# SIMPLE MODEL OF SPREADING OF TWO EPIDEMICS: PHASE TRANSITION AND PATTERN FORMATION*

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Spreading of two different diseases in small world network, with restriction that an individual can be ill only with one disease in the same time, is investigated in the frame of SIRS model. It was found that in the special range of control parameters the presence of the second disease can significantly decrease the number of individuals, who passed the first disease. The speed of propagation of the wave-front of the epidemic is calculated analytically and good agreement with numerical calculation is obtained. The influence of additional long range connections on epidemic spreading and phase transition is investigated. It is found that in special conditions spatio-temporal patterns, in particular spiral waves, can emerge in the system. Small number of additional long-range connections increases the probability of emerging of spiral waves.

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## 1. Introduction

In recent years hazards connected with the epidemics like SARS, anthrax, or with computer viruses results in a number of papers devoted to this subject (see *e.g.* [1–9]), which are based on the mathematical modeling and computer simulations of spreading processes. Epidemic spreading may be treated as one of the dynamic phenomena observed in populations described by complex networks [10]. In particular small world type of network [11] is used to model the structure of contacts between individuals

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forming the populations [1, 3, 12–16]. Different approaches, like application of the percolation theory [3,15,17,18] or mean field approximation [6,19–21], were used to obtain characteristics of spreading process. In our work we investigate the model of spreading of two different diseases, in the frame of SIRS model [19], with the restriction that an individual can be ill only with one disease in the same time. The structure of contacts between individuals in population is given by small world network. Quantities describing the spreading process, like outcome of the epidemic or speed of propagation of the wave-front of the epidemic, including the role of additional long-range connections, are calculated and discussed. Other interesting observation is that the epidemic of the first disease can be suppressed by the second one.

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.* [22–26]). 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 interesting spatio-temporal patterns, in particular of a form of spiral waves, can emerge. Such effect of epidemic spreading has not been investigated so far, to our knowledge.

## 2. The model

In our model we investigate the epidemic spreading in the population consisting of $N = L \times L$ individuals ($S_{ij}$), 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 the restriction that an individual can be ill only with one disease in the same time; this property is observed for some pairs of real diseases [19]. 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 the presence of two different diseases each individual can be in eight permitted states. Susceptible individual can be infected with the probability $\beta_1$ by neighbors who are ill with the first disease or with the probability $\beta_2$ by neighbors who are ill with the second disease. The number of ill neighbors is important only in the case when an individual has some neighbors ill with

the first disease and some other neighbors ill with the second disease. In such a case at first is checked the probability of infection with respect to a disease with which more neighbors is ill. If an individual become infected, the probability of infection with other disease is not checked, because an individual cannot be ill with both diseases in the same time.

Ill individual become unsusceptible with probability $\gamma_1$ and $\gamma_2$, 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 $\alpha_1$ and $\alpha_2$. The states of the individuals as well as their possible next states are presented in Table I.

TABLE I

The list of possible states $S_{ij}$ and permitted, subsequent states of an individual.

| $S_{ij}$ | 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 |

## 3. Results

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 the 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 (denoted by $V_1$ — the number of individuals, which were at least one time infected with the first disease and $V_2$ — the number of individuals, which were at least one time infected with the second disease) depends significantly on the values $\gamma_1$ and $\gamma_2$. In the case of a collision of two different wave fronts ill individuals cannot be infected by another disease (see Table I). Hence, the propagation process

stops, until some ill individuals become unsusceptible. When $\gamma_1$ is much smaller than $\gamma_2$, the probability that an individual will be infected with the first disease is much larger. This is so, because individuals ill with the first disease have long lasting immunity on the second disease and individuals ill with the second disease quickly recover and become unsusceptible. The width of the first wave front is much larger than width of the second wave front. In those conditions the first wave front can stop 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 $\gamma_1$ on the range of epidemic $V_2$ is shown. For $\gamma_1 > \gamma_{1C}$ almost whole population was infected with second disease and $V_2 \approx 1$. However, for $\gamma_1 < \gamma_{1C}$ the range of the 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 $\gamma_2$, there is a decrease in $\gamma_{1C}$ and the changes of $V_2$ in function of $\gamma_1$ are more abrupt. In the case when $\gamma_1$ is close to 1 the opposite situation is visible, because large enough is the difference between $\gamma_1$ and $\gamma_2$ (see inset in Fig. 1 for $p = 0$).

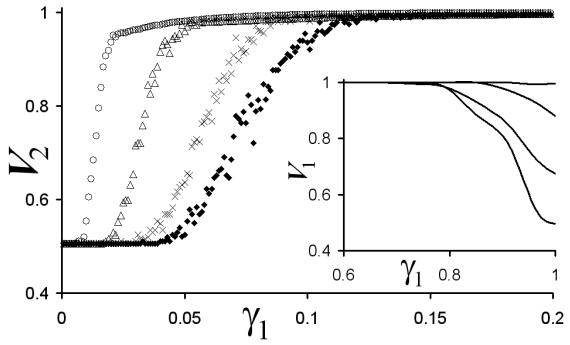


Fig. 1. The range of epidemic $V_2$ in function of $\gamma_1$ for different values of $\gamma_2$ ($\gamma_2 = 0.1$ — circles; $\gamma_2 = 0.3$ — triangles; $\gamma_2 = 0.7$ — crosses and $\gamma_2 = 1$ — diamonds). The inset shows range of epidemic $V_1$ in function of $\gamma_1$ for different values of $p$ (0; 0.0001; 0.0004; and 0.0016, from bottom to top, respectively) and $\gamma_2 = 0.5$. The values of the other parameters are: $\beta_1 = \beta_2 = 1$, $\alpha_1 = \alpha_2 = 0$, $p = 0$, $L = 100$. Results were averaged over 100 independent simulations.

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 $\gamma_1$ and $\gamma_2$ are large, the presence of short-cuts cause that the range of epidemic is greater (see inset in Fig. 1). 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 $\gamma_1$ (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 $\gamma_1$ the smaller changes of $V_2$ are observed. In intermediate region of values of parameter $\gamma_1$ initially $V_2$ slightly decreases with increasing $p$, however, for large enough $p$ an increase in $V_2$ is visible.

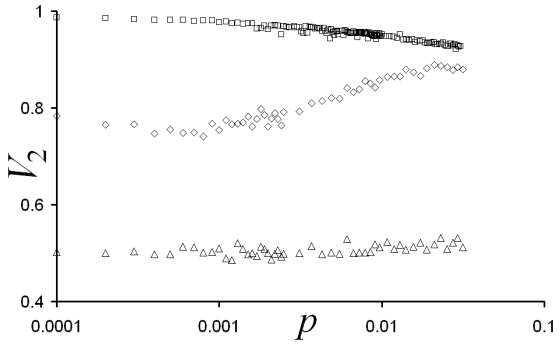


Fig. 2. The range of epidemic $V_2$ in function of $p$ for different values of $\gamma_1$ ($\gamma_1 = 0.01$ — triangles; $\gamma_1 = 0.05$ — diamonds; and $\gamma_1 = 0.1$ — squares). The values of the other parameters are: $\gamma_2 = 0.5$, $\beta_1 = \beta_2 = 1$, $\alpha_1 = \alpha_2 = 0$, $L = 100$. Results were averaged over 100 independent simulations.

The presence of short-cuts change not only the range of epidemic, but also spatial localization of individuals who passed a disease. The number $n_1$ of clusters of individuals who are unsusceptible only to the first disease ($S_{ij} = 6$) depends significantly on parameters $\gamma_1$ and $p$ (Fig. 3). It is visible that for the critical value of $\gamma_1$ the behavior of the system change abruptly

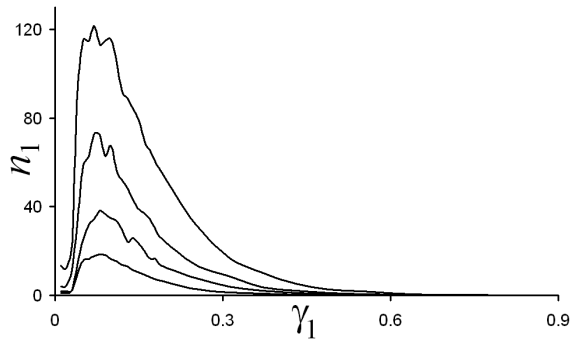


Fig. 3. The relation between the number of clusters $n_1$ of individuals in the state $S_{ij} = 6$ (individuals which have passed only first disease) and $\gamma_1$ for different values of $p$ (0, 0.0016, 0.064, 0.0256 from the bottom to the top, respectively). The values of the other parameters are: $\gamma_2 = 0.5$, $\beta_1 = \beta_2 = 1$, $\alpha_1 = \alpha_2 = 0$, $L = 100$. Results were averaged over 100 independent simulations.

*i.e.* the number of clusters $n_1$ increases rapidly (see maxima of curves in Fig. 3). For narrow range of the values of parameter $\gamma_1$ the presence of short-cuts has strong influence on spatial character of the outcome of the epidemic. For low values of $p$ individuals who passed through only first disease are grouped in a small number of clusters — for small enough $\gamma_1$ there is only one cluster. With increasing $p$ there is an increase in the number of clusters, because there is more secondary sources of epidemic. Therefore, there is an increase in length of all wave-fronts and their shape, as a result of collisions of different wave-fronts is more complicated.

## 4. Speed of epidemic

The speed $v$ of propagation of wave-front of epidemic 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.* $\beta_1 = \beta_2 = \beta$, $\gamma_1 = \gamma_2 = \gamma$ and $\alpha_1 = \alpha_2 = \alpha$ to simplify the calculation of $v$ and assume that initially two ill individuals are located in the opposite corners of the lattice. In consequence two different wave-fronts propagate to the center of the lattice with the speed $v = \beta$. After the collision a value of $v$ changes. This phenomenon is similar to a 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:

$$\begin{cases} dS(t)/dt = \alpha R(t) - \beta I(t) S(t)\,, \\ dI(t)/dt = \beta I(t) S(t) - \gamma I(t)\,, \\ dR(t)/dt = \gamma I(t) - \alpha R(t)\,, \end{cases} \tag{1}$$

where $S(t)$, $I(t)$, $R(t)$ are the probabilities that an individual is susceptible, ill and unsusceptible, respectively. We assume that the system is in a stationary state, hence we obtain: $S = \gamma/\beta$; $I = \alpha(\beta-\gamma)/\beta(\gamma+\alpha)$; $R = \gamma(\beta-\gamma)/\beta(\gamma+\alpha)$. Individuals susceptible or unsusceptible on the first disease can be infected with the second disease. The probability that one of them can be infected equals $S + R$ and this individual is infected with the probability $\beta$. Hence, the speed of propagation of the wave front is $v = \beta(S+R) = \beta(1-I)$. After calculation we obtain

$$v = \frac{\gamma}{\gamma+\alpha}\,(\beta+\alpha)\,. \tag{2}$$

Comparison of analytical calculation with a numerical simulation is shown in Fig. 4. As we see both results agree rather well. Discrepancy in results are connected, first of all, with the assumption that the system is in stationary state, what is not true in large range of values of control parameters.

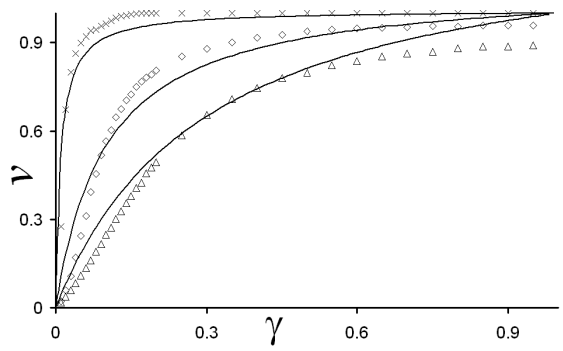


Fig. 4. Relation between the speed $v$ of wave-front and $\gamma$ ($\gamma_1 = \gamma$ and $\gamma_2 = \gamma$) for different values of $\alpha$ ($\alpha_1 = \alpha$ and $\alpha_2 = \alpha$), $\alpha = 0.01$ — crosses; $\alpha = 0.1$ — diamonds and $\alpha = 0.3$ — triangles and $p = 0$. Good agreement with analytical results (solid line) is visible. The values of the other parameters are: $\beta_1 = \beta_2 = 1$, $L = 200$. Results were averaged over 100 independent simulations.

## 5. Spiral waves

In models of epidemic spreading, where there is a non-zero probability that an unsusceptible individual can lose its immunity (SIRS model), or an ill individuals can directly become susceptible (SIS model), some interesting patterns can emerge. Temporal patterns, like oscillations of different types, were earlier observed [27, 28]. Also in the present model such patterns are observed, however, more interesting are spatio-temporal patterns which can emerge in special conditions. In the case when all parameters describing a disease are equal 1 (*i.e.* $\alpha_i = \beta_i = \gamma_i = 1;\ i = 1, 2$) as a result of collision of two wave-fronts (connected with different diseases) a spiral wave can be formed. It is surprising that 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. [29]. 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 that they cannot spoil the propagation of spiral waves ($p < L^{-1}$). Spiral waves are also formed, when some parameters $(\alpha, \beta, \gamma)$ are slightly smaller than 1, in particular the influence of change of $\gamma$ is the weakest. Then the system loses 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 surrounding pattern) increases with time and when it reaches high enough level patterns are destroyed.

In some cases individuals ill with one of the diseases vanish and spiral waves are formed only by individuals ill with the same disease (see Fig. 5 where two different spiral waves are shown). Fig. 6 illustrates more compli-

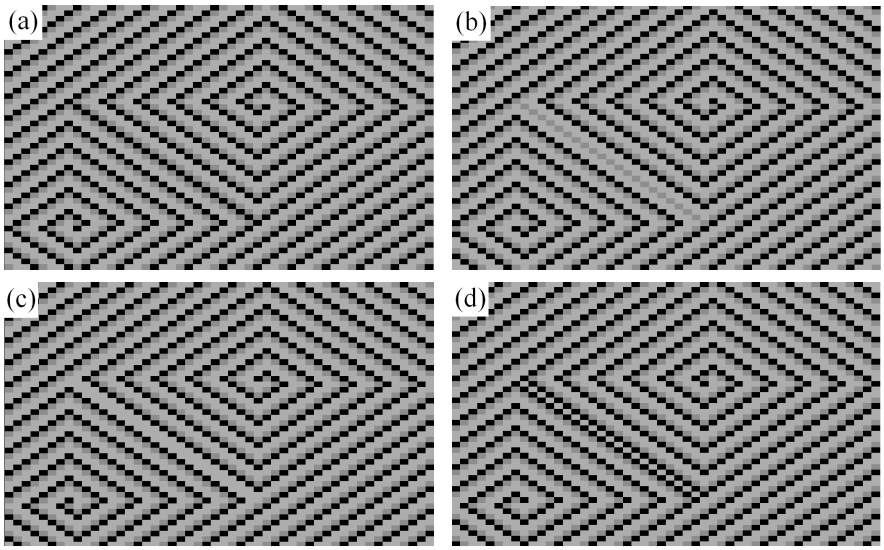


Fig. 5. Two spiral waves in the case when only one disease survive. The system after four time steps go back to its initial state (a). Individuals ill with the first disease, unsusceptible or susceptible are black, dark gray or light gray, respectively.

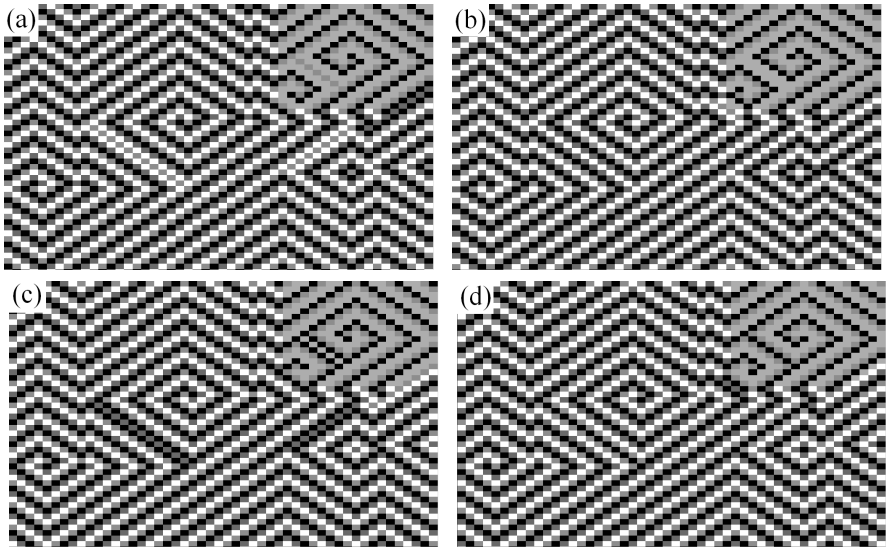


Fig. 6. Spiral waves for two diseases. Individuals ill with the first or the second disease are black or white, respectively. In the upper-right corner of the lattice individuals are ill only with the first disease, which is a result of the presence of two spiral waves in this region.

cated case with individuals ill with the first or second disease forming spiral waves in major part of the lattice. At the same time in the upper-right corner of the lattice two spiral waves, in which individuals are ill only with the first disease, are observed. Wave-fronts generated in this region built some kind of a barrier, which holds propagation of the second epidemic.

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 a long range connection is reached by the forehead of spiral wave and the second end is in the region where individuals are susceptible a spherical wave is formed there.

## 6. Conclusions

In this work 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 and with the contacts between individuals in the form of small world network, has been investigated. The outcome of epidemic, like its range as a function of removal rate $\gamma$, has been calculated. We observed that for special range of control parameters the epidemic of the first disease is suppressed by the second one. We investigated the influence of the presence of additional long-range connections on range of epidemic and spatial localization of individuals who passed a disease. The speed of propagation of the wave-front of the epidemic has been calculated analytically and good agreement with numerical calculations was obtained.

It has been found that pattern formation (*e.g.* spiral and spherical waves), commonly observed in different physical systems, can occur also in an epidemic spreading, when two different diseases propagate in a system. As it results from our observations, the necessary condition for pattern formation is the restriction that an individual ill with the first disease is unsusceptible to the second disease and vice versa. Such interactions between two pathogens and immune system of an individual is observed in the nature [19]. An interesting property of the system is that the presence of small number of additional long-range connections ($p \ll 1$) increases the probability of forming spiral waves. However, the presence of long-range connections is not necessary for pattern formation; it can emerge also for some special localization of initial sources of epidemic.

## REFERENCES

[1] C.F. Moukarzel, *Phys. Rev.* **E60**, R6263 (1999).

[2] H.K. Janssen, K. Oerding, F. van Wijland, H.J. Hilhorst, *Eur. Phys. J.* **B7**, 137 (1999).

[3] C. Moore, M.E.J. Newman, *Phys. Rev.* **E61**, 5678 (2000).

[4] R. Pastor-Satorras, A. Vespignani, *Phys. Rev. Lett.* **86**, 3200 (2001).

[5] P.M. Tarwater, C.F. Martin, *Complexity* **6**, 29 (2001).

[6] Y. Moreno, J.B. Gomez, A.F. Pacheco, *Phys. Rev.* **E68**, 035103(R) (2003).

[7] A. Grabowski, R. Kosiński, *Phys. Rev.* **E70**, 031908 (2004).

[8] R. Olinky, L. Stone, *Phys. Rev.* **E70**, 030902(R) (2004).

[9] B. Dybiec, A. Kleczkowski, C.A. Gilligan, *Acta Phys. Pol. B* **36**, 1509 (2005).

[10] N. Madar, T. Kalisky, R. Cohen, D. ben-Avraham, S. Havlin, *Eur. Phys. J.* **B38**, 269 (2004).

[11] D.J. Watts, S.H. Strogatz, *Nature* **39**, 440 (1998).

[12] M.E.J. Newman, D.J. Watts, *Phys. Rev.* **E60**, 7332 (1999).

[13] A. Ramezanpour, V. Karimipour, *Phys. Rev.* **E66**, 036128 (2002).

[14] J. Lahtinen, J. Kertesz, K. Kaski, *Physica* **A311**, 571 (2002).

[15] L.M. Sander, C.P. Warren, I.M. Sokolov, C. Simon, J. Koopman, *Math. Biosciences* **180**, 293 (2002).

[16] R.A. Kosiński, Ł. Adamowski, *Int. J. of Mod. Phys.* **C15**, 755 (2004).

[17] H. Hinrichsen, M. Howard, *Eur. Phys. J.* **B7**, 635 (1999).

[18] L. Turban, *Eur. Phys. J.* **B33**, 331 (2003).

[19] N.T.J. Bailey, *The Mathematical Theory of Infectious Diseases*, Springer, 1993.

[20] M.J. Keeling, *Proc. R. Soc. Lond.* **B266**, 859 (1999).

[21] M.A. Aguirre, G. Abramson, A.R. Bishop, V.M. Kenkre, *Phys. Rev.* **E66**, 041908 (2002).

[22] K. Kawasaki, M. Suzuki, A. Onuki, *Formation Dynamics, Statistics of Patterns*, World Scientific, 1990.

[23] T.R.J. Bossomaier, D.G. Green, *Complex Systems*, Cambridge University Press, 2000.

[24] P.G. Lind, J. Corte-Real, J.A.C. Gallas, *Phys. Rev.* **E69**, 066206 (2004).

[25] K.R. Crounse, L.O. Chua, P. Thiran, G. Setti, *Int. J. Bifurcation, Chaos* **6**, 1703 (1996).

[26] A.L. Lin, A. Hagberg, E. Meron, H.L. Swinney, *Phys. Rev.* **E69**, 066217 (2004).

[27] M. Girvan, D.S. Callaway, M.E.J. Newman, S.H. Strogatz, *Phys. Rev.* **E65**, 031915 (2002).

[28] A. Grabowski, R.A. Kosiński, *Acta Phys. Pol. B* **36**, 1579 (2005).

[29] D. He, G. Hu, M. Zhan, W. Ren, Z. Gao, *Phys. Rev.* **E65**, 055204(R), (2002).