

The SIS model for assessment of epidemic control in a social network

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Abstract

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.

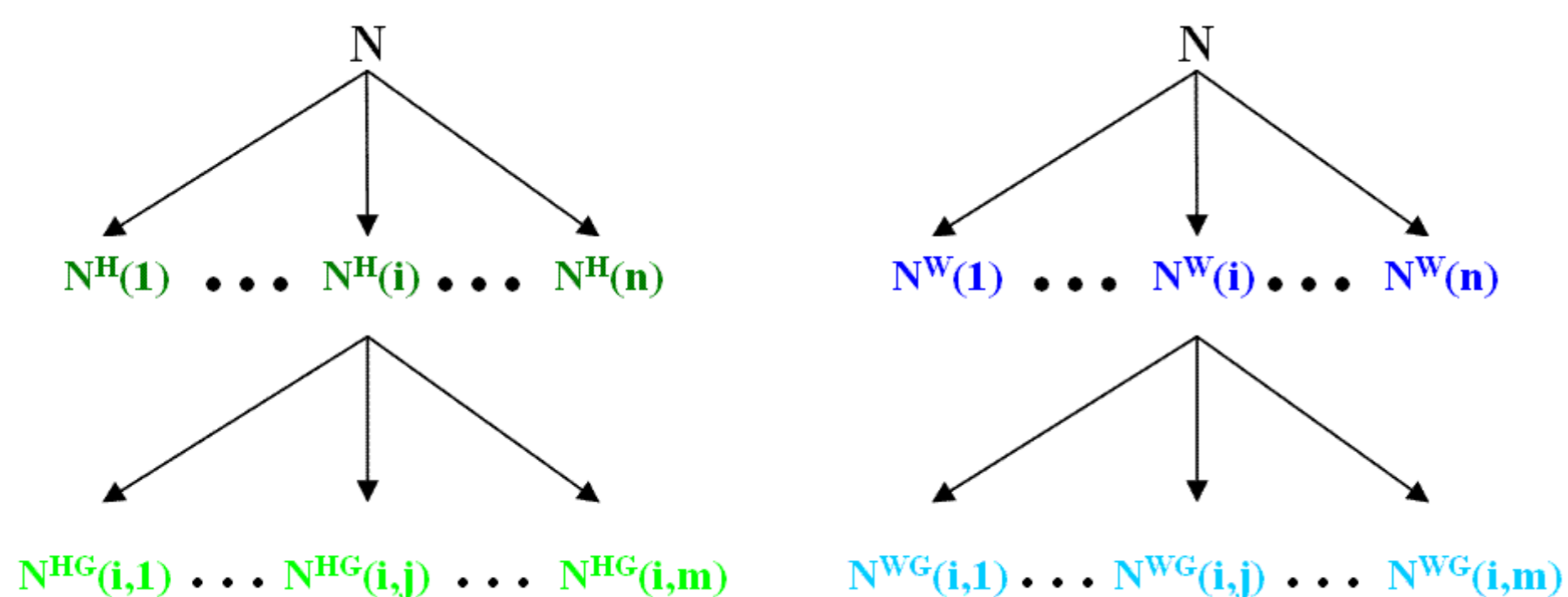
$$S \text{ (susceptible)} \rightarrow IN \text{ (infected)} \rightarrow IL \text{ (ill)} \rightarrow R \text{ (resistant)} \rightarrow S$$

The probabilities of transition between states are: $W_{S \rightarrow IN}$; $W_{IN \rightarrow IL}$; $W_{IL \rightarrow R}$ and $W_{R \rightarrow S}$

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. Cost-effectiveness of mass immunization campaigns, target vaccinations and the sick leaves is compared. Critical vaccination coverage, sufficient for the suppression of an epidemic as well as the probability that an endemic state occurs, are calculated.

The model

The population consists of N individuals, who interact with each other in a three level hierarchical social network structure. The first level of the structure consists of interactions within the smallest social groups, i.e. households N^{HG} and working teams (or school class in case of schoolchildren) N^{WG} . The second level are the interactions within larger social groups, like coworkers of a company (or people who work in the same place, e.g. in the same building) N^W and individuals who live nearby (e.g. neighbours) N^H . The third-level interactions are the interactions with the whole population (e.g. community of a city) N . The size of N^{WG} groups is drawn from power-law distribution 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. household and working team). Since the chosen groups are a part of larger social groups from the second level of the structure (N^H and N^W), an individual is automatically assigned also to the second-level groups. Such method of modeling of social interactions give nontrivial properties of real social networks, e.g. small world topology of connections, large clustering coefficient, assortative mixing and hierarchical structure [1].



To differentiate the effectiveness of pathogen transmission between the interactions in different levels of the hierarchy we introduce three equations describing the probability 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 [2,3] and has a form:

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

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

The probability of acquiring infection during second-level interactions has a form:

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

The probability p_3 of infection caused by a random contact between individuals from the same population does not depend on the localization of the individuals and we assume it has the following form:

$$p_3 = W_{S \rightarrow IN} \left(\frac{N_{IL}}{N} \right)^2$$

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 he or she is still sick. On the other hand, if he or she recovers before the sick leave period is over they do not return to work earlier. This assumption allows 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 influence of target vaccination on the process of epidemic development we introduce the parameter p_{TV} . In each time step all susceptible neighbors (S) of an ill individual belonging to the same N^{HG} and N^{WG} , are vaccinated with probability p_{TV} . After vaccination, these individuals become unsusceptible R (for simplification of the model we assumed that time needed to develop immunity is very short – not longer than one day). It should be noted, that introducing of probability p_{TV} can be also treated as a simple model of chemoprophylaxis [4]. The value of probability p_{TV} is related to the time of identification of ill individuals in population by health services.

Results

In our model it is possible to investigate influence of sick leaves on the process of epidemic spreading. Fig.1. Illustrates the relation between p_{SL} and the magnitude of the epidemic V defined as the relative number of individuals who went through the disease during epidemic, as well as the probability P_E that an endemic state occurs. For critical value of p_{SL} an abrupt decrease in V and in P_E is visible. This indicates that phase transition occurs $p_{SL} = p_{SL}^c$ and for larger values the epidemic is suppressed. The greater value of $W_{R \rightarrow S}$ (the probability that an individual loses his or hers immunity, e.g. as result of virus mutation), the more abrupt changes in V and P_E are.

Epidemic can be suppressed with use of target vaccination provided the time of identification of ill individuals is sufficiently short (see Fig.2). For p_{TV} large enough, the magnitude of the epidemic V and the number N_V of vaccines, needed to suppress the epidemic, are relatively small even for large values of $W_{R \rightarrow S}$.

The influence of routine preventive vaccination on epidemic spreading is shown in Fig.3. Abrupt decrease in V is visible for large enough number of vaccinated individuals (N_{R0} – number of individuals in the state R in time $t=0$). However, in order to suppress the epidemic the vaccination coverage has to be very high, especially for $W_{R \rightarrow S} > 0$. Surprisingly preventive vaccination can increase the probability P_E that an endemic state occurs.

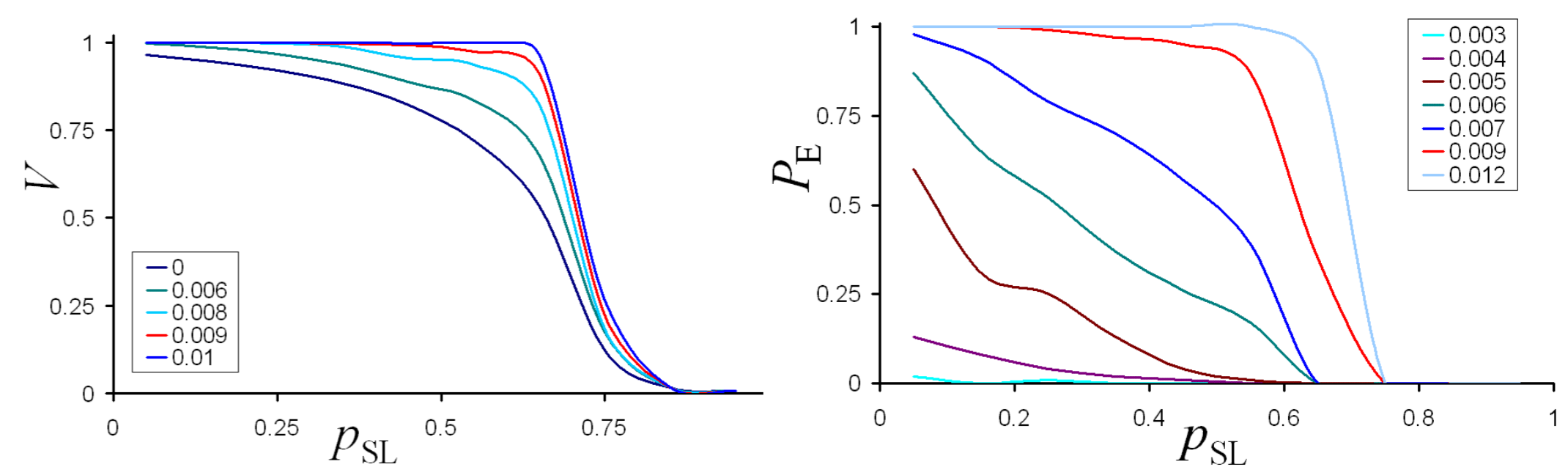


Fig.1. The influence of p_{SL} on range of epidemic V and on the probability P_E that endemic state of epidemic occurs for different values of $W_{R \rightarrow S}$.

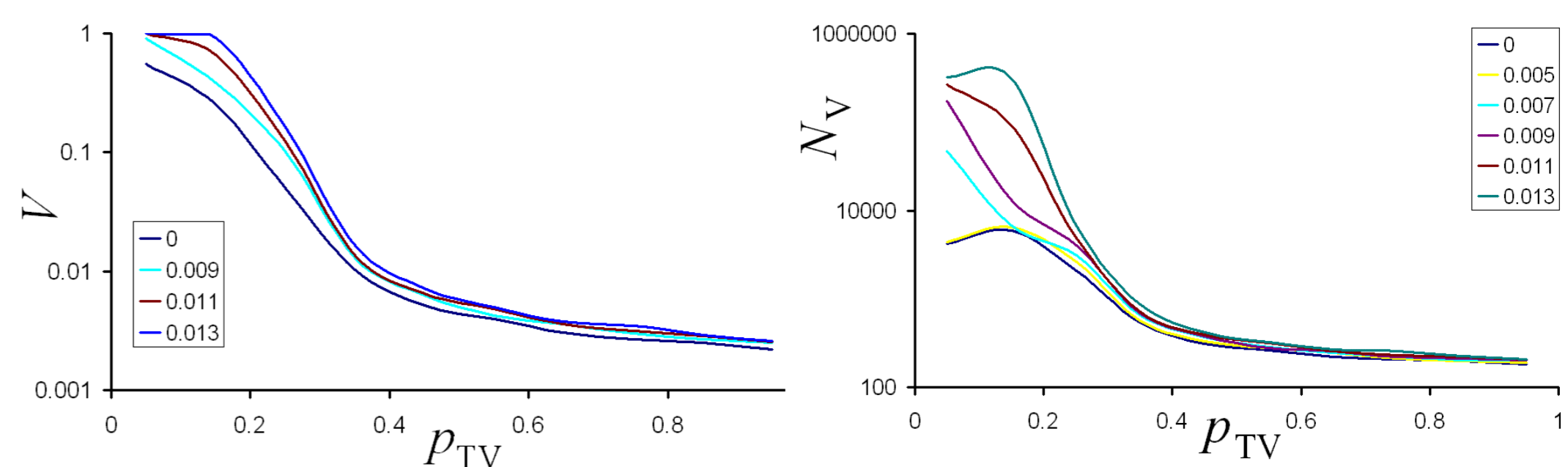


Fig.2. The influence of p_{TV} on range of epidemic V and on the number N_V of vaccines used during epidemic (time of simulation: $T=10000$ time steps) for different values of $W_{R \rightarrow S}$.

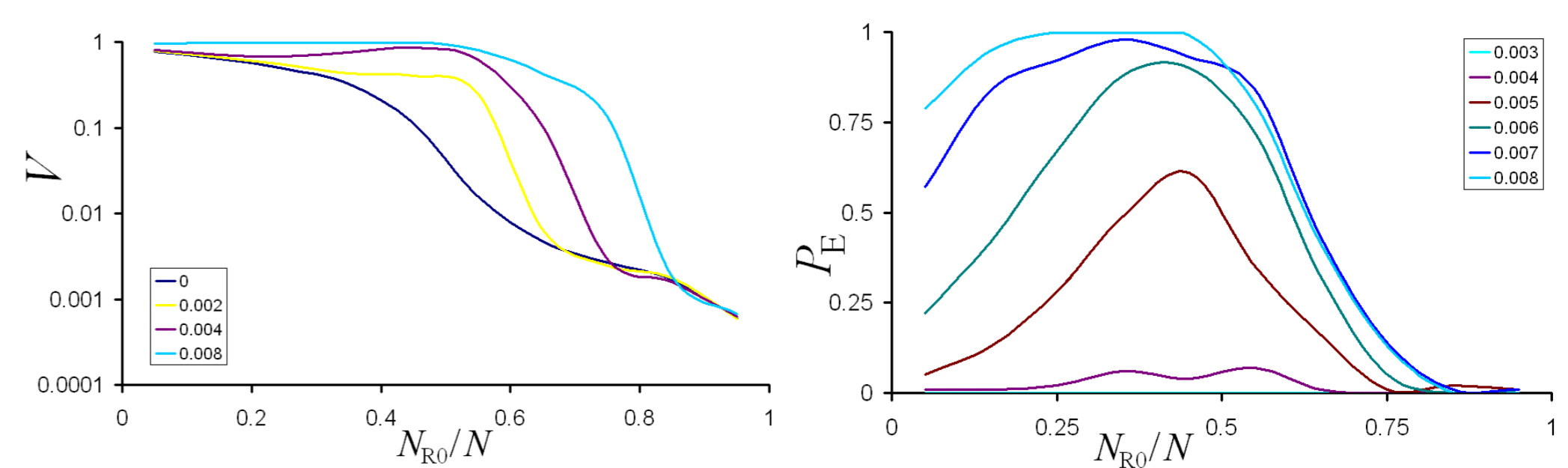


Fig.3. The influence of N_{R0} on range of epidemic V and on the probability P_E that endemic state of epidemic occurs for different values of $W_{R \rightarrow S}$.

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