

PAPER: INTERDISCIPLINARY STATISTICAL MECHANICS

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PAPER: Interdisciplinary statistical mechanics

The SIS process in populations with exponential decay

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Abstract. We study the final state of a susceptible-infected-susceptible (SIS) process whose running time is an exponentially distributed random variable. The population in which the spreading evolves is assumed to be homogeneously mixed. We show that whenever the state dependent normalized infection rates are *on average* smaller than the corresponding curing rates, the final prevalence of the process vanishes in the large population size limit, irrespectively of the mean running time of the process. We show how this statement implies similar results concerning the time evolution of the SIS and the modified SIS processes as well as the steady state of the modified SIS process. In the case of the usual SIS model, for suitably low values of the mean running time, the absence of a non-vanishing prevalence is found in the large population size limit, even if the normalized infection rate exceeds the curing rate.

Keywords: stochastic processes

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1. Introduction

Stochastic modelling is a common way to simulate real world spreading processes like biological epidemic outbreaks, information spreading in social media, virus spreading in computer networks, error propagation, etc (see [1–4] and references therein).

Consider a Markovian susceptible-infected-susceptible (SIS) process [1, 2, 5] with infection rate β and curing rate δ taking place in a population of individuals modelled by a simple, unweighted graph \mathcal{G} . Fixing the curing rate δ , it is plausible that the long time limit of the prevalence, i.e. the ratio of the infected individuals in the whole population is zero for small values of β . After increasing β one should achieve a threshold $\beta_c(\delta, \mathcal{G})$ beyond which there can be a non-zero prevalence in the long time limit. Above the threshold, the prevalence is a monotonically increasing function of β .

Although the closed form of the time evolution of the expectation value of the prevalence is not accessible due to the complexity of the problem, several mean field methods, like the heterogeneous [1, 4, 6] or the N -intertwined [7, 8] mean-field approximation have been developed which support the above presented picture and give close approximants of $\beta_c(\delta, \mathcal{G})$. It turned out that the epidemic threshold satisfies the inequality [4, 7–9]

$$\frac{\delta}{\lambda(\mathcal{G})} \leq \beta_c(\delta, \mathcal{G}), \quad (1)$$

where $\lambda(\mathcal{G})$ is the largest eigenvalue of the adjacency matrix of \mathcal{G} [10, 11]. The inequality becomes sharp in the case of complete graphs resulting in $\beta_c(\delta, K_n) = \delta/(n-1)$. For general graphs, there is a sequence of lower bounds improving (1) given by higher order mean field approximations, see [9] for details.

Unfortunately when one aims to perform a similar analysis within the theoretical framework of Markov processes one immediately runs into a serious problem if the population is finite [12, 13]. To see this, observe that the SIS process has a single absorbing state. It is the state that contains only susceptible individuals. Such a unique absorbing state has a profound consequence on the asymptotic probability distribution of the Markov process. Namely, the long time limit of the probability of finding the system in the absorbing state is equal to one [7] making it impossible to define $\beta_c(\delta, \mathcal{G})$ through the calculation of the expectation value of the prevalence in the long time limit.

In order to define the threshold by the analysis of the time evolution of the Markovian model, numerous exact results have been obtained. In [14] it was shown that (1) is a sufficient condition for the separation of two different dynamical regimes which correspond to fast and slow extinction of the spreading process. Fast extinction is characterized by an extinction time proportional to $\log(n)$, where n is the size of the population. Slow extinction time scales with $\exp(n^\alpha)$ for some positive α . An approximation of the time dependence of the prevalence has been recently obtained in [15, 16] by a ‘tanh formula’ which shows that a non-zero initial prevalence exponentially dies out below a threshold but can persist for rather long time above it. The stationary distribution of processes like the modified SIS [13] or the ε -SIS [12, 13] process on K_n show a threshold like behaviour in the $n \rightarrow \infty$ limit as well as the extinction time and the second largest eigenvalue of the infinitesimal generator of the SIS process [17, 18] on the same graph.

On the other hand, the lifetime of groups formed by individuals are usually not infinite. Several groups are created for impermanent occasions. These groups form, exist and decay in finite time. Consider for instance humans forming study classes or temporary gatherings in transportation networks; or animal groups like of shoaling fish and flocking birds. Even artificial networks, like the network of computers of a LAN party can form and decay. The study of formation and decay of such groups [19, 20] is becoming more and more important as mesoscale interaction data among humans are becoming available to researchers (see [21] and references therein). If a connected population has finite lifetime, then the definition of the epidemic process needs a modification, namely, we have to introduce a stopping time which is usually not definite but also a random variable with a given probability distribution, independent of the process itself. While groups form, decay and reappear again, newly formed groups inherit those infected individuals that have been produced previously in such processes.

In this paper, we study the effect of a stochastic finite running time on the SIS process taking place on the complete graph K_n . While the SIS process evolves, the infection can spread along all the edges of K_n , that is the connection structure of the population remains static and represents homogeneous mixing. The distribution of the running time is at first assumed to be exponential. We study the expectation value of the prevalence in the final state of the system if initially there is only one infected individual in the group. (The choice of the initial data is common in the study of the SIS process [22].) The exponential running time of the population can save the infection from extinction. The question that we are interested in is, *whether this is enough to maintain a finite average prevalence in the $n \rightarrow \infty$ limit?*

As the results of [13] suggest, it is not trivial that the stationary value of the prevalence is surely non-zero in the large population size limit even if the system does not

contain any absorbing state. In order to study the question in detail, we keep the usual assumption of the population being homogeneously mixed, that is the infection rate between the individuals is supposed to be uniform in the population. Nevertheless, we weaken the assumption of the curing and infection rates to be independent of the number of infected individuals: The infection rates β_k and curing rates δ_k that now depend on k , the number of infected individuals in the population, replace the uniform infection and curing rate. It turns out that the mean of the ratio of the normalized infection rates (defined through the relationship $\beta_k = \tau_k/n$) and the curing rates plays a distinguished role: When $\overline{\tau/\delta}$, defined by

$$\overline{\tau/\delta} = \lim_{k \rightarrow \infty} \frac{1}{k} \left(\frac{\tau_1}{\delta_2} + \dots + \frac{\tau_k}{\delta_{k+1}} \right) \quad (2)$$

is less than one, the prevalence is zero in the large n limit *irrespective* of the average running time of the process. The choice of the exponential distribution is not restrictive and the same statement can be formulated for any other distribution. We will see that the methods that enable the proof of the result can also be used to show the validity of similar statements concerning the full time evolution and the stationary state of the modified SIS process in the same parameter regime. The usual SIS model has uniform (k independent) normalized infection and curing rate. We will show that for small values of the expectation value of the stopping time, the average prevalence vanishes in the $n \rightarrow \infty$ limit, even if the normalized infection rate is larger than the curing rate.

The paper is organized as follows. In section 2, we introduce the spreading processes under consideration and show how the final prevalence of the SIS and the modified SIS processes are related to each other. Section 3 contains the main results of our paper concerning the $\overline{\tau/\delta} < 1$ parameter regime. The detailed discussion of the usual SIS model is given in section 4. Summary of the results and the outlook are presented in section 5. The necessary mathematical preliminaries and some auxiliary calculations, including new upper and lower bounds on the Schur complements of tridiagonal infinitesimal generators are omitted in the main text, they can be found in the appendix.

2. The SIS process on the complete graph

Thanks to the high symmetry of the complete graph K_n , the continuous time Markov process of a SIS dynamics is lumpable [23], i.e. the states of the process can be joined together to form a new Markovian process with a smaller state space. The lumping results in a state space $\mathcal{S} = \{0, 1, \dots, n\}$, where a given state is the total number of the infected individuals in the system. The state space and the transition rates of the SIS process on K_n is shown in figure 1. It is clear that the process has an absorbing state characterized by the absence of infected individuals. Removal of the absorbing state leads to the modified SIS (MSIS) process [13]. Note that since the MSIS dynamics has no absorbing state its asymptotic distribution has a non-trivial dependence on the infection and curing rates [13].

The master equation of the time evolution of the probabilities $p_k(t)$ corresponding to the number of infected individuals is

$$\exp(Q_n t) = \begin{pmatrix} 1 & \mathbf{0}^T \\ \mathbf{a}(t) & \exp(A_n t) \end{pmatrix}, \tag{8}$$

where

$$\mathbf{a}(t) = \delta_1 \sum_{k=1}^{\infty} \frac{t^k}{k!} A_n^{k-1} \mathbf{e}_1. \tag{9}$$

The matrix A_n is of special importance for us. It gives a connection between the SIS process and the MSIS process. The time evolution of the MSIS process is generated by \hat{Q}_n :

$$\hat{Q}_n = \begin{pmatrix} \hat{a}_1 & (n-1)\beta_1 & & & & & \\ 2\delta_2 & a_2 & 2(n-2)\beta_2 & & & & \\ & 3\delta_3 & a_3 & & & & \\ & & & \ddots & & & \\ & & & & (n-1)\beta_{n-1} & & \\ & & & n\delta_n & & a_n & \end{pmatrix}, \tag{10}$$

where the only difference between A_n and \hat{Q}_n pops up in the first entry of the main diagonal:

$$\hat{a}_1 = a_1 + \delta_1. \tag{11}$$

Thus, the two differs by only a projection of rank one:

$$\hat{Q}_n = A_n + \delta_1 P_1, \tag{12}$$

where $P_1 = \mathbf{e}_1 \mathbf{e}_1^T$. This has some important consequences. The invertibility of A_n (which we prove in section 3, see equation (36)) implies

$$\mathbf{a}(t) = \delta_1 A_n^{-1} (\exp(A_n t) - \mathbb{1}) \mathbf{e}_1, \tag{13}$$

where $\mathbb{1}$ is the $n \times n$ identity matrix. Furthermore, A_n is a tridiagonal matrix containing positive entries in the first diagonals, so it is similar to a symmetric tridiagonal matrix (see appendix A). Since the entries of $\exp(Q_n t)$ are bounded functions of t , this symmetric matrix must have non-positive eigenvalues. But A_n is invertible, so the spectrum of A_n contains only negative numbers. Therefore $\exp(A_n t)$ vanishes in the $t \rightarrow \infty$ limit. The rate of convergence is dictated by the greatest eigenvalue of A_n . This implies that in the long time limit $\mathbf{a}(t)$ approaches $-\delta_1 A_n^{-1} \mathbf{e}_1$, that is the first column of A_n^{-1} , multiplied by $-\delta_1$. This can be calculated directly, but can be stated to be equal to one by noting that the entries of the time evolution operator, i.e. $[\exp(Q_n t)]_{kl}$ give the probability that the system containing initially k infected individuals will have l infected ones after the passing of time t , so

$$\sum_{l=0}^n [\exp(Q_n t)]_{kl} = 1. \tag{14}$$

In the long time limit this equation can hold if and only if the first column of the time evolution operator contains only ones. Thus, the stationary state of the system is trivial.

Assume now that after some time t , the original process stops. The state of the system at t is given by (6). If the running time follows an exponential distribution with density $f(t) = \kappa \exp(-\kappa t)$, then the expectation value of the final state is given by

$$\int_0^{+\infty} \mathbf{p}^T(0) \exp(Q_n t) \kappa \exp(-\kappa t) dt = \kappa \mathbf{p}^T(0) (\kappa \mathbb{1} - Q_n)^{-1}. \tag{15}$$

This calculation shows that the determination of the resolvent $\mathcal{R}(Q_n; \kappa) = (\kappa \mathbb{1} - Q_n)^{-1}$ is crucial to understand the final (stochastic) state of the system. The kl entry of $\kappa \mathcal{R}(Q_n; \kappa)$ gives the average probability of observing l infected individuals when the process stops, if there were initially k infected ones. This shows that each row of $\kappa \mathcal{R}(Q_n; \kappa)$ is a probability distribution, thus its row sums are equal to one.

Integrating the product of $f(t)$ and the blocks of (8) gives that $\kappa \mathcal{R}(Q_n; \kappa)$ is of the form

$$\kappa \mathcal{R}(Q_n; \kappa) = \begin{pmatrix} 1 & 0^T \\ \mathbf{b} & \kappa \mathcal{R}(A_n; \kappa) \end{pmatrix}, \tag{16}$$

where

$$\mathbf{b} = \delta_1 A_n^{-1} (\kappa (\kappa \mathbb{1} - A_n)^{-1} - \mathbb{1}) \mathbf{e}_1 = \delta_1 \mathcal{R}(A_n; \kappa) \mathbf{e}_1, \tag{17}$$

thus $\mathcal{R}(Q_n; \kappa)$ is entirely determined by $\mathcal{R}(A_n; \kappa)$.

To study possible outbreaks in the system, we restrict our attention to the case when initially there is only one infected individual in the system, that is

$$\mathbf{p}^T(0) = (0, 1, 0, \dots, 0)^T. \tag{18}$$

Let N be the number of infected individuals at the end of the process. The expectation value of the prevalence in the final state of the system is

$$I(Q_n; \kappa) = \sum_{k=0}^n \frac{k}{n} \text{Prob}(N = k) = \kappa \sum_{k=1}^n \frac{k}{n} [\mathcal{R}(A_n; \kappa)]_{1k}, \tag{19}$$

and the probability of N being greater than zero is

$$\rho_n(\kappa) = 1 - \text{Prob}(N = 0) = \kappa \sum_{k=1}^n [\mathcal{R}(A_n; \kappa)]_{1k}. \tag{20}$$

Both $I(Q_n; \kappa)$ and $\rho_n(\kappa)$ can be calculated by the help of the first row of $\mathcal{R}(A_n; \kappa)$, which can be related to the resolvent of \hat{Q}_n , the generator of the MSIS process. To see this, note that the equality

$$\mathcal{R}(\hat{Q}_n; \kappa) - \mathcal{R}(A_n; \kappa) = \mathcal{R}(\hat{Q}_n; \kappa) (\hat{Q}_n - A_n) \mathcal{R}(A_n; \kappa), \tag{21}$$

which is called the second resolvent identity, combined with (12) gives

$$\mathbf{e}_1^T \mathcal{R}(\hat{Q}_n; \kappa) - \mathbf{e}_1^T \mathcal{R}(A_n; \kappa) = \delta_1 \mathbf{e}_1^T \mathcal{R}(\hat{Q}_n; \kappa) \mathbf{e}_1 \mathbf{e}_1^T \mathcal{R}(A_n; \kappa), \tag{22}$$

that is

$$\mathbf{e}_1^T \mathcal{R}(A_n; \kappa) = \frac{\mathbf{e}_1^T \mathcal{R}(\hat{Q}_n; \kappa)}{1 + \delta_1 [\mathcal{R}(\hat{Q}_n; \kappa)]_{11}}. \tag{23}$$

Since the entries of $\kappa\mathcal{R}(\hat{Q}_n; \kappa)$ sums up to one in any of its rows, the preceding equation gives

$$\rho_n(\kappa) = \frac{1}{1 + \delta_1[\mathcal{R}(\hat{Q}_n; \kappa)]_{11}} \tag{24}$$

and

$$I(Q_n; \kappa) = \rho_n(\kappa)I(\hat{Q}_n; \kappa), \tag{25}$$

where $I(\hat{Q}_n; \kappa)$ is the expectation value of the prevalence of the MSIS process with the same initial condition and running time distribution. As mentioned in [13], the state of the system of an SIS process for some finite time is either being in the absorbing state, or it is identical to that of the corresponding MSIS process with the same initial condition. This enables one to think about the steady state of the MSIS process as some metastate [13] or quasi-stationary distribution [25] of the SIS process. We see that such a connection translates naturally to stochastic finite running times also. Here, $I(\hat{Q}_n; \kappa)$ is the conditional expectation of the prevalence, conditioned on not being in the absorbing state of the SIS process at the stopping time.

The survival probability $\rho_n(\kappa)$ is bounded away from zero for every $0 < \kappa$. To see this, let $N(t)$ be the state of the system in a particular realization of the SIS process at some time t . We have the following inequality:

$$\begin{aligned} \text{Prob}(0 < N(t) | N(0) = 1) \\ \geq \text{Prob}(N(t') = 1 \text{ for all } 0 \leq t' \leq t) = e^{-(\delta_1 + (n-1)\beta_1)t}, \end{aligned} \tag{26}$$

where $\delta_1 + (n - 1)\beta_1$ is the overall rate of a jump in the state space from the initial state (containing a sole infected individual), see figure 1. Multiplying both sides with $\kappa \exp(-\kappa t)$ and integrating with respect to t from zero to infinity gives

$$\rho_n(\kappa) \geq \frac{\kappa}{\kappa + \delta_1 + (n - 1)\beta_1}. \tag{27}$$

If one would like to formulate a well defined $n \rightarrow \infty$ limit of the SIS process on the complete graph, one always has to regulate the infection intensity, that is we have to choose constants τ_k and set β_k as the size dependent quantity $\beta_k = \tau_k/n$ [26]. Then, the lower bound of $\rho_n(\kappa)$ is

$$\rho_n(\kappa) > \frac{\kappa}{\kappa + \delta_1 + \tau_1(1 - n^{-1})} > 0, \tag{28}$$

which remains finite in the $n \rightarrow \infty$ limit. Thus, a vanishing $\lim_{n \rightarrow \infty} I(\hat{Q}_n; \kappa)$ always implies $\lim_{n \rightarrow \infty} I(Q_n; \kappa) = 0$ and vice versa.

The introduction of a stochastic finite running time enables the study of the parameter dependence of the prevalence of the SIS process separately from the presence of the absorbing state. In the next section we show that whenever the constants τ_k are *on average* less than δ_k , then in the $n \rightarrow \infty$ limit, albeit $\rho_n(\kappa)$ remains non-zero, $I(Q_n; \kappa)$ as well as $I(\hat{Q}_n; \kappa)$ vanish. But before moving to the proof and the consequences of the statement, we illustrate the behaviour of $I(Q_n; \kappa)$ with results of numerical calculations.

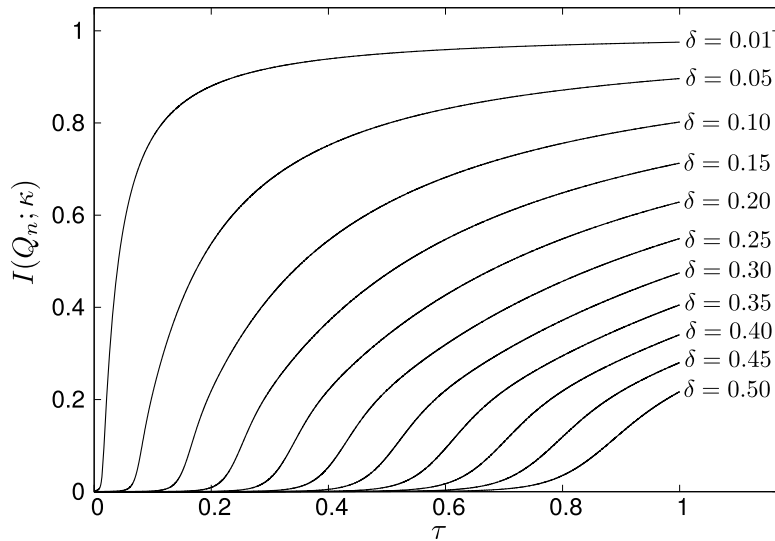


Figure 2. Average prevalence in the final state of the SIS process on K_{50} initiated by one infected individual. The resolution of the numerical calculations is $\Delta\tau = 10^{-4}$ and κ is set to 10^{-3} .

For the sake of simplicity, assume that the constants τ_k and δ_k are independent of the number of infected individuals, that is $\tau_k = \tau$ and $\delta_k = \delta$. From a practical point of view the interesting regime of the parameter space formed by τ , δ and κ is characterized by $\kappa \ll \tau, \delta$. This is the case when the timescale of the spreading process is smaller than the timescale of the decay of the group in which the spreading occurs. In order to study the effect of the finite running time on the final state of the SIS process on K_n , we set $\kappa = 10^{-3}$ and performed numerical calculation of the resolvent to obtain $I(Q_n; \kappa)$ for various values of δ . The results can be seen in figure 2, where we plot the prevalence as the function of τ . The figure shows that even a small decay rate can imply a drastic change in the final state of the system. Assuming a fixed δ , the small value of τ leads to small $I(Q_n; \kappa)$. At the beginning, increasing τ has only a little effect on the prevalence, then close to δ the situation changes and the prevalence starts to significantly increase. This rapid growth has two different phases: An initial convex phase pushes out the prevalence from the close-to-zero value, after which the forthcoming concave phase starts and leads to a moderate growth.

In order to gain insight into the $n \rightarrow \infty$ limit we fixed $\kappa = 10^{-3}$ and $\delta = 1$ and evaluated the τ dependence of the prevalence for various increasing values of the population size. Figure 3 illustrates the results. It can be seen that the width and the height of the previously mentioned convex phase become smaller and smaller as n increases. On the other hand, the concave phases seemingly accumulate in the large n limit. For sufficiently large values of τ , the difference between the curves corresponding to different population sizes becomes less and less pronounced. These numerical calculations suggest that in the large n limit the prevalence $I(Q_n; \kappa)$ remains zero if the ratio τ/δ is less than a threshold which is close to one. Above the threshold the limit of $I(Q_n; \kappa)$ is non-zero and possibly depends on κ , see figure 4.

The SIS process in populations with exponential decay

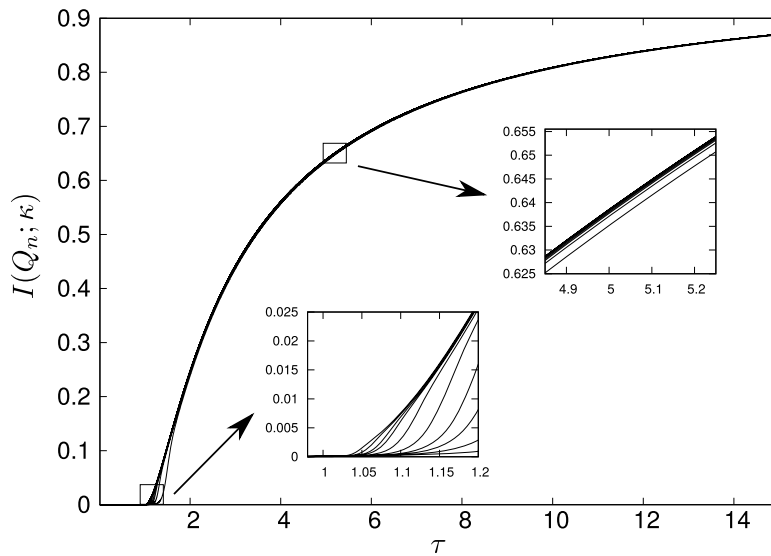


Figure 3. Average prevalence in the final state of the SIS process on K_n initiated by one infected individual. The curing rate and the decay rate of the group are set to $\delta = 1$ and $\kappa = 10^{-3}$. The resolution of the numerical calculations is $\Delta\tau = 10^{-3}$. The sizes of the populations where $I(Q_n; \kappa)$ was examined: $n = 100, 200, 300, 400, 600, 1600, 2000, 3000, 5000$. Curves corresponding to smaller n are always closer to the horizontal axis.

3. Large n limit of $\overline{\tau/\delta} < 1$

The matrix $\kappa\mathbb{1} - A_n$ is tridiagonal

$$\kappa\mathbb{1} - A_n = \begin{pmatrix} \tilde{a}_1(\kappa) & -b_2 & & & \\ -c_2 & \tilde{a}_2(\kappa) & -b_3 & & \\ & -c_3 & \ddots & \ddots & \\ & & \ddots & \ddots & -b_n \\ & & & -c_n & \tilde{a}_n(\kappa) \end{pmatrix}, \tag{29}$$

where

$$\begin{aligned} b_k &= \tau_{k-1}(k-1)(1 - (k-1)/n) \\ c_k &= k\delta_k \\ \tilde{a}_k(\kappa) &= \kappa + b_{k+1} + c_k \end{aligned} \tag{30}$$

with $b_{n+1} = 0$ and $1 \leq k \leq n$. The entries of the first row of $\mathcal{R}(A_n; \kappa) = (\kappa\mathbb{1} - A_n)^{-1}$ are given by (see appendix B)

$$[\mathcal{R}(A_n; \kappa)]_{1k} = \frac{1}{d_1(\kappa)} \frac{b_2 \cdots b_k}{d_2(\kappa) \cdots d_k(\kappa)}, \tag{31}$$

where (here and for the rest of the paper) the empty product is always understood to be equal to one and the (backward) Schur complements $d_k(\kappa)$ of $\kappa\mathbb{1} - A_n$ are defined by the recursion

The SIS process in populations with exponential decay

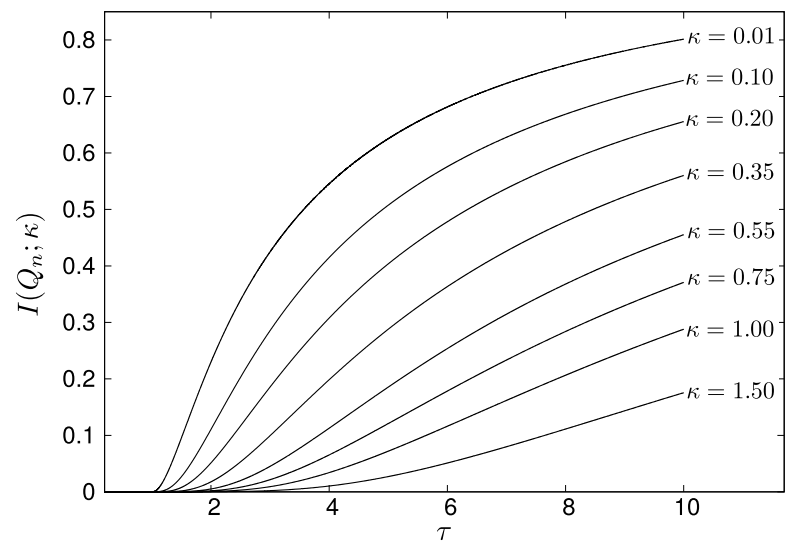


Figure 4. Average prevalence in the final state of the SIS process on K_{10000} initiated by one infected individual. The curing rate is set to $\delta = 1$. The resolution of the numerical calculations is $\Delta\tau = 2 \cdot 10^{-3}$.

$$\begin{aligned}
 d_n(\kappa) &= \tilde{a}_n(\kappa) \\
 d_k(\kappa) &= \tilde{a}_k(\kappa) - \frac{b_{k+1}c_{k+1}}{d_{k+1}(\kappa)} \quad k < n.
 \end{aligned}
 \tag{32}$$

Since the b_k 's are positive numbers, the Schur complements have to be also positive to maintain the non-negativity of the entries of $\mathcal{R}(A_n; \kappa)$. The recursion is non-linear and analytically intractable in most of the cases. Fortunately, we have the following two observations.

Firstly, members of the sequence $d_1(\kappa), \dots, d_n(\kappa)$ are monotonically increasing as functions of $\tilde{a}_k(\kappa)$ (see appendix C). Since all of the former depends linearly on κ we have

$$d_k(\kappa_1) \leq d_k(\kappa_2),
 \tag{33}$$

whenever $\kappa_1 < \kappa_2$. By equation (31) and the positivity of the Schur complements, this clearly indicates

$$[\mathcal{R}(A_n; \kappa_1)]_{1k} \geq [\mathcal{R}(A_n; \kappa_2)]_{1k}
 \tag{34}$$

for all $1 \leq k \leq n$.

Secondly, for $\kappa = 0$ the Schur complements $d_k(0)$ can be calculated explicitly and they are equal to c_k :

$$\begin{aligned}
 c_n &= a_n \\
 c_k &= c_k + b_{k+1} - b_{k+1} = a_k - \frac{b_{k+1}c_{k+1}}{c_{k+1}} \quad k < n.
 \end{aligned}
 \tag{35}$$

The determinant of a tridiagonal matrix is the product of its Schur complements (see appendix B), therefore

$$\det(-A_n) = d_1(0) \cdots d_n(0) = n! \prod_{l=1}^n \delta_l \tag{36}$$

and A_n must be invertible.

Combining equation (35) with the inequality $d_k(0) < d_k(\kappa)$ gives

$$0 \leq [\mathcal{R}(A_n; \kappa)]_{1k} \leq \frac{b_2 \cdots b_k}{c_1 c_2 \cdots c_k} \tag{37}$$

for all $0 \leq \kappa$. Thus the prevalence satisfies the inequality:

$$\begin{aligned} I(Q_n; \kappa) &= \kappa \sum_{k=1}^n \frac{k}{n} [\mathcal{R}_{1k}(A_n; \kappa)]_{1k} \\ &\leq \frac{\kappa}{\delta_1} \sum_{k=1}^n \frac{k}{n} \prod_{l=1}^{k-1} \frac{\tau_l}{\delta_{l+1}} \frac{l}{l+1} \left(1 - \frac{l}{n}\right). \end{aligned} \tag{38}$$

Using the inequality

$$\frac{l}{l+1} \left(1 - \frac{l}{n}\right) < 1 \tag{39}$$

provided that $l < n$, we arrive to

$$I(Q_n; \kappa) < \frac{\kappa}{\delta_1} \sum_{k=1}^n \frac{k}{n} \prod_{l=1}^{k-1} \frac{\tau_l}{\delta_{l+1}} < \frac{1}{n} \frac{\kappa}{\delta_1} \sum_{k=0}^{\infty} (k+1) \prod_{l=1}^k \frac{\tau_l}{\delta_{l+1}}. \tag{40}$$

The mean ratio of the normalized infection rates τ_k and the curing rates δ_k is defined as

$$\overline{\tau/\delta} = \lim_{k \rightarrow \infty} \frac{1}{k} \left(\frac{\tau_1}{\delta_2} + \cdots + \frac{\tau_k}{\delta_{k+1}} \right). \tag{41}$$

When $\overline{\tau/\delta}$ is less than one, the infinite sum on the rhs of (40) is finite. This becomes transparent when the root test and the inequality of the geometric and arithmetic mean is applied to the series. For any non-negative integer m , we have

$$\lim_{k \rightarrow \infty} \left(k^m \prod_{l=1}^k \frac{\tau_l}{\delta_{l+1}} \right)^{1/k} < \lim_{k \rightarrow \infty} \frac{k^{m/k}}{k} \left(\frac{\tau_1}{\delta_2} + \cdots + \frac{\tau_k}{\delta_{k+1}} \right) = \overline{\tau/\delta}. \tag{42}$$

With the introduction of the constants

$$\mu_m = \sum_{k=0}^{\infty} k^m \prod_{l=1}^k \frac{\tau_l}{\delta_{l+1}} \tag{43}$$

the previous result leads to

$$I(Q_n; \kappa) < \frac{1}{n} \frac{\kappa}{\delta_1} (\mu_0 + \mu_1). \tag{44}$$

This implies that

$$\lim_{n \rightarrow \infty} I(Q_n; \kappa) = \lim_{n \rightarrow \infty} I(\hat{Q}_n, \kappa) = 0 \tag{45}$$

for all $0 < \kappa$. That is, if $\overline{\tau/\delta} < 1$, the introduction of a finite running time cannot sustain a finite prevalence in the $n \rightarrow \infty$ limit because *even the MSIS process which does not have an absorbing state has a vanishing average prevalence*, which is one of the main results of our paper. We show three other consequences of the inequality (44).

First, we show that (45) implies that the average prevalence of the stationary state of the MSIS process initiated by a single infected individual is zero in the $n \rightarrow \infty$ limit whenever $\overline{\tau/\delta} < 1$. For uniform τ_k and δ_k , this has been proved in [27] or [13]. Here, we give an independent proof in the general, state dependent case.

The matrix $\mathcal{R}(\hat{Q}_n; \kappa)$ has the decomposition

$$\mathcal{R}(\hat{Q}_n; \kappa) = \sum_{k=1}^n \frac{1}{\kappa - \lambda_k(\hat{Q}_n)} \mathbf{r}_k \mathbf{l}_k^T, \tag{46}$$

where \mathbf{r}_k and \mathbf{l}_k^T are the right and left eigenvectors of \hat{Q}_n corresponding to the eigenvalues $\lambda_k(\hat{Q}_n)$ which are now indexed such that they form a descending sequence:

$$0 = \lambda_1(\hat{Q}_n) > \lambda_2(\hat{Q}_n) \geq \dots \geq \lambda_n(\hat{Q}_n). \tag{47}$$

The right and left eigenvector corresponding to the zero eigenvalue are the vector $\mathbf{1}$ containing only ones and $\boldsymbol{\pi}$, the vector that contains the stationary probabilities of the MSIS process. To calculate the prevalence, one has to calculate $\kappa \mathcal{R}(\hat{Q}_n; \kappa)$:

$$\kappa \mathcal{R}(\hat{Q}_n; \kappa) = \mathbf{1} \boldsymbol{\pi}^T + \sum_{k=2}^n \frac{\kappa}{\kappa - \lambda_k(\hat{Q}_n)} \mathbf{r}_k \mathbf{l}_k^T. \tag{48}$$

The expectation value of the prevalence in the stationary state of the MSIS process is

$$P(\hat{Q}_n) = \sum_{k=1}^n \frac{k}{n} \pi_k. \tag{49}$$

Using the n component vector

$$\mathbf{x} = (1/n, 2/n, \dots, (n-1)/n, 1)^T, \tag{50}$$

we have

$$I(\hat{Q}_n; \kappa) = P(\hat{Q}_n) + \sum_{k=2}^n \frac{\kappa}{\kappa - \lambda_k(\hat{Q}_n)} \mathbf{e}_1^T \mathbf{r}_k \cdot \mathbf{l}_k^T \mathbf{x}. \tag{51}$$

The left and right eigenvectors are of the form $\mathbf{l}_k^T = (T \mathbf{u}_k)^T$ and $\mathbf{r}_k = T^{-1} \mathbf{u}_k$, where \mathbf{u}_k is a unit vector and T is a diagonal matrix with entries $[T]_{11} = 1$ and

$$[T]_{kk} = \sqrt{\frac{b_2 \cdots b_k}{c_2 \cdots c_k}} \quad 1 < k \leq n \tag{52}$$

in the main diagonal (see appendix A). Hence the magnitude of the scalar products of (51) are bounded by

$$|\mathbf{l}_k^T \mathbf{x}| = |\mathbf{u}_k^T T \mathbf{x}| \leq \|T \mathbf{x}\|, \tag{53}$$

and

$$|\mathbf{e}_1^T \mathbf{r}_k| = |\mathbf{e}_1^T T^{-1} \mathbf{u}_k| = |\mathbf{e}_1^T \mathbf{u}_k| \leq 1, \tag{54}$$

with $\|\cdot\|$ being the Euclidean norm. We also have an upper bound on $\|T\mathbf{x}\|$:

$$\begin{aligned} \|T\mathbf{x}\| &= \sum_{k=1}^n \frac{k}{n} \sqrt{\frac{(k-1)!}{k!} \prod_{l=1}^{k-1} \left(1 - \frac{l}{n}\right) \prod_{l=1}^{k-1} \frac{\tau_l}{\delta_{l+1}}} \\ &\leq \sum_{k=1}^n \frac{1}{n} \sqrt{k \prod_{l=1}^{k-1} \frac{\tau_l}{\delta_{l+1}}} \\ &\stackrel{\text{CBS}}{\leq} \sqrt{\sum_{k=1}^n \frac{k}{n} \prod_{l=1}^{k-1} \frac{\tau_l}{\delta_{l+1}}} \\ &\leq \sqrt{\frac{1}{n} \sum_{k=1}^{\infty} k \prod_{l=1}^{k-1} \frac{\tau_l}{\delta_{l+1}}} = \sqrt{\frac{\mu_0 + \mu_1}{n}}, \end{aligned} \tag{55}$$

where μ_0 and μ_1 are defined in (43). Let $\kappa_n = |\lambda_2(\hat{Q}_n)|\alpha_n^{-1}e^{-n}$, where

$$\alpha_n = \max\{1, \tau_1 + \delta_1, \dots, \tau_n + \delta_n\}, \tag{56}$$

then

$$\begin{aligned} |I(\hat{Q}_n; \kappa_n) - P(\hat{Q}_n)| &< \sum_{k=2}^n \sqrt{\frac{\mu_0 + \mu_1}{n}} \frac{1}{1 + e^n \alpha_n |\lambda_k(\hat{Q}_n)/\lambda_2(\hat{Q}_n)|} \\ &< \sum_{k=2}^n \sqrt{\frac{\mu_0 + \mu_1}{n}} e^{-n} \\ &< \sqrt{\mu_0 + \mu_1} n^{1/2} e^{-n}, \end{aligned} \tag{57}$$

that is in the $n \rightarrow \infty$ limit $I(\hat{Q}_n; \kappa_n)$ and $P(\hat{Q}_n)$ converge to the same number. Since the magnitude of $\lambda_k(\hat{Q}_n)/\alpha_n$ is bounded from above (see appendix A) by

$$\frac{|\lambda_k(\hat{Q}_n)|}{\alpha_n} < 2n \frac{\max_{1 \leq k \leq n} \{\tau_k + \delta_k\}}{\alpha_n} < 2n, \tag{58}$$

the sequence κ_n tends to zero when n goes to infinity. We can write

$$\begin{aligned} P(\hat{Q}_n) &< \sqrt{\mu_0 + \mu_1} n^{1/2} e^{-n} + \frac{I(Q_n; \kappa_n)}{\rho_n(\kappa_n)} \\ &\stackrel{(28)}{<} \sqrt{\mu_0 + \mu_1} n^{1/2} e^{-n} + \frac{I(Q_n; \kappa_n)}{\kappa_n} (\kappa_n + \delta_1 + \tau_1 (1 - n^{-1})) \\ &\stackrel{(44)}{<} \sqrt{\mu_0 + \mu_1} n^{1/2} e^{-n} + \frac{\mu_0 + \mu_1}{n} \frac{\kappa_n + \delta_1 + \tau_1}{\delta_1}, \end{aligned} \tag{59}$$

from which

$$\lim_{n \rightarrow \infty} P(\hat{Q}_n) = 0 \tag{60}$$

readily follows.

The second consequence concerns the point-wise limit of $P(\hat{Q}_n; t)$, that is the full time evolution of the expectation value of the prevalence of the MSIS process initiated by a single infected individual. If $N(t)$ denotes the number of infected individuals, then

$$P(\hat{Q}_n; t) = \sum_{k=1}^n \frac{k}{n} \text{Prob}(N(t) = k | N(0) = 1) = \sum_{k=1}^n \frac{k}{n} [\exp(t\hat{Q}_n)]_{1k}. \quad (61)$$

The Laplace transform of $P(\hat{Q}_n; t)$ is

$$\hat{P}(\hat{Q}_n; z) = \int_0^{+\infty} P(\hat{Q}_n; t) e^{-zt} dt = \mathbf{e}_1^T \mathcal{R}(\hat{Q}_n; z) \mathbf{x}, \quad (62)$$

which is finite if $\Re[z]$, the real part of $z \in \mathbb{C}$ is positive, due to the fact that \hat{Q}_n has non-positive eigenvalues. The second resolvent identity

$$\mathcal{R}(A_n; z) - \mathcal{R}(\hat{Q}_n; z) = -\delta_1 \mathcal{R}(A_n; z) \mathbf{e}_1 \mathbf{e}_1^T \mathcal{R}(\hat{Q}_n; z) \quad (63)$$

gives

$$\mathbf{e}_1^T \mathcal{R}(\hat{Q}_n; z) \mathbf{x} = \frac{\mathbf{e}_1^T \mathcal{R}(A_n; z) \mathbf{x}}{1 - \delta_1 [\mathcal{R}(A_n; z)]_{11}}, \quad (64)$$

which enables us to give an upper bound of the magnitude of $\hat{P}(\hat{Q}_n; z)$ as follows. Since

$$[\mathcal{R}(A_n; z)]_{1k} = \frac{1}{d_1(z)} \frac{b_2 \cdots b_k}{d_2(z) \cdots d_k(z)}, \quad (65)$$

where $d_k(z)$ are the Schur complements of $z\mathbb{1} - A_n$, equation (64) can be written as

$$\mathbf{e}_1^T \mathcal{R}(\hat{Q}_n; z) \mathbf{x} = \frac{1}{d_1(z) - \delta_1} \sum_{k=1}^n \frac{k}{n} \frac{b_2 \cdots b_k}{d_2(z) \cdots d_k(z)}. \quad (66)$$

Let $z = \kappa + i\omega$ and assume that $0 < \kappa$. In appendix C, we show that $\Re[d_k(\kappa + i\omega)]$ has the lower bound

$$\Re[d_k(\kappa + i\omega)] \geq \kappa + d_k(0) = \kappa + c_k, \quad (67)$$

which results in the following upper bound:

$$\begin{aligned} \left| \sum_{k=1}^n \frac{k}{n} \frac{b_2 \cdots b_k}{d_2(z) \cdots d_k(z)} \right| &\leq \sum_{k=1}^n \frac{k}{n} \frac{b_2 \cdots b_k}{|d_2(z)| \cdots |d_k(z)|} \\ &\leq \sum_{k=1}^n \frac{k}{n} \frac{b_2 \cdots b_k}{d_2(0) \cdots d_k(0)} \\ &\leq \sum_{k=1}^n \frac{k}{n} \frac{b_2 \cdots b_k}{c_2 \cdots c_k} \\ &< \frac{\mu_0 + \mu_1}{n}, \end{aligned} \quad (68)$$

where we have used the same bounds for the sum that were used in the derivation of (44). Since the inequality

$$\Re[\delta_1 - d_1(\kappa + i\omega)] \leq \delta_1 - \kappa - c_1 = -\kappa \tag{69}$$

also holds (see appendix C) and results in

$$|\delta_1 - d_1(\kappa + i\omega)| \geq \kappa, \tag{70}$$

we see that—as a function of the variable ω —the magnitude of the Laplace transform $\widehat{P}(\widehat{Q}_n; \kappa + i\omega)$ is uniformly bounded by

$$|\widehat{P}(\widehat{Q}_n; \kappa + i\omega)| < \frac{1}{\kappa} \frac{\mu_0 + \mu_1}{n}, \tag{71}$$

that is $\widehat{P}(\widehat{Q}_n; \kappa + i\omega)$ converges uniformly to the constant zero function on the $\Re[z] = \kappa$ line of the complex plane. This implies the same convergence of the integrand of the inverse Laplace transform

$$P(\widehat{Q}_n; t) = \frac{1}{2\pi} \int_{-\infty}^{+\infty} \widehat{P}(\widehat{Q}_n; \kappa + i\omega) e^{(\kappa+i\omega)t} d\omega \tag{72}$$

for all $0 < t$, i.e. when $\overline{\tau/\delta} < 1$ the limit curve of the sequence $P(\widehat{Q}_n; t)$, that is the time evolution of the prevalence of the MSIS process initiated by a single individual vanishes in the large n limit for all time instances $0 < t$. By the absence of the absorbing state $P(Q_n; t) \leq P(\widehat{Q}_n; t)$ also holds and implies similar consequences for the large n limit of $P(Q_n; t)$.

Finally, let $f(t)$ be an arbitrary running time distribution of the process. The Fourier transform of $f(t)$ is denoted by $\widehat{f}(\omega)$. The average prevalence after the halt of the SIS dynamics initiated by only one infected individual is

$$I_f(Q_n) = \int_0^{+\infty} \mathbf{e}_1^T \exp(A_n t) \mathbf{x} f(t) dt = \int_{-\infty}^{+\infty} \widehat{f}(\omega) \mathbf{e}_1^T \mathcal{R}(A_n; -i\omega) \mathbf{x} d\omega. \tag{73}$$

The magnitude of the integrand is bounded by

$$|\widehat{f}(\omega) \mathbf{e}_1^T \mathcal{R}(A_n; -i\omega) \mathbf{x}| < \frac{1}{n} \frac{|\widehat{f}(\omega)|}{\delta_1} (\mu_0 + \mu_1), \tag{74}$$

which can be obtained by using (68) and $|d_1(-i\omega)| \geq \delta_1$ for the first Schur complement of $z\mathbb{1} - A_n$. Since $|\widehat{f}(\omega)| \leq 1$, the integrand converges uniformly to the constant zero function. Thus, whenever $\tau/\delta < 1$ holds, using any distribution of the running time of the process results in a vanishing average prevalence of the SIS process (initiated by a sole infected individual) in the final state, in the $n \rightarrow \infty$ limit.

4. Large n limit of $\delta < \tau$

In this section, we analyze the usual SIS model whose individual normalized infection rates do not depend on the number of infected individuals, that is

$$\begin{aligned} b_{k+1} &= \tau(k-1)(1-(k-1)/n), \\ c_k &= k \quad 1 \leq k \leq n, \end{aligned} \tag{75}$$

where, for the sake of simplicity, we have set $\delta = 1$. Our result here is only partial, that is, it does not cover the whole parameter space formed by κ and τ . However it remains significant, because in a huge region of the parameter space, we are able to demonstrate the absence of a finite prevalence in the $n \rightarrow \infty$ limit: We prove that *whenever* $2 \leq \tau$ and κ satisfies the inequality

$$\frac{7\tau + 1}{2} \leq \kappa, \tag{76}$$

then the prevalence $I(Q_n; \kappa)$ vanishes in the $n \rightarrow \infty$ limit. In order to prove the statement, we first give a lower bound of the Schur complements of the matrix $\kappa \mathbb{1} - A_n$. In appendix C, we show that whenever

$$\frac{3\tau + 1}{2} \leq \kappa \tag{77}$$

holds, then the Schur complements of $\kappa \mathbb{1} - A_n$ have the lower bound

$$d_k(\kappa) > \kappa - \frac{3\tau + 1}{2} + \max\{b_k, c_k\}, \tag{78}$$

see inequality (C.62) and the discussion that precedes it. Furthermore, if $2 \leq \tau$, then for all $2 < k < nx_\tau - 1$, where $x_\tau = 1 - \tau^{-1}$, the inequality $c_k < b_k$ holds, while for all $nx_\tau - 1 < k \leq n$, we have $b_k < c_k$ (see appendix C, especially inequality (C.68)). In the first case, $\max\{b_k, c_k\} = b_k$ and the product $b_k/d_k(\kappa)$ has the upper bound

$$\begin{aligned} \frac{b_k}{d_k(\kappa)} &< \frac{b_k}{\kappa - (3\tau + 1)/2 + b_k} \\ &< \frac{(k-1)(1-k/n)}{G(\kappa, \tau) + (k-1)(1-k/n)} \\ &< \frac{k-1}{G(\kappa, \tau) + k-1}, \end{aligned} \tag{79}$$

where $G(\kappa, \tau)$ stands for

$$G(\kappa, \tau) = \frac{\kappa}{\tau} - \frac{1}{2\tau} - \frac{3}{2}. \tag{80}$$

Note that $2 \leq G(\kappa, \tau)$ if and only if inequality (76) holds. In the second case, $\max\{b_k, c_k\} = c_k$, so

$$\begin{aligned} \frac{b_k}{d_k(\kappa)} &< \frac{b_k}{\kappa - (3\tau + 1)/2 + c_k} \\ &< \frac{b_k}{\kappa - (3\tau + 1)/2 + b_k} \\ &< \frac{k-1}{G(\kappa, \tau) + k-1}. \end{aligned} \tag{81}$$

Thus, the prevalence $I(Q_n; \kappa)$ has the upper bound

$$\begin{aligned}
 I(Q_n; \kappa) &< \\
 &< \frac{\kappa}{d_1(\kappa)} \frac{1}{n} + \frac{\kappa b_2}{d_1(\kappa) d_2(\kappa)} \frac{2}{n} + \frac{\kappa b_2}{d_1(\kappa) d_2(\kappa)} \sum_{k=3}^n \frac{k}{n} \prod_{l=3}^k \frac{l-1}{G(\kappa, \tau) + l - 1}.
 \end{aligned} \tag{82}$$

Assuming $2 \leq G(\kappa, \tau)$, we get

$$\sum_{k=3}^n k \prod_{l=3}^k \frac{l-1}{G(\kappa, \tau) + l - 1} < \sum_{k=3}^n k \prod_{l=3}^k \frac{l-1}{l+1}. \tag{83}$$

The sum in the rhs can be evaluated explicitly:

$$\sum_{k=3}^n k \prod_{l=3}^k \frac{l-1}{l+1} = \sum_{k=3}^n \frac{6}{k+1} = 6(H_{n+1} - H_3), \tag{84}$$

where H_k is the k^{th} harmonic number. This gives rise to the inequality

$$I(Q_n; \kappa) < \frac{\kappa}{d_1(\kappa)} \frac{1}{n} + \frac{\kappa b_2}{d_1(\kappa) d_2(\kappa)} \frac{2}{n} + \frac{6\kappa b_2}{d_1(\kappa) d_2(\kappa)} (H_{n+1} - H_3). \tag{85}$$

In the $n \rightarrow \infty$ limit, all the terms of the rhs of the preceding inequality converges to zero, thus

$$\lim_{n \rightarrow \infty} I(Q_n; \kappa) = 0, \tag{86}$$

which is the desired result. This calculation also allows to give an upper bound on the prevalence if $1 \leq G(\kappa, \tau) < 2$ holds. In that case, instead of (83), we have

$$\sum_{k=3}^n k \prod_{l=3}^k \frac{l-1}{G(\kappa, \tau) + l - 1} < \sum_{k=3}^n k \prod_{l=3}^k \frac{l-1}{l} = 2(n-2), \tag{87}$$

which can be inserted to (82). Then, using (C.11) and taking the $n \rightarrow \infty$ limit on both sides gives

$$\lim_{n \rightarrow \infty} I(Q_n; \kappa) < \frac{2\kappa\tau}{(\kappa+1)(\kappa+2)} \quad \frac{5\tau+1}{2} \leq \kappa < \frac{7\tau+1}{2}. \tag{88}$$

In figure 5, we summarize the results concerning the usual SIS model. If $\tau/\delta < 1$, then there is no non-vanishing prevalence in the large n limit, regardless of the value of κ . The same remains true if $2 < \tau/\delta$ and κ/δ is great enough to exceed $7\tau/2\delta + 1/2$. The parameter regime $1 < \tau/\delta < 2$ remained completely untouched. Nonetheless, we believe that $\tau/\delta = 2$ is not a real critical value, where the process significantly changes its behaviour, but its appearance is supposed to be more or less a result of the approximation method we used in appendix C, that is it can be eliminated from the analysis in the future, if one works out better approximation schemes of the Schur complements. The low κ/δ regime remains undiscovered. Here, the numerical results that have been presented for instance in figure 3 supports the idea of a non-vanishing prevalence in the $n \rightarrow \infty$ limit. Furthermore, we can give a theoretical argument also: To be more precise, we show that if $\delta < \tau$ holds, then an upper bound $I(Q_n; \kappa)/\kappa < B(n)$ which is

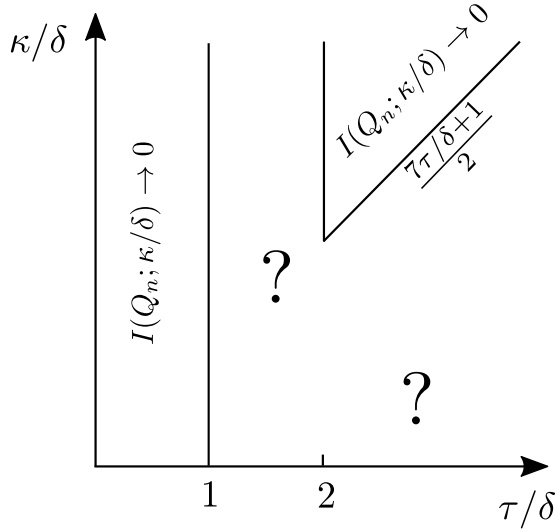


Figure 5. The prevalence $I(Q_n; \kappa)$ in the large n limit, as the function of the parameters κ/δ and τ/δ .

uniform with respect to $0 < \kappa$ and vanishes in the large n limit cannot exist. To prove, note that if $\delta < \tau$ holds, then the magnitude of the vector $\|T\mathbf{x}\|$, introduced in equation (53) has the upper bound

$$\begin{aligned} \|T\mathbf{x}\| &\leq \sqrt{\sum_{k=1}^n \frac{k}{n} \left(\frac{\tau}{\delta}\right)^{k-1}} \\ &\leq \sqrt{\frac{1}{n} \frac{(n-1)(\tau/\delta)^n - n(\tau/\delta)^{n-1}}{(\tau/\delta - 1)^2}} \\ &\leq e^{\frac{n}{2} \ln(\tau/\delta)} (\tau/\delta - 1)^{-1}, \end{aligned} \tag{89}$$

which can be shown analogously as in (55). Let κ_n be defined by

$$\kappa_n = |\lambda_2(\hat{Q}_n)|(\tau + \delta)^{-1} e^{-n(1 + \frac{1}{2} \ln(\tau/\delta))}, \tag{90}$$

where $\lambda_2(\hat{Q}_n)$ is the second largest eigenvalue of \hat{Q}_n , the infinitesimal generator of the MSIS process. Then, in the same vein as it has been presented in (57), one can show that the difference of the prevalence of the MSIS process $I(\hat{Q}_n; \kappa_n)$ and its stationary value $P(\hat{Q}_n)$ is bounded by

$$|I(\hat{Q}_n; \kappa_n) - P(\hat{Q}_n)| < \sum_{k=2}^n \frac{e^{-n}}{\tau/\delta - 1} < \frac{ne^{-n}}{\tau/\delta - 1} \tag{91}$$

that is, when $n \rightarrow \infty$, κ_n as well as the difference tend to zero. On the other hand, $I(\hat{Q}_n; \kappa_n)$ and $I(Q_n; \kappa_n)$ are related to each other by equality (25), so the same calculation that was carried out in (59) now results in

$$\begin{aligned} P(\hat{Q}_n) &< \frac{ne^{-n}}{\tau/\delta - 1} + \frac{I(Q_n; \kappa_n)}{\kappa_n} (\kappa_n + \tau + \delta) \\ &< \frac{ne^{-n}}{\tau/\delta - 1} + B(n) (\kappa_n + \tau + \delta). \end{aligned} \tag{92}$$

Thus if $B(n)$ tends to zero as $n \rightarrow \infty$, then $P(\hat{Q}_n)$ must vanish in the large n limit which is known to be not the case [27].

5. Summary and outlook

In this paper we examined the effect of finite, stochastic stopping time on the Markovian SIS and MSIS dynamics. We examined the average prevalence in the final state of these systems if the spreading process was initiated by a sole infected individual. We have shown numerical evidence that in the $n \rightarrow \infty$ limit universal behaviour occurs. We have proven this universality when the normalized infection rate is on average less than the curing rate. We have shown that the upper bounds on the magnitude of the entries of the resolvent of the infinitesimal generator of these processes lead to results concerning the full time evolution of the SIS process also. We have proven that the choice of the distribution of the stopping time, i.e. the exponential distribution was not restrictive at all in the $\bar{\tau}/\bar{\delta} < 1$ parameter regime: Similar conclusions apply for any other distribution. In case of the usual SIS model, we have shown that $\delta < \tau$ is not enough to maintain a non-vanishing average prevalence in the large n limit: If the expectation value of the exponentially distributed stopping time is not large enough, the process cannot sustain significant average prevalence.

Historically, the first mathematical epidemiological models were based on the assumption of homogeneous mixing. These models and their refinements (like for instance the age-structured models) have the advantages of being analytically tractable but they miss the capability of describing the dynamics of the contagion on the short time scale or to incorporate the non-homogeneous structure of the contact networks of populations where the spreading takes place. The data driven interest in contact networks states urgent demand for quantitative description of spreading processes evolving on time-varying contact networks. Nevertheless simulations gave crucial insights how epidemics evolve in artificial [28] or empirical [19, 29, 30] time-varying networks, the phenomenological models of these spreading processes are still in their infancy. One possible direction to break the homogeneity assumption of certain epidemiological models is considering group formation dynamics [21], where groups can form and decay in a stochastic manner. While each group is well mixed within the collection of the individuals, the whole population has a contact structure that can be very far from being studiable with the usual homogeneous models. This is where our considerations can have much impact.

To interpret the state dependent transition rates τ_k and δ_k that were used in section 3, we just mention the well known processes which can affect the transition rates of epidemiological processes, for instance immunization, state organized preventive actions, the change of behaviour of humans under pandemics, uncertainty in modelling, etc. Enabling the infection and curing rates to depend on the number of the infected individuals can have theoretical importance also. For instance, consider the sequence $\tau_k = r_k \delta_{k+1}$, where the sequence r_k is formed by i.i.d. random variables of mean r . If $r < 1$, the, the strong law of large numbers guarantees, that the SIS processes

parametrized by an arbitrary (positive) sequence δ_k and the corresponding τ_k satisfies almost surely the assumptions of the statements given in the main text.

The future directions of the theoretical research consists of further exploration of the $1 < \overline{\tau/\delta}$ parameter regime, the study of different initial conditions and the generalization of the methods presented here to other homogeneous processes formulated on the complete graph K_n .

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Appendix A. Spectral decomposition of tridiagonal matrices

Throughout the text we use tridiagonal matrices. Here, we review some of their properties. Let a_k , b_k and c_k be sequences of length n , the subscripts having values $1 \leq k \leq n$. A tridiagonal matrix M_n has the form

$$M_n = \begin{pmatrix} a_1 & -b_2 & & & \\ -c_2 & a_2 & -b_3 & & \\ & -c_3 & \ddots & \ddots & \\ & & \ddots & \ddots & -b_n \\ & & & -c_n & a_n \end{pmatrix}. \tag{A.1}$$

If none of the members of the off-diagonal sequences are equal to zero and the sign of b_k is always the same as the sign of the corresponding c_k , then the similarity transformation with the diagonal matrix T defined through

$$\begin{aligned} [T]_{11} &= 1 \\ [T]_{kk} &= \sqrt{\frac{b_2 \cdots b_k}{c_2 \cdots c_k}} \quad 2 \leq k \end{aligned} \tag{A.2}$$

turns M_n into a symmetric tridiagonal matrix with entries $-\sqrt{c_k b_k}$ in the first diagonals. Whenever λ is an eigenvalue of $T M_n T^{-1}$ with the corresponding eigenvector \mathbf{u} , the vectors $\mathbf{u}^T T$ and $T^{-1} \mathbf{u}$ are left and right eigenvectors of M_n , corresponding to the same eigenvalue. Thus, the spectral decomposition of M_n is

$$M_n = \sum_{k=1}^n \lambda_k(M_n) \cdot (T^{-1} \mathbf{u}_k)(T \mathbf{u}_k)^T. \tag{A.3}$$

The non trivial entries of the diagonal matrix that symmetrize the infinitesimal generator of the MSIS process \hat{Q}_n with individual infection rates $\beta_k = \tau_k/n$ and curing rates δ_k , are given by

$$[T]_{kk}^2 = \frac{(k-1)!}{k!} \prod_{l=1}^{k-1} \left(1 - \frac{l}{n}\right) \prod_{l=1}^{k-1} \frac{\tau_l}{\delta_{l+1}}. \tag{A.4}$$

One can calculate an upper bound of the magnitude of the eigenvalues of \hat{Q}_n with the help of Gershgorin's circle theorem [31]: An eigenvalue of \hat{Q}_n is contained within at least one interval of the real line of radius $\sum_{l \neq k} |[\hat{Q}_n]_{kl}|$ and center $[\hat{Q}_n]_{kk}$. Since \hat{Q}_n is an infinitesimal generator of a Markov process, we have $[\hat{Q}_n]_{kk} = -\sum_{l \neq k} [\hat{Q}_n]_{kl}$, thus

$$|\lambda_k(\hat{Q}_n)| < 2 \max_{1 \leq k \leq n} (b_{k+1} + c_k) < 2 \max_{1 \leq k \leq n} (\tau_k k + \delta_k k) < 2n \max_{1 \leq k \leq n} (\tau_k + \delta_k) \quad (\text{A.5})$$

which results in

$$|\lambda_k(\hat{Q}_n)| < 2n \max_{1 \leq k \leq n} \{\tau_k + \delta_k\}. \quad (\text{A.6})$$

Appendix B. Inversion of tridiagonal matrices

The canonical $n \times n$ shift matrix is

$$S_n = \begin{pmatrix} 0 & 1 & & & \\ & 0 & 1 & & \\ & & 0 & 1 & \\ & & & \ddots & 1 \\ & & & & 0 \end{pmatrix}. \quad (\text{B.1})$$

We introduce the notational convention that whenever x_1, \dots, x_n is a sequence, \hat{x} denotes the diagonal matrix whose entries in the main diagonal are the members of the sequence, i.e. $[\hat{x}]_{kk} = x_k$. The matrix M_n in (A.1) can be written in the compact form

$$M_n = \hat{a} - S_n \hat{b} - \hat{c} S_n^T. \quad (\text{B.2})$$

Assume that M_n is invertible. The so called *UDL* decomposition of M_n is a factorization of M_n such that U is an upper, L is a lower triangular matrix, both of them containing ones in their main diagonal and D is a diagonal matrix. For tridiagonal matrices, the *UDL* decomposition is relatively simple. Define the sequence d_1, \dots, d_n by the backward recursion relations

$$\begin{aligned} d_n &= a_n, \\ d_k &= a_k - \frac{b_{k+1}c_{k+1}}{d_{k+1}} \quad k < n, \end{aligned} \quad (\text{B.3})$$

then the matrices

$$\begin{aligned} U &= \mathbb{1} - S_n \hat{b} \hat{d}^{-1}, \\ L &= \mathbb{1} - \hat{d}^{-1} \hat{c} S_n^T, \\ D &= \hat{d} \end{aligned} \quad (\text{B.4})$$

give the desired decomposition. The members of the sequence d_1, \dots, d_n are called the (backward) Schur complements of M_n . Since $\det(U) = \det(L) = 1$, the determinant of M_n is equal to the determinant of D , that is

$$\det(A) = d_1 \cdots d_n. \tag{B.5}$$

Thus, an invertible M_n has non-vanishing Schur complements.

The inversion of U and L can be carried out explicitly using Gaussian elimination:

$$(L^{-1})_{kl} = \begin{cases} \frac{c_l \cdots c_{k-1}}{d_l \cdots d_{k-1}} & \text{if } k < l \\ 1 & \text{if } k = l \\ 0 & \text{if } k > l \end{cases} \tag{B.6}$$

and

$$(U^{-1})_{kl} = \begin{cases} \frac{b_l \cdots b_{k-1}}{d_l \cdots d_{k-1}} & \text{if } l < k \\ 1 & \text{if } l = k \\ 0 & \text{if } l > k \end{cases}. \tag{B.7}$$

Our primary interest concerns usually the first row of the inverse of a tridiagonal matrix. This can be calculated easily in the UDL decomposition. Since L^{-1} is a lower triangular matrix, it leaves e_1^T invariant when multiplied from the left, thus

$$[e_1^T(UDL)^{-1}]_k = [\hat{d}^{-1}U^{-1}]_{1k} = \frac{1}{d_1} \frac{b_2 \cdots b_k}{d_2 \cdots d_k} \tag{B.8}$$

where an empty product—if it appears—understood to be equal to one. More on the inversion of the tridiagonal matrices can be found in [32, 33] and references therein.

Appendix C. Monotonicity of the Schur complements and some of its consequences

The members of the sequence D_1, \dots, D_n , which satisfy the recursion

$$\begin{aligned} D_n &= a_n \\ D_k &= a_k - \frac{b_{k+1}c_{k+1}}{D_{k+1}} \quad 1 \leq k < n \end{aligned} \tag{C.1}$$

are monotone functions of the members of the sequence a_1, \dots, a_n , if the signs of b_k and the corresponding c_k are the same. To see this, note that by the backward recursion relations, D_k depends only on a_k, \dots, a_n . This gives

$$\frac{\partial}{\partial a_k} D_k(a_1, \dots, a_n) = 1 \tag{C.2}$$

and

$$\frac{\partial}{\partial a_l} D_k(a_1, \dots, a_n) = \frac{b_{k+1}c_{k+1}}{d_{k+1}^2} \frac{\partial}{\partial a_l} D_{k+1}(a_1, \dots, a_n), \tag{C.3}$$

if $k < l$. The repeated application of this formula results in

$$\frac{\partial}{\partial a_l} D_k(a_1, \dots, a_n) = \frac{b_{k+1}c_{k+1} \cdots b_l c_l}{D_{k+1}^2 \cdots D_l^2}, \tag{C.4}$$

for all $k < l \leq n$. Since $0 \leq b_{k+1}c_{k+1}$, we arrive to the desired result.

In Section 3, we study the Schur complements of the matrix $z\mathbb{1} - A_n$ where $z = \kappa + i\omega$ has a non-negative real part $0 \leq \kappa$. The matrix $z\mathbb{1} - A_n$ is tridiagonal and has the following relationship between entries in the main and first diagonals (in the notation of (A.1)):

$$\begin{aligned} a_n &= c_n \\ a_k &= z + c_k + b_{k+1} \quad 1 \leq k < n \end{aligned} \tag{C.5}$$

with the positivity prescription that $0 < b_k, c_k$. For $z = 0$, the Schur complements can be calculated explicitly:

$$a_k = c_k \quad 1 \leq k \leq n. \tag{C.6}$$

We show that the z dependent Schur complements $d_k(z)$ of $z\mathbb{1} - A_n$ satisfy the inequality

$$\Re[d_k(z)] \geq \kappa + d_k(0) = \kappa + c_k \tag{C.7}$$

for all $1 \leq k \leq n$ if $\kappa = \Re[z]$ is non-negative. The real part of the Schur complements satisfy the recursion

$$\begin{aligned} \Re[d_n(z)] &= \kappa + c_n \\ \Re[d_k(z)] &= \kappa + c_k + b_{k+1} - \frac{b_{k+1}c_{k+1}}{|d_{k+1}(z)|^2} \Re[d_{k+1}(z)]. \end{aligned} \tag{C.8}$$

Note that the relationship

$$\Re[d_n(z)] \geq \kappa + c_n \tag{C.9}$$

trivially holds. Assuming $\Re[d_{k+1}(z)] \geq \kappa + c_{k+1}$, we get

$$\begin{aligned} \Re[d_k(z)] &= \kappa + c_k + b_{k+1} - \frac{b_{k+1}c_{k+1}\Re[d_{k+1}(z)]}{\Re[d_{k+1}(z)]^2 + \Im[d_{k+1}(z)]^2} \\ &\geq \kappa + c_k + b_{k+1} - \frac{b_{k+1}c_{k+1}}{\Re[d_{k+1}(z)]} \\ &\geq \kappa + c_k + b_{k+1} - \frac{b_{k+1}c_{k+1}}{\kappa + c_{k+1}} \\ &\geq \kappa + c_k + b_{k+1} - \frac{b_{k+1}c_{k+1}}{c_{k+1}} \\ &\geq \kappa + c_k, \end{aligned} \tag{C.10}$$

which proves the desired result for all $1 \leq k < n$.

For positive κ , the Schur complements of $\kappa\mathbb{1} - A_n$ are also positive and the recursion of (C.1) tells that they are bounded from above by $\kappa + c_k + b_{k+1}$. Setting b_{n+1} to be equal to zero, we have the following estimates of the Schur complements

$$\kappa + c_k \leq d_k(\kappa) \leq \kappa + c_k + b_{k+1} \quad 1 \leq k \leq n. \tag{C.11}$$

These crude estimates can be refined. To go on, we need the following statement: Whenever M_n is an $n \times n$ tridiagonal matrix of the form (A.1), whose diagonal entries satisfy

$$0 < a_k^2 - 4b_kc_k, \tag{C.12}$$

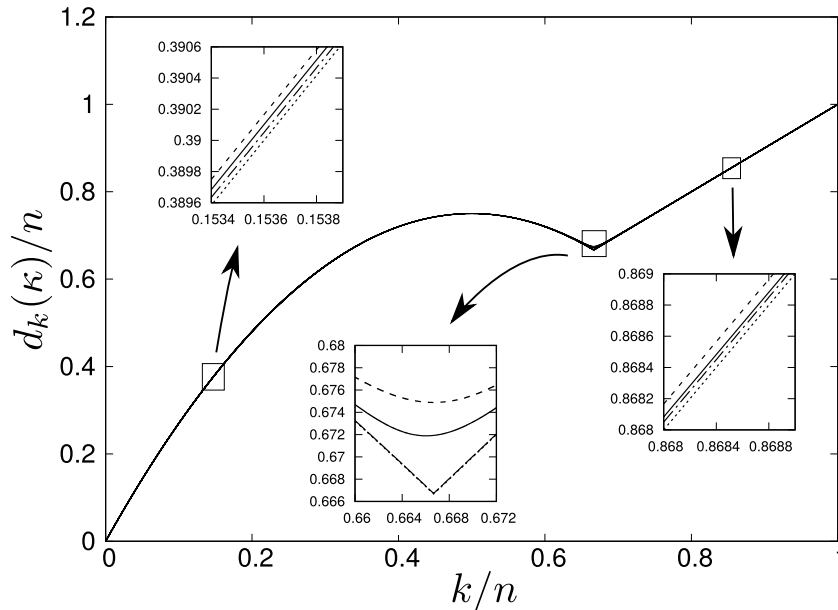


Figure C1. Depiction of the numerical solution of the Schur complements (solid); their upper bound according to (C.19) (dashed); their lower bound according to (C.42) (dotted); and the approximation of (C.70) (dotdash). The actual parameter values are set to $\delta = 1$, $\tau = 3$, $\kappa = (3\tau + 1)/2$ and $n = 10^5$.

then there exists a tridiagonal matrix F_n , whose Schur complements are given by the expression $(a_k + \sqrt{a_k^2 - 4b_k c_k})/2$ and the difference of M_n and F_n is a diagonal matrix [34]. Since the construction of F_n is used later on, we repeat the proof here. Assume that (C.12) holds and define the sequences

$$f_{\pm,k} = \frac{1}{2} \left(a_k \pm \sqrt{a_k^2 - 4b_k c_k} \right) \quad 1 \leq k \leq n. \tag{C.13}$$

The diagonal matrices \hat{f}_+ and \hat{f}_- formed of these sequences satisfy Vieta’s formulae:

$$\begin{aligned} \hat{f}_+ + \hat{f}_- &= \hat{a}, \\ \hat{f}_+ \hat{f}_- &= \hat{b}\hat{c}. \end{aligned} \tag{C.14}$$

Let F_n be defined by the UDL decomposition

$$F_n = (\mathbb{1} - S_n \hat{b} \hat{f}_+^{-1}) \hat{f}_+ (\mathbb{1} - \hat{c} \hat{f}_+^{-1} S_n^T), \tag{C.15}$$

then F_n is tridiagonal and its Schur complements are given by the sequence $f_{+,1}, \dots, f_{+,n}$. On the other hand, F_n and M_n are related to each other by

$$\begin{aligned} F_n &= \hat{f}_+ - S_n \hat{b} - \hat{c} S_n^T + S_n \hat{b} \hat{f}_+^{-1} \hat{c} S_n^T \\ &= \hat{a} - \hat{f}_- - S_n \hat{b} - \hat{c} S_n^T + S_n \hat{b} \hat{f}_+^{-1} \hat{c} S_n^T \\ &= M_n + S_n \hat{f}_- S_n^T - \hat{f}_-, \end{aligned} \tag{C.16}$$

where Vieta’s formulae have been used. By choosing $b_{n+1} = 0$ and an arbitrary c_{n+1} , the sequence $f_{-,1}, \dots, f_{-,n}$ can be extended by $f_{-,n+1} = 0$, in accordance with its definition. Introducing the diagonal matrix

$$d\hat{f}_- = S\hat{f}_-S^T - \hat{f}_- = \begin{pmatrix} f_{-,2} - f_{-,1} & & \\ & \ddots & \\ & & f_{-,n+1} - f_{-,n} \end{pmatrix}, \tag{C.17}$$

we arrive to

$$F_n = M_n + d\hat{f}_-, \tag{C.18}$$

which proves the statement.

The refined bounds can be obtained by choosing an appropriate tridiagonal matrix F_n of the form (C.15) whose Schur complements can be calculated explicitly, then using the monotonicity result will give bounds on the Schur complements of $\kappa\mathbb{1} - A_n$.

First, we state that the Schur complements of $\kappa\mathbb{1} - A_n$ have upper bounds of the form

$$d_k(\kappa) \leq \frac{1}{2} \left(\kappa + u_n + b_k + c_k + \sqrt{(\kappa + u_n + b_k + c_k)^2 - 4b_k c_k} \right), \tag{C.19}$$

where u_n is specified later. Choosing

$$a_k = \kappa + u_n + b_k + c_k, \tag{C.20}$$

one can calculate the sequences $f_{\pm,1}, \dots, f_{\pm,n}$ and the corresponding matrix F_n using (C.13) and (C.15). According to the previous statement, the Schur complements of F_n are given by

$$f_{k,+} = D_k(a_1 + df_{-,1}, \dots, a_n + df_{-,n}). \tag{C.21}$$

If u_n is such that for all $1 \leq k \leq n$ the inequality

$$\kappa + u_n + b_k + c_k + df_{-,k} > \kappa + b_{k+1} + c_k, \tag{C.22}$$

holds, then the monotonicity of the Schur complements guarantees that the inequalities

$$f_{k,+} \geq D_k(\kappa + b_2 + c_1, \dots, \kappa + b_{n+1} + c_n) = d_k(\kappa) \quad 1 \leq k \leq n, \tag{C.23}$$

where $d_k(\kappa)$ is the k th Schur complement of $\kappa\mathbb{1} - A_n$, are also satisfied, thereby (C.19) holds for all $1 \leq k \leq n$. Inequality (C.22) is true if and only if

$$u_n > \max_{1 \leq k \leq n} \{dg_k\}, \tag{C.24}$$

where the sequence g_1, \dots, g_n is given by

$$g_k = \frac{1}{2} \left(b_k - c_k + \sqrt{(\kappa + u_n + c_k + b_k)^2 - 4b_k c_k} \right). \tag{C.25}$$

Since all the g_k 's depend on u_n , inequality (C.24) is implicit in u_n . Fortunately, in the case of the SIS model, we can explicitly solve it in the $\delta \leq \tau$ parameter regime: For the sake of simplicity, set $\delta = 1$ and let

$$\begin{aligned} b_1 &= 0, \\ b_{k+1} &= \tau k \left(1 - \frac{k}{n} \right) \quad 1 \leq k \leq n, \end{aligned} \tag{C.26}$$

and

$$c_k = k. \tag{C.27}$$

If we introduce the functions

$$\begin{aligned} \mathbf{b}_n(x) &= \tau(x - n^{-1})(1 - x + n^{-1}), \\ \mathbf{c}(x) &= x, \\ \mathfrak{h}_n(\alpha, x) &= \sqrt{(\alpha + \mathbf{b}_n(x) + \mathbf{c}(x))^2 - 4\mathbf{b}_n(x)\mathbf{c}(x)}, \\ \mathfrak{g}_n(\alpha, x) &= \frac{1}{2} (\mathbf{b}_n(x) - \mathbf{c}(x) + \mathfrak{h}_n(\alpha, x)), \end{aligned} \tag{C.28}$$

then g_k is given by

$$g_k = n\mathfrak{g}_n(\alpha_n, k/n) \quad 1 \leq k \leq n, \tag{C.29}$$

where $\alpha_n = \kappa/n + u_n/n$. This enables us to approximate the differences dg_k by

$$dg_k = n \int_{k/n}^{(k+1)/n} \partial_x \mathfrak{g}_n(\alpha_n, x) dx < \max_{k/n \leq x \leq (k+1)/n} \{\partial_x \mathfrak{g}_n(\alpha_n, x)\}, \tag{C.30}$$

thus

$$\max_{1 \leq k \leq n} \{dg_k\} < \max_{1/n \leq x \leq 1} \{\partial_x \mathfrak{g}_n(\alpha_n, x)\}. \tag{C.31}$$

A short calculation gives

$$\partial_x (\mathbf{b}_n(x) - \mathbf{c}(x)) < \tau - 1 + \frac{2\tau}{n} \quad 1/n \leq x \leq 1, \tag{C.32}$$

while the calculation of an upper bound of

$$\partial_x \mathfrak{h}_n(\alpha_n, x) = \frac{\alpha_n \partial_x (\mathbf{b}_n(x) + \mathbf{c}(x))}{\mathfrak{h}_n(\alpha_n, x)} + \frac{\partial_x (\mathbf{b}_n(x) - \mathbf{c}(x))^2}{2\mathfrak{h}_n(\alpha_n, x)} \tag{C.33}$$

is a little bit more involved. Since α_n can be safely assumed to be positive, the inequality

$$\mathfrak{h}_n^2(\alpha_n, x) = \alpha_n^2 + 2\alpha_n(\mathbf{b}_n(x) + \mathbf{c}(x)) + (\mathbf{b}_n(x) - \mathbf{c}(x))^2 > \alpha_n^2 \tag{C.34}$$

holds, and the first term of the rhs of equation (C.33) is bounded by

$$\frac{\alpha_n \partial_x (\mathbf{b}_n(x) + \mathbf{c}(x))}{\mathfrak{h}_n(\alpha_n, x)} < \max_{1/n \leq x \leq 1} \{\partial_x (\mathbf{b}_n(x) + \mathbf{c}(x))\} < \tau + 1 + \frac{2\tau}{n} \tag{C.35}$$

for all $1/n \leq x \leq 1$. Fix a given x between $1/n$ and one! If $\mathbf{b}_n(x) = \mathbf{c}(x)$ holds, then the second term in (C.33) vanishes, thus zero is an upper bound on it. If $\mathbf{b}_n(x)$ is not equal to $\mathbf{c}(x)$, then there are two possibilities. If the second term of (C.33) is negative, it is bounded again by zero from above. If it is positive, then it is a monotone decreasing function of the positive α_n and in that case

$$\begin{aligned} \frac{\partial_x (\mathbf{b}_n(x) - \mathbf{c}(x))^2}{2\mathfrak{h}_n(\alpha_n, x)} &< \frac{\partial_x (\mathbf{b}_n(x) - \mathbf{c}(x))^2}{2\mathfrak{h}_n(0, x)} = \partial_x |\mathbf{b}_n(x) - \mathbf{c}(x)| \\ &< \tau + 1 - \frac{2\tau}{n}, \end{aligned} \tag{C.36}$$

for all $1/n \leq x \leq 1$. Finally, we can write

$$\partial_x \mathfrak{h}_n(\alpha_n, x) < 2\tau + 2, \tag{C.37}$$

which, together with (C.32) gives

$$\max_{1/n \leq x \leq 1} \{\partial_x \mathfrak{g}_n(\alpha_n, x)\} < \frac{3\tau + 1}{2} \tag{C.38}$$

for all $1 \leq n$. Thus, the constant sequence

$$u_n = \frac{3\tau + 1}{2} \quad 1 \leq n \tag{C.39}$$

solves inequality (C.24), thereby inequality (C.19) stands for all $1 \leq k \leq n$.

In the same vein as before, we can give a lower bound of the Schur complements of $\kappa \mathbb{1} - A_n$, but now the possible values of κ , where the lower bound is applicable is restricted. Assume that there exists l_n such that $0 < \kappa + l_n$ and l_n satisfies the implicit inequality

$$l_n < \min_{1 \leq k \leq n} \{dg_k\}, \tag{C.40}$$

where g_k is now defined by

$$g_k = \frac{1}{2} \left(b_k - c_k + \sqrt{(\kappa + l_n + c_k + b_k)^2 - 4b_k c_k} \right). \tag{C.41}$$

Then, the Schur complements of $\kappa \mathbb{1} - A_n$ are bounded from below by

$$d_k(\kappa) \geq \frac{1}{2} \left(\kappa + l_n + b_k + c_k + \sqrt{(\kappa + l_n + b_k + c_k)^2 - 4b_k c_k} \right). \tag{C.42}$$

To prove the statement, note that $0 < \kappa + l_n$ holds, so members of the sequence a_1, \dots, a_n , defined by

$$a_k = \kappa + l_n + b_k + c_k \tag{C.43}$$

satisfy inequality (C.12). Thus, the sequences

$$f_{\pm, k} = \frac{1}{2} \left(a_k \pm \sqrt{a_k^2 - 4b_k c_k} \right) \quad 1 \leq k \leq n \tag{C.44}$$

have non-negative members and the sequence $f_{+,1}, \dots, f_{+,n}$ gives the Schur complements of the matrix

$$F_n = (\mathbb{1} - S_n \hat{b} f_+^{-1}) \hat{f}_+ (\mathbb{1} - \hat{c} f_+^{-1} S_n^T). \tag{C.45}$$

Thus, the Schur complements $f_{+,1}, \dots, f_{+,n}$ satisfy the equalities

$$f_{k,+} = D_k(a_1 + df_{-,1}, \dots, a_n + df_{-,n}), \tag{C.46}$$

and whenever l_n satisfy the inequalities

$$\kappa + l_n + b_k + c_k + df_{-,k} < \kappa + b_{k+1} + c_k \tag{C.47}$$

for all $1 \leq k \leq n$, the monotonicity of the Schur complements guarantees that the inequalities

$$f_{k,+} < D_k(\kappa + b_2 + c_1, \dots, \kappa + b_{n+1} + c_n) = d_k(\kappa) \quad 1 \leq k \leq n, \quad (\text{C.48})$$

where $d_k(\kappa)$ is the k th Schur complement of $\kappa\mathbb{1} - A_n$, are also satisfied. Since the inequalities in (C.47) are equivalent to (C.40) (if the corresponding definition of the sequence g_1, \dots, g_n is given by (C.41)), the statement is proved. In case of the SIS model, inequality (C.40) can be solved explicitly. Let $\mathbf{b}_n(x)$, $\mathbf{c}(x)$, $\mathfrak{h}_n(x)$ and $\mathfrak{g}_n(\alpha, x)$ defined as in (C.28)! Then the minimum of the finite differences dg_k is bounded from below by

$$\min_{1 \leq k \leq n} \{dg_k\} > \min_{1/n \leq x \leq 1} \{\partial_x \mathfrak{g}_n(\beta_n, x)\}, \quad (\text{C.49})$$

where $\beta_n = \kappa/n + l_n/n$ is assumed to be positive. Again the derivative of $\mathfrak{g}_n(\beta_n, x)$ can be bounded from below by giving lower bounds on the derivatives of $\mathbf{b}_n(x) - \mathbf{c}(x)$ and $\mathfrak{h}_n(\beta_n, x)$. The first is easy to calculate:

$$\partial_x(\mathbf{b}_n(x) - \mathbf{c}(x)) > -\tau - 1. \quad (\text{C.50})$$

Provided that β_n is positive, lower bounds of the two terms that consists of $\partial_x \mathfrak{h}_n(\beta_n, x)$ are given by

$$\frac{\beta_n \partial_x(\mathbf{b}_n(x) + \mathbf{c}(x))}{\mathfrak{h}_n(\beta_n, x)} > \min_{1/n \leq x \leq 1} \{\partial_x(\mathbf{b}_n(x) + \mathbf{c}(x))\} > 1 - \tau, \quad (\text{C.51})$$

and

$$\begin{aligned} \frac{\partial_x(\mathbf{b}_n(x) - \mathbf{c}(x))}{2\mathfrak{h}_n(\beta_n, x)} &> \min_{1/n \leq x \leq 1} \{\partial_x |\mathbf{b}_n(x) - \mathbf{c}(x)| : \mathbf{b}_n(x) \neq \mathbf{c}(x)\} \\ &> -1 - \tau. \end{aligned} \quad (\text{C.52})$$

Finally,

$$\mathfrak{g}_n(\beta_n, x) > -\frac{3\tau + 1}{2}, \quad (\text{C.53})$$

that is the constant sequence

$$l_n = -\frac{3\tau + 1}{2} \quad (\text{C.54})$$

satisfy inequality (C.40) and if the inequality

$$\frac{3\tau + 1}{2} < \kappa \quad (\text{C.55})$$

also holds, the Schur complements of $\kappa\mathbb{1} - A_n$ are bounded from below according to (C.42).

Due to the appearance of the square root, the applicability of the bounds (C.19) and (C.42) is complicated. However, when k is large, using an arbitrary positive constant γ , the approximation

$$b_k + c_k + \sqrt{(\gamma + b_k + c_k)^2 - 4b_k c_k} \approx 2 \max\{b_k, c_k\} \quad (\text{C.56})$$

sounds reasonable. To quantify it, we introduce the difference

$$0 < \Delta_k(\gamma) = \frac{1}{2} \left(c_k + b_k + \sqrt{(\gamma + c_k + b_k)^2 - 4b_k c_k} \right) - \max\{b_k, c_k\}. \quad (\text{C.57})$$

Introducing the auxiliary sequences

$$\sigma_k = |b_k - c_k|, \tag{C.58}$$

$$\Sigma_k = b_k + c_k, \tag{C.59}$$

a short calculation gives

$$\Delta_k(\gamma) = \frac{\gamma}{2} \frac{\gamma + 2\Sigma_k}{\sigma_k + \sqrt{\gamma^2 + 2\gamma\Sigma_k + \sigma_k^2}}. \tag{C.60}$$

Replacing σ_k with Σ_k under the square root gives a lower bound

$$\frac{\gamma}{2} < \frac{\gamma}{2} \frac{\gamma + 2\Sigma_k}{\gamma + 2 \max\{b_k, c_k\}} < \Delta_k(\gamma), \tag{C.61}$$

which can be used to give a lower bound of $d_k(\kappa)$: Setting $\gamma = \kappa - (3\tau + 1)/2$ and assuming $0 < \gamma$, we obtain

$$\begin{aligned} d_k(\kappa) &> \frac{\gamma}{2} + \Delta(\gamma) + \max\{b_k, c_k\} \\ &> \gamma + \max\{b_k, c_k\} \\ &> \kappa - \frac{3\tau + 1}{2} + \max\{b_k, c_k\}. \end{aligned} \tag{C.62}$$

The replacement of σ_k with zero in the denominator of equation (C.60) results in an upper bound:

$$\Delta_k(\gamma) < \frac{\sqrt{\gamma}}{2} \sqrt{\gamma + 2\Sigma_k}. \tag{C.63}$$

Considering the SIS model, whenever $1 < \tau$ holds (the infection rate is set to be equal to one), there is at least one specific value of $1 \leq k \leq n$, such that the corresponding σ_k approaches zero, that is b_k and c_k are close to each other. That is, inequality (C.63) is the sharpest one which holds for all $1 \leq k \leq n$. Nonetheless, when σ_k is far away from zero, we can give another upper bound:

$$\begin{aligned} 2\Delta_k(\gamma) &= \int_0^\gamma \frac{\mu + \Sigma_k}{\sqrt{\mu^2 + 2\mu\Sigma_k + \sigma_k^2}} d\mu \\ &< \int_0^\gamma \frac{\mu}{\sqrt{\mu^2 + \sigma_k^2}} d\mu + \int_0^\gamma \frac{\Sigma_k}{\sigma_k} d\mu \\ &< \left[\sqrt{\mu^2 + \sigma_k^2} \right]_0^\gamma + \frac{\Sigma_k}{\sigma_k} \gamma \\ &< \gamma \left(1 + \frac{\Sigma_k}{\sigma_k} \right), \end{aligned} \tag{C.64}$$

which results in

$$\Delta_k(\gamma) < \gamma \frac{\max\{\mathbf{b}_n(x_k), \mathbf{c}(x_k)\}}{|\mathbf{b}_n(x_k) - \mathbf{c}(x_k)|}, \tag{C.65}$$

where $x_k = k/n$. One may wonder whether the ratio in the rhs of this inequality is small enough to recognize (C.65) as a useful upper bound. To answer this question, let k_n^- and k_n^+ be the smallest and the greatest of the (not necessarily integer) solutions of the inequality

$$k \leq \tau(k-1) \left(1 - \frac{k-1}{n}\right) \quad 1 \leq k \leq n. \tag{C.66}$$

A short calculation shows

$$k_n^\pm = 1 + \frac{n}{2}x_\tau \pm \frac{n}{2}\sqrt{x_\tau^2 - \frac{4}{\tau n}}, \tag{C.67}$$

where $x_\tau = 1 - \tau^{-1}$. For any $0 \leq c \leq 1/2$ and $0 \leq z \leq 1$, the inequality $1 - z < \sqrt{1 - z} < 1 - cz$ holds, thus

$$1 + \frac{2c}{(\tau - 1)} < k_n^- < 1 + \frac{2}{(\tau - 1)}, \tag{C.68}$$

and

$$nx_\tau - \frac{2}{\tau - 1} < k_n^+ < nx_\tau - \frac{2c}{\tau - 1}. \tag{C.69}$$

That is, whenever $2 < \tau$ holds, the integer solutions of the inequality $c_k < b_k$ are between 2 and $nx_\tau - 1$. In that case, choosing any k , which is sufficiently far away from 2 and nx_τ , the rhs of (C.65) remains sufficiently small. Furthermore, if κ is larger than $(3\tau + 1)/2$, the upper and lower bounds given by (C.19) and (C.42) combined with the bounds (C.61) and (C.65) give rise to quantify the approximation

$$d_k(\kappa) \approx \kappa + \max\{b_k, c_k\}. \tag{C.70}$$

Introducing the function

$$\epsilon(x) = \begin{cases} 1 + 1/|x - x_\tau| & \text{if } x < x_\tau \\ 1/|x - x_\tau| & \text{if } x > x_\tau, \end{cases} \tag{C.71}$$

which is a reasonable approximant of $\max\{b_n(x), c(x_k)\}/|b_n(x_k) - c(x_k)|$ in the large n limit, we get

$$|\kappa + \max\{b_k, c_k\} - d_k(\kappa)| \lesssim \frac{3\tau + 1 - 2\kappa}{4} + \epsilon(x)(2\kappa + 3\tau + 1), \tag{C.72}$$

if the rhs exceeds $(3\tau + 1)/2$ and

$$|\kappa + \max\{b_k, c_k\} - d_k(\kappa)| \lesssim \frac{3\tau + 1}{2}, \tag{C.73}$$

otherwise. If k is sufficiently far away from 2 and nx_τ , the rhs of these equations are rather small, when compared to the actual values of $d_k(\kappa)$: While the latter is of the order $o(k)$, the the former remains of order $o(1)$, see figure C1 for a numerical example.

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