

Characterization and control of small-world networks

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Abstract

Recently Watts and Strogatz have given an interesting model of small-world networks. Here we concretise the concept of a “far away” connection in a network by defining a *far edge*. Our definition is algorithmic and independent of underlying topology of the network. We show that it is possible to control spread of an epidemic by using the knowledge of far edges. We also suggest a model for better advertisement using the far edges. Our findings indicate that the number of far edges can be a good intrinsic parameter to characterize small-world phenomena.

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The properties of very large networks are mainly determined by the way the connections between the vertices are made. At one extreme are the regular networks where only the “local” vertices are inter-connected and the “far away” vertices are not connected while at the other extreme are the random networks where the vertices are connected at random. The regular networks display a high degree of local *clustering* and the average distance between vertices is quite large. On the other hand, the random networks show negligible local *clustering* and the average distance between vertices is quite small. The *small-world* networks [1,2] have intermediate connectivity properties but exhibit a high degree of clustering as in regular networks and small average distance between vertices as in random networks. A very interesting model for small-world networks was recently proposed by Watts and Strogatz [3]. They found that a regular network acquires the properties of a small-world network with only a very small fraction of connections or edges (about 1%) rewired to “far away” vertices. They demonstrated that several diverse phenomena like neural networks [5], power grids and collaboration graphs of film actors [4] can be modeled using small-world networks. Also the spread of an epidemic is much faster in small-world networks than in the regular networks and almost close to that of random networks.

In this paper we suggest a possible way of characterizing small-world networks. The basic ingredients of small-world networks are the “far away” connections. We introduce a notion of *far edges* in a network to identify these “far away” connections. Our definition of a *far edge* is independent of any underlying topology for a network and depends only on the way connections or edges are made. We claim that the rapid spread of an epidemic in small-world network as found by Watts and Strogatz [3] is due to these far edges. This allows us to propose a mechanism to control the epidemic using the same far edges which are responsible for the rapid spread. We further demonstrate the utility of our notion of far edges by giving an better method of advertisement.

Consider a graph (network) with n vertices and E edges. Let \mathcal{N}_{ij}^ν denote the number of distinct paths of length ν between the vertices i and j . For a simple graph, \mathcal{N}_{ij}^1 is one if there is an edge between vertices i and j else it is zero. We now concretise the idea of “far

away” connections by defining a far edge. Let an edge e_{ij} between vertices i and j be a far edge of order μ if it is an edge for which $\mathcal{N}_{ij}^{\mu+1} = 0$ and $\mathcal{N}_{ij}^l \neq 0$ for all $l \leq \mu$.

Fig. 1 shows an example of a far edge of order one. We note that none of the edges in a completely connected graph are far edges, while all edges in a tree are far edges of order one. Hence forth we will assume that a far edge has order one unless stated otherwise.

To generate small-world networks and also other type of networks we follow the procedure given in Ref. [3]. We start with a regular network consisting of a ring of n vertices with edges connecting each vertex to its k nearest neighbours. Each edge is rewired with probability p avoiding multiple edges. The $p = 1$ case corresponds to a random network. The networks obtained with $p \approx 0.01$ correspond to small-world networks [3].

We have generated several networks from regular ($p = 0$) to random ($p = 1$) case. For each network we calculate the average path length $L(p)$ and clustering coefficient $C(p)$. The quantity $L(p)$ denotes the average length of the shortest path between two vertices, and $C(p)$ denotes the average of C_v over all the vertices v , where C_v is the number of edges connecting the neighbours of v normalized with respect to the maximum number of possible edges between these neighbours [3]. Next we determine the far edges in these networks. Let \mathcal{F} denote the ratio of number of far edges with the total number of edges. We find that initially, to a good approximation, \mathcal{F} is equal to p for $p \leq 0.1$ and then it increases slowly till it saturates to a value of about 0.2 for $p = 1$. It turns out that the number far edges of order higher than one are negligible.

In Fig. 2 we plot $C(\mathcal{F})/C(0)$ and $L(\mathcal{F})/L(0)$ as functions of \mathcal{F} . This figure is similar in nature to the plot of $C(p)/C(0)$ and $L(p)/L(0)$ as functions of p (Fig. 2 of Ref. [3]). The small-world networks can be identified as those with $C(p)/C(0) \approx 1$ and $L(p)/L(0) \approx L(1)/L(0)$. From Fig. 2 we see that this corresponds to $\mathcal{F} \approx 0.01$. Thus \mathcal{F} can be used as a parameter to characterize networks which interpolate between regular and random cases. We note that \mathcal{F} is an intrinsic quantity and does not depend on the procedure of generating networks and hence should prove to be a better parameter than p .

To further investigate the importance of far edges, we consider the problem of spread of

an epidemic. Consider an epidemic starting from a random vertex (seed). We assume that at each time step all the neighbours of infected vertices are affected with probability one, which is the most infectious case, and the vertices which are already affected die and play no further role in the spread of the epidemic. Here, neighbours of a given vertex means all the vertices which are joined to it by edges. As found by Watts and Strogatz [3], the spread of an epidemic in small-world networks is almost as fast as that in the random case. We propose that the mechanism for the rapid spread of epidemic in small-world networks is due to the traversal of the disease along the far edges. Each such traversal opens a virgin area for the spread of epidemic leading to a rapid growth.

Clearly if the far edges are responsible for the rapid growth of epidemic then we should be able to effectively control the spread by preventing the traversal of epidemic along the far edges. To test this hypothesis, we propose the following mechanism to control an epidemic. We assume that we have sufficient knowledge of the network and we have identified all the far edges. We note that identification of far edges requires only the knowledge of vertices and edges and hence should be possible in many practical situations. Let τ denote the time steps elapsed between the beginning of the epidemic and its detection. Let m denote the number of vertices that can be immunized at each time step. To block a far edge we first immunize one of the two vertices connected by this far edge. Immunization is carried out by first blocking all the far edges and then immunizing at random. If the number of far edges is greater than m then blocking all the far edges will take more than one time step.

In Fig. 3 we show the fraction of vertices affected as a function of time steps for a small-world network. Curve (a) shows the uncontrolled spread of the epidemic. Curves (d) and (g) show the spread of epidemic with the control method suggested above for $\tau = 7$ and 2 respectively. For comparison we show, by curves (c) and (f), the epidemic with only random immunization for $\tau = 7$ and 2 respectively. It is obvious that the far edge control mechanism proposed here is very effective. For larger τ some of the far edges are already traversed by the epidemic, decreasing the efficiency of our control mechanism. Comparing the far edge immunization and the random immunization, we find that the far edge immunization

decreases the rate of spread of epidemic more effectively but takes longer time for completely stopping the spread (See Fig. 3, curves (d) and (g)). Further, to test the effectiveness of our method we compare the results with another method of immunization. We order the vertices by their degree. Immunization is carried out by starting with the vertex with the largest degree and then going down the degree. The results for $\tau = 7$ and 2 are shown as curves (b) and (e) in Fig. 3 respectively. We note that results for immunization using degree are similar to that of the random immunization.

Let d denote the asymptotic difference between the number of affected vertices in random and far edge immunization. We plot d as a function of m for three different values of \mathcal{F} (or p) in Fig. 4. The plot shows that the far edge immunization is most effective when m is about half the number of far edges. The reason for the decrease of d for large m is that the probability that random immunization blocks a far edge, keeps on increasing as m increases, thereby decreasing the difference between the two methods. The plot of d as a function of \mathcal{F} for different values of m is shown in Fig. 5. The figure shows that the far edge immunization is more effective for small-world networks. Also from Figs. 4 and 5 it is clear that the far edge immunization gives a substantial benefit in terms of number of unaffected vertices in the small-world case and this number can be as large as 410 which is more than 40% of the total number of vertices.

Now, we consider an interesting model of advertisement. Let r be the number of vertices or centers from where a product is advertised. The information about the product spreads by word of mouth to the neighbours with the probability q_t where t is the time elapsed from the initial advertisement. We compare the results of two different ways of choosing the initial centers. In one way the centers are chosen at random and in the other they are chosen as one of the vertex in a far edge. Fig. 6 shows the number of people informed about the product as a function of t . It is clear that the choice of centers using far edges has definite advantage over that of random choice.

To conclude we have introduced the concept of *far edges* in networks. Our definition of a far edge is in accordance with the intuitive idea of a “far away” connection between

two vertices. The advantage of our definition of far edge is that it is independent of the underlying topology of the network. Also the definition is algorithmic in nature, and allows the determination of far edges only from the knowledge of vertices and edges. We have also applied the idea of far edges to the networks which are not generated by the algorithm given in Ref. [3] and arrived at similar conclusions [6].

We have demonstrated the use of far edges in the control of the spread of an epidemic and the advertisement of products. Our simulations show that the far edges are indeed important in the spread of epidemic, particularly in the small-world networks. We have shown that the knowledge of far edges can be fruitfully utilized to control the spread of epidemic and better advertisement. Our results strongly indicate that the far edges are the key elements responsible for the special properties of small world phenomena.

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Figure Captions:

Figure 1 An example of a network consisting of a far edge. The edge between vertices ‘a’ and ‘b’ is a far edge of order one.

Figure 2 The graph of $C(\mathcal{F})/C(0)$ and $L(\mathcal{F})/L(0)$ as a function of \mathcal{F} , where C is the clustering coefficient, L is the average path length and \mathcal{F} is the ratio of the number of far edges with the total number of edges. This figure is similar in nature to the plot of $C(p)/C(0)$ and $L(p)/L(0)$ as functions of p . The small-world networks lie around $\mathcal{F} = 0.01$.

Figure 3 The graphs of fraction of vertices affected as a function of time steps. The curve (a) is the epidemic spread without immunization, the curves (c) and (f) represent the spread when the random immunization is applied (see text) for $\tau = 7$ and 2 respectively, the curve (b) and (e) shows the spread if the immunization is carried out for the vertices with highest degree first and then in descending degree for $\tau = 7$ and 2 respectively and the curves (d) and (g) are the spread when the far edge immunization is used $\tau = 7$ and 2 respectively. The simulations are carried out on a small-world network of 1000 vertices and 10000 edges. The plotted results are averaged quantities over 500 seeds for epidemic.

Figure 4 The graph of the asymptotic difference between the number of affected vertices in random and far edge immunization, d as function of number of vertices immunized in one time step, m . The three curves (a), (b) and (c) are for $\mathcal{F} = 0.0022$, 0.0084 and 0.0162 respectively. The curve (b) corresponds to small-world network. The other parameters are as in Fig. 3.

Figure 5 The graph of d as function of \mathcal{F} . The three curves (a), (b) and (c) are plotted for $m = 30$, 10 and 80 respectively. The figure shows that the immunization method suggested here is most effective in small-world networks.

Figure 6 The graph of number people informed as function of t . The curves (a) and (b) show the result for far edge centers and random centers respectively. The simulation is carried out on a small-world network with 1000 vertices and 10000 edge. The initial advertisement is done from five centers. The probability function q_t is chosen as $q_1 = 0.8$ and $q_i = 0.18$, where $i \geq 2$.

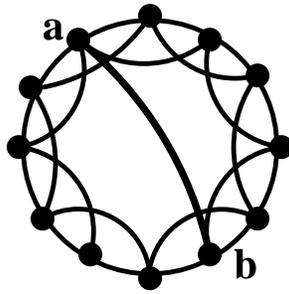


Fig. 1

(SAP & REA)

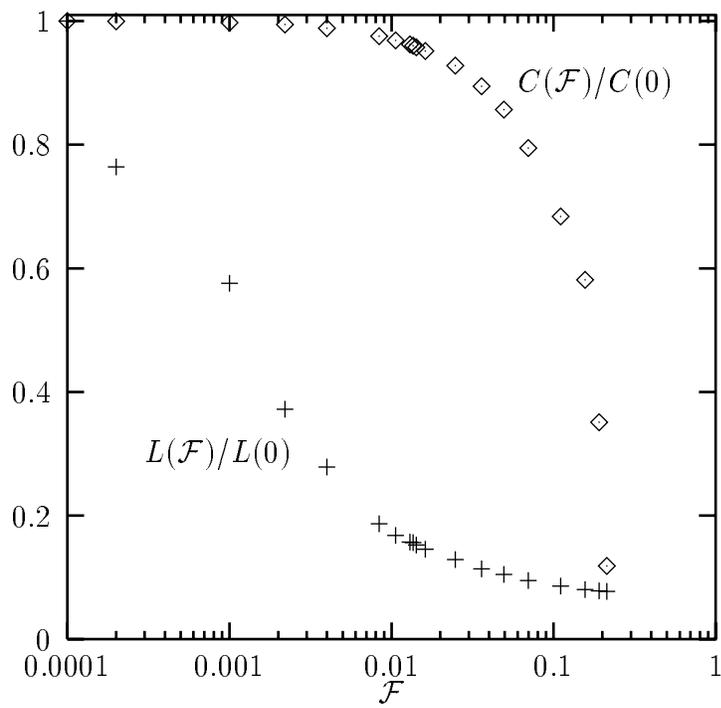


Fig. 2

(SAP & REA)

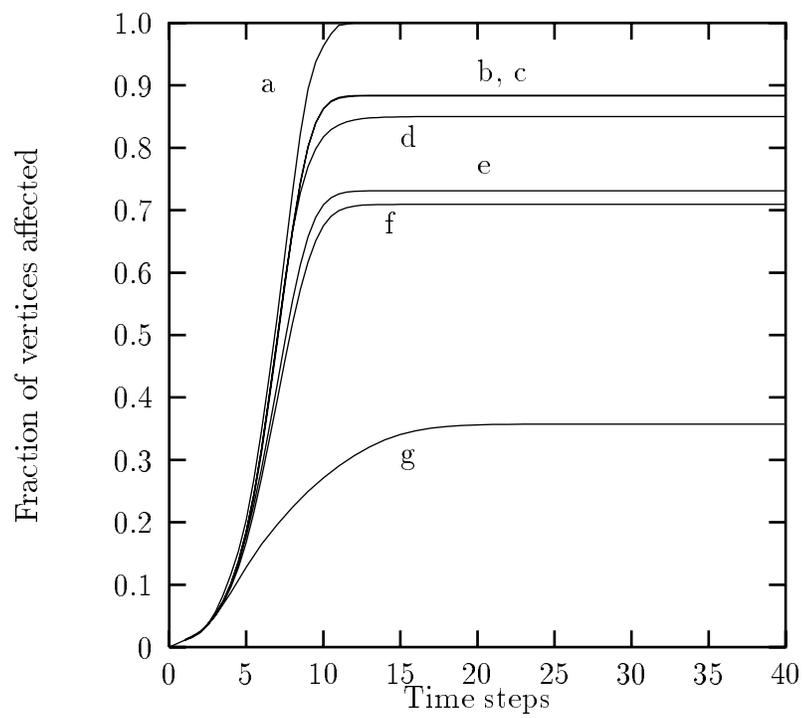


Fig. 3

(SAP & REA)

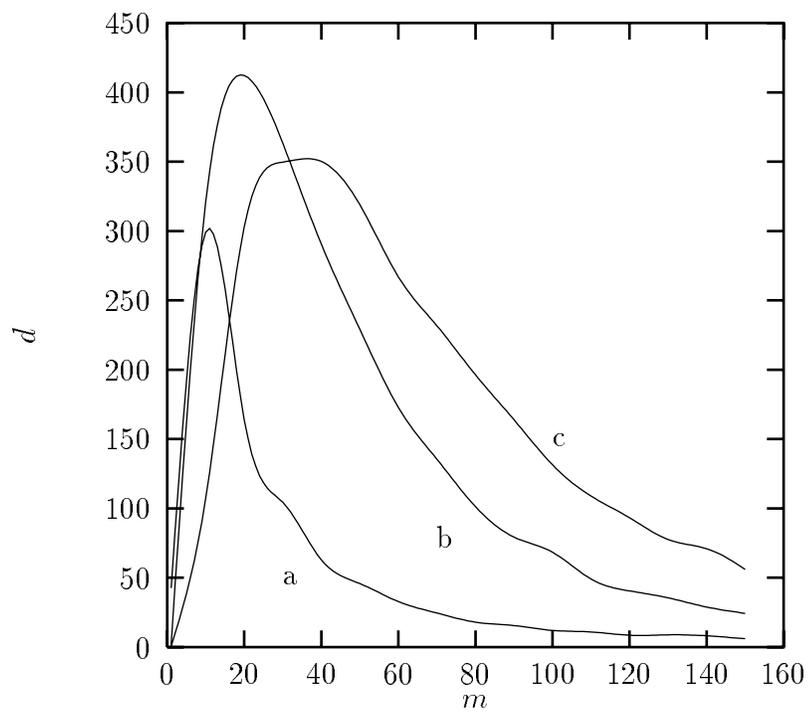


Fig. 4

(SAP & REA)

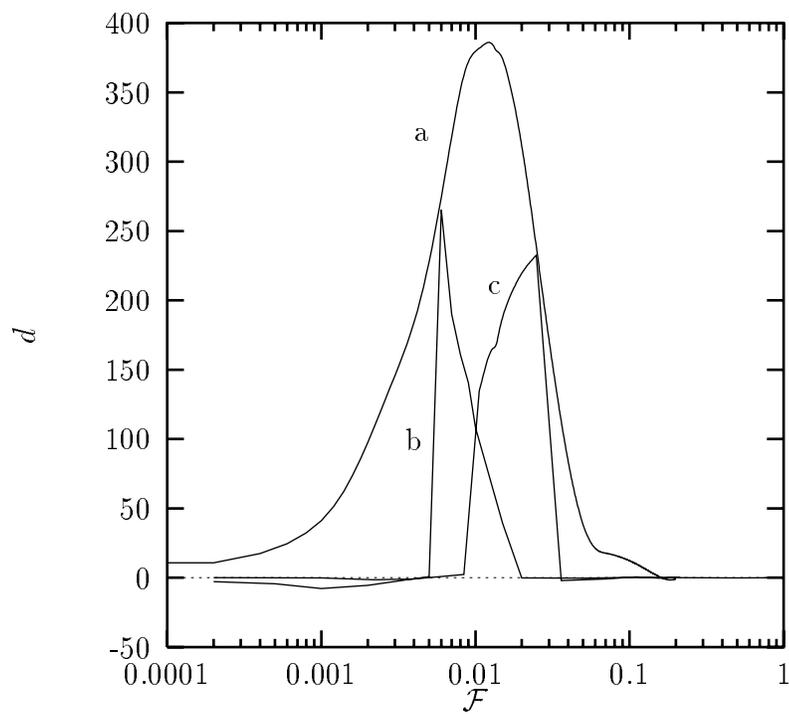


Fig. 5

(SAP & REA)

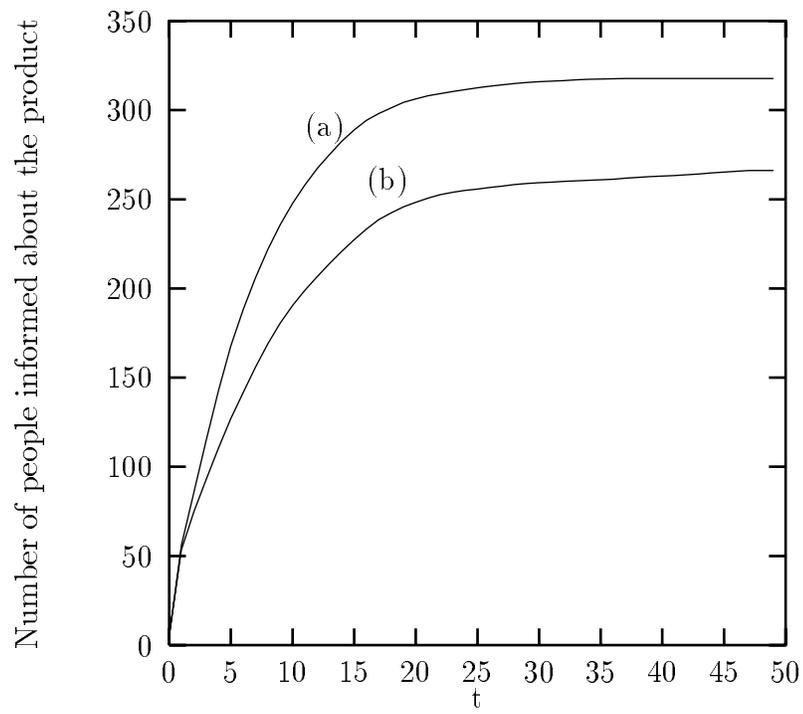


Fig. 6

(SAP & REA)