Multiple epidemic waves in delayed susceptible-infected-recovered models on complex networks

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We consider a delayed susceptible-infected-recovered epidemic model on an uncorrelated complex network and address the effect of time lag on the shape and multiple waves of epidemic curves. We show that when the transmission rate is above a threshold, a large delay can cause multiple waves with larger amplitudes in the second and subsequent waves.

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I. INTRODUCTION

The role of network heterogeneity in spreading an infectious disease has received substantial attention partially due to the need for understanding the transmission dynamics of human sexually transmitted infection (STI) through the complex web of sexual partnerships [1,2]. The population involved in an STI can normally be stratified according to their levels of sexual activity and their numbers of sexual contacts. The network heterogeneity is associated with average path lengths among any two nodes [3,4] and can sometimes be described by a power-law distribution (scale-free property), $P(k) \sim k^{-2-\gamma}$, for the probability that a given node has k connections to other nodes [5,6].

In the intensively studied susceptible-infected-recovered (SIR) models on complex networks (see, for example, Refs. [7,8] for susceptible-infected-susceptible and [9–12] for SIR), the population is divided into three classes depending on the disease status of individuals: susceptible, infected and infectious, and removed (either immunized or dead). In such a modeling framework, every node of the network is also associated with an integer to characterize its connection to other nodes, so we can let $S_k(t)$, $I_k(t)$ and $R_k(t)$ be the densities of susceptible, infected and infectious, and removed individuals with degree k at time t, respectively. We then have the normalization condition:

$$S_k(t) + I_k(t) + R_k(t) = 1.$$

Such global quantities as epidemic prevalence can be expressed by an average over the various degree classes. Mean-field theory can then be used to derive the following deterministic system (see Moreno *et al.* [10] and Yang *et al.* [12])

$$\begin{aligned} \tfrac{d}{dt} S_k(t) &= -\lambda k S_k(t)\Theta_k(t), \\ \tfrac{d}{dt} I_k(t) &= -\mu I_k(t) + \lambda k S_k(t)\Theta_k(t), \\ \tfrac{d}{dt} R_k(t) &= \mu I_k(t), \end{aligned} \tag{1}$$

where $\Theta_k(t) = \sum_h h^{-1}\varphi(h)P(h|k)I_h(t)$, $\varphi(h)$ denotes the infectivity of a node with degree h, and $P(h|k)$ denotes the conditional probability for a node with degree k to connect a node with degree h. In the uncorrelated case, one has $P(h|k) = hP(h)/\langle k\rangle$. In the above formulation, a susceptible individual acquires the infection per unit time at the transmission rate λ by one contact with a neighboring infected individual. Thus if a susceptible individual has an edge connecting to an infected individual, the disease will be transmitted to the susceptible through the edge with the rate λ. On the other hand, the infected ones will recover or become immune with the rate μ. In what follows, we set $\mu = 1$ without loss of generality.

Two special cases of Eq. (1) were considered by Moreno *et al.* and Yang *et al.*. For both cases, the networks are uncorrelated. In Moreno *et al.* [10], the case where $\varphi(h) = h$ was considered and an epidemic threshold $\lambda_c = \langle k\rangle/\langle k^2\rangle$ was introduced. It was shown that when $\lambda < \lambda_c$, the total number of recovered individuals is small enough and the epidemic prevalence is insignificant; whereas when $\lambda > \lambda_c$, the total number of recovered individuals attains a finite value and the epidemic prevalence is significant. In Ref. [12], Yang *et al.* considered the case where $\varphi(h) = B$ is a constant, and they found the threshold $\lambda_c = 1/\langle B\rangle$ and similar qualitative observations as those in Moreno *et al.* One of the contributions of the present paper is to extend the aforementioned studies to a general function $\varphi(h)$ and, in addition, to show that the force of infection $\sum_h \varphi(h)P(h)I_h(t)$ has only one peak if λ is larger than a threshold λ_c to be defined later, and this force of infection has no peak and decreases very fast when $\lambda < \lambda_c$.

Our main focus, however, is on how the incorporation of a constant time lag (an incubation period) into the model alters the aforementioned qualitative results. Although our focus here is on incubation period, we note that in disease transmission models, time delay is an important quantity for many epidemiological mechanisms. In particular, time delays can be introduced to model constant sojourn times in an infective state. We refer to van den Driessche [13] for a brief review of delay differential equations arising from disease modeling. Delayed SIR models have been extensively studied (see, for example, Refs. [14–35]). However, to our best knowledge, little has been done to address the effect of

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delay on disease transmission dynamics in scale-free and other complex networks.

When the incubation period is assumed to be a constant $\tau > 0$, we have a delayed SIR model on networks in the uncorrelated case as follows:

$$\frac{d}{dt}S_k(t) = -\lambda k S_k(t)\sum_h \frac{\varphi(h)P(h)I_h(t-\tau)}{\langle k\rangle}, \tag{2}$$

$$\frac{d}{dt}I_k(t) = \lambda k S_k(t)\sum_h \frac{\varphi(h)P(h)I_h(t-\tau)}{\langle k\rangle} - I_k(t), \tag{3}$$

$$\frac{d}{dt}R_k(t) = I_k(t). \tag{4}$$

Note that, even for the case without delay, it is difficult to obtain analytic results for epidemic models on correlated networks. For such models, only numerical simulations are done (see, for example, Refs. [38–41]). As a result, for the sake of simplicity, we focus on system (2)–(4) in this paper. To specify a solution of the system, we have to specify the initial conditions, which are assumed to be

$$\text{for any degree } k, \begin{cases} S_k(\theta) > 0 \\ I_k(\theta) \geqslant 0 \\ R_k(\theta) = 0 \end{cases} \quad \text{for} \quad \theta \in [-\tau, 0], \tag{5}$$

with $I_k(\theta) > 0$ for some k and some $\theta \in [-\tau,0]$. Note that the global averages of the three epidemic classes defined by $S(t) = \sum_k P(k)S_k(t)$, $I(t) = \sum_k P(k)I_k(t)$, $R(t) = \sum_k P(k)R_k(t)$ obey the normalization condition: $S(t) + I(t) + R(t) = 1$. We will compare our results with those in Refs. [10] and [12] when delay is ignored. It turns out that the incorporation of delay changes the dynamics qualitatively.

For the delayed SIR models in the above-cited references with the exception of Ref. [16], the demographics (death and birth) is incorporated and results obtained are similar, namely there is always a disease-free equilibrium: If the basic reproduction number is less than 1, then there is no endemic equilibrium and the disease-free equilibrium is locally or globally stable. If the basic reproduction number is larger than 1, then the disease-free equilibrium becomes unstable, and there is a unique endemic equilibrium. In most cases, the endemic equilibrium is locally or globally stable for all delays, whereas in some cases it is locally or globally stable only for small delay and Hopf bifurcation of periodic solutions may occur when the delay passes a certain critical value [17,19,20,23,25,29,35]. However, in our model any points $(S_1,0,R_1,\ldots,S_k,0,R_k,\ldots)$ with $S_k + R_k = 1$ can be an equilibrium and hence any equilibrium is unstable. To understand the limiting behavior of each solution, we first, in Sec. II A, establish the final size equation that enables us to calculate the total number of recovered during the entire course of the outbreak regardless of the transmission rate (whether there is an outbreak or not) as long as there is an initial infection. We mention that Di Liddo [16] considered the following delayed SIR model

$$\begin{aligned} \dot{S}(t) &= -\beta S(t)I(t-a), \\ \dot{I}(t) &= \beta S(t)I(t-a) - \gamma I(t), \\ \dot{R}(t) &= \gamma I(t), \end{aligned}$$

and also established result on the final size of R. Note that our model (1) is simply the counterpart of the Di Liddo model in the framework of spread on a network rather than a homogeneous population.

Recent studies have provided empirical evidence of epidemic waves in infectious diseases like the Spanish influenza of 1918–1919 (see, for example, Refs. [36,37] and references therein). Peak detection is very necessary for health resource planning. Possible explanations of epidemic waves are summarized in Merler *et al.* [37]. Here, we present another possible explanation, that is, delay induces epidemic waves. The numerical simulations in Ref. [16] have already indicated that $I(t)$ may have multiple waves (i.e., multiple local maxima) during the process. Unfortunately, no rigorous analysis was provided even in the simple case with homogeneous populations. We, in Sec. II B, introduce a threshold λ_c and consider the monotonicity of the force of infection $\sum_h \varphi(h)P(h)I_h(t)$ and show that outbreak occurs when $\lambda > \lambda_c$ whether or not delay is incorporated. An argument is provided to show that this force of infection changes its monotonicity only once when the delay is small, but multiple peaks with increasing amplitudes can take place if the delay is large. This theoretical result will be illustrated with two special cases about the infectivity and with some numerical simulations in Sec. III. Our study thus confirms both theoretically and numerically that multiple peaks can arise very naturally due to the introduction of time delay in simple SIR models, and the value of the delay for such multiple peaks to occur and the magnitude of a subsequent wave of outbreak rely on the interplay of the size and complexity of the network.

II. GENERAL ANALYTICAL RESULTS

In this section, we consider system (2)–(4) with a general infectivity function $\varphi(h)$.

A. The final size

With the initial conditions (5), we can integrate (2) on the interval $[0,t]$ to obtain

$$S_k(t) = S_k^0 e^{-\lambda k\langle k\rangle^{-1}\psi(t)} \quad \text{for} \quad t \geqslant 0, \tag{6}$$

where $S_k^0 = S_k(0)$ and $\psi(t) = \int_0^t \sum_h \varphi(h)P(h)I_h(s-\tau)ds$ and hence

$$\begin{aligned} \psi(t) &= \sum_h \varphi(h)P(h)\int_{-\tau}^{t-\tau} I_h(s)ds \\ &= \sum_h \varphi(h)P(h)\left[\int_{-\tau}^{0} I_h(s)ds + \int_0^{t-\tau} I_h(s)ds\right] \\ &= \Upsilon(t-\tau) + L, \end{aligned} \tag{7}$$

where $\Upsilon(t-\tau) = \sum_h \varphi(h)P(h)\int_0^{t-\tau} I_h(s)ds$ and $L = \sum_h \varphi(h)P(h)\int_{-\tau}^{0} I_h(\theta)d\theta$ (> 0). For $t \geqslant \tau$, with the help

TABLE I. Thresholds in the case where $\varphi(h) = \alpha h$.

Max. deg.	5	10	15	20	25	30
$\lambda_c \approx$	0.5920	0.3891	0.2987	0.2459	0.2108	0.1856

of (4) and (6), we have $\Upsilon(t-\tau)=\sum_h \varphi(h)P(h)R_h(t-\tau)$ and

$$\begin{aligned}&\frac{d}{dt}\Upsilon(t-\tau)\\&=\sum_h \varphi(h)P(h)I_h(t-\tau)\\&=\sum_h \varphi(h)P(h)[1-R_h(t-\tau)-S_h(t-\tau)]\\&=\sum_h \varphi(h)P(h)-\Upsilon(t-\tau)\\&\quad-\sum_h \varphi(h)P(h)S_h^0 e^{-\lambda h\langle k\rangle^{-1}\psi(t-\tau)}\\&=\sum_h \varphi(h)P(h)-\Upsilon(t-\tau)\\&\quad-\sum_h \varphi(h)P(h)S_h^0 e^{-\lambda h\langle k\rangle^{-1}(L+\Upsilon(t-2\tau))}.\end{aligned}\tag{8}$$

As R_k is increasing and bounded by 1, we concluded that $\lim_{t\to\infty} R_k(t)$ exists and $\lim_{t\to\infty} I_k(t)=0$. It follows from $\Upsilon(t-\tau)=\sum_h \varphi(h)P(h)R_h(t-\tau)$ that $\lim_{t\to\infty}\Upsilon(t):=\Upsilon_\infty$ exists. Using $R_h(\infty)=1-S_h(\infty)$ as well as (6) and (7), we can express $R_\infty:=\sum_h P(h)[1-S_h(\infty)]$ as

$$R_\infty=\sum_h P(h)\big[1-S_h^0 e^{-\lambda h\langle k\rangle^{-1}L}e^{-\lambda h\langle k\rangle^{-1}\Upsilon_\infty}\big].\tag{9}$$

On the other hand, since $\lim_{t\to\infty} I_k(t)=0$, we have $\lim_{t\to\infty}\frac{d}{dt}\Upsilon(t-\tau)=0$. This, together with (8), yields $G(\Upsilon_\infty)=0$, where

$$\begin{aligned}G(\Upsilon_\infty):&=\sum_h \varphi(h)P(h)-\Upsilon_\infty\\&\quad-\sum_h \varphi(h)P(h)S_h^0 e^{-\lambda h\langle k\rangle^{-1}(L+\Upsilon_\infty)}.\end{aligned}\tag{10}$$

Note that $G(\sum_h \varphi(h)P(h))<0$ and $G(0)>0$. Therefore, the concavity of G implies the existence of a unique $\Upsilon_\infty\in(0,\sum_h \varphi(h)P(h))$ such that $G(\Upsilon_\infty)=0$.

Consequently, we obtain R_∞ in terms of Eq. (9). It is important to note that we obtain R_∞ for a general infectivity function without requiring a threshold condition $\lambda>\lambda_c$ that was required in Moreno *et al.* [10] and Yang *et al.* [12] even for some special cases.

B. Multiple peaks of the force of infection

Though a measurable quantity is the total of infection $I(t)=\sum_h P(h)I_h(t)$ defined before, the quantity describing more accurately the severeness of the disease outbreak is the force of infection. The force of infection is defined as $\hat{I}(t):=\sum_h \varphi(h)P(h)I_h(t)$. Whether the epidemic has multiple peaks depends on the existence and the number of t^* such that $\frac{d}{dt}\hat{I}(t^*)=0$. In the sequel, we focus on $\hat{I}(t)$. However, our simulations to be reported below will plot both the force of infection and the total of infection and will show how they differ depending on the infectivity function φ.

First, we consider the case where $\tau=0$. Following the calculations of Ref. [10], we have

$$\begin{aligned}\hat{I}(t)&=\sum_h \varphi(h)P(h)\\&\quad-\widetilde{\phi}(t)-\sum_h \varphi(h)P(h)S_h^0 e^{-\lambda h\langle k\rangle^{-1}\widetilde{\phi}(t)}\\&:=H_0(\widetilde{\phi}(t)),\end{aligned}\tag{11}$$

where $\widetilde{\phi}(t)=\int_0^t \hat{I}(s)ds=\sum_h \varphi(h)P(h)R_h(t)$. Then $\frac{d}{dt}\hat{I}(t)=H_1(\widetilde{\phi}(t))\hat{I}(t)$, where

$$H_1(x)=-1+\sum_h \lambda h\langle k\rangle^{-1}\varphi(h)P(h)S_h^0 e^{-\lambda h\langle k\rangle^{-1}x}.$$

As $\hat{I}(t)>0$, it reaches its peaks only when $H_1(\widetilde{\phi}(t))=0$. Note that H_1 is strictly decreasing and $H_1(0)=-1+\sum_h \lambda h\langle k\rangle^{-1}\varphi(h)P(h)S_h^0$. It follows that $H_1(x)<0$ for all $x>0$ if $\lambda\frac{\langle S_k^0\varphi(k)k\rangle}{\langle k\rangle}\leqslant 1$ or, equivalently, if

$$\lambda\leqslant\lambda_c:=\frac{\langle k\rangle}{\langle S_k^0\varphi(k)k\rangle}.$$

If $\lambda>\lambda_c$, then $H_1(0)>0$. This implies that $\hat{I}(t)$ is increasing when t is close enough to 0. This, combined with $\hat{I}(t)\to 0$ as $t\to\infty$, implies that $\hat{I}(t)$ has a local maximum. As $H_1(x)$ is decreasing as a function of $x\geqslant 0$, we conclude that there exists exactly one nonzero $\widetilde{\phi}_0$ such that $H_1(\widetilde{\phi}_0)=0$. On the other hand, $\widetilde{\phi}(t)$ is strictly increasing. It follows that there exists a unique t^* such that $\widetilde{\phi}(t^*)=\widetilde{\phi}_0$. Therefore, we conclude that: *if $\lambda>\lambda_c$, the epidemic will take off and the force of infection will have exactly one peak, and if $\lambda<\lambda_c$, then the force of infection will decrease and the epidemic will not take off.* This result, when $\tau=0$, is consistent with those of Moreno *et al.* [10] and Yang *et al.* [12].

We now consider the case where $\tau>0$, and here we assume $I_k(\theta)=I_k^0(\geqslant 0)$ for $\theta\in[-\tau,0]$ and $I_k^0>0$ for some k. It follows from (3) that

$$\begin{aligned}\tfrac{d}{dt}\hat{I}(t)&=-\hat{I}(t)+\sum_h \lambda h\langle k\rangle^{-1}\varphi(h)P(h)\\&\quad\times S_h^0 e^{-\lambda h\langle k\rangle^{-1}(L+\Upsilon(t-\tau))}\hat{I}(t-\tau).\end{aligned}\tag{12}$$

We now distinguish two cases.

Case 1 : $\lambda\leqslant\lambda_c$. We claim that $\hat{I}(t)$ is decreasing in t. To verify this, we first note that

$$\begin{aligned}&\sum_h \lambda h\langle k\rangle^{-1}\varphi(h)P(h)S_h^0 e^{-\lambda h\langle k\rangle^{-1}(L+\Upsilon(t-\tau))}\\&\leqslant\sum_h \lambda h\langle k\rangle^{-1}\varphi(h)P(h)S_h^0\\&=\lambda\langle k\rangle^{-1}\langle h\varphi(h)S_h^0\rangle\\&\leqslant 1.\end{aligned}$$

As $I_h(-\tau)=I_h(0)$, it follows from (12) that $\frac{d}{dt}\hat{I}(0)\leqslant 0$. Let $t^*=\sup\{s\geqslant 0:\hat{I}(t)\text{ is decreasing on }[0,s]\}$. Then, from the above discussion it follows that $t^*\geqslant 0$. It suffices to show that $t^*=\infty$. By way of contradiction, suppose that $t^*<\infty$, then we have $\frac{d}{dt}\hat{I}(t^*)=0$ and there exists a $t^\dagger\in(t^*,t^*+\tau]$ such that $\frac{d}{dt}\hat{I}(t^\dagger)>0$. It follows that there exists a $t^\ddagger\in[t^*,t^\dagger)$

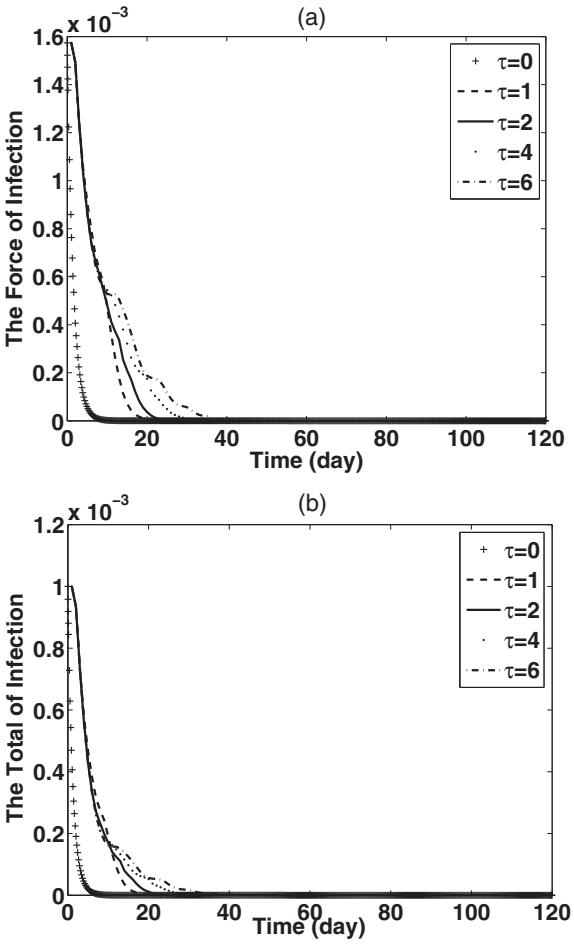

FIG. 1. (a) The evolution of $\sum_h \alpha h P(h) I_h(t)$; (b) the evolution of $\sum_h P(h) I_h(t)$ for different delays when $\lambda = 0.1$ in the case where $\varphi(h) = \alpha h$. The five curves from the left to right in (a) and (b) correspond to delay $\tau = 0, 1, 2, 4, 6$, respectively.

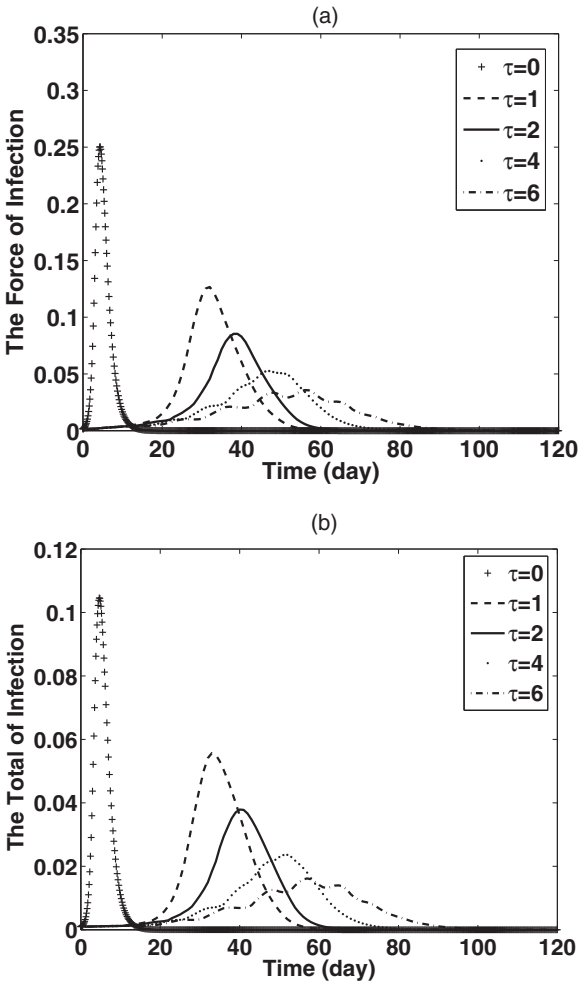

FIG. 2. (a) The evolution of $\sum_h \alpha h P(h) I_h(t)$; (b) the evolution of $\sum_h P(h) I_h(t)$ for different delays when $\lambda = 0.8$ in the case where $\varphi(h) = \alpha h$. The five curves from the left to right in (a) and (b) correspond to delay $\tau = 0, 1, 2, 4, 6$, respectively.

such that $\frac{d}{dt}\hat{I}(t^{\ddagger}) = 0$ and $\hat{I}(t^{\ddagger}) < \hat{I}(t^{\dagger})$. Note that $\hat{I}(t^{\ddagger} - \tau) \geqslant \hat{I}(t^{\dagger} - \tau)$. It follows from (12) that

$$0 < \frac{d}{dt}\hat{I}(t^{\dagger}) \leqslant \frac{d}{dt}(t^{\ddagger}) = 0,$$

a contradiction. This proves the claim. It follows that the disease will take off but the force of infection will not increase.

Case 2: $\lambda > \lambda_c$. In this case, since $I_h(0) = I_h(-\tau)$ and

$$\begin{aligned}&\sum_h \lambda h \langle k\rangle^{-1} \varphi(h) P(h) S_h^0 e^{-\lambda h \langle k\rangle^{-1}(L+\Upsilon(-\tau))}\\ &= \lambda \langle k\rangle^{-1} \langle S_k^0 k \varphi(k)\rangle\\ &> 1,\end{aligned}$$

we have $\frac{d}{dt}\hat{I}(0) > 0$, which implies that $\hat{I}(t)$ increases at the beginning of the epidemic. Since $\hat{I}(t) \to 0$ as $t \to \infty$, there exists at least one outbreak.

Assume that t^* is the first peak time and remember that $I_k(\theta) = I_k^0$ for $\theta \in [-\tau, 0]$. Then $\hat{I}(t)$ is increasing on $[-\tau, t^*]$ and $\frac{d}{dt}\hat{I}(t^*) = 0$. Using (12), we get

$$\sum_h \lambda h \langle k\rangle^{-1} \varphi(h) P(h) S_h^0 e^{-\lambda h \langle k\rangle^{-1}(L+\Upsilon(t^*-\tau))} > 1.$$

We now consider two extreme cases for the possibility of multiple outbreaks.

Case 2S (Small delay). In this case, $\hat{I}(t^*) \approx \hat{I}(t^* - \tau)$ and hence, by Eq. (12), $\sum_h \lambda h \langle k\rangle^{-1} \varphi(h) P(h) S_h^0 e^{-\lambda h \langle k\rangle^{-1}(L+\Upsilon(t^*-\tau))} \approx 1$. As $\Upsilon(t)$ is increasing in t, it is easy to have $\sum_h \lambda h \langle k\rangle^{-1} \varphi(h) P(h) S_h^0 e^{-\lambda h \langle k\rangle^{-1}(L+\Upsilon(t-\tau))} < 1$ for $t > t^*$, and so it is hard to get a $t^{**} > t^*$ such that $\frac{d}{dt}\hat{I}(t^{**}) > 0$ by (12). Therefore, multiple outbreaks are unlikely.

Case 2L (Large delay). In this case, $\hat{I}(t^*)$ is much greater than $\hat{I}(t^* - \tau)$ and hence it follows from (12) that $\sum_h \lambda h \langle k\rangle^{-1} \varphi(h) P(h) S_h^0 e^{-\lambda h \langle k\rangle^{-1}(L+\Upsilon(t^*-\tau))}$ is much greater than 1. There is a good chance of the occurrence of multiple peaks. For example, if $\sum_h \lambda h \langle k\rangle^{-1} \varphi(h) P(h) S_h^0 e^{-\lambda h \langle k\rangle^{-1}(L+\Upsilon(t^*))} > 1$ then $\hat{I}(t)$ cannot be decreasing on $[t^*, t^* + \tau]$. Otherwise, there exists $t^{\dagger} \in [t^*, t^* + \tau]$ such that $\hat{I}(t^{\dagger}) \leqslant \hat{I}(t^{\dagger} - \tau)$. Hence it follows from Eq. (12) that $\frac{d}{dt}\hat{I}(t^{\dagger}) > 0$, a contradiction. If $\hat{I}(t)$ is not decreasing on $[t^*, \infty)$ then as discussed above there exists at least another peak of the force of infection.

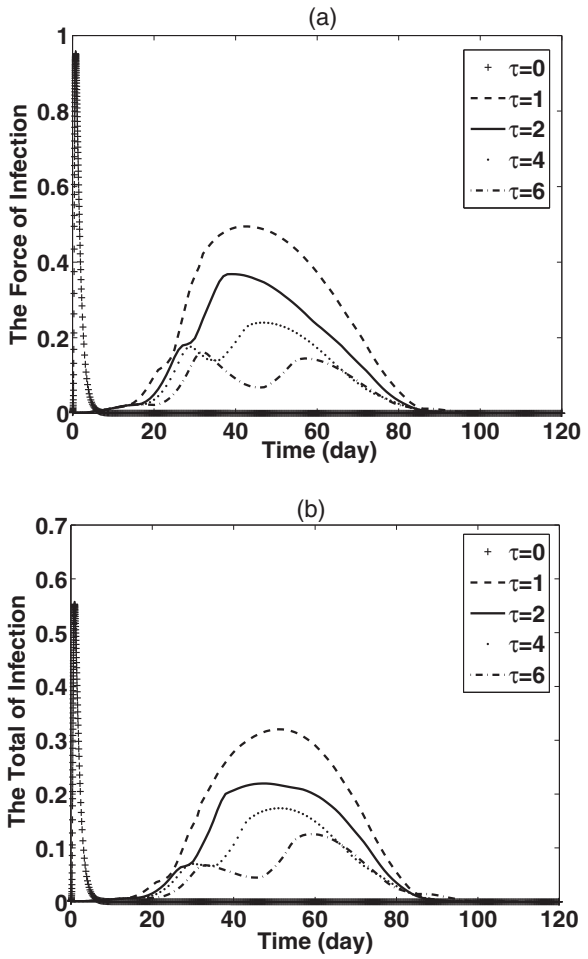

FIG. 3. (a) The evolution of $\sum_h \alpha h P(h) I_h(t)$; (b) the evolution of $\sum_h P(h) I_h(t)$ for different delays when $\lambda = 5$ in the case where $\varphi(h) = \alpha h$. The five curves from the left to right in (a) and (b) correspond to delay $\tau = 0, 1, 2, 4, 6$, respectively.

In summary, we conclude that *if* $\lambda \leqslant \lambda_c$, *there is no peak of the force of infection; if* $\lambda > \lambda_c$, *there is only one peak when the delay is small enough, while there may be multiple peaks when the delay is large*.

III. NUMERICAL SIMULATIONS: TWO CASE STUDIES

In Sec. II, we obtained some general results for system (2)–(4) on an uncorrelated network. We know that the maximum network degree plays an important role in determining whether an epidemic can take off. This becomes clear from the expression of the threshold $\lambda_c = \frac{\langle k \rangle}{\langle S_k^0 \varphi(k) k \rangle}$. On the other hand, larger delays can be a potential cause for multiple outbreaks represented by multiple peaks of the force of infection. In this section, we will support these theoretical results with numerical simulations for two special cases of the infectivity function φ. Both cases will involve only scale-free uncorrelated networks for the sake of simplicity, and it is an important issue to see that multiple waves in a single outbreak can be generated by the delay in an uncorrelated network. Recall that, in a scale-free network, $P(k) \sim k^{-2-\gamma}$. Moreover, the maximal degree is taken to be 15 in both cases. We emphasize that the simulations are numerical integration without any explicit networks. In other words, instead of simulating a spreading model on complex networks, we numerically integrate with respect to time of the group of delay differential equations (2)–(4).

Case 1: $\varphi(h) = \alpha h$. In this case, the final size R_∞ is

$$R_\infty = \sum_h P(h)\left(1 - S_h^0 e^{-\lambda h \langle k \rangle^{-1}(L + \Upsilon_\infty)}\right),$$

where $L = \sum_h \alpha h P(h) \int_{-\tau}^0 I_h(\theta) d\theta$ and Υ_∞ is the unique positive solution to

$$\alpha \langle k \rangle - \Upsilon_\infty - \sum_h \alpha h P(h) S_h^0 e^{-\lambda h \langle k \rangle^{-1}(L + \Upsilon_\infty)} = 0.$$

Moreover, the threshold $\lambda_c = \frac{\langle k \rangle}{\alpha \langle S_k^0 k^2 \rangle}$. For the case without delay ($\tau = 0$), if $S_k^0 \approx 1$ for any degree k, then $\lambda_c \approx \frac{\langle k \rangle}{\alpha \langle k^2 \rangle}$, which is consistent with the result in Ref. [10].

For the simulation below, we take $\gamma = 0.1$ and $\alpha = 0.8$. We also assume that, for any degree k, $S_k^0 = 0.999$ and $I_k^0 = 0.001$. Table I lists the thresholds for different maximum degrees. We can easily see that the threshold decreases as the maximum degree increases. This means that heterogeneity is an important factor for a disease to take off.

To consider the effect of delay, we assume that the maximal degree is 15. Then $\lambda_c \approx 0.2987$. Figure 1 shows the evolutions of $\sum_h \alpha h P(h) I_h(t)$ and $\sum_h P(h) I_h(t)$ for different delays with $\lambda = 0.1$. It turns out that there is no peak for $\sum_h \alpha h P(h) I_h(t)$ as λ is less than λ_c and $\sum_h P(h) I_h(t)$ has similar behaviors. We now consider the case where $\lambda > \lambda_c$. First, we take $\lambda = 0.8 \in (\lambda_c, \mu) = (\lambda_c, 1)$. Figure 2 shows that there is always a peak and the number of peaks increases with delay for both $\sum_h \alpha h P(h) I_h(t)$ and $\sum_h P(h) I_h(t)$. Then, we take $\lambda = 5 > \mu = 1$. Figure 3 illustrates more remarkably multiple peaks of the infection with the highest peak being determined by the size of the delay.

Case 2 : $\varphi(h) = B$, where B is a constant. In this case, the final size R_∞ is

$$R_\infty = \sum_h P(h)\left(1 - S_h^0 e^{-\lambda h \langle k \rangle^{-1}(L + \Upsilon_\infty)}\right),$$

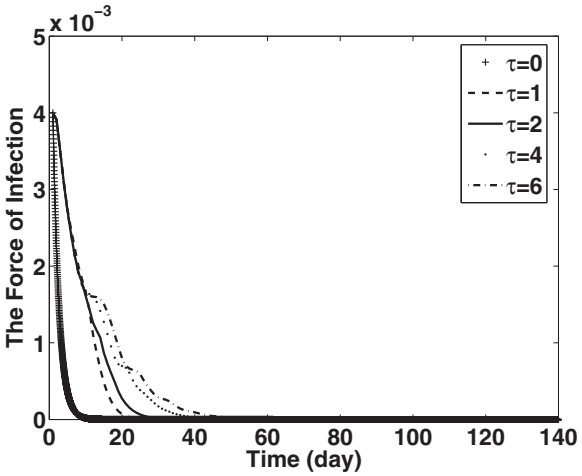

FIG. 4. The evolution of $\sum_h B P(h) I_h(t)$ for different delays when $\lambda = 0.1$ in the case where $\varphi(h) = B$. The five curves from the left to right correspond to delay $\tau = 0, 1, 2, 4, 6$, respectively.

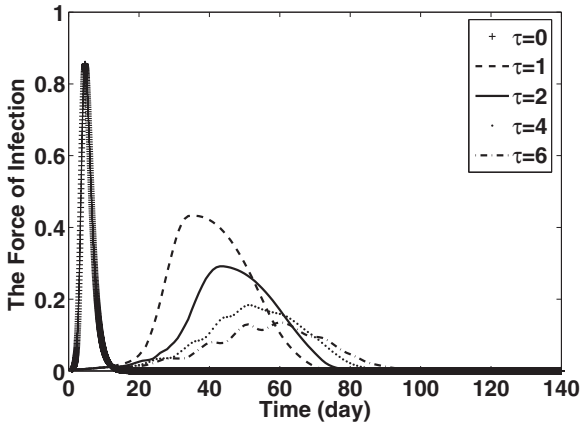

FIG. 5. The evolution of $\sum_h BP(h)I_h(t)$ for different delays when $\lambda = 0.8$ in the case where $\varphi(h) = B$. The five curves from the left to right correspond to delay $\tau = 0, 1, 2, 4, 6$, respectively.

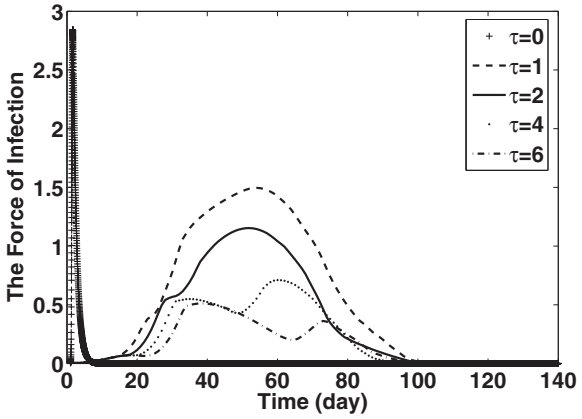

FIG. 6. The evolution of $\sum_h BP(h)I_h(t)$ for different delays when $\lambda = 5$ in the case where $\varphi(h) = B$. The five curves from the left to right correspond to delay $\tau = 0, 1, 2, 4, 6$, respectively.

where $L = \sum_h BP(h)\int_{-\tau}^{0} I_h(\theta)d\theta$ and Υ_∞ is the unique positive solution to

$$B - \Upsilon_\infty - \sum_h BP(h)S_h^0 e^{-\lambda h\langle k\rangle^{-1}(L+\Upsilon_\infty)} = 0.$$

Moreover, the threshold $\lambda_c = \frac{\langle k\rangle}{B\langle S_k^0 k\rangle}$. Note that if S_k^0 are the same for all k then λ_c is independent of the maximum degree. Moreover, if $S_k^0 \approx 1$ for any degree k then $\lambda_c \approx 1/B$, which agrees with the result in Yang *et al.* [12]. In general, λ_c depends on the maximum degree and so the heterogeneity again affects whether a disease can take off.

Since the force of infection is proportional to the total of infection, we plot only the force of infection in the simulations below. During the simulations, we take the maximal degree to be 15, $\gamma = 0.1$ and $B = 4$. We also assume that $S_k^0 = 0.999$ and $I_k^0 = 0.001$ for any degree k. Then $\lambda_c \approx 0.2503$. Similar to the case where $\varphi(h) = \alpha h$, we first take $\lambda = 0.1 \ll \lambda_c$. Figure 4 shows the evolution of $\sum_h BP(h)I_h(t)$ for different delays. We observe no increase for $\sum_h BP(h)I_h(t)$. Then we take $\lambda = 0.8 \in (\lambda_c, \mu)$. Figure 5 indicates that there is always a peak and the number of peaks increases with delay for $\sum_h BP(h)I_h(t)$. Finally, we take $\lambda = 5 > \mu$. Figure 6 shows that a higher or the highest peak seems to appear in subsequent waves when delay is increased.

We conclude the paper with a remark. In the above, we have plotted both the force of infection and the total of infection. It is interesting to note that in the first case, these two quantities may differ in their behavior depending on the infectivity function φ.

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