

Threshold and quasistationary distribution for the susceptible-infectious-susceptible model on networks

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We study the Susceptible-Infectious-Susceptible model on arbitrary networks. The well-established pair approximation treats neighboring pairs of nodes exactly while making a mean-field approximation for the rest of the network. We improve the method by expanding the state space dynamically, giving nodes a memory of when they last became susceptible. The resulting approximation is simple to implement and appears to be highly accurate, both in locating the epidemic threshold and in computing the quasistationary fraction of infected individuals above the threshold, for both finite graphs and infinite random graphs.

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I. INTRODUCTION

The spread of an infectious disease can be modeled as a stochastic process unfolding on a network. This perspective has significantly advanced our understanding of the mathematics of epidemics (see, e.g., Refs. [1–7]).

Some diseases, such as measles or polio, infect individuals at most once (with rare exceptions). Others, such as RSV or norovirus, can routinely reinfect the same individual. The mathematical implications of this recurrent reinfection are quite subtle, since it creates ongoing epidemic dynamics on a network rather than sweeping through it just once.

The simplest epidemic models with reinfection are Susceptible-Infectious-Susceptible (SIS) models [1,2,8–10]. In these models, each individual is either susceptible (S) or infectious (I). Susceptible individuals become infectious due to interactions with other infectious individuals, and infectious individuals recover and immediately become susceptible again. The key finding of SIS models is that, under the right circumstances, a disease can enter an endemic phase where it continually infects people at a rate that balances out recovery, maintaining a finite fraction of infected individuals in the population. This endemic phase is quasistationary; it persists for exponential time until the system enters its absorbing state, where no individuals are infected.

Even though the SIS model is exceedingly simple, it is surprisingly resistant to analytic treatment. Ideally, we would like to locate the epidemic transition and define the properties of the endemic state, including the quasistationary fraction of

infected individuals. But accurate characterization of either quantity has eluded the community, and, remarkably, we lack a solution even for the one-dimensional lattice. Nevertheless, numerous approximate approaches have been advanced, i.e., Refs. [1,2].

A key line of attack for SIS mirrors the development of methods to study percolation in the physics literature. In particular, the discrete-time SIS model is equivalent to a directed percolation model on a graph of nodes in space-time, with directed edges from one time step to the next corresponding to the spread or persistence of infection [9].

To compute epidemic thresholds, we typically start with simple mean-field approximations where nodes are assumed to be independent [11–14]. These approximations are inaccurate because they fail to account for correlations between neighbors.

To go beyond the mean-field approximation, we can consider the joint distribution of pairs of connected individuals, while assuming that these pairs interact with each other in a mean-field way. This is known as the pair approximation [15–19]. Unfortunately, the pair approximation is still noticeably inaccurate for SIS models, even when the network is locally tree-like, and the corresponding approximation would be asymptotically exact for percolation [20,21].

In principle, one can extend the pair approximation by considering larger clusters of connected nodes, moving to connected triples, quadruples, and so on, but in practice this is rarely done. First, the computational effort of this approach grows exponentially with the size of the cluster. Second, while any network can easily be decomposed into a set of connected pairs, i.e., the edges, decomposition into larger motifs is nontrivial and nonunique (e.g., see Ref. [22] for a triplet decomposition on regular graphs). Another approach along these lines is the star approximation [23], which yields a unique decomposition into overlapping star-shaped neighborhoods but at the expense of more complex equations.

We take a different approach in this paper. Rather than extending the pair approximation in space, we extend it in

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time. The idea is reminiscent of the dynamic cavity method [24–28], which has also been deployed to study recurrent disease models [29]. These models work by keeping track of a node’s entire history and then assuming that the histories of neighboring nodes are conditionally independent. Tracking the history of a node, however, requires computational effort that grows exponentially with the number of time steps or transitions. Thus, this approach rapidly becomes intractable just as spatial cluster methods do.

In contrast, we use a memoryless approach, but we augment the susceptible state to a chain of K states that make transitions from one to the next according to a Poisson process. This provides an approximate “clock” that estimates how long a node has been susceptible, i.e., how long ago it was last infected. These additional states let us model the correlation between this (approximate) time and the states of its neighbors. If a node was infected very recently, then its neighbors are more likely to be infected, either because they infected it or vice versa. Conversely, if a node has been susceptible for a long time, its neighbors are less likely to be infected.

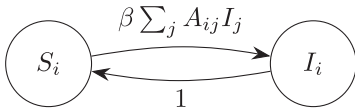
We capture these correlations by assuming that the joint distribution of each neighboring pair of nodes is the stationary distribution of the corresponding Markov process. We then, analogous to the pair approximation, assume that pairs of nodes are conditionally independent of other pairs, taking the marginals of their shared endpoints into account.

By varying the number of states K , this approach yields a sequence of approximations for the SIS model that are simple to implement on arbitrary networks. While we remain agnostic on whether this sequence approaches the exact behavior of the SIS model as $K \rightarrow \infty$, it converges quickly and is significantly more accurate than standard methods. Even using a chain of $K = 8$ susceptible states gives results that agree very closely with experiments on a variety of networks.

II. APPROXIMATING THE SIS MODEL

We consider the continuous-time SIS model, also known as the contact process [9]. At any given time, each node is either susceptible or infectious. Each infectious node infects each of its susceptible neighbors at a constant rate, which we denote β . Thus, a susceptible node with k infectious neighbors becomes infected at rate βk . Once infectious, nodes recover at a constant rate and become susceptible once again. Without loss of generality, we set the rate of recovery to 1.

We use the indicator variables S_i and I_i to denote the state of node i as susceptible or infectious, respectively. We can represent the model with the following diagram:

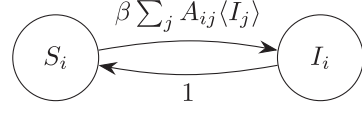


where A is the adjacency matrix so that the sum $\sum_j A_{ij} I_j$ is the number of infectious neighbors of node i .

A. The individual approximation

While the system as a whole is Markovian, any subsystem is not. Nevertheless, in the style of a mean-field approximation, let us assume that each node follows its own Markov

process, becoming infected at a rate proportional to its average number of infectious neighbors. Diagrammatically, this becomes



The stationary distribution for node i is

$$P_i(I_i) = \langle I_i \rangle = \frac{\beta \sum_j A_{ij} \langle I_j \rangle}{1 + \beta \sum_j A_{ij} \langle I_j \rangle}, \quad (1)$$

or, in matrix notation, we can write

$$\rho = \frac{\beta A \rho}{1 + \beta A \rho}, \quad (2)$$

where $\rho_i = \langle I_i \rangle = P_i(I_i)$. This simple set of self-consistent equations provides a qualitatively reasonable approximation to the full model. Linearizing (2) around $\rho = \mathbf{0}$ leads to the first-order approximation for the epidemic threshold, namely

$$\beta_c \simeq \beta_c^{(0)} = \frac{1}{\lambda(A)}, \quad (3)$$

where $\lambda(A)$ is the largest eigenvalue of A (see, e.g., Refs. [11–13]).

A regular graph provides the simplest example. If every node has $q + 1$ neighbors, as in a tree where each node has q children and a parent, then Eq. (2) predicts

$$\rho_i = \frac{\beta(q + 1) - 1}{\beta(q + 1)} \quad (4)$$

when $\beta > \beta_c^{(0)} = 1/(q + 1)$ and $\rho_i = 0$ otherwise. However, both Eq. (2) and this estimate for β_c are inaccurate, as we shall presently see.

B. The pair approximation

To improve the approximation of the previous section, let us instead assume that each connected pair of nodes is Markovian. For each connected pair (i, j) , we endeavor to find $P_{ij}(X_i, X_j)$, the joint probability that that node i is in state X_i and j in state X_j . To do this, we (incorrectly) assume that the small subsystem of (i, j) is Markovian and that infections from outside the pair arrive at their average rate. When node j is susceptible, it will be infected by nodes other than i at rate

$$\beta \sum_{k \neq i} A_{jk} P_{jk}(I_k | S_j). \quad (5)$$

Defining

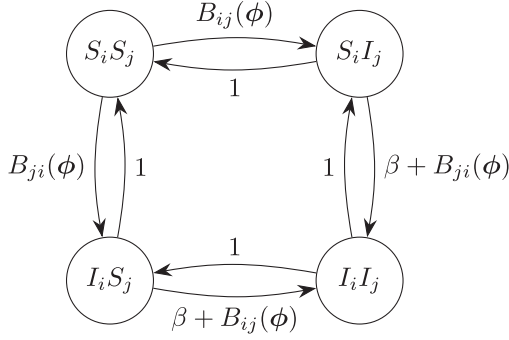
$$\phi_{jk} = \beta P_{jk}(I_k | S_j), \quad (6)$$

then the rate of infection from outside the pair can be written compactly using operator notation. Namely, by defining

$$B_{ij}(\phi) = A_{ij} \sum_{k \neq i} A_{jk} \phi_{jk}, \quad (7)$$

the rate of infection from outside the pair is $B_{ij}(\phi)$ for node j and $B_{ji}(\phi)$ for node i . The operator B is the nonbacktracking operator (see, e.g., Ref. [30]) and keeps track of how the infection spreads to the pair due to the rest of the network.

Diagrammatically, the picture we get is



If we knew ϕ , we could solve this simple system for its stationary distribution following elementary methods [31]. To do this one solves for the left-eigenvector with zero eigenvalue of the transition matrix

$$Q(B_{ij}(\phi), B_{ji}(\phi)) = \begin{pmatrix} \cdot & 1 & 1 & 0 \\ \beta + B_{ij}(\phi) & \cdot & 0 & 1 \\ \beta + B_{ji}(\phi) & 0 & \cdot & 1 \\ 0 & B_{ji}(\phi) & B_{ij}(\phi) & \cdot \end{pmatrix}, \quad (8)$$

where the diagonals are set so that the rows sum to zero. This provides an expression for the distribution P_{ij} given knowledge of ϕ . Namely, P_{ij} is the vector that solves

$$P_{ij} Q(B_{ij}(\phi), B_{ji}(\phi)) = \mathbf{0}. \quad (9)$$

Of course the whole point of this calculation is that we do not know ϕ in advance. Regardless, from its definition we write

$$\phi_{ij} = \beta P_{ij}(I_j | S_i) = \frac{\beta P_{ij}(S_i, I_j)}{P_{ij}(S_i, I_j) + P_{ij}(S_i, S_j)}. \quad (10)$$

With this, we have a closed system of equations. Equation (9) gives us the required probabilities in terms of ϕ and Eq. (10) gives us ϕ in terms of the probabilities. By explicitly inverting the matrix in Eq. (9) and inserting the resulting distribution into the definition of Eq. (10), we find

$$\phi_{ij} = \Psi_\beta(B_{ij}(\phi), B_{ji}(\phi)), \quad (11)$$

with

$$\Psi_\beta(x, y) = \beta \frac{2x + xy + x^2 + \beta x + \beta y}{(2 + x + y)(1 + x + \beta)}. \quad (12)$$

On any network of interest, this set of equations (11) can be iterated until convergence to find ϕ .

Finally, when we want to approximate the behavior of a single node i , we can assume it receives infections from each neighbor j at rate ϕ_{ij} , and thus

$$P_i(I_i) = \frac{\sum_j A_{ij} \phi_{ij}}{1 + \sum_j A_{ij} \phi_{ij}}. \quad (13)$$

While we have derived this via a conceptually different route, this approximation is equivalent to the standard pair approximation [2,16]. The main difference is that in the conventional setup for the pair approximation, one integrates a large system of nonlinear equations, whereas by solving

Eq. (11), we directly jump to the steady-state solution without needing to numerically integrate.

As seen above, the single-node mean-field approximation predicts that the endemic transition occurs at $\beta = 1/\lambda(A)$. The pair approximation improves on this estimate. Linearizing Eq. (11) at $\phi_{ij} = 0 + \varepsilon_{ij}$ we get

$$\varepsilon_{ij} = \frac{\beta(\beta + 2)}{2(\beta + 1)} B_{ij}(\mathbf{e}) + \frac{\beta^2}{2(\beta + 1)} B_{ji}(\mathbf{e}), \quad (14)$$

which changes stability at a critical value $\beta_c^{(1)}$, the solution to

$$\beta_c^{(1)} = \frac{1}{\lambda(A - \beta_c^{(1)} L/2 - I)}, \quad (15)$$

where $L = D - A$ is the graph Laplacian (see Appendix A). This fixed-point equation can be easily solved numerically by iteration.

For a $(q + 1)$ -regular random graph, Eq. (11) becomes

$$\phi = \Psi_\beta(q\phi, q\phi) = \frac{q\beta\phi}{1 + q\phi}. \quad (16)$$

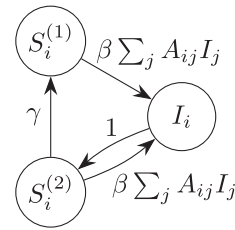
This yields $\phi = \beta - 1/q$, so this approximation predicts that the endemic threshold is

$$\beta_c^{(1)} = 1/q. \quad (17)$$

While this is not a bad prediction, it disagrees with simulations by an appreciable margin, particularly for small q . Below we give a correction derived from our approach.

C. Improved approximation for dynamical correlations

Having reviewed the mean-field and pair approximations, we turn now to our augmentation of the state space. We consider a family of variations of the SIS model, which we call the SIS^K model, with $K \geq 1$. The SIS^1 model is the SIS model. The SIS^2 model is very similar to the SIS model, except that there are two different S states, $S^{(1)}$ and $S^{(2)}$. The transitions are

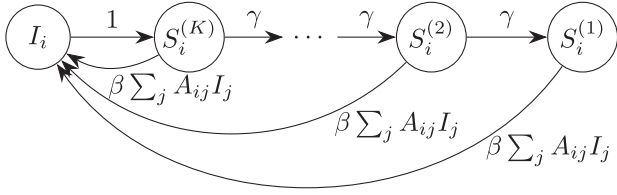


Just as for the standard SIS model, the infectious state I recovers at unit rate. Both $S^{(1)}$ and $S^{(2)}$ are infected at exactly the same rate as the standard SIS model. State I recovers to $S^{(2)}$, and then $S^{(2)}$ decays to $S^{(1)}$ at some rate γ .

Clearly nodes in state $S^{(1)}$ have been susceptible for longer, on average, than nodes in state $S^{(2)}$. However, this variant model is ultimately the same as the conventional SIS model: if we collapse or “lump” the states $S^{(1)}$ and $S^{(2)}$ together in a single S state, we recover the original SIS dynamics exactly.

The variants SIS^K with $K > 2$ are defined in the same way. We have K different S states, $S^{(1)}, \dots, S^{(K)}$. Each is infected at exactly the same rate; I always recovers to $S^{(K)}$; and each $S^{(x+1)}$ decays to $S^{(x)}$ at rate γ . $S^{(1)}$ does not decay further,

so its only transition is to become infectious. The picture is now



Again, the key point of this construction is that for all K , SIS^K is exactly equivalent to SIS if we lump the $S^{(1)}, \dots, S^{(K)}$ together into a single S state [32].

Because SIS^K has precisely the same behavior as the standard SIS model, the extra S states may seem like an unnecessary complication. A simple appeal to Occam's razor would have us do away with them. However, they turn out to be a useful conceptual tool. Their true utility becomes apparent once we start to approximate the system, rather than performing exact simulations.

While an exact simulation of SIS^K and the standard SIS model are equivalent, the approximations to them are not. Let us follow the same logic as in the pair approximation above. If nodes i and j are connected then we solve the equilibrium dynamics of the pair exactly, while assuming they are infected by their other neighbors at rate that depends on their state. When node j is in state $S^{(x)}$, it is infected by its other neighbors at rate

$$\beta \sum_{k \neq i} A_{jk} P_{jk}(I_k | S_j^{(x)}). \quad (18)$$

Defining

$$\phi_{jk}^x = \beta P_{jk}(I_k | S_j^{(x)}), \quad (19)$$

we have the rate of infection for node j from neighbors other than i to be $B_{ij}(\phi^x)$ for each $x = 1, \dots, K$.

Each node in the pair (i, j) can be in any one of $K + 1$ states and so the system consisting of the pair can be in any of $(K + 1)^2$ states. Let $Q(a, b)$ denote the transition matrix for a system of two nodes with infections from outside the pair occurring at rate a_x (resp. b_x) when the first (resp. second) node is in state $S^{(x)}$. The elements of the transition matrix are, for recovery events,

$$\begin{aligned} [Q(a, b)]_{II, IK} &= [Q(a, b)]_{II, KI} \\ &= [Q(a, b)]_{IX, KX} \\ &= [Q(a, b)]_{xI, xK} = 1, \end{aligned} \quad (20)$$

for the decay events

$$\begin{aligned} [Q(a, b)]_{IX, I(x-1)} &= [Q(a, b)]_{xI, (x-1)I} \\ &= [Q(a, b)]_{xy, (x-1)y} \\ &= [Q(a, b)]_{xy, x(y-1)} = \gamma, \end{aligned} \quad (21)$$

and for infection events

$$[Q(a, b)]_{Iy, II} = \beta + b_y, \quad (22)$$

$$[Q(a, b)]_{xI, II} = \beta + a_x, \quad (23)$$

$$[Q(a, b)]_{xy, xI} = b_y, \quad (24)$$

$$[Q(a, b)]_{xy, Iy} = a_x. \quad (25)$$

As usual, the diagonals of the transition matrix Q are set so that all rows sum to zero.

Assuming we knew the rates ϕ_{ij}^x , the stationary distribution for the system can be found by solving a linear equation, namely, the distribution P_{ij} solves

$$P_{ij} Q(B_{ij}(\phi), B_{ji}(\phi)) = 0. \quad (26)$$

On the other hand, if we knew the stationary distribution P_{ij} we could easily compute

$$\phi_{ji}^x = \frac{\beta P_{ij}(I_i, S_j^{(x)})}{P_{ij}(I_i, S_j^{(x)}) + \sum_{y=1}^K P_{ij}(S_i^{(y)}, S_j^{(x)})}. \quad (27)$$

Hence, we are again left with a set of self-consistent equations that we can solve by iteration. We alternately find P_{ij} for all connected pairs (i, j) by solving the linear equation in Eq. (26) and then we update our estimates of ϕ_{ji}^x using Eq. (27).

The extra S states effectively track how recently a node was infectious. At very short times after moving from infectious back to susceptible, a node will likely be in state $S^{(K)}$. At longer times, if it has not been reinfected, it will eventually decay back to $S^{(1)}$. Which S state a node is in thus acts like a noisy clock that “ticks” from $S^{(x+1)}$ to $S^{(x)}$ at rate γ . More precisely, the time taken to decay from $S^{(x+\gamma t)}$ to $S^{(x)}$ is a (Gamma distributed) random variable with mean t , while the variance is t/γ . Hence the clock measures time at a resolution inversely proportional to γ and the maximum time that these K states can record in this sense is K/γ .

We can tune the decay rate γ as we wish, in order for the state distribution to be as informative as possible about how long a node has been susceptible. In order for both the temporal resolution and the maximum time to improve as K grows, we propose setting γ to

$$\gamma = \beta q \sqrt{K-1}, \quad (28)$$

where q is one less than the mean degree, i.e., $q = \sum_{ij} A_{ij}/n - 1$, and we use $\sqrt{K-1}$ because there are $K-1$ transitions from $S^{(K)}$ to $S^{(1)}$. This choice clearly meets our temporal scaling requirements: $1/\gamma$ tends to zero as K gets large and additionally K/γ tends to infinity, and thus we obtain a more fine-grained resolution over a wider range of times as K increases. Of course, using $\sqrt{K}$ would work as well.

Additionally, note that the number of infections arriving at a node per unit timescales linearly with βq , whereas the number of decay events at a node (where $S^{(x+1)}$ decays to $S^{(x)}$) scales linearly with γ . For these to scale equivalently, and hence for the timescales of infections and decays to match, we require that γ is proportional to βq . Equation (28) matches our desiderata, although of course it is not the only such choice.

1. Dynamic correlation

The standard pair approximation allows us to approximate spatial correlations, i.e., $\langle I_i I_j \rangle = P_{ij}(I_i, I_j)$. Our SIS^K pair approximation also allows us to compute this and if it approximates P_{ij} more accurately, then it should approximate

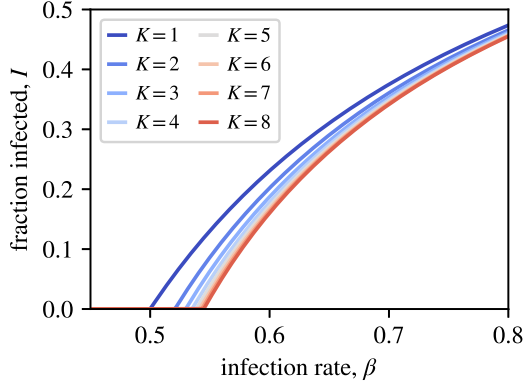


FIG. 1. Approximations for the SIS model on three-regular random graphs with increasing K . We show the predicted steady-state fraction of infectious nodes as a function of the spreading rate β . As K increases, we see a diminishing change in the predictions at K against $K - 1$. Note that, throughout this paper, we set the recovery rate to 1 without loss of generality.

the location of the transition and the proportion infected in the endemic state above the transition.

As discussed above, by increasing K , an effective memory is built into the system and our answers to temporal questions should also improve in accuracy. We see exactly this in Fig. 3. We simulated the SIS model on a large four-regular random graph at $\beta = 0.4$ (which is above the threshold), and we measured the time between infections. From these measurements, we can estimate the probability that a node will survive in the S state for more than t time units. Note that by the stan-

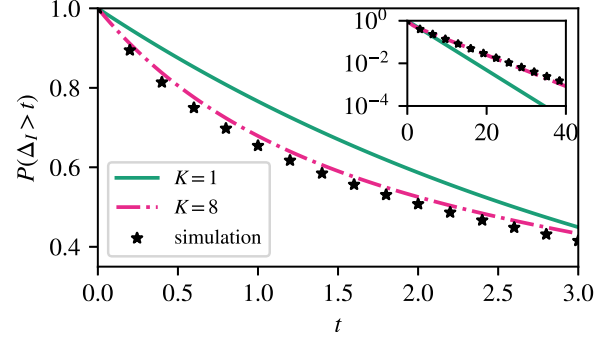


FIG. 3. Estimates of the survival function for a four-regular random graph. Let Δ_I be the time between infections in the endemic state. We show the probability that $\Delta_I > t$, i.e., that the node survives in the susceptible state for longer than t between infections. Simulations are on a graph with 50 000 nodes and $\beta = 0.4$. The inset shows the same data but on a logarithmic scale. Setting $K = 1$, i.e., using the standard pair approximation, yields a simple exponential, while the $K = 8$ approximation closely matches experiment.

dard approximation, the survival time distribution is a simple exponential. Thus, our goal is to correctly model how correlations create a mix of exponentials. In particular, susceptible nodes that were recently infected are more likely to become reinfected quickly, so the risk of reinfection decreases as t increases.

We show the survival probability on both linear and logarithmic scales in Fig. 3, and compare simulations to the $K = 1$ and $K = 8$ approximations. While setting $K = 1$ yields a simple exponential, the $K = 8$ approximation is quite accurate, closely matching both the curvature and tail of the survival time distribution.

B. Real-world examples

The uniform nature of random regular graphs makes our approximation very simple, since every edge is equivalent. However, unlike some other higher-order approximations, our approximations can be directly applied to arbitrary graphs. For any value of K , our approximation provides a system of linear equations for each edge in the network, and the set of all these equations can be solved by a simple fixed-point iteration scheme.

To explore how our approximations behave on more realistic structures, we compute them for two real-world network datasets. The first of these is a sexual contact network [33], while the second is a network of European roads [34]. In Fig. 4, we show the results of our approximation, and again these are compared with direct simulation of the SIS process. As in random regular graphs, our approximation quickly improves and converges as K increases, while $K = 8$ closely agrees with our simulation.

IV. THE $K \rightarrow \infty$ LIMIT

In our experiments so far, we found that the accuracy of our approximation increases quickly as K increases. On the other hand, increasing K also increases the complexity of our

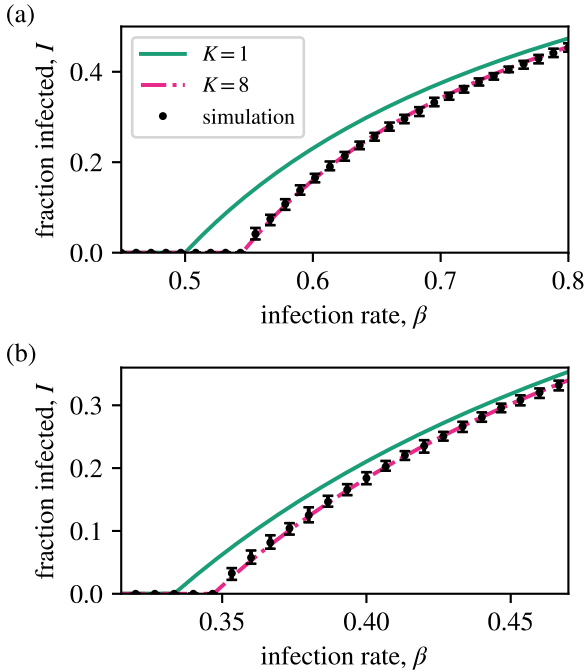


FIG. 2. Fraction of infected nodes at different rates of infection on (a) three-regular random graphs and (b) four-regular random graphs. We compare simulations on a 50 000 node network to our approximations with $K = 1$ and $K = 8$. We see a compelling improvement in the predictions.

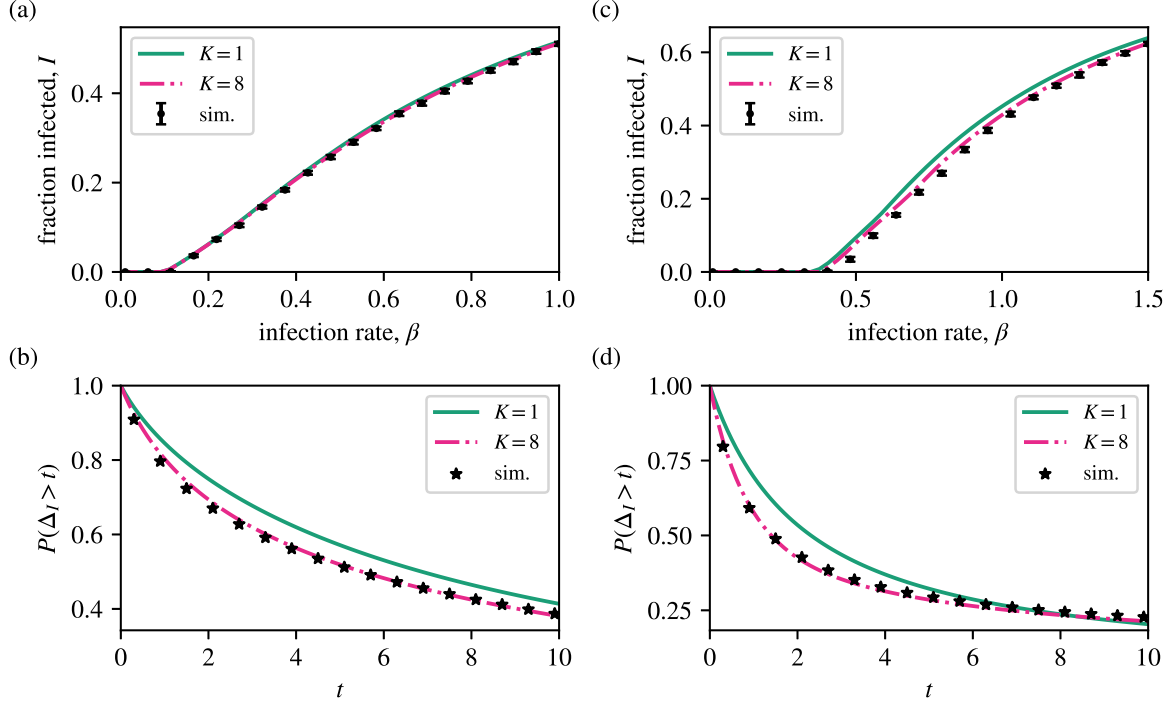


FIG. 4. Simulations on real-world networks. Panels (a) and (b) are the results of simulations on a sexual contact network of Ref. [33]. Panels (c) and (d) are the results of simulations on a network of roads, see Ref. [34]. In panels (a) and (c), we show the fraction infected after simulating for 10 000 units of time at varying values of β . In panels (b) and (d), we show the survival function at $\beta = 0.4$ and $\beta = 0.6$, respectively.

method, since in each iteration we need to solve a system of $L = (K + 1)^2$ linear equations to compute the stationary distribution of each pair, and doing this with standard linear algebra requires $O(L^3) = O(K^6)$ operations. While this growth in complexity is only polynomial, and we might be able to decrease it by exploiting the structure of the system; in practice, it is still sufficiently severe that we must settle for relatively small values such as $K = 8$.

Practicality notwithstanding, we may still inquire as to what would happen in the limit $K \rightarrow \infty$. In this case, the set of S states converges to a continuum, each one associated with the real-valued time since the node last became susceptible. The aging or decay between these states becomes deterministic, and the linear equations corresponding to finite K become differential equations.

For the random $(q + 1)$ -regular graph, all nodes and edges are statistically identical. To describe the limit $K \rightarrow \infty$ we use the following quantities and functions, which may vary with time t :

(1) S and I are the fraction of vertices that are susceptible and infectious, respectively.

(2) $g(x)$ is the density of vertices that are currently susceptible and have age x , i.e., which recovered and became susceptible at time $t - x$.

(3) $f(x, y)$ is the density of edges (i, j) where i and j are both susceptible and have ages x and y , respectively. We treat i, j and x, y as ordered pairs and count a given edge whose endpoints have ages x, y half toward $f(x, y)$ and half toward $f(y, x)$. Thus $f(x, y) = f(y, x)$ and the total fraction of edges where both endpoints are susceptible is $\iint dx dy f(x, y)$.

(4) $h(x)$ is the density of edges where one endpoint is infectious and the other is susceptible with age x , where we view these two as an unordered pair.

(5) ℓ is the fraction of edges where both endpoints are infectious.

We have several identities:

$$S + I = 1, \quad (35)$$

$$S = \int dx g(x), \quad (36)$$

$$\iint dx dy f(x, y) + \int dx h(x) + \ell = 1, \quad (37)$$

$$g(x) = \int dy f(x, y) + \frac{h(x)}{2}, \quad (38)$$

$$I = \frac{1}{2} \int dx h(x) + \ell. \quad (39)$$

To see Eqs. (38) and (39), think of choosing a random node by first choosing a uniformly random edge and then flipping a coin to choose between its two endpoints: this yields a uniformly random node since our network is regular. In Eq. (38), we use the fact that $f(x, y) = [f(x, y) + f(y, x)]/2$.

We also define $\sigma(x) = h(x)/g(x)$ as a kind of “surface-to-volume ratio” of the set of susceptible vertices, as a function of their age x . Specifically, if i is susceptible of age x , the probability that a random neighbor of i is infectious is $\sigma(x)/2$, namely, the fraction of $g(x)$ contributed by $h(x)/2$ in Eq. (38).

The time derivatives of these functions are as follows:

$$\begin{aligned} \frac{\partial f(x, y)}{\partial t} = & -\frac{\partial f(x, y)}{\partial x} - \frac{\partial f(x, y)}{\partial y} \\ & - \beta q \frac{\sigma(x) + \sigma(y)}{2} f(x, y) \\ & + \frac{1}{2} [\delta(x)h(y) + \delta(y)h(x)], \end{aligned} \quad (40)$$

$$\begin{aligned} \frac{\partial h(x)}{\partial t} = & -\frac{\partial h(x)}{\partial x} - \beta \left(1 + q \frac{\sigma(x)}{2}\right) h(x) \\ & + 2\beta q \int dy \frac{\sigma(y)}{2} f(x, y) + [2\delta(x)\ell - h(x)], \end{aligned} \quad (41)$$

$$\frac{\partial \ell}{\partial t} = \beta \int dx \left(1 + q \frac{\sigma(x)}{2}\right) h(x) - 2\ell. \quad (42)$$

Here $-\partial f/\partial x$, $-\partial f/\partial y$, and $-\partial h/\partial x$ are flow terms keeping track of the aging of susceptible vertices. The terms proportional to $\beta q \sigma/2$ are flows from $f(x, y) + f(y, x)$ to $h(x)$ and from $h(x)$ to ℓ : if an endpoint i of an edge (i, j) is susceptible with age x , then each of its q neighbors outside that edge is infectious with probability $\sigma(x)/2$ and infects i at rate β . There is an additional flow from $h(x)$ to ℓ of rate β since the disease can spread from the infectious endpoint to the susceptible one. Finally, the terms proportional to $\delta(x)$ are recovery events, where an infectious vertex becomes susceptible with age 0: the flow from ℓ to $h(0)$ has rate 2, and the flow from $h(x)$ to $f(x, 0) + f(0, x)$ has rate 1.

In Appendix B we show that solving for the steady state of Eqs. (40)–(42) is equivalent to finding auxiliary functions $w(x)$ and $\phi(x)$ that obey

$$\frac{d\phi}{dx} = -(\beta + 1)\phi(x) + \int_0^\infty \frac{\beta q \phi(u)\phi(|x - u|)}{[\gamma(u) + \phi(u)]w(u)} du, \quad (43)$$

$$\frac{dw}{dx} = \frac{\beta q \phi(x)}{\gamma(x) + \phi(x)} w(x), \quad (44)$$

$$1 = \frac{\beta}{\phi(0)} \int_0^\infty \left(1 + \frac{q\sigma(x)}{2}\right) \frac{\phi(x)}{w(x)} dx. \quad (45)$$

Unfortunately we are unsure how to solve this set of integro-differential equations except by numerical methods; we suggest this as a direction for future work. We have solved them numerically for a range of β and q , although we have some reservation about the approach. In particular, the numerical solver discretizes to a lattice $x_i = ix_{\max}/N$ for $i = 0, \dots, N$ and hence introduces artifacts both due to discretization and truncating the integrals at $x_{\max}$. Solving for multiple $x_{\max}$ and extrapolating to $x_{\max} = \infty$ appears to work well though it is not obvious how to rigorously quantify the numerical error.

To explore Eqs. (43)–(45), we extract predictions for β_c for $q = 2, 3, \dots, 12$. Results of this exercise are shown in Fig. 5. Of particular note, recall that we found (analytically) that $\beta_c^{(2)} = 1/q + 1/4q^3 + O(1/q^4)$. In agreement with this, the numerical solutions to the partial differential equation (PDE) appear consistent with

$$\beta_c^{(\infty)} = \frac{1}{q} + \frac{c}{q^3} + O\left(\frac{1}{q^4}\right) \quad (46)$$

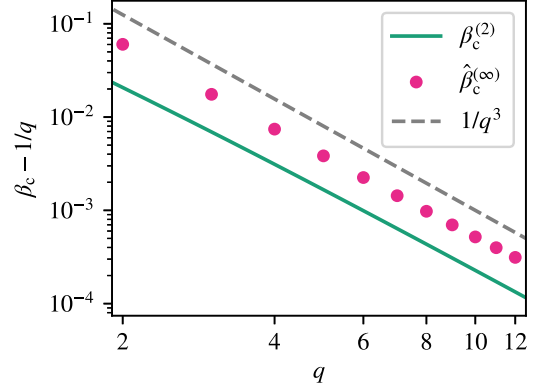


FIG. 5. Estimates of the critical spreading rate, β_c , for the random $(q + 1)$ -regular graph. The value $\beta_c^{(2)}$ is computed directly from Eq. (33). The estimate $\hat{\beta}_c^{(\infty)}$ is computed by first numerically solving the system in Eqs. (43) and (44) and then checking for consistent with Eq. (45). We show the results of a bisection search for the smallest value of β that produces a consistent solution.

for some $c > 0$. We conjecture that this is the true scaling for the critical value of the SIS model on random $(q + 1)$ -regular graphs: that is, in a large-degree approximation where we expand β_c in powers of $1/q$, we conjecture that c/q^3 is the first nonzero correction.

Finally, while these equations describe the large- K limit of our approach, we believe that they are still not exact, even on random regular graphs. In particular, we have only incorporated one kind of memory—the time since a node last recovered and became susceptible. If we further augmented the state space to track more past infections and recoveries at each node, we should approach the continuum limit of the dynamical cavity method.

V. DISCUSSION

By augmenting the state space to keep track of how long a node has been susceptible, and applying the pair approximation to these augmented states, we have derived a set of fixed-point equations to accurately approximate the behavior of the SIS model. On any finite network of interest, these equations can be iterated to convergence in order to make predictions without simulating the process, and thus approximate both the epidemic threshold and the endemic fraction of individuals. Additionally, on ensembles of networks such as random regular graphs or random graphs with a given degree distribution, these equations can be solved to approximate the SIS model on the corresponding ensemble.

Increasing the number K of susceptible states rapidly increases the accuracy of our approximation. As evidence for this, we compared our results to numerical simulation on random graphs and two real-world networks. In all cases we found good agreement between predictions and simulations for $K = 8$.

Our approach focuses on temporal correlations, namely, between the times that neighboring nodes have been susceptible and the probability their neighbors are infected. The computational cost of this method increases polynomially with K . In contrast, modeling longer-range spatial correlations

using k -tuples of nodes has complexity exponential in k . Additionally, we see no obvious and unique decomposition for arbitrary networks into sets of k -tuples, while the decomposition for the pair approximation simply corresponds to the set of edges.

While our approximations are very accurate in practice, we hypothesize that they are not exact even in the limit $K \rightarrow \infty$. To improve them further one could introduce additional states to keep track of more previous infection and recovery transitions, and thus capture more temporal correlations between a node's recent history and those of its neighbors. However, as in the dynamical cavity method the size of the state space would grow exponentially with the number of transitions. We leave this for future work.

Finally, we have focused on the SIS model but it seems hopeful that the technique we advanced will generalize to other settings, such as more sophisticated epidemic models, dynamic spin models, or lattice gases. This, again, we leave to future work.

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DATA AVAILABILITY

The data that support the findings of this article are openly available [36].

APPENDIX A: LINEAR STABILITY FOR PAIR APPROXIMATIONS

First, we derive a result, reminiscent of the Ihara-Bass formula [30], for the eigenvalues of a modified nonbacktracking operator. Specifically, let z_{ij} be an eigenvector of the operator

$$z_{ij} = wB_{ij}(z) + vB_{ji}(z) \quad (\text{A1})$$

and define

$$\mu_j = \sum_k A_{jk} z_{jk}. \quad (\text{A2})$$

Then

$$z_{ji} = \frac{w}{1+v}(\mu_i - z_{ij}) + \frac{v}{1+v}\mu_j, \quad (\text{A3})$$

and

$$\sum_j A_{ij} z_{ji} = \frac{w}{1+v}(d_i - 1)\mu_i + \frac{v}{1+v} \sum_j A_{ij} \mu_j, \quad (\text{A4})$$

where $d_i = \sum_j A_{ij}$ is the degree of node i .

Now consider the quantity

$$\begin{aligned} \sum_j A_{ij} \mu_j &= \sum_j A_{ij} \sum_k A_{jk} z_{jk} \\ &= \sum_j A_{ij} (z_{ji} + B_{ij}(z)) \end{aligned}$$

$$\begin{aligned} &= \sum_j A_{ij} \left(z_{ji} + \frac{1}{w} z_{ij} - \frac{v}{w} B_{ji}(z) \right) \\ &= \sum_j A_{ij} \left(z_{ji} + \frac{1+v}{w} z_{ij} - \frac{v}{w} \mu_i \right). \end{aligned} \quad (\text{A5})$$

Using Eqs. (A3) and (A4) we arrive at

$$\left[\left(\frac{w}{1+v} - \frac{v}{w} \right) (D - I) - \frac{1}{1+v} A + \frac{1}{w} I \right] \mu = 0, \quad (\text{A6})$$

where D is the diagonal matrix of node degrees, $D_{ii} = d_i$.

For the pair approximation to the SIS model we compare Eq. (A1) with Eq. (14) and set

$$w = \frac{\beta(\beta+2)}{2(\beta+1)} \text{ and } v = \frac{\beta^2}{2(\beta+1)},$$

and let $D = L + A$ for the Laplacian L to get

$$\left[I + \beta \left(I + \frac{\beta}{2} L - A \right) \right] \mu = 0. \quad (\text{A7})$$

Then the prediction for the critical value of β is the value that solves

$$\beta = \frac{1}{\lambda(A - \beta L/2 - I)}. \quad (\text{A8})$$

APPENDIX B: STEADY STATE OF THE PDES FOR THE $K \rightarrow \infty$ LIMIT

To find the steady-state solution (assuming it exists), we set all time derivatives to zero in Eqs. (40)–(42). We also define the following three auxiliary functions:

$$w(x) = \exp \left(\frac{\beta q}{2} \int_0^x \sigma(y) dy \right), \quad (\text{B1})$$

$$\phi(x) = h(x)w(x), \quad (\text{B2})$$

$$\gamma(x) = \int_0^\infty \frac{\phi(|x-y|)}{w(y)} dy. \quad (\text{B3})$$

To make progress, let us first consider the value of $f(x, y)$ along lines $(x, y) = (x_0 + s, y_0 + s)$. Setting $\partial f / \partial t = 0$ in Eq. (40) gives

$$\frac{df}{ds} = -\beta q \frac{\sigma(x_0 + s) + \sigma(y_0 + s)}{2} f(x_0 + s, y_0 + s), \quad (\text{B4})$$

which yields

$$\frac{f(x_0 + s, y_0 + s)}{f(x_0, y_0)} = e^{-\frac{\beta q}{2} \int_0^s [\sigma(x_0+u) + \sigma(y_0+u)] du}. \quad (\text{B5})$$

For arbitrary $x > y$, set $s = y$, $x_0 = x - y$, and $y_0 = 0$ to find

$$f(x, y) = f(x - y, 0) \frac{w(x - y)}{w(x)w(y)}. \quad (\text{B6})$$

Then, noting the boundary condition $f(x, 0) = h(x)/2$ and the symmetry in x and y , we get the steady-state

solution

$$f(x, y) = \frac{\phi(|x - y|)}{2w(x)w(y)}. \quad (\text{B7})$$

Hence if we can find $\phi(x)$ and $w(x)$ then we can immediately compute f , h , and ℓ .

From this equation for $f(x, y)$ we see that

$$\sigma(x) = \frac{2h(x)}{2 \int_0^\infty f(x, y)dy + h(x)} = \frac{2\phi(x)}{\gamma(x) + \phi(x)}, \quad (\text{B8})$$

and since $w' = \beta q \sigma w/2$ we have that

$$\begin{aligned} \frac{dw}{dx} &= \frac{dh}{dx}w(x) + \frac{dw}{dx}h(x) \\ &= w(x) \left(\frac{dh}{dx} + \frac{\beta q \sigma(x)}{2} h(x) \right). \end{aligned} \quad (\text{B9})$$

Inserting the identity for dh/dx from Eq. (41) (again, setting $\partial h/\partial t = 0$) we get

$$\frac{d\phi}{dx} = -(\beta + 1)\phi(x) + \frac{\beta q}{2} \int_0^\infty \frac{\sigma(u)\phi(|x - u|)}{w(u)} du, \quad (\text{B10})$$

$$\frac{dw}{dx} = \frac{\beta q}{2} \sigma(x)w(x), \quad (\text{B11})$$

with the boundary condition that $\phi(0) = 2\ell$.

Hence, if we can find $\phi(x)$ and $w(x)$ that solve these equations then we have putative expressions for the steady state f and h . However, we should also check that the equation for ℓ is satisfied, namely,

$$\frac{\beta}{\phi(0)} \int_0^\infty \left(1 + \frac{q\sigma(x)}{2} \right) \frac{\phi(x)}{w(x)} dx = 1. \quad (\text{B12})$$

If this holds, we have a complete set of equations for the steady state.

To solve these equations we iteratively update estimates for $w(x)$ and $\tilde{\phi}(x) = \phi(x)/\phi(0)$. [Note: $\tilde{\phi}(x)$ obeys the same differential equation as $\phi(x)$ but conveniently has $\tilde{\phi}(0) = 1$, so that we do not need to track ℓ inside the iteration.] The algorithm is as follows:

initialization.

$$\tilde{\phi}^{(0)}(x) = e^{-(\beta+1)x} \text{ and } w^{(0)}(x) = 1.$$

update.

$$\tilde{\gamma}^{(t)}(x) = \int_0^\infty \left(\frac{\tilde{\phi}^{(t)}(|x - y|)}{w^{(t)}(y)} \right) dy,$$

$$\sigma^{(t)}(x) = \frac{2\tilde{\phi}^{(t)}(x)}{\tilde{\gamma}^{(t)}(x) + \tilde{\phi}^{(t)}(x)},$$

$$c^{(t)}(x) = \frac{\beta q}{2} \int_0^\infty \left(\frac{\tilde{\phi}^{(t)}(|x - y|)\sigma^{(t)}(y)}{w^{(t)}(y)} \right) dy,$$

$$w^{(t+1)}(x) = \exp\left(\frac{\beta q}{2} \int_0^x \sigma^{(t)}(y) dy\right),$$

$$\tilde{\phi}^{(t+1)}(x) = e^{-(\beta+1)x} \left(1 + \int_0^x e^{(\beta+1)y} c^{(t)}(y) dy \right).$$

To compute the required quantities numerically we discretize to a uniform grid $x_i = ix_{\max}/N$ for $i = 0, \dots, N$. The convolutions for $\gamma^{(t)}$ and $c^{(t)}$ are approximated using a fast Fourier transform and the integrals for $w^{(t+1)}$ and $\tilde{\phi}^{(t+1)}$ are approximated using Simpson's rule. After the convergence of $\phi(x)$ and $w(x)$, we check to see if Eq. (B12) is obeyed. If so, we have found a nontrivial steady state. If (B12) is violated we assume that a nontrivial steady state does not exist, i.e., the process is subcritical.

Each value of $x_{\max}$ makes slightly different predictions. To find the critical β , we rerun calculations for a range of $x_{\max}$ and extrapolate Eq. (B12) by fitting

$$\beta \int_0^{x_{\max}} \left(1 + \frac{q\sigma(x)}{2} \right) \frac{\tilde{\phi}(x)}{w(x)} dx = \alpha_0 + \frac{\alpha_1}{x_{\max}}, \quad (\text{B13})$$

and if $\alpha_0 < 1$ we assume the process is still subcritical when $x_{\max} \rightarrow \infty$. This results in the estimates of $\beta_c^{(\infty)}$ shown in Fig. 5.

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