# Modeling the Outbreak of an Infectious Disease on a Heterogeneous Network

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**Annotation**
The outbreak of infectious diseases is a global public health threat for the international community. Modelling the propagation of epidemics in a society is one of the important fields in epidemiology science. It is essential to know the number of infected cases for estimating and controlling the spread of disease in the affected countries. In this study, we used complex network theory to model the spread mechanism of epidemic disease in social networks. We modeled a social complex network by graph theory. Individuals are considered as nodes and acquaintances between them are considered as links. Disease virus can transmit along the links between nodes (people) according to different situations. In this work, we proposed a dynamic model for simulating the outbreak of infectious disease on a social network based on the susceptible, exposed, infected and recovered (SEIR) dynamical categories. It has been tried to study the heterogeneity on the network by considering two key factors in the epidemic propagation: 1) The communications weights between individuals in the network 2) different body resistances of people based on age. The proposed dynamic model was applied on a real social network which was constructed in our previous research. We compared the proposed model with two different dynamic models. Finally, the simulations were compared with the reported data of infected cases of SARS outbreak in Hong Kong in 2003. The results indicated some similarity between our proposed model and the real reported data. Based on the results, it could be concluded that considering communications weights and body resistances of people captures the dynamic of disease spread in a proper way.

*Key words*: Social network, Infectious disease, Heterogeneity, Body resistance, Communications weights.

## 1 Introduction

Complex network theory could help to understand the spread mechanism of epidemic disease in social networks [1, 2]. Graph theory is a powerful tool for modeling complex networks [3-6]. In graph theory, individuals are considered as nodes and acquaintances between individuals are considered as links [7]. Virus transmission can occur along the existing links according to different situations. Some studies indicated that human interactions are important for the spread of infectious disease [8]. In the real-word, social networks links strengths aren't equal and some interactions have a high probability of infection transmission than others, i.e. the weight of links shows the amount of communications between individuals [7].

Prevalence of infectious diseases such as Influenza, Meningitis, Pertussis, Yellow fever and etc., since the beginning of history has been a major concern of humanity. Some of them are

widely spread in the last decade: Severe Acute Respiratory Syndrome (SARS) in 2002, Methicillin-resistant Staphylococcus aureus (MRSA) in 2005 and Ebola in 2014.

Several types of research have studied the spread of infectious disease in social networks [9-13]. Some of them, considered the complex network approach in their researches. Christian L. Althaus presented mathematical modeling that was appropriate for estimate the outbreak of Ebola in West Africa in 2014. He applied SEIR model in the simulations [14]. Abdulrahman et al. presented and analyzed a model for controlling the spread of Ebola Virus Disease (EVD) in a population by SLIR dynamic model. They defined 12 parameters in their model such as rate of public enlightenment, enhanced personal hygiene due to public enlightenment, number of quarantined individuals and availability of isolation centers. Their simulations showed that improved personal hygiene and quarantining of infectious individuals are enough to control the spread of EVD [15]. Small and Tse modeled transmission of SARS in Hong Kong with complex small world network. They used SEIR dynamic model. They supposed different probabilities of transition between the states of disease in the network. In their model, random nodes are isolated in order to control the spread of infection [16]. Colizza et al. used real airline networks data for their study because they believed that people's transportations are causes of SARS disease propagation. They considered the population of each city is classified into seven different compartments, and hospitalized as well as infectious individuals are able to transmit the infection. They concluded that their models are fit for the forecast and analysis of emerging disease spreading at the global level [17].

Some scientists have attempted to produce their database through questionnaires [18]. Previously, we recorded the communications between 100 persons during one week and constructed a sample of a social network consisting human communications. It was shown that the constructed social encounters network has a small-world topology. In the next step, the common cold outbreak was modeled with SIR dynamic model [1]. In this study, we used our previous social network for modeling SARS propagation by SEIR model.

In a disease spread network, there are some sources of heterogeneity. For example, individuals' characteristics against the disease and the level of interaction between people are some bases of inhomogeneity. In this paper, we tried to apply this heterogeneity factors in the simulations. For this purpose, we proposed a dynamic model with two important features: 1) communications levels between individuals which is reflected in the connections' weights of the network 2) different body resistances based on ages of people which is used as different thresholds for each node (people). Our suggested model considers cumulative weights of all links between each node and its infected neighbors. We named this model as Cumulative Weight model (CW). This model was compared with two different dynamics. The first one is Abramson and Kuperman's model (AK) that it is based on the number of infected neighbors for each node [19]. This model doesn't consider the amount of communication between persons. The second model is Rajabi Vishkaie et al. model (R) that it's based on the largest connection weight between one node and its infected neighbors [1]. At the end, the simulation results of these models were compared with reported data of infected cases of SARS outbreak in Hong Kong in 2003 [20].

## 2 Methods

*Dataset:* Previously, for data gathering, we used the questionnaire distribution method in a small social network. In order to construct the network structure, 15 participants were asked in the questionnaires to record their encounters in a week. Moreover, they were asked to record their contact time weekly. Based on the level of contact between the persons, we assigned different amounts for edge weight in integer values from 0 to 9: more time of weekly contacts receives more scores.

The questionnaires were distributed among the author's acquaintances. These participants were asked to distribute the questionnaires between their own acquaintances (exactly those people that the participants mentioned in their own questionnaires). In this way, the network was expanded. All the participants were asked to record their encounters with their acquaintances; also, we requested participants to record the encounters between their acquaintances. There were 15 participants. The number of all people that they recorded was 100. The produced network (database network) had 326 links between persons [1]. In this study, since we used our previous database, the run time must be 7 days but it isn't long enough to see the propagation of disease. Hence, the run time must be a multiple of 7. Therefore, the run time was chosen 98 days. (We considered the run time long enough to see the changes.)

### *2.1. Basic definitions in disease transmission*

The dynamic of the disease spread governed by SEIR model (that 'S' stands for susceptible, 'E' shows exposed persons, 'I' shows Infected person, and 'R' indicates recovered or deceased person). It has reported that SARS has incubation period between 2 to 7 days (or longer) [21]. In our model, we supposed the incubation period is 7 days ($\boldsymbol{T_{exp}}$). This means that when the SARS's virus goes inside a person body (s)he goes to exposed state (E) while (s)he doesn't have disease symptoms in this period (first week). After this incubation period, the person goes to infected state (I) which has disease symptoms such as fever, cough etc. Here, we assumed the symptoms period is about 7 days. Since CDC (Centers for Disease Control and Prevention) recommends that persons with SARS limit their interactions outside the home until 10 days after their respiratory symptoms have gotten better [22], we considered infection period ($\boldsymbol{T_{inf}}$) as 17 (10+7) days which each infected person could carry the virus. After the infected period (s)he goes to recovered state (R) in which (s)he can't transmit the infection anymore (See Fig.1).

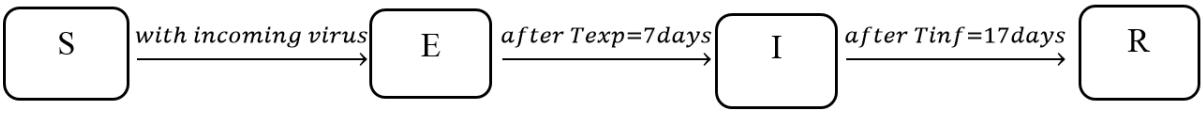


**Fig.1** Schematic of state transition by SEIR dynamic model for SARS disease, this dynamic model has four state for a society that has infectious disease: S (Susceptible), E (exposed), I (Infected) and R (Recovered)

In our network, we assumed that at the start time of the simulations, SARS viruses infected 1% of the population and before this time, nobody is sick.

We defined two time-based vectors:

$$\boldsymbol{State(t) = (State_1(t), \ldots., State_N(t))} \quad (1)$$

Where *State(t)* is a vector that indicates the conditions of each node (individuals) and *N* is the number of network's nodes.

We also defined probability vector as:

$$\boldsymbol{P(t) = (P_1(t), \ldots., P_N(t))} \quad (2)$$

Where $P_i(t)$ is a vector that I ndicates the likelihood of getting illness for the node *i* at time *t*. Generally, if the interaction between two people increases, the probability of getting illness ($P_i(t)$) will increase, too.

The states of nodes in time step t+1 (*State (t+1)*) will change according to:

$$\boldsymbol{State_i(t+1)} = \begin{cases} \boldsymbol{0\ (S)} & ; \quad \boldsymbol{P_i(t) < th} \\ \boldsymbol{1\ (E)} & ; \quad \boldsymbol{P_i(t) \geq th} \\ \boldsymbol{2\ (I)} & \\ \boldsymbol{3\ (R)} & \end{cases} \quad (3)$$

One of the important factors in the outbreak of epidemic disease is the probability of virus transmission from each infected nodes to its neighbor nodes (*q*). We assumed that *q* is between 0 and 1 with a normal distribution. Another factor in propagation is the threshold (*th*). We considered it as body resistance against the disease. If a person has more body resistance, (s)he will have a higher threshold. In this study, at first, in some simulations we considered *th* as a fixed parameter between 0 and 1 to all the individuals for obtaining results in different situations. Since the disease mortality depends on age [23], so body resistance against the virus may be different according to the people' age. Hence, in some simulations, we assigned different *th* to the network nodes based on the ages.

### *2.2. Transmission Dynamics*

We applied three different models for the dynamic of the disease spread:

1) AK model. This model is proposed by Abramson et al. and it was used for modeling infectious disease [19].

$$P_i(t) = 1 - (1 - q)^{k_{infected,i}(t)} \quad (4)$$

This model has expressed based on the number of the infected neighbors. Where $k_{infected,i}(t)$ is the number of the infected neighbors for the node *i* at time *t*, and *q* is the probability of transmission from each infected neighbor to node *i* and it is a random quantity between *0* and *1*. $P_i(t)$ is the probability of becoming ill for node i at time *t*. In simulations if $P_i(t)$ is more than *th*, node *i* will go to the E state. (*th* is the body resistance against the disease for each node.)

2) R model. This model is proposed by Rajabi et al. and it was used for modeling common cold outbreak [1].

$$P_i(t) = {}^{w_{infected,i}(t)}/_{10} \quad (5)$$

Where $P_i(t)$ is the probability of becoming ill for *i* node at time *t*. $w_{infected,i}(t)$ is the weight of the link between the node *i* and one of its infected neighbors. If the node *i* has several infected neighbors, simulations will check all of the links and if it get a connection which has a $P_i(t)$ more than *th*, the contagion transmits to the node *i* and it will go to the E state. (*th* is the body resistance against the disease for each node.) In fact, one dangerous connection (a link that its weight is more than probability *10) is sufficient to transmit the infection and the other connections will be ignored.

3) CW model. The amount of interactions between persons (*w*) and the number of infected neighbors have the main roles in disease spread. As the number of infected neighbors increase, the probability of getting illness will be increased too. Also, the probability of transmission from each infected neighbor to a susceptible node (*q*) is significant. In order to consider these factors and the heterogeneities on the network, we changed Eq. (4) (AK model) to Eq. (6). This equation comprised cumulative weights of links to each node and its infected neighbors:

$$P_i(t) = 1 - (1 - q)^{\sum w_{infected,i}(t)} \quad (6)$$

Where $P_i(t)$ is the probability of becoming ill for *i* node at time *t*. $\sum w_{infected,i}(t)$ is the sum of the edges weights between the node *i* and its infected neighbors at time *t*. If $P_i(t)$ is more than *th*, node *i* will go to the E state. (*th* is the body resistance against the disease for each node.)

## 3 Results

The diagrams of the susceptible, exposed, infected and recovered persons for three models is illustrated in Fig.2. In these three diagrams, the total number of susceptible, exposed, infected and recovered persons is equal to 100 (number of network's vertices) at each time. For all 3

dynamics, it was indicated that the number of exposed people is 1 from the first day until 7th day. During this period, the number of infected people is 0.

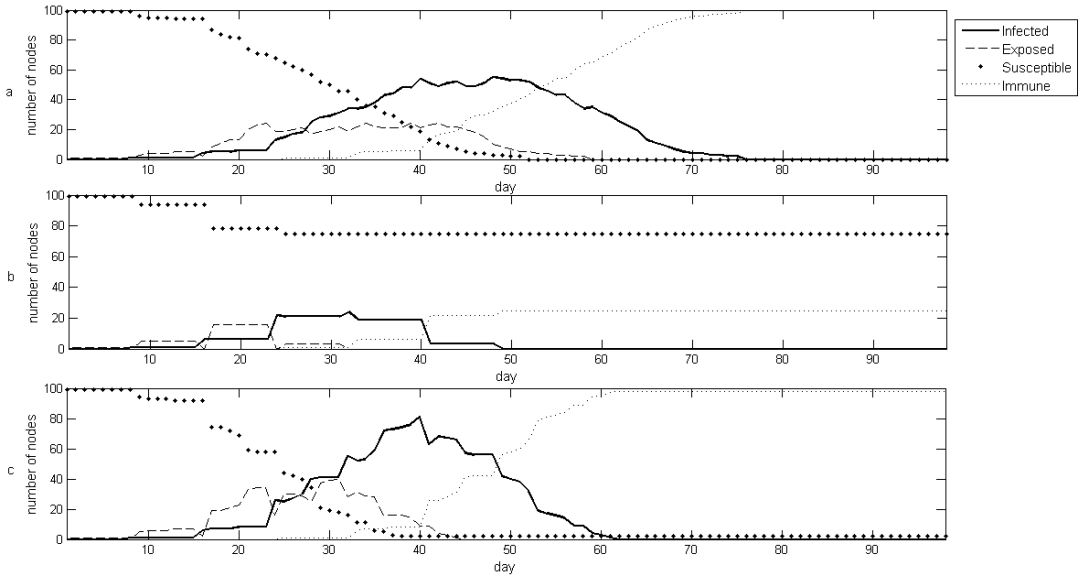


**Fig.2** Number of susceptible, exposed, infected and recovered persons for (a) AK model (Eq. (4)) with mean of the *q*=0.4 and *th*=0.5 (b) R model (Eq. (5)) with *th*=0.1 (c) CW model (Eq. (6)) with mean of the *q*=0.4 and *th*=0.5

The results of simulation for AK model with the mean of *q=0.4* and *th=0.5* are shown in Fig.2.a. As it is shown in this figure, the number of infected persons is increased from 7th day to 40 days from the beginning of the simulation, and then it will be decreased till the 46th day. After that, it will be increased till 48th day and then it will be decreased till the 77th day. The maximum number of infected persons is 55 which is reached in the 48th day. The number of susceptible persons becomes 0 at the 52nd day and it will be remained 0 until the end of run time. Therefore, all the people (100 persons in the social network) became sick in AK model.

Simulation results for R model with *th=0.1* are illustrated in Fig.2.b. As it is shown in this diagram, the number of infected persons is increased from 7th day till 32nd day of simulation cycle and then it will be decreased till the 49th day. The maximum of the infected subject is 24 in the 32nd day. The number of susceptible individuals remained in 75 from 25th day till the end of simulation cycle. In this model, 25 persons in the social network became infected.

The results of implementation for CW model with the mean of *q=0.4* and *th=0.5* are indicated in Fig.2.c. As it is shown in this figure, the number of infected people is increased from 7th day till 40th day, and then it will be decreased till 41st day quickly. Again it will increase till 42nd day, after that it will be decreased till the 62th day. The maximum number of infected people is 81 which is reached in the 40th day of simulation cycle. The number of susceptible people became 2 in the 38th day and it will remain in 2 until the end of the simulation, i.e.in CW model 98 persons became sick.

With giving different quantities to the *q* and *th* for each dynamic model, the number of susceptible people will change. The results are shown in Fig.3.

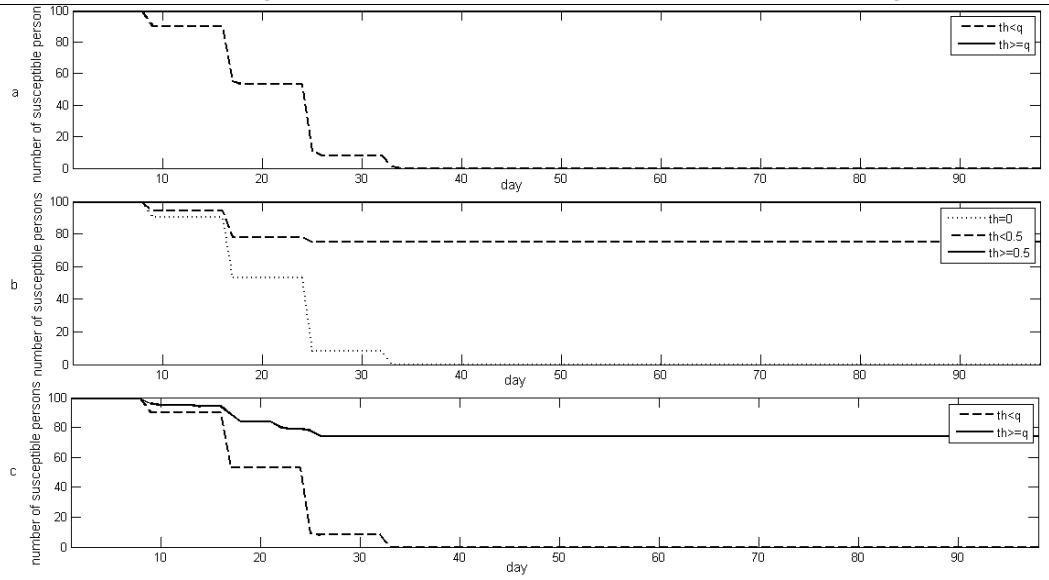


**Fig.3** (a) Number of susceptible persons for (a) AK model (Eq. (4)) when $th<q$ : $th$=0.2 & $q$=0.4 and when $th>q$ : $th$=0.8 & $q$=0.4 (b) R model (Eq. (5)) when $th<0.5$ : $th$=0.2 and when $th>0.5$ : $th$=0.8 (c) CW model (Eq. (6)) when $th<q$ : $th$=0.2 & $q$=0.4 and when $th>q$ : $th$=0.8 & $q$=0.4

In this Figure when $th \geq q$ in AK model any node won't be infected although in CW model 26 persons became ill. Also, when th<q in AK and CW model after 34 days from beginning of simulation number of susceptible people became 0. It means, in this case, all the susceptible individuals will be sick (see Fig.3.a and Fig.3.c). In Fig.3.b for R model when th=0 all the people in the network will be infected, since the number of susceptible people becomes 0 at 33rd day. In addition, when 0<th<0.5 only 25 persons became infected and it remained 25 from 25th day till the end of the simulation. Moreover, when th≥0.5 any person won't be infected.

In the real world, old people and kids have lower body resistance against the disease. In order to have a more plausible model, we consider different body resistances (*th*) in our simulations. We assign different quantities to the *th* based on the persons' ages in the network. Old people and kids were given the lower *th* and younger people were given higher *th*. The simulation results for three models in which *th* quantities are assigned based on persons' ages is shown in Fig4.

The diagram of the number of susceptible, exposed, infected and recovered people for AK model in which *th* quantities are based on individuals ages is shown in Fig.4.a. As you see the number of infected individuals is increased from 7th day till 40th day, and then it will be decreased till the 69th day. The maximum number of infected individuals is 75 which is reached in the 40th day of simulation cycle. The number of susceptible individuals became 0 in the 45th day and it will remain in 0 until the end of the simulation. Therefore, all the people in this social network became sick.

For R model as it is shown in Fig.4.b, the number of infected individuals is increased from 7th day till 56th day of simulation cycle and then it will be decreased till the 65th day. The maximum of the number of infected individuals is 8 from the 40th day till 48th day. The number of susceptible individuals remained in 89 from 41st day till the end of run time. Thus, in this model, just 11 persons in the social network became ill.

The simulation results for CW model in which *th* quantities are assigned based on ages and with the mean of $q=0.7$ is shown in Fig.4.c. As it is shown in this figure, the number of infected people is increased from 7th day till 40th day, and then it will be decreased till the 44th day. Again it is increased till 48th day and after that, it will be decreased till the 70th day of the simulation period. The maximum number of infected people is 73 which is reached in the 40th day of simulation cycle. The number of susceptible individuals became 1 in the 46th day and it will remain in 1 until the end of the simulation. So, 99 persons became infected.

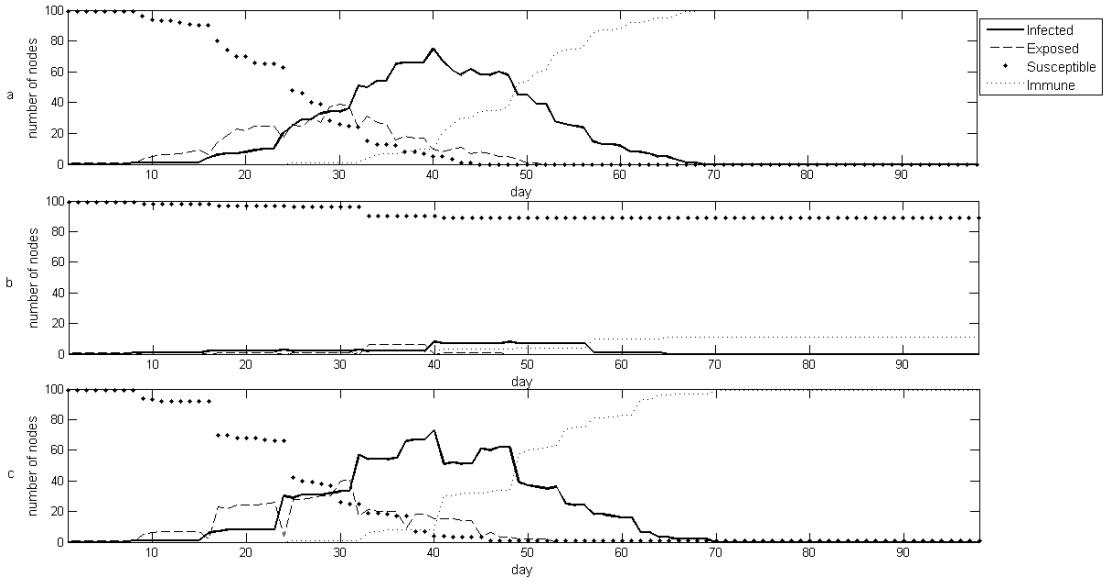


**Fig.4** Number of susceptible, exposed, infected and recovered persons giving *th* quantity based on age for (a) AK model (Eq. (4)) with mean of the $q$=0.7 (b) R model (Eq. (5)) (c) CW model (Eq.(6)) with mean of the $q$=0.7

In order to have a better comparison with real data from SARS infection data for Hong Kong since 15 February 2003 [20], the number of infected people in Fig.4 for each model is depicted in Fig.5.

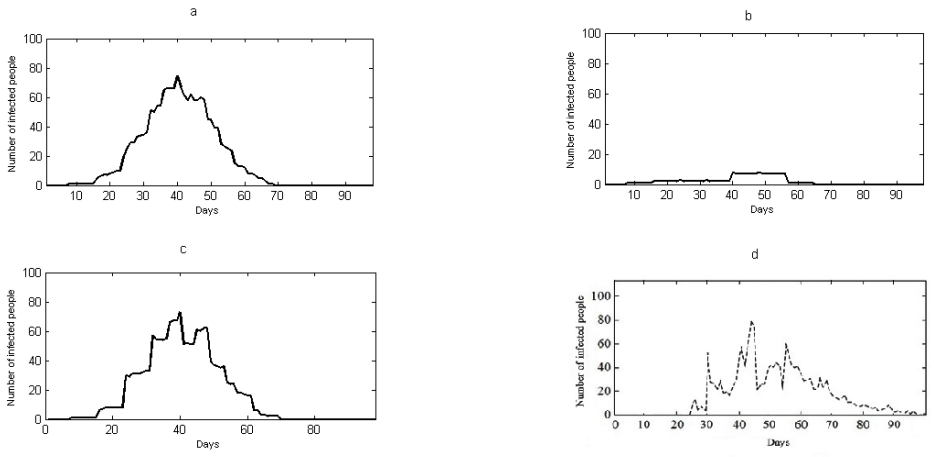


**Fig.5** Number of infected persons (a) AK model (Eq. (4)) with mean of $q$=0.7 and giving *th* quantity based on age (b) R model (Eq. (5)) and giving *th* quantity based on age (c) CW mode l (Eq. (6)) with mean of $q$=0.7 giving *th* quantity based on age (d) Daily reported SARS infection data for Hong Kong since 15 February 2003

All these diagrams have the same trend of propagation of disease. AK and CW model have higher amplitude for the number of infected people. In addition CW model has a higher slope than AK and R model.

## 4 Discussion

In this study, we examined three different dynamic models for simulating the outbreak of an infectious disease (here the SARS was modeled) on a social network. In all the simulations, at the beginning of the disease spread, 1% of people will receive disease virus, (in this study the number of nodes is 100 persons) therefore just one node will go to exposed status. According to Fig.2, in the first 7 days, there are no changes in the number of infected people. In fact, the number of infected people depends on the changing status of the first exposed person in the network. The incubation period is supposed 7 days, and the contagion couldn't transmit from the first exposed person to others in this period.

The AK model (Eq. (4)) considers these factors: the number of infected neighbors $(k_{infected,i}(t))$ and the probability of infection transmission from an infected person to its susceptible neighbors ($q$). This probability depends on the transmission rate.

In the transmission of epidemic diseases, another important factor is the weights of communications between persons [24, 25]. There are different strategies to assign a weight to a link based on the amount of communication between two nodes [26, 27]. Previously, we allocated different amounts for links weights in integer values from 0 to 10 based on the level of communication between the people and we applied this factor (*w) in* R model (Eq. (5)) [1]. According to R model, having an infected neighbor with strong communication weight is enough to become sick. In our data, most of the links have the weight equal to 1. For these links, the probability of becoming sick is equal to 0.1 (Eq. (5)) and it is not more than *th* (*th= 0.1)*. So this dynamic doesn't let the communication links with weight 1 to transmit the infection. This is the reason for the R model's result wherein a few people have been ill (see Fig.2.b). In the real-word social networks, the communications between individuals aren't equal. In order to consider heterogeneity on the network, we changed Eq. (4) (AK model) to Eq. (6) (CW model). In the proposed model the communication weights of all the infected neighbors are considered. The diagram trend of infected people in the CW model is similar to the AK because both of them consider *q*. In both of these models, almost all the people became infected. Besides, the CW model shows more fluctuations in disease propagation than the AK and R model. It may be as a result of considering the summation of communication weights.

With giving different amounts to the *q* and *th* the below results were achieved from simulations (see Fig.3). According to Fig.3.a in AK model we observe:

If $th \geq q$: any node can't be infected.

If $th < q$: all nodes will be infected.

These former statements can be obtained from the following equations:

$$th \geq q \rightarrow 1 - q \geq 1 - th \tag{7}$$

At the beginning of the disease spread just one node will be sick. So some nodes have just one infected neighbor and the remaining ones does not have any infected neighbors. So we have $k_{infected,i} = 0$ or $k_{infected,i} = 1$ .

$k_{infected,i} = 0 \rightarrow (1-q)^{k_{infected,i}} = 1,\ 1 > 1-q \overset{(7)}{\rightarrow} (1-q)^{k_{infected,i}} > 1-th \rightarrow th > 1-(1-q)^{k_{infected,i}} \rightarrow th > p_i(t)$

$k_{infected,i} = 1 \rightarrow (1-q)^{k_{infected,i}} = 1-q \overset{(7)}{\rightarrow} (1-q)^{k_{infected,i}} > 1-th \rightarrow th > 1-(1-q)^{k_{infected,i}} \rightarrow th > p_i(t)$

Therefore, node *i* doesn't become infected. It means that if $th \geq q$ at the beginning of the spread of disease, the infection does not transmit from node *i* to any node.

In R model, we observe that (see Fig.3.b):

If $th \geq 0.5$: no node will be infected.

If $th < 0.5$: some persons will be infected.

If $th = 0$: all the nodes will be infected.

In this model as *th* decreases, the probability of becoming infected will increase. In our data, the weights of links are between 0 and 10. In the beginning, there is only one infected node which its maximum weight of connected links is 5. According to Eq. (5)

($P_i(t) = w_{infected,i}(t)/10$), if $th \geq 0.5$, the infection can't transmit from first infected node to its neighbors.

In CW model, we observe that (see Fig.3.c):

If th ≥ q: some persons will be infected.

If $th < q$: all the nodes will be infected.

In an overview, when $th \geq q$ in AK model no one became sick, but in CW model some persons became sick. As CW model considers the weight of links and so it is more impressible than AK model.

In addition to aforementioned factors, the characteristics of individuals are not the same and people may have different body resistance against the disease, i.e. each person may have a particular threshold of becoming sick (*th*). For example, infection risks may be related to age. Previously Hethcote suggested that in realistic infectious disease models it would be beneficial to include the age of individuals [28]. Here we allocated different quantities for the resistance body from 0 to 1 based on the persons' age (see Fig.4 and Fig.5).

For the R model, the number of infected individuals in Fig.4 are fewer than it in Fig.2, since in our network data most of the people are young and they have high *th* (compare Fig.2 with Fig.4). And as mentioned before, the R model depends on *th* very much.

Comparing the number of infected persons in our simulated models with real data suggests that the AK and the CW showed more similarity to real result (see Fig.5). However, the CW results have more fluctuations similar to real data which is as a result of considering both q and w.

## 5 Conclusions and Future Prospect

Modeling transmission of infectious diseases in a society is one the most important field in epidemiology science. Here, we have attempted to model the transmission of SARS disease in a small population with three different models. Based on our results, weights of communications between individuals and the thresholds of body resistances of people are important factors for disease spread. The CW model which has all these factors is more similar to the reported data of infected cases of SARS outbreak in Hong Kong in 2003 [20]. Based on these results it may be concluded that the CW captures the dynamic of disease spread in a proper way.

As suggestions for the future prospect, it will be useful to model the vaccination effect of the person who has more connections with other people in a society, since in other studies it has expressed that the prevalence of the disease depends on the degree distribution [2]. Also, it has shown that quarantining infected persons can reduce the infectious in a society [16]. Considering this state in simulated models will be interesting.

We believe the model can be applied to another contagious diseases such as Ebola, influenza, AIDS and etc., because these diseases have some similar features. For example, Ebola and SARS have the analogous incubation period.

**Acknowledgment**

We want to thank Dr.Sajad Jafari for useful guidance and improvements.

## References

[1] F. R. Vishkaie, F. Bakouie, and S. Gharibzadeh(2014), "Common cold outbreaks: A network theory approach," *Communications in Nonlinear Science and Numerical Simulation,* Vol. 19, pp. 3994-4002.

[2] J. Saramäki and K. Kaski(2005), "Modelling development of epidemics with dynamic small-world networks," *Journal of Theoretical Biology*, Vol. 234, pp. 413-421.

[3] M. Newman(2010), *Networks: an introduction*: Oxford University Press.

[4] M. Kaiser, R. Martin, P. Andras, and M. P. Young(2007), "Simulation of robustness against lesions of cortical networks," *European Journal of Neuroscience*, Vol. 25, pp. 3185-3192.
[5] R. Albert(2005), "Scale-free networks in cell biology," *Journal of cell science*, Vol. 118, pp. 4947-4957.
[6] S. R. Proulx, D. E. Promislow, and P. C. Phillips(2005), "Network thinking in ecology and eVolution," *Trends in Ecology & EVolution*, Vol. 20, pp. 345-353.
[7] L. Pellis, F. Ball, S. Bansal, K. Eames, T. House, V. Isham, *et al.*(2015), "Eight challenges for network epidemic models," *Epidemics*, Vol. 10, pp. 58-62.
[8] J. M. Read, K. T. Eames, and W. J. Edmunds(2008), "Dynamic social networks and the implications for the spread of infectious disease," *Journal of The Royal Society Interface*, Vol. 5, pp. 1001-1007.
[9] S. Eubank, H. Guclu, V. A. Kumar, M. V. Marathe, A. Srinivasan, Z. Toroczkai, *et al*(2004)., "Modelling disease outbreaks in realistic urban social networks," *Nature*, Vol. 429, pp. 180-184.
[10] F. Fasina, A. Shittu, D. Lazarus, O. Tomori, L. Simonsen, C. Viboud, *et al.*(2014), "Transmission dynamics and control of Ebola virus disease outbreak in Nigeria, July to September 2014," *Euro Surveill*, Vol. 19, p. 20920.
[11] L. Danon, T. A. House, J. M. Read, and M. J. Keeling(2012), "Social encounter networks: collective properties and disease transmission," *Journal of The Royal Society Interface*, p. rsif20120357.
[12] V. Colizza, A. Barrat, M. Barthélemy, and A. Vespignani(2007), "Predictability and epidemic pathways in global outbreaks of infectious diseases: the SARS case study," *BMC medicine*, Vol. 5, p. 34.
[13] T. Yoneyama, S. Das, and M. Krishnamoorthy(2010), "A hybrid model for disease spread and an application to the sars pandemic," *arXiv preprint arXiv:1007.4523*.
[14] C. L. Althaus(2014), "Estimating the reproduction number of Ebola virus (EBOV) during the 2014 outbreak in West Africa," *PLoS currents*, Vol. 6.
[15] N. Abdulrahman, A. Sirajo, and A. Abdulrazaq(2015), "A Mathematical Model for Controlling the Spread of Ebola Virus Disease in Nigeria," *International Journal of Humanities and Management Sciences*, Vol. 3.
[16] M. Small and C. K. Tse(2005), "Small world and scale free model of transmission of SARS," *International Journal of Bifurcation and Chaos*, Vol. 15, pp. 1745-1755.
[17] V. Colizza, A. Barrat, M. Barthélemy, and A. Vespignani(2007), "Predictability and epidemic pathways in global outbreaks of infectious diseases: the SARS case study," *BMC medicine*, Vol. 5, p. 1.
[18] J. Mossong, N. Hens, M. Jit, P. Beutels, K. Auranen, R. Mikolajczyk, *et al.*(2008), "Social contacts and mixing patterns relevant to the spread of infectious diseases," *PLoS Med*, Vol. 5, p. e74.
[19] M. Kuperman and G. Abramson(2001), "Small world effect in an epidemiological model," *Physical Review Letters*, Vol. 86, p. 2909.
[20] M. Small and C. K. Tse(2005), "Clustering model for transmission of the SARS virus: application to epidemic control and risk assessment," *Physica A: Statistical Mechanics and its Applications*, Vol. 351, pp. 499-511.
[21] G. Chowell, P. W. Fenimore, M. A. Castillo-Garsow, and C. Castillo-Chavez(2003), "SARS outbreaks in Ontario, Hong Kong and Singapore: the role of diagnosis and isolation as a control mechanism," *Journal of Theoretical Biology*, Vol. 224, pp. 1-8.
[22] C. f. D. C. a. Prevention(2004), "Frequently Asked Questions About SARS".
[23] J. Gjorgjieva, K. Smith, G. Chowell, F. Sánchez, J. Snyder, and C. Castillo-Chavez(2005), "The role of vaccination in the control of SARS," *Math Biosci Eng*, Vol. 2, pp. 753-769.
[24] Y. Sun, C. Liu, C.-X. Zhang, and Z.-K. Zhang(2014), "Epidemic spreading on weighted complex networks," *Physics Letters A*, Vol. 378, pp. 635-640.

[25] K. Van Kerckhove, N. Hens, W. J. Edmunds, and K. T. Eames(2013), "The impact of illness on social networks: Implications for transmission and control of influenza," *American journal of epidemiology*, Vol. 178, pp. 1655-1662.

[26] C. Kamp, M. Moslonka-Lefebvre, and S. Alizon(2013), "Epidemic spread on weighted networks," *PLoS Comput Biol*, Vol. 9.

[27] J. Stehlé, N. Voirin, A. Barrat, C. Cattuto, L. Isella, J.-F. Pinton, *et al.*(2011), "High-resolution measurements of face-to-face contact patterns in a primary school," *PloS One*, Vol. 6.

[28] H. W. Hethcote(2000), "The mathematics of infectious diseases," *SIAM review*, Vol. 42, pp. 599-653.

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