Epidemic threshold in pairwise models for clustered networks: closures and fast correlations

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Abstract

The epidemic threshold is probably the most studied quantity in the modelling of epidemics on networks. For a large class of networks and dynamics the epidemic threshold is well studied and understood. However, it is less so for clustered networks where theoretical results are mostly limited to idealised networks. In this paper we focus on a class of models known as pairwise models where, to our knowledge, no analytical result for the epidemic threshold exists. We show that by exploiting the presence of fast variables and using some standard techniques from perturbation theory we are able to obtain the epidemic threshold analytically. We validate this new threshold by comparing it to the numerical solution of the full system. The agreement is found to be excellent over a wide range of values of the clustering coefficient, transmission rate and average degree of the network. Interestingly, we find that the analytical form of $R_0$ depends on the choice of closure, highlighting the importance of model choice when dealing with real-world epidemics. Nevertheless, we expect that our method will extend to other systems in which fast variables are present.

Keywords: network, epidemic, pairwise model, clustering, correlation, fast variables

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1 Introduction

Epidemic dynamics on networks, being susceptible-infected-susceptible (SIS), susceptible-infected-recovered/removed (SIR) or otherwise, are often modelled as continuous time Markov chains with discrete but extremely large state spaces of order $m^N$, where $m$ is the number of different disease statuses (e.g. $m = 2$ for SIS and $m = 3$ for SIR) and $N$ is the number of nodes/individuals in the network. This makes the analysis of the resulting exact system almost impossible, except for some specific network topologies such as the fully connected network, networks with considerable structural symmetry or networks with few nodes \[14, 6\] that allow for simplification.

This problem, instead, has been dealt with by focusing on mean-field models where the goal is to derive, often heuristically, a system of ordinary or integro-differential equations that describe (non-Markovian) epidemics for some average quantities, such as the expected number of nodes in various states, the expected number of links in various states or the expected number of star-like structures (focusing on a node and all of its neighbours). These methods usually rely on closures to break the dependency on higher-order moments (e.g. the expected number of nodes in a state depends on the expected number of links in certain states and so on). Such an approach has led to a number of models including heterogeneous or degree-based mean-field \[24, 23\], pairwise \[25, 12\], effective-degree \[16\], edge-based compartmental \[20\] and message passing \[10\], to name a few. These models essentially differ in the choice of variables over which the averaging is done. Perhaps the most compact model with the fewest number of equations is the edge-based compartmental model \[21\] and this works for heterogeneous networks with Markovian SIR epidemics, although extensions of it for arbitrary infection and recovery processes are also possible \[29\].

Pairwise models have been extremely popular and the very first model for regular networks and SIR epidemics \[25, 12\] has been generalised to heterogeneous networks \[4\], preferentially mixing networks \[4\], directed \[28\] and weighted networks \[26\], adaptive networks \[5, 13, 31\], and structured networks \[7\] among others. Perhaps this is due to its relative simplicity and transparency, whereby variables seem to make sense in a straightforward way and a basic understanding of the network and epidemic dynamics coupled with good bookkeeping leads to a valid and analytically tractable model. Pairwise models have been successfully used to derive analytically the epidemic threshold and final epidemic size, with these results mostly limited to networks without clustering. The propensity of contacts to cluster, i.e. that two friends of an individual/node are also friends of each other, is known to lead to many complications, and modelling epidemics on clustered networks using analytically tractable mean-field models is still limited to networks with very specific structural features \[7, 22, 17, 18, 11, 34, 27\]. However, using approaches borrowed from percolation theory \[18\] and focusing more on the stochastic process itself \[32\], some results have been obtained. For example, in \[18\] it was shown that for the susceptible-infected-recovered (SIR) epidemic on clustered networks with heterogeneous degree distributions,
the basic reproduction number is given by

\[ R_0 = \frac{\langle k^2 - k \rangle}{\langle k \rangle} T - \frac{2\langle n_\triangle \rangle}{\langle k \rangle} T^2 + \cdots, \quad (1.1) \]

where \( \langle k^i \rangle \) stands for the \( i \)th moment of the degree distribution, \( T \) is the probability of infection spreading across a link connecting an infected to a susceptible node and \( \langle n_\triangle \rangle \) denotes the average number of triangles that a node belongs to. The first positive term corresponds to what the threshold is for configuration-type networks with no clustering. The second term, which is negative, shows that clustering reduces the epidemic threshold when compared to the unclustered case.

For pairwise models, clustering first manifests itself by requiring a different and more complex closure, which makes the analysis of the resulting system, even for regular networks and SIR dynamics, challenging. Furthermore, it turns out that such closure may in fact fail to conserve pair-level relations and may not accurately reflect the early growth of quantities such as closed loops of three with all nodes being infected \([8]\). Such considerations have led to an improved closure being developed in an effort to keep as many true features of the exact epidemic process as possible \([8]\). In this paper we will focus on the classic pairwise model for regular networks with clustering, using both the simplest closure and a variant of the improved closure. We will show that by working with two fast variables corresponding to the correlations that develop during the spread of the epidemic, we can analytically determine the epidemic threshold as an asymptotic expansion in terms of the clustering coefficient.

The use of fast variables is not completely new and has been used in \([12]\) and \([3]\) but the epidemic threshold has only been obtained numerically and it was framed in terms of a growth-rate-based threshold which of course is equivalent to the basic reproduction number at the critical point of the epidemic spread. In \([3]\) a hybrid pairwise model incorporating random and clustered contacts is considered, with the analysis focused on the growth-rate-based threshold. The authors of \([3]\) managed to derive a number of results, some analytic (the critical clustering coefficient for which an epidemic can take off) and some semi-analytic, and they have shown, in agreement with most studies, that clustering inhibits the spread of the epidemic when compared to an equivalent network without clustering but with equivalent parameter values governing the epidemic process. However, no analytic expression for the threshold was provided.

More recently, in \([15]\), the epidemic threshold in a pairwise model for clustered networks with closures based on the number of links in a motif, rather than nodes, was calculated as

\[ R_0 = \frac{(n - 1)\tau}{\tau + \gamma + \tau\phi}, \quad (1.2) \]

where \( n \) is the average number of links per node, \( \phi \) is the global clustering coefficient (see later sections for a formal definition) and \( \tau \) and \( \gamma \) are the infection and recovery rates,
respectively. The expression above can be expanded in terms of $\phi$ to give

$$R_0 = \frac{(n-1)\tau}{\tau + \gamma} \left( \frac{1}{1 + \phi \frac{\tau}{\tau + \gamma}} \right) \simeq \frac{(n-1)\tau}{\tau + \gamma} \left( 1 - \phi \frac{\tau}{\tau + \gamma} + \cdots \right),$$

(1.3)

which again reflects that clustering reduces the epidemic threshold.

Building on these results, and effectively extending the work in [12, 3], our paper sets out to present a method to determine the epidemic threshold analytically and apply it in the context of pairwise models with two different closures for clustered networks. The paper is structured as follows. In Section 2 we outline the model with closures for unclustered and clustered networks discussed in Section 3. In Section 4 we briefly review existing results and approaches for the pairwise model with the simple closure and then focus on the correlation structure in terms of fast variables, showing that the epidemic threshold can be expressed via the solution of a cubic polynomial. This key solution is determined numerically and analytically as an asymptotic expansion in terms of the clustering coefficient. In Section 5 we show that our approach works for a compact version of the improved closure, thus validating and generalising our approach. Finally we conclude with a discussion of the results, including comparing the threshold to other known results and touching upon a number of possible extensions.

2 Model formulation

2.1 The network

We begin by considering a population of $N$ individuals with its contact structure described by an undirected network with adjacency matrix $G = (g_{ij})_{i,j=1,2,\ldots,N}$ where $g_{ij} = 1$ if nodes $i$ and $j$ are connected and zero otherwise. Self-loops are excluded, so $g_{ii} = 0$ and $g_{ij} = g_{ji}$ for all $i,j = 1,2,\ldots,N$. The network is static and regular, such that each individual has exactly $n$ edges or links. The sum over all elements of $G$ is defined as $||G|| = \sum_{i,j} g_{ij}$. Hence, the number of doubly counted links in the network is $||G|| = nN$. More importantly, using simple matrix operations on $G$, we can calculate the clustering coefficient of the network

$$\phi = \frac{\text{trace}(G^3)}{||G^2|| - \text{trace}(G^2)},$$

(2.1)

where $\text{trace}(G^3)$ yields six times the number of closed triples or loops of length three (uniquely counted) and $||G^2|| - \text{trace}(G^2)$, twice the number of triples (open and closed, also uniquely counted).

2.2 SIR dynamics

The standard SIR epidemic dynamics on a network is considered. The dynamics are driven by two processes: (a) infection and (b) recovery from infection. Infection can spread from
an infected/infectious node to any of its susceptible neighbours and this is modelled as a
Poisson point process with per-link infection rate $\tau$. Infectious nodes recover from infection
at constant rate $\gamma$.

2.3 The unclosed pairwise model

Let $A_i$ equal 1 if the individual at node $i$ is of type $A$ and equal zero otherwise. Then
single nodes (singles) of type $A$ can be counted as $[A] = \sum_i A_i$, pairs of nodes (pairs) of
type $A - B$ can be counted as $[AB] = \sum_{i,j} A_i B_j g_{ij}$ and triples of nodes (triples) of type
$A - B - C$ can be counted as $[ABC] = \sum_{i,j,k} A_i B_j C_k g_{ij} g_{jk}$. This method of counting means
that pairs are counted once in each direction, so $[AB] = [BA]$, and $[AA]$ is even. Using this
notation to keep track of singles, pairs and triples leads to the following system of pairwise
equations describing the SIR epidemic on a regular network:

\[
\begin{align*}
\dot{S} & = -\tau [SI], \\
\dot{I} & = \tau [SI] - \gamma [I], \\
\dot{SI} & = \tau ([SSI] - [ISI] - [SI]) - \gamma [SI], \\
\dot{SS} & = -2\tau [SSI], \\
\dot{II} & = 2\tau ([ISI] + [SI]) - 2\gamma [II].
\end{align*}
\]

We note that equations (2.4)-(2.6) contain triples which are not defined within the
entire system of equations (2.2)-(2.6). The flow between compartments and the rates are
illustrated in Fig. 1. To determine solutions of the system, we must find a way to account
for these triples in terms of pairs and singles, a method referred to as closing the system.

3 Closures

A quick inspection of the unclosed pairwise system (2.2)-(2.6) reveals that only triples of
type $[ASI]$ need closing, with $A \in \{S, I\}$. These triples, as well as triples of type $[RSI]$, are
illustrated in Fig. 2 for unclustered and clustered networks.

3.1 Closure for unclustered networks

First, we consider the situation depicted in Fig 2a. Several observations can be made. The
expected number of $A - S$ type links is $[AS]$ and the total number of links emanating from
susceptible nodes counted across the whole network is $n[S]$. Hence, the most straightforward
approximation would be to assume that $X_i$, with $i = 1, 2, \ldots, n - 1$, are independent
Figure 1: Flow diagrams showing the flux between compartments of singles (left) and compartments of pairs (right). In the compartments of pairs, straight arrows denote infections coming from within the pair (with a rate depending on a pair) or from outside the pair (with a rate depending on a triple), and curved arrows denote a recovery. The colour indicates the status of the “first” node in the pair. Symmetry allows us to conclude that some of the variables (see lighter shaded variables on the right hand side of the pairs diagram) must equal their symmetric version (e.g. $[RS] = [SR]$), so we do not need to directly calculate both quantities.

Figure 2: General setup for a central susceptible node with a given infected neighbour for (a) unclustered and (b) clustered regular networks with degree $n$. Dashed arrows indicate that the infected node may be connected to the other neighbours of the central susceptible node. Random variables $X_1, X_2, \ldots, X_{n-1}$ take values from the set $\{S, I, R\}$. 
and identically Bernoulli distributed random variables with a probability of success being equal to $p_{A|S-I} = \frac{[AS]}{n[S]}$. Averaging across the whole network leads to

$$[ASI] = [SI](n - 1)p_{A|S-I} = \frac{n - 1}{n} \frac{[AS][SI]}{[S]}.$$  (3.1)

It is important to note that the new, closed system is effectively an approximation of the exact pairwise model and one should question if the closure (3.1) conserves the properties of the stochastic process and of the counting on the network. For example, it is expected that in the closed system the number of nodes is conserved, i.e. $[S] + [I] + [R] = N$. Furthermore, the number of pairs of different types must sum to $nN$. More subtle conditions refer to the conservation of link types at node level ($[SS] + [SI] + [SR] = n[S]$) and pair level ($[SSI] + [ISI] + [RSI] = (n - 1)[SI]$), respectively. It turns out that the closure for unclustered networks (3.1) conserves these relations [14]. Finally, the validity or appropriateness of closures can be empirically assessed by looking at the initial growth rate of the number of open and closed triples, where the number of open triples comprised of three infectious nodes should grow differently to the number of such closed triples. Of course such subtle tests/comparisons are usually preceded by direct comparisons between the numerical solution of the closed pairwise system and explicit stochastic network simulations for a range of parameters. Such tests initially focus on prevalence of infection and final epidemic size but may include expected number of pairs.

### 3.2 Closures for clustered networks

#### 3.2.1 Simple closure

The presence of closed loops of length three, as illustrated in Fig. 2b, introduces some complications. Namely, a neighbour of the central susceptible that is itself connected to an infected neighbour of the central node, is less likely to be susceptible due to the added pressure from the infected neighbour, when compared to the case when the force of infection is distributed evenly, as is the case for the closure for unclustered networks (3.1). More precisely, the epidemic process on the network displays clear correlations. In [1] it has been shown that the exact SIS and SIR epidemics on networks are non-negatively correlated in the sense that $P(I_i I_j) \geq P(I_i)P(I_j)$. Here, $P(I_i I_j)$ represents the probability that nodes $i$ and $j$, connected by a link, are both infected, while $P(I_i)$ stands for the probability of node $i$ being infected. For this result to hold, all processes must be Markovian and infection rates across all links and recovery rates of all nodes have to be fixed a priori. Using the pairwise model for an SIS epidemic on an unclustered network with closure (3.1), it has been shown that the same correlation is preserved when averaging at the population level [14]. While the proof has not been executed for the pairwise SIR model, intuitively we expect to find the same correlation structure. Based on these observations, we assume that the correlation
structure in exact SIS and SIR epidemics on networks averaged at the population level is maintained. Hence, the inequalities

\[ [SI] \leq n[S] [I] \frac{N}{N}, \quad [II] \geq n[I] [I] \frac{N}{N}, \quad \text{and} \quad [SS] \geq n[S] [S] \frac{N}{N}, \]  

(3.2)

hold, where \([AB]\) and \([A]\) with \(A, B \in \{S, I\}\) represent the expected counts of pairs and singles of the corresponding types taken from the exact model, i.e., the continuous time full Markov chain.

Intuitively, this means that as the epidemic spreads on the network, infected nodes are more likely to have neighbours which are themselves infected (either those that infected the node or were infected by it), and at the ‘front’ of the epidemic we would expect to observe a ‘sea’ of susceptible nodes alongside a ‘front’ of links between susceptible and infected nodes that drives the epidemic. Hence, clustering and correlations need to be accounted for and a new \(p_{AI|S-I}^{uc}\) for clustered networks needs to be defined. This has been done in [12] and it relies on a correlation factor, \(C_{AB}\), that is able to capture the propensity of nodes of type \(A\) and \(B\) being neighbouring nodes. This is given by

\[ C_{AB} = \frac{[AB]}{n[A][B]}, \]  

(3.3)

where \(A, B \in \{S, I\}\). This effectively compares the expected number of edges of type \([AB]\) to what its value would be if nodes were labelled at random with \([A]\) nodes of type \(A\) and \([B]\) nodes of type \(B\). If \(C_{AB} > 1\), then nodes of type \(A\) and \(B\) are positively correlated, whereas if nodes of type \(A\) and \(B\) are negatively correlated, \(C_{AB} < 1\). As expected, \(C_{AB} = 1\) means that nodes are effectively labelled as type \(A\) or \(B\) at random. Equation (3.2) implies that

\[ C_{SI} \leq 1, \quad C_{II} \geq 1 \quad \text{and} \quad C_{SS} \geq 1. \]  

(3.4)

We can augment \(p_{AI|S-I}^{uc}\) to reflect these observations, leading to \(p_{AI|S-I}^{uc} = \frac{[AS]}{n[S]} C_{AI}\). However, before the closure can be expressed, open and closed loops need to be treated separately. In order to do this, we split the closure based on whether the neighbour whose state is to be determined is part of a closed loop of three nodes and thus in direct contact with an infectious node, or not. This leads to

\[ p_{AI|S-I}^{uc} = \begin{cases} 
p_{AI|S-I}^{uc} & \text{with probability } (1 - \phi), \\
p_{AI|S-I}^{uc} C_{AI} & \text{with probability } \phi, 
\end{cases} \]  

(3.5)

where \(\phi\) is defined in equation (2.1). With this in mind, the closure can be derived by averaging equation (3.1) over the unclustered and clustered parts of the network. This leads to

\[ [ASI] = (1 - \phi)(n - 1)[SI]p_{AI|S-I}^{uc} + \phi(n - 1)[SI]p_{AI|S-I}^{uc} C_{AI} \]  

(3.6)
\[
\frac{(n - 1) [AS][SI]}{n} \left( 1 - \phi \right) + \phi \frac{N[AI]}{n[A][I]}.
\] (3.7)

Framing \( p_{\text{rc}}^\text{uc} \) and \( p_{\text{rc}}^\text{c} \) more generally and independently of the network type, i.e. simply considering \( p_A \), the following statement holds:

**Proposition 1.** Consider a closure of the following form \([ASI] = (n - 1)[SI]p_A\). If \( \sum_A p_A = 1 \), where \( A \) is taken over all possible states, then \( \sum_A [ASI] = (n - 1)[SI] \).

**Proof.** \( \sum_A [ASI] = (n - 1)[SI] \sum_A p_A = (n - 1)[SI] \).

### 3.2.2 Improved closure

We note that while \( p_{\text{uc}}^\text{uc} \) satisfies the above proposition, \( p_{\text{c}}^\text{c} \) does not. In particular, we find

\[
\sum_A [ASI] = \sum_A (n - 1)[SI]p_{\text{c}}^\text{c} = \sum_A (n - 1)[SI] \frac{[AS]}{n[S]} N[AI] n[A][I] n[S]
\]

\[
= \frac{(n - 1)[SI]}{n[S]} \sum_A [AS] = \frac{(n - 1)[SI]}{n[S]} n[S] = (n - 1)[SI].
\]

However, for the clustered part of the network this is not the case. We find that

\[
\sum_A [ASI] = \sum_A (n - 1)[SI]p_{\text{c}}^\text{c} = \sum_A (n - 1)[SI] \frac{[AS]}{n[S]} \frac{N[AI]}{n[A][I]} n[S]
\]

\[
= \frac{(n - 1)N[SI]}{n^2[S][I]} \sum_A \frac{[AS][AI]}{[A]},
\]

which does not result in the desired \( (n - 1)[SI] \). This can be corrected in a straightforward way by defining

\[
p_{\text{c}}^{\text{new}} = \begin{cases} 
 p_{\text{c}}^\text{uc} & \text{with probability } (1 - \phi), \\
 \frac{p_{\text{c}}^\text{c}}{\sum_a p_{a|S-I}^\text{c}} & \text{with probability } \phi.
\end{cases}
\]

(3.8)

Hence we can now write

\[
\sum_A [ASI] = \sum_A ((1 - \phi)[ASI] + \phi[ASI])
\]

\[
= (1 - \phi)(n - 1)[SI] \sum_A p_{\text{c}}^{\text{new}} + \phi(n - 1)[SI] \sum_A p_{\text{c}}^{\text{new}}
\]

\[
= (1 - \phi)(n - 1)[SI] \sum_A \frac{[AS]}{n[S]} + \phi(n - 1)[SI] \sum_A \frac{p_{\text{c}}^{\text{new}}}{\sum_a p_{a|S-I}^\text{c}}
\]
It is informative to investigate the relationship between the various probability models that lead to different closures. This is summarised in the following proposition.

**Proposition 2.** For closures applied across the clustered part of the network and assuming that the number of nodes in state $R$ is negligible, it follows that

\[
p^{\text{new}}_{S|S-I} = \frac{[SS][I]}{[SS][I] + [II][S]}, \quad p^{c}_{S|S-I} = \frac{[SS] N[S]}{n[S] n[S][I]}, \quad p^{\text{wc}}_{S|S-I} = \frac{[SS]}{n[S]},
\]

and

\[
p^{c}_{S|S-I} \leq p^{\text{wc}}_{S|S-I} \quad \text{and} \quad p^{\text{new}}_{S|S-I} \leq p^{\text{wc}}_{S|S-I}.
\]

**Proof.** All three probabilities follow from their definitions and assuming that $A \in \{S, I\}$. Since $S - I$ links are negatively correlated (3.2), it follows that $C_{SI} = \frac{N[S]}{n[S][I]} \leq 1$ and as a result

\[
p^{c}_{S|S-I} = \frac{[SS]}{n[S]} C_{SI} \leq \frac{[SS]}{n[S]} = p^{\text{wc}}_{S|S-I}.
\]

While $p^{c}_{S|S-I}$ has a natural interpretation (it is a simple discounted variant of the probability from the unclustered network case and takes into account the observation that if the neighbour of a central susceptible node is connected to one of the infected neighbours of the same node then it is less likely that the node in question is susceptible), the interpretation of $p^{\text{new}}_{S|S-I}$ is less obvious. A close inspection reveals that $p^{\text{new}}_{S|S-I}$ can be rewritten as

\[
p^{\text{new}}_{S|S-I} = \frac{[SS][I]}{[SS][I] + [II][S]} = \frac{[SS]}{[SS] + [II][S]},
\]

However, combining $[SI] \leq n[S]\frac{I}{N}$ with $[I] \leq \frac{N[I]}{n[I]}$, as given in equation (3.2), leads to $[SI] \leq [II]\frac{[S]}{[I]}$. Finally, using the relation $[SI] \leq [II]\frac{[S]}{[I]}$ in equation (3.12) yields

\[
p^{\text{new}}_{S|S-I} = \frac{[SS]}{[SS] + [II]\frac{[S]}{[I]}} \leq \frac{[SS]}{[SS] + [SI]} = \frac{[SS]}{n[S]} = p^{\text{wc}}_{S|S-I}.
\]

Equation (3.13) illustrates that as expected $p^{\text{new}}_{S|S-I} \leq p^{\text{wc}}_{S|S-I}$. Again, this simply shows that for clustered networks and for the setup in Fig. 2, it is less likely to find neighbours who are susceptible compared with the unclustered network case. \(\square\)
Taking into account the new way of defining $p_{A|S−I}^\text{new}$, the improved closure yields

$$[ASI] = (1 - \phi)[ASI] + \phi[ASI]$$

$$= (1 - \phi)(n - 1)[SI] \frac{[AS]}{n[S]} + \phi(n - 1)[SI] \frac{[AS] C_{AI}}{\sum_a P_{a|S−I}}$$

$$= (1 - \phi) \left( \frac{n - 1}{n} \frac{[AS][SI]}{[S]} + \phi(n - 1) \frac{[AS][SI]}{[A] \sum_a [aS][aI]/[a]} \right) \sum_a P_{a|S−I}$$

$$= (n - 1) \left( (1 - \phi) \frac{[AS][SI]}{n[S]} + \phi \frac{[AS][SI]}{[A] \sum_a [aS][aI]/[a]} \right). \quad (3.14)$$

We finally note that the closures rely heavily on the assumption of how the states of the neighbours are distributed, and the assumption of independent and identically Bernoulli-distributed variables is a strong one. For clustered networks in particular, we have illustrated different ways of incorporating correlations induced by closed cycles of length three. Despite these seemingly strong assumptions, it is known that the pairwise model for unclustered networks is equivalent to the edge-based compartmental equivalent on configuration networks [19, 14] and the latter has been shown to be the limiting system of the stochastic network epidemic model [2, 9]. For clustered networks we are not aware of such results.

4 Results for the pairwise model with the simple closure

4.1 Background

Using the simple closure for clustered networks (3.7), and writing $\xi = \frac{(n-1)}{n}$, we obtain the following closed pairwise model equations describing an SIR epidemic on a clustered regular network of $N$ individuals with degree $n$:

$$[\dot{S}] = -\tau[SI],$$

$$[\dot{I}] = \tau[SI] - \gamma[I],$$

$$[\dot{SI}] = -(\tau + \gamma)[SI] + \tau\xi \frac{[SS][SI]}{[S]} \left( (1 - \phi) + \phi \frac{N[SI]}{n[S][I]} \right) - \tau\xi \frac{[SI]^2}{[S]} \left( (1 - \phi) + \phi \frac{N[I]}{n[I]^2} \right),$$

$$[\dot{SS}] = -2\tau\xi \frac{[SS][SI]}{[S]} \left( (1 - \phi) + \phi \frac{N[SI]}{n[S][I]} \right).$$

(4.1)

(4.2)

(4.3)

(4.4)
\[ [I] = 2\tau[S] - 2\gamma[I] + 2\tau\xi\frac{[SI]^2}{[S]} \left(1 - \phi + \frac{N[I]}{n[I]^2}\right). \]  

(4.5)

For model equations (4.1)-(4.5), in [12] the basic reproductive ratio \((R_0)\) is considered. Starting from the evolution equations of infectious individuals leads to

\[ [I] = \tau[S] - \gamma[I] \]
\[ = \left(\frac{\beta[S]}{N}C_{SI} - \gamma\right)[I], \]

where \(C_{SI}\) is defined in equation (3.3). Taking into account that \(\tau_n = \beta\) and that initially \([S] \approx N\), in [12] it is claimed that \(R_0 = C_{SI}\beta/\gamma\). It is important to note that this \(R_0\) is not the classical \(R_0\) in the sense of being the expected number of new infections produced by a typical infectious individual when introduced in a fully susceptible population. Rather it can be thought of as a growth-rate-based threshold, and has the same properties as the classical \(R_0\) when both are exactly one. In what follows, we will simply refer to it as \(R\) [3, 13].

Thus in order to determine \(R\) explicitly, the authors in [12] consider the early behaviour of \(C_{SI}\) and find that this variable is given by the ordinary differential equation (ODE)

\[ C_{SI} = -\tau \left(C_{SI} + C_{SI}^2 - n\xi(C_{SI} - C_{SI}^2)(1 - \phi) + n\xi C_{SI}^2 \phi \frac{[I]C_{II}}{N}\right). \]  

(4.6)

The ODE above, (4.6), however depends on the behaviour of \([I]C_{II}/N\) and in [12] it was found that

\[ \frac{[I]C_{II}}{N} \rightarrow \frac{2\tau C_{SI}}{\gamma + \beta C_{SI} - 2\xi\beta C_{SI}^2}\phi. \]  

(4.7)

Considering the quasi-equilibrium of \(C_{SI}\), referred to as \(C_{SI}^*\), in equation (4.6) together with the expression for \([I]C_{II}/N\) in equation (4.7), one finds that \(C_{SI}^*\) is given by

\[ 1 + C_{SI}^* - n\xi(1 - C_{SI}^*)(1 - \phi) + \frac{2\tau n\xi C_{SI}^*}{\gamma + \beta C_{SI}^* - 2\xi\beta C_{SI}^*\phi} = 0. \]  

(4.8)

Hence, \(R\) can be calculated as \(C_{SI}^*\beta/\gamma\), at least numerically. Variables such as \(C_{SI}\) and \(C_{II}\) describe the correlations between the states of neighbouring nodes on the network as the epidemic unfolds and these have been studied numerically in [12].

For model equations (4.1)-(4.5) and when there is no clustering present in the network structure (thus \(\phi = 0\)), a further simplification of equation (4.8) can be achieved [12]. To determine \(R = C_{SI}^*\beta/\gamma\) in this case, simply solve

\[ 1 + C_{SI}^* - n\xi(1 - C_{SI}^*) = 0 \]  

(4.9)

to find \(C_{SI}^* = \frac{n-2}{n}\) and thus \(R = \frac{(n-2)\tau}{\gamma}\).
Unfortunately when $\phi \neq 0$, according to our knowledge, the quasi-equilibrium values can only be determined numerically via equation (4.8). In what follows, we show that by working with two new variables, $\alpha = [SI]/[I]$ and $\delta = [II]/[I]$, which are still closely linked to the correlations formed during the spreading process, it is possible to obtain the epidemic threshold as the solution of a cubic equation and, more importantly, we show that this can be obtained as asymptotic expansion in powers of $\phi$.

### 4.2 Epidemic threshold

Consider the initial phase of an infection invading an entirely susceptible population in the pairwise model, described by equations (4.1)-(4.5). We find that

$$
\dot{I} = \tau[SI] - \gamma[I] = \gamma[I] \left( \frac{\tau[SI]}{\gamma[I]} - 1 \right).
$$

We know the quantity $\gamma[I]$ remains non-negative regardless of time in the epidemic process, and we choose to consider the threshold in terms of $\frac{[SI]}{[I]}$. This leads to $R = \frac{\tau[SI]}{\gamma[I]}$. When $R > 1$ an epidemic will occur, and when $R < 1$ the epidemic will die out. Although we know the values of $\tau$ and $\gamma$, to determine if an epidemic will occur a priori, we require further knowledge about the quantity $\frac{[SI]}{[I]}$ at some initial time close to $t = 0$. While this is similar to the approach taken in [12], we focus on variables such as $\frac{[SI]}{[I]}$ and $\frac{[II]}{[I]}$, and we motivate our choice below. The problem of finding the epidemic threshold can be dealt with in at least two more different but equivalent ways. First, one can carry out a simple linear stability analysis of the disease-free steady state and this is shown in Appendices C and D. Second, the threshold can also be computed as the largest eigenvalue of the next generation matrix, see section 6. However, in both cases, the variables $[SI]/[I]$ and $[II]/[I]$ turn out to play a key role and their values for small times need to determined.

### 4.3 Fast variables with the simple closure

To circumvent the problem of the ill-defined variables above, we exploit the fact that $\alpha = [SI]/[I]$ and $\delta = [II]/[I]$ are fast variables when compared to the time course of the epidemic. Fig. 3 shows clearly that $\alpha$ and $\delta$ are fast compared to the epidemic process and that they quickly converge to a quasi-equilibrium. Hence, at early times $\alpha$ and $\delta$ attain their quasi-equilibrium values, and these are the values that can be used to compute the epidemic threshold.

We continue by deriving differential equations for the variables $\alpha = [SI]/[I]$ and $\delta = [II]/[I]$. Differentiating $\alpha$ and $\delta$ and using equations (4.1)-(4.5) leads to

$$
\frac{d\alpha}{dt} = -\tau \alpha + \tau \xi \alpha \phi - \tau \xi \phi \alpha^2 - \tau \xi \alpha - \tau \alpha^2,
$$

(4.11)
Figure 3: Illustration of the dynamics of prevalence, $[I]/N$, over time ((a)-(b)), compared to that of $\alpha = \frac{[SI]}{[I]}$ ((c)-(d)) and $\delta = \frac{[II]}{[I]}$ ((e)-(f)) for the pairwise model with the simple (left column) and the improved (right column) closures. Parameter values are $N = 10000$, $n = 5$, $\phi = 0.5$ and $\tau = \gamma = 1$. 
\[
\frac{d\delta}{dt} = 2\tau\alpha - \gamma\delta + 2\tau\xi\frac{1}{n}\phi\alpha^2\delta - \tau\alpha\delta.
\] (4.12)

The detailed derivation for equations (4.11) and (4.12) can be found in Appendix A.

### 4.3.1 Fast variables without clustering

When clustering is negligible and hence \(\phi = 0\), we find that

\[
\frac{d\alpha}{dt} = -\tau\alpha + \tau\xi n\alpha - \tau\alpha^2,
\] (4.13)

\[
\frac{d\delta}{dt} = 2\tau\alpha - \gamma\delta - \tau\alpha\delta,
\] (4.14)

where \(\xi = \frac{(n-1)}{n}\). The steady states of the system (4.13)-(4.14) are given by \((\alpha_1^*, \delta_1^*) = (0, 0)\) and \((\alpha_2^*, \delta_2^*) = \left(\frac{n-2}{\gamma + (n-2)}, \frac{2\tau(n-2)}{\gamma} \right)\). Based on equation (4.10), it follows that \(R = \frac{\tau\alpha_2^*}{\gamma} = \frac{\tau(n-2)}{\gamma}\).

### 4.3.2 Fast variables with clustering

When clustering is present in the network, the differential equations for \(\alpha\) and \(\delta\) are more complex and thus steady states are harder to compute. Firstly, we set equation (4.11) equal to zero and rearrange to isolate \(\delta\), finding

\[
\delta = -1 + \xi n(1 - \phi) + \xi \phi\alpha - \alpha.
\] (4.15)

Plugging equation (4.15) into equation (4.12) leads to the following cubic equation in \(\alpha\):

\[
(2\tau\xi\phi(1 - \xi\phi))\alpha^3 + (\tau\xi n\phi - 2\tau\xi^2 n\phi(1 - \phi) - \tau n)\alpha^2 + (-n(\tau + \gamma) + \tau\xi n^2(1 - \phi) + \gamma\xi n\phi)\alpha + (\gamma\xi n^2(1 - \phi) - \gamma n) = 0.
\] (4.16)

The solution of the cubic equation (4.16) provides the steady state(s) of system (4.11)-(4.12), and allows the computation of the threshold via the formula \(R^c = \frac{\tau\alpha^*}{\gamma}\). We note that the steady state in \(\alpha\) has to be biologically plausible. \(\alpha = \frac{\left|S^B\right|}{\left|I\right|}\) restricts the steady state to be positive and to be less than \(n\), since the average number of susceptible neighbours averaged over all infected nodes needs to be less than the average degree.

### 4.4 Asymptotic expansion of the epidemic threshold

The case of \(\phi \neq 0\) can be regarded as a perturbation of the case with no clustering and we thus set out to find \(\alpha\) using a perturbation method. More precisely, we seek to find the
roots of the cubic polynomial, given in equation (4.16), in terms of an asymptotic expansion in powers of \( \phi \), that is
\[
\alpha = \alpha_0 + \phi \alpha_1 + \phi^2 \alpha_2 + \cdots. \tag{4.17}
\]

Plugging (4.17) into equation (4.16) leads to
\[
2\tau \xi \phi (1 - \xi \phi)(\alpha_0 + \phi \alpha_1 + \phi^2 \alpha_2 + \cdots)^3
+ (\tau \xi n \phi - 2\tau \xi^2 n \phi(1 - \phi) - \tau n)(\alpha_0 + \phi \alpha_1 + \phi^2 \alpha_2 + \cdots)^2
+ (-n(\tau + \gamma) + \tau \xi n^2 (1 - \phi) + \gamma \xi n \phi)(\alpha_0 + \phi \alpha_1 + \phi^2 \alpha_2 + \cdots)
+ (\gamma \xi n^2 (1 - \phi) - \gamma n) = 0. \tag{4.18}
\]

Collecting terms of order \( \phi^0 \) in (4.18) and after some algebra we find that \( \alpha_0 \) satisfies the equation below:
\[
n(\alpha_0 - (n - 2))(\tau \alpha_0 - \gamma) = 0. \tag{4.19}
\]
Hence, \( \alpha_0 = (n - 2) \). As expected, this corresponds to the unclustered case. Collecting terms of order \( \phi \) in (4.18), we find a polynomial in terms of \( \alpha_0 \) and \( \alpha_1 \):
\[
2\tau \xi \alpha_0^3 + (\tau \xi n - 2\tau \xi^2 n)\alpha_0^2 + (\gamma \xi n - \tau \xi n^2)\alpha_0 - 2\tau n \alpha_0 \alpha_1 + (\tau \xi n^2 - n(\tau + \gamma))\alpha_1 - \gamma \xi n^2 = 0. \tag{4.20}
\]

Equation (4.20) leads to
\[
\alpha_1 = \frac{\gamma \xi n^2 - 2\tau \xi \alpha_0^3 + (2\tau \xi^2 n - \tau \xi n)\alpha_0^2 + (\tau \xi n^2 - \gamma \xi n)\alpha_0}{\tau \xi n^2 - n(\tau + \gamma) - 2\tau n \alpha_0},
\]
which after substituting \( \alpha_0 = (n - 2) \) and \( \xi = \frac{(n-1)}{n} \) yields
\[
\alpha_1 = \frac{-2(n - 1)}{n^2} \left( \frac{2\tau(n - 1)(n - 2) + \gamma n}{\tau(n - 2) + \gamma} \right). \tag{4.21}
\]

To summarise, we have determined the first two coefficients \( \alpha_0 \) and \( \alpha_1 \) of the asymptotic expansion (4.17) which solves the cubic equation (4.16). Hence, the true solution is approximated by the following expansion:
\[
\alpha = (n - 2) - \phi \frac{2(n - 1)}{n^2} \left( \frac{2\tau(n - 1)(n - 2) + \gamma n}{\tau(n - 2) + \gamma} \right) + \cdots. \tag{4.22}
\]

We make several remarks. First, the epidemic threshold will be given by \( R^c = \tau \alpha / \gamma \). Second, the coefficient of the first order correction of \( \alpha \) can be rearranged in terms of \( R = \frac{n(n-2)}{\gamma} \), the threshold for the case of unclustered networks, leading to
\[
R^c = R - \phi a \frac{\tau}{\gamma} \left( \frac{aR + 1}{R + 1} \right), \tag{4.23}
\]
where $a = 2(n - 1)/n$.

Finally, it is clear that due to the first order correction being negative, we have that

$$R^c = R - \phi a \frac{\tau}{\gamma} \left( \frac{aR + 1}{R + 1} \right) \leq R = \frac{\tau(n - 2)}{\gamma}. \quad (4.24)$$

The goodness of the estimate for $\alpha$ (4.22) is tested by comparing it to the numerical solution of the cubic equation (4.16). This is done in Fig. 4 for five different values of the clustering coefficient. The asymptotic approximation performs well and only breaks down for values of clustering larger than $\simeq 0.3$. From the same figure it is clear that higher values of clustering push the critical $R^c = 1$ curve to higher values of $\tau$ and $n$. Hence, in the presence of clustering a viable epidemic requires either a denser network or a higher transmission rate, noting that the transmission rate and the recovery rate $\gamma$ are not strictly independent.

### 4.5 Numerical examples

In the previous section we have demonstrated that for the pairwise model with the simplest closure for clustered networks, the determination of the epidemic threshold involves the solution of a cubic equation. While this can be done numerically, we presented an asymptotic approximation of the solution in powers terms of powers of the clustering coefficient $\phi$. In Fig. 4 we present a systematic test of the newly determined threshold by comparing the threshold based on the numerical solution of the cubic equation (4.16) (continuous line in the $(\tau, n, 0)$ plane), the asymptotic approximation of the solution to the cubic equation (4.22) (dashed line and markers - ○) and the numerical solution of the full ODE system corresponding to the closed pairwise model (4.1)-(4.5).

The agreement between the explicit numerical solution of the closed pairwise system and threshold based on the numerical solution of the cubic equation is excellent for all clustering values and other parameter combinations. Moreover, the agreement of these results with the threshold based on the asymptotic approximation is also excellent and remains valid for values of $0 \leq \phi \leq 0.3$. Our numerical tests confirm that our analytical results are correct. The initial conditions for the closed pairwise systems were set in the following way: $[I](0) = I_0 = 1$, $[S](0) = N - I_0 = S_0$, $[SI](0) = nI_0 S_0$, $[SS](0) = nS_0 S_0$, and $[II](0) = nI_0 I_0$. The ODEs were run for a sufficiently long time ($T_{\text{max}} = 1000$) to ensure that the epidemic died out. It is worth noting that the correct numerical solution of the cubic equation can be chosen by keeping in mind that $0 \leq \alpha = \frac{|[SI]|}{[I]} \leq n$. 


Figure 4: Assessing the validity of the epidemic threshold based on the asymptotic approximation (4.22) (dashed line and markers - ○) by comparing it to the epidemic threshold based on the numerical solution of the cubic equation (4.16) (continuous lines). In the right hand column we compare both threshold curves in the $(\tau, n, 0)$ plane. In the left hand column both curves are compared to the final epidemic size based on numerical integration of the pairwise model equations with the simple closure. Parameter values are $N = 10000$, $\gamma = 1$ and from top to bottom the clustering coefficients are $\phi = 0, 0.15, 0.3, 0.45, 0.6$. 
5 Results for the pairwise model with the compact improved closure

Starting from the improved closure (3.14) but in line with Proposition 2, we adapt the closure so that the term responsible for the approximation on the clustered part of the network does not consider variables, singles or pairs involving the recovered/removed class. This leads to the new closure

\[
ASI = (n - 1) \left( (1 - \phi) \frac{[AS][SI]}{n[S]} + \phi \frac{[AS][SI][IA]}{[A] \left( \frac{[SS][SI]}{[S]} + \frac{[SI][II]}{[I]} \right)} \right),
\]

which we refer to as the compact improved closure. Plugging equation (5.1) into the exact system (2.2)-(2.6) leads to a self-consistent system that is written out in full in Appendix B.

In line with our procedure so far, we aim to find the epidemic threshold of this new pairwise system with the compact improved closure. It turns out that the approach used for the pairwise system with the simple closure is applicable to this case, and the steps and results are summarised below.

5.1 Fast variables with the compact improved closure

As we have shown before, finding the threshold relies on finding the quasi-equilibrium of \( \alpha = \frac{[SI]}{[I]} \). In Appendix B we show that this requires knowledge about the behaviour of the \( \delta = \frac{[II]}{[I]} \) variable and indeed a system of differential equations involving these two variables can be derived. This system is given below

\[
\frac{d\alpha}{dt} = -\tau \alpha - \tau \alpha^2 + \tau (n - 1) \left( (1 - \phi) \alpha + \phi \alpha \frac{(n - \delta)}{n + \delta} \right), \tag{5.2}
\]

\[
\frac{d\delta}{dt} = 2\tau \alpha - \gamma \delta + 2\tau (n - 1) \left( \frac{\phi \alpha \delta}{n + \delta} \right) - \tau \alpha \delta. \tag{5.3}
\]

As previously, the steady state of this system is of interest and apart from the trivial \((\alpha^*, \delta^*) = (0, 0)\) steady state, the quasi-equilibrium can be found by first expressing \( \delta \) as a function of \( \alpha \). This can be done by setting equation (5.2) equal to zero and rearranging, leading to

\[
\alpha = (n - 2) - (n - 1) \phi \frac{2\delta}{n + \delta}. \tag{5.4}
\]

Plugging equation (5.4) into equation (5.3) and collecting powers of \( \delta \) leads to the following cubic equation

\[
(-A - B)\delta^3 + (-n(n - 2) - A^2 - 2nB)\delta^2
\]
$$+ (-n(n-2)A + 2nA - n^2B)\delta + 2n^2(n-2) = 0,$$

(5.5)

where $A = (n-2) - 2\phi(n-1)$ and $B = \gamma/\tau$. It is worth noting that in this case it is easier to work with $\delta$, but any results can be converted in terms of $\alpha$ which is the main variable of interest.

### 5.2 Asymptotic expansion of the epidemic threshold

As in Section 4.4, we require the roots of the cubic polynomial given in equation (5.5). To do so, we express $\delta$ as an asymptotic expansion in powers of $\phi$. We substitute

$$\delta = \delta_0 + \delta_1 \phi + \delta_2 \phi^2 + \cdots.$$  

(5.6)

Plugging the expansion for $\delta$ (5.6) into equation (5.5) leads to

$$(-A-B)(\delta_0 + \delta_1 \phi + \delta_2 \phi^2 + \cdots)^3 + (-n(n-2) - A^2 - 2nB)(\delta_0 + \delta_1 \phi + \delta_2 \phi^2 + \cdots)^2$$

$$+ (-n(n-2)A + 2nA - n^2B)(\delta_0 + \delta_1 \phi + \delta_2 \phi^2 + \cdots) + 2n^2(n-2) = 0.$$  

(5.7)

Alternatively, substituting (5.4) into the differential equation for $\delta$ (5.3), setting the expression equal to zero and rearranging leads to

$$\gamma \delta (n + \delta)^2 = \tau [(n-2)(n+\delta) - 2\phi(n-1)\delta][(2-\delta)(n+\delta) + 2\phi(n-1)\delta].$$  

(5.8)

Substituting (5.6) into (5.8) and collecting terms of order $\phi^0$ yields

$$\gamma \delta_0 (n + \delta_0)^2 = \tau [(n-2)(n+\delta_0)][(2-\delta_0)(n+\delta_0)]$$

(5.9)

$$\gamma \delta_0 = \tau (n-2)(2-\delta_0)$$

(5.10)

$$\delta_0(\gamma + \tau (n-2)) = 2\tau(n-2)$$

(5.11)

$$\delta_0 = \frac{2\tau(n-2)}{\gamma + \tau(n-2)}.$$  

(5.12)

Following the same process to collect terms of order $\phi^1$, we find

$$\gamma \delta_1 [(n + \delta_0)^2 + 2(n+\delta_0)\delta_0] = \tau(n-2)(n+\delta_0)[\delta_1(2-n-2\delta_0) + 2(n-1)\delta_0]$$

$$+ \tau(2-\delta_0)(n+\delta_0)[(n-2)\delta_1 - 2(n-1)\delta_0],$$

(5.13)

which can be rearranged to yield

$$\delta_1 = \frac{2\tau(n-1)\delta_0(n-4+\delta_0)}{\gamma(n+3\delta_0) + \tau(n-2)(n+3\delta_0-4)}.$$  

(5.14)
with $\delta_0$ defined in (5.12). In summary, we have determined the first two coefficients $\delta_0$ and $\delta_1$ of the asymptotic expansion for $\delta$ given in equation (5.6). Hence, the true solution is approximated by the following expression:

$$\delta = \frac{2\tau(n-2)}{\gamma + \tau(n-2)} + \frac{2\tau(n-1)\delta_0(n-4 + \delta_0)\phi}{\gamma(n+3\delta_0) + \tau(n-2)(n+3\delta_0 - 4)} + \cdots. \tag{5.15}$$

Finally, we are able to plug (5.15) into the quasi-equilibrium point for $\alpha$, given in equation (5.4), to obtain

$$\alpha = (n - 2) - 2(n - 1)\phi \frac{\delta_0}{n + \delta_0} + \mathcal{O}(\phi^2), \tag{5.16}$$

which can be rearranged to find

$$\alpha = (n - 2) - \phi \frac{4\tau(n-1)(n-2)}{\tau(n+2)(n-2) + \gamma n}. \tag{5.17}$$

The expression for $\alpha$ (5.17) can be used to determine the epidemic threshold as follows

$$R_{cci} = \frac{\tau \alpha}{\gamma} = \frac{(n-2)\tau}{\gamma} - \phi \frac{4\tau(n-1)(n-2)}{\tau(n+2)(n-2) + \gamma n}. \tag{5.18}$$

It is straightforward to see that again $R_{cci} \leq R$, with clustering making the spread of the epidemic less likely.

### 5.3 Numerical examples

In Fig. 5 we repeat the systematic test of comparing the epidemic threshold generated via the numerical solution of the cubic equation (5.5), the epidemic threshold generated by the asymptotic expansion (5.18) and the numerical value of the final epidemic size predicted by pairwise model with the compact improved closure, over a wide range of $(\tau, n)$ values. Several observations can be made. First, it is clear that higher values of clustering push the location of threshold to higher $\tau$ and $n$ values, meaning that the limiting effect of clustering on the epidemic spread can only be overcome if either the value of the transmission rate or average degree increases. Second, the agreement between the threshold based on the numerical solution of the cubic equation (5.5) and the asymptotic expansion (5.15) is excellent over a wide range of $\phi$ values. In fact, in this case the agreement is excellent for $0 \leq \phi \leq 0.45$, with only small deviations even for $\phi = 0.6$. The agreement between the numerical solution of the pairwise model and the threshold based on the numerical solution of the cubic equation (5.5) remains excellent across all parameter values.
Figure 5: Assessing the validity of the epidemic threshold based on the asymptotic expansion (5.15) (dashed line and markers - o) by comparing it to the epidemic threshold based on the numerical solution of the cubic equation (5.5) (continuous lines). In the right hand column we compare both threshold curves in the $(\tau, n, 0)$ plane. In the left hand column both curves are compared to the final epidemic size based on numerical integration of the pairwise model equations with the compact improved closure. Parameter values are $N = 10000$, $\gamma = 1$ and from top to bottom the clustering coefficients are $\phi = 0, 0.15, 0.3, 0.45, 0.6$. 
6 Discussion

In this paper we set out to obtain an analytic epidemic threshold using pairwise models but for clustered networks. For the unclustered case this problem has been solved previously [12]. Furthermore, in [12] it was shown that one way to approach the computation of the threshold is to exploit the presence of fast variables. In particular, working out the quasi-steady state of the fast variables allowed the authors to determine the epidemic threshold analytically. However, this was done only for the case when the network is unclustered. Here, we went one step further and showed that the quasi-equilibrium can be found as an asymptotic expansion in powers of the clustering coefficient. Prior to this new result we re-derived known closures by providing extra intuition for the assumptions underlying them as well as for the motivation for deriving them.

Exploiting the presence of fast variables and combining this with elements of perturbation theory allowed us to compute the epidemic threshold for the pairwise model with two different closures that take clustering into account. Our results are in line with the findings of [15] and [18]. In [15], the epidemic threshold in a pairwise model for clustered networks with closure based on the number of links in a motif, rather than nodes, was calculated as

\[ R_0 = \frac{(n-1)\tau}{\tau + \gamma + \tau \phi} \]  

Equation (6.1) can be expanded in terms of \( \phi \) to give

\[ R_0 = \frac{(n-1)\tau}{\tau + \gamma} \left( \frac{1}{1 + \phi \frac{\tau}{\tau + \gamma}} \right) \approx \frac{(n-1)\tau}{\tau + \gamma} \left( 1 - \phi \frac{\tau}{\tau + \gamma} + \cdots \right), \]  

which again reflects our finding that clustering reduces the epidemic threshold.

Similarly but for clustered networks with heterogeneous degree distributions, in [18] it was found that

\[ R_0 = \frac{\langle k^2 - k \rangle}{\langle k \rangle} T - \frac{2\langle n_\Delta \rangle}{\langle k \rangle} T^2 + \cdots, \]  

where \( \langle k^i \rangle \) stands for the \( i \)th moment of the degree distribution, \( T \) is the probability of infection spreading across a link connecting an infected to a susceptible node and \( \langle n_\Delta \rangle \) denotes the average number of triangles that a node belongs to. The expression above again shows that clustering reduces the epidemic threshold when compared to the unclustered case. Furthermore, if the network is regular and we assume that infections and recoveries are Markovian processes with rates \( \tau \) and \( \gamma \) respectively, giving \( T = \tau/(\tau + \gamma) \), \( R_0 \) above reduces to

\[ R_0 = \frac{\tau(n-1)}{\tau + \gamma} - (n-1)\phi \left( \frac{\tau}{\tau + \gamma} \right)^2 + \cdots, \]  

where we have used the fact that a global clustering coefficient of \( \phi \) translates to a node on average being part of \( \frac{1}{2}n(n-1)\phi \) uniquely counted triangles. This in turn coincides
with equation (6.2), and this is perhaps unexpected since the first expression was obtained based on a new type of closure for pairwise models while the other expression was based on percolation theory type arguments. In [32], specific networks with household structure were used to investigate the effects of clustering and infectious period distribution on a modified version of $R_0$ referred to as $R_*$, and lower and upper bounds for the value of this quantity were found.

Our analysis confirms that clustering starves the spreading epidemic of susceptible neighbours and that the epidemic is less likely to spread if the networks are clustered, all other parameters being equal. More importantly, the epidemic threshold is model-dependent and the pairwise model with the compact improved closure leads more readily to epidemic outbreaks when compared to the pairwise model with the simple closure, see Figs. 4-5. While this ordering is true for the parameters used in this paper, it is easy to show that this relation can change if parameters are tuned accordingly. For example, looking at the limit of $\gamma \to 0$ (or $\tau/\gamma$ large limit), the two epidemic thresholds are the same if

$$\frac{2(n-1)(n-2)}{n^2} \left( \frac{2(n-1)(n-2)}{(n-2)} \right) = \frac{4(n-1)(n-2)}{(n-2)(n+2)}. \quad (6.5)$$

After some simple algebra this reduces to $n = 2$. Hence, if the $\tau/\gamma$ ratio is large we will essentially have that (i) if $n > 2$ then $R_0^c < R_0^{cci}$, and (ii) if $n < 2$ then $R_0^c > R_0^{cci}$. This highlights the difficulty of determining the epidemic threshold and emphasises the importance of model choice when modelling real-world epidemics.

The analysis of the pairwise model with the full improved closure is still outstanding and will be the subject of a separate research paper. In this case, we expect that additional fast-variables need to be identified. Intuition tells us that $[SI]$, $[I]$ may need to be extended to include $[SR]$ and $[RI]$. The computation of the true $R_0$ for pairwise models can be attempted by considering the next generation matrix approach [33]. Looking at the pairwise model with the simplest closure and ordering the variables involved in the spreading process as: $[I],[SI]$, the generation of new infectious cases at the disease-free steady state is given by

$$F = \begin{pmatrix} 0 & \tau \\ 0 & \tau(n-1)(1-\phi) + \tau\xi\alpha \phi \end{pmatrix}, \quad (6.6)$$

where the lower right term is obtained from equation (4.3) by looking at the rate of growth of $[SI]$ in terms of $[SI]$ itself, that is

$$[SI] = \tau [SI] + \tau\xi [SS] \left( (1-\phi) + \phi \frac{N [SI]}{n [I]} \right) [SI] \simeq (\tau(n-1)(1-\phi) + \tau\xi\alpha\phi) [SI].$$

Now all other transfers between compartments are summarised in the $V$ matrix, which is given below

$$V = \begin{pmatrix} \gamma & 0 \\ (\tau + \gamma) + \tau\xi\alpha\phi \end{pmatrix}, \quad (6.7)$$
where the lower right term describes the rate at which $[SI]$ pairs move out of this compartment. This is obtained from equation (4.3) by looking at the rate at which $[SI]$ pairs are depleted as shown below

$$\dot{[SI]} = - \left( (\tau + \gamma) + \tau \xi \frac{[SI]}{[S]} (1 - \phi) + \phi \tau \xi \frac{[SI]}{[S]} \frac{N[I]}{n[I]^2} \right) [SI] \simeq - (\tau + \gamma) + \tau \xi \frac{\alpha \delta \phi}{n} [SI].$$

Now $R_0$ is given by the leading eigenvalue of $F^{-1}$, which turns out to be

$$R_0 = \frac{\tau n (n - 1) - \tau (n - 1)(n - \alpha)\phi}{n(\tau + \gamma) + \tau \xi \alpha \delta \phi}.$$  \hspace{1cm} (6.8)

Obviously, this seems like a rather complicated expression since the quasi-equilibrium values for $\alpha$ and $\delta$ are needed. These are only available as asymptotic expansions in powers of $\phi$. Nevertheless, for $\phi = 0$, $R_0 = \frac{\tau (n-1)}{\tau + \gamma}$, which agrees perfectly with the two results quoted above. Now writing $R_0 = r_0 + \phi r_1$, $\alpha = \alpha_0 + \phi \alpha_1$ and $\delta = \delta_0 + \phi \delta_1$, and plugging these into equation (6.8), leads to

$$r_0 = \frac{\tau (n - 1)}{\tau + \gamma} \text{ and } r_1 = - \frac{\tau^2 (n - 1)}{(\tau + \gamma)^2} \left[ \frac{2(\tau + \gamma)}{n\tau} + \frac{(n - 1)}{n} \alpha_0 \delta_0 \right].$$

While the first term in the expansion for $R_0$ agrees with the results quoted above, the second term seems less unlikely to be equivalent to those shown above. This same approach can be used to compute $R_0$ when the compact improved closure is used. We believe that comparing these different ways of computing the epidemic threshold can contribute to reconciling different methods and will lead to more clarity and transparency between various modelling approaches.

The ODE systems for the fast variables are worth investigating in more detail. We expect that these systems will exhibit a number of steady states, some stable and some unstable. Namely, we expect the quasi-steady states to be unstable and the trivial zero steady states to be stable. However, numerical solutions of the cubic polynomials show that other equilibria exist. It will also be worthwhile to compare different models in order to identify the impact of clustering on epidemics by mapping out regions in the parameter space where its effect is strongest. It is known that when the network is dense the effect of clustering is limited and the same holds when the transmission/recovery rates are high/low, respectively. Of course there remains the issue of accounting for degree heterogeneity and this has been addressed to some extent by using percolation type approaches. Using the kind of approach that we presented in this paper may be extended to degree-heterogeneous clustered networks, but this will require more sophisticated models such as effective-degree, or compact/super-compact pairwise models [30]. These will no doubt lead to more complex systems which are more challenging to analyse. However, we hope that the results of this paper may encourage other researchers to consider and tackle the challenges posed
by modelling epidemic dynamics on clustered networks with heterogeneous degree distributions. Finally, it would be worthwhile to test our findings against explicit stochastic network simulations. This was beyond the scope of the present work, whose focus was on exploiting the presence of fast variables and the use of perturbation analysis to determine the epidemic threshold analytically.

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A Derivation of evolution equations for the fast variables with simple closure

We begin by computing differential equations for the variables \( \alpha := \frac{[SI]}{|I|} \) and \( \delta := \frac{[II]}{|I|} \). Differentiating \( \alpha = \frac{[SI]}{|I|} \) gives

\[
\frac{d\alpha}{dt} = \frac{[SI]'[I] - [SI][I]'}{|I|^2} \]

and substituting \([SI]'\) from equation (4.3) and \([I]'\) from equation (4.2), we obtain

\[
\frac{d\alpha}{dt} = - (\tau + \gamma) \frac{[SI]}{|I|} + \tau \xi \frac{[SS][SI]}{|S||I|} \left( 1 - \phi + \frac{N[SI]}{n[S]|I|} \right) - \tau \xi \frac{[SI]^2}{|S||I|} \left( 1 - \phi + \frac{N[II]}{n[I]^2} \right) - \tau \frac{[SI]^2}{|I|^2} + \gamma \frac{[SI]}{|I|}.
\]

Replacing all \( \frac{[SI]}{|I|} \) terms by \( \alpha \) and all \( \frac{[II]}{|I|} \) terms by \( \delta \) gives

\[
\frac{d\alpha}{dt} = - (\tau + \gamma) \alpha + \tau \xi \frac{[SS]}{|S|} \alpha \left( 1 - \phi + \frac{N}{n[S]|S|} \alpha \right) - \tau \xi \frac{[SI]}{|S|} \alpha \left( 1 - \phi + \frac{N}{n[I]|I|} \delta \right) - \tau \alpha^2 + \gamma \alpha
\]

\[
= - \tau \alpha + \tau \xi \frac{[SS]}{|S|} \alpha \left( 1 - \phi + \frac{N}{n[S]|S|} \alpha \right) - \tau \xi \frac{[SI]}{|S|} \alpha \left( 1 - \phi + \frac{N}{n[I]|I|} \delta \right) - \tau \alpha^2
\]

\[
= - \tau \alpha + \tau \xi \frac{[SS]}{|S|} \alpha \left( 1 - \phi \right) + \tau \xi \phi \frac{N[SS]}{n[S]^2} \alpha^2 - \tau \xi \frac{[SI]}{|S|} \alpha \left( 1 - \phi \right) - \tau \xi \frac{N[SI]}{n[S]|I|} \phi \alpha \delta - \tau \alpha^2
\]
\[
\frac{d\alpha}{dt} = -\tau\alpha + \tau\xi \frac{[SS]}{[S]}(1 - \phi)\alpha + \tau\xi\phi \frac{N[SS]}{n[S]^2}\alpha^2 - \tau\xi\frac{[SI]}{[S]}(1 - \phi)\alpha - \tau\xi\frac{N}{n[S]}\phi\alpha^2\delta - \tau\alpha^2
\]

In section 4.2 we considered an epidemic threshold and a condition for stability of the disease-free steady state. In both cases, we consider the state of the system at time zero. At time zero we assume that \([S] = N\), \([SS] = nN\), and \([SI] = 0\), therefore we substitute these values into the differential equation for \(\alpha\) to obtain

\[
\frac{d\alpha}{dt} = -\tau\alpha + \tau\xi n\frac{N}{N}\frac{nN}{nN^2}\alpha^2 - \tau\xi\frac{N}{nN}\phi\alpha^2\delta - \tau\alpha^2
\]

\[
= -\tau\alpha + \tau\xi n(1 - \phi)\alpha + \tau\xi\phi\alpha^2 - \tau\xi\frac{1}{n}\phi\alpha^2\delta - \tau\alpha^2,
\]

where \(\xi = \frac{(n-1)}{n}\). Differentiating \(\delta = \frac{[I]}{[I]}\) gives

\[
\frac{d\delta}{dt} = \frac{[II]'[I] - [II][I]'}{[I]^2} = \frac{[II]'}{[I]} - \frac{[II][I]'}{[I]^2},
\]

and substituting \([II]'\) from equation (4.5) and \([I]'\) from equation (4.2), we obtain

\[
\frac{d\delta}{dt} = 2\tau\frac{[SI]}{[I]} - 2\gamma\frac{[II]}{[I]} + 2\tau\xi\frac{[SI]^2}{[S][I]}\left(1 - \phi + \phi\frac{N[II]}{n[I]^2}\right) - \tau\frac{[SI][II]}{[I]^2} + \gamma\frac{[II]}{[I]}
\]

Replacing all \(\frac{[SI]}{[I]}\) terms by \(\alpha\) and all \(\frac{[II]}{[I]}\) terms by \(\delta\) gives

\[
\frac{d\delta}{dt} = 2\tau\alpha - 2\gamma\delta + 2\tau\xi\frac{[SI]}{[S]}\alpha \left(1 - \phi + \phi\frac{N}{n[I]}\delta\right) - \tau\alpha\delta + \gamma\delta
\]

\[
= 2\tau\alpha - \gamma\delta + 2\tau\xi\frac{[SI]}{[S]}\alpha \left(1 - \phi + \frac{N}{n[I]}\delta\right) - \tau\alpha\delta
\]

\[
= 2\tau\alpha - \gamma\delta + 2\tau\xi\frac{[SI]}{[S]}(1 - \phi)\alpha + 2\tau\xi\frac{N[SI]}{n[S][I]}\phi\alpha^2\delta - \tau\alpha\delta
\]

\[
= 2\tau\alpha - \gamma\delta + 2\tau\xi\frac{[SI]}{[S]}(1 - \phi)\alpha + 2\tau\xi\frac{N}{n[S]}\phi\alpha^2\delta - \tau\alpha\delta.
\]

At time zero we assume that \([S] = N\) and \([SI] = 0\). We substitute these values into the differential equation for \(\delta\) to obtain

\[
\frac{d\delta}{dt} = 2\tau\alpha - \gamma\delta + 2\tau\xi\frac{1}{n}\phi\alpha^2\delta - \tau\alpha\delta.
\]

Combining the differential equations for both \(\alpha = \frac{[SI]}{[I]}\) and \(\delta = \frac{[II]}{[I]}\), we have

\[
\frac{d\alpha}{dt} = -\tau\alpha + \tau\xi n(1 - \phi)\alpha + \tau\xi\phi\alpha^2 - \tau\xi\frac{1}{n}\phi\alpha^2\delta - \tau\alpha^2, \quad (A.1)
\]

\[
\frac{d\delta}{dt} = 2\tau\alpha - \gamma\delta + 2\tau\xi\frac{1}{n}\phi\alpha^2\delta - \tau\alpha\delta. \quad (A.2)
\]
B Derivation of evolution equations for the fast variables with the compact improved closure

Using the improved closure (3.14) in line with Proposition 2, which we refer to as the reduced improved closure, we find that

\[
[ASI] = (n - 1) \left( 1 - \phi \right) \frac{[ASI][SI]}{n[S]} + \phi \frac{[ASI][SI][IA]}{[A]} \sum_a [aS][aI]/[a] \tag{B.1}
\]

\[
= (n - 1) \left( 1 - \phi \right) \frac{[ASI][SI]}{n[S]} + \phi \frac{[ASI][SI][IA]}{[A]} \left( \frac{[SS][SI]}{[S]} + [SI][II] \right). \tag{B.2}
\]

Using equation (B.2) to close the original pairwise equations (2.2)-(2.6), we obtain the following system of equations:

\[
\dot{[S]} = -\tau[SI] \tag{B.3}
\]

\[
\dot{[I]} = \tau[SI] - \gamma[I] \tag{B.4}
\]

\[
\dot{[SI]} = - (\tau + \gamma)[SI] + \tau(n - 1) \left( 1 - \phi \right) \frac{[SS][SI]}{n[S]} + \phi \frac{[I][SS][SI]}{[I][SS] + [S][II]} \tag{B.5}
\]

\[
- \tau(n - 1) \left( 1 - \phi \right) \frac{[SI]^2}{n[S]} + \phi \frac{[S][SI][II]}{[I][SS] + [S][II]} \right)
\]

\[
\dot{[SS]} = -2\tau(n - 1) \left( 1 - \phi \right) \frac{[SS][SI]}{n[S]} + \phi \frac{[I][SS][SI]}{[I][SS] + [S][II]} \tag{B.6}
\]

\[
\dot{[II]} = 2\tau[SI] - 2\gamma[II] + 2\tau(n - 1) \left( 1 - \phi \right) \frac{[SI]^2}{n[S]} + \phi \frac{[S][SI][II]}{[I][SS] + [S][II]} \tag{B.7}
\]

As we have shown in the main body of the paper, the computation of the threshold requires a system of differential equations for the fast variables \( \alpha = [SI]/[I] \) and \( \delta = [II]/[I] \). We find

\[
\frac{d\alpha}{dt} = \frac{[SI]'}{[I]} - \frac{[SI][I]'}{[I]^2}
\]

and substituting \( [SI]' \) from equation (B.5) and \( [I]' \) from equation (B.4), we obtain

\[
\frac{d\alpha}{dt} = - (\tau + \gamma) \frac{[SI]}{[I]} + \tau(n - 1) \left( 1 - \phi \right) \frac{[SS][SI]}{n[S][I]} + \phi \frac{[SS][SI]}{[I][SS] + [S][II]} \tag{B.8}
\]

\[
- \tau(n - 1) \left( 1 - \phi \right) \frac{[SI]^2}{n[S][I]} + \phi \frac{[S][SI][II]}{[I]^2[SS] + [S][II]} - \gamma \frac{[SI]^2}{[I]^2} + \gamma \frac{[SI]}{[I]}
\]
Replacing all $\frac{[SI]}{[I]}$ terms by $\alpha$ and all $\frac{[II]}{[I]}$ terms by $\delta$ gives

\[ \frac{d\alpha}{dt} = - (\tau + \gamma)\alpha + \tau(n - 1) \left( (1 - \phi) \frac{[SS]}{n[S]} \alpha + \phi\alpha \frac{[SS]}{[SS] + [S]\delta} \right) \]

\[ - \tau(n - 1) \left( (1 - \phi) \frac{[SI]}{n[S]} \alpha + \phi\alpha \frac{[S]}{[SS] + [S]\delta} \right) - \tau \alpha^2 + \gamma \alpha, \]

and evaluating $\frac{d\alpha}{dt}$ at the disease-free steady state $([S], [I], [SI], [SS], [II]) = (N, 0, 0, nN, 0)$ (C.1) gives

\[ \frac{d\alpha}{dt} = - (\tau + \gamma)\alpha + \tau(n - 1) \left( (1 - \phi) \alpha + \phi\alpha \frac{nN}{nN + N\delta} \right) \]

\[ - \tau(n - 1) \left( \phi\alpha \frac{N}{nN + N\delta} \right) - \tau \alpha^2 + \gamma \alpha. \]

After simplification we find that

\[ \frac{d\alpha}{dt} = - \tau \alpha + \tau(n - 1) \left( (1 - \phi) \alpha + \phi\alpha \frac{n}{n + \delta} - \phi\alpha \frac{1}{n + \delta} \right) - \tau \alpha^2 \]

\[ = - \tau \alpha + \tau(n - 1) \left( (1 - \phi) \alpha + \phi\alpha \left( \frac{n - \delta}{n + \delta} \right) \right) - \tau \alpha^2. \]

Differentiating $\delta = \frac{[II]}{[I]}$ gives

\[ \frac{d\delta}{dt} = \frac{[II]'}{[I]} - \frac{[II][I]'}{[I]^2}, \]

and substituting $[II]'$ from equation (B.7) and $[I]'$ from equation (B.4), we obtain

\[ \frac{d\delta}{dt} = 2\tau \frac{[SI]}{[I]} - 2\gamma \frac{[II]}{[I]} + 2\tau(n - 1) \left( (1 - \phi) \frac{[SI]^2}{n[S][I]} + \phi \frac{[S][SI][II]}{[I]^2[SS] + [S][I][II]} \right) \]

\[ - \tau \frac{[SI][II]}{[I]^2} + \gamma \frac{[II]}{[I]}. \]

Replacing all $\frac{[SI]}{[I]}$ terms by $\alpha$ and all $\frac{[II]}{[I]}$ terms by $\delta$ gives

\[ \frac{d\delta}{dt} = 2\tau \alpha - 2\gamma \delta + 2\tau(n - 1) \left( (1 - \phi) \frac{[SI]}{n[S]} \alpha + \phi\alpha \frac{[S]}{[SS] + [S]\delta} \right) - \tau \alpha \delta + \gamma \delta \]

\[ = 2\tau \alpha - \gamma \delta + 2\tau(n - 1) \left( (1 - \phi) \frac{[SI]}{n[S]} \alpha + \phi\alpha \frac{[S]}{[SS] + [S]\delta} \right) - \tau \alpha \delta, \]

and evaluating $\frac{d\delta}{dt}$ at the disease-free steady state (C.1) gives

\[ \frac{d\delta}{dt} = 2\tau \alpha - \gamma \delta + 2\tau(n - 1) \left( \phi\alpha \delta \frac{N}{nN + N\delta} \right) - \tau \alpha \delta \]
\[
\begin{align*}
\frac{d\alpha}{dt} &= -\tau\alpha + \tau(n - 1)\left(1 - \phi\right)\alpha + \phi\alpha\left(\frac{n - \delta}{n + \delta}\right) - \tau\alpha^2 \quad \text{(B.16)} \\
\frac{d\delta}{dt} &= 2\tau\alpha - \gamma\delta + 2\tau(n - 1)\left(\frac{\phi\alpha\delta}{n + \delta}\right) - \tau\alpha\delta. \quad \text{(B.17)}
\end{align*}
\]

Combining the differential equations for both \(\alpha = \frac{[S]}{[I]}\) and \(\delta = \frac{[I]}{[I]}\), we have

\[
J_{df} = \begin{pmatrix}
0 & 0 & -\tau & 0 & 0 \\
0 & -\gamma & \tau & 0 & 0 \\
\frac{\partial [S]}{\partial [I]} & \frac{\partial [S]}{\partial [I]} & 0 & \frac{\partial [S]}{\partial [I]} & 0 \\
\frac{\partial [S]}{\partial [I]} & \frac{\partial [S]}{\partial [I]} & 0 & \frac{\partial [I]}{\partial [I]} & 0 \\
0 & 0 & \frac{\partial [I]}{\partial [I]} & \frac{\partial [I]}{\partial [I]} & \frac{\partial [I]}{\partial [I]}
\end{pmatrix},
\]  
(C.2)

An alternative way to determine the epidemic threshold is to consider the stability of the disease-free steady state

\[
([S], [I], [SI], [SS], [II]) = (N, 0, 0, nN, 0). \quad \text{(C.1)}
\]

When the disease-free steady state is stable, the system will always end up at the disease-free steady state and thus no epidemic will occur. When the disease-free steady state becomes unstable, there exists (at least) a second steady state whereby an epidemic will occur and \([S]\) will no longer be equal to \(N\). To determine a stability condition for the disease-free steady state \((C.1)\), we must compute the Jacobian matrix \(J\) of the system \((4.1)-(4.5)\), evaluated at the disease-free steady state, and solve to find its eigenvalues.

By computing partial derivatives of each differential equation \((4.1)-(4.5)\) with respect to each model variable \([S], [I], [SI], [SS]\) and \([II]\), and evaluating each expression at the disease-free steady state \((C.1)\), we obtain

The zero entries in \(J_{df}\) reflect the true values that the respective partial derivatives attain at the disease-free equilibrium. However, the majority of the non-zero matrix entries involve...
\( \frac{[S]}{[I]} \) and \( \frac{[I]}{[I]} \). Since \( [I] = [S] = [I] = 0 \) at the disease-free steady state, both of these quantities are ill-defined. Hence, not all entries of the Jacobian can be evaluated at the equilibrium. This issue prevents the computation of the eigenvalues of \( J_{df} \) and thus the value of the epidemic threshold. In order to progress, we need to determine the correct values for \( \alpha = \frac{[S]}{[I]} \) and \( \delta = \frac{[I]}{[I]} \). We note that the correct value of \( \alpha = \frac{[S]}{[I]} \) is also required in equation (4.10), and the threshold cannot be computed without it.

In fact, using only \( \phi = 0 \), the Jacobian at the disease-free steady state (C.1) becomes

\[
J_{df_{\text{no,clust}}} = \begin{pmatrix}
0 & 0 & -\tau & 0 & 0 \\
0 & -\gamma & \tau & 0 & 0 \\
0 & 0 & -\gamma + \tau(n - 2) & 0 & 0 \\
0 & 0 & -2\tau(n - 1) & 0 & 0 \\
0 & 0 & 2\tau & 0 & -2\gamma
\end{pmatrix}
\] (C.3)

It is straightforward to show that the eigenvalues are given by \( \lambda_1 = 0, \lambda_2 = -\gamma, \lambda_3 = \gamma(n - 2) - \gamma, \lambda_4 = 0 \) and \( \lambda_5 = -2\gamma \). The only eigenvalue that can be non-zero and non-negative is \( \lambda_3 = \gamma(n - 2) - \gamma \). Hence, we know that the disease-free steady state (C.1) is stable when \( \lambda_3 \leq 0 \) and becomes unstable when \( \lambda_3 > 0 \). Thus, the epidemic threshold is given by \( \lambda_3 = 0 \) and this can be rearranged to give \( \tau(n - 2)/\gamma = 1 \). This is equivalent to the calculation based on determining the quasi-equilibrium of the fast variables.

## D Standard linear-stability analysis for the case of the compact improved closure

To determine an epidemic threshold, we consider conditions for stability of the disease-free steady state (C.1). To do so, we compute the Jacobian matrix evaluated at the disease-free steady state as

\[
J_{df} = \begin{pmatrix}
0 & 0 & -\tau & 0 & 0 \\
0 & -\gamma & \tau & 0 & 0 \\
\frac{\partial[S]}{\partial[I]} & \frac{\partial[S]}{\partial[I]} & 0 & \frac{\partial[S]}{\partial[I]} & 0 \\
\frac{\partial[S]}{\partial[I]} & \frac{\partial[S]}{\partial[I]} & \frac{\partial[S]}{\partial[I]} & \frac{\partial[S]}{\partial[I]} & 0 \\
\frac{\partial[I]}{\partial[I]} & \frac{\partial[I]}{\partial[I]} & \frac{\partial[I]}{\partial[I]} & \frac{\partial[I]}{\partial[I]} & \frac{\partial[I]}{\partial[I]}
\end{pmatrix}
\] (D.1)

where \( \frac{\partial[S]}{\partial[I]} = 2\tau(n - 1)/\phi + \phi \cdot n/(n + 2\delta + \gamma) \), \( \frac{\partial[S]}{\partial[I]} = -\tau(n - 1)(1 - \phi + \phi \cdot n/(n + 2\delta + \gamma)) \), \( \frac{\partial[S]}{\partial[I]} = -2\tau(n - 1)(1 - \phi + \phi \cdot n/(n + 2\delta + \gamma)) \), \( \frac{\partial[S]}{\partial[I]} = -2\tau(n - 1)(\phi \cdot n/(n + 2\delta + \gamma)) \), \( \frac{\partial[S]}{\partial[I]} = 2\tau(n - 1)(\phi \cdot n/(n + 2\delta + \gamma)) \), \( \frac{\partial[S]}{\partial[I]} = -2\tau(n - 1)(\phi \cdot n/(n + 2\delta + \gamma)) \), \( \frac{\partial[I]}{\partial[I]} = 2\tau + 2\tau(n - 1)(\phi \cdot n/(n + 2\delta + \gamma)) \) and \( \frac{\partial[I]}{\partial[I]} = -2\gamma + 2\tau(n - 1)(\phi \cdot n/(n + 2\delta + \gamma)) \) cannot be fully evaluated as they contain products of the problematic variables \( \alpha = \frac{[S]}{[I]} \) and \( \delta = \frac{[I]}{[I]} \).
The Jacobian above becomes useful once analytical expressions for $\alpha$ and $\delta$ are obtained (or it could be an asymptotic expansion or even numerical values). Plugging these in the Jacobian will allow to either numerically or analytically compute the threshold. We note that using the linear-stability analysis or focusing on the initial growth rate should lead to the same threshold value, as was already shown for the for the case of the system with the simple closure in Section C.

References

[1] E Cator and P Van Mieghem. Nodal infection in markovian susceptible-infected-susceptible and susceptible-infected-removed epidemics on networks are non-negatively correlated. *Physical Review E*, 89(5):052802, 2014.

[2] Laurent Decreusefond, Jean-Stéphane Dhersin, Pascal Moyal, Viet Chi Tran, et al. Large graph limit for an sir process in random network with heterogeneous connectivity. *The Annals of Applied Probability*, 22(2):541–575, 2012.

[3] Ken TD Eames. Modelling disease spread through random and regular contacts in clustered populations. *Theoretical population biology*, 73(1):104–111, 2008.

[4] Ken TD Eames and Matt J Keeling. Modeling dynamic and network heterogeneities in the spread of sexually transmitted diseases. *Proceedings of the National Academy of Sciences*, 99(20):13330–13335, 2002.

[5] Thilo Gross, Carlos J Dommar D'Lima, and Bernd Blasius. Epidemic dynamics on an adaptive network. *Physical review letters*, 96(20):208701, 2006.

[6] Petter Holme. Three faces of node importance in network epidemiology: Exact results for small graphs. *Physical Review E*, 96(6):062305, 2017.

[7] Thomas House, Geoffrey Davies, Leon Danon, and Matt J Keeling. A motif-based approach to network epidemics. *Bulletin of Mathematical Biology*, 71(7):1693–1706, 2009.

[8] Thomas House and Matt J Keeling. The impact of contact tracing in clustered populations. *PLoS computational biology*, 6(3):e1000721, 2010.

[9] Svante Janson, Malwina Luczak, and Peter Windridge. Law of large numbers for the sir epidemic on a random graph with given degrees. *Random Structures & Algorithms*, 45(4):726–763, 2014.

[10] Brian Karrer and Mark EJ Newman. Message passing approach for general epidemic models. *Physical Review E*, 82(1):016101, 2010.
[11] Brian Karrer and Mark EJ Newman. Random graphs containing arbitrary distributions of subgraphs. *Physical Review E*, 82(6):066118, 2010.

[12] Matthew J Keeling. The effects of local spatial structure on epidemiological invasions. *Proceedings of the Royal Society of London B: Biological Sciences*, 266(1421):859–867, 1999.

[13] Istvan Z Kiss, Luc Berthouze, Timothy J Taylor, and Péter L Simon. Modelling approaches for simple dynamic networks and applications to disease transmission models. *Proc. R. Soc. A*, 468(2141):1332–1355, 2012.

[14] István Z Kiss, Joel C Miller, and Péter L Simon. *Mathematics of Epidemics on Networks*. Springer, 2017.

[15] Jinxian Li, Weiqiang Li, and Zhen Jin. The epidemic model based on the approximation for third-order motifs on networks. *Mathematical biosciences*, 2018.

[16] Jennifer Lindquist, Junling Ma, P Van den Driessche, and Frederick H Willeboordse. Effective degree network disease models. *Journal of mathematical biology*, 62(2):143–164, 2011.

[17] Joel C Miller. Percolation and epidemics in random clustered networks. *Physical Review E*, 80(2):020901, 2009.

[18] Joel C Miller. Spread of infectious disease through clustered populations. *Journal of the Royal Society Interface*, pages rsif–2008, 2009.

[19] Joel C Miller and Istvan Z Kiss. Epidemic spread in networks: Existing methods and current challenges. *Mathematical modelling of natural phenomena*, 9(2):4–42, 2014.

[20] Joel C Miller, Anja C Slim, and Erik M Volz. Edge-based compartmental modelling for infectious disease spread. *Journal of the Royal Society Interface*, 9(70):890–906, 2012.

[21] Joel C Miller and Erik M Volz. Model hierarchies in edge-based compartmental modeling for infectious disease spread. *Journal of mathematical biology*, 67(4):869–899, 2013.

[22] Mark EJ Newman. Random graphs with clustering. *Physical review letters*, 103(5):058701, 2009.

[23] Romualdo Pastor-Satorras, Claudio Castellano, Piet Van Mieghem, and Alessandro Vespignani. Epidemic processes in complex networks. *Reviews of modern physics*, 87(3):925, 2015.
[24] Romualdo Pastor-Satorras and Alessandro Vespignani. Epidemic dynamics and endemic states in complex networks. *Physical Review E*, 63(6):066117, 2001.

[25] DA Rand. Correlation equations and pair approximations for spatial ecologies. *Advanced ecological theory: principles and applications*, 100, 1999.

[26] Prapanporn Rattana, Konstantin B Blyuss, Ken TD Eames, and Istvan Z Kiss. A class of pairwise models for epidemic dynamics on weighted networks. *Bulletin of mathematical biology*, 75(3):466–490, 2013.

[27] Martin Ritchie, Luc Berthouze, and Istvan Z Kiss. Beyond clustering: Mean-field dynamics on networks with arbitrary subgraph composition. *Journal of mathematical biology*, 72(1-2):255–281, 2016.

[28] Kieran J Sharkey, Carmen Fernandez, Kenton L Morgan, Edmund Peeler, Mark Thrush, James F Turnbull, and Roger G Bowers. Pair-level approximations to the spatio-temporal dynamics of epidemics on asymmetric contact networks. *Journal of mathematical biology*, 53(1):61–85, 2006.

[29] Neil Sherborne, Joel C Miller, Konstantin B Blyuss, and Istvan Z Kiss. Mean-field models for non-markovian epidemics on networks. *Journal of mathematical biology*, 76(3):755–778, 2018.

[30] Péter L Simon and Istvan Z Kiss. Super compact pairwise model for sis epidemic on heterogeneous networks. *Journal of Complex Networks*, 4(2):187–200, 2015.

[31] András Szabó-Solticzky, Luc Berthouze, Istvan Z Kiss, and Péter L Simon. Oscillating epidemics in a dynamic network model: stochastic and mean-field analysis. *Journal of mathematical biology*, 72(5):1153–1176, 2016.

[32] Pieter Trapman. On analytical approaches to epidemics on networks. *Theoretical population biology*, 71(2):160–173, 2007.

[33] Pauline Van den Driessche and James Watmough. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical biosciences*, 180(1-2):29–48, 2002.

[34] Erik M Volz, Joel C Miller, Alison Galvani, and Lauren Ancel Meyers. Effects of heterogeneous and clustered contact patterns on infectious disease dynamics. *PLoS computational biology*, 7(6):e1002042, 2011.