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Disease dynamics on small-world and other networks

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Abstract

In 1998 Watts and Strogatz introduced the concepts of small-world networks and in the process expanded our views of computer, social, and biological networks. In this project, we built epidemics on small world and other networks. Epidemic outbreaks of communicable and sexually-transmitted diseases are modeled on small-world and two-node networks, respectively. The results of simulations are compared to those obtained from homogeneous mixing (mean field) epidemic models. Scaling relationships between transmission rates for epidemics on small-world, random and homogeneous mixing populations are established empirically. The transmission dynamics of gonorrhea in heterosexually-active populations with multiple partners is used to illustrate the spread of disease on two-node networks. Strategies for disease control are explored.

1 Introduction

Networks are inherently part of our environment and can be defined as sets of nodes interconnected by edges. We are part of networks of scientists, actors, and musicians, to name a few. However, networks are not only comprised of people and there are computer, power generator, airline, and many other type of networks. Connections between nodes in a network represent some predefined relationship (such as friendship, hub-host connections, country alliance, to list a few).

Research into networks has gone in multiple directions. For example, regular and random networks have been studied extensively. Therefore, these are the type of networks that we will use in our research.

Regular networks are networks in which each node is connected in the same way to the same number of neighbors (e.g., four connections to the nearest neighbors). These networks exhibit high levels of clustering since nearby nearby points are typically connected by a high density of links. Random networks are networks in which nodes are connected haphazardly (e.g. randomly). Hence some nodes are connected to their nearest neighbors and others to more distant parts of the networks. Hence, random networks have a relatively small characteristic path length (a measure of the average number of steps between any two nodes) and exhibit little clustering. However, such graphs also have little clustering of nearby nodes.

Watts and Strogatz (1998) introduction of the concept of small-world networks and their analysis of the relationship to random networks re-invigorated the study of networks (for an excellent overview, see Strogatz 2001). Research on small world phenomenon goes back to the 1960's [2]. The formulation and initial mathematical analysis of the properties of small-world networks is due to Manfred Kochen and Ithiel de Sola Pool (Pool and Kochen, 1978). They estimated the average number of acquaintances of an individual as well as the probability that two randomly selected members of a society were actually connected to this individual via one or two intermediaries. Their estimates were computed with models that included social level and the degree of stratification present in the population. They concluded that acquaintance chains in highly structured (cluster) populations had characteristic path lengths that were not much longer than those found in completely unstructured populations. Anatol Rapoport and collaborators worked University of Chicago colleagues worked on social stratification in networks. Their work set the ground for the theoretical work of Pool and Kochen.

Watts and Strogatz model of small-world phenomena arose from their study of networks with a mixed ""connectivity" strategy. They found that the average characteristic path length and the level of clustering are ex-

tremely sensitive to small variations (addition of long-distance connections between nodes with low probability) on the connectivity structure of a regular network. Watts and Strogatz knew that regular networks have longer characteristic path lengths and larger clustering coefficients (a measure of the fraction of a node's links that go to other nodes in its immediate vicinity) than random networks. However, they noticed that the replacement (with low probability) of a few local connections with long-term connections drastically decreases the average-path length while keeping high levels of clustering. In other words, specific perturbations on the connectivity structure of a regular network result in networks that have some of the features of random (small average-path length between nodes) and regular (clustering) networks. Watts and Strogatz showed that the replacement of one percent of the connections of a regular network with distant links was enough to trigger small-world phenomena.

In this paper, individual-based disease dynamics are implemented on fixed small-world and random networks. These networks can provide a good insight into the dynamics of disease in social networks. Results from the known mean field epidemic models and those resulting from epidemics on networks are compared. Empirical scaling relationships between both approaches are established. We also study disease dynamics on networks with two types of nodes where connections among nodes of the same type are not allowed. The connections between one type of nodes is assumed to be exponentially distributed. We apply these two-node networks to study the dynamics of gonorrhea on a heterosexually active population. In this two-sex network some males have more connections than others while females receive connections. The impact of abstinence is considered. In this network, each individual has at least one partner of the opposite sex. A deterministic model to study gonorrhea dynamics is studied for comparison purposes. Results from this deterministic model are compared with those obtained from two-node networks. It is found that abstinence plays an important role in the reduction of disease prevalence and disease persistence.

2 Characterizing Networks

Networks can be mathematically represented by an adjacency matrix T of zeros and ones where

$$T_{ij} = \begin{cases} 1, & \text{if there exists a connection between individual } i \text{ and } j \text{ where } i \neq j, \\ 0, & \text{otherwise.} \end{cases}$$

N	Total number of individuals (vertices) in the network.
r	Radius of vicinity.
k	Average number of contacts per individual.
L	Characteristic path length.
γ_ν	Clustering coefficient.

Table 1: Network-related parameters

The dimensions of the adjacency matrix is $N \times N$ where N is the total number of nodes in the network. All links are bidirectional and self-contact is not allowed (See table 1 for reference to network-related parameters). The radius of vicinity, r, gives the maximum possible number of immediate connections of an individual to the right and to the left. For a regular network, all the connections are to immediate nodes. A regular network with radius of vicinity r has a total of $2r$ connections set per node.

According to the analysis of small-world networks, the average number of contacts per individual in a random network increases with the number of long-distance random connections. For a regular network with $r = 2$, the number of connection per node (individual) is constant and equals to 4. This value increases until it reaches approximately 8 contacts per individual as the network moves from a regular to a random network via small-world networks with an increasing number of long-term connections. See figure 1.

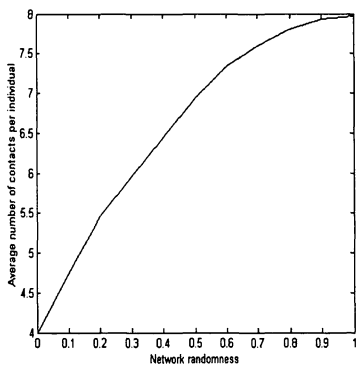

Figure 1: Relationship between the average number of contacts per individual and randomness in the networkwhen $r = 2$

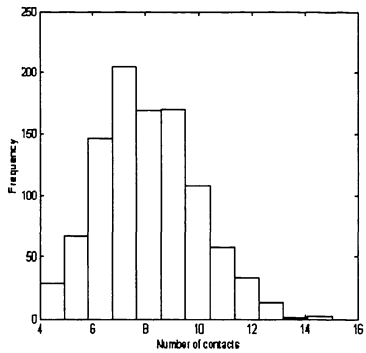

Figure 2: Histogram of the number of contacts per individual for completely random network. For the case of a regular network all the individuals have exactly $2r$ contacts

2.1 The characteristic path length and the clustering coefficient

The characteristic path length is defined as the average distance between two nodes in the network [2]. That is, it is given by the average number of edges that must be traversed in order to reach vertex i from vertex j. Watts defines the characteristic path length (L) of a graph as the median of the means of the shortest path lengths connecting each vertex $\nu \in V(G)$ to all other vertices. The algorithm to compute L is as follows: $d_\nu = d(\nu, j)$ for each $j \in V(G)$ and find d_ν for each ν. L is the median of $\{d_\nu\}$ where $\nu \in V(G)$.

When N is large, this approach is not efficient and random sampling techniques are preferred (see Huber, 1996). According to the method described in Huber, d_ν is calculated for a randomly selected subset of s vertices. L is the median of $\{d_\nu\}$

Watts and Strogatz (1998) computed the clustering coefficient in their graphs by listing all the neighbors of a vertex, counting the edges that link those neighbors, and dividing by the maximum number of edges that could be drawn among the node's neighbors. The process is repeated on all the vertices and the average taken. In contrast to the characteristic path length L, the clustering coefficient remains high until the number of random connections in the network is is rather large. Such clustering coefficient γ_ν of Γ_ν (which is the neighborhood of the vertex ν) characterizes the extent to which vertices adjacent to any vertex ν are adjacent to each other. More precisely,

$$\gamma_\nu = \frac{|E(\Gamma_\nu)|}{\binom{k_\nu}{2}}$$

where $|\ E(\Gamma_\nu)\ |$ is the number of edges in the neighborhood of ν and $\binom{k_\nu}{2}$ is the total number of possible edges in Γ_ν.

That is, given k_ν vertices in the subgraph T_ν, at most $\binom{k_\nu}{2}$ edges can be constructed in that subgraph.
The clustering coefficient of G is $\gamma = \gamma_\nu$ averaged over all $\nu \in V(G)$.

For instance, in a regular network where each node is connected to the four nearest neighbors the number of edges that link any node's neighbors is 3 and the total number of connections that can be drawn among those four neighbors is $\binom{4}{2}$. Therefore the clustering coefficient according to the definition is given by $frac36$ and is constant for all the nodes in regular networks.

3 Individual-based models for disease spread on networks

Individual-based models are simulations based on the local interactions of members of a population. They typically consist of an environment in which the interactions occur specific behaviors and characteristic parameters for the individuals[10]. In an individual-based model, the characteristics of each individual are tracked through time.

3.1 Method of creating small-world networks

Watts and Strogatz (1998) proposed the following construction algorithm for generating small world networks: The initial network is a one dimensional lattice of N nodes. Each node is connected to its $2r$ nearest neighbors. The nodes are then visited consecutively; each edge is connected to a node of its r nearest neighbors clockwise with probability 1-p and then reconnected, with probability p, to a randomly chosen node. There are no isolated nodes.

3.2 Method of creating two-node networks

We start with an initial network of N nodes where half of the nodes are different from the other half. First, each node of type T_1 is paired to a node of type T_2 to avoid isolated nodes in the network. Next, the nodes are

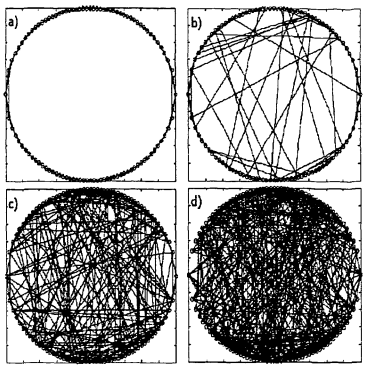

Figure 3: a) Regular network, b)Small-world network, c)Random network with p=0.5, d)Completely random network

visited consecutively; each node of type T_1 sends a number of connections exponentially distributed to nodes of type T_2.

T_{ij}	Adjacency matrix that represents connections among individuals on the network
$S_i(t)$	Vector that represents the state of the individual i at time t.
$\hat{\beta}$	Infectious rate for individual based models
β	Infectious rate for deterministic models
γ	Recovery rate
σ	Loss of immunity rate
$I(t)$	Total number of infected individuals on the network at time t
$S(t)$	Total number of susceptible individuals on the network at time t
$R(t)$	Total number of recovered individuals on the network at time t
δt	A small time increment
P_{SI}	Probability of a susceptible individual becoming infectious
P_{IR}	Probability of an infected individual becoming recovered
P_{IS}	Probability of an infected individual becoming susceptible

3.3 Modeling the epidemic process

Now that we have described ways of construction, we proceed to build simple epidemics on them. We will consider a simple case where individuals on the network could be at most in one of the following three possible epidemiological states: Susceptible (S) b) Infectious(I) c) Recovered (R).

A susceptible individual in contact with I infectious individuals may become infected during a period δt with probability:

$$P_{SI} = 1 - e^{-\hat{\beta} I \delta t} \tag{1}$$

where $\hat{\beta}$ is a constant risk of infection per unit of time.

The infectious rate is chosen according to the relationship that gives the average number of infected individuals by an infected individual that has k neighbors (Keeling & Grenfell, 1999).

$$R_o = k \frac{\hat{\beta}}{\hat{\beta} + \gamma}, \tag{2}$$

$\frac{\hat{\beta}}{\hat{\beta}+\gamma}$ determines the proportion of such infected individuals.
It is also assumed that the infectious period is exponentially distributed, that is, that the probability of recovery in $(t, t + \delta t)$ is given by:

$$P_{IR} = 1 - e^{-\gamma \delta t} \tag{3}$$

Hence $\frac{1}{\gamma}$ gives the mean infectious period.

Recovered individuals may lose immunity at the per capita rate σ, that is the probability of an individual losing immunity in $(t, t + \delta t)$ is given by:

$$P_{RS} = 1 - e^{-\sigma \delta t} \tag{4}$$

When infection do not provide permanent immunity then recovered individuals become susceptible again. The probability of such transition is given by (3). On the other hand, if immunity is permanent then $\sigma = 0$ in (4)

4 Computer simulations

Computer programs for the disease spread simulations were developed on MATLAB. Basically, three programming modules were constructed: The network-creation module, the disease-spread-simulator module, and the module to plot the network topology.

For the network-creation module, the total number of nodes (individuals), N, for the networks was fixed to 1000 and the radius of vicinity ,r, was set equal to 2.
For the disease-spread-simulator module, the recovery rate, γ, was fixed to $\frac{1}{7}$. Three infectious rates were chosen to run simulations: $\hat{\beta} = \frac{2}{7}$, $\hat{\beta} = \frac{3}{14}$ and $\hat{\beta} = \frac{2}{21}$. Simulations were run for the SIR, SIS and SIRS models with two different initial conditions: $I(0) = 5$ and $I(0) = 500$.

The randomness in the connectivity of the individuals on networks plays an important role in disease spread.
When randomness increases the actual characteristic path length between individuals decreases, the average number of contacts per individual increases for a certain r and the clustering among individuals decreases.

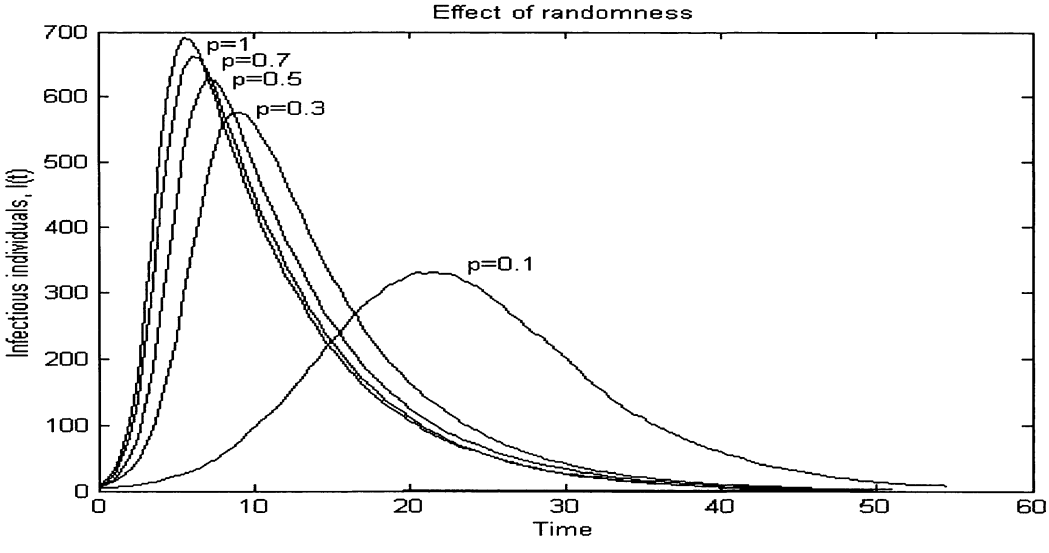

Figure 4: Randomness effect on the disease spread on a network

5 Homogeneous mixing models in closed populations without vital dynamics

The homogeneous mixing models corresponding to the cases described in Section 3.2 are widely used in epidemiology (see for example, Brauer and Castillo-Chavez, 2000). The simplest SIS model is given by

$$\frac{dS}{dt} = -\frac{\beta}{N}SI + \gamma I, \tag{5}$$

$$\frac{dI}{dt} = \frac{\beta}{N}SI - \gamma I. \tag{6}$$

If recovery provides permanent immunity then the model changes to

$$\frac{dS}{dt} = -\frac{\beta}{N}SI, \tag{7}$$

$$\frac{dI}{dt} = \frac{\beta}{N}SI - \gamma I, \tag{8}$$

$$\frac{dR}{dt} = \gamma I. \tag{9}$$

Finally, whenever acquired immunity is lost at a constant per capita rate σ the model becomes

$$\frac{dS}{dt} = -\frac{\beta}{N}SI + \sigma R \tag{10}$$

$$\frac{dI}{dt} = \frac{\beta}{N}SI - \gamma I \tag{11}$$

$$\frac{dR}{dt} = \gamma I - \sigma R \tag{12}$$

Only models (6-??) and (10-12) can support endemic equilibria.

6 Scaling relationships

In homogeneous mixing models, each individual has equal chance to contact anyone else in the population. That is, every single individual has at least a weak influence on all other individuals. In addition, homogeneous mixing models do not deal with any type of intrinsic structure. On the other hand, dynamics on networks depend strongly on the network structure. That is, two different network structures will provide different results when disease dynamics are implemented on them.

Empirical relationships between the transmission parameter (β) used in the homogeneous mixing models and that used in the stochastic individual-based model ($\hat{\beta}$) were obtained for the small-world, and in a general random network as a function of network properties. Simulation results obtained for networks with different levels of randomness including the regular networks, small-world networks, and completely random networks were analyzed. Two properties in such networks were found to play an important role in order to compare the disease spread dynamics with homogeneous mixing models of the type SIR, SIS,and SIRS. These are the average number of contacts per individual (node) and the clustering coefficient.
According to the method of network construction, the average number of contacts per individual increases with the randomness in the network and the clustering coefficient decreases significantly as high levels of randomness are achieved. In general the following relationship between the infectious rate in the deterministic model and that in the networks holds when levels of clustering in the networks are very low:

$$\beta = k\hat{\beta},$$

where k is the average number of contacts per individual on the network.

One of the main features of a small-world network is the high levels of clustering retained with the introduction of a small number of random connections. The above relationship does not hold for small-world networks. However for random networks with 40% of randomness or higher, such relationship adjusts accurately due to the low levels of clustering in such networks.

An empirical relationship for small-world and in general any random network was found by using regression on a set of data generated from stochastic simulations:

$$\beta = (-7.32 + 1.85k + 2.08\gamma_\nu)\hat{\beta},$$

where γ_ν is the clustering coefficient of the network and the data set consisted of two predictor variables: k and γ_ν and a response variable: the factor $\hat{k}$ that best fit the network results with the mean field model results. See figures 6, 7 and 8 for comparisons of the SIR, SIS and SIRS homogeneous mixing models and the results obtained from different random networks.
In the next section, two-node networks are used to study gonorrhea on a heterosexually active population.

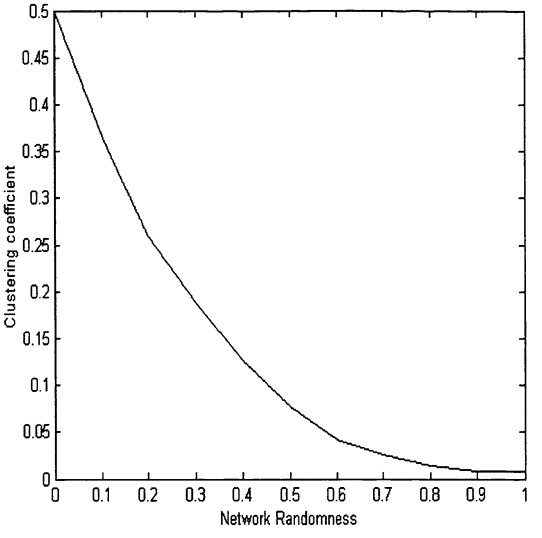

Figure 5: Randomness effect on the clustering coefficient of networks

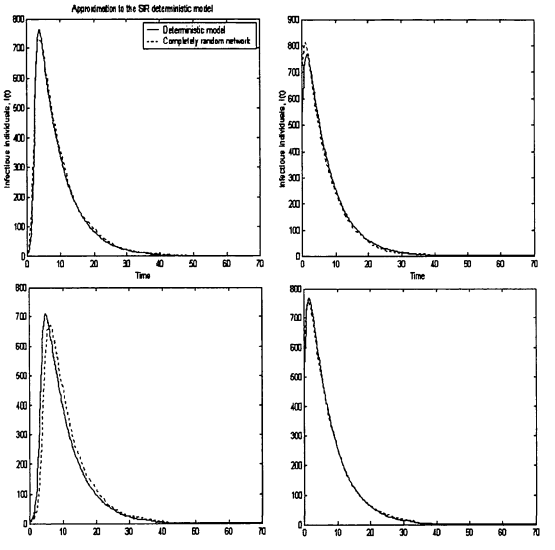

Figure 6: Comparison of the SIR homogeneous mixing model and the results obtained from a completely random network and $\gamma_\nu = 0.0077$. The infectious rates were scaled according to the relationship above. $\hat{\beta} = \frac{2}{7}$ and $\gamma = \frac{1}{7}$ for the top graphs and $\hat{\beta} = \frac{3}{14}$ and $\gamma = \frac{1}{7}$ for the bottom graphs . I(0)=5 (left graphs); I(0)=500 (right graphs)

7 Application: Gonorrhea spread in heterosexual populations

Gonorrhea is a sexually transmitted disease. Gonorrhea mostly affect teens and young adults. The CDC reports that gonorrhea rates declined steadily

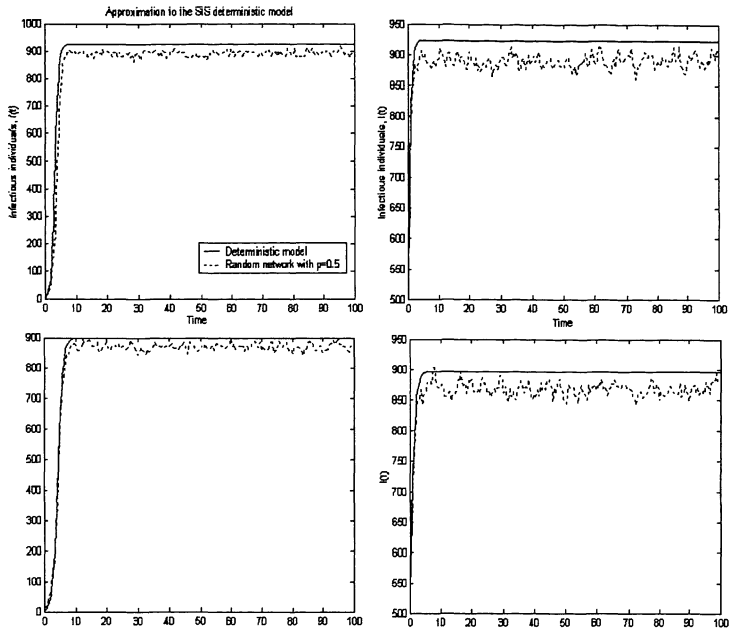

Figure 7: Comparison of the SIS homogeneous mixing model and the results obtained from a random network with $p = 0.5$ and $\gamma_\nu = 0.074$. The infectious rates were scaled according to the relationship above. $\hat{\beta} = \frac{2}{7}$ and $\gamma = \frac{1}{7}$ for the top graphs and $\hat{\beta} = \frac{3}{14}$ and $\gamma = \frac{1}{7}$ for the bottom graphs. I(0)=5 (left graphs); I(0)=500 (right graphs)

until the late 1990's([12]). However, rates stabilized between 1996 and 1997 but between increased by nine percent between 1997 and 1999. It is believed that this increase is due to the increase in gonorrhea among gay and bisexual men([12]). Studies have shown that gonorrhea can facilitate HIV transmission. If the disease is not treated on time gonorrhea can cause serious difficulties in women, like: PID (pelvic inflamatory disease), subsequent infertility and tubal pregnancie. Since women are less likely to show symptoms there are fewer cases reported from women. Men show symptoms quickly and are more likely to seek treatment. The recovery rate for men is three times greater than for women, that means it takes a man 7 days to they recover and become susceptible again and a woman takes 21 days to get treated and become susceptible. The infectious rate for men is around 2.5 greater than for women[11]. Hence, due to their asymptotic behavior females stay infectious longer than males.

We introduce temporary periods of abstinence in our stochastic and deterministic frameworks. Abstinence plays an important role in the control of sexually transmitted diseases[12]. In our simulations individuals who have just recovered go through an abstinence period. Simulations for abstinence

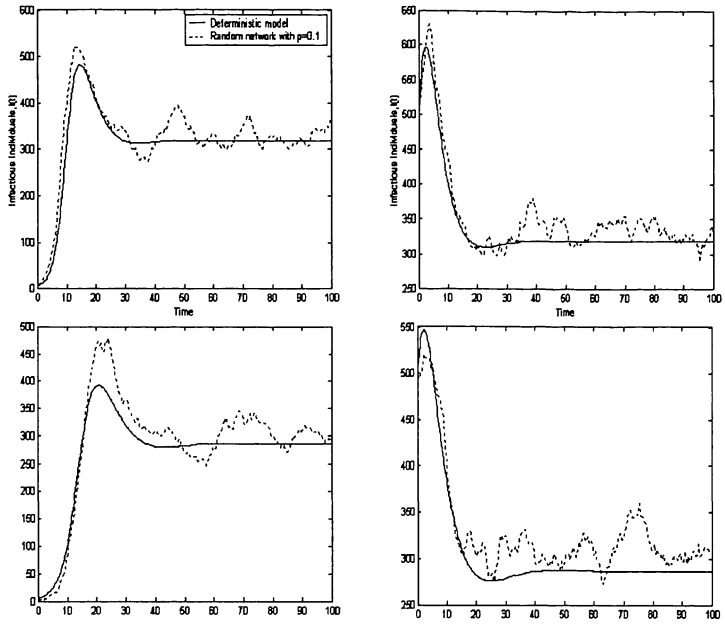

Figure 8: Comparison of the SIRS homogeneous mixing model and the results obtained from a random network with $p = 0.1$ and $\gamma_\nu = 0.366$. The infectious rates were scaled according to the relationship above. $\hat{\beta} = \frac{2}{7}$ and $\gamma = \frac{1}{7}$ for the top graphs and $\hat{\beta} = \frac{3}{14}$ and $\gamma = \frac{1}{7}$ for the bottom graphs. I(0)=5 (left graphs); I(0)=500 (right graphs)

periods of 1,2,3, and 5 weeks and efficacies of 50% up to 100% in increments of 10% are carried out, where efficacy is defined to be the proportion of the population that obey the abstinence state. Simulations results show that the actual endemic states can be lowered considerably and proportional to the abstinence periods. However, such endemic state can be lowered considerably only when the efficacy to follow such abstinence period is above 80%. Furthermore, oscillations whose amplitude depend on the abstinence period appear. In fact, the disease may be eradicated for large enough periods of abstinence that may not be realistic. Therefore, abstinence can not be a strategy to eradicate the disease, but it can control the spread in a susceptible population.

7.1 Deterministic model for gonorrhea with an abstinence state

The following deterministic model for the heterosexual transmission of gonorrhea was introduced by Hethcote[11]:

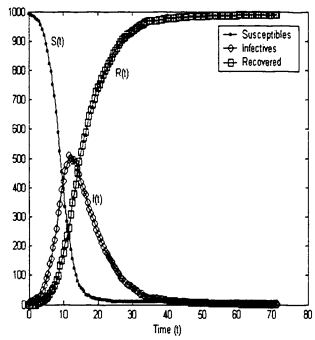

Figure 9: SIR model results for a small-world network. $\hat{\beta} = \frac{2}{7}$ and $\gamma = \frac{1}{7}$

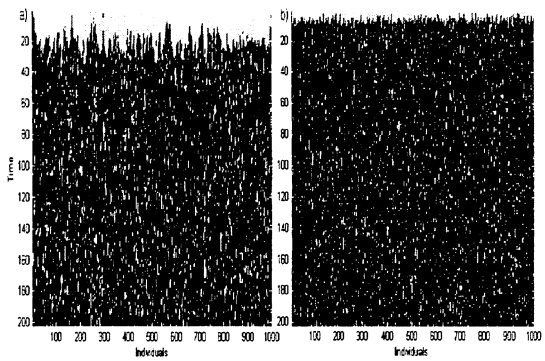

Figure 10: Life history for 200 days for individuals in two different networks on a SIS model. a) Small-world network b) Completely random network. $N = 1000$, $\hat{\beta} = \frac{2}{7}$ and $\gamma = \frac{1}{7}$, in white the susceptibles and in black the infected.

$$\frac{dI_1}{dt} = (\frac{\beta_{12}}{r})(1 - I_1)I_2 - \frac{I_1}{\gamma_1}$$
$$\frac{dI_2}{dt} = (\beta_{21}r)(1 - I_2)I_1 - \frac{I_2}{\gamma_2}$$

where $r = \frac{N_m}{N_f}$, N_m is the male populaton and N_f is the female population. The transmission rate for males is β_{12} and β_{21} for females, γ_i are the infectious period of males and females where $i = 1$ are males and $i = 2$ are females. I_1 are the males in the infected class and I_2 are the females in the infected class and the susceptible classes are $S_i = 1 - I_i$ where $i = 1$ are the male susceptible class and $i = 2$ are the female susceptible class.

A deterministic model for the heterosexual transmission of gonorrhea with

temporary periods of abstinence is introduced, as shown in Figure 11. We will consider males and females. Considering that we want to make comparisons with the two node stochastic model for gonorrhea, this model was derived taking into account the structure of the network. In the network the population size is fixed to 1,000 individuals and we divide the population in two: 500 males and 500 females. The nodes are set to be one male following a female and so on. Also, a male at least has one female partner but it can have up to five partners distributed exponentially in the male population. The female population receive the connections but do not send connections. In the deterministic model individuals can become infected with gonorrhea by having contact with an infectious individual from the opposite sex at the rate $\beta_{ij}I_i$, where $i \neq j$, $i = 1, 2$ and $j = 1, 2$. After an individual becomes infected with the disease the individual can become abstinent at a rate γ_i. While in the abstinent state the individual may still be infectious but does not transmits the disease. After a period of time they leave the abstinent state at a rate δ_i.

Individuals are classified epidemically into classes: S_i for susceptible proportions where $i = 1$ stand for male population and $i = 2$ for females, I_i for the infected males and females proportions, and A_i for males and females in abstinence respectively. We assume a constant population with no recruitment and no deaths.

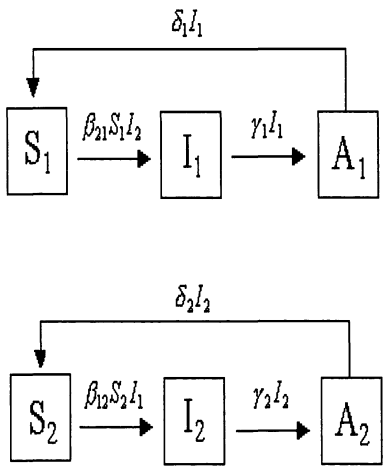

Figure 11: Deterministic model for gonorrhea with abstinence

The model equations for the proportions are:

$$\frac{dS_1}{dt} = -\beta_{21} S_1 I_2 + \delta_1 A_1 \quad (13)$$

$$\frac{dS_2}{dt} = -\beta_{12} S_2 I_1 + \delta_2 A_2 \quad (14)$$

$$\frac{dI_1}{dt} = \beta_{21} S_1 I_2 - \gamma_1 I_1 \quad (15)$$

$$\frac{dI_2}{dt} = \beta_{12} S_2 I_1 - \gamma_2 I_2 \quad (16)$$

$$\frac{dA_1}{dt} = \gamma_1 I_1 - \delta_1 A_1 \quad (17)$$

$$\frac{dA_2}{dt} = \gamma_2 I_2 - \delta_2 A_2 \quad (18)$$

where, $S_1 + I_1 + A_1 = 1$ and $S_2 + I_2 + A_2 = 1$.

7.1.1 Basic Reproductive Numbers

The basic reproductive number is defined as the average number of secondary infections caused by an infectious individual during the infectious period in a population of mostly susceptible male and female populations. Because there is only heterosexual contact we have a basic reproductive number for males R_{om} which is the number of secondary female cases produced by one infected male $R_{om} = \frac{\beta_{12}}{\gamma_1}$, and the female basic reproductive number is, $R_{of} = \frac{\beta_{21}}{\gamma_2}$, which is the number of secondary male cases produced by an infected female. The R_o for the system is then:

$$R_o = R_{om} R_{of}.$$

R_o gives the number of secondary males (females) cases that one female (male) case produce in a fully susceptible population. The disease will persist only if $R_o > 1$.

7.1.2 Equilibria and Elasticity analysis

The disease free equilibrium of system13 is $\hat{S}_i = 1$, $\hat{I}_i = 0$, and $\hat{A}_i = 0$. For $R_o < 1$ the disease free equilibrium is locally stable while for $R_o > 1$ the disease free equilibrium becomes unstable and there exist a unique endemic equilibrium.

$$\hat{I}_1 = \frac{\gamma_1\gamma_2\delta_1\delta_2(R_o - 1)}{\beta_{12}(\beta_{21}\delta_1\delta_2 + \beta_{21}\gamma_1\delta_2 + \delta_1\delta_2\gamma_1 + \gamma_1\gamma_2\delta_1)}$$

$$\hat{I}_2 = \frac{\gamma_1\gamma_2\delta_1\delta_2(R_o - 1)}{\beta_{21}(\beta_{12}\delta_1\delta_2 + \beta_{12}\gamma_2\delta_1 + \delta_1\delta_2\gamma_1 + \gamma_1\gamma_2\delta_2)}$$

$$\hat{A}_i = \frac{\gamma_i\hat{I}_i}{\delta_i}$$

where $i = 1, 2$ and $S_i = 1 - \hat{I}_i - \hat{A}_i$.

We analyzed the stability of the endemic equilibrium for both male and female populations numerically. For all parameter values that were used in the simulations the endemic equilibrium resulted stable as shown in figure12. Proving stability for endemic when $R_o > 1$ in a general case could be done by using the Routh Horwitz criteria but we were not able to find it.

In order to find what parameters were most likely to change the endemic equilibria we compared the elasticity of the R_o with respect to the transmission rate β_{ij}, $i \neq j$ and the infectious rate γ_i. Elasticity of $f(\lambda)$ with respect to λ is defined as:

$$E_\lambda(f, \lambda) \equiv |\frac{1}{f(\lambda)}\frac{\partial f(\lambda)}{\partial \lambda}|$$

In our case we have $E(R_o, \beta_{12}) = \frac{1}{R_o m}\frac{1}{\gamma_1}$ and $E(R_o, \beta_{21}) = \frac{1}{R_o f}\frac{1}{\gamma_2}$.
The elasticity analysis showed that the most significant parameter is the infectious period for females, γ_2. We can resume from the elasticity analysis that when $R_o > 1$ if we lower the infectious periods of females the endemic states will lower significantly. A strategy for control would be having females being abstinent for longer periods of time until they recover, but the problem of asymptomatic individuals takes that strategy out. After looking at various changes in the initial conditions we found that there is an endemic equilibrium for any initial conditions $I_1(0)$ and $I_2(0)$, and a disease free equilibrium when $I_1(0) = 0$ and $I_2(0) = 0$. Therefore, the endemic state of the disease is independent of any other changes in the parameters. But we can observe if individuals choose to be abstinent for longer periods of time, the endemic state of the disease would be lower than if individuals leave the abstinent state after a short period of time. Hence, we can conclude that abstinence will not make the disease die out but rather lower the chances of individuals

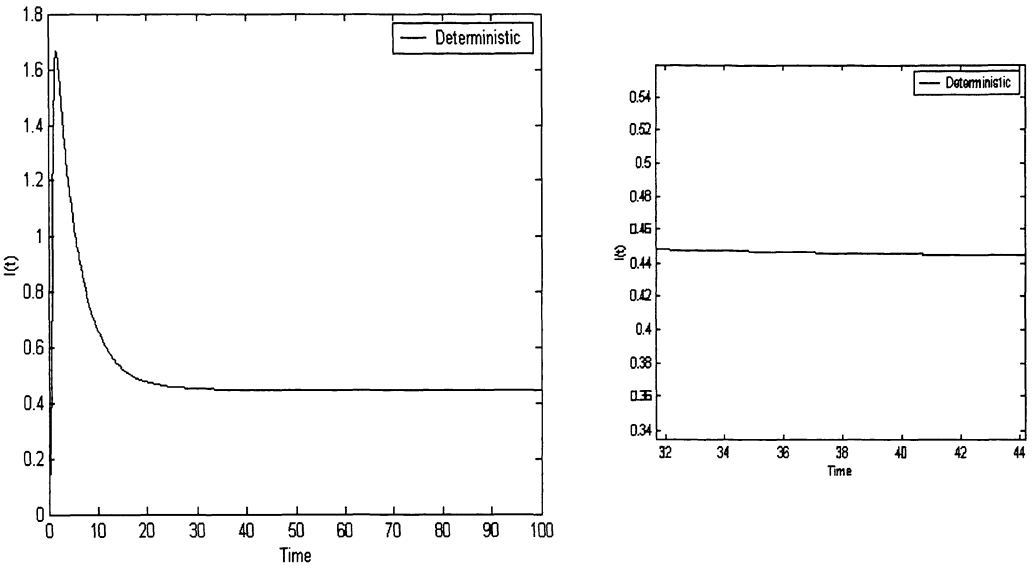

Figure 12: Endemic state

getting infected. As we see in figure 13 the endemic state of the disease for different periods of abstinence.

7.2 Networks with two types of nodes

We focus on heterosexual populations. That is, only sexual contacts among males and females are considered. The model assumes an active population where there are no isolated individuals. Everyone in the network has at least one partner. The distribution of the number of partners is exponential and given by the following relationship:

$$\frac{a}{e^{-a}} e^{-at} \tag{19}$$

where a was fixed to 0.5.

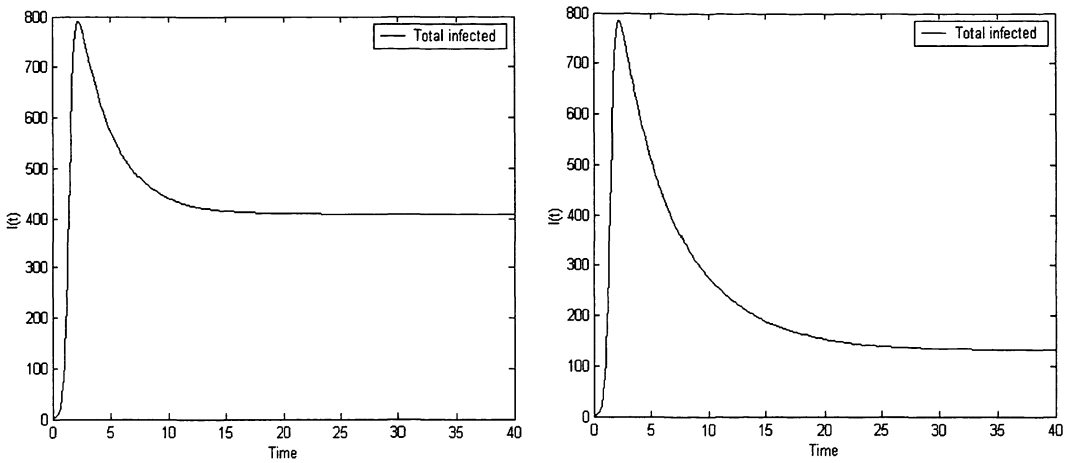

Figure 13: Endemic state of gonorrhea after different periods of abstinence,(left: one week),(right: five weeks)

7.3 Simulations for the gonorrhea spread

A computer program for the spread of gonorrhea on networks of two types of nodes is developed on MATLAB. The total number of individuals (individuals), N, for the network was fixed to 1000, half males and half females. The recovery rate, γ, is fixed to $\frac{1}{7}$ for males and to $\frac{1}{21}$ for females. The infectious rates are chosen to be $\hat{\beta} = 2.5$ and $\hat{\beta} = 1$ for males and females, respectively. Simulations are based on an SIS model. Initially, there is an infected male with four partners, $I(0) = 1$.

Simulation results show the presence of an endemic state for all the simulation runs.

7.4 Comparisons between the stochastic and deterministic models

Now we want to compare the stochastic and deterministic and see if there is any relation between them. In figure16 the period of abstinence is for one week. In figure17 the period of abstinence is five weeks. The stochastic

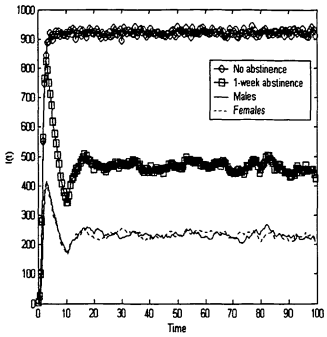

Figure 14: Comparison between the gonorrhea evolution without abstinence period and with an abstinence period of 7 days for males and females considering a 100% abstinence efficacy.

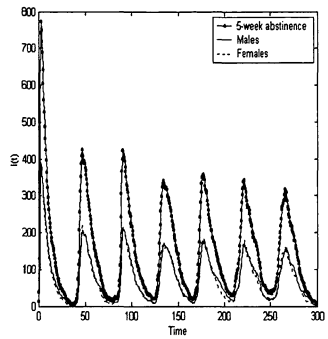

Figure 15: Gonorrhea evolution with an abstinence period of 5 weeks for males and females considering a 100% abstinence efficacy.

model show oscillations and the deterministic crosses through the middle as the average of those oscillations. We used mostly the same parameters values in the stochastic and the deterministic in order to make the comparisons. The one parameter value that changed was the infectious period. In the stochastic the infectious period γ_i was for males $\gamma_1 = 1/7$ and for females $\gamma_2 = 1/21$. Therefore, individuals in the stochastic would be infectious for 7 days for males and 21 days for females. In the deterministic model the infectious period was reduced to $\gamma_1 = 1/4$ for males and $\gamma_2 = 1/9$ for females.

8 Conclusions

Small-world networks provide a good approach to modeling individual-based disease dynamics. Such networks can provide a good insight into the dynam-

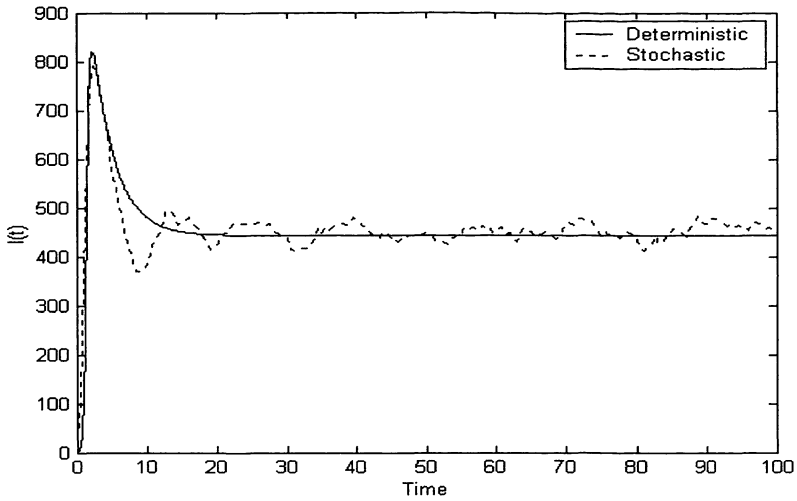

Figure 16: Stochastic vs. Deterministic

ics in social networks. Disease dynamics is only one of many dynamics that could be modeled on a small-world network environment. Disease dynamics on networks depend strongly on the network structure.

Individual-based models offer a useful approach to understand disease dynamics on networks. In this paper, we focus on small-world networks and random networks. Local dynamics yield global consequences since each individual is characterized and tracked through time.

For networks with small clustering coefficient, the relationship $\beta = k\hat{\beta}$ provides an accurate approximation between the deterministic mean field models and those based on random networks.

Empirical relationships between networks with different levels of randomness including small-world networks can be obtained by multilinear regression where the clustering coefficient and the connectivity are chosen as the predictor variables.

In sexually transmitted diseases as gonorrhea, abstinence plays an important role in the way of decreasing endemic states. Oscillations in disease evolution appear as a consequence of such control strategies.

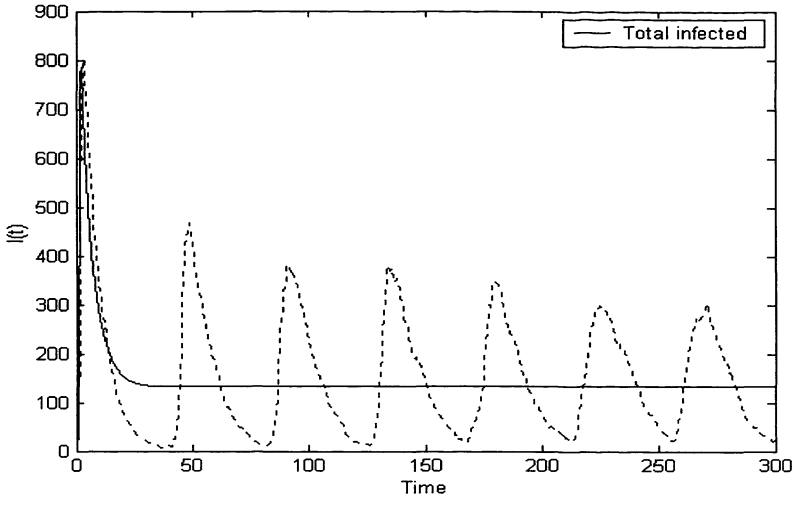

Figure 17: Stochastic vs. Determinisitic

9 Future Work

Use different infectious rates for long distance connections.

Study virulence evolution in networks.

Study disease dynamics on weighted networks where the infectious rate would depend on the distance between individuals.

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A Computer programs

The following computer code corresponds the network-creation module and the disease-spread-simulator module.

```
function y=SWNetwork(n,in,r,tspan,filename,flag2)
%//////////////////////////////////////////////
%SMALL WORLD NETWORK
%//////////////////////////////////////////////

   %INPUT
%n is the total number of nodes in the network
%r is the radious of vecinity
%OUTPUT
%cpl=characteristic path length
%cc=clustering coefficient
%in=initial number of infected individuals
%filename=name for the file where the adjacency matrix is saved
%p=probability of edge randomly changed
%flag2=1 if bidirectional connections are considered
%T(i,j)=Matrix that indicates the number of ways of node i to reach node j in
ONLY one step

   if r>((n/2)-1)
'r [the radious of vecinity] is too big'
end

   PROBS=zeros(1,n+1);
frac=1/n;

   PROBS(1,1)=0;
sumfrac=0;
for j=2:n+1,
sumfrac=sumfrac+frac;
PROBS(1,j)=sumfrac;
end

   p=0.1;

   while p<=0.1 %Loop for changing P and get different networks
T=sparse(n,n,n*5); for i=1:n,
k=i;
l=i;
a=0;
for j=1:r,
if (k+1)>n %right neighbors
k=1;
else
k=k+1;
end

   %1 percent of the edges will be set randomly
```

```
rnd1=rand;
if (rnd1>=0) & (rnd1<p)
'randomly chosen'
k2=i;
while(i==k2)
rnd1=rand;
for x=1:n+1,
if (rnd1>=PROBS(1,x)) & (rnd1<PROBS(1,x+1))
k2=x;
end
end
end
T(i,k2)=1;
else
T(i,k)=1;
end

   if (l-1)<1 %left neighbors
l=n;
else
l=l-1;
end

   %1 percent of the edges will be set randomly
rnd1=rand;
if (rnd1>=0) & (rnd1<p)
%'randomly chosen'
k2=i;
while(i==k2)
rnd1=rand;
for x=1:n+1,
if (rnd1>=PROBS(1,x)) & (rnd1<PROBS(1,x+1))
k2=x;
end
end
end
T(i,k2)=1;
else
T(i,l)=1;
end
end
end

   T;

   save(strcat(filename,'-',num2str(p),'.mat'),'T')

   if flag2==2
% Convert the network to a bidirectional network-> critical for highly random
networks
'Two way connections have been chosen...'
T2=sparse(n,n,n*5);
```

```
T2=T;
for i=1:n
con=T(i,:);
(x,y)=find(con);
for j=1:length(x)
if T(i,y(j))==1
T2(i,y(j))=1;
if i =y(j)
T2(y(j),i)=1;
end
end
end
end
T=T2;
strcat(filename,'-',num2str(p),'-bi.mat')
save(strcat(filename,'-',num2str(p),'-bi.mat'),'T')
else
'one way connections have been chosen...'
end
%

   %End of network creation

   %Parameters
b=2/7; %infectious rate
g=1/7; %recovery rate
s=0.1; %loss of immunity rate
dt=0.5;
Tsus=n-in;
Tinf=in;
Trec=0;
Tsusm=zeros(4000,2);
Tinfm=zeros(4000,2);
Trecm=zeros(4000,2);
Tsir=zeros((tspan/dt)+1,4);

   Cstatus=zeros(tspan/dt,n);

   SUSCEPTIBLE_=0;
INFECTED_=1;
RECOVERED_=2;

   t=0; %initial time

   status=zeros(n,1); %status of individual 0=suceptible,1=infected,2=recovered

   %Set the number of initial infectious individuals
for i=1:in
status(i)=1;
end
%
```

```
   Cstatus(1,:)=status(:,1)';

   'Simulating disease spread on the SW-NETWORK (SIS model)...'

   %iterations=round(tspan/dtt);
iterations=tspan;

   qt=0;

   tic
c=1;
while qt<=tspan
for i=1:n
con=T(i,:);
switch status(i)
case SUSCEPTIBLE_
Linf=0;
(x,y)=find(con);
for j=1:length(x)
if status(y(j))==INFECTED_
Linf=Linf+1;
end
end
PSI=1-exp(-b*Linf*dt);
u=rand;
if u<=PSI
status(i)=INFECTED_;
Tinf=Tinf+1;
Tsus=Tsus-1;
end

   %SIR model -permanent immunity
case INFECTED_
PIR=1-exp(-g*dt);
u=rand;
if u<=PIR
status(i)=RECOVERED_;
Trec=Trec+1;
Tinf=Tinf-1;
end

   % SIS model
%case INFECTED_
% PIS=1-exp(-g*dt);
% u=rand;
% if u<=PIS
% status(i)=SUSCEPTIBLE_;
% Tsus=Tsus+1;
% Tinf=Tinf-1;
% end

   %SIRS model -loss of immunity
```

```
case RECOVERED_
PRS=1-exp(-s*dt);
u=rand;
if u<=PRS
status(i)=SUSCEPTIBLE_;
Tsus=Tsus+1;
Trec=Trec-1;
end
end
end
Tsir(c,1)=qt;
Tsir(c,2)=Tsus;
Tsir(c,3)=Tinf;
Tsir(c,4)=Trec;

   if c==10
toc
end
if mod(c,1)==0
save(strcat('status-',num2str(p),'-',num2str(c),'.mat'),'status');
end

   Cstatus(c+1,:)=status(:,1)';

   if Tinf==0
qt
break %Exit for loop when the number of infectives are 0
end

   dgplot(strcat(filename,'-',num2str(p),'-bi.mat'),strcat('status-',num2str(p),'-',num2str(c),'
M(c) = getframe;

   qt=qt+dt;
c=c+1;
end

   movie2avi(M,'temp.avi','FPS',5,'QUALITY',90)

   toc
save(strcat('statusFinal-',num2str(p),'.mat'),'status');
save(strcat('SIRS_DATA','-',num2str(p),'-',num2str(z),'.mat'),'Tsir');
save(strcat('Cstatus-STD','-',num2str(z),'.mat'),'Cstatus');

   p=p+0.1
end %of while p<=1

   c=c-1;

   plot(Tsir(1:c,1),Tsir(1:c,2),'g');
hold on;
plot(Tsir(1:c,1),Tsir(1:c,3),'r');
plot(Tsir(1:c,1),Tsir(1:c,4),'b');
```

```
xlabel('Time steps');
legend('Susceptibles','Infected','Recovered');
```

The following is the module to plot the network topology:

```
   function DGplot(filename,filenameStatus)
load (filename,'T');
load(filenameStatus);

   T
L=length(T);
y=zeros(L,2);
x=-round(L/4);
i=1;

   TS0=zeros(L,2);
TI0=zeros(L,2);
TR0=zeros(L,2);
cons=1;
coni=1;
conr=1;
SUSCEPTIBLE_=0;
INFECTED_=1;
RECOVERED_=2;

   while i<=(L/2)+1
y(i,1)=x;
y(i,2)=sqrt((L/4)^2-x.^2);

   switch status(i)
case SUSCEPTIBLE_
TS0(cons,1)=x;
TS0(cons,2)=y(i,2);
cons=cons+1;
case INFECTED_
TI0(coni,1)=x;
TI0(coni,2)=y(i,2);
coni=coni+1;
case RECOVERED_
TR0(conr,1)=x;
TR0(conr,2)=y(i,2);
conr=conr+1;
end

   x=x+1;
i=i+1;
end

   x=round(L/4)-1;
j=i;
'iccc',i
```

```
while i<=j+((L/2)-1)
y(i,1)=x;
y(i,2)=-sqrt((L/4)^2-x.^2);

   i
if i<=L
switch status(i)
case SUSCEPTIBLE_
TS0(cons,1)=x;
TS0(cons,2)=y(i,2);
cons=cons+1;
case INFECTED_
TI0(coni,1)=x;
TI0(coni,2)=y(i,2);
coni=coni+1;
case RECOVERED_
TR0(conr,1)=x;
TR0(conr,2)=y(i,2);
conr=conr+1;
end
end
x=x-1;
i=i+1;
end

   gplot(T,y,'-o');
hold on;

   if conr>1
conr=conr-1;
T0=eye(conr,conr);
gplot(T0,TR0(1:conr,:),'-oy');
end

   if cons>1
cons=cons-1;
T0=eye(cons,cons);
gplot(T0,TS0(1:cons,:),'-og');
end

   if coni>1
coni=coni-1;
T0=eye(coni,coni);
gplot(T0,TI0(1:coni,:),'-or');
end

   cons,coni,conr

   length(y)
TS0
TI0
TR0
```

```
end
```

B Acknowlegements

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