

Solving the Dynamic Correlation Problem of the Susceptible-Infected-Susceptible Model on Networks

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The susceptible-infected-susceptible (SIS) model is a canonical model for emerging disease outbreaks. Such outbreaks are naturally modeled as taking place on networks. A theoretical challenge in network epidemiology is the dynamic correlations coming from that if one node is infected, then its neighbors are likely to be infected. By combining two theoretical approaches—the heterogeneous mean-field theory and the effective degree method—we are able to include these correlations in an analytical solution of the SIS model. We derive accurate expressions for the average prevalence (fraction of infected) and epidemic threshold. We also discuss how to generalize the approach to a larger class of stochastic population models.

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The susceptible-infected-susceptible (SIS) model is a fundamental model of outbreaks of diseases (like influenza, chlamydia, gonorrhoea, etc.) that does not give immunity upon recovery. Diseases spread over networks of people and the structure of these networks affect spreading [1]; thus, it makes sense to put the SIS model on networks. The SIS model divides the population into two classes—susceptible (S) and infected (I). Links between S and I transmit the disease (i.e., make the susceptible infected) with a rate r . Infected individuals become susceptible again with a rate g . One can reduce these two parameters to one— $\lambda = r/g$ —the rate of new infections per SI link per recovery, or the *effective infection rate*. In the thermodynamic limit $N \rightarrow \infty$, there can be a threshold, or phase-transition phenomenon when tuning λ [2]. For λ less than a critical λ_c the disease dies out spontaneously. If $\lambda > \lambda_c$ the disease will live forever—i.e., it has reached an *endemic* state where the *prevalence* ρ (fraction of infected nodes) is nonzero. Also, for finite networks, there is effectively an endemic state as the expected extinction time, even for small networks, grows extremely fast beyond λ_c [3]. The two main directions in the literature are to study extinction times in finite, homogeneous networks [4], or how the threshold depends on the network structure [5,6]. This work belongs to the latter class.

For the SIS model in a well-mixed population, the threshold happens at $\lambda_c = 1$ [7]. For the SIS model in homogeneous networks, such as the Erdős-Rényi random networks and random regular graphs, a simple approximative (mean-field) analysis gives the threshold $\lambda_c = 1/\langle k \rangle$ [5]. For the SIS model in scale-free networks [8]—where the degree distribution (the probability that a random node is connected to k other nodes) follows $P(k) \sim k^{-\gamma}$ —heterogeneous mean-field theory predicts that the epidemic threshold of the SIS model is equal to that of the susceptible-infected-recovered (SIR) model $\lambda_c^{\text{HMF,SIS}} = \lambda_c^{\text{HMF,SIR}} = \langle k \rangle / \langle k^2 \rangle$ [5,6,9,10]. In other words,

the threshold seems to be zero for $\gamma \leq 3$, and finite for $\gamma > 3$ [11]. The heterogeneous mean-field theory is a degree-based mean-field theory, which sorts the nodes into different classes in terms of the magnitude of their degrees, but all the other aspects are considered totally random (for instance, who connects whom). In other words, the heterogeneous mean-field theory neglects correlation, both from the disease dynamics (a node is more likely infected if its neighbors are infected) and the network structure. Effectively, the heterogeneous mean-field theory applies to a situation where the network is constantly rewired (or *annealed*) at a time scale faster than the disease dynamics. (See the Supplemental Material [12] for more details on the SIS and SIR models on annealed networks).

Moving beyond the heterogeneous mean-field assumption that the network is rapidly changing, we have to deal with dynamic correlations. In the heterogeneous mean field, the probability to be infected is ρ for two nodes of the same degree, but because of dynamic correlations a node is more likely to be infected if many of its neighbors are infected. This fact—the probability of having an already infected neighbor is larger than in the mean-field approximation—will reduce the effective infection rate, and thus the speed and extent of the disease propagation. One possible remedy is to consider an individual-based mean-field approximation by taking into account the full network structure correlation (still ignoring the dynamic correlations). This is also called the quenched mean-field theory [17,18]. The quenched mean-field theory gives the epidemic threshold $\lambda_c^{\text{QMF,SIS}} = 1/\Lambda_1$ [17], where Λ_1 is the largest eigenvalue of the adjacency matrix of the underlying network. According to Ref. [19], for scale-free networks,

$$1/\Lambda_1 \sim \begin{cases} 1/\sqrt{k_{\max}}, & \gamma > 5/2, \\ \langle k \rangle / \langle k^2 \rangle, & 2 < \gamma < 5/2, \end{cases} \quad (1)$$

where k_{\max} is the maximum degree in the network. Both the heterogeneous mean-field and quenched mean-field methods imply that the epidemic threshold in scale-free networks vanishes in the thermodynamic limit, but remains finite for networks of finite sizes. Some numerical studies suggest that the quenched mean-field is qualitatively correct in scale-free networks [20,21], but others point at deviations from the threshold value [10].

There are studies that do take dynamic correlations into account. This, so called, effective degree approach [22,23] is a higher order degree-based mean-field theory, explicitly considering the dynamic correlation between directly connected neighbors. It does indeed provides a much more accurate prediction of the epidemic threshold for the SIR model in uncorrelated networks, $\lambda_c^{\text{ED,SIR}} = [\langle k \rangle / (\langle k^2 \rangle - 2\langle k \rangle)]$ [22]. This works by mapping the SIR process to a bond-percolation problem [11,24], but has not yet been applied to the SIS model. Boguñá *et al.* [21] take dynamic correlation between distant neighbors into account within the framework of quenched mean-field theory. They replaced the original SIS dynamics by a modified process valid over coarse-grained times and argued that dynamic correlation changes the dynamics near the threshold. Based on a spectral approach, which takes into account the other eigenvectors of the adjacency matrix than Λ_1 , Goltsev *et al.* [25] showed that in scale-free networks with $\gamma > 5/2$, the principal eigenvector is localized. This means that above λ_c^{QMF} the epidemic is alive, but the activity is concentrated to the hubs and their immediate neighbors. This leads to the conclusion that the true threshold for the endemic state would have a finite value for scale-free networks with $\gamma > 3$ [21]. Ferreira *et al.* [26] studied the relationship between the hub lifespan and the hub infection time to discern the nature of the threshold in general epidemic models on scale-free networks. References [27,28] present yet further refinements of the quenched mean-field theory. Finally, in this literature review, we also want to mention many other approaches to understand SIS or SIR processes on networks, such as the pair-approximation method [29,30], probability generating function techniques [31,32], R_0 -based modification [33], percolation theory [31,34], and branching process [35], etc. However, all these methods depend on large sets of coupled ordinary differential equations, and are unable to provide explicit analytical solutions for the prevalence, in particular, for large-scale networks [11].

Our current understanding of the SIS dynamics on heterogeneous networks is far from complete. In this Letter, we combine the idea of the heterogeneous mean-field theory with the effective degree approach to study the effect of dynamic correlation present in static networks on the SIS epidemic dynamics. The underlying static networks can have arbitrary degree distributions. However, we focus on networks without degree-degree correlations or other (e.g., mesoscopic) structures. Our method gives a closed-form analytical solution for the prevalence ρ , from which we immediately can obtain the epidemic threshold by

solving the equation $\rho = 0$. We further show that our method matches numerical simulations better than the above-mentioned theory.

To begin, we define p_k and q_k , respectively, as the probabilities of reaching an arbitrary infected individual by following a randomly chosen edge from susceptible and infected individuals of degree k . In the framework of heterogeneous mean-field theory, where the underlying network is treated as annealed, the value of q_k is always equal to that of p_k [21]. For the earlier mentioned reasons, the dynamic correlations mean that

$$q_k > p_k. \quad (2)$$

In the spirit of the heterogeneous mean field [5], we can write down the master equation for those infected individuals of degree class k ,

$$\frac{dI_k(t)}{dt} = -I_k(t)g + rkS_k(t)p_k(t), \quad (3)$$

where $S_k(t)$ and $I_k(t)$ are the number of susceptible and infected individuals with degree k at time t , respectively. The first term represents the spontaneous recovery and the second one the newly emerged infection in class k due to the interaction with other classes. In the steady state, we have

$$p_k = \frac{g I_k}{kr S_k}. \quad (4)$$

In annealed networks, one assumes $p_k = q_k = [1/(\langle k \rangle N)] \times \sum_k k I_k$, where N is the total population size, to continue the theoretical derivation. As mentioned above, this hypothesis is not applicable for the SIS model in static networks. In what follows, we circumvent this problem following the effective degree approach. This means that we divide the network into classes representing both the state of an individual and its neighbors [36,37]. Let $S_{k,j}(t)$ [$I_{k,j}(t)$] be the density of k degree nodes that are susceptible (infected) at time t , connected to j infected neighbors. Then, $S_{k,j}(t)$ and $I_{k,j}(t)$ can be written as

$$S_{k,j}(t) = S_k(t)[1 - p_k(t)]^{k-j}[p_k(t)]^j \binom{k}{j}, \quad (5a)$$

$$I_{k,j}(t) = I_k(t)[1 - q_k(t)]^{k-j}[q_k(t)]^j \binom{k}{j}, \quad (5b)$$

where $S_k(t) = \sum_j S_{k,j}(t)$ and $I_k(t) = \sum_j I_{k,j}(t)$. Summing over all possible events, we obtain the total recovery rate $a(t) = \sum_k \sum_j I_{k,j}(t)g$ and the population-level transmission rate $b(t) = \sum_k \sum_j S_{k,j}(t)jr$. When the system is in its steady state, the total recovery rate must be equal to the total transmission rate, satisfying the *detailed balance* conditions $a = b$ and $\langle \Delta a \rangle = \langle \Delta b \rangle$, where Δa and Δb are the changing rate of a and b in the time interval dt (where only one event occurs). All possible values of these two quantities in a static network are summarized in Table I. With these preliminary results, we have

TABLE I. All possible situations in a time interval dt of the SIS process in a static network.

Situation	Probability	Δa	Δb
$S_{k,j} \rightarrow I_{k,j}$	$[S_{k,j}(t)jr]/(a+b)$	$+g$	$-jr + r(k-j)$
$I_{k,j} \rightarrow S_{k,j}$	$[I_{k,j}(t)g]/(a+b)$	$-g$	$+jr - r(k-j)$

$$\sum_k \sum_j S_{k,j} j^2 r = \sum_k \sum_j I_{k,j} j g. \quad (6)$$

Note that the formula of Eq. (6) is exact for the steady state in any static networks. Nevertheless, we want to point out that they are not applicable to irreversible processes such as the SIR model.

Using the condition that the total number of infectious neighbors of all susceptible individuals equals the total number of susceptible neighbors of all infectious individuals [38], we get the relationship $\sum_k \sum_j I_{k,j} j g = g \sum_k I_k [k - g/r]$. Combining this with Eqs. (4) and (6), gives

$$g \sum_k I_k \left[(k-1)p_k + 1 - k + \frac{g}{r} \right] = 0. \quad (7)$$

Notice that only infected individuals can determine the birth and death of susceptible individuals, but not vice versa. Any new infection or recovery event will mainly change the difference among various q_k (since the probability of finding connected infected pair with certain degrees will increase or decrease definitely due to the event). But it does not provide any valuable hints about how various p_k will differ from each other (albeit their values would change owing to the newly emerged or disappeared infected individual) for *degree-uncorrelated* random networks. In light of the maximum entropy principle [39], we assume all p_k change with the same magnitude such that they always satisfy the following approximative relations

$$p_1 \approx p_2 \approx \dots \approx p_{k_{\max}} = p. \quad (8)$$

That is to say, our method is mainly concerned about the dynamic correlations present in connected infected pairs, but neglects higher-order dynamic correlations in susceptible-susceptible or susceptible-infected pairs. We have verified that Eqs. (2) and (8) capture the most important parts of the dynamic correlations, neglecting only minor ones [12]. Substituting Eq. (8) into Eqs. (4) and (7), we obtain the iterative equation

$$I_k = \frac{\lambda k p}{1 + \lambda k p} N_k, \quad (9)$$

where p itself is a function of I_k as

$$p = 1 - \frac{I}{\lambda \sum_k I_k (k-1)}. \quad (10)$$

Combining with Eqs. (9) and (10), we can find the solution of p , which is now a function of λ , satisfying the self-consistency equation

$$(1-p)\lambda \sum_k (k-1) \frac{\lambda k p}{1 + \lambda k p} N P(k) - \sum_k \frac{\lambda k p}{1 + \lambda k p} N P(k) = 0. \quad (11)$$

Now we are able to calculate the prevalence ρ in the steady state as follows: (i) Calculate p from Eq. (11); (ii) Substitute the value of p into Eq. (9) to solve I_k ; (iii) Obtain the prevalence $\rho = (1/N) \sum_k I_k$. The results are summarized in Fig. 1, from which we can see that the estimations obtained by our approach match those from stochastic simulations quite well [12].

A nonzero stationary prevalence is obtained when the p has a nontrivial solution in the interval $0 < p \leq 1$. We denote the left-hand side of Eq. (11) by $f(p)$. It is easy to see that $p = 0$ is a trivial solution of Eq. (11). Furthermore, note that $f(p)$ is always negative for $p = 1$. Hence, the condition that p has a meaningful solution in the interval $(0, 1]$ reads as

$$\frac{d}{dp} f(p) \Big|_{p=0} \geq 0. \quad (12)$$

The value of λ satisfying the equality of the above inequality determines the epidemic threshold λ_c , whose value is given, for uncorrelated random networks, by

$$\lambda_c = \frac{\langle k \rangle}{\langle k^2 \rangle - \langle k \rangle}. \quad (13)$$

The epidemic threshold of the SIS model obtained by our method is larger than the one predicted by the heterogeneous mean-field theory [5,6,9,10], and is smaller than the threshold of the SIR model by the effective degree approach [11,22]. Equation (13) implies that the epidemic threshold of the SIS epidemic process is zero for scale-free networks with $\gamma < 3$, but finite if $\gamma > 3$. This is consistent with the conclusions of previous work using other methods [25,27]. In Fig. 2, we plot the epidemic thresholds against network size and show that, in Erdős-Rényi and scale-free

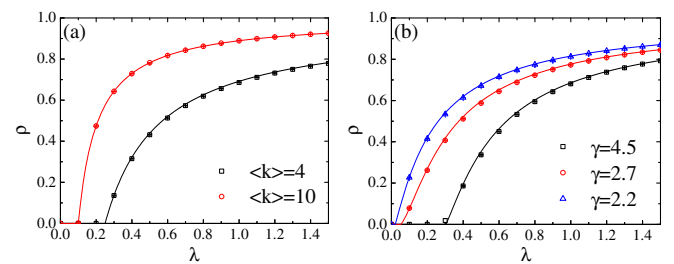


FIG. 1. The prevalence ρ is plotted as a function of the effective infection rate λ in static networks. (a) Erdős-Rényi random networks with average degree 4 and 10, (b) scale-free network with minimum degree 3 and $\gamma = 4.5, 2.7$, and 2.2 . Lines are theoretical estimations of Eqs. (9) and (11), while the points are simulation results. We use networks with $N = 10^5$ nodes and 100 randomly chosen seeds for the infection. Each data point is an average over at least 100 independent epidemic outbreaks, performed on at least 10 different network realizations.

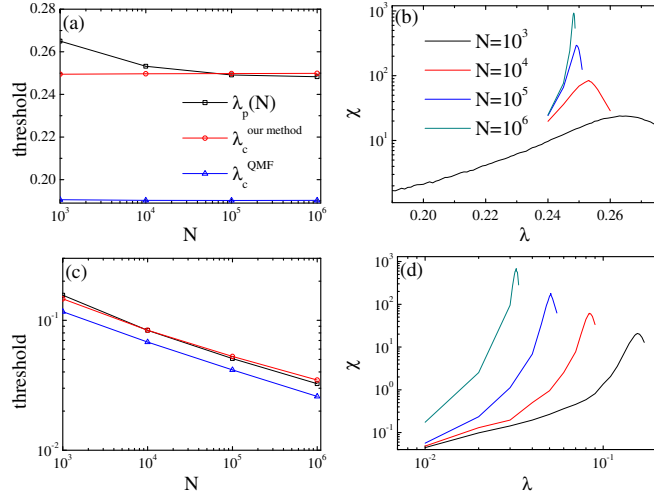


FIG. 2. (a) and (c) Epidemic threshold λ_c , obtained by our method, the quenched mean-field method, and the quasistationary state method [10,21], versus network size N . (b) and (d) Susceptibility χ as a function of the effective infection rate λ for varying network size N , where the peak values determine the $\lambda_p(N)$ in (a) and (c). Panels (a) and (b) are for Erdős-Rényi random networks with average degree $\langle k \rangle = 4$; (c) and (d) are for scale-free networks with minimum degree 3 and $\gamma = 2.7$. The results are obtained for the epidemic dynamics over at least ten different network realizations with initial infected seed number $I_0 = 1$.

random networks, the accuracy of the epidemic threshold of our method is better than that of the quenched mean-field theory, and matches the results from the quasistationary numerical simulation well [10,21].

To provide further evidence on the efficiency of our proposed approach, we consider the SIS model in a random regular graph, whose degree distribution is a Kronecker's delta function $P(k) = \delta_{kk_0}$; i.e., each node in a random regular graph has a degree of k_0 and all the other aspects are totally random. From Eqs. (4) and (7), we can acquire the epidemic prevalence

$$\rho = 1 - \frac{1}{1 + k_0(\lambda - \frac{1}{k_0-1})}, \quad (14)$$

where $\lambda = r/g$ is again the effective infection rate. From Fig. 3(a), we can see that the results from the analytical solution of Eq. (14) are in excellent agreement with those obtained from stochastic simulations in a static random regular graph.

For the epidemic threshold of the SIS model in static random regular graphs, the prediction of the quenched mean-field theory can easily be simplified to $\lambda_c^{\text{QMF}} = 1/k_0$ by applying the Perron-Frobenius theorem [10]. By means of the pair-approximation method, a more accurate estimation of epidemic threshold, $\lambda_c^{\text{pair}} = 1/(k_0 - 1)$, is reported [29]. Very recently, by combining the branching process with the probability generating function, Leventhal *et al.* obtained the same threshold $1/(k_0 - 1)$ [35]. For our case, we just need to set $\rho = 0$ in Eq. (14), and then the epidemic threshold can be obtained directly as

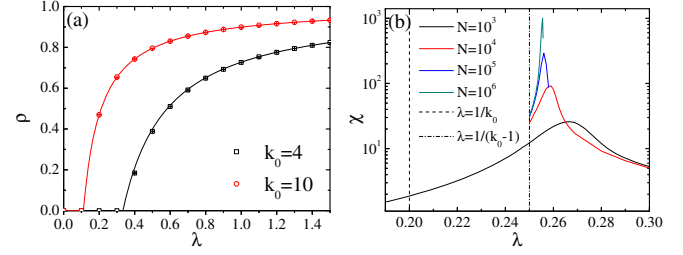


FIG. 3. (a) The epidemic prevalence ρ is plotted as a function of the effective infection rate λ in static random regular graphs with degree $k_0 = 4$ and 10. Lines are the theoretical predictions of Eq. (14), while the scatters are Monte Carlo simulation results. Other parameters are the same as those in Fig. 1. (b) Susceptibility χ as a function of the effective infection rate λ for random regular graphs with different network sizes, where k_0 is fixed to 5.

$$\lambda_c = \frac{1}{k_0 - 1}, \quad (15)$$

which is consistent with the findings in Refs. [29,35]. In Fig. 3(b), we plot the susceptibility against the effective infection rate λ in static random regular graph with degree $k_0 = 5$, and show that the susceptibility peak is closer to the theoretical prediction of Eq. (15) than to the quenched mean-field result [10], once again validating our method.

Thus, the combination of the heterogeneous mean-field theory and the effective degree method enables us to, on one hand, include dynamic correlation and network-structural correlation (to a necessary extent) to obtain more accurate predictions (than in previous works) of both the epidemic prevalence and threshold, and, on the other hand, obtain explicit expressions for these two quantities.

As a final analysis, we extended our method to the case of contact process [40]—where infected individuals meet random neighbors for possible contagion events—on random regular graph networks with degree k_0 . According to the heterogeneous mean-field theory, we have $\rho = (g/r)(I_{k_0}/S_{k_0})$. The total recovery and transmission rates of the whole population are $a = \sum_j I_{k_0,j}g$ and $b = \sum_j S_{k_0,j}jr(1/k_0)$, respectively. Equation (6) will now be replaced by $(r/k_0)\sum_j S_{k_0,j}^2 = g\sum_j I_{k_0,j}$. Then, we can straightforwardly derive the prevalence as

$$\rho = 1 - \frac{1}{1 + \lambda - \frac{k_0}{k_0-1}}. \quad (16)$$

Accordingly, the epidemic threshold of the contact process on random regular graphs is given by

$$\lambda_c = \frac{k_0}{k_0 - 1}. \quad (17)$$

This epidemic threshold is also consistent with previously reported results [41,42]; see more details in Ref. [12].

In summary, we have studied the impact of dynamic correlations, naturally arising in spreading processes on static networks, on the SIS epidemics. In particular, we take into account the dynamic correlation from infected pairs, but

ignore those from other node pairs and higher-order network structure, to derive the master equations governing the state evolution of the system. By combining the idea of the heterogeneous mean-field theory with the effective degree approach, we are able to obtain the epidemic prevalence of the SIS process in uncorrelated static networks with arbitrary degree distributions with a higher precision than other approaches. It is worth noting that the epidemic threshold can be calculated as a corollary of the epidemic prevalence. Specifically, for SIS in scale-free networks, the quenched mean-field theory predicts that the epidemic threshold is zero in the thermodynamic limit. By contrast, our theoretical results show that it remains finite for scale-free networks with $\gamma > 3$, even in the thermodynamic limit.

Our work can be generalized to more general stochastic-logistic models of density dependent population dynamics [43]. In such cases, both the infection and recovery rates usually depend on the ratio I/N in these models (and I is often interpreted as the population size, while N is the carrying capacity). Our dynamic correlation approach could be fairly straightforwardly extended to the general stochastic logistic model, where Eq. (3) is the first equation to be modified (to account for the population-dependent death rate).

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