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EPIDEMIC CONTROL IN A HIERARCHICAL SOCIAL NETWORK

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The phenomenon of epidemic spreading in a population with a hierarchical structure of interpersonal interactions is described and investigated numerically. The SIR model with incubation time is used. In our model the localization of individuals in different social groups, the effectiveness of different interpersonal interactions and the mobility of a contemporary community are taken into account. The influence of different control methods on the spreading process is investigated as a function of different initial conditions. The cost-effectiveness of mass preventive random vaccinations, target vaccinations and sick leaves are compared. A critical range of vaccinations, sufficient for suppressing of an epidemic is calculated. The results of numerical calculations are similar to the solutions of the master equation for the spreading process.

Keywords: Epidemic; vaccination.

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1. Introduction

In recent years it was discovered that a structure of different biological, technical, economical and social systems has the properties of complex networks.[1–3] The short length of the average shortest-path distance and the high value of the clustering coefficient are some of the common properties of those networks.[3–5] Social networks, which are an important example of complex networks, also have those properties. They are successfully modelled using different approaches.[6–9] In particular, small-world topology of interpersonal connections[3,10–14] and their hierarchical structure[15,16] are taken into account, e.g., the epidemic spreading in a population with a two-level structure of interpersonal interactions was analyzed in Ref. 17. Such a structure of a social network has a strong influence on dynamical phenomena in a population.

In recent years the spreading of epidemics was investigated by many authors, who used different models of interpersonal interactions.[18–24] This was motivated by the hazards of bio-terrorist attacks[25] and new types of pathogens observed lately. In our work, we investigate epidemic spreading in the human population, taking into account spatial localization of individuals, with a three-level hierarchical structure of interpersonal interactions on the basis of the SIR model.[26]

We assume that each individual belongs to some social groups:[4,27] from small ones (e.g., family or friends), to a large ones (e.g., the community of a 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, social connections within a family, among close friends etc. are most effective; however, random contacts with unknown individuals are important, too. Such a random contact is most probable for individuals, who live (or work) in the same place, e.g., in the same building. On the other hand, contemporary communities are very mobile; therefore, there is a nonzero probability of contact between two arbitrarily chosen individuals from a population. A contact like that can occur, e.g., while commuting, in the cinema or any other public place, and it can result 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 related to the mobility of a community. The hierarchical structure of interpersonal interactions described in the present paper seems to be plausible for modeling real social networks.

A group of co-workers is one of the social groups defined in our model. Therefore, it is possible to investigate the influence of the probability of obtaining sick leave and the duration of sick leave on spreading phenomena.

This article is organized as follows. The model of a network of human contacts and the probabilities of infection depending on the type of social contact, as well as the master equation, are described in Sec. 2. The results, like the influence of vaccination and sick leave on the spreading process, are described in Sec. 3. The results obtained from the numerical model and the results obtained from the solution of the master equation are compared in Sec. 4, and summarized in Sec. 5.

2. Model

In our model, each individual is in one of four permitted states: healthy 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 (this also denotes how

contagious the disease is); $W_{\mathrm{IN}\to\mathrm{IL}}$, the probability that an infected individual will 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).

The spreading process in a population can be treated as a nonstationary process, which is described by the master equation, and similar approach was applied in a number of studies.[26,28–30] The results obtained in our model will be compared with the solutions of this equation in Sec. 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

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

This simple analytical model has one serious disadvantage: it does not take into account the structure of interpersonal interactions in the human population, an important part of our model, in which the population and its structure are described as follows.

The population consists of N individuals who interact with one another in a three-level hierarchical structure of a social network. Interactions within the smallest social group, the household and the working team, constitute the first level of a hierarchical structure. Interactions within larger social groups, workers of a company (or people who work in the same place, e.g., in the same building) and individuals who live in the vicinity (e.g., neighbours), are second-level interactions. Interactions within the whole population (e.g., the community of a city) are third-level interactions.

Before the simulation the whole population is divided into smaller social groups (see Fig. 1): N^{H} — individuals who live in the vicinity and N^{W} — individuals

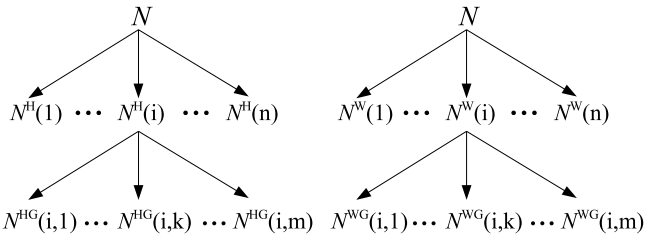


Fig. 1. The whole population (N individuals) is divided into smaller social groups (N^{H} and N^{W}), which are divided into even smaller social groups (N^{HG} and N^{WG}).

who work in the vicinity (e.g., in the same building). In addition, those groups are divided into smaller ones: N^{H} into N^{HG} — households and N^{W} into N^{WG} — co-workers (e.g., working team or people working in the same office) or members of the same school class in the case of schoolchildren.

A similar distinction between residential neighbours and work neighbours was introduced in the Solomon model, where two different networks share a common set of nodes.[31] The average sizes of the abovementioned groups, $\langle N^{\mathrm{H}}\rangle$, $\langle N^{\mathrm{HG}}\rangle$, $\langle N^{\mathrm{W}}\rangle$, $\langle N^{\mathrm{WG}}\rangle$, which determine their number, are the parameters of the model. We have Poisson distribution of sizes of N^{HG} and N^{WG} groups. Each individual is randomly assigned to two smallest social groups (i.e., household and the working team). Because the chosen groups are parts of larger social groups from the second level of the hierarchy (N^{H} and N^{W}), an individual is automatically assigned to them, too. This method of modeling interactions between humans in the population gives nontrivial properties of real social networks, e.g., small-world topology of connections, a large clustering coefficient and a hierarchical structure.[3,15,32,33]

Computations are performed for the values of all groups independently, e.g., if it was assumed initially that $\langle N^{\mathrm{WG}}\rangle = 10$, but if a certain working team has only 8 individuals, then for individuals from this group the value 8 is taken into account.

To distinguish the effectiveness of interactions between individuals at 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 with people with whom we spend most of our time are a more effective way of spreading of an epidemic than random contacts, we assume that the probability of an infection of such individuals is a simple nonlinear function[9,34] with the following form:

$$p_1 = W_{\mathrm{S}\to\mathrm{IN}}\left(\frac{1}{2}\sqrt{\frac{N_{\mathrm{IL}}^{\mathrm{HG}}}{N^{\mathrm{HG}}}} + \frac{1}{2}\sqrt{\frac{N_{\mathrm{IL}}^{\mathrm{WG}}}{N^{\mathrm{WG}}}}\right) \tag{2}$$

where $N_{\mathrm{IL}}^{\mathrm{HG}}$ is the number of ill individuals who live in the same household and $N_{\mathrm{IL}}^{\mathrm{WG}}$ is the number of ill co-workers.

Random contacts between individuals in the same groups of N^{H} and N^{W} individuals is the second level of interpersonal interactions. They are most probable for individuals living or working in the same place, e.g., in the same building. In our model, we assume that the probability of infection resulting from a random contact like that is proportional to the probability that an individual from the group is ill:

$$p_2 = W_{\mathrm{S}\to\mathrm{IN}}\left(\frac{1}{2}\frac{N_{\mathrm{IL}}^{\mathrm{H}}}{N^{\mathrm{H}}} + \frac{1}{2}\frac{N_{\mathrm{IL}}^{\mathrm{W}}}{N^{\mathrm{W}}}\right) \tag{3}$$

where $N_{\mathrm{IL}}^{\mathrm{H}}$ is the number of ill individuals who live in the vicinity and $N_{\mathrm{IL}}^{\mathrm{W}}$ is the number of ill individuals who work in the vicinity.

Random contacts between pairs of individuals who are strange to each other and who are chosen arbitrarily from the whole population is the third level of

interpersonal interactions. 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:

$$p_3 = W_{\mathrm{S}\to\mathrm{IN}} \left(\frac{N_{\mathrm{IL}}}{N} \right)^2 \tag{4}$$

where N_{IL} is the number of ill individuals in the whole population. The nonlinear factor in Eq. (4) causes the probability p_3 to initially increase very slowly and become significant for a great number of ill individuals.

It can be seen that from the point of view of each individual, his or her interpersonal interactions are hierarchical and they can be divided into three levels. Note that, as results from Eqs. (2)–(4), the probabilities p_1, p_2 and p_3 of an infection of each individual depend 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 the ill individual belongs to the working team or close family, it is smaller when the ill individual lives or works in the vivinity and it is smallest when the 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 $W_{x\to Y}$, like in the master equation (1).

Each ill (S) individual may obtain sick leave for x time steps (i.e., days) with the probability p_{SL}. An individual on sick leave does not interact with N_{W} and N_{WG} groups during sick leave. After x days this individual goes back to work, even if he or she is still sick. On the other hand, if an individual recovers earlier, he or she does not go back to work before the end of sick leave. Hence, it is possible to find optimal time of sick leave, i.e., when the number of ill individuals and the number of healthy individuals on sick leave are minimal. It is also possible to investigate the influence of the probability p_{SL} of going on sick leave on spreading phenomena.

In order to investigate the influence of target vaccination on the process of epidemic spreading we introduce the parameter p_{TV}. In each time step all susceptible S closest neighbors of the ill individual, i.e., individuals belonging to the same N^{HG} and N^{WG} groups, are vaccinated with the probability p_{TV}. After vaccination, these individuals become unsusceptible R (to simplify the model we assume that the time necessary to develop immunity is very short: no longer than one day). It should be noted that introducing the probability p_{TV} can also be treated as a simple modeling of the use of antiviral agents.[35,36] The value of the probability p_{TV} can be connected with the time health services take to identify ill individuals in the population.

3. Results

Computations were performed for different initial conditions with different numbers n of ill (IL) and randomly located individuals, and the rest of the population healthy and susceptible (S). Large values of n can be compared to, e.g., broad dispersal of pathogens during a bio-terrorist attack or to a case when counteractions of health

care services are introduced some time after beginning of an epidemic. Synchronous dynamics and the size of the population $N = 10^5$ were used. In most computations the average sizes of social groups $N^{\mathrm{HG}} = 4$, $N^{\mathrm{WG}} = 10$ and $N^{\mathrm{H}} = N^{\mathrm{W}} = 100$ were used. In order to investigate the dynamics of the spreading process and the range of an epidemic we introduce two observables: the time $t_{\max}$ when the maximal number of ill individuals is reached and the number V of individuals who have had the disease.

The time of incubation $\tau = 1/W_{\mathrm{IN}\to\mathrm{IL}}$ influences the rate of epidemic spreading only. The time $t_{\max}$ increases approximately linearly with τ. On the other hand, the dynamics of spreading process and the range of an epidemic depend significantly on the value of the parameter $W_{\mathrm{IL}\to\mathrm{R}}$. Figure 2 shows that for a critical value of $W_{\mathrm{IL}\to\mathrm{R}} = W^{\mathrm{C}}_{\mathrm{IL}\to\mathrm{R}}$ there is an abrupt decrease in the time $t_{\max}$ and the range of epidemic: this indicates that a phase transition takes place at this value. This is confirmed by a significant increase in the transient times (i.e., the time before the system reaches the point attractor) for $W_{\mathrm{IL}\to\mathrm{R}}$ slightly smaller than $W^{\mathrm{C}}_{\mathrm{IL}\to\mathrm{R}}$, which is typical behavior for a phase transition.

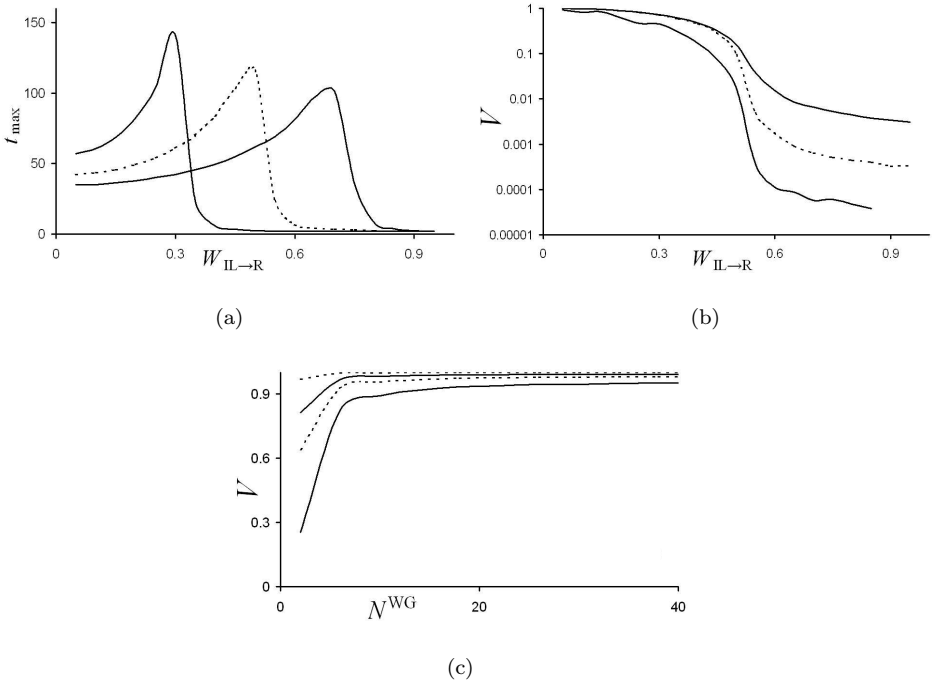
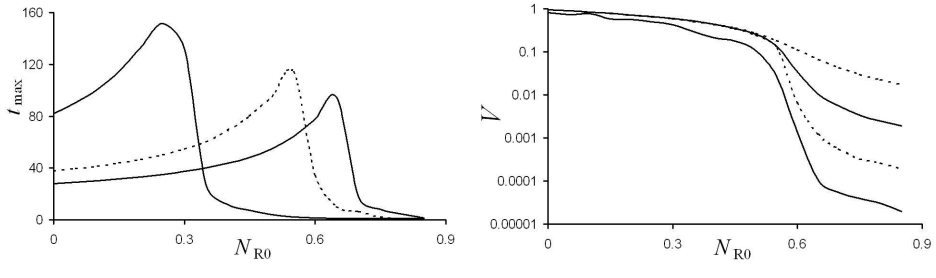


Fig. 2. The influence of the parameter $W_{\mathrm{IL}\to\mathrm{R}}$ on the time $t_{\max}$ (a) for different values of $W_{\mathrm{S}\to\mathrm{IN}}$ (0.2; 0.3 and 0.4 from left to right respectively) and the range of the epidemic V (b) for different initial conditions (the number of initially ill individuals $n = 1$; 10 and 100 from bottom to top respectively). The relation between the average size of the work group N^{WG} and V for different values of $W_{\mathrm{S}\to\mathrm{IN}}$ (0.3; 0.4; 0.5 and 0.8 from bottom to top respectively) is shown in (c). The results are averaged over 100 independent simulations. The values of the other parameters are: $W_{\mathrm{S}\to\mathrm{IN}} = 0.3$; $W_{\mathrm{IN}\to\mathrm{IL}} = 0.5$; $W_{\mathrm{IL}\to\mathrm{R}} = 0.2$; $n = 100$.

The influence of the structure of a social network, e.g., the average size of the work group N^{WG}, on the outcome of the epidemic decreases for more contagious diseases (i.e., when $W_{\mathrm{S}\to\mathrm{IN}}$ increases; see Fig. 2(c)). Significant changes in the range of the epidemic are visible only for small values of N^{WG}. We obtained similar results in our previous work for another model of a social network.[9,16]

In order to investigate the influence of mass preventive vaccination on the spreading process, at the time $t = 0$, the state of N_{R0} randomly chosen individuals is set to (R). With an increase in the number of preventively vaccinated individuals N_{R0}, there is a decrease in the rate of spreading of infection: the time $t_{\max}$ increases. This is so because an epidemic cannot spread freely in the presence of vaccinated individuals. However, for critical value $N_{\mathrm{R0}} = N_{\mathrm{RC}}$ there is an abrupt decrease in $t_{\max}$ and the range of the epidemic V: the epidemic is suppressed (see Fig. 3). This indicates that phase transition has taken place. Such phase transition is observed in percolating systems.[37] When the disease is more contagious, i.e., when the value of $W_{\mathrm{S}\to\mathrm{IN}}$ increases, the part of the population that should have been preventively vaccinated in order to suppress the epidemic also increases. Also the value of $W_{\mathrm{IL}\to\mathrm{R}}$ parameter is important: when $W_{\mathrm{IL}\to\mathrm{R}}$ decreases, the critical value N_{RC} increases significantly and the changes in the range of the epidemic for $N_{\mathrm{R0}} \approx N_{\mathrm{RC}}$ are more abrupt. The behavior of the system also depends on the initial conditions (Fig. 3(b)). With an increase in the number initially ill individuals n, there is a slight increase in N_{RC} and the changes in the range of epidemic are less abrupt. This indicates how dangerous broad dispersal of pathogens can be (e.g., as a result of a bio-terrorist attack): the range of an epidemic is relatively large even in if almost the whole population has been vaccinated.

The influence of the size of the network on the spreading process for values of $W_{\mathrm{IL}\to\mathrm{R}}$ and N_{R0} close to their critical values is shown in Fig. 4. It is visible that the changes of the range of the epidemic and relative value of the time $t_{\max}$ are



(a) (b)

Fig. 3. The influence of the relative number of preventively vaccinated individuals N_{R0} on the time $t_{\max}$ (a) for different values of $W_{\mathrm{S}\to\mathrm{IN}}$ (0.2; 0.4 and 0.6 from left to right respectively) and the range of the epidemic V (b) for different initial conditions (the number of initially ill individuals $n = 1$; 10; 100 and 1000 from bottom to top respectively). The results are averaged over 100 independent simulations. The values of the other parameters are: $W_{\mathrm{S}\to\mathrm{IN}} = 0.3$; $W_{\mathrm{IN}\to\mathrm{IL}} = 0.5$; $W_{\mathrm{IL}\to\mathrm{R}} = 0.2$; $n = 100$.

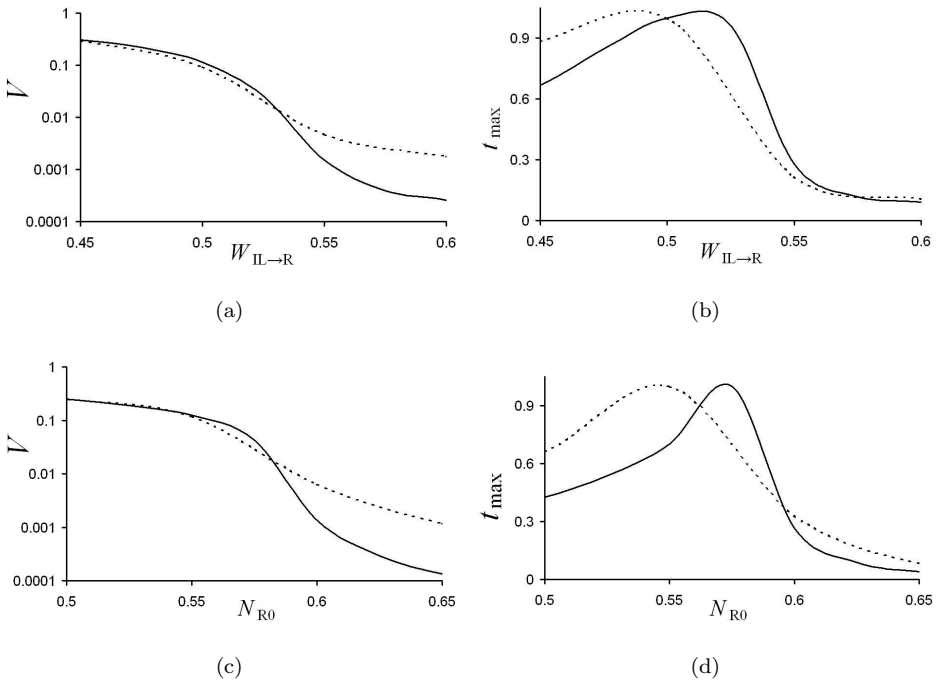


Fig. 4. The influence of the size of the system N (for $N = 10^5$-dashed line and $N = 10^6$-solid line) on the range of epidemic V (left column) and relative value of the time $t_{\max}$ (right column). The results are averaged over 100 independent simulations. The values of the other parameters are: $W_{S\to IN} = 0.3$ (a), (b); $W_{S\to IN} = 0.4$ (c), (d); $W_{IN\to IL} = 0.5$; $W_{IL\to R} = 0.2$; $n = 10$.

more abrupt in the case of larger network (solid line) — it is characteristic feature of phase transitions in finite systems.

Mass preventive vaccination is not the only method of using of vaccines. In our work, we also investigated the influence of target vaccination. Figure 5 illustrates the influence of the probability p_{TV} on the spreading process (in the simulation we assum that there was no shortage of vaccines). Like in the case of mass vaccination there is a critical value of $p_{TV} = p_{TV}^{C}$ when phase transition takes place and abrupt changes (which are more abrupt for larger networks) in $t_{\max}$ and V are visible. In addition, the influence of initial conditions is similar to the previous case (cf. Figs. 3(b) and 5(b)). Note that above the critical value, a further increase in p_{TV} did not provide better results: the changes in V and in the relative number N_V of individuals who are vaccinated are very small (see Figs. 5(b) and 5(c)). The value of p_{TV}^{C} increases when $W_{IL\to R}$ decreases. However, the value of parameter $W_{IL\to R}$ has a smaller influence on effectiveness of targeted vaccination than in the case of mass vaccination.

Because of the cost of vaccines, it is important to calculate the relative number N_V of individuals who are vaccinated (Fig. 5(c)). Although in our model we assume unlimited supplies of vaccines, during a real epidemic there can be a shortage of

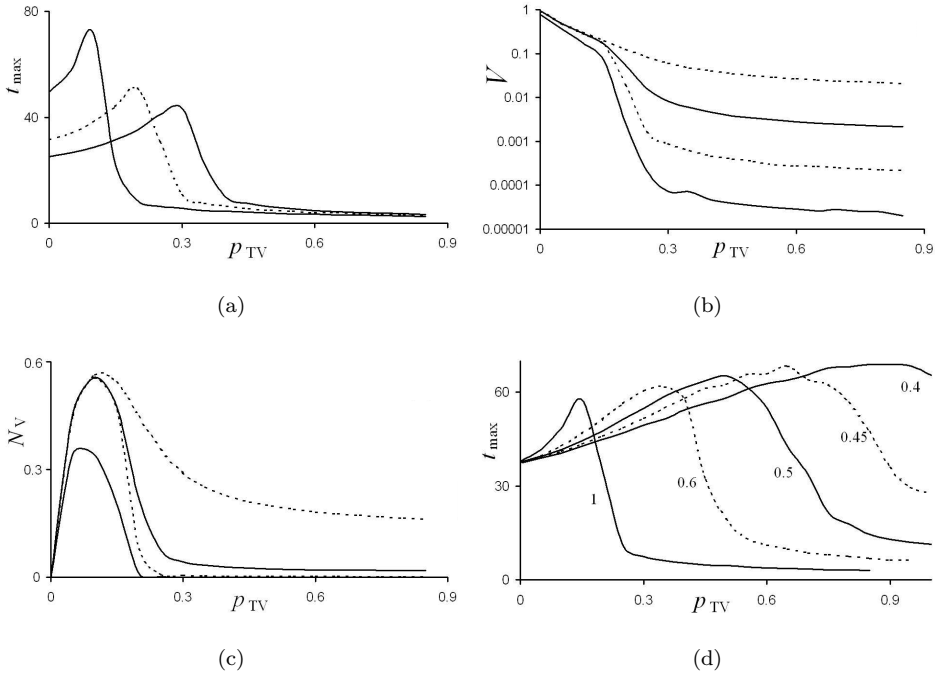


Fig. 5. The influence of the probability p_{TV} on the time $t_{\max}$ (a) for different values of $W_{\mathrm{S}\to\mathrm{IN}}$ (0.3; 0.5 and 0.7 from left to right respectively); the range of the epidemic V (b) and the relative number of vaccinated individuals N_{V} (c) for different initial conditions (the number of initially ill individuals $n = 1$; 10; 100 and 1000 from bottom to top respectively). The influence of the efficiency of vaccines p_{EV} on the relation between the time $t_{\max}$ and p_{TV} is shown in (d). The results are averaged over 100 independent simulations. The values of the other parameters are: $p_{\mathrm{EV}} = 1$; $W_{\mathrm{S}\to\mathrm{IN}} = 0.4$; $W_{\mathrm{IN}\to\mathrm{IL}} = 0.5$; $W_{\mathrm{IL}\to\mathrm{R}} = 0.2$; $n = 100$.

vaccines. The value of N_{V} quickly increases with an increase in p_{TV}. It can be seen that for $p_{\mathrm{TV}} = p_{\mathrm{TV}}^{\mathrm{C}}$ there is an abrupt decrease in N_{V} and the number of vaccines necessary to suppress an epidemic is very low. However, for large n even very quick identification of new ill individuals ($p_{\mathrm{TV}} \approx 1$) is insufficient: the range of the epidemic and the number of vaccines used remain relatively large. This result indicates that not only quick identification of new cases of infection is important. It is also very important to take counteractions at early stages of an epidemic, when very first cases of infection are identified (low n).

In order to investigate the efficiency of vaccines for preventing illnesses, we introduce the probability p_{EV} which describes the efficiency of the vaccines used (i.e., the state of a vaccinated individual is set to (R) with the probability p_{EV}). Figure 5(d) illustrates the influence of the value of p_{EV} on the $t_{\max}(p_{\mathrm{TV}})$ relation. When the time of identification of new cases of infection is short enough (i.e., p_{TV} is large enough), the epidemic can be suppressed even if the efficiency of vaccines is relatively small. However, if the efficiency of vaccines is too small, the only effect

of target vaccination is a decrease in the rate of spreading process irrespectively of the value of the probability p_{TV}.

In our model, it is possible to investigate the influence of the probability p_{SL} of going on sick leave (this is a simple method of isolating ill individuals from part of the population) on the spreading phenomena. Figure 6 illustrates the influence of the probability p_{SL} on the time $t_{\max}$ (Fig. 6(a)), the range of the epidemic V (Fig. 6(b)) and the relative number N_{NW} of individuals who do not work, because they are on sick leave (Fig. 6(c)). When p_{SL} increases, the time $t_{\max}$ also increases, because an ill individual who is on sick leave interacts strongly with his or her local neighborhood only; long-range connections are removed from the social network. Hence, the number of new sources of the epidemic is smaller and the rate of the spreading process is lower. For the critical value $p_{\mathrm{SL}} = p_{\mathrm{SL}}^{\mathrm{C}}$ there is an abrupt decrease in $t_{\max}$ and V. With an increase in the size of the network N, changes in those variables become sharper. It should be noted that the change in the range of the epidemic for $p_{\mathrm{SL}} \approx p_{\mathrm{SL}}^{\mathrm{C}}$ is much more abrupt than in the case of critical values of

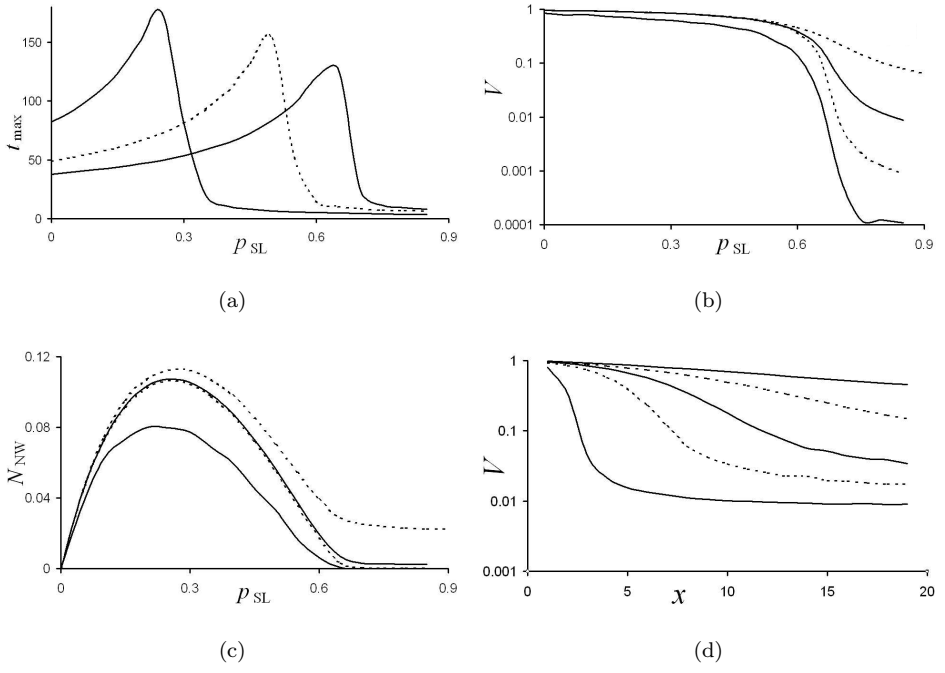


Fig. 6. The influence of the probability p_{SL} on the time $t_{\max}$ (a) for different values of $W_{\mathrm{S}\to\mathrm{IN}}$ (0.2; 0.3 and 0.4 from left to right respectively); the range of the epidemic V (b) and the maximal number of individuals N_{NW} who do not work due to sick leave (c) for different initial conditions (the number of ill individuals $n = 1$; 10; 100 and 1000 from bottom to top respectively). In Fig. 6(d) the relation between the range of the epidemic V and the duration of sick leave x (for different values of p_{SL}: 0.65; 0.7; 0.75; 0.8 and 0.9 from top to bottom, respectively) is shown. The results are averaged over 100 independent simulations. The values of the other parameters are: $W_{\mathrm{S}\to\mathrm{IN}} = 0.4$; $W_{\mathrm{IN}\to\mathrm{IL}} = 0.5$; $W_{\mathrm{IL}\to\mathrm{R}} = 0.2$; $n = 100$.

N_{R0} (cf., Fig. 3) and p_{TV} (cf., Fig. 5), which is not clearly visible in the logarithmic scale. As in the case of using vaccines, changes in the range of the epidemic V near a critical value are less abrupt for a larger number of initially ill individuals. Note that for $n = 1000$ and in the range of control parameters when an epidemic is suppressed, the maximal number N_{NW} of individuals who are on sick leave is almost ten times smaller than the number N_V of necessary vaccines (cf., Figs. 6(c) and 5(c)). The value of N_{NW} depends also on the time of incubation and increases with an increase in $W_{IN\to IL}$.

The duration of sick leave x also influences the spreading process. Fig. 6(d). illustrates the relation between the range of the epidemic V and x, for different values of p_{SL}. It can be seen that with an increase in x, there is a decrease in the critical value p_{SL}^{C}. However, for low enough p_{SL} the epidemic is not suppressed even for very long sick leave. The number of individuals who do not work N_{NW} decreases significantly as x increases (the change in N_{NW} is more rapid for greater p_{SL}) and reaches a minimum when the epidemic is suppressed. A further increase in x causes a small increase in N_{NW}.

4. Comparison with Master Equation

In the master equation, it is assumed that each individual interacts with all other individuals in the population and the interactions with all individuals are treated in the same way. In contemporary and large communities this is not true, because people interact strongly only with a small (in comparison to the size of the whole population) number of other individuals. In Fig. 7 the results obtained from analytical solutions of the master equation (1) and from the present model are compared. The two curves are similar but in the case of our model, the number of ill individuals increases faster and the maximum appears for lower values of the time than in the case of the solutions of the master equation. When only a few individuals are ill at $t = 0$, the number of infected individuals N_{IN} resulting from the master equation

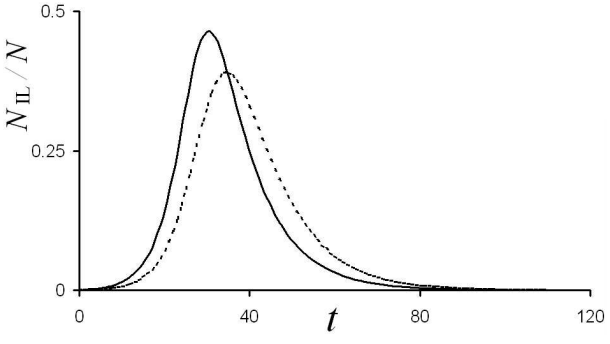


Fig. 7. Comparison of the relation $N_{IL}(t)/N$ obtained in the presented model (solid line) with the solution of the master equation (dashed line) for $W_{S\to IN} = 0.5$, $W_{IN\to IL} = 0.5$, $W_{IL\to R} = 0.1$ and $n = 100$.

increases very slowly, because P_{IL} is very low. In our model, however, strong interactions with the nearest neighbors are taken into account; consequently, the epidemic spreads faster, which explains the discrepancy between the location of the two curves.

5. Conclusions

The model of the spreading of an epidemic in the population with a three-level hierarchical structure of interpersonal interactions has been described and investigated numerically. In our model, the influence of mass preventive vaccinations on the spreading of an epidemic was investigated. We found the critical value of preventively vaccinated individuals, sufficient for the suppression of an epidemic. However, the part of the population which should be vaccinated is very large and strongly increases with probability of infection. Moreover, in the case of broad dispersal of pathogens (e.g., as a result of bio-terrorist attack) the range of the epidemic remains relatively large, even if almost the whole population is vaccinated.

Contrary to mass vaccination, target vaccination can give much better results with little demand for vaccines (or antiviral agents), i.e., when only the nearest neighbors of ill individuals are vaccinated. An epidemic can be suppressed with a relatively small amount of vaccines if new ill individuals are identified quickly enough. Especially good results can be obtained if target vaccination starts just after the appearance of the very first cases of infection, i.e., in the initial stage of an epidemic. Moreover, even if the efficiency of vaccines is lower than 100% an epidemic can also be suppressed with quick enough identification of infected individuals.

In our model we also investigated the influence of sick leave (which can be treated as a simple method of isolating ill individuals from part of the population) on spreading process. It turns out that for a critical value of the probability of going on sick leave there is an abrupt decrease in the range of the epidemic. The epidemic is suppressed and the number of individuals who do not work is very low.

Mass vaccination can be effective only in the case of well-known pathogens (e.g., in the case of annual influenza epidemics). If there is a new pathogen in a susceptible population (as a result of mutation or a bio-terrorist attack), only a quick response of health services can provide good results. In such a case, the efficiency of target vaccination of the nearest neighbors of ill individuals is high. Removing interpersonal interactions with spatially distant individuals by isolating an ill individual (e.g., at home) decreases significantly the number of new sources of the epidemic and is helpful in suppressing epidemic spreading.

Our results were compared with the solutions of the master equation. The character of both solutions is similar; however, there are discrepancies between the locations of the maxima of the relations of the number of ill individuals and time. This is so because in our model we assume a hierarchical structure of interpersonal interactions in a more plausible way than in the case of the master equation.

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