THE SIS MODEL OF EPIDEMIC SPREADING IN A HIERARCHICAL SOCIAL NETWORK*

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Dedicated to Professor Andrzej Fuliński on the occasion of his 70th birthday

The phenomenon of epidemic spreading in a population with a hierarchical structure of interpersonal interactions is described and investigated numerically. The SIS model with temporal immunity to a disease and a time of incubation is used. In our model spatial localization of individuals belonging to different social groups, effectiveness of different interpersonal interactions and the mobility of a contemporary community are taken into account. The structure of interpersonal connections is based on a scale-free network. The influence of the structure of the social network on typical relations characterizing the spreading process, like a range of epidemic and epidemic curves, is discussed. The probability that endemic state occurs is also calculated. Surprisingly it occurs, that less contagious diseases has greater chance to survive. The influence of preventive vaccinations on the spreading process is investigated and critical range of vaccinations that is sufficient for the suppression of an epidemic is calculated. Our results of numerical calculations are compared with the solutions of the master equation for the spreading process, and good agreement is found.

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1. Introduction

The structure and the dynamics of complex networks have been extensively investigated in recent years [1–11]. It was found that many real-world networks, like the web of human sexual contacts [1], e-mail networks [2], or

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Internet [3], have similar properties. They are called scale-free networks, because the probability that the number of k links connected to a node equals $P(k) \sim k^{-\gamma}$ [4]. Many authors have used this type of complex network to model a network of social contacts [9], [12–16]. In particular complex networks with a hierarchical structure, corresponding to the real structure of human communities, were studied [7,17–19], *e.g.* an epidemic spreading in a population with a two-level structure of interpersonal interactions was analyzed in Ref. [20]. A small average shortest path between nodes (individuals) and a high value of the clustering coefficient [5,6], *e.g.* the probability that "a friend of my friend is my friend" in the community is high, are the most important properties of social networks. These properties are typical for the structure of a social network and they have a strong influence on dynamical phenomena in the population.

The spreading of epidemics has been investigated by many authors with different models of interpersonal interactions [21–28]. In our work we investigate an epidemic spreading in the human population, treated as a scale-free network, taking into account spatial localization of individuals, with a three-level hierarchical structure of interpersonal interactions on the basis of SIS model [29]. Similar problem, in the frame of SIR model, were investigated earlier in Ref. [16].

We assume that each individual belongs to some social groups [7]: from the smallest one (*e.g.* family or friends), to a large one (*e.g.* community of the whole city). Interpersonal interactions among individuals in the same group are stronger than interactions among individuals from different groups. The smaller the group, the stronger an individual's influence on the other individuals in that group. From the point of view of the spreading of an epidemic most effective are social connections with the family, close friends *etc.*, however, random contacts with unknown individuals are important too. Such a random contact is most probable for individuals, who live (or work) in this same place, *e.g.* in the same building. On the other hand a contemporary community is very mobile; therefore, there is a nonzero probability of contact between two arbitrarily chosen individuals from a population. Such contact can occur *e.g.* commuting, in the cinema or in another public place, and can results in an infection of a new individual. In our model we take into account this hierarchical structure of a social network, with interpersonal connections between neighbors and contacts between random individuals referring to the mobility of a community. The hierarchical structure of interpersonal interactions described in the present paper seems to be more plausible for modeling real social networks.

This article is organized as follows. The model of a network of human contacts and probabilities of infection depending on the type of social contact, as well as the master equation, are described in Section 2. The results,

like the influence of structure of social network and preventive vaccination on spreading process, are described in Section 3. Also, in that section the probability that endemic states of epidemic occurs, depending on the parameters describing a disease (*e.g.* time of incubation), is calculated. Results obtained from numerical model and results obtained from solution of the master equation are compared in Section 4, and summarized in Section 5.

2. The model

In our model each individual has one of four permitted states: healthly and susceptible (S), infected (IN), ill (IL), healthy and unsusceptible or isolated from the rest of the population (R). The state of the individuals evolves in time and depends on their previous state and the connections or random contacts with other individuals. The probabilities of transitions between different states in one time step are described with the following parameters: $W_{\mathrm{S}\to\mathrm{IN}}$, the probability that a susceptible individual will be infected by an ill individual (it also denotes how contagious the disease is); $W_{\mathrm{IN}\to\mathrm{IL}}$, the probability that infected individual become ill (this value is connected with the average time of incubation); $W_{\mathrm{IL}\to\mathrm{R}}$, the probability that an ill individual will recover or be isolated from the rest of the population (*e.g.* in a hospital); $W_{\mathrm{R}\to\mathrm{S}}$, the probability that an unsusceptible individual lose its immunity and became healthy and susceptible (this value may be referred to the probability of the mutation of the pathogen).

The spreading process in a population can be treated as a nonstationary process, which is described by the master equation, and that approach was applied in a number of studies [14, 24, 29–33]. The results obtained in our model will be compared with the solutions of this equation in section 4. For the present case the changes in time of the probabilities $P_{\mathrm{X}}(t)$ that an individual is in one of the possible states X (where X = S, IN, IL or R) are described with the master equation

$$\begin{cases} \frac{dP_{\mathrm{S}}(t)}{dt} = W_{\mathrm{R}\to\mathrm{S}}P_{\mathrm{R}}(t) - W_{\mathrm{S}\to\mathrm{IN}}P_{\mathrm{IL}}(t)P_{\mathrm{S}}(t)\,, \\ \frac{dP_{\mathrm{IN}}(t)}{dt} = W_{\mathrm{S}\to\mathrm{IN}}P_{\mathrm{IL}}(t)P_{\mathrm{S}}(t) - W_{\mathrm{IN}\to\mathrm{IL}}P_{\mathrm{IN}}(t)\,, \\ \frac{dP_{\mathrm{IL}}(t)}{dt} = W_{\mathrm{IN}\to\mathrm{IL}}P_{\mathrm{IN}}(t) - W_{\mathrm{IL}\to\mathrm{R}}P_{\mathrm{IL}}(t)\,, \\ \frac{dP_{\mathrm{R}}(t)}{dt} = W_{\mathrm{IL}\to\mathrm{R}}P_{\mathrm{IL}}(t) - W_{\mathrm{R}\to\mathrm{S}}P_{\mathrm{R}}(t)\,. \end{cases} \tag{1}$$

This simple analytical model has one serious disadvantage — it does not take into account the structure of interpersonal interactions in the human population which is the important part of our model, where the population and its structure are described as follows. The population consists of $N = L \times L$ individuals S_{ij} localized by the indices i, j in a two-dimensional

lattice. Connections and random contacts between individuals have a hierarchical structure. The connections of each individual with k neighbors is the first level of interpersonal interactions (see Fig. 1(a)). All connections are symmetrical and have the same value. We have assumed that the network of social connections is scale-free, *i.e.* the distribution of connectivity of individuals has the form $P(k) \sim k^{-\gamma}$ ($\gamma = 3$ was used in most of computations), with k generated from the range $(k_{\min}, k_{\max})$. Initialy all individuals are not connected. Next, the connections between individuals are created with the probability $P(l)$, depending on the distance l between individuals S_{ij} and S_{nm}, where $n = i \pm l_1$; $m = j \pm l_2$ (l_1, l_2 are two independent random variables and the sign is generated with the probability 0.5):

$$P(l) \sim \frac{1}{1 + \exp\left[(l-a)/b\right]} + 0.01\frac{L-l}{L}\,. \tag{2}$$

The second term in Eq. (2) causes $P(l)$ to reach zero slowly enough. Hence, the network of the first level connections has small-world properties. The whole population is divided into local groups of $G = L_{\rm G} \times L_{\rm G}$ individuals, where the size of those groups is connected with the parameters $a = L_{\rm G}$ and $b = L_{\rm G}/4$ of the distribution (2). Thus, most connections are created between individuals located in the same local group. The structure of the network from the point of view of a certain individual is depicted in Fig. 1(a). Having created the connection between S_{ij} and S_{nm}, the connections between the individual S_{nm} and each neighbor of the individual S_{ij} are created with the probabilities $p_{\rm c}$ (Fig. 1(b)). Similarly, new connections

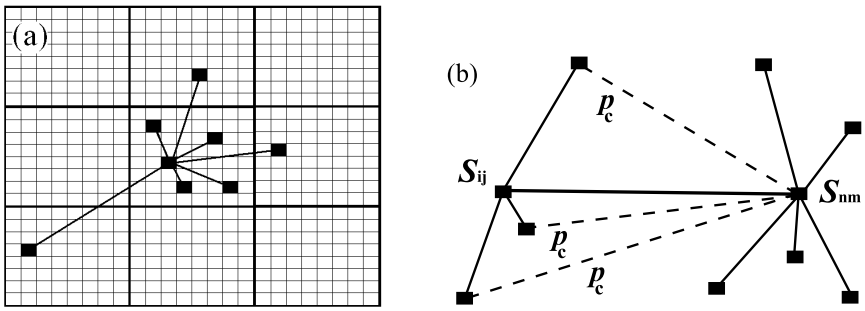

Fig. 1. An example of a network with $L = 24$ and $L_{\rm G} = 8$ (nine local groups) from the point of view of the $S_{11,13}$ individual, who is connected with $k_{11,13} = 7$ neighbors and four of those connections are located in its local group (a). When a connection between two individuals S_{ij} and S_{nm} is created (solid line), the individual S_{nm} is connected with the neighbors of the individual S_{ij}. Next, the connections between the neighbors S_{nm} and the individual S_{ij} are created (this is not shown in the figure). Each new connection (dashed line) is created with the probability $p_{\rm c}$ (b).

between S_{ij} and the neighbors of the individual S_{nm} are created, also with the probabilities p_{c}. However, each pair of individuals can be connected only once, and a new connection is added to each individual only when its actual number of connections is smaller than the value k_{ij} (where $i, j = 1, 2, ..., L$) generated with the distribution $k^{-\gamma}$. In this way desirable distribution of connectivity is obtained. It should be noted that in this procedure the value p_{c} influences the clustering coefficient C of the network [4, 34]

$$C = \left\langle \frac{2E_{ij}}{k_{ij}(k_{ij}-1)} \right\rangle , \tag{3}$$

where E_{ij} is the number of connections between neighbors of the ij-th individual. For $p_{\mathrm{c}} > 0$ the relation between clustering coefficient of an individual and its connectivity has a form $C(k) \sim k^{-\beta}$ [17].

To distinguish the effectiveness of interactions between individuals in different levels of a hierarchy we introduce three equations describing the probabilities of infection. Let us describe the effectiveness of first-level connections in the spreading of epidemics. Taking into account that interpersonal connections are a more effective way of spreading of the epidemic than random contacts, we assume that the probability of an infection of an individual by one of k neighbors is a simple nonlinear function of the number of ill neighbors [23] and has a form:

$$p_1 = W_{\mathrm{S}\to\mathrm{IN}} \sqrt{\frac{k_{\mathrm{IL}}}{k}} , \tag{4}$$

where k_{IL} is the number of neighbors in the state (IL). Note that, p_1 increases faster for a lower value of k_{IL}.

The second level of interpersonal interactions is random contacts between individuals in the same local group of G individuals. They are most probable for the individuals living (or working) in this same place *e.g.* in the same building. In our model we assumed that the probability of infection resulting from such a random contact is proportional to the power two of the probability that an individual from the local group is ill:

$$p_2 = W_{\mathrm{S}\to\mathrm{IN}} \left(\frac{G_{\mathrm{IL}}}{G} \right)^2 , \tag{5}$$

where G_{IL} is the number of ill individuals in a local group.

The third level of interpersonal interactions is random contacts between pairs of individuals who do not know each other and are chosen arbitrarily from the whole population. The probability p_3 of infection caused by such

a contact does not depend on the localization of the individuals and we assume it in the form:

$$p_3 = W_{\mathrm{S}\to\mathrm{IN}} \left(\frac{N_{\mathrm{IL}}}{N}\right)^2 , \tag{6}$$

where N_{IL} is the number of ill individuals in the whole population. The nonlinear factors in Eqs. (5), (6) cause the probabilities p_2 and p_3 to initialy increase very slowly and became significant for a great number of ill individuals.

It can be seen that from the point of view of each individual its interpersonal interactions have a hierarchical structure and they can be divided into three levels: k neighbors, individuals from the same local group and individuals from the rest of the population. Note that, as results from Eqs. (4)–(6) the probabilities p_1, p_2, p_3 of an infection of each individual depend nonlinearly on the number of ill individuals and their localization in one of the abovementioned levels. This is why the probability of an infection of a certain individual is greatest when an ill individual is one of its k neighbors, it is smaller when an ill individual belongs to the same local group and it is smallest when an ill individual is located somewhere in the rest of the population. Other probabilities of a transition between states X,Y are described by the parameters $W_{\mathrm{X}\to\mathrm{Y}}$, as in the master equation (Eq. (1)).

3. Results

Computations were performed for the initial conditions with one ill (IL) and randomly located individual and the rest of the population healthy and susceptible (S). Synchronous dynamics with assumption that an individual can change its state only ones in each time step was used. Because there are three equations describing the probabilities of infection, at first we check the influence of individuals in the first level (p_1), next the influence of the individuals in the second level (p_2) and at the end the influence of the individuals in the third level of a hierarchy (p_3).

Fig. 2 shows the influence of the localization of the source of infection in one of three levels of interpersonal interactions on the number of newly infected individuals as a function of time (epidemic curves). It can be seen that the number of newly infected individuals resulting from connections with k neighbors is approximately ten times greater for times $30 < t < 50$ than in the case of random contacts. In the first stage of the epidemic, new infections result from the interactions with k neighbors (probability p_1), whereas the possibility of infection resulting from random contacts (p_2 and p_3) becomes significant when the number of ill individuals is large enough.

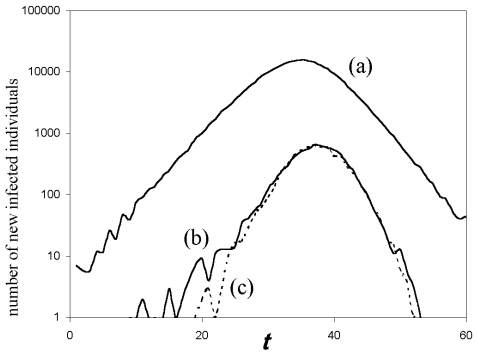

Fig. 2. Epidemic curves (the number of newly infected individuals per time step) as a function of time for different types of interpersonal interactions. Infections resulting from interpersonal connections — curve (a); infections resulting from random contacts with the individuals from a local group — curve (b); infections resulting from random contacts with individuals from the rest of the population — curve (c). The values of the other parameters are: $\gamma = 3$, $L = 500$, $L_{\mathrm{G}} = 20$, $W_{\mathrm{S}\to\mathrm{IN}} = 0.5$, $W_{\mathrm{IN}\to\mathrm{IL}} = 0.5$, $W_{\mathrm{IL}\to\mathrm{R}} = 0.5$, $W_{\mathrm{R}\to\mathrm{S}} = 0$, $k_{\min} = 8$ and $k_{\max} = 24$.

In our model it is possible to investigate the influence of the value of the clustering coefficient C (Eq. (3)) on the spreading process by changing the value of p_{c}. This problem was also discussed in earlier papers [11, 28]. The progress of the epidemic depends significantly on the value of C. As is shown in Fig. 3, the greater C, the greater the time $t_{\max}$, in which the

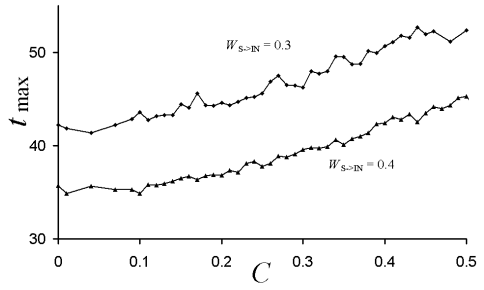

Fig. 3. The time $t_{\max}$ in which the number of ill individuals reaches a maximum value as a function of the clustering coefficient C for different values of $W_{\mathrm{S}\to\mathrm{IN}}$. Results were averaged over 1000 independent simulations — standard deviation is less than ten percent. The values of the other parameters are: $\gamma = 3$, $k_{\min} = 8, k_{\max} = 24, W_{\mathrm{IN}\to\mathrm{IL}} = 0.5, W_{\mathrm{IL}\to\mathrm{R}} = 0.5, W_{\mathrm{R}\to\mathrm{S}} = 0, L = 100, L_{\mathrm{G}} = 20$.

number of ill individuals reaches its maximum value, $w_{\max} = N_{\mathrm{IL}}(t_{\max})/N$. Moreover, the value of this maximum decreases with increasing C. Higher values of C cause the number of individuals grouped in clusters of highly

connected nodes increases. When one ill individual appears in a cluster, first individuals from that cluster are infected and next the infection spreads outside the cluster. This slows down the spreading process in the whole population. The final number V of the individuals which were infected at least one time before the epidemic dies out (*i.e.* the range of the epidemic) is not influenced by C for the case $W_{\mathrm{R}\to\mathrm{S}} = 0$, but in the case $W_{\mathrm{R}\to\mathrm{S}} > 0$ range of epidemic slightly decreases with increasing C.

Another important parameter is the time of incubation proportional in our model to $\tau = 1/W_{\mathrm{IN}\to\mathrm{IL}}$. In the case when $W_{\mathrm{R}\to\mathrm{S}} = 0$ it was found that the range of the epidemic V is influenced by τ^{-1} *i.e.* the higher the value τ^{-1}, the greater the range V, especially when the value $W_{\mathrm{S}\to\mathrm{IN}}$ is low and $W_{\mathrm{S}\to\mathrm{IN}} < W_{\mathrm{IL}\to\mathrm{R}}$. However, for high enough values of $W_{\mathrm{S}\to\mathrm{IN}}$, the range of the epidemic does not depend on time τ. On the other hand, the duration of the epidemic T and $t_{\max}$ decreases with the decreasing time of incubation — the epidemic spreads more rapidly. The maximal number of ill individuals $w_{\max}$ increases with the increasing parameter τ^{-1}. In the case $W_{\mathrm{R}\to\mathrm{S}} > 0$ the behavior of the system is more complicated because the endemic state of epidemic can occur with probability P_{E} (we define P_{E} as a probability that after 10^4 time steps the number of ill or infected individuals is greater than zero). It was found that the longer time of incubation τ, the greater probability P_{E}. When τ is small the epidemic spreads rapidly and almost whole population is quickly infected, then infected individuals become unsusceptible. There are only a few susceptible individuals which can be infected and small number of ill individuals and, therefore, epidemic dies out, because the probability that an individual will be infected is to low. On the other hand, when τ is large, the epidemic spread much slower and the number of susceptible individuals is always large enough. Hence, the probability P_{E} takes nonzero values. In Fig. 4 the influence of the parameter $W_{\mathrm{S}\to\mathrm{IN}}$ on the probability P_{E} is shown. It was found that very contagious disease has small chance to survive, especially when time of incubation is short. The maximum of P_{E} (which is a global maximum for large enough $W_{\mathrm{IN}\to\mathrm{IL}}$) is observed for $W_{\mathrm{S}\to\mathrm{IN}}$ slightly smaller than $W_{\mathrm{IL}\to\mathrm{R}}$.

Similar influence on the spreading process has the parameter $W_{\mathrm{R}\to\mathrm{S}}$ (Fig. 5). For very small values of $W_{\mathrm{S}\to\mathrm{IN}}$ epidemic quickly dies out, because the probability that a new individual will be infected is to low. In the case when $W_{\mathrm{R}\to\mathrm{S}}$ is low the endemic state can occur only for narrow range of values of $W_{\mathrm{S}\to\mathrm{IN}}$. This is so because probability of infection is low enough and susceptible individuals appear approximately in the same rate in which they are infected. If value of $W_{\mathrm{S}\to\mathrm{IN}}$ is to large, almost the whole population is quickly infected and the spreading process is stopped, because the number of susceptible individuals is to low. On the other hand, if $W_{\mathrm{S}\to\mathrm{IN}}$ increases, the probability that susceptible individuals (which appear

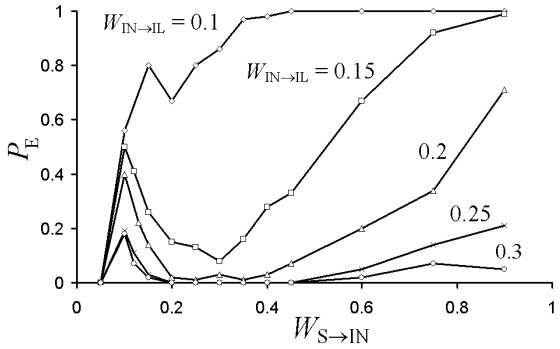

Fig. 4. The influence of the parameter $W_{S\to IN}$ on the probability P_E for different values $W_{IN\to IL}$. Results were averaged over 100 independent simulations. The values of the other parameters are as in Fig. 3 and $W_{IL\to R} = 0.2$; $W_{R\to S} = 0.002$; $p_c = 0.5$; $L = 300$.

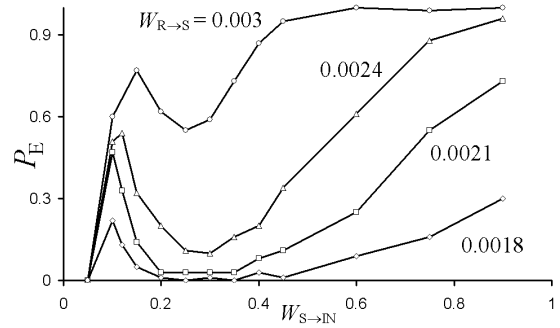

Fig. 5. The influence of the parameter $W_{S\to IN}$ on the probability P_E for different values $W_{R\to S}$. Results were averaged over 100 independent simulations. The values of the other parameters are as in Fig. 3 and $W_{IL\to R} = 0.2$; $W_{IN\to IL} = 0.2$; $p_c = 0.5$; $L = 300$.

in population), will be infected also increases, even if there is relatively small number of susceptible individuals and ill individuals. Therefore, probability P_E increases with increasing $W_{S\to IN}$ for high enough $W_{R\to S}$. An increase in $W_{R\to S}$ cause that the greater number of susceptible individuals appears in the population in one time step and there is an increase in P_E.

Changes in the parameter L_G have the strongest influence on the spatial character of the spreading process. For the lowest values of L_G there is a small number of long range connections, and infections of individuals spatially located near ill individuals are more likely. The spreading process is similar to the propagation of the wave front when secondary sources of epidemics are activated [22]. With increasing L_G, the average length of con-

nections increases. Therefore, the epidemic spreads slightly faster and the range of the epidemic is slightly smaller, which results from weaker interactions between individuals in the same local group.

The spreading process and the range of the epidemic are strongly influenced by the parameter $k_{\max}$. An increase in the value of $k_{\max}$ (and, as a result, the total number of connections in the network) accelerates the spreading process and increases the range of the epidemic V in the population (Fig. 6(a)). For higher values of $k_{\max}$ the maximal number of ill individuals $w_{\max}$ has a higher value and occurs earlier (Fig. 6(b), (c)). As results from Fig. 6 significant changes in V, $w_{\max}$ and $t_{\max}$ are observed only for low values of $k_{\max}$, then the curves saturate. It is also interesting to discuss the influence of the parameter $W_{\mathrm{S}\to\mathrm{IN}}$ on the aforementioned relations. It was found, that the higher the values of $W_{\mathrm{S}\to\mathrm{IN}}$, the smaller the influence of the parameter $k_{\max}$ on the evolution of the epidemic *i.e.* the saturation of the curves $V(k_{\max})$, $w_{\max}(k_{\max})$ and $t_{\max}(k_{\max})$ occurs for lower values of $k_{\max}$. For large enough values of the parameter $W_{\mathrm{R}\to\mathrm{S}}$, the range of epidemic is slightly influenced by $k_{\max}$ (see dashed line in Fig. 6(a) for $W_{\mathrm{R}\to\mathrm{S}} = 0.005$ and $W_{\mathrm{S}\to\mathrm{IN}} = 0.3$).

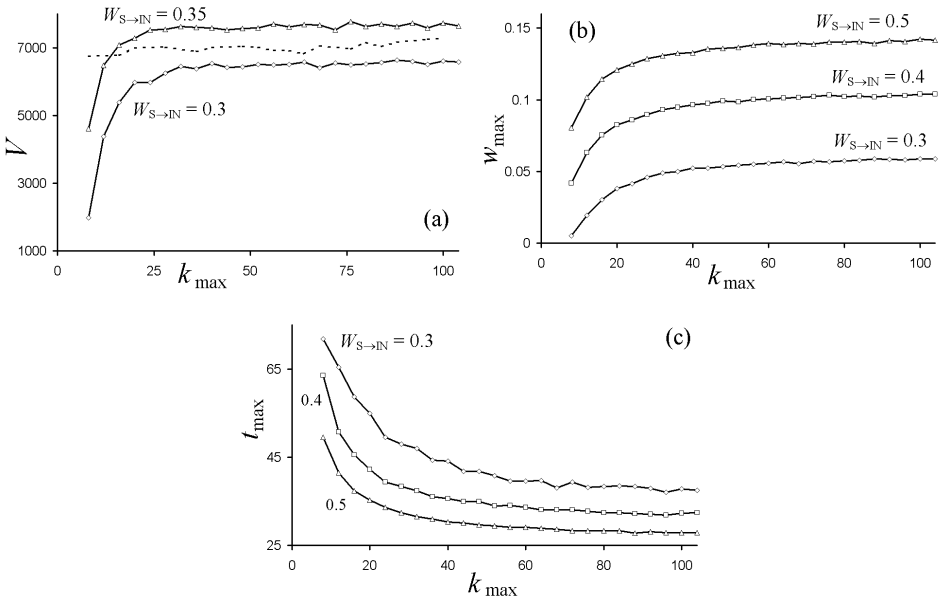

Fig. 6. The influence of the maximum number of the connectivity $k_{\max}$ on range of the epidemic V (a), the maximum value of the number of ill individuals $w_{\max}$ (b) and the time $t_{\max}$ (c), for different values of $W_{\mathrm{S}\to\mathrm{IN}}$ and $p_{\mathrm{c}} = 0.5$. Results were averaged over 1000 independent simulations — standard deviation is near to five percent. The values of the other parameters are as in Fig. 3.

The connectivity k_{start} of the individual which is the initial source of the epidemic is another important parameter which determines the time evolution of the epidemic. This parameter has a similar influence on the behavior of V, $w_{\max}$ and $t_{\max}$ as the parameter $k_{\max}$. An earlier work discusses the influence of the localization of the initial source of epidemic in the population on the spreading process [21].

It is important to investigate the influence of the preventive vaccination (the number N_{R0} of the individuals in the state (R) at time $t = 0$) on the spreading process. In Fig. 7(a) the time $t_{\max}$ (which well describe dynamic properties of the spreading process, *i.e.* the rate of spread) as a function of the number of preventive vaccinated (and randomly chosen) individuals for different values of $W_{\mathrm{S}\to\mathrm{IN}}$, is shown. For low values of N_{R0} the time of duration of the epidemic T increases, because epidemic can not spread freely. For a certain value of N_{R0}, denoted N_{RC}, the time $t_{\max}$ reaches a maximum. The abrupt decrease of the times $t_{\max}$ and T, observed for bigger values of N_{R0}, indicates that a phase transition occurs at N_{RC}. This is proved by the

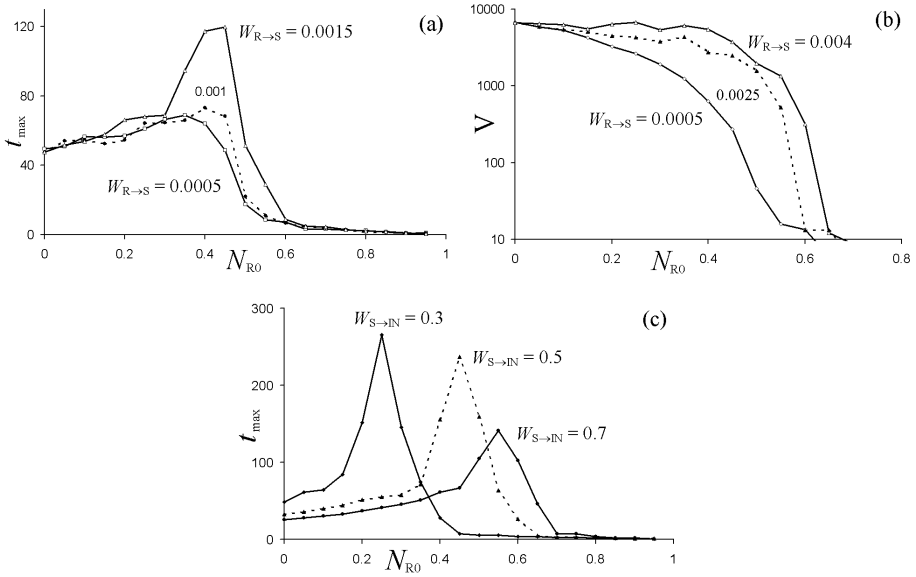

Fig. 7. The influence of the number of the preventive vaccinated individuals N_{R0} on time $t_{\max}$ (a) and range of the epidemic V (b) for different values $W_{\mathrm{R}\to\mathrm{S}}$ and $W_{\mathrm{S}\to\mathrm{IN}} = 0.3$. The relation between time $t_{\max}$ and the N_{R0} for different values of $W_{\mathrm{S}\to\mathrm{IN}}$ and $W_{\mathrm{RS}} = 0.0015$ in the case when individuals with greatest k are vaccinated is shown in (c). Results were averaged over 1000 independent simulations — standard deviation is near to five percent. The values of the other parameters are as in Fig. 3 and $p_{\mathrm{c}} = 0.5$.

significant increase of the transient times (*i.e.* the time before the system reaches the point attractor) for N_{R0} slightly smaller than N_{RC}, which is typical behavior for a phase transition. Such a phase transition is observed in percolating systems [35]. When the value of $W_{\mathrm{S}\to\mathrm{IN}}$ increases, the part of the population that should be preventively vaccinated in order to suppress an epidemic also increases. Similar influence has parameter $W_{\mathrm{R}\to\mathrm{S}}$: the greater $W_{\mathrm{R}\to\mathrm{S}}$, the greater N_{RC}. Moreover, with increasing $W_{\mathrm{R}\to\mathrm{S}}$ the range of the epidemic V decreases more abruptly (see Fig. 7(b)).

When preventive vaccinated individuals are not chosen randomly, but individuals with the greatest k are chosen, the results are similar (*cf.* Fig. 7(c) and Fig. 7(a)). However, the phase transition occurs for a lower value N_{R0}, which means that a smaller number of preventive vaccinated individuals is needed to suppress the epidemic. Note that, in this case, for values N_{R0} slightly smaller than the critical value N_{RC}, the times $t_{\max}$ and T increase quickly with N_{R0}. This means that the rate of the spreading of epidemic is much smaller, because only individuals with small k can be infected (*cf.* Fig. 6(b)).

4. Comparison with master equation

In the master equation it is assumed that each individual interacts with all other individuals in the population and interactions with all individuals are treated in the same way. In contemporary and large communities this is not true, because the people interact strongly only with a small (in comparison to the size of the whole population) number of other individuals. In Fig. 8 results obtained from the analytical solutions of the master Eq. (1) and from the present model are compared. The two curves are similar but in the case of our model, the number of the ill individuals increases faster and the maximum appears for lower values of time than in the case of the solutions of the master equation. When only one individual is ill at $t = 0$, the number of infected individuals N_{IN} resulting from the master equation increases very slowly, because P_{IL} is very small. In our model, however, strong interactions with nearest neighbors are taken into account; as a consequence the epidemic spreads faster, which explain the discrepancy between the location of the two curves. For large enough time t the solution of master equation settles in fixed point, but in the case of numerical calculation oscillations of the number of ill individuals are still observed. When the number of susceptible individuals is very low, the number of ill individuals decreases, because the probability that a new individual will be infected is low. On the other hand, when the number of ill individuals is low the number of susceptible individuals increases. Hence, when the critical value N_{S} is reached, the epidemic starts to spread. In consequence, there is an abrupt increase in N_{IL}.

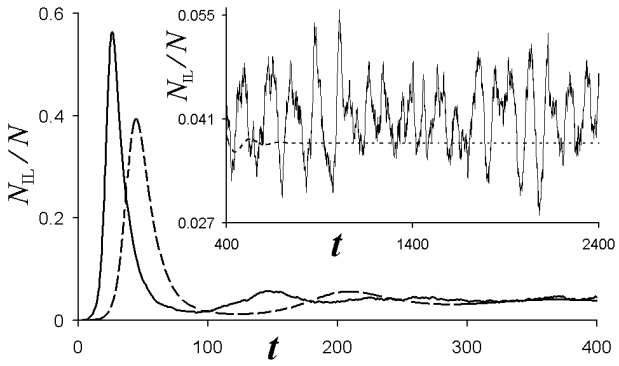

Fig. 8. Comparison of the relation $N_{\mathrm{IL}}(t)/N$ obtained in the presented model (solid line) with the solution of the master equation (dashed line) for $W_{\mathrm{S}\to\mathrm{IN}} = 0.5$, $W_{\mathrm{IL}\to\mathrm{R}} = 0.1$, $W_{\mathrm{R}\to\mathrm{S}} = 0.005$ and $p_{\mathrm{c}} = 0.5$. The values of the other parameters are as in Fig. 3.

5. Conclusions

A model of the spreading of epidemic in a population with the hierarchical structure of interpersonal interactions have been described and investigated numerically. The structure of interpersonal connections is based on a scale-free network. Spatial localization of individuals belonging to different social groups and the mobility of the individuals in the community are taken into account. It was found, that the type of interpersonal interactions has an essential influence on the spreading process. In particular, connections with the nearest neighbors (*i.e.* family or friends) are more important than random contacts between strange individuals.

Our calculations, performed for initialy one ill and randomly chosen individual, show, that epidemic spreads more slowly in a population with a higher value of the clustering coefficient C. This process depends also on the incubation time τ. With increasing values of τ the duration time T of the epidemic increases. On the other hand, an increase of the maximal number of connections in the population $k_{\max}$ causes an increase of the range of the epidemic and accelerates the spreading process. The influence of different types of initial sources of epidemic, *e.g.* massive disperse of a pathogen simulating bio-terrorist attack, on spreading process need further investigation.

In our model the influence of preventive vaccinations on the spreading of the epidemic was investigated. We found a critical value of preventively vaccinated individuals, sufficient for the suppression of the epidemic.

From all the results obtained a general conclusion emerges that an increase of the probability $W_{\mathrm{S}\to\mathrm{IN}}$ decreases the influence of all the parameters

characterizing the social network (*i.e.* $k_{\max}$, L_{G} or clustering coefficient) on the dynamics and range of the epidemic. This observation shows how dangerous are most contagious diseases.

Our results were compared with the solutions of the master equations. The character of the two solutions is similar, however, there are discrepancies between the locations of the maxima of the relations of the number of ill individuals and time. It is caused by the assumptions in our model which take into account the hierarchical structure of interpersonal interactions in a more plausible way than in the case of the master equation.

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