Spreading of infection in a two species reaction-diffusion process in networks

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We study the dynamics of the infection of a two mobile species reaction from a single infected agent in a population of healthy agents. Historically, the main focus for infection propagation has been through spreading phenomena, where a random location of the system is initially infected and then propagates by successfully infecting its neighbor sites. Here both the infected and healthy agents are mobile, performing classical random walks. This may be a more realistic picture to such epidemiological models, such as the spread of a virus in communication networks of routers, where data travel in packets, the communication time of stations in *ad hoc* mobile networks, information spreading (such as rumor spreading) in social networks, etc. We monitor the density of healthy particles $\rho(t)$, which we find in all cases to be an exponential function in the long-time limit in two-dimensional and three-dimensional lattices and Erdős-Rényi (ER) and scale-free (SF) networks. We also investigate the scaling of the crossover time t_c from short- to long-time exponential behavior, which we find to be a power law in lattices and ER networks. This crossover is shown to be absent in SF networks, where we reveal the role of the connectivity of the network in the infection process. We compare this behavior to ER networks and lattices and highlight the significance of various connectivity patterns, as well as the important differences of this process in the various underlying geometries, revealing a more complex behavior of $\rho(t)$.

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I. INTRODUCTION

Reaction-diffusion systems are systems in which there is a competition between two factors: the interaction (reaction) between the particles and their diffusion on the substrate on which they move. While such processes have been widely applied to chemical systems, they can also describe dynamical processes of nonchemical nature in physics, biology, and even communication systems. The field is vast, and with a lack of general approach for the treatment of that type of problem as well as the variety of substrates on which such reactions take place, we see that several such problems pose new challenges [1].

There have recently been new approaches examining such processes occurring on networks [2–13]. Networks describe systems from various fields, such as communication (e.g., the Internet), the social sciences, transportation, sexual contacts, ecological systems, protein and gene interactions in biology, and others. Therefore, a reaction-diffusion process occurring on a network simply depicts the interaction among such agents, and as it has been shown recently it may reveal some very rich dynamics [3,6,7,9,10,14–16]. The Erdős-Rényi (ER) [17] is a well-known simple model, which generates random graphs by setting an edge between each pair of nodes with some probability p, independent of other edges. This yields (in the limit $N\rightarrow\infty$) a Poisson distribution (for $p<1$) of the degree k of the node: $P(k)=(\langle k\rangle^k/k!)e^{-\langle k\rangle}$ with $\langle k\rangle=p(N-1)$, with $p=1$ giving the completely connected graph.

Scale-free (SF) networks, termed after the absence of characteristic typical node connectivity, exhibit many unusual properties compared to simple lattice models, random graphs, small-world (Watts-Strogatz) networks, and ER networks. They have been widely studied during recent years since they describe many real-world structures [18–21] from markedly different disciplines, and new systems are added continuously in the list. This ubiquity explains the intense interest devoted to the study of the complex network field. SF networks are defined by a degree distribution which follows a power law $P(k)\sim k^{-\gamma}$, where γ is a parameter which controls the broadness of the distribution and is characteristic of the network. The minimum degree k_{min} is the minimum connectivity that a node can have and it greatly influences the network structure. Although these systems may be very large, as counted by the number of nodes they encompass, their diameter is shown to be very small [19,21], a property which is usually referred to as the "small-world effect." The topology of such a network leads to a drastically different behavior for the above-mentioned reactions.

II. REACTION-DIFFUSION MODELS

Several types of bimolecular reaction have been studied in the literature over the past 30 years or so. Two common models include the $A+A\rightarrow 0$, as well as the $A+B\rightarrow 0$ reaction [22–24], which are two of the most fundamental annihilation processes with many applications [1]. In the first, there is only one species and two like particles react upon collision, while in the second one there are two different species and a reaction occurs only between like-unlike particles. Numerical simulations verified the generation of the depletion zone for the one species reaction [24], while the spatial segregation is a very characteristic effect for the reaction of the two species [23]. Other studies have demonstrated the existence of several temporal regimes and explained the crossovers between the early-time and long-time behaviors (see [25], e.g., for a study of the crossover time on a tubular lattice) and a wealth of other information, rendering these systems as some of the most heavily studied systems of interacting particles [1,4]

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The behavior of these reactions depends strongly on the substrate on which they are taking place. For example, in the annihilation reaction of one kind it was shown that the depletion zone does not appear when the reaction occurs in networks [4,5], as opposed to lattices. Similarly, for the two-kind reaction the segregation of the reactants does not appear in scale-free networks [4,6,7]. Thus, it has been shown that there is a marked difference in the reaction-diffusion process between lattices and networks. The presence or absence of correlations in the network construction model has also been demonstrated to affect the behavior of the reactions [5,26,27].

Another example model for diffusion-limited reaction process is the trapping problem which is $A+T\rightarrow T$, where T is a static site which acts as an irreversible trap, annihilating all A particles colliding with it. Trapping has been studied in regular lattices and in fractal spaces [28–32] and recently in small-world [8], Erdős-Rényi [9], and scale-free networks [3,9], as well as fractal and pseudofractal networks [11–13]. In the trapping reaction in low dimensions, the occurrence of $A-T$ reactions creates a depletion zone around the trap, which is a form of self-organization of reactants [33]. The depletion zone growth has been shown, by theory [31,34] and experiment [35], to exhibit a nonuniversal scaling with time for different lattice dimensionalities. However, it has also been shown [10] that the depletion zone is absent in regular, Erdős-Rényi, and scale-free networks, appearing only in very sparse networks.

Other cases in the diffusion-limited reaction scheme include three species reaction. The reversible process $A+B\leftrightarrow C$ was studied in [36]. A similar model has also been used to describe the population dynamics with mutations and migration effects in well mixed population with no spatial effects [37,38] or in scale-free networks [39]. The irreversible process $A+B\rightarrow C$ has been studied in [40,41], where the total amount of product C was found to scale with time as power law [41] for the subdiffusion process. Such reaction-diffusion processes have also been applied to dynamic processes which include the spread of epidemics and various interactions in population models in networks [15,16,42]. In the past 20 years, a large number of works extended the initial ideas and explained in detail the appearance of these effects.

The $A+B\rightarrow 2B$ autocatalytic reaction is of the irreversible chemical reaction type and there are applications of this model not only in physics but, increasingly, also in biology, social sciences, and computer science. Particle systems on lattices have been extensively studied in recent years with the voter and infection models, modeled by a Markov process, being two of the most popular [43]. It has also been analyzed in three-dimensional (3D) Euclidean structures and fractal substrates in the low-density regime [44,45], with a continuous description by the Fisher equation [46–48], which describes the system in terms of front propagation (see also [49] for the reaction under subdiffusion). While this process has been widely applied to chemical processes, we use it in our model in a completely different fashion, namely, to describe the dynamics of infection spreading in networks, such as computer networks, social networks, etc.

III. MODEL

Several models of infection spreading exist in the literature built on different algorithms, such as the susceptible-infected-recovered (SIR) model [50], the susceptible-infected-susceptible (SIS) model [51], the susceptible-infected-recovered-susceptible (SIRS) model [52], etc. These are deterministic compartmental models, based on a spreading mechanism, in which the compartments used consist of three classes: susceptible, infected, and recovered. When such processes are applied to networks, each network node can be in any of these three states, and a measure of infection is the total number of infected nodes. In contrast to these models, we use mobile species rather than a spreading process to model the infection spreading. This might offer a better description for real-world systems in which the information traverses the networks in packets, such as routers in computer networks, wireless sensor networks [53], *ad hoc* networks [54], and peer-to-peer networks [55]. Since these packets are mobile, it follows that these systems can be described by both infected and susceptible agents being mobile.

Specifically, we examine a system of healthy particles which are infected by a single "sick" particle, which is able to infect every healthy particle that it comes in contact with. Both species are mobile. The reaction stops when all healthy particles are infected. This would correspond to a reaction of type $A+B\rightarrow 2B$, which was recently studied [2] on general graphs and some important special cases, such as lollipops, cliques, four-regular graphs, and $G_{n,p}$ graphs. In this work a set of probabilistic tools was developed to obtain the upper bounds on the expected value of the infection time.

Here, we extend this model to include a broader category of networks, i.e., ER and SF networks with varying connectivities, since such systems describe many real-world structures [18–21], from a wide variety of fields, such as communication (e.g., the Internet), the social sciences, transportation, sexual contacts, ecological systems, protein and gene interactions in biology, and others. We focus on the evolution of density of healthy particles as a measure of the rate of infection on the network. We perform Monte Carlo calculations for lattices and networks and compare the corresponding behavior for these different substrates.

The problem studied here is an example of the spread of a virus in networks of routers, where the data traverse the network in packets, information spreading (such as rumor spreading) in social networks, and the communication time of stations in *ad hoc* mobile networks under the existence of a mobile infrastructure as a virtual intermediary pool for messages [2], etc. This follows since in some cases data packets traverse the network in a random fashion (for example, in wireless sensor networks [53], *ad hoc* networks [54], and peer-to-peer networks [55]). An *ad hoc* mobile network is a collection of mobile hosts, with wireless communication capabilities, forming a temporary network without the aid of any established fixed infrastructure.

Virus attacks in computer networks pose several key problems regarding intrusion propagation. Various models have been proposed for studying the effective detection and defeat of attacks (see, e.g., [56]). Intrusion propagation (the process of spread of such attacks) has mostly been investi-

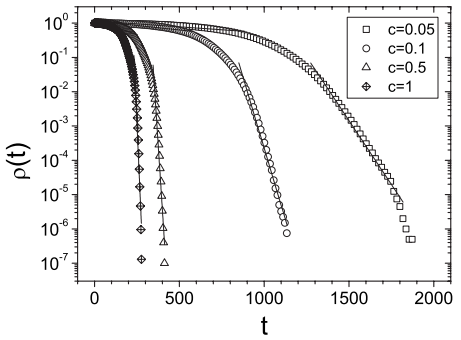

FIG. 1. $\rho(t)$ vs t for a 2D 200×200 lattice for different values of particle density c. Solid lines represent fitting for the short- and long-time regimes.

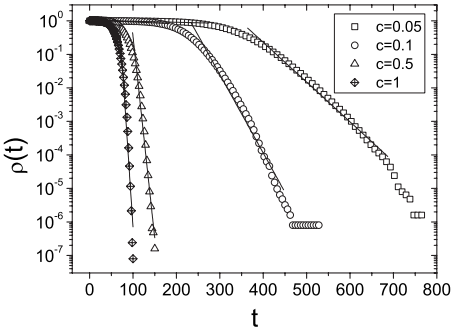

FIG. 2. $\rho(t)$ vs t for a 3D $50\times50\times50$ lattice for different values of particle density c.

gated under gossip or epidemiological models [57]. Also Nikoletseas *et al.* [58] investigated the analytic and experimental behavior of several protocols for the attack propagation problem in networks under a new model of intrusion propagation introduced there. Even and Monien studied the gossip problem in [59], where some people wish to distribute several rumors among themselves. In this case the specific persons who communicate at each time are fixed in advance by the designer of the algorithm.

We use a Monte Carlo simulation model to study the dynamics of this infection process. In our system, the infected particles have zero probability for recovery. We consider n particles with density c, which stays constant during the simulation, of which one is infected and the rest are healthy. All particles are initially placed on random sites regardless of the substrate the reaction is taking place on. They move randomly on the lattice or network and interact with each other. A lattice site or network node is allowed to have more than one particle (i.e., no excluded volume restriction). Whenever two particles of different types come in contact, the healthy particles become infected. The substrates we have used are one-dimensional (1D), two-dimensional (2D), and 3D regular lattices, Erdős-Rényi networks, and scale-free networks.

We generate ER networks as follows: given a finite set of N nodes, all the $N(N-1)/2$ pairs are considered and a link between two nodes is added with probability p. Therefore, the average degree of each node is $\langle k\rangle=p(N-1)$. SF networks are constructed using the standard configuration model [60,61]. This model introduces correlations in the range $2<\gamma\leq3$, which are, however, present in most real-world networks [26]. First, the number of nodes N and the γ parameter of the particular network is fixed, and then each node i is assigned a number of links k_i from the $k^{-\gamma}$ distribution. The value of k lies in the range from 1 to $k_{max}=N-1$ (no upper cutoff value is used for k). Each node i extends k_i "hands" toward all other nodes. We randomly select two such hands (that do not belong in the same node) and connect them, thus creating a link. No double links are allowed, so if two nodes are already connected this link is rejected. We continue this process until all nodes have reached their preassigned connectivity.

The process takes place on the largest cluster of the network, which we identify using a breadth-first search algorithm as described in [62]. Given a graph $G=(V,E)$ and a distinguished source vertex s, breadth-first search systematically explores the edges of G to record every vertex that is reachable from s. It computes the distance from s to each reachable vertex, which is the smallest number of edges. Breadth-first search is so named because it expands the frontier between discovered and undiscovered vertices uniformly across the breadth of the frontier. The algorithm discovers all vertices at distance r from s before discovering any vertices at distance $r+1$.

At least 100 independent realizations were performed, where the network is built anew in each run. We place n particles of density c on the system, with one sick particle and $(n-1)$ healthy particles. All values of the particle density are normalized to unity. The steps of the algorithm are as follows:

(1) A particle is randomly selected.

(2) It moves on a random adjacent site (or node in the case of networks).

(3) Whenever two or more particles of different types are present on the same site then all healthy particles are infected.

(4) After each particle on the system has moved once, one time step (Monte Carlo step) has been accomplished.

(5) We proceed with steps 1–4 until the density $\rho(t)$ of healthy particles reaches 0.

IV. RESULTS AND DISCUSSION

Figures 1 and 2 show the decay for the density of healthy particles in 2D and 3D lattices, respectively. We have also

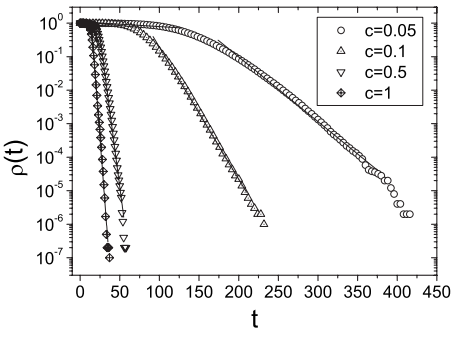

FIG. 3. $\rho(t)$ vs t for ER networks of $N=10^4$ and $\langle k\rangle=10$ for different values of particle density c.

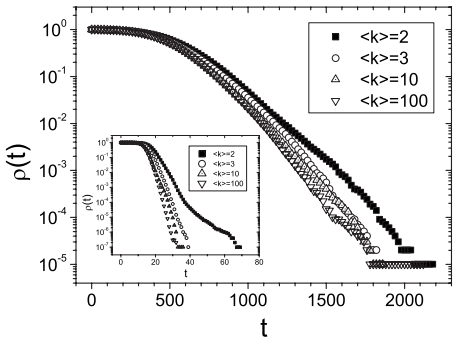

FIG. 4. $\rho(t)$ vs t for ER networks of $N=10^4$ and $c=0.01$ for different values of $\langle k\rangle$. Inset: same but for $c=1$.

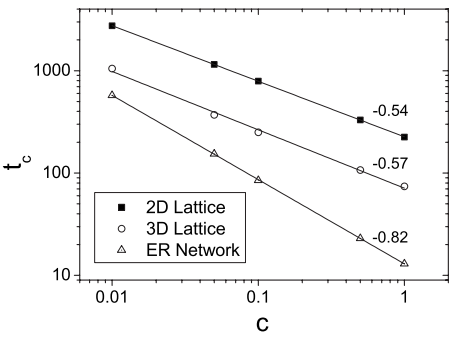

FIG. 6. Crossover time t_c from short- to long-time regimes for lattices and ER networks.

performed simulations in 1D lattices, which present the most trivial case, with the density $\rho(t)$ having a linear dependence on t and c in the form of $\rho(t)\sim \alpha ct$, where α is constant. It is apparent, however, that in both two dimensions and three dimensions, this behavior switches to an exponential function in the long-time regime. Since a 2D lattice is a more restricted geometry than three dimensions, it takes more time for the particles to become infected in the 2D case, comparing the infection times on three dimensions (see Figs. 1 and 2). The infection time is significantly reduced for networks, as we will show later.

Figure 3 show the $\rho(t)$ results vs t for ER networks for average degree $\langle k\rangle=10$. We also fit here the long-time regime with an exponential function. The picture looks similar to the one in lattices, with the infection time being reduced even more since the particles traverse the network more easily than a 2D or even 3D lattice. The density is, again, an exponential function of time in the long-time regime.

An unusual property of this process in ER networks is demonstrated in Fig. 4. We see that the connectivity of the network plays almost no role for the spread of the infection, which is shown to proceed just as quickly in both sparse and dense ER networks. The behavior of $\rho(t)$ regarding the connectivity of the network is significantly different in SF networks, as we will show later. In ER networks, the infection will spread out with roughly the same rate with $\langle k\rangle=3$ or $\langle k\rangle=100$. In addition, this is true regardless of the density of the particles that traverse the network, and the infection proceeds at the same rate for networks with different values of $\langle k\rangle$, both for $c=0.01$ (Fig. 3) and $c=1$ (inset of Fig. 3), with a small deviation for very sparse networks (i.e., $\langle k\rangle=2$). This behavior is significantly different than other reaction processes, e.g., the trapping reaction, in which the average degree was demonstrated to play in important role in the function of $\rho(t)$ for sparse networks [9], as well as the distribution of the particles on the network and the depletion zone formation [10].

The particle density c plays, of course, a large role in the spreading of the infection, with the larger density having naturally much lower infection times than low c values. In Fig. 5, we plot the slope β of the exponential long-time regime against the particle density c for 2D and 3D lattices and ER networks (Figs. 1–3, respectively). The slope β is demonstrated here to have a linear scaling with c, with the value of the slopes in Fig. 5 very close to 1 ($\approx$0.98). It then follows that $\beta\sim-\alpha ct$, where α is some constant. Therefore, for these cases, we can claim that in 2D and 3D lattices and ER networks the density $\rho(t)$, in the long-time regime, is an exponential function of time in the form of

$$\rho(t)\sim e^{-\alpha ct}. \tag{1}$$

We have also investigated the crossover time t_c from short- to long-time regime, which is the time at the intersection of the fitting of these two regimes. The scaling of t_c, with the particle density c, is presented in Fig. 6. It becomes apparent that for the 2D, 3D, and ER network case the scaling of t_c with c is a power law,

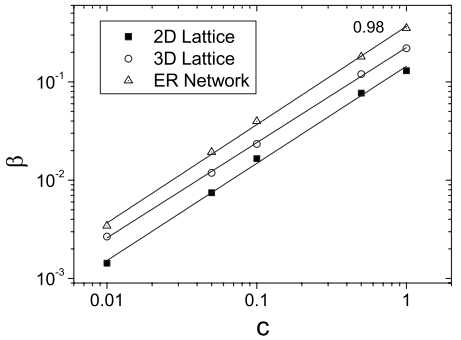

FIG. 5. Slope β of the exponential fit for the long-time regime for lattices and ER networks. All slopes are $\approx$0.98.

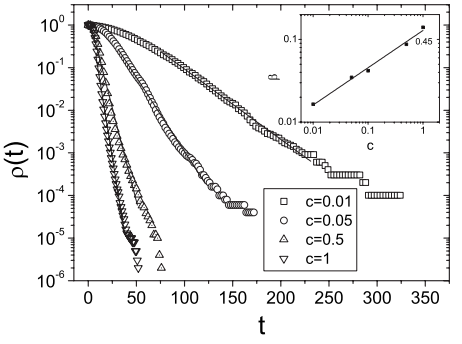

FIG. 7. $\rho(t)$ vs t for SF networks of $N=10^4$ and $\gamma=2.5$ for different values of particle density c. Inset: slope β of the exponential long-time regime vs c.

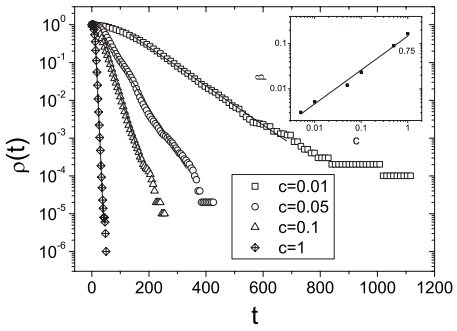

FIG. 8. $\rho(t)$ vs t for SF networks of $N=10^4$ and $\gamma=3$ for different values of particle density c. Inset: slope β of the exponential long-time regime vs c.

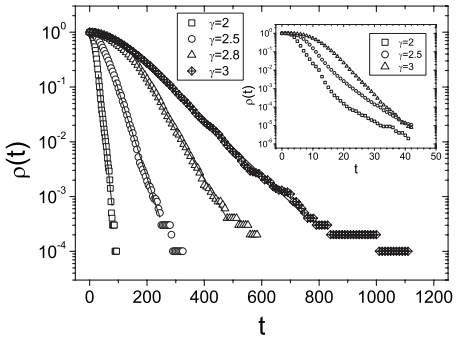

FIG. 9. $\rho(t)$ vs t for SF networks of $N=10^4$ and $c=0.01$ for different values of γ. Inset: same but for $c=1$.

$$t_c \sim c^{-\theta}. \tag{2}$$

However, while the exponent θ in Eq. (2) is roughly the same for 2D and 3D lattices ($\theta \approx 0.55$), this is not the case in ER networks ($\theta \approx 0.82$). Therefore, ER networks are shown to behave considerably different compared to their lattice counterparts, as far as the crossover time is concerned for this diffusion-limited reaction model.

We examine the density $\rho(t)$ in SF networks in Figs. 7–9. The exponential regime is also present here; however, the crossover to this behavior is almost completely absent and this regime is not limited to long-time behavior but spans almost the entire time domain in the infection process. This is an important difference compared to ER networks or lattices; in SF networks the exponential time regime is reached almost immediately after the beginning of the diffusion-limited reaction process. This phenomenon can be explained in the basis of the role of the highly connected nodes of the network (hubs). In other systems, such as lattices and ER networks, the infection process takes time to reach the exponential regime because particles cannot find each other as easily as in SF networks. Here, the infection is spread almost immediately because of the high centrality of the hubs, which become the centers of infection for the whole process as most particles meet and are infected there. Therefore, in an infection of this type, the crossover which is characteristic of lattices and ER networks is absent in SF networks, where the infection spreads very quickly reaching almost immediately the exponential time regime.

In Figs. 7 and 8 we see that $\rho(t)$ also has an exponential decay form. There are, however, deviations from the exponential behavior, especially in well-connected networks (see inset of Fig. 9). While $\rho(t)$ is an exponential function of time in SF networks, the exponent does not depend linearly on particle density, as is the case with lattice and ER networks [Eq. (1)] but is a power-law function of c. The slope is shown to depend on γ (compare the inset of Figs. 7 and 8), but an exact solution of the explicit dependence on γ is still lacking.

The role of the connectivity of the SF network is revealed in Fig. 9, where we show $\rho(t)$ for $c=0.01$ different values of γ. Here we see that the infection progresses much more quickly in a well-connected network (e.g., $\gamma=2.0$) than a sparse one (e.g., $\gamma=3.0$). This is in contrast to ER networks (Fig. 4), where it was shown that the connectivity of the network varying $\langle k \rangle$ has little or no effect to the rate of the infection process. The behavior of $\rho(t)$ in SF networks is more complex than their ER counterparts if we compare Fig. 9 with the inset, where the same quantity is shown, but for $c=1$, where it is evident that the connectivity of the network has very little effect on the infection time. Therefore, for small densities the connectivity of the network becomes the major factor in the infection process, while for high densities it plays little or no role. As mentioned above, the exact function of $\rho(t)$ and the relationship of c with γ in SF networks are still lacking. From a qualitative point of view, however, we can say that the significance of connectivity of the network in the infection process is more pronounced in lower particle densities in SF networks.

V. CONCLUSIONS

In summarizing, we have developed a model to study a two mobile species infection process, which is related to epidemiological modes, such as the spread of a virus in networks where data travel in packets, the spread of rumors in social networks, and information propagation in *ad hoc* mobile networks. 2D and 3D lattices as well as ER networks can be described with an exponential decay in the long-time regime in the form of $\rho(t) \sim e^{-\alpha c t}$. The crossover time from short to long time scales with c as a power law in these systems, with ER networks characterized by a different exponent than lattices. The connectivity of ER networks was shown to have little to no influence in the infection process, with a small deviation for very sparse networks. In SF networks the crossover is almost completely absent and the infection spreads immediately, with the highly connected nodes acting as infection centers. The behavior of $\rho(t)$ in SF networks is more complex than their ER counterparts, where the density c determines how the connectivity of the network affects the process. For low c in SF networks the connectivity is shown to severely influence the infection process, which proceeds much faster in well-connected networks, while for high values of c the infection progresses with almost the same rate in both sparse and dense SF networks.

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