Supplementary Information for *Edge-Based Compartmental Modeling for Infectious Disease Spread Part I: An Overview*

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In this supplementary information, we give additional information for the edge-based compartmental modeling approach for the spread of susceptible-infected-recovered (SIR) diseases in different types of static and dynamic networks. We give a more detailed discussion of the use of a test node and the assumption that test nodes do not cause infections. We then discuss the calculation of $\mathcal{R}_0$, the behavior of our equations at early time (showing that the thresholds they predict are the same as those given by $\mathcal{R}_0$), and for some cases we give the final size prediction. Finally, we show that the MFSH models we have used are in fact equivalent to some more familiar existing models.

1 Selection of the test node

The basis of our approach is the claim that the probability a randomly selected test node u is susceptible, infected, or recovered is equal to the proportion of the population that is susceptible, infected, or recovered. This claim implicitly assumes that the epidemic size grows deterministically: if stochastic effects could cause the outbreak to die out or even be slightly delayed, this claim is false. The probability a random node u is infected by time t depends on whether an epidemic happens and, if so, how delayed it is. So as in any ODE approach, our model is exact only once the outbreak is large enough to behave deterministically.

Our assumptions that the susceptible proportion of the population equals the probability u is susceptible, the proportion infected equals the probability u is infected, and the proportion recovered equals the probability u is recovered allow us to move our focus away from the proportion in each state. Instead we focus on the probability that u is in each state. Our goal remains to determine the course of the epidemic in the entire population, but our method will be to focus on the equivalent problem of finding the probability the randomly chosen test node u has a given status.

In order to calculate the probability that u is susceptible, infected, or recovered, we find another equivalent problem which is mathematically simpler. We make a simplifying assumption which allows us to treat neighbors of u as independent. As it stands, if w infects u, then u can infect another neighbor v, meaning the satus of v and w are not independent. We ignore this dependency, that is, we ignore transmissions from u to its neighbors. To make this mathematically precise, we prevent u from transmitting infection to its neighbors. This has no impact until after u is infected, so it has no impact on the probability u is susceptible. It may affect the state of neighbors of u once u is infected, but it has no impact on the duration of infection of u, and so it does not alter the probability that u is infected or recovered. Consequently, this alteration of u has no impact on the probability that u is in any given state. Thus our result for S, I, and R is not affected by preventing u from causing infection.

Consequently, we can calculate S, I, and R as the probability that u is susceptible, infected, or recovered under the assumption that u is prevented from causing infection. The result will give the proportion of the population that is susceptible, infected or recovered in the original epidemic.

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2 Simulation

Both static networks and networks with mean field social heterogeneity satisfy the "time homogeneity" assumption of [8]. That is, given the properties of u and v, the *a priori* probability that u would transmit infection to v if u is infected is independent of the time at which u becomes infected. Consequently, for these cases we can use the Epidemic Percolation Network (EPN) approach of [7]. In this, we consider each node u in turn. We assume that u becomes infected and select the duration of infection from the appropriate exponential distribution of mean γ. Given the duration of infection, for every node v that u might infect, we calculate the probability that u infects v, and randomly determine whether u infects v, and if so, how long it takes. We then create a directed network by assigning edges from u to each node it would infect with the edge weighted by the associated duration. This directed network is an EPN.

To simulate an epidemic, we can choose a node to be the index case. We then follow the epidemic as it passes from each node to the nodes that it would infect. If the outbreak remains small, we discard it. This can be done efficiently using Dijkstra's algorithm [5]. To quickly identify a node which sparks an epidemic, we can take the EPN and find the strongly-connected components within it. Above the epidemic threshold there is a single giant strongly-connected component. Any node from its "in-component" (including any node within the giant strongly-connected component) would spark an epidemic. We choose any of these nodes randomly and use it as the index case.

The DVD, DFD, and DC models are harder to frame in terms of the EPN framework, so we use more traditional simulation techniques. We use a Gillespie-style event-driven algorithm [6] and calculate whether the next event is a transmission, recovery, edge creation or edge breaking. For the DFD model, edges break in pairs and neighbors are swapped. These calculations are considerably slower because there are many events to track, only a few of which are directly relevant to disease transmission.

3 $\mathcal{R}_0$, early growth, and final size

In this section, we briefly turn away from the deterministic ODE methods and use branching process arguments to calculate $\mathcal{R}_0$ for each population. We then return to the ODEs and linearize the equations about the equilibrium corresponding to a fully susceptible population. We calculate the early growth rate, show that it is consistent with the branching process $\mathcal{R}_0$ above, and identify appropriate initial conditions. Finally, for most of the models, we are able to calculate a final size relation.

The typically quoted definition of $\mathcal{R}_0$ is the number of new cases caused by a single randomly infected individual in a completely susceptible population. However, a more careful definition is necessary in cases where the average individual in the population may have different properties than the average infected individual early in the epidemic. The appropriate definition of $\mathcal{R}_0$ is the number of new cases an average infected individual causes early in an outbreak [4, 16, 12, 17]. In particular, for an epidemic on a network, a single node chosen randomly in the population and then infected will have (on average) $\langle K \rangle$ neighbors to infect, while early on the typical infected node has higher degree than a randomly chosen node and has at least one neighbor which is no longer susceptible. Early in an outbreak, the probability mass function for a newly infected node in the actual degree case to have degree k is $P_n(k) = kP(k)/\langle K \rangle$, while in the expected degree case the probability density function for a newly infected node to have expected degree κ is $\rho_n(\kappa) = \kappa\rho(\kappa)/\langle K \rangle$. Consequently, we must account for the fact that such a node has higher degree than average, but we must also account for the fact that such a node cannot infect the source of its infection.

In our calculation of the early growth, we (as expected) find that if $\mathcal{R}_0 < 1$, the disease has negative growth rate. We assume that the early growth is proportional to the leading eigenvector and use this to find appropriate initial conditions. In practice this is unnecessary because effectively any appropriate initial condition (with almost all individuals and stubs being in a susceptible state) quickly converges to the leading eigenvector. For our calculation of the final size, we are often able to identify a unique equilibrium corresponding to the state of the population after the disease has spread through. For some models this is not possible. As expected, if $\mathcal{R}_0 < 1$, we find that the only equilibrium corresponds to no large scale transmission, but if $\mathcal{R}_0 > 1$ there is an additional equilibrium which we can calculate to find the final size.

Most of our calculations for the early growth and final size are done under the assumption that an epidemic occurs and in the limit that the initial proportion infected goes to zero. Thus our results are inappropriate for $\mathcal{R}_0 < 1$. In calculating ϕ_S and ϕ_R in terms of θ, we found that they take particular forms. However, the imposed initial conditions could be different. In the growing epidemic case, these early perturbations become insignificant as the number of infections becomes much larger than the initial conditions. However, in the case of a decaying epidemic, the initial number of infections is always significant compared to the later number. So the variations never disappear. Thus if the initial conditions do not satisfy the formulae we derived, the later solution does not either. This can still be handled using the edge-based compartmental modeling approach. To correct for this in the CM model (and similar models) we would need to find the equation for $\dot{\phi}_S$ and $\dot{\phi}_I$ (resulting in a system more like the DFD equations).

3.1 Actual degree models

3.1.1 CM

$\mathcal{R}_0$ In a CM network the expected number of infections a newly infected node causes is $\mathcal{R}_0 = \sum_k P_n(k)(k-1)\beta/(\beta+\gamma)$ where $\beta/(\beta+\gamma)$ is the probability a node infects a neighbor prior to recovering. The reason for the $k-1$ is that a newly infected node has one neighbor (its infector) who is not susceptible, and so there are $k-1$ susceptible neighbors. So

$$\begin{aligned}\mathcal{R}_0 &= \sum_k P_n(k)(k-1)\frac{\beta}{\beta+\gamma} \\ &= \sum_k \frac{k(k-1)P(k)}{\langle K\rangle}\frac{\beta}{\beta+\gamma} \\ &= \frac{\beta}{\beta+\gamma}\frac{\langle K^2-K\rangle}{\langle K\rangle} \\ &= \frac{\beta}{\beta+\gamma}\frac{\psi''(1)}{\psi'(1)}\end{aligned}$$

which is a well-known result for static CM networks. This calculation is in agreement with previous results for CM networks [11, 14, 2, 3, 16].

In the special case of a network with a Poisson degree distribution, the probability of selecting a higher degree node and the reduction by one in the available number of susceptible neighbors exactly balance. So for the Poisson distribution $\langle K^2-K\rangle = \langle K\rangle^2$ and $\mathcal{R}_0 = \beta\langle K\rangle/(\beta+\gamma)$. However, this does not hold for more general distributions.

Early Growth and Initial Conditions We return to the deterministic equations

$$\begin{aligned}&\dot{\theta} = -\beta\theta + \beta\frac{\psi'(\theta)}{\psi'(1)} + \gamma(1-\theta)\,, \\ &\dot{R} = \gamma I\,, \qquad S = \psi(\theta)\,, \qquad I = 1-S-R\,.\end{aligned}$$

Clearly $\theta = 1$ is an equilibrium solution corresponding to no transmission. To test its stability, we linearize about $\theta = 1$. We set $\theta = 1+\epsilon$. At leading order we find

$$\dot{\epsilon} = \left(-\beta + \beta\frac{\psi''(1)}{\psi'(1)} - \gamma\right)\epsilon$$

So at early times $\epsilon = Ce^{\lambda t}$ where

$$\lambda = \beta\frac{\psi''(1)}{\psi'(1)} - (\beta+\gamma)$$

The equilibrium loses stability as λ transitions from negative to positive, $\beta\psi''(1)/\psi'(1)(\beta+\gamma)$, which is exactly the condition for $\mathcal{R}_0$ to transition from below 1 to above 1. Both methods predict the same threshold.

To find appropriate initial conditions for S, I, and R, we could simply take $S = \psi(\theta)$, and choose any nonnegative I and R such that $1 = S + I + R$. As we solve forward, any error in I and R decays exponentially quickly. If we wish to be more precise, we note that $\dot{I} = -\dot{S} - \gamma I$, and at leading order $\dot{S} = \dot{\theta}\psi'(\theta) = \lambda C e^{\lambda t}\psi'(1)$ to leading order. We will have $I = Ke^{\lambda t}$, and we need to find K in terms of C. We get $\lambda K e^{\lambda t} = -C\lambda\psi'(1)e^{\lambda t} - \gamma K e^{\lambda t}$. Solving this gives $K = -C\lambda\psi'(1)/(\gamma+\lambda)$, so the appropriate initial condition is

$$\theta(0) = 1 + C, \qquad S(0) = \psi(\theta(0)), \qquad I(0) = -\frac{C\lambda\psi'(1)}{\gamma+\lambda}, \qquad R(0) = 1 - I(0) - S(0)$$

where C is a small, negative number.

However, in practice, there is no need to do this. I and R have no role to play in determining θ. We simply require that $I + R = 1 - \psi(\theta)$ initially. Although our initial distribution of probability to I and R may differ from the true amount, it is a small effect initially and decays exponentially. So in practice we can use any convenient assumption.

Final Size To calculate the final size, we note that as the epidemic dies out, the derivatives must all go to zero. Thus we can set $\dot{\theta} = 0$ and solve for $\theta(\infty)$. Note that (if $\mathcal{R}_0 > 1$) this has two solutions, as there are two equilibrium conditions. In one equilibrium the disease has not been introduced and $\theta = 1$, while in the other the disease has spread and died out and $\theta < 1$. We want the smaller of the solutions, which corresponds to an epidemic occuring. We solve

$$\theta(\infty) = \frac{\gamma}{\beta+\gamma} + \frac{\beta}{\beta+\gamma}\frac{\psi'(\theta(\infty))}{\psi'(1)}$$

for the smaller solution. In practice, this can be done by using a guess $\theta_1 = 0$, and then plugging θ_i into the right hand side to find θ_{i+1}. This iteration converges quickly, and if $\mathcal{R}_0 > 1$, the attracting solution is the solution we want. The total fraction of the population infected in the course of an epidemic is $R(\infty) = 1 - \psi(\theta(\infty))$.

3.1.2 Actual Degree MFSH

$\mathcal{R}_0$ To find $\mathcal{R}_0$ for the actual degree formulation of the MFSH model, we consider a newly infected node early in the epidemic. The probability it has degree k is $P_n(k)$. Because it has a new set of neighbors at each moment, we do not have to account for the fact that it cannot infect the source of its infection, nor do we have to account for the fact that once it infects a neighbor, it cannot infect the neighbor again. Thus at all times it has k susceptible neighbors, so it causes new infections at rate βk for the entire time it is infected. The average duration of infection is $1/\gamma$, so the expected number of infections caused given k is $\beta k/\gamma$. Averaging over k, we have

$$\begin{aligned}
\mathcal{R}_0 &= \sum_k P_n(k)\frac{k\beta}{\gamma} \\
&= \frac{\beta}{\gamma}\sum \frac{k^2 P(k)}{\langle K\rangle} \\
&= \frac{\beta}{\gamma}\frac{\langle K^2\rangle}{\langle K\rangle} \\
&= \frac{\beta}{\gamma}\left(\frac{\psi''(1)}{\psi'(1)} + 1\right)
\end{aligned}$$

Early Growth and Initial Conditions We begin with the equations

$$\dot{\theta} = -\beta\theta + \beta\frac{\theta^2\psi'(\theta)}{\psi'(1)} - \theta\gamma\ln\theta$$
$$\dot{R} = \gamma I\,, \qquad S = \psi(\theta)\,, \qquad I = 1 - S - R$$

we proceed similarly to the CM case. We set $\theta = 1 + \epsilon$ and at leading order we find

$$\dot{\epsilon} = \left(-\beta + \beta\frac{2\psi'(1) + \psi''(1)}{\psi'(1)} - \gamma\right)\epsilon$$

After some rearrangement, we have $\dot{\epsilon} = [\beta - \gamma + \beta\psi''(1)/\psi'(1)]\epsilon$. So $\epsilon = Ce^{\lambda t}$ where

$$\lambda = \beta\frac{\psi''(1)}{\psi'(1)} + \beta - \gamma$$

The equilibrium loses stability exactly where $\mathcal{R}_0 = 1$. The remaining calculations are identical to those of the CM case, and we find that the appropriate initial conditions are as before except that the value of λ is different

$$\theta(0) = 1 + C, \qquad S(0) = \psi(\theta(0)), \qquad I(0) = -\frac{C\lambda\psi'(1)}{\gamma + \lambda}, \qquad R(0) = 1 - I(0) - S(0)$$

(recall C is a small, negative number). As before, any reasonable initial condition with θ close to 1, $S = \psi(\theta)$ and $I + R = 1 - S$ would be acceptable.

Final Size To find the final size of an epidemic, we set $\dot{\theta}$ to zero and solve for θ. We find

$$\theta(\infty) = \exp\left[-\frac{\beta}{\gamma}\left(1 - \frac{\theta(\infty)\psi'(\theta(\infty))}{\psi'(1)}\right)\right]$$

If $\mathcal{R}_0 > 1$ this has two solutions, one with $\theta = 1$, and one with $0 < \theta < 1$, which is the solution of interest. Once this is found, the total fraction infected is $R(\infty) = 1 - \psi(\theta(\infty))$.

Note that if $\psi(x) = x^k$ for some k, this corresponds to the MFSH model with all individuals having the contact rate $k\beta$, which is the MA model and $\mathcal{R}_0 = k\beta/\gamma$. We find

$$\theta = \exp\left(-\frac{\beta}{\gamma}[1 - \theta^k]\right)$$

Rewriting the left hand side as $\theta = S^{1/k} = (1 - R(\infty))^{1/k}$ and raising both sides to the k'th power, we have

$$1 - R(\infty) = \exp\left(-\frac{k\beta}{\gamma}R(\infty)\right)$$

Which is the well known final size relation for the MA model

$$R(\infty) = 1 - \exp(-\mathcal{R}_0 R(\infty))$$

3.1.3 DFD

$\mathcal{R}_0$ To calculate $\mathcal{R}_0$ for this model, consider a randomly chosen newly infected node early in the epidemic. It has degree k with probability $P_n(k)$. Initially this node has $k - 1$ available susceptible neighbors. Let us focus instead on the one edge from the infection source. The stub may result in more infections if the edge is broken and reformed. The probability that it breaks and reforms prior to recovering is $\eta/(\eta + \gamma)$. The probability that it then causes infection prior to recovering is $\beta/(\beta + \gamma)$. At this point the stub is connected

to an infected neighbor, the same state it was at the beginning of infection and the process repeats. So the probability this stub infects at least n nodes is r^n where $r = \eta\beta/[(\beta+\gamma)(\eta+\gamma)]$. Summing this gives an expectation of $r/(1-r)$ new infections for this stub. Now consider one of the $k-1$ stubs that are not the source of infection. The probability that this stub transmits infection at least once is $\beta/(\beta+\gamma)$. After this it is like the stub that received infection. Thus the expected number of infections such a stub causes is $[\beta/(\beta+\gamma)][1+r/(1-r)]$ which can be rearranged into $[(\eta+\gamma)/\eta][r/(1-r)]$.

Adding these together, we see that a newly infected node is expected to cause

$$\begin{aligned}
\mathcal{R}_0 &= \sum_k P_n(k)\left[\frac{r}{1-r} + (k-1)\frac{r(\eta+\gamma)}{\eta(1-r)}\right] \\
&= \frac{r}{1-r}\sum_k \frac{kP(k)}{\langle K\rangle}\left[1 + (k-1)\frac{\eta+\gamma}{\eta}\right] \\
&= \frac{r}{1-r}\left(1 + \frac{\eta+\gamma}{\eta}\frac{\langle K^2-K\rangle}{\langle K\rangle}\right) \\
&= \frac{\beta\eta}{\gamma(\beta+\eta+\gamma)}\left(1 + \frac{\eta+\gamma}{\eta}\frac{\langle K^2-K\rangle}{\langle K\rangle}\right) \\
&= \frac{\beta}{(\beta+\eta+\gamma)}\left(\frac{\eta}{\gamma} + \frac{\eta+\gamma}{\gamma}\frac{\langle K^2-K\rangle}{\langle K\rangle}\right) \\
&= \frac{\beta}{(\beta+\eta+\gamma)}\left(\frac{\eta}{\gamma} + \frac{\eta+\gamma}{\gamma}\frac{\psi''(1)}{\psi'(1)}\right)
\end{aligned}$$

where we have substituted for $r = \eta\beta/[(\beta+\gamma)(\eta+\gamma)]$.

Ealry Growth and Initial Conditions Our equations are

$$\begin{aligned}
\dot\theta &= -\beta\phi_I\,, \\
\dot\phi_S &= -\beta\phi_I\phi_S\frac{\psi''(\theta)}{\psi'(\theta)} + \eta\theta\pi_S - \eta\phi_S\,, \\
\dot\phi_I &= \beta\phi_I\phi_S\frac{\psi''(\theta)}{\psi'(\theta)} + \eta\theta\pi_I - (\beta+\gamma+\eta)\phi_I\,, \\
\dot\pi_R &= \gamma\pi_I\,, \qquad \pi_S = \frac{\theta\psi'(\theta)}{\psi'(1)}\,, \qquad \pi_I = 1-\pi_R-\pi_S\,, \\
\dot R &= \gamma I\,, \qquad S(t) = \psi(\theta)\,, \qquad I(t) = 1-S-R\,.
\end{aligned}$$

Here we have a higher dimensional problem, and the equilibrium of interest is $\theta = 1$, $\phi_S = 1$, $\phi_I = 0$, $\pi_S = 1$, and $\pi_I = 0$. We set $\theta = 1+\epsilon_1$, $\phi_S = 1+\epsilon_2$, and $\phi_I = \epsilon_3$. For π_S we use $\pi_S = \theta\psi'(\theta)/\psi'(1)$. For π_I, we set $\pi_I = \epsilon_4$ and use the fact that $\dot\pi_I = -\dot\pi_S - \gamma\pi_I$. We linearize about the equlibrium. We find

$$\begin{aligned}
\dot\epsilon_1 &= -\beta\epsilon_3 \\
\dot\epsilon_2 &= -\beta\epsilon_3\frac{\psi''(1)}{\psi'(1)} + \eta\frac{2\psi'(1)+\psi''(1)}{\psi'(1)}\epsilon_1 - \eta\epsilon_2 \\
\dot\epsilon_3 &= \beta\frac{\psi''(1)}{\psi'(1)}\epsilon_3 + \eta\epsilon_4 - (\beta+\gamma+\eta)\epsilon_3 \\
\dot\epsilon_4 &= \beta\frac{\psi'(1)+\psi''(1)}{\psi'(1)}\epsilon_3 - \gamma\epsilon_4
\end{aligned}$$

which can be rewritten as the matrix equation

$$\frac{\mathrm{d}}{\mathrm{d}t}\begin{pmatrix}\epsilon_1\\ \epsilon_2\\ \epsilon_3\\ \epsilon_4\end{pmatrix} = \begin{pmatrix} 0 & 0 & -\beta & 0\\ \eta\frac{2\psi'(1)+\psi''(1)}{\psi'(1)} & -\eta & -\beta\frac{\psi''(1)}{\psi'(1)} & 0\\ 0 & 0 & \beta\frac{\psi''(1)}{\psi'(1)} - (\beta+\gamma+\eta) & \eta\\ 0 & 0 & \beta\frac{\psi'(1)+\psi''(1)}{\psi'(1)} & -\gamma\end{pmatrix}\begin{pmatrix}\epsilon_1\\ \epsilon_2\\ \epsilon_3\\ \epsilon_4\end{pmatrix}$$

The standard solution technique for this is to find the largest eigenvalue of the matrix. It is relatively straightforward to show that 0 and $-\eta$ are always eigenvalues of this matrix. The other two turn out to be the eigenvalues of the 2×2 matrix forming the lower right corner. They are

$$\lambda_{1,2} = \frac{-(2\gamma+\beta+\eta-\beta\frac{\psi''(1)}{\psi'(1)}) \pm \sqrt{(2\gamma+\beta+\eta-\beta\frac{\psi''(1)}{\psi'(1)})^2 - 4\left(\gamma(\beta+\gamma+\eta) - \gamma\beta\frac{\psi''(1)}{\psi'(1)} - \eta\beta(1+\frac{\psi''(1)}{\psi'(1)})\right)}}{2}$$

For our above expression, if $\mathcal{R}_0 = 1$, then $\psi''(1)/\psi'(1) = (\gamma-\eta)/(\gamma+\eta) + \gamma/\beta$. Placing this into our expression above, the largest eigenvalue becomes 0. If $\psi''(1)/\psi'(1)$ is larger than this threshold ($\mathcal{R}_0 > 1$) then an epidemic can occur. If it is less than this threshold, this eigenvalue goes below zero, but there is still an eigenvector of zero whose eigenvector has ϵ_3 and ϵ_4 both zero (corresponding to ϕ_I and π_I both zero). If $\mathcal{R}_0$ is less than 1, the values of ϵ_3 and ϵ_4 will decay according to the largest eigenvalue whose eigenvector has nonzero entries in the appropriate component.

For initial conditions, if $\mathcal{R}_0 > 1$, we take λ to be the largest eigenvalue, and the vector (a,b,c,d) to be the corresponding eigenvalue. Then the appropriate initial conditions are found by taking $\theta = 1+\epsilon a$, $\phi_S = 1+\epsilon b$, $\phi_I = \epsilon c$, and $\pi_I = \epsilon d$ where $\epsilon \ll 1$. Note that we must choose our eigenvector such that $a < 0$. In practice however, so long as the initial amount of infection is taken to be very small, the initial conditions need not take this exact form; the solution will quickly converge to something of this form. If $\mathcal{R}_0 < 1$, then the appropriate initial conditions come from a linear combination of the eigenvector of 0 and the decaying eigenvector. The coefficient of the 0-eigenvector will be very small unless the initial introduction infected many individuals.

We note that in the previous cases, if $\mathcal{R}_0 < 1$, then the pertubation to θ decays, and θ returns to 1. Physically, this is an unrealistic result: it says that if transmission has already happened, then as time progresses, transmission is undone. Here we do not see that. If transmission has happened, then it does not decay, corresponding to the eigenvector of 0. The reason that this model is more correct, is that in the previous models, we found a relation between ϕ_I and θ. This relation implicitly assumes that the epidemic is growing. If it is not growing, then this relation does not hold. Eliminating the assumption from those models would result in additional equations for ϕ_S and ϕ_I, and the system would look more like the DFD model.

Final Size We have not been able to find a simple expression for the final size of an epidemic in this case. The system has multiple equilibria corresponding to possible states after the disease was introduced. In the previous cases, we were able to find a closed form for the relation between θ and ϕ_I. The assumption made there was equivalent to stating that the early growth is dominated by the largest eigenvector. In the previous cases, this assumption led to an analytic relation between ϕ_I and θ. In our case, we still want to make the equivalent assumption, which gives a constraint that determines which equilibrium is the final state. However, we have not found a way to impose the constraint analytically. Instead we must solve the system using initial conditions corresponding to a small number of cases to find the correct final size. Thus we only have the final size as a numerical prediction.

3.1.4 DC

To calculate $\mathcal{R}_0$ for the DC model, we first define r_a, r_d, and r_s to be the expected number of infections caused by a stub prior to recovery given that the stub is active and connected to a node other than the source, dormant, or active but connected to the source of infection at the time of infection.

It is straightforward to show that if the stub is active and connected to a node other than the source, the probability that the edge transmits prior to breaking or recovery is $\beta/(\beta+\gamma+\eta_2)$. The probability it breaks prior to recovery is $\eta_2/(\gamma+\eta_2)$. Once it breaks, it is equivalent to a stub that was dormant at infection. Thus $r_a = \beta/(\beta+\gamma+\eta_2) + \eta_2 r_d/(\gamma+\eta_2)$.

To find r_d, we note that a dormant stub must find a neighbor prior to recovery before it can cause any transmissions. Once this happens, it is equivalent to a stub that was active at infection. Thus $r_d = \eta_1 r_a/(\gamma+\eta_1)$. Combining this with our expression for r_a, we have

$$r_a = \frac{\beta(\eta_1+\gamma)(\eta_2+\gamma)}{\gamma(\gamma+\eta_1+\eta_2)(\beta+\gamma+\eta_2)}.$$

To find r_s, we note that infection cannot happen along that stub until the stub breaks and reforms at which point it is equivalent to an active stub, so $r_s = \eta_1\eta_2 r_a/[(\gamma+\eta_1)(\gamma+\eta_2)]$.

The probability that a stub is active is $\xi = \eta_1/(\eta_1+\eta_2)$ and the probability it is dormant is $\pi = 1-\xi$. The total number of infections a node with degree k_m is expected to cause is $r_s + (k-1)\xi r_a + (k-1)(1-\xi)r_d$. Since the probability a newly infected node has degree k_m is $P_n(k_m) = k_m P(k_m)/\langle K_m\rangle$, we find

$$\begin{aligned}
\mathcal{R}_0 &= \sum_{k_m} P_n(k_m)[(k_m-1)\xi r_a + (k_m-1)(1-\xi)r_d + r_s] \\
&= \sum_{k_m} \frac{k_m P(k_m)}{\langle K_m\rangle}[(k_m-1)\xi r_a + (k_m-1)(1-\xi)r_d + r_s] \\
&= \sum_{k_m} \frac{k_m P(k_m)}{\langle K_m\rangle}\left[(k_m-1)\left(\xi + (1-\xi)\frac{\eta_1}{\gamma+\eta_1}\right) + \frac{\eta_1\eta_2}{(\gamma+\eta_1)(\gamma+\eta_2)}\right] r_a \\
&= \left(\frac{\langle K_m^2 - K_m\rangle}{\langle K_m\rangle}\frac{\eta_1}{\eta_1+\eta_2}\frac{\gamma+\eta_1+\eta_2}{\gamma+\eta_1} + \frac{\eta_1\eta_2}{(\gamma+\eta_1)(\gamma+\eta_2)}\right) r_a \\
&= \frac{\beta}{\gamma}\left[\frac{\langle K_m^2 - K_m\rangle}{\langle K_m\rangle}\frac{\eta_1}{\eta_1+\eta_2}\frac{\eta_2+\gamma}{\beta+\gamma+\eta_2} + \frac{\eta_1\eta_2}{(\gamma+\eta_1+\eta_2)(\beta+\gamma+\eta_2)}\right] \\
&= \frac{\beta}{\beta+\eta_2+\gamma}\left(\frac{\langle K_m^2 - K_m\rangle}{\langle K_m\rangle}\frac{\eta_1}{\eta_1+\eta_2}\frac{\eta_2+\gamma}{\gamma} + \frac{\eta_1\eta_2}{\gamma(\gamma+\eta_1+\eta_2)}\right) \\
&= \frac{\beta}{\beta+\eta_2+\gamma}\left(\frac{\psi''(1)}{\psi'(1)}\frac{\eta_1}{\eta_1+\eta_2}\frac{\eta_2+\gamma}{\gamma} + \frac{\eta_1\eta_2}{\gamma(\gamma+\eta_1+\eta_2)}\right)
\end{aligned}$$

Early Growth and Initial Conditions We have not attempted to calculate the early growth rate because showing the details will not be particularly informative. The method is similar to that for the DFD model. If we wish to use appropriate initial conditions, we simply begin with θ approximately 1, ϕ_S approximately π, ϕ_D approximately $\theta - \phi_S$, π_S approximately π, and ξ_S approximately ξ. We can make all the R variables 0, and then set the I variables to $I = 1-S$, $\phi_I = \theta - \phi_S - \phi_D$, $\pi_I = \pi - \pi_S$, and $\xi_I = \xi - \xi_S$. This will converge relatively quickly to the appropriate eigenvalue. Alternately, we could solve the linear system and identify the appropriate eigenvalue and use it to find the initial conditions.

Final Size As in the DFD case, we need an additional constraint to identify the appropriate equilibrium. We do not have this constraint analytically, so we must solve the ODE system numerically to find the final size.

3.2 Expected Degree Models

3.2.1 MP

$\mathcal{R}_0$ We calculate $\mathcal{R}_0$ much as in the CM network. We focus on all individuals with a given expected degree κ: these nodes have a Poisson degree distribution, and the fact that those with higher degree are more likely to become infected exactly cancels the reduction in available contacts, and so the expected number of remaining contacts of a newly infected node with expected degree κ is κ. So the expected number of infections such a node causes is $\kappa\beta/(\gamma+\beta)$. To find $\mathcal{R}_0$, we must take a weighted average over the value of κ for newly infected individuals.

The probability a newly infected individual has expected degree κ is $\rho_n(\kappa)$. So we find

$$\begin{aligned}\mathcal{R}_0 &= \int_0^\infty \rho_n(\kappa)\frac{\kappa\beta}{\gamma+\beta} \\ &= \int_0^\infty \frac{\rho(\kappa)\kappa}{\langle K\rangle}\frac{\kappa\beta}{\gamma+\beta}\,\mathrm{d}\kappa \\ &= \frac{\left\langle \hat{K}^2\right\rangle}{\langle K\rangle}\frac{\beta}{\beta+\gamma} \\ &= \frac{\Psi''(1)}{\Psi'(1)}\frac{\beta}{\beta+\gamma}\end{aligned}$$

where $\left\langle \hat{K}^2\right\rangle$ denotes the average of κ^2. It turns out $\left\langle \hat{K}^2\right\rangle = \left\langle K^2-K\right\rangle$, so this result is the same as the CM result.

Early Growth and Initial Conditions To calculate the early growth, we take

$$\begin{aligned}&\dot{\Theta} = -\beta\Theta + \beta\frac{\Psi'(\Theta)}{\Psi'(1)} + \gamma(1-\Theta)\,, \\ &\dot{R} = \gamma I\,, \qquad S = \Psi(\Theta)\,, \qquad I = 1-S-R\,.\end{aligned}$$

and set $\Theta = 1+\epsilon$. At leading order we have

$$\dot{\epsilon} = \left(-\beta + \beta\frac{\Psi''(1)}{\Psi'(1)} - \gamma\right)\epsilon$$

We find $\epsilon = Ce^{\lambda t}$ where

$$\lambda = \beta\frac{\Psi''(1)}{\Psi'(1)} - (\beta+\gamma)$$

Looking at the threshold, we see that $\lambda = 0$ exactly where $\mathcal{R}_0 = 1$.

To find appropriate initial conditions, we follow the CM case and find

$$\Theta(0) = 1+C, \qquad S(0) = \Psi(\Theta(0)), \qquad I(0) = -\frac{C\lambda\Psi'(1)}{\gamma+\lambda}, \qquad R(0) = 1-I(0)-S(0)$$

where C is a small, negative number.

Final Size The final size of epidemics in MP networks can be calculated in much the same way as for CM networks. We set $\dot{\Theta} = 0$ and find

$$\Theta(\infty) = \frac{\gamma}{\beta+\gamma} + \frac{\beta}{\beta+\gamma}\frac{\Psi'(\Theta(\infty))}{\Psi'(1)}$$

Then $S(\infty) = \Psi(\Theta(\infty))$ and $R(\infty) = 1-S(\infty)$.

3.2.2 Expected Degree MFSH

$\mathcal{R}_0$ To find $\mathcal{R}_0$ for the actual degree formulation of the MFSH model, we consider a newly infected node early in the epidemic. The probability density function for the expected degree κ is $\rho_n(\kappa)$. Because it has a new set of neighbors at each moment, we do not have to account for the fact that it cannot infect the source of its infection, nor do we have to account for the fact that once it infects a neighbor, it cannot infect the neighbor again. Thus on average it has κ susceptible neighbors, so it causes new infections at average rate $\beta\kappa$ for the entire time it is infected. The average duration of infection is $1/\gamma$, so the expected number of infections caused given k is $\beta\kappa/\gamma$. Taking the average over all κ, we have

$$\begin{aligned}\mathcal{R}_0 &= \int_0^\infty \rho_n(\kappa)\frac{\kappa\beta}{\gamma}\,\mathrm{d}\kappa \\ &= \frac{\beta}{\gamma}\int_0^\infty \frac{\kappa^2\rho(\kappa)}{\langle K\rangle} \\ &= \frac{\beta}{\gamma}\frac{\left\langle \hat{K}^2\right\rangle}{\langle K\rangle} \\ &= \frac{\beta}{\gamma}\frac{\Psi''(1)}{\Psi'(1)}\end{aligned}$$

Early Growth and Initial Conditions Our governing equations are

$$\begin{aligned}&\dot{\Theta} = -\beta + \beta\frac{\Psi'(\Theta)}{\Psi'(1)} + \gamma(1-\Theta) \\ &\dot{R} = \gamma I\,, \qquad S = \Psi(\Theta)\,, \qquad I = 1-S-R\end{aligned}$$

Setting $\Theta = 1+\epsilon$, we have

$$\dot{\epsilon} = \left(\beta\frac{\Psi''(1)}{\Psi'(1)} - \gamma\right)\epsilon$$

So $\epsilon = Ce^{\lambda t}$ where

$$\lambda = \beta\frac{\Psi''(1)}{\Psi'(1)} - \gamma$$

We see that the threshold for $\lambda = 0$ is again the same as $\mathcal{R}_0 = 1$.

To find the initial conditions, we repeat our previous approach and find

$$\Theta(0) = 1+C, \qquad S(0) = \Psi(\Theta(0)), \qquad I(0) = -\frac{C\lambda\Psi'(1)}{\gamma+\lambda}, \qquad R(0) = 1-I(0)-S(0)$$

where C is a small, negative number.

Final Size To find the final size we set $\dot{\Theta} = 0$ and find that $\Theta(\infty)$ solves

$$\Theta = \frac{\beta}{\gamma}\left(1 + \frac{\Psi'(\Theta)}{\Psi'(1)}\right) + 1$$

Then we have $R(\infty) = 1 - \Psi(\Theta(\infty))$.

3.2.3 DVD

To calculate $\mathcal{R}_0$ for the DVD population, we begin by considering a newly infected node soon after disease is introduced. Because nodes are infected with probability proportional to their expected degree, the probability density function for a node to have expected degree κ given that it is newly infected is $\rho_n(\kappa) = \kappa\rho(\kappa)/\langle K\rangle$.

Given a newly infected node with expected degree κ, the expected number of additional neighbors (other than its infector) it has is also κ (as in the static MP case). For each of those neighbors, the probability that it transmits infection prior to recovering or breaking the edge is $\beta/(\beta+\eta+\gamma)$. So the expected number of transmissions to neighbors it has when the infection occurs is $\kappa\rho(\kappa)\beta/[\langle K\rangle(\beta+\eta+\gamma)]$.

However, the node also has the opportunity to infect neighbors that it gains during its infectious period. The probability that it creates a new edge before recovering is given by considering the recovery rate γ, and the edge creation rate $\kappa\eta$. We track edge creations before recovery. The probability that at least one edge creation occurs $\kappa\eta/(\gamma+\kappa\eta)$. More generally, the probability that at least n edge creations is $[\kappa\eta/(\gamma+\kappa\eta)]^n$. If it gains at least n neighbors, the probability that it infects the n-th neighbor before recovering or breaking the edge is $\beta/(\beta+\eta+\gamma)$. So the probability that a node creates an n-th neighbor and infects that neighbor is $[\beta/(\beta+\eta+\gamma)][(\kappa\eta)/(\gamma+\kappa+\eta)]^n$

The expected number of newly created neighbors which it infects can be found by summing the probability that a node creates and infects an n-th neighbor over all n. This gives $[\beta/(\beta+\eta+\gamma)]\sum_n[\kappa\eta/(\gamma+\kappa\eta)]^n = [\beta/(\beta+\eta+\gamma)][\kappa\eta/\gamma]$. Adding the expected number of new and original neighbors infected together, the expected number of infections a node with κ causes is $[\beta/(\beta+\eta+\gamma)]\kappa[1+\eta/\gamma]$. Taking a weighted average over all κ gives

$$\begin{aligned}\mathcal{R}_0 &= \int_0^\infty \frac{\kappa\rho(\kappa)}{\langle K\rangle}\kappa\frac{\beta}{\beta+\eta+\gamma}\frac{\eta+\gamma}{\gamma}\,\mathrm{d}\kappa \\ &= \frac{\beta}{\beta+\eta+\gamma}\frac{\eta+\gamma}{\gamma}\frac{\left\langle\hat{K}^2\right\rangle}{\langle K\rangle} \\ &= \frac{\Psi''(1)}{\Psi'(1)}\frac{\beta}{\beta+\eta+\gamma}\frac{\eta+\gamma}{\gamma}\end{aligned}$$

The terms in this expression may be interpreted as follows: $\left\langle\hat{K}^2\right\rangle/\langle K\rangle$ gives the expected value of κ for a newly infected node, $\beta/(\beta+\eta+\gamma)$ gives the probability that an edge which exists at any point during the infectious period will transmit infection prior to breaking or the infectious period ending, and $(\eta+\gamma)/\gamma = 1+[\eta/\gamma]$ gives the expected number of susceptible contacts per expected degree to exist at infection (1) or be created prior to recovery (η/γ).

Early growth We take the equations

$$\begin{aligned}\dot{\Theta} &= -\beta\Theta + \beta\frac{\Psi'(\Theta)}{\Psi'(1)} + \gamma(1-\Theta) + \eta\left(1-\Theta-\frac{\beta}{\gamma}\Pi_R\right), \\ \dot{\Pi}_R &= \gamma\Pi_I\,, \qquad \Pi_S = \Psi'(\Theta)/\Psi'(1)\,, \qquad \Pi_I = 1-\Pi_S-\Pi_R\,, \\ \dot{R} &= \gamma I\,, \qquad S = \Psi(\Theta)\,, \qquad I = 1-S-R\,.\end{aligned}$$

We set $\Theta = 1+\epsilon_1$ and $\Pi_R = \epsilon_2$. We note that $\dot{\Pi}_R = \gamma\Pi_I = \gamma(1-\Pi_S-\Pi_R)$. At leading order we have

$$\begin{aligned}\dot{\epsilon}_1 &= -\beta\epsilon_1 + \beta\frac{\Psi''(1)}{\Psi'(1)}\epsilon_1 - \gamma\epsilon_1 + \eta\left(-\epsilon_1 - \frac{\beta}{\gamma}\epsilon_2\right) \\ \dot{\epsilon}_2 &= \gamma\left(-\frac{\Psi''(1)}{\Psi'(1)}\epsilon_1 - \epsilon_2\right)\end{aligned}$$

which becomes

$$\frac{\mathrm{d}}{\mathrm{d}t}\begin{pmatrix}\epsilon_1\\ \epsilon_2\end{pmatrix} = \begin{pmatrix}\beta\frac{\Psi''(1)}{\Psi'(1)} - (\beta+\gamma+\eta) & -\frac{\eta\beta}{\gamma}\\ -\gamma\frac{\Psi''(1)}{\Psi'(1)} & -\gamma\end{pmatrix}\begin{pmatrix}\epsilon_1\\ \epsilon_2\end{pmatrix}$$

The eigenvalues of a 2×2 matrix solve $\lambda^2 - T\lambda + D$ where T is the trace and D the determinant. So the dominant eigenvalue is

$$\lambda = \frac{T+\sqrt{T^2-4D}}{2}$$

If $T > 0$, then the growth rate is positive. To show that $T > 0$ implies $\mathcal{R}_0 > 1$, note that $T > 0$ implies $\beta\psi''(1)/\psi'(1) > \beta + \gamma + \eta$. From this the product of the first two factors in our expression for $\mathcal{R}_0$ is greater than 1. Because $(\eta + \gamma)/\gamma > 1$, it follows that $\mathcal{R}_0 > 1$. If $T \leq 0$, then our equations predict growth if and only if $D < 0$. To complete our argument that the equations predict growth exactly when $\mathcal{R}_0 > 1$, we must show that if $T \leq 0$, then $\mathcal{R}_0 > 1$ is equivalent to $D < 0$. We can show that

$$D = -\beta(\eta + \gamma)\frac{\Psi''(1)}{\Psi'(1)} + \gamma(\beta + \gamma + \eta)$$

From this a small amount of algebra shows $D < 0$ is equivalent to $\mathcal{R}_0 > 1$. Thus regardless of the sign of T, $\lambda > 0$ exactly when $\mathcal{R}_0 > 1$, and conversely $\lambda < 0$ exactly when $\mathcal{R}_0 < 1$. So the predicted thresholds are the same.

To find the appropriate initial conditions, we can again take any sufficiently small reasonable initial condition and the particulars of the initial condition will be unimportant. Alternately, we can note that the solution for (ϵ_1, ϵ_2) must converge to $Ce^{\lambda t}\mathbf{v}$ where $\mathbf{v}$ is the eigenvector of the eigenvalue λ. This takes the value

$$\mathbf{v} = \begin{pmatrix} \lambda + \gamma \\ \gamma\frac{\Psi''(1)}{\Psi'(1)} \end{pmatrix}$$

From this it is straightforward to find the appropriate initial conditions using the approaches seen before.

Final Size At the end of the epidemic, no infected nodes remain, and so $I(\infty) = \Phi_I(\infty) = \Pi_I(\infty) = 0$. We have $\Pi_R(\infty) = 1 - \Pi_S(\infty) = 1 - \Psi'(\Theta(\infty))/\Psi'(1)$. Setting $\dot{\Theta} = 0$ we find

$$\Theta(\infty) = \frac{\beta}{\beta + \eta + \gamma}\left(\frac{\eta + \gamma}{\gamma}\frac{\Psi'(\Theta(\infty))}{\Psi'(1)} + \frac{\eta + \gamma}{\beta} - \frac{\eta}{\gamma}\right)$$

We can solve this for $\Theta(\infty)$ using iterative methods. The total fraction infected is

$$R = 1 - S = 1 - \Psi(\Theta(\infty))$$

4 Equivalence of MFSH models with pre-existing models

The basic equations for the MFSH model used by other authors [1, 9, 10, 13, 15] are

$$\begin{aligned} \dot{S}_k &= -\beta k S_k \zeta \\ \dot{I}_k &= \beta k S_k \zeta - \gamma I_k \\ \zeta &= \frac{\sum_k kP(k)I_k}{\langle K \rangle} \end{aligned}$$

However, in the actual degree case we have derived

$$\dot{\theta} = -\beta\theta + \beta\frac{\theta^2\psi'(\theta)}{\psi'(1)} - \theta\gamma\ln\theta \tag{1}$$

$$\dot{R} = \gamma I\,, \qquad S = \psi(\theta)\,, \qquad I = 1 - S - R \tag{2}$$

It is not immediately obvious that these are equivalent. To see that they are, we first reduce the dimensions of the first system. We note that the equation for $\dot{S}_k$ has as solution

$$S_k = e^{-\beta k\int_{-\infty}^{t}\zeta(t')\,\mathrm{d}t'}$$

We set $\alpha = e^{-\beta\int_{-\infty}^{t}\zeta(t')\,\mathrm{d}t'}$ and then $S_k = \alpha^k$. Our goal is to show that in fact, α solves the same equation as θ. We begin by noting that

$$\dot{\alpha} = -\beta\zeta\alpha$$

So $\zeta = -\dot{\alpha}/\beta\alpha$

We now move to finding $\dot{\zeta}$.

$$\begin{aligned}
\dot{\zeta} &= \frac{\sum_k kP(k)\dot{I}_k}{\langle K\rangle} \\
&= \frac{\sum_k kP(k)[\beta k S_k \zeta - \gamma I_k]}{\psi'(1)} \\
&= \beta\zeta\frac{\sum_k k^2 P(k)\alpha^k \zeta}{\psi'(1)} - \frac{\sum_k kP(k)\gamma I_k}{\psi'(1)} \\
&= \beta\zeta\frac{\sum_k (k^2-k+k)P(k)\alpha^k \zeta}{\psi'(1)} - \gamma\zeta \\
&= \beta\zeta\frac{\psi''(\alpha)\alpha^2 + \psi'(\alpha)\alpha}{\psi'(1)} - \gamma\zeta \\
&= \alpha\beta\zeta\frac{\psi''(\alpha)\alpha + \psi'(\alpha)}{\psi'(1)} - \gamma\zeta \\
&= \alpha\beta\zeta\frac{\frac{\mathrm{d}}{\mathrm{d}\alpha}(\alpha\psi'(\alpha))}{\psi'(1)} - \gamma\zeta
\end{aligned}$$

We substitute $\zeta = -\dot{\alpha}/\beta\alpha$ to express this as a derivative.

$$\begin{aligned}
\dot{\zeta} &= -\dot{\alpha}\frac{\frac{\mathrm{d}}{\mathrm{d}\alpha}(\alpha\psi'(\alpha))}{\psi'(1)} + \frac{\gamma}{\beta}\frac{\dot{\alpha}}{\alpha} \\
&= \frac{\mathrm{d}}{\mathrm{d}t}\left[-\frac{\alpha\psi'(\alpha)}{\psi'(1)} + \frac{\gamma}{\beta}\ln\alpha\right]
\end{aligned}$$

We can integrate this to find

$$\zeta = 1 - \frac{\alpha\psi'(\alpha)}{\psi'(1)} + \frac{\gamma}{\beta}\ln\alpha$$

(using the fact that $\zeta \to 0$ and $\alpha \to 1$ at early time) and so $\dot{\alpha} = -\beta\alpha\zeta$ becomes

$$\dot{\alpha} = -\beta\alpha + \beta\alpha^2\frac{\psi'(\alpha)}{\psi'(1)} - \alpha\gamma\ln\alpha$$

which means that α solves the same equation as θ for the fixed degree version of the MFSH equations. Since $S_k = \alpha^k$ is the same formula as we would find for S_k in terms of θ, this shows that in fact the two systems of equations are equivalent.

We are not the first to see that the usual system can be simplified into a handful of equations, but the approach we have used to derive these equations is new. Previous authors have simply observed that the S_k equation can be solved, done so, and then used a change of variables. The resulting equations are equivalent to our own, but are written in terms of slightly different variables. The advantage of our system is that the variables connect more easily to meaningful quantities, so it can be derived directly, and it can be related to the other edge-based compartmental models.

The usual model can be altered to allow for continuous contact rates, which would yield

$$\begin{aligned}
\dot{S}_\kappa &= -\beta\kappa S_\kappa \zeta \\
\dot{I}_\kappa &= \beta\kappa S_\kappa \zeta - \gamma I_\kappa \\
\zeta &= \frac{\int_0^\infty \kappa\rho(\kappa) I_\kappa \,\mathrm{d}\kappa}{\langle K\rangle}
\end{aligned}$$

A similar approach shows that this is equivalent to our expected degree formulation of the MFSH equations.

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