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CHAOS IN AN ADAPTIVE NETWORK WITH REWIRING

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An epidemic spreading through an adaptive network, where the susceptible are able to avoid contact with the infective by rewiring their network connections, with random and regular contacts is considered. This paper uses a new mathematical model obtained by pair approximation to explore the effect of rewiring of the epidemic dynamics. We show that chaotic behavior can emerge while considering rewiring. The obtained results show that rewiring plays an very important role on the dynamical behavior of the epidemic model.

Keywords: Epidemic spreading; adaptive network; chaos

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1. Introduction

Mathematical epidemiology is a rapidly evolving field of fundamental importance in understanding communicable diseases and for identifying strategies for their prevention and control.[1–4] An alternative approach is to model a population of the susceptible and the infective and the contact patterns among them as a static random network.[5–8] This approach has generated a new category of epidemiological models in which epidemics spread from node to node by traversing network connections.[9–16]

However, there is a shortcoming of the network model that it is difficult to describe the explicit dynamical behavior of epidemics on networks. The mean outbreak size is easy to calculate, yet the dynamic epidemic incidence, that is the number of new infective at a time t, has been difficult to derive. Thus, pair-wise models have been developed to provide a means of modeling the spread of epidemics on networks.[17–19] These deterministic models form an intermediate step between mean-field and full network models by including, as their variables, all possible

configurations of pairs of individuals within the network. Pair-level models were used to investigate the outbreak of foot-and-mouth disease in the UK in 2001.[20]

In this paper, we want to investigate an adaptive network,[21] where the susceptible would be to keep away from the infective for the reason that the susceptible individuals have the ability to recognize the infective group and avoid connecting with them.

W let $[A]$ be the number of individuals of type A (either S or I to represent the susceptible, the infective, respectively); $[AB]$ the number of contacts in the network between a type A individual and a type B individual; and $[ABC]$ the number of $A-B-C$ triples within the network. For ease of book-keeping, pairs are counted once in each direction, thus $[AB]=[BA]$ and $[AA]$ is even.[11]

We consider a network with a constant number of nodes, N, and bidirectional links, L. The nodes represent individuals, which are either susceptible (S) or infective (I). In every time step and for every link connecting an infective with a susceptible (SI-link), the susceptible becomes infective with the fixed probability p. Infective recover from the disease with probability r, becoming susceptible again.

By using pair approximation,[11,22] we have the following three coupled ordinary differential equations[21]:

$$\frac{d[I]}{dt}=\beta_1[SI]-r[I]\,, \tag{1a}$$

$$\frac{d[SS]}{dt}=-2\beta_1[SSI]+r[SI]\,, \tag{1b}$$

$$\frac{d[II]}{dt}=\beta_1([ISI]+[SI])-2r[II]\,, \tag{1c}$$

where $[S]+[I]=N$ and $[SI]+[SS]+[II]=L$.

Random, one-off, contacts can be well captured with more familiar mean-field models.[1] These models, which have been applied to a huge range of infection scenarios, assume that all individuals in a population are capable of interacting, in contrast to the network paradigm that limits the number of interactions. While combined with random contacts, system (1) will be changed as:

$$\frac{d[I]}{dt}=\beta_1[SI]-r[I]-\frac{\beta_2 K}{N}[S][I]\,, \tag{2a}$$

$$\frac{d[SS]}{dt}=-2\beta_1[SSI]+r[SI]-2\frac{\beta_2 K}{N}[SS][I]\,, \tag{2b}$$

$$\frac{d[II]}{dt}=\beta_1([ISI]+[SI])-2r[II]+\frac{\beta_2 K}{N}[SI][I]\,, \tag{2c}$$

where β_2 is the rates of transmission through random contacts, K is the number of the populations of random contacts (here, $K<N$).

The advantages of a discrete-time approach are multiple in epidemic model.[23,24] Firstly, difference models are more realistic than differential ones since the epidemic

statistics are compiled from given time intervals and not continuously (a fact that can be of importance for fast-spreading epidemics). The second reason is that, the discrete-time models can provide natural simulators for the continuous cases (i.e., differential models). One can thus not only study with good accuracy the behavior of the continuous-time model, but also assess the effect of larger time steps. At last, the use of discrete-time models makes it possible to use the entire arsenal of methods recently developed for the study of mappings and lattice equations, either from the integrability and/or chaos points of view.

By using Euler method, we obtain the following models:

$$[I]_{n+1} = [I]_n + \beta_1[SI]_n - r[I]_n - b[S]_n[I]_n\,, \tag{3a}$$

$$[SS]_{n+1} = [SS]_n - 2\beta_1[SSI]_n + r[SI]_n - 2b[SS]_n[I]_n\,, \tag{3b}$$

$$[II]_{n+1} = [II]_n + \beta_1([ISI]_n + [SI]_n) - 2r[II]_n + b[SI]_n[I]_n\,, \tag{3c}$$

where $b = (\beta_2 K)/N$.

However, in the real world, humans tend to respond to the emergence of an epidemic by avoiding contacts with infective individuals. Such rewiring of the local connections can have a strong effect on the dynamics of the disease, which in turn influences the rewiring process. With probability w for every SI-link, the susceptible breaks the link to the infective and forms a new link to another randomly selected susceptible. Double- and self-connections are not allowed to form in this way. On the other hand, to close the system, we need to be able to approximate the triples that appear in terms of pairs and singletons. In the present paper, we can use the approximation[11]

$$[ABC] = \xi\frac{[AB][BC]}{B}\,, \tag{4}$$

where ξ is a parameter and A, B, $C \in \{S, I\}$. For sake of simplicity, we set $\xi = 1$ in the present paper. This leads to a system of three coupled difference equations as follows:

$$[I]_{n+1} = [I]_n + \beta_1(L - [SS]_n - [II]_n) - r[I]_n - b(N - [I]_n)[I]_n\,, \tag{5a}$$

$$\begin{aligned}[SS]_{n+1} = [SS]_n &- 2\beta_1\frac{[SS]_n(L - [SS]_n - [II]_n)}{N - [I]_n} + r(L - [SS]_n - [II]_n)\\ &- 2b[SS]_n[I]_n + w(L - [SS]_n - II_n)\,,\end{aligned} \tag{5b}$$

$$\begin{aligned}[II]_{n+1} = [II]_n &+ \beta_1\frac{(L - [SS]_n - [II]_n)^2}{N - [I]_n} + L - [SS]_n - [II]_n) - 2r[II]_n\\ &+ b(L - [SS]_n - [II]_n)[I]_n\,.\end{aligned} \tag{5c}$$

2. Numerical Simulations

In Fig. 1, the numerical results from system (5) are given. Without rewiring, there is only a single, continuous dynamical transition. That is, the disease will die out from an initial positive number. As the rewiring is switched on, this dynamic behavior is changed a lot. When $w = 0.03$, two-period solutions emerge. As w being further increased, chaotic behavior occurs [cf. Figs. 1(c) and 1(d)]. To well show the effect of rewiring, we plot the bifurcation diagram of $w - [I]_n$ in Fig. 2 with $b = 0.002$, $\beta_1 = 0.004$, $r = 0.01$, $N = 10,000$, and $L = 200,000$. There is stable two-period solutions for a large range of parameters, beyond which the system goes through a quasi-periodicity that eventually leads to chaos at $w \approx 0.422$. Also, we can obtain that there exists cascades of both period-doubling bifurcations and period-halving bifurcations in Fig. 2.

A powerful numerical tool to investigate whether the dynamical behavior is chaotic is a plot of the largest Lyapunov exponent, as a function of one of the model parameters. The largest Lyapunov exponent is the average growth rate of an infinitesimal state perturbation along a typical trajectory (orbit). We rewrite system (5) as:

$$x_{n+1} = F(x_n)\,, \tag{6}$$

where $x_n = ([I]_n, [SS]_n, [II]_n)^T$. Let $\|\cdot\|$ denote the Euclidean norm. The distance in state space between two initially close state vectors x_0 and $x_0 + \delta$ for small $\|\delta\|$

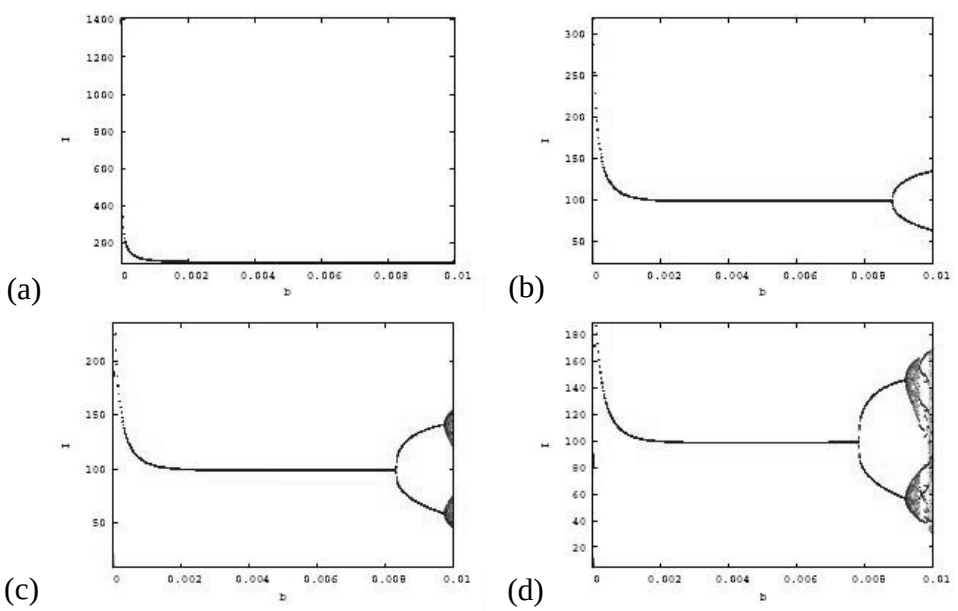

Fig. 1. Bifurcation diagram of $b-[I]_n$ as w being increased. (a) $w = 0$; (b) $w = 0.03$; (c) $w = 0.06$; (d) $w = 0.1$. The other parameter values are: $\beta_1 = 0.004$, $r = 0.01$, $N = 10,000$ and $L = 200,000$.

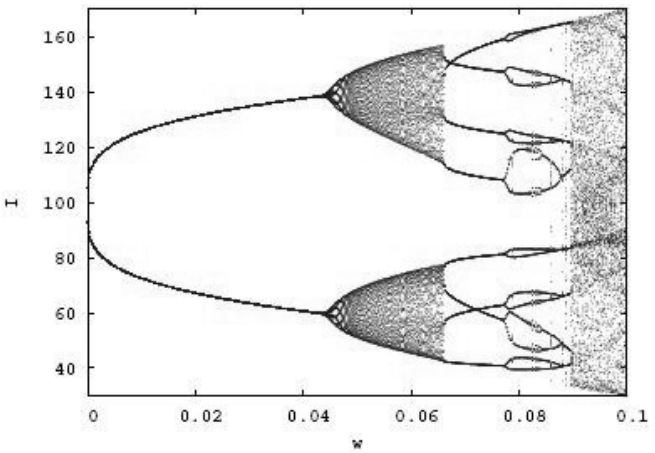

Fig. 2. Bifurcation diagram of $w - [I]_n$. The bifurcation diagram shows the long run dynamical behavior, 100 iterations after a transient of 100 iterations, as a function of the parameter w. Parameters value are used as that $b = 0.002$, $\beta_1 = 0.004$, $r = 0.01$, $N = 10,000$ and $L = 200,000$.

evolves as:

$$F^n(x_0 + \delta) - F^n(x_0) \approx DF^n(x_0)\delta = DF(x_n)DF(x_{n-1}) \cdots DF(x_1)\delta\,, \tag{7}$$

where $DF(x)$ denotes the Jacobi matrix of F evaluated at state x. For typical initial states x_0 the length of the perturbation grows exponentially as:

$$DF^n(x_0)\delta \sim e^{\lambda n}\|\delta\|\,, \tag{8}$$

motivating a definition of largest Lyapunov exponent as:

$$\lambda = \lim_{n\to\infty} \frac{1}{n} \log \frac{\|DF^n(x_0 + \delta)\|}{\|\delta\|}\,. \tag{9}$$

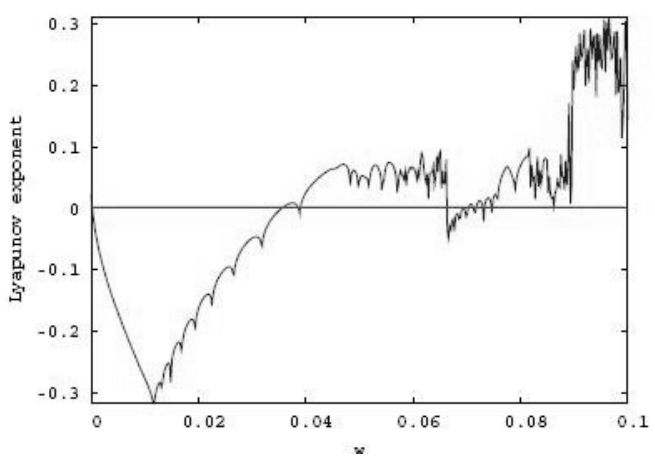

Fig. 3. The corresponding Lyapunov exponent of Fig. 2. This plot is based on 5000 iterations after a transient of 300 iterations.

For a chaotic attractor, the largest Lyapunov exponent must be positive. If it is less than 0, that implies that a stability state or a period attractor. We can obtain the corresponding Lyapunov exponent to the case that $b = 0.002$, $\beta_1 = 0.004$, $r = 0.01$, $N = 10,000$, and $L = 200,000$, which is shown in Fig. 3.

3. Conclusion and Discussion

In summary, we have shown that the interaction with random and regular contacts on an adaptive network can give rise to rich dynamics. In the present paper, we have studied only the simplest example of an adaptive network, in which the number of nodes and links remains constant and the local dynamics is simple, which is the same as in Ref. 21. Nevertheless, the rewiring can give rise to complicated dynamical features, like chaos.

To well know the relative importance of regular and random contacts, it is vital to be able to assess the level of clustering within social networks. This is not a easy task, and requires extensive surveying of social mixing behavior.[25] Information about behavior within households or workplaces, for example, though interesting and useful, is obtained from a naturally clustered subset of the population, so does not provide a sensible estimate of social clustering. Accurate surveys are needed to determine both how many regular and how many random contacts we have and also the amount of clustering displayed by the contacts.[19] On the other hand, chaos may cause the population to be out of control. But in the real world, the density of the infective needs to be under control or it will be harmful to the health of people worldwide. Thus, how to control chaos in the epidemic model is very important.

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