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The relationship between human behavior and the process of epidemic spreading in a real social network

A. Grabowski[1,a] and M. Rosińska[2]

[1] Central Institute for Labour Protection, National Research Institute, 00-701 Warsaw, Poland
[2] National Institute of Hygiene, National Center for Disease Prevention and Control, 00-701 Warsaw, Poland

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Abstract. On the basis of experimental data on interactions between humans we have investigated the process of epidemic spreading in a social network. We found that the distribution of the number of contacts maintained in one day is exponential. Data on frequency and duration of interpersonal interactions are presented. They allow us to simulate the spread of droplet-/-air-borne infections and to investigate the influence of human dynamics on the epidemic spread. Specifically, we investigated the influence of the distribution of frequency and duration of those contacts on magnitude, epidemic threshold and peak timing of epidemics propagating in respective networks. It turns out that a large increase in the magnitude of an epidemic and a decrease in epidemic threshold are visible if and only if both are taken into account. We have found that correlation between contact frequency and duration strongly influences the effectiveness of control measures like mass immunization campaigns.

1 Introduction

In recent years investigations of complex networks attracted great interest among the physics community's. Various biological, technical, economical, and social systems were shown to form complex networks [1–4]. Progress in information technology has made it possible to investigate the structure of social networks of interpersonal interactions maintained over the internet [5], e.g. e-mail networks and web-based social networks or artificial communities [6,7]) as well as telephone contact networks using the mobile phone logs [8]. Further investigations allowed to demonstrate that propagation in these networks depends greatly on the dynamics of the contacts [2,6,9]. For instance, it was found that the heavy-tailed distribution of the intercontact times between susceptible and infected individuals significantly affects the spreading of computer viruses [10]. On the other hand models of infectious diseases spread [11,12] often rely on the assumptions of mixing patterns in the population. Based on finding from web-based community studies failure to capture the important social network properties could lead to poor model performance in describing an epidemic. However, studying the statistical properties of real-world social networks remains a challenge. To gain insight into the basic properties of a network, e.g. the form of degree distribution, or the network dynamics a survey has to be conducted [13]. In the recent years the duration of social contacts were investigated using different data sources, e.g. wearable active Radio-Frequency Identification Devices (RFID) were used to detect face-to-face contacts among individuals [14,15] and a multi-country survey was conducted to collect data on daily contacts of each of participants [16]. However, the properties of network structure could be different in different countries and variable in time. Efforts are therefore made to validate reconstructions of the social network based on routinely available data [17]. Moreover, models that take into account many different features of the networks become complex. They require more input data and numerical computations take longer. For these reasons it is necessary to investigate which real network characteristics result in qualitatively different outputs and therefore are important to model.

Survey data show that contact duration and contact frequency are correlated [16]. Using experimental data enables us to study how observable in real life contact frequency, contact duration, and correlation between them influence the results of a simple epidemic model. In our assessment we highlight the results related to effectiveness of control measures, e.g. coverage thresholds, which allow to prevent epidemic spread of an infectious disease. As public health decisions are often made based on such estimations omission of important effects from the models can impact on predicted parameters and thus directly influence the adopted control measures. We therefore numerically investigated the influence of human social activity on epidemic spreading in a social network.

[a] e-mail: angra@ciop.pl

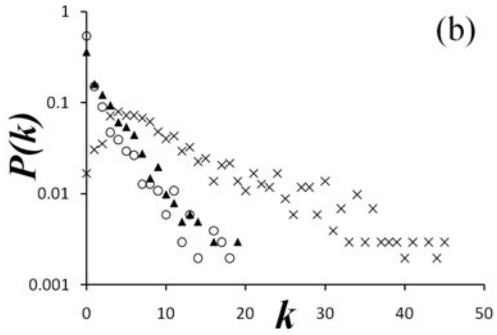

Fig. 1. Degree distribution (a) – all contacts (squares); (b) – daily contacts (crosses), rare contacts (triangles), and random contacts (circles). Results can fit exponential decay $P(k) \sim e^{-\alpha k}$ (solid line; compared with power-law – dashed line). The parameters of exponential and power-law fits were computed with maximum likelihood estimation.

2 The social network

To construct the model social network we incorporated data from contact survey conducted in Poland in the framework of POLYMOD [16]. Quota sampling was employed taking into consideration population distribution by age, sex, region and type of residence (rural, city <100 000 and city >100 000). The participants were recruited by trained interviewers visiting random households and each of participants was randomly assigned one day when they had to record all contacts. A contact was defined as a conversation in physical presence or a touch. Participants were asked to fill the total duration of all contact episodes with each contact person during the assigned day, as well as location of contacts and usual frequency of contacts with that person. Detailed description of the study is provided elsewhere [16]. In our study we aim to investigate droplet-/air-borne infections, which require physical closeness between the infected and susceptible individual, therefore we based the social network model on observed one day contact patterns, including both types of physical contacts reported in the study – conversations and touch.

The social network under investigation has a three-level structure of interpersonal interactions: (a) daily contacts (people we meet almost every day; 72.4% of all contacts); (b) rare contacts (people we meet a few times a month or less frequently; 16.3% of all contacts), and (c) random contacts (people we meet for the first time; 11.3% of all contacts). Initially the total degree distribution increases and has a maximum for $k = 5$ (squares in Fig. 1a). However for a large enough k ($k > 5$) the degree distribution has an exponential form $P(k) \sim e^{-\alpha k}$. The value of the exponent α equals 0.07, 0.09, 0.28, and 0.27 for all, daily, rare, and random contacts, respectively. The parameters of the exponential decay were computed with maximum likelihood estimation. Note that we take into account only contacts that are active for a day, the distribution of numbers of all acquaintances often have the form of a power law [5]. However in this case exponential fit gives better results (see solid and dashed lines in Fig. 1a). The small departure from the exponential curve for small k might be explained by underrepresentation of single person households in the survey [16].

Table 1. Weight distributions for different types (frequency) of contacts. Each element of the table contains the percentage of contacts of specific type (frequency) and weight (duration), e.g. 14% of all contacts are shorter than five minutes.

Weight id (value)	All	Daily	Rare	Random
1 (5/(24 × 60))	0.14	0.10	0.20	0.31
2 (10/(24 × 60))	0.18	0.14	0.26	0.34
3 (40/(24 × 60))	0.19	0.17	0.30	0.21
4 (2.5/24)	0.19	0.20	0.20	0.10
5 (4/24)	0.30	0.40	0.03	0.04

A contact's weight depends on the total time spent with a person in a day: (1) less than five minutes; (2) 5 to 15 min; (3) 15 min to 1 h; (4) 1 to 4 h and (5) more than 4 h. Table 1 presents weight distribution for different types of contacts; duration of contact is highly correlated with its frequency.

3 The model

3.1 Network model

The following generic procedure of creating a network was used. The desired number of connections of an individual k_i is drawn from experimental distribution. Initially no individuals are connected. Next, connections between individuals are created. However, each pair of individuals can be connected only once, and a new connection is added to the ith individual only when its actual number of connections is smaller than the value k_i (this value is drawn from proper distribution depending on the type of the network, see Tab. 2).

The pair of nodes that can be connected is chosen at random – i.e. we randomly choose two nodes, i and j from all nodes which actual number of connection is smaller than desired number of connection k_i. If they are not connected we create connection between them. For some or all connections (depending on the network model, see Tab. 2) we additionally apply 'clustering' procedure. This means that after creating a connection between individuals i and j, if possible a new connection is created between the ith individual and an individual randomly selected from available (i.e. having less connections than the desired number) neighbors of the jth individual (see Fig. 2). Iteration of this procedure allows to obtain the desired distribution of connectivity (actual number of connections of a node equals the value which was drown from desired distribution). In the case of dynamic network (model c and d in Tab. 2) at first daily connections are created, next rare connections and at the end random connections.

Such a procedure allows us to create a network with properties typical for social networks, e.g. small value of average shortest path and large clustering. For a

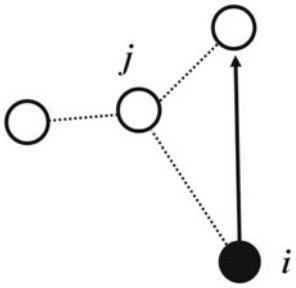

Fig. 2. After creation of connection between individuals (i and j) additional connection between ith individual and a nearest neighbor of jth individual (pointed by arrow) is added (if it is possible). This procedure increases the number of connections between the neighbors of an individual (the number of triangles in the networks). Hence, the value of the clustering coefficient increases.

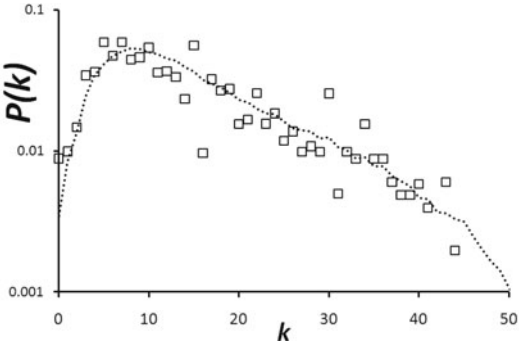

Fig. 3. Degree distribution of a static (boxes) and a dynamic (dashed line) network. In the case of the dynamic network the results are averaged over 10^3 different realizations of the network. The size of the network: 10^6 individuals.

large network with 10^6 nodes the value of the clustering coefficient is $C \approx 0.4$ and the average shortest path equals $\langle l \rangle \approx 5.6$. The clustering coefficient C of the network describes the probability that neighbors of an individual are connected

$$C = \left\langle \frac{2E_{\mathrm{i}}}{k_{\mathrm{i}}(k_{\mathrm{i}}-1)} \right\rangle \qquad (1)$$

where E_{i} is the number of connections between neighbors of the ith individual, k_i is connectivity of an individual and $\langle \ldots \rangle$ means averaging over all individuals.

The network is assortatively mixed by degree and has a hierarchical structure. This is so because in the procedure of creating the network, individuals with small values of k quickly obtain all connections. Next, individuals with a larger k cannot create connections with individuals with a smaller k – individuals with high connectivity (hubs) have to create connections between themselves, creating in this way connections between groups (highly interconnected because the value of the clustering coefficient is high) of individuals with a small k. Thus, small groups are organized into increasingly larger groups in a hierarchical manner.

Once the connection between individuals i and j has been created its weight, w_{ij}, is generated with the proper distribution $P(w)$ (the form of weight distribution depends on the type of contacts, see Table 1 for more details) – connections are symmetrical, i.e. $w_{ij} = w_{ji}$. Note that this model allows us to generate a network whose structure is based on experimental data and allows us to take into account the real distribution of contact frequency and duration. To simplify the model we do not consider changes in human behavior during an epidemic [18,19].

To investigate the influence of contact frequency we made computations for two types of network: static and dynamic. All networks include 10^6 individuals. In the case of a static network all contacts are created at the beginning of a simulation according to $P_{ALL}(k)$ distribution (squares in Fig. 1a). The network is static because its structure does not change during the simulation. In the case of a dynamic network daily contacts are created at the beginning of simulation only; rare and random contacts are re-created in each time step. The number of daily, random and rare contacts are drawn from $P_{daily}(k)$ (crosses in Fig. 1b), $P_{rare}(k)$ (triangles in Fig. 1b) and $P_{random}(k)$ (circles in Fig. 1b) distribution, respectively.

Even in the case of a dynamic network a large part of connections (daily contacts constitute 72.4% of all contacts) does not change during the simulation. At the beginning of each time step all rare and random contacts are removed and then for each individual a desired number of rare and random contacts is drawn from experimental distributions (see Fig. 1b). Next, individuals are connected according to the above-mentioned procedure. Note that the rare contacts are preferentially drawn from neighbors of neighbors due to clustering procedure, thus these contacts are more likely to be repeated over time then random contacts.

Figure 3 compares the degree distribution of static and dynamic networks. In the case of a static network degree distribution has the same form as experimental degree distribution (cf. Fig. 1a). A dynamic network changes over time, therefore degree distribution is averaged over 10^3 different realizations of the network (i.e. time steps). As a result of averaging (note that the number of individuals in the network is approximately 10^3 times greater than the number of people taking part in the experiment) the form of degree distribution is slightly different than in the case of a static network. It is worth noticing that in a dynamic network the maximal degree is higher than in the case of a static network, however the number of individuals with a high degree ($k > 45$) is very low (there is a rapid decrease in $P(k)$ for $k > 45$).

Because we have data on contact frequency and duration, we can distinguish four different models summarized in Table 2: (a) we do not consider data on contact duration and frequency (a simple static network with uniform weights); (b) we consider only data on contact duration (a static network with real weight distribution); (c) we consider only data on contact frequency (a dynamic network with uniform weight distribution); (d) we consider data on contact duration and frequency (a dynamic network with real weight distribution) – in this case correlation between contact frequency and duration are taken into account.

Table 2. The comparison of different network models.

Model	a	b	c	d
Distribution of number of contacts	P_{ALL}	P_{ALL}	P_{daily}; P_{rare}; P_{random};	P_{daily}; P_{rare}; P_{random};
clustering procedure	All connections	All connections	Daily and rare connections	Daily and rare connections
Network dynamics	None	None	Rare and random connections re-created at each step	Rare and random connections
Distribution of weights	Uniform	All (see Tab. 1)	Uniform	Daily, Rare, Random (see Tab. 1)

3.2 Epidemic spreading model

In the literature there are many models of airborne/droplet-borne epidemic spread [20–22]. However, to understand better the influence of duration and dynamics of contacts on the spreading process, we used a simple SIR model [23]. In our model, each individual is in one of three permitted states: healthy and susceptible (S), ill (I), healthy and unsusceptible or isolated from the rest of the population (R).

In SIR models based on differential equations it is often assumed that an increase in the probability that randomly chosen individual is ill P_I is linearly proportional to the number of ill individuals [24]. In a homogeneous population the process of epidemic spreading can be described with differential equations:

$$\begin{cases} \frac{dP_S}{dt} = -\beta P_S P_I \langle k \rangle \\ \frac{dP_I}{dt} = \beta P_S P_I \langle k \rangle - \gamma P_I \\ \frac{dP_R}{dt} = \gamma P_I \end{cases} \quad (2)$$

where P_S, P_I and P_R are densities of susceptible, infected and recovered individuals, respectively ($P_S + P_I + P_R = 1$) and $\langle k \rangle$ is the averaged number of neighbors (in totally mixed population $\langle k \rangle$ equals the size of population $N - 1$). In the first order approximation the probability of a susceptible to become infected in the time step $t + 1$, p_I, is directly proportional to the average number of infected neighbors in the population at the time t

$$p_I = \beta \langle k \rangle P_I(t). \quad (3)$$

In case of heterogeneous networks, mixing patterns vary and a microscopic (i.e. taking into account local interactions) version of the above equation is used relating the probability of infection not to all infected in the population, but to infected in the immediate neighborhood (Eq. (2)). We consider an initial state such that each individual is assigned one of the three states, susceptible, infected or recovered. The probability of acquiring the infection by a susceptible individual in the following time step (one day) is then directly proportional to the number of infected neighbours. However, to distinguish the effectiveness of interactions between individuals we take into account the weights of connections w_{ij}. For a susceptible individual i the probability of infection in one time step, p_i is given by:

$$p_i = \beta \sum_{j}^{k_i^I} w_{ij} \quad (4)$$

where β is probability of infection per day of contact, k_i^I is the number of ill neighbors of the ith individual. The probability of transition between states I and R (recovery rate) equals γ.

To investigate the influence of weights of connections on the spreading process we made computations for two distributions of weights, real and uniform $w_{ij} = \text{const}$. To obtain more comparable results the average weight was the same in both distributions. Note that for $w_{ij} = \text{const}$. the probability p_i increases linearly with increasing k_i^I ($p_i = \beta k_i^I \langle w \rangle$).

4 Results

Computations were performed for initial conditions with one ill (I) randomly located individual and the rest of the population healthy and susceptible (S). To investigate the dynamics of the spreading process and the magnitude of an epidemic, we introduced two observables: the time t_{max} when the maximal number of ill individuals is reached (i.e. peak timing) and the magnitude of the epidemic (i.e. final attack rate), V, defined as the proportion of individuals who had the disease during the epidemic. Figure 4 illustrates the relation between the control parameter β describing a disease and the observables V and t_{max}. We compared the model outcomes for the four cases defined above.

For the static network the results are approximately the same for real and uniform weight distribution. In comparison to static networks in the dynamic network (with uniform weight distribution) the epidemic spreads slightly faster (lower values of t_{max}) and the magnitude of the epidemic V is slightly larger. However, if both the real weight and the real frequency distribution (black triangles in Fig. 4), are considered the differences are much greater, the epidemic spreads faster and the magnitude of epidemic is much larger, e.g. for $\beta = 0.5$, $V = 0.5$ and $V = 0.7$ for static and dynamic networks, respectively. Notably the epidemic threshold is also much lower [9,24–26]. Note that in the SIR model epidemic threshold depends on β and γ, however in our case γ is fixed. Therefore we consider only the value of β. For β below epidemic threshold V is independent on the size of the system N, for β greater than epidemic threshold V is proportional to N. In other words epidemic threshold is a critical value of β above which a virus may spread, and below which it cannot. The epidemic threshold equals approximately $\beta = 0.25$, if correlation between contact frequency and

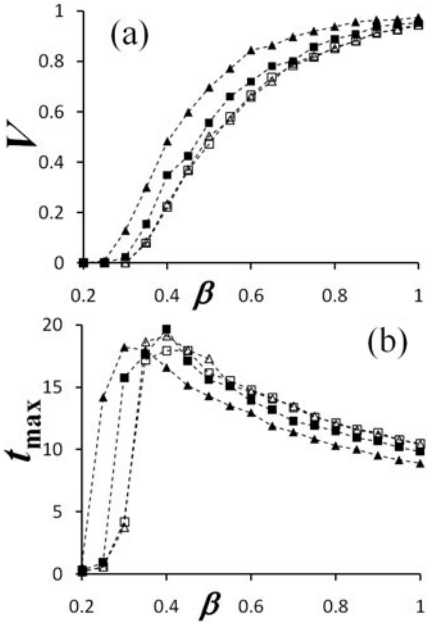

Fig. 4. Influence of the parameter β on the average magnitude of the epidemic V (a), the time t_{max} (b) for different types of networks (static – white markers, dynamic – black markers) and different distributions of weights (real triangles, uniform – squares). Recovery rate equals 1. Results were averaged over 10^4 independent simulations. The size of the network: 10^6 individuals.

duration is taken into account (black triangles) and approximately 0.3 in the other cases.

The increase in epidemic size and efficiency of spread when including the dynamically changing random contacts is expected. This procedure effectively increased the probability of emergence of secondary outbreaks in distant parts of the network. However, an increase in magnitude of the epidemic is observed if we account for the presence of frequent and long-lasting contacts between individuals (i.e. daily contact longer than at least four hours), but only in presence of dynamically changing rare and random contacts. These contacts constitute the core of the network and their presence increases the probability of infection due to the large weights assigned to them (Eq. (4)). As a result these connections are efficient in the process of epidemic spread even for lower values of the parameter β and therefore the epidemic threshold decreases.

Figure 5 shows that for lower values of the recovery rate the results are similar. A large increase in the magnitude of the epidemic (and a much lower value of the epidemic threshold) is visible only if correlation between contact frequency and duration is considered (a dynamic network with a real distribution of weights – black triangles). Note that the lower recovery rate the greater influence of rewiring on epidemic spreading. For $\gamma = 1$ the structure of the network does not change during the time when a node is in the state I, however for $\gamma = 0.1$ the neighborhood of a node changes in average ten times during it is in the state I. As result the difference between results for dynamic and static network is slightly larger

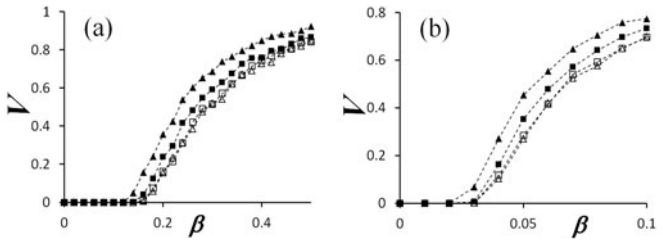

Fig. 5. Influence of the parameter β on the average magnitude of the epidemic V for recovery rate equal (a) 0.5 and (b) 0.1, respectively. Results were averaged over 10^4 independent simulations. The size of the network: 10^6 individuals.

for low values of γ (cf. Figs. 4a and 4b). Note that the rewiring process is limited to the rare and random contacts only (72.4% of all contacts, i.e. daily contact, do not change during simulation), hence in our case the influence of rewiring on epidemic spreading is relatively small.

In contrast to our results inclusion of human dynamics in communication network resulted in a slower rumor spread. The influence of human social activity on dynamic phenomena in complex networks is likely to depend on type of spreading process, e.g. spreading is accelerated in the case of epidemic and decelerated in the case of rumor spreading [6,8]. In reference [8] the process of rumor propagation in mobile communication network is investigated on the basis of SI model (i.e. recovery rate equals 0). Note that there are some differences in the network structure. The mobile communication network consists of highly interconnected communities – connections inside community are strong. Different communities are connected by weak connections. Such property is not observed in our network. Moreover in the case of mobile communication network the strongest connection is approximately 10^6 times stronger than the weakest connection – therefore there is very large increase in weight of connections between communities after averaging. In our case the strongest connection is only 48 times greater than the weakest connection. These differences cause the influence of real distribution of weights to have opposite effect.

Finally, it should be noted that the considered dynamics of a network may directly impact the effectiveness of control measures such as mass immunization campaigns. Figure 6 shows the relationship between the magnitude of the epidemic V and the proportion of individuals vaccinated at initial time N_R (i.e. the relative number of individuals who are in the state R in the time $t = 0$) for $\beta = 0.5$. Figure 6a shows results for random vaccination (vaccinated individuals were randomly chosen) and Figure 6b shows results for target vaccination (individuals with highest value of k were vaccinated – in the case of dynamic networks the number of daily contacts were taken into account).

With an increase in the number of preventively vaccinated individuals N_R, there is a decrease in the rate of infection spreading (the time t_{max} increases). This is so because an epidemic cannot spread freely in the presence of vaccinated individuals. However, for the critical value $N_R = N_{RC}$ there is an abrupt decrease in the magnitude

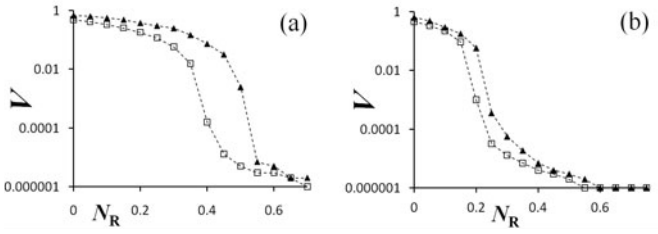

Fig. 6. Relationship between the average magnitude of the epidemic V and the number of preventively vaccinated individuals N_R for different types of networks (static with uniform distribution of weights – squares, dynamic with real distribution of weights – triangles). Recovery rate equals 1. Results were averaged over 10^4 independent simulations. The size of the network: 10^6 individuals. (a) random vaccination; (b) target vaccination (individuals with highest value of k were vaccinated).

of the epidemic V: the epidemic is suppressed. This phenomenon describes vaccinated populations for many diseases and is known as herd immunity [27]. It can be regarded as a phase transition. Such phase transitions are observed in percolating systems [24,28,29]. In the model accounting for real distribution of weights and frequency of contacts herd immunity level for random vaccination is much higher (i.e. a much larger number of individuals have to be vaccinated to suppress the epidemic). This is of note given that herd immunity level is often used as target vaccination coverage in planning mass immunization campaigns.

Simulating effects of immunization targeted to individuals with the largest contact networks the herd immunity level (N_R) is generally lower than for random vaccination (see Fig. 6b), 0.2–0.3 as compared to 0.4–0.5, implying greater efficiency of this targeted approach. Additionally, the difference between the static and dynamic network is not as striking although still the heard immunity level remains higher for the dynamic network with real life weight distribution. The coverage of approximately 0.2 targeted to individuals with the largest number of contacts implies vaccinating individuals with 10 or more contacts (data not shown). In practical terms this translates to vaccinating school children and teenagers (aged 7–18) and adult population working or studying in larger teams.

5 Conclusions

We have found that human dynamics significantly influences the dynamic processes in a social network. We investigated the process of epidemic spreading in a social network numerically. Our results indicate that on their own weight distribution or contact frequency slightly influence the process of epidemic spreading, and this influence decreases for lower values of the recovery rate. A large increase in the magnitude of the epidemic V is visible if and only if both are taken into account. Similarly, a decrease in the epidemic threshold is visible if and only if correlation between contact frequency and duration is considered. The presence of frequent and long-lasting contacts enhances the propagation process, increases the magnitude of the epidemic as well as herd immunity level. Hence, to build more plausible models of dynamic phenomena in complex networks the correlation between contact frequency and duration should be taken into account.

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