

# Spreading dynamics on small-world networks with connectivity fluctuations and correlations

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Infectious diseases and computer malwares spread among humans and computers through the network of contacts among them. These networks are characterized by wide connectivity fluctuations, connectivity correlations, and the small-world property. In a previous work [Phys. Rev. Lett. **96**, 038702 (2006)] I have shown that the connectivity fluctuations together with the small-world property lead to a spreading law characterized by an initial power law growth with an exponent determined by the average node distance on the network. Here I extend these results to consider the influence of connectivity correlations which are generally observed in real networks. I show that assortative and disassortative connectivity correlations enhance and diminish, respectively, the range of validity of this spreading law. As a corollary I obtain the region of connectivity fluctuations and degree correlations characterized by the absence of an epidemic threshold. These results are relevant for the spreading of infectious diseases, rumors, and information among humans and the spreading of computer viruses, email worms, and hoaxes among computer users.

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## I. INTRODUCTION

Halting an epidemic outbreak in its early stages requires a detailed understanding of the progression of the number of new infections (incidence). Current mathematical models predict that the incidence grows exponentially during the initial phase of an epidemic outbreak [1–5]. Within this exponential growth scenario infectious diseases are characterized by the average reproductive number, giving the number of secondary infections generated by a primary case, and the average generation time, giving the average time elapse between the infection of a primary case and its secondary cases [1,2]. In turn, vaccination strategies are designed in order to modify the reproductive number and the generation time [1,2,6,7].

I have recently shown, however, that this picture dramatically changes when the graph underlying the spreading dynamics is characterized by a power law degree distribution [8,9], where the degree of a node is defined as the number of its connections. The significant abundance of high-degree nodes (hubs) carries as a consequence that most nodes are infected in a time scale of the order of the disease generation time. Furthermore, the initial incidence growth is no longer exponential but it follows a power law growth  $n(t) \sim t^{D-1}$ , where  $D$  is the characteristic distance between nodes on the graph. Yet, these predictions are limited to uncorrelated graphs and the susceptible-infected model.

In this work I extend the theory of age-dependent branching processes [4,5,10] to consider the topological properties of real networks. First, I generalize my previous study [8,9] to include degree correlations. This is a fundamental advance since real networks are characterized by degree correlations [11–14] that may significantly affect the system's behavior [15–17]. Second, I consider the susceptible-infected-removed (SIR) model that provides a more realistic description of real epidemic outbreaks [1], allowing us to obtain conclusions about the impact of patient isolation and immunization strategies on the final outbreak size. Finally, I survey our current knowledge about different networks underlying

the spreading of infectious diseases and computer malwares and discuss the impact of their topology on the spreading dynamics.

## II. POPULATION STRUCTURE

Consider a population of  $N$  susceptible agents (humans, computers, etc.) and an infectious disease (human disease, computer malware, etc.) spreading among them. The potential disease transmission channels are represented by an undirected graph, where nodes represent susceptible agents and edges represent disease transmission channels. For example, when analyzing the spreading of sexually transmitted diseases the relevant graph is the web of sexual contacts [18], where nodes represent sexually active individuals and edges represent sexual relationships.

The degree of a node is the number of edges connecting this node to other nodes (neighbors) in the graph. Given the finite size of the population there is a maximum degree  $k_{\max}$ , where  $k_{\max}$  is at most  $N-1$ . I denote by  $p_k$  the probability distribution that a node has degree  $k$ . The results obtained in this work are valid for arbitrary degree distributions. Nevertheless, recent studies have shown that several real networks are characterized by the power law degree distribution

$$p_k = \frac{k^{-\gamma}}{\sum_{s=1}^{k_{\max}} s^{-\gamma}} \quad (1)$$

with  $\gamma > 2$  [18–23]. Therefore, I focus the discussion on this particular case.

Real networks are characterized by degree correlations between connected nodes as well. Networks representing technological and biological systems exhibit disassortative (negative) correlations with a tendency to have connections between nodes with dissimilar degrees [11,13]. In contrast, social networks are characterized by assortative (positive) degree correlations with a tendency to have connections among nodes with similar degrees [12]. To characterize the

degree correlations I consider the probability distribution  $q(k'|k)$  that a neighbor of a node with degree  $k$  has degree  $k'$ . It is important to note that the probability distributions  $p_k$  and  $q(k'|k)$  are related to each other by the detailed balance condition [24]

$$kp_k q(k'|k) = k' p_{k'} q(k|k'). \quad (2)$$

Although  $q(k'|k)$  contains all the information necessary to characterize the degree correlations it is difficult to analyze. A more intuitive measure which often appears in the analytical calculations [15,16] is the average neighbor excess degree [11]

$$K_k = \sum_{k'=2}^{\infty} q(k'|k)(k' - 1). \quad (3)$$

The empirical data indicate that [11,23,25,26]

$$K_k = ck^\nu, \quad (4)$$

where  $c$  is obtained from the detailed balance condition (2), resulting in

$$c = \frac{\langle k(k-1) \rangle}{\langle k^{1+k} \rangle}. \quad (5)$$

When the degree correlations are disassortative the nearest neighbors of a low- (high-) degree node tend to have larger (smaller) degree. In this case  $K_k$  decreases with increasing  $k$ . In contrast, when the degree correlations are assortative the nearest neighbors of a low/high degree node tend to have proportional degrees. In this case  $K_k$  increases with increasing  $k$ . Therefore, disassortative and assortative correlations are characterized by  $\nu < 0$  and  $\nu > 0$ , respectively.

Real networks also exhibit the small-world property [27], meaning that the average distance  $D$  between two nodes in the graph is small or it grows at most as  $\log N$ . For instance, social experiments such as the Bacon and Erdős numbers [28] or the Milgram experiment [29] reveal that social actors are separated by a small number of acquaintances. This property is enhanced on graphs with a power law degree distribution (1) with  $2 < \gamma \leq 3$  [30–32]. In this case the average distance between two nodes grows as  $\log \log N$ , receiving the name of ultrasmall world [32].

Given the graph underlying the spreading of an infectious disease, let us consider an epidemic outbreak starting from a single node (the root, patient zero, or index case). In the worst case scenario the disease propagates to all the nodes that could be reached from the root using the graph connections. Thus, the outbreak is represented by a spanning or causal tree from the root to all reachable nodes. The generation of a node on the tree corresponds with the topological or hopping distance from the root. This picture motivates the introduction of the following branching process:

*Definition 1. Annealed spanning tree with degree correlations.* Consider a graph with degree probability distribution  $p_k$  and average degree  $\langle k \rangle$ , neighbor degree distribution  $q(k'|k)$  given a node with degree  $k$ , detailed balance condition (2), and average distance between nodes  $D$ . The annealed spanning tree (AST) associated with this graph is the

branching process satisfying the following properties.

(1) The process starts from a single node, the root, at generation  $d=0$ . The root generates  $k$  sons with probability distribution  $p_k$ .

(2) Each son at generation  $1 \leq d < D$  generates  $k' - 1$  other sons with probability distribution  $q(k'|k)$ , given its parent node has degree  $k$ .

(3) A son at generation  $d=D$  does not generate new sons.

The term ‘‘annealed’’ means that we are not analyzing the true (quenched) spanning tree on the graph but a branching process with similar statistical properties. From the mathematical point of view the AST is a generalization of the Galton-Watson branching process [10] to the case where (i) the reproductive number of a node depends on the reproductive number of its ancestor and (ii) the process is truncated at generation  $D$ . This mathematical construction has been previously introduced to analyze the percolation properties of graphs with degree correlations [12].

The sharp truncation of the branching process at generation  $D$  is an approximation. In the original graph there are some nodes beyond the average distance between nodes  $D$  and their average degree decreases with increasing generation. Therefore, a more realistic description is obtained defining  $q(k'|k)$  as generation dependent [33,34] and truncating the branching process when the number of generations equals the graph diameter. Yet an analytical treatment of this more realistic model is either unfeasible or results in equations that must be solved numerically, casting doubts on its advantage with respect to direct simulations on the original graph. To allow for an analytical understanding I truncate the branching process at generation  $d=D$ , where  $D$  represents the average distance between nodes  $D$  in the original graph. Furthermore, I assume that  $q(k'|k)$  is the same for all generations  $0 \leq d \leq D$ . At this point it is worth noticing that all results derived below are exact for the AST but an approximation for the original graph.

### III. SIR MODEL OF DISEASE SPREADING

The AST describes the case where all neighbors of an infected node are infected and at the same time. More generally a node infects a fraction of its neighbors and these infections take place at variable times. The susceptible  $\rightarrow$  infected  $\rightarrow$  removed model is an appropriate framework to consider the timing of the infection events [1]. The time scales for the transitions susceptible  $\rightarrow$  infected and infected  $\rightarrow$  removed are characterized by the distribution function of infection and removal times  $G_I(\tau)$  and  $G_R(\tau)$ , respectively. For example,  $G_I(\tau)$  is the probability that the infection time is less than or equal to  $\tau$ .

Consider an infected node  $i$  and a susceptible neighbor  $j$ . The probability  $b(t)$  that  $j$  is infected by time  $t$  given  $i$  was infected at time zero is the combination of two factors. First, the infection time should be smaller than  $t$  and, second, the removal time of the ancestor  $i$  should be larger than the infection time. More precisely

$$b(t) = \int_0^t dG_I(\tau)[1 - G_R(\tau)]. \quad (6)$$

From  $b(t)$  I obtain the probability that  $j$  gets infected no matter at what time

$$r = \lim_{t \rightarrow \infty} b(t) \quad (7)$$

and the distribution function of the generation times

$$G(\tau) = \frac{1}{r} b(\tau). \quad (8)$$

In the original Kermack-McKendrick formulation of the SIR model [35] the disease spreads at a rate  $\lambda$  from infected to susceptible nodes and infected nodes are removed at rate  $\mu$ . In this case the infection and removal rates  $\lambda$  and  $\mu$  are exponentially distributed,  $G_I(\tau) = 1 - e^{-\lambda\tau}$  and  $G_R(\tau) = 1 - e^{-\mu\tau}$ , resulting in

$$r_{\text{SIR}} = \frac{\lambda}{\mu + \lambda}, \quad (9)$$

$$G_{\text{SIR}}(\tau) = 1 - e^{-(\mu+\lambda)\tau}. \quad (10)$$

Some of the results obtained in this work are valid for any generation time distribution. We focus, however, on the SIR model with constant rate of infection and removal (9), (10).

At this point we can extend the AST definition to account for the variable infection times.

*Definition 2. Age-dependent AST with degree correlations.* The age-dependent AST is an AST where nodes can be in two states, susceptible or infected, and (1) an infected node (primary case) infects each of its neighbors (secondary cases) with probability  $r$ ; (2) The generation times, the times elapsed from the infection of a primary case to the infection of a secondary case, are independent random variables with probability distribution  $G(\tau)$ .

The age-dependent AST is a generalization of the Bellman-Harris [10] and Crum-Mode-Jagers [4,5] age-dependent branching processes. The key new elements are the degree correlations and the truncation at a maximum generation, allowing us to consider the topological properties of real networks.

#### IV. SPREADING DYNAMICS AND FINAL OUTBREAK SIZE

Let  $I(t)dt$  be the average number of nodes that are infected between times  $t$  and  $t+dt$  given that patient zero (the root) was infected at time zero. This magnitude is known in the epidemiology literature as the incidence [1]. Consider an age-dependent AST and a constant infection and removal rate (9), (10). Making use of the tree structure I obtain (see the Appendix )

$$I(t) = \sum_{d=1}^D z_d \left( \lambda \frac{(\lambda t)^{d-1}}{(d-1)!} e^{-(\lambda+\mu)t} \right), \quad (11)$$

where

$$z_d = \begin{cases} \langle k \rangle, & d=1, \\ \sum_{k=1}^{\infty} p_k k(k-1) K_k^{d-2}, & d>1, \end{cases} \quad (12)$$

is the average number of nodes  $z_d$  at generation  $d$ , satisfying the normalization condition

$$1 + \sum_{d=1}^D z_d = N. \quad (13)$$

The interpretation of (11) is the following. If we count the time in units of 1 then on average  $z_d$  new nodes are found at each generation. Since the infection times are variable, however, nodes at the same generation may be infected at different times. This contribution is taken into account by the factor between large parentheses in (11), giving the probability density of the sum of  $d$  generation times.

Independent of the particular  $d$  dependence of  $z_d$ , from (11) it follows that the incidence decays exponentially for long times with a decay time  $1/(\lambda+\mu)$ . This result is a consequence of the population finite size, i.e., sooner or later most of the nodes are infected and the number of new infections decays. In contrast, the factor remaining after excluding the exponential decay is an increasing function of time and it dominates the spreading dynamics at short and intermediate times. I obtain the following result determining the speed of the initial growth.

*Theorem 1.* Consider the normalized incidence

$$\rho(t) = \frac{I(t)}{N}. \quad (14)$$

If there is some integer  $d_c \leq D$  and real numbers  $a$  and  $b > 0$  such that for all  $d > d_c$

$$\langle p_k k(k-1) K_k^{d-2} \rangle \sim k_{\text{max}}^{a+b(d-2)} \quad (15)$$

when  $k_{\text{max}} \rightarrow \infty$  then

$$\rho(t) = \lambda \frac{(\lambda t)^{D-1}}{(D-1)!} e^{-(\lambda+\mu)t} \left[ 1 + O\left(\frac{t_0}{t}\right) \right], \quad (16)$$

where

$$t_0 = \frac{1}{\lambda} \frac{D-1}{k_{\text{max}}^b}. \quad (17)$$

The symbol  $O(t_0/t)$  indicates that (16) is valid asymptotically when  $t \gg t_0$  and it represents correction terms of the order of  $t_0/t$ . The demonstration of this result is straightforward. From (15) it follows that for all  $d > d_c$  the average number of nodes at generation  $d$  (12) is of the order of  $z_d \sim k_{\text{max}}^{a+b(d-2)}$ . Therefore, in the limit  $k_{\text{max}} \rightarrow \infty$  the sums in (11) and (13) are dominated by the  $d=D$  term and corrections are given by the ratio between the  $d=D-1$  and  $d=D$  terms.

The initial dynamics is characterized by a power law growth with an exponent determined by the average distance  $D$ . The characteristic time  $t_0$  marks the time scale when this polynomial growth starts to be manifested. This time is particularly small for graphs with a large maximum degree and satisfying the small world property, i.e.,  $D$  is small. For instance, let us consider a power law degree distribution (1) with  $\gamma > 2$  and degree correlations (4). The values of  $\gamma$  and  $\nu$  for which the condition (15) is satisfied are given in Fig. 1, together with the exponent  $b$ . Disassortative degree correlations ( $\nu < 0$ ) may invalidate the condition (15), indicating that strong disassortative correlations may lead to deviations from the Theorem 1 prediction. This observation is in agree-

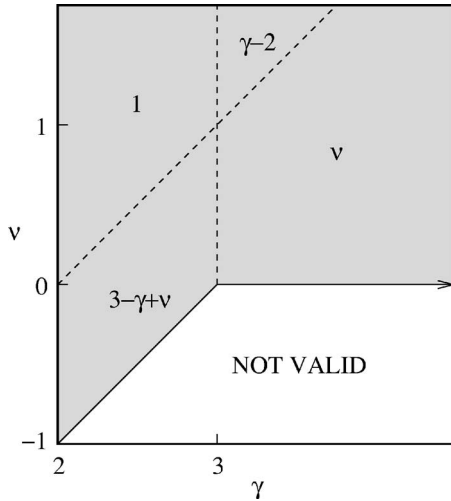


FIG. 1.  $\gamma$ - $\nu$  plane showing the regions where theorem 1 is valid (shaded region) for the case of a power law degree distribution (1) and degree correlations (4). The labels indicate the exponent  $b$  in (15).

ment with a previous study focusing on the epidemic threshold [16]. In contrast, for assortative degree correlations ( $\nu > 0$ ) the condition (15) is satisfied for all  $\gamma > 2$ . In other words, assortative correlations enhance the degree fluctuations, extending the validity of Theorem 1 to the  $\gamma > 3$  region.

Focusing on the final size of the outbreak I obtain the following corollary.

*Corollary 1.* Consider the average total number of infected nodes

$$N_I = N \int_0^\infty dt \rho(t). \quad (18)$$

If the conditions of Theorem 1 are satisfied then

$$N_I = N \left( \frac{\lambda}{\lambda + \mu} \right)^D \left[ 1 + O\left( \frac{(\lambda + \mu)t_0}{D-1} \right) \right]. \quad (19)$$

From this corollary it follows that by increasing the rate of node removal, because of patient isolation or immunization, we just obtain a gradual decrease in the final outbreak size. This implies that the concept of epidemic threshold loses sense since the outbreak size remains proportional to the population size for all removal rates. This conclusion is in agreement with previous studies for the cases  $\gamma, \nu = 0$  [12,36,37] and  $2 < \gamma \leq 3, \nu$  [15,16]. The above corollary extends these studies to the region  $\gamma > 3$ , demonstrating that when  $\nu > 0$  there is not an epidemic thresholds for any value of  $\gamma$ .

## V. DISCUSSION

Theorem 1 proposes a different law of spreading dynamics characterized by an initial power law growth. In essence the power law growth is a consequence of the small-world property and the divergence of the average reproductive number. Its origin is better understood by analyzing the con-

tribution of nodes at a distance  $d$  from the root. The distribution of infection times of nodes at generation  $d$  is given by the distribution of the sum of  $d$  generation times. For the case of a constant infection rate this distribution is a gamma distribution, which is characterized by an initial power law with exponent  $d-1$ . This is the standard result for stochastic processes defined by a sequence of  $d$  steps happening at a constant rate. The total incidence is then obtained by superimposing the contribution of each generation  $d$ , weighted by the average number of nodes at that generation. Since most nodes are found at generation  $d=D$  then the contribution from that generation dominates the incidence progression, resulting in a power law growth with exponent  $D-1$ . The small-world properties simply implies that  $D$  is small and the resulting power law growth can be distinguished from an exponential growth. The validity of this regime is restricted to time scales that are large enough such that an appreciable number of nodes at generation  $d$  are infected, and it is followed by an exponential decay after most nodes at that generation are infected.

To understand the relevance of this spreading law for real epidemic outbreaks, in the following I analyze the validity of the conditions of Theorem 1 for real networks underlying the spreading of human infectious diseases and computer malwares.

*Sexually transmitted diseases.* Sexual contacts are a dominant transmission mechanism of the HIV virus causing AIDS. There are several reports indicating that the web of sexual contacts is characterized by a power degree distribution. The current debate is if the exponent  $\gamma$  is smaller or larger than 3 [18,38–40]. In either case, it is known that social networks are characterized by assortative degree correlations [12], which extends the validity of Theorem 1 to  $\gamma > 3$  (see Fig. 1). There is also empirical evidence indicating that the number of AIDS infections grows as a power law in time for several populations [1,41,42]. When this empirical evidence is put together with that for sexual networks we obtain a strong indication that Theorem 1 provides the right explanation for the observed power law growth.

*Airborne diseases.* Airborne diseases require contact or proximity between two individuals for their transmission. In this case the graph edges represent potential contact or proximity interactions among humans and the degree of an individual is given by the number of people with who he/she interacts within a certain period of time. Recent simulations of the Portland urban dynamics [43] shows that the number of people an individual contacts within a day follows a wide distribution up to about 10 000 contacts. A report for the 2002–2003 SARS epidemic shows a wide distribution as well, in this case for the number of secondary cases generated by a primary SARS infection case. Although these data are not sufficient to make a definitive conclusion, it provides a clear indication that the number of proximity contacts a human undergo within a day is widely distributed. This observation together with the high degree of assortativity of social networks opens the possibility that the spread of airborne diseases within a city is described by Theorem 1.

*Computer malwares.* Email worms and other computer malwares such as computer viruses and hoaxes spread through email communications. The email network is actu-



ally directed, i.e., the observation that user  $A$  has user  $B$  on his/her address book does not imply the opposite. This is an important distinction since the detailed balance condition (2) is valid for graphs with undirected edges. There is, however, a significant reciprocity, meaning that if user  $A$  has user  $B$  on his/her address book then with high probability the opposite takes place as well. Thus, in a first approximation we can represent email connections by undirected links or edges and, in such a case, the detailed balance condition (2) holds. Recent studies of the email network structure within university environments indicate that they are characterized by a power law degree distribution with  $\gamma \approx 2$  [21,22]. Therefore, the spreading of computer malwares represents another scenario for the application of Theorem 1. Further research is required to determine the influence of the lack of reciprocity among some email users.

In conclusion, Theorem 1 characterizes the spreading dynamics on complex networks with wide connectivity fluctuations. Its Corollary 1 determines the region of connectivity fluctuations and degree correlation where there is not an epidemic threshold. The empirical data indicates that the Theorem conditions are satisfied for several networks underlying the spreading of infectious diseases among humans and computer malwares among computers. Therefore, I predict that Theorem 1 is a spreading law of modern epidemic outbreaks.

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#### APPENDIX: ITERATIVE APPROACH

Let  $P_n^{(d,k)}(t)$  be the probability distribution of the number of infected nodes at time  $t$  (including those that have been recovered),  $n$ , on a branch of the AST of Definition 1, given that the branch is rooted at a node at generation  $d$  and its has degree  $k$ . In particular  $P_n^{(0,k)}(t)$  is the probability distribution of the total number of infected nodes at time  $t$ , given that patient zero (the root of the tree) became infected at time zero and its has degree  $k$ . Based on the tree structure we can develop an iterative approach to compute  $P_n^{(d,k)}(t)$  recursively.

*Proposition 1.* Let  $i$  be a node at generation  $d$  with degree  $k$  and let us denote by  $j$  its neighbors on the next generation  $d+1$ , where  $j \in \{1, \dots, k\}$  if  $d=0$ ,  $j \in \{1, \dots, k-1\}$  if  $0 < d < D$ , and  $j \in \{\emptyset\}$  if  $d=D$ . Then

$$P_n^{(0,k)}(t) = \sum_{n_1=0}^{\infty} \cdots \sum_{n_k=0}^{\infty} \delta \sum_{i=1}^k n_{i+1,n} \prod_{i=1}^k \sum_{k'=2}^{k_{\max}} q(k'|k) \times \left( r \int_0^t dG(\tau) P_{n_i}^{(d+1,k')}(\tau) + \delta_{n_i,0} \{1 - r - r[1 - G(t)]\} \right), \quad (\text{A1})$$

$$P_n^{(d,k)}(t) = \sum_{n_1=0}^{\infty} \cdots \sum_{n_{k-1}=0}^{\infty} \delta \sum_{i=1}^{k-1} n_{i+1,n} \prod_{i=1}^{k-1} \sum_{k'=1}^{k_{\max}} q(k'|k) \times \left( r \int_0^t dG(\tau) P_{n_i}^{(d+1,k')}(\tau) + \delta_{n_i,0} \{1 - r - r[1 - G(t)]\} \right), \quad (\text{A2})$$

$$P_n^{(D,k)}(t) = \delta_{n,1}. \quad (\text{A3})$$

*Proof.* Let  $n_i$  be the number of infected nodes in the branch rooted at node  $i$ , and  $n_j$  the number of infected nodes in the branches rooted at each of its neighbors  $j$ . Then

$$n_i = 1 + \sum_j n_j. \quad (\text{A4})$$

Since node  $i$  and its neighbors lie on a tree then  $n_j$  are uncorrelated random variables. Furthermore, all nodes at generation  $d$  have the same statistical properties, i.e.,  $n_j$  are identically distributed random variables. Let  $Q_n^{(d+1,k)}(t)$  be the probability distribution of  $n_j$ , given node  $j$  is at generation  $d+1$  and its ancestor has degree  $k > 0$ . With probability  $1 - r$  the node  $j$  is not infected at any time and with probability  $1 - G(t)$  it is not yet infected at time  $t$  given it will be infected at some later time. Thus

$$Q_0^{(d+1,k)}(t) = 1 - r + r[1 - G(t)]. \quad (\text{A5})$$

On the other hand, with probability  $r$  node  $j$  will be infected at some time  $\tau$ , with distribution function  $G(\tau)$ , and continue the spreading dynamics to subsequent generations. Once node  $j$  is infected, the number of infected nodes in the branch rooted at node  $j$  is a random variable with probability distribution  $P_n^{(d+1,k')}(\tau)$ , given node  $j$  has degree  $k'$ . Therefore,

$$Q_n^{(d+1,k)}(t) = r \sum_{k'} q(k'|k) \int_0^t dG(\tau) P_n^{(d+1,k')}(\tau) \quad (\text{A6})$$

for  $n > 0$ . From Eqs. (A4)–(A6) we finally obtain Eqs. (A1)–(A3). ■

Let  $I(t)dt$  be the average number of nodes that are infected between times  $t$  and  $t+dt$  (incidence), i.e.,

$$I(t) = \frac{d}{dt} \sum_{k=0}^{\infty} p_k \sum_{N=0}^{\infty} P_n^{(0,k)}(t) n. \quad (\text{A7})$$

Using the recursive relations for  $P_n^{(d,k)}(t)$  (A1)–(A3) we obtain the following result.

*Proposition 2.*

$$I(t) = \sum_{d=1}^D r^d z_d \frac{dG^{\star d}(t)}{dt}, \quad (\text{A8})$$

$$G^{*d}(t) = \int_0^t dG(\tau_1) \int_0^{\tau_1} dG(\tau_2) \cdots \int_0^{\tau_{d-1}} dG(\tau_d) \quad (\text{A9})$$

is the  $d$ -order convolution of  $G(\tau)$ , giving the probability distribution function of the sum of  $d$  generation times.

*Proof.* To obtain  $n(t)$  we are going to make use of the generation function

$$F^{(d,k)}(x,t) = \sum_{n=0}^{\infty} P_n^{(d,k)}(t)x^n. \quad (\text{A10})$$

Using the recursive equations (A1)–(A3) for  $P_n^{(d,k)}(t)$  we obtain

$$F^{(0,k)}(x,t) = x \left( 1 - r + r[1 - G(t)] + r \sum_{k'=1}^{k_{\max}} q(k'|k) \int_0^t dG(\tau) F^{(1,k')}(x,t-\tau) \right)^k, \quad (\text{A11})$$

$$F^{(d,k)}(x,t) = x \left( 1 - r + r[1 - G(t)] + r \sum_{k'=1}^{k_{\max}} q(k'|k) \int_0^t dG(\tau) F^{(d+1,k')}(x,t-\tau) \right)^{k-1}, \quad (\text{A12})$$

$$F^{(0,k)}(x,t) = x. \quad (\text{A13})$$

We denote by

$$M^{(d,k)}(t) = \frac{\partial F^{(d,k)}(1,t)}{\partial x} \quad (\text{A14})$$

the mean number of infected nodes on the branch rooted at a node at layer  $d$  with degree  $k$ . Making use of the recursive relations (A11)–(A13) we obtain

$$M^{(0,k)}(t) = 1 + (1-r) \int_0^t dG(\tau) M^{(1,k)}(t-\tau), \quad (\text{A15})$$

$$M^{(d,k)}(t) = 1 + (1-r) \int_0^t dG(\tau) M^{(d+1,k)}(t-\tau), \quad (\text{A16})$$

$$M^{(D,k)}(t) = 1. \quad (\text{A17})$$

Iterating the recursive relations (A15)–(A17) from  $d=D$  to  $d=0$  we obtain

$$M^{(0,k)}(t) = 1 + \sum_{d=1}^D r^d G^{*d}(t) k \sum_{k_1} q(k_1|k) (k_1-1) \sum_{k_2} q(k_2|k_1) \times (k_2-1) \cdots \sum_{k_{d-1}} q(k_{d-1}|k_{d-2}) (k_{d-1}-1). \quad (\text{A18})$$

Note that from (A10) and (A14) it follows that

$$\sum_{n=1}^{\infty} P_n^{(0,k)}(t) n = M^{(0,k)}(t). \quad (\text{A19})$$

Substituting (A18) into (A19) and the result into (A7) we obtain (A8) with

$$z_d = \sum_k p_k k \sum_{k_1} q(k_1|k) (k_1-1) \sum_{k_2} q(k_2|k_1) (k_2-1) \cdots \times \sum_{k_{d-1}} q(k_{d-1}|k_{d-2}) (k_{d-1}-1). \quad (\text{A20})$$

Finally, using the detailed balance condition (2) we reduce (A20) to (12). ■

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- [1] R. M. Anderson and R. M. May, *Infectious Diseases of Humans* (Oxford University Press, New York, 1991).
- [2] O. Diekmann and J. Heesterbeek, *Mathematical Epidemiology of Infectious Diseases: Model Building, Analysis and Interpretation* (John Wiley & Sons, New York, 2000).
- [3] M. Barthélemy, A. Barrat, R. Pastor-Satorras, and A. Vespignani, Phys. Rev. Lett. **92**, 178701 (2004).
- [4] P. Jagers, *Branching Processes with Biological Applications* (Wiley, London, 1975).
- [5] C. J. Mode and C. K. Sleeman, *Stochastic Processes in Epidemiology* (World Scientific, Singapore, 2000).
- [6] N. M. Ferguson *et al.*, Nature (London) **425**, 681 (2003).
- [7] C. Fraser, S. Riley, R. Anderson, and N. M. Ferguson, Proc. Natl. Acad. Sci. U.S.A. **101**, 6146 (2004).
- [8] A. Vazquez, DIMACS Series in Discrete Mathematics and Theoretical Computer Science, **70**, 163 (2006).
- [9] A. Vazquez, Phys. Rev. Lett. **96**, 038702 (2006).
- [10] T. E. Harris, *The Theory of Branching Processes* (Springer-Verlag, Berlin, 2002).
- [11] R. Pastor-Satorras, A. Vazquez, and A. Vespignani, Phys. Rev. Lett. **87**, 258701 (2001).
- [12] M. E. J. Newman, Phys. Rev. Lett. **89**, 208701 (2002).
- [13] S. Maslov and K. Sneppen, Science **296**, 910 (2002).
- [14] P. L. Krapivsky and S. Redner, Phys. Rev. E **63**, 066123 (2001).
- [15] M. Bogaña, R. Pastor-Satorras, and A. Vespignani, Phys. Rev. Lett. **90**, 028701 (2003).
- [16] A. Vazquez and Y. Moreno, Phys. Rev. E **67**, 015101(R) (2003).
- [17] A. Vazquez and M. Weigt, Phys. Rev. E **67**, 027101(R) (2003).
- [18] F. Liljeros, C. R. Edling, L. A. N. Amaral, H. E. Stanley, and Y. Berg, Nature (London) **411**, 907 (2001).
- [19] A.-L. Barabási and R. Albert, Science **286**, 509 (1999).
- [20] M. Faloutsos, P. Faloutsos, and C. Faloutsos, Comput. Commun. Rev. **29**, 251 (1999).
- [21] H. Ebel, L.-I. Mielsch, and S. Bornholdt, Phys. Rev. E **66**,

- R35103 (2002).
- [22] J.-P. Eckmann, E. Moses, and D. Sergi, Proc. Natl. Acad. Sci. U.S.A. **101**, 14333 (2004).
- [23] A. Barrat, M. Barthelemy, R. Pastor-Satorras, and A. Vespignani, Proc. Natl. Acad. Sci. U.S.A. **101**, 3747 (2004).
- [24] M. Boguñá and R. Pastor-Satorras, Phys. Rev. E **68**, 036112 (2003).
- [25] A. Vazquez, Phys. Rev. E **67**, 056104 (2003).
- [26] S. Batiston and M. Catanzaro, Eur. Phys. J. B **38**, 345 (2004).
- [27] D. J. Watts and S. H. Strogatz, Nature (London) **393**, 440 (1998).
- [28] D. J. Watts, *Small Worlds: The Dynamics of Networks between Order and Randomness* (Princeton University Press, Princeton, NJ, 1999).
- [29] S. Milgram, Psychol. Today **2**, 60 (1967).
- [30] F. Chung and L. Lu, Proc. Natl. Acad. Sci. U.S.A. **99**, 15879 (2002).
- [31] B. Bollobás and O. M. Riordan, in *Handbook of Graphs and Networks: From the Genome to the Internet*, edited by S. Bornholdt and H. G. Schuster (Wiley-VCH, Weinheim, 2003), pp. 1–34.
- [32] R. Cohen and S. Havlin, Phys. Rev. Lett. **90**, 058701 (2003).
- [33] R. Cohen, D. Dolev, S. Havlin, T. Kalisky, O. Mokryn, and Y. Shavitt, e-print cond-mat/0305582.
- [34] P. Echenique, J. Gomez-Gardenes, Y. Moreno, and A. Vazquez, Phys. Rev. E **71**, 035102 (2005).
- [35] W. O. Kermack and A. G. McKendrick, Proc. R. Soc. London, Ser. A **115**, 700 (1927).
- [36] R. Pastor-Satorras and A. Vespignani, Phys. Rev. Lett. **86**, 3200 (2001).
- [37] C. Moore and M. E. J. Newman, Phys. Rev. E **61**, 5678 (2000).
- [38] J. H. Jones and M. S. Handcock, Nature (London) **423**, 605 (2003).
- [39] J. H. Jones and M. S. Handcock, Proc. R. Soc. London, Ser. B **270**, 1123 (2003).
- [40] A. Schneeberger, C. H. Mercer, S. A. Gregson, N. M. Ferguson, C. A. Nyamukapa, R. M. Anderson, A. M. Johnson, and G. P. Garnett, Sex Transm. Dis. **31**, 380 (2004).
- [41] R. Brookmeyer and M. H. Gail, *AIDS Epidemiology: A Quantitative Approach* (Oxford University Press, New York, 1994).
- [42] B. Szendrői and G. Csányi, Proc. R. Soc. London, Ser. B **271**, S364 (2004).
- [43] S. Eubank, H. Guclu, V. S. A. Kumar, M. Marathe, A. Srinivasan, Z. Toroczcai, and N. Wang, Nature (London) **429**, 180 (2004).