Universality of the SIS prevalence in networks

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Abstract

Epidemic models are increasingly used in real-world networks to understand diffusion phenomena (such as the spread of diseases, emotions, innovations, failures) or the transport of information (such as news, memes in social on-line networks). A new analysis of the prevalence, the expected number of infected nodes in a network, is presented and physically interpreted. The analysis method is based on spectral decomposition and leads to a universal, analytic curve, that can bound the time-varying prevalence in any finite time interval. Moreover, that universal curve also applies to various types of Susceptible-Infected-Susceptible (SIS) (and Susceptible-Infected-Removed (SIR)) infection processes, with both homogenous and heterogeneous infection characteristics (curing and infection rates), in temporal and even disconnected graphs and in SIS processes with and without self-infections. The accuracy of the universal curve is comparable to that of well-established mean-field approximations.

1 Introduction to SIS epidemics on networks

Epidemic processes on a network can approximately describe an amazingly large variety of real-world processes [1], such as the spread of a disease, a digital virus, a message in an on-line social network, an emotion, the propagation of a failure or an innovation and other diffusion phenomena on networks (competing opinions, social contagion [2]). While the study of epidemics dates back to the great Bernoulli’s, the investigation of the role of the underlying graph on the dynamics of the susceptible-infected-susceptible (SIS) process was only initiated 15 years ago with the seminal paper of Pastor-Satorras and Vespignani [3]. The relatively new field of network science [4, 5, 6, 7, 8, 9] aims to study the interplay between dynamic processes on a graph and the characteristics of that underlying graph.

In [10], we discussed the “Local rule-Global emergent properties” (LrGep) class, where the collective action of the local rules executed at each node in the network gives rise to a complex, emergent global network behavior. Prominent examples of the LrGep-class are epidemic models and more general reaction-diffusion processes [11], the Ising spin model [12], the Kuramoto coupled-oscillator model [13], cellular automata [14], sandpiles as models for self-organized criticality [15, 16] and opinion models [17, 18]. The fascinating binding of these LrGep class members is that many LrGep models feature a phase transition [19], they all depend heavily on the underlying topology and many processes in nature seem well described by LrGep models. The simplicity of the local rules disguises the overwhelming
complexity of the global emergent network behavior that these local rules create. Even one of the simplest members of the LrGep class, the SIS process, is intricate and not sufficiently understood. However, the Markovian SIS process on networks allows for the highest degree of analytic treatment, which is a major motivation for the continued effort towards its satisfactory understanding. Here, we report on a universal property of the SIS prevalence and we propose a new analytic approximation with an accuracy comparable to the well-established mean-field models [1].

We consider an unweighted, undirected graph \( G \) containing a set \( \mathcal{N} \) of \( N \) nodes and a set \( \mathcal{L} \) of \( L \) links. The topology of the graph \( G \) is represented by a symmetric \( N \times N \) adjacency matrix \( A \). In an SIS epidemic process [20, 21, 22, 23, 11, 24], the viral state of a node \( i \) at time \( t \) is specified by a Bernoulli random variable \( X_i(t) \in \{0, 1\} \): \( X_i(t) = 0 \), when node \( i \) is healthy, but susceptible and \( X_i(t) = 1 \), when node \( i \) is infected. A node \( i \) can only be in one of these two states: infected, with probability \( \Pr[X_i(t) = 1] \) or healthy, with probability \( \Pr[X_i(t) = 0] \), but susceptible to the infection. We assume that the curing process for node \( i \) is a Poisson process with rate \( \delta_i \) and that the infection rate over the link \( (i, j) \) is a Poisson process with rate \( \beta_{ij} \). Only when node \( i \) is infected, it can infect each node \( k \) of its healthy direct neighbors with rate \( \beta_{ik} \). All Poisson curing and infection processes are independent. This description defines the continuous-time, Markovian heterogeneous SIS epidemic process on a graph \( G \). We do not consider non-Markovian epidemics [25, 26] and assume that the infection characteristics in the graph, i.e. all curing and infection rates, are independent of time. The fraction of infected nodes is defined as

\[
S(t) = \frac{1}{N} \sum_{i=1}^{N} X_i(t) \tag{1}
\]

and its expectation, called the prevalence or the order parameter, equals

\[
y(t) = E[S(t)] = \frac{1}{N} \sum_{i=1}^{N} \Pr[X_i(t) = 1] \tag{2}
\]

exploiting the property \( E[X_i] = \Pr[X_i = 1] \) of a Bernoulli distribution, which enables to avoid computations with the probability operator in favor of the easier, linear expectation operator. In that setting, the exact Markovian heterogeneous SIS governing equation [27] [8] for the infection probability of node \( i \) is

\[
\frac{dE[X_i(t)]}{dt} = E \left[ -\delta_i X_i(t) + (1-X_i(t)) \sum_{k=1}^{N} \beta_{ki} a_{ki} X_k(t) \right] \tag{3}
\]

When node \( i \) is infected at time \( t \) and \( X_i(t) = 1 \), only the first term on the right-hand side between the brackets \( [\cdot] \) affects and decreases with rate \( -\delta_i \) the change in infection probability with time \( \frac{d\Pr[X_i(t) = 1]}{dt} \) (left-hand side in [3]). When node \( i \) is healthy, \( X_i(t) = 0 \) and \( (1-X_i(t)) \) only the second term between the brackets \( [\cdot] \) increases \( \frac{d\Pr[X_i(t) = 1]}{dt} \) by a rate \( \sum_{k=1}^{N} \beta_{ki} a_{ki} X_k(t) \) due to all its infected, direct neighbors. We define the Bernoulli random vector \( w(t) = (X_1(t), X_2(t), \ldots, X_N(t)) \) at time \( t \), the nodal curing vector \( \tilde{\delta} = (\delta_1, \delta_2, \ldots, \delta_N) \) and the weighted adjacency matrix \( \tilde{A} \) with element \( \tilde{a}_{ij} = \beta_{ij} a_{ij} \), that can change with time \( t \) as in temporal networks [28]. If \( \beta_{ki} = \beta_{ik} \), the corresponding heterogeneous SIS prevalence differential equation is (see Theorem [1] in Appendix [A])

\[
N \frac{dy(t)}{dt} = -E \left[ \tilde{\delta}^T w(t) \right] + E \left[ (w(t))^T \tilde{Q}(t) w(t) \right] \tag{4}
\]
where the time-depending, weighted Laplacian $\tilde{Q}(t) = \tilde{\Delta}(t) - \tilde{A}(t)$ is an $N \times N$ positive semi-definite symmetric matrix, with the diagonal matrix $\tilde{\Delta}(t) = \text{diag}(\tilde{d}_1, \tilde{d}_2, \ldots, \tilde{d}_N)$ and the infection strength of node $k$ is $\tilde{d}_k = \sum_{i=1}^{N} \beta_{ki} a_{ki}$. In a homogeneous SIS process, where all $\beta_{ij} = \beta$ and $\delta_j = \delta$, (11) simplifies [29] to,

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

where $\tau = \frac{\beta}{\delta}$ is the effective infection rate, $t^* = t\delta$ is the normalized time, $Q = \Delta - A$ is the Laplacian of the graph $G$ with $\Delta = \text{diag}(d_1, d_2, \ldots, d_N)$ and $d_i$ is the degree of node $i$. The corresponding governing equation for the prevalence of the SIR process is deduced in [30]. Assume in a temporal network that the infection characteristics do not change, but only links in the graph change at time $t$: $A(t - \varepsilon) = A_1$ and $A(t + \varepsilon) = A_2$ for any arbitrarily small real $\varepsilon > 0$. Since the number $N$ of nodes does not change, the number of infected nodes is continuous at time $t$. Thus, the Bernoulli vector $w(t) = \lim_{\varepsilon \to 0} w(t - \varepsilon) = \lim_{\varepsilon \to 0} w(t + \varepsilon)$ is continuous at time $t$ and the prevalence differential equation (11) shows that

$$
\left. \frac{dy(t)}{dt} \right|_{t+\varepsilon} - \left. \frac{dy(t)}{dt} \right|_{t-\varepsilon} = \frac{1}{N} E \left[ (w(t))^T (\tilde{Q}_2 - \tilde{Q}_1) w(t) \right]
$$

implying that the derivative of the prevalence is likely not continuous at the time when the topology changes. On the other hand, the derivative $\frac{dy(t)}{dt}$ is continuous when the topology does not change (nor the infection characteristics). Thus, the SIS prevalence on temporal networks may show a discontinuous slope at time $t$, from which a topology change at that time $t$ may be inferred.

![Figure 1](image.png)

Figure 1: A sketch of an epidemic state in a graph at time $t^*$, described by (5), illustrates the three sets: (a) the set of infected nodes containing $NS(t^*) = 7$ nodes (in black), (b) the set of susceptible nodes (in green) and (c) the cut-set (in red): the number of links with one infected node and here equal to $w^T(t^*) Q w(t) = 6$ links.
2 The cut-set

The evolution of the nodal infection, described by (3), reflects the “local rule” of the SIS process, whereas the SIS prevalence differential equation (4) describes the “global emergent properties”. In (3), (4) and (5), the second, non-linear term quantifies the coupling between process and underlying topology. Fig. 1 illustrates that this physical interaction is embedded in the cut-set \((w(t))^T Q w(t)\), which equals the number of links with one end node infected at time \(t\). Given that one node is infected initially and that the effective infection rate \(\tau\) is well above the epidemic threshold \(\tau_c\), the early infection spreads as the ripples in a pool caused by throwing a stone in the water. First the direct neighbors become infected, then the neighbors of those neighbors and so on. This early spread can be specified by the expansion of the graph [8, p. 371], a graph metric which determines the number of nodes at \(k\) hops from the initial node. In this early phase, the epidemic grows exponentially with time and the cut-set boundary is analogous to “concentric circles in a pool” around the initially infected node [31]. After some time, infected nodes cure and move in Fig. 1 to the set of susceptible nodes. When the number of successive shells around the initial node exceeds the average hopcount [8, p. 360 & Chapter 16], i.e. number of links of the shortest path between two arbitrary nodes, the finite size of the graph prevents exponential increase in the number of nodes reached from an initial node. Hence, two effects, curing and finite graph structure, limit the growth of an epidemic. After the early phase, the cut-set as well as its border line between infected and healthy nodes cease to resemble simple geometric concentric circles and start exhibiting a complicated shape. Determining the largest cut-set, which corresponds to the fastest possible viral increase (see (11)) in the network, is NP-hard, as well as finding the smallest cut-size that is related [32, p. 95] to the isoperimetric constant \(\eta\), which upper bounds the epidemic threshold \(\tau_c \leq \frac{1}{\eta}\), as shown by Ganesh et al. [33]. In spite of its computational difficulty, the key to understanding an infectious spread lies in the cut-set, which is the place to prevent epidemic spread. The latest dynamic control strategies [34] target the reduction of the cut-set \((w(t))^T Q w(t)\) at each time \(t\).

3 Universality of the tanh-formula

Our major new result concerns “universality”: the time-varying prevalence \(y(t)\) of any Markovian SIS process, be it homogeneous or heterogeneous in its infection or/and curing rates, in temporal or even disconnected graphs, with or without self-infections, can be upper and lower bounded by a single, universal curve. To simplify the explanation, we concentrate on a homogeneous SIS process and refer to Appendices E and F for the other cases.

Our method, which is entirely different from the mean-field concept, is based on the spectral decomposition of the cut-set \((w(t))^T Q w(t)\) and of the Bernoulli state vector \(w\), whose components \(w_j = X_j\) are only zero or one. Physically, the dynamics (5) of the SIS epidemics, characterized by the Bernoulli vector \(w\) and the Laplacian matrix \(Q\), is mapped onto the Laplacian eigenspace, determined by the underlying graph \(G\). As shown in Appendix C, the Bernoulli vector \(w\) is projected onto the \(N\) orthogonal axes formed by the real, normalized Laplacian eigenvectors \(x_1, x_2, \ldots, x_N\) belonging to the eigenvalues \(\mu_1 \geq \mu_2 \geq \cdots \geq \mu_N = 0\), respectively, and obeying the orthogonality condition \(x_k^T x_m = 1\) if \(k = m\), otherwise \(x_k^T x_m = 0\). The coordinates in the Laplacian eigenvector basis, \(\zeta_j = w^T x_j\) for
1 ≤ j ≤ N, completely specify the Bernoulli vector \( w \). The relative success of the method is, in contrast to the adjacency matrix, due to the knowledge of one eigenvector \( x_N = \frac{1}{\sqrt{N}} u \) belonging to the eigenvalue \( \mu_N = 0 \) and where \( u = (1, 1, \ldots, 1) \) is the all-one vector. The corresponding coordinate is \( \zeta_N = \frac{1}{\sqrt{N}} u^T w = \sqrt{N} S \), by the definition (2) written in vector form as \( S = \frac{u^T w}{N} \). Since \( w \) is a zero-one vector, the largest scalar product \( \zeta_j = w^T x_j \) is \( \zeta_N \), which means that the Bernoulli vector \( w \) is most close to the eigenvector \( x_N \). In addition, the norm of the Bernoulli vector equals the sum of its components, \( w^T w = u^T w = NS \), which allows to specify the second most influential coordinate \( \zeta_{N-1} \).

We consider a graph \( G \) consisting of \( k \) connected components, where the \( k \) smallest Laplacian eigenvalues are zero, but \( \mu_{N-k} > 0 \) (see Appendix [13]). For a connected graph \( (k = 1) \), the second smallest eigenvalue \( \mu_{N-1} > 0 \) of the Laplacian \( Q \), coined by Fiedler [35] the algebraic connectivity, is studied over the last decades [32]. After spectral decomposition, the differential equation (5) becomes

\[
\frac{dy(t^*; \tau)}{dt^*} = (\tau \mu_{N-k} - 1) y(t^*; \tau) - \tau \mu_{N-k} y^2(t^*; \tau) - \Psi_k(t^*; \tau)
\]

where the remainder \( \Psi_k(t^*; \tau) \) is explicitly given in (21). If \( \Psi_k(t^*; \tau) \) equals a constant \( c \), then (7) reduces to a Riccati differential equation, which can be solved exactly. Assuming that we can bound \( \Psi_k(t^*; \tau) \) in a normalized time interval \([t_1^*, t_2^*]\) by two constants, \( c_L(k) \leq \Psi_k(t^*; \tau) \leq c_U(k) \), then the prevalence \( y(t^*) \) can be bounded in \([t_1^*, t_2^*]\), for the same initial condition \( y_0 \), by

\[
T(t^*|y_0, \mu_{N-k}, c_L(k)) \leq y(t^*) \leq T(t^*|y_0, \mu_{N-k}, c_U(k))
\]

where our “tanh-formula” is

\[
T(t|y_0, s, c) = \frac{1}{2} \left( 1 - \frac{1}{s} \right) + \frac{\Xi}{2} \tanh \left( \frac{s \Xi}{2} t + \Omega_0 \right)
\]

with the Laplacian normalized effective infection rate \( s = \mu_{N-k} \) and

\[
\Omega_0 = \arctanh \left( \frac{2y_0 - (1 - \frac{c}{s})}{\Xi} \right)
\]

and

\[
\Xi = \sqrt{\left( 1 - \frac{1}{s} \right)^2 - \frac{4c}{s}}
\]

Fig. [2] draws the tanh-formula (8) as a function of normalized time \( t^* \) for various \( c \in [-1, 0] \), in two characteristic regimes above \((\tau \mu_{N-k} \) high\) and below \((\tau \mu_{N-k} \) small\) the epidemic threshold.

We argue in Appendix [D] that, for \( k = 1 \), a rough estimate for \( c \approx -y_0 \). Our tanh-formula (8) approximates the total contribution \( \Psi_k(t^*; \tau) \), containing the less influential Bernoulli vector coordinates in the Laplacian eigenspace, by a constant \( c \). Extensive simulations [36] on various graph types and infection characteristics, compared with the N-Intertwined Mean-Field Approximation (NIMFA, [37]), demonstrate that the tanh-formula (8) has an overall performance comparable to mean-field approximations.
Figure 2: The tanh-formula (8) as a function of normalized time $t^*$ for various values of $c = \{-1, -0.9, -0.8, \ldots, 0\}$. Two characteristic regimes are shown: (above) $s = \tau \mu_{N-k}$ below the epidemic threshold and initial condition $y_0 = 1$, where all are nodes infected, and (below) $s = \tau \mu_{N-k}$ above the epidemic threshold and initial condition $y_0 = 0.1$.

4 Potential of the tanh-formula (8)

We discuss the tanh-formula (8) further. First, (8) contains three parameters: the initial condition $y_0$, $s = \tau \mu_{N-k}$ and $c$ that all depend upon the underlying graph. Remarkably, the expectation of a complicated dynamic process – the prevalence is an expectation – is approximately characterized by only three parameters. Second, the tanh-formula (8) generalizes the classical Kermack and McKendrick expression of 1927 by incorporating the graph. Assuming “homo geneous mixing”, equivalent to regarding the underlying network as a complete graph $K_N$, Kermack and McKendrick [38] demonstrated that the SIR prevalence is described by the time-derivative of a simplified variant of our tanh-formula (8). The correspondence with our tanh-formula (8) is not so surprising: in the complete graph $K_N$ with algebraic connectivity $\mu_{N-1} = N$, the complicated remainder reduces to its simplest possible form: $\Psi_1(t^*; \tau) = \tau N \text{Var}[S(t^*; \tau)]$. In $K_N$, the fraction of infected nodes $S(t^*; \tau)$ is close to a Gaussian random variable [39] above the epidemic threshold $\tau_c$ and the variance $\text{Var}[S(t^*; \tau)] \approx \frac{1}{N}$ is almost constant in the metastable regime. Generally, in sufficiently large networks and above the epidemic...
threshold, the average total infection “force” is balanced in equilibrium by the average total healing “force” and the individual infection state $X_j$ of node $j$ is only weakly dependent on $X_k$ of node $k$. Under these conditions of weakly dependence, the Central Limit Theorem states that the fraction $S$ of infected nodes tends to a Gaussian with mean $y = E[S]$ and standard deviation $\sigma = \sqrt{\text{Var}[S]}$. The tanh-formula does not include the eventual die-out of the SIS epidemic in any finite network.

Third, the parameter $c$ approximates the complicated remainder $\Psi_k(t^*; \tau)$. The important bounding assumption $c_L(k) \leq \Psi_k(t^*; \tau) \leq c_U(k)$ leads to the prevalence envelope, illustrated in Fig. 3 and akin to Fig. 4, which encloses (see also Fig. 4) roughly 68% of all realizations (i.e., all possible real-world measurements of an SIS epidemic) assuming Gaussian fluctuations around the prevalence – which is, as mentioned above, a good approximation for dense graphs as $K_N$ sufficiently above the epidemic threshold. In absence of sufficiently clean data of a real-word SIS prevalence, Fig. 4 plots 50 random realizations of $S(t^*)$ out of $10^6$ with the same infection characteristics and on the same graph as in Fig. 3. Since only the realizations that have reached the metastable state after a start with one initially infected, randomly chosen node, are observable, the prevalence is rescaled to $y = \frac{N_m y_m + N_{\text{cured}}}{N} = \frac{N_m}{N} y_m$, which practically means for large real-world networks that the SIS process can only be caused by a cascade of mainly curing events in succession, which is a very rare event. Consequently, once the process has reached the metastable state, the epidemic remains in the network for a very long time [40, 41, 42], which practically means for large real-world networks that the SIS epidemics remains in the metastable state. Hence, for large $N$ and for effective infection rates $\tau > \tau_c$, the tanh-formula models the “real” SIS epidemic very well, although it ignores absorption.

Figure 3: The prevalence envelope in a normalized time interval $[0, 50]$ for an instance of an Erdös-Rényi random graph $G_p(N)$ with $N = 50$ nodes, link density $p = 0.4$ and spectral radius $\lambda_1 = 20.8$. Initially, one random node was infected. The infection rate $\beta = 0.15$ and the curing rate $\delta = 1$, leading to a normalized effective infection rate $x = \frac{\lambda_1}{\tau_c} = \lambda_1 \tau = 3.12$. 

$y_m$
where the index \( m \) refers to those realizations that reach the metastable state, and \( d \) those that die out fast \[^{[44]}\] and never reach the metastable state. Usually, the die-out probability, given an initial number of infected nodes, is unknown, which complicates, as demonstrated in Fig. 4 the proper normalization in reality, where often only one realization of a spreading process (e.g. of a disease) is measured over time. Fortunately, NIMFA \[^{[29]}\] upper bounds the prevalence, ignoring that realizations die out, while the tanh-formula \[^{[8]}\] can fit, upper or lower bound data to infer from the parameters \((y_0, \tau_{\mu N-k}, c)\) insights in the epidemic.

![Figure 4: A random selection of 50 realizations out \( 10^6 \) realizations, shown in Fig. 3. Conditioned on 28 realization that did not die out, the prevalence (red) has been renormalized as well as the tanh-formula (8) upper (green) and lower bound (blue) in Fig. 3](image)

Fourth, the tanh-formula \[^{[8]}\] can be used in temporal networks by gluing the different time-regimes in which the network is unaltered: at time \( t \), where the topology changes, we impose continuity in the prevalence, \( y(t-\varepsilon) = y(t+\varepsilon) \) for \( \varepsilon \to 0 \), but allow discontinuity in the derivatives as in \[^{[6]}\]. The tanh-formula \[^{[8]}\] is the more accurate, the better \( \Psi_k(t^*; \tau) \) can be approximated by a constant. The smaller the time interval \([t_1^*, t_2^*]\), the better \( \Psi_k(t^*; \tau) \) is approximated by its mean \( c = \frac{1}{t_2^*-t_1^*} \int_{t_1^*}^{t_2^*} \Psi_k(t^*; \tau) \, dt^* \). By dividing an experiment in small time intervals, in which the data is fitted by the tanh-formula \[^{[8]}\], and by “continuously gluing” the intervals that determine \( y_0 \), a set \( \{\tau_{\mu N-k}, c\} \) for each interval is obtained. Theory prescribes that all \( \tau_{\mu N-k} \) over the intervals should hardly differ, which can serve as an accuracy indication or a verification that the epidemic process is Markovian SIS-like. The set of \( c \) values then approximates the non-constant remainder \( \Psi_k(t^*; \tau) \), that depends on both the epidemic process and the underlying graph.

Finally, just as higher order mean-field methods can increase the accuracy, our spectral approach can be improved. Instead of bounding the remainder \( \Psi_k(t^*; \tau) \) by a constant, which is the zero-order approximation in the Taylor expansion of \( \Psi_k(t^*; \tau) \), a polynomial in the prevalence \( y \) seems promising \[^{[36]}\], which suggests that the method may be refined further. Thus, we believe that it is worthwhile to research \( \Psi_k(t^*; \tau) \) in depth to find sharper approximations. Another extension towards more realistic
non-Poissonian infection and curing processes stands on the agenda for further research.

In summary, besides the powerful mean-field approximations, we have demonstrated the potential of a spectral method for the prevalence to unravel properties of SIS (SIR) epidemics on networks.

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A The basic differential equation of the SIS prevalence

**Theorem 1** Let \( \tilde{Q}(t) = \tilde{\Delta}(t) - \tilde{A}(t) \) denote the time-depending, weighted Laplacian, which is an \( N \times N \) positive semi-definite symmetric matrix, with the diagonal matrix \( \tilde{\Delta}(t) = \text{diag}(\tilde{d}_1, \tilde{d}_2, \ldots, \tilde{d}_N) \) and the infection strength of node \( k \) is \( \tilde{d}_k = \sum_{i=1}^{N} \beta_{ki} a_{ki} \). If the link infection rate is the same in both directions, \( \beta_{ki} = \beta_{ik} \), then the corresponding heterogeneous SIS prevalence differential equation is

\[
N \frac{dy(t)}{dt} = -E \left[ \delta^T w(t) \right] + E \left[ (w(t))^T \tilde{Q}(t) w(t) \right]
\]

If the link infection rate is not the same in both directions, \( \beta_{ki} \neq \beta_{ik} \), a Laplacian representation is not possible and we end up with

\[
N \frac{dy(t)}{dt} = -E \left[ \delta^T w(t) \right] + E \left[ (u - w(t))^T \tilde{A} w(t) \right]
\]

In a homogeneous SIS epidemic process, where all \( \beta_{ij} = \beta \) and \( \delta_j = \delta \), then \([7]\) simplifies to the differential equation,

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

**Proof:** Summing the Markovian heterogeneous SIS governing equation \([3]\) for the infection probability of node \( i \) over all nodes and omitting the time-dependence in \( X_i(t) \) to shorten the equations, yields

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

After rewriting in matrix notation and in terms of the prevalence \([2]\), we obtain

\[
N \frac{dy(t; \tau)}{dt} = -E \left[ \delta^T w(t) \right] + E \left[ (\tilde{A}u)^T w(t) - w(t)^T \tilde{A} w(t) \right]
= -E \left[ \delta^T w(t) \right] + E \left[ (u - w(t))^T \tilde{A} w(t) \right]
\]

We define the weighted Laplacian as \( \tilde{Q} = \tilde{\Delta} - \tilde{A} \), where the diagonal matrix \( \tilde{\Delta} = \text{diag}(\tilde{d}_1, \tilde{d}_2, \ldots, \tilde{d}_N) \) and the strength of node \( k \) is \( d_k = \sum_{i=1}^{N} \beta_{ki} a_{ki} \). In order to benefit from the basic Laplacian property \( \tilde{Q} u = 0 \) of a constant row and column sum, we confine ourselves to a symmetric weighted adjacency matrix \( \tilde{A} = (\tilde{A})^T \), implying that \( \beta_{ij} = \beta_{ji} \). Thus, the infection rate of a link is only link dependent and the same in both directions: from node \( i \) to node \( j \) and vice versa. Consequently, the weighted Laplacian is symmetric, \( \tilde{Q} = (\tilde{Q})^T \). Under this symmetry restriction, we have

\[
(u - w(t))^T \tilde{A} w(t) = (u - w(t))^T (\tilde{\Delta} - \tilde{Q}) w(t)
= (u - w(t))^T \tilde{\Delta} w(t) - u^T \tilde{Q} w(t) + (w(t))^T \tilde{Q} w(t)
= (w(t))^T \tilde{Q} w(t)
\]

because

\[
(u - w(t))^T \tilde{\Delta} w(t) = \sum_{j=1}^{N} (1 - X_j(t)) X_j(t) \tilde{d}_j = 0
\]
since \((1 - X_j(t)) X_j(t) = 0\) as \(X_j(t) \in \{0, 1\}\). The homogeneous case \((5)\), as discussed in \(29\), follows directly from \((1)\) with normalized time \(t^* = t\delta\).

Perhaps surprising, the exact governing equations \((1)\) and \((5)\) of the SIS prevalence are formally easier than their mean-field counterpart (see \(8\) p. 467). By using the basic definition \(Q = BB^T\) of the \(N \times N\) Laplacian \(32\) p. 14] in terms of the \(N \times L\) incidence matrix \(B\), we directly find \(32\) p. 72] that, for any \(N \times 1\) vector \(z\),

\[
z^T Q z = (B^T z)^T B^T z = \sum_{l \in L} (z_{l^+} - z_{l^-})^2
\]

where each link \(l \) joins two end nodes \(l^+\) and \(l^-\). In particular, for \(z = w\), we observe that

\[
w^T Q w = \sum_{l \in L} (X_{l^+} - X_{l^-})^2
\]

If both end of a link \(l\) are either infected or healthy, then \(X_{l^+} - X_{l^-} = 0\) and such a link \(l\) does not contribute to the sum. Hence, only links with one end infected and the other end healthy, for which \((X_{l^+} - X_{l^-})^2 = 1\), contribute precisely a unit amount to \(w^T Q w\). In other words, \(w^T Q w\) equals the number of links in the cut-set.

**B Kernel of the Laplacian \(Q\) of a graph with \(k\) disconnected components**

A graph \(G\) has \(k\) components (or clusters) if there exists a relabeling of the nodes such that the adjacency matrix has the structure

\[
A = \begin{bmatrix}
A_1 & O & \cdots & O \\
O & A_2 & \vdots \\
\vdots & \ddots \\
O & \cdots & A_k
\end{bmatrix}
\]

where the square submatrix \(A_m\) is the \(n_m \times n_m\) adjacency matrix of the connected component \(m\) containing \(n_m\) nodes. The total number of nodes in \(G\) equals \(N = \sum_{m=1}^{k} n_m\). The corresponding Laplacian is

\[
Q = \begin{bmatrix}
Q_1 & O & \cdots & O \\
O & Q_2 & \vdots \\
\vdots & \ddots \\
O & \cdots & Q_k
\end{bmatrix}
\]

Since \(Q_m u_{n_m} = 0\) for each connected component \(m\) and the (unscaled) \(n_m \times 1\) all-one eigenvector \(u_{n_m}\) is the only eigenvector belonging to eigenvalue \(\mu_{n_m} = 0\) (due to the connectivity of the connected component \(m\)), we find that the general representation of an eigenvector \(x_N (m)\) of \(Q\) belonging to the eigenvalue \(\mu_N = 0\) with multiplicity \(k\) is

\[
x_N^T (m) = (\eta_{m1} u_{n1}^T, \eta_{m2} u_{n2}^T, \ldots, \eta_{mk} u_{nk}^T)
\]
The subspace of the $N$-dimensional space spanned by the eigenvectors of a matrix belonging to the zero eigenvalue is called the kernel or null space of that matrix. Each of the $k$ possible $N \times 1$ eigenvectors $x_N(m)$ of $Q$ have the form \((\mathbf{12})\) and can thus be specified by a $k \times 1$ vector $\eta_m = (\eta_{m1}, \eta_{m2}, \ldots, \eta_{mk})$ for $1 \leq m \leq k$. Any pair \(\{x_N(m), x_N(s)\}\) of such (normalized) eigenvectors, represented by the vectors $\eta_m = (\eta_{m1}, \eta_{m2}, \ldots, \eta_{mk})$ and $\eta_s = (\eta_{s1}, \eta_{s2}, \ldots, \eta_{sk})$, must be orthogonal \((32)\), which leads to the set of \(\binom{k}{2}\) non-linear equations

$$x_N(m) x_N(s)^T = \sum_{j=1}^{k} \eta_{mj} \eta_{sj} n_j = \delta_{ms}$$

Let us define the vector $y_m = (y_{m1}, y_{m2}, \ldots, y_{mk})$ with $y_{mj} = \sqrt{m_j \eta_{mj}}$, then the above orthogonality condition reduces to the “ordinary” orthogonality condition for the set of $k$ vectors $y_1, y_2, \ldots, y_k$,

$$y_m^T y_s = \sum_{j=1}^{k} y_{mj} y_{sj} = \delta_{ms}$$

This means that any set of $k$ orthogonal vectors $y_1, y_2, \ldots, y_k$, that spans the $k$-dimensional space, can be used to produce $k$ eigenvectors $x_N(m)$, with $1 \leq m \leq k$, of the kernel of $Q$. The corresponding $k \times k$ matrix $Y_k$, with the vectors $y_1, y_2, \ldots, y_k$ in the columns, is an orthogonal matrix, whose properties with respect to graphs are studied in \([46]\). Clearly, the simplest set is the set $e_1, e_2, \ldots, e_k$ of the basis vectors for which $Y_k = I$. Since any other $N \times 1$ eigenvectors of $Q$ belonging to a positive eigenvalue of $Q$ is orthogonal to each kernel eigenvector $x_N(m)$ that belongs to the zero eigenvalue $\mu_N = 0$, we observe that there exist a $k$-fold infinity of such eigenvector sets, depending on our choice of the set of $k$ vectors $y_1, y_2, \ldots, y_k$.

Since the Bernoulli vector $w$ has only zero and one components, the scalar product $w^T x_N(m)$ is maximized if one the vectors $y_m$ is equal to the $k \times 1$ all-one vector $u_k$. This observation suggests us to construct the set of orthogonal eigenvectors $y_1, y_2, \ldots, y_k$, with one of them, say $y_1 = u_k$, equal to the all-one vector $u_k$. Basically, this means that all orthogonal vectors $y_1, y_2, \ldots, y_k$ are eigenvectors of the adjacency matrix of the complete graph $K_k$, because the unscaled largest adjacency eigenvector (of any regular graph and thus also of $K_k$) is $x_1(K_k) = u_k$.

Barik et al. \([17]\) have shown that only regular graphs, such as the complete graph $K_N$, for $N = 4k$ and $k \in \mathbb{N}_0$, and the regular bipartite graph $K_{2k,2k}$, are diagonalizable by a symmetric Hadamard matrix. An $n \times n$ Hadamard matrix $H_n$ contains as elements either $-1$ and $1$ and obeys $H_n H_n^T = n I_n$. The normalized matrix $\frac{1}{\sqrt{n}} H_n$ is an orthogonal matrix, from which it follows that $\det H_n = n^{n/2}$, which is maximal among all $n \times n$ matrices with elements in absolute value less than or equal to $1$, which includes all orthogonal matrices. Indeed, let $H_n = [u^T \bar{H}]$ so that $H_n e_1 = u_n$. Consider the diagonal matrix $D = I - e_1 e_1^T$, then

$$H_n D H_n^T = H_n H_n^T - H_n e_1 (H_n e_1)^T = n I_n - u_n u_n^T = n I - J$$

Hence, the Laplacian matrix of the complete graph $K_n$ is $Q K_n = n I - J = H_n D H_n^T$. Since $K_n$ is a regular graph, the eigenvectors of the Laplacian $Q$ and the adjacency matrix $A$ are the same \((32)\). In conclusion, any Hadamard matrix with first column $H_n e_1 = u_n$ provides the orthogonal eigenvector matrix for the complete graph $K_n$.  

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In summary, by choosing an $k \times k$ normalized Hadamard matrix $Y_k = \frac{1}{\sqrt{k}} H_k$ with first column $H_k e_1 = u_k$, all components $\eta_{mj} = \frac{y_{mj}}{\sqrt{n_j}}$ in (12) are determined, leading, with $(Y_k)_{mj} = \eta_{mj}$, to the matrix $Y_k = \text{diag} \left( \frac{1}{\sqrt{n_j}} \right) Y_k = \frac{1}{\sqrt{k}} \text{diag} \left( \frac{1}{\sqrt{n_j}} \right) H_k$. Moreover, the scalar product $w^T \xi$ is maximal among all normalized vectors $\xi$ for $\xi = x_N (1)$; in particular, $w^T x_N (1) = \frac{w^T u_k}{\sqrt{N}} = \frac{w^T u_k}{\sqrt{N}}$.

C The quadratic form $z^T Q z$ in a graph with $k$ disconnected components and its spectral decomposition

C.1 The vector $z$ is real

Since the eigenvectors of $Q$ constitute an orthogonal basis, any $N \times 1$ real vector $z$ can be expressed as a linear combination of eigenvectors $x_1, x_2, \ldots, x_N$ of $Q$,

$$z = \sum_{j=1}^N \alpha_j x_j$$

where $\alpha_j = z^T x_j$ and $x_j$ is the eigenvector belonging to the $j$-th largest Laplacian eigenvalue $\mu_j$. In terms of the orthogonal matrix $X$ with eigenvector in its columns $[46]$, which satisfies the orthogonality conditions $XX^T = X^T X = I$ so that $X^{-1} = X^T$, we have

$$\alpha = X^T z \text{ and } z = X \alpha$$

illustrating the one-to-one relation between the coordinates of $z$ expressed in a certain basis and its coordinates $\alpha$ expressed in the basis of eigenvectors of $Q$. The quadratic form equals

$$z^T Q z = \sum_{k=1}^N \sum_{m=1}^N \alpha_k \alpha_m x_k^T Q x_m = \sum_{k=1}^N \alpha_k^2 \mu_k$$

(13)

When the graph $G$ is disconnected into $k$ connected components (Appendix [33], there holds [32] p. 74] that $\mu_{N-j} = 0$ for $0 \leq j \leq k-1$. In other words, the $k$ smallest eigenvalues of the Laplacian $Q$ are zero, whereas all the others are positive $\mu_1 \geq \mu_2 \geq \ldots \geq \mu_{N-k} > 0$. Thus,

$$z^T z = \sum_{j=1}^N \alpha_j^2 = \sum_{j=1}^{N-k} \alpha_j^2 + \sum_{j=N-k+1}^N \alpha_j^2$$

Further, as shown in (12) above for $k > N - m$ and writing $z$ as a $k$-block vector,

$$z^T = (\tilde{z}_1^T, \tilde{z}_2^T, \ldots, \tilde{z}_k^T)$$

where $\tilde{z}_j$ is an $n_j \times 1$ vector corresponding to the block structure of the connected components in the adjacency matrix, the projection of the vector $z$ onto the kernel vectors $x_N (m)$ for $1 \leq m \leq k$ of the Laplacian $Q$ is

$$z^T x_N (m) = \sum_{j=1}^k \eta_{mj} \left( u_{mj}^T \tilde{z}_j \right)$$
so that
\[ \sum_{j=N-k+1}^{N} \alpha_j^2 = \sum_{m=1}^{k} \left( \sum_{j=1}^{k} \eta_{mj} (u_{nj}^T \tilde{z}_j) \right)^2 \]

In conclusion, for a graph \( G \) with \( k \) connected components, the Euclidean norm of the vector \( z \) can be written as
\[ z^T z = \frac{1}{N} (u^T z)^2 + \sum_{m=2}^{k} \left( \sum_{j=1}^{k} \eta_{mj} (u_{nj}^T \tilde{z}_j) \right)^2 + \sum_{j=1}^{N-k} \alpha_j^2 \]  
(14)

where each element \( \eta_{mj} \) of the matrix \( \Upsilon_k = \frac{1}{\sqrt{k}} \text{diag} \left( \frac{1}{\sqrt{n_j}} \right) H_k \) can be determined, as shown in Appendix B.

C.2 The Bernoulli random vector \( w \) is a binary vector

The Bernoulli vector \( w \) is a so-called binary vector, because each component \( w_k = X_k \) is either zero or one. For such vectors, we observe with (1) that
\[ w^T w = \sum_{k=1}^{N} X_k^2 = \sum_{k=1}^{N} X_k = u^T w = NS \]

Let us consider the eigenvector decomposition
\[ w = \sum_{j=1}^{N} \zeta_j x_j \]  
(15)

where \( \zeta_j = w^T x_j \) is the \( j \)-the coordinate of the Bernoulli vector \( w \) along the \( j \)-th eigenvector \( x_j \) in the eigenspace of \( Q \). For a graph \( G \) with \( k \) connected components, (14) leads to
\[ w^T w = \frac{1}{N} (u^T w)^2 + \sum_{m=2}^{k} \left( \sum_{j=1}^{k} \eta_{mj} (u_{nj}^T w_j) \right)^2 + \sum_{j=1}^{N-k} \zeta_j^2 \]
or, with \( w^T w = u^T w = NS \),
\[ \sum_{j=1}^{N-k} \zeta_j^2 = N (S - S^2) - \sum_{m=2}^{k} \left( \sum_{j=1}^{k} \eta_{mj} (u_{nj}^T w_j) \right)^2 \]  
(16)

Next, since \( \mu_{N-j} = 0 \) for \( 0 \leq j \leq k - 1 \), the quadratic form (13) becomes
\[ w^T Q w = \sum_{j=1}^{N-k} \zeta_j^2 \mu_j \]  
(17)

Introducing the square of the coordinate, obtained from (16),
\[ \zeta_{N-k}^2 = N (S - S^2) - \sum_{m=2}^{k} \left( \sum_{j=1}^{k} \eta_{mj} (u_{nj}^T w_j) \right)^2 - \sum_{j=1}^{N-k-1} \zeta_j^2 \]
Rewritten,
\[ w^T Q w = \mu_{N-k} N (S - S^2) + R_k \]
where the correction \( R_k \) is
\[ R_k = \sum_{j=1}^{N-k-1} \left( \frac{\zeta_j^2}{\nu_{j}} - \frac{\mu_{N-k}}{\nu_{j}} \right) \sum_{m=2}^{k} \left( \sum_{j=1}^{k} \eta_{mj} \left( u_{nj} x_{\tilde{w}_j} \right) \right)^2 \]
where \( \tilde{w}_j \) here is the \( j \)-th \( n_j \times 1 \) block vector of \( w \) according to the component structure of the graph \( G \). Thus, \( u_{nj} \tilde{w}_j \) equals the number of infected nodes at time \( t \) in the \( j \)-th connected component of \( G \) with \( n_j \) nodes and, with \( N = \sum_{m=1}^{k} n_m \), the fraction \( S \) of infected nodes in \( G \) (at time \( t \)) is a “weighted” average over the \( k \) components of \( G \)
\[ S = \frac{\sum_{j=1}^{k} u_{nj} \tilde{w}_j}{\sum_{m=1}^{k} n_m} \]

The first term in \( R_k \) is non-negative, as well as the second term. In a connected graph \( G \) where \( k = 1 \), the second term in (19) vanishes so that \( R_1 \) is non-negative, but only zero for the complete graph \( K_N \). When \( k = 1 \), the general expression \( R_k \) in (19) reduces to our previous expression in [29] in terms of the algebraic connectivity \( \mu_{N-1} \). Clearly, the second term in (19) only appears when a graph is disconnected into \( k \) connected components. When \( k \) is large, then \( R_k \) is likely negative, and certainly for \( k = N - 1 \), in which case the first in (19) term vanishes.

For a given graph \( G \), all parameters related its Laplacian eigenstructure, such as the eigenvalues \( \mu_j \) and the elements \( \eta_{mj} \) of the Hadamard related matrix \( \Upsilon_k = \frac{1}{\nu_{j}} \text{diag} \left( \frac{1}{\sqrt{n_j}} \right) H_k \), are known. Only the coordinates \( \zeta_j \) for \( 1 \leq j \leq N-k \) and kernel coordinates \( u_{nj} x_{\tilde{w}_j} \) for \( 1 \leq j \leq k \) in (19) depend on the SIS process via the Bernoulli vector \( w(t) \), that depends upon its initial value at \( w(0) \) at time \( t = 0 \). Indeed, if the initially infected nodes only appear in one component, say \( m = 1 \), so that the vector \( \tilde{w}_1 (0) \neq 0 \), then all other vectors \( \tilde{w}_m = 0 \) for all components \( m > 1 \), because evidently, an infection can only spread in a connected component.

C.3 A Fourier analysis interpretation of Laplacian eigenvectors

If \( x_k \) is an eigenvector of \( Q \) belonging to eigenvector \( \mu_k \), then the fundamental Laplacian quadratic form in (11) becomes
\[ \mu_k = x_k Q x_k = \sum_{l \in \mathcal{L}} \left( (x_k)_{l+} - (x_k)_{l-} \right)^2 \]
which implies that the variation of eigenvector components at both ends of a link increases with the Laplacian eigenvalue. When interpreting the eigenvector \( x_k \) as a function \( (x_k)_i \) of the nodal components
i at frequency $\mu_k$, the above suggests that a high frequency Laplacian eigenvector oscillates more (over a link) than a low frequency Laplacian eigenvector. The suggestion is correct for a ring graph \[32\] p. 116-123, because the orthogonal eigenvector matrix $X$ of the ring graph is the Fourier matrix (with the usual cosine and sine as eigenfunctions). While a general theorem valid for any graph that the Laplacian eigenvector $x_k$ possesses more sign changes with increasing $\mu_k$ – a reflection of higher oscillatory behavior with increasing frequency – seems missing\[1\] the intuition of the Fourier decomposition of a signal hints that the “Fourier coefficients” $\zeta_k = \mathbf{w}^T x_k$ are generally expected to decrease with higher index $k$. If correct and if $\zeta_k^2 \mu_k$ decreases generally with $\mu_k$, then the first sum of $R_k$ in \[19\] would generally consists of decreasing positive terms. This interpretation may lead to sharp approximations of $R_k$.

D  Governing equation of the homogeneous SIS prevalence in graph with $k$ disconnected components

Invoking the definition \[2\] of the prevalence $y = E[S]$, $E[S - S^2] = y - E[S^2]$ and $E[S^2] = y^2 + \text{Var}[S]$, \[18\] becomes

$$\frac{1}{N} E[w^T Q w] = \mu_{N-k} E[S] - \mu_{N-k} E[S^2] + \frac{E[R_k]}{N}$$

$$= \mu_{N-k} y - \mu_{N-k} y^2 - \mu_{N-k} \left( \text{Var}[S] - \frac{E[R_k]}{N \mu_{N-k}} \right)$$

(20)

Using \[5\], the spectral representation of the SIS prevalence governing differential equation is

$$\frac{dy(t^*; \tau)}{dt^*} = (\tau \mu_{N-k} - 1) y(t^*; \tau) - \tau \mu_{N-k} y^2(t^*; \tau) - \Psi_k(t^*; \tau)$$

where the remainder

$$\Psi_k(t^*; \tau) = \tau \mu_{N-k} \left( \text{Var}[S(t^*; \tau)] - \frac{E[R_k(t^*; \tau)]}{N \mu_{N-k}} \right)$$

(21)

For a connected graph (i.e. with $k = 1$ connected component), we again find the earlier result in \[29\] eq. (12)]. Introducing \[19\], the explicit form of the remainder is

$$\frac{\Psi_k(t^*; \tau)}{\tau \mu_{N-k}} = \text{Var}[S(t^*; \tau)] + \frac{1}{N} \sum_{m=2}^{\infty} \left( \sum_{j=1}^{k} \eta_{mj} \left( u^T w_j \right) \right)^2 - \frac{1}{N} \sum_{j=1}^{N-k-1} \left( \mu_j \mu_{N-k} - 1 \right) E[\zeta_j^2]$$

(21)

illustrating that $\Psi_k(t^*; \tau)$ is likely positive for large $k$, i.e. in a graph with many connected components.

For small time, the Taylor expansion yields

$$y(t^*; \tau) = y_0(t^*; \tau) + \left. \frac{dy(t^*; \tau)}{dt^*} \right|_{t^*=0} t^* + O(t^{*2})$$

Invoking the differential equation \[7\] and $y(0; \tau) = y_0$ leads to

$$y(t^*; \tau) = y_0 + \left( y_0 (\tau \mu_{N-k} - 1) - y_0^2 \mu_{N-k} - \Psi_k(0; \tau) \right) t^* + O(t^{*2})$$

\[1\] Any symmetric matrix can be reduced by orthogonal Householder reflections to a tri-band matrix, whose eigenvector structure consists of orthogonal polynomials and is computed in \[8\] p. 565-573, \[32\].
where \( \Psi_1(0; \tau) \) is small (because \( \text{Var}[S(0; \tau)] = 0 \) since \( S(0) \) is deterministic). The tanh-approximation \( 5 \), on the other hand, replaces \( \Psi_k(t^*; \tau) \) by \( c \) (in any time interval) so that

\[
T(t^* | y_0, \tau \mu_{N-k}, c) = y_0 + y_0 \left\{ \tau \mu_{N-k}(1 - y_0) - \left( 1 + \frac{c}{y_0} \right) \right\} t^* + O(t^{*2})
\]

The initial slope is non-negative when \( \tau \geq \frac{1}{\mu_{N-k}} \left( \frac{1 + \frac{c}{y_0}}{1 - y_0} \right) \). For a connected graph \( (k = 1) \), the tanh-prevalence \( T(t^* | y_0, \tau \mu_{N-1}, c) \) increases for \( \tau > \tau_c \) for all time \( t^* \geq 0 \) and, hence,

\[
1 - \frac{1}{\mu_{N-1}} \left( 1 + \frac{c}{y_0} \right) < \tau_c
\]

or

\[
c > y_0 \tau_c \mu_{N-1} (1 - y_0) - y_0 \geq y_0 \frac{\mu_{N-1}}{\lambda_1} (1 - y_0) - y_0
\]

because the lower bound for the epidemic threshold obeys \( \tau_c \geq \frac{1}{\lambda_1} \) (see e.g. \[1, 29\]). Since \( \mu_{N-1} < \lambda_1 \) (except for the complete graph and regular multipartite graphs), the positive-slope condition would suggest that \( c \gg -y_0 \) for almost all graphs. The value \( c \approx -y_0 \) is also approximately deduced from the Kermack and McKendrick \[38\] analysis for SIR. Simulations \[36\] seem to agree roughly with \( c \approx -y_0 \).

For large time, the tanh-formula \( 5 \) reduces to

\[
\lim_{t^* \to \infty} T(t^* | y_0, \tau \mu_{N-k}, c) = \frac{1}{2} \left( 1 - \frac{1}{\tau \mu_{N-k}} \right) + \frac{1}{2} \sqrt{\left( 1 - \frac{1}{\tau \mu_{N-k}} \right)^2 - \frac{4c}{\tau \mu_{N-k}}} = \frac{1}{2} \left( 1 - \frac{1}{\tau \mu_{N-k}} \right) + \frac{1}{2} \sqrt{\left( 1 - \frac{1}{\tau \mu_{N-k}} \right)^2 - \frac{4c}{\tau \mu_{N-k}}}
\]

which corresponds to the metastable state of the SIS process. For some extremal values of \( c \), \( 22 \) shows that

\[
\lim_{t^* \to \infty} T(t^* | y_0, \tau \mu_{N-k}, -1) = 0
\]

\[
\lim_{t^* \to \infty} T(t^* | y_0, \tau \mu_{N-k}, 0) = \begin{cases} 1 - \frac{1}{\tau \mu_{N-k}} & \tau > \frac{1}{\mu_{N-k}} \\ 0 & \tau < \frac{1}{\mu_{N-k}} \end{cases}
\]

For \( c_{\text{real}} = \frac{\tau \mu_{N-k}}{1} \left( 1 - \frac{1}{\tau \mu_{N-k}} \right)^2 \geq 0 \), that guarantees a real prevalence \( (\Xi = 0 \text{ in } 10) \), the prevalence does not depend on time any more and \( T(t^* | y_0, \tau \mu_{N-k}, c_{\text{real}}) = \frac{1}{2} \left( 1 - \frac{1}{\tau \mu_{N-k}} \right) \). Moreover, for \( \tau < \frac{1}{\mu_{N-k}} \), positive \( c > 0 \) are not physical since the prevalence can become negative. For \( \tau > \frac{1}{\mu_{N-k}} \), a positive \( c < c_{\text{real}} \) can be possible.

Similarly to \[29\] for the case \( k = 1 \) of a connected graph, \( T(t^* | y_0, s, c) \) in \[8\] obeys the Riccati differential equation

\[
\frac{dT}{dt^*} = (s - 1) T - s T^2 - c
\]

For the same initial prevalence \( y(0) = \tilde{y}_k(0) = y_0 \) and given the constants \( c_L(k) \) and \( c_H(k) \), that satisfy \( c_L(k) \leq \Psi_k(t^*; \tau) \leq c_H(k) \), the prevalence \( y(t^*) \) at normalized time \( t^* \) is bounded by the relatively simple expression \[8\]

\[
\begin{cases}
  y(t^*) \geq T(t^* | y_0, \tau \mu_{N-k}, c_L(k)) & \text{if } \Psi_k(t^*; \tau) \leq c_L(k) \text{ for } t^* \in [t_1^*, t_2^*] \\
y(t^*) \leq T(t^* | y_0, \tau \mu_{N-k}, c_U(k)) & \text{if } \Psi_k(t^*; \tau) \geq c_U(k) \text{ for } t^* \in [t_1^*, t_2^*]
\end{cases}
\]


The upper bound $\Psi_k (t^*; \tau) \leq c_U (k)$ implies that $y (t^*) \geq T (t^* \| y_0, \tau \mu_{N-k}, c_U (k))$ and $T (t^* \| y_0, \tau \mu_{N-k}, c_U (k))$ is real if the discriminant in (10) is positive,
\[
\left( 1 - \frac{1}{\tau \mu_{N-k}} \right)^2 \geq \frac{4c}{\tau \mu_{N-k}} \geq 4\Psi_k (t^*; \tau) = 4 \left( \text{Var} [S (t^*; \tau)] - \frac{E [R_k (t^*; \tau)]}{N \mu_{N-k}} \right).
\]
If $\text{Var} [S (t^*; \tau)] \geq \frac{E [R_k (t^*; \tau)]}{N \mu_{N-k}}$ (i.e. $c_L (k) \geq 0$), then the inequality is equivalent to the lower bound for the effective infection rate
\[
\tau \geq \frac{1}{\mu_{N-k}} \left( 1 - 2 \sqrt{\text{Var} [S (t^*; \tau)] - \frac{E [R_k (t^*; \tau)]}{N \mu_{N-k}}} \right) \geq \frac{1}{\mu_{N-k}}
\] (23)
that guarantees to operate in the endemic regime when $t^*$ is sufficiently large. We observe that, the more connected components a graph on $N$ nodes has, the larger $k$ and $\mu_{N-k}$ and, consequently, the lower $\frac{1}{\mu_{N-k}}$. Physically, the larger $k$, the fewer nodes a connected component has (on average $k/N$) and the larger the epidemic threshold of a connected component should become, because $\tau_c > \tau_c^{(1)} = \frac{1}{\lambda_1}$ increases with decreasing $\lambda_1$ and the spectral radius $\lambda_1 \geq E [D]$, which, in dense graphs, increases with $N$ on average. Hence, we expect that the epidemic threshold in a graph with $k$ connected components increases and (23) would imply that $\text{Var} [S (t^*; \tau)] - \frac{E [R_k (t^*; \tau)]}{N \mu_{N-k}} \to \frac{1}{4}$ for sufficiently large $t^*$.

In summary, the analysis generalizes the previous derivations in [29] to graphs with $k$ connected components, as e.g. in temporal networks. We can thus conclude that, for any graph $G$ with $k$ connected components with a fixed topology in some non-zero time interval, the prevalence $y (t^*)$ in that time interval can be bounded by the curve $T (t^* \| y_0, s, c)$ in (8) with three parameters: (a) the initial condition $y_0$ or value of the prevalence at the beginning of the time interval, (b) a Laplacian normalized rate $s = \tau \mu_{N-k}$ and (c) a constant $c$. Implicitly, the computation of the prevalence also assumes that the number $N$ of nodes in the graph $G$ is known. The prevalence is only non-zero when the effective infection rate $\tau$ exceeds the epidemic threshold $\tau_c$. Moreover, it is known that $\tau > \tau_c > \tau_c^{(1)}$, where the NIMFA threshold $\tau_c^{(1)} = \frac{1}{\lambda_1}$ and $\lambda_1$ is the largest eigenvalue of the adjacency matrix of the graph $G$. Hence, the adjacency normalized effective infection rate $x = \lambda_1 \tau$ allows us to compare epidemics in different graphs for sufficiently long time: when $x \leq 1$, the epidemic will die out, whereas $x > 1 + \varepsilon$ with $\varepsilon$ a correction due to the mean-field approximation, the epidemic will be persistent. However, the correction $\varepsilon$ is unknown. On the other hand, (23) tells us that, when the Laplacian normalized rate $s = \tau \mu_{N-k} \geq \xi$, where $\xi = \frac{1}{1 - 2 \sqrt{\text{Var} [S (t^*; \tau)] - \frac{E [R_k (t^*; \tau)]}{N \mu_{N-k}}}} > 1$, we are surely in the endemic regime where the prevalence $y (t^*) > 0$ (for a sufficiently large $t^*$). Unfortunately, computing $\xi$ is difficult, so that determining which effective infection rate $\tau$ leads to persistent infections, is complicated.

In conclusion, an accurate determination of the SIS epidemics threshold regime will likely stay on the scientific agenda for future research.

E Governing equation of the heterogeneous SIS prevalence in graph with $k$ disconnected components

The expression (18) is valid for the weighted Laplacian $\overline{Q}$ with eigenvectors $\overline{x}_1, \overline{x}_2, \ldots, \overline{x}_N = u$ belonging to eigenvalues $\overline{\mu}_1 \geq \overline{\mu}_2 \geq \ldots \geq \overline{\mu}_N = 0$, respectively and with the scalar product $\overline{\zeta}_k = w^T \overline{x}_k$,
since the kernel space of $\tilde{Q}$ is the same as that of the unweighted Laplacian $Q$. Invoking the definition of the prevalence $y = E[S]$, $E[S - S^2] = y - E[S^2]$ and $E[S^2] = y^2 + \Var[S]$, (18) becomes

$$\frac{1}{N}E\left[w^T \tilde{Q} w\right] = \tilde{\mu}_{N-k}y - \tilde{\mu}_{N-k}y^2 - \tilde{\mu}_{N-k} \left(\Var[S] - E[\tilde{R}_k] \frac{E[\tilde{R}_k]}{N \tilde{\mu}_{N-k}}\right)$$

Introduced into (14), which we write as,

$$\frac{dy(t; \tau)}{dt} = -\frac{1}{N}E\left[ (\delta_{av}u + \tilde{\delta} - \delta_{av}u)^T w(t) \right] + \frac{1}{N}E\left[ (w(t))^T \tilde{Q} w(t) \right]$$

$$= -\delta_{av}y - \frac{1}{N}E\left[(\tilde{\delta} - \delta_{av}u^T) w(t) \right] + \tilde{\mu}_{N-k}y - \tilde{\mu}_{N-k}y^2 - \tilde{\mu}_{N-k} \left(\Var[S] - E[\tilde{R}_k] - E\left[(\tilde{\delta} - \delta_{av}u^T) w(t) \right]\right)$$

where $\left(\tilde{\delta} - \delta_{av}u^T\right) w(t)$ is now an additional correction due to heterogeneous, node-depending curing rates. Normalizing the time $\tilde{t} = \delta_{av}t$ with respect to average curing rate $\delta_{av} = \frac{\tilde{\delta}u}{N}$ and realizing that the eigenvalues of the weighted Laplacian are function of the heterogeneous infection rates $\beta_{ij}$, we have

$$\frac{dy(\tilde{t}; \tau)}{d\tilde{t}} = (\frac{\tilde{\mu}_{N-k}}{\delta_{av}} - 1) y - \frac{\tilde{\mu}_{N-k}}{\delta_{av}}y^2 - \tilde{\Psi}_k(\tilde{t}; \tau)$$

where the remainder is

$$\tilde{\Psi}_k(\tilde{t}; \tau) = \frac{\tilde{\mu}_{N-k}}{\delta_{av}} \left(\Var[S] - E[\tilde{R}_k] - E\left[(\tilde{\delta} - \delta_{av}u^T) w(t) \right]\right)$$

In summary, the spectral representation of the heterogeneous SIS prevalence governing differential equation in a graph with $k$ connected components is

$$\frac{dy(t^*; \tau)}{d\tilde{t}} = (\frac{\tilde{\mu}_{N-k}}{\delta_{av}} - 1) y - \frac{\tilde{\mu}_{N-k}}{\delta_{av}}y^2 - \tilde{\Psi}_k(t^*; \tau)$$

Just as in the homogeneous case (Appendix D), we may proceed by a bounding procedure to find that the heterogeneous SIS prevalence also can be bounded by a tanh-expression of the form (8), though with different coefficients and an even more complicated $\tilde{\Psi}_k(t^*; \tau)$.

### F Governing equation of the $\varepsilon$–SIS prevalence in graph with $k$ disconnected components

The differential equation for the average fraction of infected nodes $y$ in the $\varepsilon$–SIS process is [8, p. 455]

$$\frac{dy(t^*; \tau)}{d\tilde{t}} = \varepsilon^* - (1 + \varepsilon^*) y(t^*; \tau) + \frac{\tau}{N}E\left[w^T (t^*; \tau) Q w (t^*; \tau) \right]$$

(24)

where $\varepsilon$ is the constant self-infection rate for each node [18]. If $\tau = 0$, the differential equation (24) for the $\varepsilon$-SIS prevalence shows that

$$\frac{dy(t^*; 0, \varepsilon^*)}{d\tilde{t}} = \varepsilon^* - (1 + \varepsilon^*) y(t^*; 0, \varepsilon^*)$$

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with solution
\[
y(t^*; 0, \varepsilon^*) = \frac{\varepsilon^*}{1 + \varepsilon^*} + \left(y_0 - \frac{\varepsilon^*}{1 + \varepsilon^*}\right)e^{-(1+\varepsilon^*)t}
\]

As in previous Section [12] after using \( y = E[S], E[S - S^2] = y - E[S^2] \) and \( E[S^2] = y^2 + \text{Var}[S] \) and (13), the differential equation (24) of the \( \varepsilon \)-SIS prevalence becomes
\[
\frac{dy(t^*; \tau, \varepsilon^*)}{dt^*} = (\tau\mu_{N-k} - (1 + \varepsilon^*))y(t^*; \tau, \varepsilon^*) - \tau\mu_{N-k}y^2(t^*; \tau, \varepsilon^*) - \Psi_k(t^*; \tau, \varepsilon^*)
\]
where
\[
\Psi_k(t^*; \tau, \varepsilon^*) = \tau\mu_{N-k}\left(\text{Var}[S(t^*; \tau)] - \frac{E[R_k(t^*; \tau)]}{N\mu_{N-k}}\right) - \varepsilon^*
\]
(25)

Again, by bounding \( c_L (k; \varepsilon^*) \leq \Psi_k(t^*; \tau, \varepsilon^*) \leq c_U (k; \varepsilon^*) \), a variant of the tanh-formula (8) applies
\[
\tilde{T}(t^*| y_0, s, c; \varepsilon^*) = \frac{1}{2} \left(1 - \frac{1 + \varepsilon^*}{s}\right) + \frac{\Upsilon}{2} \tanh\left(\frac{s\Upsilon}{2} t^* + \text{arctanh}\left(\frac{2y_0 - (1+\varepsilon^*)}{\Upsilon}\right)\right)
\]
(26)
where \( s = \tau\mu_{N-k} \) and
\[
\Upsilon = \sqrt{\left(1 - \frac{1 + \varepsilon^*}{s}\right)^2 - 4c/s}
\]
which clearly reduces to (26) for \( \varepsilon^* = 0 \).

F.1 Extremal values of the \( \varepsilon \)-SIS prevalence

When the prevalence attains an extremum \( y(p; \tau, \varepsilon^*) \) at time \( t^* = p \), obeying \( \frac{dy(t^*; \tau, \varepsilon^*)}{dt^*} \bigg|_{t^* = p} = 0 \), then
\[
\tau\mu_{N-k}y^2(p; \tau, \varepsilon^*) - (\tau\mu_{N-k} - (1 + \varepsilon^*)) y(p; \tau, \varepsilon^*) + \Psi_k(p; \tau, \varepsilon^*) = 0
\]

There are only real solutions for the “time-extremal” prevalence,
\[
y_{\pm}(p; \tau, \varepsilon^*) = \frac{(\tau\mu_{N-k} - (1 + \varepsilon^*)) \pm \sqrt{(\tau\mu_{N-k} - (1 + \varepsilon^*))^2 - 4\tau\mu_{N-k}\Psi_k(p; \tau, \varepsilon^*)}}{2\tau\mu_{N-k}}
\]
and
\[
y_{\pm}(p; \tau, \varepsilon^*) = \left(1 - \frac{1 + \varepsilon^*}{\tau\mu_{N-k}}\right) \frac{1}{2} \left\{1 \pm \sqrt{1 - \frac{\Psi_k(p; \tau, \varepsilon^*)}{\tau\mu_{N-k}}}\right\}
\]
(27)
provided the discriminant \( (\tau\mu_{N-k} - (1 + \varepsilon^*))^2 - 4\tau\mu_{N-k}\Psi_k(p; \tau, \varepsilon^*) \geq 0 \), which is equivalent to
\[
\frac{1}{4} \left(1 - \frac{1 + \varepsilon^*}{\tau\mu_{N-k}}\right)^2 \geq \frac{\Psi_k(p; \tau, \varepsilon^*)}{\tau\mu_{N-k}} = \text{Var}[S(p; \tau)] - \frac{E[R_k(p; \tau)]}{N\mu_{N-k}} - \frac{\varepsilon^*}{\tau\mu_{N-k}}
\]

This inequality leads to a lower bound for the effective infection rate,
\[
\tau_{\varepsilon^*} \geq \frac{1}{\mu_{N-k} - 2\sqrt{\text{Var}[S(p; \tau)] - \frac{E[R_k(p; \tau)]}{N\mu_{N-k}} - \frac{\varepsilon^*}{\tau\mu_{N-k}}}} > \tau(0)
\]
For small self-infection rates \( \varepsilon^* \), we can demonstrate the last inequality, which implies that the effective infection rate to guarantee an endemic regime lies higher for the \( \varepsilon \)-SIS model than for the classical
(ε = 0) SIS-model, which is consistent with the analysis in [8, p. 457-458]. Although surprising at first glance, we need to realize that the steady state in the ε = 0 SIS-model is the overall healthy state, to which the ε-SIS must converge if ε → 0, irrespective of the effective infection rate τ. The existence of an absorbing state implies in a finite graph that the epidemics eventually dies out (i.e. the dynamic process will surely hit the absorbing state in the 2^N large Markov state graph). This peculiar limit ε → 0 is further illustrated in [48, Fig. 5 and 6].

F.2 The absorbing state

The "time-extremal" prevalence y± (p; τ, ε∗) at time t∗ = p in (27) can only be zero if (a) the negative sign applies and (b) Ψk (p; τ, ε∗) = 0, in which case y− (p; τ, ε∗) = 0 for all effective infection rates τ. However, if y± (p; τ, ε∗) = 0, then S(p) = 0, which implies that Bernoulli vector w(p) = 0. The definition (25) of the remainder Ψk (p; τ, ε∗) and the specific expression (19) for the spectral correction Rk illustrate that Ψk (p; τ, ε∗) = −ε∗ if S(p) = 0. Consequently, y− (p; τ, ε∗) = 0 and Ψk (p; τ, ε∗) = 0 can only be satisfied if ε∗ = 0, thus only in the classical SIS process. This singular condition, y− (p; τ, ε∗) = 0 and Ψk (p; τ, ε∗) = 0, which holds irrespective of the effective infection rate τ, corresponds to the absorbing state which is attained at time p. If ε∗ > 0, there cannot be an absorbing state and the negative sign solution y− (p; τ, ε∗) in (27), which is decreasing in τ, does not exist. Moreover, Markov theory [8] states the ε-SIS Markovian chain possesses a unique steady-state, which corresponds to y+ (p; τ, ε∗) in (27). When bounding Ψk (t∗; τ, ε∗) ≥ cL so as to prevent that Ψk (p; τ, ε∗) → 0, then the limit ε∗ → 0 in (27) will correspond to the metastable state of the SIS process,

\[
y± (p; τ, 0) = \frac{1}{2} \left( 1 - \frac{1}{τμN−k} \right) \left\{ 1 + \sqrt{1 - \frac{cL}{τμN−k}} \right\}
\]

which is precisely equal to the tanh-formula’s steady-state (22).