Fractal scale-free networks resistant to
disease spread

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Abstract. The conventional wisdom is that scale-free networks are prone to
epidemic propagation; in the paper we demonstrate that, on the contrary, disease
spreading is inhibited in fractal scale-free networks. We first propose a novel
network model and show that it simultaneously has the following rich topological
properties: scale-free degree distribution, tunable clustering coefficient, ‘large-
world’ behavior, and fractal scaling. Existing network models do not display
these characteristics. Then, we investigate the susceptible–infected–removed
(SIR) model of the propagation of diseases in our fractal scale-free networks by
mapping it to the bond percolation process. We establish the existence of non-
zero tunable epidemic thresholds by making use of the renormalization group
technique, which implies that power law degree distribution does not suffice to
characterize the epidemic dynamics on top of scale-free networks. We argue that
the epidemic dynamics are determined by the topological properties, especially
the fractality and its accompanying ‘large-world’ behavior.

Keywords: exact results, growth processes, network dynamics, random graphs,
networks

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1. Introduction

In recent years, there has been much interest in the study of the structure and dynamics of complex networks [1]–[4]. One aspect that has received considerable attention is the epidemic spreading taking place on top of networks [5], which is relevant to computer virus diffusion, information and rumor spreading, and so on. In the study of epidemic spreading, the notion of thresholds is a crucial problem since it finds an intermediate practical application in disease eradication and vaccination programs [6, 7]. In homogeneous networks, there is a non-zero infection threshold: if the spreading rate is above the threshold, the infection spreads and becomes endemic; otherwise the infection dies out quickly. However, recent studies demonstrate that the threshold is absent in heterogeneous scale-free networks [8]–[11]. Thus, it is important to identify what characteristics of network structure determine the presence or absence of epidemic thresholds.

Already, the influences of most structural properties on disease dynamics have been studied; these include the degree distribution [9]–[11], the clustering coefficient [12], and degree correlations [13]. However, these features do not suffice to characterize the architecture of a network [14]. Very recently, by introducing and applying a box-covering (renormalization) technique, Song et al found the presence of fractal scaling in a variety of real networks [15, 16]. Examples of fractal networks include the WWW, actor collaboration networks, metabolic networks, and yeast protein interaction networks. The fractal topology is often characterized through two quantities: fractal dimension $d_B$ and degree exponent of the boxes $d_k$, both of which can be calculated using the box-counting algorithm [17, 18]. The scaling of the minimum possible number of boxes $N_B$ of linear size $\ell_B$ required to cover the network defines the fractal dimension $d_B$, namely $N_B \sim \ell_B^{-d_B}$. Similarly, the degree exponent of the boxes $d_k$ can be found via $k_B(\ell_B) / k_{\text{hub}} \sim \ell_B^{-d_k}$, where $k_B(\ell_B)$ is the degree of a box in the renormalized network, and $k_{\text{hub}}$ the degree of the most-connected node inside the corresponding box. Interestingly, for fractal scale-free networks...
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Fractal scale-free networks have degree distribution $P(k) \sim k^{-\gamma}$, the two exponents, $d_B$ and $d_k$, are related to each other through the following universal relation: $\gamma = 1 + d_B/d_k$ [15].

Fractality is now acknowledged as a fundamental property of a complex network [14]. It relates to a lot of aspects of network structure and dynamics running on the network. For example, in fractal networks the correlation between degree and betweenness centrality of nodes is much weaker than that in non-fractal networks [19]. In addition, several studies uncovered that fractal networks are not assortative [16,20,21]. The peculiar structural nature of fractal networks makes them exhibit distinct dynamics. It is known that fractal scale-free networks are more robust than non-fractal ones against malicious attacks on hub nodes [16,21]. On the other hand, fractal networks and their non-fractal counterparts also display disparate phenomena of other dynamics, such as cooperation [22,23], synchronization [21], transport [24], and first-passage time [25,26]. Despite the ubiquity of the fractal feature and its important impacts on dynamical processes, the dynamics of disease outbreaks in fractal networks has been far less investigated.

In this current paper, we focus on the effects of fractality on the dynamics of disease in fractal scale-free networks. Firstly, we propose an algorithm for creating a class of fractal scale-free graphs by introducing a control parameter $q$. Secondly, we give in detail a scrutiny of the network architecture. The analysis results show that this class of networks have unique topologies. They are simultaneously scale-free, fractal, ‘large-world’, and have tunable clustering coefficient. Thirdly, we study a paradigmatic epidemiological model [6,7], namely the susceptible–infected–removed (SIR) model on the proposed fractal graphs. By mapping the SIR model to a bond percolation problem and using the renormalization group theory, we find the existence of non-zero epidemic thresholds as a function of $q$. We also provide an explanation for our findings.

2. Network construction and topological properties

This section is devoted to the construction and the relevant structural properties of the networks under consideration, such as degree distribution, clustering coefficient, average path length (APL), and fractality.

2.1. Construction algorithm

The proposed fractal networks have two categories of bonds (links or edges): iterative bonds and non-iterated bonds which are depicted as black and red lines, respectively. The networks are constructed in an iterative way as shown in figure 1. Let $F_t$ ($t \geq 0$) denote the networks after $t$ iterations. Then the networks are built in the following way: for $t = 0$, $F_0$ is two nodes (vertices) connected by an iterative edge. For $t \geq 1$, $F_t$ is obtained from $F_{t-1}$. We replace each existing iterative edge in $F_{t-1}$ either by a connected cluster of links on the top right of figure 1 with probability $q$, or by the connected cluster on the bottom right with complementary probability $1 - q$. The growing process is repeated $t$ times, with the fractal graphs obtained in the limit $t \rightarrow \infty$. Figure 2 shows a network after three-generation growth for a specific case of $q = 0.6$.

Next we compute the numbers of total nodes and edges in $F_t$. Let $L_v(t)$, $L_i(t)$ and $L_n(t)$ be the numbers of nodes, iterative edges, and non-iterated edges created at step $t$, respectively. Note that each of the existing iterative edges yields two nodes and four
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Figure 1. Iterative construction method for the fractal networks. Each iterative link is replaced by a connected cluster on the right-hand side of the arrow. The red link is a non-iterated link.

Figure 2. Sketch of a network after three iterations for the particular case of $q = 0.6$.

new iterative edges; at the same time this original iterative edge itself is deleted, which means that $L_i(t)$ is also the total number of iterative edges at time $t$. Then we have $L_i(t) = 2L_i(t - 1)$ and $L_v(t) = 4L_i(t - 1)$ for all $t > 0$. Considering the initial condition $L_i(0) = 1$, one can obtain $L_i(t) = 4^t$ and $L_v(t) = 2 \times 4^{t-1}$. Thus the number of total nodes $N_t$ present at step $t$ is

$$N_t = \sum_{t_i=0}^{t} L_v(t_i) = \frac{2 \times 4^t + 4}{3}. \tag{1}$$

On the other hand, at each construction step, each of the existing iterative edges may yield one non-iterated link with probability $1 - q$, so the expected value of $L_n(t)$ is

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$(1-q) L_i(t-1)$ for $t \geq 1$, i.e., $L_n(t) = (1-q)^t$. Therefore, the total number of edges $E_t$ present at step $t$ is

$$E_t = L_i(t) + \sum_{i=1}^{t} L_n(t_i) = \frac{(4-q) 4^t - (1-q)}{3}. \quad (2)$$

The average node degree after $t$ iterations is $\langle k \rangle_t = 2 E_t / N_t$, which approaches $4 - q$ in the infinite $t$ limit.

2.2. Degree distribution

When a new node $u$ enters the system at step $t_u$ ($t_u \geq 1$), it has two iterative edges. At the same time, with probability $1-q$ one non-iterated edge is created and linked to node $u$. Let $L_i(u, t)$ be the number of iterative links emanating from node $u$ at step $t$, then $L_i(u, t_u) = 2$. Notice that at any subsequent step each iterative edge of $u$ is broken and generates two new iterative edges linked to $u$. Thus $L_i(u, t) = 2 L_i(u, t-1) = 2^{t-t_u+1}$.

We define $k_u(t)$ as the degree of node $u$ at time $t$; then we have

$$k_u(t) = \begin{cases} 2^{t-t_u+1}, & \text{with probability } q, \\ 2^{t-t_u+1} + 1, & \text{with probability } 1-q, \end{cases} \quad (3)$$

where the last term 1 in the second formula represents the non-iterated link connected to node $u$. For the initial two nodes created at step 0, neither of them has a non-iterated link; both nodes have a degree of $2^t$.

Equation (3) indicates that the degree spectrum of the networks is not continuous. It follows that the cumulative degree distribution \cite{27, 28} is given by $P_{\text{cum}}(k) = N_{t,k} / N_t$, where $N_{t,k}$ is the number of nodes whose degree is not less than $k$. When $t$ is large enough, we find $P_{\text{cum}}(k) \approx k^{-2}$. So the degree distribution follows a power law form with the exponent $\gamma = 3$, which is independent of $q$. The same degree exponent has been obtained in the famous Barabási–Albert (BA) model \cite{8}.

2.3. Clustering coefficient

The clustering coefficient \cite{29} of a node $u$ with degree $k_u$ is the probability that a pair of neighbors of $u$ are themselves connected, which is given by $C(k_u) = 2b_u/[k_u(k_u-1)]$, where $b_u$ is the number of existing connections between the $k_u$ neighbors of $u$. For our networks, the clustering coefficient $C(k)$ for a single node with degree $k$ can be evaluated exactly. Note that except for those nodes born at step $t$, all existing links among the neighbors of a given node are non-iterated ones, whose number is easy to calculate. For the initial two nodes, the expected existing non-iterated links among the neighbors number $(1-q)2^{t-1}$. For each of those nodes created at step $\phi$ ($0 < \phi < t$), there are on average $(1-q)2^{t-\phi}$ non-iterated links among its neighbors. Finally, for the nodes generated at step $t$, some of them have a degree of 3, and the number of links between the neighbors of each is 2; the others have a degree of 2, and their clustering coefficient is zero. Thus, there is a one-to-one correspondence between the clustering coefficient $C(k)$ of the node and its

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Figure 3. The average clustering coefficient $C$ of the whole network as a function of $q$. It can be tuned systematically by changing $q$. The squares are the simulation results, and the line represents the analytical expression given by equation (5).

degree $k$:

$$C(k) = \begin{cases} 
(1 - q)/(k - 1) & \text{for } k = 2^m (2 \leq m \leq t), \\
(1 - q)/k & \text{for } k = 2^m + 1 (2 \leq m \leq t), \\
0 & \text{for } k = 2, \\
2/k & \text{for } k = 3.
\end{cases}$$

(4)

Using equation (4), we can obtain the clustering $\bar{C}_t$ of whole the network at step $t$, which is defined as the average clustering coefficient of all individual nodes. Then we have

$$\bar{C}_t = \frac{1 - q}{N_t} \left[ \frac{L_v(0)}{2^t - 1} + \sum_{r=1}^{t-1} \frac{qL_v(r)}{K_r - 1} + \sum_{r=1}^{t-1} \frac{(1 - q)L_v(r)}{K_r + 1} + \frac{2L_v(t)}{K_t + 1} \right],$$

(5)

where $K_r = 2^{t-r+1}$ and $K_t = 2$. In the infinite network order limit ($N_t \to \infty$), $\bar{C}_t$ converges to a non-zero value $C$ as a function of parameter $q$. In the two extreme cases of $q = 0$ and $q = 1$, $C$ takes the values 0 and 0.5435 [30], respectively. When $q$ increases from 0 to 1, $C$ grows from 0 to 0.5435; see figure 3. Thus, the parameter $q$ in our model introduces the clustering effect by allowing the formation of triangles. Furthermore, the relation between $C$ and $q$ is almost linear, as depicted in figure 3.

2.4. Average path length

Shortest paths play an important role both in the transport and communication within a network and in the characterization of the internal structure of the network [31]–[34]. Let $d_{ij}$ represent the shortest path length from node $i$ to $j$; then the average path length (APL) $d_t$ of $F_t$ is defined as the mean of $d_{ij}$ over all couples of nodes in the network, and the maximum value $D_t$ of $d_{ij}$ is called the diameter of the network.

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For general $q$, it is difficult to derive a closed formula for the APL $d_t$ of network $F_t$. But for two limiting cases of $q = 0$ and $q = 1$, both the networks are deterministic ones. In the large $t$ limit, $d_t^0 \approx \frac{8}{21} 2^t$ [30] and $d_t^1 \approx \frac{11}{21} 2^t$ [23]. On the other hand, for the large $t$ limit, $N_t \sim 4^t$, so both $d_t^0$ and $d_t^1$ grow as a square power of the number of network nodes.

By construction, it is obvious that $d_t^0$ and $d_t^1$ are the lower and upper bounds of APL for network $F_t$, respectively. As a matter of fact, if we denote the specific $q = 1$ case of the network as $F_t^1$ (it has no non-iterated edges, and thus the maximum APL), then one can obtain $F_t$ from $F_t^1$ by adding non-iterated edges in a certain way. The lesser the parameter $q$, the greater the number of added non-iterated edges. In the case of $q = 0$, the network is the densest one with the minimum APL. Therefore, the APL is a decreasing function of $q$. As $q$ drops from 1 to 0, $d_t$ increases from $\frac{8}{21} 2^t$ at $q = 1$ to $\frac{11}{21} 2^t$ at $q = 0$. From above arguments, we can conclude that for the full range of $q$ between 0 and 1, the APL $d_t$ has an exponential growth with network size, which indicates that the networks considered are not small worlds.

2.5. Fractal dimension

To determine the fractal dimension, we follow the mathematical framework introduced in [16]. We are concerned with three quantities, network size $N_t$, network diameter $D_t$, and degree $k_u(t)$ of a given node $u$. By construction, we can easily see that in the infinite $t$ limit, these quantities grow obeying the following relation: $N_t \sim 4 N_{t-1}$, $D_t = 2 D_{t-1}$, $k_u(t) \sim 2 k_u(t-1)$. Thus, for large networks, $N_t$, $k_u(t)$ and $D_t$ increase by factors of $f_N = 4$, $f_k = 2$ and $f_D = 2$, respectively.

From above obtained microscopic parameters demonstrating the mechanism for network growth, we can derive the scaling exponents: the fractal dimension $d_B = \ln f_N / \ln f_D = 2$ and the degree exponent of boxes $d_k = \ln f_k / \ln f_D = 1$. According to the scaling relation of fractal scale-free networks, the exponent of the degree distribution satisfies $\gamma = 1 + (d_B / d_k) = 3$, giving the same $\gamma$ as was obtained in the direct calculation of the degree distribution.

The fractality behavior can also be easy to see by tiling the system using a renormalization procedure as follows. We can ‘zoom out’ (i.e. renormalize) from the network by replacing each connected cluster of bonds on the right of figure 1 by a single ‘super’-link, in a way that reverses the process of network growth; see figure 1. This has the effect of rescaling the diameter; it reduces the diameter by a factor of 2 by carrying a cluster of bonds with diameter 2 into a single ‘super’-link with unit length. At the same time, the number of nodes in the rescaled network decreases by a factor of 4. Thus, the fractal dimension is $d_B = \ln 4 / \ln 2 = 2$.

3. The SIR model on the networks

As demonstrated in the preceding section, the networks exhibit simultaneously many interesting properties: the scale-free phenomenon, tunable clustering, ‘large-world’ behavior, and fractality. To the best of our knowledge, these features have not been reported for previous network models. Hence, it is of scientific interest and great significance to investigate dynamic processes taking place upon the model to inspect the

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effect of network topologies on the dynamics. Next we will focus on studying the SIR model of epidemics, which is one of the most important disease models and has attracted much attention within the physics community; see [5] and references therein.

The standard SIR model [6,7] assumes that each individual can be in any of three exclusive states: susceptible (S), infected (I), or removed (R). The dynamics of disease transmission on a network can be described as follows. Each node of the network stands for an individual and each link represents the connection along which the individuals interact and the epidemic can be transmitted. At each time step, an infected node transmits infection to each of its neighbors independently with probability \( \lambda \); at the same time the infected node itself becomes removed with a constant probability 1, whereupon it cannot catch the infection again, and thus will not infect its neighbors any longer.

The above-described SIR model for disease dynamics is equivalent to a bond percolation process with bond occupation probability equal to the infection rate \( \lambda \) [35,36]. The connected clusters of nodes in the bond percolation process then correspond to the groups of individuals that would be infected by a disease outbreak starting with any individual within the cluster that it belongs to. It was shown that in scale-free networks such as the Barabási–Albert (BA) network, for any vanishingly small \( \lambda \), there exists an extensive spanning cluster or ‘giant component’, implying that scale-free networks demonstrate an absence of epidemic thresholds. However, we will show that for our networks, epidemic thresholds are present, the values of which depend on the parameter \( q \).

According to the mapping between the SIR dynamics and the bond percolation problem, the epidemic threshold corresponds precisely to the percolation threshold. In our case, the recursive construction of the networks makes it possible to study the percolation problem by using the real-space renormalization group technique [37–39] to obtain an exact solution for the percolation (epidemic) thresholds. Here we are interested in only the percolation phase transition point, excluding the size of the giant cluster. Let us describe the procedure in application to the network considered. Supposing that the network growth stops at a time step \( t \to \infty \), then we spoil the network in the following way: for an arbitrary link present in the undamaged network, it is retained in the damaged network with probability \( \lambda \). Then we invert the transformation shown in figure 1 and define \( n = t - \tau \) for this inverted transformation, which is in fact a decimation procedure [39]. Further, we introduce the probability \( \lambda_n \) that if two nodes are connected in the undamaged network at \( \tau = t - n \), then at the \( n \)th step of the decimation for the damaged network, there exists a path between these vertices. Here, \( \lambda_0 = \lambda \). According to the network construction and the analysis in [39], it is easy to obtain the following recursive relation for \( \lambda_n \):

\[
\lambda_{n+1} = q \left( \lambda_n^2 + \lambda_n^2 - \lambda_n^2 \times \lambda_n^2 \right) + (1 - q)\left[ \lambda_n^2 + 5\lambda_n^4(1 - \lambda_n)^2 + 8\lambda_n^3(1 - \lambda_n)^2 + 2\lambda_n^2(1 - \lambda_n)^3 \right].
\]

(6)

Equation (6) has five roots (i.e., fixed points), among which two are invalid: one is greater than 1, and the other is less than 0. The other three fixed points are as follows: two stable fixed points at \( \lambda = 0 \) and 1, and an unstable fixed point at \( \lambda_c \) that is the percolation threshold. We omit the expression for \( \lambda_c \) as a function of \( q \), because it is very lengthy. We show the dependence of \( \lambda_c \) on \( q \) in figure 4, which indicates that the threshold \( \lambda_c \) increases almost linearly as \( q \) increases. When \( q \) grows from 0 to 1, \( \lambda_c \) increases from 0.5 to \( (\sqrt{5} - 1)/2 \approx 0.618 \).

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Therefore, there exists a percolation threshold $\lambda_c$ such that for $\lambda > \lambda_c$ a giant component appears; for $\lambda < \lambda_c$ there are only small clusters. This means that for the SIR model the epidemic prevalence undergoes a phase transition at a non-zero epidemic threshold $\lambda_c$. If the infection rate $\lambda > \lambda_c$, the disease spreads and becomes persistent in time; otherwise, the infection dies out gradually. The existence of epidemic thresholds in our networks is in sharp contrast with the null threshold found in a wide range of stochastic scale-free networks of the Barabási–Albert (BA) type [9]–[11].

Why are the present scale-free networks not prone to disease propagation like previously studied uncorrelated BA type scale-free networks? We argue that the presence of finite epidemic thresholds in our networks rests on their two-dimensional fractal structure with diameter increasing as a square power of the network order, a property analogous to that of a two-dimensional regular lattice [40]. The ‘large-world’ feature resulting from the fractality stops the diffusion of diseases, and makes the behaviors of disease spreading in our networks similar to those of regular lattices. Thus, the fractal topology provides protection against disease spreading.

4. Conclusions

In the present work, we have introduced a new model for fractal networks and provided a detailed analysis of the structural properties, which are related to the model parameter $q$. The model exhibits a rich topological behavior. The degree distribution is a power law with the degree exponent asymptotically approaching 3 for large network order. The clustering coefficient is changeable, and can be systematically tuned over a large range by altering $q$. In particular, the networks are topologically fractal with a fractal dimension of 2 for all $q$. Along with the fractality, the networks display a ‘large-world’ phenomenon; their average path length increases approximatively as a square power of the number of nodes.
We have also investigated the effects of the particular topological characteristics on the SIR model for disease spreading dynamics. Strikingly, we found that epidemic thresholds are recovered for all networks regardless of the value of $q$, which is in contrast to the conventional wisdom that being prone to disease propagation is an intrinsic feature of scale-free networks. We concluded that the dominant factor suppressing epidemic spreading is the fractal structure accompanied by a 'large-world' behavior. The peculiar structural properties and epidemic dynamics make our networks unique within the category of scale-free networks. Our study is helpful for designing real-life networked systems robust against epidemic outbreaks, and for better understanding of the influences of structure on the propagation dynamics.

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References

[1] Albert R and Barabási A-L, 2002 Rev. Mod. Phys. 74 47
[2] Dorogovtsev S N and Mendes J F F, 2002 Adv. Phys. 51 1079
[3] Newman M E J, 2003 SIAM Rev. 45 167
[4] Boccaletti S, Latora V, Moreno Y, Chavez M and Hwang D-U, 2006 Phys. Rep. 424 175
[5] Barthélémy M, Barrat A, Pastor-Satorras R and Vespignani A, 2005 J. Theor. Biol. 235 275
[6] Bailey N T. 1975 The Mathematical Theory of Infectious Diseases 2nd edn (New York: Macmillan)
[7] Hethcote H W, 2000 SIAM Rev. 42 599
[8] Barabási A-L and Albert R, 1999 Science 286 509
[9] Pastor-Satorras R and Vespignani A, 2001 Phys. Rev. Lett. 86 3200
[10] Pastor-Satorras R and Vespignani A, 2001 Phys. Rev. E 63 066117
[11] Moreno Y, Pastor-Satorras R and Vespignani A, 2002 Eur. Phys. J. B 26 521
[12] Serrano M and Boguñá M, 2006 Phys. Rev. Lett. 97 088701
[13] Boguñá M, Pastor-Satorras R and Vespignani A, 2003 Phys. Rev. Lett. 90 028701
[14] Costa L da F, Rodrigues F A, Travieso G and Boas P R V, 2007 Adv. Phys. 56 167
[15] Song C, Havlin S and Makse H A, 2005 Nature 433 392
[16] Song C, Havlin S and Makse H A, 2006 Nat. Phys. 2 275
[17] Song C, Gallos L K, Havlin S and Makse H A, 2007 J. Stat. Mech. P03006
[18] Kim J S, Goh K-I, Kahng B and Kim D, 2007 Chaos 17 026116
[19] Kitsak M, Havlin S, Paul G, Riccaboni M, Pammolli F and Stanley H E, 2007 Phys. Rev. E 75 056115
[20] Yook S-H, Radicchi F and Ortmanns H M, 2006 Phys. Rev. E 72 045105
[21] Zhang Z, Zhou S G and Zou T, 2007 Eur. Phys. J. B 56 259
[22] Hinczewski M, 2007 Phys. Rev. E 75 061104
[23] Hinczewski M and Berker A N, 2006 Phys. Rev. E 73 066126
[24] Gallos L K, Song C, Havlin S and Makse H A, 2007 Proc. Nat. Acad. Sci. 10 7746
[25] Condamin S, Bénichou O, Tejedor V, Voituriez R and Klafter J, 2007 Nature 450 77
[26] Rozenfeld H D, Havlin S and ben-Avraham D, 2007 New J. Phys. 9 175
[27] Dorogovtsev S N, Goltsev A V and Mendes J F F, 2002 Phys. Rev. E 65 066122
[28] Zhang Z, Zhou S G, Zou T, Chen L C and Guan J H, 2007 Eur. Phys. J. B 60 259
[29] Watts D J and Strogatz H, 1998 Nature 393 440
[30] Zhang Z, Zhou S G, Zou T and Guan J H, 2008 unpublished

doi:10.1088/1742-5468/2008/09/P00008
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[31] Fronczak A, Fronczak P and Holyst J A, 2004 Phys. Rev. E 70 056110
[32] Holyst J A, Sienkiewicz J, Fronczak A, Fronczak P and Suchecki K, 2005 Phys. Rev. E 72 026108
[33] Dorogovtsev S N, Mendes J F F and Oliveira J G, 2006 Phys. Rev. E 73 056122
[34] Zhang Z Z, Chen L C, Zhou S G, Fang L J, Guan J H and Zou T, 2008 Phys. Rev. E 77 017102
[35] Grassberger P, 1982 Math. Biosci. 63 157
[36] Newman M E J, 2002 Phys. Rev. E 66 016128
[37] Migdal A A, 1975 Zh. Eksp. Teor. Fiz. 69 1457
    Migdal A A, 1976 Sov. Phys. JETP 42 743 (translation)
[38] Kadanoff L P, 1976 Ann. Phys., NY 100 359
[39] Dorogovtsev S N, 2003 Phys. Rev. E 67 045102
[40] Newman M E J, 2000 J. Stat. Phys. 101 819

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