

Epidemic Oscillations in a Meta-Population Model

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Abstract: The individual mobility and the underlying spatial structure of populations play an important role in epidemic spreading processes. In this study, effects of these two factors on epidemic dynamics in metapopulation systems are investigated by using a discrete Susceptible-Infective-Recovered-Susceptible (SIRS) model. Our extensive numerical Monte Carlo simulations show that both irregular and regular oscillations can be observed, depending on the life cycles of epidemic diseases. Furthermore, the amplitudes of regular oscillations are enlarged by decreasing the move cycle. It is also found that heterogeneous connectivity patterns among subpopulations result in a global infection under fast movement, compared to homogeneous connectivity patterns.

Keywords: Epidemic, metapopulation, oscillation, SIRS model

INTRODUCTION

Epidemic dynamics has been extensively investigated in recent years. Many of them have focused on the spreading behaviours in the populations by using a network approach, where individuals are represented by nodes and the contacts between any pair of individuals are mapped to links. Various network connectivity patterns, like small world network and scale free network, have been manifested to play an important role in the study of the influence of the network topology upon the dynamics of epidemics. The underlying assumption in these models is that the population is localized in one region and the connections between individuals are static. Given the situation that individuals are distributed in relatively isolated regions or subpopulations connected by some degree of migration through corridors between these regions, then another approach called metapopulation modelling is being revamped in studying epidemic dynamics for such large scale populations. Diekmann and Heesterbeek (1999) study the mathematical epidemiology of infectious diseases: model building, analysis and interpretation. Anderson and May (1991) have a research of the infectious diseases of humans: dynamics and control. Bailey (1975) studies the mathematical theory of infectious diseases and its applications.

The meta-population approach is a basic theoretical framework to describe population dynamics in ecology. Populations of many species occupy patches of high

quality habitat and use the intervening habitat for movement from one patch to another. Such a collection of interacting populations of the same species is called a metapopulation, which means a population of populations. The classical metapopulation theory focuses on the processes of local extinction, recolonization and regional persistence and emphasizes the importance of connectivity between seemingly isolated populations. It has been found that the spatial structure of populations has remarkable impacts on the system's evolution. The meta-population approach can serve as a paradigm for studying the epidemic dynamics of spatially structured populations with well-defined social units (e.g., cities, towns, villages, etc.) connected through individuals' movement. Recently, this method has been successfully applied to study the dynamics of epidemic processes in metapopulation models characterized by heterogeneous connectivity patterns, as the outcome of the influences of network heterogeneity and mobility process on the invasion threshold. It is assumed in that model that individuals are homogeneously mixed in each subpopulation and the mobility rate is topologically dependent. Besides, there is another interesting model called dynamical domain network, which is also very promising for modelling the interactions between the subpopulations.

In this study, effects of these two factors on epidemic dynamics in meta-population systems are investigated by using a discrete Susceptible-Infective-Recovered- Susceptible (SIRS) model. Our extensive

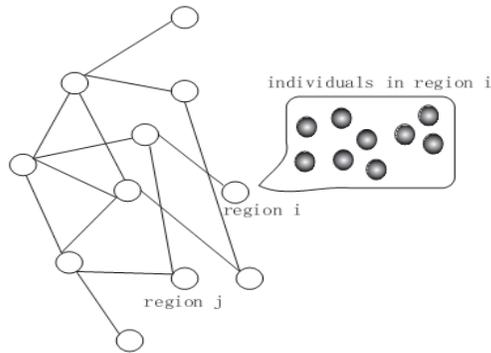


Fig. 1: Schematic representation of a metapopulation model R

numerical Monte Carlo simulations show that both irregular and regular oscillations can be observed, depending on the life cycles of epidemic diseases. Furthermore, the amplitudes of regular oscillations are enlarged by decreasing the move cycle. It is also found that heterogeneous connectivity patterns among subpopulations result in a global infection under fast movement, compared to homogeneous connectivity patterns.

METAPOPULATION MODEL

In the real world, the populations are localized discretely in different regions, such as cities, urban areas, or other defined geographical regions. Individuals can transfer from one region to another through traffic routes. This way, individuals in different regions mutually interact, which makes a disease propagating among large scale population possible. At the microscopic layer of the meta population system, individuals randomly contact each other and move on top of the spatial structure of the meta population system at the macroscopic layer. The macroscopic structures specifying the coupling between the subpopulations are in some cases homogeneous. Examples can be found in road transportation networks. It has been manifested that the connectivity distribution of such road networks follows an exponential. In other cases, spatial networks are often found to exhibit high heterogeneity. A typical example is given by the airline network which shows scale free property.

A Motivated by the above findings, we consider here the epidemic dynamics in metapopulation systems with complex network topologies (Fig. 1). This way, a metapopulation system is treated as the combination of many subpopulations which are represented by M nodes in the networks. In this study, we explore two kinds of network structures: small-world network and scale-free network. The epidemic model is defined as follows. Each individual can be in three different states. Following the original terminology and epidemiological literature, these three classes correspond to susceptible (S), infected (I) and recovered (R). Susceptible elements

can transit to the infected category through contagion by an infected one. Infected elements pass to the recovered state automatically after a recovery time τ_i . Recovered elements return to the susceptible state after a tolerance time τ_R . The contagion only occurs possibly for the susceptible elements by an infected element. Note that the elements in the refractory state are immune and cannot be infected. Here we study the discrete SIRS model, in which each element is characterized by a time counter $\tau_i(t) = 0, 1, \dots, \tau_i + \tau_R = \tau$ describing its phase in the disease cycle. The epidemiological state π_i of the element i is decided on its state in the following way:

$$\begin{aligned} \pi_i &= S \text{ if } \tau_i(t) = 0, \\ \pi_i &= I \text{ if } \tau_i(t) \in (1, \tau_i), \\ \pi_i &= R \text{ if } \tau_i(t) \in (\tau_i + 1, \tau) \end{aligned} \quad (1)$$

The state of an element depends on its current phase as well as on the state of its neighbours in the network. A susceptible element keeps $\tau_i(t) = 0$ until it becomes infected. Once infected, it goes (deterministically) over the disease cycle which lasts one period τ . During the first τ_i time steps, it is contagious and can potentially transmit the disease to its susceptible neighbours. Then it passes to the recovery stage automatically and remains in state R for τ_R time steps. During this stage, it is immune but not contagious. After finishing the cycle, it returns to the susceptible state. We indicate as $N_S(t)$, $N_I(t)$ and $N_R(t)$, respectively, the numbers in the three states at time t , with the total number of $N_S(t) + N_I(t) + N_R(t) = N$ being constant in time. Additionally, in order to include the individuals' mobility, it is possible to assume that each individual can transfer from one region to another with probability p_j but does not move with probability $1 - p_j$. This means, individuals perform a movement every $1/p_j$ time steps on average. In contrast to the stochastic moving process, we model the movement in a deterministic way by letting each individual perform a movement every τ_M . Besides, initial phases of individuals' movement are assumed to be randomly distributed in $[1, \tau_M]$. On the other hand, the destinations should be decided for those who complete one move cycle at some time instant. To take into account the preference of people's movement to central cities in the real world, we assume the probability that a region j is chosen to be the destination of individuals inside regions depends upon its degree, i.e:

$$d_j = \frac{k_j}{\sum_{i \in M_s} k_i} \quad (2)$$

where,

M_S = The number of neighbours of region s

Furthermore, inside the local regions, we suppose that the individuals randomly contact each other with certain probability p_0 at any time. In this sense, the

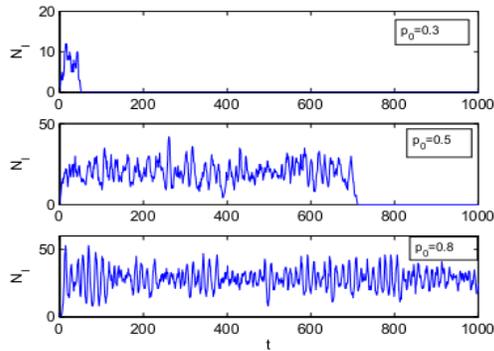


Fig. 2: Time series of the fraction of infected elements for different p_0 in one subpopulation without individuals moving in or moving out of the subpopulation. The contact network is generated with homogeneous connectivity with $N = 100$; the spreading begins with one infected individual. Parameters are: $\tau_1 = 3$ and $\tau_R = 5$

contact patterns in subpopulations can be treated as time-varying Erdős-Rényi random networks which are not required to be connected. By means of random networks, a susceptible element i in subpopulation s with $k_{s,i}$ neighbours is infected with probability $q = k_{s,inf}/k_{s,i}$ where $k_{s,inf}$ is the number of the infected elements in $k_{s,i}$ neighbours. Then the i th element will be infected with probability 1 if all of its neighbours are infected. Another reasonable choice is a fixed infecting probability with which the susceptible is infected by each of its neighbours. We have tested this and found that this criterion gives qualitative similar results for small q . An example of spreading dynamics in a single region without individuals moving between regions is shown in Fig. 2. On this condition, the individuals do not move in or move out of the regions. They can, however, randomly contact other individuals in the same region with probability p_0 at each time step. That is, the individuals are just locally movable. The subplots of Fig. 2 show different dynamic patterns for different p_0 . Similar behaviour can be observed for $\tau_1 = 8$; $\tau_R = 5$. To go deep into the differences of these epidemic processes, we can regard the regional individuals which may be infected as nodes of a network in which the links are the potentially infectious contacts among individuals but changing in time. It is clear that the prevalence of infection is influenced by transmissibility of such contact networks. In our model, the transmissibility depends on the probability p_0 , as it determines the contact rate. The larger p_0 is, the more each individual can contact. Furthermore, the local mobility of individuals also contributes to effective contacts. However, such transient contacts are too short to ensure disease transmission and therefore cannot lead to a wider spread of disease, in comparison with static contact networks. The numerical results Fig. 2 correspondingly show that the epidemics cannot spread among the population for small p_0 . As a comparison, oscillations almost exist for a large p_0 in our simulations.

NUMERICAL SIMULATIONS

We have performed extensive numerical simulations of the described metapopulation model. Populations with $N = 10^4$; 10^5 to 10^6 individuals have been explored, with $M = 100$ to 1000 regions and $\langle k \rangle = 6$ to 10. The individuals are initially distributed randomly in M regions. The spatial structures of metapopulation systems have been considered to be homogeneous. For the comparison and numerical simulation, we use the configurations with $N=10^4$, $M = 100$, $\tau_1 = 8$; $\tau_R = 5$ and $\langle k \rangle = 6$ throughout this study. Qualitatively similar results can be obtained for $\tau_1 \geq 3$. In the case of $\tau_1 = 1$, we get irregular oscillations under individuals' movement, which will be shown in the next part. Typical realizations start with the generation of the spatial network and the initialization of the state of the individuals. An initial fraction of 0.01 infected and the rest susceptible was used in all the results shown here. Other possible initial conditions have been explored as well and no changes have been observed in the behaviour. After a transient a stationary state is achieved. In order to inspect the dynamics of epidemic spreading in metapopulation system with complex topologies, we firstly study the case with homogeneous connections between the subpopulations. As in the Watts-Strogatz (WS) small-world model, the homogenous networks we study are random networks built upon a ring network with k -nearest neighbours. Each link connecting a vertex to a neighbour in the clockwise sense is then rewired at random, with a probability $p \in (0,1]$ to any vertex of the network. Any self-connections and multiple connections are not allowed. It should be noted that this procedure may produce a disconnected network. We only use the connected ones for our analysis.

We adopt Monte Carlo simulations for epidemic processes where the state of each individual can be tracked in time. We start the epidemics from a randomly selected region with a fraction of 0.01 infected as the seeds to propagate. Additionally, in the realization of individual's mobility, the individuals are initialized randomly in the move cycle τ_M . At each time step, the states of the individuals evolve by Eq. (1). They choose one of the neighbours of their habitat by Eq. (2) to transfer when they complete the movement cycle. The long-term behaviour of the fraction of the infected individuals is shown to oscillate for a short move cycle after a transient (Fig. 3).

In this case, the contact probability in each region is assumed to be $p_0 = 0.5$. Compared with the epidemic dynamics among the populations in a single region (Fig. 2), the global oscillation in a metapopulation system is partially attributed to individuals' movement. Other smaller contact probabilities have been tested (e.g., $p_0 = 0.01$) and oscillations have also been detected for a rapid

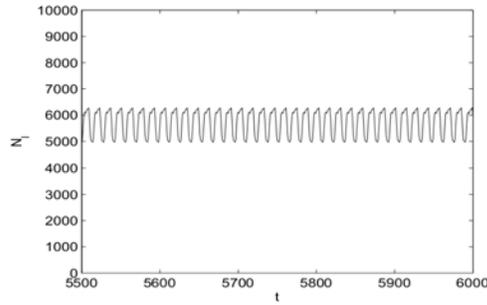


Fig. 3: The epidemics evolve in time, parameters: $\tau_I = 8$, $\tau_R = 5$ and $\tau_M = 1$

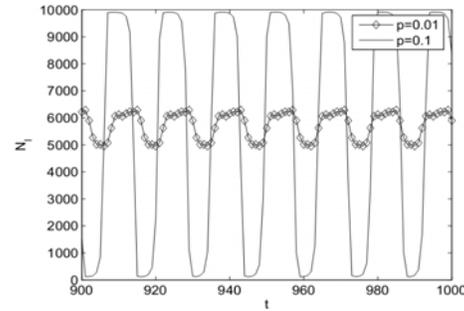


Fig. 6: Comparison of the oscillation amplitudes for various p in small-world network model. In these cases, $\tau_M = 1$

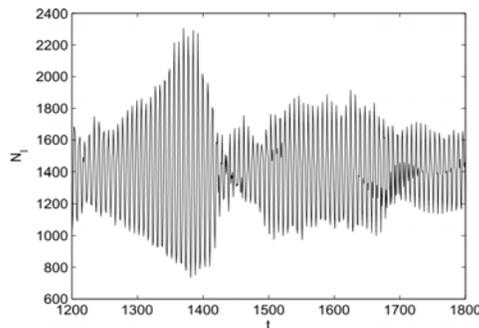


Fig. 4: Irregular oscillations in moving populations parameters: $\tau_I = 1$, $\tau_R = 5$ and $\tau_M = 5$

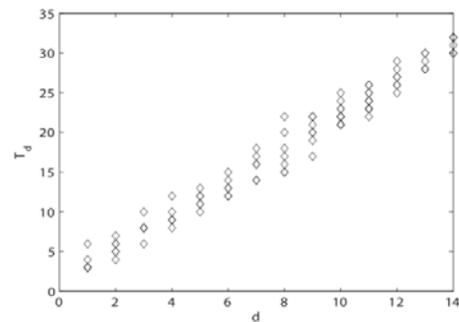


Fig. 7: T_d vs. d in the case of small-world spatial structure, parameters are: $\tau_I = 8$, $\tau_R = 5$, $\tau_M = 1$, $p = 0.01$ and $p_0 = 0.5$, respectively

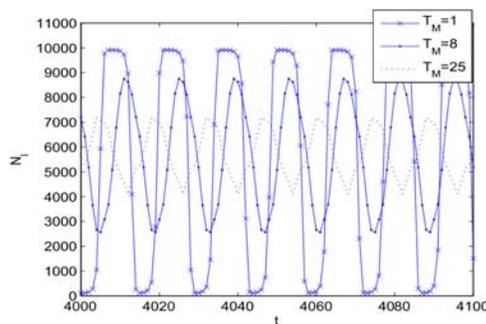


Fig. 5: The amplitudes of oscillations for different τ_M in metapopulation system with small-world connections in which $p = 0.01$; other parameters; $\tau_I = 8$, $\tau_R = 5$

movement. As mentioned above, the life cycle of the diseases can have an impact on the system dynamics. When τ_I is significantly small, then the periodic oscillations will be replaced by irregular fluctuation (Fig. 4).

In addition, the amplitude of the oscillations is shown to decrease with the increase of the move cycle (Fig. 5), which accordingly implies that the prevalence of the epidemics can be enlarged by individuals' rapid move. On the other hand, from Fig. 5 that, the period of the oscillations is slightly affected by movement, showing an intrinsic property of the SIRS dynamics.

According to the Watts-Strogatz model, the small-world property will emerge during the network structure change from a regular network to an entirely random

network by continuously increasing rewiring probability p from 0 to 1. This is a transition on topology. Similarly, a transition on dynamics can also be manifested by analyzing the change of the oscillation amplitudes while increasing the rewiring probability from $p = 0.01$ to $p = 1.0$. Figure 6 shows the oscillations of the epidemic incidence in the case of rewiring probability $p = 0.01$ and $p = 0.1$. Other values of rewiring probability ($p > 0.1$) have been also tested and the oscillations have been found to be almost the same as that in the case of $p = 0.1$ except for the phase (not shown here). From the numerical results, the variation of the oscillation amplitude we observe here is similar to the transition of small-world networks for increasing rewiring probability. Therefore, the change of the oscillation amplitude can be related to the topological properties of the spatial structures.

In order to explore the epidemic spreading process, firstly we define the distance d_i as the number of connections along the shortest path starting from the initially infected region to region i . Then denote the quantity T_d be the infection time that the infected individuals spend to arrive at the regions whose d_i is equal to d for the first time.

In Fig. 7, the time for the regions to be infected has a linear dependence on the distance d_i in the small-world network with $p = 0.01$. As the rewiring probability grows, the strip of time distribution becomes more flat (Fig. 8).

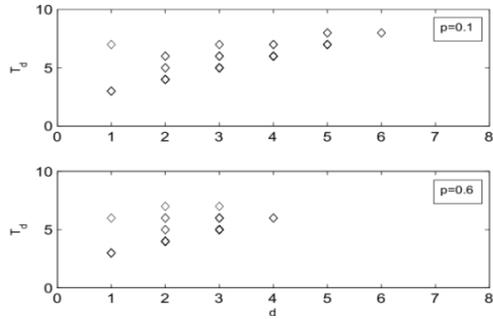
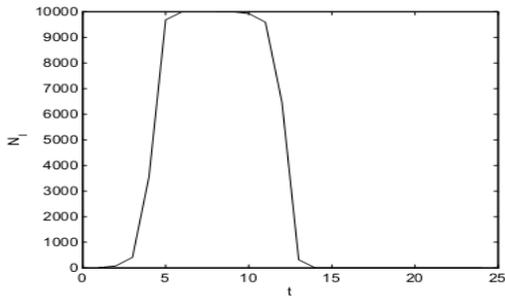
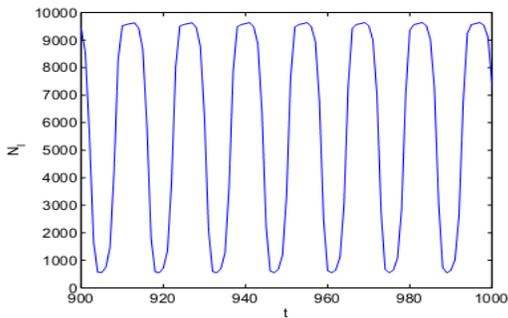


Fig. 8: T_{\max} vs. p_j , other parameters are the same as above setting



(a)



(b)

Fig. 9: Time series of epidemics in subpopulations with scale free network connections, one of the hubs in the scale-free network is selected to be the initially infected region, parameters: $\tau_1 = 8$, $\tau_R = 5$, $\tau_M = 1$ and (a) $\tau_M = 1$ (b) $\tau_M = 5$

In other words, the infection time T_d turns out to be approximately uniform, regardless of the distances. We recall that the clustering coefficient decreases rapidly, while the characteristic path length slightly changes as the randomness of network connections increases by changing $p = 0.01$ to $p = 1$. These results indicate the relationship between network topology and the dynamic processes.

Taking into account of heterogeneity of connections, we also study the epidemic dynamics in a metapopulation system with scale-free network topologies. Meanwhile, to get an insight into the

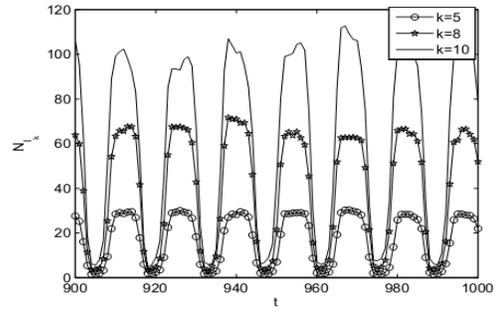


Fig. 10: The epidemic behaviours in subpopulations with different degree k , parameters are: $\tau_1 = 8$, $\tau_R = 5$, $\tau_M = 5$

topological fluctuations of the spatial structures, we use the quantity introduced in:

$$N_{ik} = \frac{1}{M_k} \sum N_{ij} \quad (3)$$

where, M_k is the number of subpopulations with degree k and N_{ik} represents the average number of infected individuals in regions with degree k . We generate scale-free networks by using the evolving model. In contrast to homogeneous networks, the epidemic oscillations can disappear here for a rapid movement for $\tau_M = 1$ (Fig. 9a).

For large τ_M , the incidence of epidemics oscillates periodically, as Fig. 9 shown. Note that the average distance between two nodes is smaller in scale-free networks than in random ones. In our simulations, we set the distance $d_i = 1, 2$ and 3 , respectively. Consequently, epidemics in scale-free networks can propagate more rapidly than in homogeneous networks by individuals' movement. Meanwhile, it is hard to find out a dependence of T_d on d . On the other hand, from Fig. 10, the quantity N_{ik} is also shown to be coherent in phase for the subpopulations with different degrees, which is similar to that in homogeneous networks.

CONCLUSION

Here we have performed extensive simulations on epidemic spreading in a metapopulation system with SIRS on individuals' dynamics. The connecting patterns and individuals' mobility are taken into consideration in the metapopulation model. The system shows long term sustained oscillations under individuals' move both in homogeneous and heterogeneous networks. In particular, the oscillation amplitude of the prevalence in subpopulations with homogeneous connectivity patterns is found to be enlarged by decreasing the move cycle, while the impact of movement on the amplitude is not apparent in heterogeneous networks. However, the global prevalence can happen to subpopulations with heterogeneous networks' rapid move. In our daily life, individuals' mobility can benefit regional economic

development which may accelerate the epidemic spreading. Therefore, there is a tradeoff between throttling the disease diffusion and maintaining economics for controlling individuals' movement.

Interestingly, a prominent increase of the oscillation amplitude of the prevalence with the increase of rewiring probability accords with the transition of small-world networks. Moreover, the heterogeneous distribution of the infection time transit to a homogeneous distribution when the spatial network structure goes through regular networks to random ones, which implies the underlying interactions between dynamic processes and connection patterns.

ACKNOWLEDGMENT

P. Li was supported by the National Natural Science Foundation of China under Grant No 61104224, D.Hu and Z.Lei was supported by the Science and

Technology Planning Project of Zhong Shan, under Grant No 20114A235 and 20114A230.

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