Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.
Critical properties of the SIS model on the clustered homophilic network

F.L. Santos, M.L. Almeida, E.L. Albuquerque, A. Macedo-Filho, M.L. Lyra, U.L. Fulco

Abstract

The spreading of epidemics in complex networks has been a subject of renewed interest of several scientific branches. In this regard, we have focused our attention on the study of the susceptible–infected–susceptible (SIS) model, within a Monte Carlo numerical simulation approach, representing the spreading of epidemics in a clustered homophilic network. The competition between infection and recovery that drives the system either to an absorbing or to an active phase is analyzed. We estimate the static critical exponents $\beta/\nu$, $1/\nu$ and $\gamma/\nu$, through finite-size scaling (FSS) analysis of the order parameter $\rho$ and its fluctuations, showing that they differ from those associated with the contact process on a scale-free network, as well as those predicted by the heterogeneous mean-field theory.

© 2020 Elsevier B.V. All rights reserved.

1. Introduction

During the last decades, the scientific community has been paying a lot of attention to the important issue of how diseases spread across a population. Several epidemiological models have been proposed to date to address this question and to make relevant contributions to the public-health domain around the world. Within this perspective, a classic epidemic susceptible–infected–susceptible (SIS) model plays an essential role in modeling infectious diseases in which recovered individuals do not acquire any long-lasting immunity to the disease and are again susceptible to infection upon recovery. In this context, the SIS model can be used to describe sexually transmitted diseases, such as syphilis or gonorrhea, in which individuals are treated and recovered, although immediately susceptible anew.

It seems to be a crucial issue to take a very close look at the people’s pattern of interactions to better understand how a spreading disease has been mostly taking place. It is known that numerous infectious diseases in the world are transmitted by vectors, by direct contact between healthy and diseased individuals or by animals. The infectious diseases are caused by spread pathogenic microorganisms, such as bacteria, viruses, parasites or fungi. When these diseases become epidemics, they spread rapidly and many deaths can happen in a short time somewhere on the planet. Thus, the scientific community devotes efforts to understand the transmission of this disease and to have greater control over them.

In this work, we intend to investigate an epidemic spreading process in a more realistic system with the architecture of a complex network with an added clustering degree. Complex networks describe a wide range of systems in nature...
and society. An important aim of network modeling is to incorporate in the interaction among individuals the already known characteristics of epidemics [1–5]. Each new model scientifically proposed for the study of an epidemic process is an attempt to approach the reality of this epidemic or one of its characteristics through a combination between the proposed model and the own network where the epidemic is developing. Here, we will simulate an epidemic process based on the SIS model in a complex network called homophilic network (HN).

The homophilic network is a complex network introduced in 2013 by Almeida et al. [6,7], who modified the Barabási–Albert (BA) network model [8], introducing a similarity factor influencing the connection occurrence. While in the Barabási–Albert model the links between sites depend only on the connectivity, in the homophilic one we have the similitude factor. It denotes the tendency of sites to link with others, which are similar to themselves. In particular, homophily refers to how the preferential attachment privileges the links between new sites and those having a high number of the nearest neighbors and high similitude jointly. Concerning the clustering coefficient, BA-like models have a low value which is quite different from what many real-world systems have exhibited. For instance, social networks are highly clustered, meaning that everyone scarcely knows each other.

Aiming to capture this feature, a new model for complex networks was recently proposed [9] based on the formation of triangles (cycles of three nodes linked one another). The new model, known as clustered scale-free network, is built based on the sense of increasing the probability that two nearest neighbors of a given node are themselves connected, tuning up a high clustering coefficient for the network constructed.

In this paper, we will assume that in a time step each individual occupies a site (vertex) of the network and is identified as an individual in one of two possible states: healthy (inactive) and infected (active). The dynamics of interaction between the individuals obey local Markovian rules. If they are healthy and one or more connected sites in the network are occupied by infected (active) persons, they will be infected with probability $\lambda$ (control parameter). On the other hand, if they are infected, they will simply turn healthy. In this context, the system evolves to a stationary state with a finite density of infected individuals $\rho$ for low values of the control parameter $\lambda$ with a critical point $\lambda_c$ above it the system falls in an absorbing vacuum state. At the critical point $\lambda_c$ the system exhibits a phase transition. The stationary density of infected individuals $\rho$ is the order parameter that vanishes in the absorbing state and is strictly positive in the active one.

Our main focus here is the investigation of the properties of the SIS model on a clustered homophilic network (CHN) within a Monte Carlo numerical simulation approach. Numerical simulations in regular networks have shown that the phase transition between active and inactive regimes belongs to the directed percolation universality class [10]. A previous study of the contact process (CP) model on the homophilic network unveiled that the numerical values of the critical exponents, obtained from the heterogeneous mean-field theory and from a standard scale-free network model, were different from those found on the homophilic one [11].

Within this context, Pastor-Satorras [12,13] showed that scale-free networks will have a phase transition to $\rho = 0$ at $\lambda = 0$. To get around this issue, we reformulated the homophilic network to include an additional clustering process, named the clustered homophilic network. In order to extract the critical indexes, a finite-size scaling analysis will be employed to characterize a set of critical exponents $(1/\nu, \beta/\nu, \beta$ and $\gamma/\nu)$, whose universality class differs from those associated to the CP model on the unclustered homophilic network.

Complex networks have been useful to describe many biological and physical systems [14–18], as well as epidemic processes [19–21]. Recently, there is a widespread understanding that our global social interactions reside in the world of networks, mainly after the rapid way that SARS-CoV-2 spread worldwide. The community has learned that the term “network”, for example, the internet network, has changed our lives a lot. In addition, more attention has been devoted to the study of social [22], cultural phenomena and the spread of viruses on computers [23], and epidemic diseases [19,20] in complex networks. Recently, the CP and SIS models were shown to share the same universality class in the deterministic Apollonian network [11,19,20], while the former model in the homophilic network exhibited a phase transition [11]. Pastor-Satorras [12,13] showed that there was no phase transition in the SIS model in a random scale-free network model. However, real complex networks display some degree of local clustering besides the scale-free character. Therefore, the main motivation of the present work is to investigate the possible occurrence of an absorbing state phase transition in the SIS model in the clustered homophilic network, and to unveil its universal characteristics.

This work is organized as follows. In Section 2 we present the scale-free homophilic network and its new clustered variant. The model and the algorithm used in the numerical simulations are described in Section 3. Section 4 reports our main numerical results, while the summary and conclusions of this work is the main topic of Section 5.

### 2. The homophilic network

In this section, we describe the homophilic network (HN) and its clustered variant CHN. To take into account the different site skills to compete for links, a term called similitude is introduced and defined as $S_{ij} = |\eta_i - \eta_j|$, where $\eta_i$ ($\eta_j$) is an intrinsic characteristic of site $i$ ($j$). Note that $S_{ij}$ increases significantly the link formation among similar sites at a higher rate than amidst dissimilar ones (see Fig. 1a).

The homophilic network HN can be generated according to the following rules [11]:

(a) the network begin with $m_0$ sites characterized by $\eta_i$’s, which are unchanged in time and chosen as randomly values ranging from 0 to 1;
Fig. 1. Schematic representation of how the scale-free homophilic model and the clustered homophilic network CHN grow. (a) The NH: In time step \( t_0 \) the number of sites is \( m_0 = 2 \). In the following time steps \( t_1, t_2, \) and \( t_3 \), each arriving site makes only one new connection \( m = 1 \), with the pre-existing site defined according to Eq. (1). (b) The CHN: In the preferential attachment step (step (a)), the new site \( j \) chooses a site \( i \) to be linked with a probability proportional to its number of nearest neighbors and the similitude between them. In the triad formation step (b) the new site \( j \) chooses a site \( s \) in the neighborhood of the site \( i \), linked to it in the previous preferential attachment step. Here the symbol \( \times \) stands for “not allowed to attach to”.

(b) at every time step, a new site \( j \) with \( \eta_j \) attributed randomly connects to other \( m \)'s (\( \leq m_0 \)) pre-existing sites by undirected and unweighted new links (multiple leaks between two sites are not allowed);

(c) the connections are established by considering the linking probability depending on the degree of site \( i \), labeled \( k_i \), and on the similitude \( S_{ij} \), defined by:

\[
\Pi_i = \frac{(1 - S_{ij})k_i}{\sum_j (1 - S_{ij})k_j}.
\]  

(d) after \( t \) time steps this procedure results in a network with \( N = t + m_0 \) sites and \( mt \) links.

From this perspective, the structure of the built network presents some interesting properties, namely:

(a) it is scale-free, i.e., the degree distribution decays as the power law \( P(k) \sim k^{-\theta} \), with \( \theta = -2.84(1) \);

(b) it is small-world, i.e., the average shortest path length, \( \langle d \rangle \), between two sites increases logarithmically with the network size \( N \);

(c) the clustering coefficient obeys \( \langle C \rangle \sim N^{-0.746(2)} \), meaning that the likelihood of finding two nearest neighbors of a given site connected themselves, decreases as the network size \( N \) grows.

The procedure to build the clustered homophilic network CHN is similar to the HN one, except that, after an initial preferential attachment (PA) step we insert a triad formation step: the new connection is linked to one of the neighbors of the site attached to it in the previous PA step (see Fig. 1b). We will follow the steps employed in [9], here briefly described: a triad formation step is inserted after a preferential attachment one, where every newly added node \( j \) in the network links not only to node \( i \), but also to a randomly chosen neighbor of \( i \) (node \( s \) according to Fig. 1b), leading to the formation of lots of triads in the network, increasing the overall clustering coefficient. A parameter \( m \) is used to decide the number of connections a newly added node will have in the network.

3. The SIS model and its numerical implementation

In this section we describe the SIS model on the clustered homophilic network. We build networks with size \( N = 320, 640, 1280, 2560, 5120, 10240 \) and \( 20480 \), using 2000 steps for the relaxation time. The running time used to let the system reaches the steady state depends on the network size in such a way that one combines the smallest \( N \) with the largest running times. For example, for \( N = 320 \) we use 12 000 runs, decreasing by 1000 for each \( N \) up to reach 6000 runs for the largest network size. The simulations were performed according to the following steps:

(a) it is assumed that every single site of the network represents an individual who can only exist in one out of two possible discrete states, namely, healthy (inactive) or infected (active). Within this assumption, it is unequivocally associated with each site a variable \( \sigma_i(t) \), meaning the state of the site at a time \( t \). Therefore, \( \sigma_i(t) \) takes the value 1(0) if the individual is active (inactive).

(b) we initialize all sites of the clustered homophilic network as infected.
(c) at each elementary time step of the dynamics, one site is picked at random in the network and its state is verified. In the case of $\sigma_i = 0$, this site will be infected with probability $\lambda$ if it is connected to one or more infected neighbors. Otherwise, in the case of $\sigma_i = 1$, the site will be healed and become again healthy with probability 1. Time is increased after a Monte Carlo loop composed by $N$ elementary steps.

(d) at any time $t$, we compute the density of active particles as

$$\rho(t) = \frac{1}{N} \sum_i \sigma_i(t).$$

In the absence of any infected site, the system is trapped in the absorbing state. To leave this state and to allow the model’s dynamics to continue, we replace a randomly chosen inactive individual by an active one whenever the system visits the vacuum state. Such reflective boundary condition has been already employed in the literature and does not affect the critical behavior (see [24,25]). Then, steps (c) and (d) are repeated as long as necessary until the system reaches a statistically stationary regime (steady-state).

4. Results and discussions

Considering the effect of clustering in the homophilic network, we now investigate the possible occurrence of an absorbing state phase transition in the SIS model. Fig. 2 displays the order parameter $\rho$ as a function of the infection rate $\lambda$ for different CHN sizes. These data suggest that there is indeed a transition at a finite infection rate. For small values of $\lambda$, we have $\rho \to 0$, meaning that the disease-free scenario takes control and the fraction of infected individuals dies out dramatically. On the other hand, for large values of $\lambda$, the system reaches a stationary density with a finite fraction of infected individuals.

Firstly, we determine the critical point $\lambda_c$ in terms of the order parameter fluctuation $\chi$ defined as [26]:

$$\chi = N(\langle \rho^2 \rangle - \langle \rho \rangle^2).$$

By plotting $\chi$ as a function of $\lambda$ in Fig. 3, one can notice that it exhibits a maximum (peak) at a value $\lambda^*$, which is interpreted as a pseudo-critical point in a network of fixed size $N$. In the limit of infinite network size, the maximum diverges while $\lambda^*$ tends to the critical point $\lambda_c$.

For purposes of analyzing the behavior of the critical point as the network size $N$ grows, we show in Fig. 4 the value of the effective critical point $\lambda^*$ estimated from the peaks of the order parameter fluctuations $\chi$ against the inverse of $N$. From the position of these peaks, we can obtain the epidemic outbreak threshold $\lambda_c$ in the asymptotic limit $1/N \to 0$.

Performing a nonlinear regression of the numerical data by means of the scaling relation $\lambda = a + b \cdot N^{-c}$, where $a$ stands for the critical threshold value $\lambda_c$, and $c$ for the critical exponent $1/\nu$, we can obtain from the best fit of the data (the full red curve in Fig. 4) the following estimated values for these three parameters, namely: $\lambda_c = 0.068$, $b = 0.619$ and $1/\nu = 0.254$. 

---

**Fig. 2.** The density of infected individuals $\rho$ as a function of the infection rate $\lambda$ for different CHN sizes. The curves have a sigmoid shape with a marked inflection point featuring the criticality of the phase transition.
Fig. 3. The order parameter fluctuations $\chi$ as a function of the infection rate $\lambda$ for different CHN sizes. The $\chi$-curves diverge at the critical threshold in the infinite network size limit.

Fig. 4. The value of the effective critical point $\lambda_c$ estimated from the peak of the order parameter fluctuations $\chi$ as a function of the inverse network size $1/N$. In the thermodynamic limit, $1/N \rightarrow 0$, and then we obtain, by applying a nonlinear regression for the numerical data, $\lambda_c = 0.068(9)$ and $1/\nu = 0.254$.

At criticality, the position $\lambda^*$ for which the height of the order parameter fluctuations has a maximum, is expected to depend on the network size $N$ through the scaling form:

$$\lambda^* - \lambda_c \sim \left( \frac{1}{N} \right)^{1/\nu}. \quad (4)$$

This relation provides another way to estimate the critical exponent ratio $1/\nu$. From this, and using a linear regression in logarithmic scale (see Fig. 5), we obtain the estimate $1/\nu = 0.25(4)$, which is in perfect agreement with the value previously evaluated by the nonlinear fitting depicted in Fig. 4.

Now, we consider the relation between the density of active sites $\rho$ and the network size $N$ at the critical point. It shall scale according to:

$$\rho(\lambda_c, N) \sim N^{-\beta/\nu}. \quad (5)$$

where $\beta$ is associated with the decay of the order parameter $\rho$ at a certain distance from the critical point, while $\nu$ is related to the growth of correlations in the vicinity of the critical point. In Fig. 6 we plot, in logarithmic scale, the order
Fig. 5. Plot of the peak position of the order parameter fluctuations $\langle \lambda^* - \lambda_c \rangle$ as a function of the inverse network size on the CHN. The full black line indicates the best linear regression, and from its slope we obtain the exponent $1/\nu$ according to Eq. (4).

Fig. 6. The scaling behavior of the order parameter $\rho(\lambda_c, N)$ as plotted in logarithmic scale against the network size $N$ in the vicinity of the critical point. The slope is an estimate of the exponents ratio $\beta/\nu$.

parameter $\rho(\lambda_c, N)$ against the network size $N$ to obtain the critical exponent ratio $\beta/\nu$. From the linear fitting procedure, the estimated value was found to be $\beta/\nu = 0.88(4)$.

The last critical exponent ratio measured in this work is given by the height of the peak of the order parameter fluctuations which is expected, at criticality, to scale as a power-law of the network size as:

$$\chi_{\text{peak}}(N) \sim N^{\gamma/\nu},$$

where $\gamma$ is the characteristic critical exponent associated with the divergence of the order parameter fluctuations. In Fig. 7 we present the values of $\chi_{\text{peak}}(N)$, computed at the susceptibility maximum, as a function of $N$. Applying to these data such a power-law fitting, we find $\gamma/\nu = 0.51(2)$ as our best estimate.

Once having obtained the critical point $\lambda_c$ and the critical ratio of $1/\nu$, $\beta/\nu$ and $\gamma/\nu$, we check the accuracy of these estimated values by performing a data collapse analysis. The collapsing hypothesis implies that if the order parameter and its fluctuations data, obtained from different network sizes, are rescaled properly to be plotted against $(\lambda - \lambda_c)N^{1/\nu}$, they will fall on a single curve when the correct values of the critical points and critical exponents are used, as depicted in Fig. 8. Fig. 8a displays a fine data collapse of the order parameter for distinct network sizes $N$. In this case, the best collapse occurs for $\lambda_c = 0.068(9)$, $\beta/\nu = 0.88(4)$ and $1/\nu = 0.25(4)$, confirming the accuracy of the critical properties previously estimated. On the other hand, Fig. 8b shows a very good data collapse using previous results obtained from
Fig. 7. Log–log plot showing the power-law behavior for the order parameter fluctuations peak values as a function of the network size $N$. The solid black line represents a linear fit of these data, whose slope gives the exponent ratio $\gamma/\nu$.

Fig. 8. (a) Data collapse of the order parameter $\rho N^{\beta/\nu}$ against the control parameter $(\lambda^* - \lambda_c)N^{\beta/\nu}$ for several values of the network size $N$. The best data collapse is given by the critical exponents: $\beta/\nu = 0.88$, $1/\nu = 0.25$ and $\lambda_c = 0.068$. (b) Data collapse of the order parameter fluctuations for different network sizes using the same set of critical quantities obtained from the finite-size scaling (FSS) analysis. All data fall over a single universal curve, making stronger the accuracy of the estimated critical properties. The statistical error bars are smaller than the symbols sizes.

the maximum point and peak position of the order parameter fluctuations. To get this single universal curve we have used $\lambda_c = 0.068$, $\gamma/\nu = 0.51$ and $1/\nu = 0.25$, which are the values formerly estimated.

Our numerical simulations can be compared with those reported in the scientific literature, emphasizing the SIS model in complex networks. It is well known that its critical behavior depends on the topological structure [12,27–31]. In Table 1 we compare our results in the CHN network with $\theta = 2.84$ with the HMF theory and the non-correlated configuration model (UCM) for $\theta = 2.25$ and 2.75 [27] networks and in the Apollonian network with $\theta = 2.585$ [20]. Our results were obtained through extensive simulation and appropriate linear adjustments and data collapse, for values of $N = 320, 640, 1280, 2560, 5120, 10240$ and 20480. We obtained the critical exponents $\beta/\nu = 0.88(4)$, $nu = 4.0(8)$ and $\gamma/\nu = 0.51(2)$. These are distinct from those predicted by the HMF theory for $\theta = 2.84$ which are $\beta/\nu = 0.54$, $nu = 2.19$ and $\gamma/\nu = -0.09$ as well as the others found in Table 1. Therefore, the CHN seems to belong to a new universality class. Exact as well as mean-field theory predicts $\lambda_c = 0$ for uncorrelated quenched random networks with power-law distributed connectivities. The triad formation process introduces correlations in the network topology. The reported finite value of the critical infection rate indicates that these underlying correlations play a relevant role in the SIS model and extensions of the standard theoretical approaches would be in order to correctly capture their influence.
Table 1

| Model                  | $\beta/\nu$         | $\nu$            | $\gamma/\nu$       |
|------------------------|---------------------|------------------|---------------------|
| HMF, $\theta = 2.25$  | $1/(\theta - 1)$   | $\theta - 1)/(\theta - 2)$| $(\theta - 3)/(\theta - 2)$ |
| UCN, $\theta = 2.75$ | 0.58(1)             | 2.4(1)           | 0.16(2)             |
| AN, $\theta = 2.585$ | 0.56(7)             | 2.1(4)           | 0.39(4)             |
| CHN, $\theta = 2.84$ | 0.88(4)             | 4.0(4)           | 0.51(2)             |

5. Summary and conclusions

In this paper we have investigated the critical behavior of the SIS epidemic model on the clustered homophilic network CHN that mimics the underlying habitat where spreading disease takes place. By applying finite-size scaling analysis to the data obtained from extensive numerical simulations, we showed that the SIS dynamics model on the CHN presents a clear absorbing state phase transition at a critical infection rate $\lambda_c = 0.068(9)$. We computed some relevant critical exponents governing the singular behavior associated with the critical point. From the scaling relations, we obtained that $\beta/\nu = 0.88(4)$, $1/\nu = 0.25(4)$ and $\gamma/\nu = 0.51(2)$. It is worth stressing out that these values are quite different from those reported for the contact process in an unclustered homophilic network [7], as well as from those predicted by quenched mean-field (QMF) theory considering scale-free networks characterized by $P(k) \sim k^{-\theta}$, with the same degree exponent $\theta$ [26].

No doubt, these findings indicate that clustering is a relevant mechanism affecting the universal behavior of nonequilibrium phase-transitions taking place in homophilic complex networks. Therefore, it should be carefully taken into account when modeling real epidemic processes.

CRediT authorship contribution statement

**F.L. Santos:** Formal analysis, Applied methodology, Analysis and investigation of the available data. **M.L. Almeida:** Formal analysis, Applied methodology, Analysis and investigation of the available data. **E.L. Albuquerque:** Conceptualization, Formal analysis, Writing the paper for publication and revised the manuscript. **A. Macedo-Filho:** Formal analysis, Applied methodology, Analysis and investigation of the available data. **M.L. Lyra:** Conceptualization, Formal analysis, Writing the paper for publication and revised the manuscript. **U.L. Fulco:** Conceptualization, Formal analysis, Writing the paper for publication and revised the manuscript.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgments

The authors would like to thank D.C. Jotha for his computational assistance. This work was partially financed by the Brazilian Research Agencies CAPES and CNPq, and by the Alagoas State Research Agency FAPEAL. We would like to thank the Núcleo de Processamento de Alto Desempenho of the Universidade Federal do Rio Grande do Norte - NPAD/UFRN to allow us to access their computer facilities. Each author had participated sufficiently in the work to take public responsibility for appropriate portions of the content. All authors read and approved the final manuscript.

References

[1] E. Alfinito, M. Beccaria, A. Fachechi, G. Macorini, Europhys. Lett. 117 (2017) 18002.
[2] A.G. Dickman, R. Dickman, Amer. J. Phys. 83 (2015) 468.
[3] A.A. Alemi, M. Bierbaum, C.R. Myers, J.P. Sethna, Phys. Rev. E 92 (2015) 052801.
[4] R. Pastor-Satorras, C. Castellano, P. Van Mieghem, A. Vespignani, Rev. Modern Phys. 87 (2015) 925.
[5] R. Pastor-Satorras, A. Vespignani, Phys. Rev. E 65 (2002) 036104.
[6] M.L. Almeida, G.A. Mendes, G.M. Viswanathan, L.R. da Silva, Eur. Phys. J. B 86 (2013) 38.
[7] A.M. dos Santos, M.L. Almeida, G.A. Mendes, L.R. da Silva, Internat. J. Modern Phys. C 26 (2015) 1550097.
[8] A.L. Barabási, R. Albert, Science 286 (1999) 509.
[9] P. Holme, B.J. Kim, Phys. Rev. E 65 (2002) 026107.
[10] J. Marro, R. Dickman, Nonequilibrium Phase Transitions in Lattice Models, Cambridge University Press, Cambridge, 1999.
[11] M.L. Almeida, A. Macedo-Filho, G.A. Mendes, L.R. da Silva, E.L. Albuquerque, U.L. Fulco, J. Stat. Mech. (2016) 043202.
[12] R. Pastor-Satorras, A. Vespignani, Phys. Rev. Lett. 86 (2001) 3200.
[13] R. Pastor-Satorras, A. Vespignani, Phys. Rev. E 63 (2001) 066117.
[14] W. Wang, Ze-Xun Wang, Shi-Min Cai, Phys. Rev. E 98 (2018) 052312.
[15] W. Wang, M. Tang, H.E. Stanley, L.A. Braunstein, Rep. Progr. Phys. 80 (2017) 036603.
[16] R. Albert, A.L. Barabási, Rev. Modern Phys. 74 (2002) 47.
[17] J.S. Shiner, M. Davison, Chaos Solitons Fractals 21 (2004) 1.
[18] G.A. Mendes, L.R. da Silva, H.J. Herrmann, Physica A 391 (2012) 362.
[19] L.F. da Silva, R.N. Costa Filho, D.J.B. Soares, A. Macedo-Filho, U.L. Fulco, E.L. Albuquerque, Physica A 392 (2013) 1532.
[20] L.F. da Silva, R.N. Costa Filho, A.R. Cunha, A. Macedo-Filho, M. Serva, U.L. Fulco, E.L. Albuquerque, J. Stat. Mech. (2013) P05003.
[21] C. Castellano, R. Pastor-Satorras, Phys. Rev. Lett. 105 (2010) 218701.
[22] L.A.N. Amaral, A. Scala, M. Barthelemy, H.E. Stanley, Proc. Natl. Acad. Sci. USA 97 (2000) 149.
[23] R. Pastor-Satorras, A. Vespignani, Evolution and Structure of the Internet: A Statistical Physics Approach, Cambridge University Press, Cambridge, 2004.
[24] U.L. Fulco, D.N. Messias, M.L. Lyra, Phys. Rev. E 63 (2001) 066118.
[25] A.M. Filho, G. Corso, M.L. Lyra, U.L. Fulco, J. Stat. Mech. (2010) P04027.
[26] H. Hong, M. Ha, H. Park, Phys. Rev. Lett. 98 (2007) 258701.
[27] H. Hong, M. Ha, H. Park, Phys. Rev. Lett. 98 (2007) 258701.
[28] S.C. Ferreira, R.S. Ferreira, C. Castellano, R. Pastor-Satorras, Phys. Rev. E 84 (2011) 066102.
[29] C. Castellano, R. Pastor-Satorras, Phys. Rev. Lett. 96 (2006) 038701.
[30] M. Karsai, R. Juház, F. Iglói, Phys. Rev. E 73 (2006) 036116.
[31] F. Caccioli, L. DallAsta, J. Stat.Mech. (2009) P10004.