IEEE Access
Multidisciplinary | Rapid Review | Open Access Journal

Received January 18, 2019, accepted February 3, 2019, date of publication February 11, 2019, date of current version March 12, 2019.

Digital Object Identifier 10.1109/ACCESS.2019.2898639

The Computational Approach for the Basic Reproduction Number of Epidemic Models on Complex Networks

JUNYUAN YANG[1] AND FEI XU[2]
[1]Complex Systems Research Center, Shanxi University, Taiyuan 030006, China
[2]Department of Mathematics, Wilfrid Laurier University, Waterloo, ON N2L 3C5, Canada

Corresponding author: Junyuan Yang (yangjunyuan00@126.com)

This work was supported in part the National Natural Science Foundation of China under Grant 61573016 and Grant 61203228, in part by the Shanxi Scholarship Council of China under Grant 2015-094, in part by the Shanxi Scientific Data Sharing Platform for Animal Diseases, and in part by the Startup Foundation for High-Level Personal of Shanxi University.

ABSTRACT The basic reproduction number plays an important role in exploring the dynamics of the epidemic models. Such value has been extensively used in the estimation of how severe an epidemic outbreak. Although several methods for calculating the basic reproduction number has been proposed, there isn't an effectively universal method to estimate such value. In this paper, we propose a general approach to calculate the explicit formulation of the basic reproduction number by the renewal equation. We apply such a method to estimate the basic reproduction number of many epidemic models on complex networks consisting of mean-field models, pairwise models, and edge-based compartmental models.

INDEX TERMS Complex networks, the basic reproduction number, the renewal equation.

I. INTRODUCTION

The concept of the basic reproduction number $\mathcal{R}_0$ [1], [2] has been defined as the numbers of secondary cases produced by a typical infected individual in a completely susceptible population. $\mathcal{R}_0$ is an epidemiologic metric for the study of dynamics of infectious diseases and is extensively used indicator of how severe an epidemic outbreak can be. $\mathcal{R}_0$ has the property coupling the process of contagion and the contact patterns of the population. In classical epidemic models, $\mathcal{R}_0$ marks the threshold property, the disease breaks out if $\mathcal{R}_0 > 1$, otherwise, it dies out. Furthermore, the final outbreak size, incidence and time to the peak prevalence of disease are always functions of $\mathcal{R}_0$. Therefore, many epidemiologists used $\mathcal{R}_0$ to design the control strategies including vaccination, sanitation, quarantine, etc.

Indeed, $\mathcal{R}_0$ equips with the important mission in study of epidemic dynamics. Many researches adopted some methods to estimate the value of $\mathcal{R}_0$. Generally, the definition of $\mathcal{R}_0$ couples three factors: the likelihood of infection per contact with a susceptible, the contact rate, and infectious duration. From these three points, there exist three main methods to estimate $\mathcal{R}_0$: calculation of the spectral radius of the next generation operator [3]–[5], analysis of the data on the time series of cases or stochastic processes [6]–[8], and analysis local stability of the disease-free equilibrium [9]–[12].

As we know, individuals contact pattern is an key factor to evaluation of $\mathcal{R}_0$. Such property has been addressed epidemic models on contact networks [6]–[12]. Network-based epidemic models concern the contact relationship between nodes (individuals or agents). Up to now, there are four-type epidemic models on networks: mean-field models [10]–[13], the pairwise models [16], [17], the edge-based compartmental models [15], [17] and the bond percolation [18]. For disunity of network-based models, there are not a universal method to estimate $\mathcal{R}_0$ until now. In view of previous studies, $\mathcal{R}_0$ for mean-field models are generally solved by the calculation the spectral radius of the next generation matrix $\mathcal{K} = FV^{-1}$ to estimate $\mathcal{R}_0$, where F denotes the transmission matrix and V represents the transition matrix; there are two ways to determine $\mathcal{R}_0$ for some pairwise models: directly given by the definition of $\mathcal{R}_0$, analysis the local stability of the disease-free equilibrium or the positivity of the endemic equilibrium. For any method, the process of calculation $\mathcal{R}_0$ is tedious and it is hard to give a reasonable interpretation. This paper is conducted to proposing an effectively universal

The associate editor coordinating the review of this manuscript and approving it for publication was Dominik Strzalka.

method to calculate $\mathcal{R}_0$ in terms of model parameters by renewal equation and Laplace transformation. We will show that $\mathcal{R}_0$ completely determines whether or not the disease based on networks breaks out or dies out.

II. RENEWAL EQUATIONS AND ITS PROPERTIES

The classical renewal equation on positive half-line is defined by

$$f(t) = q(t) + \int_0^t f(t-s)G(s)ds = q(t) + f * G, \quad t \geq 0. \tag{1}$$

where $q(t)$ is a measurable function and bounded on any finite interval, and $G(s)$ is a distribution function on $\mathbb{R}_+$, named by the forcing function and the distribution generating the renewal equation. $*$ denotes the convolution of two functions f and G. Taking Laplace transform on both sides of (1), we have the following lemma.

Lemma 1: Let $\widehat{f(\lambda)} = \int_0^\infty e^{-\lambda t} f(t)dt$ *and* $\widehat{G(\lambda)} = \int_0^\infty e^{-\lambda t} G(t)dt$ *be Laplace transforms for* f *and* G*, respectively. Then the following relation holds*

$$\widehat{f(\lambda)} = \frac{\widehat{q(\lambda)}}{1 - \widehat{G(\lambda)}} = \widehat{q(\lambda)} \sum_{n=1}^{\infty} \widehat{G_n(\lambda)}, \tag{2}$$

where

$$\widehat{G_n(\lambda)} = \widehat{G^{*n}(\lambda)} = \widehat{G(\lambda)} * \widehat{G(\lambda)} * \widehat{G(\lambda)} \cdots * \widehat{G(\lambda)}.$$

From Lemma 1, we conclude that if $0 < \widehat{G(0)} < 1$, then $\widehat{f(0)}$ is convergent as $n \to \infty$, otherwise, it is divergent if $0 < \widehat{G(0)} < 1$.

Now, we are in a position to adopt the renewal equation to calculate $\mathcal{R}_0$ for a SIS epidemic model. As we know, a classical SIS-type epidemic model obeys the following equations

$$\frac{dS(t)}{dt} = \Lambda - \beta S(t)I(t) - \mu S(t) + \gamma I(t), \tag{3}$$

$$\frac{dI(t)}{dt} = \beta S(t)I(t) - (\mu + \alpha + \gamma)I(t), \tag{4}$$

where S and I denote the density of the susceptible and infected individuals, Λ represents the input rate, α denotes the disease-caused mortality, μ is the natural death rate, β represents the transmission rate. $\mathcal{R}_0$ is defined as the expected number of secondary cases that an infected individual infects if he or she enters a entirely susceptible population. Observe that system (3)-(4) has a disease-free equilibrium $E_0 = (S^0, 0) = (\frac{\Lambda}{\mu}, 0)$. Linearizing equation (4) around the disease-free equilibrium E_0, one arrives at

$$\frac{dI(t)}{dt} = \beta S^0 I(t) - (\mu + \alpha + \gamma)I(t). \tag{5}$$

Solving equation (5) yields

$$\begin{aligned} I(t) &= I_0 e^{-(\mu+\alpha+\gamma)t} + \int_0^t \beta S^0 I(s) e^{-(\mu+\alpha+\gamma)(t-s)} ds \\ &= q(t) + \int_0^t I(t-s)G(s)ds, \end{aligned} \tag{6}$$

where $q(t) = I_0 e^{-(\mu+\alpha+\gamma)t}$ and $G(t) = \beta S^0 e^{-(\mu+\alpha+\gamma)t}$. Hence, (6) is a renewal equation. From Lemma 1, together with [14, Th. 2.6], it follows that equation (6) has the solution in form of

$$I(t) = I_0 e^{\xi^* t}(1 + \Omega(t)), \tag{7}$$

where

$$I_0 \geq 0 \quad \text{and} \lim_{t \to +\infty} \Omega(t) = 0.$$

The parameter ξ^* denotes the intrinsic Malthusian parameter of the infected population determining the growth or reduction of the infected population. The following lemma establishes the relations between ξ^* and $\widehat{G(0)}$.

Lemma 2: Let ξ^* *be defined in* (7)*. The following relationships hold.*

(1) $\xi^* > 0$ *if and only if* $\widehat{G(0)} > 1$;
(2) $\xi^* < 0$ *if and only if* $\widehat{G(0)} < 1$;
(3) $\xi^* = 0$ *if and only if* $\widehat{G(0)} = 1$.

Therefore, we can define the basic reproduction number

$$\mathcal{R}_0 = \widehat{G(0)}. \tag{8}$$

From Lemma 2, $\mathcal{R}_0$ determines whether or not the disease persists or eradicates. Now, we are going to extend this method to calculate the basic reproduction number $\mathcal{R}_0$ on complex networks.

III. APPLICATIONS

As we mentioned in Section I, there are three kinds of epidemic models on complex networks: mean-fields models, pairwise models and edge-compartmental models. Now, we are in a position to calculate the basic reproduction number $\mathcal{R}_0$ of the three such models. Epidemic models on complex networks mainly concern on the relations of the infected nodes or the variations of the infected edges. In the following, we pay much efforts on deriving the variation of infected edges and building the renewal equations associated to infected edges.

A. $\mathcal{R}_0$ of MARKOVIAN EPIDEMIC MODELS ON COMPLEX NETWORKS

A mean-field SIS epidemic model on complex networks proposed by Wang and Dai [19] takes in the form of

$$\begin{cases} \dfrac{dS_k(t)}{dt} = -\beta k S_k(t)\Theta(t) + \gamma I_k(t), \\ \dfrac{dI_k(t)}{dt} = \beta k S_k(t)\Theta(t) - \gamma I_k(t), \end{cases} \tag{9}$$

with initial condition

$$S_k(0) = S_{k0} \geq 0, \quad I_k(0) = I_{k0} \geq 0,$$

where the total population is divided into two classes: susceptible nodes and infected nodes with degree k, denoted by $S_k(t)$ and $I_k(t)$, respectively. Here, β and γ denote the transmission rate and recovery rate, respectively, and $p(l|k)$ represents the probability of some node with degree k connecting a node

with degree l. The average number of infected individuals with degree l connecting with susceptible node with degree with k is formulated in terms of

$$\Theta(t) = \sum_{k=1}^{n} p(l|k) I_l(t).$$

If we assume that the network is of degree unrelated, then $p(l|k) = \frac{lp(l)}{\langle k \rangle}$, where $\langle k \rangle = \sum_{k=1}^{n} kp(k)$. For a mean-field SIS epidemic model on complex networks, $\Theta(t)$ denotes the variation of the infected vertexes. Obviously, system (9) always has a disease-free equilibrium $E_0 = (S_k^0, 0) = (1, 0)$, for each $n \in \mathbb{N}$. From system (9), we can easily derive the infected edges in the disease invasion phase

$$\frac{d\Theta(t)}{dt} = \frac{\beta\langle k^2 \rangle}{\langle k \rangle}\Theta(t) - \gamma\Theta(t), \quad \Theta(0) = \Theta_0. \tag{10}$$

Solving (10), we have

$$\begin{aligned}\Theta(t) &= \Theta_0 e^{-\gamma t} + \frac{\beta\langle k^2 \rangle}{\langle k \rangle}\int_0^t \Theta(s) e^{-\gamma(t-s)} ds \\ &= \Theta_0 e^{-\gamma t} + \frac{\beta\langle k^2 \rangle}{\langle k \rangle}\int_0^t \Theta(t-s) e^{-\gamma s} ds. \end{aligned} \tag{11}$$

Denote

$$q^M(t) = \Theta_0 e^{-\gamma t} \quad \text{and } G^M(t) = \frac{\beta\langle k^2 \rangle}{\langle k \rangle} e^{-\gamma t}$$

Thus, equation (11) can be rewritten as

$$\Theta(t) = q^M(t) + \int_0^t \Theta(t-s) G^m(s) ds. \tag{12}$$

Clearly, equation (12) is a renewal equation. From the similar definition for $\mathcal{R}_0$ in (8), the basic reproduction number $\mathcal{R}_0^M$ can be defined by

$$\mathcal{R}_0^M = \widehat{G_M(0)} = \frac{\langle k^2 \rangle}{\langle k \rangle}\frac{\beta}{\gamma},$$

which coincides the definition by Wang and Dai [19].

For the SIS homogeneous pairwise model, the model couples the nodes and the edges in terms of

$$\begin{aligned}\frac{d[S](t)}{dt} &= -\beta[SI](t) + \gamma[I](t), \\ \frac{d[I](t)}{dt} &= \beta[SI](t) - \gamma[I](t), \\ \frac{d[SS](t)}{dt} &= 2\gamma[SI](t) - 2\beta[SS](t)\frac{n-1}{n}\frac{[SI](t)}{[S](t)}, \\ \frac{d[II](t)}{dt} &= -2\gamma[II](t) + 2\beta[SI](t)\left(1 + \frac{n-1}{n}\frac{[SI](t)}{[S](t)}\right), \\ \frac{d[SI](t)}{dt} &= \gamma[II](t) - \gamma[SI](t) - \beta[SI](t)\left(1 + \frac{n-1}{n}\right. \\ &\quad \left.\times\frac{[SI](t)}{[S](t)}\right) + \beta[SS](t)\frac{n-1}{n}\frac{[SI](t)}{[S](t)}, \end{aligned} \tag{13}$$

where $[A](A = S, I)$ denote the expected values of susceptible and infected values, $[AB](A = S, I; B = S, I)$ represent the expected number of edges in a given status. In model (16), there are two constraints conditions:

$$2[SI](t) + [II](t) + [SS](t) = nN, \quad [S](t) + [I](t) = N,$$

where N is a positive constant.

Obviously, system (16) has a disease-free equilibrium $E_0 = ([S]^0, 0, [SS]^0, 0, 0) = (N, 0, nN, 0, 0)$. Since we focus on the variations of the infected edges on complex networks, we need to linearize $[SI]$ and $[II]$ edges around the disease invasion phase

$$\begin{aligned}\frac{d[II](t)}{dt} &= -2\gamma[II](t) + 2\beta[SI](t), \\ \frac{d[SI](t)}{dt} &= \gamma[II](t) + [\beta(n-2) - \gamma][SI](t). \end{aligned} \tag{14}$$

Solving (14), we obtain

$$[II](t) = [II]_0 e^{-2\gamma t} + 2\beta\int_0^t [SI](s) e^{-2\gamma(t-s)} ds, \tag{15}$$

and

$$\begin{aligned}[SI](t) = [SI]_0 e^{-\gamma t} + \int_0^t (\gamma[II](s) + \beta(n-2) \\ \times[SI](s)) e^{-\gamma(t-s)} ds. \end{aligned} \tag{16}$$

Substituting (15) into (16), we have

$$\begin{aligned}[SI](t) &= [SI]_0 e^{-\gamma t} + \gamma[II]_0 \int_0^t e^{-\gamma(t+s)} ds \\ &\quad + \int_0^t \left(2\beta\gamma\int_0^s [SI](a) e^{-2\gamma(s-a)} da \right. \\ &\quad \left. + \beta(n-2)[SI](s)\right) e^{-\gamma(t-s)} ds \\ &= [SI]_0 e^{-\gamma t} + \gamma[II]_0 \int_0^t e^{-\gamma(t+s)} ds \\ &\quad + \int_0^t \left(2\beta\gamma\int_0^{t-s} [SI](a) e^{-2\gamma(t-s-a)} da \right. \\ &\quad \left. + \beta(n-2)[SI](t-s)\right) e^{-\gamma s} ds. \end{aligned} \tag{17}$$

From equation (18), it is hard to define the last term as a convolution style. Define

$$q^W(t) = [SI]_0 e^{-\gamma t} + \gamma[II]_0 \int_0^t e^{-\gamma(t+s)} ds.$$

In order to derive the basic reproduction number $\mathcal{R}_0^W$, we take Laplace transform on both sides of (18) to obtain

$$\begin{aligned}\widehat{[SI](\lambda)} &= \widehat{q^W(\lambda)} + 2\beta\gamma\int_0^\infty e^{-\lambda t}\int_0^t\int_0^{t-s} [SI](a) \\ &\quad \times e^{-2\gamma(t-s-a)} da e^{-\gamma s} ds dt + \frac{\beta(n-2)}{\gamma}\widehat{[SI](\lambda)} \\ &= \widehat{q^W(\lambda)} + 2\beta\gamma\int_0^\infty e^{-(\lambda+\gamma)s}\int_0^\infty\int_0^s [SI](a) \\ &\quad \times e^{-2\gamma(t-a)} da e^{-\lambda t} dt ds + \frac{\beta(n-2)}{\gamma}\widehat{[SI](\lambda)} \\ &= \widehat{q^W(\lambda)} + 2\beta\gamma\int_0^\infty e^{-(\lambda+\gamma)s} ds\int_0^\infty e^{-(2\gamma+\lambda)t} dt \end{aligned}$$

$$\times \int_0^\infty e^{-\lambda a}[SI](a)da + \frac{\beta(n-2)}{\gamma}\widehat{[SI](\lambda)}$$
$$= \widehat{q^W(\lambda)} + \left(\frac{2\beta\gamma}{(\lambda+\gamma)(2\gamma+\lambda)} + \frac{\beta(n-2)}{\gamma}\right)\widehat{[SI](\lambda)}.$$

Therefore, the basic reproduction number can be defined by the last term of the above equation provided $\lambda = 0$

$$\mathcal{R}_0^W = \frac{\beta(n-1)}{\gamma} = \frac{\beta}{\gamma}\frac{n^2-1}{n},$$

which is agreement with the definition by Kiss *et al.* [17].

For Edge-based compartmental model, Miller [15] propose a model in terms of

$$\begin{aligned}
\frac{d\theta(t)}{dt} &= -\beta\phi_I(t),\\
\phi_I(t) &= \theta(t) - (1-\rho)\frac{\psi'(\theta(t))}{\langle k\rangle} - \frac{\gamma}{\beta}(1-\theta(t)),\\
S(t) &= (1-\rho)\psi(\theta(t)),\\
I(t) &= 1 - S(t) - R(t),\\
\frac{dR(t)}{dt} &= \gamma I(t),
\end{aligned} \tag{18}$$

with initial condition

$$\theta(0) = 1, \quad R(0) = 0.$$

where $\psi(x) = \sum_k p(k)x^k$ is the probability generating function of the degree distribution, θ denotes the probability of a randomly chosen partner v of a randomly chosen individual u has not transmitted infection to u, ϕ_I stands the probability that v is infected and has not transmitted infection to u; ρ represents the fraction of an arbitrary initial infection.

Linearizing system (18) at disease free equilibrium $E_0 = (1, 0)$, we have for $\rho = 0$

$$\frac{d\theta(t)}{dt} = -\beta\phi_I(t), \tag{19}$$
$$\phi_I(t) = (1 - \frac{\psi''(1)}{\langle k\rangle} + \frac{\gamma}{\beta})\theta(t). \tag{20}$$

Solving (19) and (20), we have

$$\begin{aligned}
\theta(t) &= \theta_0 e^{-(\beta+\gamma)t} + \beta\frac{\psi''(1)}{\langle k\rangle}\int_0^t \theta(a)e^{-(\beta+\gamma)(t-a)}da\\
&= q^{EM}(t) + \int_0^t G^{EM}(a)\theta(t-a)da,
\end{aligned} \tag{21}$$

where $q^{EM}(t) = \theta_0 e^{-(\beta+\gamma)t}$ and $G^{EM}(t) = \beta\frac{\psi''(1)}{\langle k\rangle}e^{-(\beta+\gamma)(t)}$. Hence the basic reproduction number for edge-based compartmental model is given by

$$\mathcal{R}_0^{EM} = \frac{\psi''(1)}{\langle k\rangle}\frac{\beta}{\beta+\gamma},$$

which is consistent with the value estimated by Miller [15].

B. $\mathcal{R}_0$ OF NON-MARKOVIAN EPIDEMIC MODELS ON COMPLEX NETWORKS

Since exponentially distributed infectious period with constant transmission rates are not realistic, infection age accounting for general transmission and recovery processes has been integrated into some epidemic models describing non-Markovian transmission process on complex networks [9], [17]. An SIS epidemic model with non-markovian on complex networks proposed by Yang *et al.* [9] reads

$$\begin{aligned}
\frac{dS_k(t)}{dt} &= -kS_k(t)\Theta(i) + \int_0^\infty \gamma(a)i_k(t,a)da,\\
\frac{\partial i_k(t,a)}{\partial t} + \frac{\partial i_k(t,a)}{\partial a} &= -\gamma(a)i_k(t,a),\\
i_k(t,0) &= kS_k(t)\Theta(i(t,\cdot)),
\end{aligned} \tag{22}$$

where the total population is divided into two classes: the density of susceptible nodes and the density of the infected nodes, denoted by $S_k(t)$ and $i_k(t,a)$, respectively. $\beta(a)$ and $\gamma(a)$ denote the transmission rate and recovery rate with respect to the infection age a.

Integrating the second equation of (22) along the characteristic line, we have for each $k \in \mathbb{N}$

$$i_k(t,a) = \begin{cases} b_k(t-a)\pi(a), & t > a,\\ i_{k0}(a-t)\dfrac{\pi(a)}{\pi(a-t)}, & t \le a, \end{cases} \tag{23}$$

where $\pi(a) = e^{-\int_0^a \gamma(s)ds}$ and $b_k(t) = S_k(t)\Theta(i(t,\cdot))$.

Similarly, $\Theta(i(t,\cdot)$ denotes the variations of the infected vertexes. From the expression of $\Theta(i(t,\cdot))$, it follows that

$$\Theta(i(t,\cdot)) = \frac{1}{\langle k\rangle}\sum_{k=1}^n k^2p(k)\int_0^t \beta(a)S_k(t-a)\Theta(i(t-a,\cdot)) \times \pi(a)da + q^{NM}(t), \tag{24}$$

where $q^{NM}(t) = \frac{1}{\langle k\rangle}\sum_{k=1}^n kp(k)\int_0^\infty \beta(a+t)\frac{\pi(a+t)}{\pi(a)}da$. Linearizing at the disease-free equilibrium $E_0^P = (1, 0)$, we obtain that

$$\begin{aligned}
\Theta(i(t,\cdot)) &= \frac{1}{\langle k\rangle}\sum_{k=1}^n k^2p(k)\int_0^t \beta(a)\Theta(i(t-a,\cdot))\pi(a)da\\
&\quad + q^{NM}(t)\\
&= \int_0^t G^{NM}(a)\Theta(i(t-a,\cdot))da + + q^{NM}(t),
\end{aligned} \tag{25}$$

where $G^{NM}(t) = \frac{1}{\langle k\rangle}\sum_{k=1}^n k^2p(k)\beta(t)\pi(t)$. The next generation operator reads as

$$\begin{aligned}
\mathcal{R}_0^{NM} &= \widehat{G^{NM}(0)} = \frac{1}{\langle k\rangle}\sum_{l=1}^n l^2p(l)\int_0^\infty \beta(s)\pi(s)ds\\
&= \frac{\langle k^2\rangle K^{NM}}{\langle k\rangle},
\end{aligned}$$

where we denote $K^{NM} = \int_0^\infty \beta(a)e^{-\int_0^a \gamma(s)ds}da$.

For a SIR pairwise network model with non-markovian recovery, the model proposed by Röst *et al.* [16] takes in the form of

$$\begin{aligned}
\frac{d[S](t)}{dt} &= -\beta[SI](t),\\
\frac{d[SS](t)}{dt} &= -2\beta\frac{n-1}{n}\frac{[SS](t)[SI](t)}{[S](t)},\\
\frac{d[I](t)}{dt} &= \beta[SI](t)-\beta\int_0^t[SI](t-a)f(a)da-\int_t^\infty\psi(a-t)\\
&\quad\times\frac{f(a)}{\xi(a-t)}da,\\
\frac{d[SI](t)}{dt} &= \beta\frac{n-1}{n}\frac{[SS](t)[SI](t)}{[S](t)}-\beta\frac{n-1}{n}\frac{[SI](t)}{[S](t)}[SI](t)\\
&\quad-\beta[SI](t)-\beta\frac{n-1}{n}\int_0^t\frac{[SS](t-a)[SI](t-a)}{[S](t-a)}\\
&\quad\times e^{-\int_{t-a}^t(\beta\frac{n-1}{n}\frac{[SI](s)}{[S](s)}+\beta)ds}f(a)da-\int_t^\infty\frac{n}{N}[S]_0\\
&\quad\times\psi(a-t)e^{-\int_0^t(\beta\frac{n-1}{n}\frac{[SI](s)}{[S](s)}+\beta)ds}\frac{f(a)}{\xi(a-t)}da, \quad (26)
\end{aligned}$$

where A and $[AB]$ have the same biological meaning as in system (16). The recovery process is a non-Markovian with a cumulative distribution function $F(a)$ and probability function $f(a)$. $\xi(a) = 1 - F(a)$ represents the survival function.

Using the similar approach, we obtain the linearized equation at the disease-free equilibrium $E_0 = (N, nN, 0, 0)$ with respect to infected edges as follows

$$\begin{aligned}
\frac{d[SI](t)}{dt} &= \beta(n-1)[SI](t)-\beta[SI](t)\\
&\quad-\beta(n-1)\int_0^t[SI](t-a)e^{-\beta a}f(a)da. \quad (27)
\end{aligned}$$

Solving (27) yields

$$\begin{aligned}
[SI](t) &= q^{NP}(t)+\int_0^t\Big(\beta(n-1)[SI](s)-\beta(n-1)\\
&\quad\times\int_0^t[SI](s-a)e^{-\beta a}f(a)da\Big)e^{-\beta(t-s)}ds, \quad (28)
\end{aligned}$$

where $q^{NP} = [SI]_0e^{-\beta t}$. Taking Laplace transformation on both sides of (28), we arrive at

$$\begin{aligned}
\widehat{[SI](\lambda)} &= \widehat{q^{NP}(\lambda)}+\beta(n-1)\frac{\widehat{[SI](\lambda)}}{\lambda+\beta}\\
&\quad\times\left(1-\int_0^\infty f(a)e^{-(\lambda+\beta)a}da\right).
\end{aligned}$$

Therefore, the basic reproduction number is defined by

$$\mathcal{R}_0^{NP} = \left(\frac{n^2-n}{n^2}\right)\left(1-K^{NP}\right),$$

where $K^{NP} = \int_0^\infty f(a)e^{-\beta a}da$. $\mathcal{R}_0^{NP}$ coincides with the definition of $\mathcal{R}_0^{NP}$ by Röst *et al.* [16].

TABLE 1. The formulation of $\mathcal{R}_0$.

Models	Markovian	Non-markovian
Mean-field model	$\frac{\beta}{\gamma}\frac{\langle k^2\rangle}{\langle k\rangle}$	$K^{NM}\frac{\langle k^2\rangle}{\langle k\rangle}$
Pairwise model	$\frac{\beta}{\gamma}\left(\frac{n^2-n}{n}\right)$	$(1-K^{NP})\left(\frac{n^2-n}{n}\right)$
Edge-compartmental model	$\frac{\beta}{\beta+\gamma}\frac{\langle k^2-k\rangle}{\langle k\rangle}$	-

IV. CONCLUSION

Roughly speaking, $\mathcal{R}_0$ is the spectral radius of a next generation operator $\mathcal{K}$. In review of the obtained results, there are two main pathways to estimate $\mathcal{R}_0$: calculation of the spectral radius of $\mathcal{K} = FV^{-1}$, where F and V are defined in Section I; direct estimation from the definition of $\mathcal{R}_0$. We have seen that the calculation process for the former one is tedious, while the later one is hard to be tractable. In this paper, we proposed an effectively universal method to compute the explicit formulation of the basic reproduction number $\mathcal{R}_0$ of epidemic models on complex networks. Actually, the epidemic models based on networks mainly concern with the variations of the edges (the relationship between nodes). Therefore, the steps for the calculation of $\mathcal{R}_0$ is as follows:

- Calculate the disease-free equilibrium of the proposed model;
- Linearize the proposed model in disease related edges phase;
- Solve the linearizing system and obtain the renewal equation;
- Take Laplace transformation and obtain the reproduction number.

Following the mentioned processes, we have calculated the explicit expressions of $\mathcal{R}_0$ for epidemic models on complex networks including mean-field models, pairwise models and edge-compartmental models, even if epidemic models with markovian and non-markovian distribution processes. Lemma 2 ensures that if $\mathcal{R}_0 < 1$ then the disease-free equilibrium E_0 is locally asymptotically stable. The formulation $\mathcal{R}_0$ for epidemic models on complex networks takes in Table 1.

Clearly, the proposed method in this paper is more concise than the method by analyzing the positivity of the endemic equilibrium in [17] and [19], and it is more specific and tractable than the one by the definition of the next operator [16], [17]. Furthermore, we clarify that the proposed method is effective for compartmental models on complex networks, but it is useless for the individual-based epidemic models [8]. However, this method is not limited to the simple structures including SIS and SIR. In fact, some models with sophisticated structures such as SIRS, vertical transmission, even if competing models are useful as long as one captures the variations of the disease related edges and follows the steps in shadow area.

ACKNOWLEDGMENT

The authors would like to thank the editor and the anonymous reviewers for their constructive comments and suggestions, which improve the quality of this paper.

REFERENCES

[1] P. L. Delamater, E. J. Street, T. F. Leslie, Y. T. Yang, and K. H. Jacobsen, "Complexity of the basic reproduction number (R0)," *Emerg. Infect. Dis.*, vol. 25, no. 1, pp. 1–4, Jan. 2019.

[2] O. Diekmann and J. A. P. Heesterbeek, *Mathematical Epidemiology of Infectious Diseases: Model Building, Analysis and Interpretation*. Chichester, U.K.: Wiley, 2000.

[3] P. van den Driessche and J. Watmough, "Reproduction numbers and subthreshold endemic equilibria for compartmental models of disease transmission," *Math. Biosci.*, vol. 180, no. 12, pp. 29–48, Nov./Dec. 2002.

[4] P. van Den Driessche and J. Watmough, "Further notes on the basic reproduction number," *Mathematical Epidemiology* (Lecture Notes in Mathematics), vol. 1945. Berlin, Germany: Springer, pp. 159–178, 2008.

[5] O. Diekmann, J. A. P. Heesterbeek, and J. A. J. Metz, "On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations," *J. Math. Biol.*, vol. 28, no. 4, pp. 365–382, 1990.

[6] N. G. Becker and K. Dietz, "Reproduction numbers and critical immunity levels for epidemics in a community of households," in *Proc. Athens Conf. Appl. Probab. Time Ser. Anal.* (Lecture Notes in Statistics), vol. 114, C. C. Heyde, Y. V. Prohorov, R. Pyke, S. T. Rachev, Eds. New York, NY, USA: Springer, 1996, pp. 267–276.

[7] E. Volz and L. A. Meyers, "Epidemic thresholds in dynamic contact networks," *J. Roy. Soc. Interface*, vol. 6, pp. 233–241, Jul. 2008.

[8] M. J. Keeling and B. T. Grenfell, "Individual-based perspectives on R_0," *J. Theor. Biol.*, vol. 203, pp. 51–61, Mar. 2000.

[9] J. Y. Yang, Y. M. Chen, and F. Xu, "Effect of infection age on an SIS epidemic model on complex networks," *J. Math. Biol.*, vol. 73, pp. 1227–1249, Nov. 2016.

[10] A. L. Barabási and R. Albert, "Emergence of scaling in random networks," *Science*, vol. 286, pp. 509–512, Oct. 1999.

[11] Z. Jin, G.-Q. Sun, and H. Zhu, "Epidemic models for complex networks with demographics," *Math. Biosci. Eng.*, vol. 11, no. 6, pp. 1295–1317, Dec. 2014.

[12] R. Pastor-Satorras and A. Vespignani, "Epidemic spreading in scale-free networks," *Phys. Rev. Lett.*, vol. 86, pp. 3200–3203, Apr. 2001.

[13] Y. Wang, Z. Jin, Z. Yang, Z.-K. Zhang, T. Zhou, and G.-Q. Sun, "Global analysis of an SIS model with an infective vector on complex networks," *Nonlinear Anal., Real World Appl.*, vol. 13, pp. 543–557, Apr. 2012.

[14] M. Iannelli and F. Milner, *The Basic Approach to Age-Structured Population Dynamics: Models, Methods and Numerics* (Lecture Notes on Mathematical Modelling in the Life Sciences). Dordrecht, The Netherlands: Springer, 2016. [Online]. Available: https://link.springer.com/book

[15] J. C. Miller, "Mathematical models of SIR disease spread with combined non-sexual and sexual transmission routes," *Infectious Disease Model.*, vol. 2, pp. 35–55, Feb. 2017.

[16] G. Röst, Z. Vizi, and I. Z. Kiss, "Pairwise approximation for SIR-type network epidemics with non-Markovian recovery," *Proc. Roy. Soc. Math. Phys. Eng. Sci.*, vol. 474, no. 2210, Feb. 2018, Art. no. 20170695.

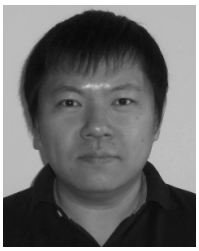
[17] I. Z. Kiss, J. C. Miler, and P. L. Simon, *Mathematics Epidemics Netw. from Exact to Approx. Models, Interdiscipl. Appl. Mathematics*. Cham, Switzerland: Springer, 2017. Available: [Online]. Available: https://link.springer.com/book

[18] M. E. J. Newman, "Spread of epidemic disease on networks," *Phys. Rev. E, Stat. Phys. Plasmas Fluids Relat. Interdiscip. Top.*, vol. 66, no. 1, Jul. 2002, Art. no. 016128.

[19] L. Wang and G.-Z. Dai, "Global stability of virus spreading in complex heterogeneous networks," *SIAM J. Appl. Math.*, vol. 68, no. 5, pp. 1495–1502, 2008.

JUNYUAN YANG held a Postdoctoral position at the Department of Mathematics, Wilfrid Laurier University, from 2012 to 2013. From 2014 to 2015, he was a Visiting Scholar with the Department of Mathematics, University of Florida. He is currently an Associate Professor with the Complex Systems Research Center, Shanxi University, China. He has over 50 publications in various international journals. His main research interests include mathematical biology, epidemiology, complex networks, and game theory. He is an active Researcher, and he is working on the study of dynamics of the epidemic models with Markovian and non-Markovian on complex networks.

Dr. Yang has served as an Assistant Editor for some academic journals.



FEI XU received the Ph.D. degree from Western University (University of Western Ontario), London, ON, Canada, in 2008.

From 2010 to 2014, he held a Postdoctoral position at the Department of Mathematics, Wilfrid Laurier University. Since 2015, he has been with the Wilfrid Laurier University. He has published over 40 papers in academic journals. His research interests include biomathematics, game theory, nonlinear dynamics, differential equations, data analytic, sociology, and mathematical modeling.