An effective degree model for epidemics on dynamic networks

Michael Taylor,[1,*] Timothy J. Taylor,[1,2] and Istvan Z. Kiss[1]

[1] School of Mathematical and Physical Sciences, Department of Mathematics, University of Sussex, Brighton BN1 9QH, UK
[2] Centre for Computational Neuroscience and Robotics, University of Sussex, Brighton BN1 9QH, UK

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In this paper we present a new ODE based framework for modelling disease transmission on dynamic contact networks. We adapt and extend the effective degree model for a static network to account for the random creation and deletion of links between individuals. The resulting set of ODEs is solved numerically and results are compared to those obtained using individual-based stochastic network simulations. We show that the ODEs display excellent agreement for the evolution of both the disease and the network, and is able to accurately capture the epidemic threshold for a wide range of parameters. Using the proposed model we show that mild epidemics can be controlled while keeping the contact network well connected, and this is in contrast with severe epidemics, where successful control via link removal leads to a disconnected network.

The rise in the popularity and relevance of networks as a tool for modelling complex systems is well illustrated by the ever increasing body of research concerned with the spread of diseases within host populations exhibiting non-trivial contact structures [1, 2]. Networks offer an intuitive and relatively simple modelling framework which enables us to relax the strong implicit assumptions of more classical ODE-based approaches and to account for complexities in the contact structure of the host population [3–7]. This approach has shown that epidemic thresholds not only depend upon the infectiousness of the pathogen, or even simply the mean number of contacts per individual, but also upon the exact structure of the host population [8, 9]. In addition to its inherent theoretical value, this paradigm has immediate practical benefits, as the primary role of public health services is to put measures in place to bring diseases below their epidemic threshold. These measures depend heavily upon disrupting the transmission of a disease through vaccination and also more directly, through the closure of public services, or even quarantine and curfews in extreme cases. Hence the knowledge of how the structure of the host population is contributing to the spread of a disease would help to increase the efficacy of any intervention [10].

Despite advances in both rigorous and non-rigorous analysis of networks, a key assumption in many network models is that contacts are fixed for the duration of an epidemic and that the disease propagates with a constant intensity across links. This will not be true for many diseases, especially those with long infectious periods, or diseases that become endemic. Indeed human contact patterns are well described by short repeated events, with individuals having a number of contacts best described by some appropriate time dependent random variable [11]. Furthermore, individuals and the communities they belong to are likely to change their contact behaviour as a result of natural evolution and endogenous or exogenous perturbations such as a disease outbreak [12].

Recently a number of studies have attempted to relax this assumption by allowing the networks to evolve over time [13, 14]. Thus the dynamics of the disease is coupled with the dynamics of the network itself, with both potentially acting as a feedback mechanism for the other [15, 16]. A number of micro modelling studies have used individual based simulations to investigate how diseases propagate on a network that evolves over time, often in response to the disease itself [17, 18]. Other papers have built macro ODE-based models that describe the coevolution of networks and the diseases that spread along them [19, 20]. All these studies confirm that dynamic networks and the coupling between the two dynamics lead to a richer spectrum of behavior than is found for epidemics on static networks, such as bi-stability and oscillations.

A crucial feature of allowing the co-evolution of disease and network is the interplay and feedback between both dynamics, however this interdependence is difficult to measure empirically. In this paper we propose a dynamic network model that is based on link activation-deletion, be it random or adapted to be link-type dependent [21]. This dynamic network coupled with the simple SIS disease dynamics, leads to the full model. We study this system and explore to what extent a macro ODE-based model proposed for static networks is flexible enough to be adapted to the dynamic network case. Specifically, we focus on the SIS effective degree model for a static network as described in detail by Lindquist et al. [22]. The static model is governed by a closed set of ODEs, and here we modify it to allow for the random creation and deletion of links over time. The modified dynamic effective degree model is also governed by a closed set of ODEs, which is then solved and compared to results from individual based simulations and its ability to accurately predict the epidemic threshold over a range of parameters is investigated.

The effective degree modelling approach for SIS type disease dynamics [22] not only categorizes the disease state of each individual as susceptible (S) or infected (I) but also describes the state of their immediate neighbour-

*Corresponding author: mt264@sussex.ac.uk

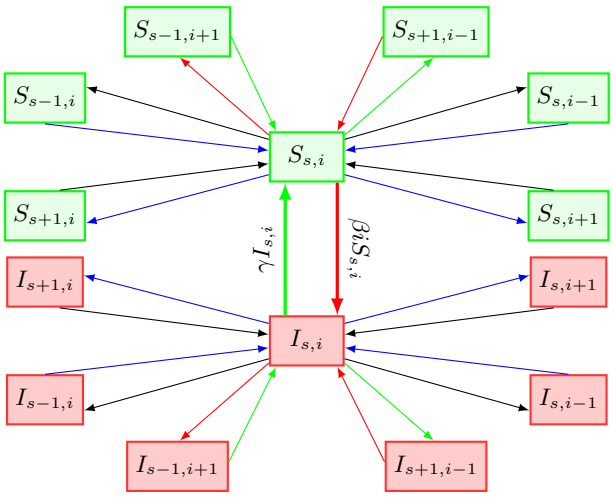

FIG. 1: Flow chart showing transitions in the dynamic SIS effective degree model. The directed red, green, blue and black lines represent changes in state of an individual via infection, recovery, link creation and link deletion respectively. The thick lines represent changes to the individual, and thin lines represent changes to that individual's immediate neighbourhood. In relation to nodes of type $X_{si}, X \in \{S, I\}$, infection of neighbours occurs at rate sG_X, recovery of neighbours at rate γi, creation of a susceptible (infectious) link at rate $\alpha(M-(s+i))P_{S(I)}$ and deletion of a susceptible (infectious) link at rate $\omega s(i)$, where:
$G_S = \beta\frac{\sum_{k=1}^{M}\sum_{j+l=k} jlS_{jl}}{\sum_{k=1}^{M}\sum_{j+l=k} jS_{jl}}$, $G_I = \beta\frac{\sum_{k=1}^{M}\sum_{j+l=k} l^2S_{jl}}{\sum_{k=1}^{M}\sum_{j+l=k} jI_{jl}}$
and $P_X = \frac{\sum_{k=0}^{M}\sum_{j+l=k}(M-(j+l))X_{jl}}{\sum_{k=0}^{M}\sum_{j+l=k}(M-(j+l))(S_{jl}+I_{jl})}$.

hood. This is achieved by keeping track of the number of susceptible and infected neighbours that belongs to a given node. For example, S_{si} represents the number of susceptible individuals that have s susceptible and i infected neighbours. This gives rise to more states and equations than would be seen in a standard pairwise model, where equations are given at the population level for all types of singles and pairs [23]. For example if a S_{si} type node became infected via one of its i infectious neighbours, this individual would move to state I_{si} as only the status of the node itself is changing. However, if one of the i infected neighbours of an S_{si} type node recovered then the node would enter the $S_{s+1,i-1}$ class, whereas infection of one of the s neighbouring susceptible nodes moves the S_{si} type node into the $S_{s-1,i+1}$ class.

Lindquist et al. [22] defined γ to be the per node recovery rate, β the per link infection rate and M the maximum nodal degree of a network with N nodes. They then derived the following system of $\sum_{k=1}^{M} 2(k+1) = M(M+3)$ equations:

$$\begin{aligned}\dot{S_{si}} &= -\beta i S_{si} + \gamma I_{si} + \gamma[(i+1)S_{s-1,i+1} - iS_{si}] \\ &\quad + \beta\frac{\sum_{k=1}^{M}\sum_{j+l=k} jlS_{jl}}{\sum_{k=1}^{M}\sum_{j+l=k} jS_{jl}}[(s+1)S_{s+1,i+1} - sS_{si}],\end{aligned} \tag{1}$$

$$\begin{aligned}\dot{I_{si}} &= \beta i S_{si} - \gamma I_{si} + \gamma[(i+1)I_{s-1,i+1} - iI_{si}]] \\ &\quad + \beta\frac{\sum_{k=1}^{M}\sum_{j+l=k} l^2S_{jl}}{\sum_{k=1}^{M}\sum_{j+l=k} jI_{jl}}[(s+1)I_{s+1,i+1} - sI_{si}],\end{aligned} \tag{2}$$

for $\{(s,i) : s, i \geq 0, 1 \leq k = s+i \leq M\}$. This is the SIS effective degree model for a *static* contact network.

In oder to adapt this model to describe SIS dynamics on a *dynamic* contact network, we introduce two new parameters: ω, the per link deletion rate and α, the per non-link, or more precisely the per *potential* link creation rate. These rates could also be made to be link-type dependent, i.e. ω_{SI} would be the per SI link deletion rate. For the dynamic network cae, the system size will increase slightly from $M(M+3)$ to $\sum_{k=0}^{M} 2(k+1) = (M+1)(M+2)$ equations to account for nodes of the type $X_{0,0}$ where $X \in \{S, I\}$. In the static case, these nodes were dynamically unimportant as they could neither infect nor become infected by other nodes. However in the dynamic model, they could connect to other nodes in the system and so enter states $X_{1,0}$ or $X_{0,1}$ depending on the state of the node with which they have just formed a new link.

The total number of links in the system at time t, $\Lambda(t)$, and potential links, $\Phi(t)$ can easily be calculated from the effective degree formulation as

$$\begin{aligned}\Lambda(t) &= \sum_{k=0}^{M}\sum_{j+l=k}(j+l)(S_{jl}+I_{jl}), \\ \Phi(t) &= \sum_{k=0}^{M}\sum_{j+l=k}(M-(j+l))(S_{jl}+I_{jl})],\end{aligned}$$

with the mean nodal degree given by $\langle k(t)\rangle = \frac{\Lambda(t)}{N}$. At the equilibrium, $\alpha\Phi = \omega\Lambda$ which gives us the mean nodal degree:

$$\langle k\rangle^* = \frac{\alpha}{\alpha+\omega}M. \tag{3}$$

Note that Eq. (3) does not depend on the system size, N, but rather on the maximum nodal degree, M. This is important because in the static model, M is simply given by the node or nodes with the highest degree whilst in the dynamic case, however, M can be considered as a carrying capacity, whereby no node can have more than M links. This subtle but important difference means that in the dynamic case, M itself can be regarded as a parameter which controls the potential level of network saturation.

When adding the terms that govern link creation and deletion to Eqs. (1) and (2) it is far simpler to construct the terms that govern deletion of existing links than those for the creation of new links. Links to nodes of type X_{si} where $X \in \{S, I\}$ are cut at a rate proportional to their degree, so individuals will leave X_{si} through link deletion at a rate $\omega(s+i)$ and will either enter the $X_{s-1,i}$ or $X_{s,i-1}$ classes depending on the state of the nodes to

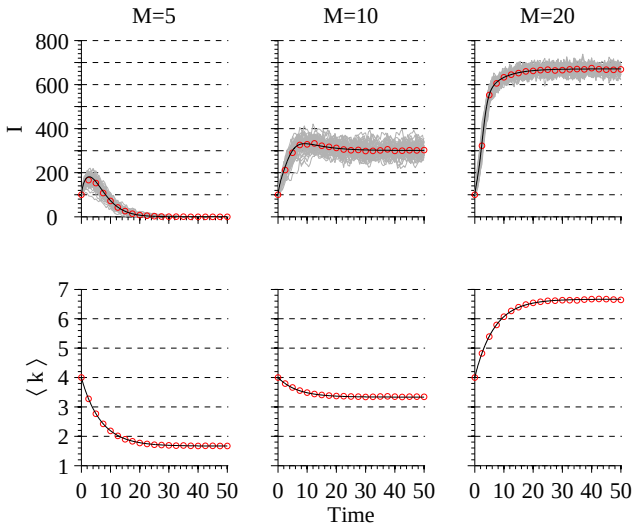

FIG. 2: Time evolution of $I(t) = \sum_{k=0}^{M}\sum_{j+l=k} I_{jl}(t)$ and $\langle k\rangle(t) = \frac{\Lambda(t)}{N}$ for three different values of M. Results from the ODE are given by solid lines and those from simulation by points. In all cases $N = 1000$, $I_0 = 100$, $\alpha = 0.05$, $\omega = 0.1$, $\beta = 0.5$ and $\gamma = 1$. The initial network is a regular random graph with $k = 4$. In each case, mean values from the stochastic simulations were found by averaging over 100 repetitions, with the individual realisations plotted in grey.

which they were previously connected. Similarly individuals can enter state X_{si} if they were in states $X_{s,i+1}$ or $X_{s+1,i}$ and a link to an infected or susceptible node was deleted respectively.

When creating new links to nodes of type X_{si}, there are $M-(s+i)$ stubs remaining, so nodes will transition out of this state at a rate $\alpha(M-(s+i))$ and will either enter the $X_{s+1,i}$ or $X_{s,i+1}$ classes depending on the state of the node to which they have just connected. The rate at which nodes enter the X_{si} class from either $X_{s-1,i}$ or $X_{s,i-1}$ depends not only on the number of stubs still available in the node in question, but also on the probability that the newly created link attaches to a node of state S or I respectively. So nodes enter X_{si} from $X_{s-1,i}$ at the rate $\alpha P_S(M-(s-1+i))$, and nodes enter X_{si} from $X_{s,i-1}$ at rate $\alpha P_I(M-(s+i-1))$, where $P_X = \frac{\sum_{k=0}^{M}\sum_{j+l=k}(M-(j+l))X_{jl}}{\sum_{k=0}^{M}\sum_{j+l=k}(M-(j+l))(S_{jl}+I_{jl})}, X \in \{S, I\}$ is the probability of picking an available stub belonging to nodes of type X where $X \in \{S, I\}$. The full set of transitions captured by this model is shown in Fig. 1.

The addition of these terms to Eqs. (1) and (2) transforms the SIS effective degree model for a static network into one that captures the spread of SIS type diseases on a dynamic contact network and is described by the following system of $(M+1)(M+2)$ equations:

$$\begin{aligned}
\dot{S_{si}} &= -\beta i S_{si} + \gamma I_{si} + \gamma[(i+1)S_{s-1,i+1} - iS_{si}] \\
&\quad +\beta\frac{\sum_{k=0}^{M}\sum_{j+l=k} jlS_{jl}}{\sum_{k=0}^{M}\sum_{j+l=k} jS_{jl}}[(s+1)S_{s+1,i+1} - sS_{si}] \\
&\quad -\omega[(s+i)S_{si} - (i+1)S_{s,i+1} - (s+1)S_{s+1,i}] \\
&\quad -\alpha(M-(s+i))S_{si} + \alpha(M-(s-1+i))P_S S_{s-1,i}, \\
&\quad +\alpha(M-(s+i-1))P_I S_{s,i-1} \\
\dot{I_{si}} &= \beta i S_{si} - \gamma I_{si} + \gamma[(i+1)I_{s-1,i+1} - iI_{si}] \\
&\quad +\beta\frac{\sum_{k=1}^{M}\sum_{j+l=k} l^2 S_{jl}}{\sum_{k=1}^{M}\sum_{j+l=k} jI_{jl}}[(s+1)I_{s+1,i+1} - sI_{si}] \\
&\quad -\omega[(s+i)I_{si} - (i+1)I_{s,i+1} - (s+1)I_{s+1,i}] \\
&\quad -\alpha(M-(s+i))I_{si} + \alpha(M-(s-1+i))P_S I_{s-1,i} \\
&\quad +\alpha(M-(s+i-1))P_I I_{s,i-1},
\end{aligned} \tag{5}$$

for $\{(s,i) : s,i \geq 0, 0 \leq k = s+i \leq M\}$. This system is the *dynamic SIS* effective degree model.

As shown in Fig. 2, the ODEs given by Eqs. (4) and (5) closely capture the time evolution of an epidemic as predicted by stochastic simulations. The only parameter that is varied in Fig. 2 is M, and it is interesting to note the effect it has on the evolution of the disease. As per Eq. (3), the mean nodal degree at equilibrium is dependent on M, and hence, given the same initial network configuration and values of α and ω, the network either loses or gains links as the system evolves. Thus varying the carrying capacity alone leads to different outcomes depending on whether the network can reach a level of connectedness that allows an epidemic to spread and become established. Allowing M to become an active model parameter that is able to control the outcome of an epidemic has potentially interesting real world implications. The number of contacts per person is a natural, countable property unlike the other model parameters, such as ω, which are more difficult to infer. Therefore local constraints that limit the maximum number of contacts per person could be potentially used as a metric when promoting safe behaviour at a population level in the event of an outbreak or other public health crisis.

In Fig. 3, for a given value of α, M and β, the epidemic threshold has been calculated from the ODEs in terms of ω and compared to that predicted by simulations. The agreement is excellent and this is strong evidence that the dynamic effective degree model accurately captures the evolution of an epidemic on a network with random link creation and deletion. When considering the (β, ω) parameter space used for the threshold plot in Fig. 3, there are three distinct regions that are worth noting. Firstly, given an initial starting network, it is possible to calculate the threshold value of β in the static network case. For the regular random graph with $k = 4$ used here, that value is $\beta^* \approx 0.36$. For values of $\beta < 0.36$, the relative time scales of disease and network evolution are crucial in determining whether or not an epidemic will occur. In this situation, the network needs to quickly evolve to become more densely connected in order for there to be an outbreak, and as a result the system is very sensitive to stochastic effects and initial conditions. This would be

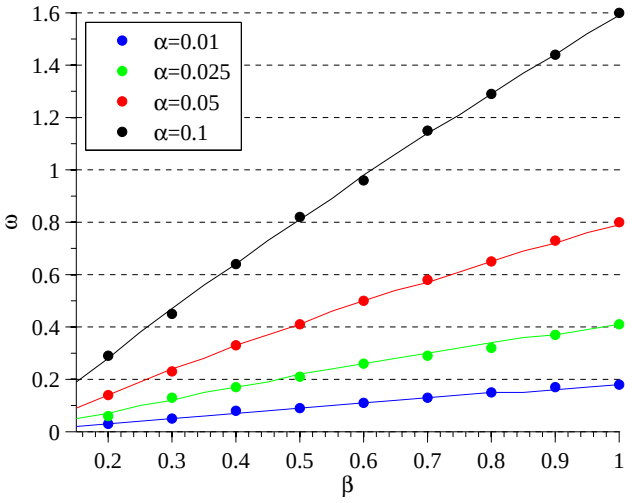

FIG. 3: Epidemic threshold plot in the (β, ω) parameter space for four distinct values of α. Results from the ODE are given by solid lines and those from simulation by solid points. In each case, $N = 1000$, $I_0 = 10$, $M = 20$ and $\gamma = 1$. The initial network is a regular random graph with $k = 4$.

exacerbated on an initial network with a heterogeneous degree distribution, where the placement of the initial infected individuals becomes crucial in determining the likelihood of an epidemic. The second area of interest is when the disease is highly infectious and as a result requires a high value of ω to drive the epidemic below threshold. For a given α and M, Eq. (3) allow us to calculate the value of ω needed to drive the equilibrium average degree below two. If a network has $\langle k \rangle^* < 2$ then it becomes fragmented, with many nodes becoming unconnected. In these situations, the value ω needed to prevent an epidemic virtually destroys the network. In terms of real world implications, a large value of ω corresponds to a situation of strict quarantine and curfew. In between these two cases lies a region within which an epidemic would take hold naturally, given the initial network, but which can be prevented by a value of ω that leaves the network well connected. In the proposed model, the creation and deletion of links happens at random and as such the network evolves independently of the disease, although the evolution of the disease is dependent upon the network. Indeed this leads to observing dynamic networks with degree distributions close to Poisson with the mean given by Eq. (3), and this is confirmed by results from both the ODEs and simulations.

In summary, this paper has proposed an effective degree model for epidemics on dynamic networks. We have shown that this provides a reliable modelling framework that can be used for the analytical and semi-analytical study of coupled disease and network dynamics. In future work, this modelling framework could be adapted and extended to account for individuals cutting and creating links with knowledge of the state of others in the population, i.e., link-type dependent network dynamics. This two-way feedback will lead to more sophisticated network properties such as degree correlations, high clustering or even network fragmentation. In such cases ODE models need to be used with care, making sure that the agreement with simulations remains valid. Besides modelling epidemics, this framework could also be used to study the spread of information, beliefs and new ideas within populations, and as such could have implications across a wide range of disciplines.

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