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Chapter 4

Epidemic Propagation Dynamics on Complex Networks

Xinchu Fu*[1], Zengrong Liu[2], Michael Small[3] and Chi Kong Tse[3]

[1]*Department of Mathematics, Shanghai University, Shanghai 200444, P. R. China*

[2]*Institute of Systems Biology, Shanghai University, Shanghai 200444, P. R. China*

[3]*Department of Electronic and Information Engineering, Hong Kong Polytechnic University, Hung Hom, Kowloon, Hong Kong*

Dedicated to Professor Youzhong Guo on the occasion of his 75th birthday

This paper provides a partial summary of our recent work on propagation dynamics of complex networks, mainly on constructing and studying network models of disease spreading and related propagation problems. Traditional compartmental models of disease spreading categorize individuals from a population based on their current pathology. These methods provide a population-based description that offers a smooth continuous and exponential response to the presence of an infectious agent. In many cases the available data is inconsistent with the standard models of disease spreading and can be more readily explained using a discrete agent-based model of spreading on complex networks. Moreover, models for diseases spreading are not just limited to SIS or SIR. For instance, for the spreading of AIDS/HIV, the susceptible individuals can be classified into different cases according to their immunity, and similarly, the infected individuals can be sorted into different classes according to their infectivity. In addition, some diseases may develop through several stages, or with mobility property, or with mutually exclusive feature (multi-strain epidemics). So in this paper, in order to better study the dynamical behavior of epidemics, we discuss different epidemic models on complex networks, and provide a mathematical analysis of the epidemic dynamics and spreading behavior, obtaining the epidemic threshold for each case. Some other related diffusion and propagation processes, such as information transmission dynamics, traffic flows, contact processes, etc., are also briefly discussed.

*Corresponding author. E-mail: xcfu@shu.edu.cn
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1. Introduction

Disease transmission has been extensively studied by the Markov chain and mean-field compartment models. While in the recent decade, great progress has been made by using new results from network science. When disease transmission[12] is modelled over networks,[14,16,18] it is usual to model the infectivity (that is, the rate of transmission between infected and susceptible nodes) by assuming that transmission is equally likely over all links. For an idealized model this is the natural way to consider infectivity. However, when the underlying complex network is scale-free, the situation becomes unrealistic in the extreme tail of the distribution. While it has frequently been observed that real human, social and disease transmission networks exhibit scale-free properties over several orders of magnitude, the tail of the distribution observed from data is always bounded. It is an open question whether these real networks are close to scale-free or only scale-free over a finite domain (note that any real network is of finite size so the degree is bounded).[20] In[25] for example, the observation of a scale-free transmission mechanism for avian influenza is tempered by the fact that the finite available data necessarily limits inference to a bounded distribution. Moreover, when considering transmission of a disease in a finite time period it is natural to suppose that there exists an upper bound on the infectivity of a highly connected individual. It is also quite reasonable to suppose that highly connected (and therefore highly visible) nodes in the network would be the focus of an immunization scheme (even for very limited control measures). Hence, in this paper we consider the case where the infectivity is a non-decreasing, but sub-linear, function of the node degree.

The standard network SIS compartment model (Susceptible-Infected-Susceptible) assumes that each infected node will contact every neighbor once within one time step,[3] that is, the infectivity is equal to the connectivity, or the node degree. In,[27] it is assumed that every individual has the equal infectivity A, in which, at every time step, each infected individual will generate A contacts, where A is a constant. Joo and Lebowitz[10] examined cases where the transmission of infection between nodes depends on their connectivity, and a saturation function $C(k)$ which reduces the infection transmission rate across an edge going from a node with high connectivity k was introduced.

Based on these results, in the present model, we take a more realistic approach. We assume the infectivity is piece-wise linear: when the degree k of a node is relatively small, its infectivity is proportional to k, e.g., αk; when k is big, say, surpasses a constant A/α, then its infectivity is, say, A. We further discuss this model with respect to the effects of various immunization schemes.

Our motivation for this study is the observation that transmission of SARS (Severe Acute Respiratory Syndrome), most notably in Hong Kong during 2003, exhibits characteristics typical of a small world or scale-free network.[21–24] During the SARS outbreak of 2003 several clusters of secondary infections were observed and

traced back to a single primary infection. This can either be explained by assuming a highly infectious source or by assuming a highly connected source. The latter case leads naturally to a scale-free model of transmission, and the question of under which conditions a real disease transmitted on an apparently scale-free network will have a finite threshold. It has also recently been observed that the spatial-temporal distribution of avian influenza outbreaks naturally induces a scale-free network connectivity.[25] In this work, the available data exhibits a power law over three orders of magnitude, but nonetheless, the tail of the distribution is bounded because the data is finite.

Of course, the SIS model used here was chosen because it is relatively simple, and also widely applicable. It may also be related to influenza vaccination problems[25] and strategies for dealing with computer viruses[7] among others.

This paper provides a partial summary of our recent series works on propagation dynamics of complex networks, mainly including construction and study of complex network models of disease spreading and related propagation problems.[45,47,53,55,56,58] A more complete list of research papers reflecting our recent works is referred to.[21–25,39–59]

2. The epidemic threshold for SIS model with piecewise linear infectivity

Individuals can be classified into three states, S-susceptible, I-infected and R-recovered (removed). Here we first consider the SIS model.

Let $S_k(t)$ and $I_k(t)$ be the densities of susceptible and infected nodes with degree k at time t, then

$$S_k(t) + I_k(t) = 1,$$

and the mean-field equations for infected nodes with degree k can be written as

$$\frac{dI_k(t)}{dt} = \lambda k(1 - I_k(t))\Theta(k,t) - I_k(t) \tag{2.1}$$

here we take a unit recovery rate, λ is the infection rate, and according to,[4,15,17,18,26] $\Theta(k,t)$ can be written in general as

$$\Theta(k,t) = \sum_{k'} \frac{\varphi(k')P(k'|k)I_{k'}}{k'} \tag{2.2}$$

where $\varphi(k)$ denotes the infectivity of a node with degree k, and $P(k'|k)$ stands for the probability for a node with degree k pointing to a node with degree k'.

An epidemic threshold for (2.1) is the critical value λ_c of the infection rate λ, if λ is below λ_c, the disease will gradually die out, while if λ is above λ_c, the disease will spread on the network.

In,[4,15,17,18] $\varphi(k) = k$, and then the epidemic threshold $\lambda_c = 0$ for sufficiently large networks. If $\varphi(k) = \alpha k$, the threshold λ_c also vanishes. In,[26] $\varphi(k) = A$,

where A is a constant, that means every node has the same infectivity, no matter its degree, small or large. In this case, $\lambda_c = \frac{1}{A} > 0$, a positive threshold.

For simplicity, we suppose that the connectivity of nodes is uncorrelated, then $P(k'|k) = k'P(k')/\langle k\rangle$, where $\langle k\rangle = \sum_k kP(k)$. Then (2.2) becomes

$$\Theta = \frac{1}{\langle k\rangle}\sum_{k'}\varphi(k')P(k')I_{k'} \tag{2.3}$$

where for scale-free node distribution $P(k) = C^{-1}k^{-2-\gamma}, 0 < \gamma \leq 1$, where $C \approx \zeta(2+\gamma)$ is approximately (as some degrees may not appear in a real network) the Riemann's zeta function.[8,25] Note that $\Theta(k,t)$ represents the probability that any given link points to an infected node. For simplified uncorrelated cases, $\Theta(k,t) = \Theta(t)$ doesn't depend on k.

2.1. *Piecewise linear infectivity*

Rather than a piecewise constant infectivity used in,[10] we here take a more realistic piecewise linear infectivity,

$$\varphi(k) = \min(\alpha k, A) \tag{2.4}$$

where α and A are positive constants, $0 < \alpha \leq 1$.

By imposing steady state $\frac{dI_k(t)}{dt} = 0$, from (2.1) we have

$$I_k = \frac{\lambda k\Theta}{1+\lambda k\Theta} \tag{2.5}$$

Substitute I_k in (2.3) by (2.5), we obtain a self-consistency equation as follows:

$$\Theta = \frac{\lambda\Theta}{\langle k\rangle}\sum_{k'}\frac{k'\varphi(k')P(k')}{1+\lambda k'\Theta} \equiv f(\Theta) \tag{2.6}$$

Obviously, $\Theta \equiv 0$ is a solution of (2.6), i.e., $f(0) = 0$. Note that

$$f(1) < 1, f'(\Theta) > 0, f''(\Theta) < 0,$$

therefore, a nontrivial solution exists only if

$$\frac{df(\Theta)}{d\Theta}|_{\Theta=0} > 1 \tag{2.7}$$

The value of λ yielding the inequality (2.7) defines the critical epidemic threshold λ_c:

$$\lambda_c = \frac{\langle k\rangle}{\langle k\varphi(k)\rangle} = \frac{\sum\limits_k kP(k)}{\sum\limits_k k\varphi(k)P(k)} \tag{2.8}$$

Approximating the sum in (2.8) on discrete k by continuous integration, and suppose the size of the network is sufficiently large, we can calculate λ_c as

$$\lambda_c = \frac{\int_m^{+\infty} k^{-1-\gamma}dk}{\int_m^{A/\alpha}\alpha k^{-\gamma}dk + \int_{A/\alpha}^{+\infty}Ak^{-1-\gamma}dk} = \begin{cases} \frac{\frac{1-\gamma}{\alpha m}}{(\frac{A}{\alpha m})^{1-\gamma}-\gamma}, & 0 < \gamma < 1, \\ \frac{\frac{1}{\alpha m}}{1+\log\frac{A}{\alpha m}}, & \gamma = 1, \end{cases} \tag{2.9}$$

where m is the minimum connectivity of the network, and $\alpha m < A$.

We remark that when $A \to +\infty$, from the above formula (2.9), $\lambda_c \to 0$, this is consistent with the fact that $\varphi(k)$ approaches to the linear infectivity $\varphi(k) = \alpha k$; and when $\alpha m \geq A$, we can calculate that $\lambda_c = 1/A$, this is consistent with $\varphi(k) = A$ for all k.

From (2.9), we have a positive epidemic threshold λ_c if

$$\alpha m < \frac{1}{\gamma^{\frac{1}{1-\gamma}}} A \ (0 < \gamma < 1) \ \text{ or } \ \alpha m < eA \ (\gamma = 1)$$

If $A \geq \gamma^{\frac{1}{1-\gamma}} m$ $(0 < \gamma < 1)$ or $A \geq e^{-1} m$ $(\gamma = 1)$, then λ_c is always positive.

2.2. *Piecewise smooth and nonlinear infectivity*

In some cases, the infectivity may take the following piecewise smooth function

$$\varphi_1(k) = \min(\alpha k^{\beta}, A), \ 0 \leq \beta \leq 1, \alpha > 0.$$

In this case, the epidemic threshold

$$\lambda_c' = \begin{cases} (\frac{A\beta}{\beta-\gamma}(\frac{\alpha m^{\beta}}{A})^{\frac{\gamma}{\beta}} - \frac{\alpha m^{\beta}}{\gamma(\beta-\gamma)})^{-1}, & \beta \neq \gamma \\ (\frac{m\alpha}{\beta}\log\frac{A}{\alpha m^{\beta}} + \frac{m\alpha}{\beta})^{-1}, & \beta = \gamma \end{cases}$$

then we have positive λ_c' if $(\alpha m^{\beta})^{\frac{\gamma}{\beta}-1} > \frac{1}{\gamma(\beta-\gamma)} A^{\frac{\gamma}{\beta}}$ and $\beta > \gamma$ or $\alpha m^{\beta} < eA$ $(\beta = \gamma)$

We can also discuss the epidemic threshold for a smooth nonlinear infectivity, e.g.,

$$\varphi_2(k) = \frac{ak^{\beta}}{1 + bk^{\beta}}, \ \ 0 \leq \beta \leq 1, \ a > 0, \ b \geq 0,$$

The details are discussed in.[47] We may also consider the effects of finite scale-free networks on the above discussions.[20,47]

3. Model with different immunities and infectivities

The SIS and SIR models cannot correctly interpret all kinds of diseases. For instance, for the spreading of AIDS/HIV, the susceptible individuals can be classified into different cases according to their immunity, and similarly, the infected individuals can be sorted into different classes according to their infectivity.

To better explore the mechanism of epidemic spreading on complex networks, we suppose that the S and I states can be subdivided into subclasses according to their different immunities, different infectivities and so on. That is, our models can describe S_iIR, SI_iR and $SI_{i,1}I_{i,2}, \cdots, I_{i,n}R$, $i = 1, 2, \cdots, n$. In order to make the models more reasonable, we also consider the birth and death of individuals. By using the method as in,[13] we assume that all individuals are distributed on the network, and each node of the network is empty or occupied by at most one individual.

The numbers 0,1,2,3 denote that the node has no individual, a healthy (susceptible) individual, an infected individual and a recovered individual respectively. Each node can change its state with a certain rate. An empty node can give birth to a healthy (susceptible) individual at the rate δ. The susceptible individual can be infected at a rate which is proportional to the number of infected individuals in the neighborhood or die at certain rate α. The infected individual can be cured at certain rate μ or die at certain rate β. If an individual dies, that node will become an empty node again.

3.1. *Multiple susceptible individuals*

We consider the susceptible individuals with several different cases according to their age or immunities. $S_{i,k}, i = 1, \cdots, n$ denote the density of the susceptible individuals with degree k and also belong to the i-th case, I_k and R_k denote the density of the infected individuals and the recovered individuals with degree k, respectively, then

$$\begin{cases} \frac{dS_{i,k}}{dt} = \delta_i(1 - \sum_{i=1}^{n} S_{i,k} - I_k - R_k) - \lambda_i S_{i,k} k\Theta - \alpha_i S_{i,k} \\ \frac{dI_k}{dt} = k\Theta \sum_{i=1}^{n} \lambda_i S_{i,k} - (\beta + \mu) I_k \\ \frac{dR_k}{dt} = \mu I_k - \gamma R_k \end{cases} \qquad i = 1, \cdots, n$$

Where $(1 - \sum_{i=1}^{n} S_{i,k} - I_k - R_k)$ is the density of empty nodes which will give birth to nodes with degree k, and $\delta_i, \lambda_i, \alpha_i$ are the birth rates, infectivity rates, and the natural death rates for the i-th case susceptible individuals respectively, β, μ are the natural death rate and the rate from $I \to R$ for infected individuals, and γ is the natural death rate of recovered individuals. Θ takes the form for uncorrelated networks, as discussed above.

Similar to the analysis in Section 2, we have the following inequality

$$\sum_{i=1}^{n} \frac{\lambda_i \delta_i}{\alpha_i} > \frac{\langle k \rangle (\beta + \mu)(1 + \sum_{i=1}^{n} \frac{\delta_i}{\alpha_i})}{\langle k^2 \rangle}$$

3.2. *Multiple infected individuals*

We suppose that the infected individuals are classified into several different cases according to their infectivity rates or natural death rates. Let $I_{i,k}, i = 1, \cdots, n$

denote the i-th infected individual with degree k, then

$$\begin{cases} \frac{dS_k}{dt} = \delta(1 - \sum_{i=1}^{n} I_{i,k} - S_k - R_k) - S_k k \sum_{i=1}^{n} \lambda_i \Theta_i - \alpha S_k \\ \frac{dI_{i,k}}{dt} = p_i S_k k \sum_{i=1}^{n} \lambda_i \Theta_i - (\beta_i + \mu_i) I_{i,k} \qquad i = 1, \cdots, n \\ \frac{dR_k}{dt} = \sum_{i=1}^{n} \mu_i I_{i,k} - \gamma R_k \end{cases}$$

Here the new infected individuals will come into the i-th infectivity individuals with probability p_i, so $\sum_{i=1}^{n} p_i = 1$. Other parameters are similar to those in Section 3.1, and

$$\Theta_i = \frac{\Sigma_k k p(k) I_{i,k}}{\langle k \rangle} \quad i = 1, \cdots, n$$

We have

$$\sum_{i=1}^{n} \frac{\lambda_i p_i}{\mu_i + \beta_i} > \frac{\langle k \rangle (\delta + \alpha)}{\langle k^2 \rangle \delta}$$

3.3. *Multiple-staged infected individuals*

As was discussed in,[56] each case of infected individuals can also develop in several stages. So we now discuss the multiple-staged infected individuals models. Let $I_{i,j}, i = 1, \cdots, n, j = 1, \cdots, m$ denote the i-th infected individual which is in the j-th stage.

In order to simplify the computation, we do not consider the natural death rate for $I_{i,j}, i = 1, \cdots, n, j = 1, \cdots, m$, but only suppose that they only go into R state with certain rates. Then the dynamics equations are:

$$\begin{cases} \frac{dS^{(k)}}{dt} = \delta(1 - \sum_{i=1}^{n} \sum_{j=1}^{m} I_{i,j}^{(k)} - S^{(k)} - R^{(k)}) - S^{(k)} k \sum_{i=1}^{n} \sum_{j=1}^{m} \lambda_{i,j} \Theta_{i,j} - \alpha S^{(k)} \\ \frac{dI_{i,1}^{(k)}}{dt} = p_i S^{(k)} k \sum_{i=1}^{n} \sum_{j=1}^{m} \lambda_{i,j} \Theta_{i,j} - \mu_{i,1} I_{i,1}^{(k)} \\ \frac{dI_{i,j}^{(k)}}{dt} = \mu_{i,j-1} I_{i,j-1}^{(k)} - \mu_{i,j} I_{i,j}^{(k)}, \quad i = 1, \cdots, n, \ j = 2, \cdots, m \\ \frac{dR^{(k)}}{dt} = \sum_{i=1}^{n} \mu_{i,m} I_{i,m}^{(k)} - \gamma R^{(k)} \end{cases}$$

Here the individuals' degree k is given as the superscripts to differentiate from the subscripts i, j. The infectivity rates for $I_{i,j}$ on susceptible individuals are $\lambda_{i,j}$, and $\mu_{i,j}$ are the rates of the transformation $I_{i,j} \to I_{i,j+1}, i = 1, \cdots, n, j = 1, \cdots, m-2$, and $\mu_{i,m}$ are the rates of the transformation $I_{i,m} \to R$. Here we suppose that each $I_{i,j}$ can infect susceptible individuals, and new infected individuals will come into the i-th infectivity individuals with probability p_i, so we also have $\sum_{i=1}^{n} p_i = 1$.

$\Theta_{i,j}$ are given by:

$$\Theta_{i,j} = \frac{\Sigma_k k p(k) I_{i,j}^{(k)}}{\langle k \rangle}, \quad i = 1, \cdots, n, j = 1, \cdots, m$$

Then the threshold for the multiple-staged infected model:

$$\sum_{i=1}^{n} \sum_{j=1}^{m} \frac{p_i}{\mu_{i,j}} \lambda_{i,j} > \frac{(\delta + \alpha)\langle k \rangle}{\delta \langle k^2 \rangle}$$

From the above three inequalities, we can obtain the relationship between thresholds of epidemic and the parameters, such as the degree distribution, birth rate, death rate, and so on. In particular, the thresholds for each case are zero when the size of network is sufficiently large, that is, $\langle k^2 \rangle = \Sigma_k k^2 p(k) \to \infty$.

4. SIS model with population mobility

Most previous research on epidemic spreading assumed that a node is an individual, as a result, the deeper structure of networks were neglected, such as the mobility of individuals between different cities was ignored. Most recently, Vittoria Colizza et al studied the behavior of two basic types of reaction-diffusion processes ($B \to A$ and $B + A \to 2B$),[28] they supposed that a node of the network can be occupied by any number of individuals and the individuals can diffuse along the link between nodes. The two basic reaction-diffusion processes can be used to model the spreading of epidemic diseases with SIS model.[28] In the epidemic terminology, a node can be viewed as a city, i.e, all people have the same degree k if they live in the same city (the node with degree k), and the diffusion of particles among different nodes can be considered as the movement of people among different cities. They supposed that the infection may happen inside a city, however, the infection may also happen in different cities by other media, e.g., for the Avian Influenza, in different regions poultry can be infected by migratory birds even though the poultry have no mobility.

We suppose that the infection can also happen in different cities, and study the effect of this kind of epidemic spreading on the epidemic threshold. This can be done by introducing a probability of spreading of the infection to the neighboring nodes without the need of diffusion of infected particles. In fact, as we will show, this mechanism is in part equivalent to the diffusion of the particles.

4.1. *Epidemic spreading without mobility of individuals*

In order to find out the effect of mobility of individuals, we first assume that mobility is zero, then

$$\begin{cases} \frac{dI_k(t)}{dt} = \alpha k S_k \Theta_i + \beta S_k I_k - \mu I_k \\ \frac{dS_k(t)}{dt} = -\alpha k' S_{k'} \Theta_i - \beta S_k I_k + \mu I_k \end{cases} \quad i = 1, 2$$

Here we should note that the total density $S_k + I_k$ is not changed because there is no mobility of individuals among different cities, so we can let $S_k + I_k = 1$. Then we have

$$\frac{\alpha}{\mu - \beta}\frac{\langle k^2\rangle}{\langle k\rangle} > 1$$

this condition demonstrates that epidemic diseases will always become endemic for a heterogeneous network with sufficiently large size.

For the case Θ_1, we can get

$$\frac{\alpha}{\mu - \beta} > 1$$

this case suggests that the epidemic threshold is irrelevant to the topology of the network, which is similar to the results in.[29,30]

In the following subsections, we take into account the mobility of individuals in different cities, so the individuals' degrees may change, that is, the total density $S_k + I_k$ is not an invariant, but the average density $n = \Sigma_k P(k)(S_k + I_k)$ is.

4.2. *Spreading of epidemic diseases among different cities*

Similar to,[28] we denote the size of the network as V, and N_S and N_I are the numbers of susceptible and infective individuals respectively, so the total number of individuals in the network is $N = N_S + N_I$ and $n = N/V$ is the average density of people. Because the number of individuals on each node is a random non-negative integer, set a_i and b_i as the numbers of S and I stores on node i. In order to take into account the heterogeneous quality of networks we have to explicitly consider the presence of nodes with very different degree k. A convenient representation of the system is therefore provided by the following quantities:

$$S_k = (\Sigma_{i|k_i=k} a_i)/v_k, \qquad I_k = (\Sigma_{i|k_i=k} b_i)/v_k$$

where v_k is the number of nodes with degree k and the sums run over all nodes i having degree k_i equal to k.

Just as in,[28] we also assume that the mobility of people is unitary time rate 1 along one of the links departing from the node in which they are at a given time. This implies that at each time step an individual occupying a node with degree k will travel to another city with probability $1/k$.

Now the dynamics of epidemic spreading can be described as follows:

$$\begin{cases} \frac{dI_k(t)}{dt} = -I_k(t) + k\Sigma_{k'} P(k'|k)\frac{1}{k'}[(1-\mu)I_{k'}(t) + \alpha k' S_{k'}\Theta_i] \\ \frac{dS_k(t)}{dt} = -S_k(t) + k\Sigma_{k'} P(k'|k)\frac{1}{k'}[S_k(t) + \mu I_{k'}(t) - \alpha k' S_{k'}\Theta_i] \end{cases} \qquad i = 1, 2$$

For the case of Θ_1, the threshold for the average density is

$$n_{c_1} = \frac{\mu\langle k\rangle^2}{\alpha\langle k^2\rangle}$$

For the case of Θ_2, the threshold is

$$n_{c_2} = \frac{\mu\langle k\rangle^3}{\alpha\langle k^2\rangle^2}$$

We conclude that the epidemic is always endemic for sufficiently large heterogeneous networks, moreover, the prevalence of epidemics with infection rate $\alpha k\Theta_2$ is greater than the infection rate $\alpha k\Theta_1$.

4.3. *Epidemic spreading within and between cities*

Now we assume that the epidemic disease not only occurs within individual cities but also between connected cities. And we also consider two types of epidemic spreadings inside the same cities. In the case of type 1, we consider that each a_i individuals may be infected by all the b_i individuals in the same cities, in this case, the epidemic rate is β when the spreading of the epidemic disease happen in the same cities. In the case of type 2, we consider that each individual has a finite number of contacts with others, in this case the epidemic rate has to be rescaled by the total number of individuals in city i, i.e., β/n_i is the epidemic rate in the same cities, where $n_i = a_i + b_i$ is the total number of individuals in the city i.

4.3.1. *The epidemic rate is β inside the same cities*

In this case, the number of infected individuals generated by the infection taking place in node of the degree class k is $\beta S_k I_k$. Let $T_k = S_k I_k$, we have

$$T = \Sigma_k P(k) T_k = \Sigma_k P(k) S_k I_k$$

Then the dynamics of epidemic spreading can be written as

$$\begin{cases} \frac{dI_k(t)}{dt} = -I_k(t) + k\Sigma_{k'} P(k'|k)\frac{1}{k'}[(1-\mu)I_{k'}(t) + \beta T_k + \alpha k' S_{k'}\Theta_i] \\ \frac{dS_k(t)}{dt} = -S_k(t) + k\Sigma_{k'} P(k'|k)\frac{1}{k'}[S_k(t) + \mu I_{k'}(t) - \beta T_k - \alpha k' S_{k'}\Theta_i] \end{cases} \qquad i = 1, 2$$

For the case of Θ_1, the threshold for the prevalence of epidemic is

$$n_{c_3} = \frac{\mu\langle k\rangle^2}{(\alpha+\beta)\langle k^2\rangle}$$

For the case of Θ_2, the threshold for the prevalence of epidemic disease is

$$n_{c_4} = \frac{\mu\langle k\rangle^3}{(\alpha\langle k^2\rangle + \beta\langle k\rangle)\langle k^2\rangle}$$

4.3.2. *The epidemic rate is β/n_i inside the same cities*

In this case, the number of infected individuals generated by the infection taking place in node of the degree class k is $\beta\frac{S_k I_k}{S_k+I_k}$, we also let $T_k = \frac{S_k I_k}{S_k+I_k}$.

We can obtain

$$T = \Sigma_k P(k) T_k = \Sigma_k P(k)\frac{S_k I_k}{S_k + I_k} = \frac{IS}{n}$$

For the Θ_1 case, the prevalence of epidemic disease takes place if $(\beta-\mu)\langle k\rangle^2 + \alpha n\langle k^2\rangle > 0$, i.e.,

$$n_{c_5} = \begin{cases} 0 & \beta/\mu > 1 \\ \frac{(\mu-\beta)\langle k\rangle^2}{\alpha\langle k^2\rangle} & \beta/\mu < 1 \end{cases}$$

For the Θ_2 case,

$$n_{c_6} = \begin{cases} 0 & \beta/\mu > 1 \\ \frac{(\mu-\beta)\langle k\rangle^3}{\alpha\langle k^2\rangle^2} & \beta/\mu < 1 \end{cases}$$

From the above equalities, we can find that the epidemic always occurs whatever the size of networks, when $\beta/\mu > 1$.

5. Multi-strain epidemic models

There are cases of multi-strain epidemics in the real world which we wish to examine, i.e., different strains of the same pathogen transmitting on the same network. For example, the human immunodeficiency virus (HIV) (which can cause AIDS) has many genetic varieties, and can be divided into some strains, such as strain HIV-1 and strain HIV-2.[31] On the one hand, being the same virus, there are many similarities between HIV-1 and HIV-2, such as the modes of HIV-1 and HIV-2 transmission are the same - sexual contact, sharing needles etc. On the other hand, there exist many differences between HIV-1 and HIV-2, for example, HIV-2 seems to weaken the immune system more slowly than HIV-1.

Some multi-strains epidemic dynamics problems on fully mixed species have been generally investigated in.[32–35] During disease spreading processes, a kind of pathogen sometime generates many strains with different spreading features, hence researches on multi-strain epidemic dynamics possess practical significance.

For multi-strain epidemic models, the manner of interaction between two particles with different strain have many types, including co-infection, which means two strains can host in one particle, invasion, which means one strain can host the particle with the other strain, and perfect cross-immunity, which means that two strains are perfectly competing and nodes infected with one strain cannot be infected by the other strain. Previous results have shown that different interaction mechanisms have different effects, such as co-infection, and can induce complex dynamical behaviors, such as chaotic attractors.[36] Super-infection may generate so-called strain replacement phenomenon,[35] but perfect cross-immunity can not produce similar phenomenon even with perfect vaccination (the vaccine provides full protection against all strains[35]). Strain replacement shows that the phenomenon that one strain with smaller basic reproduction number can become prevalent in a long time.

In this section, two-strain epidemic models on complex networks with scale-free connectivity is discussed. We assume that the network has saturated infectivity. The two kinds of strains of the same pathogen can be denoted by strain I and

strain J. It may be the case that the two strains have different spreading rates. Let the spreading rate of the strain I be λ_1, and that of the strain J be λ_2. Each individual is represented by a node of the network, and can be in three discrete states, either susceptible or infected by the strain I or J, which allows that infection mechanism to belong to the SIS type,[37] that is, susceptible nodes may be infected owing to contact with an infected node, and infected nodes also may recover into the susceptible state, the recovery rates are β_1 and β_2 for strain I and strain J respectively.

Let $i_k(t)$ and $j_k(t)$ represent the densities at time t of nodes in class with degree k infected by strain I and strain J respectively.

We will focus on two kinds of interaction mechanisms between two strains without co-infection, one is perfect cross-immunity, and the other is super-infection. Then the two-strain models can be written as follows:

Perfect cross-immunity mechanism:

$$\begin{cases} \frac{di_k(t)}{dt} = -\beta_1 i_k(t) + \lambda_1 k[1 - i_k(t) - j_k(t)]\Theta_1(t) \\ \frac{dj_k(t)}{dt} = -\beta_2 j_k(t) + \lambda_2 k[1 - i_k(t) - j_k(t)]\Theta_2(t) \end{cases} \tag{5.1}$$

where the probability $0 \leqslant \Theta_1(t) \leqslant 1$ describes a link pointing to an individual infected by the strain I. According to,[45] it satisfies the equality

$$\Theta_1(t) = \sum_{k'} \frac{P(k'|k)}{k'} \varphi(k') i_{k'}(t) \tag{5.2}$$

Similarly, the probability $0 \leqslant \Theta_2(t) \leqslant 1$ describes a link pointing to an individual infected by the strain J, which satisfies the equality

$$\Theta_2(t) = \sum_{k'} \frac{P(k'|k)}{k'} \varphi(k') j_{k'}(t) \tag{5.3}$$

Those nodes with degree k have saturated infectivity, $\varphi(k)$, which satisfies the following three conditions:

(i) $\varphi(k) \leqslant k$; (ii) $\varphi(k)$ is monotonously increasing; (iii) $\lim\limits_{k\to\infty} \varphi(k) = A > 0$.

According to the physical meaning of $\varphi(k)$, the above constraints are reasonable and recover some previous setting about the function. It can be shown that the piecewise linear infectivity introduced in[45] is a special case for the saturated infectivity defined above.

Super-infection mechanism:

For this mechanism, we assume that when nodes infected by the strain I contact nodes infected by strain J they may be reinfected by strain I. The transmission process is asymmetric, that is, strain J cannot reinfect the node infected by strain I. This process is referred to as super-infection. And the asymmetric transmission rate is denoted by δ.

Similar to the perfect cross-immunity mechanism, the two-strain model with super-infection can be described as follows:

$$\begin{cases} \frac{di_k(t)}{dt} = -\beta_1 i_k(t) + \lambda_1 k[1 - i_k(t) - j_k(t)]\Theta_1(t) + \delta k j_k(t)\Theta_1(t) \\ \frac{dj_k(t)}{dt} = -\beta_2 j_k(t) + \lambda_2 k[1 - i_k(t) - j_k(t)]\Theta_2(t) - \delta k j_k(t)\Theta_1(t) \end{cases} \tag{5.4}$$

Now we present detailed analysis on the above models.

5.1. *The two-strain epidemic model with perfect cross-immunity*

Assume the network is uncorrelated about node degree. Therefore

$$P(k'|k) = \frac{k'P(k')}{< k >},$$

so the model (5.1) can be transformed into

$$\begin{cases} \frac{di_k(t)}{dt} = -\beta_1 i_k(t) + \lambda_1 k[1 - i_k(t) - j_k(t)]\Theta_1(t) \\ \frac{dj_k(t)}{dt} = -\beta_2 j_k(t) + \lambda_2 k[1 - i_k(t) - j_k(t)]\Theta_2(t) \end{cases} \tag{5.5}$$

where

$$\Theta_1(t) = \frac{\sum_{k'} \varphi(k')P(k')i_{k'}(t)}{\langle k \rangle} \tag{5.6}$$

and

$$\Theta_2(t) = \frac{\sum_{k'} \varphi(k')P(k')j_{k'}(t)}{\langle k \rangle} \tag{5.7}$$

In the above model, we can see that the system (5.5) has four parameters, which can be reduced to three if using typical time-scale transformation. So this multi-parameters property invokes some different dynamical behaviors, which are different from the case of one strain epidemic. In the following analysis, we find that composed parameter $\sigma_i = \frac{\lambda_i}{\beta_i}, i = 1, 2$ are important, which is referred to as the effective spreading rates for strain I and strain J respectively. It is reasonable to assume that $\sigma_1 \neq \sigma_2$. But the recovery rates β_1, β_2 can not be all eliminated. Hence, the case $\sigma_1 = \sigma_2$ will also be discussed.

5.2. *The case $\sigma_1 \neq \sigma_2$*

5.2.1. *Basic Reproduction Numbers (BRNs)*

In order to obtain the existence of non-trivial equilibria, we define the following two parameters

$$R_1 = \frac{\sigma_1 \langle k\varphi(k) \rangle}{\langle k \rangle}, \qquad R_2 = \frac{\sigma_2 \langle k\varphi(k) \rangle}{\langle k \rangle} \tag{5.8}$$

They are the basic reproduction numbers for the strain I and the strain J respectively. The number R_1 gives the average value of secondary infectious cases

produced by the infected individual with strain I during the entire infectious period in a purely susceptible population. The number R_2 has similar meaning. The two BRNs are related by the effective spreading rates. If $\sigma_1 = \sigma_2$, then $R_1 = R_2$. Otherwise, they are different.

Below we list some basic results:

A1. There is always a disease-free equilibrium $E_0 = (1, 0, 0)$;

A2. There is a strain one exclusive equilibrium $E_1 = (s_1^*, i^*, 0)$, if and only if $R_1 > 1$;

A3. There is a strain two exclusive equilibrium $E_2 = (s_2^*, 0, j^*)$, if and only if $R_2 > 1$.

In fact, by imposing steady state $\frac{di_k(t)}{dt} = 0$ and $\frac{dj_k(t)}{dt} = 0$, from (5.5) we have

$$i_k = \frac{\sigma_1 k\Theta_1}{1 + \sigma_1 k\Theta_1 + \sigma_2 k\Theta_2}, \qquad j_k = \frac{\sigma_2 k\Theta_2}{1 + \sigma_1 k\Theta_1 + \sigma_2 k\Theta_2}. \tag{5.9}$$

Substitute (5.9) into (5.6) and (5.7), we can get two self-consistent equations as follows:

$$\Theta_1 = \frac{\sigma_1}{\langle k\rangle} \sum_{k'} \frac{k'\varphi(k')P(k')\Theta_1}{1 + \sigma_1 k'\Theta_1 + \sigma_2 k'\Theta_2} \tag{5.10}$$

and

$$\Theta_2 = \frac{\sigma_2}{\langle k\rangle} \sum_{k'} \frac{k'\varphi(k')P(k')\Theta_2}{1 + \sigma_1 k'\Theta_1 + \sigma_2 k'\Theta_2} \tag{5.11}$$

Obviously, $(\Theta_1, \Theta_2) = (0, 0)$ is a trivial solution of equations (5.10) and (5.11). Since $\sigma_1 \neq \sigma_2$, it can be shown that the equations have no positive solutions. So we need only to consider the other two cases, $\Theta_1 = 0$ and $\Theta_2 = 0$. When $\Theta_2 = 0$, we can only focus on nontrivial solutions of (5.10). Note that (5.10) can be reduced to

$$1 = \frac{\sigma_1}{\langle k\rangle} \sum_{k'} \frac{k'\varphi(k')P(k')}{1 + \sigma_1 k'\Theta_1} \equiv f(\Theta_1). \tag{5.12}$$

Because

$$f(1) < 1$$

and

$$f'(\Theta_1) < 0,$$

a nontrivial solution of (5.12) exists if and only if

$$f(0) > 1 \tag{5.13}$$

So we have $R_1 = f(0)$, i.e., $R_1 = \frac{\sigma_1 <k\varphi(k)>}{<k>} = \frac{\lambda_1 <k\varphi(k)>}{\beta_1 <k>}$. Similarly, by letting $\Theta_1 = 0$, we can get the above result about R_2.

5.2.2. *Asymptotic stability of equilibria*

Now we examine the asymptotic property of the equilibria E_0, E_1 and E_2.

We rewrite (5.5) as the following matrix form

$$\frac{dU(t)}{dt} = A * U - N(U) \tag{5.14}$$

According to block property of matrices, the zero solution E_0 is locally asymptotically stable, if and only if $R_1 \leq 1$ and $R_2 \leq 1$.

Perturbing the steady state i_k^* so that $i_k = \varepsilon_k + i_k^*$ and omitting higher powers of ϵ_k gives the linearization matrix, which determines the stable state.

It can be shown that the condition $R_1 < 1$ cannot ensure the local stability of E_1. To make E_1 stable, $R_2 < R_1$ must hold.

According to symmetry, we have that E_2 is locally stable if and only if $R_1 < R_2$.

When $R_1 < 1$ or $R_2 < 1$ holds, there are no more than two equilibria, system (5.5) is globally stable.

5.2.3. *Invasion Reproduction Numbers(IRNs)*

The IRNs can be written as below:

$$R_3 = \frac{\lambda_2\beta_2}{\lambda_1\beta_1} = \frac{R_2}{R_1}, \qquad R_4 = \frac{\lambda_1\beta_1}{\lambda_2\beta_2} = \frac{R_1}{R_2}. \tag{5.15}$$

According to biological interpretation,[32] R_3, the IRN of strain one can be considered as the number of secondary cases that one infected individual will produce in a population where strain two is at equilibrium and measures the invasion capability of strain one. Similarly, R_4 is the measure for strain two.

If $R_3 > 1$, the strain I spread eventually, but the other strain J cannot spread. If $R_4 > 1$ (e.g., $R_3 < 1$), the strain I cannot spread eventually. As for $R_3 = R_4 = 1$, it can be shown numerically that it is a very special case (we omit the details here).

While $R_3 \ll 1$ (that is $R_4 \gg 1$), the proportion eventually infected by the strain I is not affected by the strain J. In other words, however large the spreading rate of the strain J is, the eventually infected proportion is the same, and it is determined by R_1, that is, the spreading rate λ_1, the recovery rate β_1 and network structure. Of course, when $R_3 \gg 1$ (that is $R_4 \ll 1$), a corresponding result can be also obtained. So we can study only one strain with a much bigger spreading rate, thus the two strains model can be reduced to the one strain model, which is referred as *strain dominance*.

But when the invasion reproduction numbers are close to 1, we prefer to use the two strains model (5.5) to obtain accurate results about its dynamical behavior for prediction epidemic spreading and optimal containment strategies.

5.2.4. *Uniform immunization strategy*

According to,[38] uniform immunization (or random immunization) strategy is an effective immunization strategy. A selective uniform immunization is proposed to immunize individuals who may be infected by the epidemic with greater spreading rate. If $R_1 > R_2$, one can approximatively use $\lambda_1(1-\varepsilon)$ to substitute λ_1. ε denotes the immunized proportion or immunized rate. The immunized system is as follows:

$$\begin{cases} \frac{di_k(t)}{dt} = -\beta_1 i_k(t) + \lambda_1(1-\varepsilon)k[1-i_k(t)-j_k(t)]\Theta_1(t) \\ \frac{dj_k(t)}{dt} = -\beta_2 j_k(t) + \lambda_2 k[1-i_k(t)-j_k(t)]\Theta_2(t) \end{cases} \tag{5.16}$$

Note the above system is just the same as (5.5) if we regard $\lambda_1(1-\varepsilon)$ as a new λ_1. So the BRN for strain J is still R_2, while the BRN for strain I can be written as

$$\hat{R}_{1c} = R_1(1-\varepsilon) < R_1.$$

Therefore, one know that if the immunization rate ε satisfies

$$R_1(1-\varepsilon) < R_2,$$

the strain I evolves from spreading to eliminating eventually. Further, if $1 < R_2 < R_1$, the strain J varies from eliminating to spreading eventually. This alternate spreading phenomenon is actually strain replacement, which is useful to control epidemic outbreak by immunization.[32]

5.3. *The case $\sigma_1 = \sigma_2$*

For the case $\sigma_1 \neq \sigma_2$ we give a relatively complete analysis, however we still cannot confirm the existence of positive equilibria or the coexistence of two strains. Now we turn to consider the other case, that is, $\sigma_1 = \sigma_2 = \sigma$. In this case, the self-consistent equations still hold. We can find the equilibrium solutions, which can be divided into two cases:

B1. When $R \leq 1$, there is disease-free equilibrium $E_0' = (1, 0, 0)$;

B2. When $R > 1$, there are infinitely many equilibria, the parameter points (Θ_1, Θ_2) of the equilibria form a line segment which connects two endpoints: $(0, \bar{\Theta})$ and $(\bar{\Theta}, 0)$, where $\bar{\Theta}$ satisfies

$$1 = \frac{\sigma}{\langle k \rangle} \sum_{k'} \frac{k'\varphi(k')P(k')}{1+\sigma k'\bar{\Theta}}. \tag{5.17}$$

At the level of the equilibrium solution, Θ_1, Θ_2 and i_k, j_k are in one-to-one correspondence. So we can transform the above continuous problem to discrete mapping problem.

The self-consistent mapping:

$$F : x \to \frac{\sigma x}{\langle k \rangle} \sum_{k'} \frac{k'\varphi(k')P(k')}{1+\sigma k'x}$$

The mapping F has the following properties:

(a) There is always the zero solution, that is, $F(0) = 0$ holds. Further, when $R \leq 1$, $x = 0$ is locally stable.

(b) When $R > 1$, there is always a non-zero solution, $x = \bar{x}$, such that $F(\bar{x}) = \bar{x}$ holds. And $x = \bar{x}$ is also locally stable.

We have the following stability results for equilibria:

C1. When $R \leq 1$, the disease-free equilibrium $E_0' = (1, 0, 0)$ is locally asymptotically stable.

C2. When $R > 1$, all non-zero equilibria are locally asymptotically stable; but the zero solution is unstable.

5.4. *The two-strain epidemic model with super-infection*

5.4.1. *The model*

Super-infection is the concurrent or subsequent multiple infection of a host with the same parasite, which may be with identical or different strains.[34] Now we only consider the latter case, that is, super-infection only occurs between different strains. Similar to the case with perfect cross-immunity, we focus on the uncorrelated networks with super-infection mechanism

$$\begin{cases} \frac{di_k(t)}{dt} = -\beta_1 i_k(t) + \lambda_1 k[1 - i_k(t) - j_k(t)]\Theta_1(t) + \delta k j_k(t)\Theta_1(t) \\ \frac{dj_k(t)}{dt} = -\beta_2 j_k(t) + \lambda_2 k[1 - i_k(t) - j_k(t)]\Theta_2(t) - \delta k j_k(t)\Theta_1(t) \end{cases} \tag{5.18}$$

$(\Theta_1, \Theta_2) = (0, 0)$ is a trivial equilibrium. So there is always disease-free equilibrium K_0 (the zero solution) for the system (5.18). Omitting the hight order terms, we confirm that K_0 is locally asymptotically stable when $R_1 \leq 1, R_2 \leq 1$.

We now discuss the nontrivial equilibria. Assuming $\Theta_1 \neq 0, \Theta_2 = 0$, then when $R_1 > 1$, there exists the strain one exclusive equilibrium K_1.

Secondly, assuming $\Theta_2 \neq 0, \Theta_1 = 0$, then there exists the strain two exclusive equilibrium K_2, if and only if $R_2 > 1$.

From the above analysis, we conclude that the two basic reproduction numbers are the same as the case with perfect cross-immunity.

Finally, assuming that $\Theta_2 \neq 0, \Theta_1 \neq 0$, in order to find the positive solutions, we need to discuss the equations determining two implicit functions, which are not easy to be solved. We can obtain that when $R_2 \leq R_1$, there is no positive solutions for the system under consideration. So in what follow, we assume $H : R_2 > R_1$.

To simplify our discussion, we assume that $\beta_1 = \beta_2 = 1$.

Case 1: $0 < p_r \leq 1$ and $0 < p_b \leq 1$;

Since $R_2 > R_1$ and $\beta_1 = \beta_2 = 1$, we get $\lambda_2 > \lambda_1$. So $g'(\Theta_1) < 0$. We have the IRN for positive epidemic state:

$$R_5 = g(0) + g(p_b) - g(0)g(p_b).$$

When $R_5 > 1$, there exists one positive epidemic state for the system.

Case 2: $0 < p_r \leq 1$ and $p_b > 1$;
Similar to the Case 1, we have the IRN:

$$R_6 = g(0) + g(1) - g(0)g(1).$$

Case 3: $p_r > 1$ and $0 < p_b \leq 1$;
The IRN is

$$R_7 = g(p_t) + g(p_b) - g(p_t)g(p_b).$$

Case 4: $p_r > 1$ and $p_b > 1$.
The corresponding IRN can be written as

$$R_8 = g(p_t) + g(1) - g(p_t)g(1).$$

Finally, we remark here that for the case $\beta_1 \neq \beta_2$, theoretical analysis would be quite difficult. We leave this for future research.

At the moment the type A H1N1 influenza has become a pandemic, and the threat of future outbreaks of other emerging diseases or of a human-transmissible version of the H5N1 avian influenza still remain. The problem of how best to respond to disease transmission on a network currently remains unaddressed. And many problems need to be further studied by using the method presented in this paper. Most new approaches are needed for more realistic situations, e.g., on a directed network we may need to distinguish degree distribution between in-degrees and out-degrees, as the infectivity and immunization scheme choice will depend on these quantities. More precisely, in a directed network, the infectivity will depend on out-degree distribution, while the choice of immunization scheme will depend on in-degree distribution.

References

1. R. Albert and A-L. Barabási, Statistical mechanics of complex netowrks. *Reviews of Modern Physics*. **74** (2002) 47-97.
2. F. Brauer. The Kermack-McKendrick epidemic model revisited. *Math. Biosci*. **198** (2005) 119-131.
3. M. Barthelémy, A. Barrat, R. Pastor-Satorras, and A. Vespignani, Velocity and hierarchical spread of epidemic outbreaks in scale-free networks. *Phys. Rev. Lett.*, **92** (2004) 178701.
4. M. Boguñá, R. Pastor-Satorras, and A. Vespignani, Absence of epidemic threshold in scale-free networks with degree correlations. *Phys. Rev. Lett.* **90** (2003) 028701.
5. D.S. Callaway, M.E.J. Newman, S.H. Strogatz, and D.J. Watts, Network robustness and fragility: percolation on random graphs. *Phys. Rev. Lett.* **85** (2000) 5468-5471.
6. R. Cohen, K. Erez, D. ben-Avraham, and S. Havlin, Resilience of the Internet to random breakdowns. *Phys. Rev. Lett.* **85** (2000) 4626-4628.
7. R. Cohen, S. Havlin, and D. ben-Avraham, Efficient immunization strategies for computer networks and populations. *Phys. Rev. Lett.*, **91** (2003) 24791.
8. M. L. Goldstein, S. A. Morris and G. G. Yen, Problems with fitting to the power-law distribution. *Eur. Phys. J. B.* **41** (2004) 255-258.

9. A.N. Hill, I.M. Longini Jr. The critical vaccination fraction for heterogeneous epidemic models. *Math. Biosci.* **181** (2003) 85-106.
10. J. Joo, J.L. Lebowitz, Behavior of susceptible-infected-susceptible epidemics on heterogeneous networks with saturation. Preprint, 2006.
11. N. Madar, T. Kalisky, R. Cohen, D. ben-Avraham, and S. Havlin, Immunization and epidemic dynamics in complex networks. *Eur. Phys. J.*, **B 38** (2004) 269C276.
12. W.O. Kermack and A.G. McKendrick, Proc. Roy. Soc., **A 115** (1927) 700-721.
13. J.Z. Liu, et al, The spread of disease with birth and death on networks. J. Stat. Mech., (2004) P08008.
14. R.M. May and A.L. Lloyd, Phys. Rev. E, **64** (2001) 066112.
15. R.M. May and A.L. Lloyd, Infection dynamics on scale-free networks. *Phys. Rev*, **E 65** (2002) 035108.
16. M.E.J. Newman, Phys. Rev. E, **66** (2002) 016128.
17. R. Pastor-Satorras and A. Vespignani, Epidemic dynamics and endemic states in complex networks. *Phys. Rev.*, **E 63** (2001) 066117.
18. R. Pastor-Satorras and A. Vespignani, Epidemic spreading in scale-free networks. *Phys. Rev. Lett.*, **86** (2001) 3200-3203.
19. R. Pastor-Satorras and A. Vespignani, Immunization of complex networks. *Phys. Rev.*, **E 65** (2002) 036104.
20. R. Pastor-Satorras and A. Vespignani, Epidemic dynamics in finite size scale-free networks. *Phys. Rev.*, **E 65** (2002) 035108.
21. M. Small, P. Shi and C.K. Tse. Plausible models for propagation of the SARS virus. *IEICE Trans. on Fundamentals of Electr., Commun. and Comput. Sci.*, **E87-A** (2004) 2379-2386.
22. M. Small and C.K. Tse. Small world and scale free model of transmission of SARS. *Int. J. of Bifurcat. and Chaos*, **15** (2005) 1745-1755.
23. M. Small and C.K. Tse, Clustering model for transmission of the SARS virus: application to epidemic control and risk assessment. *Physica A*, **351** (2005) 499-511.
24. M. Small, C.K. Tse and D. Walker. Super-spreaders and the rate of transmission of the SARS virus. *Physica D*, **215** (2006) 146-158.
25. M. Small, D.M. Walker and C.K. Tse, Scale free distribution of avian influenza outbreaks. Phys. Rev. Lett., **99** (2007) 188702.
26. R. Yang, J. Ren, W.-J. Bai, T. Zhou, M.-F. Zhang, B.-H. Wang, Epidemic spreading and immunization with identical infectivity. (2006) arXiv: physics/0611095.
27. T. Zhou, J.-G. Liu, W.-J. Bai, G.R. Chen, and B.-H. Wang, Behaviors of susceptible-infected epidemics on scale-free networks with identical infectivity. *Phys. Rev. E* **74** (2006) 056109.
28. V. Colizza, R. Pastor-Satorras, A. Vespignani, Reaction-diffusion processes and metapopulation models in heterogeneous networks. *Nature Physics*, **3** (2007) 276.
29. T. Zhou, J.-G. Liu, et al, Behaviors of susceptible-infected epidemics on scale-free networks with identical infectivity. Phys. Rev. E, **74** (2006) 056109.
30. R. Yang, B.-H.Wang, et al, Epidemic spreading on heterogeneous networks with identical infectivity. Phys. Lett. A, **364** (2007) 189.
31. M. Balter, New HIV strain could pose health threat. *Science*, **281** (5382) (1998) 1425-1426.
32. D.H. Thomasey, M.Martcheva, Serotype replacement of vertically transmitted disease through perfect vaccination. *J. Bio. Sys.*, **16** (2) (2008) 255-277.
33. M. NuNo, Z.Feng, M. Martcheva, and C. Castillo-Chavez, Dynamics of two-strain influenza with isolation and partial cross-immunity. *SIAM J. Appl. Math.*, **65** (3) (2005) 964-982.

34. L.M.Cai, X.Z.Li, J.Y. Yu, A two-strain epidemic model with super infection and vaccination. *Math. Appl.*, **20** (2007) 328-335.
35. M. Martcheva, T.R. Horst, Progression age enhanced backward bifurcation in an epidemic model with infection and perfect vaccination. *Math. Biosci.*, **195** (2005) 23-46.
36. M.Kamo, A. Sasaki, the effect of cross-immunity and seasonal forcing in a multi-strain epidemic model. *Physica D*, **165** (2002) 228-241.
37. F. Brauer, The Kermack-McKendrick epidemic model revisited. *Math. Biosci.*, **198** (2005) 119-131.
38. N. Madar, T. Kalisky, R. Cohen, D. ben-Avrham and S. Havlin, Immunization and epidemic dynamics in complex networks. *Eur. Phys. J.* **B 38** (2004) 269-276.
39. J.-Z. Wang, Z.-R. Liu, J.-H. Xu, Epidemic spreading on uncorrelated heterogeneous networks with non-uniform transmission. *Physica A*, **382** (2007) 715-721.
40. Jiazeng Wang and Zengrong Liu, A Markovia model of the susceptible-infected-removed spreading in networks. Preprint.
41. Jiazeng Wang and Zengrong Liu, Mean-field level analysis of epidemics in directed networks. Preprint.
42. Hai-Feng Zhang, Xin-Chu Fu, The SIR model's epidemic dynamics on complex networks with the effect of immunization. *J. Shanghai Univ. Nat. Sci.*, **13** (2) (2007) 189-192. (in Chinese)
43. M. Small, D.M. Walker, C.K. Tse and X.C. Fu, Scale free distribution of avian influenza outbreaks. 2007 China Symposium on Circuits and Systems, (Guangzhou, China, 15-17 June, 2007). Special Session (in Chinese).
44. Haifeng Zhang, Michael Small and Xinchu Fu, Global behavior of epidemic transmission on heterogeneous networks via two distinct routes. *Nonlinear Biomedical Physics*, 2008, 2:2, DOI: 10.1186/1753-4631-2-2)
45. Xinchu Fu, Michael Small, David M. Walker, Haifeng Zhang, Epidemic dynamics on scale-free networks with piecewise linear infectivity and immunization. *Physical Review E*, **77** (2008) 036113.
46. M. Small, C.K. Tse and X.C. Fu, Transmission of infectious agents on networks. In: Análisis No Lineal De Series Temporales, ed. Grupo de Investigación Interdisciplinar en Sistemas Dinámicos, 2008. In press.
47. Haifeng Zhang and Xinchu Fu, Spreading of epidemics on scale-free networks with nonlinear infectivity. *Nonlinear Analysis TMA*, **70** (2009) 3273-3278.
48. Hai-Feng Zhang, Ke-Zan Li, Xin-Chu Fu and Bing-Hong Wang, An effcient control strategy of epidemic spreading on scale-free networks. *Chinese Physics Letters*, **26** (2009) 068901.
49. Kezan Li and Xinchu Fu, The hybrid models combining complex dynamical networks and epidemic networks. *Draft*, 2009.
50. Ying-Ying Shao and Xin-Chu Fu, The SEIR model with immunization delays. *Draft*, 2009.
51. Qingchu Wu, Xinchu Fu, Estimating method of epidemic threshold value on SW networks. *Preprint*, 2009.
52. Qingchu Wu, Xinchu Fu, et al, Responsive immunizations and epidemic outbreaks in correlated complex networks. *Preprint*, 2009.
53. Qingchu Wu, Xinchu Fu, Zengrong Liu and Michael Small, Two-strain epidemic models on scale-free networks with saturated infectivity. *Preprint*, 2009.
54. Qingchu Wu and Xinchu Fu, A mixed epidemic model on scale-free networks with saturated infectivity. *Preprint*, 2009.
55. Hai-Feng Zhang, Michael Small, Xin-Chu Fu and Bing-Hong Wang, Dynamical be-

havior of SIS model on heterogeneous networks with population mobility. *Chinese Physics B*, **18** (2009) 3633-3640.

56. Haifeng Zhang, Michael Small and Xinchu Fu, Staged progression model for epidemic spread on homogeneous and heterogeneous networks. To appear in *J. of Syst. Sci. and Complexity*. (Accepted in May 2009)
57. Kezan Li, Michael Small, Haifeng Zhang and Xinchu Fu, Epidemic outbreaks on networks with effective contacts. *Nonlinear Anal. Real World Appl.* (In press. DOI: 10.1016/j.nonrwa.2009.01.046)
58. Haifeng Zhang, Michael Small and Xinchu Fu, Different epidemic models on scale-free networks. *Commun. in Theoret. Phys.*, **52** (2009) 180-184.
59. Ying-Ying Shao, Meng Liu, and Xin-Chu Fu, Epidemic dynamics of SIR model with piecewise linear infectivity and immunizations on scale-free networks. To appear in *J. of Dynamics and Control*, (Accepted in Sept. 2008) (in Chinese)