Explosive phase transition in susceptible-infected-susceptible epidemics with arbitrary small but nonzero self-infection rate

Piet Van Mieghem [*]

Delft University of Technology, Faculty of Electrical Engineering, Mathematics and Computer Science,
P.O. Box 5031, 2600 GA Delft, The Netherlands

The ε–susceptible-infected-susceptible (SIS) epidemic model on a graph adds an independent, Poisson self-infection process with rate ε to the "classical" Markovian SIS process. The steady state in the classical SIS process (with $\varepsilon = 0$) on any finite graph is the absorbing or overall-healthy state, in which the virus is eradicated from the network. We report that there always exists a phase transition around $\tau_c^\varepsilon = O(\varepsilon^{-\frac{1}{N-1}})$ in the ε-SIS process on the complete graph K_N with N nodes, above which the effective infection rate $\tau > \tau_c^\varepsilon$ causes the average steady-state fraction of infected nodes to approach that of the mean-field approximation, no matter how small, but not zero, the self-infection rate ε is. For $\tau < \tau_c^\varepsilon$ and small ε, the network is almost overall healthy. The observation was found by mathematical analysis on the complete graph K_N, but we claim that the phase transition of explosive type may also occur in any other finite graph. We thus conclude that the overall-healthy state of the classical Markovian SIS model is unstable in the ε-SIS process and, hence, unlikely to exist in reality, where "background" infection $\varepsilon > 0$ is imminent.

I. INTRODUCTION

The network science [1] definition of a network rests upon the duality between the network's *structure*, called the graph or topology, and the network's *function*, also called the process or service that runs over the graph. One may also simplistically regard a network as consisting of "hardware" (the structure) and "software" (the function). Grip on complexity in many processes today starts by understanding the interplay between structure and function. For most functions in complex networks, the governing equations are beyond reach; just think, for example, about the processes in the brain, biological and chemical interactions, even man-made networks such as the Internet, steered by transfer control protocol (TCP), and the stock market. The principle of science, top-down analysis ($\alpha\nu\alpha\lambda\upsilon\epsilon\iota\nu$: release completely), and bottom-up synthesis ($\sigma\upsilon\nu\tau\iota\theta\epsilon\nu\alpha\iota$: place together) suggests us to embrace the simple models that we understand. Undoubtedly, one of the simplest functions on a graph is diffusion, in particular, the spread of items described as an infection [2,3]. Recently, for a broad range of dynamics, Hens *et al.* [4] disentangled the function from the network structure by regarding propagation characteristics. Of all epidemic models, the simplest one is, perhaps, the Markovian susceptible-infected-susceptible (SIS) process that can be exactly described for any network [5], but only solved for small graphs, roughly up to 15 nodes. Among all those graphs, the analysis can be pushed further only in surprisingly few graphs, such as the star and the complete graph [6], but the time dynamics in even those graphs is analytically beyond reach [7]. The present paper confines the

function to SIS epidemics and the structure to the complete graph. We first describe the function and then report the phenomenon, which can occur in any graph.

In a graph G with N nodes, the viral state of a node i at time t is specified by a Bernoulli random variable $X_i(t) \in \{0, 1\}$: $X_i(t) = 0$ for a healthy node and $X_i(t) = 1$ for an infected node. A node i at time t can be in one of the two states: *infected*, with probability $v_i(t) = \Pr[X_i(t) = 1]$, or *healthy*, with probability $1 - v_i(t)$, but susceptible to the virus. The curing process per node i is a Poisson process with rate δ and the infection rate per link is a Poisson process with rate β. Besides infections over links from infected neighbors with rate β, the node i can also infect itself by a Poisson process with self-infection rate ε. Only when a node is infected, it can infect its direct, healthy neighbors. All Poisson processes are independent. This is the continuous-time description[1] of the self-infectious Markovian susceptible-infected-susceptible (ε-SIS) process on a contact graph. The ε-SIS model reduces to the "classical" SIS model when the self-infection rate is $\varepsilon = 0$.

The ε-SIS epidemic process can model information spread in social networks, where individuals themselves can generate information, which is spread over links to neighbors. Hill *et al.* [8] modeled happiness of persons by an ε-SIS infection over a social contact network. In a similar vein, obesity

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*p.f.a.vanmieghem@tudelft.nl

[1]In the ε-SIS heterogenous setting, the curing rate δ_i and self-infection rate ε_i are coupled to a node i and the infection rate β_{ij} specifies the link from node i to node j. In any continuous-time Markovian process, the interevent times are exponentially distributed [24, p. 210]. Thus, a self-infection rate ε means that, on average $1/\varepsilon$ time units, a self-infection event occurs.

is known to have a self-infected component and its spread has been described [9,10] by an ε-SIS type of infection on social networks. The self-infection nodal process can also be considered as a "drift field" [11] that drives the infection in each node with the same strength. From a biological point of view, when the virus dies out, the population will start losing immunity against that virus, i.e., the curing rate δ will decrease with time. A sudden reappearance of the virus may wipe out the whole population, because the curing rate δ has become very low. Hence, the existence of very few infected nodes on average keeps the population fit against the virus, meaning that the curing rate δ remains more or less constant because their immunity system is constantly challenged. The infectious environment may be modeled by a self-infection process, usually with a small infection rate ε as a "background" or "imminent" infection, in addition to the disease or viral spread with infection rate β under study. Related studies on contagion in networks consider a slightly more complex local rule than in ε-SIS, with either memory of infection doses [12] or a spontaneous self-infection dependent on a fixed number of infected neighbors [13].

Earlier, the ε-SIS model was introduced in [14], mainly motivated to compute a realistic steady state of the SIS epidemic on any finite graph [15], which is different from the uninformative absorbing state in the classical SIS process. The existence of an absorbing state in the classical SIS process causes a significant complication [11,6], leading to a metastable or quasistationary regime and to an unrealistically long absorption time [16]. In the ε-SIS model with $\varepsilon > 0$, there is no absorbing state and the Markov process is irreducible in a connected contact graph, which implies that there is a unique steady state, which is, for specially chosen self-infection rate ε as explained in [15], very close to observations and to the SIS mean-field steady state.

II. EXPLOSIVE PHASE TRANSITION

Here, we report curious steady-state behavior of the ε-SIS process with arbitrarily small self-infection rate $\varepsilon > 0$, that does not exist in the classical $\varepsilon = 0$ SIS model. The observation of a remarkable phase transition, increasingly
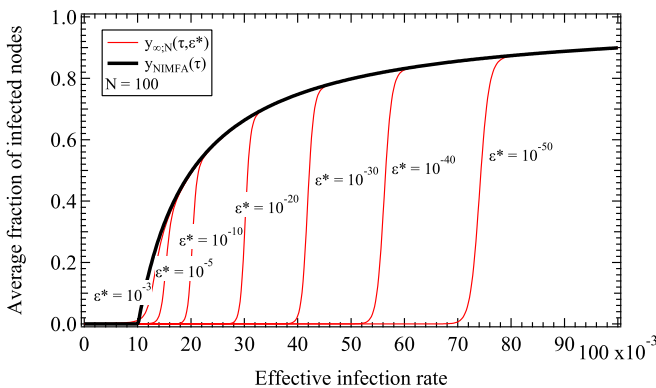
FIG. 1. The steady-state average fraction $y_{\infty;N}(\tau, \varepsilon^*)$ of infected nodes vs the effective infection rate τ in the complete graph K_N with $N = 100$ nodes for various normalized self-infection rates $\varepsilon^* = \{10^{-3}, 10^{-5}, 10^{-10}, 10^{-20}, 10^{-30}, 10^{-40}, 10^{-50}\}$.
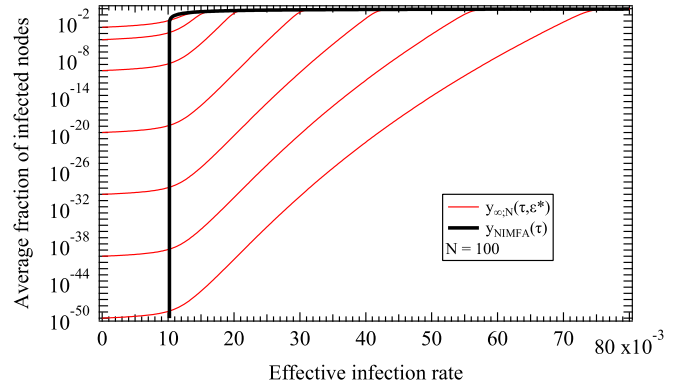
FIG. 2. The steady-state average fraction $y_{\infty;N}(\tau, \varepsilon^*)$ of infected nodes on log scale vs the effective infection rate τ in the complete graph K_N with $N = 100$ nodes for the same normalized self-infection rates $\varepsilon^* = \{10^{-3}, 10^{-5}, 10^{-10}, 10^{-20}, 10^{-30}, 10^{-40}, 10^{-50}\}$ as in Fig. 1. Each curve for ε^* is distinguishable, because $y_{\infty;N}(0, \varepsilon^*) = \frac{\varepsilon^*}{1+\varepsilon^*}$.

explosive with the size N of the graph, was only possible by a purely analytical study presented in Appendix, for which we have limited ourselves to the complete graph K_N. In particular, we show that there always exists a phase transition around τ_c^ε in the ε-SIS process on K_N, above which the effective infection rate $\tau = \frac{\beta}{\delta} > \tau_c^\varepsilon$ causes the average steady-state fraction $y_{\infty;N}(\tau, \varepsilon^*)$ of infected nodes, briefly called the prevalence, to approach the N-intertwined mean-field (NIMFA) prevalence [17]

$$y_{\infty;N}^{(1)}(\tau) = 1 - \frac{1}{(N-1)\tau} \quad \tau > \frac{1}{N-1} = \tau_c^{(1)} \qquad (1)$$

no matter how small, but not zero, the self-infection rate $\varepsilon^* = \frac{\varepsilon}{\delta}$ is. The phase transition τ_c^ε is a zero of an Nth order polynomial in τ (Theorem 4 in Appendix A 4), but can be bounded by

$$\frac{1}{e}\left(\frac{10^{-s}}{\varepsilon^*(N-1)!}\right)^{\frac{1}{N-1}} < \tau_c^\varepsilon < \left(\frac{10^{-s}}{\varepsilon^*(N-1)!}\right)^{\frac{1}{N-1}}$$

where s specifies an agreed level for the onset of the phase transition at which $y_{\infty;N}(\tau, \varepsilon^*) = 10^{-s}$ is first reached, when τ is gradually increased from $\tau = 0$ at $y_{\infty;N}(0, \varepsilon^*) = \frac{\varepsilon^*}{1+\varepsilon^*}$ on. Figure 1 illustrates the steady-state prevalence in a complete graph[2] with $N = 100$ nodes. Figure 1 indicates that the classical $\varepsilon = 0$ SIS process is unlikely to model reality of both biological and digital viral items, where "infectious noise" with $\varepsilon > 0$ exists. In addition, the phase transition can hardly be simulated for extremely small self-infection rate ε^*. Although the observation of a rather explosive phase transition is derived from the complete graph K_N, we believe that it may occur in any graph.[3]

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[2]A similar plot for $N = 500$ appeared earlier in [16, Fig. 6] as a curiosity.

[3]Analytic intractability prevents us from demonstrating the claim. Perhaps the star graph, whose analysis [6] is already considerably more complex than for the complete graph, might be beyond reach.
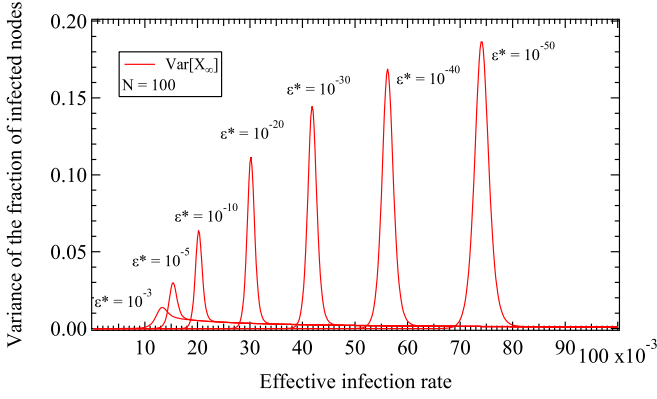
FIG. 3. The steady-state variance $\mathrm{Var}[X_\infty]$ of the fraction X_∞ of infected nodes vs the effective infection rate τ in the complete graph K_N with $N = 100$ nodes for the same normalized self-infection rates as in Fig. 1.

The shape of the red curves in Fig. 1 for relatively small normalized self-infection rates $\varepsilon^* = 10^{-q}$ with $2 < q < 5$ seems to correspond well with simulations of the classical SIS process *on any graph* and may hint at the analytic behavior of the steady-state prevalence around the epidemic threshold, which is linear in $(\frac{1}{\tau_c^{(1)}} - \frac{1}{\tau})$ in NIMFA [18], but unknown in general for the classical SIS process. For large power law graphs, Mountford *et al.* [19, Theorem 1.1] have specified the exponent of the metastable prevalence in terms of the power law degree exponent. Figure 2 illustrates that, above the NIMFA epidemic threshold $\tau_c^{(1)} = \frac{1}{N-1}$, the steady-state prevalence $y_{\infty;N}(\tau, \varepsilon^*)$ increases almost exponentially with the effective infection rate τ, from about $y_{\infty;N}(0, \varepsilon^*) = \frac{\varepsilon^*}{1+\varepsilon^*}$ up to the NIMFA steady-state in (1).

The variance of the fraction of infected nodes in K_N for $N = 100$, computed via (A12) and corresponding to the same graph and ε-SIS parameters as in Fig. 1, is presented in Fig. 3. We can choose the parameter s in (A18) so that τ_c^ε coincides with the maximum of the variance. By scaling $\tau = x\tau_c^\varepsilon$ in the variance (A12), all peaks align at $x = 1$ for sufficiently large $N > 50$ and small ε.

The scaling of the steady-state prevalence $y_{\infty;N}(\frac{x}{N-1}, \varepsilon^*)$ with N is drawn in Fig. 4 versus normalized effective infection rate $x = \frac{\tau}{\tau_c^{(1)}} = (N-1)\tau$. For this normalization, the NIMFA steady-state prevalence becomes $y_{\infty;N}^{(1)}(\frac{x}{N-1}) = 1 - \frac{1}{x}$, which is independent of N. Figure 4 illustrates that the phase transition in the steady-state prevalence $y_{\infty;N}(\frac{x}{N-1}, \varepsilon^*)$ from about zero towards $1 - \frac{1}{x}$ becomes increasingly steep and seems to resemble[4] a "product rule" type phase transition [20, Figs. 1, 12, and 16]. Figure 5 shows the scaling of the steady-state prevalence $y_{\infty;N}(\frac{x}{N-1}, \frac{e^{-zN}}{\sqrt{2\pi N}})$ as derived in Appendix A5, versus the normalized effective infection rate $x = (N-1)\tau$ for the same values of N as in Fig. 4. Figure 5 illustrates that $y_{\infty;N}(\frac{x}{N-1}, \frac{e^{-zN}}{\sqrt{2\pi N}})$ tends to a universal curve in z for large
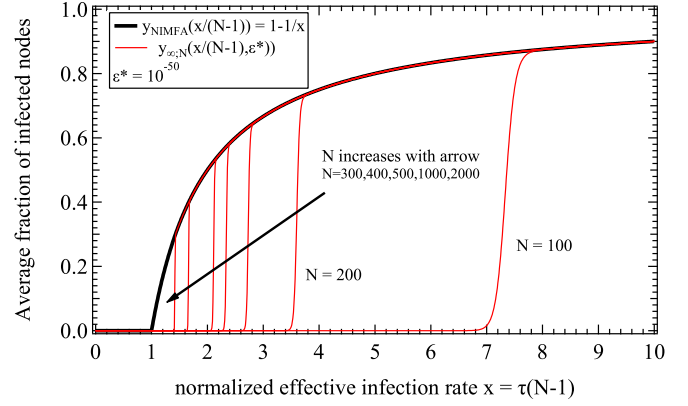

FIG. 4. The steady-state prevalence $y_{\infty;N}(\frac{x}{N-1}, \varepsilon^*)$ and NIMFA $y_{\infty;N}^{(1)}(\tau) = 1 - \frac{1}{x}$ vs normalized effective infection rate $x = \tau(N-1) = \frac{\tau}{\tau_c^{(1)}}$ for various $N \in \{100, 200, 300, 400, 500, 1000, 2000\}$ and normalized self-infection rate $\varepsilon^* = 10^{-50}$.

N, close to $(1 - \frac{1}{x})\theta[x - x_c(z)]$, where $\theta(u)$ is Heavyside's step function with a jump at $x_c(z) = (N-1)\tau_c^{\varepsilon(z)}$. The scaled self-infection rate $\varepsilon^*(z) = \frac{e^{-zN}}{\sqrt{2\pi N}}$ depends exponentially on the size N of the complete graph. Clear phase transitions at $x_c(z)$ occur slightly above $x = e^{1+z}$, as derived in Appendix A5, and the approximate exponential law for the phase transition point $\tau_c^{\varepsilon(z)} \simeq \frac{e^{1+z}}{N-1}$ for $y_{\infty;N}(\frac{x}{N-1}, \frac{e^{-zN}}{\sqrt{2\pi N}})$ has been verified for various other values of z ranging from $z = 0.1$ to 2 (not shown in Fig. 5).

The observations in Figs. 1–5 suggest that the classical SIS steady state is instable in the following sense: by adding arbitrarily small "infection noise ε," there always exists a critical effective infection strength τ_c^ε depending on the self-infection rate ε above which the absorbing state is destroyed ($\tau > \tau_c^\varepsilon$) and switched to the mean-field steady state. Below the threshold τ_c^ε, on the other hand, the probability of infection is about $\frac{\varepsilon}{1+\varepsilon}$ and, thus, small for small $\varepsilon > 0$, but not entirely zero. The phenomenon can be regarded as a stochastic instability.
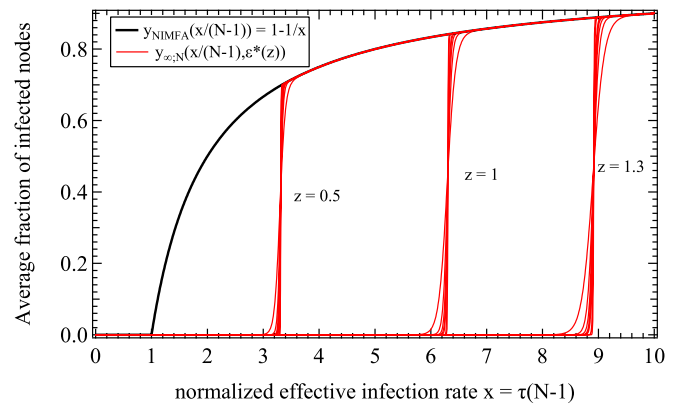

FIG. 5. The steady-state prevalence $y_{\infty;N}(\frac{x}{N-1}, \varepsilon^*(z))$ with $\varepsilon^*(z) = \frac{e^{-zN}}{\sqrt{2\pi N}}$ and NIMFA $y_{\infty;N}^{(1)}(\tau) = 1 - \frac{1}{x}$ vs normalized effective infection rate $x = \tau(N-1) = \frac{\tau}{\tau_c^{(1)}}$ for various $N \in \{100, 200, 300, 400, 500, 1000, 2000\}$ as in Fig. 4 for $z = 0.5, 1, 1.3$.

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[4] Explosive phase transitions can be continuous as well as discontinuous [20]. Since $y_{\infty;N}(\tau, \varepsilon^*)$ is differentiable in τ and $\varepsilon^* > 0$ for finite N, but $\lim_{N\to\infty} y_{\infty;N}(\tau, \varepsilon^*)$ is a step function, Fig. 12 in [20] points to a type II explosive, phase transition.

In the theory of equilibrium processes (see, e.g., the review of Schnakenberg [21]) two processes balance each other on average, which implies for the ε-SIS process that the average number of cured and healthy nodes in the graph remains about constant over time. The individual nodes of the graph, however, continue alternating to the infectious state, and the process fluctuations around the mean, here the prevalence [22, Fig. 7], tend to a Gaussian as proved by Feller [23] and specified for Markov processes in [24, Theorem 9.3.7 on p.196].

In the classical SIS process, the curing process has a slight advantage over the infection process, because the curing process has the power to destroy the infection process, whereas the reverse is not possible. Indeed, let m be the number of infected nodes at some time in the metastable state ($\tau > \tau_c$). If the curing process generates a sequence of m healings, each of them so rapidly that the infection process has no occasion to create a single new infectious event, then the infection is eradicated and the actual steady state, the absorbing state, of the classical SIS process is reached and lasts forever. Eventually and after an average time $E[T] = O(e^{N \ln \frac{\tau}{\tau_c}})$—the precise average time $E[T]$ to absorption for the complete graph for $\tau > \tau_c$ is given in [16]—the epidemic disappears in the classical SIS Markovian process, due to a rare event of m consecutive healings before an infection is generated. The situation is comparable to winning in a casino m successive games, which is possible, but its probability sharply decreases with m. Alternatively, one needs to play for an unrealistically long time to win. For the classical SIS Markovian process, such a rare event is extremely hard to simulate in graphs of realistic size N, and impossible if simulation acceleration is not available like in our ε-SIS process here.

In the ε-SIS process with self-infection rate $\varepsilon > 0$, the inequality between curing and infection is removed and neither of the processes can destroy the other. Even if a rare succession of m healings occurs and the infection is momentarily removed from the graph, the self-infection process will again create an infection, which means that the zero infection state is not absorbing (in the sense that the process cannot leave that state anymore). We would then expect that, in the steady state, the balance between the number of infected and cured nodes is about constant forever. However, we found that the steady state appears in two "flavors." For any arbitrary small, but not zero, self-infection rate $\varepsilon > 0$, the steady state lies in the realm of the classical SIS process, without virtually an infection in the graph when the effective infection rate $\tau < \tau_c^\varepsilon$. On the other hand, when $\tau > \tau_c^\varepsilon$, the steady-state fraction of infected nodes jumps to a significantly higher ratio, roughly equal to that predicted by a mean-field analysis. It appears that, when $\tau > \tau_c^\varepsilon$, a reversed rare event of successive infections occurs starting from an almost healthy graph up to an equilibrium between the number of infected and healthy nodes in a fair competition between infection and curing process, well described by mean-field theory. For a given strength ε of the self-infection, the effective link infection strength τ below τ_c^ε has insufficient power to cause a spread. For $\tau > \tau_c^\varepsilon$ and $\tau_c^\varepsilon > \tau_c^{(1)}$ above the classical SIS mean-field epidemic threshold $\tau_c^{(1)}$, the infection violently spreads over the entire network, irrespective of how improbable, but not impossible, that infection is; thus, the mere existence

of an infection ($\varepsilon > 0$) causes the endemic state above a certain link propagation strength $\tau > \tau_c^\varepsilon > \tau_c^{(1)}$. Alternatively, the stationary or equilibrium regime favors the least energy or lowest potential in a physical system, that can generally be represented by a convex Lyapunov function, such as the Kullback-Leibner divergence for a Markov process [21]. Rare fluctuations far from equilibrium are pulled forcefully back to the stationary or equilibrium state. We argue that the almost exponential growth in Fig. 2 is a fingerprint of a multiplicative process with many infection events in a row, that create on the linear scale in Fig. 1 the jump around τ_c^ε. Such a multiplicative process agrees with the product rule type phase transition [20, Figs. 1, 12, and 16] mentioned above.

As illustrated in Fig. 5 with exponentially small self-infection rate $\varepsilon^*(z) = \frac{e^{-zN}}{\sqrt{2\pi N}}$, the "exponential sensitivity" of the steady state in the simple ε-SIS model came as a surprise. Zooming out from the ε-SIS toy model to real dynamics (viz., the climate), whose processes and their interactions are incomparably more complex, the observations in Figs. 1–5 caution against the unexpected consequences of small perturbations of steady-state behavior. Evolution in biology is believed to happen by certain, rare mutations that result in a superior species, that relatively rapidly replaces the older one by the principle of the survival of the fittest. Such rare mutations are generated at low rate, comparable here to the self-infection rate ε, but just as with the explosive phase transition in ε-SIS they create a phase of new species.

APPENDIX: ε-SIS EPIDEMICS ON THE COMPLETE GRAPH K_N

We confine ourselves to the complete graph K_N because exact computations are possible. The time T to reach the absorbing state in an SIS epidemic is an exponential random variable with mean $E[T]$, that is precisely computed in [16] for the complete graph. The analysis of the steady-state fraction of infected nodes in the ε-SIS epidemic process on K_N is presented here, after reviewing earlier work in [14, Appendix], [7], and [24, Sec. 17.3].

1. Review of our previous analyses

The number of infected nodes $M(t)$ in an ε-SIS epidemic at time t in the complete graph K_N is a continuous-time Markov process on $\{0, 1, \ldots, N\}$ states with the following rates:

$$M \mapsto M + 1 \text{ at rate } (\beta M + \varepsilon)(N - M),$$
$$M \mapsto M - 1 \text{ at rate } \delta M.$$

Every infected node heals with rate δ, whereas every healthy node (of which there are $N - M$) has exactly M infected neighbors each actively transferring the virus with rate β, in addition to the self-infection rate ε. This Markov process $M(t)$ is, in fact, a birth and death process with birth rate $\lambda_j = (\beta j + \varepsilon)(N - j)$ and death rate $\mu_j = j\delta$, whose steady-state probabilities $\pi_0, \ldots, \pi_N$, where

$\pi_j = \lim_{t\to\infty} \Pr[M(t) = j]$, can be computed exactly [24, p. 209] as

$$\pi_0 = \frac{1}{1 + \sum_{k=1}^{N} \prod_{m=0}^{k-1} \frac{(\beta m + \varepsilon)(N-m)}{(m+1)\delta}}, \tag{A1}$$

$$\pi_j = \pi_0 \prod_{m=0}^{j-1} \frac{(\beta m + \varepsilon)(N-m)}{(m+1)\delta}, \quad 1 \leqslant j \leqslant N. \tag{A2}$$

Simplified, in terms of the normalized self-infection rate $\varepsilon^* = \frac{\varepsilon}{\delta}$, the steady state with j infected nodes in the ε-SIS process on K_N has probability

$$\pi_j = \pi_0 \binom{N}{j} \varepsilon^* \tau^{j-1} \frac{\Gamma\left(\frac{\varepsilon^*}{\tau} + j\right)}{\Gamma\left(\frac{\varepsilon^*}{\tau} + 1\right)} \tag{A3}$$

and the healthy steady-state probability (A1) becomes

$$\pi_0 = \frac{1}{\sum_{k=0}^{N} \binom{N}{k} \tau^k \frac{\Gamma\left(\frac{\varepsilon^*}{\tau} + k\right)}{\Gamma\left(\frac{\varepsilon^*}{\tau}\right)}}. \tag{A4}$$

In [7], we have derived a linear, second order differential equation for the probability generating function $\varphi(x, t) = E[z^{M(t)}]$ and demonstrated that a time-dependent analytic solution will be difficult to find. Therefore, we return here again to the "simple" steady state of the ε-SIS process on K_N, that is analytically characterized by (A3) and (A4).

The average steady-state fraction of infected nodes, in short the steady-state prevalence, is with (A3)

$$y_{\infty;N}(\tau, \varepsilon^*) = \frac{1}{N} \sum_{j=0}^{N} j \pi_{j;N} = \frac{\pi_{0;N}}{N} \sum_{j=1}^{N} j \binom{N}{j} \tau^j \frac{\Gamma\left(\frac{\varepsilon^*}{\tau} + j\right)}{\Gamma\left(\frac{\varepsilon^*}{\tau}\right)} \tag{A5}$$

while a more general form, for any regular graph [24, Eq. (17.20) on p. 456], leads for K_N to

$$y_{\infty;N}(\tau, \varepsilon^*) = \frac{\frac{1}{N} E\left[w_\infty^T (J - I) w_\infty\right] - \frac{\varepsilon^*}{\tau}}{N - 1 - \frac{1+\varepsilon^*}{\tau}} \tag{A6}$$

where the $N \times 1$ vector w_∞ has as ith component the steady-state infection $X_{\infty,i} \in \{0, 1\}$ of node i. For $\tau = 0$, we find that $\pi_j = \pi_0 \binom{N}{j} (\varepsilon^*)^j$ and $\pi_0 = \frac{1}{(1+\varepsilon^*)^N}$, and thus $y_{\infty;N}(0, \varepsilon^*) = \frac{\varepsilon^*}{1+\varepsilon^*}$, which also follows from (A6). When $\frac{\varepsilon^*}{\tau} = \frac{\varepsilon}{\beta} = 1$, then $\pi_j = \pi_0 \frac{N!}{(N-j)!} \tau^j$, which almost reduces to the steady state of a Markov model with a forbidden absorbing state [6]. Using $j\binom{N}{j} = N\binom{N-1}{j-1}$ and (A4), we obtain

$$y_{\infty;N}(\tau, \varepsilon^*) = \frac{\sum_{k=1}^{N} \binom{N-1}{k-1} \tau^k \Gamma\left(\frac{\varepsilon^*}{\tau} + k\right)}{\sum_{k=0}^{N} \binom{N}{k} \tau^k \Gamma\left(\frac{\varepsilon^*}{\tau} + k\right)}. \tag{A7}$$

A recursion in N for the steady-state prevalence $y_{\infty;N}(\tau)$ is derived in [14, Appendix]:

$$y_{\infty;N}(\tau, \varepsilon^*) = \frac{1}{1 + \frac{1}{\varepsilon^* + (N-1)\tau y_{\infty;N-1}(\tau, \varepsilon^*)}}, \tag{A8}$$

that is equivalent to[5]

$$y_{\infty;N}(\tau, \varepsilon^*) = 1 - \frac{1}{1 + \varepsilon^* + (N-1)\tau y_{\infty;N-1}(\tau, \varepsilon^*)}.$$

Explicitly, for the first values of the number N of nodes in complete graph K_N, we list[6]

$$y_{\infty;1}(\tau, \varepsilon^*) = \frac{\varepsilon^*}{1 + \varepsilon^*},$$

$$y_{\infty;2}(\tau, \varepsilon^*) = \varepsilon^* \frac{1 + \varepsilon^* + \tau}{(1 + \varepsilon^*)^2 + \varepsilon^* \tau},$$

$$y_{\infty;3}(\tau, \varepsilon^*) = \varepsilon^* \frac{(1 + \varepsilon^*)^2 + (2 + 3\varepsilon^*)\tau + 2\tau^2}{(1 + \varepsilon^*)^3 + \varepsilon^*\{(3 + 3\varepsilon^*)\tau + 2\tau^2\}},$$

$$y_{\infty;4}(\tau, \varepsilon^*) = \varepsilon^* \frac{(1+\varepsilon^*)^3 + [3 + 9\varepsilon^* + 6(\varepsilon^*)^2]\tau + (6 + 11\varepsilon^*)\tau + 6\tau^3}{(1+\varepsilon^*)^4 + 6\varepsilon^*\{(1+\varepsilon^*)^2\tau + (8 + 11\varepsilon^*)\tau + 6\tau^3\}}.$$

Since $\pi_j = 0$ in (A3) when the self-infection rate is zero, $\varepsilon^* = 0$, for $1 \leqslant j \leqslant N$ and, consequently, $\pi_0 = 1$ (due to the fact that $\sum_{j=0}^{N} \pi_j = 1$), we retrieve the classical Markovian SIS process (with $\varepsilon^* = 0$) in which, indeed, the absorbing or overall-healthy state is the steady state for any graph with finite size N.

2. Mean-field approximation for ε-SIS epidemics on the complete graph K_N

The heterogeneous Markovian ε-SIS governing equation for node i in a graph G equals [24, Sec. 17.3]

$$\frac{dE[X_i(t)]}{dt} = E\left[-\delta_i X_i(t) + [1 - X_i(t)]\left\{\sum_{k=1}^{N} \beta_{ki} a_{ki} X_k(t) + \varepsilon_i\right\}\right]. \tag{A9}$$

The ε-SIS governing equation (A9) states that the change over time of the probability of infection $E[X_i(t)] = \Pr[X_i(t) = 1]$ of node i equals the average of two competing random variables: (a) if the node i is infected ($X_i = 1$), then $E[X_i(t)]$ decreases over time t with rate equal to the curing rate δ_i; and (b) if node i is healthy ($X_i = 0$) it can be infected with infection rate β_{ki} over any direct link a_{ki} from each infected neighbor k plus its own self-infection with rate ε_i. The total number of infected neighbors of node i is $\sum_{k=1}^{N} a_{ki} X_k$, where the adjacency matrix element a_{ki} is the explicit reference to the underlying contact graph over which the epidemic spreads.

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[5]Suppose that we equate $y_{\infty;N-1}(\tau) = y_{\infty;N}(\tau) = y$; then the above variant of the recursion (A8) leads to the quadratic equation (A14) in Theorem 2 below with $R_N(\tau) = 1$.

[6]Introducing the generating function of the Stirling numbers $S_m^{(k)}$ of the first kind [25],

$$\frac{\Gamma(x + m)}{\Gamma(x)} = \prod_{k=0}^{m-1} (x + k) = \sum_{k=0}^{m} S_m^{(k)} (-1)^{m-k} x^k,$$

the steady-state prevalence (A7) can be written as

$$y_{\infty;N}(\tau, \varepsilon^*) = \frac{\sum_{q=0}^{N} \left[\sum_{k=q}^{N} \binom{N-1}{k-1} S_k^{(k-q)} (\varepsilon^*)^{k-q}\right] (-1)^q \tau^q}{\sum_{q=0}^{N} \left[\sum_{k=q}^{N} \binom{N}{k} S_k^{(k-q)} (\varepsilon^*)^{k-q}\right] (-1)^q \tau^q}.$$

If the graph is fixed and undirected, then (A9) reduces to

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

$$- \sum_{k=1}^{N} \beta_{ki}a_{ki}E[X_i(t)X_k(t)], \qquad (A10)$$

which shows the complicating joint probabilities $E[X_iX_j] = \Pr[X_i = 1, X_j = 1]$. The mean-field approximation lies in assuming independence between infection states so that $E[X_iX_j]$ is replaced by $E[\widetilde{X}_i]E[\widetilde{X}_j]$, where the tilde in $\widetilde{X}_i$ reflects the approximative step of NIMFA. The differential equation (A10) in $v_i(t) = E[\widetilde{X}_i(t)]$ of the heterogeneous ε-SIS NIMFA equation for node i becomes

$$\frac{dv_i(t)}{dt} = \varepsilon_i - (\delta_i + \varepsilon_i)v_i(t) + [1 - v_i(t)]\sum_{k=1}^{N} \beta_{ki}a_{ki}v_k(t), \qquad (A11)$$

which is the same as replacing the random variable X_i in (A9) by its mean $E[\widetilde{X}_i]$.

We confine ourselves to the homogeneous setting in which all curing rates $\delta_i = \delta$, all nodal self-infection rates $\varepsilon_i = \varepsilon$, and all link infection rates $\beta_{ki} = \beta$ are the same. In addition, the graph is the complete graph K_N and we limit ourselves to the steady state $v_{i\infty} = \lim_{t \to \infty} v_i(t)$, where $\frac{dv_{i\infty}(t)}{dt} = 0$ and symmetry in K_N dictates that $v_{i\infty} = v_\infty = y_\infty$. Under these conditions (A11) simplifies to

$$0 = \varepsilon - (\delta + \varepsilon)v_\infty + (1 - v_\infty)\beta(N-1)v_\infty.$$

Let $\tau = \frac{\beta}{\delta}$ and $\varepsilon^* = \frac{\varepsilon}{\delta}$; then we arrive at the quadratic equation in $y_\infty = v_\infty$:

$$y_\infty^2 - \left(1 - \frac{1 + \varepsilon^*}{(N-1)\tau}\right)y_\infty - \frac{\varepsilon^*}{(N-1)\tau} = 0,$$

which is (A14) with $R_N(\tau) = 1$. The mean-field prevalence y_∞ does not exhibit a phase transition for small self-infection rates ε.

The ε-SIS steady state follows from (A6) with $J = u.u^T$, where u is the all-one vector, with $w_\infty^T(J - I)w_\infty = (w_\infty^T u)^2 - w_\infty^T w_\infty = (w_\infty^T u)^2 - w_\infty^T u$ and with $w_\infty^T u = NX_\infty$ as

$$y_{\infty;N}(\tau, \varepsilon^*) = \frac{NE[X_\infty^2] - E[X_\infty] - \frac{\varepsilon^*}{\tau}}{N - 1 - \frac{1 + \varepsilon^*}{\tau}}$$

where the random variable X_∞ denotes the steady infection state of any node (by symmetry of the complete graph in the steady state). Since $E[X_\infty] = y_{\infty;N}(\tau, \varepsilon^*)$ and $E[X_\infty^2] = y_{\infty;N}^2(\tau, \varepsilon^*) + \text{Var}[X_\infty]$, we find

$$y_{\infty;N}^2(\tau, \varepsilon^*)$$
$$- \left(1 - \frac{1 + \varepsilon^*}{N\tau}\right)y_{\infty;N}(\tau, \varepsilon^*) + \text{Var}[X_\infty] - \frac{\varepsilon^*}{N\tau} = 0.$$

The quadratic equation is a general feature of SIS epidemics on a graph, resulting in a Riccati type of differential equation for the time-variant prevalence as shown in [26]. Subtraction from the quadratic equation (A14), derived below in terms of $R_N(\tau) = \frac{y_{\infty;N-1}(\tau, \varepsilon^*)}{y_{\infty;N}(\tau, \varepsilon^*)}$, leads to an expression for the variance of the fraction of infected nodes in K_N:

$$\text{Var}[X_\infty] = \frac{1 + \varepsilon^*}{\tau N}\left(\frac{N}{(N-1)R_N(\tau)} - 1\right)\{y_{\infty;N}(\tau, \varepsilon^*)$$
$$- y_{\infty;N}(0, \varepsilon^*)\}. \qquad (A12)$$

3. Properties of the steady-state prevalence $y_{\infty;N}(\tau)$ in K_N

It follows from the recursion (A8) that

$$\frac{1}{y_{\infty;N}(\tau, \varepsilon^*)} - \frac{1}{y_{\infty;N-1}(\tau, \varepsilon^*)} = \frac{\tau[(N-2)y_{\infty;N-2}(\tau, \varepsilon^*) - (N-1)y_{\infty;N-1}(\tau, \varepsilon^*)]}{[\tau(N-1)y_{\infty;N-1}(\tau, \varepsilon^*) + \varepsilon^*][\tau(N-2)y_{\infty;N-2}(\tau, \varepsilon^*) + \varepsilon^*]}.$$

If the right-hand side is negative, so is the left-hand side. Hence, the implication between two inequalities

$$\{(N-2)y_{\infty;N-2}(\tau, \varepsilon^*) < (N-1)y_{\infty;N-1}(\tau, \varepsilon^*)\} \implies \{y_{\infty;N-1}(\tau, \varepsilon^*) < y_{\infty;N}(\tau, \varepsilon^*)\} \qquad (A13)$$

holds for all N.

Theorem 1. The steady-state prevalence $y_{\infty;N}(\tau, \varepsilon^*)$ in the complete graph K_N satisfies the inequality $y_{\infty;N-1}(\tau, \varepsilon^*) < y_{\infty;N}(\tau, \varepsilon^*)$ for $\tau > 0$, for $\varepsilon^* > 0$ and for any $N \geqslant 2$.

Since $y_{\infty;N}(0, \varepsilon^*) = \frac{\varepsilon^*}{1+\varepsilon^*}$ for any N, Theorem 1 excludes $\tau = 0$ and provides strict inequalities $y_{\infty;1}(\tau, \varepsilon^*) < y_{\infty;2}(\tau, \varepsilon^*) < \cdots < y_{\infty;N}(\tau, \varepsilon^*) < \cdots$ for $\tau > 0$.

Proof by induction. (a) We start by demonstrating that the inequality $y_{\infty;N-1}(\tau, \varepsilon^*) < y_{\infty;N}(\tau, \varepsilon^*)$ holds for $N = 2$. Indeed, from the explicit evaluation, we deduce for $\tau > 0$ that

$$y_{\infty;1}(\tau, \varepsilon^*) = \frac{\varepsilon^*}{1 + \varepsilon^*}, \quad y_{\infty;2}(\tau, \varepsilon^*) = \frac{\varepsilon^*(1 + \varepsilon^*) + \varepsilon^*\tau}{(1 + \varepsilon^*)^2 + \varepsilon^*\tau} = \frac{\varepsilon^*}{1 + \varepsilon^*}\frac{(1 + \varepsilon^*) + \tau}{(1 + \varepsilon^*) + \frac{\varepsilon^*}{1+\varepsilon^*}\tau} > \frac{\varepsilon^*}{1 + \varepsilon^*} = y_{\infty;1}(\tau, \varepsilon^*).$$

Also the inequality $(N-2)y_{\infty;N-2}(\tau, \varepsilon^*) < (N-1)y_{\infty;N-1}(\tau, \varepsilon^*)$ holds for $N = 3$, namely, $y_{\infty;1}(\tau, \varepsilon^*) < 2y_{\infty;2}(\tau, \varepsilon^*)$.

(b) *The induction hypothesis*: Assume that $(N-2)y_{\infty;N-2}(\tau, \varepsilon^*) < (N-1)y_{\infty;N-1}(\tau, \varepsilon^*)$ holds for $N = n$.

(c) We need to verify that inequality $(N - 2)y_{\infty;N-2}(\tau, \varepsilon^*) < (N - 1)y_{\infty;N-1}(\tau, \varepsilon^*)$ also holds for $N = n + 1$. Now, the induction hypothesis for $N = n$ in (b),

$$(n - 2)y_{\infty;n-2}(\tau, \varepsilon^*) < (n - 1)y_{\infty;n-1}(\tau, \varepsilon^*),$$

implies by (A13) that

$$y_{\infty;n-1}(\tau, \varepsilon^*) < y_{\infty;n}(\tau, \varepsilon^*).$$

Since $(\frac{n-1}{n})y_{\infty;n-1}(\tau, \varepsilon^*) < y_{\infty;n-1}(\tau, \varepsilon^*)$, the above inequality translates to $(\frac{n-1}{n})y_{\infty;n-1}(\tau, \varepsilon^*) < y_{\infty;n}(\tau, \varepsilon^*)$ and is equivalent to

$$(n - 1)y_{\infty;n-1}(\tau, \varepsilon^*) < ny_n(\tau, \varepsilon^*),$$

which is the inequality to be verified for $N = n + 1$. This proves the induction argument and Theorem 1. ∎

Theorem 2. The steady-state prevalence $y_{\infty;N}(\tau, \varepsilon^*)$ in the complete graph K_N satisfies the quadratic equation for $\tau > 0$:

$$y_{\infty;N}^2(\tau, \varepsilon^*) - \left(1 - \frac{1 + \varepsilon^*}{(N - 1)\tau R_N(\tau)}\right)y_{\infty;N}(\tau, \varepsilon^*)$$

$$- \frac{\varepsilon^*}{(N - 1)\tau R_N(\tau)} = 0 \tag{A14}$$

where $R_N(\tau) = \frac{y_{\infty;N-1}(\tau,\varepsilon^*)}{y_{\infty;N}(\tau,\varepsilon^*)}$, and its solution

$$y_{\infty;N}(\tau, \varepsilon^*) = \left(1 - \frac{1 + \varepsilon^*}{(N - 1)\tau R_N(\tau)}\right)\frac{1}{2}\left\{1 + \sqrt{1 + \frac{4\varepsilon^* R_N(\tau)}{(N - 1)\tau\left[R_N(\tau) - \frac{(1+\varepsilon^*)}{(N-1)\tau}\right]^2}}\right\}. \tag{A15}$$

Proof. Denoting the ratio $R_N(\tau) = \frac{y_{\infty;N-1}(\tau,\varepsilon^*)}{y_{\infty;N}(\tau,\varepsilon^*)}$, we rewrite the recursion (A8) as

$$y_{\infty;N}(\tau, \varepsilon^*) = 1 - \frac{1}{\frac{\varepsilon^*}{y_{\infty;N}(\tau,\varepsilon^*)} + (N - 1)\tau R_N(\tau, \varepsilon^*)}. \tag{A16}$$

Next, we rewrite the recursion (A16) as the quadratic equation (A14), whose solution is

$$y_{\infty;N}(\tau, \varepsilon^*) = \frac{1}{2}\left(1 - \frac{1 + \varepsilon^*}{(N - 1)\tau R_N(\tau)}\right) \pm \frac{1}{2}\sqrt{\left(1 - \frac{1 + \varepsilon^*}{(N - 1)\tau R_N(\tau)}\right)^2 + \frac{4\varepsilon^*}{(N - 1)\tau R_N(\tau)}}.$$

The discriminant of the quadratic equation (A14) is larger than $(1 - \frac{1+\varepsilon^*}{(N-1)\tau R_N(\tau)})^2$, which excludes the minus sign because $y_{\infty;N}(\tau, \varepsilon^*) \geqslant 0$. After some manipulations we find (A15). ∎

For $R_N(\tau) = 1$ and self-infection rate $\varepsilon^* = 0$, the steady-state prevalence $y_{\infty;N}(\tau, \varepsilon^*)$ in (A15) of the ε-SIS process on K_N reduces to the steady-state fraction $y_{\infty;N}^{(1)}(\tau) = 1 - \frac{1}{(N-1)\tau}$ in (1) of infected nodes in NIMFA for the complete graph K_N. We have shown in [27] that NIMFA always upper bounds the probability of nodal infection, hence $y_{\infty;N}^{(1)}(\tau) \geqslant y_{\infty;N}(\tau; 0)$. The argument suggests us to consider the condition that $R_N(\tau) = \frac{y_{\infty;N-1}(\tau,\varepsilon^*)}{y_{\infty;N}(\tau,\varepsilon^*)} = 1$ as equivalent to the mean-field approximation, that assumes independence between viral states of different nodes [27]. The second factor $\frac{1}{2}\{\ldots\}$ in (A15) is only slightly larger than 1 for $\tau \geqslant \tau_c^{(1)} = \frac{1}{N-1}$. However, the NIMFA quadratic equation of the steady-state prevalence [(A14) with $R_N(\tau) = 1$] indicates that NIMFA provides an envelope below or at which $y_{\infty;N}(\tau, \varepsilon^*)$ lies.

While intuition would hint that $R_N(\tau) \lessapprox 1$, at least for large N, as we did in [14, Appendix], the truth is surprisingly different: computations show that $1 - R_N(\tau)$ is near to $y_{\infty;N}(\tau, \varepsilon^*)$ for $\varepsilon^* = 10^{-a}$ and $a = 3, 5, 10, 20, 30, 40, 50$.

4. Estimate of the onset τ_c^ε of the ε-SIS phase transition for small self-infection rates ε

Theorem 3. For small self-infection rate ε, the steady-state prevalence $y_{\infty;N}(\tau, \varepsilon^*)$ in the complete graph K_N is up to order $O(\varepsilon^3)$

$$y_{\infty;N}(\tau, \varepsilon^*) = \frac{\varepsilon^*}{\tau}\sum_{k=1}^{N}\frac{(N - 1)!\tau^k}{(N - k)!} + \left(\frac{\varepsilon^*}{\tau}\right)^2\sum_{k=1}^{N}\frac{(N - 1)!\tau^k}{(N - k)!}\sum_{j=1}^{k-1}\frac{1}{j} + O(\varepsilon^3). \tag{A17}$$

Proof. For small ε, we expand $\Gamma(k + \frac{\varepsilon^*}{\tau})$ in a Taylor series around $z = k$:

$$\Gamma\left(k + \frac{\varepsilon^*}{\tau}\right) = \Gamma(k) + \Gamma'(k)\frac{\varepsilon^*}{\tau} + O(\varepsilon^2) = \Gamma(k)\left\{1 + \psi(k)\frac{\varepsilon^*}{\tau} + O(\varepsilon^2)\right\}$$

where the digamma function is $\psi(z) = \frac{\Gamma'(z)}{\Gamma(z)}$ (see [25, Sec. 6.3]). For integer $z = k \geqslant 1$, $\psi(k) = -\gamma + \sum_{j=1}^{k-1}\frac{1}{j} \geqslant 0$ so that

$$\Gamma\left(k + \frac{\varepsilon^*}{\tau}\right) = (k - 1)!\left\{1 + \left(-\gamma + \sum_{j=1}^{k-1}\frac{1}{j}\right)\frac{\varepsilon^*}{\tau} + O(\varepsilon^2)\right\}$$

while

$$\Gamma\left(\frac{\varepsilon^*}{\tau}\right) = \frac{\tau}{\varepsilon^*}\Gamma\left(\frac{\varepsilon^*}{\tau}+1\right) = \frac{\tau}{\varepsilon^*}\left[1 - \gamma\frac{\varepsilon^*}{\tau} + \frac{\Gamma''(1)}{2}\left(\frac{\varepsilon^*}{\tau}\right)^2 + O(\varepsilon^3)\right] = \frac{\tau}{\varepsilon^*} - \gamma + \frac{\Gamma''(1)}{2}\frac{\varepsilon^*}{\tau} + O(\varepsilon^2)$$

and $\Gamma''(1) = \frac{d^2\Gamma(z)}{dz^2}|_{z=1} = 1.97811$ (see [28, Appendix]). Substituted in steady-state prevalence, (A7) gives us

$$y_{\infty;N}(\tau,\varepsilon^*) = \frac{(N-1)!\sum_{k=1}^{N}\frac{\tau^k}{(N-k)!}\{1 + \psi(k)\frac{\varepsilon^*}{\tau} + O(\varepsilon^2)\}}{\Gamma\left(\frac{\varepsilon^*}{\tau}\right) + N!\sum_{k=1}^{N}\frac{\tau^k}{k(N-k)!}\{1 + \psi(k)\frac{\varepsilon^*}{\tau} + O(\varepsilon^2)\}}$$

$$= \frac{(N-1)!\sum_{k=1}^{N}\frac{\tau^k}{(N-k)!} + (N-1)!\varepsilon^*\sum_{k=1}^{N}\frac{\tau^{k-1}}{(N-k)!}\psi(k) + O(\varepsilon^2)}{\frac{\tau}{\varepsilon^*} - \gamma + \frac{\Gamma''(1)}{2}\frac{\varepsilon^*}{\tau} + \varepsilon^* N!\sum_{k=1}^{N}\frac{\tau^k}{k(N-k)!} + (\varepsilon^*)^2 N!\sum_{k=1}^{N}\frac{\tau^{k-1}}{k(N-k)!}\psi(k) + O(\varepsilon^3)}.$$

Up to order $O(\varepsilon^2)$, we have

$$y_{\infty;N}(\tau,\varepsilon^*) = \frac{(N-1)!\sum_{k=1}^{N}\frac{\tau^k}{(N-k)!} + (N-1)!\varepsilon^*\sum_{k=1}^{N}\frac{\tau^{k-1}}{(N-k)!}\psi(k) + O(\varepsilon^2)}{\frac{\tau}{\varepsilon^*} - \gamma + \varepsilon^*\left(\frac{\Gamma''(1)}{2}\frac{1}{\tau} + N!\sum_{k=1}^{N}\frac{\tau^k}{k(N-k)!}\right) + O(\varepsilon^2)}$$

$$= \frac{1}{\tau}\frac{\varepsilon^*(N-1)!\sum_{k=1}^{N}\frac{\tau^k}{(N-k)!} + (N-1)!(\varepsilon^*)^2\sum_{k=1}^{N}\frac{\tau^{k-1}}{(N-k)!}\psi(k) + O(\varepsilon^3)}{1 - \varepsilon^*\left[\frac{\gamma}{\tau} - \varepsilon^*\left(\frac{\Gamma''(1)}{2}\frac{1}{\tau^2} + N!\sum_{k=1}^{N}\frac{\tau^{k-1}}{k(N-k)!}\right)\right] + O(\varepsilon^3)}.$$

Expanding the denominator with the geometric series results in

$$y_{\infty;N}(\tau,\varepsilon^*) = \frac{1}{\tau}\left(\varepsilon^*(N-1)!\sum_{k=1}^{N}\frac{\tau^k}{(N-k)!} + (N-1)!(\varepsilon^*)^2\sum_{k=1}^{N}\frac{\tau^{k-1}}{(N-k)!}\psi(k) + O(\varepsilon^3)\right)$$

$$\times \left[1 + \varepsilon^*\frac{\gamma}{\tau} - (\varepsilon^*)^2\left(\frac{\Gamma''(1)}{2}\frac{1}{\tau^2} + N!\sum_{k=1}^{N}\frac{\tau^{k-1}}{k(N-k)!}\right)\right].$$

Simplifying further up to $O(\varepsilon^3)$ demonstrates (A17). ∎

Theorem 4. For a small self-infection rate ε in an ε-SIS epidemic on the complete graph K_N, the phase transition in the steady-state prevalence $y_{\infty;N}(\tau,\varepsilon^*)$ lies in between

$$\frac{1}{e}\left(\frac{10^{-s}}{\varepsilon^*(N-1)!}\right)^{\frac{1}{N-1}} < \tau_c^{\varepsilon} < \left(\frac{10^{-s}}{\varepsilon^*(N-1)!}\right)^{\frac{1}{N-1}} \quad (A18)$$

where s specifies an agreed level for the onset of the phase transition at which $y_{\infty;N}(\tau,\varepsilon^*) = 10^{-s}$ is first reached, when τ is gradually increased from $\tau = 0$ at $y_{\infty;N}(0,\varepsilon^*) = \frac{\varepsilon^*}{1+\varepsilon^*}$ on.

Proof. Equating $y_{\infty;N}(\tau,\varepsilon^*) = 10^{-s}$ and invoking (A17) for $\tau > 0$ up to first order in ε^* results in

$$\frac{10^{-s}}{\varepsilon^*} = \sum_{k=1}^{N}\frac{(N-1)!\tau^{k-1}}{(N-k)!}, \quad (A19)$$

which shows that the solution at $\tau = \tau_c^{\varepsilon}$ is a zero of a polynomial of degree $N-1$ in τ, which cannot be expressed analytically in closed form for $N > 5$. After transforming $\sum_{k=1}^{N}\frac{(N-1)!\tau^{k-1}}{(N-k)!} = (N-1)!\tau^{N-1}\sum_{k=0}^{N-1}\frac{\tau^{-j}}{j!}$, the bounds $1 < \sum_{k=0}^{N-1}\frac{\tau^{-j}}{j!} < e^{\frac{1}{\tau}}$ for $\tau > 0$ illustrate that the zero τ_c^{ε} satisfies

$$(N-1)!\tau^{N-1} < \frac{10^{-s}}{\varepsilon^*} \quad \text{and} \quad \frac{10^{-s}}{\varepsilon^*} < (N-1)!\tau^{N-1}e^{\frac{1}{\tau}}$$

or, after inversion,

$$\left(\frac{10^{-s}}{\varepsilon^*(N-1)!}\right)^{\frac{1}{N-1}}e^{-\frac{1}{\tau_c^{\varepsilon}(N-1)}} < \tau_c^{\varepsilon} < \left(\frac{10^{-s}}{\varepsilon^*(N-1)!}\right)^{\frac{1}{N-1}}.$$

Clearly, the smaller $e^{-\frac{1}{\tau_c^{\varepsilon}(N-1)}}$, the sharper the bounds are. Since the onset of the ε-SIS epidemic threshold τ_c^{ε} exceeds, for small ε, the NIMFA epidemic threshold $\tau_c^{(1)} = \frac{1}{N-1}$ and, thus, $(N-1)\tau_c^{\varepsilon} > 1$, we have that $1 > e^{-\frac{1}{\tau_c^{\varepsilon}(N-1)}} > e^{-1} = 0.367$, which demonstrates Theorem 4. ∎

The bounds (A18) enable rapid numerical determination of the zero τ_c^{ε}. The phase transition at $\tau = \tau_c^{\varepsilon}$ obeys (A19):

$$\tau = \left(\frac{10^{-s}}{\varepsilon^*(N-1)!}\right)^{\frac{1}{N-1}}\left(\frac{1}{\sum_{k=0}^{N-1}\frac{\tau^{-j}}{j!}}\right)^{\frac{1}{N-1}},$$

which we rewrite as an iterative system in $m \geqslant 1$:

$$\phi_m = \left(\frac{\frac{10^{-s}}{\varepsilon^*(N-1)!}}{1 + \sum_{k=1}^{N-1}\frac{\phi_{m-1}^{-j}}{j!}}\right)^{\frac{1}{N-1}}$$

with initial value $\phi_0 = \left(\frac{10^{-s}}{\varepsilon^*(N-1)!}\right)^{\frac{1}{N-1}}$. The Lagrange series of (A19) is another analytic approach, that we omit here.

5. Scaling of the steady-state prevalence $y_{\infty;N+1}(\tau,\varepsilon^*)$

We consider here the complete graph K_{N+1} to simplify the computations, because $N-1 \rightarrow N$, so that the bounds[7]

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[7]Since the maximum of the terms in $\sum_{k=0}^{N}\frac{\tau^{-j}}{j!}$ with $\tau = \frac{x}{N}$ occurs at $j = [\frac{N}{x}]$, after which the terms start decreasing, the approximation $e^{\frac{1}{\tau}} \approx \sum_{k=0}^{N}\frac{\tau^{-j}}{j!}$ is only reasonable if $x \geqslant 1$, which is in agreement with Sec. A 7.

derived in the proof of Theorem 4 for small ε^* are

$$\varepsilon^* N! \tau^N < y_{\infty;N+1}(\tau, \varepsilon^*) < \varepsilon^* N! \tau^N e^{\frac{1}{\tau}}.$$

Using the mean-field scaling $\tau = \frac{x}{N}$ and Stirling's approximation $N! = \sqrt{2\pi N} N^N e^{-N + \frac{\theta}{12N}}$ for $0 < \theta < 1$ (see [25, 6.1.38]), we find for K_{N+1} that

$$\varepsilon^* \sqrt{2\pi N} e^{(-1 + \ln x)N} < y_{\infty;N+1}\left(\frac{x}{N}, \varepsilon^*\right)$$
$$< \varepsilon^* \sqrt{2\pi N} e^{(\frac{1}{x} - 1 + \ln x)N} e^{\frac{\theta}{12N}}.$$

We also know that the NIMFA steady-state prevalence $y_{\infty;N+1}^{(1)}(\frac{x}{N}) = 1 - \frac{1}{x}$ upper bounds the steady-state prevalence $y_{\infty;N+1}(\frac{x}{N}, \varepsilon^*)$ and, above $x > 1$, the NIMFA upper bound is considerably sharper than the above upper bound. Furthermore, if we choose $\varepsilon^* = \frac{e^{-zN}}{\sqrt{2\pi N}}$, then

$$e^{(-1 + \ln x - z)N} < y_{\infty;N+1}\left(\frac{x}{N}, \frac{e^{-zN}}{\sqrt{2\pi N}}\right) < 1 - \frac{1}{x}. \quad (A20)$$

The scaled inequality (A20) means that, for effective infection rate τ below the NIMFA epidemic threshold $\tau_c^{(1)} = \frac{1}{N}$ in K_{N+1}, corresponding to $x = 1$, the steady-state prevalence $y_{\infty;N+1}(\tau, \varepsilon^*) < \sqrt{2\pi N} e^{\frac{\theta}{12N}} \varepsilon^*$ is thus negligibly small for small ε^*, but $y_{\infty;N+1}(\tau, \varepsilon^*)$ starts to increase exponentially in τ with "rate" at least $-1 + \ln x - z$ for $x > 1$ until the NIMFA prevalence $y_{\infty;N+1}^{(1)}(\tau) = 1 - \frac{1}{x}$ is reached. Thus, for $-1 + \ln x - z > 0$ or $x > e^{z+1}$, the lower bound in inequality (A20) equals the maximum possible and indicates that an estimate of the phase transition lies around $x \approx e^{z+1}$, which is confirmed by numerical computations in Fig. 5.

6. Prevalence $y_{\infty;N}(\tau)$ in terms of confluent hypergeometric functions

Since $\frac{\varepsilon^*}{\tau} + k > 0$ in any term of (A7), we use, as in [7], Euler's integral for the Gamma function $\Gamma(z) = \int_0^\infty e^{-u} u^{z-1} du$ for $\mathrm{Re}(z) > 0$ in (A4),

$$\frac{\Gamma\left(\frac{\varepsilon^*}{\tau}\right)}{\pi_{0;N}} = \sum_{k=0}^N \binom{N}{k} \tau^k \Gamma\left(\frac{\varepsilon^*}{\tau} + k\right)$$
$$= \sum_{k=0}^N \binom{N}{k} \tau^k \int_0^\infty e^{-u} u^{\frac{\varepsilon^*}{\tau} + k - 1} du,$$

resulting in the inverse of the probability that the virus is extinct in the steady state of the ε-SIS process on K_N:

$$\frac{1}{\pi_{0;N}} = \frac{1}{\Gamma\left(\frac{\varepsilon^*}{\tau}\right)} \int_0^\infty e^{-u} u^{\frac{\varepsilon^*}{\tau} - 1} (1 + \tau u)^N du. \quad (A21)$$

Let $x = \tau u$ in (A21); then $u = \frac{1}{\tau} x$:

$$\frac{1}{\pi_{0;N}} = \frac{1}{\tau^{\frac{\varepsilon^*}{\tau}} \Gamma\left(\frac{\varepsilon^*}{\tau}\right)} \int_0^\infty e^{-\frac{1}{\tau} x} x^{\frac{\varepsilon^*}{\tau} - 1} (1 + x)^N dx,$$

which can be rewritten as a confluent hypergeometric function [25, 13.2.5]

$$U(a, b, z) = \frac{1}{\Gamma(a)} \int_0^\infty e^{-zx} x^{a-1} (1 + x)^{b-a-1} dx,$$

$$\mathrm{Re}(b) > \mathrm{Re}(a) > 0, \text{ and } \mathrm{Re}(x) > 0$$

as[8]

$$\frac{1}{\pi_{0;N}} = \frac{1}{\Gamma\left(\frac{\varepsilon^*}{\tau}\right)} \sum_{k=0}^N \binom{N}{k} \tau^k \Gamma\left(\frac{\varepsilon^*}{\tau} + k\right)$$

$$= \tau^{-\frac{\varepsilon^*}{\tau}} U\left(\frac{\varepsilon^*}{\tau}, N + 1 + \frac{\varepsilon^*}{\tau}, \frac{1}{\tau}\right). \quad (A22)$$

The steady-state prevalence, after decreasing $k \to k - 1$ in the numerator of (A7), becomes

$$y_{\infty;N}(\tau, \varepsilon^*) = \tau \frac{\sum_{k=0}^{N-1} \binom{N-1}{k} \tau^k \Gamma\left(\frac{\varepsilon^*}{\tau} + 1 + k\right)}{\sum_{k=0}^N \binom{N}{k} \tau^k \Gamma\left(\frac{\varepsilon^*}{\tau} + k\right)}$$

and invoking (A22) leads to

$$y_{\infty;N}(\tau, \varepsilon^*) = \frac{\varepsilon^*}{\tau} \frac{U\left(\frac{\varepsilon^*}{\tau} + 1, N + 1 + \frac{\varepsilon^*}{\tau}, \frac{1}{\tau}\right)}{U\left(\frac{\varepsilon^*}{\tau}, N + 1 + \frac{\varepsilon^*}{\tau}, \frac{1}{\tau}\right)}. \quad (A23)$$

There are a number of "contiguous" relations [25, 13.4.15-13.4.20], coined by Gauss for three term relations between hypergeometric functions, that provide recursion relations as in (A8).

Rather than using the theory of confluent hypergeometric functions, we concentrate on the integral representation (A21):

$$y_{\infty;N}(\tau, \varepsilon^*) = \tau \frac{\int_0^\infty e^{-u} u^{\frac{\varepsilon^*}{\tau}} (1 + \tau u)^{N-1} du}{\int_0^\infty e^{-u} u^{\frac{\varepsilon^*}{\tau} - 1} (1 + \tau u)^N du}. \quad (A24)$$

Splitting the denominator into two integrals after using $(1 + \tau u)^N = (1 + \tau u)^{N-1}(1 + \tau u)$, we find that

$$y_{\infty;N}(\tau, \varepsilon^*) = \frac{1}{1 + \frac{\int_0^\infty e^{-u} u^{\frac{\varepsilon^*}{\tau} - 1} (1 + \tau u)^{N-1} du}{\tau \int_0^\infty e^{-u} u^{\frac{\varepsilon^*}{\tau}} (1 + \tau u)^{N-1} du}}. \quad (A25)$$

Similarly, since $\tau \int_0^\infty e^{-u} u^{\frac{\varepsilon^*}{\tau}} (1 + \tau u)^{N-1} du = \int_0^\infty e^{-u} u^{\frac{\varepsilon^*}{\tau} - 1} \tau u (1 + \tau u)^{N-1} du$ and $\tau u (1 + \tau u)^{N-1} = (1 + \tau u)^N - (1 + \tau u)^{N-1}$, we have

$$y_{\infty;N}(\tau, \varepsilon^*) = 1 - \frac{\int_0^\infty e^{-u} u^{\frac{\varepsilon^*}{\tau} - 1} (1 + \tau u)^{N-1} du}{\int_0^\infty e^{-u} u^{\frac{\varepsilon^*}{\tau} - 1} (1 + \tau u)^N du}. \quad (A26)$$

Invoking (A21), we find

$$y_{\infty;N}(\tau, \varepsilon^*) = 1 - \frac{\pi_{0;N}}{\pi_{0;N-1}}. \quad (A27)$$

Since $y_{\infty;N}(\tau, \varepsilon^*) > 0$ for $\varepsilon^* > 0$, we establish that $\pi_{0;N-1} > \pi_{0;N}$ for all N and τ. Alternatively, using $\frac{\partial}{\partial z}(1 + \tau u)^z = (1 + \tau u)^z \log(1 + \tau u) \geqslant 0$ for $u \geqslant 0$ for any real z and thus also for $z = N$, (A21) shows that $\frac{1}{\pi_{0;N}}$ increases (even strictly monotonously if $\varepsilon^* > 0$) with N and $\frac{1}{\pi_{0;N}} \to \infty$ when $N \to \infty$. Hence, the viral extinction probability $\pi_{0;N}$ decreases with N and tends to zero with $N \to \infty$, which implies that the

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[8]Alternatively, with Kummer's transformation [25, 13.1.29], $U(a, b, z) = z^{1-b} U(1 + a - b, 2 - b, z)$, we have

$$\frac{1}{\pi_0} = \tau^N U\left(-N, 1 - N - \frac{\varepsilon^*}{\tau}, \frac{1}{\tau}\right).$$

absorbing state is almost surely impossible in an infinitely large complete graph, provided the self-infection rate $\varepsilon^* > 0$.

Partial integration of the numerator in (A24),

$$\int_0^\infty e^{-u} u^{\frac{\varepsilon^*}{\tau}} (1 + \tau u)^{N-1} du$$

$$= \frac{1}{N\tau} \int_0^\infty e^{-u} u^{\frac{\varepsilon^*}{\tau}} (1 + \tau u)^N du$$

$$- \frac{\varepsilon^*}{\tau} \frac{1}{N\tau} \int_0^\infty e^{-u} u^{\frac{\varepsilon^*}{\tau}-1} (1 + \tau u)^N du,$$

yields

$$N y_{\infty;N}(\tau, \varepsilon^*) + \frac{\varepsilon^*}{\tau} = \frac{\int_0^\infty e^{-u} u^{\frac{\varepsilon^*}{\tau}} (1 + \tau u)^N du}{\int_0^\infty e^{-u} u^{\frac{\varepsilon^*}{\tau}-1} (1 + \tau u)^N du}.$$

Introducing (A25) shows that

$$\tau \left(\frac{1}{y_{\infty;N}(\tau, \varepsilon^*)} - 1 \right) = \frac{1}{(N-1) y_{\infty;N-1}(\tau, \varepsilon^*) + \frac{\varepsilon^*}{\tau}},$$

which is again equivalent to the recursion (A8).

7. Large N asymptotics

We let $\tau = \frac{a}{N}$ in $\sum_{k=0}^N \binom{N}{k} \tau^k \Gamma(\frac{\varepsilon^*}{\tau} + k) = \int_0^\infty e^{-u} u^{\frac{\varepsilon^*}{\tau}-1} (1 + \tau u)^N du$, because

$$\left(1 + \frac{au}{N} \right)^N = e^{au} \left[1 + O\left(\frac{1}{N} \right) \right]$$

illustrates that a linear scaling of τ leads to a finite, nontrivial result[9] for large N. Hence, we obtain, for $0 < a < 1$,

$$\int_0^\infty e^{-u} u^{\frac{N\varepsilon^*}{a}-1} \left(1 + \frac{au}{N} \right)^N du$$

$$= \int_0^\infty e^{-u(1-a)} u^{\frac{N\varepsilon^*}{a}-1} \left[1 + O\left(\frac{1}{N} \right) \right] du$$

$$= \frac{\Gamma\left(\frac{N\varepsilon^*}{a} \right)}{(1-a)^{\frac{N\varepsilon^*}{a}}} \left[1 + O\left(\frac{1}{N} \right) \right]$$

while the integral diverges for $a > 1$. In order to have a same exponent of $(1 + \tau u)$, we consider (A25)

$$y_{\infty;N}(\tau, \varepsilon^*) = \frac{1}{1 + \frac{\int_0^\infty e^{-u} u^{\frac{\varepsilon^*}{\tau}-1} (1+\tau u)^{N-1} du}{\tau \int_0^\infty e^{-u} u^{\frac{\varepsilon^*}{\tau}} (1+\tau u)^{N-1} du}}$$

$$= \frac{1}{1 + \frac{N}{a} \frac{\int_0^\infty e^{-u} u^{\frac{N\varepsilon^*}{a}-1} \left(1+\frac{a}{N} u\right)^{N-1} du}{\int_0^\infty e^{-u} u^{\frac{N\varepsilon^*}{a}} \left(1+\frac{a}{N} u\right)^{N-1} du}}.$$

With

$$\frac{N}{a} \frac{\int_0^\infty e^{-u} u^{\frac{N\varepsilon^*}{a}-1} \left(1+\frac{a}{N} u\right)^{N-1} du}{\int_0^\infty e^{-u} u^{\frac{N\varepsilon^*}{a}} \left(1+\frac{a}{N} u\right)^{N-1} du}$$

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[9]All other scalings $\tau = \frac{a}{N^\alpha}$ do not, unless $\alpha = 1$. A linear scaling also agrees with NIMFA.

$$= \frac{N}{a} \frac{\int_0^\infty e^{-u(1-a)} u^{\frac{N\varepsilon^*}{a}-1} \left[1 + O\left(\frac{1}{N}\right)\right] du}{\int_0^\infty e^{-u(1-a)} u^{\frac{N\varepsilon^*}{a}} \left[1 + O\left(\frac{1}{N}\right)\right] du}$$

$$= \frac{N}{a} \frac{\Gamma\left(\frac{N\varepsilon^*}{a}\right)(1-a)^{\frac{N\varepsilon^*}{a}+1}}{(1-a)^{\frac{N\varepsilon^*}{a}} \Gamma\left(\frac{N\varepsilon^*}{a}+1\right)} = \frac{(1-a)}{\varepsilon^*}$$

we have, provided $0 < a < 1$ (thus below the NIMFA epidemic threshold, significantly), in the thermodynamic limit for $N \to \infty$,

$$y_{\infty;N}\left(\frac{a}{N}, \varepsilon^* \right) \to \frac{1}{1 + \frac{(1-a)}{\varepsilon^*}} = \frac{\varepsilon^*}{1 + \varepsilon^* - \tau N}$$

where the right-hand side is an upper bound for $y_{\infty;N}(\tau, \varepsilon^*)$, as shown in [24, p. 486], consistent with Theorem 1. Due to the integral convergence constraint $a < 1$, we cannot deduce the explosive phase transition τ_c^ε for $N \to \infty$.

More elegantly, we take the limit $N \to \infty$ of a variant of the recursion (A8),

$$y_{\infty;N}(\tau, \varepsilon^*) = 1 - \frac{1}{1 + \varepsilon^* + (N-1)\tau y_{\infty;N-1}(\tau, \varepsilon^*)},$$

denote the "size limit prevalence" by $y = \lim_{N\to\infty} y_{\infty;N}(\tau(N), \varepsilon^*(N))$, and obtain

$$y = 1 - \frac{1}{1 + E + Ty}$$

where $T = \lim_{N\to\infty} (N-1)\tau(N)$ and $E = \lim_{N\to\infty} \varepsilon^*(N)$. The two extremes, $T = 0$ and ∞, lead to

$$\frac{E}{1+E} \leqslant y \leqslant 1.$$

The bounds emphasize that large E (irrespective of T) results in the less interesting case of a prevalence approaching 1. Rewriting $y = 1 - \frac{1}{1+E+Ty}$ as a quadratic equation

$$Ty^2 + (1 + E - T)y - E = 0$$

leads, after maintaining only the positive root, to

$$y = \frac{1}{2} \left[1 - \left(\frac{1+E}{T} \right) + \sqrt{ \left(1 - \frac{1+E}{T} \right)^2 + 4\frac{E}{T} } \right],$$

which means that roughly the ratio $r = \frac{1+E}{T}$ is determining

$$y = \frac{1}{2} \left(1 - r + \sqrt{(1+r)^2 - \frac{4}{T}} \right).$$

The finite T case, in between the extremes $T = 0$ and ∞, is the more interesting situation. If $T = 1$, which represents the

$\varepsilon = 0$ SIS epidemic threshold around $\tau \to 0$, then

$$y = \frac{E}{2} \left\{ \sqrt{1 + \frac{4}{E}} - 1 \right\}$$

where $\lim_{E \to \infty} \frac{E}{2} \{\sqrt{1+\frac{4}{E}} - 1\} = 1$ and $\lim_{E \to 0} \frac{E}{2} \{\sqrt{1+\frac{4}{E}} - 1\} = 0$. Moreover, y is increasing in E with a derivative $\lim_{E \to 0} \frac{dy}{dE} = \infty$, while $\lim_{E \to \infty} \frac{dy}{dE} = 0$, again illustrating that around $\varepsilon \to 0$ the changes in the "size limit prevalence" are phenomenal and explosive.

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