



Generating function approach to the effective degree SIR model

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Abstract

The effective degree SIR model describes the dynamics of diseases with lifetime acquired immunity on a static random contact network. It is typically modeled as a system of ordinary differential equations describing the probability distribution of the infection status of neighbors of a susceptible node. Such a construct may not be used to study networks with an infinite degree distribution, such as an infinite scale-free network. We propose a new generating function approach to rewrite the effective degree SIR model as a nonlinear transport type partial differential equation. We show the existence and uniqueness of the solutions that are biologically relevant. In addition we show how this model may be reduced to the Volz model with the assumption that the infection statuses of the neighbors of a susceptible node are initially independent to each other. This paper paves the way to study the stability of the disease-free steady state and the disease threshold of the infinite dimensional effective degree SIR models.

Keywords Contact network · Effective degree SIR Model · Well-posedness

Mathematics Subject Classification 93D30 · 35A01 · 35A02

1 Introduction

Network disease models (Kiss et al. 2017) have recently attracted much attention, because they can describe realistic human contact patterns, while classical compartmental disease models (see, e.g., Hethcote 2000; Kermack and McKendrick 1927) assume random mixing (each pair of individuals has identical contact rates). Network disease models describe population contacts as networks, where the nodes represent individuals and the edges represent contacts.

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One approach to model disease spread on networks is the node-based approach, which, like compartmental models, traces the fraction (or number) of individuals with an infection status (e.g., susceptible, infectious, etc). The effective degree SIR model (Lindquist et al. 2011) is a good example of this approach. The effective degree model keeps track of the infectious status of both the nodes and their neighbors, and can thus describe the correlation of infection status of neighboring nodes. This model is an improvement of the Pastor-Satorras and Vespignani model (Pastor-Satorras and Vespignani 2002), which does not keep track of the infectious status of neighbors, and thus cannot incorporate this correlation.

The effective degree SIR model, and other models of the node-based approach, classify the nodes by their degree (the number of edges attached to a node) using a system of ordinary differential equations (ODEs). To apply the qualitative theories of ODEs to study the system, it is commonly assumed that the network has a maximum degree. This may be reasonable for real networks, which are finite. However, for theoretical studies, the underlying population is typically assumed to be infinite with a Poisson or power-law degree distribution (see, e.g., Pastor-Satorras and Vespignani 2002). Such networks do not have a finite maximum degree, and thus ODE analysis may not easily be applied to such networks.

In this paper, we propose a novel generating function approach to rewrite the system of ODEs for the effective degree SIR model as a partial differential equation (PDE). The state variables of the effective degree model are probability distributions of the infection status of neighbors given a central node. The distribution uniquely corresponds to a probability generating function (Johnson et al. 2005). We thus derive an equation that governs the time evolution of the probability generating function. This approach will allow us to study infinite degree distributions (without a maximum degree). We aim to use the new PDE model to study the stability of the disease steady state and derive the disease threshold condition in infinite dimensional systems. We also hope to generalize this approach to study the disease threshold of SIS effective degree models in the future.

The edge-based approach is also commonly used for disease models on networks. Well studied examples of this approach include the Volz model (Volz 2008) and the Miller model (Miller 2011), which are equivalent. The importance of these models is that, with a simple independence assumption that the infection status of the neighbors of a susceptible node are independent to each other, the disease dynamics of an SIR model can be described by a very simple system of ODEs. The pair approximation models (Eames and Keeling 2002) are another commonly used approach for disease models on networks. Like the edge-based approach, these models classify the edges by the infection status of the nodes. However, unlike the edge-based approach, these models sometimes also consider the degree of the nodes connected by an edge. Kiss et al. (2017) discussed the relationships of these approaches, and found that, if the initial condition of the disease on the network satisfies the independent assumption, the pair approximation SIR models may be simplified to the Volz-Miller models.

The effective degree model is also found (Kiss et al. 2017; Miller and Kiss 2014) to be equivalent to the Volz-Miller models if we assume that the infection states of the neighbors of susceptible nodes are always independence. In this paper, we will show that, like the pair approximation models, as long as the initial condition satisfies the

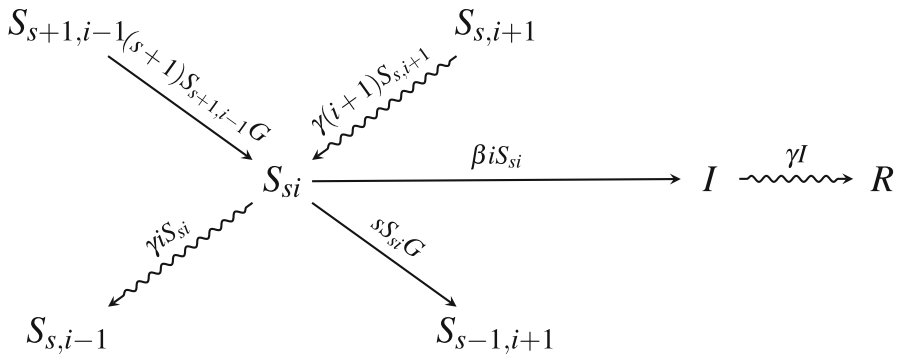


Fig. 1 The flow between compartments for the Effective Degree SIR model, where $G = \beta \frac{\sum_{s,i} s i S_{si}}{\sum_{s,i} s S_{si}}$. In this diagram the straight arrows represent infection and the curvy arrows represent recovery

independence condition, the effective degree SIR model may also be simplified to the Volz-Miller model. Even with the equivalence of these models, the effective degree models and the pair approximation models are also useful because they may be used to study the disease dynamics if the independence assumption does not hold.

We will review the effective degree model in Sect. 2, derive the generating function approach in Sect. 3, show in Sect. 4 that the model is well-posed, reduce the effective degree model to the Volz model in Sect. 5, and give some discussions and concluding remarks in Sect. 6.

2 Effective degree model

The Effective Degree model (Lindquist et al. 2011) considers an SIR model on a contact network, in which susceptibles are infected by neighbouring infectious individuals with a per link transmission rate β , and infectious individuals recover to full immunity at a rate γ . This model compartmentalizes nodes by both their state and by the number of neighbours that it has in each state. Hence, S_{sir} is the fraction of the population that is susceptible, having s susceptible, i infected, and r recovered neighbours. The system is simplified by observing that the recovered individuals cannot transmit the disease or be infected, hence we only track the susceptible and infected neighbours of a node. We call $k = s + i$ the *effective degree* of a node, which decreases with time as neighbours become infected and then recover.

The new compartments are denoted S_{si} . Denote I as the total fraction of infected nodes, and notice that once a central node is infected, its neighbours no longer influence its status, hence there is no reason to track the neighbours of infected nodes. A node in the class S_{si} has i infected neighbours and so is infected at a rate βi , and enters the I class. This node may also move to the class $S_{s,i-1}$ at a rate γi upon the recovery of an infectious neighbour. Finally, this node can move to the class $S_{s-1,i+1}$ upon the

infection of a susceptible neighbour. This occurs at the rate

$$\beta \frac{\sum_{s,i} si S_{si}}{\sum_{s,i} s S_{si}} s,$$

where the fraction is the probability that a susceptible neighbour of this node has an infectious neighbour. Figure 1 presents a flow chart for this model. Thus a simplified version of the effective degree model presented in Lindquist et al. (2011) is

$$\dot{S}_{si} = -\beta i S_{si} + \gamma[(i + 1)S_{s,i+1} - i S_{si}] + \beta \frac{\sum_{s,i} si S_{si}}{\sum_{s,i} s S_{si}} [(s + 1)S_{s+1,i-1} - s S_{si}], \tag{1a}$$

$$S = \sum_{s,i} S_{si}, \quad \dot{I} = \beta \sum_{s,i} i S_{si} - \gamma I. \tag{1b}$$

The original model also tracks I_{si} , however, the model can be simplified by tracking the total proportion of infected nodes $I = \sum_{s,i} I_{si}$ (see, e.g., Kiss et al. (2017)).

This model’s disease threshold condition, i.e. the requirement for an epidemic to occur, is that the basic reproduction number $\mathcal{R}_0 > 1$, where

$$\mathcal{R}_0 = \frac{\beta}{\beta + \gamma} \frac{\langle k(k - 1) \rangle}{\langle k \rangle}. \tag{2}$$

Note that $\langle k(k - 1) \rangle / \langle k \rangle$ is the average excess degree of a node, i.e., if an edge is chosen at random and followed to a node, this is the average number of remaining edges leaving that node. Considering the disease just begins to spread in a completely susceptible population. An infectious node must be infected by a neighbor, while all the other neighbors are still susceptible. Thus, the number of neighbors that are subject to infection is the effective degree of the newly infected node. Each node may be infected independently with the probability $\frac{\beta}{\beta + \gamma}$ (that transmission happens before the infectious node recovers). Thus, the basic reproduction number is the average number of secondary infections caused by an infectious individual in a fully susceptible population. The disease free equilibrium is unstable if $\mathcal{R}_0 > 1$, and asymptotically stable if $\mathcal{R}_0 < 1$.

Remark 1 If $\sum_{s,i} s S_{si} = 0$ then the third term in (1a) is undefined. However, notice that this implies $S_{si} = 0$ for all $s \geq 1$, which corresponds to a network in which no susceptible node has susceptible neighbours. The third term in (1a) defines the state transition due to the infection of a neighbour, but this cannot occur if all nodes have no susceptible neighbours. Thus define $\sum_{s,i} si S_{si} / \sum_{s,i} s S_{si} = 0$ if $\sum_{s,i} s S_{si} = 0$.

3 Generating function approach

We notice that the form of \mathcal{R}_0 presents no problem for an infinite dimensional degree distribution as long as the mean and variance are well defined, e.g. Poisson. The

effective degree model presented above calculates \mathcal{R}_0 using a next-generation matrix method (van den Driessche and Watmough 2002) for *finite dimensional systems*. Hence, a different approach is required to extend the effective degree SIR model to infinite dimensional systems. In the next section, we present a generating function approach to do just this.

3.1 Model formulation

Given a sequence $\{S_{si}\}$, define the associated generating function $S : [0, 1]^2 \rightarrow \mathbb{R}$

$$S(x, y) = \sum_{s \geq 0, i \geq 0} x^s y^i S_{si}. \tag{3}$$

For the solution $S_{si}(t)$ to (1a), we derive a PDE by taking a derivative with respect to time of (3), and substituting (1a) for \dot{S}_{si}

$$S_t = \sum_{s,i} x^s y^i \dot{S}_{si} \tag{4}$$

$$\begin{aligned} &= -\beta \sum_{s,i} x^s y^i i S_{si} + \gamma \sum_{s,i} x^s y^i [(i + 1)S_{s,i+1} - i S_{si}] \\ &\quad + \frac{\sum_{s,i} \beta s i S_{si}}{\sum_{s,i} s S_{si}} \sum_{s,i} x^s y^i [(s + 1)S_{s+1,i-1} - s S_{si}]. \end{aligned} \tag{5}$$

Notice that by taking derivatives in x and y of (3), e.g.

$$S_x = \sum_{s,i} x^{s-1} y^i s S_{si}, \tag{6}$$

and shifting indices, we arrive at the PDE formulation of the effective degree model

$$S_t = -(\beta + \gamma) \left(y - \frac{\gamma}{\beta + \gamma} \right) S_y + \frac{S_{xy}(t, 1, 1)}{S_x(t, 1, 1)} \beta (y - x) S_x. \tag{7}$$

We only consider the case where initially $S_{xy}(0, 1, 1)/S_x(0, 1, 1)$ is well-defined and non-zero, as this is the more interesting non-linear case. If this value is zero, then Equation (7) can be simply analyzed (please see Remark 1).

Remark 2 In Sect. 4.1, it will be shown that the characteristic curves flow out of the unit square. It is this property that allows the problem to be well-posed whilst not having boundary conditions.

Definition 1 A solution of (7) is *biologically relevant* if it is a power series with non-negative coefficients S_{si} .

The coefficients S_{si} of the power series of S represent *proportions* of the population that are susceptible, hence negative coefficients are nonsense.

3.2 Steady-state solutions

The purpose of this section is to characterize disease-free solutions. More precisely, we have the following lemma.

Lemma 1 *Any function $S = S^*(x)$, i.e. a function of x , is a disease-free steady-state solution to (7). Furthermore, these comprise the entire family of biologically relevant steady-state solutions.*

Proof Suppose $S = S(x, y)$ is a steady-state solution to (7). Then $S_{xy}(1, 1)/S_x(1, 1) = A$, a constant. Furthermore, S being biologically relevant implies that $A \geq 0$, as both S_{xy} and S_x have power series with non-negative coefficients. Equation (7) then becomes

$$-(\beta + \gamma) \left(y - \frac{\gamma}{\beta + \gamma} \right) S_y + A\beta(y - x)S_x = 0, \tag{8}$$

which can be solved via the method of characteristics. Assume that $A \geq 0$, and define the characteristic curves parameterized by τ as the solutions to

$$\frac{dx}{d\tau} = A\beta(y - x) \tag{9a}$$

$$\frac{dy}{d\tau} = -(\beta + \gamma) \left(y - \frac{\gamma}{\beta + \gamma} \right) \tag{9b}$$

$$\frac{dS}{d\tau} = 0. \tag{9c}$$

Solving the equation for y using an integrating factor gives

$$y(\tau) = \left(y_0 - \frac{\gamma}{\beta + \gamma} \right) e^{-(\beta+\gamma)\tau} + \frac{\gamma}{\beta + \gamma}, \tag{10}$$

where $y(0) = y_0$. Substitute $y(\tau)$ into the x equation to obtain

$$\frac{dx}{d\tau} + A\beta x = A\beta \left(y_0 - \frac{\gamma}{\beta + \gamma} \right) e^{-(\beta+\gamma)\tau} + A\beta \frac{\gamma}{\beta + \gamma}, \tag{11}$$

which can be solved again using an integrating factor, resulting in

$$\begin{aligned} x = & x_0 e^{-A\beta\tau} + \frac{A\beta}{A\beta - \beta - \gamma} \left(y_0 - \frac{\gamma}{\beta + \gamma} \right) \left(e^{-(\beta+\gamma)\tau} - e^{-A\beta\tau} \right) \\ & + \frac{\gamma}{\beta + \gamma} \left(1 - e^{-A\beta\tau} \right) \end{aligned} \tag{12}$$

The third characteristic from (9) is

$$\frac{dS}{d\tau} = 0, \tag{13}$$

thus the solution is constant along the curve $(x(\tau), y(\tau))$, i.e.,

$$S(x(\tau), y(\tau)) = c \tag{14}$$

In particular, for all $(x_0, y_0) \in [0, 1]^2$,

$$S(x(\tau), y(\tau)) = S(x_0, y_0) \tag{15}$$

where $(x(\tau), y(\tau))$ and (x_0, y_0) lie along the same characteristic curve. Fix (x, y, τ) , and write x_0 and y_0 in terms of those variables, i.e.

$$S(x, y) = S(x_0(\tau, x, y), y_0(\tau, x, y)) \tag{16}$$

where

$$x_0 = xe^{A\beta\tau} - \frac{A\beta}{A\beta - \beta - \gamma} \left(y - \frac{\gamma}{\beta + \gamma} \right) \left(e^{A\beta\tau} - e^{(\beta+\gamma)\tau} \right) - \frac{\gamma}{\beta + \gamma} \left(e^{A\beta\tau} - 1 \right) \tag{17a}$$

$$y_0 = \left(y - \frac{\gamma}{\beta + \gamma} \right) e^{(\beta+\gamma)\tau} + \frac{\gamma}{\beta + \gamma}. \tag{17b}$$

Take derivatives in x and y of (16)

$$S_x(x, y) = \frac{\partial S}{\partial x_0} \frac{\partial x_0}{\partial x} \tag{18}$$

$$S_{xy}(x, y) = \frac{\partial S}{\partial x_0} \frac{\partial^2 x_0}{\partial x \partial y} + \left(\frac{\partial^2 S}{\partial x_0^2} \frac{\partial x_0}{\partial y} + \frac{\partial^2 S}{\partial x_0 \partial y_0} \frac{\partial y_0}{\partial y} \right) \frac{\partial x_0}{\partial x}. \tag{19}$$

Substituting this into the expression for A gives the compatibility condition

$$A = \frac{S_{xy}(s, 1, 1)}{S_x(s, 1, 1)} \tag{20}$$

$$= \frac{\frac{\partial^2 S}{\partial x_0^2} \frac{\partial x_0}{\partial y} + \frac{\partial^2 S}{\partial x_0 \partial y_0} \frac{\partial y_0}{\partial y}}{\frac{\partial S}{\partial x_0}} \Bigg|_{x=y=1} \tag{21}$$

$$= \frac{\frac{\partial^2 S}{\partial x_0^2} \frac{A\beta}{A\beta - \beta - \gamma} \left(e^{(\beta+\gamma)\tau} - e^{A\beta\tau} \right) + \frac{\partial^2 S}{\partial x_0 \partial y_0} e^{(\beta+\gamma)\tau}}{\frac{\partial S}{\partial x_0}} \Bigg|_{x=y=1} \tag{22}$$

The LHS of equation (22) is a constant, and the RHS depends on τ . Since the RHS is a linear combination of functions, the only solution for the RHS to be τ independent is for $A = 0$. When $A = 0$, equation (8) gives $S_y = 0$ and so

$$S = S^*(x). \tag{23}$$

□

Definition 2 A *disease-free steady state* of the PDE (7) is a biologically relevant solution that is y -independent, i.e. $S = S^*(x)$.

Such a steady-state solution has power series coefficients $S_{si} = 0$ for all $i > 0$. Hence, the network is disease-free.

3.3 A change of variables

To properly introduce the topology of the functional space, we consider the change of variables

$$w = x - \Gamma, \quad z = y - \Gamma, \quad \tilde{S}(t, w, z) = S(t, x, y) \tag{24}$$

and

$$\tilde{S}(t, w, z) = \sum_{m \geq 0, n \geq 0} w^m z^n s_{mn}(t), \tag{25}$$

where $\Gamma := \gamma / (\beta + \gamma)$. We will show later that the point $(x, y) = (\Gamma, \Gamma)$ is the fixed point (i.e, a constant solution in time) of the characteristics, where all the characteristic lines emerge.

Substitute $S = \tilde{S}$ into (7) to derive the PDE in terms of the new variables

$$\tilde{S}_t = -(\beta + \gamma)z\tilde{S}_z + \beta \frac{\tilde{S}_{wz}(t, 1 - \Gamma, 1 - \Gamma)}{\tilde{S}_w(t, 1 - \Gamma, 1 - \Gamma)}(z - w)\tilde{S}_w \tag{26}$$

with initial condition

$$\tilde{S}(0, w, z) = \tilde{S}_0(w, z). \tag{27}$$

Relate the disease-free equilibrium to the PDE (26) by $\tilde{S}(w) = S^*(x)$.

The relationship between the coefficients of S and \tilde{S} is determined by substitution of $x = w + \Gamma$ and $y = z + \Gamma$ into S :

$$S(t, w + \Gamma, z + \Gamma) = \sum_{s,i} (w + \Gamma)^s (z + \Gamma)^i S_{si}(t) \tag{28a}$$

$$= \sum_{m,n} w^m z^n s_{mn}(t). \tag{28b}$$

Use of the binomial theorem, or a Taylor expansion at $w = z = 0$, gives the desired relation

$$s_{mn}(t) := \sum_{s \geq m, i \geq n} \binom{s}{m} \binom{i}{n} \Gamma^{s+i-m-n} S_{si}(t). \tag{29}$$

Remark 3 If $S(t, x, y)$ is biologically relevant according to definition 1, then the coefficients of $\tilde{S}(t, w, z)$ are non-negative. This is clear by consideration of equation (29).

Remark 4 An equivalent change of variable $x = \Gamma + (1 - \Gamma)w$ and $y = \Gamma + (1 - \Gamma)z$ would normalize the unit square to $[-\Gamma/(1 - \Gamma), 1]^2$, and the PDE becomes

$$\tilde{S}_t = -(\beta + \gamma)z(1 - \Gamma)\tilde{S}_z + \beta(1 - \Gamma)^2 \frac{\tilde{S}_{wz}(t, 1, 1)}{\tilde{S}_w(t, 1, 1)}(z - w)\tilde{S}_w$$

This has the advantage that w and z are probability generation functions of a Bernoulli random variable with a success probability $1 - \Gamma$, i.e., the probability that an infectious node eventually transmits. Thus, $\tilde{S}(t, w, z)$ has a clearer biological meaning as the probability generating function for the join distribution of the number of susceptible and infectious neighbors (of a susceptible node) that eventually transmitted.

Define the square $R = [-\Gamma, 1 - \Gamma]^2$, and let X be the space of functions $\tilde{S} : [0, \infty) \times R \rightarrow \mathbb{R}$ with \tilde{S} defined in (25), equipped with the norm

$$\|\tilde{S}\|_X = \sum_{m,n} (1 + m)|s_{mn}| < \infty. \tag{30}$$

This change of variables ensures that the space of solutions is equipped with a norm that is mathematically tractable.

4 Well-posedness

The system (26) is well-posed if the solution with a given initial condition exists and is unique. In Sect. 4.1, we find the general solution of the system using the method of characteristics; in Sect. 4.2 we show that this solution is uniquely determined.

From here on, confusion between the w, z and x, y variables should be minimal, and so we drop the “tilde” notation, i.e. $S = S(t, w, z)$.

4.1 General solution via the method of characteristics

Write the PDE (26) in the form

$$S_t + (\beta + \gamma)zS_z + \beta(w - z)\phi'(t)S_w = 0 \tag{31}$$

where $\phi(t)$ is given by

$$\phi(t) = \int_0^t \frac{S_{wz}(\tau, 1 - \Gamma, 1 - \Gamma)}{S_w(\tau, 1 - \Gamma, 1 - \Gamma)} d\tau \tag{32}$$

such that

$$\phi'(t) = \frac{S_{wz}(t, 1 - \Gamma, 1 - \Gamma)}{S_w(t, 1 - \Gamma, 1 - \Gamma)}. \tag{33}$$

By treating $\phi'(t)$ as a known function, equation (31) can be solved by the method of characteristics. Assume that $\phi'(t) \neq 0$, because otherwise the PDE is linear and easily solved. The characteristics are given by

$$\frac{dS}{dt} = 0 \tag{34a}$$

$$\frac{dz}{dt} = (\beta + \gamma)z \tag{34b}$$

$$\frac{dw}{dt} = \beta(w - z)\phi'(t). \tag{34c}$$

Notice that $w = z = 0$, i.e. $x = y = \Gamma$ is the fixed point of (34).

The solution to the system (34) is constant in time along the characteristic curve $(w(t), z(t))$, i.e.

$$\begin{aligned} S(t, w(t), z(t)) &= S_0(w_0, z_0) \\ z(t) &= z_0 e^{(\beta+\gamma)t} \\ w(t) &= w_0 e^{\beta\phi(t)} \\ &\quad + z_0 \left(e^{(\beta+\gamma)t} - e^{\beta\phi(t)} - (\beta + \gamma)e^{\beta\phi(t)} \int_0^t e^{(\beta+\gamma)\tau - \beta\phi(\tau)} d\tau \right) \end{aligned} \tag{35}$$

where $(w_0, z_0) = (w(0), z(0))$. In terms of w and z , the solution is

$$S(t, w, z) = S_0(w_0, z_0) \tag{36a}$$

$$z_0(t, z) = z e^{-(\beta+\gamma)t} \tag{36b}$$

$$w_0(t, w, z) = w e^{-\beta\phi(t)} + z\eta(t, \phi(t)) \tag{36c}$$

where

$$\eta(t, \phi(t)) := e^{-(\beta+\gamma)t} - e^{-\beta\phi(t)} + (\beta + \gamma)e^{-(\beta+\gamma)t} \int_0^t e^{(\beta+\gamma)\tau - \beta\phi(\tau)} d\tau. \tag{37}$$

We have found the general solution to the system (26) in terms of ϕ and η . In the following subsection we determine ϕ and η , and show that this solution is unique.

4.2 Existence and uniqueness

As long as ϕ and η are unique, then w_0 given by (36c) is unique, and hence the solution to the system (26) is unique. The solution (36) to the PDE (26) was derived assuming that $\phi(t)$ was a known function. However, this is only a solution if $\phi(t)$ satisfies the *compatibility condition*

$$\phi'(t) = \frac{S_{wz}(t, 1 - \Gamma, 1 - \Gamma)}{S_w(t, 1 - \Gamma, 1 - \Gamma)} \tag{38}$$

By taking partial derivatives of (36a) and a time derivative of (37) the compatibility condition becomes

$$\begin{pmatrix} \phi'(t) \\ \eta'(t) \end{pmatrix} = \begin{pmatrix} f(t, \phi, \eta)e^{-(\beta+\gamma)t} + h(t, \phi, \eta)\eta \\ f(t, \phi, \eta)\beta e^{-(\beta+\gamma)t-\beta\phi} + [h(t, \phi, \eta)\beta e^{-\beta\phi} - \beta - \gamma]\eta \end{pmatrix} \tag{39}$$

where $\phi(0) = 0$ and $\eta(0) = 0$ by definition, and

$$\begin{aligned} f(t, \phi, \eta) &= \left. \frac{\frac{\partial^2 S_0}{\partial w_0 \partial z_0}}{\frac{\partial S_0}{\partial w_0}}(w_0, z_0) \right|_{w=z=1-\Gamma} \\ h(t, \phi, \eta) &= \left. \frac{\frac{\partial^2 S_0}{\partial w_0^2}}{\frac{\partial S_0}{\partial w_0}}(w_0, z_0) \right|_{w=z=1-\Gamma} \end{aligned} \tag{40}$$

From here on, we will assume the following is true:

Assumption 1 $S_0(w, z)$ is biologically relevant, $S_0 \in C^3(R)$, and $\frac{\partial S_0}{\partial w} \neq 0$.

Lemma 2 Under assumption 1, $\phi'(t) > 0$, where $\phi'(t)$ is given by (39).

Proof First, under the assumption that S_0 is biologically relevant, the functions $f, h > 0$ for all time, where f and h are given by equation (40).

To see this, notice that since S_0 is a biologically relevant initial condition, it has a power series with positive coefficients, and thus its derivatives will also have this property. The functions f and h are quotients where the numerator and denominator are derivatives of S_0 evaluated at a point. Hence, if the point that these functions are evaluated at is greater than or equal to zero, then f and h are quotients of power series with positive coefficients evaluated at a positive point, i.e. $f, h > 0$. Thus it suffices to show that

$$\begin{aligned} &(w_0, z_0) \Big|_{w=z=1-\Gamma} \\ &= (1 - \Gamma) \left(e^{-(\beta+\gamma)t} + (\beta + \gamma)e^{-(\beta+\gamma)t} \int_0^t e^{(\beta+\gamma)s-\beta\phi(s)} ds, e^{-(\beta+\gamma)t} \right) > 0. \end{aligned} \tag{41}$$

Indeed, each coordinate is positive, as $\Gamma < 1$, and the remaining terms are non-negative.

Next, we want to show that $\phi' > 0$. From the system (39), it suffices to prove that $\eta(t) \geq 0$. Notice that at $t = 0$, we have $\eta(0) = 0$ from (37), and

$$\eta'(0) = f(0, \phi, \eta)\beta. \tag{42}$$

Since $f, \beta \geq 0$ there are three possibilities:

- i. η is zero for all t .

- ii. η increases away from zero initially.
- iii. $\eta'(0) = 0$, and η is zero until just after some time $t_1 > 0$, where it increases away from 0 because

$$\eta'(t_1) = f(t_1, \phi, \eta)\beta e^{-(\beta+\gamma)t_1-\beta\phi} \tag{43}$$

In any case, $\eta(t) \geq 0$, and so $\phi'(t) > 0$. □

Lemma 3 *If assumption 1 is satisfied, then characteristic curves given by (36b) and (36c) flow out of the square $R = [-\Gamma, 1 - \Gamma]^2$ in finite time. This implies that $(w_0, z_0) \in R$ for all $(w, z) \in R$.*

Proof We first observe from the (34b) that z grows exponentially in time, and thus the characteristic lines hit the boundary of R in finite time. We then show that, once hit the boundary, the characteristic lines flow out by calculating the normal component p of the characteristic flow through the boundary of the square R as

$$p = \mathbf{n} \cdot \left(\frac{dw}{dt}, \frac{dz}{dt} \right), \tag{44}$$

where \mathbf{n} is the outward unit normal. For example, along the left boundary $w = -\Gamma$, $z \in (-\Gamma, 1 - \Gamma)$ we have

$$p = (-1, 0) \cdot (\beta\phi'(t)(-\Gamma) - \beta\phi'(t)z, \frac{dz}{dt}) \tag{45a}$$

$$= \beta\phi'(t)(z + \Gamma) \tag{45b}$$

$$> 0 \tag{45c}$$

because $\phi' > 0$ by lemma 2 and $z > -\Gamma$. Thus the characteristic lines flow out of this boundary.

The idea is similar for the remaining boundaries. □

Remark 5 Thanks to this Lemma, our PDE model (31) does not need a boundary condition. This is because, for any time $t > 0$ and $w, z \in R$, the solution to the PDE at (t, w, z) is defined by the initial condition S_0 at the point (w_0, z_0) traced back along the characteristic line passing through (w, z) . This lemma guarantees that $(w_0, z_0) \in R$, and thus the solution is defined at (t, w, z) .

Theorem 1 *If $S_0(w, z)$ satisfies assumption 1, then the system (39) has a unique solution defined for all time $t \in [0, \infty)$.*

Proof Since $S_0 \in C^3(-\Gamma, 1 - \Gamma)$, we have that f and h are continuously differentiable in t, ϕ , and η , for all (t, ϕ, η) such that $w_0(t, 1 - \Gamma, 1 - \Gamma)$ and $z_0(t, 1 - \Gamma, 1 - \Gamma)$ are in the square R . Rewrite the system (39) as

$$y' = F(t, y). \tag{46}$$

For $F(t, y)$ continuous on the closure \bar{E} of an open (t, y) -set E , ODE existence and uniqueness (Picard-Lindelöf) and extension theorems (see for example Hartman 1982) give the existence and uniqueness of the solution $y(t)$ on some maximal interval J given by one of the following

1. $J = [t_0, \delta]$ with $\delta < \infty$ and $(\delta, y(\delta)) \in \partial E$
2. $J = [t_0, \delta)$ with $\delta < \infty$ and $|y(t)| \rightarrow \infty$ as $t \rightarrow \delta$
3. $J = [t_0, \infty)$

Eliminate the first possibility by recognizing that the only way to hit the boundary ∂E is for (w_0, z_0) to be outside the square R at some time t . Lemma 3 guarantees that this cannot happen.

To eliminate the second possibility, $S_0 \in C^3(-\Gamma, 1 - \Gamma)$ gives f and h in (40) as continuous, and defined on a compact set. Hence f and h are bounded. Let $M := \max(|f|, |h|)$, then by considering (39), we get the bounds

$$\phi(t) \leq \frac{e^{M\beta t}}{\beta} \tag{47a}$$

$$\eta(t) \leq e^{M\beta t} - 1. \tag{47b}$$

The only remaining possibility for the interval of existence is $J = [0, \infty)$. □

5 Reduction to the Volz model

The goal of this section is to show that by assuming solutions given by a multinomial distribution, the effective degree model reduces to the Volz model (Volz 2008).

Assume a generating function solution to (26) of the form

$$S(t, w, z) = \sum_k p_k S_k(t) (p_S(t)(w + \Gamma - 1) + p_I(t)(z + \Gamma - 1) + 1)^k \tag{48}$$

where $S_k(t)$ is the proportion of susceptible nodes having degree k at time t , $k = s + i$ is the effective degree of a node, and $p_S(t)$ and $p_I(t)$ are the probabilities of a susceptible node having a susceptible/infected neighbour at time t , respectively.

Our goal now is to show that there exist solutions of the form (48) satisfying the PDE (26). Substitute (48) into (26),

$$\begin{aligned} & \sum_{k=0}^{\infty} p_k k S_k (p_S(w + \Gamma - 1) + p_I(z + \Gamma - 1) + 1)^{k-1} \\ & \quad \times ((w + \Gamma - 1)p'_S + (z + \Gamma - 1)p'_I) \\ & \quad + p_k S'_k (p_S(w + \Gamma - 1) + p_I(z + \Gamma - 1) + 1)^k \\ & = -(\beta + \gamma)z \sum_{k=0}^{\infty} k p_k S_k (p_S(w + \Gamma - 1) + p_I(z + \Gamma - 1) + 1)^{k-1} p_I \end{aligned}$$

$$\begin{aligned}
 & + \beta \frac{\sum_{k=0}^{\infty} k(k-1)p_k S_k p_I p_S}{\sum_{k=0}^{\infty} k p_k S_k p_S} (z-w) \sum_{k=0}^{\infty} k p_k S_k (p_S(w + \Gamma - 1) \\
 & + p_I(z + \Gamma - 1) + 1)^{k-1} p_S.
 \end{aligned} \tag{49}$$

Compare coefficients of $(p_S(w + \Gamma - 1) + p_I(z + \Gamma - 1) + 1)^{k-1}$:

$$\begin{aligned}
 & (k S_k p'_S + S'_k p_S)w + (k S_k p'_I + S'_k p_I)z + (\Gamma - 1)k S_k (p'_S + p'_I) \\
 & + (\Gamma - 1)S'_k (p_S + p_I) + p_k S'_k \\
 & = -(\beta + \gamma)z k S_k p_I + \beta \frac{\sum_{k=0}^{\infty} k(k-1)S_k}{\sum_{k=0}^{\infty} k S_k} (z-w)k S_k p_S p_I.
 \end{aligned} \tag{50}$$

Now compare coefficients of $w, z,$ and constant terms

$$k S_k p'_S + S'_k p_S = -\beta \frac{\sum_{k=0}^{\infty} k(k-1)p_k S_k}{\sum_{k=0}^{\infty} k p_k S_k} k S_k p_S p_I \tag{51a}$$

$$k S_k p'_I + S'_k p_I = -(\beta + \gamma)k S_k p_I + \beta \frac{\sum_{k=0}^{\infty} k(k-1)p_k S_k}{\sum_{k=0}^{\infty} k p_k S_k} k S_k p_S p_I \tag{51b}$$

$$(\Gamma - 1)S'_k (p_S + p_I) + (\Gamma - 1)k S_k (p'_S + p'_I) + S'_k = 0 \tag{51c}$$

Add equations (51a) and (51b) to find

$$k S_k (p'_S + p'_I) + S'_k (p_S + p_I) = -(\beta + \gamma)k S_k p_I. \tag{52}$$

Divide (51c) by $(1 - \Gamma)$, and add it to equation (52) to get an ODE for S_k :

$$S'_k(t) = -\beta k S_k(t) p_I(t) \tag{53}$$

which has a solution

$$S_k(t) = \exp \left\{ - \int_0^t \beta p_I(\tau) d\tau \right\}^k, \tag{54}$$

or

$$S_k = S_1^k. \tag{55}$$

Replacing S'_k in (51) with (53) and S_k with (55) gives

$$p'_I = \beta p_I^2 - (\beta + \gamma)p_I + \beta \frac{\sum_{k=0}^{\infty} k(k-1)p_k S_1^k}{\sum_{k=0}^{\infty} k p_k S_1^k} p_S p_I \tag{56a}$$

$$p'_S = \beta p_I p_S - \beta \frac{\sum_{k=0}^{\infty} k(k-1)p_k S_1^k}{\sum_{k=0}^{\infty} k p_k S_1^k} p_S p_I \tag{56b}$$

$$S'_1(t) = -\beta S_1(t) p_I(t) \tag{56c}$$

Comparison with (Volz 2008) shows that we have reduced the PDE effective degree model to the Volz model ($S_1 = \theta$ in their formulation). This computation leads to the following theorem

Theorem 2 *A function given by (48) is a solution to the PDE (26) if and only if p_S , p_I , and S_1 satisfy the Volz model (Volz 2008).*

6 Discussion

Using a generating function approach, we derive a single partial differential equation from the system of ordinary differential equations for the network effective degree SIR model. The power series coefficients of solutions correspond to the compartments of the original model. This new approach extends the effective degree SIR model to be able to include networks having infinite degrees, e.g. theoretical Poisson and scale-free networks.

The resulting PDE (7) or (26) is a transport equation governing the probability distribution of the number of susceptible and infectious neighbors of a susceptible node. Mathematically, this PDE is interesting for two reasons: its lack of boundary conditions, and its non-local non-linearity coming from the term $\frac{S_{xy}(t, 1, 1)}{S_y(t, 1, 1)}$ on the RHS of (7). We've shown the existence and uniqueness of solutions to the model (for fairly unrestrictive initial conditions) by consideration of the compatibility condition.

If the infection status of a neighbor of a susceptible node is initially independent to that of other neighbors, then the numbers of susceptible, infectious or recovered neighbors of a susceptible are multinomially distributed. If this independence is initially true, the effective degree model guarantees that this independence holds for all future time, because the multinomial generating function is a solution to the PDE. We've also shown that, under this assumption, the effective degree model reduces to the Volz model (Volz 2008).

The results of this paper setup the PDE effective degree SIR model for further study. As a future research direction, we will consider the linear and nonlinear stability of the disease-free equilibrium, and will show that the stability of the PDE model is governed by the same disease threshold condition as the ODE model.

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References

- Eames KTD, Keeling MJ (2002) Modeling dynamic and network heterogeneities in the spread of sexually transmitted diseases. *Proc Natl Acad Sci* 99:13330–13335
- Hartman P (1982) *Ordinary Differential Equations*, 2nd edn. Birkhäuser, Boston, Basel, Stuttgart
- Hethcote HW (2000) The mathematics of infectious diseases. *SIAM Rev* 42:599–653
- Johnson NL, Kemp AW, Kotz S (2005) *Univariate discrete distributions*, vol 444. John Wiley & Sons, New Jersey

- Kermack W, McKendrick A (1927) A contribution to the mathematical theory of epidemics. *Proc R Soc Lond A* 115:700–721
- Kiss IZ, Miller JC, Simon PL (2017) *Mathematics of Epidemics on Networks*. Springer International Publishing AG, Switzerland
- Lindquist J, Ma J, van den Driessche P, Willeboordse F (2011) Effective degree network disease models. *J Math Biol* 62:143–164
- Miller JC (2011) A note on a paper by Erik Volz: SIR dynamics in random networks. *J Math Biol* 62:349–358
- Miller JC, Kiss IZ (2014) Epidemic spread in networks: Existing methods and current challenges. *Math Model Nat Phenom* 9:4–42
- Pastor-Satorras R, Vespignani A (2002) Epidemic dynamics in finite size scale-free networks. *Phys Rev E* 65:035108(R)
- van den Driessche P, Watmough J (2002) Reproduction numbers and subthreshold endemic equilibria of compartmental models for disease transmission. *Math Biosci* 180:29–48
- Volz E (2008) SIR dynamics in random networks with heterogeneous connectivity. *J Math Biol* 56:293–310

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