Spreading of infectious diseases on complex networks with non-symmetric transmission probabilities

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Abstract

We model the spread of a SIS infection on Small World and random networks using weighted graphs. The entry $w_{ij}$ in the weight matrix $W$ holds information about the transmission probability along the edge joining node $v_i$ and node $v_j$. We use the analogy between the spread of a disease on a network and a random walk performed on this network to derive a master equation describing the dynamics of the process. We find conditions under which an epidemic does not break out and investigate numerically the effect of a non-symmetric weight distribution of the initially infected individual on the dynamics of the disease spread.

Keywords: epidemic models, random walks, Small World networks, SIS model, complex networks
1 Introduction

The study of epidemiological models has been a subject of great interest for many years. The aim is to model the spread of a particular infectious disease, reproducing the actual dynamics of the disease and designing strategies to control and possibly eradicate the infection. Several approaches to tackle this problem have been undertaken. The majority of epidemic models are based on a compartmental model in which the individuals are grouped according to their disease status [1], [2]. The basic models describe the number of individuals that are susceptible to \((S(t))\), infected with \((I(t))\) and recovered from \((R(t))\) a particular disease at time \(t\). The difference in responses between individuals, the influence of the topological structure of the system and many other complex aspects of the progression of the disease are neglected in this approach. Although the simplicity of the model means a loss of information and reality, it enables us to get a first glimpse of the inner workings of the dynamics of the disease spread and makes calculations of threshold values and equilibria possible. The assumptions of this model lead to two standard sets of differential equations that have provided the foundation of the majority of mathematical epidemiology:
1) The Susceptible-Infectious-Removed (SIR) model:

\[
\begin{align*}
\frac{dS}{dt} &= \beta N - \lambda S - \rho S \\
\frac{dI}{dt} &= \lambda S - \delta I - \rho I \\
\frac{dR}{dt} &= \delta I - \rho R
\end{align*}
\]

(1)

2) The Susceptible-Infectious-Susceptible (SIS) model:

\[
\begin{align*}
\frac{dS}{dt} &= \delta I - \lambda S \\
\frac{dI}{dt} &= \lambda S - \delta I,
\end{align*}
\]

(2)

where \( N \) is the population size, \( \beta \) is the birth rate, \( \rho \) is the natural death rate, \( \delta \) is the recovery rate and \( \lambda \) is the infection rate. The SIR model is a suitable model for infectious diseases that confer lifelong immunity, for example measles or whooping cough. The SIS model is mainly used to model the spread of sexually transmitted diseases, such as chlamydia or gonorrhoea, where repeated infections are common. In these models a random mixing assumption is made: each individual has a small and equal chance of coming into contact with any other individual. Many modifications to this basic approach have been made to account for more heterogeneities. One approach is to further subdivide the population into subpopulations, with different mixing rates in these groups. This means that the parameter \( \beta \) in the above equations is replaced by a matrix, describing the transmission of infection between different groups. Nevertheless,
the random mixing assumption, at least within the subgroups, remains unchanged. In reality, however, it is usually the case that the number of contacts of an individual is much smaller than the population size and random mixing does not occur so that the above model can only serve as a relatively crude approximation. Models that incorporate network structure avoid the need to rely on the random mixing assumption. They do so by assigning each person a fixed amount of contacts. Networks thus capture the permanence of interactions. A network (or graph) is comprised of a set of nodes and a set of edges. An edge is a connection (or bond) that links two nodes. Not all nodes in a network are connected directly by one edge. Nodes that are connected by one edge are called neighbors. We introduce following network quantities:

- $V = \{v_i\}$, the set of nodes.
- $E = \{e_{ij}\}$, the set of edges. $e_{ij}$ is the edge running from node $v_i$ to node $v_j$.
- $k_i$, the degree of node $v_i$, i.e., the number of neighbors of node $v_i$.
- $< k >$, the average degree of the nodes.
- $k_{\text{max}}$, the maximum degree found in the network.
- $P(k)$, the degree distribution, i.e., $P(k)$ is the percentage of nodes in the network that have degree $k$.

In a network used to model the spread of a SIS infection, the nodes represent individuals that are either infected by or susceptible to the disease under con-
sideration. Edges represent interactions between individuals: The disease can only spread from one node to the next if there is an edge connecting them. If we assign a weight \( \varrho_{ij} \), representing the probability of node \( v_i \) to infect node \( v_j \), to each edge \( e_{ij} \), we obtain a weighted graph. The weight matrix \( \mathbf{W} = \{ w_{ij} \} \) is defined as follows:

\[
w_{ij} = \begin{cases} 
\varrho_{ij}, & \text{if node } v_i \text{ is a neighbor of node } v_j \\
0, & \text{else.}
\end{cases}
\]

This matrix gives information about the network topology, the relations between individuals in it and the characteristic of the disease under consideration.

The use of networks in Mathematical Epidemiology has grown exponentially since the middle of the 20th century. In recent times, starting with the work of Pastor-Satorras and Vespignani, there has been a burst of activity on understanding the effects of the network topology on the rate and pattern of the disease spread \([3]-[7]\). Amongst others the main network types studied are the following:

1) Regular lattices

In regular lattices each vertex is connected to its \( k \) nearest neighbors, to form either rings (one-dimensional) or grids (two-dimensional) \([8]\).

2) Random graphs

The term random graph refers to the disordered nature of the arrangements
of links between different nodes. Erdős and Rényi (ER), in their first paper, proposed a model to generate random graphs with N nodes and K links [9]. Starting with N disconnect nodes, these random graphs are generated by connecting couples of randomly selected nodes, prohibiting multiple connections, until the number of edges is K.

An alternative model for ER graphs is created by connecting each pair of nodes with probability $0 < p < 1$. This procedure results in graphs having different amount of edges present but the two models show strong similarities and coincide in the limit of large N. random graphs have degree distribution approximately Poisson with parameter $< k >$.

3) Small World networks

The study of dynamical processes over real networks has pointed out the existence of shortcuts, i.e. links that connect different areas of the graph, thus speeding up the communication between otherwise distant nodes. This is known as the Small World property. It is mathematically characterized by a relatively short average path length that depends at most logarithmically on the network size. This property is observed in a variety of real networks including random graphs. To distinguish between random and Small World networks, the Small World property is often associated with the presence of clustering. Watts and Strogatz have proposed to define Small World networks as networks having both, a short average path length (like random graphs) and a high clustering
coefficient [10].

4) Scale free networks

Networks that have a power law degree distribution: \( P(k) \sim k^{-\gamma}, \) \( 2 < \gamma < 3 \) are called scale free [11]. In comparison, in a random graph, \( P(k) \) decays faster than exponentially. In scale free networks we often find a significant amount of nodes with very high degree. In the context of disease spread on a graph, these nodes are aptly called super spreaders. In scale free networks the average degree \( < k > \) is no longer a relevant variable and one expects fluctuations in \( < k^2 > \) to play an important role.

In their work, Shirley and Rushton investigated how the speed of the disease spread is influenced by certain topological characteristics of the graph [12]. They found that an epidemic spreads fastest on a scale free network, followed by random graphs and is slowest on regular lattices. Small World graphs lay in between random and regular graphs. In their model, the transmission probability between nodes is homogeneous throughout the network, i.e., \( w_{ij} = w \). However, when modelling the spread of a disease on a network one should, along with complex topological features, take into account heterogeneity in the intensity strength between nodes: not every individual is susceptible to infection or capable to infect its neighbors to the same degree. The complexity in the capacity and intensity of the connections also plays an important role in other real networks like scientific collaboration networks, air-transportation networks,
internet clusters and other large infrastructure systems. Newman showed that weighted networks can in many cases be analyzed by using a simple mapping from the weighted graph to an unweighted multi graph and then applying standard techniques for unweighted networks [13]. Simonsen et al. studied diffusion on unweighted and weighted networks and discovered that the eigenvalues of the transfer matrix describing the process can be used to recover large scale topological features of the system [14]–[15]. Most recently, Vasquez introduced a type-network representation of a Small World weighted graph to take into account the population heterogeneity in a very general approach [16]. He obtained a recursive equation for the probability distribution of the outbreak size as a function of time and demonstrated that the expected outbreak size and its progression in time are determined by the largest eigenvalue of a certain matrix (reproductive number matrix) and the characteristic distance between individuals.

In this work, we focus on the effect of heterogeneities in the transmission probabilities on the dynamics of the disease spread. We use the strong analogy between the spread of a disease on the network and a random walk performed on that network to derive a master equation describing the dynamics of the process. We find conditions under which an epidemic does not break out and investigate numerically the effect of a non-symmetric weight distribution on the dynamics of the disease spread.

This paper is organized as follows: In section 2, the epidemic models (SIS) is
described. Section 3 contains the derivation of the master equation describing
the dynamic process. Section 4 is concerned with identifying conditions that
prevent the outbreak of an epidemic. In section 5, we discuss our numerical
simulations and their results. We close with an overview of the main results
and a discussion in section 6.

2 The model

In this paper we will consider the standard SIS epidemic models without birth
and death on a Small World graph.

2.1 The epidemic model

At each time $t$, the population is divided into two categories: susceptibles,
$S(t) \geq 0$, and infectious, $I(t) \geq 0$. We normalize so that $S(t) + I(t) = 1$
for all times $t$. Susceptible members are virgin territory for the disease, whilst
infectious members are both infected and capable of infecting others with whom
they are in direct contact, i.e., their neighbors. After being infected for time
$\tau_{Inf}$, an individual returns to the susceptible class. In this paper we take
$\tau_{Inf} = 1$.

2.2 The Network

In our networks, the vertices of the graph represent the individuals of the pop-
ulation under consideration, the edges describe the contact patterns between
individuals. The adjacency matrix $A$ of this network is defined by
\[ a_{ij} = \begin{cases} 
1, & \text{if node } v_i \text{ and } v_j \text{ are joined by an edge} \\
0, & \text{else} 
\end{cases} \]

and gives information about neighbor relations in the network. Assigning a weight \( w_{ij} \) to the edge connecting node \( v_i \) and node \( v_j \), representing the probabilities for the disease to spread from node \( v_i \) to node \( v_j \), will result in a weighted graph giving information about

1. The network topology describing the population and the relations between individuals in it and

2. The characteristics of the disease under consideration.

This information is encoded in the weight matrix \( \mathbf{W} \), defined in the previous section.

The weights play the important role of conveying susceptibility and transmissibility levels of individuals. For example, consider nodes \( v_i \) and \( v_j \) with \( w_{ij} \) being relatively large in comparison to \( w_{ji} \). This could either mean that node \( v_i \)'s capability to transmit the disease is very large and \( v_j \)'s very low or that node \( v_i \) is not very susceptible to transmission of the disease from node \( v_j \) while \( v_j \) can easily catch the disease from \( v_i \).
3 Random Walks and Epidemics

In this section we will describe the model implemented to simulate the spread of an infectious disease throughout a population and derive a master equation governing the dynamics of the system. The design of the model was inspired by the work of Alves et al [17]. There is an strong analogy between the model of an epidemic on a network and a random walk performed on this network that can be described as follows:

Suppose we want to follow the spread of an SIS epidemic on a social network. The individuals of the network are represented as nodes. At each node we place a certain amount of random walkers. Each random walker represents the possibility of an infection to happen: If a walker moves along the edge $e_{ij}$ from the infected node $v_i$ to the susceptible node $v_j$, node $v_j$ has been infected with the disease. If no walker moves to $v_j$, the node remains susceptible. The probability of the walker moving from $v_i$ to $v_j$ is given by the entry $w_{ij}$ of the weight matrix $W$. There is an artificial component to this model: The number of walkers placed at each node at time $t_0$ depends on the length of our experiment and the infection time $\tau_{inf}$. Since each walker represents the possibility of one infection, we need to have a sufficient amount of walkers at each node at time $t_0$ so that we do not ’run out’ of walkers (the ability to infect) at some time $t$ before the end of our experiment. If we want to model the spread of a SIS infection for $T_{max}$ time steps, we need to place at least $\left\lceil \frac{T_{max}}{\tau_{inf}+1} \right\rceil k_j$ walkers at node $v_j$. This is to ensure that, when the node is infected, an infection can
happen along each bond emanating from our node throughout the duration of the experiment.

Let us define following quantities:

- Let $N = T_{max} \sum_j k_j$ be the total number of random walkers participating. (Here, the sum is taken over all nodes.)
- Let $W = \{w_{ij}\}$ be the matrix of transmission probabilities described earlier: $w_{ij}$ is the probability of infected individual $v_i$ to infect susceptible individual $v_j$.
- Let $I(t) = \{I_i(t)\}$, where

$$ I_i(t) = \begin{cases} 1 & \text{if } v_i \text{ is infected} \\ 0 & \text{else.} \end{cases} $$

$I(t)$ is called the infection matrix. The number of non-zero elements of $I(t)$ is the total number of infected individuals at time $t$.

We model the spread of the infection through the population via a random walk performed on the network:

At $t = t_0$, we place $T_{max}k_i$ walkers at each node $v_i$. Each walker represents the possibility of an infection to happen. Choosing $T_{max}k_i$ walkers (and hence $N = T_{max} \sum_j k_j$) ensures that no node looses the ability to infect its neighbors (provided the node is infected) before the maximum number of time steps $T_{max}$ (i.e., the end of the experiment) is reached. At each time step $t$, at a generic node $v_i$, one of two things may happen:
1. If node $v_i$ is infected: $k_i$ walkers may each make one move: The walkers are allowed to move between adjacent vertices. What edge, out of the possible outgoing ones, a walker chooses to move along is picked at random with probability equal to the weight assigned to this directed edge. If the walker moves to a susceptible neighboring node, that node will be infected with the disease. Walkers from infected neighboring nodes may enter. After one time step the status of $v_i$ is reset to susceptible ($I_i(t-1) = 1, I_i(t) = 0$).

2. If node $v_i$ is susceptible: No walkers leave $v_i$ but walkers from neighboring infected nodes may enter. As soon as a walker enters the susceptible node $v_i$, it is considered infected ($I_i(t - 1) = 0, I_i(t) = 1$). At the next time step $k_i$ walkers of node $v_i$ may move to infect any of the susceptible neighbors of node $v_i$.

Let us denote by $\eta_i(t)$ the percentage of walkers at node $v_i$ at time $t$. This is the quantity that will give us information about the dynamics of the disease spread. We note following properties of $\eta_i(t)$:

- If node $v_i$ is susceptible at time $t$, $\eta_i(t + 1) - \eta_i(t) \geq 0$ and the larger this difference is, the more susceptible to infection is the node.

- If node $v_i$ is infected at time $t$, walkers are leaving but may also enter from neighboring nodes so $\eta_i(t + 1) - \eta_i(t)$ does not give any useful information.

- The change in walker density from time $t$ to time $t + 1$ for the susceptible
node $v_i$ satisfies:

$$0 \leq |\eta_i(t + 1) - \eta_i(t)| \leq \sum_{m \in \text{Neigh},} \frac{k_m}{\sum_j k_j}.$$ 

If

$$0 \leq |\eta_i(t + 1) - \eta_i(t)| = \sum_{m \in \text{Neigh},} \frac{k_m}{\sum_j k_j},$$

all neighboring nodes are infected and all walkers from those nodes move to $v_i$.

- The change in walker density from time $t$ to time $t+1$ for the infected node $v_i$ satisfies:

$$0 \leq |\eta_i(t + 1) - \eta_i(t)| \leq \frac{k_i}{\sum_j k_j}.$$ 

If

$$0 \leq |\eta_i(t + 1) - \eta_i(t)| = \frac{k_i}{\sum_j k_j},$$

all $k_i$ walkers leave node $v_i$ and no walkers enter.

- Hence, for a generic node $v_i$, we have

$$0 \leq |\eta_i(t + 1) - \eta_i(t)| \leq \sum_{m \in \text{Neigh},} \frac{k_{\text{max}}}{\sum_j k_j} + \frac{k_{\text{max}}}{\sum_j k_j}$$

$$= \frac{1}{\sum_j k_j} [k_{\text{max}}^2 + k_{\text{max}}]$$

(3)

- Nodes that are extremely susceptible to infection at time $t$ have large $|\eta_i(t + 1) - \eta_i(t)|$ values $(\gg \frac{k_i}{\sum_j k_j})$. Infected nodes that satisfy $|\eta_i(t + 1) - \eta_i(t)| \approx \frac{k_i}{\sum_j k_j}$, are very infectious. Hence $|\eta_i(t + 1) - \eta_i(t)|$ quantifies the extend to which node $v_i$ participates in the spread of the disease under consideration.
• Since the infection time $\tau_{inf}$ is one and $|\eta_i(t+1) - \eta_i(t)| \leq \sum_{m \in N_i} \frac{k_m}{\sum_j k_j}$, for $T_{max} \geq 3$, we have \(^1\):

$$\eta_i(T_{max}) \leq \frac{1}{\sum_m k_m} \left[ (T_{max} - \left[ \frac{(T_{max} - 3)}{2} \right]) k_{max} + \left[ \frac{(T_{max} - 2)}{2} \right] (k_{max})^2 \right]$$

and for $T_{max} \geq 1$:

$$\frac{1}{\sum_m k_m} \left[ (T_{max} - \left[ \frac{T_{max}}{2} \right]) k_{max} + \left[ \frac{T_{max}}{2} \right] \right] \leq \eta_i(T_{max})$$

• From the derivation of these bounds, we can conclude that a generic node $v_i$ satisfies:

$$\frac{1}{\sum_m k_m} \left[ (t - \left[ \frac{t}{2} \right]) k_{max} + \left[ \frac{t}{2} \right] \right] \leq \eta_i(t) \leq \frac{1}{\sum_m k_m} \left[ (t - \left[ \frac{(t - 3)}{2} \right]) k_{max} + \left[ \frac{(t - 2)}{2} \right] (k_{max})^2 \right]$$

• Nodes with large $\eta_i(T_{max})$ values have been infected with great intensity but have transmitted the disease with low frequency.

• Nodes with small $\eta_i(T_{max})$ values have rarely been infected themselves but if infected have contracted the disease to many of their neighbors.

• $\sum_i \eta_i(t) = 1$ for all $t$.

3.1 The Master Equation

We now derive the equation governing the dynamics of the disease spread. This equation is called the master equation. The change in the walker density is the difference between the relative number of walkers entering, $J_i^-(t)$, and leaving, $J_i^+(t)$, the same vertex over the time interval $t \rightarrow t + 1$. Hence,

\(^1\)See appendix A and B
\[ \eta_i(t + 1) = \eta_i(t) + J_i^-(t) - J_i^+(t). \] (4)

For the moment, let us presume that node \( v_i \) is infected. Then the edge current on the directed edge from vertex \( v_i \) to a neighboring vertex \( v_j \) is given by:

\[
C_{ij}(t) = \frac{k_i}{\sum_j k_j \sum_{m \in \text{Neigh}_i} w_{im}} \frac{\eta_i(t_0)}{w_{ij}} \frac{\eta_i(t)}{T_{\text{max}}} \frac{\sum_{m \in \text{Neigh}_i} w_{im}}{w_{ij}}.
\] (5)

The edge current is the fraction of walkers moving along this edge according to the weight distribution emanating from that node. Note that \( \frac{\eta_i(t)}{T_{\text{max}}} \frac{\sum_{m \in \text{Neigh}_i} w_{im}}{w_{ij}} \) is the probability of a walker deciding on the edge from vertex \( v_i \) to vertex \( v_j \).

Since no walkers are leaving if the node is susceptible, we must have:

\[ J_i^-(t) = \sum_{j \in \text{Neigh}_i} I_j(t) C_{ji} \] (6)

and

\[ J_i^+(t) = \sum_{j \in \text{Neigh}_i} I_i(t) C_{ij}. \] (7)

and upon substitution into equation (4), we obtain
\[ \eta_i(t + 1) = \eta_i(t) + \sum_{j \in \text{Neigh}_i} I_j(t)C_{ji} - \sum_{j \in \text{Neigh}_i} I_i(t)C_{ij} \]

\[ = \eta_i(t) + \sum_{j \in \text{Neigh}_i} I_j(t)\frac{\eta_j(t_0)}{T_{\text{max}}} \frac{w_{ji}}{\sum_{m \in \text{Neigh}_j} w_{jm}} - I_i(t)\frac{\eta_i(t_0)}{T_{\text{max}}}. \quad (8) \]

With the matrix \( T \) defined as follow:

\[ T_{ij} = \begin{cases} \frac{w_{ji}}{\sum_{m \in \text{Neigh}_j} w_{jm}} & \text{if } v_j \text{ is a neighbor of } v_i \\ 0 & \text{else,} \end{cases} \]

we can rewrite equation (8) in matrix notation.

\[ \eta(t + 1) = \eta(t) + I(t) \cdot T \frac{\eta(t_0)}{T_{\text{max}}} - I(t) \cdot \frac{\eta(t_0)}{T_{\text{max}}} \]

\[ = \eta(t_0) + \frac{1}{T_{\text{max}}} (\sum_{\tau = t_0}^t I(\tau)) \cdot T \eta(t_0) - (\sum_{\tau = t_0}^t I(\tau)) \cdot \eta(t_0) \]

\[ = \eta(t_0) + \frac{1}{T_{\text{max}}} (\sum_{\tau = t_0}^t I(\tau)) \cdot (T \eta(t_0) - \eta(t_0)). \quad (9) \]

Equation (9) is the desired master equation. This equation governs the dynamics of the disease spread. We can see that \( \eta(t + 1) \) depends on the initial walker distribution, the sum of the infection matrices from time \( t_0 \) to \( t \), which tells us which nodes have not been infected yet and the matrix \( T \), which is called the transfer matrix.
4 Conditions for non-outbreak

If $\eta(t + 1) \approx \eta(t_0)$ for all $t$, an outbreak of the epidemic does not take place. From equation (9) we can see that this is the case if either

1. $\sum_{\tau=t_0}^{t} I(\tau) \approx 0$

or

2. $T\eta(t_0) \approx \eta(t_0)$.

In this paper we will concentrate on the first condition. We investigate how the distribution of incoming and outgoing weights of the initial node affects the dynamics of the disease spread. We define a weighted difference $D_i$ and a nodal entropy $S_i$ to quantify the difference between incoming and outgoing transmission probabilities of a generic node $v_i$. There are two ways in which condition 1 can be realized:

1) The epidemic does not break out: either non or relatively few neighbors of the initially infected node get infected by it and they, in turn, do not infect a significant number of their neighbors until, after a short number of time steps, there are no infected individuals in the population. This happens if the majority of the transmission probabilities stay below the threshold value of the network.

2) Infection of susceptible individuals takes place over a long period of time but infected individuals stay localized around the initially infected node.

To investigate when this may happen, we introduce the following difference measure: For node $v_i$, we define
\[ D_i = \frac{\sum_{j \in \text{Neigh}_i} (w_{ij} - w_{ji})}{k_i}, \]
i.e., \( D_i \) describes the difference between outgoing \((w_{ij})\) and incoming \((w_{ji})\) transmission probabilities of node \( v_i \). If \( D_i < 0 \), the sum of the outgoing weights is smaller than the sum of incoming weights and vice versa if \( D_i > 0 \). Nodes with \( D_i < 0 \) are more likely to be infected often but infect their neighbors with low intensity. Nodes with \( D_i > 0 \) are more infectious but less susceptible to infection. We expect some sort of localization of the disease spread around the initial node \( v_1 \) to occur if \( D_1 \ll 0 \) and the average of the weights of the other nodes are sufficiently small. Let us also define an entropy describing the diversity of the differences of incoming and outgoing weights of a node:

Let

\[ P_i(d) = \% \text{ of edges } e_{ij} \text{ of node } v_i \text{ with } w_{ij} - w_{ji} = d. \]

Then \( P_i(d) \) is a probability distribution on the differences of incoming and outgoing weights of node \( v_i \) (i.e., \( \sum_d P_i(d) = 1 \) for all \( i \)) and we utilize this distribution by defining the following nodal entropy in the standard way: At node \( v_i \), we let

\[ S_i^D = -\sum_d P_i(d) \log(P_i(d)). \]
$S_i^D$ gives information about the disorder of these differences: The larger the value of $S_i^D$, the large the variety of differences in incoming and outgoing weights.

5 Numerical investigation on a Small World Graph

5.1 The network

We create a Small World graph in the following way: $N$ points lying in the square $[0, 1] \times [0, 1]$ are randomly selected. These points represent our population. We choose the following network defining quantities:

- $r$: The short distance radius
- $p_r$: The probability of short distance bond formation
- $R$: The long distance radius
- $p_R$: The probability of long distance bond formation
- $w_r$: The transmission probability along short distance bonds.
- $w_R$: The transmission probability along short distance bonds.

Points lying a distance less than $r$ away from each other are connected with probability $p_r$. Points lying a distance more than $R$ away from each other are connected with probability $p_R$. Transmission of the disease along short distance bonds occurs with probability $p_r$, transmission along long distance bonds occurs with probability $p_R$. 

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5.2 Sensitivity to initial conditions

To see when $\sum_{\tau=t_0}^{t} I(\tau) \approx 0$, let us set up our Small World graph as follows:

The population size $N$ is 2000. All transmission probabilities across bonds, regardless of the nature of the bond, except for node one, are held fixed at 3% (i.e., $p_r = p_R$). This value is chosen because our numerical simulations with homogeneous transmission probabilities showed that an epidemic does rarely break out for values around 3%. We will look at two different scenarios to evaluate the influence of long distance connections in the network:

1. $p_R$ is chosen small enough so that the average long distance degree of a node lies at 0.09 (Small world graph).

2. $p_R = 0$, no long distance connections are permitted (random graph).

$p_r$ is chosen so that the average short distance degree is 25. Node one, the initially infected, has degree $k_1 = 20$ with 19 short distance connections and one long distance connection in case one and 20 connections in case 2. We define the participation ratio $PR(t)$ at time $t$ as the percentage of the population that has been infected at least once during the time interval $[0, t]$ and the diameter $Diam(t)$ as the maximum distance from the initially infected node where an infected individual can be found during the time interval $[0, t]$. We investigate how the diameter and the participation ratio change with varying $D_1$ and $S_1^P$ values, i.e. how the non-symmetry of the transmission probabilities of the initial node and the extend to which it is not symmetric effect the dynamics of the epidemic. The diameter and participation ratio are measured at the last time
step $T_{max}$. $T_{max}$ is chosen large enough so that either the epidemic has spread through all the population, resulting in a maximal diameter and participation ratio, an equilibrium is reached (participation ratio and diameter approach some limit) or the epidemic has died out for some $t < T_{max}$. Our numerical simulations resulted in figure number 1.

We can see that the plots look very similar for graphs without long distance connections and Small World graphs. As expected, the introduction of long distance connections between nodes drives up the diameter and participation ratio.

We note that the plots of the diameter and participation ratio in both cases are almost symmetric about the line $D_1 = 0$. For small values of $S_1^D$ we observe a basin around $D_1 = 0$: diameter and participation ratio stay low as we hoped. But note that, as $S_1^D$ increases, the behavior around $D_1 \approx 0$ becomes increasingly erratic and unpredictable. As we move away from $D_1 \approx 0$ regions but stay in areas where $S_1^D$ is relatively low, the diameter increases.

We can see that the infection reaches the outskirts of our domain $[0, 1] \times [0, 1]$. participation ratios climb as high as 40%. Again, as $S_1^D$ increases we notice the smoothness of the plots breaking and see disorder taking over. Looking at the participation ratio plots we notice an oddity: At $D_1 \approx 0.4$ and $S_1^D \approx 0.65$ we see a sharp spike much higher than any surrounding participation ratio values. This behavior is surprising and needs further investigation.
6 Results and Discussion

We have used the analogy between the spread of a disease on a network and a random walk performed on this network to derive a master equation describing the dynamics of the process. We found two conditions under which an epidemic does not break out. One of these conditions strongly depends on the transmission probabilities of the initial node. This leads to the consideration of a non-symmetric weight matrix \( W \). The majority of research concerning epidemic modeling on networks assumes that the transmission probability matrix \( W \) is symmetric. This means that node \( v_i \) infects neighboring node \( v_j \) with the same probability as node \( v_j \) infects node \( v_i \). We have focused on a more realistic setting where we take into account the heterogeneity in the intensity strength between nodes, i.e., a non-symmetric weight matrix \( W \). In particular, we focus on the initially infected individual showing these heterogeneities, leaving all other nodes with homogeneous transmission probabilities. We chose these small enough so that in case of a fully homogeneous network an epidemic would not break out. We numerically investigated the effect of the non-homogeneous weight distribution of the initial node. To quantify the heterogeneity in transmission probabilities, we defined two nodal quantities:

1. The difference \( D_i = \frac{\sum_{j \in \text{Neigh}_i} (w_{ij} - w_{ji})}{k_i} \), describes the difference between outgoing \( (w_{ij}) \) and incoming \( (w_{ji}) \) transmission probabilities of node \( v_i \).

2. The entropy \( S_i^D = -\sum_d P_i(d) \log(P_i(d)) \), where

\[
P_i(d) = \% \text{ of edges } e_{ij} \text{ of node } v_i \text{ with } w_{ij} - w_{ji} = d,
\]
describing the disorder in the differences $d$.

Through our numerical simulations we obtained surface profiles of diameter and participation ratio for a variety of $D_1$ and $S_1^D$ values for graphs with long distance connections and without. Graphs without long distance connections are just random graphs. Introduction of long distance connections creates Small World graphs. We obtained the following results:

- The quasi-symmetry around $D_1 \approx 0$ for low $S_1^D$ values shows that regardless of whether incoming weights of node 1 are larger than outgoing or vice versa, the dynamics of the disease spread is very similar.

- An increase in $S_1^D$ values leads to a breakdown of symmetry and unpredictable dynamics. This breakdown of symmetry occurs at $S_1^D$ values about 0.5.

In summary, we have shown that the introduction of even very few non-symmetric transmission probabilities along edges of an otherwise homogeneous network changes the dynamic of the disease spread. For a larger disorder in the differences between incoming and outgoing weights of the initially infected, the dynamics becomes unpredictable. The threshold value of this disorder measured by the entropy $S_1^D$ lies at about 0.5. It is quite apparent that a realistic model of the spread of an infectious disease throughout a population should take into consideration the heterogeneities in transmission probabilities between individuals. We have taken a step towards this direction by investigating the effect of the introduction of a single node with heterogeneous transmission probabilities.
Further research opportunities present themselves. One should investigate the effect of heterogeneities in more than one node. One should try and find a measure quantifying the extend of anti-symmetry in the network as a whole and investigate if there exist certain threshold values determining if an epidemic breaks out or not. We also plan to analyze the second condition for a non-outbreak found in this paper. This will involve a thorough investigation of the properties of the transfer matrix $T$. Another direction of research would be to run the simulation on scale-free networks to see if we find the same symmetry about $D_1 = 0$, if a breakdown of this symmetry occurs and if so, what the $S_1^D$ threshold value is.

Network analysis in the context of epidemic modelling has helped us create more realistic settings to investigate the spread of infectious diseases throughout a population. The study of non-symmetric weight matrices may prove useful in creating even more realistic models taking into account the variability of transmission probability between individuals of the population and could lead to more accurate threshold values predicting the outbreak of an epidemic, hence enabling us to develop better methods to prevent or eradicate the disease at hand.

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7 Appendix

7.1 A

Derivation of lower bound

\[
\frac{1}{\sum_m k_m} \left[ (T_{\text{max}} - \left\lfloor \frac{T_{\text{max}}}{2} \right\rfloor) k_{\text{max}} + \frac{T_{\text{max}}}{2} \right] \leq \eta_i(T_{\text{max}})
\]

First, we make some observations:

\(\tau_{inf} = 1\) means that a generic node \(v_i\) can be infected by its neighbor at most every second time step. Since a node can only loose walkers when it is infected and node \(v_{in}\) is the only infected individual at time \(t_0\), we conclude that \(v_{in}\) is the node that potentially has the least number of walkers at time \(T_{\text{max}}\). We also need to assume that node \(v_{in}\) is of maximum degree \(k_{\text{max}}\). The following chain of events will result in node \(v_{in}\) having the smallest number of walkers at the end of the experiment:

- **Time** \(t_0\): \(v_{in}\) is infected, \(v_j\) is susceptible.
  \[\eta_{in}(t_0) = T_{\text{max}} \frac{k_{\text{max}}}{\sum_m k_m}.\]

- **Time** \(t_1\): All \(k_{in} = k_{\text{max}}\) walkers leave \(v_{in}\).
  \[\eta_{in}(t_1) = (T_{\text{max}} - 1) \frac{k_{\text{max}}}{\sum_m k_m},\]
  \(v_{in}\) turns susceptible.

- **Time** \(t_2\): To keep loosing walkers at the largest rate, the node needs to be infected at every second time step. We assume that only one walker

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causes the infection at each time, keeping the amount of walkers entering at a minimum.

\[ \eta_{in}(t_2) = (T_{\text{max}} - 1) \frac{k_{\text{max}}}{\sum_m k_m} + \frac{1}{\sum_m k_m}. \]

- When infected, the node looses all \( k_{in} \) walkers in the next time interval.

\[ \eta_{in}(t_3) = (T_{\text{max}} - 2) \frac{k_{\text{max}}}{\sum_m k_m} + \frac{1}{\sum_m k_m}. \]

- When susceptible, one walker enters, causing infection.

\[ \eta_{in}(t_4) = (T_{\text{max}} - 2) \frac{k_{\text{max}}}{\sum_m k_m} + 2 \frac{1}{\sum_m k_m} \ldots \]

- Hence, for \( t = T_{\text{max}} \), we obtain:

\[ \eta_{in}(T_{\text{max}}) = \frac{1}{\sum_m k_m} \left[ (T_{\text{max}} - \left\lfloor \frac{T_{\text{max}}}{2} \right\rfloor) k_{\text{max}} + \left\lceil \frac{T_{\text{max}}}{2} \right\rceil \right]. \]

7.2 B

**Derivation of upper bound**

\[ \eta(T_{\text{max}}) \leq \frac{1}{\sum_m k_m} \left[ (T_{\text{max}} - \left\lfloor \frac{(T_{\text{max}} - 3)}{2} \right\rfloor) k_{\text{max}} + \left\lceil \frac{(T_{\text{max}} - 2)}{2} \right\rceil (k_{\text{max}}^2) \right] \]

Let \( v_j \) be a neighbor of the initially infected individual \( v_{in} \). If we assume that \( v_j \) and all its neighbors are of maximum degree \( k_{\text{max}} \) and notice that only walkers sitting at infected nodes can move, we conclude that the most walkers can accumulate at a node \( v_j \), neighbor to \( v_{in} \). The following chain of events will result in \( v_j \) obtaining the largest number of walkers over the time span of the experiment:
• **Time** $t_0$: $v_i$ is infected, $v_j$ is susceptible.

$$
\eta_j(t_0) = T_{\text{max}} \frac{k_{\text{max}}}{\sum_m k_m}.
$$

• **Time** $t_1$: All walkers leave node $v_i$ and move to node $v_j$. Node $v_j$ gets infected. Node $v_i$ turns susceptible.

$$
\eta_j(t_1) = (T_{\text{max}} + 1) \frac{k_{\text{max}}}{\sum_m k_m}.
$$

• **Time** $t_2$: All $k_{\text{max}}$ walkers leave node $v_j$:

$$
\eta_j(t_2) = (T_{\text{max}}) \frac{k_{\text{max}}}{\sum_m k_m},
$$

infected each neighbor. The node returns to the state of susceptibility.

• **Time** $t_3$: Assuming that all walkers of all infected neighboring nodes will move to $v_j$, we obtain:

$$
\eta_j(t_3) = (T_{\text{max}}) \frac{k_{\text{max}}}{\sum_m k_m} + \sum_{i \in N_j} \frac{k_i}{\sum_m k_m}
$$

$$
= (T_{\text{max}}) \frac{k_{\text{max}}}{\sum_m k_m} + \frac{(k_{\text{max}})^2}{\sum_m k_m},
$$

since we assumed all neighbors of $v_j$ to be of maximum degree.

• We now assume that step 2 and 3 repeat until the end of the experiment.

• **Time** $t_4$:

$$
(T_{\text{max}} - 1) \frac{k_{\text{max}}}{\sum_m k_m} + \frac{(k_{\text{max}})^2}{\sum_m k_m}.
$$

• **Time** $t_5$:

$$
(T_{\text{max}} - 1) \frac{k_{\text{max}}}{\sum_m k_m} + 2 \frac{(k_{\text{max}})^2}{\sum_m k_m} . . .
$$
\[ \eta_j(T_{\text{max}}) = \frac{1}{\sum m k_m} \left[ (T_{\text{max}} - \left\lceil \frac{(T_{\text{max}} - 3)}{2} \right\rceil k_{\text{max}} + \left\lceil \frac{(T_{\text{max}} - 2)}{2} \right\rceil (k_{\text{max}})^2 \right]. \]
References


