Consistent Approximation of Epidemic Dynamics on Degree-heterogeneous Clustered Networks

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Abstract. Realistic human contact networks capable of spreading infectious disease, for example studied in social contact surveys, exhibit both significant degree heterogeneity and clustering, both of which greatly affect epidemic dynamics. To understand the joint effects of these two network properties on epidemic dynamics, the effective degree model of Lindquist et al. \cite{30} is reformulated with a new moment closure to apply to highly clustered networks. A simulation study comparing alternative ODE models and stochastic simulations is performed for SIR (Susceptible–Infected–Removed) epidemic dynamics, including a test for the conjectured error behaviour in \cite{42}, providing evidence that this novel model can be a more accurate approximation to epidemic dynamics on complex networks than existing approaches.

Keywords: Networks, Epidemiology, Moment Closure, SIR, Clustering

1 Introduction

Networks offer an unprecedented opportunity to represent and model contacts between interacting units at all scales ranging from proteins and individuals to countries via air transportation. This additional degree of freedom has lead to extensive modelling and analysis in mathematical epidemiology and has allowed the development of a number of network-based models, which are either based or parametrised by real network data, if available, or by using synthetic or theoretical networks which reflect and reproduce some observed local or global properties of real-world networks \cite{24, 6, 41, 29}. Idealised networks often offer a greater degree of analytical tractability, which in turn offers clearer insight into the impact of network properties on epidemic outbreak threshold, final epidemic size or prevalence and on effectiveness or choice of control measures.

The many degrees of freedom offered by networks come at the cost of computational and mathematical complexity. In order to handle this, and for a systematic investigation and understanding of network processes, it is desirable not to rely solely on stochastic simulations. As a result a number of differential equation-based models have been developed including pairwise \cite{44, 22, 18, 50}, PGF or edge-based compartmental models \cite{35, 32} and effective degree models \cite{30, 13}, to name just a few. The goal of all these models is the same: to derive a set of low-dimensional system of ordinary differential equations (ODEs) where variables correspond to some average quantity from the stochastic process—e.g. expected prevalence—over time. All such mean-field models require choice of a ‘state space’. For example, pairwise models initially concentrate on the expected number of nodes in different states, while effective degree models concentrate on the expected number of star like structures, i.e. a central node with all its neighbours, and the possible state that these can be in. Once chosen, evolution equations for these variables are derived. This, often heuristic, step still involves a precise book-keeping which in general yields a dependency on higher order states or moments, e.g. for pairwise models the expected number of infected nodes depends on the

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expected number of edges where one node is infected and the other is susceptible. These newly introduced higher-order structures or moments require further equations and hence to curtail the dependency on higher-order moments and the fast growth in the number of equations ‘closures’ are needed. This amounts to approximating higher-order moments in terms of lower order ones. The performance of mean-field models is then often tested by direct comparison to results based on explicit stochastic network simulations. If the process is successful and the mean-field model works well, the analysis of the stochastic epidemic on networks is mapped into analysing a system of ODEs. This can be done using dynamical systems tools and such analysis often leads to analytical results which explicitly reveal the interaction between network and disease characteristics. More importantly, it shows how the fundamental properties of the network impact on growth rate, epidemic threshold, final epidemic size and so on.

Degree or contact heterogeneity and degree-based mixing is well accounted for in existing differential equation models, but epidemics on networks which are clustered (i.e. where two nodes with a common neighbour are highly likely to be neighbours of each other [53]) pose more of a challenge. A major factor of this difficulty is the non-unique way in which global or network-level clustering can be achieved, and the same level of clustering can be achieved while keeping the degree distribution the same but using different distributions or different sets of motifs [40, 28, 14, 45].

In general, clustered bond percolation-type [12, 33, 53, 21] or PGF-based models [51, 46, 18] consider specific forms of clustering (e.g. non-overlapping triangles) while pairwise models [22, 16, 23, 18] usually work best in the case where clustering is ‘randomly’ distributed, as is the case when using rewiring algorithms such as the big-V [19, 3, 14], which precludes certain kinds of interaction between clustering and degree heterogeneity. The question of how to approximate epidemic dynamics on a more general complex network remains open, and is the topic of this paper.

In general, for clustered networks it is difficult to derive accurate differential equation models for epidemics. In this paper, we present an approach based on generalisation of the effective degree model to clustered networks, and we show that this newly-derived model outperforms the state-of-the-art models and display excellent agreement with results based on stochastic simulations for a range of degree distributions and clustering values.

The paper is structured as follows: In Sect. 2 an overview of relevant complex network concepts and terminology are presented; Sect. 3 introduces methods of generating clustered networks with a specific degree distribution; in Sect. 4 existing ODE models are introduced and discussed; Sect. 5 presents the extension of ODE models to dynamics on clustered networks including our novel ODE model; Sect. 6 details the simulation study performed to compare between models and test the closure performance against conjectured errors.

2 Model Definitions

2.1 Networks

A network (or graph) is a pair \( G = (\mathcal{N}, \mathcal{L}) \) where \( \mathcal{N} \) is a size-\( N \) set of nodes and \( \mathcal{L} \subseteq \mathcal{N} \times \mathcal{N} \) is a set of links. The information about a network can be usefully encoded in terms of an adjacency matrix, which has elements \( G_{ab} = 1 \) if \( (a, b) \in \mathcal{L} \), and zero otherwise. We will consider simple undirected networks without self edges meaning that \( G_{aa} = 0 \) and \( G_{ab} = G_{ba}, \forall a, b \in \mathcal{N} \). The degree of node \( a \) is the number of links that it participates in, i.e.

\[
k_a := \sum_{b \in \mathcal{N}} G_{ab} .
\]

We assume that all degrees are integers between 1 and the maximum degree \( M \). We will use angled brackets to refer to mean values of functions of degree across the network, i.e. for an arbitrary function \( f \),

\[
\langle f(k) \rangle := \frac{1}{N} \sum_{a \in \mathcal{N}} f(k_a) .
\]

One particularly important such expectation is the probability generating function (PGF) which is \( \psi(x) := \langle x^k \rangle \).
A final network property of interest in this work is the clustering coefficient,

$$\phi := \frac{\sum_{a,b,c \in N} G_{ab}G_{bc}G_{ca}}{\sum_{a,b,c \neq a \in N} G_{ab}G_{bc}} \in [0, 1].$$

Our interest is in networks with large size ($N \gg 1$), degree distributions with a non-infinite variance ($\langle k \rangle^2 \leq \langle k^2 \rangle < \infty$), and clustering coefficients that can be non-zero but are not particularly large ($\phi \in [0, 0.3]$).

### 2.2 Epidemic Dynamics

We will consider SIR (Susceptible–Infective–Removed) dynamics. At the individual level, an individual $a$ has a random state $X_a \in \{S, I, R\}$. Individuals in state $I$ move to state $R$ at rate $\gamma$, and individuals in state $S$ move to state $I$ at a rate $\tau$ multiplied by the number of $I$ individuals they are linked to on the network.

At the population level, we will consider expected total node, pair and triple counts in given disease state configurations,

$$[A] = \mathbb{E} \sum_{a \in N} 1\{X_a = A\},$$

$$[AB] = \mathbb{E} \sum_{a,b \in N} 1\{X_a = A \& X_b = B\} G_{ab},$$

$$[ABC] = \mathbb{E} \sum_{a,b,c \neq a \in N} 1\{X_a = A \& X_b = B \& X_c = C\} G_{ab}G_{bc},$$

where $A, B, C \in \{S, I, R\}$ and $1$ is the indicator function. We will distinguish notationally between closed and open triples,

$$[ABC]_\triangle = \mathbb{E} \sum_{a,b,c \in N} 1\{X_a = A \& X_b = B \& X_c = C\} G_{ab}G_{bc}G_{ca},$$

$$[ABC]_\land = [ABC] - [ABC]_\triangle,$$

which will be particularly important later. We will also consider more detailed states—in particular $[A_s,i]$ represents the expected number of nodes in state $A$ with $s$ susceptible and $i$ infective contacts.

Another important definition is the correlation between states

$$C_{AB} = \frac{N}{\langle k \rangle} \frac{[AB]}{[A][B]},$$

which expresses how much more likely an $[AB]$ edge is over the null model. The understanding of this is key to the role of networks in shaping epidemic dynamics [23].

Our aim in this paper is to find methods for approximation of the expected population-level behaviour of the epidemic dynamics that are, as much as possible, logically consistent, well motivated, and accurate.

### 3 Models of Network Generation

Typically, a full epidemic network is not directly available from data and so a standard method is to work with probabilistic models for the network that respect certain observable summary statistics—in our case, the degree distribution and clustering coefficient. We will now present several such algorithms in outline form—more detail on these can be found in relevant papers and textbooks, e.g. Newman [38].

In $n$-regular networks, all nodes have the same degree i.e. $k_a = n, \forall a \in N$. A typical such network can be generated by starting with a network that is atypical but assuredly $n$-regular, for example a one-dimensional ($n/2$)-nearest neighbour. The network is then rewired by removing two edges $(a, b)$ and $(c, d)$ sampled (without replacement) uniformly at random from $\mathcal{L}$, and then
adding new edges \((a, c)\) and \((b, d)\) (Fig. 1 \(\text{③} \rightarrow \text{④}\)). Performing a large number of such ‘edge swaps’ will result in a \(n\)-regular network that is representative of this class of graphs.

Fig. 1: The configuration model starts with a set of nodes with half-edges \((\text{①})\) and constructs a network by pairing these up to form a full-edge at random, resulting in configurations like \(\text{②} \& \text{③}\). \(\text{②}\) shows an example where the configuration model can lead to self and duplicate edges, where as \(\text{③}\) gives a valid configuration. \(\text{④} \rightarrow \text{⑤}\) will rewire a network such that local structure is lost, for example performing many of these edge swaps yields a network with both negligible clustering and degree assortativity. In addition this rewiring introduces the small world property to a lattice.

The clustering of a network may be increased by looking for V-shaped configurations \((\text{⑥})\) and performing the rewiring \(\text{⑥} \rightarrow \text{⑦}\) if it increases the overall clustering coefficient of the network.

Erdős-Rényi networks [10], often referred to simply as ‘random graphs’ due to their importance, are defined such that each possible link is present with independent probability \(q\). This leads to a binomial degree distribution although practically this will always be very well approximated by a Poisson distribution with mean \(\langle k \rangle = (N - 1)q\).

In the configuration model (CM), nodes are given a number of ‘stubs’, which are then paired uniformly at random [37]. This construction is useful for the development of asymptotic results, but can cause problems for a given finite value of \(N\) due to the presence of ‘defects’—self-edges, multiple links between nodes, and stubs that are not eventually paired with others.

Networks with a given degree distribution can be generated following the procedure described in [9], which generates a sample of a given degree distribution that is graphical (meaning that a network with this degree distribution can be constructed without defects such as self or multiple edges) and then directly samples a statistically independent network. This algorithm is better behaved than the Configuration Model as it always produces a simple graph without defects, backtracking or rejections.

Algorithms that generate clustered heterogeneous networks using one random process typically generate special network topologies. We therefore generate clustered networks via a two-step process in which we first generate a network of the required degree distribution, and then increase the clustering coefficient using a rewiring method known as the ‘big-V’ [19, 3, 14]. This involves finding a ‘V’ configuration in the network (Fig. 1 \(\text{⑥}\)) and proposing a rewiring which produces a triangle and a separate edge (Fig. 1 \(\text{⑦}\)). This rewiring is then performed provided the clustering coefficient is increased. This method preserves the degree distribution whilst increasing the clustering coefficient (empirically) up to between \(\phi = 0.3\) and \(\phi = 0.4\) before the acceptance ratio of proposed moves critically slows down. The origin node, \(c\), is selected with a weight proportional to \(k_c(k_c - 1)\) so that the expected proportion of possible triangles present around each node does not depend on its degree [48].

4 Epidemic Dynamics on Locally Tree-like Networks

In the limit as \(\phi \rightarrow 0\) while \(N \rightarrow \infty\), it is possible to obtain results for the expected population-level epidemic dynamics that are known to be asymptotically exact.
4.1 Simple SIR Model

It is possible to show [49] that the following equations hold for an arbitrary network:

\[
\frac{d}{dt}[S] = -\tau[S]I, \quad \frac{d}{dt}[I] = \tau[S]I - \gamma[I].
\]  

There are two limits in which we can accurately approximate the pair variable \([SI]\) in terms of the node variables: (i) for an \(n\)-regular graph, as \(n \to N - 1\); and (ii) for an ER graph with mean degree \(\langle k \rangle\). In each case we take \([SI] \approx \langle k \rangle [S][I]/N\) with \(\beta := \tau \langle k \rangle\) to end up with a special case of the classic model in mathematical epidemiology introduced almost a century ago by Kermack and McKendrick [26], often called the simple SIR model,

\[
\frac{d}{dt}[S] = -\beta N[S][I], \quad \frac{d}{dt}[I] = \beta N[S][I] - \gamma[I].
\]

Homogeneous mixing in a population is a poor assumption and more structural information is required to focus on the underlying network of contacts.

4.2 Pairwise Model

The pairwise model was one of the first steps toward this more realistic contact structure, and primarily concerns \(n\)-regular graphs. In this approach, we continue to write down equations in the form (10),

\[
\begin{align*}
\frac{d}{dt}[SS] &= -2\tau[ISS], \\
\frac{d}{dt}[SI] &= \tau\left([ISS] - [ISI] - [IS]\right) - \gamma[IS], \\
\frac{d}{dt}[II] &= 2\tau\left([ISI] + [IS]\right) - 2\gamma[II].
\end{align*}
\]

The closed pairwise model [23, 43] is gained by taking the unclosed ODEs (10) and approximating the number of triples, \([ABC]\), in the system using a moment closure originally attributed to Kirkwood [27],

\[
[ABC] \approx \frac{n - 1}{n} \frac{[AB][BC]}{[B]}.
\]

4.3 Effective Degree (ED) Model

Ball and Neal [2] introduced the notion of an effective degree. In the Ball and Neal construction, individuals (nodes) begin with a number of unpaired half-links/stubs—an effective degree—and a contact network is constructed along with SIR epidemic dynamics.

The dynamics occur as follows: stubs of infected nodes randomly connect with stubs of susceptible nodes at rate \(\tau\), reducing the effective degree of both nodes by one and infecting the susceptible node; infected nodes become recovered at rate \(\gamma\), at which point they connect their remaining stubs uniformly at random to any remaining unpaired stubs in the network thus reducing their effective degree to zero. This process results in a configuration model network [37].

Following Ball and Neal [2], Lindquist et al. [31] developed an effective degree model categorising each node by its own disease state (S, I or R) along with the number of neighbours in each disease state resulting in the network being separated into classes representing the state of a node and its neighbours. For example, \(S_{s,i}\) denotes the star motif corresponding to a susceptible node where \(s\) and \(i\) are the number of susceptible and infected neighbours respectively.

Analogously to the Pairwise model (see Sect. 4.2), ODEs describing the time evolution of network motifs may be written down; however for the Effective Degree model there are \(M(M + 3)\) equations representing the time evolution of each of the possible star motifs (where \(M\) is the maximum degree) compared to the equations of the pairwise model (12-14) which represent the time evolution of all node and all edge combinations, of which there are 5 (\([S], [I], [SS], [SI], [II]\)).
The susceptible node at the centre of a $S_{s,i}$ star experiences a force of infection, $\tau$, from each of its $i$ infected neighbours with infection resulting in the transition from an $S_{s,i}$ star to an $I_{s,i}$ star. Each of the $i$ infectious neighbours recover at rate $\gamma$ and thus transition from the $S_{s,i}$ class to the $S_{s,i-1}$ class at rate $\gamma i[S_{s,i}]$. By the same reasoning the rate at which transitions from $S_{s,i+1}$ to $S_{s,i}$ occur is $\gamma(i+1)[S_{s,i+1}]$. Infection of one of the $s$ susceptible neighbours results in a transition from $S_{s,i}$ to $S_{s-1,i+1}$ occurring at a rate of

$$\frac{\sum_j \tau_{jl}[S_{j,l}]}{\sum_j \tau_{jl}[S_{j,l}]} s[S_{s,i}], \quad (16)$$

where above and henceforth notation of the form $\sum_{k=1}^{M} \sum_{j+k=l}$ is adopted to aid brevity. The rate derives from the fact that new infections are generated at rate $\sum_j \tau_{jl}[S_{j,l}]$ which in turn causes the effective degree of the susceptible neighbours of the now infected $[S_{j,l}]$ to change at rate $\sum_j \tau_{jl}[S_{j,l}]$. Put in the notation of Sect. 4.3 this can be expressed as the rate at which transitions $[ISS] \rightarrow [IIS]$ occur which is $\tau[ISS]$ where,

$$[ISS] = \sum_j [IS, j] S = \sum_j j[l][S_{j,l}] . \quad (17)$$

As we wish to express the rate at which a susceptible neighbour of a $S_{s,i}$ star becomes infected, i.e. the rate of $[ISS_{s,i}] \rightarrow [IIS_{s-1,i+1}]$, we must account for the probability that the $S$–$S$ link in the $[ISS]$ triple will connect to a $S_{s,i}$ star which is given by the following identity,

$$[ISA_{s,i}] = [ISA] \frac{s[A_{s,i}]}{\sum_j A_{j,l}} , \quad A \in \{S, I\} . \quad (18)$$

Putting this all together then yields the rate given in (16). Mutatis mutandis the rate of transition from $S_{s+1,i-1}$ to $S_{s,i}$ is obtained.

Using the above rates the set of ODEs describing the time evolution of star motifs with a central susceptible are obtained as (19) below. Following similar reasoning allows (20) below to be derived with the extra term $\gamma[I_{s,i}]$ corresponding to the recovery of the infectious individual at the centre of the star:

$$\frac{d[S_{s,i}]}{dt} = \gamma ((i + 1)[S_{s,i+1}] - i[S_{s,i}]) + \frac{\sum_j \tau_{jl}[S_{j,l}]}{\sum_j \tau_{jl}[S_{j,l}]} (s + 1)[S_{s+1,i-1}] - s[S_{s,i}] - \tau i[S_{s,i}], \quad (19)$$

$$\frac{d[I_{s,i}]}{dt} = \gamma ((i + 1)[I_{s,i+1}] - i[I_{s,i}]) + \frac{\sum_j \tau_{jl}[S_{j,l}]}{\sum_j \tau_{jl}[S_{j,l}]} (s + 1)[I_{s+1,i-1}] - s[I_{s,i}] - \gamma[I_{s,i}] + \tau i[I_{s,i}]. \quad (20)$$

The constraints upon $s$ and $i$ are given by $\{(s, i) : s \geq 0, i \geq 0, s + i \leq M\}$. Figure 2 summarises these two sets of equations governing the system in graphical form.

It is noteworthy to remark that the closure in this model is more implicit than that of the pairwise model, i.e. rather than approximating higher order states with lower order states one makes the assumption that the infectious pressure on the susceptible neighbours of the central node is equal to the population average.

### 4.4 Probability Generating Function (PGF) Methods

PGF models \[52, 56\] provide low-dimensional representations of epidemic dynamics on configuration models and have been proved to be asymptotically exact \[7, 5, 4, 20\].

In the simplest form of PGF model, given in \[34\], epidemic dynamics can be captured by one differential equation,

$$\frac{d\theta}{dt} = -\tau \theta + \gamma (1 - \theta) + \frac{\psi'(\theta)}{\psi'(1)} . \quad (21)$$

The Volz variable $\theta$ represents the probability that a ‘test node’ with one link remains susceptible, and its use is responsible for the massive simplicity of this model.
5 Dynamics on Clustered Networks

5.1 Pairwise Model

The Kirkwood closure of Sect. 4.2 can be extended to clustered networks by introducing a correlation term, $C_{CA} = \frac{N}{\langle k \rangle} [CA]$, that accounts for the number of transitive links between A and C by measuring the observed $[CA]$ compared to the number of A–C pairs one would expect given independence between C and A, $\langle k \rangle [A][C]$. If $C_{CA} = 1$ then nodes of type C and nodes of type A are connected at random. Given a clustering coefficient, $\phi$, the clustered Kirkwood closure is

$$[ABC] \approx n^{-1} \frac{[AB][BC]}{[B]} ((1 - \phi) + \phi C_{AC}).$$

(22)

5.2 Clustered PGF

House and Keeling introduced the clustered PGF model, which consists of the following differential equations with $[S] = N\psi(\theta)$ and $Y = \sum_k k[I_k]$:

$$\frac{d\theta}{dt} = -\tau \frac{[SI]}{N\psi(\theta)}, \quad \frac{d[I]}{dt} = \tau [SI] - \gamma [I], \quad \frac{dY}{dt} = \gamma [I] - \gamma Y,$$

(23)

together with (12), (13) and (14), and the closures

$$[ISS] \approx \frac{[SI][SS]\psi'(\theta)}{N^2 \psi(\theta)^2} \left(1 - \phi + \phi \langle k \rangle \frac{[SI]}{N^2 \psi(\theta)^2}\right).$$

(24)

$$[ISI] \approx \frac{[SI]^2 \psi'(\theta)}{N\psi(\theta)^2} \left(1 - \phi + \phi \langle k \rangle \frac{[II]}{Y^2}\right).$$

(25)

The clustered PGF is the main model that attempts to deal with epidemics on clustered heterogeneous networks and as such is the main direct comparator to our new approach.

5.3 A New Model

The ED degree model (Sect. 4.3) approximates the epidemic dynamics on a network of arbitrary degree distribution extremely well; however, in the large network limit the clustering coefficient of...
the configuration model network it explains tends to zero, which does not realistically explain real world contact networks. A new set of equations extending the effective degree model to clustered networks is now presented.

An exact version of the ED model may be written as follows,

\[
\frac{d[S_{s,i}]}{dt} = -\tau_i[S_{s,i}] + \gamma((i+1)[S_{s,i+1}] - [S_{s,i}]) - \tau[ISS_{s,i}]_\triangle - \tau[ISS_{s,i}]_\wedge + \\
\tau[ISS_{s+1,i-1}]_\triangle + \tau[ISS_{s+1,i-1}]_\wedge,
\]

\[
\frac{d[I_{s,i}]}{dt} = \tau_i[S_{s,i}] + \gamma((i+1)[I_{s,i+1}] - [I_{s,i}]) - \tau[ISI_{s,i}]_\triangle - \tau[ISI_{s,i}]_\wedge + \tau[ISI_{s+1,i-1}]_\triangle \\
+ \tau[ISI_{s+1,i-1}]_\wedge - \gamma[I_{s,i}] + \tau((s+1)[I_{s+1,i-1}] - s[I_{s,i}]),
\]

where we separate out transmission events happening within and outside of the neighbourhood of the star: \([ISS_{s,i}]_\triangle\) denotes a closed triple forming a triangle (e.g. edge ① Fig. 3) and \([ISS_{s,i}]_\wedge\) denotes an unclosed triple (e.g. edge ② Fig. 3).

Table 1: Counts of possible triangles around a node in state \(A_{s,i}\).

| Triangle states | \([SSA_{s,i}]_\triangle\) | \([ISA_{s,i}]_\triangle\) | \([IIA_{s,i}]_\triangle\) |
|-----------------|-----------------|-----------------|-----------------|
| Combinations    | \(\frac{1}{2}s(s-1)\) | \(i\)           | \(\frac{1}{2}i(i-1)\) |

In a network with a clustering coefficient of zero then \([ABC]_\triangle = 0\) and a closure on the \([ABC]_\wedge\) terms will yield the ED model \([31]\); however an effective closure for clustered networks must be made on both \(\triangle\) and \(\wedge\) terms.

Given a clustering coefficient, \(\phi\), there are an expected \(\phi k(k-1)/2\) triangles around a given degree-\(k\) node. If we decompose the effective degree (c.f. Sect. [4.3]) of a node, \(k\), into its susceptible and infected neighbours such that \(s + i = k\) then Table 1 enumerates the expected number of triangles involving different states, given no correlations between states.

\([ISS_{s,i}]_\triangle = \phi is[S_{s,i}]\) as the clustering coefficient, \(\phi\), gives the expected ratio of edges connecting a node’s neighbours together to the maximum possible number of such edges (Table 1). Correlations between the states cannot be ignored, therefore the correlation between nodes of state A and B, \(C_{AB} = \frac{N}{k} \langle AB \rangle [A][B]\), is introduced which is used to account for how many \([AB]\) pairs there are compared to how many one would expect from random mixing, \(\langle k \rangle [A] [B] / N\). This yields the closure equation \([31]\), and mutatis mutandis \([33]\) is obtained.
The original ED closure for the $\wedge$ terms must now be modified to account for the infection events that happen within the $\Delta$ closure. The new closure is achieved by taking the original expressions and, for $A \in \{S, I\}$, using the identity $[ISA]_\Delta + [ISA]_\wedge = [ISA]$ along with the original and the $\Delta$ closure. For example,

\[
[ISS] = \sum_{j,l} [IS_{j,l}S] = \sum_{j,l} jl[S_{j,l}], \tag{28}
\]

\[
[ISS]_\Delta = \sum_{j,l} [IS_{j,l}S]_\Delta = \sum_{j,l} \phi_c S_{j,l}, \tag{29}
\]

\[
\Rightarrow [ISS]_\wedge = \sum_{j,l} [IS_{j,l}S]_\wedge = \sum_{j,l} jl(1 - \phi_c)S_{j,l}. \tag{30}
\]

Using identity (18), one gains the final clustered effective degree closures (31-34) which when substituted into (26-27) yield the clustered ED model.

\[
[ISS_{s,i}]_\Delta = \phi_c S_{s,i}, \tag{31}
\]

\[
[ISS_{s,i}]_\wedge = \frac{\sum_{j,l} jl(1 - \phi_c)S_{j,l}}{\sum_{j,l} S_{j,l}} s_{s,i}, \tag{32}
\]

\[
[ISI_{s,i}]_\Delta = \phi_c I_{s,i}, \tag{33}
\]

\[
[ISI_{s,i}]_\wedge = \frac{\sum_{j,l} (l - 1)(1 - \phi_c)S_{j,l}}{\sum_{j,l} I_{j,l}} s_{s,i}. \tag{34}
\]
6 Simulation Study

To test the accuracy of the clustered ED model we perform a simulation study.

6.1 Methodology

First, unclustered networks are generated for three different degree distributions according to the methods presented in Sect. 3. The big-V algorithm (Fig. 1) was then applied to each unclustered network to generate a series of networks with approximate clustering coefficients \( \phi \in \{0.05, 0.10, 0.15, 0.20, 0.25, 0.30\} \).

Numerical simulation was performed using an individual-based analogue of Gillespie’s algorithm [11], which generates a statistically correct trajectory with regard to the master equation of the underlying stochastic process. Simulations were performed for 20 uniquely generated networks of \( 10^5 \) nodes with the dynamics being simulated on each network 5 times, for a total of 100 epidemics per network type. To account for the large stochastic variability at the beginning of an epidemic, we shifted the time-origins of each of the 100 epidemics to coincide at the point where 500 individuals are infected before averaging the dynamics, which we then compare to each differential equation model.

We choose to generate and simulate networks of mean degree four as a rigorous model comparator—closures perform better as \( \langle k \rangle \) increases as the behaviour tends towards the simple SIR model since these networks are closer to complete graphs.

6.2 Results

The results in Figs. 4–6 show that our novel clustered ED model generally outperforms the clustered PGF approach in terms of errors in expected prevalence, with exceptions to this only occurring after the epidemic peak.

Fig. 4: Simulated epidemic dynamics for 4-regular networks with \( \gamma = 1, \tau = 2, N = 10^5 \).
Approximate epidemic dynamics on heterogeneous clustered networks

Fig. 5: Simulated epidemic dynamics for Erdős-Rényi networks with $\langle k \rangle = 4, \gamma = 1, \tau = 2, N = 10^5$.

Fig. 6: Simulated epidemic dynamics for Negative binomial networks with $\langle k \rangle = 4, r = 5, \gamma = 1, \tau = 2, N = 10^5$. We use the formulation where we are counting $k$ successes given $r$ failures.

Work by Pellis et al. [42] argued (based on a rigorous analysis of finite-size networks) that we should expect moment closure to be exact when (i) triangles are not overlapping and (ii) recovery happens after a constant time (or not at all as when $\gamma = 0$). When the clustering coefficient is small, the proportion of overlapping triangles will be $O(\phi^2)$, and so for small values of $\gamma$ we expect errors in the prediction of prevalence of infection to be $O(\phi^2)$, while for larger values we expect them to be $O(\phi)$. We found that in general, obtaining accurate assessments of absolute error in predictions of prevalence was numerically challenging, and it is likely that deeper theoretical understanding of the source of errors would be required to perform a definitive computational analysis of this question. Nevertheless, we were able to obtain the results shown in Fig. 7, which demonstrate that as expected errors lie between the $O(\phi)$ and $O(\phi^2)$ lines.
In this paper, we have shown how to combine effective degree approaches to epidemics on heterogeneous networks with moment closure approaches to clustering. Numerical results suggest that the errors introduced in so doing may be better than $O(\phi)$ but worse than $O(\phi^2)$, meaning that the potential for improvements remains. In particular, the closure due to Kirkwood \cite{27} could be replaced by the improved closure in \cite{17} or the maximum entropy method \cite{47}. In particular, in the large $\phi$ limit we expect graphs to be dominated by cliques \cite{8}, whose differential equation limit is described by equations such as those written in \cite{1}.

Fig. 7: Error between the clustered ED model and expected prevalence of the stochastic simulations detailed in Sec. 6.1 is plotted against errors of order $\phi$, $\phi^2$, and $\phi^3$ plotted in red, green, and blue respectively.

7 Discussion

In this paper, we have shown how to combine effective degree approaches to epidemics on heterogeneous networks with moment closure approaches to clustering. Numerical results suggest that the errors introduced in so doing may be better than $O(\phi)$ but worse than $O(\phi^2)$, meaning that the potential for improvements remains. In particular, the closure due to Kirkwood \cite{27} could be replaced by the improved closure in \cite{17} or the maximum entropy method \cite{47}. In particular, in the large $\phi$ limit we expect graphs to be dominated by cliques \cite{8}, whose differential equation limit is described by equations such as those written in \cite{1}.
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