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Impact of commuting on disease persistence in heterogeneous metapopulations

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ABSTRACT

We use a stochastic metapopulation model to study the combined effects of seasonality and spatial heterogeneity on disease persistence. We find a pronounced effect of enhanced persistence associated with strong heterogeneity, intermediate coupling strength and moderate seasonal forcing. Analytic calculations show that this effect is not related to the phase lag between epidemic bursts in different patches, but rather with the linear stability properties of the attractor that describes the steady state of the system in the large population limit.

1. Introduction

Demographic stochasticity due to the probabilistic nature of events such as births, deaths, mating and disease transmission, plays a major role in the dynamics of small populations. Its impact was acknowledged more than fifty years ago by Bartlett (Bartlett, 1957), who introduced the concept of critical community size. Originally defined as the population size above which the expected time to fade out after an epidemic exceeds a certain period, it is usually taken in more general terms as the threshold population for (a given definition of) disease persistence. It became a central concept in epidemiology, much revisited in several attempts to provide less arbitrary definitions and to reconcile theoretical estimates with data (Keeling and Grenfell, 2002; Nåsell, 2004). Threshold levels of host abundance are equally important in ecology, a context in which the idea of the stochastic Allee effect was introduced (Lande, 1998) to represent demographic stochasticity.

The fact that many natural populations experience annual abundance troughs establishes an obvious connection between average population size and extinction probability, on one hand, and seasonality, on the other (Yorke et al., 1979). Indeed, the annual and multiannual incidence patterns of many infectious diseases show that seasonality is a key ingredient in the overall dynamics of these diseases. Despite the mathematical difficulties involved, theoretical studies have therefore tried to take seasonality into account ever since the earliest efforts (Soper, 1929). The complex interplay between seasonal forcing and the system's nonlinearities is nowadays reasonably well understood, setting the stage for the additional layer of complexity that arises from demographic stochasticity (Stone et al., 2007; Conlan and Grenfell, 2007; Mantilla-Beniers et al., 2010).

Another key ingredient for population persistence is spatial structure and heterogeneity. Spatial structure was first addressed using reaction-diffusion equations that successfully modelled the spread of the epizootic in animal borne diseases (Källén et al., 1985). In these models, inspired by physical systems, the interactions are local and the population is distributed on a plane. More recently, developments that explore the role of individual mobility and long range interactions have come up in the form of metapopulation models, where a number of typically weakly interacting units represent well mixed homogeneous population patches (Grenfell and Harwood, 1997; Hanski, 1998; Levins, 1969; Lloyd and May, 1996; Riley, 2007; Balcan et al., 2009). A long standing idea associated with the concept of metapopulation is that persistence is favoured in a fragmented population, provided that movement between patches accompanies spatial dispersion (Bolker and Grenfell, 1995; Hanski, 1999). This idea has recently been shown to be less straightforward than previously thought (Hagenaars et al., 2004; Jesse and Heesterbeek, 2011).

Among many aspects treated in these studies on spatially extended systems, the degree of synchrony of population abundance oscillations has received special attention as it has been considered the main determinant of persistence (Grenfell

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et al., 1995; Blasius et al., 1999; Grassly and Fraser, 2006). With few exceptions associated with chaotic oscillations, it has been found that a small amount of coupling between population patches is enough to induce synchrony (Earn et al., 1998; Viboud et al., 2006; Grenfell et al., 2001; Lloyd and May, 1996). Although they are usually called spatially heterogeneous, the metapopulation models in these studies assume the same, or very similar, parameter values for the different population patches, and we will refer to them as 'uniform', keeping the term 'heterogeneous' for extended systems that include significant parameter variation across different patches. In line with available data for large urban populations (Viboud et al., 2006; Mantilla-Beniers et al., 2010), the synchronized oscillations in uniform systems are moreover found to be in phase between patches, or, in the case of the 2-year cycle typical of measles, in phase opposition. This is in contrast with the results for heterogeneous systems, where synchronous states may correspond to intermediate phase lags (Blasius et al., 1999; Rozhnova et al., 2012).

In this paper we present an extensive computational study of the combined influence of seasonality and heterogeneity on disease persistence. The basic unit of our model, which we will call a 'city', is formed by a number of individuals undergoing well-mixed stochastic infection dynamics whose parameters are specific to that city and may present seasonal variation. The number of individuals that interact in this way may comprise commuters from another city, as well as the residents of that city. Disease persistence is measured over sets of stochastic simulations of the model. We find that it depends in a nonintuitive way both on the level of seasonality and on the magnitude of the flow of commuters, with a pronounced enhancement of persistence induced by strong heterogeneity at intermediate coupling strengths. We also find that the epidemic phase lags generated by city heterogeneity have no significant effect on disease persistence.

For the unforced case, an analytic description of the incidence fluctuations based on van Kampen's expansion was shown to give good quantitative results for moderate system sizes (Rozhnova et al., 2011, 2012). Using this approximation, summarized in the Supplementary Material (SM), it can be seen that this increase in persistence is instead related with the stability properties of the attractor that describes the steady state of the system in the large population limit.

2. Methods

2.1. Model

In this section, we briefly present the metapopulation susceptible-infectious-recovered (SIR) stochastic model introduced in Rozhnova et al. (2011, 2012) to describe several interacting cities, which are population patches where interactions between individuals are taken to be well mixed.

The SIR model consists of three classes of individuals: susceptibles, infected and recovered. We denote their number among the residents of city k by S_k, I_k, R_k, respectively. These numbers change due to birth, death, infection and recovery, which in the stochastic version of the model are taken as stochastic events with certain rates. As usual when working with time scales for which there are no major demographic changes, we assume that the number of individuals that reside in city k, N_k, is fixed, so that S_k and I_k together completely determine the state of city k. The birth/death rate μ is taken to be constant, and infected individuals recover also at a constant rate γ. When a given disease spreads in a city, the rate of infection is proportional to the number of encounters between susceptibles and infected that take place in that city, which in turn, assuming that in the city the population is well mixed, is proportional to the product of the number of susceptibles and the number of infected in that city. Now these numbers should take into account the flow of commuters from and to that city. In the simplest version of the model, we will assume that the coupling between cities 1 and 2 may be described by a single parameter, f, which is the fraction of the number of residents of each class of city 1 (respectively, 2) that are present in city 2 (respectively, 1) at any given time. The parameter f must be interpreted as the overall fraction of time that an individual from one city spends in the other city, averaged over all types of stays with their typical frequencies and durations. In general, f should be taken class and city dependent (see SM), but we will explore here only the simplest case.

The usual SIR rate of infection then becomes, for susceptible residents of city 1 while in city 1,

$$\frac{\beta_1(1-f)S_1[(1-f)I_1+fI_2]}{M_1},$$

where β_1 is a parameter that reflects the urban characteristics of city 1 through the rate of encounters they elicit, and $M_1=(1-f)N_1+fN_2$ is the number of individuals present in city 1 at any given time. The rate of infection of susceptible residents of city 1 while in city 2 will be given by

$$\frac{\beta_2 fS_1[(1-f)I_2+fI_1]}{M_2},$$

with $M_2=(1-f)N_2+fN_1$. Similar expressions hold for the rates of infections taking place in city 2.

Our mechanistic model thus leads us to represent the interaction between population patches as a weighted distribution of their respective forces of infection. Along with other metapopulation models based on a description of the underlying mobility patterns (Keeling and Rohani, 2002; Keeling et al., 2010), it extends the traditional phenomenological modelling of interacting population patches by means of a single coupling parameter (Lloyd and May, 1996), with the important difference that the parameters β_k are allowed to differ from patch to patch, so that spatial heterogeneity does not come from 'patchiness' of the population only.

The parameter β_k may be time dependent to represent seasonal variability of social intercourse, or of other ingredients such as for instance weather conditions that influence the rate of infectious contacts. We will consider a time dependence of the form $\beta_k(t)=\beta_k^0(1+\epsilon\cos 2\pi t)$, where t is the time measured in years and ϵ represents the amplitude of seasonal forcing. More realistic forcing terms that include a representation of school term calendars are commonly found in the literature on childhood infectious diseases (e.g. Keeling and Grenfell (2002)), but we expect the overall picture revealed by varying β_k^0 and ϵ to be largely independent of the particular form of the periodic forcing.

With these assumptions, the stochastic process is governed by the master equation for the time evolution of $P_{\mathbf{n}}(t)$, the probability distribution for finding the system in state $\mathbf{n}$ at time t (van Kampen, 1981):

$$\frac{dP_{\mathbf{n}}(t)}{dt}=\sum_{\mathbf{n}'\neq\mathbf{n}}\sum_{\alpha}[T_\alpha(\mathbf{n}|\mathbf{n}')P_{\mathbf{n}'}(t)-T_\alpha(\mathbf{n}'|\mathbf{n})P_{\mathbf{n}}(t)], \quad (1)$$

where $\mathbf{n}$ denotes the state of the system given by the numbers of infected and susceptibles in each city and $T_\alpha(\mathbf{n}|\mathbf{n}')$, are the (possibly time dependent) transition rates from the state $\mathbf{n}'$ to the state $\mathbf{n}$ that result from the birth–death, recovery and infection processes. These rates are given explicitly in the SM.