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Model Hierarchies in Edge-Based Compartmental Modeling for Infectious Disease Spread

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Abstract

We consider the family of edge-based compartmental models for epidemic spread developed in [11]. These models allow for a range of complex behaviors, and in particular allow us to explicitly incorporate duration of a contact into our mathematical models. Our focus here is to identify conditions under which simpler models may be substituted for more detailed models, and in so doing we define a hierarchy of epidemic models. In particular we provide conditions under which it is appropriate to use the standard mass action SIR model, and we show what happens when these conditions fail. Using our hierarchy, we provide a procedure leading to the choice of the appropriate model for a given population. Our result about the convergence of models to the Mass Action model gives clear, rigorous conditions under which the Mass Action model is accurate.

1 Introduction

The spread of epidemics through populations is affected by many factors such as the infectiousness of the disease, the duration of infection, the distribution of contacts through the population, and the typical duration of contacts. Typically for predicting epidemic spread or intervention effectiveness, we want an accurate, but simple, model that captures the relevant effects.

In our earlier work [11], we introduced the edge-based compartmental modeling approach for the spread of SIR diseases in populations with different contact dynamics. Table 1 summarizes the models and their underlying assumptions. In particular we showed that edge-based compartmental models can capture contact duration and social heterogeneity (variation in contact levels) simultaneously in mathematically and conceptually simple terms. The models we studied all assumed that the population was made up of individuals who were identical except for their contact levels. We modeled the population as a network, with nodes representing individuals joined by edges representing potentially transmitting contacts. We also assumed a simple disease, with transmission occurring at rate β per edge and recovery occurring at rate γ . In this paper we investigate the relationships between models and how to choose the simplest model appropriate for a given population.

The contact duration falls into three possibilities: it can be permanent, finite, or fleeting. In the permanent case, a contact that exists at any time has always existed and will always exist. In the finite case contacts may change over time. In the fleeting case, contacts are so brief that over any macroscopic time scale an individual samples a very large number of

neighbors, and so it is safe to assume that the total contact time with infected individuals matches its expected value.

The distribution of contact levels can be split into two types. In the first class of models we discuss (expected degree models), we assign an expected degree to a node, with different nodes having different values of $\langle k \rangle$. The probability that an edge exists between two nodes is proportional to the expected degrees of each node. Edges are created independently of one another, so the existence of an edge between u and v does not alter whether an edge can exist between u and w . Note that the expected degree can take any non-negative real value, and we assume that the distribution of $\langle k \rangle$ is given by the probability density function $P(k)$. If the contacts are permanent, then we arrive at a “Mixed Poisson” network: a node with a given expected degree has its actual degree chosen from a Poisson distribution. These are also called Chung-Lu Networks after [4]; these are a type of inhomogeneous random graphs [2, 5], and are almost identical to network classes introduced in [3,17]. If existing contacts break at rate λ and (independently) an individual with expected degree $\langle k \rangle$ forms new contacts at rate μ , then at any time the networks will be Mixed Poisson networks, but the structure will vary in time. The degree of any individual varies in time, but has average equal to its expected degree. These are the Dynamic Variable Degree networks. If the duration is so short that at every time the contacts are newly selected, then this is the Mean Field Social Heterogeneity model (in the expected degree formulation).

In the second class of models (actual degree models) we assign an actual integer number of contacts k , the degree, to each individual though in the case of the dormant contact model not all of these contacts must be active at all times. We think of an individual as having k stubs (or half-edges) which pair randomly with stubs of other nodes to form edges. In this case the existence of an edge between u and v removes an available stub from u , and so it affects the probability of an edge between u and w . The distribution of degrees is given by the probability mass function $P(k)$. If the contacts are permanent, this is a Configuration Model network [16]. If individuals break contacts in such a way that they immediately form a new contact with other individuals who are simultaneously breaking a contact, then degrees do not change: this is the Dynamic Fixed Degree model. If contacts break so quickly that at each moment the contacts are newly selected, then this is the Mean Field Social Heterogeneity model (in the fixed degree formulation). In a final model, the Dormant Contact model, individuals have a given number k_m (assigned randomly to each individual) of possible contacts, referred to as “stubs”. A given stub is either active (involved in a contact) or inactive (not in a contact). Inactive stubs join with other stubs at rate λ_1 and active stubs dissolve their contact at rate λ_2 . This Dormant Contact model reduces to any of the other expected or actual degree models in appropriate limits.

The distinction between the expected degree models and the actual degree models becomes apparent when we calculate the probability that an individual is susceptible. For the expected degree models, there is a continuum of risk levels and so we will have to calculate the per-expected degree probability of not having been infected. In contrast for the actual degree models the risk is discretized. The calculation is slightly different in each case, but the underlying concepts are the same. The expected degree models tend to be marginally more difficult conceptually, but they are simpler mathematically.

$\langle K \rangle$ is large while the rate of transmission per edge scales such that β is of order $1 / \langle K \rangle$ and the recovery rate γ is fixed, then the probability that any given edge transmits even once is small. The probability it transmits twice is negligible: the disease “sees” an edge at most once, so whether it is permanent, fleeting, or finite has no impact on disease spread. Thus we may treat the model as if the contacts are fleeting, which simplifies the equations. If further the contact distribution is such that the contact levels are generally close to the mean, then

we can neglect variation in contact levels, and turn to the simple mass action model of [6,1]. The precise condition required for this is somewhat technical: we must have $|\langle K^4 \rangle - \langle K \rangle^4| / \langle K \rangle^4 \ll 1$ and $\langle K \rangle \gg 1$ with $\langle K \rangle$ fixed. When this does not hold, there can be a significant deviation away from the mass action model.

In this paper we begin by providing a flow chart leading to selection of the appropriate model for a given population. Following the flow chart, we introduce the hierarchy of the models which underlies the flow chart. We describe the precise assumptions of the models and sketch their derivation. Throughout we assume that the epidemic is started by a very small initial proportion infected. We demonstrate some of the simpler parts of the hierarchy. After that, we consider some of the more difficult aspects of the hierarchy. We finally discuss some of the implications and limitations of our approach. In particular, we note that we give a simple heuristic for when the Mass Action equations are appropriate. In the Supplementary Information (SI) we provide more rigorous justifications for the claims in the hierarchy section. Because of its importance, we include the rigorous proof of conditions under which the Mass Action equations hold in the main text.

2 Model Selection

In Figure 1 we present a flow chart that can be used to select the appropriate model for a given population. The conditions depend on the degree distribution and the rate at which edges change. These are relatively straightforward to measure for a population [14,24,19]. From the observations of population and disease parameters, we can choose the simplest model to accurately represent disease spread in a given population. The equations for each model are developed in Section 3.

Most existing modeling of infectious disease spread are based on the Mass Action model [6]. This flow chart gives appropriate conditions under which this model is reasonable. In general, we must have most degrees close to the average degree, but at the same time either contacts must have high turnover or the probability of transmission per contact is very low. If these conditions hold, it is appropriate to use the MA model.

Before using this flow chart it is always prudent to be sure that the population does not violate other assumptions of the models. For example, different contact structure between age groups may require more consideration. If there are important features not captured by these models it may be possible to develop a custom model that captures the relevant detail (see [12,23]). Otherwise we may not be able to rely on edge-based compartmental models and may have to use simulation. Some features, such as non-constant rates of infection or recovery or a latent period can be captured by straightforward generalizations of the models.

Although most of these models are new, some of these (or closely related models) have been applied previously. An early version of the CM model was introduced in [21], and an earlier, closely related approach which only gives final size information has been widely used (*e.g.*, [15,9,10]). The DFD model has been used by [22,20] to study syphilis and HIV infections. The MFSH models have been used widely, primarily to understand HIV spread [1,7,8,13, 18]

The questions asked are somewhat vague in the sense that whether a number is large or not is somewhat a matter of opinion. In Section 3 we address this in more detail. We show how the results of the models converge as the parameter values change.

3 The model hierarchy

In this section we investigate the hierarchy of Figure 2 underlying the flow chart in Figure 1. We first give a brief overview of the standard simple model, the Mass Action model. We then consider the hierarchy of models. We find it convenient to consider the expected degree models before the actual degree models. We consider the models roughly in order of increasing complexity, explaining the underlying assumptions and sketching the derivation of the equations from [11]. If we can reduce the model to another model, we explain why this should happen and give some details of the mathematical explanation. More complete derivations are in the SI.

The Mean Field Social Heterogeneity models require further attention. We have two formulations, one in terms of expected degree $\langle K \rangle$ and the other in terms of actual degree k . We will not address this initially while we discuss the main structure of the hierarchy, but at the end of this section we show that the two formulations are mathematically equivalent in the sense that each can be derived from the other. So we do not distinguish between the two models in the hierarchy of Figure 2. We will also show that all other models reduce to Mean Field Social Heterogeneity in the large $\langle K \rangle$ limit if $\langle K \rangle$ and $\langle K^4 \rangle / \langle K \rangle^4$ are both constant. In turn we have conditions under which the Mean Field Social Heterogeneity model reduces to the Mass Action model. Given any model with $\langle K \rangle$ and $\langle K^4 \rangle / \langle K \rangle^4$ fixed, $\langle K \rangle$ and $\langle K^4 \rangle / \langle K \rangle^4$ 1, we arrive at the Mass Action model¹.

3.0.1 The Mass Action model—The Mass Action (MA) model assumes that all individuals have the same rate of contact formation k (or β) and edges (contacts) are sufficiently short-lived that we may neglect contact duration. The transmission rate per contact is γ , and so the combined transmission rate is $\beta\gamma$. If we take S , I , and R to be the proportion of the population that is infected, then figure 3 leads to

$$\dot{S} = -\beta IS, \quad \dot{I} = \beta IS - \gamma I, \quad \dot{R} = \gamma I$$

However, this may be simplified somewhat by noting that $S + I + R = 1$. We replace the equation for I with $I = 1 - S - R$

$$\dot{S} = -\beta IS, \quad \dot{I} = 1 - S - R, \quad \dot{R} = \gamma I$$

3.1 Expected degree models

We now study the expected degree models for which each node has an expected degree assigned using the probability distribution function $P(k)$. We allow k to be a continuous variable. At any given time, the probability that two nodes u and v share an edge is proportional to $k_u k_v$, and each edge is assigned independently of all others.

We briefly sketch the approach used to derive equations in these networks. Full details are in [11]. We define i as a function of time such that $1 - i$ represents the per-unit probability of having been infected. To be precise let u and v be nodes such that v has a slightly larger than u : $k_v = k_u + \epsilon$ with $\epsilon \rightarrow 0$. By calculating the increased risk to v , we can arrive at a

¹It is worth noting that in the *expected degree* formulations the average fourth power of the degree and the average fourth power of the expected degrees are not equal, but if the ratio with $\langle K \rangle^4$ tends to 1 for either, it does for the other as well.

formula for $s(u, t)$ the probability an individual with a given u is susceptible. The probability that u is susceptible is $s(u, t)$ and the probability that v is still susceptible is $s(u + v, t) = [1 - (1 - s(u, t))]s(v, t) + \mathcal{O}(t^2)$. So in the small t limit, $(1 - s(u, t))$ is the probability that the small amount of extra v has ever contributed an edge that has transmitted to v . Taking $t \rightarrow 0$, we have $s(u, 0) = (1 - s(u, 0))s(u, 0)$. So $s(u, t) = \exp[-(1 - s(u, 0))t]$ and the probability a random node is susceptible is $S(t) = \int_0^\infty s(u, t) \rho(u) du$ where

$$\Psi(x) = \int_0^\infty e^{-\kappa(1-x)} \rho(\kappa) d\kappa$$

The difference in the various expected degree models is in how long edges last: they may be permanent, fleeting or finite.

For each system, we sketch the derivation of the equations. A full derivation and comparison with simulations for each model appear in [11]. Our focus in this paper is on understanding how the systems relate to one another rather than details of the derivation. We begin by considering permanent edges.

3.1.1 Mixed Poisson—In the Mixed Poisson (MP) model, the population is assumed to be static, so that if a contact ever exists, then it has always existed and will always exist. The relevant flow diagram is in figure 4. We set s , i , and r to be the per-unit probabilities of having a susceptible, infected, or recovered neighbor that has not transmitted. For the MP model, $1 = s + i + r$. We can solve for $s = (1 - i)/\Psi(1)$, and show that $r = (1 - i)/\Psi(1)$ from which we can find i in terms of s . So $\dot{s} = -\beta s i$ becomes an equation just in terms of s . Putting this all together, the governing equations are

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

3.1.2 Mean Field Social Heterogeneity [expected degree formulation]—We now consider the opposite limit in which edges are fleeting. The Mean Field Social Heterogeneity (MFSH) model [1,7,8,13,18] generalizes the MA model by allowing for variations in contact rate among the people. At any given time the node is expected to have $\langle k \rangle$ edges, but they change over rapidly. The relevant flow diagram is shown in figure 5. This introduces some new variables, s , i , and r which are the probabilities a new contact is with a susceptible, infected or recovered individual. Unlike the MP case, $1 = s + i + r$. Through some simplifications similar to the MP case, we find $r = (1 - i)/\Psi(1)$, so $i = 1 - s - r = 1 - s - (1 - i)/\Psi(1) = (1 - s)/\Psi(1)$. This leads to a similar system of governing equations

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

These equations differ in only one term from the MP equations.

The only difference in the assumptions of the MFSH model and the MA model is that the MA model assumes all contact rates are the same. Indeed, if all expected degrees are the same $\langle k \rangle$, then $\Psi(x) = \exp[-\langle k \rangle(1 - x)]$. We set $R = (1 - s)/\Psi(1)$, $I = 1 - S - R$, and $S =$

(). We first note that with this (), we find $\dot{S} = -I$. Setting $\dot{R} = 0$ and taking the time derivatives of S and R , we see that $\dot{S} = -IS$ and $\dot{R} = I$. Thus we have arrived at the MA equations.

More generally we expect that if the variation in contact rate is sufficiently small, the MFSH model should behave like the MA model. In Section 3.3.3 we discuss this further.

We note that in [11], the final size derivation for this model has a sign error. Setting $\dot{R} = 0$, and solving, we find $\Theta = \frac{\beta}{\gamma} \left(-1 + \frac{\Psi'(\Theta)}{\Psi'(1)} \right) + 1$. Solving this equation and setting $R(\infty) = 1 - S(\infty)$ gives the final size.

3.1.3 Dynamic Variable-Degree—In the Dynamic Variable-Degree (DVD) model, an individual may create or terminate edges at any time. A node with expected degree $\langle k \rangle$ creates edges at rate $\gamma \langle k \rangle$. Any existing edge breaks at rate η , so a node with expected degree $\langle k \rangle$ will on average have $\langle k \rangle$ edges, though the value fluctuates. The flow diagram in figure 6 is similar to the previous diagrams. We can use the flow diagram to find a differential equation for $\langle k \rangle$, which can be solved in terms of R . However, we cannot analytically solve for R in terms of $\langle k \rangle$, so we require additional equations. The governing equations are

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

The new variables Π_S , Π_I , and Π_R give the probabilities that a newly formed edge will connect to a susceptible, infected, or recovered node respectively.

If the changeover rate is sufficiently fast we anticipate that the model should reduce to the MFSH model. This statement can be made precise by considering a susceptible node u . The node u is constantly creating new edges and breaking existing edges. Its risk of infection depends on how many infected neighbors it has. If edges are long-lasting, then knowing that u has not been infected suggests that its neighbors may still be susceptible. However, if an edge to an infected neighbor is likely to break before the neighbor transmits, then knowing that u is still susceptible says little about current neighbors. In mathematical terms this means if η/β is large, we anticipate that the DVD model reproduces the MFSH model. This is made more precise in the SI.

In the opposite limit, we would expect that having small η leads to an effectively static network, so the MP model should result. In fact this is true, but the precise condition is somewhat more subtle than might be anticipated. It is not enough that η/β and η/γ be very small because the epidemic can last for many generations. In practice the static model will work well at early times, but may fail at later times as the contact structure accumulates changes. We set t_0 to be a time early enough that the number of infections by time t_0 is very small, but large enough that shortly thereafter the total number of infections is no longer negligible. At some later time t the MP model will be reasonable if $(t - t_0)$ is small. A more precise condition that the MP model is reasonable if $(\beta I + \gamma R)/r \ll 1$ where r is the early exponential growth rate is also described in the SI.

We show convergence of the DVD model to the MP and MFSH heterogeneity model as $\eta \rightarrow 0$ or $\eta \rightarrow \infty$ in figure 7. The technical mathematical details showing this convergence are in the SI.

3.2 Actual degree models

For our second class of models, we assume that each node has an actual degree k assigned using the probability mass function $P(k)$. The degree must be a non-negative integer. Each individual is given k stubs, and at any time those stubs may join in pairs with stubs of other nodes to form edges. In most models we assume that the stubs are always in pairs (though the partner may change), so the degree is k . In the Dormant Contact model, we allow stubs to be active or dormant, and thus take k_m [distributed according to $P(k_m)$] to be the maximum degree of a node, with k_a and k_d the active and dormant degrees respectively $k_a + k_d = k_m$. In all of these models, we use $\psi(t)$ to denote the probability that a stub has not transmitted infection from a neighbor to its node by time t , and S , I , and R to denote the probabilities that a stub has not transmitted infection and currently connects to a node of the given status. The probability a node with a given k is susceptible is ψ^k , and taking a weighted average over all k , we find $S(t) = \sum_k \psi^k(t)$ where

$$\psi(x) = \sum_k P(k) x^k$$

As above, we sketch the derivations of the equations. Full details of the derivations and comparison with simulation are in [11]. We begin again with permanent contacts.

3.2.1 Configuration Model—The Configuration Model (CM) networks are similar to the MP networks. In a CM network, the exact degree of an individual is assigned. A node is given k stubs, assigned using the probability mass function $P(k)$. Once all stubs are assigned to nodes, stubs are randomly paired into edges. The resulting network is static. We define ψ to be the probability that the neighbor along a random stub from u has not transmitted infection to u (so $1 - \psi$ is the probability the neighbor has transmitted). Using the flow diagram in figure 8, we find that

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

This is similar to the MP model. In fact, it is possible to show that a MP network is a special case of the CM networks. In MP networks, a node u with expected degree $\langle k \rangle_u$ has its actual degree chosen from a Poisson distribution of mean $\langle k \rangle_u$ (in the limit of a large network). Thus

in an MP network the probability a node has degree k is $P(k) = \int_0^\infty [e^{-\kappa} \kappa^k / k!] \rho(\kappa) d\kappa$. Thus $\psi(x) = \sum_k P(k) x^k = \int_0^\infty e^{-\kappa} (\sum_k \kappa^k x^k / k!) \rho(\kappa) d\kappa = \Psi(x)$, and so the MP model emerges as a special case of the CM model.²

3.2.2 Mean Field Social Heterogeneity [actual degree formulation]—In the actual degree version of Mean Field Social Heterogeneity (MFSH), each individual has some number of stubs k assigned independently of other individuals. Stubs change edges quickly so that the neighbor at any given time has no bearing on who the neighbor is later. We use the flow diagram in figure 9. The new variables S , I , and R represent the proportion of all stubs that belong to susceptible, infected, or recovered nodes. A newly formed edge

²In finite networks, there are some correlations between degrees that are different between the models, but this disappears in the large network limit.

connects a node to a susceptible, infected, or recovered node with probabilities β_s , β_i , and β_r respectively. We can find that $\beta_r = -\beta_s / \beta_i$ from which we can find β_r in terms of β_s . This gives β_i in terms of β_s which allows us to find a differential equation for β_s in terms of β_s . The governing equations are

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

Using techniques similar to those for the expected degree formulation of the MFSH model, we can show that if all degrees are the same, then this reduces to the MA model. Similarly, if the degrees are sufficiently close to the mean degree in the sense that $\langle K^4 \rangle / \langle K \rangle^4 \rightarrow 1$ and both β_s and $\langle K \rangle$ are fixed, then the solution again converges to that of the MA model. In section 3.3.1 we show that in fact this model is equivalent to the expected degree formulation of the MFSH model.

3.2.3 Dynamic Fixed-Degree—In the Dynamic Fixed-Degree (DFD) model a node is given k stubs, which are paired with stubs of other nodes into edges. As time progresses, an edge may break, and the freed stubs immediately form edges with stubs from other edges that break at the same time, a process we refer to as “edge swapping”. The rate any edge breaks is η . From figure 10, the resulting equations are

$$\begin{aligned}\dot{\theta} &= -\beta\varphi_i, \\ \dot{\varphi}_s &= -\beta\varphi_i\varphi_s \frac{\psi''(\theta)}{\psi'(\theta)} + \eta\theta\pi_s - \eta\varphi_s, \\ \dot{\varphi}_i &= \beta\varphi_i\varphi_s \frac{\psi''(\theta)}{\psi'(\theta)} + \eta\theta\pi_i - (\beta + \gamma + \eta)\varphi_i, \\ \dot{\pi}_r &= \gamma\pi_i, \quad \pi_s = \frac{\theta\psi'(\theta)}{\psi'(1)}, \quad \pi_i = 1 - \pi_r - \pi_s, \\ \dot{R} &= \gamma I, \quad S(t) = \psi(\theta), \quad I(t) = 1 - S - R.\end{aligned}$$

Unlike previous models, these equations cannot be simplified into a single equation for β_s .

The DFD model plays the same role in the actual degree case that DVD model played in the expected degree case. It experiences similar limiting behavior. If β_s / β_i is large, we recover the MFSH model. Alternately the CM is an accurate approximation so long as $(t - t_0)$ is small where t_0 is a time around when the epidemic begins to infect significant numbers. Again, a more precise condition that $(\beta_i + \beta_r) / r \rightarrow 1$ where r is the early exponential growth rate is described in the SI.

Figure 11 shows the convergence of this model to the CM and MFSH models as $\beta_s / \beta_i \rightarrow 0$ or $\beta_s / \beta_i \rightarrow \infty$.

3.2.4 Dormant Contacts—We finally move to the Dormant Contact (DC) model which captures all the previous expected and actual degree models as limiting cases. In the DC model, each node is given k_m stubs [with k_m chosen using $P(k_m)$]. However, only a fraction of them are active. At any given time, the node will have k_a active stubs and k_d dormant stubs, so $k_m = k_a + k_d$ is the maximum number of active stubs. Active stubs become dormant at rate γ_2 and dormant stubs become active at rate γ_1 . We define $\psi(x) = \sum_{k_m} P(k_m) x^{k_m}$. Using figure 12, the governing equations are

$$\begin{aligned}
\dot{\theta} &= -\beta\varphi_I \\
\dot{\varphi}_S &= -\beta\varphi_I\varphi_S\frac{\psi''(\theta)}{\psi'(\theta)} + \eta_1\frac{\pi_S}{\pi}\varphi_D - \eta_2\varphi_S \\
\dot{\varphi}_I &= \beta\varphi_I\varphi_S\frac{\psi''(\theta)}{\psi'(\theta)} + \eta_1\frac{\pi_I}{\pi}\varphi_D - (\eta_2 + \beta + \gamma)\varphi_I \\
\dot{\varphi}_D &= \eta_2(\theta - \varphi_D) - \eta_1\varphi_D \\
\dot{\xi}_R &= -\eta_2\xi_R + \eta_1\pi_R + \gamma\xi_I, \quad \xi_S = (\theta - \varphi_D)\frac{\psi'(\theta)}{\psi'(1)}, \quad \xi_I = \xi - \xi_S - \xi_R \\
\dot{\pi}_R &= \eta_2\xi_R - \eta_1\pi_R + \gamma\pi_I, \quad \pi_S = \varphi_D\frac{\psi'(\theta)}{\psi'(1)}, \quad \pi_I = \pi - \pi_S - \pi_R \\
\xi &= \frac{\eta_1}{\eta_1 + \eta_2}, \quad \pi = \frac{\eta_2}{\eta_1 + \eta_2} \\
\dot{R} &= \gamma I, \quad \dot{S} = \psi(\theta), \quad \dot{I} = 1 - S - R
\end{aligned}$$

The new variable φ_D represents the probability that a stub has not transmitted infection to its node and is currently dormant. The variables φ_S , φ_I and φ_R now measure the proportion of all stubs which are both dormant and belong to a susceptible, infected, or recovered node, with $\varphi = \varphi_S + \varphi_I + \varphi_R = \psi'(\theta)/(\psi'(1) + \psi'(0))$ the probability a stub is dormant. The ratios φ_S/φ , φ_I/φ , and φ_R/φ give the probabilities that a newly formed edge connects to a susceptible, infected, or recovered node. The variables ξ_S , ξ_I and ξ_R give the probabilities that a stub is active and belongs to a susceptible, infected, or recovered node, with $\xi = \xi_S + \xi_I + \xi_R = \psi'(\theta)/(\psi'(1) + \psi'(0))$ the probability a stub is active.

It is relatively straightforward to see that if η_1 is much larger than η_2 , then the proportion of time a stub is dormant is tiny, $\varphi \approx 0$. Consequently at any moment a node is expected to have $k_a = (1 - \varphi)k_m \approx k_m$ active contacts. As η_2/η_1 shrinks, this approximation improves. So in this limit, the DC model should reduce to the DFD model with edges breaking at rate η_2 . Indeed, in the equations above, if we take $\eta_2 = 0$ and assume $\eta_1 \gg 0$, then φ_D is negligibly small, so the $\dot{\varphi}_D$ terms drop out of the $\dot{\varphi}_S$ and $\dot{\varphi}_I$ equations. The values of φ_S and φ_I both go to zero as η_1 grows, but a little more care shows that φ_S/φ approaches ξ_S and φ_I/φ approaches ξ_I and the ξ variables solve the same equations in this limit as the φ variables in the DFD equations. Thus the DC equations reduce to the DFD equations in this limit. More details are in the SI.

In the opposite limit, if η_2 is large compared to η_1 , then stubs spend most time dormant, $\varphi \approx 1$. If the number of stubs is sufficiently large, the expected number of active stubs $k_a = \mathbb{E}[k_a] = k_m$ will not be negligible. The variation in the number of active stubs will be significant relative to its expected value k_a . However, $k_m \gg k_a$ so the number of dormant stubs $k_d = k_m - k_a$ can be approximated as k_m . The rate that dormant stubs become active is η_1 , so the rate at which the node forms new edges is approximately $\eta_1 k_d = \eta_1 k_m$ which itself is η_2 , while each existing edge breaks at rate η_2 . This is the assumption underlying the DVD model. A careful analysis of the equations show that indeed we can reduce them to the DVD model if $\eta_2 \gg \eta_1$. More details are in the SI.

Alternately, we can have the DC model converge to the MFSH model if $\langle K \rangle \gg 1$, as long as $\langle K \rangle$ remains fixed. In this case we find that the MFSH model is a good approximation, using β as the transmission rate. The underlying argument of this is that as $\langle K \rangle$ increases, the probability of transmission per edge decreases. Consequently, it becomes unimportant whether the edge has long or short duration because it is incredibly rare for the disease to try to transmit along the same edge twice. The total infectiousness of an individual is then given by the number of active edges times the per-edge infection rate. We can assume that only a proportion φ of the stubs are active at any time, and we can absorb this into β . So an individual with k stubs causes transmission at rate $k\beta$ where $\beta = \beta\varphi$. This is independent of how (or if) the edges are changing in time.

Figure 13 shows how the DC model reduces in the various limits.

3.3 Further Analysis of Mean Field Social Heterogeneity models

In this section we show that the two MFSH models are equivalent. We also show that all the non-Mass Action models described here reduce to the MFSH model if the average number of contacts is large, and the probability of infection per edge scales like $1 / \langle K \rangle$. Technically, our results show that there is no difference between the results of any of the (non-MA) models in this limit, so any of these models would be appropriate, but generally the equations of the MFSH model are simplest so we use it.

3.3.1 Equivalence of Mean Field Social Heterogeneity formulations—There are two formulations of the MFSH model, one with expected degrees and the other with actual degrees.

In the expected degree formulation, each individual has a given expected degree (which varies by individual). On average, at any given moment of time the individual has contacts. The exact number of contacts may vary from moment to moment, but how many contacts exist at one moment and who those contacts are with are independent from any other moment. Thus over a short period of time, we may safely assume that the total number of contacts with infected individuals matches the expected number.

In the actual degree formulation, each individual is assigned an actual degree k and given k stubs. At any time each of those k stubs forms an edge with a stub from another node, but who the stub connects to changes rapidly. Again, over a short period of time, we may assume that the number of contacts with infected individuals matches the expected number.

Actual degree formulation as a special case of expected degree formulation: Because contacts are very short, over any time interval, the total contact time is well-approximated based on the expected amount of contact at any given moment. Thus whether the individuals have the same number of contacts at each moment, or whether the amount varies from moment to moment, the effect over any macroscopic time interval is the same. Thus the actual degree model should be a special case of the expected degree model. In the expected degree formulation, the probability that a node with expected contacts is susceptible is $\exp[-(1 - \beta)]$ and in the actual degree formulation the probability that a node with k contacts is susceptible is $e^{-\beta k}$. If $\beta = k$, we expect these to equal, and so we anticipate $\beta = \exp(-1)$, or $\beta = 1 + \ln$. Plugging this into the expected degree equations, we arrive at the actual degree equations. So the two methods are equivalent subject to a change in variables.

Expected degree formulation as a limiting case of the actual degree formulation: The fact that the actual degree formulation leads to the expected degree formulation is based on similar reasoning. The underlying additional idea is that any continuous distribution can be approximated by a sufficiently well-refined discrete distribution. We then need a way to take a well-refined discrete distribution and rescale it so that all the probability is massed at integer values. To do this we make the observation that for the MFSH models, we can multiply every individual's (expected or actual) degree by L with no impact on the epidemic so long as we also divide the transmission rate by L . The resulting equations remain unchanged.

To make this more precise, consider a given β and $\rho(k)$ and assume L is large. We approximate the continuous distribution of β using a discrete distribution where the probability of k/L is $\int_{k/L}^{(k+1)/L} \rho(\kappa) d\kappa$. Increasing L gives a finer scale approximation. We

then multiply every k/L by L , to get $P(k) = \int_{k/L}^{(k+1)/L} \rho(\kappa) d\kappa$ where k is an integer. So long as we divide by L , the spread of the epidemic on a population with the given $P(k)$ and the rescaled will closely approximate the epidemic spread in the original expected degree population, with the approximation improving as L increases. We find that the actual degree equations converge to the expected degree equations as $L \rightarrow \infty$. Full details are in the SI.

As an example we take the uniform distribution from 0 to 10 for $\rho(x)$. So $\rho(x) = (1 - \exp[-10(1-x)]) / [2(1-x)]$. We initially start with a discrete distribution on the integers 0

through 9 with weight $1/10$ on each. So our initial $\psi(x)$ is $\sum_{k=0}^9 x^k / 10 = (x^{10} - 1) / (10(x - 1))$ with $\psi(0) = 1$. We then refine the distribution. For given L we have

$\psi(x) = \sum_{k=0}^{10L-1} x^k / 10L = (x^{10L} - 1) / [10L(x - 1)]$, and we take $\psi_k = \psi(k/L)$. Convergence is shown in figure 14.

3.3.2 Reduction of all models to Mean Field Social Heterogeneity model at large average degree—

One particularly important limit corresponds to people having many contacts, but a low probability of transmitting per contact before recovering. That is, $\langle K \rangle$ is large, and $\beta / \langle K \rangle$ is comparably small. In this limit, we expect that the duration of contact becomes unimportant, because the infection is unlikely to cross an edge more than once so the disease has no way to know how long the edge lasts. Heterogeneity in contact levels may still play an important role. If the heterogeneity is sufficiently small, it is reasonable to expect that the MA SIR model is appropriate.

In section 4.2 we rigorously derive the MFSH model from the DC model assuming $\langle K \rangle$ with fixed $\langle K^4 \rangle / \langle K \rangle^4$ and fixed $\beta / \langle K \rangle$. A similar proof will apply for the other models, or we can simply argue that the DC model reduces to all of the others, and careful attention to detail shows that they inherit this limiting case.

3.3.3 Reduction of MFSH to MA model if $\langle K^4 \rangle / \langle K \rangle^4 \rightarrow 1$ and $\beta / \langle K \rangle$ fixed—It is straightforward to show that the MFSH model becomes the MA SIR model if every node has the same contact rate. However more generally, we would expect that if the contact rates are sufficiently close together, the model should behave like the SIR model. In fact this holds if $\langle K^4 \rangle / \langle K \rangle^4 \rightarrow 1$ with $\langle K \rangle$ fixed. Intuitively, if the number of nodes with higher or lower contact rates is very small, their contribution to the spread of the disease is not significantly different from the contribution of an average node, and so the disease should spread as if the contact rate were homogeneous. We do not have a good intuitive explanation for why the precise condition relies on $\langle K^4 \rangle / \langle K \rangle^4$. However, we prove it rigorously in section 4.3.

We expect this case to be particularly relevant if the average degree is large and infectiousness is low. Combining this with the previous result, we conclude that all models converge to the MA model under appropriate conditions as $\langle K \rangle \rightarrow \infty$.

4 Rigorous proof of convergence to Mass Action equations

In this section, we give a rigorous proof of one of the most significant results. Namely, if $\langle K \rangle$ is large, but $\langle K^4 \rangle / \langle K \rangle^4$ is approximately 1 and $\beta / \langle K \rangle$ and γ are small compared to $\langle K \rangle$, then regardless of which model we use, the result is well-approximated by the MA equations.

We first show some examples, and then provide the details needed to prove the result for the DC model. The same result for simpler models can be derived by the same manner (though

in some cases the proof will be simpler). For our proof, we assume we have a sequence of populations and diseases indexed by n such that $\langle K \rangle \rightarrow \infty$, $\langle K^4 \rangle / \langle K \rangle^4 \rightarrow 1$ with $\langle K \rangle$ fixed. We show that this converges to the mass action equations using $\beta = \langle K \rangle$.

We do the proof in two steps. First, we show that in the limit of large $\langle K \rangle$ and constant $\langle K^4 \rangle / \langle K \rangle^4$, the DC equations converge to the MFSH equations. We then show that the MFSH equations converge to the MA equations if additionally $\langle K^4 \rangle / \langle K \rangle^4 \rightarrow 1$.

4.1 Example

As examples, we consider several different population structures using the MP model in figure 16. In the first, we take an exponential distribution of expected degrees. To vary the average degree, we change the decay rate of the exponential.

$$\rho(\kappa) = \frac{e^{-\kappa/\langle K \rangle}}{\langle K \rangle}$$

This does not satisfy the conditions that $\langle K^4 \rangle / \langle K \rangle^4 \rightarrow 1$. In the other examples, we take a uniform distribution, with expected degrees chosen uniformly from $(\langle K \rangle - \langle K \rangle^\alpha, \langle K \rangle + \langle K \rangle^\alpha)$. That is,

$$\rho(\kappa) = \begin{cases} 0 & \kappa < \langle K \rangle - \langle K \rangle^\alpha \\ \frac{1}{2\langle K \rangle^\alpha} & \langle K \rangle - \langle K \rangle^\alpha < \kappa < \langle K \rangle + \langle K \rangle^\alpha \\ 0 & \kappa > \langle K \rangle + \sqrt{\langle K \rangle} \end{cases}$$

This satisfies the condition for any $\alpha < 1$. Using $\langle K^4 \rangle$ to denote the average of the 4th power of the expected degree³, we have $\langle K^4 \rangle = \langle K \rangle^4 + 2\langle K \rangle^{2+2\alpha} + \langle K \rangle^4 / 5$. If $\alpha = 1$, then $\langle K^4 \rangle / \langle K \rangle^4$ does not approach 1. The distribution would not make sense for $\alpha > 1$ since some nodes would have negative expected degree.

For all distributions, we take $\alpha = 2 / \langle K \rangle$ and $\beta = 1$. The corresponding MA model has $\beta = 2$. In figure 17 we plot the results for the exponential distribution. This distribution does not satisfy the conditions, and we see that the solutions do converge, but not to the MA model. In figure 18, we take the uniform distribution from $\langle K \rangle - \langle K \rangle^\alpha$ to $\langle K \rangle + \langle K \rangle^\alpha$. We again see that when the conditions are not satisfied (the exponential distribution and the distribution with $\alpha = 1$), the solutions may still converge, but not to the corresponding MA model. However, when the conditions are satisfied, the solutions converge to the MA model.

4.2 DC to MFSH

We now begin our proof that the DC model converges to the MA model. We begin by showing that it converges to the MFSH model. We will use the following result several times in the proof: Assume $g(t)$ is a function of time and

$$\dot{g} = -c(t)g + f(t)$$

³Our condition that $\langle K^4 \rangle / \langle K \rangle^4 \rightarrow 1$ applies equally for the fourth power of actual or expected degree since at leading order they are the same.

If $\alpha(t) > 0$ for all time and $|f| \leq F$, and further $|g(0)| \leq F/C$, then for all time $|g(t)| \leq F/C$. To see this, assume that f and g are positive. If g is increasing beyond F/C , then $-cg + f$ must be negative, violating the assumption that g is increasing. Similar results hold for negative values of f and g . Our proofs skip some of the intermediate steps. Complete details of all the algebra is in the SI.

We want to prove that if degrees increase while the infection rate simultaneously decreases, with β constant, then we must arrive at the Mass Action equations. We do this for the Dormant Contact model. We introduce the notation $\alpha(1)$ to denote a function whose value goes to zero as $\langle K \rangle \rightarrow \infty$. Our result above implies that if $f = \alpha(1)$ and c is bounded away from 0 as $\langle K \rangle$ changes, then $g = \alpha(1)$. We will show that neglecting small terms in the large degree limit the DC model becomes

$$\begin{aligned} \dot{\theta} &= -\tilde{\beta}\theta\zeta \\ \dot{R} &= \gamma I, \quad S = \psi(\theta), \quad I = 1 - S - R \end{aligned}$$

where $\psi = 1 - \frac{\theta}{\langle K \rangle} - \frac{1}{\langle K \rangle} \ln \theta$.

We define $\theta = I + \alpha(1)$. This is the proportion of all stubs which belong to infected nodes. We prove a series of results:

1. $\theta = 1 + \alpha(1)$.
2. $D = \alpha(1)$.
3. $\frac{1}{\langle K \rangle} R = \frac{2}{\langle K \rangle} R + \alpha(1)$
4. $\varphi_S = \xi \frac{\psi'(\theta)}{\psi'(1)} + \alpha(1)$.
5. $I = \alpha(1)$ $I = \alpha(1)$.
6. $I = \alpha(1)$
7. $\theta = 1 + \alpha(1)$.
8. $\zeta = 1 - \theta \frac{\psi'(\theta)}{\psi'(1)} + (\gamma/\tilde{\beta}) \ln \theta + \alpha(1)$

If we drop the error terms in the final two results, then we have the equations governing the Mean Field Social Heterogeneity model in the actual degree case, with θ playing the role of the transmission rate. The existence of the error terms means that at earlier time, the approximation is better, and it can deviate more as time increases. The solution converges uniformly for any t less than any arbitrary chosen value T as $\langle K \rangle$ grows. So for large enough $\langle K \rangle$, the region over which the MFSH model gives an accurate approximation will include the entire period of the epidemic.

1. $\theta = 1 + \alpha(1)$ —To estimate the probability a stub receives at least one transmission, we first estimate the expected number of transmissions a stub receives, which gives a bound on the probability of at least one transmission. Each time a stub receives infection, this corresponds to a transmission from the neighboring node through one of its stubs. So the expected number of inward transmissions equals the expected number of outward transmissions. This is an upper bound on the probability a stub receives an inward transmission, $1 - \frac{1}{\langle K \rangle}$. After some straightforward calculations, we can show that

$$\frac{\beta}{\beta + \eta_2 + \gamma} \frac{1}{1 - \frac{\eta_1 \eta_2}{(\eta_2 + \gamma)(\eta_1 + \gamma)}}$$

is a bound on $1 - \frac{\beta}{\beta + \eta_2 + \gamma}$. So long as $\frac{\beta}{\beta + \eta_2 + \gamma} > 0$, this is $\alpha(1)$ since $\frac{\beta}{\beta + \eta_2 + \gamma} = \alpha(1)$. Thus $\frac{\beta}{\beta + \eta_2 + \gamma} = 1 + \alpha(1)$.

If $\frac{\beta}{\beta + \eta_2 + \gamma} = 0$, this argument can be salvaged by a cruder bound. At time t after the initial time, the expected number of transmissions is bounded by $t = t\alpha(1)$. So choosing T as described above, we will still be able to prove uniform convergence for $t \leq T$ since $\frac{\beta}{\beta + \eta_2 + \gamma} = 1 + T\alpha(1) = 1 + \alpha(1)$ if T is held fixed. If T is chosen to be large enough initially, we have uniform convergence during the dynamic phase of the epidemic.

2. $\varphi_D = \pi + \alpha(1)$ —We show $\varphi_D - \pi = \alpha(1)$:

$$\begin{aligned} \frac{d}{dt}(\varphi_D - \pi) &= \eta_2(\theta - \varphi_D) - \eta_1 \varphi_D \\ &= \eta_2[1 + o(1)] - (\eta_1 + \eta_2)\varphi_D \\ &= (\eta_1 + \eta_2)\pi - (\eta_1 + \eta_2)\varphi_D + o(1) \\ &= -(\eta_1 + \eta_2)(\varphi_D - \pi) + o(1) \end{aligned}$$

In the next to last step we used the fact that $\theta = \pi / (1 + \eta_2)$ so $\eta_2 = \pi / (1 + \eta_2)$. So $\varphi_D - \pi$ is at most $\alpha(1) / (1 + \eta_2) = \alpha(1)$.

3. $\eta_1 \pi_R = \eta_2 \xi_R + \alpha(1)$ —We set $x_S = \pi_S - \pi$, $x_I = \pi_I - \pi$, and $x_R = \pi_R - \pi$. Using the previous results we can show that $x_S = \alpha(1)$. Since the sum $x_S + x_I + x_R$ equals $\pi - \pi$ which is zero, we conclude $x_I = -x_R + \alpha(1)$. Some algebra shows $\dot{x}_R = -(\pi + \eta_2)x_R + x_I = -(\pi + \eta_2 + \eta_1)x_R + \alpha(1)$, from which the result follows quickly.

4. $\varphi_S = \xi \frac{\psi'(\theta)}{\psi'(1)} + o(1)$ —We have

$$\begin{aligned} \frac{d}{dt} \left[\varphi_S - \xi \frac{\psi'(\theta)}{\psi'(1)} \right] &= \dot{\varphi}_S - \xi \dot{\theta} \frac{\psi''(\theta)}{\psi'(\theta)} \\ &= -\eta_2 \varphi_S + \eta_1 \frac{\pi_S}{\pi} \varphi_D - \beta \varphi_I \frac{\psi'(\theta)}{\psi'(\theta)} \left(\varphi_S - \xi \frac{\psi'(\theta)}{\psi'(1)} \right) \\ &= -\eta_2 \varphi_S + \eta_1 \pi \frac{\psi'(\theta)}{\psi'(1)} - \beta \varphi_I \frac{\psi'(\theta)}{\psi'(\theta)} \left(\varphi_S - \xi \frac{\psi'(\theta)}{\psi'(1)} \right) + o(1) \end{aligned}$$

where we used the fact that $\pi_S = \pi_D - \pi / (1 + \eta_2)$ and $\pi_D = \pi + \alpha(1)$ and that $\pi / (1 + \eta_2) = 1$. Since $\pi - \pi / (1 + \eta_2) = \pi$, we have

$$\begin{aligned} \frac{d}{dt} \left[\varphi_S - \xi \frac{\psi'(\theta)}{\psi'(1)} \right] &= -\eta_2 \left(\varphi_S - \xi \frac{\psi'(\theta)}{\psi'(1)} \right) - \beta \varphi_I \frac{\psi''(\theta)}{\psi'(\theta)} \left(\varphi_S - \xi \frac{\psi'(\theta)}{\psi'(1)} \right) + o(1) \\ &= -\left(\eta_2 + \beta \varphi_I \frac{\psi''(\theta)}{\psi'(\theta)} \right) \left(\varphi_S - \xi \frac{\psi'(\theta)}{\psi'(1)} \right) + o(1) \end{aligned}$$

Since $\frac{\psi''(\theta)}{\psi'(\theta)}$ is nonnegative, the coefficient of $\varphi_S - \xi \frac{\psi'(\theta)}{\psi'(1)}$ in the final expression is at least η_2 , so we conclude that $\varphi_S = \xi \frac{\psi'(\theta)}{\psi'(1)} + \alpha(1)$.

5. $\pi_I = \pi\zeta + o(1)$ $\xi_I = \xi\zeta + o(1)$ —We first define $q = (-D + \dots) / (1)$. Since $\dots = 1 + o(1)$ and $D = \dots + o(1)$, we have $-D + \dots = o(1)$. Since $0 \dots (1)$, we have $q = o(1)$. We will show that $I - \dots - q = o(1)$ and so $I = \dots + o(1)$.

$$\begin{aligned} \frac{d}{dt}(\pi_I - \pi\zeta - q) &= -\dot{\pi}_S - \dot{\pi}_R - \pi \dot{\zeta} - \dot{q} \\ &= -\frac{d}{dt} \left(\varphi_D \frac{\psi'(\theta)}{\psi'(1)} \right) - \gamma\pi_I - \eta_2 \xi_R + \eta_1 \pi_R - \pi \left(-\frac{d}{dt} \left(\theta \frac{\psi'(\theta)}{\psi'(1)} \right) - \gamma\zeta \right) - \frac{d}{dt} \left((-\varphi_D + \pi\theta) \frac{\psi'(\theta)}{\psi'(1)} \right) \\ &= -\gamma(\pi_I + \pi\zeta - q) + o(1) \end{aligned}$$

So $I - \dots - q = o(1)$, and since $q = o(1)$, $I = \dots + o(1)$. A similar proof shows that $I = \dots + o(1)$.

6. $\varphi_I = \xi\theta\zeta + o(1)$ —We will actually show that $I + S = \dots + \dots^2 (1) / (1) + o(1)$. Once this result is shown, we use the fact that $S = \dots (1) / (1) + o(1) = \dots^2 (1) / (1) + o(1)$ to get our final result. We begin by taking the derivative of $I + S - [\dots + \dots^2 (1) / (1)]$

$$\frac{d}{dt} \left(\varphi_I + \varphi_S - \xi \left[\theta\zeta + \theta^2 \frac{\psi'(\theta)}{\psi'(1)} \right] \right) = \dot{\varphi}_I + \dot{\varphi}_S - \xi \left[\dot{\theta}\zeta + \theta \dot{\zeta} + \dot{\theta} \theta^2 \frac{\psi''(\theta)}{\psi'(1)} + 2 \dot{\theta} \theta \frac{\psi'(\theta)}{\psi'(1)} \right]$$

After some manipulations (see SI) we have

$$\frac{d}{dt} \left(\varphi_I + \varphi_S - \xi \left[\theta\zeta + \theta^2 \frac{\psi'(\theta)}{\psi'(1)} \right] \right) = -(\eta_2 + \gamma) \left(\varphi_I + \varphi_S - \xi \left[\theta\zeta + \theta^2 \frac{\psi'(\theta)}{\psi'(1)} \right] \right) + o(1)$$

So $I + S - [\dots + \dots^2 (1) / (1)] = o(1)$. Because $\dots = 1 + o(1)$ and $S = \dots (1) / (1) + o(1)$ it follows then that $I = \dots + o(1)$.

7. $\dot{\theta} = -\tilde{\beta}\theta\zeta + \beta o(1)$ —This step is trivial. Since $I = \dots + o(1)$, we have

$$\begin{aligned} \dot{\theta} &= -\beta\varphi_I \\ &= -\tilde{\beta}\theta\zeta + \beta o(1) \end{aligned}$$

8. $\zeta = 1 - \varphi \frac{\psi'(\theta)}{\psi'(1)} + (\gamma/\tilde{\beta}) \ln \theta + o(1)$ —We make the observation that $\dot{\zeta} = -\frac{d}{dt} [\theta \psi'(\theta) / \psi'(1)] - \gamma\zeta$. We have

$$\begin{aligned} \dot{\zeta} - \frac{d}{dt} \left[1 - \theta \frac{\psi'(\theta)}{\psi'(1)} + (\gamma/\tilde{\beta}) \ln \theta \right] &= -\frac{d}{dt} \left[\theta \frac{\psi'(\theta)}{\psi'(1)} \right] - \gamma\zeta + \frac{d}{dt} \left[\theta \frac{\psi'(\theta)}{\psi'(1)} \right] - \frac{\gamma\dot{\theta}}{\tilde{\beta}\theta} \\ &= -\gamma\zeta - \frac{\gamma\dot{\theta}}{\tilde{\beta}\theta} \end{aligned}$$

From our previous result we have $\dots = -[\dots + o(1)] / \dots$. Substituting this in we have

$$\dot{\zeta} - \frac{d}{dt} \left[1 - \theta \frac{\psi'(\theta)}{\psi'(1)} + (\gamma/\tilde{\beta}) \ln \theta \right] = -\frac{\gamma \dot{\theta}}{\tilde{\beta} \theta} - \frac{\gamma \beta o(1)}{\tilde{\beta} \theta} - \frac{\gamma \dot{\theta}}{\tilde{\beta} \theta} = o(1)$$

Our result follows immediately.

So the DC model reduces to the actual degree formulation of the MFSH model. Because the expected degree formulation contains the actual degree formulation as a special case, it suffices now to prove that the expected degree formulation converges to the MA model in the appropriate limit.

4.3 MFSH to MA

Our proof that the MFSH model converges to the MA model does not require that $\langle K \rangle$, but it does require $\langle K^4 \rangle / \langle K \rangle^4 \rightarrow 1$. However, in order to conclude that the other models approach the MA model, we need the conditions of the previous section to hold as well.

We first make the observation that if $\langle K^4 \rangle / \langle K \rangle^4 \rightarrow 1$, then $\langle K^2 \rangle / \langle K \rangle^2 \rightarrow 1$ as well. By Jensen's inequality, we have $\langle K \rangle^2 \leq \langle K^2 \rangle$ and $\langle K^2 \rangle \leq \sqrt{\langle K^4 \rangle}$. So $1 = \langle K \rangle^2 / \langle K \rangle^2 \leq \langle K^2 \rangle / \langle K \rangle^2 \leq \sqrt{\langle K^4 \rangle} / \langle K \rangle^2 = \sqrt{\langle K^4 \rangle / \langle K \rangle^4} \rightarrow 1$. So $\langle K^2 \rangle / \langle K \rangle^2$ is bounded from below by 1 and from above by something converging to 1.

We first prove two lemmas. We define s_1 and s_2 by

$$s_1 = \frac{1}{\langle K \rangle} \frac{\Psi''(\Theta)}{\Psi'(1)} - S$$

$$s_2 = \frac{1}{\langle K \rangle} \Psi'(\Theta) - S$$

We show s_1 and s_2 go to zero. We have

$$\lim_{n \rightarrow \infty} |s_1| = \lim_{n \rightarrow \infty} \left| \frac{\Psi''(\Theta)}{\langle K \rangle^2} - S \right|$$

$$= \lim_{n \rightarrow \infty} \left| \frac{\int_0^\infty (e^{-\kappa(1-\Theta)} - S) (\kappa^2 - \langle K^2 \rangle) \rho(\kappa) d\kappa}{\langle K \rangle^2} \right|$$

(see SI for details). We now use the Cauchy-Schwarz inequality to bound this. We have

$$\left| \frac{\int_0^\infty (e^{-\kappa(1-\Theta)} - S) (\kappa^2 - \langle K^2 \rangle) \rho(\kappa) d\kappa}{\langle K \rangle^2} \right| \leq \frac{\left| \int_0^\infty (e^{-\kappa(1-\Theta)} - S)^2 \rho(\kappa) d\kappa \right|^{1/2} \left| \int_0^\infty (\kappa^2 - \langle K^2 \rangle)^2 \rho(\kappa) d\kappa \right|^{1/2}}{\langle K \rangle^2}$$

$$\leq \frac{\left| \int_0^\infty (\kappa^2 - \langle K^2 \rangle)^2 \rho(\kappa) d\kappa \right|^{1/2}}{\langle K \rangle^2}$$

where we use the fact that $|\exp[-\kappa(1-\Theta)] - S| \leq 1$ to show that the first term in the numerator of the first equation is at most 1. All that remains is to expand the numerator and bound it.

$$\frac{|\int_0^\infty (\kappa^2 - \langle K^2 \rangle)^2 \rho(\kappa) d\kappa|^{1/2}}{\langle K \rangle^2} = \frac{|\int_0^\infty (\kappa^4 - 2\kappa^2 \langle K^2 \rangle + \langle K^2 \rangle^2) \rho(\kappa) d\kappa|^{1/2}}{\langle K \rangle^2} \\ = \left| \frac{\langle K^4 \rangle - \langle K^2 \rangle^2}{\langle K \rangle^4} \right|^{1/2}$$

But our assumption is that as $n \rightarrow \infty$, both $\langle K^4 \rangle / \langle K \rangle^4$ and $\langle K^2 \rangle / \langle K \rangle^2$ go to 1. Thus this goes to zero as $n \rightarrow \infty$. This completes the proof that $s_1 \rightarrow 0$.

To show that $s_2 \rightarrow 0$ is similar.

$$|s_2| = \left| \frac{1}{\langle K \rangle} \Psi'(\Theta) - S \right| \\ = \left| \frac{\int_0^\infty (e^{-\kappa(1-\Theta)} - S) (\kappa - \langle K \rangle) \rho(\kappa) d\kappa}{\langle K \rangle} \right| \\ \leq \frac{|\int_0^\infty (e^{-\kappa(1-\Theta)} - S) \rho(\kappa) d\kappa|^{1/2} |\int_0^\infty (\kappa - \langle K \rangle) \rho(\kappa) d\kappa|^{1/2}}{\langle K \rangle} \\ \leq \frac{|\langle K^2 \rangle - \langle K \rangle^2|^{1/2}}{\langle K \rangle}$$

and as $n \rightarrow \infty$, this tends to 0 as well.

We have our bounds on s_1 and s_2 , so we are now ready to complete the proof that the Mean Field Social Heterogeneity model converges to the Mass Action model as $n \rightarrow \infty$.

We define $I_I = -\dot{I} / \dot{I} = 1 - (\dot{I} / \dot{I}) = (1 - \dot{I} / \dot{I})$. In [11] we saw that this represents the probability that a new neighbor is infected. We expect I_I to be very close to I and set $y = I_I - I$. Then

$$\dot{y} = \dot{I}_I - \dot{I} \\ = -\dot{\Theta} \frac{\Psi''(\Theta)}{\Psi'(\Theta)} + \gamma \dot{\Theta} / \beta - [-\dot{S} - \dot{R}] \\ = -\dot{\Theta} \frac{\Psi''(\Theta)}{\Psi'(\Theta)} + \gamma \dot{\Theta} / \beta - [-\dot{\Theta} \Psi'(\Theta) - \gamma I] \\ = \beta \Pi_I \frac{\Psi''(\Theta)}{\Psi'(\Theta)} - \gamma \Pi_I - \beta \Pi_I \Psi'(\Theta) + \gamma I \\ = \beta(I+y) \frac{\Psi''(\Theta)}{\Psi'(\Theta)} - \gamma y - \beta(I+y) \Psi'(\Theta) \\ = \beta \langle K \rangle (I+y) [(S+s_1) - (S+s_2)] - \gamma y \\ = \beta(I+y) (s_1 - s_2) - \gamma y$$

We know that $I+y$ is at most 1, and s_1 and s_2 both tend to zero as n increases, so we have $\dot{y}$ approaches $-\gamma y$ as $n \rightarrow \infty$. Note also that at early time I_I and I are both very close to 0 so y begins as a very small number. It is straightforward to conclude that y can never grow larger than the maximum value of s_1 which tends to zero as $n \rightarrow \infty$. Consequently, $y \rightarrow 0$ as $n \rightarrow \infty$.

We finally have

$$\begin{aligned}
 \dot{S} &= -\beta \Pi_I \Psi'(\Theta) \\
 &= -\widehat{\beta} \Pi_I (S + s_2) \\
 &= -\widehat{\beta} I S - \widehat{\beta}_y S - \widehat{\beta}_y s_2
 \end{aligned}$$

Since y and s_2 both tend to 0 as $n \rightarrow \infty$, we have $\dot{S} = -\widehat{\beta} I S$. This means that we have the Mass Action model in the limit.

5 Discussion

We have shown a number of relations between the various edge-based compartmental models for infectious disease spread originally derived in [11]. We used this to develop a flow chart (figure 1) which can guide the choice of appropriate model for a given population.

We note that while the edge-based compartmental models allow us to capture effects that were previously inaccessible through analytic techniques, there are still many effects that are not captured by the models considered here. Our flow chart does not address these. It is always prudent to consider the disease and population to ensure that the assumptions of our models are not strongly violated. A number of adaptations of the mass action SIR models exist for populations in which the disease has multiple stages, or the population has important substructures. In our next paper [12] we show that the general edge-based compartmental modeling approach can be used to accommodate many of these effects.

There are two interesting open (and related) questions which we call attention to. Firstly, we showed that if $\langle K \rangle \rightarrow \infty$, $\langle K^4 \rangle / \langle K \rangle^4 \rightarrow 1$ with $\langle K \rangle$ fixed, the models converge to the Mass Action model. However, there are many cases where $\langle K^4 \rangle / \langle K \rangle^4$ approaches some other value, and our calculations appear to converge. It would be interesting to identify what the relevant reduced equations are in this limit. Secondly, it is perhaps surprising that the condition for convergence to the Mass Action model depends on $\langle K^4 \rangle / \langle K \rangle^4 \rightarrow 1$ rather than $\langle K^2 \rangle / \langle K \rangle^2 \rightarrow 1$. It can be shown analytically that if $\langle K^2 \rangle / \langle K \rangle^2 \rightarrow 1$ but $\langle K^4 \rangle / \langle K \rangle^4 \neq 1$ then the early exponential growth rate is higher than the Mass Action model. Our numerical calculations suggest that there is an early phase that deviates from Mass Action, but after that phase the solution is indistinguishable. The early phase becomes shorter as $\langle K \rangle$ grows. So we believe that there is pointwise convergence although not uniform convergence. The underlying explanation for this is that in this limit there is a vanishingly small proportion of nodes with very high degree. They are quickly infected in an epidemic and then removed from the active population, leaving behind a population that follows the Mass Action dynamics. We have not investigated either of these questions in any detail.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Fig. 1. Flow chart for model choice

Depending on the how the edge turnover rates compare to the infection and recovery rates, as well as the degree distribution, we arrive at different models. The model abbreviations are as in Table 1. This flow chart assumes that all structure in the population is due to heterogeneity in contact levels.

Fig. 2.

The hierarchy structure. The edges summarize the relations between the models. A solid arrow from one model to another represents that the target model can be derived as a limiting case of the base model. We give heuristic explanations in the text for these and more rigorous derivations in the SI. A dashed arrow denotes that the target model can be derived as a special case of the base model. The dashed arrows are straightforward and the justification is given in the main text.

Fig. 3.

The flow diagram for the MA model. S is the proportion susceptible, I the proportion infected, and R the proportion recovered. The flux from I to R is γI , and the flux from S to I is $\hat{\beta}IS$.

Fig. 4.

The MP flow diagram. (Left) edge quantities: Φ_S , Φ_I , Φ_R , and $1 - \Theta$ are the per-unit probabilities of having a neighbor which is susceptible, infected and has not transmitted, recovered and did not transmit, or which has transmitted respectively. (Right) individual quantities: S the probability of being susceptible, I the probability of being infected, and R the probability of being recovered.

Fig. 5.

The MFSH flow diagram (for the expected degree formulation). (Left) the variables Φ_S , Φ_I , and Φ_R are the per-unit probabilities for an individual to be connected to a susceptible, infected, or recovered node. However, because the edges are fleeting, these change over quickly, and are thus always equal to $P_{i;s}$, $P_{i;i}$, and $P_{i;r}$. The variable $1 - \Theta$ remains the per-unit probability of having received infection. (Center) The variables Π_S , Π_I , and Π_R are the proportion of contacts at any given time which are made by susceptible, infected, or recovered individuals respectively. (Right) The variables S , I , and R remain the proportion of individuals who are susceptible, infected, or recovered.

Fig. 6.

DVD flow diagram. The variables have the same meanings as in the MFSH case, but because edges have nonzero duration, we must include the possibility that a partner changes status while an edge exists. Note that $\Phi_S = \frac{\psi'(\Theta)}{\psi'(1)}$. The fluxes in and out of Φ_I lead to a differential equation which can be integrated to give an expression for Φ_I in terms of Φ_S and Φ_R . This leads to a differential equation for Θ in terms of Φ_S and Φ_R .

Fig. 7. Convergence of DVD to MP and MFSH models

We consider the DVD model with varying values of η . The probability density function for the expected degree is $p(x) = e^{-x} / [\exp(10) - 1]$ for $x \in (0, 10)$ and 0 otherwise. This yields $\langle x \rangle = [1 - \exp(-10)] / [10(1 - x)]$. We take $\beta = 0.2$ and $\gamma = 1$. As η decreases (top), the MP model results, while as η increases (bottom) the MFSH model results. Note the difference in axes from top to bottom.

Fig. 8.

The flow diagram for the CM model. (Left) edge quantities: The variables ϕ_S , ϕ_I and ϕ_R are the probabilities that a neighbor of a given node has not yet transmitted to that node and has the given status. The variable $1 - \theta = \phi_S + \phi_I + \phi_R$ is the probability that the neighbor has not yet transmitted. We can solve for ϕ_I in terms of θ , which leads to a simple differential equation for θ in terms of t . (Right) individual quantities S , I , and R are the proportions in each state.

Fig. 9.

The MFSH flow diagram (for the actual degree formulation). (Left) the variables ϕ_S , ϕ_I , and ϕ_R denote the probabilities that a stub has not yet transmitted infection to an individual and is currently connected to a partner of a given type. These sum to θ , the probability the stub has not yet transmitted infection. Each of these is θ times the corresponding π_S , π_I or π_R . (Center) the proportion of stubs that belong to susceptible, infected, or recovered individuals or equivalently, the probability a new edge is with an individual of each type. (Right) The proportion of the population in each state.

Fig. 10.

The DFD flow diagram. (Left) the arrows at the top of the diagram represent breaking edges. The total rate at which stubs that have not yet transmitted infection break is $\eta\theta$. The proportions of these that then join up with individuals of a given type is proportion to the corresponding π variable. We cannot find a simple relation giving π_I so our equations cannot simplify as much as in previous models.

Fig. 11. Convergence of DFD to CM and MFSH models

We consider the DFD model with varying values of β . The degrees are $k = 6$, $k = 8$, and $k = 10$, with probability $1/3$ each. This yields $\langle x \rangle = (x^6 + x^8 + x^{10})/3$. We take $\gamma = 0.2$ and $\delta = 1$. As β decreases (top), the CM model results, while as β increases (bottom) the MFSH model results. Note the difference in axes from top to bottom.

Fig. 12.

The flow diagram for the DC model. Now when a stub ceases to be in an edge, it enters a dormant phase until it finds a new partner. The new variable ϕ_D represents the probability a stub has not yet transmitted infection to an individual and is dormant. The new variables ξ_S, ξ_I, ξ_R represent the proportion of all stubs which belong to nodes of a given type and are part of edges (*i.e.*, are active). We set $\pi_S = \xi_S + \xi_I + \xi_R$ to be the proportion of stubs that are active. The variables π_S, π_I, π_R are now the proportion of stubs which belong to nodes of a given type and are available to form edges (*i.e.*, are dormant). We set $\xi_S = \pi_S + \pi_I + \pi_R$ to be the proportion of stubs that are dormant. The probabilities a new edge is with an individual of each type are ξ_S/ξ , ξ_I/ξ and ξ_R/ξ . Again, we are not able to significantly simplify the resulting equations.

Fig. 13. Convergence of DC to DFD, DVD, and MFSH models

We take $\beta = 1$. (top) We consider the convergence of the DC model to the DFD model as η_1 increases. We take $\beta(x) = (x^2 + x^8)/2$. For the DC model we take $\eta_2 = 1$ and vary η_1 . As η_1 increases, the DC model converges to the DFD model result for $\eta_1 = 1$. (middle) We consider the convergence of the DC model to the DVD model as η_1 decreases and the degrees increase to compensate. For the DC model, we have $\beta(x) = (x^{1/\eta_1} + x^{4/\eta_2})/2$ where $\eta_2 = \eta_1 / (\eta_1 + \eta_2)$. We use $\eta_2 = 1$ and vary η_1 . For the DVD model, we take $\beta = 1$ and $\beta(x) = (\exp[-(1-x)] + \exp[-4(1-x)])/2$. (bottom) We consider the convergence of the DC model to the MFSH model with $\eta_1 = \eta_2$ and $\eta_1, \eta_2 \rightarrow \infty$. We take $\beta(x) = (x^2 + x^8)/2$. The MFSH model has the same β , but uses $\beta = \beta/2$ as the transmission rate. Note that for all three calculations, the $\eta_1 = 1$ curves are the same.

Fig. 14. Convergence of actual degree formulation of MFSH to expected degree formulation
 The actual degree formulation of the MFSH model for the appropriate discrete distribution (described in text) converges to the expected degree formulation of the MFSH model with contact rates chosen uniformly from 0 to 10.

Fig. 15. Convergence to MFSH

(top) Convergence of the Expected Degree models assuming a uniform distribution from 0 to 2 $\langle K \rangle$. (bottom) Convergence of the Actual Degree models assuming a third each with $\langle K \rangle / 2$, $\langle K \rangle$, and $3 \langle K \rangle / 2$ stubs. The solid curves correspond to $\langle K \rangle = 1$ (top) and $\langle K \rangle = 2$ (bottom). The dotted curves correspond to various larger values of $\langle K \rangle$. Because β decreases to balance the increase in $\langle K \rangle$, the MFSH curves are the same for all $\langle K \rangle$.

Fig. 16. The expected degree distributions for our examples of convergence as $\langle K \rangle$, with and without $\langle K^4 \rangle / \langle K \rangle^4 \rightarrow 1$.

Fig. 17. Sample convergence as $\langle K \rangle$. We use the exponential distribution described in figure 16. The distribution of expected degrees decays exponentially and $\langle K^4 \rangle / \langle K \rangle^4$ does not approach 1 as $\langle K \rangle$ grows. The solutions appear to converge, but they do not converge to the MA SIR model.

Fig. 18. Sample convergence as $\langle K \rangle$. We use the uniform distribution described in figure 16. In the first, second, and third rows, we use $\alpha = 1, 0.85, 0.5$ respectively. The condition fails for $\alpha = 1$ and the solutions do not converge to the MA model, but they converge to the MA model for smaller

Table 1

The basic models and their underlying assumptions. Contacts are assumed to form and break at some rate which can vary from zero (permanent) to infinite (fleeting). Depending on the process governing contact formation, we may know the actual degree k of an individual or we may know its expected degree.

Model	Contact Duration	Heterogeneity type
Configuration Model (CM)	Permanent	Actual degree k
Mixed Poisson (MP)	Permanent	Expected degree
Dynamic Variable-Degree (DVD)	Finite	Expected degree
Dynamic Fixed-Degree (DFD)	Finite	Actual degree k
Mean Field Social Heterogeneity (MFSH)	Fleeting	Contact rate k or
Dormant Contact (DC)	Finite active and dormant	Maximum degree k_m
Mass Action (MA)	Fleeting	None