Influence of Network Heterogeneity on Chaotic Dynamics of Infectious Diseases

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Abstract: It is well known that the topology of contacts among individuals can sharply influence the persistence of a disease. Much less is known on how the network structure influences the dynamical properties in the important case of seasonal diseases. Aim of this work is to study a periodically forced SIR contact process with demography in host populations modeled through complex networks (of either Erdős-Rényi or scale-free type). We systematically perform one-parameter bifurcation analyses with respect to the strength of seasonality, and find that the epidemiological regime is largely independent of the network class. However, the structure of the host networks does matter. In fact, the heterogeneity of the network degree distribution emerges as a key element in determining the epidemic temporal patterns. In particular, we find that the dynamical complexity is maximal (i.e., chaotic) at intermediate values of the heterogeneity.

Keywords: Chaos, networks, bifurcations, epidemic models, Lyapunov exponent.

1. INTRODUCTION

With no doubts, the discipline of science where the complex network concept has most favorably spread is epidemiology. It is in fact quite spontaneous and does not require much empirical evidence to recognize that individuals cannot be treated "as average" in terms of pathogen transmissions. For example, in sexually transmitted or in childhood diseases – just to limit our focus on human populations – the social behavior of each individual varies her/his risk of infection, thus it can enhance or reduce its role of potential spreader in the population. This is why the extensive use of ODE approaches to epidemics, rooted in the pioneering work by Kermack and McKendrick (1927), is currently facing a deep revision in the light of the emerging complex network paradigm. Particularly studied in this new context is the problem of disease persistence (Pastor-Satorras and Vespignani, 2001a, 2001b), because of its crucial importance for public health policies. However, another undesirable characteristic of epidemiological time-series is their apparently erratic nature. Seasonality is a key component of many human (Grassly and Fraser, 2006) and animal infectious diseases (Altizer et al., 2006) and it is one of the many possible causes of irregular outbreaks observed in field data (see for example Earn et al., 2000). We do not enter the debate about the extent at which the observed dynamics can be sufficiently well explained by deterministic models evolving on a strange attractor. Some researchers in fact propose an interplay of such models with stochasticity (Rohani et al., 2002) while others opted for transient dynamics Bauch and Earn (2003). Rather, here we focus on the effects played by the network structure on the emergence of chaos in simple, yet nonlinear, epidemiological models. We both have analyzed via bifurcation analysis how seasonality can affect the dynamics of infectious diseases in the SIR (Kuznetsov and Piccardi, 1994) and the SIRC model (Casagrandi et al., 2006), both models being based on the hypotheses of well-mixed host populations. In the present study we concentrate on a single contact process – the SIR model with demography – over three different network topologies.

2. EPIDEMIC DYNAMICS ON NETWORKS

We model the population of individuals as a network composed of N nodes. The i-th node has degree k_i, i.e., it is connected by $0 < k_{min} \leq k_i \leq k_{max}$ links to other nodes. The network is characterized by its degree distribution p_k ($k = k_{min}, \ldots, k_{max}$), where $0 \leq p_k \leq 1$ is the fraction of nodes having exactly degree k. We denote by μ and σ, respectively, the mean value and the standard deviation of the degree distribution. Thus μ is the *average degree* of the network.

The epidemiological problem studied here is the simple and traditional, yet seasonally forced, SIR contact process (Anderson and May, 1992). At any time instant t, each node i is in one of the three possible states: Susceptible (or infectable), Infective (and infected), or Recovered (i.e., permanently immune). During a short time interval Δ, an infective node can recover with probability $\gamma\Delta$ while a susceptible node (say i) can become infective with probability $\rho\Delta n_i$, where n_i is the number of infectives among its neighbors. It is particularly important to notice that we also account for hosts' demography. In fact, we assume that each node can die with probability $\eta\Delta$, irrespective of its degree and its current state, while it can give birth to another node with the same probability ($\eta\Delta$). The newborn nodes are attached to the network with rules that are detailed below, but always in a susceptible state. The birth and death rates are assumed to be equal, in

order to simulate populations that exhibit only stochastic fluctuations around the constant value N. As will be discussed in the next section, although the birth-death process underlying the epidemics does not drastically alter the initial number of nodes in the network, it has the power of strongly modifying its degree distribution.

To obtain a model that describes the disease dynamics over the entire network, we follow the approach by Pastor-Satorras and Vespignani (2001a, 2001b) and subdivide the nodes into classes that depend on their current state and their degree. We therefore introduce $2d$ state variables $S_k(t)$ and $I_k(t)$ (where $d = k_{max} - k_{min} + 1$) that describe the fraction of nodes with degree k that are, respectively, in susceptible and infective state at time t. Note that the fraction of nodes in recovered state are simply $R_k(t) = 1 - S_k(t) - I_k(t)$. Letting $\Delta \to 0$, we derive the following set of $2d$ differential equations:

$$\begin{aligned} \dot{S}_k &= \eta - \eta S_k - \beta S_k(k/\mu)\tilde{I}, \\ \dot{I}_k &= \beta S_k(k/\mu)\tilde{I} - (\eta + \gamma) I_k, \end{aligned} \tag{1}$$

where $\beta = \rho\mu$ is the so-called *transmission rate*, while

$$\tilde{I} = \sum_{k=k_{min}}^{k_{max}} \frac{k p_k}{\mu} I_k \tag{2}$$

is the expected proportion of infectives at time t among the neighbors of a node, because $q_k = {}^{kp_k}/_{\mu}$ is the degree distribution of the neighbors (e.g., Boccaletti et al., 2006). The global disease prevalence at time t is therefore given by

$$I(t) = \sum_{k=k_{min}}^{k_{max}} p_k I_k(t). \tag{3}$$

In the simple case of strictly homogeneous network ($p_\mu = 1$, $p_k = 0$ for all $k \neq \mu$), it is straightforward to verify that model (1) reduces to the standard SIR model:

$$\begin{aligned} \dot{S} &= \eta - \eta S - \beta S I, \\ \dot{I} &= \beta S I - (\eta + \gamma) I, \end{aligned} \tag{4}$$

with $R(t) = 1 - S(t) - I(t)$. Provided that β is not too small ($\beta > \eta + \gamma$), system (4) has a unique, strictly positive equilibrium which is globally asymptotically stable. The long-term behavior of the epidemic is therefore trivially constant.

To account for seasonality, we adopt the typical hypothesis formulated in mathematical biosciences (Dietz, 1976) that the transmission rate $\beta = \beta(t)$ of the disease is periodically varying through time, with period equal to 1 year:

$$\beta(t) = \beta_0 \left[1 + \varepsilon \cos(2\pi t)\right]. \tag{5}$$

The two coefficients $\beta_0 > 0$ and $0 \leq \varepsilon \leq 1$ are the the so-called *average transmission rate* and *strength of seasonality*.

The properties of epidemiological ODE models with seasonal transmission rates of type (5) have been extensively studied in the past, particularly the SIR model (Smith, 1983; Aron and Schwartz, 1984; Kuznetsov and Piccardi, 1994), but also variations (e.g., Casagrandi et al., 2006). Bifurcation analyses have shown that, by varying β_0 and ε, periodic solutions with periods multiple of 1 year, as well as chaotic behavior, can easily be obtained. Both regular and irregular oscillations are suited to explain evidences emerging from large sets of available data (Schaffer and Kot, 1985; Olsen et al., 1988).

The basic scenario giving rise to the emergence of chaos in (4)-(5) is the cascade of period-doubling bifurcations. By keeping β_0 fixed, a stable periodic solution with period of 1 year is obtained for sufficiently small ε. By increasing ε, a sequence of bifurcations leads to cycles with period 2, 4, etc., until chaos is reached at a threshold $\varepsilon_{\text{chaos}}$. Although different phenomena can be detected at different values of β_0, the above scenario is the most common in a range of values for β_0 which is reasonable for a number of diseases (Olsen et al., 1988).

3. METHOD OF ANALYSIS

Aim of this work is to study how the appearance of chaos – more technically, the minimum strength of seasonality $\varepsilon_{\text{chaos}}$ leading to chaos – depend on the heterogeneity of network topology. To this end, we therefore need to (i) introduce the different network topologies we work with, (ii) define indices of heterogeneity that can serve to synthesize our results, and (iii) identify a method for inspecting $\varepsilon_{\text{chaos}}$ for various levels of network heterogeneity.

3.1 Networks topologies

In the present study we deal with three different networks topologies. Two of them are the most widely used types of networks in epidemiology, namely (a) the *Erdős-Rényi* and (b) the *scale-free* networks. The third network type (c), *evolved scale-free network*, is uncommon only because the discovery that scale-free networks lose their structure while evolving under birth-death processes is very recent (Moore et al., 2006). The degree distribution of such networks and the method used to construct them are as follows:

(a) The *Erdős-Rényi (ER) network* (1959) is obtained by randomly connecting N nodes with a pre-specified number of links. For this reason, such networks are often, yet improperly, denoted as *random networks*. The degree distribution of an ER network with large N's and average degree μ is given by a Poisson distribution (e.g., Newman, 2003):

$$p_k = \frac{e^{-\mu}\mu^k}{k!}. \tag{6}$$

It can be shown that, under suitable assumptions, degree distributions of type (6) remain unaltered if the population evolves through a birth-death process. A formal proof is available in Moore et al. (2006). Here it suffices to notice that the number of links is preserved. On the one hand, in fact, if a randomly selected node (say i) dies, all its k_i links are removed thus, on average, $\eta\Delta\mu$ links are lost per unit time. On the other hand, however, if the degree of each newborn node is extracted by the same Poisson distribution (6) which characterizes the original network prior the birth event, then on average $\eta\Delta\mu$ links are added per unit time.

(b) *Scale-free networks* (SFn) are highly heterogeneous networks because they have very few nodes (the hubs) linked to many others, but a large number of nodes with scarce connections. In recent years, SFn have