang[a]

[a] School of Life Sciences, Lanzhou University, Lanzhou 730000, China
[b] School of Mathematics and Statistics, Lanzhou University, Lanzhou 730000, China

ARTICLE INFO

Article history:
Received 9 August 2013
Received in revised form
19 December 2013
Accepted 21 January 2014
Available online 18 February 2014

Keywords:
Invasion control
Releasing disease
Pair approximation
Cellular automata
Eco-epidemiological model

ABSTRACT

Many investigations reveal that diseases and pathogens have a certain role in promoting the control of biological invasion. They are mostly based on mean-field assumption. Only few of them have considered the local spatial effect that is more close to the reality. In this research, we developed a local pair-dynamic model based on the SI (susceptible infected) framework among competitive interaction system in which invasive species is the superior competitor. By analyzing the values of several important parameters in steady state, we find that such a disease-introduction control measure does have a reversing effect on biological invasion, but this effect can only occur under specific conditions such as: (1) native species in the target invasion system should have a limited birth rate because too large colonization ability will correspond to a worse controlling result, (2) this kind of control measure will be more effective on the invaders with stronger competitive power and (3) in terms of chosen pathogens, we should select those with high infectiousness and moderately virulent diseases. These results are confirmed by cellular automata simulation, and the verdicts are different with cases under mean-field approximation. Our findings might provide meaningful guidance for invasion control before large-scale control programmes.

1. Introduction

Biological invasion has caused a grave threat to the ecosystem function, resource availability, human health and economic sustainable development (Lonsdale, 1999; Stanton, 2005). It is also considered to be an important reason leading to the loss of biodiversity (Kenis et al., 2009; Oliveira and Hilker, 2010; Stanton, 2005; Xu et al., 2004). Ehler (1998) once called for increased research of the mechanism underlying biological invasion and better linking to control measures. Then numerous empirical or theoretical studies on invasion subject have gradually increased. Despite such efforts, ecologists still pursue some questions like why the number of an invasive species can be dramatically larger than that of a native resident and what actually we can be down to slow down or even reverse the invasive spread (Colautti et al., 2004; Fagan et al., 2002; Marco et al., 2002). Currently, some evidences implied that disease has an important role in determining invasion's outcome, and as a kind of biological control measures, it is also regarded as promising compared with those time-consuming, costly and unsafely classical methods such as culling and trapping programs (Courchamp and Sugihara, 1999; Cleaveland et al., 1999).

Residents are usually threatened by invaders in a variety of ways, including predation, parasitism, competition, and changes in habitat (Fahrig, 2003; Hart and Gardner, 1997; Roques and Hamel, 2007), among which competition is more common (Coleman and Levine, 2007; Parker et al., 1999; Richardson et al., 2004). As invaders are normally free from their natural enemies such as predators and pathogens, the competitive advantage will lead native species to be in danger or even become extinct (Petren et al., 1993; Petren and Case, 1996; Parker et al., 1999). As for animals, large species will replace small one, which is referred to as interference competition (Roughgraden et al., 1984). As for plants, competitive process is often carried out by inhibiting the seeding's germination and settlements to reduce their rival's possessive ability (Parker et al., 1999; Yurkonis and Meiner, 2004). Thus, competition is always regarded as one of the main relationships between native species and invaders.

Given the two points above, we will construct a mathematical model in which we let disease-introduction happen among one established invasive system suffering competitive relationship between resident and aggressor. Indeed, the diffusion propagation of invasive organisms and non-invasive organisms are identical with respect to mechanisms, and both contain intra-specific or inter-specific interactions (Petrovskii and Li, 2006). Therefore, to

* Corresponding author. Tel.: + 86 931 8490983; fax: +86 931 8912481.
E-mail addresses: yangyh2010@lzu.edu.cn (Y. Yang), weideli@lzu.edu.cn, weideli8888@hotmail.com (W. Li).

0304-3800/$ – see front matter © 2014 Elsevier B.V. All rights reserved.
http://dx.doi.org/10.1016/j.ecolmodel.2014.01.023

establish our mathematic model, actually we need a population dynamic model, or specifically the eco-epidemiology one. Compartmental method, namely ordinary differential equations, is always prevalent and applied to bio-invasion control studies (Courchamp and Sugihara, 1999; Oliveira and Hilker, 2010). These works discussed the potential impacts of disease in a predator–prey system. In these studies, invaders are unlikely to be fully eradicated. Hilker et al. (2005) take the spatial factor into consideration. A reaction–diffusion approach was provided to analyze the traveling wave of aggressors which were governed by strong Allee effect as well as an infectious disease. This kind of approach is initially developed by Fisher (1937), which presents a partial differential system that is continuous in time and space. Although the framework is ideally suitable for time-space continuous population dynamics and could generate one stably progressive wave, neither neighbor correlations nor stochastic factors were included ever, containing both of which may produce some new message. In this research, we mainly focus on these two points for which computer simulations are usually regarded as the most suitable method (Su and Hui, 2011; Webb et al., 2007a). Besides, in order to bridge the gap between computer simulation and mathematical analysis, we propose a pair approximation (PA) model. This was initially used in statistical physics (Tainaka, 1993) and first established for theoretical ecological research by Matsuda et al. (1992). This approach has been extended to many biological fields, such as host–parasite dynamics (Su et al., 2009; Webb et al., 2007a,b), the effect of spatial structure (Harada et al., 1995; Hiebeler, 2005; Matsuda et al., 1992; Yuko and Yoh, 1994), eco-epidemiology (Hui et al., 2006; Su and Hui, 2011) and the evolution of virulence (Boots and Sassaki, 1999, 2000, 2001). However, it is rarely applied into biological invasion but Ellner et al. (1998).

Our paper begins by examining bifurcations for several critical parameters and their independent or joint influence on equilibrium density in an established PA model. Then direct computer simulation (cellular automation(CA) is performed to track control processes and confer the PA's results. For exploring the importance of space on the control effect, especially the local space, we finally compare some of their ecological outcomes with those under mean-field situation (MFT) where individuals are completely mixed. Throughout the whole process, we are mainly concerned with the following two questions: (1) which properties should the invasion system possess have to be suitable for the disease-introduction control measure? If it is solved, then (2) how does a specific disease relate to the effectiveness of this method?

2. Method

2.1. Local situation (PA model)

We perform a lattice-structured habitat on a regular network of sites, among which one individual can occupy an arbitrary grid point at any time. Each square is in one of the four states: empty (o), native species (n), susceptible invader (s) or infectious invader (i). The later three kinds of individuals (n, s, i) can produce offsprings into empty patches. We also assume that susceptible invaders have a competitive advance compared with native species and they can replace the adjacent local residents directly at a certain rate. While they contract disease, susceptible invaders will lose their competitive power and even may have a changed capability of occupying empty sites. Additionally, because our purpose is to introduce disease rather than prevent it, infectious invaders will be set to be uncured. For the sake of brevity, we choose susceptible infected framework, namely SI. Let p_σ denote the probability that a randomly chosen lattice site is in state of $\sigma(\sigma \in \{o, n, s, i\})$. We call it global (or singlet) density. Doublet density $p_{\sigma\sigma'}$ represents the probability that a randomly chosen pair of nearest-neighbor site is in state $\sigma\sigma'$. Then we design local density $q_{\sigma/\sigma'}$ ($\sigma, \sigma' \in \{o, n, s, i\}$) as the conditional probability that a randomly chosen nearest neighbor of a site in state σ' is in state σ. By definition, doublet densities can be produced by global densities and local densities as $p_{\sigma\sigma'} = p_{\sigma'\sigma} = p_{\sigma'}q_{\sigma/\sigma'} = p_\sigma q_{\sigma'/\sigma}$. State transfer relationships of our system are illustrated in Fig. 1 and those for pair sites are also of great importance, especially in PA model. They are presented as follows:

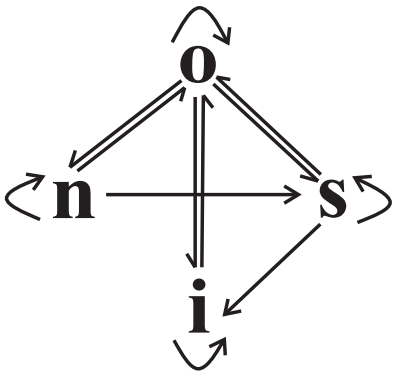

Fig. 1. Events determining the dynamic transition. Arrows represent the possible transitional directions of states for next time step.

- Birth process (Fig. 2(a)). Each individual attempts to deposit its offsprings into empty nearest-neighbor sites. r_σ ($\sigma = i, n, s$) are the intrinsic birth rates and respectively marked as r_j ($j = 1, 2, 3$) for convenience in our models. When it comes to pair states (we always assume the right site is under transmitting in the following content), like the σ–o pair, the newborn individual can not only come from its pair site σ but also the other neighborhoods (green circles in Fig. 2(a)). In o–o case, since the pair site is also empty, right transmitting site can only be filled with the other neighborhoods' offspring. Therefore, birth rate of σ–o is $r_\sigma[\theta + (1-\theta)q_{\sigma/o\sigma}]$ while that of o–o is $r_\sigma(1-\theta)q_{\sigma/oo}$ (here $\theta = 1/z$, where z is the neighbor size. It conveys the probability of interaction with the pair site as one of adjacent neighbors. Here we set $z = 4$).
- Death process. All individuals die at a constant possibility (d) which is independent of neighboring sites' states.
- Competition process (Fig. 2(b)). Susceptible competitor, namely s, can colonize patches originally occupied by native species at the rate f. Pair $\sigma - n$ ($\sigma = o, n, i$) and s–n are similar to birth process.
- Infection process. Disease transmission happens at the proportion of β through local interaction and that among pair sites is presented in Fig. 2(c). Furthermore, aggressors carrying disease also suffer an additional disease-induced death rate α (referred to as virulence).

All above facts are sufficient to yield following set of differential equations.

$$p'_{oo} = 2(d+\alpha)p_{io} + 2dp_{so} + 2dp_{no} - 2(1-\theta)p_{oo}[r_3 q_{s/oo} + r_2 q_{n/oo} + r_1 q_{i/oo}], \quad (1)$$

$$p'_{ss} = 2f[\theta + (1-\theta)q_{s/ns}]p_{ns} + 2r_3[\theta + (1-\theta)q_{s/os}]p_{os} - 2dp_{ss} - 2\beta(1-\theta)q_{i/ss}p_{ss} \quad (2)$$

$$p'_{nn} = 2r_2[\theta + (1-\theta)q_{n/on}]p_{on} - 2dp_{nn} - 2fq_{s/nn}p_{nn}(1-\theta), \quad (3)$$