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Stochastic amplification and childhood diseases in large geographical areas

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Abstract

We study the spread of childhood infectious diseases in geographically detailed population focussing on stochastic amplification. We use an individual based SIR model with demography where individuals reside in small geographical areas representing a portion of land, and long-distance transmission among different geographical areas occurs due to mobility of individuals. Mobility is implemented using the recently introduced radiation model [Simini et al., Nature 484, 96 (2012)]. Parameterizing the model for measles, we observe that some features of the data available for this disease can be understood within the framework of stochastic amplification, but also that the interplay between mobility and disease dynamics influences the resulting time-series.

Key words: stochastic amplification, human mobility, measles, SIR

1 Introduction

Stochastic fluctuations around the equilibrium for epidemiological models have been extensively studied in recent years [1, 2]. Substantial research has shown that the time series observed for various childhood diseases can be understood in terms of stochastic amplification, that is the increase in amplitude and regularization of the fluctuations around the equilibrium value observed for finite populations in epidemiological models driven by stochasticity. Such studies have shown that the frequency and amplitude observed for recurrent epidemics in available datasets for various infectious diseases could be understood within this framework [2, 3]. The work we present here investigates this idea in a more realistic setup by using realistic geographically detailed populations, where individuals move

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around different geographical areas following a mobility model recently introduced that was shown to reproduce observed human mobility patterns [4]. We show that some of the features observed in real datasets can be understood in terms of stochastic amplification; however we also show that further fine tuning is needed to properly reproduce the observed data.

2 Methods

We perform individual based simulation of a SIR model with demography, in which we assume equal death and birth rates, so that the population size is kept constant. Individual based simulations for this kind of compartmental models show fluctuations of the number of infective individuals around the endemic equilibrium [2]; these fluctuations have a preferred frequency which can be easily determined calculating their power spectrum. Spatial and temporal correlations enhance the coherence and amplitude of such fluctuations [5, 6]. This phenomenon, known as stochastic amplification, has been suggested as an explanation for the incidence patterns observed for some childhood diseases.

Our aim is to study this phenomenon using the detailed geographic distribution of human population provided by the Gridded Population of the World database [7], which provides estimates for the population of a given geographical area on a regular grid of cells of angular size 2.5 arc-minutes. This corresponds roughly to 5 km at the equator. Each of these cells is considered by us as a well mixed population, and disease evolution in described by a simple individual based SIR model. Since the geographical areas we consider include population sizes of the order of tens of millions of individuals, we use parallel computation which grants unlimited complexity. A simulated annealing technique is used to partition the map under consideration into regions that are similar in population size and compact in shape. Each of these regions is then assigned a different node of a computer cluster.

Interactions among cells is introduced by moving individuals among the cells: when an individual leaves a cell, it ceases to participate to the dynamics of that cell, while it starts to participate to the dynamics of the new cell it moves to. Thus, by moving around, individuals can spread the disease from their cell of origin to new cells, leading to long-distance transmission. The way individuals move among different cells reflects the recently introduced radiation model for human mobility described in ref. [4]. This model, which gives the mobility fluxes among a set of locations, is influenced by a single parameter N_c/N which corresponds to the fraction of individuals in the population that participates to such long-distance displacements (N is the total population, N_c is the total number of individuals moving, or *commuters*). This parameter controls the level of long-distance transmission and hence can be estimated by data on human mobility. Following previous studies [8], individuals are assigned a set of preferred locations that they visit with a frequency compatible with the fluxes provided by the radiation model, allowing us to limit

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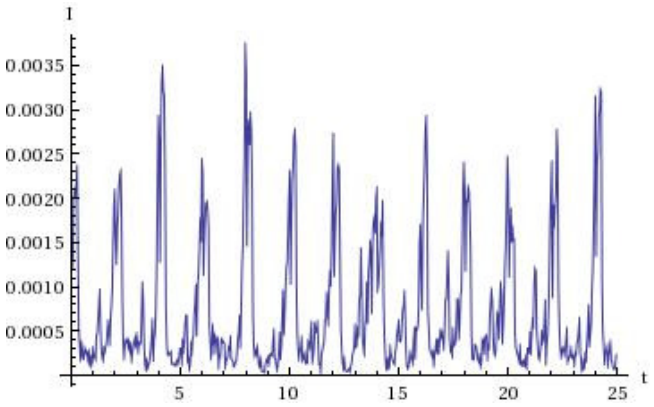

Figure 1: Infective incidence as a function of time for a grid cell in the city of London; time is in years. Simulations parameters: $\beta_0 = 1.175$ days^{-1}, $\beta_1 = 0.35$, $\gamma = 1/13$ days^{-1}, $\mu = 5.5 \times 10^{-5}$ days^{-1}, $N_c/N = 0.1$

the computational requirements of our simulations.

3 Results

We considered parameters corresponding to measles, a disease that received considerable attention [9] and was shown to be compatible with the stochastic amplification mechanism [2]. Since there are available datasets for England, we performed simulations for the geographical area of the British Isles that we assumed to be isolated: no imports from outer regions were considered. We introduced seasonality by modulating the contact rate through term-time forcing:

$$\beta(t) = \beta_0 \left[1 + \beta_1 \ \mathrm{Term}(t)\right]$$

where $\mathrm{Term}(t)$ is a function taking values $+1$ during school terms and -1 otherwise [9].

From the time series of the number of infective individuals for different locations in the area, we calculated the corresponding power spectra which we found to be in agreement with those calculated in previous studies [2]. The time series also show regular biennial cycles (see figure 1) for some location as well as propagation of waves from big centres towards less populated areas. We found however that the regularity of the sequence of peaks observed in the time series for some locations depends on the intensity of human mobility, controlled by the parameter N_c/N. Low populated cities are characterized by extinctions of the disease that must be imported by another location, however if N_c/N is low, such import events might be considerably delayed, leading to a separation among peaks larger than biennial

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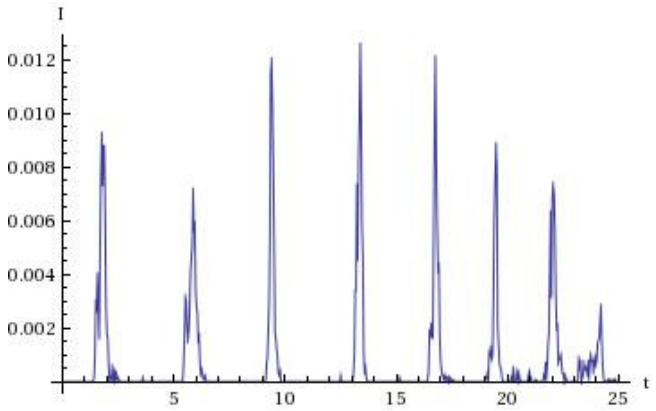

Figure 2: Infective incidence as a function of time for a grid cell in the city of York; time is in years. Due to the low N_c/N, peaks appear temporally more separated than the biennial sequence observed for London. Simulations parameters: same as in figure 1, except $N_c/N = 0.02$

(see figure 2). When N_c/N is high, long-distance transmission becomes efficient, but on the other hand this leads to higher infectiveness and broader peaks.

Although some of the features observed can also be interpreted in terms of broadness of the power spectra, we think that additional features should be considered in order to get a better agreement. An improved description for measles is that obtained using an SEIR model: our first attempts show that this change influences the time series but does not solve the discrepancies observed with data. Instead, we believe that more realistic recovery profiles using multiple infective classes would lead to a higher regularization of the epidemic sequence while reducing inter-epidemic infectivity. Also, contact restriction on individuals a few days after being infectives would also act to reduce the overall infectiveness observed in simulations.

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