Edge Removal in Random Contact Networks and the Basic Reproduction Number

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Abstract

Understanding the effect of edge removal on the basic reproduction number $\mathcal{R}_0$ for disease spread on contact networks is important for disease management. The formula for the basic reproduction number $\mathcal{R}_0$ in random network SIR models of configuration type suggests that for degree distributions with large variance, a reduction of the average degree may actually increase $\mathcal{R}_0$. To understand this phenomenon, we develop a dynamical model for the evolution of the degree distribution under random edge removal, and show that truly random removal always reduces $\mathcal{R}_0$. The discrepancy implies that any increase in $\mathcal{R}_0$ must result from edge removal changing the network type, invalidating the use of the basic reproduction number formula for a random contact network. We further develop an epidemic model incorporating a contact network consisting of two groups of nodes with random intra- and inter-group connections, and derive its basic reproduction number. We then prove that random edge removal within either group, and between groups, always decreases the appropriately defined $\mathcal{R}_0$. Our models also allow an estimation of the number of edges that need to be removed in order to curtail an epidemic.

Keywords network, SIR model, basic reproduction number, disease dynamics, edge removal, multi-group, network evolution

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1 Introduction

The problem of mathematically describing the progression of an SIR disease is usually simplified by assuming that individuals in the population are well mixed [4]. Transmission events are then governed by a mass-action law, with the underlying assumption that encounters between any two individuals in the population occur with equal probability. Individuals belong to one of three states: susceptible, infective, and removed/recovered with immunity; the fraction of the population contained in each state is denoted by S, I, and R, respectively. A pairing of (or contact between) individuals is sufficient for the disease to jump from an infective to a susceptible, and all such pairings are assumed to happen with equal likelihood. If β is the rate in time at which pairings leading to infection occur, and γ is the rate of recovery, then the familiar Kermack-McKendrick SIR model, which is a special case of a much more general model presented in [7], is

$$S' = -\beta SI\,,\ I' = \beta SI - \gamma I\,,\ R' = \gamma I\,. \tag{1}$$

However, in a real world population certain pairings almost never happen, while other pairings are exceedingly common. Encounters between family members, spouses, and friends, for example, are far more probable than the average random pairing. By accounting for these close-knit connections in the population structure, one can expect to model disease spread more realistically. There are other pairings which occur less frequently but reliably, for example encounters with doctors or nurses in a clinic or hospital. These latter pairings become particularly important during an epidemic, and while the rate of transmission for such pairings may be different from the rate for more casual encounters, pairings with hospital employees cannot be prevented during time of disease.

A contact network is a network representation of the contact structures in a population, where individuals are represented by nodes, and if there are contacts between two individuals, there is an edge connecting the two corresponding nodes; see, for example, [13]. Following [13], we will call such

a random network a network of configuration type if there is a degree distribution P_k, $k = 0, 1, 2, \ldots$ such that a randomly chosen network node (a vertex) has, with probability P_k, k connections to other nodes. To construct a random graph with these properties, first chose the desired number of vertices N, then draw a degree sequence $\{k_i\}$ from the distribution and attach k_i "stubs" to the $i-$th node. Finally, randomly choose pairs of these stubs from two nodes that are not already neighbours, and connect them to form edges. The process of stub connection is repeated until no edge can be formed. Any remaining stubs are then discarded.

Disease threshold conditions, i.e., conditions that determine whether a disease can invade a population, are of tremendous public health interest. Traditionally, the basic reproduction number $\mathcal{R}_0$, which is the average number of secondary infections caused by a typical infectious individual during one's course of infection in a completely susceptible population, is the most commonly used one. Disease can invade if and only if $\mathcal{R}_0 > 1$. For network models, another commonly used threshold is the critical transmissibility T_c. Disease can invade if and only if the per edge transmission probability $T > T_c$. Assuming exponentially distributed waiting times for transmission and recovery events, $T = \frac{\beta}{\beta+\gamma}$. For disease dynamics on configuration type contact networks, the basic reproduction number $\mathcal{R}_0$ and the critical transmissibility T_c are defined below.

Using bond percolation theory, Newman [13] studied the final state of an epidemic on a random contact network of configuration type without degree correlation or clustering. The critical transmissibility was shown to be

$$T_c = \frac{\langle k \rangle}{\langle k^2 \rangle - \langle k \rangle}.$$

Newman further found that the disease may cause an epidemic if and only if the transmissibility along an edge is large enough, which is equivalent to $R_0 > 1$, where

$$\mathcal{R}_0 = T\left(\frac{\langle k^2 \rangle}{\langle k \rangle} - 1\right) = T\left(\langle k \rangle - 1 + \frac{\mathrm{Var}\,[k]}{\langle k \rangle}\right). \tag{2}$$

Here $\langle k \rangle$, $\langle k^2 \rangle$, and $\mathrm{Var}\,[k] = \langle k^2 \rangle - \langle k \rangle^2$ are the average, the second moment, and the variance of the degree distribution. Note that, at the beginning of an epidemic on a random network, because the degree distribution of a node found by following a random edge is $\{kP_k/\langle k \rangle\}$ where P_k is the

network degree distribution, the factor inside the parentheses is the average number of transmissible neighbors of the node after it is infected by one of its neighbors. Thus $\mathcal{R}_0$ is the basic reproduction number. Equation (2) shows that, in contact network models, $\mathcal{R}_0$ depends on both the transmissibility and the degree distribution. $\mathcal{R}_0 > 1$ is equivalent to the transmissibility threshold condition

$$T > T_c = \frac{\langle k \rangle}{\langle k^2 \rangle - \langle k \rangle} .$$

The first model describing the disease dynamics on contact networks was developed by Pastor-Satarrass and Vespignani [15] and is related to work of Anderson and May [10]. The Pastor-Satarrass and Vespignani model divides the population into degree classes (the number of contacts that an individual has), and assumes random mixing among these classes. It yields a larger basic reproduction number than (2), and thus predicts a faster growth of disease epidemic, because this model does not consider the fact that the disease cannot transmit along an edge more than once until one of its nodes recovers and becomes susceptible again. A few extensions of this model (see, for example, [1, 9], keep track of the number of "effective" (i.e., transmissible) neighbors, and yield basic reproduction numbers as in (2). These models employ a large number of equations and are therefore difficult to analyze. For SIR epidemics, Volz [17] developed a much simpler model tracking the number of edges that connect nodes of different infection status. This model was further simplified by Miller [12], who arrived at an effectively one-dimensional model. Both models yield basic reproduction numbers equivalent to (2). These papers thus confirm that, under the assumption of random contact networks with no degree correlation and clustering, the basic reproduction number of an SIR epidemic is given by (2). Lindquist et al. [9] showed that for diseases with no accquired immunity (SIS), the disease threshold is different from (2). However, for simplicity, in this present work we restrict ourselves to SIR epidemics.

It is a question of some significant practical relevance how the basic reproduction number will behave if the network is altered. These alteration may be caused by finding or losing friends, a change of jobs, or more interestingly, interventions such as vaccination, isolation and quarantine, and social distancing. It is a challenge to study the disease spread on an evolving network, because of the coupling of the disease dynamics and the network evolution dynamics. In this paper, we study the effect of dropping contacts (edges) before an epidemic. Doing so effectively decouples the two dynamics.

Because the transmission probability T is independent of the network structure, Equation (2) shows that the change of $\mathcal{R}_0$ is determined by the change of $\langle k \rangle$ and $\mathrm{Var}\,[k]$.

1.1 Counterintuitive results from a simple analysis of bi-modal networks: a paradox?

Common sense states that R_0 should decrease as edges are severed (this is the basic tenet of quarantine and isolation). In other words, one would expect $\mathcal{R}_0$ to be an increasing function of the average degree $\langle k \rangle$, and this is indeed true if the degree distribution of the random contact network is Poisson. In that case, $\langle k^2 \rangle = \langle k \rangle^2 + \langle k \rangle$, and $\mathcal{R}_0 = \frac{\beta}{\beta+\gamma} \langle k \rangle$. However, for other distributions this calculation does not apply. As x edges are randomly removed, $\langle k \rangle$ decreases with x. $\mathrm{Var}\,[k]$ is also a function of x, and therefore

$$\frac{d}{dx}\mathcal{R}_0 = T\left[\left(1 - \frac{\mathrm{Var}\,[k]}{\langle k \rangle^2}\right)\frac{d}{dx}\langle k \rangle + \frac{1}{\langle k \rangle}\frac{d}{dx}\mathrm{Var}\,[k]\right]. \tag{3}$$

This suggests that, if the variance of the degree distribution is kept constant, then $\mathcal{R}_0$ could increase when $\langle k \rangle^2 < \mathrm{Var}\,[k]$, i.e., when the variance is large, dropping edges may accelerate the epidemic. In fact, this effect becomes exaggerated if $\mathrm{Var}\,[k]$ increases with edge removal.

To illustrate this possibility we present a simple example of a network with bimodal degree distribution, i.e., a fraction p of the nodes with degree k_1, the other $(1-p)$ with degree $k_2 > k_1$. Assuming that T and p remain constant, we ask how $\mathcal{R}_0$ responds to changes in k_1 and k_2 in the following two scenarios.

1.1.1 Constant variance

Let k_1 and k_2 both increase by the same amount, keeping $d = k_2 - k_1$ constant. Consequently, the variance, $Var[k] = d^2 p(1-p)$ stays constant while the average degree increases.

Using $k_2 = k_1 + d$, we can write Equation (2) as

$$\mathcal{R}_0 = T\left(\frac{k_1^2 p + (k_1+d)^2(1-p)}{k_1 p + (k_1+d)(1-p)} - 1\right)$$

As we are interested only in the sign of $\frac{\partial \mathcal{R}_0}{\partial k_1}$, we discard the positive-valued factors T and the denominator of the derivative after differentiation. This

yields

$$\frac{\partial}{\partial k_1}\mathcal{R}_0 \propto 2[k_1 + d(1-p)]^2 - [k_1^2 p + (k_1+d)^2(1-p)]$$
$$\propto k_1^2 + 2k_1 d(1-p) + d^2(1-p)(1-2p)\,.$$

We can read off this formula that $\mathcal{R}_0$ decreases with $k_1 > 0$ if $p > 1/2$ and

$$0 < k_1 < d\left[(p-1) + \sqrt{p(1-p)}\right]\,.$$

Note that the right hand side is positive if $p > 1/2$. Conversely, $\mathcal{R}_0$ will increase as k_1 decreases inside the computed range.

1.1.2 Constant high degree

Here we let k_1 change, while k_2 is held constant. Thus, the standard deviation increases linearly with k_1, while the variance $(k_2 - k_1)^2 p(1-p)$ increases. Again, we have

$$\mathcal{R}_0 = T\left(\frac{k_1^2 p + k_2^2(1-p)}{k_1 p + k_2(1-p)} - 1\right)$$

and as we increase k_1,

$$\frac{\partial}{\partial k_1}\mathcal{R}_0 \propto k_1^2 p^2 + 2k_1 k_2 p(1-p) - k_2^2 p(1-p)$$

a quadratic in k_1. Thus, $\mathcal{R}_0$ decreases with increasing $k_1 > 0$ if

$$k_1 < k_2 \frac{\sqrt{1-p} - (1-p)}{p}\,.$$

We found that by manipulating the edge distribution in certain ways, $\mathcal{R}_0$ as defined above can increase in value despite a decreasing $\langle k \rangle$. This contradicts the basic tenet of quarantine — or does it? By decreasing the total number of edges in the network we are, in a sense, limiting the number of paths available to the disease, so one would expect a reduced growth rate. We leave the resolution of this paradox to the end of the paper, but give a hint. The problem with the above reasoning is that the removal of edges subject to the rules given above leaves us with networks which are no longer configuration type (see the discussion at the end), and in a network which

is not of this class, R_0 as defined above is no longer the basic reproduction number — one has to use a different definition.

In Section 2 we provide an argument which shows that R_0 will always decrease if the edge removal is truly random. In fact, we can show that Newman's R_0 is a Lyapunov functional for the system relative to a variable measuring random edge removal.

In Section 3 we extend the Miller network SIR model [11] to describe a population split into two subnetworks in order to allow random edge removal from just a subset of the entire population. We then derive the basic reproductive number $\mathcal{R}_0$ for the model and prove that $\mathcal{R}_0$ will indeed always decrease under random edge removal. The result from Section 2 is a critical ingredient in this analysis.

In summary, the conclusion of our work is that the basic tenet of quarantine holds rigorously for the models under consideration, and that conceivable exceptions are based on logical errors as are common in probabilistic models. We have chosen to include this "paradox" for motivational and pedagogical reasons.

2 Random edge removal in a random network

In this section we discuss random edge removal and its effect on the disease dynamics. Here we discuss two processes: one is to simply uniformly choose an edge for removal, the other is to first uniformly choose a node (disregarding its degree) and then uniformly choose one of its edges for removal. In the latter approach the edges are not uniformly chosen for removal. In fact, edges of low degree nodes will have a larger probability for removal than edges of high degree nodes. However, the second scenario may be more relevant for disease dynamics, as edge removal decisions are normally individual based rather than edge based.

2.1 Uniform edge removal

Assume that a fraction p of the edges will be removed. Because we assume that these edges are uniformly chosen for removal, each edge removal is thus a Bernoulli trial with success probability p. Assuming that the contact network has a degree distribution P_k (i.e., the probability that a node has degree k is

P_k), its probability generating function is

$$G(x) = \sum_{k=0}^{\infty} x^k P_k \,,$$

After the removal, the probability generating function for the degree distribution is then

$$G_r(x) = G(p + x(1-p))$$

Thus, after removal, the average degree is

$$\langle k \rangle_r = \frac{d}{dx} G_r(1) = (1-p)G'(1) = (1-p)\langle k \rangle \ ,$$

where $\langle k \rangle$ is the average degree before removal. In addition, the second moment is

$$\langle k(k-1) \rangle_r = \frac{d^2}{dx^2} G_r(1) = (1-p)^2 G''(1) = (1-p)^2 \langle k(k-1) \rangle \ .$$

Thus, the basic reproduction number $\mathcal{R}_0$ as a function of the removal probability p is

$$\mathcal{R}_0(p) = \frac{\beta}{\beta+\gamma} \frac{\langle k(k-1) \rangle_r}{\langle k \rangle_r} = (1-p) \frac{\beta}{\beta+\gamma} \frac{\langle k(k-1) \rangle}{\langle k \rangle}$$

which is a decreasing function of p. That is, truly random edge removal reduces $\mathcal{R}_0$.

2.2 Edge removal of a random node

When a random edge of a uniformly chosen node is removed, the above moment generating function method cannot be easily applied. In this case, we develop a model for the dynamics of degree distribution with edge removal.

Given the transmission probability T along an edge, the basic reproduction number (2) is only a function of the network degree distribution N_k (the *number* of nodes of degree k). We thus need to model how degree distribution evolves with edge removal. Let $N = \sum_{k=0}^{\infty} N_k$ be the total number of nodes, and $L = \sum_{k=0}^{\infty} k N_k$ be the sum of degrees (twice the number of edges) in the network. Here we use a simplified network evolution model [8] to describe

the evolution of the network degree distribution. For simplification, we pick the time scale such that the edge removal process occurs with rate one.

The probability that a random node of nonzero degree, selected for edge removal, has degree $k \geq 1$ is $N_k/(N - N_0)$. Having selected one of its edges, the probability that the neighbor has degree $k \geq 1$ is proportional to the sum of the degrees of all nodes in N_k, i.e., kN_k/L. Both nodes reduce their degree by one, thus entering N_{k-1} if they are in N_k. Hence, the dynamics of the degree distribution N_k can be modeled as

$$\frac{d}{d\tau}N_k = \frac{1}{N - N_0}\left(N_{k+1} - N_k\right) + \frac{1}{L}[(k+1)N_{k+1} - kN_k]\,,\ k \geq 1\,, \tag{4}$$

$$\frac{d}{d\tau}N_0 = \frac{N_1}{N - N_0} + \frac{N_1}{L}\,, \tag{5}$$

2.3 The rate of change of the basic reproduction number

From Equation (2),

$$\mathcal{R}_0 = T\left(\frac{\langle k^2\rangle}{\langle k\rangle} - 1\right) = T\left(\frac{\sum_{k=0}^{\infty} k^2 N_k}{\sum_{k=0}^{\infty} kN_k}\right)\,. \tag{6}$$

Using Equations (4) and (5), it can be derived that (see Appendix A),

$$\frac{d}{d\tau}\mathcal{R}_0 = 2T\left(\frac{1}{L} - \frac{1}{N - N_0}\right) < 0\,. \tag{7}$$

In fact, because by definition τ is a measure of the number of edges removed, given that the initial total degree is $L(0)$, the total degree at time τ is $L(\tau) = L(0) - 2\tau$. During the initial phase of edge removal, the probability that a node loses all its edges is small, and thus N_0 can be treated as a constant. We can thus solve Equation (7) approximately for small τ,

$$\mathcal{R}_0(\tau) - \mathcal{R}_0(0) = -T\left[\log\left(1 - \frac{2\tau}{L(0)}\right) + \frac{2\tau}{N - N_0}\right]\,. \tag{8}$$

Note that this fails as a good approximation of $\mathcal{R}_0$ when N_0 becomes large.

Equation (7) shows that the basic reproduction number $\mathcal{R}_0$ will decrease under random edge removal. This holds for any random network without degree correlation and clustering, regardless of degree distribution. Thus

we find some disparity with the examples of bimodal degree distributions in section 1.

Yet, for the first bimodal example, the number of edges removed from the low and high degree nodes are proportional to $\Delta k_1 p$ and $\Delta k_1(1-p)$, respectively. Clearly, when $p > 0.5$, the number of edges removed from the low degree nodes exceed those from high degree nodes. For the second example where the high degree is fixed while the low degree is reduced, only low-to-low edges can be removed. On the other hand, equations (4)–(5) are only applicable for random edge removal from the whole network, i.e., every node has the same probability to be selected for edge removal. We address this problem in Section 3

3 Edge removal from part of the network

The apparent paradox presented at the end of Section 2 arises because the unintuitive results presented in Section 1 are derived using the basic reproduction number formula suitable only for random contact networks generated from configuration models, yet the edge removal scenarios presented in Section 1 break the configuration model assumption. Thus, if we derive the correct formula for $\mathcal{R}_0$ for contact networks that are not of configuration model type, for example networks resulting from edge removal restricted to a component of a network, we should still see $\mathcal{R}_0$ decrease with random edge removal. In this section, we verify this conjecture.

We model edge removal from a network consisting of two groups of nodes, A and B, with random intra- and inter-group edges. Let N_A and N_B be the number of nodes in each group. For a node in group A, its edge is labeled either AA or AB if it connects to a neighbor in group A or B, respectively. The BB and BA edges are similarly labeled for target nodes in group B. We assume that the intra- and inter-group connections are random with no degree correlation. Further, for individual nodes, there must be no correlation between the number of intra and inter-group edges. Since each connection between group A and B is both of type AB and BA, the total number of AB edges must equal the total number of BA edges.

We assume that edge removal in either group, and between groups, may occur at different rates. In this section, we model the disease dynamics, derive the basic reproduction number, and study the change of the basic reproduction number with edge removal.

3.1 Disease dynamics

First we need to model the disease dynamics. We extend the Miller model [11], which describes the SIR disease dynamics on a random network without degree correlation and clustering, to two randomly connected subnetworks.

3.1.1 The Miller Model

Consider a susceptible node with degree k. This node remains susceptible as long as none of its k edges has transmitted disease. Let $\theta(t)$ be the probability that such an edge has not transmitted disease by time t, then the probability that this node remains susceptible is θ^k. We are interested in how fast this susceptible node becomes infected, which is solely determined by the dynamics of θ. In addition, while this node remains susceptible, the infection events along each edge are independent of each other. Thus, to understand the dynamics of θ, we can restrict the analysis to one of its edges, and assume that transmission can only be passed through this edge to the susceptible node. That is, the edge can be considered as if it was connected to a degree-1 susceptible node.

Let P_k be the degree distribution, which is generated by the probability generating function

$$\Psi(x) = \sum_{n=0}^{\infty} x^k P_k \,. \tag{9}$$

Then the probability that a randomly selected node remains susceptible at time t is $\Psi(\theta)$. Thus, the fraction of nodes that are susceptible at time t is

$$S = \Psi(\theta) \,. \tag{10}$$

We now describe the dynamics of θ. Let $P_I(t)$ be the probability that the neighbor connected by this edge is infectious at time t, and β be the transmission rate along an random edge. Then βP_I is the attack rate on the edge, and θ is the survival probability, thus,

$$\theta' = -\beta P_I \theta \,.$$

Let $\phi = P_I \theta$, which is the probability that a random edge connects a (degree-1, see the paragraph above) susceptible node to an infectious node. Then, the above equation becomes

$$\theta' = -\beta \phi \,. \tag{11}$$

We need to describe the dynamics of ϕ, i.e., the class of edges connecting a degree-1 suceptible node to a infectious node. An edge leaves class ϕ either because transmission occurred along it (with rate β), or the infectious node recovers (with rate γ). An edge of a susceptible node enters class ϕ because its other neighbor becomes infected, which happens at a rate $-h'(t)$, where $h(t)$ is the probability that we arrive at a susceptible node when following a random edge that has not transmitted disease. Thus,

$$\phi' = -\beta\phi - \gamma\phi - h'(t)\,.$$

We now model $h(t)$. Note that the probability that we arrive at a given node when following a random edge is proportional to its degree (because of the random network assumption). The probability that we arrive at a degree-k node is then $q_k = kP_k/\sum_{k=0}^{\infty} kP_k = kP_k/\Psi'(1)$. Thus, the probability that this node is susceptible is $\theta^{k-1}q_k$ and we arrive at

$$h(t) = \sum_{k=0}^{\infty} \theta^{k-1}\frac{kP_k}{\Psi'(1)} = \frac{\Psi'(\theta)}{\Psi'(1)}\,. \tag{12}$$

The equation for ϕ' can now be rewritten as

$$\phi' = -(\beta+\gamma)\phi - \frac{\Psi''(\theta)}{\Psi'(1)}\theta' = -(\beta+\gamma)\phi + \beta\phi\frac{\Psi''(\theta)}{\Psi'(1)}\,. \tag{13}$$

The dynamics of the disease are thus determined by (11) and (13). The fraction of nodes which are infectious at time t changes according to

$$I'(t) = -S' - \gamma I = \beta\phi\Psi'(\theta) - \gamma I\,. \tag{14}$$

3.1.2 Our two-group model

We now extend the Miller model to describe the disease dynamics on our two-group network. Assume that, in this two-group network, the distribution P_{AA} of the number of AA edges attached to a node in group A is generated by the probability generating function $\Psi_{AA}(x)$, defined as

$$\Psi_{AA}(x) = \sum_{i=0}^{\infty} P_{AA}(i)x^i\,,$$

The distributions P_{AB}, P_{BA}, and P_{BB} and their generating functions $\Psi_{AB}(x)$, $\Psi_{BA}(x)$ and $\Psi_{BB}(x)$ can be similarly defined. Thus, the balance condition equating the number of AB and BA edges can be written as

$$N_A \Psi'_{AB}(1) = N_B \Psi'_{BA}(1)\,, \tag{15}$$

where $\Psi'_{ij}(1)$, i, j =A and B, are the average number of neighbors in each type.

Let α be the rate of transmission along edges between A and B; β_A and β_B be the corresponding transmission rates within groups A and B, respectively. As before, we define γ to be the per-infective recovery rate.

For a susceptible node in group A, let $\theta_{AA}(t)$ be the probability that one of its edges has never transmitted disease by time t. The expression for θ_{AA} is similar to the Miller model. That is, if $\phi_{AA}(t)$ denotes the probability that an edge connected to a susceptible node in group A is connected to an infectious node and the edge has not transmitted disease, then

$$\theta'_{AA} = -\beta_A \phi_{AA}\,, \tag{16}$$

and

$$\phi'_{AA} = -(\beta + \gamma)\phi_{AA} - h'_{AA}(t)\,,$$

where $h_{AA}(t)$ is the probability that we arrive at a susceptible node in group A when following a random AA edge.

However, the dynamics of h_{AA} must reflect the fact that a neighbor of a group A node can be in either group A or group B. By the independence assumption made on the intra- and inter-group connections, the generating function for the degree distribution of a given node in group A is the product of the generating functions for its AA and AB degree distributions. Thus, having followed a random AA edge to arrive at a different node in group A, the probability that this node has i neighbors in A and j neighbors in B is

$$\left[iP_{AA}(i) / \sum_{k=0}^{\infty} kP_{AA}(k) \right] P_{AB}(j)\,,$$

and

$$\begin{aligned} h_{AA}(t) &= \left[\sum_{i=0}^{\infty} \theta_{AA}^{i-1} \frac{iP_{AA}(i)}{\sum_{k=0}^{\infty} kP_{AA}(k)} \right] \left[\sum_{j=0}^{\infty} \theta_{AB}^{j} P_{AB}(j) \right] \\ &= \frac{\Psi'_{AA}(\theta_{AA})}{\Psi'_{AA}(1)} \Psi_{AB}(\theta_{AB})\,. \end{aligned}$$

Hence, the dynamics of ϕ_{AA} becomes

$$\begin{aligned}\phi'_{AA} =& -\gamma\phi_{AA} - \beta_A\phi_{AA} + [-h'_{AA}(t)] \\ =& -\gamma\phi_{AA} - \beta_A\phi_{AA} + \beta_A\phi_{AA}\frac{\Psi''_{AA}(\theta_{AA})}{\Psi'_{AA}(1)}\Psi_{AB}(\theta_{AB}) + \\ & \alpha\phi_{AB}\frac{\Psi'_{AA}(\theta_{AA})}{\Psi'_{AA}(1)}\Psi'_{AB}(\theta_{AB})\,. \end{aligned} \tag{17}$$

We can follow the same reasoning and derive the probability that an edge of a group A susceptible node connected to a group B node has not transmitted disease at time t, $\theta_{AB}(t)$, and the probability that an edge of a susceptible group A node connected to an infectious node in group B yet has not transmitted disease by time t, $\phi_{AB}(t)$. In like manner we define $\theta_{BA}(t)$, $\phi_{BA}(t)$, $\theta_{BB}(t)$ and $\phi_{BB}(t)$.

$$\theta'_{AB} = -\alpha\phi_{AB}\,, \tag{18}$$

$$\theta'_{BA} = -\alpha\phi_{BA}\,, \tag{19}$$

$$\theta'_{BB} = -\beta_B\phi_{BB}\,, \tag{20}$$

$$\begin{aligned}\phi'_{BB} =& -\gamma\phi_{BB} - \beta_B\phi_{BB} + \beta_B\phi_{BB}\frac{\Psi''_{BB}(\theta_{BB})}{\Psi'_{BB}(1)}\Psi_{BA}(\theta_{BA}) \\ & \alpha\phi_{BA}\frac{\Psi'_{BB}(\theta_{BB})}{\Psi'_{BB}(1)}\Psi'_{BA}(\theta_{BA})\,. \end{aligned} \tag{21}$$

$$\begin{aligned}\phi'_{AB} =& -\gamma\phi_{AB} - \alpha\phi_{AB} + \alpha\phi_{BA}\frac{\Psi''_{BA}(\theta_{BA})}{\Psi'_{BA}(1)}\Psi_{BB}(\theta_{BB}) + \\ & \beta_B\phi_{BB}\frac{\Psi'_{BA}(\theta_{BA})}{\Psi'_{BA}(1)}\Psi'_{BB}(\theta_{BB})\,, \end{aligned} \tag{22}$$

$$\begin{aligned}\phi'_{BA} =& -\gamma\phi_{BA} - \alpha\phi_{BA} + \alpha\phi_{AB}\frac{\Psi''_{AB}(\theta_{AB})}{\Psi'_{AB}(1)}\Psi_{AA}(\theta_{AA}) + \\ & \beta_A\phi_{AA}\frac{\Psi'_{AB}(\theta_{AB})}{\Psi'_{AB}(1)}\Psi'_{AA}(\theta_{AA})\,, \end{aligned} \tag{23}$$

Consider a node in group A that has i neighbors in A and j neighbors in B. The probability that the node is susceptible is $\theta^i_{AA}\theta^j_{AB}$. The probability that a random node in group A is not infected from A is then $\sum_{i=0}^{\infty}\theta^i_{AA}P_{AA}(i)$, and similarly for infections from B. Thus, the fraction of susceptible nodes

in A is

$$S_A = \left[\sum_{i=0}^{\infty} \theta_{AA}^i P_{AA}(i)\right]\left[\sum_{j=0}^{\infty} \theta_{AB}^j P_{AB}(j)\right] = \Psi_{AA}(\theta_{AA})\Psi_{AB}(\theta_{AB})\,,$$

Similarly,

$$S_B = \Psi_{BB}(\theta_{BB})\Psi_{BA}(\theta_{BA})\,,$$

The fractions of infectious individuals in groups A and B change according to

$$\begin{aligned} I'_A &= -S'_A - \gamma I_A \\ &= \beta_A \phi_{AA} \Psi'_{AA}(\theta_{AA})\Psi_{AB}(\theta_{AB}) + \alpha \phi_{AB} \Psi_{AA}(\theta_{AA})\Psi'_{AB}(\theta_{AB}) - \gamma I_A\,. \quad (24) \\ I'_B &= -S'_B - \gamma I_B \\ &= \beta_B \phi_{BB} \Psi'_{BB}(\theta_{BB})\Psi_{BA}(\theta_{BA}) + \alpha \phi_{BA} \Psi_{BB}(\theta_{BB})\Psi'_{BA}(\theta_{BA}) - \gamma I_B\,. \quad (25) \end{aligned}$$

Equations (16)–(25) give the full model. Note that the dynamics of I_A and I_B are determined by the dynamics of θ and ϕ.

3.2 Comparison with stochastic simulations

To verify our model, we compare the numerical solutions of the model (17)–(25) to stochastic simulations of the underlying epidemic process. Given a network degree distributions for group A, we construct a random network without degree correlation and clustering using the configuration model [2, 3, 14]. Specifically, each node is assigned a number of stubs from the given degree distribution, then stubs from two different nodes which are not already neighbors are randomly connected to form an edge; this process is repeated until no stubs can be connected. Group B is constructed similarly. Then each node in A and B is assigned a number of stubs from the AB and BA degree distributions, respectively, and pairs of stubs from A and B are then connected at random to form inter-group edges, until none remain. Notice that the balance condition (15) must be satisfied in the choice of the AB and BA degree distributions. Each node is then labeled with a infection status, i.e., one of susceptible, infectious, and recovered. An infectious node stays infectious for an exponentially distributed time with mean $1/\gamma$, then its status is changed to recovered. A susceptible node stays susceptible for an exponentially distributed time with mean $1/(\beta i)$ where i is the number of

its infectious neighbors, then its status is changed to infectious. Recovered nodes remain recovered. The status of each node is updated until there is no infectious node or a given terminal time is reached. The simulation is implemented using the Gillespie algorithm [5, 6].

To compare the stochastic simulations with our ODE model (16)–(25), the degree distributions are fed into the model together with identical initial infections. The ODE model is then numerically solved, and $I(t) = I_A + I_B$ is compared with the average of the epidemic curves from the stochastic simulations.

Figure 1 shows that, on contact networks with various degree distributions in groups A and B, the epidemic curves from the ODE model agrees well with the ensemble averages of the epidemic curves from the stochastic processes.

3.3 Basic reproduction number

For $i, j =$A or B, let

$$\mathcal{R}_{ij} = \frac{\beta_{ij}}{\beta_{ij} + \gamma} \frac{\Psi''_{ij}(1)}{\Psi'_{ij}(1)} \,,$$

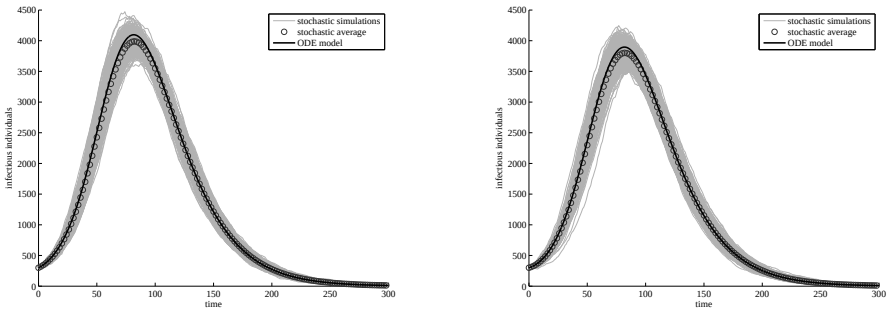
and

$$r_{ij} = \frac{\beta_{ij}}{\beta_{ij} + \gamma} \Psi'_{ij}(1) \,,$$

where $\beta_{AA} = \beta_A$, $\beta_{BB} = \beta_B$, $\beta_{AB} = \beta_{BA} = \alpha$. Note that $\beta_{ij}/(\beta_{ij} + \gamma)$ is the transmission probability along an edge between Groups i and j. At the beginning of an epidemic, $\Psi''_{ij}(1)/\Psi'_{ij}(1)$ is the average number of transmissible neighbors in Group j of a newly infected node in Group i that was infected by a node in Group j, and thus $\mathcal{R}_{ij}$ is the number of secondary infections in Group j caused by infectious nodes in Group i who have been infected by ones in Group j. Similarly, r_{ij} is the number of secondary infections in Group i caused by infectious nodes in Group i who have been infected by ones *not* in Group j. Thus the matrix

$$G = \begin{bmatrix} \mathcal{R}_{AA} & 0 & r_{AB} & 0 \\ 0 & \mathcal{R}_{BB} & 0 & r_{BA} \\ 0 & r_{BB} & 0 & \mathcal{R}_{BA} \\ r_{AA} & 0 & \mathcal{R}_{AB} & 0 \end{bmatrix} \tag{26}$$

is the second generation matrix. Its first two rows contain the secondary infections caused by nodes in group A and B who have been infected by



(a) Poisson random network (b) Bimodal random network

Figure 1: The epidemic curves of the ODE model (16)–(25) vs the ensemble averages of the stochastic simulations on two random networks: (a) Poisson distributed (λ =4, 2, 4, 10 for edge types AA, AB, BB, and BA, respectively), and (b) bimodal (with degrees fixed at 3, 2, 7, 10 for edge types AA, AB, BB, and BA, respectively). The population size of group A is $N_A = 25000$, and group B $N_B = 5000$, transmission rates $\beta_A = \beta_B = \alpha = 0.014$, recovery rate $\gamma = 0.05$. Note that the constraint (15) is satisfied.

nodes in the same group, respectively; and the last two rows are for cross-infected infectious nodes in A and B, respectively.

Using the second generation matrix method [16], the basic reproduction number for the two-group model is computed in Appendix B to be the spectral radius of the matrix G, i.e., $\mathcal{R}_0 = \rho(G)$. In general, this is not equivalent to Equation (2) because here the network has more structure than a random network.

Note that, when the network is bipartite (i.e., every edge inter-connects nodes in two groups), $\mathcal{R}_{AA}$, $\mathcal{R}_{BB}$, r_{AA}, and r_{BB} all vanish. In this case,

$$\mathcal{R}_0 = \sqrt{\mathcal{R}_{AB}\mathcal{R}_{BA}}.$$

It is shown in Appendix C Lemma 1 that $\mathcal{R}_0 > \mathcal{R}_{AA}$, $\mathcal{R}_0 > \mathcal{R}_{BB}$, and $\mathcal{R}_0 > \sqrt{\mathcal{R}_{AB}\mathcal{R}_{BA}}$, i.e., $\mathcal{R}_0$ is larger than the basic reproduction number of the disease when restricted to each component.

Assume that each type of edge is randomly removed (by choosing a node randomly then removing a random edge from that node), and this may occur with different rates for each type. Then the random edge removal model (4)–(5) describes the evolution for the degree distribution for each type of edge. From Appendix A, $\langle k \rangle = \Psi'_{ij}(1)$ and $\langle k^2 \rangle / \langle k \rangle = \Psi''_{ij}(1)/\Psi'_{ij}(1)$ for all $i, j =$A and B decrease with edge removal. In Appendix C it is shown that, because of this,

$$\frac{d}{d\tau} R_0 < 0$$

with edge removal. As a special case, removing edges from any part of the network reduces $\mathcal{R}_0$.

4 Discussion and Conclusions

It would appear obvious that in any population if the total number of potentially disease-causing contacts were to drop before an epidemic (say, as a result of vaccinations and the closing down of public places), so should the basic reproduction number. This follows from reasoning that if the average individual has fewer contacts, then the disease has fewer channels available by which to spread. Indeed, we proved in Section 2 that if the edge removal process is truly random, as given by the system of differential equations (4) and (5), then the basic reproduction number $\mathcal{R}_0$ decreases, as expected, when edges are removed.

In contrast, the mathematical arguments in the introduction seemed to indicate that for certain networks, we may see the opposite effect. In the case of a simple bimodal degree distribution, we described schemes for modifying the network in such a way that the average degree decreases, yet $\mathcal{R}_0$ as defined by (2) increases. Apparently all that is required is an edge-deletion process that causes either a tandem decrease in both the low degree k_1 and the high degree k_2, or a decrease in k_1 while k_2 remains fixed.

The reason for the "paradox" becomes clear if we pay closer attention to the definition of the basic reproduction number. The calculations from the introduction make use of the fact that for a random contact network of configuration type the basic reproduction number $\mathcal{R}_0$ is defined in (2).

However, as already indicated in the introduction, once we begin to remove edges subject to constraints the network changes character. Say, for example, we are ensuring that k_1 and k_2 change (or stay fixed) in a very particular way. To accomplish this, the edge deletion process must be selective rather than haphazard, and thus the network is no longer of the configuration type.

The $\mathcal{R}_0$ defined in (2) applies to networks which are randomly constructed from a given degree distribution. So by artificially manipulating the degree distribution, we in fact simulate a reconstruction of the network, rather than simple edge deletion. For this reason we cannot use this formula to illustrate a before and after picture of the basic reproduction number in networks which have had a few edges selectively deleted, but are otherwise structurally the same. In fact we are modeling a reorganization of the network, where the degree distribution is altered slightly and then the entire network is rebuilt.

As an example, consider the reorganization of a bimodal network where k_1 decreases and k_2 stays fixed. Imagine trying to decrement the degree of a low-degree node, chosen at random. We are required to select one of its edges and delete it. However, it is forbidden to remove edges belonging to a high-degree node. So if it happens that a high-to-low edge is selected, only the low-degree half may be discarded. The other half, a stub belonging to the high-degree node, must be reconnected somewhere else. The only way to "find a node" for it is to identify a second to-be-removed high-to-low edge, discard the low-degree half and then connect the leftover high degree stubs together, thus creating a new high-to-high edge. In this way we are reducing the average degree, while leaving k_2 fixed, as intended. However, in the end we have increased the proportion of edges that link high-degree nodes together, and the effect of this restructuring seems to outweigh the effect of a reduction in $\langle k \rangle$ for certain choices of k_1 and k_2. In a roundabout way, this illustrates the relative importance of connections amongst high-degree individuals to the spread of disease on networks.

The key point is, of course, that the definition of $\mathcal{R}_0$ in (2) loses its meaning once we introduce changes to the network that render the network "non-random". At that point one needs a different definition for a basic reproduction number.

To incorporate nonuniform edge removal, in Section 3, we extended the Miller model [11] model for a contact network consisting two groups of nodes with random intra- and inter-group connections; the transmission rates may differ inside and between the different parts of the network; the edges could be randomly removed from any part of the network. We then derive the basic

reproduction number as appropriate for this scenario, and were able to show that $\mathcal{R}_0$ will always decrease under edge removal. However this model may not be suitable for dividing a random network into two subgroups, because the resulting inter-group degree distributions may be correlated to intra-degree distributions, as confined by the total degree distribution (unless for a Poisson random network). On the other hand, this model can be extended to such cases by deriving the inter-group degree distribution from the total degree distribution and intra-degree distributions.

Most importantly, the dynamical system approach for the evolution of degree distribution under random edge removal allows us to estimate the point at which $\mathcal{R}_0$ drops below unity along the edge removal dynamics. That is, it tells us how many edges must be randomly removed to curtail an epidemic.

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A The rate of change of the basic reproduction number

The expression 7 is obtained as follows. From Equation (6), the rate of change of $\mathcal{R}_0$ along solutions to Equations (4) and (5) is

$$\frac{d}{d\tau}\left(T\frac{\sum_{k=0}^{\infty}k^2N_k}{\sum_{k=0}^{\infty}kN_k}\right) = T\left(\frac{\sum_{k=0}^{\infty}k^2N_k'}{L} - \frac{(\sum_{k=0}^{\infty}k^2N_k)(\sum_{k=0}^{\infty}kN_k')}{L^2}\right) \quad (27)$$

We substitute (4) in place of the N_k' terms. Notice that because of the coefficients k and k^2, all of the N_0' terms vanish in this substitution. We first

compute

$$
\begin{aligned}
&\frac{1}{L}\sum_{k=0}^{\infty} k^2 N_k' \\
=&\frac{1}{L(N-N_0)}\sum_{k=0}^{\infty} k^2(N_{k+1}-N_k) + \frac{1}{L^2}\sum_{k=0}^{\infty} k^2[(k+1)N_{k+1} - kN_k] \\
=&\frac{1}{L(N-N_0)}\sum_{k=1}^{\infty}[(k-1)^2 N_k - k^2 N_k] + \frac{1}{L^2}\sum_{k=1}^{\infty}[k(k-1)^2 N_k - k^3 N_k] \\
=&\frac{1}{L(N-N_0)}\sum_{k=1}^{\infty}(1-2k)N_k + \frac{1}{L^2}\sum_{k=1}^{\infty} k(1-2k)N_k \\
=&\frac{2}{L} - \frac{2}{N-N_0} - \frac{2}{L^2}\sum_{k=1}^{\infty} k^2 N_k \,.
\end{aligned}
$$

Then we compute

$$
\begin{aligned}
\sum_{k=0}^{\infty} kN_k' &= \frac{1}{N-N_0}\sum_{k=0}^{\infty} k(N_{k+1}-N_k) + \frac{1}{L}\sum_{k=0}^{\infty}[k(k+1)N_{k+1} - k^2 N_k] \\
&= \frac{1}{N-N_0}\sum_{k=1}^{\infty}[(k-1)N_k - kN_k] + \frac{1}{L}\sum_{k=1}^{\infty}[k(k-1)N_k - k^2 N_k] \\
&= -\frac{1}{N-N_0}\sum_{k=1}^{\infty} N_k - \frac{1}{L}\sum_{k=1}^{\infty} kN_k \\
&= -2\,.
\end{aligned}
$$

Thus,

$$
\frac{1}{L^2}\left(\sum_{k=0}^{\infty} k^2 N_k\right)\left(\sum_{k=0}^{\infty} kN_k'\right) = -\frac{2}{L^2}\sum_{k=0}^{\infty} k^2 N_k\,.
$$

It thus follows that

$$
\begin{aligned}
\frac{d}{d\tau}\mathcal{R}_0 &= T\left[\frac{2}{L} - \frac{2}{N-N_0} - \frac{2}{L^2}\sum_{k=1}^{\infty} k^2 N_k + \frac{2}{L^2}\sum_{k=0}^{\infty} k^2 N_k\right] \\
&= 2T\left(\frac{1}{L} - \frac{1}{N-N_0}\right)\,.
\end{aligned}
$$

Note that

$$\frac{1}{L} - \frac{1}{N - N_0} = \frac{1}{\sum_{k=1}^{\infty} kN_k} - \frac{1}{\sum_{k=1}^{\infty} N_k} < 0\,.$$

Thus,

$$\frac{d}{d\tau}\mathcal{R}_0 < 0\,.$$

B The basic reproduction number of the two-group model

To compute the basic reproduction number $\mathcal{R}_0$ for the two-group model (16)–(21), we employ the second generation matrix method [16]. This method identifies $\mathcal{R}_0$ as the dominant eigenvalue of the second generation matrix FV^{-1}. Here, for a general disease model with some susceptible and infected classes at the disease, we restrict our attention to the infected classes about the disease free equilibrium. The matrix F is the new infection matrix, whose ij entry is the rate of new infections entering class j caused by class i, and V is the transition matrix whose ij entry is the rate at which class i transfers to class j. And thus the ij entry of V^{-1} is the amount of time staying in class i starting from class j. This implies that the ij entry of the second generation matrix FV^{-1} is the average number of secondary infections in class i caused by class j. For our model, the ϕ classes are treated as "infected" classes.

To determined the matrices F and V, we linearize (17)–(23) about the disease-free equilibrium ($\phi_{AA} = \phi_{BB} = \phi_{AB} = \phi_{BA} = 0$ and $\theta_{AA} = \theta_{BB} = \theta_{AB} = \theta_{BA} = 1$) to get

$$\dot{\phi}_{AA} = -(\beta_A + \gamma)\phi_{AA} + \beta_A\phi_{AA}\frac{\Psi''_{AA}(1)}{\Psi'_{AA}(1)} + \alpha\phi_{AB}\Psi'_{AB}(1)$$

$$\dot{\phi}_{BB} = -(\beta_B + \gamma)\phi_{BB} + \beta_B\phi_{BB}\frac{\Psi''_{BB}(1)}{\Psi'_{BB}(1)} + \alpha\phi_{BA}\Psi'_{BA}(1)$$

$$\dot{\phi}_{AB} = -(\alpha + \gamma)\phi_{AB} + \alpha\phi_{BA}\frac{\Psi''_{BA}(1)}{\Psi'_{BA}(1)} + \beta_B\phi_{BB}\Psi'_{BB}(1)$$

$$\dot{\phi}_{BA} = -(\alpha + \gamma)\phi_{BA} + \alpha\phi_{AB}\frac{\Psi''_{AB}(1)}{\Psi'_{AB}(1)} + \beta_A\phi_{AA}\Psi'_{AA}(1)$$

In matrix form, this is

$$\frac{d}{dt}\begin{bmatrix}\phi_{AA}\\ \phi_{BB}\\ \phi_{AB}\\ \phi_{BA}\end{bmatrix} = (F-V)\begin{bmatrix}\phi_{AA}\\ \phi_{BB}\\ \phi_{AB}\\ \phi_{BA}\end{bmatrix}.$$

Here the terms related to new infections give

$$F = \begin{bmatrix} \beta_A \frac{\Psi''_{AA}(1)}{\Psi'_{AA}(1)} & 0 & \alpha \Psi'_{AB}(1) & 0 \\ 0 & \beta_B \frac{\Psi''_{BB}(1)}{\Psi'_{BB}(1)} & 0 & \alpha \Psi'_{BA}(1) \\ 0 & \beta_B \Psi'_{BB}(1) & 0 & \alpha \frac{\Psi''_{BA}(1)}{\Psi'_{BA}(1)} \\ \beta_A \Psi'_{AA}(1) & 0 & \alpha \frac{\Psi''_{AB}(1)}{\Psi'_{AB}(1)} & 0 \end{bmatrix},$$

and the terms not related to new infections give

$$V = \begin{bmatrix} \beta_A + \gamma & 0 & 0 & 0 \\ 0 & \beta_B + \gamma & 0 & 0 \\ 0 & 0 & \alpha + \gamma & 0 \\ 0 & 0 & 0 & \alpha + \gamma \end{bmatrix}.$$

Thus, FV^{-1} is the matrix specified in (26), and the basic reproduction number is its spectral radius.

C The monotonicity of the basic reproduction number of the two-group model

The characteristic equation of this matrix is a fourth order polynomial

$$\begin{aligned} f(x) =& (\mathcal{R}_{AA} - x)[(\mathcal{R}_{BB} - x)(x^2 - R_{AB}\mathcal{R}_{BA}) + r_{BB}\mathcal{R}_{AB}r_{BA}] + \\ & r_{AA}r_{AB}[(\mathcal{R}_{BB} - x)\mathcal{R}_{BA} - r_{BB}r_{BA}] \\ =& 0\,. \end{aligned} \tag{28}$$

The basic reproduction number $\mathcal{R}_0$ is thus the the largest root of $f(x) = 0$.

Lemma 1. *$\mathcal{R}_0 > \mathcal{R}_{AA}$, $\mathcal{R}_0 > \mathcal{R}_{BB}$, and $\mathcal{R}_0 > \sqrt{\mathcal{R}_{AA}\mathcal{R}_{BB}}$.*

Proof. Because of the symmetry of the system on A and B, i.e., exchanging A and B yields the same system, without loss of generality, we assume that $\mathcal{R}_{AA} \geq \mathcal{R}_{BB}$ (otherwise, we switch A and B in the proof).

$$f(\mathcal{R}_{AA}) = r_{AA}r_{AB}[(\mathcal{R}_{BB} - \mathcal{R}_{AA})\mathcal{R}_{BA} - r_{BB}r_{BA}] < 0$$

because both terms in the bracket are negative. Note that $f(\infty) = \infty$. Thus, the largest root of $f(x)$ satisfies $\mathcal{R}_0 > \mathcal{R}_{AA}$. Because of symmetry on A and B, $\mathcal{R}_0 > \mathcal{R}_{BB}$. Since $f(\mathcal{R}_0) = 0$, from (28),

$$\begin{aligned}\mathcal{R}_0^2 - \mathcal{R}_{AB}\mathcal{R}_{BA} =& \frac{(\mathcal{R}_0 - \mathcal{R}_{AA})r_{BB}\mathcal{R}_{AB}r_{BA}}{(\mathcal{R}_{AA} - \mathcal{R}_0)(\mathcal{R}_{BB} - \mathcal{R}_0)} + \\ & \frac{r_{AA}r_{AB}[(\mathcal{R}_0 - \mathcal{R}_{BB})\mathcal{R}_{BA} + r_{BB}r_{BA}]}{(\mathcal{R}_{AA} - \mathcal{R}_0)(\mathcal{R}_{BB} - \mathcal{R}_0)}.\end{aligned}$$

Note that each term on the right hand side is positive because $\mathcal{R}_0 > \mathcal{R}_{AA}$ and $\mathcal{R}_0 > \mathcal{R}_{BB}$. Thus, $\mathcal{R}_0 > \sqrt{\mathcal{R}_{AA}\mathcal{R}_{BB}}$. □

As stated in Section 3, when the four types of edges are randomly removed (possibly with different rates), for all $i, j =$A and B, $\frac{d}{d\tau}\mathcal{R}_{ij} < 0$ and $\frac{d}{d\tau}r_{ij} < 0$ with edge removal (where, as in Section 2, τ is the time in the edge removal process). We differentiate the expression $f(\mathcal{R}_0) = 0$ with respect to τ, which is equivalent to the number of edges removed, to investigate how $\mathcal{R}_0$ changes

under this process. The multivariable chain rule gives

$$\begin{aligned}\frac{d}{d\tau}\mathcal{R}_0 = &-\mathcal{R}'_{AA}\frac{(\mathcal{R}_{BB}-\mathcal{R}_0)(\mathcal{R}_0^2-R_{AB}\mathcal{R}_{BA})+r_{BB}\mathcal{R}_{AB}r_{BA}}{f'(\mathcal{R}_0)}\\ &-\mathcal{R}'_{BB}\frac{(\mathcal{R}_{AA}-\mathcal{R}_0)(\mathcal{R}_0^2-R_{AB}\mathcal{R}_{BA})+r_{AA}\mathcal{R}_{BA}r_{AB}}{f'(\mathcal{R}_0)}\\ &-\mathcal{R}'_{AB}\frac{[-\mathcal{R}_{BA}(\mathcal{R}_{BB}-\mathcal{R}_0)+r_{BB}r_{BA}](R_{AA}-R_0)}{f'(\mathcal{R}_0)}\\ &-\mathcal{R}'_{BA}\frac{[-\mathcal{R}_{AB}(\mathcal{R}_{AA}-\mathcal{R}_0)+r_{AA}r_{AB}](R_{BB}-R_0)}{f'(\mathcal{R}_0)}\\ &-r'_{AA}\frac{r_{AB}[(\mathcal{R}_{BB}-\mathcal{R}_0)\mathcal{R}_{BA}-r_{BB}r_{BA}]}{f'(\mathcal{R}_0)}\\ &-r'_{BB}\frac{r_{BA}[(\mathcal{R}_{AA}-\mathcal{R}_0)\mathcal{R}_{AB}-r_{AA}r_{AB}]}{f'(\mathcal{R}_0)}\\ &-r'_{AB}\frac{r_{AA}[(\mathcal{R}_{BB}-\mathcal{R}_0)\mathcal{R}_{BA}-r_{BB}r_{BA}]}{f'(\mathcal{R}_0)}\\ &-r'_{AB}\frac{r_{BA}[(\mathcal{R}_{AA}-\mathcal{R}_0)\mathcal{R}_{AB}-r_{AA}r_{AB}]}{f'(\mathcal{R}_0)}\,.\end{aligned}$$

Because $\mathcal{R}_0$ is the largest root of $f(x)$, which is a fourth order polynomial opening upward, $f'(\mathcal{R}_0) > 0$. Again, because $\mathcal{R}_0 \geq \mathcal{R}_{AA}$ and $\mathcal{R}_0 \geq \mathcal{R}_{BB}$, the last six fractions are all negative. And since $f(\mathcal{R}_0) = 0$,

$$\begin{aligned}&(\mathcal{R}_{BB}-\mathcal{R}_0)(\mathcal{R}_0^2-R_{AB}\mathcal{R}_{BA})+r_{BB}\mathcal{R}_{AB}r_{BA}\\ &\qquad\qquad = -\frac{r_{AA}r_{AB}[(\mathcal{R}_{BB}-R_0)\mathcal{R}_{BA}-r_{BB}r_{BA}]}{\mathcal{R}_{AA}-\mathcal{R}_0} < 0\,.\end{aligned}$$

Similarly.

$$(\mathcal{R}_{BB}-\mathcal{R}_0)(\mathcal{R}_0^2-R_{AB}\mathcal{R}_{BA})+r_{BB}\mathcal{R}_{AB}r_{BA} < 0\,.$$

Thus, the coefficients of all $\mathcal{R}_{ij}$ and r_{ij} for $i, =$A and B are all positive. This implies that, if all the derivatives $\mathcal{R}'_{ij} \leq 0$ and $r'_{ij} \leq 0$, and at least one is strictly negative, then

$$\frac{d}{d\tau}\mathcal{R}_0 < 0\,.$$

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