8 Epidemic Spreading in Complex Networks with Degree Correlations

Marián Boguñá[1], Romualdo Pastor-Satorras[2], and Alessandro Vespignani[3]

[1] Department de Física Fonamental, Universitat de Barcelona, Av. Diagonal 647, 08028 Barcelona, Spain
[2] Department de Física i Enginyeria Nuclear, Universitat Politècnica de Catalunya, Campus Nord, 08034 Barcelona, Spain
[3] Laboratoire de Physique Théorique (UMR du CNRS 8627), Bâtiment 210 Université de Paris-Sud 91405 ORSAY Cedex, France

Abstract. We review the behavior of epidemic spreading on complex networks in which there are explicit correlations among the degrees of connected vertices.

8.1 Introduction

Complex networks arising in the modeling of many social, natural, and technological systems are often growing and self-organizing objects characterized by peculiar topological properties [1, 2]. Many empirical evidences have prompted that most of the times the resulting network's topology exhibits emergent phenomena which cannot be explained by merely extrapolating the local properties of their constituents. Among these phenomena, two of them appear ubiquitous in growing networks. The first one concerns the *small-world* property [3], that is defined by an average path length—average distance between any pair of vertices—increasing very slowly (usually logarithmically) with the network size, N. The second one finds its manifestation in the *scale-free* (SF) degree distribution [1]. This implies that the probability $P(k)$ that a vertex has degree k—it is connected to k other vertices—is characterized by a power-law behavior $P(k) \sim k^{-\gamma}$, where $2 < \gamma \leq 3$ is a characteristic exponent.

The statistical physics approach [1, 2] has been proved a very valuable tool for the understanding and modeling of these emergent phenomena in growing networks and has stimulated a more detailed topological characterization of several social and technological networks. In particular, it has been recognized that many of these networks possess non-trivial degree correlations [4, 5, 6, 7]. The use of statistical physics tools has also evidentiated several surprising results concerning dynamical processes taking place on top of complex networks. In particular, the absence of the percolation [8, 9] and epidemic [10, 11, 12, 13, 14, 15] thresholds in uncorrelated scale-free (SF) networks has hit the community because of its potential practical implications. The absence of the percolative threshold, indeed, prompts to an exceptional tolerance to random damages [16]. On the other hand, the lack of any epidemic threshold makes SF networks the ideal media for the propagation of infections, bugs, or unsolicited information [10].

R. Pastor-Satorras, M. Rubi, A. Diaz-Guilera (Eds.): LNP 625, pp. 127–147, 2003.

While the study of uncorrelated complex networks is a fundamental step in the understanding of the physical properties of many systems[17], yet correlations may drastically change the obtained results, as several recent works addressing the effect of correlations in epidemic spreading have shown [18, 19, 20, 21, 22].

Here we want to provide a review of recent results concerning the epidemic spreading in random correlated complex networks. We will consider the susceptible-infected-susceptible (SIS) and susceptible-infected-removed (SIR) models [23, 24, 25] and we will provide an analytical description that includes two vertices degree correlations in the dynamical evolution of the infection prevalence. This will allow us to relate the presence or absence of epidemic threshold to the eigenvalue spectra of certain connectivity matrices of the networks. In particular, in the case of scale-free networks it is possible to show that for the SIS model, a SF degree distribution $P(k) \sim k^{-\gamma}$ with $2 < \gamma \leq 3$ in unstructured networks with any kind of degree correlations is a sufficient condition for a null epidemic threshold in the thermodynamic limit. For the SIR model, the same sufficient condition applies if the minimum possible degree of the graph is $k_{min} \geq 2$. The SIR model with $k_{min} = 1$ has always a null threshold unless the SF behavior is originated only by minimum degree vertices. In other words, under very general conditions, the presence of two-point degree correlations does not alter the extreme weakness of SF networks to epidemic diffusion. The present results are derived from the divergence of the nearest neighbors average degree [4], which stems from the degree detailed balance condition [21], to be satisfied in all physical networks.

8.2 Correlated Complex Networks

In the following we shall consider unstructured undirected networks, in which all vertices within a given degree class can be considered statistically equivalent. Thus our results will not apply to structured networks in which a distance or time ordering can be defined; for instance, when the small-world property is not present [26, 27]. We will consider in particular the subset of undirected *Markovian* random networks [21], that are completely defined by the degree distribution $P(k)$ ant the conditional probability $P(k' \,|\, k)$ that a vertex of degree k is connected to a vertex of degree k'. These two functions can have any form and are assumed to be normalized, i.e.

$$\sum_k P(k) = \sum_{k'} P(k' \,|\, k) = 1. \tag{8.1}$$

The term “Markovian” refers to the fact that, in our approximation, all higher-order correlation functions can be obtained as a combination of the two fundamental functions $P(k)$ and $P(k' \,|\, k)$. In fact, this approximation represents a natural step towards a more complex description of real networks. In this sense, the Erdös-Rényi (ER) model [28] (defined starting from a set of N vertices that are connected in pairs with an independent probability p) can be viewed as the zero-th order approximation, where the average degree is the only fixed parameter. The ER model is thus defined by the ensemble of all possible networks with a given average degree, but completely random at all other respects. The first-order approximation has been recently introduced by realizing that many real-world networked systems possess a more complex degree distribution than that predicted by the ER model (a Poisson distribution [29]). In this approximation the whole degree distribution, $P(k)$, is chosen as a constrain whereas the rest of properties are left at random [30, 31, 32, 17]. Even though this approximation represents a quantitative step forward, it only takes into account local properties and, therefore, it neglects possible correlations among different vertices, correlations that, on the other hand, are present in real networks [4, 7]. Thus, it is quite natural to introduce the second-order approximation as that with a fixed degree distribution, $P(k)$, and a two-point correlation function, $P(k' \mid k)$, but totally random to all other respects. As we will see, in this case the approximation must be carefully made since, due to the two-point correlation constrain, the fundamental functions $P(k)$ and $P(k' \mid k)$ must satisfy a peculiar detailed balance condition.

The degree distribution usually identifies two kinds of networks. A first class, which includes classical models of random graphs [28], is characterized by an exponentially bounded degree distribution. A second one refers to SF networks in which the degree distribution takes the form $P(k) \sim Ck^{-\gamma}$, usually with $2 < \gamma \leq 3$ [1, 2]. In this case the network shows a very high level of degree heterogeneity, signalled by unbounded degree fluctuations. Indeed, the second moment of the degree distribution, $\langle k^2 \rangle$, diverges in the thermodynamic limit $k_c \to \infty$, where k_c is the maximum degree of the network. It is worth recalling that, in growing networks, k_c is related to the network size N as $k_c \sim N^{1/(\gamma-1)}$ [2]. Noticeably, it is the large degree heterogeneity of SF networks that is at the origin of their extreme weakness towards epidemic spreading.

8.2.1 Assortative and Disassortative Mixing

A direct study of the conditional probability $P(k' \mid k)$ in data from real networks usually yields results that are very noisy and difficult to interpret. In order to characterize the degree correlations, it is more useful to work with the average nearest neighbors degree (ANND) of the vertices of degree k [4], defined by

$$\bar{k}_{nn}(k) \equiv \sum_{k'} k' P(k' \mid k), \tag{8.2}$$

and to plot it as a function of the degree k. When two-point correlations are not present in the network, the conditional probability takes the form $P^{nc}(k' \mid k) = k' P(k') / \langle k \rangle$, and the ANND reads $\bar{k}^{nc}_{nn}(k) = \langle k^2 \rangle / \langle k \rangle$, which is independent on k. On the contrary, an explicit dependence of $\bar{k}_{nn}(k)$ on k necessary implies the existence of non-trivial correlations, as often measured in real networks [4, 7]. For instance, in many social networks it is observed that vertices with high degree connect more preferably to highly connected vertices; a property referred to as "assortative mixing". In this case, $\bar{k}_{nn}(k)$ is an increasing function of k. On the opposite side, many technological and biological networks show "disassortative mixing"; i.e. highly connected vertices are preferably connected to vertices with low degree and, consequently, $\bar{k}_{nn}(k)$ is a decreasing function of k. Then, the ANND provides an easy and powerful way to quantify two-point degree correlations, avoiding the fine details contained in the full conditional probability $P(k' \mid k)$.

8.2.2 Degree Detailed Balance Condition

A key relation holding for all physical networks is that all edges must point from one vertex to another. This rather obvious observation turns out to have important implications since it forces the fundamental functions $P(k)$ and $P(k' \mid k)$ to satisfy the following degree detailed balance condition [21]

$$kP(k' \mid k)P(k) = k'P(k \mid k')P(k'). \tag{8.3}$$

This condition states that the total number of edges pointing from vertices with degree k to vertices of degree k' must be equal to the total number of edges that point from vertices with degree k' to vertices of degree k. This relation is extremely important since it constraints the possible form of the conditional probability $P(k' \mid k)$ once $P(k)$ is given. It may be surprising that such a detailed balance condition exists since, in fact, networks are the result of a multiplicative random process and, in principle, detailed balance conditions only holds for systems driven by additive noise [33]. In fact, the usual detailed balance condition is the same as (8.3) without the prefactors k and k'. These very prefactors account for the multiplicative character of the network and (8.3) can be viewed as a closure condition that guarantees the existence of the network. There is a simple way to derive this condition. Let N_k be the number of vertices with degree k. Obviously, $\sum_k N_k = N$ and, consequently, we can define the degree distribution as

$$P(k) = \frac{N_k}{N}. \tag{8.4}$$

The function $P(k)$ alone does not define completely the topology of the network, because it says nothing about how vertices are connected to each other. Thus, we need to define additionally the *matrix of connections* among vertices of different degrees. Let $N_{k,k'}$ be a symmetric matrix measuring the total number of edges between vertices of degree k and vertices of degree k', when $k \neq k'$, and two times the number of self-connections, when $k = k'$. It is not difficult to realize that this matrix fulfills the identities

$$\sum_{k'} N_{k,k'} = kN_k. \tag{8.5}$$

$$\sum_{k} \sum_{k'} N_{k,k'} = \langle k \rangle N. \tag{8.6}$$

The first of this relations simply states that the number of edges emanating from all vertices of degree k is kN_k, while the second indicates that the sum of all the vertices's degrees is equal to two times the number of edges. The identity (8.6) allows us to define the joint probability

$$P(k,k') = \frac{N_{k,k'}}{\langle k \rangle N}, \tag{8.7}$$

where the symmetric function $(2 - \delta_{k,k'})P(k,k')$ is the probability that a randomly chosen edge connects two vertices of degrees k and k'. The correlation coefficient computed from this joint probability has been recently used in [7] in order to quantify two-point degree correlations. The transition probability $P(k' \,|\, k)$, defined as the probability that an edge from a k vertex points to a k' vertex, can be easily written as

$$P(k' \,|\, k) = \frac{N_{k',k}}{kN_k} \equiv \frac{\langle k \rangle \, P(k,k')}{kP(k)}, \tag{8.8}$$

from where the detailed balance condition arises as a consequence of the symmetry of $P(k,k')$ (or $N_{k,k'}$).

From the degree detailed balance condition it is possible to derive some general exact results concerning the behavior of $\bar{k}_{nn}(k,k_c)$ and of $\left\langle \bar{k}_{nn} \right\rangle_{k_c} = \sum_k P(k)\bar{k}_{nn}(k,k_c)$ in SF networks [22]. In these two functions we have now made explicit the k_c dependence originated by the upper cut-off of the k sum and that must be taken into account since it is a possible source of divergences in the thermodynamic limit. The results that we will derive will turn out to be fundamental in determining the epidemic spreading behavior in these networks. Let us start by multiplying by a k factor both terms of (8.3) and summing over k' and k. We obtain

$$\left\langle k^2 \right\rangle = \sum_{k'} k' P(k') \sum_{k} kP(k \,|\, k'), \tag{8.9}$$

In SF networks with $2 < \gamma < 3$ we have that the second moment of the degree distribution diverges as $\left\langle k^2 \right\rangle \sim k_c^{3-\gamma}$ [4]. We thus obtain from (8.9), using the definition (8.2),

$$\sum_{k'} k' P(k') \bar{k}_{nn}(k',k_c) \simeq \frac{C}{(3-\gamma)} k_c^{3-\gamma}, \tag{8.10}$$

---

[4] For $\gamma = 3$ the second moment diverges as $\left\langle k^2 \right\rangle \sim \ln k_c$ but the argument, though more involved, is still valid.

where C is the constant prefactor from the degree distribution. In the case of disassortative mixing [7], the function $\bar{k}_{nn}(k', k_c)$ is decreasing with k' and, since $k'P(k')$ is an integrable function, the l.h.s. of (8.10) has no divergence related to the sum over k'. This implies that the divergence must be contained in the k_c dependence of $\bar{k}_{nn}(k', k_c)$. In other words, the function $\bar{k}_{nn}(k', k_c) \to \infty$ for $k_c \to \infty$ in a non-zero measure set. In the case of assortative mixing, $\bar{k}_{nn}(k', k_c)$ is an increasing function of k' and, depending on its rate of growth, there may be singularities associated to the sum over k'. Therefore, this case has to be analyzed in detail. Let us assume that the ANND grows as $\bar{k}_{nn}(k', k_c) \simeq \alpha k'^{\beta}$, $\beta > 0$, when $k' \to \infty$. If $\beta < \gamma - 2$, again there is no singularity related to the sum over k' and the previous argument for disassortative mixing holds. When $\gamma - 2 \leq \beta < 1$ there is a singularity coming from the sum over k' of the type $\alpha k_c^{\beta-(\gamma-2)}$. However, since (8.10) comes from an identity, the singularity on the l.h.s. must match both the exponent of k_c and the prefactor on the r.h.s. In the case $\gamma - 2 \leq \beta < 1$, the singularity coming from the sum is not strong enough to match the r.h.s. of (8.10) since $\beta - (\gamma - 2) < 3 - \gamma$. Thus, the function $\bar{k}_{nn}(k', k_c)$ must also diverge when $k_c \to \infty$ in a non-zero measure set. Finally, when $\beta > 1$ the singularity associated to the sum is too strong, forcing the prefactor to scale as $\alpha \simeq r k_c^{1-\beta}$ and the ANND as $\bar{k}_{nn}(k', k_c) \simeq r k_c^{1-\beta} k'^{\beta}$. It is easy to realize that $r \leq 1$, since the ANND cannot be larger than k_c. Plugging the $\bar{k}_{nn}(k', k_c)$ dependence into (8.10) and simplifying common factors, we obtain the identity at the level of prefactors

$$\frac{r}{2 - \gamma + \beta} = \frac{1}{3 - \gamma}. \tag{8.11}$$

Since $\beta > 1$ and $r < 1$, the prefactor in the l.h.s. of (8.11) is smaller than the one of the r.h.s. This fact implies that the tail of the distribution in the l.h.s. of (8.10) cannot account for the whole divergence of its r.h.s. This means that the sum is not the only source of divergences and, therefore, the ANND must diverge at some other point.

In summary, the function $\bar{k}_{nn}(k', k_c)$ must diverge when $k_c \to \infty$ in a non-zero measure set independently of the correlation behavior. The large k_c singularity of the ANND can then be used to evaluate the quantity

$$\left\langle \bar{k}_{nn} \right\rangle_N = \sum_k P(k) \bar{k}_{nn}(k, k_c), \tag{8.12}$$

where we have explicitly considered k_c as a growing function of the network size N. The r.h.s. of this equation is a sum of positive terms and diverges with k_c at least as $\bar{k}_{nn}(k, k_c)$ both in the disassortative or assortative cases. In other words, *all SF networks with* $2 < \gamma \leq 3$ *must present a* $\left\langle \bar{k}_{nn} \right\rangle_N \to \infty$ *for* $N \to \infty$. This statement is independent of the structure of the correlations present in the network. The quantity $\left\langle \bar{k}_{nn} \right\rangle_N$ is particularly useful in model analysis and real data measurements. Degree correlation functions can be measured in several networks, but measurements are always performed in the presence of a finite k_c

that does not allow to exploit the singularity of the function $\bar{k}_{nn}(k, k_c)$. The most convenient way to exploit the infinite size singularity is therefore to measure the $\langle \bar{k}_{nn} \rangle_N$ for increasing network sizes.

The quantity $\langle \bar{k}_{nn} \rangle_N$ is very important in defining the properties of spreading processes in networks since it measures the number of individuals that can be infected in a few contagions. We shall discuss this point in relation to some specific epidemic models in the next sections.

8.3 The SIS Model

As a first prototypical example for examining the properties of epidemic dynamics in SF networks we consider the susceptible-infected-susceptible (SIS) model [23], in which each vertex represents an individual of the population and the edges represent the physical interactions among which the infection propagates. Each individual can be either in a susceptible or infected state. Susceptible individuals become infected with probability λ if at least one of the neighbors is infected. Infected vertices, on the other hand, recover and become susceptible again with probability one. A different recovery probability can be considered by a proper rescaling of λ and the time. This model is conceived for representing endemic infections which do not confer permanent immunity, allowing individuals to go through the stochastic cycle susceptible → infected → susceptible by contracting the infection over and over again.

8.3.1 Uncorrelated Homogeneous Networks

In uncorrelated homogeneous networks, in which each vertex has more or less the same number of connections, $k \simeq \langle k \rangle$, a general result states the existence of a finite epidemic threshold, separating an infected (endemic) phase, with a finite average density of infected individuals, from a healthy phase, in which the infection dies out exponentially fast [25]. This is for instance the case of random networks with exponentially bounded degree distribution.

This result can be recovered by considering the dynamical evolution of the average density of infected individuals $\rho(t)$ (the prevalence) present in the network. The SIS model in homogeneous uncorrelated networks at a mean-field level is described by the following rate equation [11]

$$\frac{d\rho(t)}{dt} = -\rho(t) + \lambda \langle k \rangle \rho(t) \left[1 - \rho(t)\right] . \tag{8.13}$$

In this equation we have neglected higher order terms, since we are interested in the onset of the endemic state, close to the point $\rho(t) \sim 0$. Also, we have neglected correlations among vertices. That is, the probability of infection of a new vertex—the second term in (8.13)—is proportional to the infection rate λ, to the probability that a vertex is healthy, $1-\rho(t)$, and to the probability that a edge in a healthy vertex points to an infected vertex. This last quantity, assuming the *homogeneous mixing hypothesis*[5], is approximated for homogeneous networks as $\langle k \rangle \rho(t)$, i.e. proportional to the average number of connections and to the density of infected individuals. From (8.13) it can be proved the existence of an epidemic threshold $\lambda_c = \langle k \rangle^{-1}$ [25], such that $\rho = 0$ if $\lambda < \lambda_c$, while $\rho \sim (\lambda - \lambda_c)$ if $\lambda \geq \lambda_c$. In this context, it is easy to recognize that the SIS model is a generalization of the *contact process* model, widely studied as the paradigmatic example of an absorbing-state phase transition to a unique absorbing state [34].

8.3.2 Uncorrelated Complex Networks

For general complex networks, in which large degree fluctuations and correlations might be allowed, we must relax the homogeneous hypothesis made in writing (8.13) and work instead with the relative density $\rho_k(t)$ of infected vertices with given degree k; i.e. the probability that a vertex with k edges is infected. Following [10, 11], the rate equation for $\rho_k(t)$ can be written as

$$\frac{d\rho_k(t)}{dt} = -\rho_k(t) + \lambda k \left[1 - \rho_k(t)\right] \Theta_k(t). \tag{8.14}$$

In this case, the creation term is proportional to the spreading rate λ, the density of healthy sites $1 - \rho_k(t)$, the degree k, and the variable $\Theta_k(t)$, that stands for the probability that an edge emanating from a vertex of degree k points to an infected site. In the case of an uncorrelated random network, considered in [10, 11], the probability that a edge points to a vertex with k connections is equal to $kP(k)/\langle k \rangle$ [17]. This yields a $\Theta_k = \Theta^{\mathrm{nc}}$ independent of k that reads as

$$\Theta^{\mathrm{nc}} = \frac{1}{\langle k \rangle} \sum_{k'} k' P(k') \rho_{k'}(t). \tag{8.15}$$

Substituting the expression (8.15) into (8.14), it is possible to find the steady state solution where Θ^{nc} is now a function of λ alone [10, 11] by the following self-consistent equation:

$$\Theta^{\mathrm{nc}} = \frac{1}{\langle k \rangle} \sum_{k} k P(k) \frac{\lambda k \Theta^{\mathrm{nc}}}{1 + \lambda k \Theta^{\mathrm{nc}}}. \tag{8.16}$$

A non-zero stationary prevalence ($\rho_k \neq 0$) is obtained when the r.h.s. and the l.h.s. of (8.16), expressed as a function of Θ^{nc}, cross in the interval $0 < \Theta^{\mathrm{nc}} \leq 1$, allowing a nontrivial solution. It is easy to realize that this corresponds to the inequality

---

[5] The homogeneous mixing hypothesis [23] states that the force of the infection (the *per capita* rate of acquisition of the disease by the susceptible individuals) is proportional to the density of infected individuals.

$$\frac{d}{d\Theta^{\mathrm{nc}}}\left(\frac{1}{\langle k\rangle}\sum_k kP(k)\frac{\lambda k\Theta^{\mathrm{nc}}}{1+\lambda k\Theta^{\mathrm{nc}}}\right)\Bigg|_{\Theta^{\mathrm{nc}}=0} \geq 1 \tag{8.17}$$

being satisfied. The value of λ yielding the equality in (8.17) defines the critical epidemic threshold λ_c, that is given for uncorrelated random networks by

$$\lambda_c^{\mathrm{nc}} = \frac{\langle k\rangle}{\langle k^2\rangle}. \tag{8.18}$$

In uncorrelated and infinite SF networks with $\gamma \leq 3$, we therefore have $\left\langle k^2\right\rangle = \infty$, and correspondingly $\lambda_c^{\mathrm{nc}} = 0$. This is a very relevant result, signalling that the high heterogeneity of SF networks makes them extremely weak with respect to infections. These results have several implications in human and computer virus epidemiology [35].

8.3.3 Correlated Complex Networks

For a general network in which the degrees of the vertices are correlated, the above formalism is not correct, since we are not considering the effect of the degree k into the expression for Θ_k. This effect can be taken into account, however, for Markovian networks, whose correlations are completely defined by the conditional probability $P(k' \,|\, k)$. In this case, it is easy to realize that the correct factor Θ_k can be written as

$$\Theta_k(t) = \sum_{k'} P(k' \,|\, k)\rho_{k'}(t), \tag{8.19}$$

that is, the probability that an edge in a vertex of degree k is pointing to an infected vertex is proportional to the probability that any edge points to a vertex with degree k', times the probability that this vertex is infected, $\rho_{k'}(t)$, averaged over all the vertices connected to the original vertex. Equations (8.14) and (8.19) define together the mean-field equation describing the SIS model on Markovian complex networks,

$$\frac{d\rho_k(t)}{dt} = -\rho_k(t) + \lambda k\left[1-\rho_k(t)\right]\sum_{k'} P(k' \,|\, k)\rho_{k'}(t). \tag{8.20}$$

It must be stressed that this equation is valid only if the network has no structure; i.e. the only relevant variable is the degree k. This implies that all vertices within a given degree class are statistically equivalent. This is not the case, for instance, of regular lattices or structured networks [26, 27] in which a spatial ordering is constraining the connectivity among vertices. The exact solution of (8.20) can be difficult to find, depending on the particular form of $P(k' \,|\, k)$. However, it is possible to extract the value of the epidemic threshold by analyzing the stability of the steady-state solutions. Of course, the healthy state $\rho_k = 0$ is one solution. For small ρ_k, we can linearize (8.20), getting

$$\frac{d\rho_k(t)}{dt} \simeq \sum_{k'} L_{kk'} \rho_{k'}(t). \tag{8.21}$$

In the previous equation we have defined the Jacobian matrix $\mathbf{L} = \{L_{kk'}\}$ by

$$L_{kk'} = -\delta_{kk'} + \lambda k P(k' \,|\, k), \tag{8.22}$$

where $\delta_{kk'}$ is the Kronecker delta symbol. The solution $\rho_k = 0$ will be unstable if there exists at least one positive eigenvalue of the Jacobian matrix $\mathbf{L}$. Let us consider the *connectivity matrix* $\mathbf{C}$, defined by $C_{kk'} = kP(k' \,|\, k)$. Using the symmetry condition (8.3), it is easy to check that if v_k is an eigenvector of $\mathbf{C}$, with eigenvalue Λ, then $P(k)v_k$ is an eigenvector of the transposed matrix $\mathbf{C}^T$ with the same eigenvalue. From here it follows immediately that all the eigenvalues of $\mathbf{C}$ are real. Let Λ_m be the largest eigenvalue of $\mathbf{C}$. Then, the origin will be unstable whenever $-1 + \lambda \Lambda_m > 0$, which defines the epidemic threshold

$$\lambda_c = \frac{1}{\Lambda_m}, \tag{8.23}$$

above which the solution $\rho_k = 0$ is unstable, and another non-zero solution takes over as the actual steady-state—the endemic state.

It is instructive to see how this general formalism recovers previous results [10, 11], implicitly obtained for random uncorrelated networks. For any random network, in which there are no correlations among the degrees of the vertices, we have that the connectivity matrix is given by $C^{\mathrm{nc}}_{kk'} = kP(k'/k) \equiv kk'P(k')/\langle k \rangle$, since the probability that an edge points to a vertex of connectivity k' is proportional to $k'P(k')$. It is easy to check that the matrix $\{C^{\mathrm{nc}}_{k'k}\}$ has a unique eigenvalue $\Lambda^{\mathrm{nc}}_m = \langle k^2 \rangle / \langle k \rangle$, corresponding to the eigenvector $v^{\mathrm{nc}}_k = k$, from where we recover the now established result (8.18).

8.3.4 Correlated Scale-Free Networks

The absence of an epidemic threshold in SF uncorrelated networks is an extremely important question that prompts to a possible weakness of many real-world networks. Particularly important is for the case of digital viruses spreading on the Internet [10, 4] and sexually transmitted diseases diffusing on the web of sexual contacts [36]. Both these networks show, in fact, SF properties that would imply the possibility of major epidemic outbreaks even for infections with a very low transmission rate. Immunization policies as well must be radically changed in the case that a network has a null epidemic threshold [37, 38, 39].

In view of the relevance of this framework, it is extremely important to study to which extent the presence of correlations are altering these results. The main question is therefore which conditions on the degree correlations of SF networks preserve the lack of a critical threshold. In the case of correlated networks, we have shown that the epidemic threshold is the inverse of the largest eigenvalue of the connectivity matrix $\mathbf{C}$. The absence of an epidemic threshold thus corresponds to a divergence of the largest eigenvalue of the connectivity matrix $\mathbf{C}$ in the limit of an infinite network size $N \to \infty$. In order to provide some general statement on the conditions for such a divergence we can make use of the Frobenius theorem for non-negative irreducible matrices [40]. This theorem states the existence of the largest eigenvalue of any non-negative irreducible matrix, eigenvalue which is simple, real, positive, and has a positive eigenvector. In our case the matrix of interest is the connectivity matrix that is non-negative and irreducible. The irreducible property of the connectivity matrix is a simple consequence of the fact that all the degree classes in the network are accessible. That is, starting from the degree class k it is always possible to find a path of edges that connects this class to any other class k' of the network. If this is not the case it means that the network is built up of disconnected irreducible subnetworks and, therefore, we can apply the same line of reasoning to each subnetwork[6]. From the Frobenius theorem [40] it can be proved that the maximum eigenvalue, Λ_m, of any non-negative irreducible matrix, $A_{kk'}$, satisfies the inequality

$$\Lambda_m \geq \min_k \frac{1}{\psi(k)} \sum_{k'} A_{kk'} \psi(k'), \tag{8.24}$$

where $\{\psi(k)\}$ is any positive vector. In particular, by setting $\mathbf{A} = \mathbf{C}^2$ and $\psi(k) = k$ we obtain the inequality

$$\Lambda_m^2 \geq \min_k \sum_{k'} \sum_{\ell} k' \ell P(\ell \,|\, k) P(k' \,|\, \ell). \tag{8.25}$$

This inequality relates the lower bound of the largest eigenvalue Λ_m to the degree correlation function and allows to find a sufficient condition for the absence of the epidemic threshold. By noting that $\sum_{k'} k' P(k' \,|\, \ell) = \bar{k}_{nn}(\ell, k_c)$, we obtain the inequality

$$\Lambda_m^2 \geq \min_k \sum_{\ell} \ell P(\ell \,|\, k) \bar{k}_{nn}(\ell, k_c). \tag{8.26}$$

The r.h.s. of this equation is a sum of positive terms, and by recalling the divergence of the ANND with k_c, we readily obtain that $\Lambda_m \geq \infty$ for all networks with diverging $\langle k^2 \rangle$ both in the disassortative or assortative cases[7]. The divergence of Λ_m implies on its turn that the SIS epidemic threshold vanishes, in the thermodynamic limit, in all SF networks with assortative or disassortative mixing if the degree distribution has a diverging second moment; *i.e. a SF degree*

---

[6] Notice that being irreducible is not equivalent to being fully connected at the vertex to vertex level, but at the class to class level.

[7] One may argue that, since we are calculating a minimum for k, if the transition probability $P(\ell \,|\, k_0)$ is zero at some point k_0, this minimum is zero. In this case it is possible to show that repeating the same argument with $\mathbf{C}^3$ instead of $\mathbf{C}^2$ provides us an inequality that avoids this problem, [21].

distribution with exponent $2 < \gamma \leq 3$ *is a sufficient condition for the absence of an epidemic threshold for the SIS model in unstructured networks with arbitrary two-point degree correlation function.*

In physical terms, the absence of the epidemic threshold is related to the divergence of $\langle \bar{k}_{nn} \rangle_N$ in SF networks. In homogeneous networks, where $\langle \bar{k}_{nn} \rangle_N \simeq \langle k \rangle$, the epidemic spreading properties can be related to the average degree. In SF networks, however, the focus shifts to the possibility of infecting a large number of individuals in a finite number of contagions. The fact that an infected vertex has a very low degree is not very important if a hub of the network that provides connectivity to a large number of vertices is a few hops away. The infection can, in this case, very easily access a very large number of individuals in a short time. In SF networks is the ANND that takes into account more properly the level of degree fluctuations and thus rules the epidemic spreading dynamics.

It is worth stressing that the divergence of $\langle \bar{k}_{nn} \rangle_N$ is ensured by the degree detailed balance condition alone, and it is a very general result holding for all SF networks with $2 < \gamma \leq 3$. On the contrary, the SF behavior with $2 < \gamma \leq 3$ is a sufficient condition for the lack of epidemic threshold only in networks with general two-point degree correlations and in absence of higher-order correlations. The reason is that the relation between the epidemic threshold and the maximum eigenvalue of the connectivity matrix only holds for these classes of networks. Higher order correlations, or the presence of an underlying metric in the network [27], can modify the rate equation at the basis of the SIS model and may invalidate the present discussion.

8.4 The SIR Model

The susceptible-infected-removed model (SIR) [23] represents the other paradigmatic example of epidemic dynamics. Unlike in the SIS model, in this case infected individuals fall, after some random time, into a removed state where they cannot neither become infected again nor infect other individuals. This model tries to mimic real epidemics where individuals, after being infected, acquire permanent immunity or, in the worst case, die.

The SIR model is defined as follows. Individuals can only exist in three different states, namely, susceptible, infected, or removed. Susceptible individuals become infected with probability λ if at least one of their neighbors is infected. On the other hand, infected individuals spontaneously fall in the removed state with probability μ, which without lack of generality we set equal to unity. The main difference between both models is that whereas in the SIS, for $\lambda > \lambda_c$, the epidemics reaches a steady state, in the SIR the epidemics always dies and reaches eventually a state with zero density of infected individuals. The epidemic prevalence is thus defined in this case as the total number of infected individuals in the whole epidemic process.

8.4.1 Uncorrelated Homogeneous Networks

In a homogeneous system, the SIR model can be described in terms of the densities of susceptible, infected, and removed individuals, $S(t)$, $\rho(t)$, and $R(t)$, respectively, as a function of time. These three quantities are linked through the normalization condition

$$S(t) + \rho(t) + R(t) = 1, \tag{8.27}$$

and they obey the following system of differential equations:

$$\begin{aligned} \frac{dS}{dt} &= -\lambda \langle k \rangle \rho S, \\ \frac{d\rho}{dt} &= -\rho + \lambda \langle k \rangle \rho S, \\ \frac{dR}{dt} &= \rho. \end{aligned} \tag{8.28}$$

These equations can be interpreted as follows: infected individuals decay into the removed class at a unity rate, while susceptible individuals become infected at a rate proportional to both the densities of infected and susceptible individuals. Here, λ is the microscopic spreading (infection) rate, and $\langle k \rangle$ is the number of contacts per unit time that is supposed to be constant for the whole population. In writing this last term of the equations we have assumed again, as in the case of the SIS model, the homogeneous mixing hypothesis [23],

The most significant prediction of this model is the presence of a nonzero epidemic threshold λ_c [24]. If the value of λ is above λ_c, $\lambda > \lambda_c$, the disease spreads and infects a finite fraction of the population. On the other hand, when λ is below the threshold, $\lambda < \lambda_c$, the total number of infected individuals (the epidemic prevalence), $R_\infty = \lim_{t\to\infty} R(t)$, is infinitesimally small in the limit of very large populations. In order to see this point, let us consider the set of equations (8.28). Integrating the equation for $S(t)$ with the initial conditions $R(0) = 0$ and $S(0) \simeq 1$ (i.e., assuming $\rho(0) \simeq 0$, a very small initial concentration of infected individuals), we obtain

$$S(t) = e^{-\lambda \langle k \rangle R(t)}. \tag{8.29}$$

Combining this result with the normalization condition (8.27), we observe that the total number of infected individuals R_∞ fulfills the following self-consistent equation:

$$R_\infty = 1 - e^{-\lambda \langle k \rangle R_\infty}. \tag{8.30}$$

While $R_\infty = 0$ is always a solution of this equation, in order to have a nonzero solution the following condition must be fulfilled:

$$\frac{d}{dR_\infty} \left(1 - e^{-\lambda \langle k \rangle R_\infty} \right) \Big|_{R_\infty = 0} \geq 1. \tag{8.31}$$

This condition is equivalent to the constraint $\lambda \geq \lambda_c$, where the epidemic threshold λ_c takes the value $\lambda_c = \langle k \rangle^{-1}$. Performing a Taylor expansion at $\lambda = \lambda_c$ it is then possible to obtain the epidemic prevalence behavior $R_\infty \sim (\lambda - \lambda_c)$ (valid above the epidemic threshold). From the point of view of the physics of non-equilibrium phase transition, it is easy to recognize that the SIR model is a generalization of the *dynamical percolation* model, that has been extensively studied in the context of absorbing-state phase transitions [34].

8.4.2 Uncorrelated Complex Networks

In order to take into account the heterogeneity induced by the presence of vertices with different degree, we consider the time evolution of the magnitudes $\rho_k(t)$, $S_k(t)$, and $R_k(t)$, which are the density of infected, susceptible, and removed vertices of degree k at time t, respectively [14, 15]. These variables are connected by means of the normalization condition

$$\rho_k(t) + S_k(t) + R_k(t) = 1. \tag{8.32}$$

Global quantities such as the epidemic prevalence can be expressed as an average over the various degree classes; for example, we define the total number of removed individuals at time t by $R(t) = \sum_k P(k) R_k(t)$, and the prevalence as $R_\infty = \lim_{t\to\infty} R(t)$. At the mean-field level, for random uncorrelated networks, these densities satisfy the following set of coupled differential equations:

$$\frac{d\rho_k(t)}{dt} = -\rho_k(t) + \lambda k S_k(t) \Theta^{\mathrm{nc}}(t), \tag{8.33}$$

$$\frac{dS_k(t)}{dt} = -\lambda k S_k(t) \Theta^{\mathrm{nc}}(t), \tag{8.34}$$

$$\frac{dR_k(t)}{dt} = \rho_k(t). \tag{8.35}$$

The factor $\Theta^{\mathrm{nc}}(t)$ represents the probability that any given edge points to an infected vertex and is capable of transmitting the disease. This quantity can be computed in a self-consistent way: The probability that an edge points to an infected vertex with degree k' is proportional to $k'P(k')$. However, since the infected vertex under consideration received the disease through a particular edge that cannot be used for transmission anymore (since it points back to a previously infected individual) the correct probability must consider one less edge. Therefore,

$$\Theta^{\mathrm{nc}}(t) = \frac{1}{\langle k \rangle} \sum_k (k-1) P(k) \rho_k(t). \tag{8.36}$$

The equations (8.33), (8.34), (8.35), and (8.36), combined with the initial conditions $R_k(0) = 0$, $\rho_k(0) = \rho_k^0$, and $S_k(0) = 1 - \rho_k^0$, completely define the SIR model on any uncorrelated complex network with degree distribution $P(k)$. We will consider in particular the case of a homogeneous initial distribution of infected individuals, $\rho_k^0 = \rho^0$. In this case, in the limit $\rho^0 \to 0$, we can substitute $\rho_k(0) \simeq 0$ and $S_k(0) \simeq 1$. Under this approximation, (8.34) and (8.35) can be directly integrated, yielding

$$S_k(t) = e^{-\lambda k \phi(t)}, \qquad R_k(t) = \int_0^\infty \rho_k(\tau) dt, \tag{8.37}$$

where we have defined the auxiliary function

$$\phi(t) = \int_0^t \Theta^{\mathrm{nc}}(\tau) d\tau = \frac{1}{\langle k \rangle} \sum_k (k-1) P(k) R_k(t). \tag{8.38}$$

In order to get a closed relation for the total density of infected individuals, it results more convenient to focus on the time evolution of the averaged magnitude $\phi(t)$. To this purpose, let us compute its time derivative:

$$\frac{d\phi(t)}{dt} = 1 - \frac{1}{\langle k \rangle} - \phi(t) - \frac{1}{\langle k \rangle} \sum_k (k-1) P(k) e^{-\lambda k \phi(t)}, \tag{8.39}$$

where we have introduced the time dependence of $S_k(t)$ obtained in (8.37). Once solved (8.39), we can obtain the total epidemic prevalence R_∞ as a function of $\phi_\infty = \lim_{t\to\infty} \phi(t)$. Since $R_k(\infty) = 1 - S_k(\infty)$, we have

$$R_\infty = \sum_k P(k) \left(1 - e^{-\lambda k \phi_\infty}\right). \tag{8.40}$$

For a general $P(k)$ distribution, (8.39) cannot be generally solved in a closed form. However, we can still get useful information on the infinite time limit; i.e. at the end of the epidemics. Since we have that $\rho_k(\infty) = 0$, and consequently $\lim_{t\to\infty} \mathrm{d}\phi(t)/\mathrm{d}t = 0$, we obtain from (8.39) the following self-consistent equation for ϕ_∞:

$$\phi_\infty = 1 - \frac{1}{\langle k \rangle} - \frac{1}{\langle k \rangle} \sum_k (k-1) P(k) e^{-\lambda k \phi_\infty}. \tag{8.41}$$

The value $\phi_\infty = 0$ is always a solution. In order to have a non-zero solution, the condition

$$\frac{d}{d\phi_\infty} \left(1 - \frac{1}{\langle k \rangle} - \frac{1}{\langle k \rangle} \sum_k (k-1) P(k) e^{-\lambda k \phi_\infty} \right) \Bigg|_{\phi_\infty = 0} \geq 1 \tag{8.42}$$

must be fulfilled. This relation implies

$$\frac{\lambda}{\langle k \rangle} \sum_k k(k-1) P(k) \geq 1, \tag{8.43}$$

which defines the epidemic threshold

$$\lambda_c^{\mathrm{nc}} = \frac{\langle k \rangle}{\langle k^2 \rangle - \langle k \rangle}, \tag{8.44}$$

below which the epidemic prevalence is null, and above which it attains a finite value. It is interesting to notice that this is precisely the same value found for the percolation threshold in generalized networks [8, 9]. This is hardly surprising since, as it is well known [41], the SIR model can be mapped to a bond percolation process.

8.4.3 Correlated Complex Networks

In order to work out the SIR model in Markovian networks it is easier to consider the rate equations for the quantities $N_k^I(t)$ and $N_k^R(t)$, defined as the number of infected and removed individuals of degree k, present at time t, respectively. From this two quantities we can easily recover the densities $\rho_k(t)$ and $R_k(t)$ as

$$\rho_k(t) = \frac{N_k^I(t)}{N_k}, \qquad R_k(t) = \frac{N_k^R(t)}{N_k}, \qquad S_k(t) = 1 - \rho_k(t) - R_k(t), \tag{8.45}$$

where $N_k = NP(k)$ is the number of vertices with degree k. The rate equations for $N_k^I(t)$ and $N_k^R(t)$ are then given by

$$\frac{dN_k^I(t)}{dt} = -N_k^I(t) + \lambda S_k(t) \Gamma_k(t), \tag{8.46}$$

$$\frac{dN_k^R(t)}{dt} = N_k^I(t), \tag{8.47}$$

where we have defined the function

$$\Gamma_k(t) \equiv \sum_{k'} N_{k'}^I(t)(k'-1)P(k \,|\, k') \tag{8.48}$$

In this case, the creation of new infected individuals—the second term in the r.h.s. of (8.46)—is proportional to the number of infected individuals of degree k', $N_{k'}^I(t)$, the probability that a vertex of degree k is susceptible, $S_k(t)$, and the average number of edges pointing from these infected vertices to vertices of degree k, $(k'-1)P(k \,|\, k')$, all averaged for all the vertices of degree k'. This last term takes into account that one of the edges is not available for transmitting the infection, since it was used to infect the vertex considered. Dividing (8.46) by N_k and making use of the detailed balance condition (8.3) we find the rate equations for the relative densities as

$$\frac{dR_k(t)(t)}{dt} = \rho_k(t), \tag{8.49}$$

$$\frac{d\rho_k(t)}{dt} = -\rho_k(t) + \lambda k S_k(t) \Theta_k(t), \tag{8.50}$$

where, for a Markovian network, the factor $\Theta_k(t)$ takes the form

$$\Theta_k(t) = \sum_{k'} \rho_{k'}(t) \frac{k'-1}{k'} P(k' \mid k) \tag{8.51}$$

Again, it must be stressed that no structure is allowed in the network in order for this equations to represent a valid formulation of the SIR model.

In order to extract information about the epidemic threshold, we proceed similarly to the SIS model, performing a linear stability analysis. For time $t \to 0$, that corresponds to small ρ_k, $R_k \simeq 0$ and $S_k \simeq 1$, (8.50) and (8.51) can be written as

$$\frac{d\rho_k(t)}{dt} \simeq \sum_{k'} \tilde{L}_{kk'} \rho_{k'}(t), \tag{8.52}$$

where the Jacobian matrix $\tilde{\mathbf{L}} = \{\tilde{L}_{kk'}\}$ can be written as

$$\tilde{L}_{kk'} = -\delta_{kk'} + \lambda \frac{k(k'-1)}{k'} P(k' \mid k). \tag{8.53}$$

In order to infect a finite fraction of individuals, we need the solution $\rho_k = 0$ to be unstable, which happens if there is at least one positive eigenvalue of the Jacobian matrix $\tilde{\mathbf{L}}$. Defining the matrix $\tilde{\mathbf{C}} = \{\tilde{C}_{kk'}\}$, with elements

$$\tilde{C}_{kk'} = \frac{k(k'-1)}{k'} P(k' \mid k), \tag{8.54}$$

we know from the Frobenius theorem, since it is positive and provided that it is reducible at the degree class level, that it has a largest eigenvalue $\tilde{\Lambda}_m$ that is real and positive. Thus, the solution $\rho_k = 0$ of (8.52) is stable whenever $-1 + \lambda \tilde{\Lambda}_m < 0$. This relation defines the epidemic threshold for the SIR model in Markovian networks

$$\lambda = \frac{1}{\tilde{\Lambda}_m}. \tag{8.55}$$

In the case of a random uncorrelated network, we have that $\tilde{C}^{\mathrm{nc}}_{kk'} = k(k'-1)P(k')/\langle k \rangle$. It can be easily seen that this matrix has a unique eigenvalue $\tilde{\Lambda}^{\mathrm{nc}}_m = \langle k^2 \rangle / \langle k \rangle - 1$, corresponding to the eigenvalue $\tilde{v}^{\mathrm{nc}}_k = k$, thus recovering the previous result (8.44) obtained for this kind of networks.

The relation between the SIR model and percolation in correlated complex networks can be closed by noticing that the relevant parameter in this last problem is the largest eigenvalue of the matrix $\mathbf{C}^{\mathrm{perc}} = \{C^{\mathrm{perc}}_{kk'}\}$, with elements $C^{\mathrm{perc}}_{kk'} = (k'-1)P(k' \mid k)$, as pointed out in [42]. It is easy to check that if v^{perc}_k is an eigenvector of $\mathbf{C}^{\mathrm{perc}}$ with eigenvalue Λ, then $\tilde{v}_k = k\, v^{\mathrm{perc}}_k$ is an eigenvector of $\tilde{\mathbf{C}}$ with the same eigenvalue. Then, the eigenvalues of $\mathbf{C}^{\mathrm{perc}}$ and $\tilde{\mathbf{C}}$ coincide, yielding in this way the same description and an identical critical point.

8.4.4 Correlated Scale-Free Networks

The discussion of the absence of epidemic threshold of the SIS in SF networks with any sort of degree correlations can be easily extended to the SIR model, taking again advantage of the Frobenius theorem. In this case, in the general inequality given by (8.24), we set $\mathbf{A} = \tilde{\mathbf{C}}^2$ and $\psi(k) = k$, obtaining

$$\tilde{\Lambda}_m^2 \geq \min_k \sum_{k'} \sum_{\ell} (\ell - 1) P(\ell \,|\, k)(k' - 1) P(k' \,|\, \ell). \tag{8.56}$$

Given that $\sum_{k'}(k'-1)P(k' \,|\, \ell) = \bar{k}_{nn}(\ell, k_c) - 1$, the previous inequality reads

$$\tilde{\Lambda}_m^2 \geq \min_k \sum_{\ell} (\ell - 1) P(\ell \,|\, k) \left[\bar{k}_{nn}(\ell, k_c) - 1 \right]. \tag{8.57}$$

As in the case of the SIS model, the divergence of the ANND with k_c in the thermodynamic limit, ensures the divergence of the eigenvalue $\tilde{\Lambda}_m$. Therefore, a SF degree distribution with diverging second moment is a sufficient condition for the absence of an epidemic threshold also for the SIR model if the minimum degree of the network is $k_{min} \geq 2$. The only instance in which we can have an infinite $\bar{k}_{nn}(\ell, k_c)$ with a finite eigenvalue $\tilde{\Lambda}_m$ is when the divergence of $\bar{k}_{nn}$ is accumulated in the degree $k = 1$ and results canceled by the term $\ell - 1$ in (8.57). This situation happens when the SF behavior of the degree distribution is just due to vertices with a single edge that form star-like structures by connecting on a few central vertices. Explicit examples of this situation are provided in [27, 42].

8.5 Conclusions

In this paper we have reviewed the analytical treatment of the epidemic SIS and SIR models in complex networks at different levels of approximation, corresponding to the different levels in which degree correlations can be taken into account. At the zero-th level, in which all the vertices are assumed to have the same degree (homogeneous networks), we observe the presence of an epidemic threshold, separating an active or endemic phase from an inactive or healthy phase, that is inversely proportional to the average degree $\langle k \rangle$. At this level of approximation, both models render the same result, thus showing a high degree of universality. At the first order approximation level, in which vertices are allowed to have a different degree, drawn from a specified degree distribution $P(k)$, but are otherwise random, we obtain epidemic thresholds that are inversely proportional to the degree fluctuations $\langle k^2 \rangle$. The remarkable fact about this result is that the epidemic threshold vanishes for SF networks with characteristic exponent $2 < \gamma \leq 3$ in the limit of an infinitely large network. Finally, in the second order approximation level, in which degree correlations are explicitly controlled by the conditional probability $P(k' \,|\, k)$ that a vertex of degree k is connected to a vertex of degree k', our analysis yields that the epidemic threshold in the SIS and SIR models is inversely proportional to the largest eigenvalue of the connectivity matrices $C_{kk'} = kP(k' \,|\, k)$ and $\tilde{C}_{kk'} = k(k'-1)P(k' \,|\, k)/k'$, respectively. In the case of the SIR model we recover the mapping with percolation at the level of correlations exclusively among nearest neighbor vertices. The analysis of the divergence of the average nearest neighbors degree $\bar{k}_{nn}(k, k_c)$ with the degree cut-off k_c allows us to establish the general result that any SF degree distribution with diverging second moment is a sufficient condition for the vanishing of the epidemic threshold in the SIS model. The same sufficient condition holds in the SIR model with $k_{min} \geq 2$. The SIR model with $k_{min} = 1$ always shows the absence of an epidemic threshold with the exception of the peculiar case in which the divergence of the average nearest neighbor degree is accumulated only on the nodes of minimum degree. These results have extremely important consequences, since they imply that correlations are not able to stop an epidemic outbreak in SF networks, in opposition to previous claims, and indicates that a reduction of epidemic incidence can only be obtained by means of carefully crafted immunization strategies [37, 38, 39], or trivially through finite size effects [43].

Acknowledgments

This work has been partially supported by the European commission FET Open project COSIN IST-2001-33555. R.P.-S. acknowledges financial support from the Ministerio de Ciencia y Tecnología (Spain).

References

1. R. Albert and A.-L. Barabási. Statistical mechanics of complex networks. *Rev. Mod. Phys.* **74**, 47–97 (2002)
2. S.N. Dorogovtsev and J.F.F. Mendes. Evolution of networks. *Adv. Phys.* **51**, 1079–1187 (2002)
3. D.J. Watts and S.H. Strogatz. Collective dynamics of ‘small-world’ networks. *Nature* **393**, 440–442 (1998)
4. R. Pastor-Satorras, A. Vázquez, and A. Vespignani. Dynamical and correlation properties of the Internet. *Phys. Rev. Lett.* **87**, 258701 (2001)
5. A. Vázquez, R. Pastor-Satorras, and A. Vespignani. Large-scale topological and dynamical properties of Internet. *Phys. Rev. E* **65**, 066130 (2002)
6. S. Maslov and K Sneppen. Specificity and stability in topology of protein networks. *Science* **296**, 910–913 (2002)
7. M.E.J. Newman. Assortative mixing in networks. *Phys. Rev. Lett.* **89**, 208701 (2002)
8. D.S. Callaway, M.E.J. Newman, S.H. Strogatz, and D.J. Watts. Network robustness and fragility: percolation on random graphs. *Phys. Rev. Lett.* **85**, 5468–5471 (2000)
9. R. Cohen, K. Erez, D. ben Avraham, and S. Havlin. Resilience of the Internet to random breakdowns. *Phys. Rev. Lett.* **85**, 4626 (2000)

10. R. Pastor-Satorras and A. Vespignani. Epidemic spreading in scale-free networks. *Phys. Rev. Lett.* **86**, 3200–3203 (2001)
11. R. Pastor-Satorras and A. Vespignani. Epidemic dynamics and endemic states in complex networks. *Phys. Rev. E* **63**, 066117 (2001)
12. R.M. May and A.L. Lloyd. Infection dynamics on scale-free networks. *Phys. Rev. E* **64**, 066112 (2001)
13. R. Pastor-Satorras and A. Vespignani. Epidemics and immunization in scale-free networks. In *Handbook of Graphs and Networks: From the Genome to the Internet,* S. Bornholdt and H.G. Schuster, editors, 113–132. Wiley-VCH, Berlin (2002)
14. Y. Moreno, R. Pastor-Satorras, and A. Vespignani. Epidemic outbreaks in complex heterogeneous networks. R*Eur. Phys. J. B* **26**, 521–529 (2002)
15. M.E.J. Newman. Spread of epidemic diseases on networks. *Phys. Rev. E* **64**, 016128 (2002)
16. R.A. Albert, H. Jeong, and A.-L. Barabási. Error and attack tolerance of complex networks. *Nature* **406**, 378–382 (2000)
17. M.E.J. Newman. Random graphs as models of networks. In *Handbook of Graphs and Networks: From the Genome to the Internet,* S. Bornholdt and H.G. Schuster, editors. Wiley-VCH, Berlin (2002)
18. V.M. Eguíluz and K. Klemm. Epidemic threshold in structured scale-free networks. *Phys. Rev. Lett.* **89**, 108701 (2002)
19. C.P. Warren, L.M. Sander, and I.M. Sokolov. Geography in a scale-free network model. *Phys. Rev. E* **66**, 056105 (2002)
20. Ph. Blanchard, Ch.-H. Chang, and T. Krueger. Epidemic thresholds on scale-free graphs: the interplay between exponent and preferential choice, (2002). e-print cond-mat/0207319
21. M. Boguñá and R. Pastor-Satorras. Epidemic spreading in correlated complex networks. *Phys. Rev. E* **66**, 047104 (2002)
22. M. Boguñá, R. Pastor-Satorras, and A. Vespignani. Absence of epidemic threshold in scale-free networks with degree correlations, (2002). e-print cond-mat/0208163
23. R.M. Anderson and R.M. May, *Infectious diseases in humans*, (Oxford University Press, Oxford, 1992)
24. J.D. Murray, *Mathematical biology*, (Springer Verlag, Berlin, 1993). 2nd edition
25. O. Diekmann and J.A.P Heesterbeek, *Mathematical epidemiology of infectious diseases: model building, analysis and interpretation*, (John Wiley & Sons, New York, 2000)
26. K. Klemm and V.M. Eguíluz. Highly clustered scale-free networks. *Phys. Rev. E* **65**, 036123 (2002)
27. Y. Moreno and A.Vázquez. Disease spreading in structured scale-free networks, (2002). e-print cond-mat/0210362
28. P. Erdös and P.Rényi. On random graphs. *Publicationes Mathematicae* **6**, 290–297 (1959)
29. B. Bollobás. Degree sequences of random graphs. *Discrete Math.* **33**, 1–19 (1981)
30. E.A. Bender and E.R. Canfield. The asymptotic number of labeled graphs with given degree distribution. *Journal of Combinatorial Theory A* **24**, 296–307 (1978)
31. M. Molloy and B. Reed. A critical point for random graphs with a given degree sequence. *Random Struct. Algorithms* **6**, 161 (1995)
32. M. Molloy and B. Reed. The size of the giant component of a random graph with a given degree sequence. *Combinatorics, Probab. Comput.* **7**, 295 (1998)
33. N.G. van Kampen, *Stochastic processes in chemistry and physics*, (North Holland, Amsterdam, 1981)

34. J. Marro and R. Dickman, *Nonequilibrium phase transitions in lattice models*, (Cambridge University Press, Cambridge, 1999)
35. A.L. Lloyd and R.M. May. How viruses spread among computers and people. *Science* **292**, 1316–1317 (2001)
36. F. Liljeros, C.R. Edling, L.A.N. Amaral, H.E. Stanley, and Y. Aberg. The web of human sexual contacts. *Nature* **411**, 907–908 (2001)
37. R. Pastor-Satorras and A. Vespignani. Immunization of complex networks. *Phys. Rev. E* **65**, 036104 (2001)
38. Z. Dezsö and A.-L. Barabási. Halting viruses in scale-free networks. *Phys. Rev. E* **65**, 055103(R) (2002)
39. R. Cohen, D. ben-Avraham, and S. Havlin. Efficient immunization of populations and computers, (2002). e-print cond-mat/0209586
40. F.R. Gantmacher, *The theory of matrices*, volume II, (Chelsea Publishing Company, New York, 1974)
41. P. Grassberger. On the critical behavior of the general epidemic process and dynamical percolation. *Math. Biosci.* **63**, 157–172 (1983)
42. A. Vázquez and Y. Moreno. Resilience to damage of graphs with degree correlations, (2002)
43. R. Pastor-Satorras and A. Vespignani. Epidemic dynamics in finite size scale-free networks. *Phys. Rev. E* **65**, 035108 (2002)