The nonlinear dynamics of epidemics and networks

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Contents

1 Introduction

Since the onset of COVID19 over two years ago disease transmission and epidemiology have gained much relevance. In particular, focus has been placed on designing mathematical models which can accurately predict the spread of disease both in space and time. These models have been especially important as many public policy decisions have been influenced by their predictions. [13, 16]

However most systems are often presented from a computational perspective and epidemic spread patterns are predicted using Monte-Carlo methods [9]. Thus, introducing concepts from nonlinear science to understand these models from a more analytic or theoretical perspective could be of great interest.

This paper introduces the mathematics and dynamics of disease transmission, going over basic models in section 2 such as the famous SIR equations, and a particular system is analysed with the lens of weakly nonlinear theory. The complexity is gradually increased so that it can account for more spacial and temporal factors. In section 3 we model spatial effects by introducing diffusion into the problem, and find an asymptotic solution to a simplified case of the equations. Finally, we model transmission in nonhomogenous media by introducing Network Science. Equations are derived to model the emergence of outbreaks on complex

networks, and different approximations are studied, with a particular focus on how the spread depends on the structure of the network.

In the context of nonlinear dynamics the most relevant sections are 2.3 where we present an application of multi-timescale methods to an epidemiology problem, section 3.2 where techniques from perturbation theory are used to obtain an asymptotic formula for the wavefront, and section 4 presenting epidemics on networks.

2 Basic compartmental models

To model epidemic outbreaks at a basic level we divide a population into several compartments, and model the spread of the disease with interaction terms that 'connect' the compartments. The number of compartments will depend on the particular characteristics of a disease. There are four fundamental ways of doing this, with varying levels of complexity, and none of them account for spatial effects. We name the different models with the initials of the compartments they connect. [1]

1. **SI model**: we have two compartments: *susceptible*[1] (S) and *infected* people (I). The transmission from susceptible to infected is modeled with a $\sim SI$ term, giving rise to the following equations [23]

$$\partial_t S = -\beta SI \qquad \partial_t I = \beta SI$$

   Here β is a disease specific parameter that roughly models how infectious the pathogen is.

2. **SIS model**. Same compartments as SI model, but infected individuals can become susceptible after infection. This is modelled with a $\sim \gamma I$ term. The governing equations for SIS are [6]:

$$\partial_t S = -\beta SI + \gamma I \qquad \partial_t I = \beta SI - \gamma I$$

3. **SIR model**. Three compartments: susceptible, infected and recovered individuals (R). Once recovered/immune, an individual is always immune. This is generally the first model used to study an outbreak (most pathogens fit into this model), and thus we will look at it in detail in section 2.1.

4. **SIRS model**. Same compartments as SIR but recovered individuals can loose immunity and become susceptible. We will look at this system in section 2.2.

To model each system we will need an ODE for the size of each of the compartments. However all systems are subject to a holonomic constraint: the total number of individuals in the system is conserved [1]. This will reduce the dimensionality of the system by one degree of freedom.

This simplification makes the first two systems into one dimensional first order equations, which are separable and fully integrable, so they are not of particular interest an analytical solution can be obtained[6].

However, SIR and SIRS will become two dimensional non linear systems of equations with no known analytic solutions [6]. Hence, we will explore them in more detail in the upcoming sections to get acquainted with the mathematics of epidemiology before moving on to spatial considerations.

For diseases were a vaccine is available the standard procedure is incorporating a 'vaccinated' compartment and vaccinate susceptible individuals in a linear way. Mathematically this will just shrink the susceptible group. For more details on the role vaccination plays in compartmental models please see [11].

2.1 SIR model: an introduction to mathematical epidemiology

The SIR model is the most basic tool used to describe the spread of many diseases [10]. Whilst it has many drawbacks (the lack of spatial dependencies being one of them) it captures many characteristics of real world

[1]Simply defined as individuals who can get infected, meaning they have not gained immunity from natural infection or vaccination

pathogens with a simple set of ordinary differential equations. Although they are non-linear and have no analytic solution they are easy and cheap to integrate numerically [10]. As described previously, a population with N members is divided into three disjoint compartments: S the susceptible individuals, I the infected individuals and R the recovered individuals. The governing equations are [23]:

$$\frac{dS}{dt} = -\beta SI \quad \frac{dI}{dt} = \beta SI - \gamma I \quad \frac{dR}{dt} = \gamma I \tag{1}$$

As they are constrained by $N = S + I + R =$ constant we can focus on the first two equations and ignore the last one. We see they are first order equations and the nonlinearity comes from an interaction term of the form SI. In particular, this means that $I = 0$, the null state, is a fixed point of the system for any value of S and R, known in the literature as the *disease free* equilibrium. The initial conditions are usually of the form

$$S(0) = N - I_0 \quad I(0) = I_0 \quad R(0) = 0$$

with $I_0 << N$. We can nondimensionalise using $\tilde{t} = \gamma t$ as the nonlinear time and $S/N \ldots$ as the nonlinear population densities. This gives us (dropping the $\sim$) the following nondimensional system

$$S_t = -\frac{\beta N}{\gamma} SI \quad I_t = \frac{\beta N}{\gamma} SI - I$$

We can define $R_0 = \frac{\beta N}{\gamma}$. This is the famous *basic reproduction number*. We can see why it is important by linearizing the above around the fixed point $(S, I) = (1, 0)$ (the disease free equilibrium)

$$S_t = -R_0 I \qquad I_t = R_0 I - I = (R_0 - 1)I$$

This will have exponential growth when $R_0 > 1$ and hence we see why the reproduction number is the crux of the SIR model, as we can predict if there will be epidemic spread or not just by looking at single quantity. Indeed, much of public health policy is aimed at driving this number down.

2.2 The SIRS model

The SIRS model is very similar to the aforementioned SIR, with a small change that has a significant effect on the behaviour of the system. We incorporate a mechanism for individuals to migrate from the recovered compartment to the susceptible compartment. Physically, this means that recovered patients will loose immunity with time. How fast this happens is controlled by the parameter δ. The governing equations are similar to those presented before [10]:

$$\frac{dS}{dt} = -\beta SI + \delta R \quad \frac{dI}{dt} = \beta SI - \gamma I \quad \frac{dR}{dt} = \gamma I - \delta R \tag{2}$$

However, this model has a surprise waiting for us: a fixed point with non-zero infections!. This is known in epidemiology as the *endemic* equilibrium. This equilibrium is (again, as the system is constrained it suffices to look at the first two equations) [10]

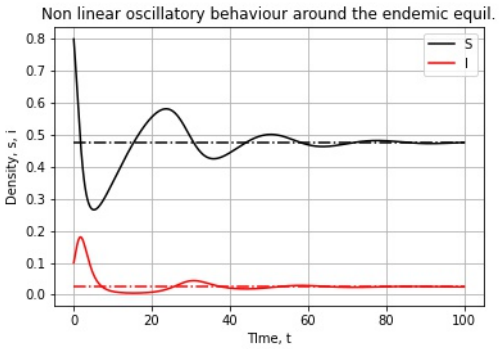
$$S^* = \frac{\gamma}{\beta} = \frac{1}{\sigma} \quad I^* = \frac{N - \frac{\gamma}{\beta}}{1 + \frac{\gamma}{\delta}} = \frac{N - \frac{1}{\sigma}}{1 + q}$$

where we have defined the *contact number* $\sigma = \frac{\beta}{\gamma}$ and the ratio $q = \frac{\gamma}{\delta}$. The careful reader might have noticed that for this equilibrium to have physical significance we require all the components to be non-negative (as they represent a number of individuals). Focusing on I^* this means

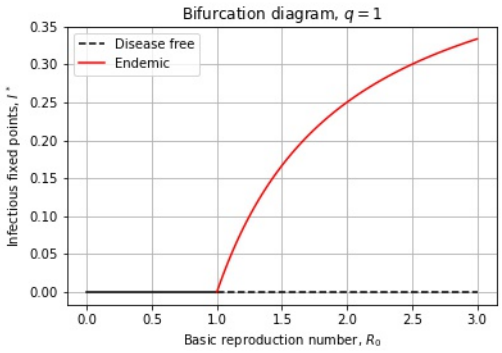
$$N - \frac{\gamma}{\beta} > 0 \iff \frac{N\beta}{\gamma} = R_0 > 1$$

with R_0 the basic reproduction number as defined for the SIR model. This in turns means that the disease free equilibrium $(N, 0, 0)$ is unstable, which suggests a bifurcation happens at the critical value $R_0 = 1$ where the endemic equilibrium appears (and we expect it to be stable) and the disease free equilibrium becomes unstable as the reproduction number is increased.

Indeed we can verify that the requirement for linear stability of the endemic equilibrium is $R_0 > 1$ [1], which is the same as the requirement for the existence of the fixed point. Therefore the endemic equilibrium is always stable. A bifurcation diagram is available in Figure 1b. This stability suggests there might be an oscillatory solution around this equilibrium that eventually converges, and that we might be able to apply weakly nonlinear techniques. Indeed, direct simulation confirms this, as evident in Figure 1a



(a) Oscillatory behaviour around the endemic equilibrium for an SIRS system.



(b) Bifurcation plot for the SIRS system, focusing on the infectious fixed points. Note the (pitchfork) bifurcation at $R_0 = 1$. We represent stability with solid lines and instability with dashed lines.

Figure 1: Visualisations for the SIRS system

2.3 Weakly nonlinear analysis of SIRS

This sections assumes that $N = 1$. This is equivalent to working with population densities. We will also require that $\sigma = \frac{\beta}{\gamma} > 1$, so that the endemic equilibrium exists and is stable. We can see from the linearization that the system is close to a damped harmonic oscillator. We can write the equations for small perturbations from the endemic equilibrium as

$$S = S^* + \varepsilon x \quad I = I^* + \varepsilon y \quad R = R^* + \varepsilon z$$

with ε a small parameter. Upon substitution to the governing equation (2) we get,

$$\frac{dx}{dt} = -\varepsilon\sigma xy + \frac{z}{q} \quad \frac{dy}{dt} = \frac{\sigma - 1}{q + 1}x + \varepsilon\sigma xy \quad \frac{dz}{dt} = y - \frac{z}{q} \tag{3}$$

as $S + I + R = 1$ we have that the perturbation variables are constrained by $x + y + z = 0$ and it suffices to study the last two equations, and noting that $x = -y - z$ we reduce the above to

$$\frac{dy}{dt} = -(y + z)\left(\frac{\sigma - 1}{q + 1} + \varepsilon\sigma y\right) \quad \frac{dz}{dt} = y - \frac{z}{q}$$

We will write this system as a nonlinear harmonic oscillator for z. From the second equation above we can take a derivative

$$y = \frac{dz}{dt} + \frac{z}{q} \Longrightarrow \frac{dy}{dt} = \frac{d^2z}{dt^2} + \frac{1}{q}\frac{dz}{dt}$$

So equating this with the second equation in (3)

$$\frac{d^2z}{dt^2} + \frac{1}{q}\frac{dz}{dt} = \frac{dy}{dt} = -(y+z)\left(\frac{\sigma-1}{q+1} + \varepsilon\sigma y\right)$$

Substituting the expression for y we get an equation that only involves z and its derivatives.

$$\frac{d^2z}{dt^2} + \frac{1}{q}\frac{dz}{dt} = \frac{dy}{dt} = -\left(\frac{dz}{dt} + \frac{z}{q} + z\right)\left(\frac{\sigma-1}{q+1} + \varepsilon\sigma\left(\frac{dz}{dt} + \frac{z}{q}\right)\right)$$

after some algebra, we can group the above to

$$\frac{d^2z}{dt^2} + \left(\frac{\sigma-1}{q+1} + \frac{1}{q}\right)\frac{dz}{dt} + \frac{\sigma-1}{q}z = -\varepsilon\sigma\left(\left(\frac{dz}{dt}\right)^2 + z\frac{dz}{dt}\frac{q+2}{q} + z^2\frac{q+1}{q^2}\right) \tag{4}$$

this is indeed a nonlinear harmonic oscillator, and we can apply ideas from weakly nonlinear analysis. We will define the following quantities, which will be helpful for our analysis

$$\omega^2 = \frac{\sigma-1}{q} \quad G = \frac{q\sigma-1}{q(q+1)} \quad k = \frac{q+2}{q} \quad \delta = \varepsilon\sigma$$

So we can write the system as

$$\frac{d^2z}{dt^2} + G\frac{dz}{dt} + \omega^2 z = -\delta\left(\left(\frac{dz}{dt}\right)^2 + z\frac{dz}{dt}k + z^2\frac{q+1}{q^2}\right) \tag{5}$$

We can non-dimensionalize using $t = \omega\bar{t}$ (z is a relative value so it is non-dimensional), and dropping "-"

$$\frac{d^2z}{dt^2} + G/\omega\frac{dz}{dt} + z = -\delta\left(\left(\frac{dz}{dt}\right)^2 + \frac{k}{\omega}z\frac{dz}{dt} + \frac{q+1}{\omega^2q^2}z^2\right) \tag{6}$$

For convenience we will discard the $\sim z^2$ term as with the parameter values we use in practice it will be small (it is roughly $\sim \frac{1}{\sigma-1}$ when compared to the first nonlinearity). We hope to revisit this assumption in the future. We can also define $B = G/\omega$ and $\kappa = \frac{k}{\omega}$. We can find the characteristic time for the other nonlinearities and the linear damping using a naive expansion. Note that as the nonlinearities are quadratic we have to go $\mathcal{O}(\delta^2)$ to find these times. We find that

1. Characteristic time for damping is $\sim \frac{1}{B}$
2. Characteristic time for $\dot{z}^2$ nonlinearity is $\sim \frac{1}{\delta^2}$
3. Characteristic time for $\dot{z}z$ nonlinearity is $\sim \frac{1}{(\kappa\delta)^2}$

Now, recall that $\sigma > 1$ and we will choose $q > 1$. Therefore we require $q \sim \sigma - 1$ for the characteristic time of the first two nonlinearities to be similar, so we will assume it. Lastly, to equate the characteristic time of the damping with the characteristic time of the nonlinearities we require that $B \sim \delta^2 t$ (which we can control with ε). Hence with this in mind we define the slow time $T = \delta^2$ and $\mu = \frac{B}{\delta^2}$, and the reduced system is without $\sim z^2$ will be

$$\frac{d^2z}{dt^2} + \mu\delta^2\frac{dz}{dt} + z = -\delta\left(\left(\frac{dz}{dt}\right)^2 + z\frac{dz}{dt}\kappa\right)$$

We will find a multi-timescale series expansion solution of the form

$$z = \theta_0(t,T) + \delta\theta_1(t,T) + \delta^2\theta_2(t,T) + \dots$$

For $\mathcal{O}(1)$ we obtain a standard harmonic oscillator so $\theta_0 = \{A(T)e^{it} + cc\}$. For the first order term $\mathcal{O}(\delta)$ we have the following system

$$\frac{\partial^2\theta_1}{\partial t^2} + \theta_1 + \left(\frac{\partial\theta_0}{\partial t}\right)^2 + \kappa\theta_0\frac{\partial\theta_0}{\partial t} = 0$$

We can substitute $\partial_t\theta_0 = \{iA(T)e^{it} + cc\}$ resulting in

$$\left(\frac{\partial\theta_0}{\partial t}\right)^2 = \{-A^2e^{2it} + AA^* + cc\} \qquad \kappa\theta_0\frac{\partial\theta_0}{\partial t} = \kappa\{iA^2e^{2it}\}$$

$$\frac{\partial^2\theta_1}{\partial t^2} + \theta_1 = -\{-A^2e^{2it} + AA^* + cc\} - \kappa\{iA^2e^{2it}\}$$

So we can solve for θ_1 using several ansatzs: $\theta_1(t,T) = \{\alpha e^{2it} + C(T)e^{it} + cc\} + \beta$ with

$$\alpha = \frac{A^2}{3}(\kappa i - 1) + cc \quad \beta = -AA^* = -2AA^* \in \mathbb{R}$$

Now we can go to $\mathcal{O}(\delta^2)$, where we will finally be able to derive an evolution equation for $A(T)$. The equations are

$$\frac{\partial^2\theta_2}{\partial t^2} + \theta_2 + 2\frac{\partial^2\theta_0}{\partial t\partial T} + 2\frac{\partial\theta_0}{\partial t}\frac{\partial\theta_1}{\partial t} + \mu\frac{\partial\theta_0}{\partial t} + \kappa\theta_1\frac{\partial\theta_0}{\partial t} + \kappa\theta_0\frac{\partial\theta_1}{\partial t} = 0$$

We know analyse each term

$$2\frac{\partial^2\theta_0}{\partial t\partial T} = \{2i\frac{\partial A}{\partial T}e^{it} + cc\} \quad \mu\frac{\partial\theta_0}{\partial t} = \{\mu iAe^{it} + cc\}$$

$$2\frac{\partial\theta_0}{\partial t}\frac{\partial\theta_1}{\partial t} = \{4\alpha A^*e^{it} + cc\} + \text{NRT}$$

$$\kappa\theta_1\frac{\partial\theta_0}{\partial t} = \{(-i\alpha\kappa A^* + i\beta\kappa A)e^{it} + cc\} + \text{NRT}$$

$$\kappa\theta_0\frac{\partial\theta_1}{\partial t} = \{2i\kappa\alpha A^*e^{iat} + cc\} + \text{NRT}$$

Cancelling the resonant terms ($\sim e^{it}$) gives us the desired evolution equation

$$2i\frac{\partial A}{\partial T} + \mu iA + 4\alpha A^* - i\alpha\kappa A^* + i\beta\kappa A + 2i\kappa\alpha A^* = 0$$

inserting the values for α and β gives us the following equation for the envelope

$$\frac{dA}{dT} = -\frac{\mu}{2}A - \left(\frac{i\kappa^2}{6} + \frac{2i}{3} - \frac{\kappa}{2}\right)A^2A^* \tag{7}$$

And hence we have applied concepts from weakly nonlinear analysis to mathematical epidemiology. To obtain equations we can simulate numerically we have to write $A = re^{i\phi}$. When $\phi(0) = 0$ we will see that the correction to the phase will depend on r_0^2.

3 Incorporating spatial dependence: a diffusive SIR model

In this section we will explore how an epidemic spread in space, with a particular emphasis on how travelling epidemic "waves" might appear. To introduce space into the problem, we will start from the same equations as before (1 and 2), but now we will assume the infected individuals can travel in space. The resulting model will account for the epidemic spread in time as well as in space with a diffusive-like term.

Instead of working with the total number of individuals $N, S, I, R, \dots$ we will work with a density $i(x,t) = I(x,t)/N, \dots$ as in previous sections. As for our modelling assumptions, we require that the total population density $n(x)$ is constant in space and time. We will model individuals travelling with a *contact distribution* or kernel $k(x)$ satisfying

$$k(-x) = k(x) \quad \int_{-\infty}^{\infty} k(x)dx = 1 \quad \text{and} \quad k(x) \geq 0$$

The interpretation for this operator is that $k(y-x)$ is the share of infected individuals at position y which come into contact with susceptibles at position x. Mathematically, the contact kernel is just a symmetric probability density function. Hence, the spatial SIRS model will have the following governing equations [10, 1]

$$\frac{\partial s}{\partial t} = -\beta \int k(x-y)i(y,t)dy + \delta r \cdot s(x,t) \quad \frac{\partial i}{\partial t} = \beta \int k(x-y)i(y,t)dy \cdot s(x,t) - \gamma i(x,t) \quad \frac{\partial r}{\partial t} = \gamma i(x,t) + \delta r \quad (8)$$

With the integrals over $\mathbb{R}$. To get the equations in a more tractable form we can perform the following Taylor expansion [22]

$$i(x-y,t) = i(x,t) - y i_x(x,t) + \frac{1}{2} y^2 i_{xx}(x,t) + \dots$$

then [1]

$$\int k(x-y)i(y,t)dy = \int k(y)i(x-y,t)dt \approx \int \left\{ k(y)i(x,t)dy - yk(y)i_x(x,t) + y^2/2k(y)i_{xx}(x,t) \right\} dy$$

In the first integral, we get $i(x,t)$ as the contact kernel is normalized. The second one turns out to be identically zero as the contact kernel is symmetric [6]. Finally, we define

$$D = \beta/2 \int k(y)y^2 dy$$

so that the equations in 8 simplify to [10]

$$\partial_t s = -(\beta i + D i_{xx})s + \delta r \quad i_t = (\beta i + D i_{xx})s - \gamma i \quad r_t = \gamma i - \delta r$$

As in the simpler cases studied previously, we can reduce the above system to 2 dimensions by noting that $s + i + r = 1$ so that the first equation can be discarded and

$$i_t = (\beta i + D i_{xx})(1 - i - r) - \gamma i \quad r_t = \gamma i - \delta r \quad (9)$$

In particular, for a finite domain we can linearize the system and perform separation of variables to find that the effective reproduction number is the same as for the SIR system describe in section 2.1, $R_0 = \frac{N\beta}{\gamma}$, so that when the population density is uniformly distributed on the domain spatial effects and diffusion have no effect on epidemic spread [11, 23]. However this is not consistent with experimental data, thus motivating the modelling of epidemics using complex networks as presented in section 4. However, before moving directly to that type of modelling it is interesting to discuss the existence of travelling wave solutions in systems like 9.

3.1 Travelling Waves in epidemic models

In this section we will discuss the existence of travelling wave solutions for the diffusive SIS system. Although the existence of travelling waves on the more complicated SIRS system is of greater interest, due to time and space constraints we will discuss a reduced SIS system here (with no recovered compartment). A brief sketch of the SIRS travelling wave problem will be given at the end of section. For more details please refer to Lin et. al. [10, 6, 1]. The governing equations for the diffusive SIS model are simpler than those previously derived for the SIRS:

$$\partial_t s = -(\beta i + D i_{xx})s + \gamma i \quad i_t = (\beta i + D i_{xx})s - \gamma i \tag{10}$$

Subject to the constraint that $1 = i + s$, we reduce the system to a single PDE:

$$i_t = (\beta i + D i_{xx})(1 - i) - \gamma i \tag{11}$$

We will assume that there is initial epidemic growth, meaning $\beta > \gamma$. The system can be simplified by dropping the $i\partial_x^2 i$ term, which gives us a slightly simpler system

$$i_t - D i_{xx} = \beta i(1 - i) - \gamma i \tag{12}$$

This is the Fisher-KPP (Kolmogorov, Petrovsky and Piskunov) equation [15], which is well studied. We can non-dimensionalize via

$$u = \frac{\gamma}{\beta} i \quad x' = \sqrt{\beta/D}x \quad t' = \beta t$$

The non-dimensional equation will be

$$\partial_t u - \partial_{xx}^2 u = u(1 - u) \tag{13}$$

However, for the time being we will stick with the dimensional equation, and we will return to this when studying the asymptotic solution in the next section. For the time being we seek a travelling wave solution of the form $i(x, t) = f(\xi)$ with $\xi = x - ct$. Upon substitution we get the following differential equation [5]

$$Df'' + cf' + \beta f(1 - f) - \gamma f = 0$$

It is best to think of this equation as the two dimensional ODE system

$$\begin{cases} f' = g \\ g' = \frac{1}{D}\left[-cf' - \beta f(1 - f) + \gamma f\right] \end{cases} \tag{14}$$

This system has two fixed points, one corresponding to the disease free equilibrium $(0, 0)$ and the other corresponding to the endemic equilibrium $(\frac{\beta-\gamma}{\beta}, 0)$. The linearization around the disease free equilibrium has Jacobian matrix

$$J(0, 0) = \begin{pmatrix} 0 & 1 \\ \frac{\gamma-\beta}{D} & -\frac{c}{D} \end{pmatrix}$$

this has eigenvalues

$$\lambda_{\pm}^0 = \frac{-c \pm \sqrt{c^2 - 4D(\beta - \gamma)}}{2D}$$

we can see that for this to have real roots (and be a stable node) we require $c \geq c_{\min} = 2\sqrt{D(\beta - \gamma)}$. The reason why we impose the real roots requirement is that we have a physical requirement that $f > 0$ and a complex solution would allow for oscillations, which would mean f takes on negative values. As f represents a density of infected people it cannot be negative [10]. Turning our attention to the endemic equilibrium $(\frac{\beta-\gamma}{\beta}, 0)$, it will have Jacobian matrix

$$J\left(\frac{\beta - \gamma}{\beta}, 0\right) = \begin{pmatrix} 0 & 1 \\ \frac{\beta-\gamma}{D} & -\frac{c}{D} \end{pmatrix}$$

with eigenvalues

$$\lambda_{\pm}^{1} = \frac{-c \pm \sqrt{c^2 + 4D(\beta - \gamma)}}{2D}$$

So we verify the endemic equilibrium is a saddle point. Thence, there exists a separatrix joining the endemic equilibrium with the disease free equilibrium when $c \geq c_{\min}$, see Figure 2 [15]. This separatrix will be a trajectory in the phase space which lies entirely in the $f > 0, g < 0$ quadrant. Hence we have determined that there exists a minimum wave speed for epidemic front [5].

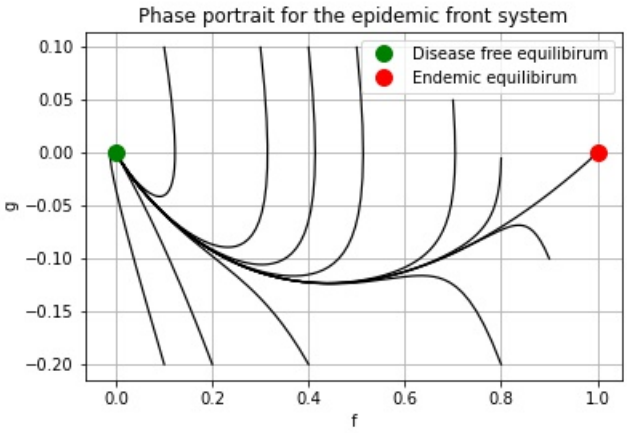


Figure 2: Phase space trajectories for $c \geq c_{\min}$. Note the separatrix joining the two fixed points

For the more complicated SIRS system Li et. al. [10] suggested a similar approach but made use of the Hopf Bifurcation theorem to establish the result. The details of this are beyond the scope of this paper, but a detailed analysis is available in any of [1, 6, 23]

3.2 Asymptotic solution of the epidemic front

We now return to the non-dimensional Fisher-KPP equation [15]

$$\partial_t u - \partial_{xx}^2 u = u(1 - u)$$

Proceeding as before, we can try a traveling wave solution of the form $u(x, t) = f(x - ct)$, which gives us the same ODE as before but in non-dimensional form:

$$f'' + cf' + f(1 - f) = 0$$

The analysis in the previous section indicates that the minimum wave speed will be $c = 2$. Hence, motivated by this we can define a small parameter $\varepsilon = \frac{1}{c^2} \leq 0.25$. We can then try an asymptotic solution using the following change of variables [7]

$$f(\xi) = h(\eta) \quad \eta = \sqrt{\varepsilon}\xi$$

Then the equations get mapped to [7]

$$\varepsilon \frac{d^2 h}{d\eta^2} + \frac{dh}{d\eta} + h(1 - h) = 0$$

We will require that the center of the front is located at $\xi = 0$. This means that $h(0) = \frac{1}{2}$, and we require that h decays to zero at positive infinity. We will seek a series solution of the form

$$h(\eta) = h_0 + \varepsilon h_1 + \ldots$$

The zeroth order equation will be

$$\mathcal{O}(1) \Longrightarrow \frac{dh_0}{d\eta} = -h_0(1-h_0)$$

We can solve this equation with the boundary condition $h_0(0) = 0.5$ with [7]

$$h_0(\eta) = \frac{1}{1+e^{\eta}}$$

Moving up to first order we get

$$\mathcal{O}(\varepsilon) \Longrightarrow \frac{dh_1}{d\eta} + h_1(1-2h_0) = -\frac{d^2h_0}{d\eta^2}$$

Subject to $h_1(0) = 0$. The solution to this problem is [7]

$$h_1(\eta) = \frac{e^{\eta}}{(1+e^{\eta})^2} \log\left[\frac{4e^{\eta}}{(1+e^{\eta})^2}\right]$$

assembling the series solution we obtain $h(\eta)$

$$h(\eta) = \frac{1}{1+e^{\eta}} + \varepsilon \frac{e^{\eta}}{(1+e^{\eta})^2} \log\left[\frac{4e^{\eta}}{(1+e^{\eta})^2}\right]$$

now we can undo the transformation to obtain the shape of the wavefront: $\xi = \varepsilon^{1/2}\eta$ and $\varepsilon = 1/c^2$ giving us

$$f(\xi) = \frac{1}{1+e^{\xi/c}} + \frac{1}{c^2} \frac{e^{\xi/c}}{(1+e^{\xi/c})^2} \log\left[\frac{4e^{\xi/c}}{(1+e^{\xi/c})^2}\right]$$

In Figure 3 we compare this asymptotic solution to the a solution obtained by numerically solving the boundary value problem. The asymptotic solution is extremely accurate, even for small values of $c \sim 2$.

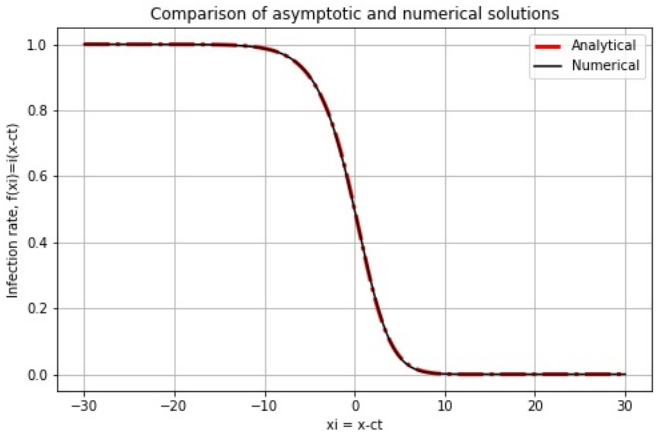


Figure 3: Comparison between the numerical solution and the asymptotic solution for $c = 2$. Numerically, the relative error stays below 1% in the entire domain.

4 Epidemics on networks

In the previous section we explored the role of spatial dependence has in epidemic spread. However, we did this under the strong underlying assumptions that the spread was isotropic, i.e. equal in all directions.

However, diseases are transmitted via human networks, which are complex and not isotropic. For this reason, it is reasonable to introduce networks to the study of epidemics. The ultimate aim for this section is to find expressions of the R_0 basic reproduction number that depend on the structure of the network. This is useful because it can help predict epidemic spread accurately if we have information about the social network where it is spreading. Instead of diving directly into epidemics it is a good idea to spend some time describing networks themselves, and the mathematical tools we use to describe them.

4.1 A brief introduction to network science

A network is essentially a collection of N nodes and L edges between them. In the simplest case, when a network is undirected (meaning nodes are connected symmetrically by an edge, ie. information can travel both ways) and unweighted (meaning every edge is as important as any other edge) we can compress all the information about a graph on the adjacency matrix $A \in \mathbb{R}^{N\times N}$, defined by [2]

$$A_{ij} = \begin{cases} 1 & \text{if there is a link between nodes } i, j \\ 0 & \text{otherwise} \end{cases}$$

Using this, we can define the degree of node i by [12]

$$k_i = \text{number of neighbors of node } i = \sum_j A_{ij} = \sum_i A_{ij}$$

4.2 Epidemics on networks: an example of nonlinear dynamics on a system with many degrees of freedom

Now we have defined all the necessary tools, and we can start to think how an epidemic would spread on a graph were each person is a node and we place an edge between two people if for example they have been in close proximity in the last 7 days (or some other rule). We can simulate an epidemic that starts from a single node by drawing random numbers for each of its neighbors and choosing if they get infected if say the number is greater than β. Then we repeat the process with the newly infectious nodes and iterate with each time step [9]. This is a Monte-Carlo like method that only returns a single realization of how an epidemic spreads on a particular graph. We are far more interested in how an epidemic spreads on average, which means we would need to repeat this procedure many times.

However, we can also obtain the average behaviour theoretically! To this end we define $x_i(t)$, the event that node i is infected at time t. This models a Bernoulli random variable, meaning that the expectation is the same as the probability that the random variable is one, so we can claim $\langle x_i(t)\rangle = \Pr(x_i(t) = 1)$. Hence the objective is to find a governing equation for $\langle x_i(t)\rangle$. If we assume there is no recovery (so an SI system), then a node stays infected forever once it is infected. So a node will be infected at time $t + \Delta t$ if either

1. It was already infected at time t
2. It got infected from one of its neighbours between t and $t + \Delta t$

We can write this mathematically by saying [8]

$$\Pr(x_i(t + \Delta t) = 1) = \Pr(x_i(t) = 1) + \pi_i(t, \Delta t)$$

where $\pi_i(t, \Delta t)$ is the probability that node i is infected by a neighbor between times t and $t + \Delta t$. For this to happen, we require $x_i(t) = 0$, $x_j(t) = 1$ and $A_{ij} = 1$ so that i and j are neighbors. If all those conditions are met, then we can model that probability as in the direct simulation case and assign it a probability of transmission of $\beta\Delta t$. Hence, when we account for all the neighbors of i, we can get an expression for π_i [19]

$$\pi_i(t, \Delta t) = \beta\Delta t \sum_{j=1}^{N} A_{ij} \Pr(x_i(t) = 0, x_j(t) = 1) + \mathcal{O}(\Delta t^2)$$

where $\Pr(x_i(t) = 0, x_j(t) = 1)$ is the joint probability that $x_i(t) = 0$ and $x_j(t) = 1$. Then

$$\Pr(x_i(t + \Delta t) = 1) = \Pr(x_i(t) = 1) + \beta \Delta t \sum_{j=1}^{N} A_{ij} \Pr(x_i(t) = 0, x_j(t) = 1) + \mathcal{O}(\Delta t^2)$$

As discussed above, we can take expectations so that

$$\langle x_i(t + \Delta) \rangle = \langle x_i(t) \rangle + \beta \Delta t \sum_{j=1}^{N} A_{ij} \langle (1 - x_i(t)) x_j(t) \rangle + \mathcal{O}(\Delta t^2)$$

now, dividing by Δt and taking the limit $\Delta t << 1$, we get a system of N non linear ODEs:

$$\frac{d\langle x_i \rangle}{dt} = \beta \sum_{j=1}^{N} A_{ij} \langle (1 - x_i(t)) x_j(t) \rangle \tag{15}$$

However (and tragically) the product of expectations in the summation means this can not be solved in this form, so we will need a simplifying assumption. On another note, using the same steps outlined here we can obtain equations for the network SIR model, where we allow recovery and s_i is the event that node i is susceptible [9, 8]

$$\frac{d\langle s_i \rangle}{dt} = -\beta \sum_{j=1}^{N} A_{ij} \langle s_i(t) x_j(t) \rangle \quad \text{and} \quad \frac{d\langle x_i \rangle}{dt} = \beta \sum_{j=1}^{N} A_{ij} \langle s_i(t) x_j(t) \rangle - \gamma \langle x_i(t) \rangle \tag{16}$$

4.3 Naive approximation

To get an equation in a form we can solve/simulate, as a starting model we can assume

$$\langle x_j(1 - x_i) \rangle = \langle x_j \rangle \langle 1 - x_i \rangle$$

This is known as the *Naive approximation* [8]. Under this assumption, we have in the case of the SI system

$$\frac{d\langle x_i \rangle}{dt} = \beta \langle 1 - x_i(t) \rangle \sum_{j=1}^{N} A_{ij} \langle x_j(t) \rangle$$

and in the case of the SIR system:

$$\frac{d\langle s_i \rangle}{dt} = -\beta \langle s_i(t) \rangle \sum_{j=1}^{N} A_{ij} \langle x_j(t) \rangle \quad \text{and} \quad \frac{d\langle x_i \rangle}{dt} = \beta \langle s_i(t) \rangle \sum_{j=1}^{N} A_{ij} \langle x_j(t) \rangle - \gamma \langle x_i \rangle$$

The above equations *can* be integrated numerically (and the animation that comes with my report is from this equations), and furthermore we can linearize around the disease free equilibrium ($\langle s_i \rangle = 1$ and $\langle x_i \rangle = 0$) obtaining linear system

$$\frac{d\langle s_i \rangle}{dt} = -\beta \sum_{j=1}^{N} A_{ij} \langle x_j(t) \rangle \quad \text{and} \quad \frac{d\langle x_i \rangle}{dt} = \beta \sum_{j=1}^{N} A_{ij} \langle x_j(t) \rangle - \gamma \langle x_i \rangle$$

The second equation can be expressed in vector form using the adjacency matrix

$$\frac{d\langle \mathbf{x} \rangle}{dt} = \beta A \langle \mathbf{x} \rangle - \gamma \langle \mathbf{x} \rangle = (A - \gamma I) \langle \mathbf{x} \rangle$$

where I is the identity matrix. The system will be dominated by the largest eigenvalue of the matrix $A - \gamma I$, which we know will be real because of the spectral theorem (recall A is symmetric), and hence the epidemic will grow as $\sim \exp(\lambda_1 - \gamma)t$, where λ_1 is the largest eigenvalue of A. Thus we have found an effective reproduction number $\lambda_1 - \gamma$ that will depend on the structure of the network.

4.4 Degree based analysis

Another approach to getting analytic is result is to assume that nodes of similar degrees will have the same probability of being infected [20]. Mathematically, this means that

$$k_i = k_j \implies \langle x_i \rangle = \langle x_j \rangle$$

So let ϕ_k indicate if the nodes of degree k are infected. Using a similar analysis as for the average infection, for any time t, we will have that $\Pr(\phi_k(t + \Delta t) = 1)$ if $\Pr(\phi_k(t) = 1)$ or if the nodes of degree k get infected from a neighbor between t and $t + \Delta t$. Denote this transmission event as B. Let $\Pr(\theta(k, k') = 1)$ be the probability that a node of degree k has a neighbour of degree k'. Then we can model $\Pr(B)$ with [20]

$$\Pr(B) = k\beta\Delta t \sum_{k'=1}^{k_{max}} \Pr(\theta(k, k') = 1, \phi_k(t) = 0, \phi_{k'}(t) = 1)$$

This can be simplified using conditional probabilities

$$\Pr(\theta(k, k') = 1, \phi_k(t) = 0, \phi_{k'}(t) = 1) =$$

$$= \Pr(\phi_{k'}(t) = 1 | \theta(k, k') = 1, \phi_k(t) = 0) \Pr(\theta(k, k') = 1 | \phi_k(t) = 0) \Pr(\phi_k(t) = 0)$$

And furthermore by independence [20] (the details of this are not important)

$$\Pr(\theta(k, k') = 1 | \phi_k(t) = 0) = \Pr(\theta(k, k') = 1)$$

$$\Pr(\phi_{k'}(t) = 1 | \theta(k, k') = 1, \phi_k(t) = 0) = \Pr(\phi_{k'-1} = 1)$$

Hence we have factorised the probability in the following way:

$$\Pr(\theta(k, k') = 1, \phi_k(t) = 0, \phi_{k'}(t) = 1) = \Pr(\phi k' - 1 = 1) \Pr(\theta(k, k') = 1) \Pr(\phi_k(t) = 0)$$

Putting everything back together and switching from probabilities to expectations (which we can do since we are working with Bernouilli variables), and taking the limit as $\Delta t \to 0^+$

$$\frac{d\langle \phi_k \rangle}{dt} = k\beta(1 - \langle \phi_k \rangle) \sum_{k'=1}^{k_m} \langle \theta(k, k') \rangle \langle \phi_{k'-1} \rangle$$

To get the equations in a form we can solve we need some information on $\theta(k, k')$. For this, suppose we have the *degree distribution* of the network. This means that for each degree k we know p_k, the share of nodes with degree k. Then $\langle \theta(k, k') \rangle = \frac{k' p_{k'}}{\bar{k}}$ [2] Hence, the governing equation is [12]

$$\frac{d\langle \phi_k \rangle}{dt} = k\beta(1 - \langle \phi_k \rangle) \sum_{k'=1}^{k_m} \frac{k' p_{k'}}{\bar{k}} \langle \phi_{k'-1} \rangle \tag{17}$$

To get an effective reproduction number we linearize around the disease free equilibrium $\langle \phi_k \rangle << 1$,

$$\frac{d\langle \phi_k \rangle}{dt} = k\beta \sum_{k'=1}^{k_m} \frac{k' p_{k'}}{\bar{k}} \langle \phi_{k'-1} \rangle$$

We can simplify this by defining

$$\Theta = \sum_{k'=1}^{k_m} \frac{k' p_{k'}}{\bar{k}} \langle \phi_{k'-1} \rangle \quad \implies \quad \frac{d\langle \phi_k \rangle}{dt} = k\beta\Theta$$

and furthermore, taking the derivative of Θ and using the original equation we get

$$\frac{d\Theta}{dt} = \beta\Theta \sum_{k'=1}^{k_m} \frac{k' p_{k'}}{\bar{k}}(k'-1)$$

But the sum turns out to be [2]

$$\sum_{k'=1}^{k_m} \frac{k' p_{k'}}{\bar{k}}(k'-1) = \frac{1}{\bar{k}} \sum_{k'=1}^{k_m} [k'^2 p_{k'} - k'] = \frac{\bar{k^2}}{\bar{k}} - 1$$

So the epidemic growth depends on the variance of the degree distribution! Furthermore, we can solve those linear equations, to recover

$$\Theta(t) = \Theta_0 \exp(t/\tau) \quad \Longrightarrow \quad \langle \phi_k(t) \rangle = \frac{k\beta\Theta_0}{\tau} \left(\exp(t/\tau) - 1\right) + \langle \phi_k(0) \rangle \tag{18}$$

where the characteristic spread time τ is $\frac{1}{\tau} = \beta \left(\frac{\bar{k^2}}{\bar{k}} - 1 \right)$. The dependence of the initial growth (and the reproduction number) on the variance of the degree distribution is a remarkable result. It is a convenient way to estimate "how bad" an outbreak will be quickly and cheaply, and it sheds light into the role network structure plays in epidemic spread. In particular, this results predicts that networks that have a wide range of degrees will have faster spread.

4.5 Pair approximation

The naive approximation relies on the assumption that we can factorise the expectation in the governing equations. This will be a terrible assumption for most real networks [2, 12]. A possible idea is to derive another evolution equation for $\langle (1-x_i)x_j \rangle$ (or equivalently $\langle x_i x_j \rangle$) in much the same way we did for $\langle x_i \rangle$. We only need to do this when nodes i and j are neighbours, so this incorporates L unknowns into our system, where L is the number of edges. This approach is known as the *pair approximation*[17]. So we need to compute $\Pr(x_i(t+\Delta t) = 1, x_j(t+\Delta t) = 1)$. Focusing again on the SI model, we can use the same reasoning as for the first master equation, by noting that

$$\Pr(x_i(t+\Delta t) = 1, x_j(t+\Delta t) = 1) = \Pr(x_i(t) = 1, x_j(t) = 1) + \Pr(x_i(t) = 1, x_j \to 1) +$$
$$+ \Pr(x_i \to 1, x_j(t) = 1) + \Pr(x_i \to 1, x_j \to 1)$$

Where the notation $x_i \to 1$ means that node i gets infected between time t and $t + \Delta t$. The first term in the sum will get absorbed into the derivative in the limit for Δt small, and the final term will be of order $\mathcal{O}((\Delta t)^2)$, so it will also disappear in the limit. Hence we have to focus in the middle terms. Using the same reasoning as previously, we can write them as [14]

$$\Pr(x_i(t) = 1, x_j \to 1) = \beta \Delta t \sum_{l=1}^{N} A_{jl} \Pr(x_i(t) = 1, x_j(t) = 0, x_l(t) = 1)$$

and similarly for the other term

$$\Pr(x_i \to 1, x_j(t) = 1) = \beta \Delta t \sum_{l=1}^{N} A_{il} \Pr(x_i(t) = 0, x_j(t) = 1, x_l(t) = 1)$$

so putting everything back together we obtain (recall $s_i = 1 - x_i$)

$$\frac{d\langle x_i x_j \rangle}{dt} = \beta \sum_{l=1}^{N} [A_{jl} \langle x_i s_j x_l \rangle + A_{il} \langle s_i x_j x_l \rangle] \Longrightarrow$$

$$\frac{d\langle s_i x_j\rangle}{dt} = \frac{d\langle (1-x_i)x_j\rangle}{dt} = \beta\sum_{l=1}^{N} A_{jl}\langle s_j x_l\rangle - \beta\sum_{l=1}^{N}[A_{jl}\langle x_i s_j x_l\rangle + A_{il}\langle s_i x_j x_l\rangle] = \beta\sum_{l=1}^{N}[A_{jl}\langle s_i s_j x_l\rangle + A_{il}\langle s_i x_j x_l\rangle]$$

Our next step to obtaining tractable equations is to approximate $\langle s_i s_j x_l\rangle$ and the other similar term: [17]

$$\langle s_i s_j x_l\rangle \approx \frac{\langle x_l s_j\rangle\langle s_i s_j\rangle}{\langle s_j\rangle} \quad \text{and} \quad \langle s_i x_j x_l\rangle \approx \frac{\langle s_i x_l\rangle\langle s_i x_j\rangle}{\langle x_j\rangle}$$

Thus have derived a closed system of $N + L$ ODEs which we can simulate:

$$\frac{d\langle x_i\rangle}{dt} = \beta\sum_{j=1}^{N}\langle s_i x_j\rangle \quad \text{and} \quad \frac{d\langle s_i x_j\rangle}{dt} = \beta\sum_{l=1}^{N}\left[A_{jl}\frac{\langle x_l s_j\rangle\langle s_i s_j\rangle}{\langle s_j\rangle} + A_{il}\frac{\langle s_i x_l\rangle\langle s_i x_j\rangle}{\langle x_j\rangle}\right]$$

This is known as the pair approximation, and it is in fact a state of the art model for the epidemic spread on networks. [17, 18]

4.6 Comparison of approximation and numerical results

Three different approximations to the governing equation have been derived. The first and third are of similar nature, where we approximate the nonlinear terms. The naive approximation will work well when the nodes of the graph are uncorrelated, as that is when the expectation can be factored. This is true for graphs that look like trees. We say these networks low high *clustering or transitivity*, and clustering is just a measure of the number of triangles on a graph. However most real social networks have high clustering [2, 21], rendering the naive approximation ineffective. Indeed, Newman [12] compared both the naive and the pair approximation on networks with different transitivity, as shown in Figure 4, clearly showing how superior the pair approximation (denoted as second order) is to the naive approximation (first order) when used on graphs with high clustering.

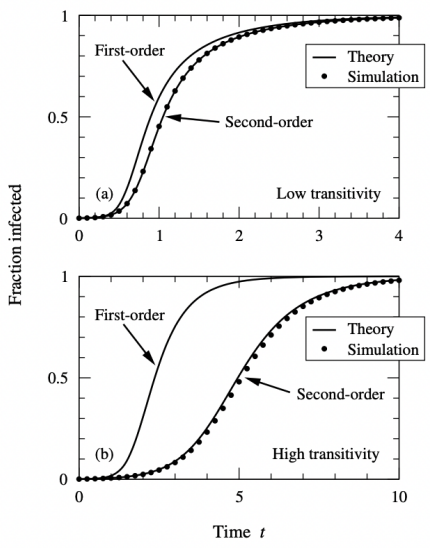


Figure 4: From Newman (2018) [12]. Comparison of the pair approximation (second order) and the naive approximation (first order) with simulations using Monte-Carlo. We observe the naive approximation dramatically fails for networks with high clustering. Here the abundance of triangles means nodes are not independent from their neighbor's neighbors

Turning our attention to the degree-based approximation, it has clear shortcomings when applied to graphs where there is a significant fraction of nodes with high degree, and they might be dispersed in opposite corners of the domain. This means that even though one node of high degree might be infected, the remaining ones are physically far away and thus have very low probability of being infected, and the degree assumption fails completely. However this approximation, provides us with a useful metric to measure initial epidemic spread, the variance of the degree distribution (which we often know). Thus it can be useful as a first estimate but public health decisions should not be made just from the degree based approximation.

5 Conclusions, future work and final remarks

All in all we explored the mathematics of epidemiology gradually increasing the role space has on the disease spread. Along the way, we successfully implemented techniques from weakly nonlinear analysis to the SIRS epidemic model, obtaining the envelope equations for the oscillations around the endemic state. Furthermore, in the case of uniform spatial dependence the conditions for the existence of travelling wave solutions for the SI system were derived, alongside a successful asymptotic expression for the wavefront.

Network Science was introduced with the aim of modelling the spread of disease on complex and nonhomogeneous media, such as human social networks. The governing equations for the average infection were derived, but they are intractable, so several alternative approximations were offered to obtain tractable equations, culminating in a state of the art model, the pair approximation [17]. Through these approximations we were able to obtain effective reproductive numbers that dependend on the structure of the network.

In the future it would be interesting to study if contact terms of the form SI^2 or S^2I are a good fit with experimetnal data, instead of the SI term standard models use. This would be beneficial for the weakly nonlinear analysis, as the nonlinearity would be cubic instead of quadratic, and $\mathcal{O}(\delta)$ would be sufficient for the analysis, potentially simplifying the algebra required for the problem. On the topic of the weakly nonlinear analysis, it would be interesting to perform the analysis with the $\sim z^2$ term as well to better capture the physics of the problem.

Moreover, further study into the existence and properties of travelling waves in the more complicated SIRS system would also be appropiate, continuing the work started by Li et. al. [10] and others [1, 6]

As for the network models, gaining a more theoretical understanding of the systems of equations that arise would be beneficial, as due to the large numbers of degrees of freedom they are often simulated numerically. In particular, large real networks often exhibit community structure, and comprehending how this affects epidemic growth is crucial if we are to use these models to establish public policy, exploiting the role communities plays in the spread when implementing lockdowns or even vaccination roll outs. Another possible avenue of study is incorporating stochastic methods, such as random walks to predict the spread, as in [4, 3]

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