Epidemic spread in networks: Existing methods and current challenges

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Abstract

We consider the spread of infectious disease through contact networks. We assume that the disease spreads through contacts and individuals recover into an immune class. The networks are assumed to be of Configuration Model type. We discuss a number of existing models for disease spread, and show relations between the underlying assumptions. The distinctions between the underlying assumptions are subtle, and in many cases this subtlety will be irrelevant. We show that in appropriate cases the models reduce to one another. We compare the benefits and disadvantages of the different models, and their application to other populations. Finally, we discuss ongoing challenges for network-based epidemic modeling.

1 Introduction

Mathematical models of infectious disease spread have played a significant role in improving our understanding of epidemics and our intervention strategies [1]. These models make simplifying assumptions in order to arrive at tractible equations.

Recently, considerable research has focused on eliminating some of the assumptions that go into most mathematical models. There has been a particular emphasis on understanding how contact networks influence the spread of disease [33, 30, 21, 22, 32, 13, 14, 15]. In this paper we focus on research into two aspects of contact networks: partnerships have nonzero duration and different individuals have different numbers of partners. We will focus our attention on "SIR" diseases, diseases in which individuals begin susceptible, become infected through partnerships with infected individuals, and may eventually recover into an immune class.

The models we investigate in this paper consider the spread of an infectious disease through a population whose partnerships are static. The structure of the population is "Configuration Model": each individual is assigned a number of stubs (its degree, k), and then finds partners for each stub randomly from the available stubs. So if $P(k)$ is the probability a random individual u has k partners, the probability that a partner v has k' partners is $k'P(k')/\langle K\rangle$ where $\langle K\rangle$ is the average degree — there is a size bias because an individual's partners are selected proportional to their degrees.

Several competing approaches have been developed to study SIR disease spread through networks. These models have varying levels of complexity and detail. Each model makes assumptions about the independence of infected individuals. The assumptions are subtley different. The resulting models are closely related, and in appropriate cases are identical. We introduce these models and provide their derivations. Then we discuss their advantages and disadvantages for different epidemiological questions. We present more recent directions of network-based infectious disease modeling and end with a discussion of current and ongoing challenges. We include an appendix which shows the relationships between the models by demonstrating how one can derive some models from others.

2 The existing models

We study five different models having three different basic approaches. In all cases, the disease is assumed to transmit at rate β from an infected individual to its partner. If the partner is susceptible, the transmission results in immediate infection. Infected individuals recover at rate γ.

The three approaches are characterized as "pairwise" models, "effective degree" models, and "edge-based comparmental" models. The pairwise models observe that the rate susceptible individuals become infected is proportional to the number of patnerships between susceptible and infected individuals. The main effort of these models in is tracking how the number of such partnerships change in time. The "effective degree" models focus on the number of individuals with different numbers of partnerships. These models "discard" edges once it is clear that they will no longer play a role in transmission (for example, if an individual's partner recovers, the edge can be discarded without altering any future transmissions). Consequently, the models stratify individual's by their "effective" degree. Finally, the "edge-based compartmental" models focus on the probability that an edge has transmitted, creating compartments showing the probability a partner has transmitted or if it has not yet transmitted, whether it is susceptible, infected, or recovered.

There are three assumptions that will play a prominent role in our discussion. This type of assumption has typically been referred to as a"closure" in the context of pairwise models. They are used to truncate an otherwise infinite system of equations into a finite system. The different models we will discuss use different closures. Mathematically a closure represents the statement that at some scale the distribution of infections in the network are random; there is no information contained in these scales. Any of these closure assumptions can be violated by the initial condition. As time progresses however, the disease spreads as a random process, and so information about the initial condition is lost. So even if the initial condition does not satisfy any given closure, its later dynamics will be well-described by our models. Of course, this is subject to the requirement that the closure used is actually consistent with the dynamics.

Before discussing the models, we introduce our three closures. Using the notation of the pairwise section, $[S_k]$ is the total number of susceptibles of degree k, $[A_pS_k]$ is the total number of partnerships involving a susceptible individual of degree k and the other partner with degree p of status A (A may be any of S, I, or R), and $[A_pS_kB_q]$ is the total number of "triples" involving a central susceptible individual of degree k and two of its partners, one with degree p of status A and one with degree q of status B.

The "triples closure" says that $[A_pS_kB_q] = (k-1)[A_pS_k][S_kB_1]/k[S_k]$. This says that the expected number of triples of a given type is exactly equal to what we would conclude assuming independence of each pair. We note that this permits the number of pairs of each type to depend on the degree of, for example, the central individual. So the assumption is that if we know all information about the frequency of pairs (in which at least one node is susceptible), there is no information to be gained by looking at triples in which the central individual is susceptible. We can rephrase this assumption as follows: given a susceptible individual u, if the statuses of partners of u are all independent conditional on the degree of u, then the triples closure holds.

To define the "star closure", we consider stars centered at susceptible individuals, that is collections of n individuals all sharing a common susceptible partner. We define $X_{s,i}$ to be the number of stars centered at susceptible individuals for which s of the periphery individuals are susceptible and i infected. The assumption of the star closure is that there is no information contained in the distribution of structures that are not stars: we assume that by knowing the frequency of each type of star we can calculate all important quantities. An equivalent expression of the star closure is that if we choose a susceptible individual u, the rate at which a susceptible neighbor v of u becomes infected is independent of the status of any other neighbors of u.

Finally, we define the "pair closure". This assumption says that the probability a partner v of a susceptible degree k individual u is susceptible is independent of the degree of u. Further, the probability that v is infected is also independent of the degree of u. However, it could be possible that the choice of initial infections means that the status of partners of u could be dependent, for example if the infection were introduced by choosing some random nodes and then infecting half of their partners.

All of these closures are consistent with the dynamics of the disease. In other words, (aside from stochastic variation), if the initial conditions satisfy a given closure, then the closure will be satisfied at all later time. We note that if we made equivalent assumptions centered about an infected or recovered indvidual, these

closures are not consistent with the disease dynamics. However, we will only need to make such assumptions with susceptible individuals at the center. No one of these closures implies another. However, if given a susceptible individual u, the probability a partner is susceptible, infected, or recovered is independent of the degree of u and independent of the status of any partners of u, then all three closures hold. Except in rare circumstances we expect this assumption to hold at the initial time (and therefore at all later times).

2.1 Pairwise models

Pairwise models are based on "pair approximation" methods. If we set $[SS]$ to be the number of partnerships in the population between susceptible individuals and $[SI]$ to be the number of partnerships between infected and susceptible individuals, we see that there is a flux from $[SS]$ to $[SI]$ resulting from susceptible individuals becoming infected from another partnership. Determining the rate of this flux has been one of the central challenges of pair approximation models.

We begin by considering the basic pairwise model, and then investigate a more recent version which reduces the number of equations required.

2.2 The basic pairwise model

Our initial goal is to track individuals of degree k as they pass through different infection classes. We set $[S_k]$, $[I_k]$, and $[R_k]$ to be the number of susceptible, infected, and recovered individuals of degree k in the population. Because recovery occurs at rate γ, it is straightforward to see that the flux from $[I_k]$ to $[R_k]$ is $\gamma[I_k]$. The rate of infection follows easily: each edge between a susceptible and an infected individual transmits independently at rate β, so the flux from $[S_k]$ to $[I_k]$ is $\beta[S_kI]$ where $[S_kI]$ is the total number of edges from susceptible individuals of degree k to infected individuals.

So we find that we need to focus on partnerships in order to predict the epidemic dynamics. This is the reason that the basic pairwise model [6] focuses on pairs or partnerships among nodes. We set $[S_kS_{k'}]$ to be the total number of edges between susceptible individuals of degrees k and k'. This is given by the sum over all susceptible individuals of degree k of the number of susceptible partners of degree k'. This definition gives an implicit direction, so that we can think of this as the total number of edges from susceptible individuals of degree k to a susceptible individuals of degree k'. If $k = k'$, each edge is counted twice.

We define $[S_kI_{k'}]$ and $[I_kS_{k'}]$ to correspond to the number of edges from susceptible degree k individuals to infected degree k' individuals and the number of edges from infected degree k individuals to susceptible degree k' individuals respectively. Note that $[S_kI] = \sum_{k'}[S_kI_{k'}]$. We similarly define $[S_kR_{k'}]$, $[I_kI_{k'}]$, $[R_kS_{k'}]$, $[I_kR_{k'}]$ $[R_kI_{k'}]$ and $[R_kR_{k'}]$.

Putting this together, we get the flow diagrams in figure 1. We note that when a susceptible individual in an edge of interest transitions to being infected, one possibility is that the infection comes from outside that edge. These are denoted by dashed lines in the flow diagram. As such, we must track the number of triples that could give rise to these transitions. Note that in all cases, the central individual of the relevant triple is susceptible.

In principle we can write down equations for triples which will depend on groupings of four nodes. This results in a cascade of equations dealing with progressively larger collections of nodes. To keep our equations finite, we make the triples closure: $[A_pS_kB_q] = (k-1)[A_pS_k][S_kB_1]/k[S_k]$.

We note that this closure can fail to be satisfied if unusual initial conditions are chosen. For example, if we introduce the disease by looking at each degree 5 individual, and then leave it susceptible, but randomly choose to infect either all of its degree 4 partners OR all its degree 3 neighbors, the independence assumption made above will break down. However, if we assume that at $t = 0$ the infected individuals have been chosen without regard to their susceptible partners, then these approximations will hold. This is what we observe if the disease is introduced into a small proportion and we set $t = 0$ to be a later time when a sufficiently large proportion is infected that the dynamics are deterministic.

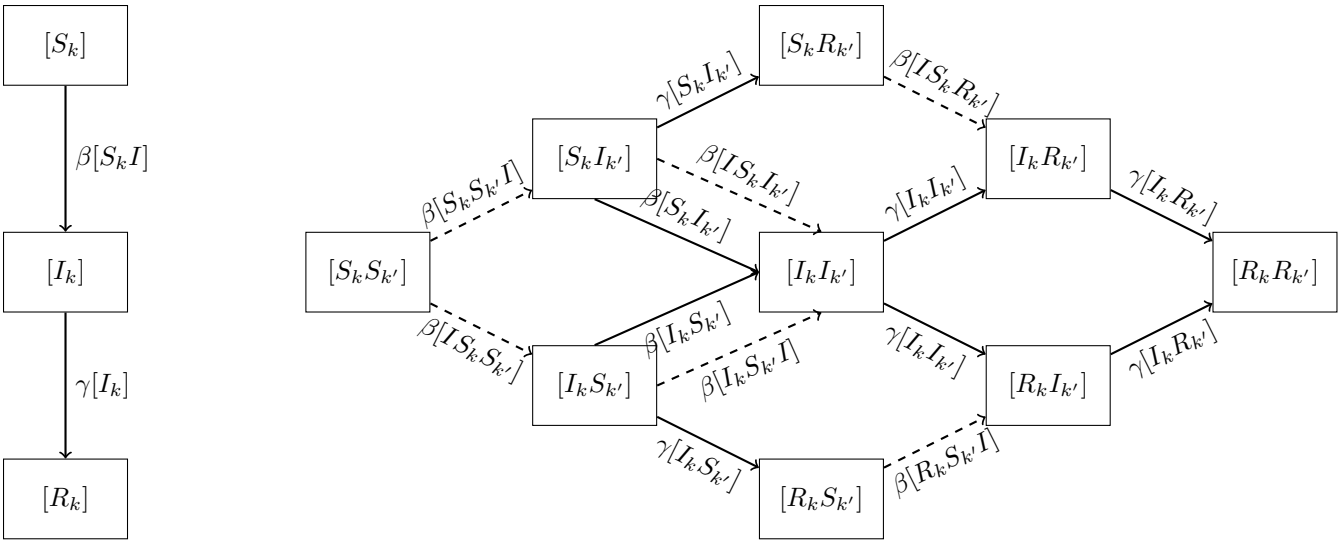

Figure 1: The flow diagram underlying the basic pairwise model. Dashed lines denote transitions that rely on infection coming from a source outside the edge of interest.

Using the closure and the flow diagram, we arrive at

$$[S] = \sum_k [S_k] \tag{1}$$

$$[I] = \sum_k [I_k] \tag{2}$$

$$[R] = \sum_k [R_k] = N - [I] - [S] \tag{3}$$

$$[IS_k] = [S_kI] = \sum_{k'} [S_kI_{k'}] \tag{4}$$

$$[\dot{S_k}] = -\beta[S_kI] \tag{5}$$

$$[\dot{I_k}] = \beta[S_kI] - \gamma[I_k] \tag{6}$$

$$[\dot{R_k}] = \gamma[I_k] \tag{7}$$

$$[S_k\dot{S}_{k'}] = -\beta\frac{k'-1}{k'}\frac{[S_kS_{k'}][S_{k'}I]}{[S_{k'}]} - \beta\frac{k-1}{k}\frac{[IS_k][S_kS_{k'}]}{[S_k]} \tag{8}$$

$$[S_k\dot{I}_{k'}] = \beta\frac{k'-1}{k'}\frac{[S_kS_{k'}][S_{k'}I]}{[S_{k'}]} - \gamma[S_kI_{k'}] - \beta\frac{k-1}{k}\frac{[IS_k][S_kI_{k'}]}{[S_k]} - \beta[S_kI_{k'}] \tag{9}$$

$$[S_k\dot{R}_{k'}] = \gamma[S_kI_{k'}] - \beta\frac{k-1}{k}\frac{[IS_k][S_kR_{k'}]}{[S_k]} \tag{10}$$

We do not require $[S_kR_{k'}]$ or $[R_k]$ directly, so we can avoid calculating them. They are included here for completeness. Solving this system (without $[S_kR_{k'}]$ or $[R_k]$) requires $2K^2 + 2K$ differential equations where K is the total number of distinct degrees in the population. A particular weakness of these equations is that in many important limiting cases, K tends to ∞. However, this approach is flexible enough that if the population structure changes such that our assumption $[A_mS_nI] = \frac{n-1}{n}[A_mS_n][S_nI]/[S_n]$ breaks down, we can use a different closure and the equations will still be correct. Depending on the details of how the assumption breaks down, the models below may not be able to accomodate this.

We note that all of these variables can be divided by N (the population size), so that the quantities may

be interpreted as the proportion of the population in each class. In a sense, this is more natural because we use continuous variables to represent our population size. This also allows us to use a universal calculation rather than having to recalculate if we study a different population size with the same degree distribution. We may think of this as equivalent to using intensive quantities rather than extensive quantities.

2.2.1 A compact pairwise model

A recent paper [9] developed some pairwise equations with a significantly reduced dimension. It was derived for a multiple disease model, but we focus on the restriction to just a single disease.

We begin with the pairwise model using the triples closure, but we additionally make the pairs closure: We assume that if v is a partner of a susceptible individual u, then the probability v is susceptible or infected is independent of the degree of u (or other partners of u). This assumption is distinct from the triples closure: we could begin by selecting a portion of the population and infecting all or their neighbors. The triples closure would fail, but the doubles closure would not.

We define $\langle I\rangle_k$ to be the proportion of partners of degree k susceptible individuals that are infected. We have (for all k)

$$\langle I\rangle_k = \frac{[S_k I]}{k[S_k]}$$

The pair closure assumption means that this is independent of k, and indeed it can be shown that if $\langle I\rangle$ is independent of k at time 0, then the basic pairwise equations predict it will remain independent. Using $\langle I\rangle$ for $\langle I\rangle_k$, we conclude that

$$\langle I\rangle = \frac{[SI]}{\sum_k k[S_k]}$$

Since we know that $\sum k[S_k] = [SS] + [SI] + [SR]$, this becomes

$$\langle I\rangle = \frac{[SI]}{[SS] + [SI] + [SR]}$$

We similarly conclude that

$$[S_k S_{k'}] = [SS_k]\frac{k'[S_{k'}]}{\sum_{k''} k''[S_{k''}]}$$

This further leads us to

$$\frac{\sum_{k'}(k'-1)[S_k S_{k'}]}{[S_k S]} = \frac{\sum_{k'} k'(k'-1)[S_{k'}]}{\sum_{k''} k''[S_{k''}]}$$

which we define to be $\langle k\rangle$. This is the average "excess degree" of a susceptible individual. That is, if we reach a susceptible individual by following an edge this is the expected number of other edges that susceptible individual has. It appropriately accounts for the probability of reaching an individual along an edge is proportional to its degree.

Using these assumptions the flux from $[S_k]$ to $[I_k]$ changes from $\beta[S_k I]$ to $\beta k\langle I\rangle[S_k]$. Since $\langle I\rangle = [SI]/\sum_k k[S_k]$ we have

$$\begin{aligned}
[\dot{S_k}] &= -\beta k\langle I\rangle[S_k] \\
[\dot{I_k}] &= \beta k\langle I\rangle[I_k] - \gamma[I_k]
\end{aligned}$$

We no longer need $[S_k I]$ explicitly for our equations at the singles level. So at the pairs level we can sum our equations for $[S_k S_{k'}]$, $[S_k I_{k'}]$, and $[S_k R_{k'}]$. We find $\sum_{k,k'}(k-1)[S_k S_{k'}][IS_k]/k[S_k] = \langle k\rangle\langle I\rangle[SS]$. Similarly if we switch k and k' in this equation, we get the same result. So we arrive at

$$\begin{aligned}
[\dot{SS}] &= -2\beta\langle k\rangle\langle I\rangle[SS] \\
[\dot{SI}] &= \beta\langle k\rangle\langle I\rangle[SS] - (\beta+\gamma)[SI] - \beta\langle k\rangle\langle I\rangle[SI] \\
[\dot{SR}] &= \gamma[SI] - \beta\langle k\rangle\langle I\rangle[SR]
\end{aligned}$$

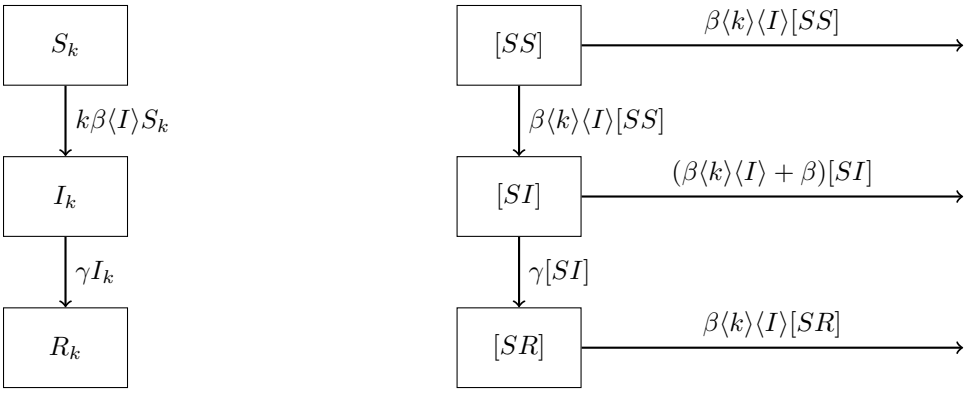

Figure 2: The flow diagram for the reduced system of [9]. The $[SS]$, $[SI]$, and $[SR]$ compartments correspond to the sum of the $[S_kS_{k'}]$, $[S_kI_{k'}]$ and $[S_kR_{k'}]$ compartments of the basic pairwise model.

Our final system is

$$[S] = \sum_k [S_k] \tag{11}$$

$$[I] = \sum_k [I_k] \tag{12}$$

$$[R] = N - [S] - [I] \tag{13}$$

$$[\dot{S_k}] = -\beta k \langle I\rangle [S_k] \tag{14}$$

$$[\dot{I_k}] = \beta k \langle I\rangle [I_k] - \gamma [I_k] \tag{15}$$

$$[\dot{R_k}] = \gamma [I_k] \tag{16}$$

$$[\dot{SS}] = -2\beta \langle k\rangle \langle I\rangle [SS] \tag{17}$$

$$[\dot{SI}] = \beta \langle k\rangle \langle I\rangle [SS] - (\beta+\gamma)[SI] - \beta\langle k\rangle\langle I\rangle [SI] \tag{18}$$

$$[\dot{SR}] = \gamma [SI] - \beta \langle k\rangle \langle I\rangle [SR] \tag{19}$$

$$\langle I\rangle = \frac{[SI]}{\sum_k k[S_k]} = \frac{[S_kI]}{k[S_k]} = \frac{[SI]}{[SS]+[SI]+[SR]} \tag{20}$$

$$\langle k\rangle = \frac{\sum_k k(k-1)[S_k]}{\sum_k k[S_k]} \tag{21}$$

We do not need to calculate $[SR]$ or $[R_k]$ directly. The number of equations required to solve this has reduced to $2K+2$ differential equations. This model is represented by a reduced flow diagram, shown in figure 2.

Similarly to the Basic Pairwise Model, all of the bracketed quantities may be divided by the population size N so that the equations are in terms of intensive quantities.

change S_k t
$[S_k]$ in figure

2.3 Effective degree models

We now turn to "effective degree" models. We define an "inactive" edge to be an edge which we know cannot cause infection. We then stratify individuals by the number of active edges they have. Depending on how much thought we put into this, we can arrive at different models. For example, if we simply say that an edge involving a recovered individual will never successfully transmit infection, we arrive at one model. On the other hand, if we go further and note that if an edge has ever transmitted infection in either direction, it cannot transmit infection, we will arrive at a different model.

Other models can be derived, but we focus our attention on two models which have been studied already.

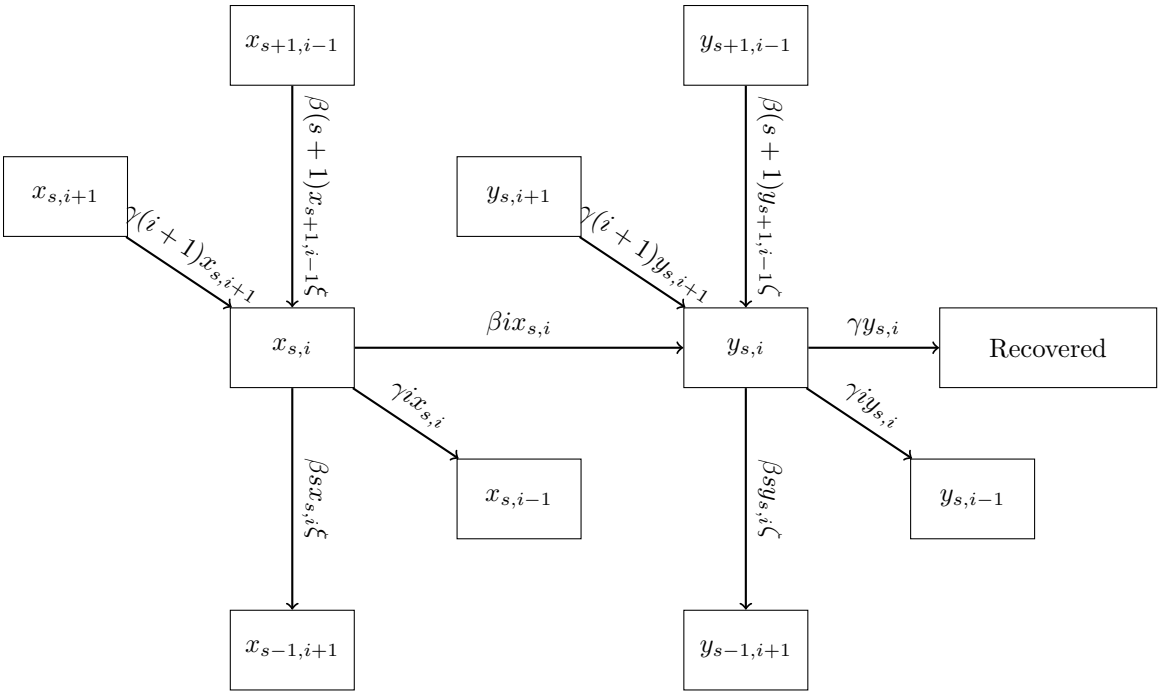

Figure 3: The flow diagram underlying the effective degree model of [16]. We include just the fluxes involving the $x_{s,i}$ or $y_{s,i}$ compartments. Fluxes between other compartments exist but are not included. Given a susceptible individual u, if v is a susceptible partner of u, the variable ξ is the expected number of infected partners of v. Given an infected individual u, if v is a susceptible partner of u, then ζ is the expected number of infected partners of v (including u). We have made some small changes to the variables as used by [16]: their $G = \beta\xi$ and their $H = \beta\zeta$.

2.3.1 The basic effective degree model

In [16], individuals are stratified by the number of partners they have that are either susceptible or infected. We make the star closure. Thus if we consider a susceptible individual u, and look at a susceptible partner v, the rate at which v is assumed to become infected is independent of any other partner of u, and is independent of our choice of u.

We use $x_{s,i}$ to denote the number of susceptible individuals with s susceptible and i infected partners. Recovery of a partner implies that an individual transitions from the $x_{s,i}$ compartment to the $x_{s,i-1}$ compartment. Similarly, we use $y_{s,i}$ to denote the number of infected individuals with s susceptible and i infected partners. Thus an edge is active if it does not include a recovered individual. We assume that the maximum possible effective degree is M. The minimum effective degree is 0, so even if all nodes begin with 10 partners, our equations must consider 0 to 10 effective partners and must include all possible ways those ten effective partners may be infected or susceptible.

We introduce the variables ξ and ζ: given a susceptible partner v of a node u, these are the expected number of infected partners of v given that u is susceptible or infected respectively. Since the status of u does not affect other partners of v, we anticipate that $\zeta = 1 + \xi$ (as for previous models, this will fail if the initial condition violates this assumption). For the calculation of ξ, we need to account for the fact that the selection of v is weighted by the number of susceptible partners of v, and for the calculation of ζ, we need to account for the fact that the selection of v is weighted by the number of infected partners of v. We have

$$\xi = \frac{\sum_{k=1}^{M}\sum_{j+l=k} jlx_{j,l}}{\sum_{k=1}^{M}\sum_{j+l=k} jx_{j,l}} \quad \text{and} \quad \zeta = \frac{\sum_{k=1}^{M}\sum_{j+l=k} l^2x_{j,l}}{\sum_{k=1}^{M}\sum_{j+l=k} lx_{j,l}}$$

These differ from G and H in [16] by a factor of β. The denominator of ζ appears different from the denominator of H in [16], but only because $\sum_{j,l} jy_{j,l} = \sum_{j,} lx_{j,l}$. The final equations are

$$\dot{x}_{s,i} = -\beta i x_{s,i} + \gamma\left[(i+1)x_{s,i+1} - i x_{s,i}\right] + \beta\xi\left[(s+1)x_{s+1,i-1} - s x_{s,i}\right] \tag{22}$$

$$\dot{y}_{s,i} = \beta i x_{s,i} - \gamma y_{s,i} + \gamma\left[(i+1)y_{s,i+1} - i y_{s,i}\right] + \beta\zeta\left[(s+1)y_{s+1,i-1} - s y_{s,i}\right] \tag{23}$$

$$\xi = \frac{\sum_{k=1}^{M}\sum_{j+l=k} jl x_{j,l}}{\sum_{k=1}^{M}\sum_{j+l=k} j x_{j,l}} \tag{24}$$

$$\zeta = \frac{\sum_{k=1}^{M}\sum_{j+l=k} l^2 x_{j,l}}{\sum_{k=1}^{M}\sum_{j+l=k} l x_{j,l}} \tag{25}$$

$$S = \sum x_{s,i} \tag{26}$$

$$I = \sum y_{s,i} \tag{27}$$

This is a system with $2(M+1)^2$ differential equations where M is the maximum degree. Careful consideration can remove about half of these equatons since the $\dot{x}_{s,i}$ equations do not depend on the $y_{s,i}$ terms. So we can sum the $\dot{y}_{s,i}$ equations to arrive at a single equation for $\dot{I} = \beta\sum i x_{s,i} - \gamma I$. So this can be reduced to $(M+1)^2 + 1$ differential equations.

This model makes an implicit closure assumption. The closure is different from that used to arrive at the basic pairwise equations. It assumes that given a susceptible individual u, the infected partners of u are randomly chosen from the infected individuals in the population (weighted appropriately by number of edges). Similarly the susceptible partners of u are randomly chosen from the susceptible individuals in the population (again, weighted by number of edges). However, this makes no assumption that the number of susceptible or infected partners u has is chosen from a random distribution. This allows for a biased initial condition in which the initial infections are distributed such that some individuals have fewer infected partners while others have more than could be explained by a random distribution. If we look at stars within the population (central nodes and their immediate neighbors), the model makes no assumptions about their distribution, but instead it allows us to calculate how their dynamics will change, subject to the assumption that the infected partners of u are randomly chosen from infected indviduals and susceptible partners of u are randomly chosen from susceptible individuals. Although this is different from the triples closure of the basic pairwise model, it is consistent with it.

As in the other models, we may switch to intensive quantities by dividing the x and y variables by N.

2.3.2 The compact effective degree model

A recent paper [2] that preceded the Basic Effective Degree model above used a different approach to consider "active edges". In this case, an edge is active if it has never transmitted infection in either direction and neither partner is in the recovered class. So some edges that remain in the Basic Effective Degree model drop out of this compact effective degree model.

We no longer track the number of susceptible or infected partners an individual has. Instead, we will calculate the probability a partner along an active edge is infected, and we assume that we can take this probability to be universal. Thus we assume that the statuses of partners of a susceptible individual u are independent of one another and of the degree of u. We are making both the triples and the pairs closure.

We use x_j and y_j to denote the proportion of susceptible and infected individuals with j active edges. We can calculate $\langle I\rangle$, the probability that v is infected given that the edge is "active". A little consideration will show that the probability a partner along an active edge is infected is $\langle I\rangle = \sum jy_j / \sum j(y_j + x_j)$.

Thus on average for susceptible individuals with j active edges, we conclude that the rate of becoming infected is $\beta j\langle I\rangle$. Similarly the rate at which suc an individual loses an active edge because of the partner

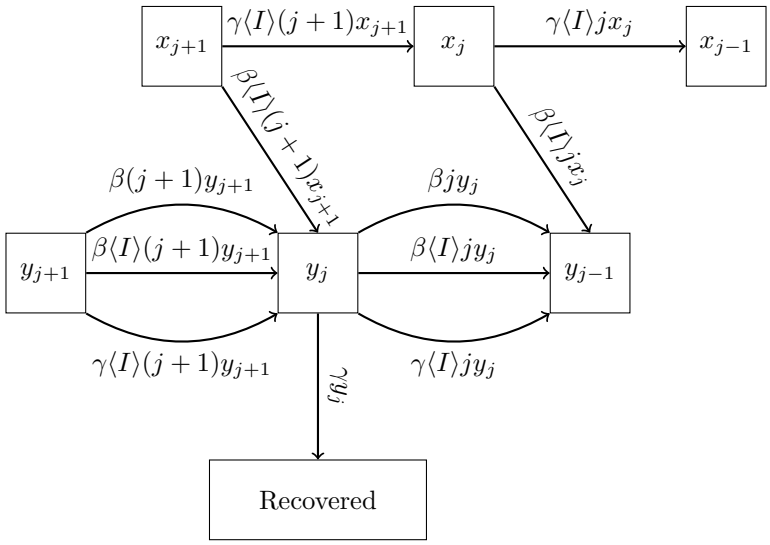

Figure 4: The flow diagram that underlies the model of [2]. Only the fluxes into and out of y_j and x_j are included. Fluxes between other compartments exist, but are not included. An active edge is eliminated if the partner recovers or if it transmits infection in either direction. The quantity $\langle I\rangle$ represents the probability an active edge joins an individual with an infected partner.

recovering is $\gamma j\langle I\rangle$. The flow diagram in figure 4 shows the relevant transitions. The resulting equations are

$$\dot{x}_j = \gamma\langle I\rangle\big((j+1)x_{j+1} - jx_j\big) - \beta\langle I\rangle jx_j \tag{28}$$

$$\dot{y}_j = (\beta + \beta\langle I\rangle + \gamma\langle I\rangle)\big((j+1)y_{j+1} - jy_j\big) + \beta\langle I\rangle(j+1)x_{j+1} - \gamma y_j \tag{29}$$

$$\langle I\rangle = \frac{\sum jy_j}{\sum j(x_j + y_j)} \tag{30}$$

This system has significantly fewer equations, just $2(M+1)$ equations. Unlike the earlier system, we cannot eliminate the equations for the y_j variables because they feed into the calculation of $\langle I\rangle$.

These equations are difficult to derive directly from equations (22)–(27) (the definitions of active edges are different). However, we can show that the $\dot{x}_j$ equation follows naturally (because the two definitions of active edges coincide if one individual is still susceptible). We define $x_k = \sum_{i+s=k} \tilde{x}_{s,i}$ where $\tilde{x}_{s,i}$ is the variable from [16]. We find

$$\dot{x}_l = \sum \gamma(i+1)\tilde{x}_{s,i+1} - \gamma i\tilde{x}_{s,i} - \beta i\tilde{x}_{s,i} = \sum_{s+j=l+1} \gamma j\tilde{x}_{s,j} - \sum_{s+i=l} \gamma i\tilde{x}_{s,i} - \sum_{s+i=l} \beta i\tilde{x}_{s,i}$$

We set $\langle I\rangle_l = \sum_{s+i=l} i\tilde{x}_{s,i} / \sum_{s+i=l} l\tilde{x}_{s,i}$ and make the assumption that $\langle I\rangle_l$ is in fact independent of l (that is, the probability a partner of a susceptible individual u is infected is independent of the number of active edges u has), then our equation reduces to

$$\dot{x}_l = \gamma\langle I\rangle(l+1)x_{l+1} - \gamma\langle I\rangle x_l - \beta\langle I\rangle x_l$$

So the systems appear consistent, though we would need to show that the two definitions of $\langle I\rangle$ coincide to complete the argument.

Again, we may switch to intensive quantities by dividing all x and y variables by N.

2.4 EBCM models

We finally turn to a third approach which has been called the "Edge-based compartmental modeling" approach. It is also sometimes referred to as a "pgf" approach because it relies on probability generating functions. The "pgf" terminology may be more descriptive because the pairwise equations also are based on compartmental models of edges. Nevertheless, we will stick to the "EBCM" language.

In an early version of this modeling approach [35], it was shown that a system with a finite number of equations was possible, but the derivation was challenging. The central variable was $\theta(t)$, the probability that a degree 1 individual is susceptible, and the observation was made that an individual with degree k is susceptible with probability θ^k. We will use this same variable, but give it a different definition, first introduced in [25] and refined in [26]. Effectively it is the probability an edge has not yet transmitted infection to an individual u, conditional in some sense on the assumption that u has not transmitted infection along the edge previously. This will be made more precise.

2.4.1 The basic EBCM model

The EBCM modeling approach results in a system with significantly fewer equations than the other models. Mathematically, it is generally easier to work with, but this comes with a slight conceptual complexity. Rather than tracking individuals (or partnerships) through the population, we track the probability that a partner of u has not infected u conditional on certain assumptions that we will make clearer.

To simplify the explanation, we make analogy to the "price taker" assumption of economics. A firm that is a price taker is typically one of many firms producing the same product, and it does not produce enough product to have any measurable impact on the price. Therefore, we can calculate the price ignoring the impact of the firm, and then calculate the impact of that price on what the firm does.

When we assume that a stochastic process is behaving deterministically on some large aggregate scale, we are making a similar assumption. In particular, for a disease spreading through a population, if we can assume that the aggregate dynamics are deterministic, then we are implicitly assuming that whether a particular individual is infected or not (and when that infection occurs) has no influence on the dynamics of the epidemic. Not only does the individual's infection not have any measurable aggregate-scale impact, but also the infections traced back to that individual have no measurable aggregate-scale impact. Thus we might make an "infection taker" assumption: we will look at how the aggregate dynamics affect the randomly chosen individual and explicitly assume the randomly chosen individual has no impact on the aggregate dynamics (or indeed even the local dynamics). Fundamentally, we are deriving a mean-field model, but using a slightly unusual approach.

Before begining the derivation, we note that in the previous models it was perhaps more natural to derive them in terms of quantities such as the total number of infected individuals or the total number of edges of a certain type. Here we think in terms of frequency: we calculate the proportion of the population that is susceptible by calculating the probability a random individual is susceptible.

Consider a randomly chosen individual u in the population. We refer to u as a "test node". Because we assume the dynamics are deterministic, the probability that u is in any given state is equal to the propotion of the population that is in each state. So rather than calculating the proportion of the population in each state, we shift to an equivalent question of calculating the probability that the test node u is in each state. If we look at neighbors of u, their statuses are dependent on one another through u: if v is infected, then another neighbor w of u is more likely to be infected because of the potential transmission path from v to w through u. This would complicate our analysis. So we remove these transmission paths by explicitly preventing transmissions from u. This has no impact on the status of u so we are shifting to a second equivalent problem: what is the probability that a randomly chosen individual is in each state given that the individual is prevented from transmitting to its partners? By changing how u affects its local environment, we are deviating from conventional mean-field approaches, but the final question we answer is equivalent to calculating the proportion of the population in each state. So long as we can assume that no single individual has an aggregate-scale impact, our approach will accurately predict the dynamics.[1]

[1] In fact, this explains why final sizes from epidemic simulations in smaller populations are often very similar even if the

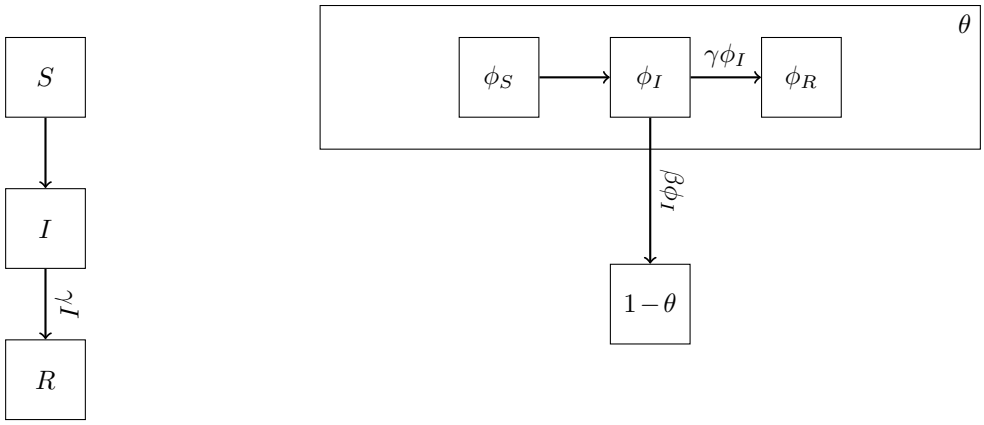

Figure 5: The flow diagram for the basic Edge-Based Compartmental Model. We do not calculate the flux from ϕ_S to ϕ_I or from S to I directly because we calculate ϕ_S and S directly. The relevant fluxes can be found by differentiating ϕ_S or S. Using the relative fluxes from ϕ_I, we can calculate ϕ_R in terms of θ.

Let us now consider a randomly chosen individual u in the population. We define X_k to be the number of individuals which are initially susceptible and have degree k. We define $\theta(t)$ to be the probability that an edge to a test node u has not yet transmitted to u by time t assuming that u was initially susceptible [so $\theta(0) = 1$]. We assume that θ is independent of the degree of u. The probability that u is susceptible at time t is thus $S(t) = \sum_k X_k \theta(t)^k / N$. We define this function to be $\psi(\theta)$. To find $R(t)$ and $I(t)$, the probability u is recovered or infected respectively, at time t we take $\dot{R} = \gamma I$, and $I = 1 - S - R$

To complete our system, we need to know θ. This is the probability that a partner v of u has not yet transmitted to u given that u was initially susceptible. We break θ into three compartments: ϕ_S, ϕ_I, and ϕ_R giving the probability that v is susceptible, infected, or recovered and has not transmitted to u. We know that $\phi_S + \phi_I + \phi_R = \theta$. It is straightforward to see that $\dot{\theta} = -\beta\phi_I$. Our goal will be to find ϕ_I in terms of θ to reduce our system to just this single differential equation.

We begin by calculating ϕ_S in terms of θ. If v was initially susceptible, the probability it has degree k is $kX_k / \sum kX_k$. Assuming it was initially susceptible with degree k, the probability that it is still susceptible is θ^{k-1} (each partnership other than the one with u has a chance to transmit to v). So the probability that v is susceptible at time t is $\phi_S(0) \sum_k kX_k \theta^{k-1} / \sum_k kX_k = \phi_S(0)\psi'(\theta)/\psi'(1)$.

We now find ϕ_R. The rate at which an infected neighbor recovers is γ. The rate at which an infected neighbor transmits to u is β. Thus, we find that $\dot{\phi}_R = \gamma\dot{\theta}/\beta$. Taking $\phi_R(0)$ as given and $\theta(0) = 1$, we conclude $\phi_R(t) = \phi_R(0) + \gamma(1-\theta)/\beta$.

We finally conclude that $\phi_I = \theta - \phi_S(0)\psi'(\theta)/\psi'(1) - \gamma(1-\theta)/\beta - \phi_R(0)$. Thus we arrive at

$$\dot{\theta} = -\beta\theta + \beta\phi_S(0)\frac{\psi'(\theta)}{\psi'(1)} + \gamma(1-\theta) + \beta\phi_R(0) \tag{31}$$

$$S = \psi(\theta)\,, \qquad I = 1 - S - R\,, \qquad \dot{R} = \gamma I \tag{32}$$

3 Model Selection

3.1 Relationship of models

The models are based on different assumptions about the independence of partners. Although the assumptions are formally different, the distinction is in a sense unnatural. If the initial infections are randomly

dynamics are still highly stochastic: an individual's infection may have a significant impact on the aggregate number infected at any given time and therefore be important dynamically, even if it has little impact on the final size.

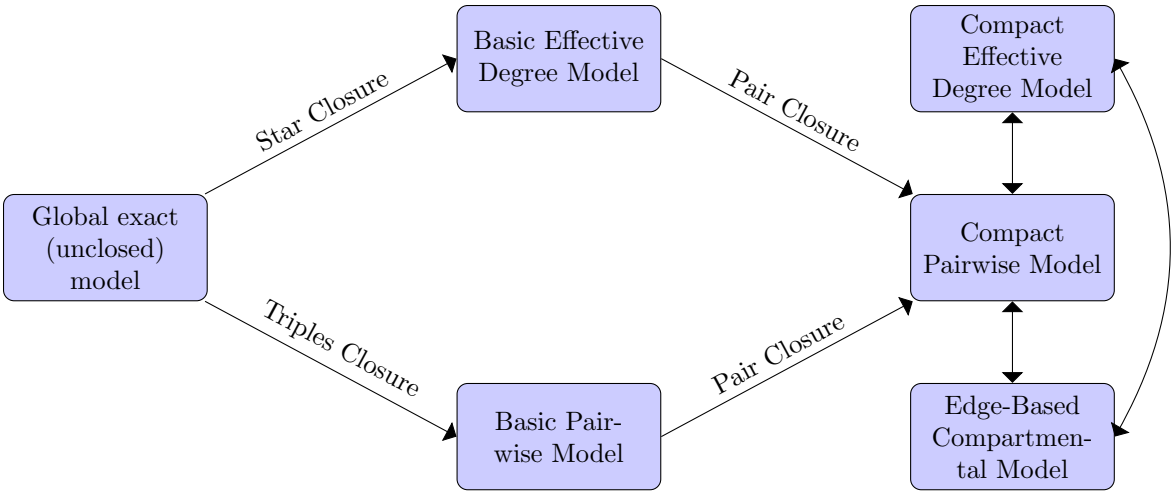

Figure 6: The hierarchy of models. Labeled edges denote the additional assumptions required to find the mathematically simpler model. Unlabeled (bidirectional) edges imply that the two models are equivalent and each can be derived from the other. The unclosed exact model will reduce to either the Basic Effective Degree model of [16] or the Basic Pairwise Model [6] depending on the closure used. Each ofthese then reduces to the Compact Pairwise Model of [9]. The Compact Pairwise Model, the Compact Effective Degree Model [2], and the Edge-based Compartmental model [26] are all equivalent.

selected, all the assumptions are satisfied, and the models give identical predictions. If the disease is introduced as a few infections, and allowed to spread until enough infections have occurred that the dynamics are deterministic, then all the assumptions will be satisfied, and the models give identical predictions (technically it is possible that stochastic effects cause one assumption to be valid but not another, but this will be a rare event).

In order for the assumptions of any of the models to be violated, this must be built into the initial condition. Somehow the initial infections must be chosen based on who their neighbors are. Even if the initial condition fails to satisfy assumptions of some of the models as time progresses information about the initial condition is lost. As the disease spreads, the violated assumptions are may eventually be satisfied and the underlying assumptions of the corresponding model hold.

Formally speaking, some of the models make stronger assumptions than others, and we prove in the appendix that it is possible to derive these models from the models having weaker assumptions. In the case in which we if we take a random susceptible individual u we find that the statuses of partners of u are independent of the degree of u and the status of one another, then we find that all of the models are appropriate. We can reduce all of the models to the EBCM model, having just a single ODE driving the dynamics.

We have discussed five distinct models of epidemic spread in static configuration model networks. These models have varying mathematical and conceptual complexity. A natural question is which model is appropriate for a given application.

3.2 Model robustness to assumptions

All of these models make some form of simplifying assumption (a closure) in the derivation. At the heart of each of the assumptions is some sort of independence assumption. Each assumption can be shown to be valid if there is no information content in certain scales. Since the epidemic is a stochastic process, whatever information may be imposed by the initial conditions is lost over time. We anticipate that all models should behave similarly at later time, and should fit observed dynamics closely.

Model	Minimal number of Differential Equations	Analytic final size calc?	Early growth calc?
Basic Pairwise	$2(K^2+K)$	No	$K^2 \times K^2$ eigenvalue problem
Condensed pairwise	$2K+2$	No	Analytic calculation
First effective degree model	$(M+1)^2+1$	No	$(M+1)^2-1 \times (M+1)^2-1$ eigenvalue problem
Second effective degree model	$2(M+1)$	No	$(M+1) \times (M+1)$ eigenvalue problem
Edge Based Compartmental model	1	Yes	Analytic calculation

Table 1: Comparison of the models for a population having K distinct degrees with maximum degree $M \geq K$.

3.3 Robustness to Configuration Model assumptions

These models have been derived by assuming that the population is a static Configuration Model network. This assumption is unrealistic: individuals change partners, and there may be correlations between the degrees of partners.

3.3.1 Degree correlations

If we allow for correlations to exist between the degrees of individuals, then the effective degree approaches will have to keep extra information. It is not enough to know how many active edges an individual has, we need to know how many partners the individual began with in order to know the risk from those active edges. This significantly increases the dimensionality of these models.

The simplifications that go into deriving the compact pairwise model from the basic pairwise model fail if an individual's degree affects its choice of partner. So we will not be able to use this model (though other simplifications may be possible).

The Edge-Based Compartmental Model presented here was derived assuming that the probability a partner of indivdual u has not transmitted to it, θ is independent of the degree of u. Clearly if there are degree correlations, this will not be true. However, depending on how those correlations occur, a modification of the EBCM approach may work. Details of how this can be done are in [27]. It requires allowing θ to be a function of k as well as t. So rather than a single equation for θ, we find K equations where K is the number of distinct degrees.

The basic pairwise model remains valid if degree correlations are put in place.

3.3.2 Dynamic networks

There are multiple ways we can include partner turnover in the model. One of the most obvious is to allow an individual's partnerships to end and be replaced by new partnerships. Another is to allow individuals to have intrinsic rates of creating new partnerhships while they end existing partnerships independently. Other models are clearly possible.

Again, the effective degree models would require a significant increase in dimension to account for partnership turnover. We would need to track the number of inactive edges in some way.

The Edge-Based Compartmental Modeling approach is well-suited for many dynamic networks. Many examples were developed in [26], and in [28] it was shown that a hierarchy of models could be constructed based on the assumptions about partner turnover. It was shown that a widely-used model [17, 18, 29, 33] that

assumes that at every moment partnerships are re-selected randomly arises as a special case of this hierarchy, and indeed the standard mass action model can be recovered in appropriate limits. One particular case that should be noted is that it is possible to model some forms of "serosorting" where individuals actively select partners of similar infection status.

Depending on how the partnership turnover is modeled, the compact pairwise model may be robust enough to accomodate it. The EBCM approach for dynamic networks requires introducing additional variables that are equivalent to $[SS]$, $[SI]$, $[SR]$ and related quantities. So we hypothesize that an attempt to adapt this model to a dynamic network is likely to lead to the same equations found by the EBCM approach.

It is relatively simple to adapt the basic pairwise model to most dynamic networks considered in previous generalizations of the EBCM model.

3.4 Final size relations and early growth

3.4.1 Early growth

One of the most important questions to answer about a disease is whether it is capable of causing an epidemic in a given population. To answer this, we typically look at R_0, the average number of infections caused by an early infectious individual, and try to find conditions under which $R_0 < 1$. However, for models which calculate the dynamic growth of an epideic, it is difficult to extract R_0 from the model. Instead, we look at an equivalent measure, the early growth rate.

We assume the epidemic is initialized with an infinitesimally small proportion infected and grows proportionally to e^{rt}, and try to calculate the value of r. If $r > 0$, epidemics are possible. Otherwise they are not.

For the basic pairwise model, we take the $[S_k I_{k'}]$ equations from equations (1)–(10). Because we assume the amount of infection is small, the $[IS_k][S_k I_{k'}]$ term is negligibly small. The other terms however are not negligible. The resulting system yields a $K^2 \times K^2$ eigenvalue problem whose dominant eigenvalue determines the stability of the system.

For the compact pairwise model, equations (11)–(21), we can note that so long as the amount of infection is very small, the denominatr of $\langle I \rangle$ may be treated as constant, so $\langle I \rangle$ is proportional to $[SI]$. Substituting this into the equation for $[SI]$, we discard the $\beta \langle k \rangle \langle I \rangle [SI]$ term. Assuming that all but an infinitesimal proportion of the population is susceptible (so $[SS]\langle I \rangle = [SI]$), we arrive at $[\dot{SI}] = \beta \langle k \rangle (0)[SI] - (\beta + \gamma)[SI]$, and so we conclude that

$$r = \beta \langle k \rangle (0) - (\beta + \gamma)$$

For our first effective degree model, equations (22)–(27), the original paper [16] showed that the early growth rate could be derived from a $(M+1)^2 - 1 \times (M+1)^2 - 1$ eigenvalue problem.

For the reduced effective degree model, equations (28)–(30), we can convert the y_j equations into an $(M+1) \times (M+1)$ eigenvalue problem.

Finally, for the EBCM model, we set $\theta = 1 - \epsilon e^{rt}$ and solve for r. The equation for $\dot{\theta}$ becomes

$$-r\epsilon e^{rt} = -\beta + \beta \epsilon e^{rt} + \beta \frac{\psi'(1) - \epsilon e^{rt} \psi''(1)}{\psi'(1)} + \gamma \epsilon e^{rt}$$

After some rearranging,

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

We note that $\langle k \rangle (0) = \psi''(1)/\psi'(1)$, so the two analytic formulae we can derive are identical.

3.4.2 Final size

To search for a final size relation, we set all derivatives to 0 and look to see if we can find an equation for the final size. The only case in which a clear solution exists is the EBCM model. We can trivially arrive at an equation which gives the fixed point of θ. Using this fixed point, we immediately have $S = \psi(\theta)$, and taking $I = 0$, we conclude that $R = 1 - \psi(\theta)$.

4 Current Challenges

There are a number of questions that remain unanswered for epidemic spread in networks. Two that appear particularly important and difficult are the spread of disease through non-Configuration Model networks (in particular clustered networks) and the spread of SIS disease: that is diseases in which individuals return to a susceptible state. In both cases the challenge is similar: the independence assumptions made in our earlier models are not valid.

4.1 Clustered networks

Social contact networks violate the assumptions of a Configuration Model networks in a number of ways. One of the clearest violations which is particularly important for the modeling of infectious disease spread is that social contact networks often have clustering: the partners of an individual are likely to be partners of one another.

As a consequence, all of the closure approximations applied above fail. There have been a number of attempts to study disease spread in clustered networks, but most rely on some sort of approximation [19, 20, 24, 34, 3, 5, 7] A select few are able to avoid such approximation, but only by defining their way out of it. If we restrict our attention to a subset of clustered networks which contain very specific motifs, then we can make analytic progress [23, 31, 36, 12] However, these networks have very specific restrictions. Despite the specific structure, the number of equations in the corresponding ODE systems increases and it becomes difficult to extract analytic results from this.

We forsee that closures at higher structure levels are possible [11] and that the appropriate closures must preserve the structure of cycles within the population. However, this requires knowledge of the motif structure, and may require specialized generation algorithms.

It is possible to generate clustered networks with identical cluster "density" but very different large scale structure, and hence very different epidemic dynamics [8]. Thus clustering alone is not a sufficient metric to describe the network structure. Different motif types (*e.g.*, full connected triangles, squares, and fully connected squares with a missing diagonal) may lead to similar levels of clustering but different overall epidemic dynamics.

Our goal of course is a model which can take an arbitrary social network and accurately predict the dynamics of an epidemic spreading through the network. However, in the immediate future, we forsee that any approximate closure models will generally be developed on a case-by-case basis, lacking the generality of SIR models on configuration model networks.

4.2 SIS models

The key simplification underlying the EBCM model was that in calculating the status of individual u, we can ignore the possibility of transmissions from u to its partners. For an SIS epidemic, this assumption fails. In a Configuration Model SIR epidemic, the partners of an individual u are independent until u becomes infected. Then the partners are no longer independent; however, once infected the status of u can no longer be influenced by its partners. So that dependence of partners is irrelevant. In an SIS epidemic, once infected, u can return to a susceptible state and be reinfected. The dependence of partners of u is no longer negligible.

Indeed, the fact that u can alter its future infection probability appears to underlie some initially surprising results [4]. It was proven that certain networks are able to sustain an epidemic even though simple mean-field models suggested they could not. The apparent reason is that high degree individuals maintain an island of infection about them by being continually reinfected (and occassionally managing to have infection spread along paths to other high degree individuals).

Our closure assumptions fail once an individual has an opportunity to infect its neighbors, so we must adapt our approaches. One of our challenges in modeling SIS disease is to identify appropriate closures which satisfy the dynamics. It should be noted that although the first effective degree model, equations (22)–(27), does not perform particularly well in comparison to the other SIR models, this was in many ways an unfair

comparison. It is a modification of a model initially developed for application to SIS epidemics. For SIS epidemics it is likely to outperform the more specialized SIR models.

5 Discussion

There are multiple approaches used to model the dynamic spread of SIR epidemics in networks. These approaches have been developed under the assumption of a Configuration Model network. They perform well and give identical results under reasonable initial conditions. We have identified the precise conditions under which the models are equivalent. The models have varying complexity, with the simplest analytic model being the Edge-based Compartmental Model of [26, 25]

Some of the approaches can be adapted to population which are assortatively or disassortatively mixed. Other features such as dynamic partnerships or link weights can also be accounted for by some of these approaches.

There are two important restrictions to the approaches developed so far. These models all make some degree of assumption of independence of neighbors of a susceptible individual u. In a network with short cycles, this assumption breaks down. Similarly for an SIS disease, infection could travel from one partner of u to another through u. Even once u returns to susceptible, we can no longer treat the partners as truly independent. We can find very special classes of clustered networks for which our SIR models can be adapted (by assuming very special motifs exist), but the number of equations grows rapidly, and the approach does not appear to be generally applicable. For SIS epidemics, there does not appear to be any analytic exact model, except in the limit of partnerships having negligible duration.

A Appendix

In this appendix we show that under reasonable assumptions, the models presented in this paper are in fact equivalent. We have three subtly different closure approximations making slightly different assumptions about the independence of partners. Depending on which assumptions hold, different models result, but all ultimately become identical in appropriate limits. We will show that by making appropriate assumptions, we can derive some of the models from others.

A.1 An example

In several cases, the technique we use is a careful application of integrating factors. We demonstrate this technique with a different physical problem for which most people's intuition is stronger.

Let us assume there is a single release of a radioactive isotope into the environment. The isotope may be in the air (A), in soil (S), or in biomass (B). It decays in time with rate τ. Assume the fluxes between the compartments are as in figure 7.

Then the equations are

$$\begin{aligned}
\dot{A} &= -\tau A + c_{SA}S - c_{AS}A + c_{BA}B - c_{AB}A \\
\dot{S} &= -\tau S - c_{SA}S + c_{AS}A - c_{SB}S - c_{BS}B \\
\dot{B} &= -\tau B - c_{BA}B + c_{AB}A - c_{SB}S + c_{BS}B
\end{aligned}$$

However if we define the variables a, s, and b to be the proportion of the remaining isotope that is in each compartment, then the decayed class disappears, and we are left

$$\begin{aligned}
\dot{a} &= c_{SA}s - c_{AS}a + c_{BA}b - c_{AB}a \\
\dot{s} &= -c_{SA}s + c_{AS}a - c_{SB}s - c_{BS}b \\
\dot{b} &= -c_{BA}b + c_{AB}a - c_{SB}s + c_{BS}b
\end{aligned}$$

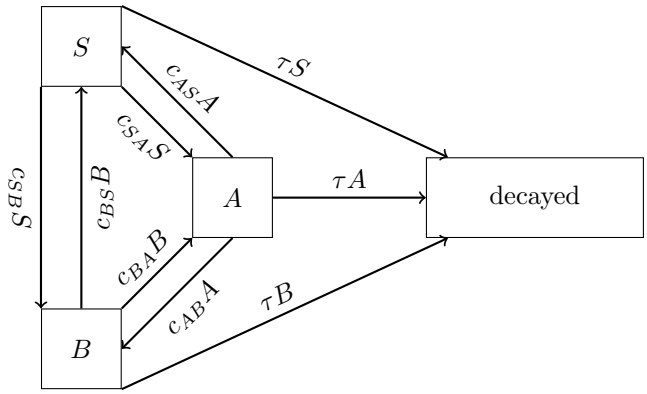

Figure 7: The flow diagram for the flux of a radioactive isotope between soil, biomass and air. The isotope decays at rate τ. This model provides a useful example of a technique that will be used in later derivations.

Physically this change of variables is fairly obvious. We are calculating the probability an isotope is in a given compartment conditional on it having not yet decayed. Mathematically we can get to this through an integrating factor of $\alpha e^{\tau t}$. We set $a = A\alpha e^{\tau t}$, $b = B\alpha e^{\tau t}$, and $c = C\alpha e^{\tau t}$ with α chosen so that the initial amounts sum to 1. If we multiply the $\dot{A}$ equation by $e^{\tau t}$, we end up with $\frac{\mathrm{d}}{\mathrm{d}t}(Ae^{\tau t}) = (c_{SA}S - c_{AS}A + c_{BA}B - c_{AB}A)e^{\tau t}$. Using the variable changes, we immediately arrive at the $\dot{a}$ equation. The other equations transform similarly. So using an integrating factor to eliminate the decay term is equivalent to transforming into variables that measure the proportion of undecayed isotopes that are in each compartment.

In general, so long as all compartments have an identical decay rate and the terms in the equations are homogeneous of order 1, then it is possible to use an integrating factor in this way to define a change of variables. This will be a key step in deriving the EBCM approach from the other models.

A.2 Pairwise to EBCM

We presented two pairwise models. We showed that the first reduces to the second if we make the assumption that if u is susceptible, then the probabilities a partner v is either infected or susceptible are independent of the degree of u or the status of any other partner of u. Mathematically, this states that $\langle I\rangle_k = [S_k I]/k[S_k]$ and $\langle S\rangle_k = [S_k S]/k[S_k]$ are independent of k.

It was previously noted [10] that if we make the generic assumption that $[A_k B] = [AB]k[A_k]/\sum_l l[A_l]$ where $[A_k B]$ represents the number of partnerships between individuals of status A having k partners and individuals of status B, then a pairwise approach can be used to derive an early version of the EBCM model [35]. In general, this assumption is inconsistent with the observation that the status of an individual's partners can be influenced by the individual once it is infected, and this is more likely to occur if the indvidual has high degree. However, in the particular case where status A is susceptible, the assumption is consistent: regardles of the degree of an individual, it has no impact on the status of its neighbors so long as it remains susceptible. We do not need the general form of the closure for our derivation, just the particular case with $A = S$.

In the derivation of the compact pairwise model, we claimed but did not prove that if $\langle I\rangle_k$ is independent of k to begin with, it remains so. To show this, we show that if $\langle I\rangle_k$ and $\langle S\rangle_k$ are independent of k at one

time, then their derivative are independent of k, and so they remains k-independent at all time. We have

$$\begin{aligned}
\dot{\langle I\rangle}_k &= \frac{\dot{[S_kI]}}{k[S_k]} - \frac{[S_kI]k[\dot{S_k}]}{k^2[S_k]^2} \\
&= \frac{\sum_{k'}\dot{[S_kI_{k'}]}}{k[S_k]} - \langle I\rangle_k\frac{k[\dot{S_k}]}{k[S_k]} \\
&= \frac{\sum_{k'}\beta\frac{k'-1}{k'}\frac{[S_kS_{k'}][S_{k'}I]}{[S_{k'}]} - \gamma[S_kI_{k'}] - \beta\frac{k-1}{k}\frac{[IS_k][S_kI_{k'}]}{[S_k]} - \beta[S_kI_{k'}]}{k[S_k]} - \langle I\rangle_k\frac{k(-\beta[S_kI])}{k[S_k]} \\
&= \frac{-(\gamma+\beta-\beta(k-1)\langle I\rangle_k)[S_kI] + \sum_{k'}\beta(k'-1)[S_kS_{k'}]\langle I\rangle_{k'}}{k[S_k]} - \langle I\rangle_k\frac{k(-\beta[S_kI])}{k[S_k]} \\
&= -(\gamma+\beta)\langle I\rangle_k - \beta(k-1)\langle I\rangle_k^2 + \frac{\sum_{k'}\beta(k'-1)[S_kS_{k'}]\langle I\rangle_{k'}}{k[S_k]} + \beta k\langle I\rangle_k^2 \\
&= -(\gamma+\beta)\langle I\rangle_k - \beta(k-1)\langle I\rangle_k^2 + \frac{\beta[S_kS]\sum_{k'}k'(k'-1)[S_{k'}]\langle I\rangle_{k'}}{k[S_k]\sum k''[S_{k''}]} + \beta k\langle I\rangle_k^2 \\
&= -(\gamma+\beta)\langle I\rangle_k - \beta(k-1)\langle I\rangle_k^2 + \frac{[S_kS]}{k[S_k]}\frac{\beta\sum_{k'}k'(k'-1)[S_{k'}]\langle I\rangle_{k'}}{\sum k''[S_{k''}]} + \beta k\langle I\rangle_k^2 \\
&= -(\gamma+\beta)\langle I\rangle_k - \beta(k-1)\langle I\rangle_k^2 + \langle S\rangle_k\frac{\beta\sum_{k'}k'(k'-1)[S_{k'}]\langle I\rangle_{k'}}{\sum k''[S_{k''}]} + \beta k\langle I\rangle_k^2 \\
&= -(\gamma+\beta)\langle I\rangle_k + \beta\langle I\rangle_k^2 + \langle S\rangle_k\frac{\beta\sum_{k'}k'(k'-1)[S_{k'}]\langle I\rangle_{k'}}{\sum k''[S_{k''}]}
\end{aligned}$$

So we see that if $\langle I\rangle_k$ and $\langle S\rangle_k$ are independent of k at a given time, then the derivative of $\langle I\rangle_k$ is also independent of k. A similar calculation shows that the derivative of $\langle S\rangle_k$ is independent of k. Thus we conclude that if at any time $\langle I\rangle_k$ and $\langle S\rangle_k$ are independent of time, they remain so for future time.

We are now in a position to reduce the second pairwise model to the EBCM model. Before doing so, we note that the derivation is simplest if we think of the pairwise variables as normalized by N. In particular, S becomes the susceptible proportion of the population.

We begin our derivation with the observation that for all k $[\dot{S_k}] = -\beta k\langle I\rangle[S_k]$. So $[S_k](t) = [S_k](0)e^{-\beta k\int_0^t\langle I\rangle(\tau)\,\mathrm{d}\tau}$. We define $\theta(t) = e^{-\beta\int_0^t\langle I\rangle(\tau)\,\mathrm{d}\tau}$. Then $[S_k](t) = [S_k](0)\theta(t)^k$. If we define $\psi(\theta) = \sum_k[S_k](0)\theta^k$, then $S(t) = \psi(\theta)$ and $\langle k\rangle = \theta\psi''(\theta)/\psi'(\theta)$. We have $\dot{\theta} = -\beta\langle I\rangle\theta$.

We return to the equations

$$\begin{aligned}
\dot{[SS]} &= -2\beta\langle k\rangle\langle I\rangle[SS] \\
\dot{[SI]} &= \beta\langle k\rangle\langle I\rangle[SS] - (\beta+\gamma)[SI] - \beta\langle k\rangle\langle I\rangle[SI] \\
\dot{[SR]} &= \gamma[SI] - \beta\langle k\rangle\langle I\rangle[SR]
\end{aligned}$$

We observe that each of these equations has a term which represents infection of the first individual in the partnership from a source outside the partnership (with the same coefficient, $\beta\langle k\rangle\langle I\rangle$, each time). In the EBCM approach, the test node is prevented from causing infection. This allows us to ignore infections to the test node from individuals other than the partner along a given edge. Mathematically, this shows up as ignoring infections of the first individual in the partnership from outside the partnership. So we use an integrating factor to eliminate this term. We define $F(t)$ such that $F'(t) = \beta\langle k\rangle\langle I\rangle$. There is an arbitrary constant in F which we will choose later. Then multiplying through by $e^{F(t)}$ we find

$$\begin{aligned}
\frac{\mathrm{d}}{\mathrm{d}t}([SS]e^{F(t)}) &= -\beta\langle k\rangle\langle I\rangle[SS]e^{F(t)} \\
\frac{\mathrm{d}}{\mathrm{d}t}([SI]e^{F(t)}) &= \beta\langle k\rangle\langle I\rangle[SS]e^{F(t)} - (\beta+\gamma)[SI]e^{F(t)} \\
\frac{\mathrm{d}}{\mathrm{d}t}([SR]e^{F(t)}) &= \gamma[SI]e^{F(t)}
\end{aligned}$$

We define $\phi_S = [SS]e^{F(t)}$, $\phi_I = [SI]e^{F(t)}$, and $\phi_R = [SR]e^{F(t)}$. The equations become

$$\begin{aligned}
\dot{\phi}_S &= -\beta\langle k\rangle\langle I\rangle\phi_S = -\dot{\theta}\frac{\psi''(\theta)}{\psi'(\theta)}\phi_S \\
\dot{\phi}_I &= \beta\langle k\rangle\langle I\rangle\phi_S - (\beta+\gamma)\phi_I = -\dot{\phi}_S - (\beta+\gamma)\phi_I \\
\dot{\phi}_R &= \gamma\phi_I
\end{aligned}$$

We can solve the equation for ϕ_S. By directly substituting in, we can check that $\phi_S = \frac{\psi'(\theta)}{\psi'(1)}\phi_S(0)$.

We choose the arbitrary constant in $F(t)$ such that $\phi_S(0)+\phi_I(0)+\phi_R(0) = 1$. We note that the equations suggest that there is some quantity being moved between compartments. We have $\frac{\mathrm{d}}{\mathrm{d}t}\phi_S+\phi_I+\phi_R = -\beta\phi_I$: if we introduce compartments, we have flux from ϕ_S to ϕ_I, from ϕ_I to ϕ_R, and from ϕ_I to an as-yet-undefined compartment (which will become $1-\theta$).

Since $\langle I\rangle = [SI]/([SS]+[SI]+[SR]) = \phi_I/(\phi_S+\phi_I+\phi_R)$, we conclude that

$$\begin{aligned}
\frac{\mathrm{d}\theta}{\mathrm{d}t} &= -\beta\langle I\rangle\theta \\
&= -\beta\phi_I\frac{\theta}{\phi_S+\phi_I+\phi_R} \\
&= \frac{\frac{\mathrm{d}\phi_S+\phi_I+\phi_R}{\mathrm{d}t}}{\phi_S+\phi_I+\phi_R}\theta
\end{aligned}$$

Since our initial condition has $\theta(0) = 1 = \phi_S(0)+\phi_I(0)+\phi_R(0)$, direct substitution into this equation shows that $\theta = \phi_S+\phi_I+\phi_R$ is the solution. So $\dot{\theta} = -\beta\phi_I$.

Because $\dot{\phi}_R = -\gamma\dot{\theta}/\beta$, we can show that $\phi_R = \phi_R(0) + \frac{\gamma}{\beta}(1-\theta)$.

Using our equations for $\dot{\theta}$, our solutions for ϕ_S and ϕ_R, and our relation $\theta = \phi_S+\phi_I+\phi_R$, we immediately arrive at the EBCM equations (31)–(32).

A.3 Effective degree

A.3.1 Deriving compact pairwise model from [16]

The effective degree model of [16] can be used to derive a pairwise model closely related to the compact pairwise model we introduced. Once the appropriate additional closure assumption is made, it becomes the compact pairwise model. It cannot recover the basic pairwise model because the two models have slightly different underlying assumptions (the effective degree model makes the "star closure" while the basic pairwise model makes the "triples closure".

We begin by defining

$$\begin{aligned}
[ss] &= \sum_{s,i} s x_{s,i} \\
[si] &= \sum_{s,i} i x_{s,i}\,.
\end{aligned}$$

These will represent the number of susceptible-susceptible edges (counted twice, once with each partner as the first individual) and the number of susceptible-infected edges.

We define

$$\begin{aligned}
[ssi] &= \sum_{s,i} s i x_{s,i} \\
[isi] &= \sum_{s,i} i(i-1) x_{s,i}\,.
\end{aligned}$$

These will represent the number of triples of the corresponding types. We find that

$$\begin{aligned}\xi &= \frac{\sum_{s,i} isx_{s,i}}{\sum_{s,i} sx_{s,i}} \\ &= \frac{[ssi]}{[ss]}\end{aligned}$$

For ζ we find

$$\begin{aligned}\zeta &= \frac{\sum_{s,i} i^2 x_{s,i}}{\sum_{s,i} ix_{s,i}} \\ &= \frac{\sum_{s,i} i(i-1)x_{s,i} + ix_{s,i}}{\sum_{s,i} ix_{s,i}} \\ &= \frac{[isi]}{[si]} + 1\end{aligned}$$

We have

$$\begin{aligned}[\dot{ss}] &= \sum s\dot{x}_{s,i} \\ &= \sum s(-\beta i x_{s,i}) + \sum s\gamma(i+1)x_{s,i+1} - \sum s\gamma i x_{s,i} + \sum s\beta\xi(s+1)x_{s+1,i-1} - \sum s\beta\xi s x_{s,i} \\ &= \beta\sum six_{s,i} + \gamma\sum s(i+1)x_{s,i+1} - \gamma\sum six_{s,i} + \beta\xi\sum(s+1)^2 x_{s+1,i-1} - \beta\xi\sum(s+1)x_{s+1,i-1} - \beta\xi\sum s^2 x_{s,i} \\ &= -\beta\sum six_{s,i} - \beta\xi\sum(s+1)x_{s+1,i-1} \\ &= -\beta\xi\sum sx_{s,i} - \beta\xi[ss] \\ &= -2\beta\xi[ss] \\ &= -2\beta[ssi]\end{aligned}$$

Going from the third to the fourth line, we used the fact that $\sum s(i+1)x_{s,i+1} = \sum six_{s,i}$ and $\sum(s+1)^2 x_{s+1,i-1} = \sum s^2 x_{s,i}$. We further have

$$\begin{aligned}[\dot{si}] &= \sum i\dot{x}_{s,i} \\ &= \sum i(-\beta i x_{s,i}) + \gamma\sum i(i+1)x_{s,i+1} - \gamma\sum i^2 x_{s,i} + \beta\xi\sum i[(s+1)x_{s+1,i-1} - sx_{s,i}] \\ &= -\beta\sum i^2 x_{s,i} + \gamma\sum(i+1)^2 x_{s,i+1} - \gamma\sum i^2 x_{s,i} - \gamma\sum(i+1)x_{s,i+1} + \beta\xi\sum(i-1)(s+1)x_{s+1,i-1} + \beta\xi\sum(s+1)x_{s+1} \\ &= -\gamma[si] - \beta\sum i^2 x_{s,i} + \beta\xi\sum(s+1)x_{s+1,i-1} \\ &= -\beta\zeta[si] - \gamma[si] + \beta\xi[ss] \\ &= -\beta[isi] - \beta[si] - \gamma[si] + \beta[ssi]\end{aligned}$$

These are the equations of the full pairwise model at the level of pairs without using any closure.

Using the effective degree model, we can derive equations for the rate of change of $[isi] = \sum i(i-1)x_{s,i}$ and the rate of change of $[ssi] = \sum six_{s,i}$ simply by using the derivative of $x_{s,i}$. It is at this level that the star closure appears. We find that the change in $[isi]$ and $[ssi]$ both depend on the frequency of groupings of four nodes, but only in stars: only with a single node having three partners. In the unclosed pairwise model, we find that groupings of four individuals in a path rather than just a star will influence the triples. The star closure assumes that at this scale we can safely average the transmission. It is worth noting that using the star closure, we will see a cascade involving ever larger stars, but if the maximum degree is bounded, the equations will eventually be closed.

We additionally introduce the pairs closure. For an individual u, with susceptible partner v, the expected number of active edges of v other than the one with u is defined to be $\langle k\rangle = \sum s(i+s-1)x_{s,i}/\sum sx_{s,i}$. We assume that this is independent of whether u is susceptible or infected, so it should also satisfy $\langle k\rangle = \sum i(i+s-1)x_{s,i}/\sum ix_{s,i}$. The probability a partner of v (other than u) is infected is defined to be $\langle I\rangle = \sum six_{s,i}/\sum s(i+s-1)x_{s,i}$ in the case u is susceptible and $\langle I\rangle = \sum i(i-1)x_{s,i}/\sum i(i+s-1)x_{s,i}$. We assume that both expressions for $\langle I\rangle$ are the same, that is the value is independent of the status of u. We conclude that $[ssi] = [ss]\langle k\rangle\langle I\rangle$ and $[isi] = [is]\langle k\rangle\langle I\rangle$ (note that $\langle k\rangle\langle I\rangle = \xi$).

Substituting these expressions into the equations yields the compact pairwise model, equations (11)–(21).

A.3.2 Deriving EBCM from the compact effective degree model

We begin with the equations of [2]. These equations focus on individuals. We want to focus on edges instead, so we need to translate the equations to be in terms of the edges. As before, we will assume the variables are normalized by N so that x_j represents the population proportion that is susceptible with j active partnerships, rather than the number of individuals that are susceptible with j partnerships. We begin by defining some auxiliary variables: $\alpha_S = \sum jx_j$ and $\alpha_I = \sum jy_j$.

We find that

$$\begin{aligned}
\dot{\alpha}_j &= \sum_{j=0}^{\infty} j\dot{y}_j \\
&= \sum_{j=0}^{\infty} j\big(\beta + \langle I\rangle(\beta+\gamma)\big)(j+1)y_{j+1} - \sum_{j=0}^{\infty} j\big(\beta + \langle I\rangle(\beta+\gamma)\big)jy_j + \sum_{j=0}^{\infty} j(j+1)\beta\langle I\rangle x_{j+1} - \sum_{j=0}^{\infty} j\gamma y_j \\
&= -\gamma\left(\sum_{j=0}^{\infty} jy_j\right) + \big(\beta + \langle I\rangle(\beta+\gamma)\big)\left(\sum_{j=0}^{\infty} j(j+1)y_{j+1} - \sum_{j=0}^{\infty} i^2y_j\right) + \langle I\rangle\beta\sum_{j=0}^{\infty} j(j+1)x_{j+1} \\
&= -\gamma\alpha_I + \big(\beta + \langle I\rangle(\beta+\gamma)\big)\left(\sum_{j=0}^{\infty} (j+1)^2y_{j+1} - \sum_{j=0}^{\infty} (j+1)y_{j+1} - \sum_{j=0}^{\infty} j^2y_j\right) + \langle I\rangle\beta\sum_{j=0}^{\infty} j(j+1)x_{j+1} \\
&= -\gamma\alpha_I + \big(\beta + \langle I\rangle(\beta+\gamma)\big)\left(\sum_{j=1}^{\infty} j^2y_j - \sum_{j=1}^{\infty} jy_j - \sum_{j=0}^{\infty} j^2y_j\right) + \langle I\rangle\beta\sum_{j=0}^{\infty} j(j+1)x_{j+1} \\
&= -\gamma\alpha_I + \big(\beta + \langle I\rangle(\beta+\gamma)\big)\left(-\sum_{j=1}^{\infty} jy_j\right) + \langle I\rangle\beta\sum_{j=0}^{\infty} j(j+1)x_{j+1} \\
&= -\gamma\alpha_I - \big(\beta + \langle I\rangle(\beta+\gamma)\big)\alpha_I + \langle I\rangle\beta\sum_{j=0}^{\infty} j(j+1)x_{j+1} \\
&= -\langle I\rangle(\beta+\gamma)\alpha_I - (\beta+\gamma)\alpha_I + \langle I\rangle\beta\sum_{j=0}^{\infty} j(j+1)x_{j+1}
\end{aligned}$$

and

$$\begin{aligned}
\dot{\alpha}_S &= \sum_{j=0}^{\infty} j\dot{x}_j \\
&= \gamma\langle I\rangle \left(\sum_{j=0}^{\infty} j(j+1)x_{j+1} - \sum_{j=0}^{\infty} j^2 x_j \right) - \beta\langle I\rangle \sum j^2 x_j \\
&= \gamma\langle I\rangle \left(\sum_{j=0}^{\infty} (j+1)^2 x_{j+1} - \sum_{j=0}^{\infty} (j+1)x_{j+1} - \sum_{j=0}^{\infty} j^2 x_j \right) - \beta\langle I\rangle \sum_{j=0}^{\infty} j^2 x_j \\
&= \gamma\langle I\rangle \left(\sum_{j=1}^{\infty} j^2 x_j - \sum_{j=1}^{\infty} j x_j - \sum_{j=0}^{\infty} j^2 x_j \right) - \beta\langle I\rangle \sum_{j=0}^{\infty} j^2 x_j \\
&= \gamma\langle I\rangle \left(-\sum_{j=1}^{\infty} j x_j \right) - \beta\langle I\rangle \sum_{j=0}^{\infty} j^2 x_j \\
&= -\gamma\langle I\rangle \alpha_S - \beta\langle I\rangle \sum_{j=0}^{\infty} j^2 x_j \\
&= -\gamma\langle I\rangle \alpha_S - \beta\langle I\rangle \sum_{j=0}^{\infty} j(j-1)x_j - \beta\langle I\rangle \sum_{j=0}^{\infty} j x_j \\
&= -\gamma\langle I\rangle \alpha_S - \beta\langle I\rangle \alpha_S - \beta\langle I\rangle \sum_{j=0}^{\infty} j(j-1)x_j \\
&= -(\beta+\gamma)\langle I\rangle \alpha_S - \beta\langle I\rangle \sum_{j=0}^{\infty} j(j-1)x_j
\end{aligned}$$

In both cases we use an integrating factor of $e^{F(t)}$ where $F'(t) = (\beta+\gamma)\langle I\rangle$. We set $\phi_S = \alpha_S e^{F(t)}$ and $\phi_I = \alpha_I e^{F(t)}$. We note that $F'(t) = (\beta+\gamma)\phi_I/(\phi_S+\phi_I) = -\frac{\mathrm{d}}{\mathrm{d}t}\ln(\phi_S+\phi_I)$ because $\frac{\mathrm{d}}{\mathrm{d}t}(\phi_S+\phi_I) = -(\beta+\gamma)\phi_I$. We have freedom in our choice of F up to an additive constant, and we will discuss this constant later. Thus our integrating factor is $e^{F(t)} = C/(\phi_S+\phi_I)$. After substituting for e^F and $\langle I\rangle = \alpha_I/(\alpha_I+\alpha_S) = \phi_I/(\phi_I+\phi_S)$, we have

$$\begin{aligned}
\dot{\phi}_I &= -(\beta+\gamma)\phi_I + \frac{C\phi_I}{(\phi_S+\phi_I)^2}\beta \sum_{j=0}^{\infty} j(j-1)x_j \\
\dot{\phi}_S &= -\beta\frac{C\phi_I}{(\phi_S+\phi_I)^2} \sum_{j=0}^{\infty} j(j-1)x_j
\end{aligned}$$

Our equations are suggestive of a conserved quantity, and seeking to arrive at the EBCM equations, we introduce ϕ_R and $\theta = \phi_S + \phi_I + \phi_R$. We set $\phi_R(0) = 0$ (this represents the fact that at the initial condition the effective degree model has discarded all information about recovered individuals. We choose the arbitrary constant in $F(t)$ such that $\phi_S(0) + \phi_I(0) = 1$. We find that $\phi_S = C\alpha_S/(\phi_S+\phi_I)$. Using $t = 0$ we get $\phi_S(0) = C\alpha_S$ and so $C = \phi_S(0)/\alpha_S$. We may assume

$$\begin{aligned}
\dot{\phi}_R &= \gamma\phi_I \\
\dot{\theta} &= -\beta\phi_I
\end{aligned}$$

Our attention now turns to finding an expression for the sum $\sum_{j=0}^{\infty} j(j+1)x_{j+1}$ in terms of our new variables.

We make the ansatz that $x_j = \sum_{k\geq j} x_k(0)\binom{k}{j}\phi_R^{k-i}(\phi_S+\phi_I)^i$. Intuitively, we are claiming that a susceptible individual has effective degree j if it initially had effective degree $k \geq j$, all but j neighbors have recovered without transmitting to it, and those j neighbors who have not yet recovered are either susceptible or infected but have also not transmitted to it. We must show that this satisfies the equations for x_y. It clearly satisfies the initial condition. We find

$$\begin{aligned}
\dot{x}_j &= \sum_{k\geq j} x_k(0)\binom{k}{j}\left(\dot{\phi}_R(k-j)\phi_R^{k-j-1}(\phi_S+\phi_I)^j + (\dot{\phi}_S+\dot{\phi}_I)j\phi_R^{k-j}(\phi_S+\phi_I)^{j-1}\right)\\
&= \sum_{k\geq j} x_k(0)\binom{k}{j}\left(\gamma\phi_I(k-j)\phi_R^{k-j-1}\frac{(\phi_S+\phi_I)^{j+1}}{\phi_S+\phi_I} - (\beta+\gamma)\phi_I j\phi_R^{k-j}\frac{(\phi_S+\phi_I)^j}{\phi_S+\phi_I}\right)\\
&= \frac{\phi_I}{\phi_S+\phi_I}\sum_{k\geq j} x_k(0)\left(\gamma\binom{k}{j+1}(j+1)\phi_R^{k-j-1}(\phi_S+\phi_I)^{j+1} - (\beta+\gamma)j\binom{k}{j}\phi_R^{k-j}(\phi_S+\phi_I)^j\right)\\
&= \langle I\rangle\left(\gamma x_{j+1} - (\beta+\gamma)jx_j\right)
\end{aligned}$$

which is our original equation for $\dot{x}_j$. So our ansatz satisfies the differential equation and the initial condition, so it is correct. For future note, we define $\psi(\theta) = \sum x_k(0)\theta^k$ so that

$$\sum_j x_j = \sum_k\sum_j x_k(0)\binom{k}{j}\phi_R^{k-j}(\phi_S+\phi_I)^j = \sum_k x_k(0)(\phi_S+\phi_I+\phi_R)^k = \psi(\theta)$$

Note that $\alpha_S(0) = \sum kx_k(0) = \psi'(1)$. Thus the arbitrary constant we used is $C = \phi_S(0)/\psi'(1)$.

We now turn to $\sum_{j=0}^\infty j(j-1)x_j$, the remaining term. We use our formula $x_j = \sum_k x_k(0)\binom{k}{j}\phi_R^{k-j}(\phi_S+\phi_I)^j$. Before doing this, we derive an identity:

$$\begin{aligned}
\sum_{j=0}^\infty\binom{k}{j}j(j-1)a^{k-j}b^j &= b^2\sum_i\binom{k}{i}i(i-1)a^{k-j}b^{j-2}\\
&= b^2\frac{\mathrm{d}^2}{\mathrm{d}b^2}\sum_j\binom{k}{j}a^{k-j}b^j\\
&= b^2\frac{\mathrm{d}^2}{\mathrm{d}b^2}(a+b)^k\\
&= k(k-1)b^2(a+b)^{k-2}
\end{aligned}$$

So

$$\begin{aligned}
\sum_{j=0}^\infty j(j-1)x_j &= \sum_{j=0}^\infty j(j-1)\sum_k x_k(0)\binom{k}{j}\phi_R^{k-j}(\phi_S+\phi_I)^j\\
&= \sum_k x_k(0)\sum_j j(j-1)\binom{k}{j}\phi_R^{k-j}(\phi_S+\phi_I)^j\\
&= \sum_k x_k(0)(\phi_S+\phi_I)^2k(k-1)(\phi_S+\phi_I+\phi_R)^{k-2}\\
&= (\phi_S+\phi_I)^2\psi''(\theta)
\end{aligned}$$

So we have

$$\begin{aligned}
\dot{\phi}_S &= -\beta\phi_I\frac{\psi''(\theta)}{\psi'(1)}\phi_S(0)\\
\dot{\phi}_I &= -(\beta+\gamma)\phi_I + \beta\phi_I\psi''(\theta)\\
\dot{\phi}_R &= \gamma\phi_I\\
\dot{\theta} &= -\beta\phi_I
\end{aligned}$$

We can solve for $\phi_S = \frac{\psi'(\theta)}{\psi'(1)}\phi_S(0)$. Following the approaches previously, we arrive at the EBCM equations (31)–(32). The $\phi_R(0)$ term does not appear: we take it to be zero because the effective degree model ignores edges to recovered individuals.

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