Stochastic spreading processes on a network model based on regular graphs

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Abstract. The dynamic behaviour of stochastic spreading processes on a network model based on k-regular graphs is investigated. The contact process and the susceptible-infected-susceptible model for the spread of epidemics are considered as prototype stochastic spreading processes. We study these on a network consisting of a mixture of 2- and 3-fold coordinated randomly-connected nodes of concentration \( p \) and \( 1 - p \), respectively, with \( p \) varying between 0 and 1. Varying the parameter \( p \) from \( p = 0 \) (3-regular graph of infinite dimension) to \( p = 1 \) (2-regular graph - 1D chain) allows us to investigate their behaviour under such structural changes. Both processes are expected to exhibit mean-field features for \( p = 0 \) and features typical of the directed percolation universality class for \( p = 1 \). The analysis is undertaken by means of Monte Carlo simulations and the application of mean-field theory. The quasi-stationary simulation method is used to obtain the phase diagram for the processes in this environment along with critical exponents. Predictions for critical exponents obtained from mean-field theory are found to agree with simulation results over a large range of values for \( p \) up to a value of \( p = 0.95 \), where the system is found to sharply cross over to the one-dimensional case. Estimates of critical thresholds given by mean-field theory are found to underestimate the corresponding critical rates obtained numerically for all values of \( p \).

Keywords: Network Epidemics, SIS Model, Contact Process, Critical Exponents

1 Introduction

The spread of epidemics poses a threat to biological populations as well as to computer networks and investigations into its dynamics and mechanisms are therefore of great current interest. One common class of epidemic models considers individuals to be in one of two possible states: susceptible (S) or infected (I). In this paper, we consider both the Contact Process (CP) [1] and the SIS model, two models of disease propagation via nearest neighbour contact, in which
a disease is passed on to healthy nearest neighbours stochastically at a rate $\lambda$ specific to the model while infected sites spontaneously recover at rate $\epsilon$.

These Markovian spreading processes have attracted wide attention in the past due to their applicability to phenomena as diverse as autocatalytic chemical reactions, spreading of rumours and transport in disordered media [2]. As the rates $\lambda$ and $\epsilon$ are varied, an epidemic will be in one of two distinct states: an invasive regime (active state) in which it is present with a non-zero probability of ultimate survival and one in which this probability is zero thus leading to a state which allows no further evolution because the disease has died out (absorbing state).

These two regimes are known to be connected by a continuous phase transition thereby rendering them of conceptual interest for investigations into this kind of critical phenomenon of non-equilibrium statistical mechanics (see [3] for a review). The critical behaviour for these models in one-, two- and three-dimensional lattices has been investigated very accurately [4] and is found to be characteristic of the Directed Percolation (DP) universality class. From a range of studies, critical thresholds for the phase transition as well as critical exponents of predicted power-law scaling relations are known to high precision.

With the growing interest in complex networks among the statistical physics community in recent years [5, 6], the question of the behaviour of dynamic processes on such topologically disordered structures has arisen [7, 8]. Particularly motivated by the fact that networked structures are ubiquitous in nature, the effects of these environments on, for example, the spread of a disease are of immediate interest. In a series of papers [9–13], the behaviour of the CP and the SIS model on a range of networks has been considered and even comparisons with data of computer virus outbreaks have been attempted [11]. As networks in general are infinite-dimensional objects, the dynamical mean-field (MF) approximation is expected to become exact in these cases in principle rendering many models tractable by analytical means. Both Monte Carlo (MC) simulations and the MF approximation have been used in previous investigations and produced such astonishing results as the absence of an epidemic threshold infection rate for infinite scale-free networks [11].

In this paper, we propose to investigate the behaviour of the CP and the SIS model as two paradigmatic stochastic spreading processes on networks of k-regular graph topology. The model network considered in this investigation consists of a mixture of 2- and 3-fold coordinated randomly-connected nodes of concentration $p$ and $1 - p$, respectively. Varying the parameter $p$ from $p = 0$ to $p = 1$ transforms the system from a 3-regular graph of infinite dimension to a 2-regular graph, i.e. a 1D chain. While both the CP and the SIS model are expected to exhibit mean-field features for $p = 0$, the processes effectively take place in a one-dimensional environment for $p = 1$ which is a very well-studied regime of the DP universality class. It is our aim to investigate the behaviour of both the critical rates and accessible critical exponents for this crossover from an infinite- to a one-dimensional case thereby probing the validity of the MF approximation in this setting. The analysis is undertaken by means of Monte
Carlo simulations using the quasi-stationary (QS) simulation method [14] and the application of mean-field theory [10].

This paper is structured as follows. Section 2 outlines the definitions and some properties of the processes considered. The MF approximation and the QS simulation method are described in section 3. We present and discuss our results in section 4 and summarise our findings in section 5.

2 Background

As outlined in the previous section, both the CP and the SIS model are simple toy models for the spread of an infectious disease by nearest-neighbour contact. In these models defined on a network, nodes represent susceptible or infected individuals surrounded by their neighbours connected via links along which the epidemic may spread. Proliferation of the disease to nearest neighbour sites happens at a transmission rate $\lambda$ while recovery is spontaneous at rate $\epsilon$ making the sequence of events an individual can cycle through Susceptible $\rightarrow$ Infected $\rightarrow$ Susceptible.

The CP and the SIS model are very similar, the difference being the exact mechanism of the spreading of infection. In the case of the CP, a site attempts to transmit its disease at rate $\lambda/k$ to a randomly selected neighbour where $k$ denotes the number of nearest neighbours. If the selected neighbour is already infected, proliferation fails. For the SIS model, transmission to any non-infected neighbour happens, in contrast, at rate $\lambda$ independent of the connectivity of the nodes. Thus, the spreading mechanism in the CP effectively compensates for the local connectivity present in the network through a suitable reduction of the spreading rate through a particular link.

Once suitable initial states for all sites have been chosen, the above rules dynamically evolve the spread of a disease in the network. A typical initial condition is the state of a fully-infected system from which the system relaxes very quickly. For very long times, and formally as time $t \rightarrow \infty$ and for an infinite number of sites in the network $N \rightarrow \infty$, the system is expected to be in one of two states: An active state in which there remains a finite density of infected sites or an absorbing state in which the disease has died out and that therefore admits no further time evolution. Depending on the value of the transmission and recovery rates $\lambda$ and $\epsilon$, ultimately the system will be in one of the two possible states. More precisely, there exists a continuous phase transition between these regimes as one fixes one of the rates and varies the other. This transition takes the system from a phase where the density of infected sites (order parameter) $\rho$ is zero to one where it continuously grows from zero as the transmission rate (control parameter, assuming $\epsilon$ fixed) is increased.

Without loss of generality, one can perform a rescaling of time and set one of the two rates to unity for convenience. In the following, the recovery rate is assumed to be $\epsilon = 1$ and the critical point is therefore characterised by a critical transmission rate $\lambda_c$ alone.
There exist a range of well-established scaling relations for various observables in these models of which we present those relevant for this investigation. The density of infected sites in the thermodynamic limit as $t \to \infty$, the order parameter, is expected to scale as

$$\lim_{t \to \infty} \langle \rho(t) \rangle = \mathcal{P} \sim |\lambda - \lambda_c|^\beta$$

(1)

thereby defining the order parameter critical exponent $\beta$ where $\langle \ldots \rangle$ denotes averaging over realisations of the process. Order parameter fluctuations are known to follow

$$V = N \left( \rho^2 - \langle \rho^2 \rangle \right) \sim |\lambda - \lambda_c|^{-\gamma}$$

(2)

Both the models under consideration are known to belong to the directed percolation (DP) universality class [2]. Accordingly, the critical exponents defined above are those characteristic of this universality class. Above the upper critical dimension, $d_u = 4$, of these models, fluctuations are expected to be Gaussian and MF theory should be exact. Therefore, these processes taking place in infinite-dimensional networks are expected to exhibit exponents predicted by mean-field theory.

3 Methods

3.1 Mean-Field Approximation

Both the CP and the SIS model can be described by the master equation which reflects the conservation of probability flow [3]. In the following, we will first outline the case of the SIS model and then consider the extension to the simpler case of the CP.

In the dynamical MF approximation, which neglects density fluctuations and statistical correlations between the densities at different sites, and, for the moment, disregarding the structure of the network completely, the master equation for the SIS model takes the form

$$\partial_t \rho(t) = -\rho(t) + \lambda k (1 - \rho(t)) \rho(t)$$

(3)

where $\rho(t)$ denotes the density of infected sites at time $t$ averaged over realisations of the process which is identical to the probability of a site of the system to be infected at time $t$. This equation describes the rate of change of the average density in the network which is equal to the flow of density into and out of any site with time and makes for the destruction and the creation terms above. The destruction term due to the vanishing of infection at unit rate is proportional to the density $\rho(t)$. The creation term is due to the possible infection by infected neighbouring sites in the case that the vertex under consideration is not infected. Accordingly, it is proportional to the probability that a site is not infected, $(1 - \rho(t))$, the probability that a neighbouring site is infected $\rho(t)$, the local connectivity $k$ and the spreading rate $\lambda$. 
The master equation (3) can be extended in order to take into account the structure of the underlying network at the level of the node degree (connectivity) distribution (as developed by Pastor-Satorras and Vespignani [9]). It is clear that, unless one assumes a homogeneous network with \( \langle k \rangle = k \) for all \( k \), the expression will decouple into a set of equations for the densities of infected vertices characterised by a certain connectivity \( k \). We can write for each \( k \),

\[
\partial_t \rho_k = -\rho_k + \lambda k (1 - \rho_k(t)) \Theta_k(t),
\]

(4)

where \( \Theta_k(t) \) is the probability that an edge emanating from a vertex of degree \( k \) is connected to an infected site. The infection term as described above now incorporates the probability that a site of degree \( k \) is connected to an infected vertex \( \Theta_k(t) \). One can interpret \( \Theta_k(t) \) as the mean density of neighbouring infected nodes and consequently \( k \Theta_k(t) \) as the mean number of infected nearest neighbours [9].

Networks which are Markovian are statistically described by their degree distribution \( P(k) \) and the conditional probability \( P(k'|k) \) that an edge of a node of degree \( k \) is connected to a vertex of degree \( k' \). For such systems, we can write

\[
\Theta_k(t) = \sum_{k'} P(k' | k) \rho_{k'}(t)
\]

(5)

where the sum runs over all degrees \( k' \). For uncorrelated networks, which we will exclusively consider in this investigation, this becomes [7]

\[
\Theta(t) = \sum_{k'} \frac{k' P(k')}{\langle k \rangle} \rho_{k'}(t)
\]

(6)

which does not depend on \( k \) any longer. Substituting this expression into the rate equation (4) and imposing stationarity (\( \partial_t \rho_k = 0 \)) one obtains

\[
\rho_k = \frac{k \lambda \Theta}{1 + k \lambda \Theta}
\]

(7)

where \( \rho_k \) and \( \Theta(\lambda) \) are the time-independent values for the mean density at sites of degree \( k \) and the mean density of infected neighbouring sites. Multiplying by \( P(k) \) and summing over \( k \) yields

\[
\frac{1}{\lambda} = \frac{1}{\langle k \rangle} \sum_k \frac{P(k) k^2}{1 + k \lambda \Theta} = f(\lambda \Theta)
\]

(8)

where \( f(x) \) is a monotonically decreasing function of \( x \). This equation only has a (unique) solution for \( \Theta(\lambda) \) different from zero for \( \lambda > \lambda_c \) where \( \lambda_c \) is the threshold value for the transmission rate that makes \( \Theta \) smallest. As by definition the mean density of infected nearest neighbours \( \Theta \geq 0 \) in the active regime and \( f(x) \) is a monotonically decreasing function we have (effectively setting \( \Theta = 0 \))

\[
\lambda_c^{SIS} = \frac{1}{f(0)} = \frac{\langle k \rangle}{\langle k^2 \rangle}
\]

(9)
for the critical threshold [15].

In order to obtain an expression for the order parameter we start by combining equations (6) and (7) and arrive at a self-consistency equation for $\overline{\Theta}$ (equivalent to equation (8))

$$\overline{\Theta} = \sum_k k P(k) \frac{k \overline{\Theta}}{\langle k \rangle} \frac{k \lambda \overline{\Theta}}{1 + k \lambda \overline{\Theta}}$$

(10)

that can in principle be solved for $\overline{\Theta}$ which in turn allows one to obtain the order parameter in the MF approximation from

$$\rho = \sum_k \rho_k$$

(11)

Critical exponents can be extracted from MF theory by considering the leading behaviour of the relevant expressions. For example, combining the last expression Eq. (11) and Eq. (7) and expanding in $\lambda - \langle k \rangle / \langle k^2 \rangle$ in analogy to the scaling form $\rho \sim (\lambda - \lambda_c)^\beta$, one obtains the MF value for the order parameter exponent $\beta = 1$. Similarly, one obtains $\gamma = 0$ for the corresponding fluctuations.

In the case of the CP where the effective spreading rate is inversely proportional to the number of links connected to an infected site, Eq. (5) has to be modified and reads

$$\Theta_k(t) = \sum_{k'} P(k'|k) \rho_{k'}(t)$$

(12)

which for uncorrelated networks leads to $\Theta^{nc}(t) = \rho(t)/\langle k \rangle$ where $\rho$ is the average density of infected sites averaged over degrees. Following the procedure as for the SIS model, the critical threshold rate is found to be degree independent and given by [12]

$$\lambda_c^{CP} = 1$$

(13)

while the critical exponents are identical to those for the SIS model.

### 3.2 Monte Carlo Simulation: Quasi-Stationary Simulation

Both processes under consideration can be simulated effectively via time-dependent Monte Carlo simulations. Once initial conditions have been chosen, the system is evolved according to the appropriate rules with a simulation that selects possible events according to their prescribed rates and ensures that they happen in exponentially-distributed time intervals.

In contrast to lattices, networks are characterised by the existence of long range links. This implies that a dynamical process will very strongly feel the size of the system in a finite representation of the network leading to much stronger finite-size effects than experienced in lattices. While the most precise method of determining the critical point in lattices is spreading from a single seed while
ensuring that the infection never reaches the boundary of the system, this is virtually impossible in networks. Thus, finite-size effects have to be systematically exploited in order to make a prediction about the infinite system using networks of different sizes. This is possible using the finite-size scaling hypothesis which predicts that values of observables in systems of size $L$ are controlled by the ratio $L/\xi_{\perp}$, where $\xi_{\perp}$ is the spatial correlation length. In order to use this scaling behaviour, one requires a stationary average value of these observables and analyses their values for different system sizes. Problematically, due to the existence of the absorbing state no such true stationary state exists in a finite system. Fortunately, the processes under consideration evolve such that some observables attain quasi-stationary (QS) values.

Most notably, the density of infected sites averaged over surviving realisations of the process $\langle \rho(t) \rangle$ exhibits this behaviour after an initial transient starting from the initial state of a fully-infected system [2]. This quasi-stationary density $\overline{\rho}$ is expected to scale systematically in accordance with the finite-size scaling hypothesis. According to recent investigations into the finite-size scaling behaviour above the upper critical dimension [16, 13] the prediction is

$$\overline{\rho} \sim L^{d\beta/2} G \left( L^{d/2}(\lambda - \lambda_c) \right) = N^{\beta/2} G \left( N^{1/2}(\lambda - \lambda_c) \right)$$

(14)

where $L^d = N$, the number of sites in the system and $G(x)$ an appropriate scaling function. A similar expression with $\beta$ replaced by $\gamma$ is valid for the associated fluctuations.

In principle the QS state can be investigated via a conventional simulation in which the system is stochastically evolved in time from a fully-infected initial condition. The density of infected sites conditioned on survival can then be analysed and a temporal average over the duration of the QS state is an estimator for $\overline{\rho}$. This method is however plagued by a range of problems [17] which led de Oliveira and Dickman to propose a simulation method which samples the QS state directly [18].

In this QS simulation method, the absorbing state is eliminated and its probability weight is redistributed over the active states according to the history of the process. It can then be shown that the true stationary state of the resulting modified process corresponds to the quasi-stationary state of the original one. This method is ideally suited for our study as a single realisation of the network is investigated in one QS simulation run enabling us to analyse sample-to-sample fluctuations between realisations and find a critical point by use of the scaling relation Eq. (14).

4 Results and Discussion

4.1 MF Solution

The mean-field theory for the CP and the SIS model on networks can be applied to our network with two types of nodes of connectivity $k_1$ and $k_2$ present with
probabilities $P(k_1)$ and $P(k_2)$ respectively. For the SIS model, the self-consistency equation, Eq. 10, for the stationary value of $\Theta$, the average density of infected nearest neighbours, takes the form

$$\frac{\langle k \rangle}{\lambda} = \frac{k_1^2 P(k_1)}{1 + k_1 \lambda \overline{\sigma}} + \frac{k_2^2 P(k_2)}{1 + k_2 \lambda \overline{\sigma}}$$

which can be solved for $\overline{\sigma}$ giving

$$\overline{\sigma} = \frac{1}{2 \lambda} \left( \lambda - \frac{k_1 + k_2}{k_1 k_2} \right) + \frac{1}{\lambda} \sqrt{\frac{1}{4} \left( \lambda - \frac{k_1 + k_2}{k_1 k_2} \right)^2 + \frac{\langle k^2 \rangle \lambda - \langle k \rangle}{k_1 k_2 \langle k \rangle}}$$

where $\overline{\sigma}$ is only defined for transmission rates $\lambda > \lambda_c = \frac{\langle k \rangle}{\langle k^2 \rangle}$ as explained above. Using the definition of the average stationary density $\rho = \sum_k P(k) \rho_k$ and finally substituting $P(k_1) = p$ and $P(k_2) = 1 - p$, the order parameter in the MF approximation is given by

$$\overline{\rho} = \lambda \overline{\sigma} \left( \frac{p k_1}{1 + k_1 \lambda \overline{\sigma}} + \frac{(1 - p) k_2}{1 + k_2 \lambda \overline{\sigma}} \right)$$

The critical exponents given by the leading order contributions of the relevant expressions are found to be the standard MF exponents [12] as outlined above. This solution is plotted for the special case $k_1 = 2$ and $k_2 = 3$ with $p$ varying from 0 to 1 in Fig. 1. As expected, MF theory qualitatively reproduces the
features of the phase transition: There exists a critical rate $\lambda_c$ below which the stationary density is zero while it grows continuously for values above. For the CP, an analogous analysis yields a similar solution.

No direct comparison with numerical predictions for the order parameter is shown in Fig. 1 because of severe finite-size effects for networks which shift the simulation curve well above its true asymptotic position. We will however compare the critical threshold rate as well as some critical exponents in the next section.

4.2 Simulation Results

![Fig. 2. The critical threshold $\lambda_c$ obtained from MF theory (lines) and MC simulation (circles and squares) for the CP (circles and solid line) and the SIS model (squares and dashed line) for various values of $p$. Inset shows the critical exponents $\beta$ (circles) and $\gamma$ (squares) as a function of $p$.](image)

The critical thresholds $\lambda_c$ for a range of values of $p$ for both the CP and the SIS model were obtained via the QS simulation method outlined above. We used networks of sizes ranging from $N = 256 – 32768$ in QS simulation runs up to $10^8$ time steps averaging over no less than 100 and up to a maximum of 1000 network realisations (the latter were required to minimise errors in light of strong sample-to-sample fluctuations for large values of $p$). The resulting critical thresholds are shown in Fig. 2 along with the MF predictions obtained from Eqs. (9) and (13). As expected from the definition of the two models, the CP threshold exceeds the one of the SIS model for a particular value of $p$ which can be attributed to the
reduction of the effective transmission rate by the local coordination number as in Eq. (12). Also, in the two cases of homogeneous connectivity, \( p = 0 \) and \( p = 1 \), the thresholds for the two models are expected to be simply related by a factor of 3 and 2 (equal to the connectivity), respectively, as can be seen from Fig. 2. Note that the critical threshold for the SIS model in the quasi one-dimensional case \((p = 1)\), \( \lambda_c = 1.65 \), is almost identical to the threshold for the CP on the 3-regular random network \((p = 0)\) \( \lambda_c = 1.63 \).

Turning to the MF predictions in comparison to the MC results, one notes that they underestimate the true critical thresholds for all values of \( p \) and for both models. The difference between the MF approximation and the simulation results is more pronounced for the CP as compared to the SIS model.

![Fig. 3. The density of infection \( \rho \) (upper panel) and the corresponding fluctuations \( V = N (\bar{\rho}^2 - \rho^2) \) (lower panel) for various network sizes for the case \( p = 0.5 \) in the SIS model. Solid lines are best-fit regression lines to the scaling forms defined in Eq. (14).](image)

The exponents \( \beta \) and \( \gamma \) were obtained by fitting data for the density of infected sites \( \rho \) and the corresponding fluctuations \( V = N (\bar{\rho}^2 - \rho^2) \) to the finite-size scaling form of Eq. (14). A typical set of data points is shown in Fig. 3 for the case \( p = 0.5 \) for the SIS model along with best-fit regression lines. Both quantities as a function of network size \( N \) show power-law behaviour with the expected MF exponents \( \beta/2 = 0.5 \) and \( \gamma = 0 \) indicating the validity of the MF approximation for this case. These values of critical exponents are plotted in the inset of Fig. 2. As \( p \) is further increased, strong sample-to-sample fluctuations for values beyond \( p = 0.95 \) render a precise analysis very complicated. For \( p = 1 \),
the well-established 1D finite-size scaling exponents are recovered ($\beta/\nu_\perp = 0.253$ for $\rho(N)$ and $\gamma/\nu_\perp = 0.498$ for $V(N)$ [2]) as can be seen from the figure. In the transition region, error bars for exponents are large and our results give slight preference to the scenario of a discontinuous change in exponent values. However, we feel that a very rapid yet continuous change of exponents towards the 1D values cannot be excluded.

5 Conclusion

We have investigated the CP and the SIS model, two paradigmatic stochastic spreading processes, in a network model which interpolates smoothly between an infinite-dimensional 3-regular random network and a linear chain through the variation of a single parameter $p$. The MF approximation yields a prediction for the critical threshold rate of an epidemic outbreak and critical exponents associated with the corresponding absorbing state phase transition. For no value of $p$ does MF theory predict the true critical threshold as calculated from MC simulations. The predictions for critical exponents agree perfectly with simulations for a very wide range of $p$ up to $p = 0.95$. Beyond this point, the analysis is complicated by strong sample-to-sample fluctuations. For $p = 1$ one recovers the established exponents for the one-dimensional case indicating a sudden crossover. While not being able to investigate the nature of this transition precisely, our simulations favour the scenario of a discontinuous change in the scaling exponents which reflects the abrupt change of the dimensionality of the network.

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