Modelling and Analysis of Delayed SIR Model on Complex Network

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Abstract Complex networks are often used to model the network of individuals for analysing various problems in human networks e.g. information diffusion and epidemic spreading. Various epidemic models have been proposed for analyzing and understanding the spreading of infectious diseases in human networks. In the classical epidemiological model, a susceptible person becomes infected instantly after getting in contact with the infected person. However, this scenario is not realistic. In reality, a healthy person takes some time to get infected after becoming in contact with the infected person. Therefore, efforts are needed for creating more realistic models to study the dynamics of epidemics in the human population.
In order to handle delays in the infection process, we propose an epidemic model *SIR with delay* in human networks, which are modelled as a complex network. We introduce a parameter, *delay in infection* to handle the process of infected nodes not getting infected instantly. The critical threshold is derived for epidemic spreading on complex network considering delay in infection. We perform simulations of our *SIR with delay* model on three different underlying dynamic network topologies, which represents the real world scenario, where humans constantly create new connections. The simulation results are in accordance with our theoretical derivations which shows that increment of delay decreases the critical threshold of epidemic spreading rate and the disease persists for longer time.

Key words: Complex Network, SIR , Delay, Epidemic Spreading

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1 Introduction

Dynamics processes such as information diffusion [16] and epidemic spreading [2] in human networks is often studied through complex systems [20] [14]. The underlying network in these system consists of nodes representing individuals and edges as the connections. Specifically, the problem of spreading of infectious diseases is an important topic as it can help in improving public health policies.

Various models have been proposed for understanding the epidemic spread in human networks [2] [22]. In this course of research and development, to find the patterns of spread, Satorras and Vespignani [20] investigated to understand the effect of network structure for epidemic spreading.

The two most widely studied models to explain the epidemic spreading in human population is the i) susceptible-infected-removed (SIR) [7] and ii) susceptible-infected-susceptible (SIS) model [6]. The theoretical approach of epidemiological model is based on compartmental concept, in which population is categorized into compartments. In SIR model, there are three compartments i) susceptible (denoted by S), ii) infected (I), and iii) recovered (R). Individuals in the susceptible compartment represent the group of healthy persons that can be infected when get in contact with infected persons. If a person gets infected, it is transferred into infected compartment. Finally, after recovery an infected person is moved into recovered compartment. In case of SIS model, there are only two compartments i) susceptible (denoted by S), and ii) infected (I), where, a recovered individual can again become susceptible unlike the SIR model, where an individual is considered forever recovered and not being prone to susceptible from a disease. Subsequently, various variations of these models have been proposed. For example, Takeuchi et al. [19] proposed a delayed SIR epidemic model to analyze the propagation of vector diseases. Later, Wang et al.[21] investigated the delayed SIR model with time delay in incubation with some carrying capacity. This carrying capacity decides the growth of susceptible individual in absence of disease.

Most of the studies have not focused on the underlying network structure, which also plays an important part in epidemic spreading [2] [6] [21] [25]. In summary, except works like [10], these studies consider the underlying networks as static which is unrealistic from real world scenario where the network is dynamics. By the term dynamics we mean, the connections between individuals. That is new connections are formed and some of the connections disappeared with time. Thus, rigorous studies are need of time for understanding the epidemic dynamics to show how dynamic human interaction impact epidemic spreading.

In this paper, we study epidemic spreading in human population using *SIR with delay*, by modelling human networks as complex networks. To incorporate the delay, we introduce a new parameter, which we call as *delay in infection*. This parameter handles the real world scenario where people generally do not get infected instantly when they come in contact with the infected person. We also derive the critical threshold for epidemic spreading

by considering delay in the infection. In particular, we evaluated our model to investigate the effect of delay using following three types of dynamic networks:

I **Random graph:** Classical random graph was proposed by Erdos-Renye [5]. A random graph, is created when a node is connected with another nodes with connecting probability. It is represented by $G(N, p)$, where N is number of nodes and p is connection probability.

II **Random geometric graph:** A random geometric graph (RGG) is an undirected graph created by randomly distributed nodes in the space and two nodes are connected if the distance between two nodes is in a given range called connectivity radius. A random geometric graph (RGG) [15], denoted by $G(N, r)$ is an undirected graph created by randomly distributed N nodes in the space and two nodes are connected if the distance between two nodes is in a given range r. Consider N nodes located at random positions, $\mathscr{X} = \{X_1, ..., X_N\}$, where X_i are i.i.d. s uniformly distributed random variable in the 2-dimensional region. The nodes i, j are connected if $|X_i - X_j| \leq r$.

III **Random geometric graph with mobility:** A random geometric graph with mobile agents is similar to RGG but in RGG, position of each node changes uniformly, while in RGG with mobile agents, position of only mobile nodes changes and rest of nodes is static.

The simulation results being obtained using our model in accordance with the theoretical derivations. That is, increment of delay decreases the critical threshold and the disease persists for longer time.

The remainder of this paper is organized as follows: Section II discusses the current state of arts regarding traditional disease models and underline network topology. Section III explains the effect of delay in SIR epidemic model. Section IV presents simulation and result analysis. In this section we have simulated the model for number of times to get the result. Finally, Section V describe conclusions and outlines some of our future directions.

2 Current State of The Art

In this section, we discuss two streams of relevant literature at the intersection of which our work lies. The first set of works are related to SIR and SIS model and their modifications. The second set of works related to epidemic spreading which have analysed using various different types of networks.

2.1 Mathematical Modelling of Epidemic Spreading

In 1760, Daniel Bernoulli [3] proposed the first mathematical approach for epidemics study for the spread of infectious diseases. In this course of action, the classical SIR model is proposed by Kermack and McKendrick [7] as follows:

$$\frac{dS(t)}{dt} = -\beta S(t)I(t) \tag{1}$$

$$\frac{dI(t)}{dt} = \beta S(t)I(t) - \mu I(t) \tag{2}$$

$$\frac{dR(t)}{dt} = \mu I(t) \tag{3}$$

where, $S(t), I(t), R(t)$ is the fraction of susceptible, infected and recovered population at time t. However, the classical SIR epidemic model does not consider the heterogeneity and topology of complex network.

Later, Anderson and May proposed various models to describe the disease-related deaths and disease-reduced reproduction that had great impact on the population size [2].

There have been numerous works which explored the SIS and SIR models for understanding the epidemic spreading. For example, Shi et al. [17] studied the behaviour of the SIS epidemic model by including the propagation vector and observe that the propagation vector reduced the epidemic threshold and lead the disease spreading. C.Xia et.al [23] presented a model based on the SIR model, to investigate the impact of infection delay and propagation vector on the spreading behaviors in complex networks, however, they did not explain the impact of infection delay on susceptible.

In addition, various modified SIR and SIS models have been proposed as well. For example, Anurag et.al. proposed the modified SIR model by considering the standard SIR rumor spreading model with degree dependent tie strength of nodes and nonlinear spread of rumor by introducing two parameters named as nonlinear exponent and degree dependent tie strength exponent [18]. Some of the works have introduced delays in the models, such as [9], [24].

In Time delay models, a delay parameter (τ) is introduced during infection, which subsequently modifies the above Eq. (1- 3) to follow:

$$\frac{dS(t)}{dt} = -\beta S(t)I(t-\tau) \tag{4}$$

$$\frac{dI(t)}{dt} = \beta S(t)I(t-\tau) - \mu I(t) \tag{5}$$

$$\frac{dR(t)}{dt} = \mu I(t) \tag{6}$$

In addition to SIR and SIS models, Pastor-Satorras et.al. [14] describe the epidemic process in complex network by applying degree based mean-field approach.

2.2 Network structure

Due to stochasticity of epidemic spreading, different underlying network structure have been used to show the different spreading patterns. The advancement in the area of complex networks sets the base for the epidemic

dynamics and initiated several related studies [13]. For example, a lot of emergent events in social networks and biological networks are pretended using the concept of complex networks [1]. Therefore, disease spreading pattern in human population can be seen and analyzed by using the different topological structure [12] [13].

Satorras and Vespignani [20] proposed the epidemic spreading model on scale free network to analyze the absence of epidemic threshold and its associated critical behaviour. Their proposal was based on computer virus spreading on communication and social networks. Yamir Moreno et.al. [10] presented a new epidemiological framework characterized by a highly heterogeneous response of the system to the introduction of infected individuals with different connectivity considering underlying scale free network. Xiang Li and Xiaofan Wang [8] proposed the general spreading dynamical behaviors in small-world evolving networks when control strategies are applied to suppress the propagation of diseases, viruses, and disasters.

But realistic model should include some time delay as delay plays an important role in the dynamics of epidemic. For instance, it can be the incubation period of the infectious disease, the infectious period of patients, and the immunity period of recovery of the disease with time delay. However, very less attention has been given to the epidemic models with time delays on heterogeneous networks as most of the dynamical process on networks are done without considering delay in process.

3 Proposed Methodology

In this section, we explain the *SIR with delay* model. Let graph G(N, E) defines the network of N nodes that represents the total population and E denotes the connections between nodes representing the interaction between individuals through which epidemic spreads. The propagation of disease is explained as: Each healthy node takes a time delay of τ to get infected. Thus, a node which gets in contact with the infected person at time $(t-\tau)$ becomes infected after the time delay of τ. The infection rate is represented by β and μ represents the recovery rate where, β and μ are $\in [0, 1]$.

Let $S_k(t), I_k(t)$ and $R_k(t)$ be the fraction of the susceptible, infected and recovered nodes at time t which are having degree of k. Let $p(k)$ be the degree distribution of the network, during the epidemic process, the node with degree k will be $kp(k)$ where, $k \in [1, N-1]$.

The transition rules of nodes' from one state to other state is defined as:

1. A healthy node after becoming infected moves from susceptible to infected state.
2. A node may be recovered spontaneously at any time with rate μ. Recovery of a node doesnot require any contact. μ =1 is considered for each time stamp a node will be recovered.
3. Once a node will get recovered it will never be infected nor become susceptible.

4. In addition, we are not considering demography that is birth and death of nodes, therefore, the total number of nodes will be constant through out the transition.

Now, using the mean-field equation for dynamics of epidemic on network, based on the above transitions:

$$\frac{dS_k(t)}{dt} = -\beta k S_k(t)\Omega_k(t-\tau) \tag{7}$$

$$\frac{dI_k(t)}{dt} = \beta k S_k(t)\Omega_k(t-\tau) - I_k(t) \tag{8}$$

$$\frac{dR_k(t)}{dt} = I_k(t) \tag{9}$$

Where, Eq. 7 describes the rate of change of susceptible nodes and Eq. 8 refers to rate of change of infected nodes while Eq. 9 explains the rate of change of recovered nodes. The dynamics of SIR are coupled through the function $\Omega(t)$ that describes the probability that an any given link of susceptible node connected to an infected node of degree k at time t as shown in Eq. (10,11,12). Here, the heterogeneous uncorrelated network [13] is considered, hence, $\Omega_k(t)$ can be defined as:

$$\Omega_k(t) = \sum_{k=1}^{k_{max}} P(k^{'}|k) I_k(t) \tag{10}$$

$$\Omega_k(t-\tau) = \sum_{k=1}^{k_{max}} P(k^{'}|k) I_k(t-\tau) \tag{11}$$

$$\Omega_k(t-\tau) = \frac{\sum_{k=1}^{k_{max}} k^{'} P(k^{'}) I_k(t-\tau)}{\langle k \rangle} \tag{12}$$

where $P(k^{'}|k)$ is degree-degree correlation and $I_k(t)$ is density of infected nodes at time t having degree k.

3.1 Dynamical behaviors of the model

Eq. (7 - 9) represent nonlinear dynamical system of epidemic spreading, where at any time t,

$$S_k(t) + I_k(t) + R_k(t) = 1$$

. Therefore, from Eq.7 and Eq.9

$$\frac{dS_k(t)}{dR_k(t)} = \frac{-\beta k S_k(t) \sum_{k=1}^{k_{max}} P(k^{'}|k) I_k(t-\tau)}{I_k(t)} \tag{13}$$

After setting non-negativity and boundness of solution by using [11] as $S_k(0) > 0$, $I_k(s) = 0$ for $s \in [-\tau, 0]$ and $R_k(0) = 0$ where $0 \leq \tau \leq t$. Delay(τ) should always be less than current time of spreading, because if τ is greater than t that creates negativity. integrating both side of Eq.13

$$S_k(t) = e^{-\beta k \sum_{k=1}^{k_{max}} P(k^{'}|k)\tau R_k(t)}$$

Let

$$\theta_k(t) = \frac{\sum_{k=1}^{k_{max}} k^{'} P(k^{'}) R_k(t)}{\langle k \rangle}$$

$$S_k(t) = e^{-\beta k \tau \theta_k(t)} \tag{14}$$

As epidemic arrives at steady state when $t \to \infty$ hence $I_k(\infty) = 0$. Therefore, normalized condition for steady state is,

$$S_k(\infty) = 1 - R_k(\infty)$$

$$S_k(\infty) = e^{-\beta k \tau \theta_k(\infty)} \tag{15}$$

$$R_k(\infty) = 1 - e^{-\beta k \tau \theta_k(\infty)} \tag{16}$$

Negative exponent in Eq. 15 shows that the number of susceptible nodes are decreasing and converted into recovered nodes.Therefore,

Therefore,

$$\theta_k(\infty) = \frac{\sum_{k=1}^{k_{max}} k^{'} P(k^{'}) R_k(\infty)}{\langle k \rangle}$$

$$\theta_k(\infty) = \frac{\sum_{k=1}^{k_{max}} k^{'} P(k^{'})(1 - e^{-\beta k \tau \theta_k(\infty)})}{\langle k \rangle}$$

$$= G(\theta_k(\infty))$$

where $G(\theta_k(\infty))$ is a continuous, and increasing function for $\theta_k(\infty)$,

$$\theta_k(\infty) = \frac{\sum_{k=1}^{k_{max}} k^{'} P(k^{'}(1 - e^{-\beta k \sum_{k=1}^{k_{max}} P(k^{'}|k)\tau R_k(\infty)})}{\langle k \rangle}$$

Above equation will have value 0 when $R_k(\infty) = 0$, which provides the disease-free state and the disappearance of epidemics. Therefore, to make equation with a solution, put the limit between 0 and 1 and following condition must hold,:

$$\frac{dG(\theta_k(\infty))}{d\theta_k(\infty)}\Big|_{\theta_k(\infty)=0} > 1 \tag{17}$$

After solving Eq. 17 we get

$$\beta = \frac{\langle k \rangle}{\langle k^2 \rangle \tau} > 1$$

Therefore , the critical spreading rate under heterogeneous network can defined as

$$\beta > \beta_c = \frac{\langle k \rangle}{\langle k^2 \rangle \tau} \tag{18}$$

From the Eq. 18 if $\tau \to 0$, then $\beta_c \to \infty$. To make value of β_c countable $\tau + 1$ is used in place of τ.

$$\beta > \beta_c = \frac{\langle k \rangle}{\langle k^2 \rangle (\tau + 1)}$$

If there is no infection delay ($\tau = 0$) then the delayed SIR will become the standard SIR model and the critical threshold is $\beta_c = \frac{\langle k \rangle}{\langle k^2 \rangle}$ which is similar to that in [13].

Therefore, critical threshold is inversely proportional to delay, as delay increases critical threshold decreases. If the delay is too large then epidemic will die out automatically and disease will not spread out.

4 Experimental Results

In this section, we first explain our experimental settings and next, we discuss the results of our simulations performed using *SIR with delay* model performed using three different types of underlying networking topologies.

4.1 Experimental setup

We have simulated the critical threshold of epidemic outbreak by considering the delay in the SIR using following three different types of underlying networks:

I SIR model with delay considering Random Network
II SIR model with delay considering Random Geometric Network
III SIR model with delay considering Random Geometric Network with mobile agents.

Each type of network consists of 2000 nodes that represent the human population in an area of 2500m X 2500m square region, where connecting probability of a node with other nodes is considered 0.2 for random network model($G(N, p)$. For random geometric networks with or without mobile agents, the connecting radius, r of 2m is kept for a possibility of creating a connection with other nodes in the region. The rationale behind keeping this distance small is that as generally infectious diseases like chicken pox and tuberculosis spread when two persons get in contact at a short distance. For random geometric network with mobile agents, velocity (v) of nodes has been assigned random from the range of [3, 100] km/h. The logic behind keeping the varying velocity is due to the fact that some individuals prefer to walk and others tend to move by vehicles. The expected length between two random points is (0.521*square length of simulation area)[4]. Various parameters for simulations are listed in Table 1.

Table 1 Simulation Parameter

Name of Parameter	Value
Nodes	2000
Simulation Area	2500m x 2500m
Square Length(a)	2500m
Connectivity Radius(r) (RGG)	2m
Spreading Rate(β)	0.6
Recovery Rate(μ)	0.1
Delay(τ)	[0,10,20,....100]
Connectivity probability (E-R model)	0.2
Expected length between two random point	0.521*a
Velocity(v) (Mobile RGG)	Random(3,100)

4.2 Results

We perform different simulation to explain the proposed delayed SIR on complex network by using different parameters (see Table 1). We focus on the effect of delay on the dynamics of epidemics on heterogenous networks. In simulation if $\tau = 0$, the critical threshold will become $\beta_c = \frac{\langle k \rangle}{\langle k^2 \rangle}$

4.2.1 Effect of Delay on SIR considering underlying Network as Random Network:

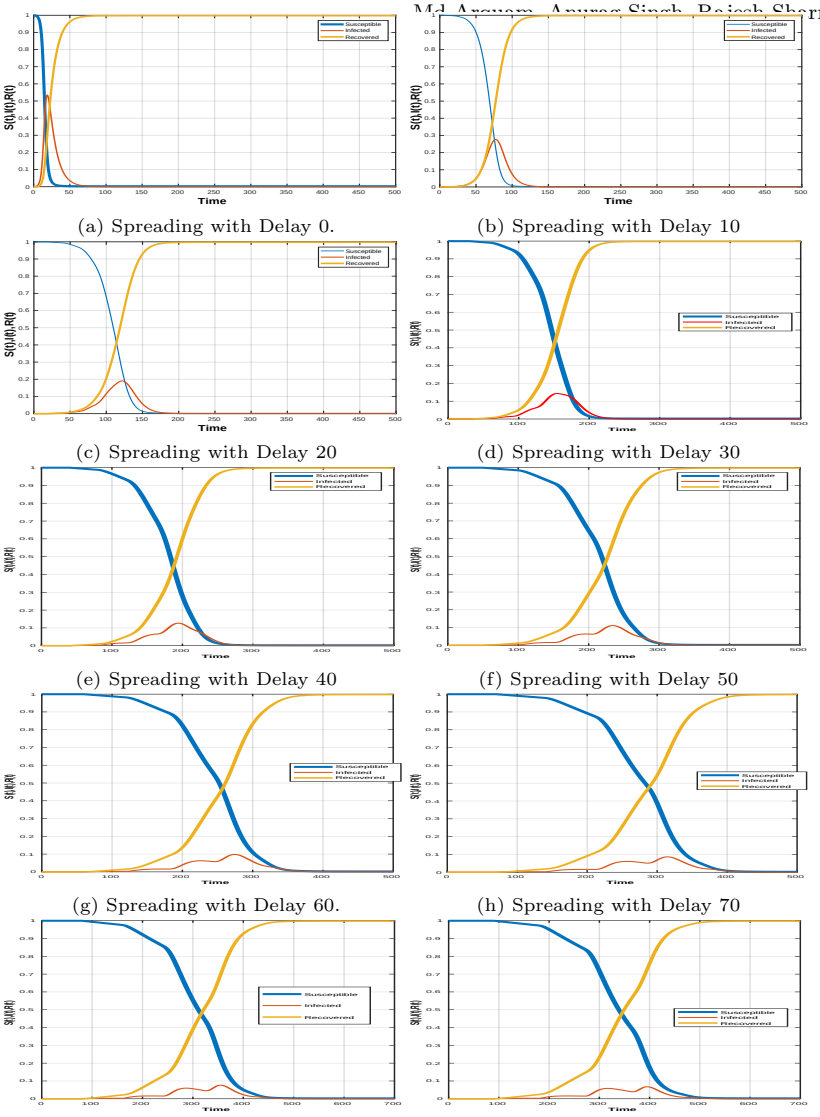
Fig. 1 shows the results of the epidemic spreading with delay in case of random networks. Fig. 1(a) to 1(j) shows the epidemic dynamics with different values of τ ($\tau = 0$ to 90, with an interval of 10). We observed that with the increase in delay, there is decrease in critical threshold but the time scale for the existance of disease increases.

4.2.2 Effect of Delay on SIR considering underlying Network as Random Geometric Network:

The results of the epidemic spreading with delay in case of random geometric networks is shown in Fig. 2. Fig. 2(a) to 2(j) shows the epidemic dynamics with different values of τ starting with τ= 0 to 90, with an interval of 10. Apart from the decrease in the critical threshold we observed that epidemic spreading in case of random geometric networks does not differ too much from random networks as the average degree of random network as well as in case of random geometric network is almost constant.

4.2.3 Effect of Delay on SIR considering underlying Network as Random Geometric Network with mobile agents:

Fig. 3 shows the results of the epidemic spreading with delay by considering random geometric networks with mobile agents. Fig 3(a) to 3(j) shows the epidemic dynamics with different values of τ starting with τ= 0 to 90, with an interval of 10. We observed that in case of random geometric networks



(a) Spreading with Delay 0.

(b) Spreading with Delay 10

(c) Spreading with Delay 20

(d) Spreading with Delay 30

(e) Spreading with Delay 40

(f) Spreading with Delay 50

(g) Spreading with Delay 60.

(h) Spreading with Delay 70

(i) Spreading with Delay 80.

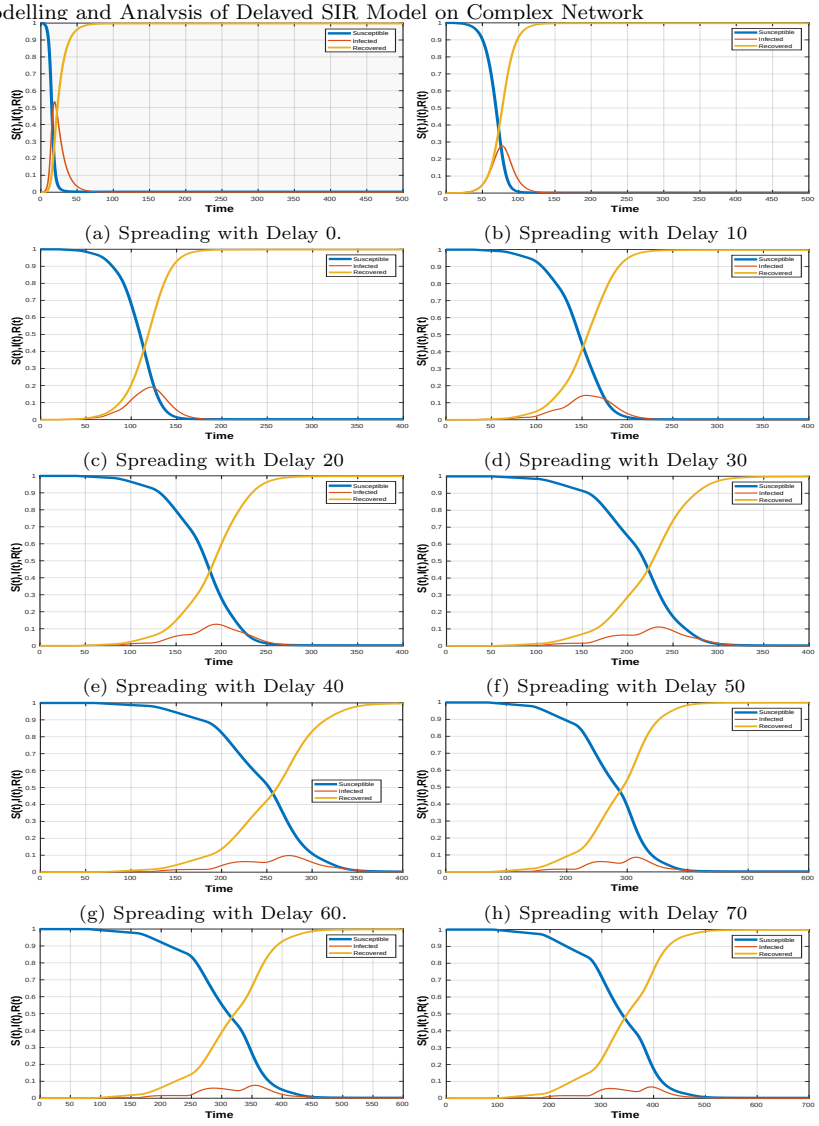
(j) Spreading with Delay 90

Fig. 1 Effect of delay in SIR considering underlying Random Network (E-R)

with mobile agents, spreading pattern is different from above two networks. In particular, the epidemic spreading varies on the node's location because the average degree of a node is stochastic in random geometric networks with mobile agents. That is the number of neighbors it comes in contact at any particular location.

4.2.4 Effect of Delay on Epidemic Threshold

It may also be seen that delay in infection decreases the critical threshold of spreading rate as shown in Fig 4. This shows that critical threshold in



(a) Spreading with Delay 0. (b) Spreading with Delay 10

(c) Spreading with Delay 20 (d) Spreading with Delay 30

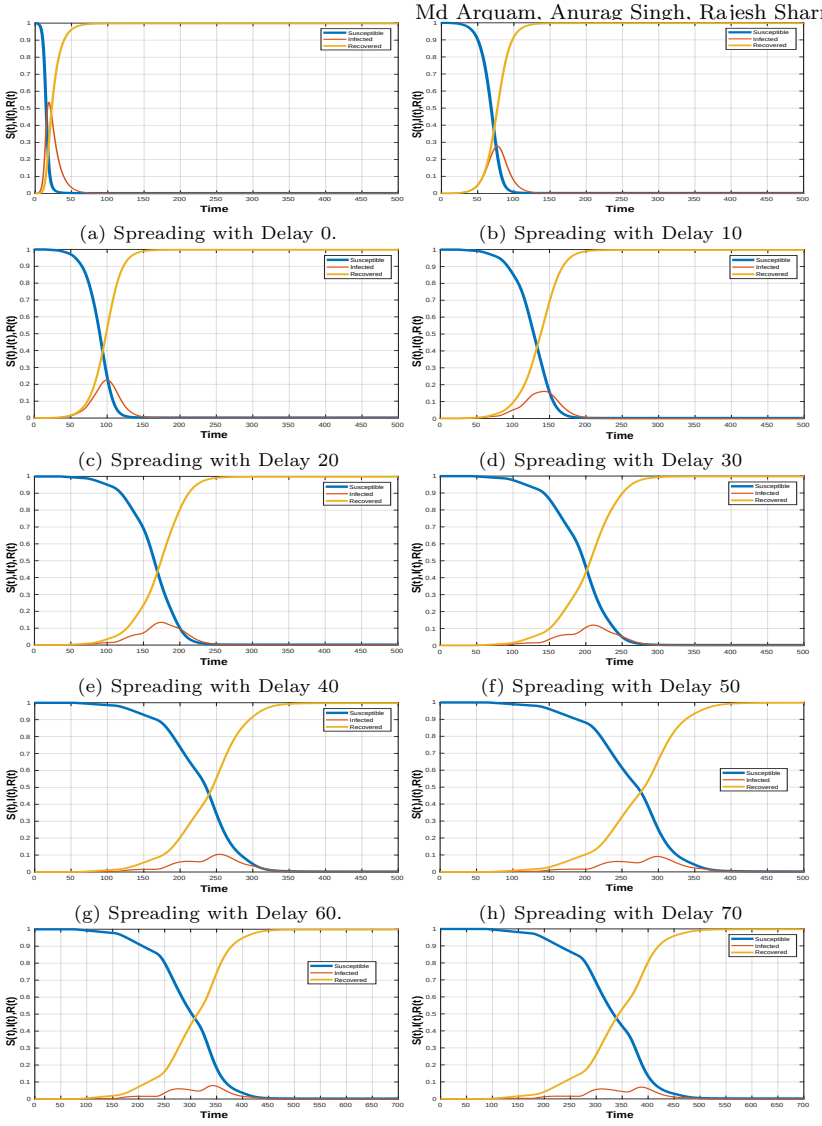
(e) Spreading with Delay 40 (f) Spreading with Delay 50

(g) Spreading with Delay 60. (h) Spreading with Delay 70

(i) Spreading with Delay 80. (j) Spreading with Delay 90

Fig. 2 Effect of delay in SIR considering underlying Random Geometric Network

SIR model is higher on a random network as compared to random geometric network with mobility and random geometric network. The critical spreading rate for having an outbreak in random graph is high, which increases with value of delay. Therefore, if the delay will be increased in the random graph then for an outbreak to happen in the network, a higher spreading rate is required. It may be concluded that higher delay may be used to stop the epidemic outbreak against the given spreading rate.



(a) Spreading with Delay 0. (b) Spreading with Delay 10

(c) Spreading with Delay 20 (d) Spreading with Delay 30

(e) Spreading with Delay 40 (f) Spreading with Delay 50

(g) Spreading with Delay 60. (h) Spreading with Delay 70

(i) Spreading with Delay 80. (j) Spreading with Delay 90

Fig. 3 Effect of delay in SIR considering underlying Random Geometric Network with Mobile agents

5 Conclusion and Future work

Classical epidemiological models unable to describe the spreading pattern of infectious diseases and effect of delay in spreading. Underlying network shows the contact pattern between human population. Delay in infection plays an important role in epidemic spreading. We obtained the spreading threshold that is inversely proportional to delay (τ). We have simulated the delayed SIR model considering 3 different underlying network as Random Network,

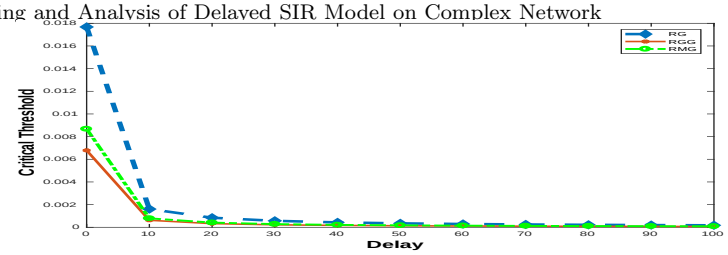


Fig. 4 Effect of delay on critical threshold on RG,RGG, RMG

Random Geometric Network with and without mobile agents. Simulations also show that delay decreases the critical threshold value of spreading rate. By considering a different delay in conversion from susceptible to infected nodes for different instances we have found that diseases persist for longer time in human population as delay increases the duration of existence of diseases. Simulation also shows that if delay is much larger then epidemic die out automatically.

We plan to include various future directions for this work. We plan to use additional dynamic networks for our future study. Another direction could be to use larger and real networks for understanding the epidemic behavior. Important, we plan to include infection delay and recovery delay simultaneously in our model, in our future studies.

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