Simple model of spreading of two epidemics - phase transition and pattern formation

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I. Introduction

In recent years hazards connected with the epidemics like SARS, anthrax, or with computer viruses, increase the role of mathematical modeling and computer simulations of spreading process and result in number of papers devoted to this subject (see e.g. [1-5]). Epidemic spreading may be treated as one of the dynamic phenomena observed in populations described by complex networks [6,7]. In particular small world type of network is used to model the structure of contacts between individuals forming the populations. In our work we investigate the model of spreading of two different diseases with the restriction that an individual can be ill only with one disease in the same time in the frame of SIRS model [1].

S *(susceptible)* ⟶ ***I*** *(ill)*⟶ ***R*** *(unsusceptible)* ⟶ ***S*** *(susceptible)*

Quantities describing the spreading process, like outcome of the epidemic or speed of propagation of the wave-front of the epidemic, with the regard of the role of additional long-range connections, are calculated and discussed. Other interesting observations are suppressing of the epidemic of first disease by the second one and emerging of spatio-temporal patterns.

II. The Model

In our model we investigate the epidemic spreading in the population consisting of $N=LxL$ individuals, located in two-dimensional lattice and connected (with the short range connections) with four nearest neighbors. The network has small-world properties, because long-range connection is added to each individual with probability p. All connections are symmetric. Each individual can become ill with one of two different diseases, however with restriction that an individual can be ill only with one disease in the same time; this property is observed for some pairs of diseases [1]. In this way an individual who is ill with the first disease is unsusceptible to the second disease. We assumed that an individual can be susceptible, ill or unsusceptible (the SIRS model) and because of presence of two different diseases each individual can be in eight permitted states.

S_{ii}	First disease	Second disease	Possible next states
1	susceptible	susceptible	2,4
2	susceptible	ill	3
3	susceptible	unsusceptible	1,4,5
4	ill	susceptible	6
5	ill	unsusceptible	4,6,8
6	unsusceptible	susceptible	1,2,7
7	unsusceptible	ill	2,3,8
8	unsusceptible	unsusceptible	1,3,6

Susceptible individual can be infected with the probability β_1 by neighbors who are ill with the first disease and with probability β_2 by neighbors who are ill with the second disease. Ill individual become unsusceptible with probability γ_1 and γ_2, for the case of first and second disease respectively. In our model we assumed also that an individual can lose its immunity to a disease with probability α_1 and α_2. The states of the individuals as well as their possible next states are presented in Table 1.

III. Results. The Phase Transition

Computations were performed for the initial conditions with two ill individuals in the states 2 and 4, respectively, and the rest of population in the state 1. Ill individuals were located in opposite corners of the lattice. In the case of a lack of additional shortcuts ($p = 0$) the spreading process is similar to the propagation of the two different wave fronts which, after some time steps, spread over the whole population, meeting together in the middle of the lattice.

For $\alpha_1 = \alpha_2 = 0$ most of ill individuals are on the forehead of the wave front. In this condition the range of epidemic (V_i – the number of individuals, which were at least on time infected by i-th disease) depends significantly on the values γ_1 and γ_2. In the case of a collision of two different wave fronts ill individuals can not be infected by another disease. Hence, the propagation process stop, until some ill individuals become unsusceptible. When γ_1 is much smaller than γ_2 , the probability that an individual will be infected with first disease is much larger. In those conditions the first wave front can stopped the second one. Thus, the second epidemic can be suppressed by the first one – it is like "fighting fire with fire". In Fig.1 the influence of γ_1 on the range of epidemic V_2 is shown. For $\gamma_1 > \gamma_{1C}$ almost whole population was infected by second disease, V_2=1. However, for $\gamma_1 < \gamma_{1C}$ the range of second disease is restricted only to half of population. As we see for critical value of $\gamma_1 = \gamma_{1C}$ occurs a phase transition. When there is a decrease in γ_2 , there is a decrease in γ_{1C} and the changes of V_2 in function of γ_1 are more abruptly. In the case when γ_1 is close to 1 the opposite situation is visible, because the difference between γ_1 and γ_2 is large enough (see inset in Fig.1. for p=0).

It is interesting to investigate the influence of the presence of the long-range connections also for the case $\alpha_1=\alpha_2=0$. In the case when γ_1 and γ_2 are large, the presence of short-cuts cause that the range of epidemic is greater (Fig.2). For large enough p almost whole population will go through both diseases. However, for $\gamma_1 \ll \gamma_2$ additional long-range connections slightly influence the range of epidemic. We can distinguish three different ranges of the parameter γ_1 (Fig.2). In the first range, before phase transition $\gamma_1 < \gamma_{1C}$, the presence of short-cuts do not influence V_2. For $\gamma_1 > \gamma_{1C}$ the range of epidemic decreases with increase in p. The greater γ_1 the smaller changes of V_2 are observed. In intermediate region of values of parameter γ_1 initially V_2 decreases with increasing p , however for large enough p an increase in V_2 is visible.

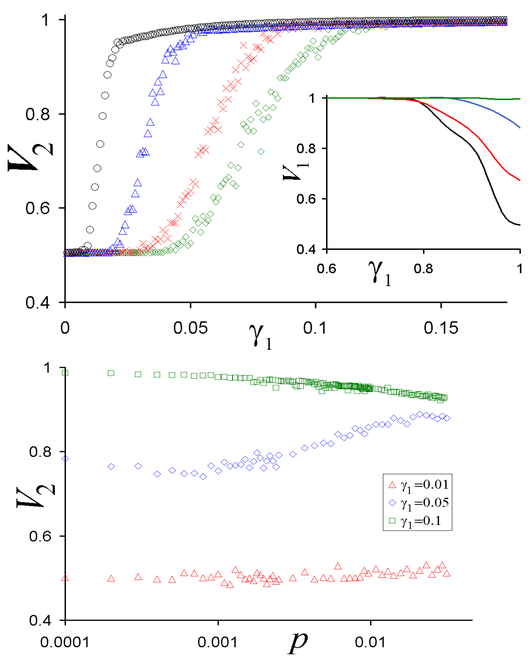

Fig1. The range of epidemic V_2 in function of γ_1 for different values of γ_2 .(γ_2=0.1 - circles; γ_2=03 - triangles; γ_2=0.7 - crosses and γ_2=1 - diamonds) The values of the other parameters are: $\beta_1=\beta_2=1$, $\alpha_1=\alpha_2=0$, $p=0$.
Inset: The range of epidemic V_1 in function of γ_1 for different values of p (0; 0.0001; 0.0004; and 0.0016, from bottom to top respectively) The values of the other parameters are: γ_2=0.5, $\beta_1=\beta_2=1$, $\alpha_1=\alpha_2=0$. Results were averaged over 100 independent simulations.

Fig.2. The range of epidemic V_2 in function of p for different values of γ_1 (γ_1=0.01 - triangles; γ_1=0.05 - diamonds; and γ_1=0.1 - squares) The values of the other parameters are: γ_2=0.5, $\beta_1=\beta_2=1$, $\alpha_1=\alpha_2=0$, p=0. Results were averaged over 100 independent simulations.

IV. Speed of Epidemic Spreading

The speed of propagation of wave-front of epidemic v in the case of one disease is connected only with probability of infection. However in the case of two epidemics this equation is more complicated. We assume symmetric values of parameters, i.e. $\alpha_1=\alpha_2=\alpha$, $\beta_1=\beta_2=\beta$ and $\gamma_1=\gamma_2=\gamma$ to simplicity the calculation of v and assume that initially two ill individuals are located in opposite corners of the lattice. In consequence two different wave-fronts propagate to center of the lattice with speed $v=\beta$. After collision value of v changes. This phenomenon is similar to phenomenon of diffusion of two different liquids. To calculate v we use master equation. Before the collision, in one half of the lattice all individuals are ill with the same disease, therefore:

$$dS(t)/dt = \alpha R(t) - \beta I(t)S(t)$$
$$dI(t)/dt = \beta I(t)S(t) - \gamma I(t)$$
$$dR(t)/dt = \gamma I(t) - \alpha R(t) \qquad (1)$$

where $S(t)$, $I(t)$, $R(t)$ are the probabilities that an individual is susceptible, ill and unsusceptible, respectively. In each time step the relation $S(t) + I(t) + R(t) = 1$ is true. We assume that system is in stationary state, hence we obtain:

$S = \gamma\,\beta^{-1}$; $I = \alpha\,(\beta-\gamma)\,[\,\beta\,(\gamma+\alpha)\,]^{-1}$; $R = \gamma\,(\beta-\gamma)\,[\,\beta\,(\gamma+\alpha)\,]^{-1}$.

Individuals susceptible and unsusceptible on first disease can be infected with second disease. The probability that one of them can be infected equals $S+R$ and this individual is infected with probability β. Hence, the speed of propagation of the wave-front is $v = \beta\,(S+R) = \beta\,(1-I)$. After calculation we obtain

$$v = \gamma\,(\beta+\alpha)\,(\gamma+\alpha)^{-1} \qquad (2)$$

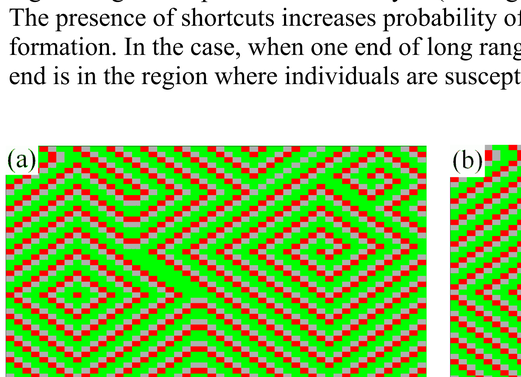

Fig.3.
Relation between the speed v of wave-front and γ ($\gamma_1=\gamma$ and $\gamma_2=\gamma$) for different values ofα ($\alpha_1=\alpha$ and $\alpha_2=\alpha$), α=0.01 – crosses; α=0.1 –diamonds and α=0.3 – triangles and p=0. Good agreement with analytical results (solid line) is visible. The values of the other parameters are: $\beta_1=\beta_2=1$. Results were averaged over 100 independent simulations.

V. Pattern Formation

The other aspect of the spreading process studied in our work is the pattern formation. Spatial pattern formation is observed in many physical, chemical and biological systems. The study of pattern formation and pattern dynamics enable to detect and describe many interesting nonlinear phenomena in these systems (e.g. phase transitions) and characteristics of local interactions (see e.g. [8,9]). To biological systems belongs also the population (a kind of complex system) investigated in the present paper.

Invasion of a pathogen in the population, which starts from an infected individual and with certain probability reaches its neighbors, may be treated as a propagation of an excitation in the system. We found that in special conditions in the system under consideration, i.e. all parameters describing diseases are equal 1 ($\alpha_1=\alpha_2=1$; $\beta_1=\beta_2=1$; $\gamma_1=\gamma_2=1$), interesting spatio-temporal patterns, in particular of a form of spiral waves, can emerged. Such effect of epidemic spreading has not been investigated so far, to our knowledge. Spiral waves emerge as a result of collision of two wave-fronts connected with different diseases and with specific mutual orientation. The additional long-range connections do not destroy spiral waves, moreover they can increase the probability of emerging of spiral waves, as was observed earlier in Ref. [10]. This is so, because with an increase in the number of shortcuts there are more secondary sources of epidemic and more collisions of different wave-fronts. However, a fraction of shortcuts should be so small ($p \ll 1$) that they cannot spoil the propagation of spiral waves. Spiral waves are also formed, when some parameters (α, β, γ) are slightly smaller than 1, in particular the influence of change of is the weakest. The system lose its deterministic character, and patterns are noised. The level of noise (i.e. the number of individuals which are not in the proper states in comparison to pattern) increases with time and when it reaches high enough level patterns are destroyed (see Fig.7).

The presence of shortcuts increases probability of creating spiral waves, but it is not their only influence on pattern formation. In the case, when one end of long range connection is reached by forehead of spiral wave and the second end is in the region where individuals are susceptible a spherical wave can be formed there.

Fig.4. Spiral waves in the case when only one disease survive. Individuals ill with first disease are black. In the left part of the lattice there is a source of spherical wave, which is caused by the end of a shortcut. The second end of this shortcut is near the source of spiral wave. In this way spiral wave and shortcut induce the formation of spherical wave. For better clarity the case when only one disease survive is depicted.

Fig.5.The process of formation of the spiral wave after collision of wave-fronts of two different epidemics. The first figure illustrates early phase of collision. Next figures show zoomed fragment of the lattice where spiral-wave is forming. They correspond to the state of the system in first time steps after collision. The last figure shows the final state of the system.

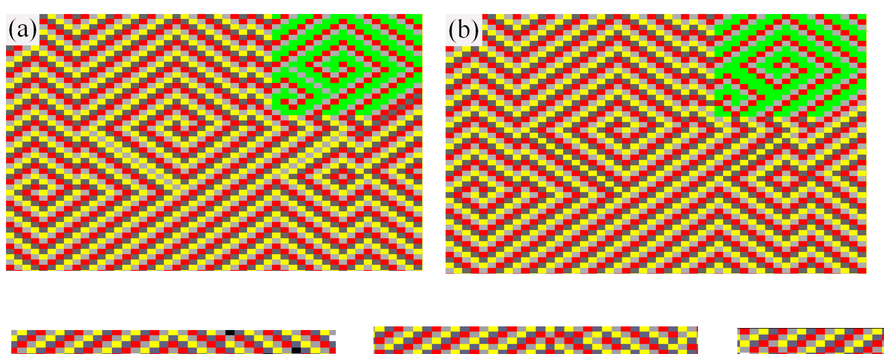

Fig.6. Spiral waves for two diseases. In the upper-right corner of the lattice individuals are ill only with first disease, as result of presence of two spiral waves in this region. Wave-fronts generated in this region built some kind of a barrier, which hold propagation of the second epidemic.

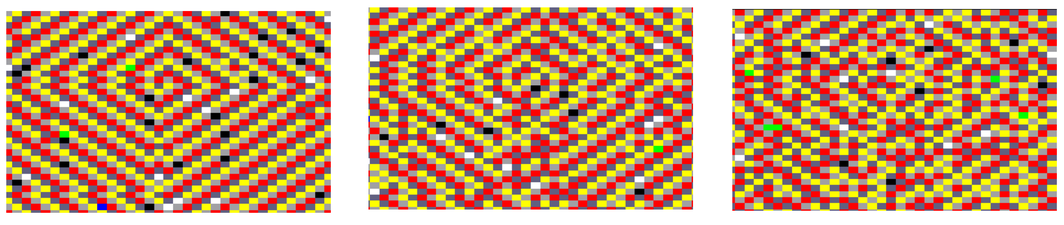

Fig.7. Effect of the noise in the case of not deterministic system ($\alpha_{1,2}<1$). Spiral wave is formed, but the level of the noise increases with time. When the level of the noise is high enough the pattern is destroyed.

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