Integral equations for the heterogeneous, Markovian SIS process with time-dependent rates in time-variant networks

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Abstract

We reformulate the governing equation for the nodal infection probability of the exact heterogeneous SIS process with time-dependent infection and curing rates on any network into two equivalent integral equations, that provide a different view on and a more natural description of the SIS dynamic process in temporal or time-variant networks. From the integral equations, we deduce general bounds.

1 Introduction

A huge number of papers has studied the SIS epidemic process on networks [1], mainly because of three reasons. First, the SIS epidemic models a simplified version of a diffusion process, which describes a kind of transport between nodes of its underlying graph [2]. Transport of items between nodes is major function of a network and is often approximated to "first order" by an epidemic type of stochastic diffusion, such as real viruses and diseases in a population, malware in digital communication networks and the spread of information (ideas, opinions, news, alarms, etc.) in social networks. Second and as earlier mentioned in [3], the SIS epidemic process is one of the simplest members of a particularly popular class of dynamic processes on networks called the "Local rule-Global emergent properties" (LrGep) class, where the collective action of the local rules executed at each node gives rise to a complex, emergent global behavior. Some examples of the LrGep-class are epidemic models (such as SIS and SIR) and more general reaction-diffusion processes [1], the Ising spin model [4], the Kuramoto coupled-oscillator model [5], sandpiles as models for self-organized criticality [6, 7, 8] and opinion models [9, 10]. Many LrGep models depend heavily on the underlying network topology and feature, in general, a phase transition [11]. The phase transition of the SIS and SIR epidemic models are similar to that of the Kuramoto-type of synchronization models, an observation that has received attention in human brain networks (see e.g. [12]). Third, we believe that the Markovian SIS epidemic model has the highest potential to analytically study that phase transition in networks. Pursuing the latter is still a worthwhile endeavor, because, so far, there does not exist (apart from asymptotic

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studies [13]) an exact analysis, not even for the complete graph [14]. Before continuing, we remark that the "phase transition" in SIS and SIR epidemics occurs due to a change in the parameters (ratio of infection to curing rate) of the model, which might be different from a phase transition due to the non-linear dynamics of the process. Both the SIS and SIR Markovian model on any network are linear processes (as any Markov process [15]) and linear dynamic processes do not exhibit jumps, limit cycles nor chaotic behavior that may occur in some non-linear processes [16].

Here, the most general description of a heterogeneous Markovian SIS process with time-dependent rates on a time-variant network is recast into integral equations. Our main results are the derivations of integral equations (7) and (9) from the SIS governing differential equation for the nodal infection probability, from which bounds are deduced in Section 4.

2 Markovian SIS governing equation

The graph G of the network consists of the set $\mathcal{N}$ of N nodes and the set $\mathcal{L}$ of L links and is represented by an $N \times N$ adjacency matrix A with elements $a_{ij} = 1$ if there is a directed link from node i to j, else $a_{ij} = 0$. The exact *heterogeneous* Markovian SIS governing equation [17, 15, 18] for the infection probability $E[X_i] = \Pr[X_i = 1]$ of node i is

$$\frac{dE[X_i(t)]}{dt} = E\left[-\delta_i X_i(t) + (1 - X_i(t)) \sum_{k=1}^{N} \beta_{ki} a_{ki} X_k(t)\right] \tag{1}$$

where the Bernoulli random variable $X_i \in \{0, 1\}$ defines the two possible states, the infected state $X_i = 1$ and the healthy state $X_i = 0$ of node i. The nodal curing is a Poisson process with rate δ_i, while the infection from node k towards node i is also described by a Poisson process with rate β_{ki}. All Poisson processes are independent. When node i is infected at time t and $X_i(t) = 1$, only the first term in (1) on the right-hand side between the brackets [.] affects and decreases with rate $-\delta_i$ the change in infection probability with time $\frac{d\Pr[X_i(t)=1]}{dt}$ (left-hand side in (1)). When node i is healthy and $X_i(t) = 0$, only the second term between the brackets [.] increases $\frac{d\Pr[X_i(t)=1]}{dt}$ by a rate $\sum_{k=1}^{N} \beta_{ki} a_{ki} X_k(t)$ due to all its infected, direct neighbors. We define the nodal curing vector $\widetilde{\delta} = (\delta_1, \delta_2, \ldots, \delta_N)$ and the weighted adjacency matrix $\widetilde{A}$ with element $\widetilde{a}_{ij} = \beta_{ij} a_{ij}$. Both the topology and the rates may dependent upon time; thus $\widetilde{A}$ is generally a time-variant, asymmetric matrix with zero elements on the diagonal ($\widetilde{a}_{ii} = 0$ for all $1 \leq i \leq N$).

An epidemic spreads along *infectious links*, which are network links with one end node infected and the other end node healthy. The probability that there is an infectious link directed from node i to j is

$$\Pr[X_i = 1, X_j = 0] = E[X_i(1 - X_j)] = E[X_i] - E[X_i X_j] \tag{2}$$

which vanishes if $i = j$. With the definition (2), the SIS governing equation (1) becomes

$$\frac{dE[X_i(t)]}{dt} + \delta_i(t) E[X_i(t)] = \sum_{k=1}^{N} \widetilde{a}_{ki}(t) \Pr[X_k(t) = 1, X_i(t) = 0] \tag{3}$$

These joint probabilities $\Pr[X_k = 1, X_i = 0]$ in a Markovian SIS process can be determined precisely as shown in [17, 15], but require the knowledge of the joint probabilities over all triples of nodal

states, which in turn requires all combinations of four nodal states and so on. Eventually, the exact determination leads to 2^N linear differential equations and this exponentially large number in N is a finger print of the NP-hard nature of SIS epidemics on networks.

Using in (3) the conditional probability $\Pr[X_k = 1, X_i = 0] = \Pr[X_k = 1|X_i = 0] \Pr[X_i = 0]$ and $1 - E[X_i] = \Pr[X_i = 0]$, an alternative form of the heterogeneous Markovian SIS governing equation is

$$\frac{dE[X_i(t)]}{dt} + \{\delta_i(t) + b_i(t)\} E[X_i(t)] = b_i(t) \tag{4}$$

where the overall infection rate of the healthy node i from all its neighbors is

$$b_i(t) = \sum_{k=1}^{N} \widetilde{a}_{ki}(t) \Pr[X_k(t) = 1|X_i(t) = 0] \tag{5}$$

which will play a crucial role in the sequel. If $\frac{dE[X_i(t)]}{dt} = 0$ at a time $t = \theta > 0$, then the infection probability $\Pr[X_i(\theta) = 1] = E[X_i(\theta)]$ of node i is extremal and equal to

$$\Pr[X_i(\theta) = 1] = \frac{b_i(\theta)}{\delta_i(\theta) + b_i(\theta)} \tag{6}$$

which resembles [19] the steady-state infection probability of node i in the N-Intertwined Mean-Field Approximation (NIMFA) [20], that led to a partial fraction expansion [21].

3 An integral equation for Markovian SIS epidemics

In appendix A, we reformulate a linear differential equation of the type of the SIS differential equation (4) into an integral equation. We apply (21) to the SIS differential equation (4) and deduce the integral equation

$$\Pr[X_i(t) = 0] = \int_x^t \delta_i(s) e^{-\int_s^t \{\delta_i(u) + b_i(u)\} du} ds + \Pr[X_i(x) = 0] e^{-\int_x^t \{\delta_i(u) + b_i(u)\} du} \tag{7}$$

connecting the healthy probability of node i at any two time points t and x. The integral representation (7) may more naturally describe temporal networks [22], whose topology (and thus adjacency matrix) changes over time. The complicating quantity in (7) is $b_i(t)$ defined in (5), where the infectious treat $\widetilde{a}_{ki}(u) \Pr[X_k(u) = 1|X_i(u) = 0]$ towards the healthy node i from its neighbor k, depends on the product of three independent factors: the link existence $a_{ki}(u)$ at time u which enables the transmission, the infection rate $\beta_{ki}(u)$ at time u which characterizes the strength of the infectious activity and the likeliness $\Pr[X_k(u) = 1|X_i(u) = 0]$ of transmission over link $k \to i$, because the SIS process only spreads over infectious links. The positive integrand in (7) shows that the probability that node i is healthy is always positive in a finite graph, provided that the curing rate $\delta_i(s)$ is not zero at all times $s \in (0, t]$ and the infection rates β_{ij} are finite. When the time t increases while $x < t$ is fixed and the curing rate $\delta_i(u) \geq \delta_{i;\min} > 0$, the last term containing the influence of $\Pr[X_i(x) = 0]$ at time x on the actual healthy probability at time t disappears exponentially fast.

The asymptotic time regime follows from (7) as

$$\lim_{t \to \infty} \Pr[X_i(t) = 0] = \lim_{t \to \infty} \frac{\int_x^t \delta_i(s) e^{\int_x^s \{\delta_i(u) + b_i(u)\} du} ds}{e^{\int_x^t \{\delta_i(u) + b_i(u)\} du}}$$

Using de Hospital's rule and assuming that the limits $\lim_{t\to\infty}\delta_i(t)=\delta_{i;\infty}$ and $\lim_{t\to\infty}\widetilde{a}_{ki}(t)=\widetilde{a}_{ki;\infty}$ in (5) exist, we find that

$$\lim_{t\to\infty}\Pr\left[X_i(t)=0\right]=\frac{\delta_{i;\infty}}{\delta_{i;\infty}+\sum_{k=1}^{N}\widetilde{a}_{ki;\infty}\lim_{t\to\infty}\Pr\left[X_k(t)=1|X_i(t)=0\right]}$$

which has the form (6) of an "extremal" probability. The governing equation (1) does not contain information to conclude that $\lim_{t\to\infty}\Pr\left[X_k(t)=1|X_i(t)=0\right]=0$, but, in any finite graph [23, 15], the SIS epidemic process ends in the absorbing, all-healthy state where $\lim_{t\to\infty}\Pr\left[X_i(t)=0\right]=1$ for any node $i\in\mathcal{N}$. In other words, the infection has disappeared in the network after sufficiently long time.

The homogeneous case with fixed rates ($\beta_{ij}(t)=\beta$, $\delta_i(t)=\delta$ and effective infection rate $\tau=\frac{\beta}{\delta}$) is the simplest version of the general expression (7),

$$\begin{aligned}\Pr\left[X_i(t)=0\right]=\delta\int_x^t e^{-\delta(t-s)}e^{-\beta\sum_{k=1}^{N}a_{ki}\int_s^t\Pr[X_k(u)=1|X_i(u)=0]du}ds\\ +\Pr\left[X_i(x)=0\right]e^{-\delta(t-x)}e^{-\beta\sum_{k=1}^{N}a_{ki}\int_x^t\Pr[X_k(u)=1|X_i(u)=0]du}\end{aligned}\tag{8}$$

The homogeneous NIMFA reformulation, where $v_k(u)$ approximates the nodal infection probability $\Pr\left[X_k(u)=1\right]$ by assuming independence such that $v_k(u)=\Pr\left[X_k(u)=1\right]=\Pr\left[X_k(u)=1|X_i(u)=0\right]$, follows from (8) with $\overline{v}_k(u)=1-v_k(u)$ as

$$\overline{v}_i(t)=\delta\int_x^t e^{-(\delta+\beta d_i)(t-s)}e^{\beta\sum_{k=1}^{N}a_{ki}\int_s^t\overline{v}_k(u)du}ds+\overline{v}_i(x)\,e^{-(\delta+\beta d_i)(t-x)}e^{\beta\sum_{k=1}^{N}a_{ki}\int_x^t\overline{v}_k(u)du}$$

For an SIS Markovian process with fixed rates, we know [24] that $E\left[X_iX_j\right]\geq E\left[X_i\right]E\left[X_j\right]$, so that $\Pr\left[X_k(u)=1|X_i(u)=0\right]\leq\Pr\left[X_k(u)=1\right]$. Then, (8) shows that NIMFA lower bounds the healthy nodal probability, $\Pr\left[X_i(t)=0\right]\geq\overline{v}_i(t)$, as found earlier [3].

3.1 An alternative integral equation to (7)

We demonstrate in Appendix B that the integral equation (7) is equivalent to

$$\ln\left(\frac{\Pr\left[X_i(t)=0\right]}{\Pr\left[X_i(x)=0\right]}\right)=\int_x^t\delta_i(u)\frac{\Pr\left[X_i(u)=1\right]}{\Pr\left[X_i(u)=0\right]}du-\int_x^t b_i(u)\,du\tag{9}$$

A general integrated governing equation is presented in (28) in Appendix B.

By the mean-value theorem (see e.g. [15, Chapter 5]), there exist a point in time $\xi\in[x,t]$ for which (9) equals

$$\frac{\ln\left(\frac{\Pr[X_i(t)=0]}{\Pr[X_i(x)=0]}\right)}{t-x}=\delta_i(\xi)\frac{\Pr\left[X_i(\xi)=1\right]}{1-\Pr\left[X_i(\xi)=1\right]}-b_i(\xi)\tag{10}$$

If the interval $[x,t]$ is not large, we may assume that $\Pr\left[X_i(\xi)=1\right]$ at a particular (unknown) time ξ is a good approximation for $\Pr\left[X_i(u)=1\right]$ at any time point $u\in(x,t)$. Thus, for a suitably chosen timestep $h=t-x$, we may transform the continuous-time setting from (10) or from (27) into a discrete time approximation,

$$\Pr\left[X_i(t_{k+1})=0\right]=\Pr\left[X_i(t_k)=0\right]e^{h\left(\frac{\delta_i(t_k)\Pr[X_i(t_k)=1]}{1-\Pr[X_i(t_k)=1]}-b_i(t_k)\right)}$$

where the $(k+1)$-th time slot is expressed in terms of the k-th time slot, in which the infection probability $\Pr[X_i(u)=1] = \Pr[X_i(t_k)=1]$ is constant for all $u \in [t_k, t_k+h)$ in time slot k and $t_{k+1} = t_k + h$. Such approximation might be valuable for an actual disease spread over a time-variant contact network, especially when additional information about the conditional probability $\Pr[X_k(t_k)=1|X_i(t_k)=0]$ can be obtained.

After solving (10) for $\Pr[X_i(\xi)=1]$, we obtain

$$\Pr[X_i(\xi)=1] = \frac{b_i(\xi) + \frac{\ln\left(\frac{\Pr[X_i(t)=0]}{\Pr[X_i(x)=0]}\right)}{t-x}}{\delta_i(\xi) + b_i(\xi) + \frac{\ln\left(\frac{\Pr[X_i(t)=0]}{\Pr[X_i(x)=0]}\right)}{t-x}} \tag{11}$$

and (11) resembles the extremal probability (6), but now complemented by the differential quotient $\frac{\ln(\Pr[X_i(t)=0])-\ln(\Pr[X_i(x)=0])}{t-x}$ that involves the infection probabilities at the beginning time x and ending time t of the interval $[x,t]$. This generalized form (11) for a particular (unknown) time ξ may suggest that each infection probability at time t obeys, to first order, the extremal probability form $\Pr[X_i(t)=1] = \frac{r_i(t)}{\delta_i(t)+r_i(t)}$, in which $r_i(t)$ is a correction to the infectious link activity $b_i(t) = \sum_{k=1}^{N} \widetilde{a}_{ki}(t) \Pr[X_k(t)=1|X_i(t)=0]$ at time t towards node i, when healthy, from all its infected neighbors.

The SIS process possesses a *quasi-stationary regime*, also called the metastable regime. In the metastable regime, the nodal infection or healthy probability hardly changes anymore. Hence, if both time x and t belong to the metastable regime, then $\Pr[X_i(t)=0] \simeq \Pr[X_i(x)=0] \simeq \Pr[X_i(u)=0]$ for $u \in [x,t]$, so that (11) reduces to the equilibrium form given by the extremal probability (6). However, it is possible that $\Pr[X_i(t)=0] = \Pr[X_i(x)=0]$ for a time x and t in the *transient regime* as reported[1] in [18], but where $\Pr[X_i(u)=0]$ for $u \in [x,t]$ is not equal to $\Pr[X_i(t)=0] = \Pr[X_i(x)=0]$ so that the extremal probability form (11) may not be a good approximation for those intermediate time points $u \in [x,t]$. The SIS process in some graphs may be not unimodal, implying that there is more than one finite time θ at which extremes (satisfying $\frac{dE[X_i(t)]}{dt} = 0$ and (6)) are reached.

4 Bounds

In addition to a general convexity bound (29) derived in Appendix B, we will deduce an upper and lower bound for $\Pr[X_i(t)=1]$ by bounding

$$\gamma_i(x,t) \int_x^t \widetilde{a}_{ik}(u)\,du \le \int_x^t \widetilde{a}_{ki}(u) \Pr[X_k(u)=1|X_i(u)=0]\,du \le \alpha_i(x,t) \int_x^t \widetilde{a}_{ik}(u)\,du$$

where

$$\alpha_i(x,t) = \max_{k\in\mathcal{N}_i \text{ and } u\in[x,t]} \Pr[X_k(u)=1|X_i(u)=0] \tag{12}$$

$$\gamma_i(x,t) = \min_{k\in\mathcal{N}_i \text{ and } u\in[x,t]} \Pr[X_k(u)=1|X_i(u)=0] \tag{13}$$

and $\mathcal{N}_i$ denotes the set of neighbors of node i. Thus, $\alpha_i(x,t) \le 1$ (and similarly for $\gamma_i(x,t) \le 1$) is the probability of occurrence of the most (least) infectious link incident to a healthy node i during

[1] Non-unimodality was first observed by Joel Miller and communicated to me.

the time interval $[x,t]$. The definition (12) for fixed and small $x=c$ and large $t=T$ suggests that the higher $\alpha_i(c,T)$ is, the better node i is reachable. Hence, $\alpha_i(c,T)$ can be considered as a centrality or importance measure for node i, such as betweenness, closeness and the diagonal element of the pseudo-inverse of the Laplacian [2] and suggests that $\alpha_i(c,T)$ may be approximated by topology information from the adjacency matrix A. By the non-negative SIS correlation property [24], we have $\Pr[X_k(u)=1|X_i(u)=0] \leq \Pr[X_k(u)=1]$, so that

$$\alpha_i(x,t) \leq \max_{k\in\mathcal{N}_i \text{ and } u\in[x,t]} \Pr[X_k(u)=1] \tag{14}$$

The minimum $\gamma_i(x,t)$ in (7) produces the upper bound

$$\Pr[X_i(t)=0] \leq \int_x^t \delta_i(s)\, e^{-\int_s^t \left\{\delta_i(u)+\gamma_i(x,t)\widetilde{d}_i(u)\right\}du} ds + \Pr[X_i(x)=0]\, e^{-\int_x^t \left\{\delta_i(u)+\gamma_i(x,t)\widetilde{d}_i(u)\right\}du}$$

where $\widetilde{d}_i(u)=\sum_{k=1}^N \widetilde{a}_{ik}(u)$ is the time-dependent, weighted degree of node i. The maximum $\alpha_i(x,t)$ in (7) produces the lower bound

$$\Pr[X_i(t)=0] \geq \int_x^t \delta_i(s)\, e^{-\int_s^t \left\{\delta_i(u)+\alpha_i(x,t)\widetilde{d}_i(u)\right\}du} ds + \Pr[X_i(x)=0]\, e^{-\int_x^t \left\{\delta_i(u)+\alpha_i(x,t)\widetilde{d}_i(u)\right\}du}$$

We use

$$\int_x^t \delta_i(s)\, e^{\int_x^s \left\{\delta_i(u)+\alpha_i(x,t)\widetilde{d}_i(u)\right\}du} ds = e^{\int_x^t \left\{\delta_i(u)+\alpha_i(x,t)\widetilde{d}_i(u)\right\}du} - 1 - \alpha_i(x,t) \int_x^t \widetilde{d}_i(s)\, e^{\int_0^s \left\{\delta_i(u)+\alpha_i(x,t)\widetilde{d}_i(u)\right\}du} ds$$

in the above bounds for $\Pr[X_i(t)=0]$ to obtain bounds for the probability $\Pr[X_i(t)=1]$ that node i is infected at time t:

$$\Pr[X_i(t)=1] \geq \gamma_i(x,t) \int_x^t \widetilde{d}_i(s)\, e^{-\int_s^t \left\{\delta_i(u)+\gamma_i(x,t)\widetilde{d}_i(u)\right\}du} ds + \Pr[X_i(x)=1]\, e^{-\int_x^t \left\{\delta_i(u)+\gamma_i(x,t)\widetilde{d}_i(u)\right\}du}$$

$$\Pr[X_i(t)=1] \leq \alpha_i(x,t) \int_x^t \widetilde{d}_i(s)\, e^{-\int_s^t \left\{\delta_i(u)+\alpha_i(x,t)\widetilde{d}_i(u)\right\}du} ds + \Pr[X_i(x)=1]\, e^{-\int_x^t \left\{\delta_i(u)+\alpha_i(x,t)\widetilde{d}_i(u)\right\}du}$$

The first integral over s in the above bounds is always positive, but generally difficult to evaluate. The inequalities for the homogenous case with constant rates simplify to

$$\Pr[X_i(t)=1] \geq \frac{\gamma_i(x,t)\,\beta d_i}{\delta+\gamma_i(x,t)\,\beta d_i} + e^{-(\delta+\gamma_i(x,t)\beta d_i)(t-x)} \left(\Pr[X_i(x)=1] - \frac{\gamma_i(x,t)\,\beta d_i}{\delta+\gamma_i(x,t)\,\beta d_i}\right) \tag{15}$$

$$\Pr[X_i(t)=1] \leq \frac{\alpha_i(x,t)\,\beta d_i}{\delta+\alpha_i(x,t)\,\beta d_i} + e^{-(\delta+\alpha_i(x,t)\beta d_i)(t-x)} \left(\Pr[X_i(x)=1] - \frac{\alpha_i(x,t)\,\beta d_i}{\delta+\alpha_i(x,t)\,\beta d_i}\right) \tag{16}$$

with equality at $t=x$. The upper bound (analogously for the lower bound) only exceeds $\frac{\alpha_i(x,t)\beta d_i}{\delta+\alpha_i(x,t)\beta d_i}$ for finite time t provided the infection probability at time x is $\Pr[X_i(x)=1] > \frac{\alpha_i(x,t)\beta d_i}{\delta+\alpha_i(x,t)\beta d_i}$. These bounds (15) and (16) resemble the time-dependent solution of a general continuous-time two-state Markov process [15, p. 231], which is also the underlying basis for NIMFA as explained in [21, Sec. 2.1 and Fig. 1.].

After sufficiently large time $t=T$ to ignore the initial condition at time x (i.e. $e^{-\delta T}\Pr[X_i(x)=1] << 1$), the inequalities (15) and (16) simply with $\tau=\frac{\beta}{\delta}$ to

$$\frac{\gamma_i(x,T)\,\beta d_i}{\delta+\gamma_i(x,T)\,\beta d_i} \leq \Pr[X_i(T)=1] \leq \frac{\alpha_i(x,T)\,\beta d_i}{\delta+\alpha_i(x,T)\,\beta d_i} \tag{17}$$

Similarly, the extremal probability (6) for constant rates can be bounded as

$$\frac{\gamma_i(x,\theta)\,\beta d_i}{\delta+\gamma_i(x,\theta)\,\beta d_i} \le \Pr[X_i(\theta)=1] \le \frac{\alpha_i(x,\theta)\,\beta d_i}{\delta+\alpha_i(x,\theta)\,\beta d_i}$$

The correspondence with (17) indicates that the quasi-stationary (or metastable) SIS regime attained after time T operates around the maximal value within bounds depending on infectious links. The smaller the difference $\alpha_i(x,T)-\gamma_i(x,T)$, the sharper and more promising the above bounds will be. Also, (17) supports the earlier claim based upon (11) that the nodal infection probability can be approximated by $\Pr[X_i(t)=1] \approx \frac{r_i}{\delta_i(t)+r_i}$.

Defining the maximum possible infection probability $v_{\max}=\max_{k\in\mathcal{N} \text{ and } u\in[0,T]}\Pr[X_k(u)=1]$ in the interval $[0,T]$, then the upper bound on $\alpha_i(x,t)$ in (14) shows that $\alpha_i(0,T)\le v_{\max}$ and the upper bound in (17) is

$$\Pr[X_i(T)=1] \le \frac{v_{\max}\tau d_i}{1+v_{\max}\tau d_i}$$

whereas the corresponding upper bound for the steady-state infection probability $v_{i\infty}$ in NIMFA [15, Theorem 17.4.2 on p. 464] obeys

$$v_{i\infty} \le \frac{\tau d_i}{1+\tau d_i}$$

If $v_{\max}$ occurs at node i, the above upper bound becomes $v_{\max} \le \frac{v_{\max}\tau d_i}{1+v_{\max}\tau d_i}$ and, equivalently for $\tau \ge \frac{1}{d_i}$,

$$v_{\max} \le 1-\frac{1}{\tau d_i} \le 1-\frac{1}{\tau d_{\max}}$$

For any regular graph with degree r, we thus observe that $\Pr[X_i(T)=1] \le 1-\frac{1}{r\tau}$, while equality holds in NIMFA. Despite the lack of a companion of (14) for the minimum $\gamma_{\min}=\min_{i\in\mathcal{N}}\gamma_i(0,t)$ in (13), we can proceed similarly,

$$\frac{\gamma_{\min}\tau d_i}{1+\gamma_{\min}\tau d_i} \le \Pr[X_i(T)=1]$$

If the minimum possible infection probability $v_{\min}$ in the interval $[0,T]$ occurs at node k, then

$$\frac{\gamma_{\min}\tau d_{\min}}{1+\gamma_{\min}\tau d_{\min}} \le \frac{\gamma_{\min}\tau d_k}{1+\gamma_{\min}\tau d_k} \le v_{\min}$$

from which an upper bound for the effective infection rate follows as

$$\tau \le \frac{1}{\gamma_{\min} d_{\min}} \frac{v_{\min}}{1-v_{\min}} \tag{18}$$

More equations for the effective infection rate τ of this type are presented in Appendix C.

5 Summary

Integral equations (7) and (9) for the most general Markovian setting of SIS epidemics are derived. The analysis illustrates that only the N first, linear Markovian equations (1, 4, 3) for the nodal infection probabilities alone out of the 2^N other equations for joint probabilities are elegantly bounded by the extremal probability form (6,11) and the bounds resemble the behavior of the corresponding N non-linear NIMFA equations.

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A Linear first order differential equation

We rewrite (4) as

$$\frac{df\left(t\right)}{dt}+g\left(t\right)f\left(t\right)=g\left(t\right)-\delta_{i}\left(t\right) \tag{19}$$

where $f\left(t\right)=E\left[X_{i}\left(t\right)\right]$ and $g\left(t\right)=\delta_{i}\left(t\right)+b_{i}\left(t\right)=G\left(f\left(t\right)\right)$, where $G\left(.\right)$ is an unknown function.

Since the dependence of $g\left(t\right)$ on $f\left(t\right)$ is unknown, the solution of the differential equation (19) (see e.g. [25, Sec. 5.2])

$$f\left(t\right)=\exp\left(-\int_{0}^{t}g\left(u\right)du\right)\left\{\int_{0}^{t}\exp\left(\int_{0}^{s}g\left(u\right)du\right)\left(g\left(s\right)-\delta_{i}\left(s\right)\right)ds+f\left(0\right)\right\} \tag{20}$$

expresses $f\left(t\right)$ in terms of $g\left(t\right)$ and thus results in an integral equation for $f\left(t\right)$, rather than in its solution. After integrating the first term,

$$\int_{0}^{t}\exp\left(\int_{0}^{s}g\left(u\right)du\right)g\left(s\right)ds=\exp\left(\int_{0}^{t}g\left(u\right)du\right)-1$$

and some rearrangements, we arrive at

$$1-f\left(t\right)=\int_{0}^{t}\delta\left(s\right)\exp\left(-\int_{s}^{t}g\left(u\right)du\right)ds+\left\{1-f\left(0\right)\right\}\exp\left(-\int_{0}^{t}g\left(u\right)du\right) \tag{21}$$

We rewrite the differential equation (4) with the definition $b_{i}\left(t\right)$ in (5) as

$$\frac{d\Pr\left[X_{i}\left(t\right)=0\right]}{dt}+\left\{\delta_{i}\left(t\right)+b_{i}\left(t\right)\right\}\Pr\left[X_{i}\left(t\right)=0\right]=\delta_{i}\left(t\right) \tag{22}$$

Only if δ is a constant (independent of time) and assuming that $G\left(h\left(t\right)\right)=\delta_{i}+b_{i}\left(t\right)$ exists and is known, then the above differential equation becomes with $h\left(t\right)=\Pr\left[X_{i}\left(t\right)=0\right]=1-f\left(t\right)$,

$$\frac{dh\left(t\right)}{dt}+G\left(h\left(t\right)\right)h\left(t\right)=\delta_{i}$$

whose solution is

$$\int_{h(0)}^{h(t)} \frac{dx}{\delta_i - xG(x)} = t$$

Let $\frac{dH(x)}{dx} = \frac{1}{\delta_i - xG(x)}$, then $H(h(t)) - H(h(0)) = t$ and the explicit solution is $h(t) = H^{-1}(t + H(h(0)))$.

For the particular example where $g(t) = \alpha f(t)$, the differential equation (19) becomes a generalized "logistic" or non-linear Bernoulli differential equation $\frac{df(t)}{dt} - \alpha f(t) = -\delta - \alpha f^2(t)$, which describes, in the mean-field approximation, the probability $v(t)$ of infection in a node at time t in a regular graph G with degree r obeying

$$\frac{dv(t)}{dt} = r\beta(t) v(t) (1 - v(t)) - \delta(t) v(t) \tag{23}$$

where the infection rate $\beta(t)$ and the curing rate $\delta(t)$ are general non-negative real functions of time t. By the same technique "variation of a constant", we find in [26] the solution

$$v(t) = \frac{\exp\left(\int_0^t (r\beta(u) - \delta(u)) du\right)}{\frac{1}{v_0} + r\int_0^t \beta(s) \exp\left(\int_0^s (r\beta(u) - \delta(u)) du\right) ds} \tag{24}$$

where v_0 is the initial fraction of infected nodes.

B The function $p_i(t, q)$ and proofs of (9)

We give two proofs of (9): after introducing the function $p_i(t; q)$ in (25), the first proof transforms the integral equation (7) into (9), while the second proof of (9) is based on the governing SIS equation (1).

Proof 1: Let us denote

$$p_i(t; q) = \Pr[X_i(t) = 0] e^{\int_q^t \{\delta_i(u) + b_i(u)\} du} \tag{25}$$

where q is an arbitrary point in time, then the integral equation (7) can be recast as

$$p_i(t; q) - p_i(x; q) = \int_x^t \delta_i(s) e^{\int_q^s \{\delta_i(u) + b_i(u)\} du} ds$$

After invoking the definition (25), we obtain an integral equation in $p_i(t; q)$,

$$p_i(t; q) - p_i(x; q) = \int_x^t \delta_i(s) \frac{p_i(s; q)}{\Pr[X_i(s) = 0]} ds \tag{26}$$

Since the integrand is non-negative, then $p_i(t; q) \geq p_i(x; q)$ for $t > x$ and $p_i(t; q)$ is non-decreasing in time t.

Given $p_i(x; q)$, we can iterate the integral equation (26). After n iterations, we obtain the series of multiple integrals,

$$\frac{p_i(t; q)}{p_i(x; q)} = 1 + \int_x^t \frac{\delta_i(t_1) dt_1}{\Pr[X_i(t_1) = 0]} + \int_x^t \frac{\delta_i(t_1) dt_1}{\Pr[X_i(t_1) = 0]} \int_x^{t_1} \frac{\delta_i(t_2) dt_2}{\Pr[X_i(t_2) = 0]} + \cdots + \int_x^t \frac{\delta_i(t_1) dt_1}{\Pr[X_i(t_1) = 0]} \cdots \int_x^{t_{n-1}} \frac{p_i(t_n; q)}{p_i(x; q)} \frac{\delta_i(t_n) dt_n}{\Pr[X_i(t_n) = 0]}$$

Using the multiple integral formula (which can be verified by partial integration)

$$\int_x^t \frac{\delta_i(t_1)\,dt_1}{\Pr[X_i(t_1)=0]}\int_x^{t_1}\frac{\delta_i(t_2)\,dt_2}{\Pr[X_i(t_2)=0]}\cdots\int_x^{t_{n-1}}\frac{\delta_i(t_n)\,dt_n}{\Pr[X_i(t_n)=0]}=\frac{1}{n!}\left(\int_x^t\frac{\delta_i(s)\,ds}{\Pr[X_i(s)=0]}\right)^n$$

yields

$$\frac{p_i(t;q)}{p_i(x;q)}=1+\sum_{k=1}^{n-1}\frac{1}{k!}\left(\int_x^t\frac{\delta_i(s)\,ds}{\Pr[X_i(s)=0]}\right)^k+\int_x^t\frac{\delta_i(t_1)\,dt_1}{\Pr[X_i(t_1)=0]}\cdots\int_x^{t_{n-1}}\frac{p_i(t_n;q)}{p_i(x;q)}\frac{\delta_i(t_n)\,dt_n}{\Pr[X_i(t_n)=0]}$$

The last integral can be bounded as

$$\int_x^t\frac{\delta_i(t_1)\,dt_1}{\Pr[X_i(t_1)=0]}\cdots\int_x^{t_{n-1}}\frac{p_i(t_n;q)}{p_i(x;q)}\frac{\delta_i(t_n)\,dt_n}{\Pr[X_i(t_n)=0]}\le\frac{p_i(t;q)}{p_i(x;q)}\frac{1}{n!}\left(\int_x^t\frac{\delta_i(s)\,ds}{\Pr[X_i(s)=0]}\right)^n$$

and illustrates that the series converges in any finite interval when $n\to\infty$. Hence,

$$\frac{p_i(t;q)}{p_i(x;q)}=1+\sum_{k=1}^{\infty}\frac{1}{k!}\left(\int_x^t\frac{\delta_i(s)\,ds}{\Pr[X_i(s)=0]}\right)^k=\exp\left(\int_x^t\frac{\delta_i(s)\,ds}{\Pr[X_i(s)=0]}\right)$$

Introducing the definition (25) of $p_i(t;x)$ results in

$$\frac{\Pr[X_i(t)=0]\,e^{\int_x^t\{\delta_i(u)+b_i(u)\}du}}{\Pr[X_i(x)=0]}=\exp\left(\int_x^t\frac{\delta_i(s)\,ds}{\Pr[X_i(s)=0]}\right)\tag{27}$$

which can be rewritten as (9). □

Proof 2: We can generalize (9) to any integrable function $h(x)=\frac{dH(x)}{dx}$ as

$$H(\Pr[X_i(t)=0])-H(\Pr[X_i(x)=0])=\int_x^t h(\Pr[X_i(u)=0])\frac{d\Pr[X_i(u)=0]}{du}du$$

Introducing the governing SIS equation (22) for the healthy probability $\Pr[X_i(u)=0]$ leads to the general formula

$$H(q)|_{\Pr[X_i(x)=0]}^{\Pr[X_i(t)=0]}=\int_x^t h(\Pr[X_i(u)=0])\left(\delta_i(u)\Pr[X_i(u)=1]-\sum_{k=1}^{N}\widetilde{a}_{ki}(u)\Pr[X_k(u)=1,X_i(u)=0]\right)du\tag{28}$$

where the special case $h(x)=\frac{1}{x}$ leads to (9). □

Lemma 1 *Provided that the curing rate* $\delta_i(t)\ge\frac{1}{t+\frac{1}{\delta_i(0)}}$*, the function* $p_i(t;q)$*, defined in (25), is convex for all time* t*.*

Proof: Differentiating (26) with respect to time t yields

$$\frac{dp_i(t;q)}{dt}=\frac{\delta_i(t)}{\Pr[X_i(t)=0]}p_i(t;q)$$

A second differentiation, in which we use the above first order differential, leads to

$$\frac{d^2p_i(t;q)}{dt^2}=\left\{\frac{d}{dt}\left(\frac{\delta_i(t)}{\Pr[X_i(t)=0]}\right)+\left(\frac{\delta_i(t)}{\Pr[X_i(t)=0]}\right)^2\right\}p_i(t;q)$$

and

$$\frac{d^2 p_i(t;q)}{dt^2} = \left\{ \delta_i(t) + \frac{\delta_i'(t)}{\delta_i(t)} \Pr[X_i(t)=0] - \frac{d\Pr[X_i(t)=0]}{dt} \right\} \frac{\delta_i(t)\, p_i(t;q)}{(\Pr[X_i(t)=0])^2}$$

Introducing the governing equation (22) yields

$$\frac{d^2 p_i(t;q)}{dt^2} = \left\{ \frac{\delta_i'(t)}{\delta_i(t)} + \delta_i(t) + b_i(t) \right\} \frac{\delta_i(t)\, p_i(t;q)}{\Pr[X_i(t)=0]}$$

which is always non-negative provided $\frac{\delta_i'(t)}{\delta_i(t)} + \delta_i(t) \geq 0$, which is, after integration, equivalent to the condition stated in Lemma 1. □

As a consequence of Lemma 1, we conclude for a constant curing rate $\delta_i(t) = \delta_i$ that $p_i(t;q)$ is always convex. Applying the convexity definition (see e.g. [15, p. 101]),

$$p_i(\alpha u + (1-\alpha) v; q) \leq \alpha p_i(u;q) + (1-\alpha) p_i(v;q)$$

with $\alpha \in [0,1]$ where $\delta_i(t)$ obeys the condition in Lemma 1, then the expression (27), after choosing $x = u$, leads to a bound involving the healthy probability where the time $\widetilde{\xi} = \alpha u + (1-\alpha) v \in [u, v]$:

$$e^{\int_u^{\alpha u+(1-\alpha)v} \frac{\delta_i(s)ds}{\Pr[X_i(s)=0]}} \leq \alpha + (1-\alpha)\, e^{\int_u^v \frac{\delta_i(s)ds}{\Pr[X_i(s)=0]}} \tag{29}$$

C The prevalence and effective infection rate

Summing (3) over all nodes and rewritten in terms of the prevalence

$$y(t) = \frac{1}{N} \sum_{i=1}^{N} \Pr[X_i(t)=1] = E\left[\frac{1}{N} \sum_{i=1}^{N} X_i(t) \right] \tag{30}$$

defined as the expected fraction of infected nodes in a graph at time t, yields

$$\frac{dy(t)}{dt} + \frac{1}{N} \sum_{i=1}^{N} \delta_i(t)\, E[X_i(t)] = \frac{1}{N} \sum_{i=1}^{N} \sum_{k=1}^{N} \widetilde{a}_{ki}(t) \Pr[X_k = 1, X_i = 0]$$

Only when all curing rates $\delta_i(t) = \delta(t)$ are equal, we find a differential equation in the prevalence

$$\frac{dy(t)}{dt} + \delta(t)\, y(t) = \frac{1}{N} \sum_{i=1}^{N} \sum_{k=1}^{N} \widetilde{a}_{ki}(t) \Pr[X_k = 1, X_i = 0]$$

Earlier, we have demonstrated in [27] and further studied in [28, 29], that

$$\frac{dy(t^*;\tau)}{dt^*} = -y(t^*;\tau) + \frac{\tau}{N} E\left[w(t^*;\tau)^T Q w(t^*;\tau) \right] \tag{31}$$

where $t^* = \delta t$ is the scaled time, $Q = \Delta - A$ is the Laplacian of the graph G with $\Delta = \mathrm{diag}(d_1, d_2, \ldots, d_N)$ and d_i is the degree of node i in G, and the Bernoulli vector $w = (X_1, X_2, \ldots, X_N)$. If we confine ourselves for simplicity to fixed rates and a time-invariant topology, then the comparison with (31) shows that the average number of links in the cut-set between healthy and infected nodes is

$$E\left[w^T Q w \right] = \sum_{i=1}^{N} \sum_{k=1}^{N} a_{ki} \Pr[X_k = 1, X_i = 0] \tag{32}$$

which is investigated further in [30].

In the metastable regime, the homogeneous case with fixed rates ($\beta_{ij}(t) = \beta$, $\delta_i(t) = \delta$) and time-invariant topology $a_{ki}(t) = a_{ki}$ in (6) with (5) satisfies to a very good approximation

$$\Pr[X_i(\theta) = 1] = \frac{\tau \sum_{k=1}^{N} a_{ki} \Pr[X_k(\theta) = 1 | X_i(\theta) = 0]}{1 + \tau \sum_{k=1}^{N} a_{ki} \Pr[X_k(\theta) = 1 | X_i(\theta) = 0]}$$

from which the effective infection rate $\tau = \frac{\beta}{\delta}$ is solved as

$$\tau = \frac{\Pr[X_i(\theta) = 1]}{\sum_{k=1}^{N} a_{ki} \Pr[X_k(\theta) = 1, X_i(\theta) = 0]} \tag{33}$$

The effective infection rate τ can also be solved[2] from (31) with (32) in terms of the prevalence as

$$\tau = \frac{y(\theta)}{\frac{1}{N} \sum_{i=1}^{N} \sum_{k=1}^{N} a_{ki} \Pr[X_k(\theta) = 1, X_i(\theta) = 0]} \tag{34}$$

We revisit the deduction[3] from [27] and rewrite (34) as

$$\tau^{-1} = \frac{\sum_{k=1}^{N} \Pr[X_k(\theta) = 1] \sum_{i=1}^{N} a_{ki} \Pr[X_i(\theta) = 0 | X_k(\theta) = 1]}{\sum_{k=1}^{N} \Pr[X_k(\theta) = 1]}$$

for a time θ, somewhere in the metastable regime (see Section 3.1). Now

$$\Pr[X_i(t) = 0 | X_k(t) = 1] = \frac{\Pr[X_i(t) = 0]}{\Pr[X_k(t) = 1]} \Pr[X_k(t) = 1 | X_i(t) = 0]$$

appears as the probability that node i is healthy given an infected neighbor k, which opposes the "dual" conditional probability $\Pr[X_k(t) = 1 | X_i(t) = 0]$ above and both conditional probabilities are only equal when $\Pr[X_i(t) = 0] = \Pr[X_k(t) = 1]$, i.e. when both nodes have equal probability to be in an opposite state at any time t. Upper and lower bounding in the usual way leads to

$$\min_{1 \le k \le N} \sum_{i=1}^{N} a_{ki} \Pr[X_i(\theta) = 0 | X_k(\theta) = 1] \le \tau^{-1} \le \max_{1 \le k \le N} \sum_{i=1}^{N} a_{ki} \Pr[X_i(\theta) = 0 | X_k(\theta) = 1]$$

Using the degree $d_i = \sum_{j=1}^{N} a_{ij}$ and confining to the lower bound, we have

$$\tau^{-1} \ge \min_{1 \le k \le N} \sum_{i=1}^{N} a_{ki} \Pr[X_i(\theta) = 0 | X_k(\theta) = 1] \ge \min_{1 \le k \le N} \left(\min_{(i,l) \in \mathcal{L}} \Pr[X_i(\theta) = 0 | X_l(\theta) = 1] \, d_k \right)$$

After a similar treatment of the upper bound, we arrive at

$$d_{\min} \min_{(i,l) \in \mathcal{L}} \Pr[X_i(\theta) = 0 | X_l(\theta) = 1] \le \tau^{-1} \le d_{\max} \max_{(i,l) \in \mathcal{L}} \Pr[X_i(\theta) = 0 | X_k(\theta) = 1] \tag{35}$$

We remark that $\gamma_{\min} = \min_{i \in \mathcal{N}} \gamma_i(\theta) = \min_{(i,l) \in \mathcal{L} \text{ and } u \in [0,\theta]} \Pr[X_k(u) = 1 | X_i(u) = 0]$, expressed in "dual" conditional probability, appears in (18) and is different from the above.

[2] Also by summing (33) over all nodes after obvious manipulations.

[3] Earlier, (34) has already appeared in the proof of Theorem 17.3.2 in [15, p. 458] and in [27]. The last part of the proof in [15, p. 459] was, unfortunately erroneous and thus also Theorem 17.3.2, as reported in [28].