# An Upper Bound for the Epidemic Threshold in Exact Markovian SIR and SIS Epidemics on Networks

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*Abstract*— Exploiting the power of the expectation operator and indicator (or Bernoulli) random variables, we present the exact governing equations for both the SIR and SIS epidemic models on *networks*. Although SIR and SIS are basic epidemic models, deductions from their exact stochastic equations without making approximations (such as the common mean-field approximation) are scarce. An exact analytic solution of the governing equations is highly unlikely to be found (for any network) due to the appearing pair (and higher order) correlations. Nevertheless, the maximum average fraction $y_I$ of infected nodes in both SIS and SIR can be written as a quadratic form of the graph's Laplacian. Only for regular graphs, the expression for the maximum of $y_I$ can be simpled to exhibit the explicit dependence on the spectral radius. From our new Laplacian expression, we deduce a general upper bound for the epidemic SIS threshold in any graph.

## I. INTRODUCTION

Although the Susceptible-Infected-Removed (SIR) and the Susceptible-Infected-Susceptible (SIS) model are basic corner-stones in epidemics (see e.g. [1]–[6]), exact stochastic equations for SIR have, to the best of our knowledge, not been published yet for an arbitrary network, while for SIS, we refer to [7]–[9]. A network is described by an adjacency matrix $A$, with degree vector $D = (d_1, d_2, \ldots, d_N)$ where $d_k$ is the degree of node $k$. For simplicity, we assume an undirected network ($A = A^T$) that does not change over time. In addition to the many applications ranging from cyber security over information diffusion [10] to biological diseases [1], [5], we explore these (relatively) simple epidemic processes on graphs to understand the influence of the topology of complex networks [11] on properties of a dynamic process. First, we describe both the SIS and SIR model on any network in a stochastic, Markovian setting and refer for non-Markovian SIS epidemics to [12], [13].

In a SIS epidemic process, the viral state of a node $i$ at time $t$ is specified by a Bernoulli random variable $X_i(t) \in \{S, I\}$: $X_i(t) = S$ for a healthy, but susceptible node and $X_i(t) = I$ for an infected node. A node $i$ at time $t$ can be in one of the two states: *infected*, with probability $v_i(t) = \Pr[X_i(t) = I]$ or *healthy*, with probability $1 - v_i(t)$, but susceptible to the infection. We assume that the curing process per node $i$ is a Poisson process with rate $\delta$ and that

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the infection rate per link is a Poisson process with rate $\beta$. Obviously, only when a node is infected, it can infect its direct neighbors, that are still healthy. Both the curing and infection Poisson process are independent. The effective infection rate is defined by $\tau = \frac{\beta}{\delta}$. This is the general continuous-time description of the simplest type of a SIS epidemic process on a network.

In the SIR model, a node can be in one of the three states. When a node $j$ is healthy, but susceptible to the virus, at time $t$, his state $Y_j = S$. A node $j$ can be infected, $Y_j = I$, by its direct neighbors that are infected. The infection is modelled by a Poisson process with rate $\beta$. Finally, an infected node $j$ can be cured, after which it is removed from the infection process, $Y_j = R$. The curing is modelled by a Poisson process with rate $\delta$. All Poisson processes are independent. This formulation describes a continuous-time SIR process on a graph.

There exist other formulations of the SIR process. For example, the discrete-time counter part, in which a node is removed at the end of each time-slot and infected neighbors can infect a susceptible node with probability $p$, is termed a Reed-Frost process and is related to bond percolation [14]. Draief and Massoulié [15] show that a Reed-Frost process is related to the growth of an Erdős-Rényi graph. The SIR process is also related to a Markov discovery process on a graph (see [16, p. 349-351]). Newman [14] has presented a generating function approach for SIR, though implicitly assuming a mean-field approximation. The above Markovian description of SIS and SIR, based on independent Poisson processes, seems the most general one that still allows us to write the general governing equations for any graph. Deviating from a Markov process, by choosing other than the exponential interaction time (for infection and/or curing, see [12], [13]) or by incorporating dependencies between the infection and curing process, will complicate the analysis considerably. This argument provides the main motivation to explore how far we can push the analysis to obtain physical insight.

## II. GOVERNING EQUATIONS

In this paper, we analyse the SIR and SIS process rigorously and exploit the power of the (linear) expectation operator $E[.]$ and the indicator random variable $1_x$ (which equals one if the condition $x$ is true, else it is zero) to remain closer to the physics of the epidemic process. The SIR governing equation for the probability that a node $j$ is

infected reads

$$\frac{d\Pr[Y_j = I]}{dt} = E\left[-\delta 1_{\{Y_j=I\}} + 1_{\{Y_j=S\}}\beta\sum_{k=1}^{N} a_{kj}1_{\{Y_k=I\}}\right]$$
(1)

where the time-dependence of $Y_j(t)$ has been omitted for simplicity. In words, the change in the probability that a node $j$ is infected at time $t$ equals the expectation of (a) the rate $\beta$ times the number of infected neighbors (specified by the adjacency matrix element $a_{kj}$), given that node $j$ is susceptible minus (b) the rate $\delta$ given that the infected node is cured (and thereafter removed). Next, the dynamic process that removes nodes satisfies

$$\frac{d\Pr[Y_j = R]}{dt} = E\left[\delta 1_{\{Y_j=I\}}\right] = \delta\Pr[Y_j = I]$$
(2)

which says that the time-derivative of the probability that a node $j$ is removed from the process equals the expectation of the rate $\delta$, given that node $j$ is infected. Finally, a node is either healthy but susceptible, infected, or cured (and removed); in other words, $1_{\{Y_j=S\}} + 1_{\{Y_j=I\}} + 1_{\{Y_j=R\}} = 1$.

The first equation (1) is complicating due to the interaction with other infected nodes in the network, but (1) is of exactly the same form as the corresponding SIS governing equation [17],

$$\frac{d\Pr[X_j = I]}{dt} = E\left[-\delta 1_{\{X_j=I\}} + 1_{\{X_j=S\}}\beta\sum_{k=1}^{N} a_{kj}1_{\{X_k=I\}}\right]$$

However, in the SIS process, there are only two nodal states (or compartments) possible so that $1_{\{X_j=S\}} + 1_{\{X_j=I\}} = 1$, which leads to fewer equations than in the SIR process. We proceed by rewriting equation (1) using $E\left[1_{\{Y_j=S\}\cap\{Y_k=I\}}\right] = \Pr[Y_j = S, Y_k = I]$,

$$\frac{d\Pr[Y_j = I]}{dt} = -\delta\Pr[Y_j = I] + \beta\sum_{k=1}^{N} a_{kj}\Pr[Y_j = S, Y_k = I]$$

After invoking the law of total probability [16, p. 27],

$$\Pr[Y_k = I] = \Pr[Y_j = S, Y_k = I] + \Pr[Y_j = I, Y_k = I]$$
$$+ \Pr[Y_j = R, Y_k = I]$$

the SIR governing equation (1) becomes

$$\frac{d\Pr[Y_j = I]}{dt} = \beta\sum_{k=1}^{N} a_{kj}\Pr[Y_k = I] - \delta\Pr[Y_j = I]$$
$$- \beta\sum_{k=1}^{N} a_{kj}\Pr[Y_j = I, Y_k = I]$$
$$- \beta\sum_{k=1}^{N} a_{kj}\Pr[Y_j = R, Y_k = I]$$
(3)

The first two terms on the right-hand side in (3) describe the spread of the infection from infected neighbors minus the nodal curing, while the third term excludes infection spread to an infected or removed node $j$. This last term grows over time, because (2) illustrates that the probability to become removed is non-decreasing over time. Relation (3) explains the bell-shape of $\Pr[Y_j(t) = I]$ as a function

of time $t$: initially the third term is small and near to exponential growth arises from the first and second term. As the number of removed nodes increases over time, the third term counteracts the initial growth and forces its decline towards extinction (for large $t$). The SIS differential equation corresponding to (3) is

$$\frac{d\Pr[X_j = I]}{dt} = \beta\sum_{k=1}^{N} a_{kj}\Pr[X_k = I] - \delta\Pr[X_j = I]$$
$$- \beta\sum_{k=1}^{N} a_{kj}\Pr[X_j = I, X_k = I]$$
(4)

The governing equations (3) and (4) lead to the following comparison: On the same network under the same infection and curing rates and starting from one infected node, the infection probability $\Pr[Y_j = I]$ in SIR epidemics is a lower bound for the infection probability $\Pr[X_j = I]$ in SIS epidemics. By starting the two processes on a same network with the same initially infected node, the additional positive term $\sum_{k=1}^{N} a_{kj}\Pr[Y_j = R, Y_k = I]$ in (3) shows that, at any time, $\Pr[Y_j = I] \leq \Pr[X_j = I]$ for any node $j \in G$. Physically, the removal process in SIR cannot increase the spread of infection in the network with respect to SIS epidemics. As a consequence, the $N$-intertwined mean-field approximation (NIMFA) [18] upper bounds, besides SIS, also SIR epidemics.

Another interesting observation, also made in [19], is that the removal process in SIR epidemics prevents that a node can be infected twice, which implies that the SIR infection process spreads over the network as a growing discovery tree (without loops). Above the epidemic threshold, most nodes are infected once (and discovered), while below the epidemic threshold, the SIR infection tree dies out before infecting most nodes once. Thus, in contrast to SIS epidemics, SIR infection travels from a node $i$ to a node $j$ along a path, and not a walk. The tree spreading property of SIR epidemics naturally maps SIR epidemics into a time-depending Bellman-Harris branching process [20] on a network.

## III. JOINT PROBABILITIES

There are two ways to proceed from (3): either we deduce the governing equations for the two-pair probabilities as in [17], followed by higher order joint probabilities until all $2^N$ SIS and $3^N$ SIR linear Markov equations are established or we try to "close" the equations [3, p. 653-654], as coined in epidemiology. Here, we propose a new method to compute all equations for higher order joint probabilities. Indeed, interchanging the derivative and expectation operator in (1) yields

$$\frac{d1_{\{X_j=I\}}}{dt} = -\delta 1_{\{X_j=I\}} + 1_{\{X_j=S\}}\beta\sum_{k=1}^{N} a_{kj}1_{\{X_k=I\}}$$
(5)

Strictly speaking, the derivative of an indicator does not exist, but we agree to *formally* define it by the random variable

equation (5). Next, making the same reversal of operators,

$$\Xi = \frac{d}{dt} E \left[ \prod_{j=1}^{n} 1_{\{X_j=I\}} \right] \overset{\text{formally}}{=} E \left[ \frac{d}{dt} \prod_{j=1}^{n} 1_{\{X_j=I\}} \right]$$

$$= E \left[ \sum_{m=1}^{n} \prod_{j=1; j \neq m}^{n} 1_{\{X_j=I\}} \frac{d1_{\{X_m=I\}}}{dt} \right]$$

substituting (5) and executing the $E[.]$ returns the correct result[1],

$$\Xi = -\delta n E \left[ \prod_{j=1}^{n} 1_{\{X_j=I\}} \right]$$

$$+ \beta \sum_{m=1}^{n} \sum_{k=1}^{N} a_{km} E \left[ 1_{\{X_k=I\}} \prod_{j=1; j \neq m}^{n} 1_{\{X_j=I\}} \right]$$

$$- \beta \sum_{m=1}^{n} \sum_{k=1}^{N} a_{km} E \left[ 1_{\{X_k=I\}} \prod_{j=1}^{n} 1_{\{X_j=I\}} \right]$$

For each combination of $n$ out of $N$ states, such a differential equation for the joint probability

$$E \left[ \prod_{j=1}^{n} 1_{\{X_j=I\}} \right] = \Pr[X_1 = I, X_2 = I, \ldots, X_n = I]$$

can be written. The expectation in the last summation contains, except when $\left(1_{\{X_j=I\}}\right)^2 = 1_{\{X_j=I\}}$ occurs, a product of $n+1$ different random variables $X_j$, for which a new differential equation is needed as outlined above. A similar method applies for a product of different indicators, $\prod_{j=1}^{n_1} 1_{\{Y_j=I\}} \prod_{j=n_1+1}^{n} 1_{\{Y_j=R\}}$, where we define from (2) that $\frac{d1_{\{Y_j=R\}}}{dt} = \delta 1_{\{Y_j=I\}}$. The analysis also shows that the derivative of the $n$-th order joint probability includes joint probabilities of order $n+1$, except if all nodes ($n = N$) are included and that an exact description thus requires governing equations for all $1 \leq n \leq N$ joint probabilities, resulting in $2^N$ SIS and $3^N$ SIR linear Markov equations.

The most evident way of closure, which is an approximation method, is to assume independence between nodes and states. For example, if we close the first-order equations such as (3) by replacing $\Pr[X_j = I, X_k = I]$ by the product $f(\Pr[X_j = I]) g(\Pr[X_k = I])$, where $f$ and $g$ are functions, we transform the set of linear equations in first-order, $\Pr[X_m = I]$, and second-order, $\Pr[X_j = I, X_k = I]$, variables to non-linear equations, though with less variables (only first-order probabilities). This type of approximation is also termed a mean-field approximation, that assumes independence between the infection state of any two nodes.

## IV. PROPERTIES FROM FIRST-ORDER EQUATIONS

In the sequel, we continue to explore what can be deduced from the first-order equations above without either higher-order deduction nor closure. We first review a known result on the epidemic threshold for the SIS process that also applies to the SIR process: The epidemic threshold of the

[1]The formal method can be made mathematically rigorous (using the framework of stochastic differential equations).

SIR and corresponding SIS process on any graph $G$ is lower bounded by

$$\tau_c \geq \frac{1}{\lambda_1} \tag{6}$$

where $\lambda_1$ is the largest eigenvalue of the adjacency matrix $A$. Directly from (3) and (4), we deduce that

$$\frac{d\Pr[Y_j(t) = I]}{dt} \leq \beta \sum_{k=1}^{N} a_{kj} \Pr[Y_k(t) = I] - \delta \Pr[Y_j(t) = I]$$

(and similarly for $\Pr[X_j(t) = I]$). The lower bound (6) the follows by a similar argument as in [12]. The lower bound (6) for the epidemic threshold also holds for directed graphs. Since the SIR infection probability lower bounds that of SIS in a same graph (with same initial conditions), $\tau_{c;SIS} \leq \tau_{c;SIR}$, which was earlier noted by Parshani et al. [19].

For SIS epidemics, the lower bound (6) was earlier proved in [8], though in a much less general and elegant form. More importantly, the **lower** bound $\tau_c^{(1)} = \frac{1}{\lambda_1}$ appeared as the **exact** epidemic threshold in NIMFA, where the superscript (1) in $\tau_c^{(1)}$ refers to the first order mean-field approximation. We deem it important to underline the difference: in the exact SIS and SIR model, the epidemic threshold $\tau_c$ is lower bounded by $\tau_c^{(1)} = \frac{1}{\lambda_1}$, while in approximate analyses (mean-field), the epidemic threshold is found to be equal to $\tau_c^{(1)} = \frac{1}{\lambda_1}$. For some graphs (such as the complete graph), the first order mean-field approximation $\tau_c^{(1)}$ is very sharp, while for other graphs (such as the star), $\tau_c^{(1)} = \frac{1}{\lambda_1}$ is less accurate [21].

The **lower** bound $\tau_c^{(1)} = \frac{1}{\lambda_1}$ is of great practical use: if the effective infection rate $\tau$ can be controlled such that $\tau \leq \tau_c^{(1)}$ or the network can be designed to lower the spectral radius $\lambda_1$ of a graph [22], then the network is safeguarded from long-term, massive infection. The lower bound (6) cautions the widely cited belief of a zero-epidemic threshold in scale-free networks [23]: any finite network must have a strictly positive epidemic threshold. Even when the mean-field epidemic threshold $\tau_c^{(1)} \to 0$ when $\lim_{N \to \infty} \lambda_1 = \infty$, it may be possible, due to the *lower* bound in (6), that the exact threshold $\tau_c > 0$ is non-zero. An upper bound for $\frac{d\Pr[X_j(t)=I]}{dt}$ (and similarly for SIR) follows from the Hölder inequality [16, p. 90] with $\frac{1}{p} + \frac{1}{q} = 1$ and $p > 1$,

$$E\left[1_{\{X_i=I\}} 1_{\{X_k=I\}}\right] \leq \left(E\left[1_{\{X_i=I\}}^p\right]\right)^{1/p} \left(E\left[1_{\{X_k=I\}}^q\right]\right)^{1/q}$$

substituted into (4) as

$$\frac{d\Pr[X_j = I]}{dt} \geq \beta \sum_{k=1}^{N} a_{kj} \Pr[X_k = I] - \delta \Pr[X_j = I]$$

$$- \beta (\Pr[X_i = I])^{1/p} \sum_{k=1}^{N} a_{kj} (\Pr[X_k = I])^{1-1/p}$$

and the right-hand side can be maximized with respect to $p$. Unfortunately, the steady-state solution of the above set of $N$ non-linear equations equals $\Pr[X_j = I] = 0$ for any node $j$ and any $p > 1$. Recently, Boguñá et al. [24] have proposed an approximate, coupling type of argument to deduce an

upper bound for the epidemic threshold. Although their new method is ingenious and physically convincing, a proven upper bound is still lacking. Below, we fill this gap by presenting a new and general upper bound for the epidemic threshold $\tau_c$ in any network in Theorem 2 below.

By definition, the steady-state is attained for the time $t \to \infty$ at which the derivatives of the probabilities do not change anymore. If $\frac{d\Pr[Y_j=R]}{dt} = 0$ in (2) for any node $j$, then $\Pr[Y_j = I] = 0$ implying that there are no infected nodes anymore in the network. In both SIS (due to the absorbing state [8], [9]) and SIR epidemics, the infectious disease eventually disappears from the network! Consequently, the time-dependent (SIR) or metastable/quasi-stationary (SIS) behavior is physically of interest. The final part expresses the exact prevalence in terms of the graph's Laplacian $Q = \Delta - A$ (see e.g. [25]) and is proven in Appendix A:

*Theorem 1:* Denoting the (random) vector $w_I = \left(1_{\{Y_1=I\}}, 1_{\{Y_2=I\}}, \ldots, 1_{\{Y_N=I\}}\right)$ and similarly for $w_R$, the average number of infected nodes (or prevalence) satisfies for SIR epidemics

$$\frac{dy_I}{dt^*} = -y_I + \frac{\tau}{N}E\left[w_I^T Q w_I - w_I^T A w_R\right] \tag{7}$$

while for SIS epidemics (denoted by a tilde)

$$\frac{d\widetilde{y_I}}{dt^*} = -\widetilde{y_I} + \frac{\tau}{N}E\left[\widetilde{w_I^T} Q \widetilde{w_I}\right] \tag{8}$$

where $t^* = \delta t$ is the scaled time and $Q = \Delta - A$ is the Laplacian of the graph with $\Delta = \text{diag}(d_1, d_2, \ldots, d_N)$.

From (2), we see that the average fraction of removed nodes satisfies $\frac{dy_R}{dt^*} = y_I$. Apart from the steady-state, also the maximum in (7) occurs at $\frac{dy_I}{dt^*} = 0$ and, at that value of time $t^*$, it satisfies

$$y_{I\,\max} = \frac{\tau}{N}E\left[w_I^T Q w_I - w_I^T A w_R\right] \tag{9}$$

illustrating that the corresponding $\widetilde{y}_{I\,\max}$ in SIS is larger (because, in SIS, $w_R = 0$ and $\widetilde{w}_I$ is not smaller on average than $w_I$). In a regular graph, each node has degree $r$ and $Q = rI - A$ so that (9) simplifies to

$$y_{I\,\max} = \frac{\tau}{N}E\left[r w_I^T w_I - w_I^T A(w_I + w_R)\right]$$

Since $w_I^T w_I = \sum_{j=1}^{N}\left(1_{\{Y_j=I\}}\right)^2 = \sum_{j=1}^{N}1_{\{Y_j=I\}} = N Z_I$ and, thus $y_I = NE\left[w_I^T w_I\right]$, we have

$$y_{I\,\max} = \frac{\tau}{N}\frac{E\left[w_I^T A(w_I + w_R)\right]}{r\tau - 1} \tag{10}$$

which illustrates (in agreement with (6) because $\lambda_1 = r$) that $y_{I\,\max} = 0$ when $\tau < \frac{1}{r}$ because $E\left[w_I^T A(w_I + w_R)\right] \geq 0$ and $y_I \geq 0$. Only for regular graphs, the epidemic threshold in both SIS and SIR epidemics appears directly from the exact equation (10). For special regular graphs such as the complete graph, we can elaborate (10) even further. The natural extension from regular graphs to any graph is to bound the degree vector as $d_{\min}u \leq D \leq d_{\max}u$ and (14) becomes

$$\begin{cases} \frac{dy_I}{dt^*} \geq (\tau d_{\min} - 1) y_I - \frac{\tau}{N}E\left[w_I^T A(w_I + w_R)\right] \\ \frac{dy_I}{dt^*} \leq (\tau d_{\max} - 1) y_I - \frac{\tau}{N}E\left[w_I^T A(w_I + w_R)\right] \end{cases}$$

from which, for any graph, we find that

$$\frac{\tau}{N}\frac{E\left[w_I^T A(w_I + w_R)\right]}{\tau d_{\max} - 1} \leq y_{I\,\max} \leq \frac{\tau}{N}\frac{E\left[w_I^T A(w_I + w_R)\right]}{\tau d_{\min} - 1}$$

illustrating, with (6), that the epidemic threshold obeys $\frac{1}{d_{\max}} \leq \frac{1}{\lambda_1} \leq \tau_c$. Since $E\left[w_I^T A(w_I + w_R)\right]$ can still be zero for $\tau > \frac{1}{d_{\min}}$, we cannot conclude that $\tau_c \leq \frac{1}{d_{\min}}$. In summary, a regular graph exhibits similar properties as derived from mean-field or deterministic analyses. The larger the heterogeneity in degree distribution as in most real-world networks [11], the larger we may expect that approximate analyses deviate (see e.g. [21] for a star graph).

An upper bound for the SIS epidemic threshold, proven in Appendix B, is

*Theorem 2:* The SIS epidemic threshold $\tau_c$ in graph $G$ is upper bounded by

$$\tau_c \leq \frac{1}{d_{\min}\left(1 - \varepsilon_G\right),} \tag{11}$$

where $\varepsilon_G = \lim_{\widetilde{y_I}\downarrow 0}\max_{(k,l)\in\mathcal{L}}\Pr[X_k = I | X_l = I]$.

The conditional probability $\varepsilon_G$ in Theorem 2 can be upper bounded by $\varepsilon_G \leq \varepsilon_{K_N}$, because just at the onset of infection ($\widetilde{y_I} \downarrow 0$), the maximum conditional infection probability $\varepsilon_G$ on a link $(k, l)$ in the graph $G$ is largest in the complete graph. Exact computations on the complete graph [9], [21] demonstrate that $\tau_c = \frac{1}{N}\left(1 + \frac{c}{\sqrt{N}} + O\left(N^{-1}\right)\right)$ for a constant $c$, implying that $\varepsilon_{K_N} = O\left(\frac{1}{\sqrt{N}}\right)$ for large $N$. Hence, for large $N$, Theorem 2 leads to the upper bound

$$\tau_c \leq \frac{1}{d_{\min}}\left(1 + O\left(\frac{1}{\sqrt{N}}\right)\right) \tag{12}$$

for any graph[2]. Theorem 2 (and its proof) also emphasizes the role of the joint probability of infection at end nodes of a same link, which laid at the basis of the pairwise approximation [26] and is considered as a significant improvement over first-order mean-field approximations.

The upper bound (12) is sharp for regular graphs, although (12) can be large for realistic networks with broad (e.g. power law) degree distribution. The general upper bound (11) and lower bound (6) are, of course, less tight than specific upper and lower bounds of particular classes of graphs, such as regular trees, whose values are found in [26, Table II] based on the work of Pemantle [27], extended by Liggett [28].

Finally, after tedious manipulations, the governing equation of the variance of the fraction of infected nodes in SIS

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[2]For large $N$, a lower bound for $\tau_c$ cannot be of the form

$$\frac{1}{d_{\min} - x}$$

where $x$ is a fixed integer independently of $N$, because for the complete graph $K_N$, $\frac{1}{d_{\min}-x} = \frac{1}{N-1-x} = \frac{1}{N}\left(1 + \frac{1+x}{N} + O\left(N^{-2}\right)\right)$ which is smaller than the exact threshold.

epidemics is

$$\frac{d\mathrm{Var}\left[\widetilde{Z}_I\right]}{dt^*} = \frac{2\tau\left\{E\left[\widetilde{Z}_I\widetilde{w}_I^T Q\widetilde{w}_I\right] - \widetilde{y}_I E\left[\widetilde{w}_I^T Q\widetilde{w}_I\right]\right\}}{N}$$
$$+ \frac{1}{N}\left(\widetilde{y}_I + \frac{\tau}{N}E\left[\widetilde{w}_I^T Q\widetilde{w}_I\right]\right) - 2\mathrm{Var}\left[\widetilde{Z}_I\right]$$

The variance is extremal when $\frac{d\mathrm{Var}\left[\widetilde{Z}_I\right]}{dt^*} = 0$, thus

$$\mathrm{Var}\left[\widetilde{Z}_I\right]\Big|_{ex} = \frac{\tau}{N}\left\{E\left[\widetilde{Z}_I\widetilde{w}_I^T Q\widetilde{w}_I\right] - \widetilde{y}_I E\left[\widetilde{w}_I^T Q\widetilde{w}_I\right]\right\}$$
$$+ \frac{1}{2N}\left(\widetilde{y}_I + \frac{\tau}{N}E\left[\widetilde{w}_I^T Q\widetilde{w}_I\right]\right) \quad (13)$$

The last term is never larger than $\frac{1}{N}$. If the fraction of infected nodes $\widetilde{Z}_I$ and the sum over all links with precisely one end infected, $\widetilde{w}_I^T Q\widetilde{w}_I = \sum_{l\in\mathcal{L}}\left(1_{\{X_{l+}=I\}} - 1_{\{X_{l-}=I\}}\right)^2$, were independent, then the maximum variance $\mathrm{Var}\left[\widetilde{Z}_I\right]\Big|_{ex} < \frac{1}{N}$ would be minimal. However, (8) shows that $\widetilde{Z}_I$ and $\widetilde{w}_I^T Q\widetilde{w}_I$ are dependent, implying that $\mathrm{Var}\left[\widetilde{Z}_I\right]\Big|_{ex} < 1$ can be significant. For regular graphs,

$$\mathrm{Var}\left[\widetilde{Z}_I\right]\Big|_{ex} = \frac{E\left[\widetilde{Z}_I\widetilde{w}_I^T A\widetilde{w}_I\right] - \widetilde{y}_I E\left[\widetilde{w}_I^T A\widetilde{w}_I\right]}{N\left(r - \frac{1}{\tau}\right)}$$
$$+ \frac{1}{2N}\left(\frac{\frac{\tau}{N}E\left[\widetilde{w}_I^T A\widetilde{w}_I\right] - \widetilde{y}_I\left(1 + \tau r\right)}{\tau r - 1}\right)$$

shows that the maximum variance occurs for $\tau$ around the epidemic threshold $\tau_c \geq \frac{1}{r}$. The fact that the fraction of infected nodes in SIS epidemics is found to vary most around the epidemic threshold, where the process exhibits a phase transition (for large $N$), agrees with the general physical theory of phase transitions [29].

## V. Summary

Based on the exact continuous-time, Markovian equations for SIS and SIR epidemics, expressed in terms of Bernoulli random variables, we have proposed a new method to deduce the differential equations for any joint probability. Besides revisiting the known facts that the infection probability in SIS epidemics always upper bounds that in SIR epidemics and that for both models, the epidemic threshold is lower bounded by the inverse of the spectral radius, we present a first order differential equation of the average SIS prevalence over time containing the Laplacian of the graph, that elegantly expresses the maximum average prevalence $y_{I\max}$ in regular graphs in terms of the spectral radius (or degree). From this new expression (8), the SIS epidemic threshold in any graph is upper bounded by (12), which complements the result in [24]. Finally, using our framework with Bernoulli random variables, the variance of the SIS prevalence is computed and found to be maximal around the epidemic threshold.

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*A. Proof of Theorem 1*

Summing (1) over all nodes $j$ yields

$$\frac{d}{dt}E\left[\sum_{j=1}^{N}1_{\{Y_j=I\}}\right]=E\left[\beta\sum_{k=1}^{N}1_{\{Y_k=I\}}\sum_{j=1}^{N}a_{kj}1_{\{Y_j=S\}}-\delta\sum_{j=1}^{N}1_{\{Y_j=I\}}\right]$$

Using $1_{\{Y_j=S\}}=1-1_{\{Y_j=I\}}-1_{\{Y_j=R\}}$, the first sum $S$ becomes{

$$S=\sum_{k=1}^{N}1_{\{Y_k=I\}}\sum_{j=1}^{N}a_{kj}1_{\{Y_j=S\}}$$
$$=\sum_{k=1}^{N}1_{\{Y_k=I\}}\left\{\sum_{j=1}^{N}a_{kj}-\sum_{j=1}^{N}a_{kj}1_{\{Y_j=I\}}-\sum_{j=1}^{N}a_{kj}1_{\{Y_j=R\}}\right\}$$
$$=\sum_{k=1}^{N}d_k1_{\{Y_k=I\}}-\sum_{k=1}^{N}\sum_{j=1}^{N}a_{kj}1_{\{Y_j=I\}}1_{\{Y_k=I\}}$$
$$-\sum_{k=1}^{N}\sum_{j=1}^{N}a_{kj}1_{\{Y_k=I\}}1_{\{Y_j=R\}}$$
$$=D^Tw_I-w_I^TAw_I-w_I^TAw_R$$

Further, denote by $Z_I=\frac{1}{N}\sum_{j=1}^{N}1_{\{Y_j=I\}}$ the fraction of infected nodes in the SIR process and by $y_I=E[Z_I]$, then

$$N\frac{dy_I}{dt}=-N\delta y_I+\beta E\left[D^Tw_I-w_I^TAw_I-w_I^TAw_R\right]$$

or, in terms of the effective infection rate $\tau=\frac{\beta}{\delta}$ in units of $t^*=\delta t$,

$$\frac{dy_I}{dt^*}=-y_I+\frac{\tau}{N}E\left[D^Tw_I-w_I^TAw_I-w_I^TAw_R\right] \quad (14)$$

Using $D=\Delta u$, where $\Delta=\text{diag}(d_1,d_2,\ldots,d_N)$ and $u=(1,1,\ldots,1)$ is the all-one vector, we can rewrite

$$D^Tw_I-w_I^TAw_I=u^T\Delta w_I+w_I^T\Delta w_I-w_I^T\Delta w_I-w_I^TAw_I$$
$$=(u-w_I)^T\Delta w_I+w_I^T(\Delta-A)w_I$$

Since $1_{\{Y_j=I\}}1_{\{Y_j=I\}}=1_{\{Y_j=I\}}$,

$$(u-w_I)^T\Delta w_I=\sum_{j=1}^{N}\left(1-1_{\{Y_j=I\}}\right)d_j1_{\{Y_j=I\}}$$
$$=\sum_{j=1}^{N}\left(1_{\{Y_j=I\}}-1_{\{Y_j=I\}}1_{\{Y_j=I\}}\right)d_j=0$$

Finally, introducing the Laplacian matrix $Q=\Delta-A$, we arrive[3] at (7). The SIS variant (8) is similarly proved. $\square$

*B. Proof of Theorem 2*

From (8) at $\frac{d\widetilde{y_I}}{dt^*}=0$, we find that

$$\tau^{-1}=\frac{E\left[\widetilde{w_I}^TQ\widetilde{w_I}\right]}{N\widetilde{y_I}}=\frac{E\left[\widetilde{w_I}^TQ\widetilde{w_I}\right]}{E\left[\widetilde{w_I}^Tu\right]}$$

Introducing the basic Laplacian property $\widetilde{w_I}^TQ\widetilde{w_I}=\sum_{l\in\mathcal{L}}\left(1_{\{X_{l+}=I\}}-1_{\{X_{l-}=I\}}\right)^2$, where the link $l$ points

[3]Alternative expressions can be obtained using $u=w_I+w_S+w_R$ and $Qu=0$.

from node $l^+=i\to l^-=j$ and $\mathcal{L}$ is the set of links of $G$, yields

$$E\left[\widetilde{w_I}^TQ\widetilde{w_I}\right]=\sum_{l\in\mathcal{L}}E\left[\left(1_{\{X_{l+}=I\}}-1_{\{X_{l-}=I\}}\right)^2\right]$$
$$=2\sum_{l\in\mathcal{L}}E\left[1_{\{X_{l+}=I\}}-1_{\{X_{l+}=I\}}1_{\{X_{l-}=I\}}\right]$$
$$=2\sum_{l\in\mathcal{L}}E\left[1_{\{X_{l+}=I\}}\left(1-1_{\{X_{l-}=I\}}\right)\right]$$
$$=2\sum_{l\in\mathcal{L}}E\left[1_{\{X_{l+}=I\}}1_{\{X_{l-}=S\}}\right]$$
$$=2\sum_{l\in\mathcal{L}}\Pr\left[X_{l+}=I,X_{l-}=S\right]$$

Further, we can write

$$E\left[\widetilde{w_I}^TQ\widetilde{w_I}\right]=\sum_{i=1}^{N}\sum_{j=1}^{N}a_{ij}\Pr\left[X_i=I,X_j=S\right]$$
$$=\sum_{i=1}^{N}\Pr\left[X_i=I\right]\sum_{j=1}^{N}a_{ij}\Pr\left[X_j=S|X_i=I\right]$$

to obtain

$$\tau^{-1}=\frac{\sum_{i=1}^{N}\Pr\left[X_i=I\right]\sum_{j=1}^{N}a_{ij}\Pr\left[X_j=S|X_i=I\right]}{\sum_{i=1}^{N}\Pr\left[X_i=I\right]}$$

The inequality [30]

$$\min_{1\le k\le n}\frac{a_k}{q_k}\le\frac{a_1+a_2+\cdots+a_n}{q_1+q_2+\cdots+q_n}\le\max_{1\le k\le n}\frac{a_k}{q_k}$$

where $q_1,q_2,\ldots,q_n$ are positive real numbers and $a_1,a_2,\ldots,a_n$ are real numbers leads to

$$\begin{cases}\tau^{-1}\ge\min_{1\le i\le N}\sum_{j=1}^{N}a_{ij}\Pr\left[X_j=S|X_i=I\right]\\\tau^{-1}\le\max_{1\le i\le N}\sum_{j=1}^{N}a_{ij}\Pr\left[X_j=S|X_i=I\right]\le d_{\max}\end{cases}$$

Using the degree $d_i=\sum_{j=1}^{N}a_{ij}$, we proceed with the lower bound,

$$\tau^{-1}\ge\min_{1\le i\le N}\sum_{j=1}^{N}a_{ij}\Pr\left[X_j=S|X_i=I\right]$$
$$\ge\min_{1\le i\le N}\left(\min_{(k,l)\in\mathcal{L}}\Pr\left[X_k=S|X_l=I\right]d_i\right)$$
$$=\min_{(k,l)\in\mathcal{L}}\Pr\left[X_k=S|X_l=I\right]d_{\min}$$

We define the epidemic threshold $\tau_c$ as that value of $\tau$ when the prevalence (or order parameter) $\widetilde{y_I}=\frac{1}{N}\sum_{i=1}^{N}\Pr\left[X_i=I\right]$ approaches zero from above, denoted as $\widetilde{y_I}\downarrow 0$, so that

$$\tau_c^{-1}=\lim_{\widetilde{y_I}\downarrow 0}\frac{E\left[\widetilde{w_I}^TQ\widetilde{w_I}\right]}{N\widetilde{y_I}} \quad (15)$$

and

$$\tau_c^{-1}\ge d_{\min}\lim_{\widetilde{y_I}\downarrow 0}\min_{(k,l)\in\mathcal{L}}\Pr\left[X_k=S|X_l=I\right]$$

The definition (15) of the epidemic threshold becomes increasingly precise for large $N$. Finally, since $\Pr\left[X_k=S|X_l=I\right]=1-\Pr\left[X_k=I|X_l=I\right]$, we arrive at (11). $\square$