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THE SIS MODEL FOR ASSESSMENT OF EPIDEMIC CONTROL IN A SOCIAL NETWORK*

Andrzej Grabowski

Central Institute for Labour Protection – National Research Institute
Czerniakowska 16, 00-701 Warsaw, Poland

Magdalena Rosińska

National Institute of Hygiene, National Center for Disease Prevention & Control
Chocimska 24, 00-791 Warsaw, Poland

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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 SIS model with incubation time and temporal immunity to a disease, is used. In our model location in social structure, effectiveness of different types of interactions and mobility of contemporary communities are taken into account. The influence of control measures on the spreading process is investigated as a function of initial conditions. The cost-effectiveness of mass immunizations campaigns, target vaccinations and the sick leaves is compared. A critical vaccinations coverage, sufficient for suppressing an epidemic as well as the probability that endemic state occurs, are calculated. The results of numerical calculations are similar to the solutions of the master equation for the spreading process.

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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, 2]. 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 [2, 3]. Social networks, which are an important example of complex networks, also have those properties. They are successfully modeled using different approaches [4,5], in particular, small-world topology

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of interpersonal connections [2, 6] and their hierarchical structure [7, 8] are taken into account, *e.g.* epidemic spreading in a population with a two-level structure of interpersonal interactions was analyzed in Ref. [9]. 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 [10–16]. 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 SIS model [18].

We assume that each individual belongs to some social groups [3, 19]: from small ones (*e.g.* family or friends), to large ones (*e.g.* the community of a whole city). Interpersonal interactions among individuals in the same group are more intense 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 (household), among close friends *etc.* are most effective; however, random contacts with unknown individuals are important, too. Such 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, *e.g.* 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. The 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); $W_{\mathrm{R}\to\mathrm{S}}$, 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 [18,20,21]. 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 $P_X(t)$ that an individual is in one of the possible states X (where $X = \mathrm{S}$, IN, IL or R) are described with the master equation

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

This simple analytical model has one serious disadvantage: it does not take into account the structure of interpersonal interactions in the human population, 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 each other 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.* neighbors), are second-level interactions. Interactions within the whole population (*e.g.* the community of a city) are third-level interactions.

Before the simulation whole population is divided into smaller social groups (see Fig. 1): N^{H} — individuals who live in the vicinity and N^{W} — co-workers of a company (or people who work in the same place, *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} — working teams (or school class in case of school children). Similar distinction between residential neighbors and work neighbors was introduced in the Solomon model, where two different networks share a common set of nodes [23]. The average sizes of the abovementioned groups $\langle N^{\mathrm{W}}\rangle$, $\langle N^{\mathrm{H}}\rangle$, $\langle N^{\mathrm{HG}}\rangle$ which determine their number, are the parameters of the model. However, the size of working-team is drawn from power-law distribution $P(N^{\mathrm{WG}}) \sim (N^{\mathrm{WG}})^{-4}$ in the range $N^{\mathrm{HG}} \in (10, 100)$ in order to obtain scale-free distribution of connectivity $P(k) \sim k^{-3}$. Each individual is randomly assigned to two first-level social groups (*i.e.* households and working team). Because the chosen groups are parts of larger social groups from the second level of the structure (N^{H} and N^{W}), an individual is automatically assigned to second-level groups, too. This method of modeling of social interactions gives nontrivial properties of real social networks, *i.e.* small-world topology of connections, a large clustering coefficient, a hierarchical structure, positive degree correlations and scale-free distribution of connectivity [2, 7, 22].

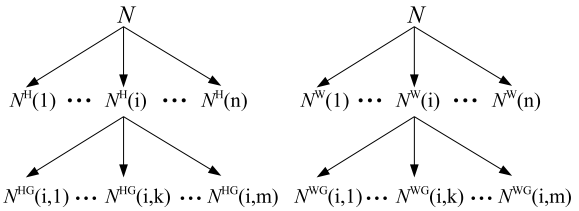

Fig. 1. The whole population (N individuals) is divided into smaller social groups (N^{H} and N^{W}), which are divided into smallest social groups (N^{HG} and N^{WG}).

To differentiate the effectiveness of pathogen transmission between the interactions in different levels of the hierarchy we introduce three equations describing the probabilities of acquiring infection. As close contacts are more likely to result in infection spread we assume that the probability of transmission of the infection between household members and between coworkers is a simple nonlinear function [5, 24] and has a 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 acquiring infection during second-level interactions 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 do not know 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 has the following 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 the greatest if the ill individual belongs to the working team or household, it is smaller if an ill individual lives or works in the vicinity and it is the smallest if the ill individual is located elsewhere in the rest of the population. Other probabilities of a transition between states X, Y are described by the parameters $W_{\mathrm{X}\to\mathrm{Y}}$, like in the master equation (Eq. (1)).

Each (IL) individual may go on sick leave for x time steps (*i.e.* days) with the probability p_{SL}. A person on sick leave does not interact with N^{W} and N^{WG} groups throughout the duration of sick leave. We assume that after x days this person comes back to work, even if she or he is still sick. On the other hand, when an individual recovers before the sick leave period is over they do not return to work earlier. This assumption allow us 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 possible

also to investigate the influence of 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 development we introduce the parameter p_{TV}. In each time step all susceptible closest neighbors (S) of the ill individual 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 be also treated as a simple model of chemoprophylaxis [25,26]. The value of the probability p_{TV} is related to the time of identification of ill individuals in population by health services.

3. Results

Computations were performed for different initial conditions with random location and different numbers n of ill (IL) 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 bio-terrorist attack or to a case when public health preventive measures are delayed with respect to the 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 $N^{\mathrm{HG}} = 4$ 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 magnitude of epidemic V defined as relative number of individuals who went through the disease during epidemic.

The time of incubation $\tau = 1/W_{\mathrm{IN}\to\mathrm{IL}}$ influences the rate of the epidemic spread only. The time $t_{\max}$ increases approximately linearly with τ. On the other hand, the dynamics of spreading process and the magnitude of epidemic depend significantly on the value of the parameter $W_{\mathrm{IL}\to\mathrm{R}}$. Fig. 2 illustrates 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 magnitude of the epidemic. The changes become sharper, when the system size increases — it indicates that a phase transition occurs at this value, because it is characteristic feature of phase transitions in finite systems. 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.

In order to investigate the influence of routine preventive vaccination on the spreading process, at the time $t = 0$ the state of $N_{\mathrm{R}0}$ randomly chosen individuals is set to R. With an increase in the number of preventively

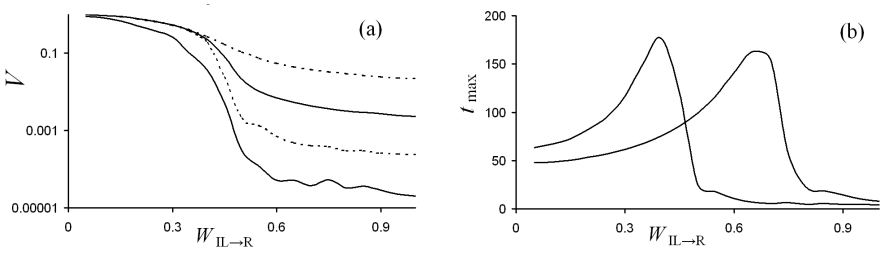

Fig. 2. The influence of the parameter $W_{\mathrm{IL}\to\mathrm{R}}$ on the magnitude of epidemic V (a) for different initial conditions (the number of initially ill individuals $n = 1; 10; 100$ and 1000 from bottom to top, respectively) and time $t_{\max}$ (b) for different values of $W_{\mathrm{S}\to\mathrm{IN}}$ (0.2 and 0.3 from left to right, respectively). Results were averaged over 100 independent simulations. The values of the other parameters are: $W_{\mathrm{S}\to\mathrm{IN}} = 0.2$; $W_{\mathrm{IN}\to\mathrm{IL}} = 0.5$; $W_{\mathrm{IL}\to\mathrm{R}} = 0.2$; $n = 10$.

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 magnitude of epidemic V: the epidemic is suppressed (see Fig. 3). This phenomenon was described 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 [28]. 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. In addition, 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 magnitude of 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 magnitude of epidemic are less abrupt. This indicates substantial risk with broad dispersal of pathogens (*e.g.* as a result of a bio-terrorist attack): the magnitude of an epidemic is relatively large even if almost whole population was vaccinated.

In the case $W_{\mathrm{R}\to\mathrm{S}} > 0$ the behavior of the system is more complicated. There is a non zero probability P_{E} of occurrence of an endemic state (we define P_{E} as a probability that after 10^5 time steps the number of ill or infected individuals is greater than zero). The influence of routine preventive vaccination on the magnitude of epidemic V and the probability P_{E} for values $W_{\mathrm{R}\to\mathrm{S}} > 0$ is shown in Fig. 3(a) and Fig. 3(d), respectively. It is visible that the greater $W_{\mathrm{R}\to\mathrm{S}}$ the greater V and N_{RC}. It indicates that mass routine vaccination is not optimal in the case of easily mutating pathogens.

The value of the probability P_{E} increases with $W_{\mathrm{R}\to\mathrm{S}}$ increasing. Surprisingly, the use of routine preventive vaccination can also increase the probability P_{E} for certain values of N_{R0}. When the average number of new infected individuals equals the average number of individuals who lost their immunity, the endemic state occurs. Such condition can occur when the epidemic spreads slowly enough, *i.e.* when the number of resistant (R) individuals is large enough (see Fig. 3(a)). However, for values $N_{\mathrm{R0}} > N_{\mathrm{RC}}$ the probability that endemic state occurs reaches zero.

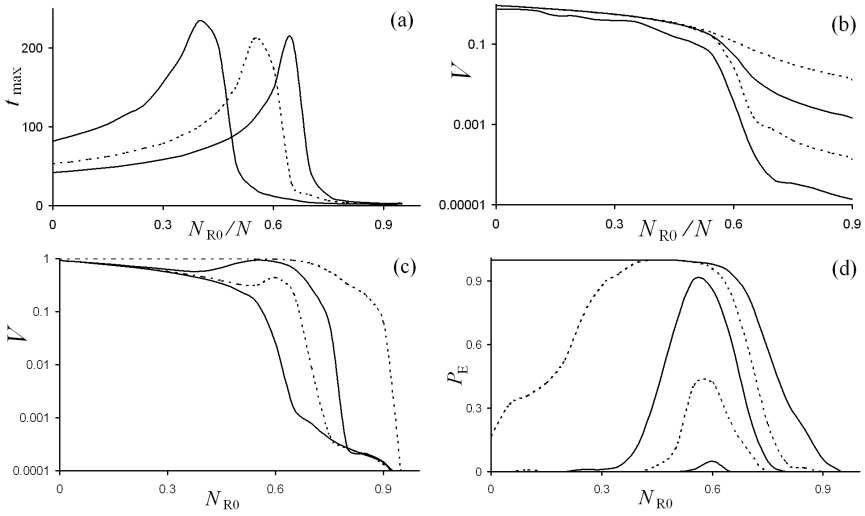

Fig. 3. The influence of the number of preventive vaccinated individuals N_{R0} on the time $t_{\max}$ (a) for different values of $W_{\mathrm{S}\to\mathrm{IN}}$ $(0.2; 0.3$ and 0.4 from left to right, respectively) and the magnitude of epidemic V (b) for different initial conditions (the number of initially ill individuals $n = 1; 10; 100$ and 1000 from bottom to top, respectively) and (c) for different values of $W_{\mathrm{R}\to\mathrm{S}}$ $(0; 0.0005; 0.0016$ and 0.006 from bottom to top, respectively). The value of the probability P_{E} for different values of $W_{\mathrm{R}\to\mathrm{S}}$ $(0.0005; 0.001; 0.0016$ and 0.006 from bottom to top, respectively) is shown in (d). The results are averaged over 100 independent simulations. The values of the other parameters are: $W_{\mathrm{S}\to\mathrm{IN}} = 0.3, W_{\mathrm{IN}\to\mathrm{IL}} = 0.5, W_{\mathrm{IL}\to\mathrm{R}} = 0.2, W_{\mathrm{R}\to\mathrm{S}} = 0, n = 10$.

The routine preventive vaccination is not the only method of using vaccines. In our work, we also investigated the influence of target vaccination. Fig. 4 illustrates the influence of the probability p_{TV} on the spreading process (in the simulation we assume that there was no shortage of vaccines). Like in the case of mass vaccination there is a critical value of $p_{\mathrm{TV}} = p_{\mathrm{TV}}^{\mathrm{C}}$ when phase transition takes place and abrupt changes in $t_{\max}$ and V are visible. In addition, the influence of initial conditions is similar to the previous case (*cf.* Fig. 3(b) and Fig. 4(b)). Note that above the critical value, a fur-

ther 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 Fig. 4(b) and Fig. 4(c)). The value of $p_{\mathrm{TV}}^{\mathrm{C}}$ increases when $W_{\mathrm{IL}\to\mathrm{R}}$ decreases. However, the value of the parameter $W_{\mathrm{IL}\to\mathrm{R}}$ has a smaller influence on effectiveness of target vaccination than in the case of routine preventive vaccination.

Because of the cost of vaccines, it is important to calculate the relative number N_{V} of individuals who are vaccinated (Fig. 4(c)). Although in our model we assume unlimited supplies of vaccines, during a real epidemic a shortage of vaccines is quite likely. 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 magnitude of the epidemic and the number of vaccines used remain relatively large. This result suggests that quick identification of new cases of infection is not the only important measure. It is also crucial to take action at early stages of an epidemic, when the very first cases of infection are identified (low n).

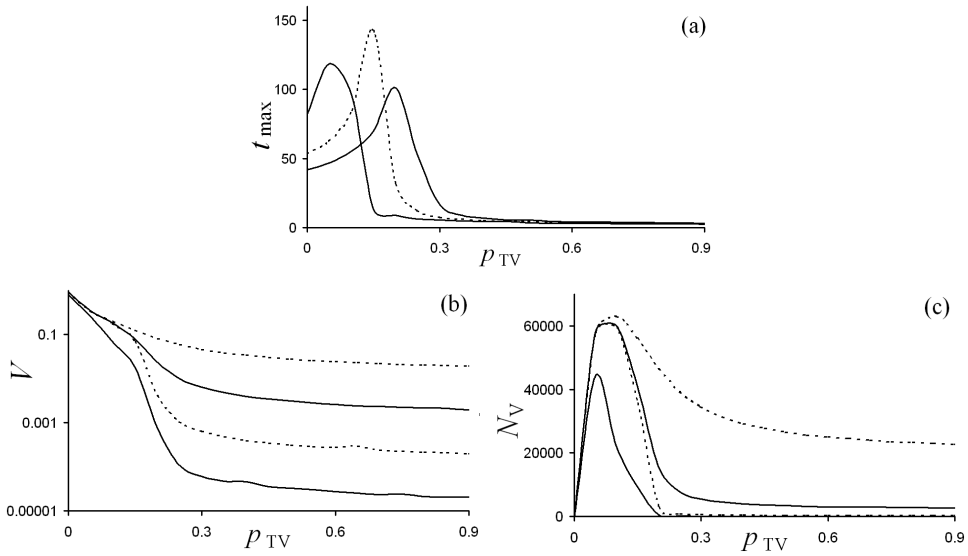

Fig. 4. The influence of the probability p_{TV} on the time $t_{\max}$ (a) for different values of $W_{\mathrm{S}\to\mathrm{IN}}$ (0.2, 0.3 and 0.4 from left to right, respectively); the magnitude of the epidemic V (b) and the relative number of vaccinated individuals N_{V} (c) for different initial conditions (the number of initially ill individuals $n = 1, 10, 100$ and 1000 from bottom to top, respectively). The results are averaged over 100 independent simulations. The values of the other parameters are: $W_{\mathrm{S}\to\mathrm{IN}} = 0.3, W_{\mathrm{IN}\to\mathrm{IL}} = 0.5, W_{\mathrm{IL}\to\mathrm{R}} = 0.2, n = 10$.

When the value of the parameter $W_{S\to S}$ increases, there is an increase in magnitude of epidemic and in p_{TV}^{C}. Moreover, for greater values of $W_{R\to S}$, the changes in V for values of the probability p_{TV} in the proximity of the critical value are more abrupt (Fig. 5). The probability that endemic state occurs reaches zero for $p_{TV} > p_{TV}^{C}$. Note that, there is significant increase in p_{TV}^{C} only for very large values of $W_{R\to S}$ and P_E monotonically decreases with p_{TV} increasing - this indicates that target vaccination is a very effective control method even in the case of easily mutating pathogens.

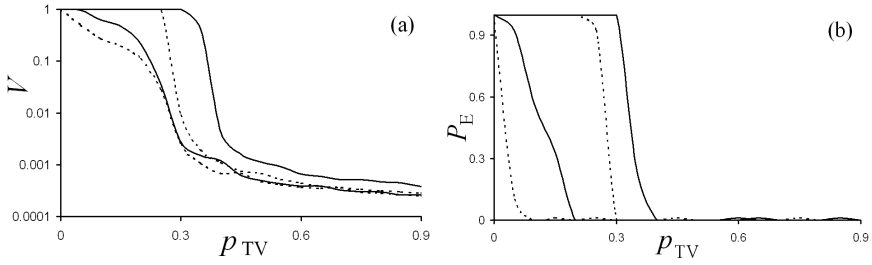

Fig. 5. The influence of the probability p_{TV} on the magnitude of the epidemic V (a) and the probability P_E (b) for different values of $W_{R\to S}$ (0.003, 0.006, 0.015 and 0.035 from bottom to top, respectively). The results are averaged over 100 independent simulations. The values of the other parameters are: $W_{S\to IN} = 0.4$, $W_{IN\to IL} = 0.5, W_{IL\to R} = 0.2, n = 10$.

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. Fig. 6 illustrates the influence of the probability p_{SL} on the time t_{max} (Fig. 6(a)), magnitude 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 its 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 $p_{SL} = p_{SL}^{C}$ there is an abrupt decrease in t_{max} and V. It should be noted that the change in the magnitude of epidemic for $p_{SL} \approx p_{SL}^{C}$ is much more abrupt than in the case of critical values of N_{R0} (*cf.* Fig. 3) and p_{TV} (*cf.* Fig. 4), which is not clearly visible in the logarithmic scale. As in the case of using vaccines, changes in the magnitude of the epidemic V in the proximity of the 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.* Fig. 6(c) and Fig. 4(c)). The value of N_{NW} depends also on the time of incubation and increases with an increase in $W_{\mathrm{IN}\to\mathrm{IL}}$.

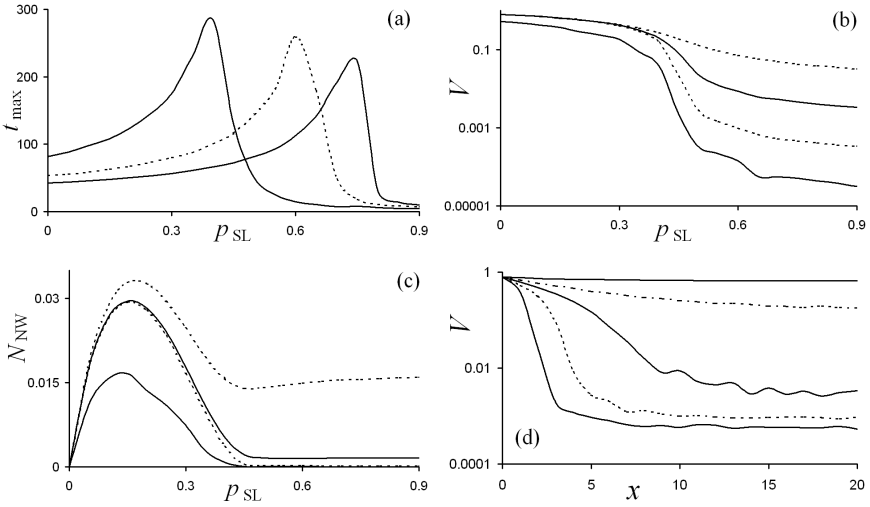

Fig. 6. The influence of the probability p_{SL} on the time $t_{\max}$ (a) for different values of $W_{\mathrm{S}\to\mathrm{IN}}$ (0.2, 0.3 and 0.4 from left to right, respectively); the magnitude of epidemic V (b) and maximal number of individuals N_{NW} who do not work due to sick leave (c) for different 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 magnitude of the epidemic V and the duration of sick leave x (for different values of p_{SL}: 0.1, 0.3, 0.4, 0.5 and 0.6 from top to bottom, respectively) is shown. The results are averaged over 100 independent simulations. The values of the other parameters are: $W_{\mathrm{S}\to\mathrm{IN}} = 0.2, W_{\mathrm{IN}\to\mathrm{IL}} = 0.5, W_{\mathrm{IL}\to\mathrm{R}} = 0.2, n = 10$.

The duration of sick leave x also influences the spreading process. Fig. 6(d) illustrates the relation between the magnitude 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 $p_{\mathrm{SL}}^{\mathrm{C}}$. 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 for $p_{\mathrm{SL}} > p_{\mathrm{SL}}^{\mathrm{C}}$ (the change in N_{NW} is more rapid for greater p_{SL}) and reaches a value close to minimum when the epidemic is suppressed. A further increase in x causes only a slight decrease in N_{NW}. On the other hand, when $p_{\mathrm{SL}} < p_{\mathrm{SL}}^{\mathrm{C}}$, the value of N_{NW} increases quickly with increasing x.

The influence of going on sick leave on the magnitude of epidemic and the probability that an endemic state occurs, for different values of $W_{\mathrm{R}\to\mathrm{S}}$ is shown in Fig. 7. When there is an increase in $W_{\mathrm{R}\to\mathrm{S}}$, there is a slight increase

in $p_{\mathrm{SL}}^{\mathrm{C}}$ and the changes in V for values of the probability $p_{\mathrm{SL}} \approx p_{\mathrm{SL}}^{\mathrm{C}}$ are more abrupt. Similarly as in the case of routine preventive vaccination, for certain values of p_{SL} an increase in P_{E} is visible. If the epidemic spreads slowly enough, *i.e.* if value of the probability p_{SL} is large enough (see Fig. 6(a)), the probability that endemic state occurs increases.

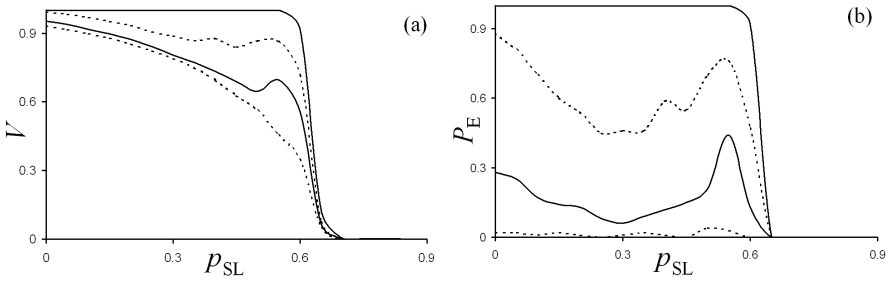

Fig. 7. The influence of the probability p_{SL} on the magnitude of the epidemic V (a) and the probability P_{E} (b) for different values of $W_{\mathrm{R}\to\mathrm{S}}$ (0.0024, 0.0032, 0.004 and 0.006 from bottom to top, respectively). The results are averaged over 100 independent simulations. The values of the other parameters are: $W_{\mathrm{S}\to\mathrm{IN}} = 0.3, W_{\mathrm{IN}\to\mathrm{IL}} = 0.5, W_{\mathrm{IL}\to\mathrm{R}} = 0.2, n = 10$.

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 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. 8 the results obtained from 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 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 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. 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}.

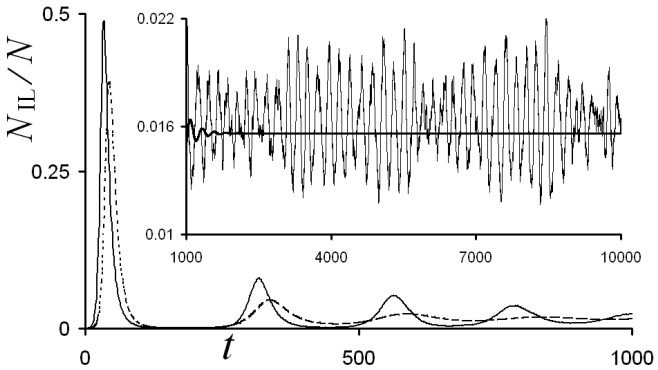

Fig. 8. Comparison of the relation $N_{\mathrm{IL}}(t)/N$ obtained in the presented model (solid line) with the solution of the master equation (dashed line) for $W_{\mathrm{S}\to\mathrm{IN}} = 0.5$, $W_{\mathrm{IN}\to\mathrm{IL}} = 0.5, W_{\mathrm{IL}\to\mathrm{R}} = 0.2, W_{\mathrm{R}\to\mathrm{S}} = 0.002$.

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 routine preventive vaccinations on the spreading of an epidemic was investigated. We found the critical coverage of preventive vaccination, sufficient for suppression of an epidemic. However, the vaccine coverage is very high and strongly increases with infectivity of a disease. Moreover, in the case of broad dispersal of pathogens (*e.g.* as a result of a bio-terrorist attack) the magnitude of the epidemic remains relatively large, even if almost whole population is vaccinated.

Contrary to routine preventive 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 number of vaccines if new ill individuals are identified quickly enough. Particularly good results can be obtained if the target vaccination starts just after the appearance of the very first cases of infection, *i.e.* in initial stage of an epidemic.

In our model we also investigated the influence of sick leave (which can be treated as a simple method of isolating ill individuals from a part of the population) on the process of an epidemic spread. It turns out that for

a critical value of the probability of going on sick leave there is an abrupt decrease in the magnitude of the epidemic. The number of individuals who do not work necessary to suppress the epidemic is very low.

Routine preventive vaccination can be effective only in the case of well-known pathogens (*e.g.* in the case of childhood diseases such as measles). If there is a new pathogen in a susceptible population (as a result of mutation or a bio-terrorist attack), only a quick public health response can provide good results. In such 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 the epidemic spread.

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. Our model provides an opportunity to study the influence of absences from work including preventive closing of workplaces and schools as well as targeted vaccinations on the spread of an epidemic. This is of particular interest since these measures are frequently implemented in practice.

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