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Procedia Environmental Sciences 13 (2012) 1010 – 1018

Procedia Environmental Sciences

The 18th Biennial Conference of International Society for Ecological Modelling

Using Cellular Automata to study the effect of competition for epidemic diseases

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Abstract

Cellular Automata (CA) has shown to be a valuable approach in ecological modeling, in particular when dealing with local interactions between species and their environment. A stochastic cellular automata model, which included two competitors (the inferior species which is immune to a disease and the superior one which is sensitive to the disease), is constructed. Through time series analysis and spatial pattern analysis, the influence of competition effect upon the behavior of epidemic diseases has been investigated to know whether the competition effect is in favor of epidemics control. Then, some strategies for introducing competitors to the infectious system are explored. The result shows that introducing some right competitors into the infection region may be a considerable policy. The population with high colonization rate, low extinction rate and long colonization radius is introduced preferably. The result may give us some suggestions for epidemic control in conservation of wild populations.

Keywords: Cellular Automata; Spatial pattern; Competition model; Eco-epidemiology; Time series analysis

1. Introduction

Eco-epidemiology is a new branch in mathematical biology which considers both ecological and epidemiological issues simultaneously [1,2]. Both theoretical and empirical researches on eco-epidemiology had multiplied recently, resulting in the prosperity of the important topic [3-9]. In general,

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1878-0296 © 2011 Published by Elsevier B.V. Selection and/or peer-review under responsibility of School of Environment, Beijing Normal University.
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the classical approach for modeling the spread of disease is based on the ordinary differential equation, which is dependent on the mean-field assumption that ignores space completely [10].

While theoretical and field experiments have suggested that the outcome of eco-epidemiology is significantly affected by spatial factors [11], the spatial-temporal dynamics and pattern of eco-epidemiological systems have hardly been considered. Fortunately, they have begun to be considered by many ecologists owing to the utilization of new techniques and the availability of more powerful computers [12]. Malchow et al. [13] and Su et al. [14] have considered the spatial dynamics of epidemic in predator-prey system, but literatures with regard to spatial competition model with disease are rare. Most realistic models focusing on the spatial aspects of epidemic dynamics are based on following two basic frameworks [15]: one based on reaction-diffusion models [13,16], which can imply the temporal heterogeneity and spatial heterogeneity simultaneously, and the other based on Cellular Automata [17], which is appropriate for studying spatial dynamics and patterns [18].

Cellular Automata (CA) is characterized by a regular lattice, an interaction neighborhood template, a set of elementary states, and a local space- and time-independent transition rule which is applied to each cell (or patch) in the lattice. It has been proved to be not only a fascinating topic by itself, but a valuable tool in various branches of science. CA models have been proposed for a large number of biological applications [14,19-23,]. The advantage of CA lies in that it can reflect temporal dynamic and spatial dynamic simultaneously [15,19,24-26]. Cellular Automata for simulating infectious diseases has been used to discover the spread of the disease or to work out remedial plans, and specialized models for different diseases have been presented in the past [27-29].

An explicit competition model based on CA framework is set up and an infectious disease is introduced to the superior competitor population. We focus on two questions: Can the competition effect be in favor of epidemics control? If it is so, what kind of competitor should better be chosen? These will be discussed by time series analysis and spatial pattern analysis.

2. Model Construction

The constructed model contains two populations, S1 and S2. S1 is the inferior population which is the loser in local competition; S2 is the superior population in local competition. In addition, we assume the superior is susceptible to some diseases for some reasons. Therefore, a cell of the CA model includes four states: empty patches, patches occupied by the inferior S1, patches occupied by the infected superior IS2 and patches occupied by the healthy superior S2, denoted by figures '0', '-1', '1', '2' respectively. Then the transitional rules of these patch states will be: First, these populations can colonize empty patches. Second, only healthy superior S2 can invade a patch containing S1; when it does so, S1 is excluded immediately. Third, population S2 and IS2 can prevent invasion by the inferior S1, and S1 only can prevent invasion by IS2. Besides, each local population has a risk to extinction in a patch. The transition structure can be implied by the transition rules [30], thus the corresponding transition matrix is

$$T=\begin{pmatrix} 1-C_x-C_y-C_I & E_x & E_y & E_I \\ C_x & 1-E_x-C_y & 0 & 0 \\ C_y & C_y & 1-E_y-a & R \\ C_I & 0 & a & 1-E_I-R \end{pmatrix}. \quad (1)$$

Where C_x and E_x are the colonization probability and the extinction probability of the population S1. C_y and E_y represent the colonization probability and the extinction probability of the healthy population S2. C_I ($C_I \leq C_y$) and E_I ($E_I \geq E_y$) are the colonization probability and the extinction probability of the infected

superior IS2. Parameters *a* and *R* denote the infection probability of disease and the recovery rate of the superior population infected by disease respectively.

T is the transition matrix of patch-occupancy (PO) model. A CA model, directly comparable to the PO model, can be constructed by applying the same transition matrix to a local neighborhood, not to the entire landscape. In order to obtain our CA model, the local interaction is introduced into the transition matrix. Generally, the colonization probability of a population on a patch has a positive correlation with the number of neighboring patches occupied by the population, and the infection probability of disease has a similar relation. But the extinction probability of the population does not have such relation. Therefore, we gain:

$$C_x = \frac{c_1 N_x}{(2d_1+1)^2-1},\quad C_y = \frac{c_2 N_y}{(2d_2+1)^2-1},\quad C_I = \frac{c_3 N_I}{(2d_3+1)^2-1},\quad a = \frac{\beta N_I}{(2D+1)^2-1},\qquad (2)$$

$$E_x = e_1,\quad E_y = e_2,\quad E_I = e_3.$$

Where, related parameters and special symbols in (2) are explained together in Table 1. According to transition matrix and local interaction, the stochastic CA model is completed finally. The corresponding transition probabilities are showed in Table 2.

Table 1. The meaning of related parameters and special symbols

parameter	range	meaning	parameter	range	meaning
c_1	[0, 1]	the colonization rate of the inferior population	L	N	the length of square lattice , N denotes the set of all natural numbers
c_2	[0, 1]	the colonization rate of the healthy superior	d_1	[1, L]	the colonization radius of the inferior population
c_3	[0, c_2]	the colonization rate of the infected superior	d_2	[1, L]	the colonization radius of the healthy superior
e_1	[0, 1]	the extinction rate of the inferior population	d_3	[1, d_2]	the colonization radius of the infected superior
e_2	[0, 1]	the extinction rate of the healthy superior	D	[1, L]	the transmission radius of the disease
e_3	[e_2, 1]	the extinction rate of the superior population infected by disease	N_x	[0,$(2d_1+1)^2$-1]	the number of neighbours which are occupied by the inferior population
R	[0, 1]	the recovery rate of the superior population infected by disease	N_y	[0,$(2d_2+1)^2$-1]	the number of neighbours which are occupied by the healthy superior
β	[0, 1]	the infection rate of disease	N_I	[0,$(2d_3+1)^2$-1]	the number of neighbours which are occupied by the infected superior

Table 2. The transition probability of our stochastic CA model

states transition	transition probability	states transition	transition probability

$0 \to -1$	$\frac{c_1 N_x}{(2d_1+1)^2-1}$	$2 \to 0$	e_2
$0 \to 2$	$\frac{c_2 N_y}{(2d_2+1)^2-1}$	$2 \to 1$	$\frac{\beta N_I}{(2D+1)^2-1}$
$0 \to 1$	$\frac{c_3 N_I}{(2d_3+1)^2-1}$	$2 \to 2$	$1-e_2-\frac{\beta N_I}{(2D+1)^2-1}$
$0 \to 0$	$1-\frac{c_1 N_x}{(2d_1+1)^2-1}-\frac{c_2 N_y}{(2d_2+1)^2-1}-\frac{c_3 N_I}{(2d_3+1)^2-1}$	$1 \to 0$	e_3
$-1 \to 0$	e_1	$1 \to 2$	R
$-1 \to 2$	$\frac{c_2 N_y}{(2d_2+1)^2-1}$	$1 \to 1$	$1-R-e_3$
$-1 \to -1$	$1-e_1-\frac{c_2 N_y}{(2d_2+1)^2-1}$		

3. Numerical simulation and results

In the research, *x(t), y(t)*, and *I(t)* represent the frequencies of the inferior competitor, the healthy superior, and the infected superior at time *t* respectively. The equilibriums of the model cannot be obtained directly due to demographic stochasticity (Table 1). Fortunately, we may solve this problem from simulations. To get the frequencies of populations when a simulation had reached equilibrium, the average frequency of each population in total grids are calculated. The simulations are run 1000 time steps on a two-dimensional space of 100×100 patches with fixed boundary condition. 15 simulations are run for each initial condition in order to maintain the reality and accuracy of the frequencies. The frequencies *x(t), y(t)* and *I(t)* are the average values of 15 times. Then the corresponding time series are obtained. All equilibrium calculations begin with initial conditions consisting of fixed frequencies of the three populations randomly distributed across the landscape. Although these related figures may be changed more or less in different values of parameters, results will not vary. The reason for selecting the values of parameters is that these situations are representative.

The influence of competition effect upon the spread of epidemic is illustrated in Fig.1. If the inferior is absent in the system, only 620 time steps are needed for the disease to spread across landscape from a local region (Fig.1b-Fig.1c). The ratio of the infected superior to the total superior, *I(t)/[I(t)+y(t)*], is about 0.87 when the system has been stable but with some slight fluctuation (red line in time series diagram, Fig.1a). To be convenient, the ratio is called the infection ratio. However, when a competitor joins in original system, the bad situation will change. It needs longer time to run through the whole superior population once the disease breaks out (Fig.1d-Fig.1f). Moreover, the infection ratio is reduced from 0.87 to 0.64 (Fig.1a). This can be explained that the inferior may be inclined to compete with the infected superior who has lost competitive advantage, while the healthy superior would like to exist in the region where the inferior population exists. With time process, a cricoids spatial wave may emerge until the disease spreads across the whole landscape. We may say that introducing an inferior competitor is in favour of epidemic control. Aside, when a competitor joins in original system, the

fluctuation of time dynamic curve may become more obvious (Fig.1a). In other words, the complexity of the system may be increased.

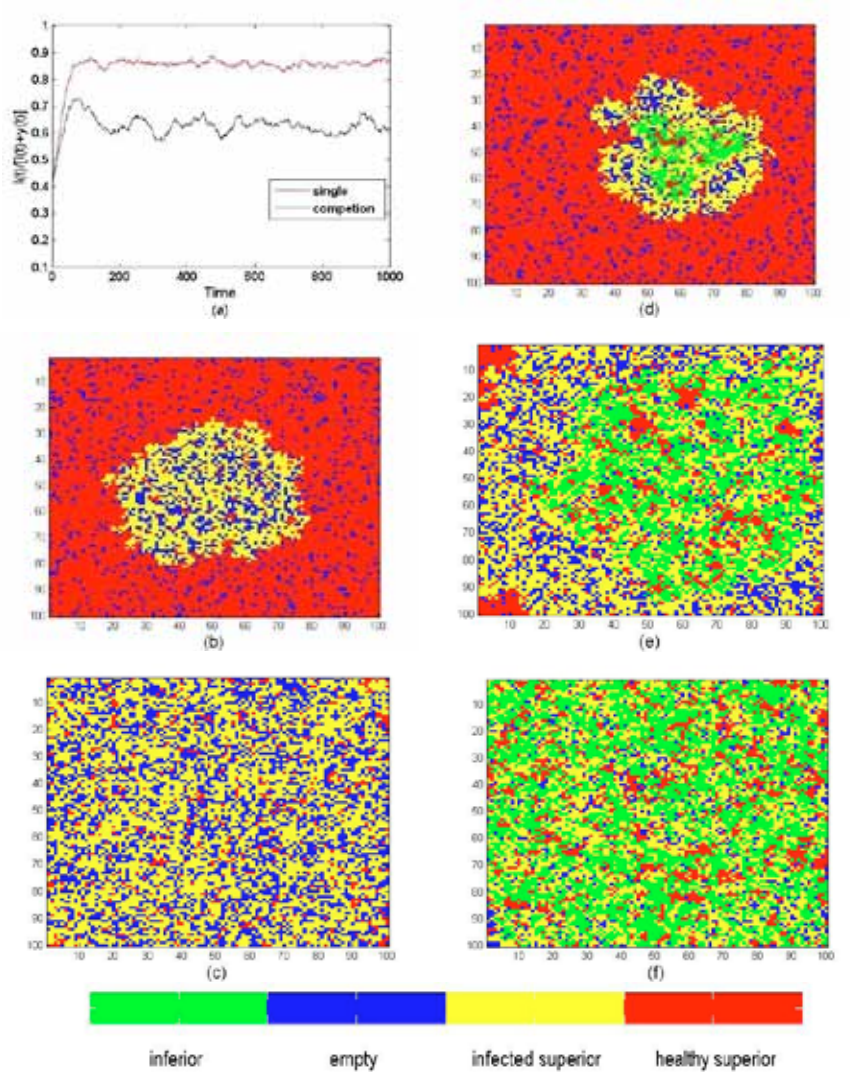
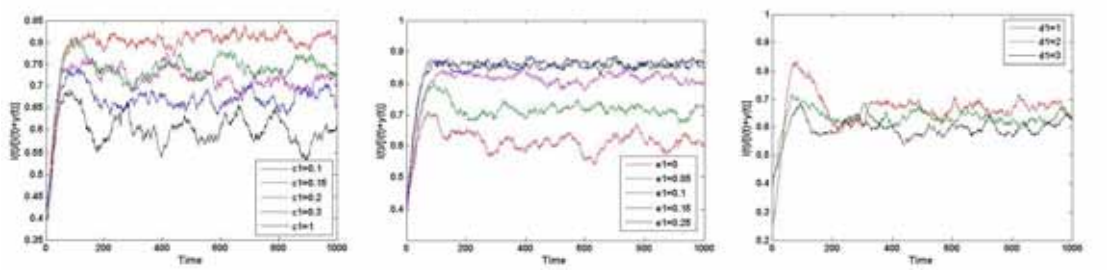


Fig. 1 Influence of competition effect upon the behaviour of epidemic. The first diagram (a) is about time series. The rest of left column (b, c) represents the spatial patterns in which only the superior exist at time 250, 620, respectively; the right column (d, e, f) represents the spatial patterns in which the inferior also exist at time 250, 620, 1000, respectively. Related parameters: c_1=0.5, c_2=0.1, c_3=0.05, e_1= 0.01, e_2=0.01, e_3=0.02, d_1=d_2=d_3=D=1, β=0.2, r=0.01.

Then, there is a question: What kind of competitor we choose is better in epidemic control? For discussing the question, the colonization rate c_1 (or the extinction rate e_1, or the colonization radius d_1) of the inferior population are changed to explore the dynamical behaviours of the system. The influence of these parameters for the system dynamics is given in Fig.2. The left panel (a) implies that increase of the colonization rate c_1 can decrease the infection ratio. With the climb of parameter e_1, the infection

ratio increases continuously until the inferior dies out (b). Same as c_1, the rise of the colonization radius d_1 is not in favour of the disease spreading, although the variation is slight (c). In addition, the increase of c_1 and decrease of e_1 will strengthen dynamic complexity. The different spatial distributions with different related parameters (c_1, e_1, d_1) are illustrated in Fig.3. The two sub-figures with same initial distribution are obtained after 1000 runs. All populations (S1, S2 and IS2) are in well distribution in the landscape, and there are no large regions occupied by any population. By comparison to Fig.3a, the number of patches occupied by the inferior S1 increases clearly with the change of these parameters (the rise of parameter c_1, the fall of parameter e_1 and the going up of parameter d_1) in Fig.3b, and the number of patches containing the healthy superior S2 has also increased. What is more, the number of the infected surrounded by the competitor S1 also increases. The increase of inferior competitor improves the immunity of the system for the epidemic disease, and the infected population may be insulated from the healthy population, thus the disease is not easy to infect the healthy individuals. It may be the reason why the infection ratio drops as the rise of parameter c_1, the fall of parameter e_1 or the going up of parameter d_1.



a b c

Fig. 2 Time series of infection ratio with different parameter values. The left diagram (a) is about the colonization rate (c_1 =0.1, 0.15, 0.2, 0.3, 1) of the inferior. The middle panel (b) represents time series following different extinction rate (e_1=0, 0.05, 0.1, 0.15, 0.25). The right panel (c) is about the colonization radius (d_1=1, 2, 3). Different parameter: e_1=0.01, d_1=1 in (a), c_1=0.1, d_1=1 in (b) and c_1=0.5, e_1=0.01 in (c). Other parameters: c_2=0.1, c_3=0.05, e_2=0.01, e_3=0.02, d_2=d_3=D=1, β=0.2, r=0.01.

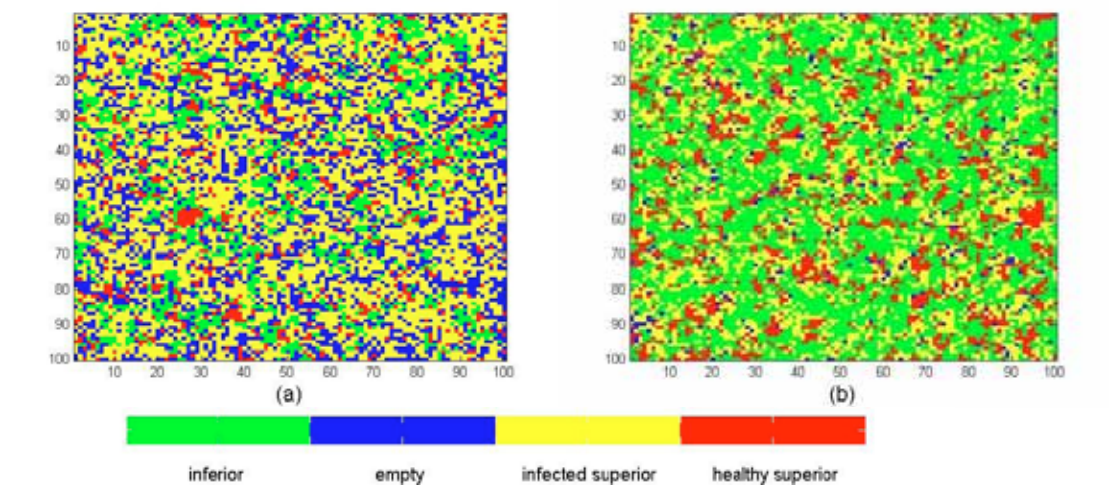


Fig.3 The spatial patterns with different parameter values (c_1, e_1, d_1). Special parameters: (a) c_1=0.2, e_1=0.03, d_1=1; (b) c_1=1, e_1=0, d_1=3; other parameters: c_2=0.1, c_3=0.05, e_2=0.01, e_3=0.02, d_2=d_3=D=1, β=0.2, r=0.01.

Table 3. Correlations between key parameters and dynamic complexity, epidemic spreading

Vary of parameters	Epidemic spreading	Dynamic complexity
Increasing of c_1	Negative	Negative
Decreasing of e_1	Negative	Negative
Increasing of d_1	Negative	Not obvious

4. Summary

Although epidemics have been researched on in many different fields by different means, the aim of most papers is controlling the disease effectively and developing suitable vaccination policies [13,14,31-34]. Three most widespread methods are curing the infected individual, isolating the infected and inoculating the susceptible. These methods are useful in the fight against AIDS, SARS, measles, rabies and so on [35,36]. However, these ways are not suitable to wildlife species with many difficulties in distinguishing between the healthy individuals and the infected, in searching and catching the infected.

This research implies that it may be a considerable policy to introduce an inferior competitor into a region with an infected population. The influence of key parameters for dynamical complexity and epidemic control is listed in Table 3. Although the increase of c_1, d_1 and decrease of e_1 will strengthen dynamic complexity, these variations may not accelerate the disease spreading. Therefore, the result suggests that in order to effectively control the spreading of epidemics, the introduced competitor-inferior species with high colonization rate, low extinction rate and long colonization radius is being favored. What is more, the selected population is immune to the epidemic disease. Besides, the more complicated dynamic will lead to the more difficult disease spreading (Table 3). In other words, increasing dynamic complexity of system may be a practical way of epidemic control. And the ways of increasing dynamic complexity of system may include introducing the right predators, competitors and the like.

Competition introduced to infectious system has been discussed in few papers, and one-host-two-pathogen system was the common thinking of this kind of theoretical research [37-39]. Venturino [40] discussed an ODE system which had similar construct as our model. His result was various depending on the relation of model parameters. Our research has one common result: the introduction of a competitor has the effect of removing the disease, only with the slight difference that he didn't meet the spatial part, although the system was deterministic. We not only touch the spatial-temporal dynamics of the system, but also broaden the result to a stochastic system in this research. While research on the conditions of dynamical complexity and the elimination of introduced competitor are other interesting topics, the corresponding results of our research may imply a novel view to epidemic control and conservation of wild life.

Acknowledgements

This research was supported by National Natural Science Foundation of China (40930533), National Science and Technology Support Program of China (2009BAC53B03), and the Scientific Research

Foundation for the Returned Overseas Chinese Scholars, Ministry of Education. We are grateful for the comments, reviews and encouragements from Dr. F Hilker, Prof GZ Du.

References

[1] Bairagi N, Roy PK, Chattopadhyay J. Role of infection on the stability of a predator-prey system with several response functions-a comparative study. *J Theor Biol* 2007; **248**: 10-25.

[2] Su M, Hui C, Zhang YY, Li ZZ. How does the spatial structure of habitat loss affect the eco-epidemic dynamics? *Ecol Mode* 2009; **220**:51-9.

[3] Hadeler KP, Feedman HI. Predator-prey populations with parasitic infection. *J Math Biol* 1989; **27**: 609-631.

[4] Venturino E. The influence of diseases on Lotka-Volterra systems. *Rocky Mountain J Math* 1993; **24**(1): 381-402.

[5] Gulland FMD. The impact of infectious diseases on wild animal populations: a review. In: Grenfell BT, Dobson AP , editors, *Ecology of infectious diseases in natural population*, Cambridge: Cambridge University Press. 1995, p. 20-51.

[6] Daley DJ, Gani J. *Epidemic modelling: an introduction, Cambridge studies in mathematical biology*. Canbridge: Cambridge University Press; 1999.

[7] Delgado M, Molina-Becerra M, Suarez A. Relating disease and predation: equilibria of an epidemic model. *Math. Methods Appl Sci* 2005; **28**: 349-62.

[8] Greenhalgh D, Haque M. A predator-prey model with disease in the prey species only. *Math Methods Appl Sci* 2007;**30**: 911-29.

[9] Zou W, Xie J H, Xiong ZL. Stability and Hopf bifurcation for an eco-epidemiology model with Holling-III functional response and delays. *Int J Biomath* 2008;**1**(3): 377-89.

[10] Law R, Diechmann U, Metz JAJ. Introduction. In: Diechmann U, Law R, Metz JAJ, editors, *The geometry of ecological interactions: simplifying spatial complexity,* Cambridge: Cambridge University Press, 2000,p.1 - 6.

[11] Johansen A. Spatio-temporal self-organisation in a model of disease spreading. *Physica D* 1994;**78**: 186-93.

[12] Levin SA, Grenfell B, Hastings A, Perelson AS. Mathematical and computational challenges in population biology and ecosystems science. *Science* 1997; **275**: 334-43.

[13] Malchow H, Hiker FM, Sarkar RR, Brauer K. Spatiotemporal patterns in an excitable plankton system with lysogenic viral infection. *Math Compu Model* 2005;**42**: 1035-48.

[14] Su M, Hui C, Zhang YY, Li ZZ. Spatiotemporal dynamics of the epidemic transmission in a predator-prey system. *Bull Math Biol* 2008;**70**: 2195-210.

[15] Tilman D, Kareiva P. *Spatial ecology: The role of space in population dynamics and interspecific interactions.* Princeton: Princeton University Press;1997.

[16] Cruickshank I, Gurney WSC, Veitch AR. The characteristics of epidemic and invasions with thresholds. *Theor Popul Biol* 1999; **56**: 279-92.

[17] Rhodes CJ, Anderson RM. Epidemic thresholds and vaccination in a lattice models of disease spread. *Theor Popul Biol* 1997;**52**:101-18.

[18] Dieckmann U, Law R, Metz JAJ. *The geometry of ecological interactions: simplifying spatial complexity*. Cambridge: Cambridge University Press; 2000.

[19] Wolfram S. Statistical mechanics of cellular automata. *Reviews of Modern Physics* 1983;**55**: 601-44.

[20] Iba Y. A stochastic cellular automaton model for recurrent epidemics: study of spatio-temporal patterns. *Proc Inst Statist Math* 1988;**36**: 69-88.

[21] Boerlijst MC. *Self structuring: A substrate for evolution*. Ph.D. Thesis, University of Utrecht;1994.

[22] Bagnoli F, Bezzi M. Species formation in simple ecosystems. *Intern J Mod Phys C* 1998;**9**(4): 1-17.

[23] Hui C, Li ZZ. Dynamical complexity and metapopulation persistence. *Ecol Model* 2003;**164**: 201-09.

[24] Hui C, Li ZZ. Distribution patterns of metapopulation determined by Allee effects. *Popul Ecol* 2004;**46**: 55-63.

[25] Hui C, McGeoch MA. Spatial patterns of prisoner's dilemma game in metapopulations. *Bull Math Biol* 2007;**69**: 659-76.

[26] Hanski I. Spatially realistic theory of metapopulation ecology. *Naturwissenschaften* 2001;**88**: 372-81.

[27] White SH, del Rey AM, Sanchez GR. Modeling epidemics using cellular automata. *Appl Math Comp* 2007;**186**(1):193-202.

[28] Gaylord RJ, Nishidate K. M*odeling Nature - Cellular Automata Simulations with Mathematica.* New York: Springer Verlag;1994.

[29] Doran RJ, Laffan SW. Simulating the spatial dynamics of foot and mouth disease outbreaks in feral pigs and livestock in Queensland, Australia, using a susceptible-infected-recovered cellular automata model. *Preventive Veterinary Medicine* 2005;**70**:133-52.

[30] Caswell H, Etter R. Cellular Automaton models for competition in patchy environments: facilitation, inhibition, and tolerance. *Bull Math Biol* 1999; **61**: 625-49.

[31] Kermack WO, McKendrick AG. Contributions to the mathematical theory of epidemics II-The problem of endemicity. *Proc R Soc* 1932; **138**A: 55-83.

[32] Anderson MY, May RM. *Infectious disease of humans, dynamics and control.* Oxford: Oxford University Press;1992.

[33] Chattopadhyay J, Sarkar RR, Ghosal G. Removal of infected prey prevent limit cycle oscillations in an infected prey-predator system- a mathematical study. *Ecol Model* 2002;**156**: 113-21.

[34] Carlsson-Grane U. Disease dynamics, host specificity and pathogen persistence in isolated host populations. *Oikos* 2006; **112**: 174-84.

[35] Bell DM, World Health Organization Working Group on Prevention of International and Community Transmission of SARS. Public health interventions and SARS spread 2003. *Emerg Infect Dis* 2004; **10**(11): 1900-6.

[36] Ferguson NM, Cummings DAT, Frascr C, Cajka JC, Cooley PC, Burke DS. Strategies for mitigating an influenza pandemic. *Nature* 2006;**442**(7107): 448-52.

[37] Saunders IW. Epidemics in competition. *J Math Biol* 1981;**11**(3):311-8.

[39] Ackleh AS, Allen LJS. Competitive exclusion and coexistence for pathogens in an epidemic model with variable population size. *J Math Biol* 2003; **47**(2):153-68.

[39] Sinha S, Misra OP, Dhar J. A two species competition model under the simultaneous effect of toxicant and disease. *Nonl Anal RWA* 2010;**11(**2):1131-42.

[40] Venturino E. The effects of disease on competing species. *Math Biosci* 2001;**174**:111-31.