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Burst of virus infection and a possibly largest epidemic threshold of non-Markovian susceptible-infected-susceptible processes on networks

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Since a real epidemic process is not necessarily Markovian, the epidemic threshold obtained under the Markovian assumption may be not realistic. To understand general non-Markovian epidemic processes on networks, we study the Weibullian susceptible-infected-susceptible (SIS) process in which the infection process is a renewal process with a Weibull time distribution. We find that, if the infection rate exceeds $1/\ln(\lambda_1+1)$, where λ_1 is the largest eigenvalue of the network's adjacency matrix, then the infection will persist on the network under the mean-field approximation. Thus, $1/\ln(\lambda_1+1)$ is possibly the largest epidemic threshold for a general non-Markovian SIS process with a Poisson curing process under the mean-field approximation. Furthermore, non-Markovian SIS processes may result in a multimodal prevalence. As a byproduct, we show that a limiting Weibullian SIS process has the potential to model bursts of a synchronized infection.

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I. INTRODUCTION

The susceptible-infected-susceptible (SIS) epidemic process is a basic model for virus spreading on networks [1]. We consider a graph G where N nodes are connected by L links, specified by an adjacency matrix A. In the SIS model, a node j of the network can be in either of two states: susceptible $X_j(t)=0$ or infected $X_j(t)=1$, for $j=1,\ldots,N$. A susceptible node can be infected by an infected neighbor with rate β, while an infected node can be cured with rate δ. By tuning the effective infection rate $\tau \triangleq \beta/\delta$, the SIS process experiences a phase transition at the epidemic threshold τ_c. For $\tau>\tau_c$, the infection can persist on the network for a very long time [2], and for $\tau<\tau_c$ the process quickly enters the all-healthy state. For simplicity, most research (implicitly) assumes that the process is Markovian, which means that both the infection and curing process are Poisson processes. The length of the time interval between two adjacent events (infection or curing in the SIS process) is exponentially distributed in the Poisson process. With Poisson infection and curing processes, the SIS process is thus a Markov process with 2^N states [3]. Under the Markovian assumption, the epidemic threshold τ_c can be approximately obtained by mean-field approximations, such as the Heterogeneous Mean-Field approximation [4] and the N-Intertwined Mean-Field Approximation (NIMFA) [5]. The NIMFA threshold is a lower bound of the exact threshold [6]. However, an epidemic process is not necessarily Markovian, and the infection attempts do not happen uniformly with time t as in a Poisson process. For example, the infection time of online information spread is found to be log-normal distributed [7]. To model general non-Markovian epidemic processes, we consider a renewal infection process [8]. In the renewal infection process, the distribution of the infection time T, which is the time interval between two adjacent infection attempts of an infected node, is replaced by a more general distribution. We adopt the Weibull distribution as in [6,9]:

$$f_T(x)=\frac{\alpha}{b}\left(\frac{x}{b}\right)^{\alpha-1}e^{-(x/b)^{\alpha}} \tag{1}$$

for $x\geqslant 0$, with the expectation

$$E[T]=b\Gamma\left(1+\frac{1}{\alpha}\right)$$

where α is a shape parameter, $\Gamma(x)$ is the Gamma function, and $b=[\beta\Gamma(1+\frac{1}{\alpha})]^{-1}$ because the average infection time $E[T]$ is fixed to the inverse of the infection rate $1/\beta$ in order to compare different α regimes. Furthermore, the distribution function is

$$F_T(x)=\Pr[T\leqslant x]=1-e^{-(x/b)^{\alpha}} \tag{2}$$

for $x\geqslant 0$. We refer to this model as a *Weibullian SIS process*. In the Weibullian SIS process, the shape parameter α controls the infection process. The Weibull distribution is heavy-tailed when $\alpha<1$, exponential when $\alpha=1$ and hence Markovian, and Gaussian-like when $\alpha>1$. Furthermore, tuning the shape parameter α dramatically shifts the epidemic threshold, and the epidemic threshold increases with the distribution changing from heavy-tailed to Gaussian-like [6,9].

The Weibullian SIS process is capable of modeling various kinds of non-Markovian epidemic processes by choosing a suitable shape parameter α. For example, the Weibullian SIS process with a heavy-tailed infection time ($\alpha<1$) predicts a smaller epidemic threshold τ_c, compared to a Markovian SIS process, which agrees with the fact that a heavy-tailed interaction time leads to a longer persistence of infection in reality [10]. However, the shape parameter α is generally not known for a real-life epidemic process, which raises two questions: (1) How small should the effective infection rate τ be to ensure that there is no epidemic on the network? and (2) how large should the effective infection rate τ be to ensure a persistence of infection on the network? Obviously, since

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the Markovian SIS process is a special case ($\alpha = 1$) of the Weibullian SIS process, neither of the answers to those two questions is the NIMFA epidemic threshold $1/\lambda_1$, where λ_1 is the largest eigenvalue of the adjacency matrix A of the network. In this paper, we make a first step to understand those questions. Under the mean-field approximation, we find that the largest epidemic threshold of the Weibullian SIS process is $\frac{1}{\ln(\lambda_1+1)}$, which is obtained when $\alpha \to \infty$. Since the Weibullian SIS process is able to model a general epidemic process, we argue that the infection can persist on the network when the effective infection rate $\tau > \frac{1}{\ln(\lambda_1+1)}$ for any infection process. Simulation results in Fig. 3 seem to support our claim.

Another motivation of our paper is that an infinite shape parameter α leads to a model for synchronized spreading phenomena. For the Weibullian SIS process with a finite α, the prevalence and the epidemic threshold can be calculated approximately by the renewal theory under the assumption that the number of infection events equals the number of curing events in the steady state [9]. Thus, the Markovian and non-Markovian process can be treated within a same framework [9]. When $\alpha \to \infty$, the distribution of the infection time becomes a Dirac delta function at the average infection time $1/\beta$. The infection attempts happen periodically, and the constant steady or metastable prevalence vanishes. This extreme nonuniform distribution of the infection attempts leads to a failure of the method proposed in [9]. The Weibullian SIS process with $\alpha \to \infty$ has the potential to be applied to some realistic situations. For example, a computer virus can be controlled to infect computers periodically, and it is also technically possible for a virus to burst at the same time point. Many computer viruses burst periodically because the developers of a virus spend time on improving the virus before each burst. Thus, the virus development life cycle and the underlying network collectively determine whether the infection can persist or not. Another example is the seasonal influenza H3N2 where the infection emerges at each influenza season and the prevalence is at a low level between seasons [11]. In those situations, either the infection is synchronized or the infection time interval is sharply Gaussian-like distributed. The Weibullian SIS process with $\alpha \to \infty$ can be applied to approximate those resurgent epidemic processes. Currently, the Weibullian SIS model with $\alpha \to \infty$ has not been researched, and we present here some initial results.

In the following part of this paper, we first study the Weibullian SIS process with $\alpha \to 0$ to show that the epidemic threshold can be very small, and then we study the process with $\alpha \to \infty$ under the mean-field approximation. Numerical and simulation results are also presented.

II. THE WEIBULLIAN SIS PROCESS WITH $\alpha \to 0$ AND ∞

In the Weibullian SIS process, the distribution of the infection time between two adjacent infection attempts of an infected node is a Weibull distribution with an expectation $1/\beta$, and the distribution of the infected time duration is exponential with an expectation $1/\delta$. If $\alpha = 1$, then the Weibullian SIS process reduces to the Markovian SIS process. If the probability of the occurrence of an infection attempt decreases with time, then the process can be modeled by the process with a suitable shape parameter $\alpha < 1$. Otherwise, the infection can be modeled by $\alpha > 1$.

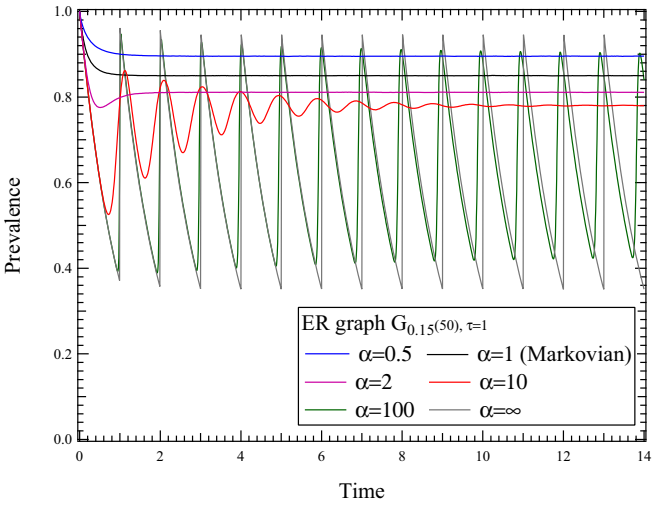


FIG. 1. The prevalence of the Weibullian SIS process on an Erdős-Rényi (ER) network $G_{0.15}(50)$ obtained by averaging over 10^5 realizations. The effective infection rate $\tau = 1$ is around 8.5 times the NIMFA threshold ($1/\lambda_1 = 0.1173$). The minimum prevalence decreases with the shape parameter α. For $\alpha < 1$, the prevalence is a trivial single-peak function with time. For small $\alpha > 1$, the prevalence oscillates but eventually becomes approximately constant. When $\alpha \to \infty$, the metastable state prevalence is no longer constant.

When $\alpha \to 0$, the Weibullian SIS epidemic threshold is zero, and for an arbitrary small α the mean-field epidemic threshold given in [9] tends to zero (see Appendix A).

If $\alpha \to \infty$, then the distribution function (2) of the infection time for $T \neq 1/\beta$ tends to

$$\lim_{\alpha\to\infty} F_T(x) = \lim_{\alpha\to\infty} 1 - e^{-[\beta x \Gamma(1+1/\alpha)]^\alpha} = \begin{cases} 1 & \text{for } x > \frac{1}{\beta} \\ 0 & \text{for } x < \frac{1}{\beta}. \end{cases} \tag{3}$$

The distribution function is right-continuous, and then $F_T(1/\beta) = \lim_{x\to(1/\beta)^+} F_T(x) = 1$ at the discontinuity $x = \frac{1}{\beta}$. Thus, the probability distribution of the time interval between two adjacent infection events is $\Pr[T = 1/\beta] = 1$ and $\Pr[T \neq 1/\beta] = 0$.

Figure 1 shows the time-dependent prevalence $y(t) \triangleq \frac{1}{N}\sum_{i=1}^{N} E[X_i(t)]$, which is the average fraction of the infected nodes in the Weibullian SIS process. Initially, all nodes are infected. For $\alpha \leqslant 1$, the prevalence $y(t)$ monotonically decreases to the metastable state, and for $\alpha > 1$ the prevalence $y(t)$ fluctuates with a decaying amplitude. When $\alpha \to \infty$, the prevalence $y(t)$ is no longer steady, but periodically changes. There is a huge gap between the maximum and the minimum prevalence. With the increase of α, the amplitude increases, but the minimum prevalence decreases as shown in Fig. 1. The persistence of the infection needs a higher effective infection rate τ for a larger α. Figure 1 reveals that a non-Markovian infection process may lead to a multimodal prevalence, a function with multiple local maxima over time. The multimodal prevalence represents the resurgence of the epidemic. Previously, the resurgent phenomenon was found in the susceptible-infected and the susceptible-infected-recover model [12,13].

As mentioned above, the infected nodes infect their neighbors precisely every $1/\beta$ time unit when $\alpha \to \infty$, and then it is hard to study the process with only one time parameter t as done in the Markovian SIS process. To investigate this process analytically, we divide the time t into time intervals of length $1/\beta$ with index $n = 0,1,\ldots$. The infection state of node j is $X_j(t^* + n/\beta)$ at time t^* of the n-th time interval, where $n \geqslant 0$ and $t^* \in [0,1/\beta)$. At $t = 0$, the initially infected nodes are seeded, and the first infection attempt of each infected node happens at the start of the second time interval $t = 1/\beta$. Thus, the infection attempts always happen at the start of each time interval ($t^* = 0$). If a healthy node has an infected neighbor when $t^* \to 1/\beta$, then the healthy node will be infected at the start of the next time interval. The probability that node j is healthy at the end of the nth time interval and has at least one infected neighbor is

$$\lim_{t^*\to 1/\beta} \Pr\left[X_j\left(t^* + \frac{n}{\beta}\right) = 0, \sum_{i\in\mathcal{N}_j} X_i\left(t^* + \frac{n}{\beta}\right) \geqslant 1\right] \quad (4)$$

where $\mathcal{N}_j$ is the set of the neighbors of node j, while the probability that node j is infected at the end of the nth time interval is

$$\lim_{t^*\to 1/\beta} E\left[X_j\left(t^* + \frac{n}{\beta}\right)\right]. \quad (5)$$

The probability that node j is infected at the start of the $(n+1)$th time interval $E\{X_j[(n+1)/\beta]\}$ is thus the sum of (4) and (5), and we obtain Eq. (6):

$$E\left[X_j\left(\frac{n+1}{\beta}\right)\right] = \lim_{t^*\to 1/\beta}\left\{\Pr\left[X_j\left(t^* + \frac{n}{\beta}\right) = 0, \sum_{i\in\mathcal{N}_j} X_i\left(t^* + \frac{n}{\beta}\right) \geqslant 1\right] + E\left[X_j\left(t^* + \frac{n}{\beta}\right)\right]\right\}. \quad (6)$$

Equation (6) is not analytically solvable. Here, we apply a mean-field approximation to solve (6), similar as in NIMFA for the Markovian SIS process. We assume that the infection state between neighbors is independent at the end of each time interval, i.e., $\lim_{t^*\to 1/\beta} E[X_i(t^* + n/\beta)X_j(t^* + n/\beta)] = \lim_{t^*\to 1/\beta} E[X_i(t^* + n/\beta)]E[X_j(t^* + n/\beta)]$. Under this assumption, we denote the approximate value of $E[X_j(t)]$ by $v_j(t)$, and the infection probabilities by a column vector $\mathbf{v}(t) \triangleq [v_1(t),\ldots,v_N(t)]^T$. Thus, the mean-field infection probability at $t^* = 0$ of the $(n+1)$th time interval follows (7):

$$v_j\left(\frac{n+1}{\beta}\right) = \lim_{t^*\to 1/\beta}\left(\left[1 - v_j\left(t^* + \frac{n}{\beta}\right)\right]\left\{1 - \prod_{i\in\mathcal{N}_j}\left[1 - v_i\left(t^* + \frac{n}{\beta}\right)\right]\right\} + v_j\left(t^* + \frac{n}{\beta}\right)\right). \quad (7)$$

In each time interval, an infected node can be cured at any time point with a equal probability during $t^* \in [0,1/\beta)$, because the curing process is Poissonian. The governing equation of the infection probability of node j for $j = 1,\ldots,N$ is

$$\frac{dv_j(t^* + n/\beta)}{dt^*} = -\delta v_j\left(t^* + \frac{n}{\beta}\right)$$

for $t^* \in [0,1/\beta)$. Given the initial condition $v_j(n/\beta)$, the solution of the equation above is

$$v_j\left(t^* + \frac{n}{\beta}\right) = v_j\left(\frac{n}{\beta}\right)e^{-\delta t^*} \quad (8)$$

for $t^* \in [0,1/\beta)$.

Substituting (8) evaluated at $t^* \to 1/\beta$ of the nth time interval, thus $\lim_{t^*\to 1/\beta} v_j(t^* + \frac{n}{\beta}) = v_j(n/\beta)e^{-1/\tau}$ into (7), we obtain a recursion of the infection probability at $t^* = 0$ of each time interval (9):

$$v_j\left(\frac{n+1}{\beta}\right) = \left[1 - v_j\left(\frac{n}{\beta}\right)e^{-1/\tau}\right]\left\{1 - \prod_{i\in\mathcal{N}_j}\left[1 - v_i\left(\frac{n}{\beta}\right)e^{-1/\tau}\right]\right\} + v_j\left(\frac{n}{\beta}\right)e^{-1/\tau}. \quad (9)$$

Equation (9) has a similar form as the discrete-time SIS process, which has been studied in [14]. In the metastable state $n \to \infty$, the infection probability $v_j(n/\beta)$ at the start of each time interval $t^* = 0$ is constant. We can check whether the infection probability $\lim_{n\to\infty} v_j(n/\beta)$ is zero or not, to obtain the epidemic threshold. Consequently, we arrive at the following result.

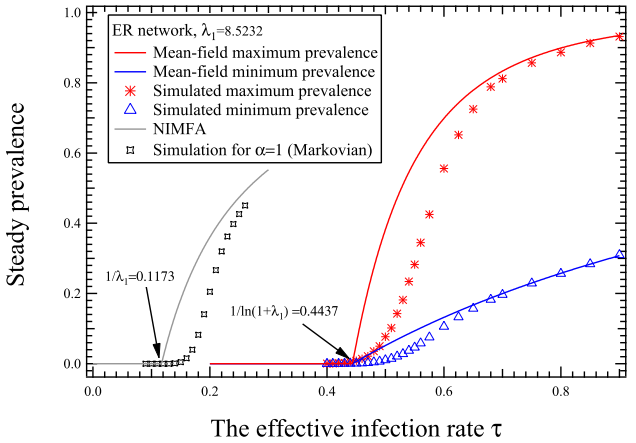
Theorem 1. The mean-field epidemic threshold of the Weibullian SIS process on a connected network with $\alpha \to \infty$ obtained by (8) and (9) is

$$\tau_c^{(1)} = \frac{1}{\ln(1+\lambda_1)}. \quad (10)$$

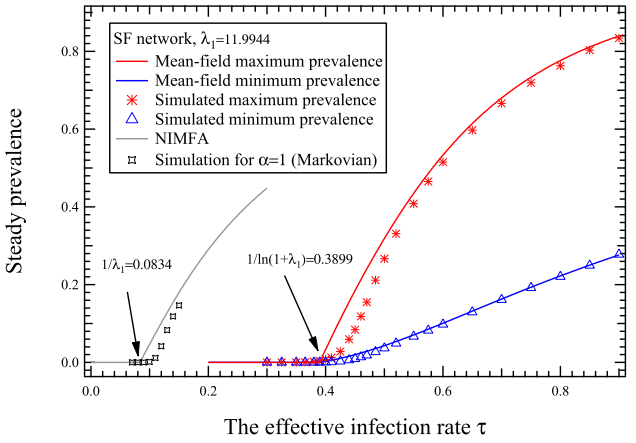
If the effective infection rate $\tau > \tau_c^{(1)}$, then infection can persist on the network with a nonzero steady periodic infection probability $\mathbf{v}_\infty(t^*) \triangleq \lim_{n\to\infty} \mathbf{v}(t^* + n/\beta)$, and $\mathbf{v}_\infty(t^*) = \mathbf{v}_\infty(0)e^{-\delta t^*}$ for $t^* \in (0,1/\beta]$. If $\tau < \tau_c^{(1)}$, then the epidemic process enters the all-healthy state in the long run $\lim_{t\to\infty} \mathbf{v}(t) = \mathbf{0}$.

The proof of Theorem 1 is in Appendix B. The superscript (1) in $\tau_c^{(1)}$ refers to a first-order mean-field approximation. The epidemic threshold (10) has a similar form as the NIMFA epidemic threshold [3] and the discrete-time SIS [15] threshold $1/\lambda_1$, but with a logarithmic relation to the largest eigenvalue λ_1 of the adjacency matrix A of the network. Furthermore, the term $1 + \lambda_1$ in the logarithmic function ensures that the epidemic threshold (10) is positive for $\lambda_1 > 0$ in any finite-size connected network. The threshold (10) of a scale-free network with a finite average degree [16,17] converges to zero in the thermodynamic limit $N \to \infty$.

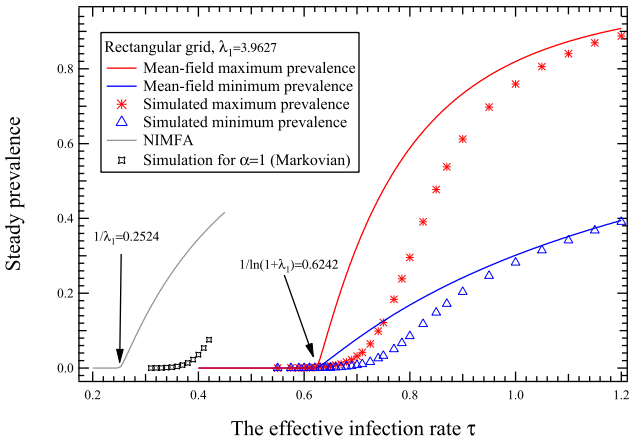
When the effective infection rate $\tau < \tau_c^{(1)}$, the infection probability $v_j(t)$ of each node decreases to zero in the long run. We represent $x_i \leqslant y_i$ and $x_i < y_i$ for all i by the vector relationship $[x_1,\cdots,x_n]^T \preceq [y_1,\cdots,y_n]^T$ and $[x_1,\cdots,x_n]^T \prec$



(a)



(b)



(c)

FIG. 2. Steady maximum prevalence and minimum prevalence of three different networks under the mean-field approximation and simulation. The prevalence is obtained by averaging over 10^5 realizations of simulation with all nodes infected initially to prevent the inaccuracy caused by the early die-out [18]. The simulation runs for a long enough time (50 time units with $\delta = 1$), and the maximum and minimum prevalence are plotted, which are selected from the last complete time period. The networks are (a) the ER network $G_{0.15}(50)$ corresponding to Fig. 1; (b) a Barabási-Albert scale-free network with size $N = 500$, and number of links $L = 1491$; and (c) a rectangular grid with size $N = 484$, $L = 924$.

$[y_1, \cdots, y_n]^T$, respectively. If $\tau < \tau_c^{(1)}$, then the infection probability $\mathbf{v}(t)$ is upper bounded by an exponentially decreasing function with time t, which is

$$\mathbf{v}(t) \prec (e^{-\delta}(\lambda_1 + 1)^{\beta})^t \mathbf{z}$$

where $\mathbf{z}$ is a constant vector of which every element is positive (see Appendix C). Furthermore, the mean-field prevalence $y^{(1)}(t) \triangleq \frac{1}{N}\sum_{i=1}^{N} v_i(t)$ is upper bounded by $y^{(1)}(t) < [e^{-\delta}(\lambda_1 + 1)^{\beta}]^t c$, where c is a positive value.

When $\tau > \tau_c^{(1)}$, the steady infection probability reaches a maximum $\mathbf{v}_\infty(0)$ at the start of each time interval $t^* = 0$, and a minimum $\mathbf{v}_\infty(0^-) \triangleq \lim_{t^* \to 1/\beta} \mathbf{v}_\infty(t^*)$ at the end of each time interval $t^* \to 1/\beta$. The steady maximum infection probability $\mathbf{v}_\infty(0)$ can be obtained by solving (9) numerically. Since $\mathbf{v}_\infty(0) = \mathbf{v}_\infty(0^-)e^{1/\tau}$, the ratio between the maximum and minimum steady infection probability is

$$\frac{\mathbf{v}_\infty(0)}{\mathbf{v}_\infty(0^-)} = e^{1/\tau} < \lambda_1 + 1. \tag{11}$$

The last inequality holds because the effective infection rate τ is above the mean-field threshold $\tau > \tau_c^{(1)}$. The inequality (11) indicates that the burst of the infection in the steady state is restricted by the underlying network, specifically, the largest eigenvalue λ_1 of the adjacency matrix A.

III. NUMERICAL AND SIMULATION RESULTS

We evaluate the mean-field method by comparing the approximation with the simulation of the exact Weibullian SIS process. The simulation is performed on an Erdős Rényi network, a scale-free network, and a rectangular grid network.

Figure 2 presents the prevalence of the Weibullian SIS process with $\alpha \to \infty$ in the long run, together with the NIMFA and the Markovian prevalence. The numerical solution of Eqs. (8) and (9) approximates the simulation results well, and the phase transition of the simulated process happens around the mean-field threshold $\tau_c^{(1)}$. Among all the three different

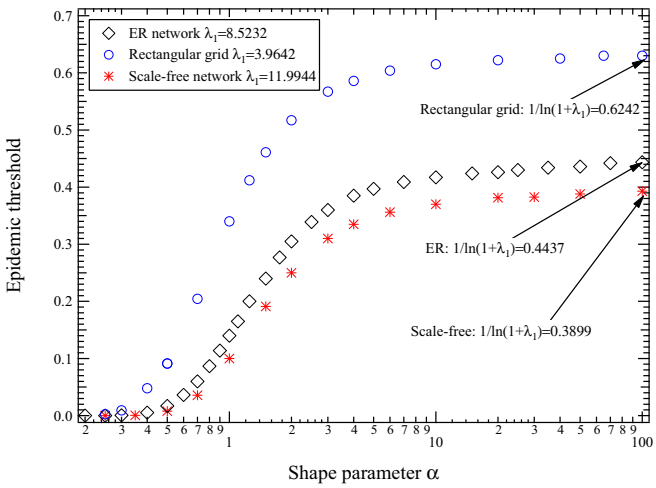


FIG. 3. The epidemic threshold vs the Weibull shape parameter α. The thresholds are obtained by simulation of 10^5 realizations. The simulation setup is the same as that in Fig. 2. The threshold is chosen as the value of the effective infection rate τ which leads to the maximum prevalence being around 0.001 at the last period.

networks, the accuracy of the mean-field approximation is worst in the rectangular grid network with a minimum largest eigenvalue $\lambda_1 = 3.9627$, and best in the scale-free network with a largest $\lambda_1 = 11.9944$. The simulations in Fig. 2 also show that the mean-field threshold $\frac{1}{\ln(1+\lambda_1)}$ is, just as for NIMFA, a lower bound.

Figure 3 shows the epidemic threshold of the Weibullian SIS process with different shape parameter α. As mentioned above, the epidemic threshold can be approximately zero, which agrees with the simulation results. With the increase of α, the epidemic threshold τ_c converges approximately to $\tau_c^{(1)}$.

IV. CONCLUSION

As a general model, the Weibullian SIS process can model a general non-Markovian SIS process by choosing a suitable shape parameter α. We study the process in the extreme situation $\alpha \to 0$ and ∞ to obtain an understanding of the influence of the underlying network on a general epidemic process. For an SIS process with an unknown infection process, our results reveal that the certainty about the extinction of infection is not possible even if the effective infection rate τ is small, but the infection can always persist on the network if the effective infection rate $\tau > 1/\ln(\lambda_1 + 1)$. Additionally, we obtain the properties of the synchronized epidemic process, i.e., the Weibullian SIS process with $\alpha \to \infty$, by the mean-field method.

ACKNOWLEDGMENT

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APPENDIX A: WHEN THE SHAPE PARAMETER $\alpha \to 0$

We consider the Weibullian SIS process with an infection rate $\beta > 0$. When $\alpha \to 0$, the distribution function of the infection time T is

$$\lim_{\alpha\to 0} F_T(x) = \lim_{\alpha\to 0} 1 - e^{-[\beta x \Gamma(1+1/\alpha)]^\alpha} = 1 - e^{-\lim_{\alpha\to 0}\Gamma(1+1/\alpha)^\alpha}. \tag{A1}$$

Since $\Gamma(1+1/\alpha)^\alpha = e^{\alpha \ln \Gamma(1+1/\alpha)}$, we invoke [19, (6.1.40)] the asymptotic formula $\ln\Gamma(z) \sim (z-\frac{1}{2})\ln z + O(z)$, and then we obtain

$$\alpha \ln \Gamma\left(1+\frac{1}{\alpha}\right) = \alpha\left(\frac{1}{\alpha}+\frac{1}{2}\right)\ln\left(\frac{1}{a}+1\right) + O(1) = -\ln\alpha - \frac{\alpha}{2}\ln\alpha + O(1).$$

Thus,

$$\lim_{\alpha\to 0}\Gamma\left(1+\frac{1}{\alpha}\right)^\alpha = \exp\left[\lim_{\alpha\to 0}\left(-\ln\alpha - \frac{\alpha}{2}\ln\alpha\right)\right] = \infty.$$

From the calculation above, $\lim_{\alpha\to 0} F_T(x) = 1$ for $x > 0$, i.e., $\lim_{\alpha\to 0}\Pr[T = 0] = 1$. Thus, when $\alpha \to 0$ and $\beta > 0$, an infected node asymptotically almost surely infects its neighbor consistently and all nodes will be infected. By a similar method, we can verify that the mean-field threshold $\frac{1}{\Gamma(1+1/\alpha)[\Gamma(\alpha+1)]^{1/\alpha}\lambda_1^{1/\alpha}}$ given in [9] tends to zero for an arbitrary small α.

APPENDIX B: PROOF OF THEOREM 1

Proof. We denote Eq. (9) by a function $\Phi : [0,1]^N \to [0,1]^N$ that $\mathbf{v}(n/\beta) = \Phi\{\mathbf{v}[(n-1)/\beta]\}$ and $v_j(n/\beta) = \Phi_j\{\mathbf{v}[(n-1)/\beta]\}$. We may verify that

$$\frac{\partial\Phi_j(\mathbf{x})}{\partial x_i}\Big|_{\mathbf{x}=\mathbf{0}} = \begin{cases} e^{-1/\tau} & \text{if } a_{ji} = 1 \text{ or } j = i \\ 0 & \text{if } a_{ji} = 0 \end{cases},$$

which is the element of the Jacobian matrix $J_\Phi(\mathbf{0})$ of the function Φ at $\mathbf{0}$ in the jth row and ith column. Thus, the Jacobian matrix is $J_\Phi(\mathbf{0}) = e^{-1/\tau}(A + I)$, and we assume that $\lambda_{\max}$ is the largest eigenvalue of the Jacobian $J_\Phi(\mathbf{0})$ in absolute value.

Since the network is connected, the matrix $J_\Phi(\mathbf{0})$ is irreducible. Thus, $\lambda_{\max}$ is the largest eigenvalue of $J_\Phi(\mathbf{0})$ by the Perron-Frobenius theorem [20], and then

$$\lambda_{\max} = e^{-1/\tau}(\lambda_1 + 1).$$

For the dynamical system $\mathbf{x}(n) = \Phi[\mathbf{x}(n-1)]$ in the form of (9), Ahn and Hassibi [14, Theorem 5.1] have indicated that $\mathbf{0}$ is globally stable and that $\lim_{n\to\infty}\mathbf{x}(n) = \mathbf{0}$ for any $\mathbf{x}(0) \in [0,1]^N$ when $\lambda_{\max} < 1$, while if $\lambda_{\max} > 1$ then there exists one and only one nonzero globally stable point such that $\mathbf{0} \prec \lim_{n\to\infty}\mathbf{x}(n)$ for any $\mathbf{x}(0) \in [0,1]^N$ and $\mathbf{x}(0) \neq \mathbf{0}$. Thus, the maximum infection probability $\mathbf{v}(n/\beta)$ of each time interval governed by Eq. (9) converges to $\mathbf{0}$ when $\lambda_{\max} < 1$, and $\mathbf{v}(n/\beta)$ converges to the unique nonzero constant infection probability $\mathbf{v}_\infty(0)$ when $\lambda_{\max} > 1$ and $\mathbf{v}(0) \neq \mathbf{0}$. Thus, $\lambda_{\max} = 1$ is the critical point at which the phase transition happens. Let $\lambda_{\max} = 1$, and we obtain

$$\tau = \frac{1}{\ln(\lambda_1 + 1)},$$

which is the epidemic threshold of the Weibullian SIS process with $\alpha \to \infty$. In each time interval in the steady state, the infection probability is $\mathbf{v}_\infty(t^*) = \mathbf{v}_\infty(0)e^{-\delta t^*}$, which follows from (8).

APPENDIX C: WHEN THE EFFECTIVE INFECTION RATE IS SMALL, $\tau < \tau_c^{(1)}$

From (9), the infection probability at $t^* = 0$ of the nth time interval is upper bounded by (C1):

$$v_j\left(\frac{n}{\beta}\right) \leqslant v_j\left(\frac{n-1}{\beta}\right)e^{-1/\tau} + \left[\sum_{i=1}^N a_{ji}v_i\left(\frac{n-1}{\beta}\right)e^{-1/\tau}\right]. \tag{C1}$$

From the inequality above, we have

$$\mathbf{v}\left(\frac{n}{\beta}\right) \preceq e^{-1/\tau}(A+I)\mathbf{v}\left(\frac{n-1}{\beta}\right) \preceq [e^{-1/\tau}(A+I)]^n\mathbf{v}(0) = \sum_{i=1}^N [e^{-1/\tau}(\lambda_i+1)]^n\mathbf{u}_i\mathbf{u}_i^T\mathbf{v}(0)$$

where $\lambda_1 \geqslant \cdots \geqslant \lambda_i \geqslant \cdots \geqslant \lambda_N$ are the eigenvalues of the matrix $e^{-1/\tau}(A + I)$, and $\mathbf{u}_i$ is the corresponding eigenvector

of λ_i. Thus, there exists a constant vector $\mathbf{z}$ where every element is positive, such that

$$\mathbf{v}\left(\frac{n}{\beta}\right) \preceq [e^{-1/\tau}(\lambda_1+1)]^n \mathbf{z}. \tag{C2}$$

We consider the inequality above for general $t = n/\beta + t^*$. Since $\mathbf{v}(t^* + n/\beta) = \mathbf{v}(n/\beta)e^{-\delta t^*}$ and $n = \beta(t - t^*)$, we have

$$\mathbf{v}(t) = \mathbf{v}\left(\frac{n}{\beta}\right)e^{-\delta t^*} \preceq [e^{-1/\tau}(\lambda_1+1)]^{\beta(t-t^*)}e^{-\delta t^*}\mathbf{z} \quad = [e^{-\delta}(\lambda_1+1)^\beta]^{t-t^*}(e^{-\delta})^{t^*}\mathbf{z} \quad \prec [e^{-\delta}(\lambda_1+1)^\beta]^t \mathbf{z}.$$

If the effective infection rate τ is below the mean-field epidemic threshold $\tau_c^{(1)}$, then $e^{-\delta}(\lambda_1+1)^\beta < 1$, and $[e^{-\delta}(\lambda_1+1)^\beta]^t\mathbf{z}$ is exponentially decreasing with time t.

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