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Epidemic spreading in scale-free networks

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Abstract

The Internet, as well as many other networks, has a very complex connectivity recently modeled by the class of scale-free networks. This feature, which appears to be very efficient for a communications network, favors at the same time the spreading of computer viruses. We analyze real data from computer virus infections and find the average lifetime and prevalence of viral strains on the Internet. We define a dynamical model for the spreading of infections on scale-free networks, finding the absence of an epidemic threshold and its associated critical behavior. This new epidemiological framework rationalize data of computer viruses and could help in the understanding of other spreading phenomena on communication and social networks.

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Many social, biological, and communication systems can be properly described by complex networks whose nodes represent individuals or organizations, and links mimic the interactions among them [1,2]. Particularly interesting examples are the Internet and the world-wide-web, which have been extensively studied because of their technological and economical relevance [3–5]. These studies have revealed, among other facts, the *scale-free nature* of these networks [3,5]. This results in the power-law distribution $P(k) \sim k^{-\gamma}$ for the probability that a node of the network has k connections to other nodes, with an exponent γ that ranges between 2 and 3. The importance of local clustering is indeed the key ingredient in the modeling of these networks with the recent introduction of scale-free (SF) graphs [6].

In view of the wide occurrence of complex networks in nature it is of great interest to inspect the effect of their features on epidemic and disease spreading [7], and more in general in the context of the nonequilibrium phase transitions typical of these phenomena [8]. The study of epidemics on these networks finds an immediate practical application in the understanding of computer virus spreading [9,10], and could also be relevant to the fields of epidemiology [11] and pollution control [12].

In this Letter, we analyze data from real computer virus epidemics, providing a statistical characterization that points out the importance of incorporating the peculiar topology of scale-free networks in the theoretical description of these infections. With this aim, we study by large scale simulations and analytical methods the susceptible-infected-susceptible [11] model on SF graphs. We find the absence of an epidemic threshold and its associated critical behavior, which implies that SF networks are prone to the spreading and the persistence of infections whatever spreading rate the epidemic agents possess. The absence of the epidemic threshold—a standard element in mathematical epidemiology [11]—radically changes many of the standard conclusions drawn in epidemic modeling. The present results are also relevant in the field of absorbing-state phase transitions and catalytic reactions [8].

The epidemiological analysis of computer viruses has been the subject of a continuous interest in the computer science community [10,13–15], following mainly approaches borrowed from biological epidemiology [11]. The standard model used in the study of computer

virus infections is the susceptible-infected-susceptible (SIS) epidemiological model. Each node of the network represents an individual and each link is a connection along which the infection can spread to other systems. This model relies on a coarse grained description of individuals in the population. Individuals exist only in two discrete states, “healthy” or “infected”. At each time step, each susceptible (healthy) node is infected with rate ν if it is connected to one or more infected nodes. At the same time, infected nodes are cured and become again susceptible with rate δ , defining an effective spreading rate $\lambda = \nu/\delta$. Without lack of generality, we can set $\delta = 1$. The updating can be performed both with parallel and sequential dynamics [8]. In models with local connectivity (Euclidean lattices and mean-field models), the most significant result is the general prediction of a nonzero epidemic threshold λ_c [8,11]. If the value of λ is above the threshold, $\lambda \geq \lambda_c$, the infection spreads and becomes persistent. Below it, $\lambda < \lambda_c$, the infection dies out exponentially fast. The epidemic threshold is actually equivalent to a critical point in a nonequilibrium phase transition. In this case, the critical point separates an active phase with a stationary density of infected nodes from a phase with only healthy nodes and null activity. In particular, it is easy to recognize that the SIS model is a generalization of the contact process (CP) model, that has been extensively studied in the context of absorbing-state phase transitions [8]. Statistical observations of virus incidents in the wild, on the other hand, indicate that all viruses that are able to pervade, spread much slower than exponentially, and saturate to a very low level of persistence, affecting just a tiny fraction of the total number of computers [10]. This fact is in striking contradiction with the theoretical predictions unless in the very unlikely chance that *all* computer viruses have an effective spreading rate tuned just infinitesimally above the threshold. This points out that the view obtained so far with the modeling of computer virus epidemics is very instructive but not completely adequate to represent the real phenomenon.

In order to gain further insight on the spreading properties of viruses in the wild, we have analyzed the prevalence data reported by the Virus Bulletin [16] from February 1996 to March 2000. We have analyzed in particular the *surviving probability* of homogeneous groups

of viruses, classified according to their infection mechanism [9]. We consider the total number of viruses of a given strain that born and die within our observation window. Hence, we calculate the surviving probability $P_s(t)$ of the strain as the ratio of viruses still alive at time t after their birth and the total number of observed viruses. Fig. 1 shows that the surviving probability suffers a sharp drop in the first two months of a virus' life. This is a well-known feature [10,13] indicating that statistically only a small percentage of viruses give rise to a significant outbreak in the computer community. Fig. 1, on the other hand, shows for larger times a clean exponential tail, $P_s(t) \sim \exp(-t/\tau)$, where τ represents the characteristic life-time of the virus strain [17]. The numerical fit of the data yields $\tau \simeq 14$ months for boot and macro viruses and $\tau \simeq 6 - 9$ months for file viruses. These characteristic times are impressively large if compared with the interval in which anti-virus software is available on the market (usually within days or weeks after the first incident report) and indicate that the viral persistence time scale is more related to the implementation of prophylactic safety measures than to the timely availability of the specific anti-virus. These external factors, however, are not possibly competing on the short time scale of the viruses spread (days or weeks), and again we face the very puzzling question of why viruses seem to have access to persistent low prevalence levels but never grow exponentially.

The key point to understand the puzzling properties exhibited by computer viruses resides in the capacity of many of them to be borne by electronic mail as an apparently innocuous attachment [10]. Having this property in mind, it is easy to realize that the topology of the connections between individuals cannot be correctly represented by an Euclidean lattice, or a mean-field model. In this sense, these connections should instead have essentially the topology of the Internet, through which electronic mail travels. The scale-free connectivity of the Internet implies that each node has a statistically significant probability of having a very large number of connections compared to the average connectivity $\langle k \rangle$ of the network. That opposes to conventional random networks (local or nonlocal) in which each node has approximately the same number of links $k \simeq \langle k \rangle$ [18]. The fact that all virus strains qualitatively show the same statistical features indicates that very likely all of them

spread on networks with connectivity properties analogous to those of the Internet [19]. It is then natural to foresee that scale-free properties should be included in a theory of epidemic spreading of computer viruses.

To address the effects of scale-free connectivity in epidemic spreading we study the SIS model on the SF network. We consider the graph generated by using the algorithm devised in Refs. [6]: We start from a small number m_0 of disconnected nodes; every time step a new vertex is added, with m links that are connected to an old node i with probability $k_i / \sum_j k_j$. After iterating this scheme a sufficient number of times, we obtain a network composed by N nodes with connectivity distribution $P(k) \sim k^{-3}$ and average connectivity $\langle k \rangle = 2m$. In this work we take $m = 3$. We have performed numerical simulations on graphs with number of nodes ranging from $N = 10^3$ to $N = 8.5 \times 10^6$ and studied the variation in time and the stationary properties of the density of infected nodes ρ in surviving infections; i.e. the virus prevalence. Initially we infect half of the nodes in the network, and iterate the rules of the SIS model with parallel updating. After an initial transient regime, the system stabilizes in a steady state with a constant average density of infected nodes. The prevalence is computed averaging over at least 100 different starting configurations, performed on at least 10 different realizations of the random networks.

The first arresting evidence from simulations is the *absence* of an epidemic threshold, i.e., $\lambda_c = 0$. In Fig. 2 we show the virus prevalence in the steady state that decays with decreasing λ as $\rho \sim \exp(-C/\lambda)$, where C is a constant. This implies that for any finite value of λ the virus can pervade the system with a finite prevalence, in sufficiently large networks. In all networks with bounded connectivity the steady state prevalence is always null below the epidemic threshold; i.e. all infections die out. Further evidence to our results is given by the total absence of scaling of ρ with the number of nodes that is, on the contrary, typical of epidemic transitions in the proximity of a finite threshold [8]. This allows us to exclude the presence of any spurious results due to network finite size effects. The present result can be intuitively understood by noticing that for usual lattices, the higher the node's connectivity, the smaller the epidemic threshold. In a SF network the unbounded

fluctuations in connectivity ($\langle k^2 \rangle = \infty$) plays the role of an infinite connectivity, annulling thus the threshold.

Finally, we analyze the spreading of infections starting from a localized virus source. We observe that the spreading growth in time has an algebraic form, that is in agreement with real data that never found an exponential increase of a virus in the wild. Noteworthy, by applying the definition of surviving probability $P_s(t)$ used to analyze real data, we recover in our model the same exponential behavior in time (see Fig. 3a). The characteristic lifetime depends on the spreading rate and the network sizes, allowing us to relate the average lifetime of a viral strain with an effective spreading rate and the Internet size [20]. At the same time, the divergence of lifetimes for larger networks points out the possible increasing of the viruses lifetime during the eventual expansion of the Internet.

We can also approach the system analytically by writing the mean-field equation governing the time evolution of $\rho(t)$. In order to take into account connectivity fluctuations, we consider the relative density $\rho_k(t)$ of infected nodes with given connectivity k ; i.e the probability that a node with k links is infected. The dynamical mean-field reaction rate equations can be written as [8]

$$\partial_t \rho_k(t) = -\rho_k(t) + \lambda k(1 - \rho_k(t))\Theta(\lambda). \quad (1)$$

The creation term considers the probability that a node with k links is healthy ($1 - \rho_k(t)$) and gets the infection via a connected node. The probability of this event is proportional to the infection rate, the number of connections, and the probability $\Theta(\lambda)$ that any given link points to an infected node. By imposing stationarity ($\partial_t \rho_k(t) = 0$) we find the stationary densities

$$\rho_k = \frac{k\lambda\Theta(\lambda)}{1 + k\lambda\Theta(\lambda)}, \quad (2)$$

denoting that the higher the node connectivity, the higher the probability to be infected. This inhomogeneity must be taken into account in the self-consistent calculation of $\Theta(\lambda)$. Indeed, the probability that a link points to a node with s links is proportional to $sP(s)$.

In other words, a randomly chosen link is more likely to be connected to a node with high connectivity, yielding

$$\Theta(\lambda) = \sum_k \frac{kP(k)\rho_k}{\sum_s sP(s)}. \quad (3)$$

Since ρ_k is on its turn function of $\Theta(\lambda)$, we obtain a consistency equation that allows to find $\Theta(\lambda)$. Finally we can evaluate the behavior of ρ by solving the second consistency relation

$$\rho = \sum_k P(k)\rho_k, \quad (4)$$

that expresses the average density of infected nodes in the system. In the SF model considered here, we have a connectivity distribution $P(k) = 2m^2/k^{-3}$, where k as approximated as a continuous variable [6]. In this case, integration of Eq.(3) allows to write $\Theta(\lambda)$ as

$$\Theta(\lambda) = \frac{e^{-1/m\lambda}}{\lambda m} (1 - e^{-1/m\lambda})^{-1}, \quad (5)$$

from which, using Eq.(4), we find at lowest order in λ :

$$\rho = 2e^{-1/m\lambda} + h.o. . \quad (6)$$

This very intuitive calculation recovers the numerical findings and confirms the surprising absence of any epidemic threshold or critical point in the model; i.e $\lambda_c = 0$. Finally, as a further check of our analytical results, we have numerically computed in our model the relative densities ρ_k , recovering the predicted dependence upon k of Eq.(2) (see Fig. 3b). It is also worth remarking that the present framework can be generalized to networks with $2 < \gamma \leq 3$, recovering qualitatively the same results [21].

The emerging picture for epidemic spreading in complex networks emphasizes the role of topology in epidemic modeling. In particular, the absence of epidemic threshold and critical behavior in a wide range of scale-free network provide an unexpected result that changes radically many standard conclusions on epidemic spreading. This indicates that infections can proliferate on these scale-free networks whatever spreading rates they may have. These

very bad news are, however, balanced by the exponentially small prevalence for a wide range of spreading rates ($\lambda \ll 1$). This point appears to be particularly relevant in the case of technological networks such as the Internet and the world-wide-web that show scale-free connectivity with exponents $\gamma \simeq 2.5$ [4,5]. For instance, the present picture fits perfectly with the observation from real data of computer virus spreading, and could solve the long standing problem of the generalized low prevalence of computer viruses without assuming any global tuning of the spreading rates. The peculiar properties of scale-free networks also open the path to many other questions concerning the effect of immunity and other modifications of epidemic models. As well, the critical properties of many nonequilibrium systems could be affected by the topology of scale-free networks. Given the wide context in which scale-free networks appears, the results obtained here and the proposed investigations could have intriguing implications in biology and social systems.

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FIGURES

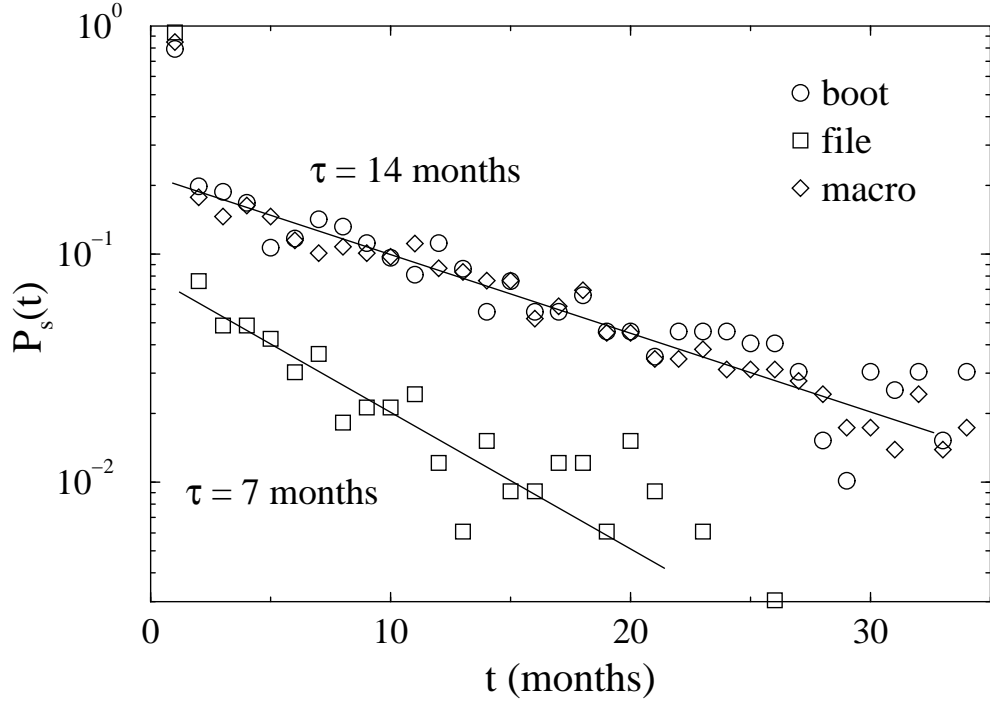


FIG. 1. Surviving probability for viruses in the wild. The 814 different viruses analyzed have been grouped in three main strains [9]: file viruses infect a computer when running an infected application; boot viruses also spread via infected applications, but copy themselves into the boot sector of the hard-drive and are thus immune to a computer reboot; macro viruses infect data files and are thus platform-independent. It is evident in the plot the presence of an exponential decay, with characteristic time $\tau \simeq 14$ months for macro and boot viruses and $\tau \simeq 7$ months for file viruses.

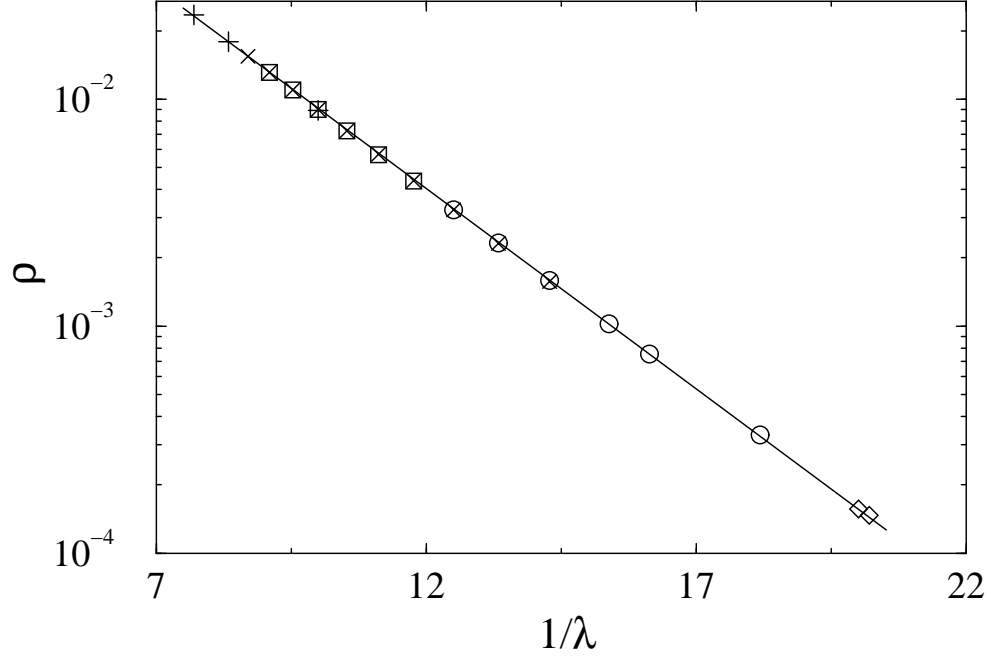


FIG. 2. Persistence ρ as a function of $1/\lambda$ for different network sizes: $N = 10^5$ (+), $N = 5 \times 10^5$ (□), $N = 10^6$ (×), $N = 5 \times 10^6$ (○), and $N = 8.5 \times 10^6$ (◇). The linear behavior on the semi-logarithmic scale proves the stretched exponential behavior predicted for ρ . The full line is a fit to the form $\rho \sim \exp(-C/\lambda)$.

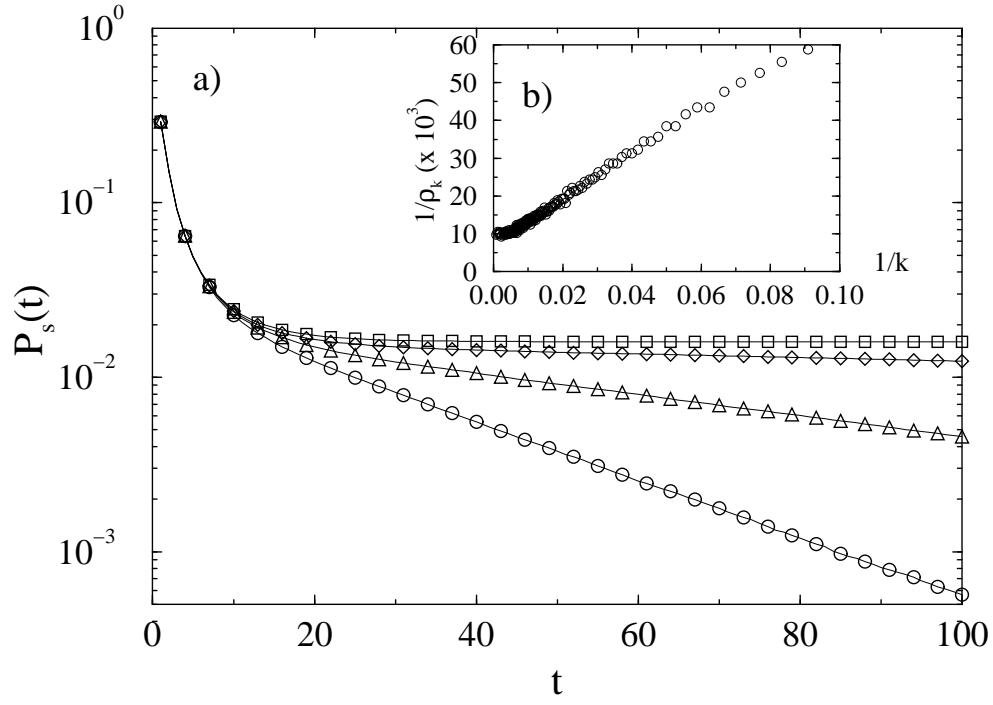


FIG. 3. a) Surviving probability $P_s(t)$ for a spreading rate $\lambda = 0.065$ in scale-free networks of size $N = 5 \times 10^5$ (\square), $N = 2.5 \times 10^4$ (\diamond), $N = 1.25 \times 10^4$ (\triangle), and $N = 6.25 \times 10^3$ (\circ). The exponential behavior, following a sharp initial drop, is compatible with the data analysis of Fig. 1. b) Relative density ρ_k versus k^{-1} in a SF network of size $N = 5 \times 10^5$ and spreading rate $\lambda = 0.1$. The plot recovers the form predicted in Eq.(2).