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Epidemics with pathogen mutation on small-world networks

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Abstract

We study the dynamical behavior of the epidemiological model with pathogen mutation on small-world networks, and discuss the influence of the immunity duration τ_R, the cross-immunity threshold h_{thr}, and system size N on epidemic dynamics. A decaying oscillation occurs because of the interplay between the immune response and the pathogen mutation. These results have implications for the interpretation of longitudinal epidemiological data on strain abundance, and they will be helpful to assess the threat of highly pathogenic and mutative viruses, such as avian influenza.

Keywords: Small world; Epidemics; Oscillation; Pathogen mutation

1. Introduction

Over the past 8 years, cases of human infection with highly pathogenic and mutative viruses, such as avian influenza, have raised international concern [1]. Mathematical models of viral transmission and control are important tools for assessing the threat of viruses and the best means of containing an outbreak, because they can integrate epidemiological and biological data to give quantitative insights into patterns of disease spread [2]. Examples include the design and evaluation of childhood disease immunization programmes [3,4], predicting the demographic impact of the HIV epidemic in different regions [5], and analyzing the spread and control of the 2001 foot-and-mouth epidemic in Britain [6–9].

Following this philosophy, a lot of mathematical epidemiological models for the spread of disease are researched [10–17]. However, most mathematical models for the spread of disease have not incorporated the pathogen mutation, which is an important issue in epidemiology. The interplay between the memory immune response and pathogen mutation affects epidemic dynamics. Recently, Girvan et al. proposed a simple model of epidemics with pathogen mutation [18]. This model is based on the uniform mixing assumption, but this assumption is unrealistic. In fact, individuals tend to make contact with household members, workplace colleagues, and friends at a much higher rate than with random strangers. Hence, socio-spatial structure has an important impact on transmission dynamics [19]. This kind of socio-spatial structure can be described by small-world networks [19,20]. Behavior of epidemics also shows characteristics of small-world networks [14]. In small-world networks, the nodes represent populations and links mimic the close contact among them.

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Though the small-world networks are not a realistic representation of social networks, small-world networks capture two principal features of real social interactions. Firstly, they are highly clustered, which implies that any two neighbors of a given site have a large probability of being in turn mutual neighbors. Secondly, they show the "small-world" property, i.e., the diameter of the graph (that is the shortest chain of links connecting any two vertices) increases very slowly, in general, logarithmically with the network size [12,20,21].

In this paper, we simulate the dynamical behavior of epidemics with pathogen mutation on small-world networks and investigate the influence of interplay between the memory immune response and pathogen mutation on the epidemic dynamics. We find a decaying oscillatory behavior depending on the immunity duration τ_R and the cross-immunity threshold h_{thr}. These results have implications for the interpretation of longitudinal epidemiological data on strain abundance, and they will be helpful to assess the threat of highly pathogenic and mutative viruses, such as avian influenza.

2. Model and method

2.1. Small-world networks

The system consists of N individuals. These individuals can be regarded as the nodes of a small-world network, and contacts between linked individuals are expressed by links of the small-world network. A one-dimensional small-world network can be established as follows [21]: the starting point is a ring with N nodes, in which each node is symmetrically connected with its $2K$ nearest neighbors. Then, for every node, each link connected to a clockwise neighbor is rewired to a randomly chosen node with probability p, and preserved with probability $1-p$. Self-connections and multiple connections are prohibited, and realizations for which the small-world network becomes disconnected are discarded. As advanced above, the parameter p measures the randomness of the resulting small-world network. Being independent of the value of network randomness p, the average number of links per site $\langle k\rangle$ is always $2K$. In this work, we took the total number of nodes $N=10^5$, the number of average nearest neighbor $\langle k\rangle = 2K = 4$. It has been pointed out that the onset of small-world behavior takes place at the network randomness $p^* \sim 1/N$ in a one-dimensional small-world network [12]. Since we took $N=10^5$, the network randomness should be larger than 10^{-5}. We chose $p=0.01$, well within the small-world regime.

2.2. Epidemiological model with pathogen mutation

In epidemiological models, the individuals are traditionally categorized into three states: susceptible to infection S, infected I, and immune R. Each individual is described by a single dynamical variable adopting one of these three states. A susceptible individual can pass to the infected state through contagion by an infected one. An infected individual passes to the immune state after an infection time τ_I. Similarly, an immune individual returns to the susceptible state after immunity duration τ_R [13,18,22]. This kind of system is usually called SIRS. When the pathogen strain mutates, a new pathogen strain will occur in the system; the individuals immunized to previous pathogens are also the susceptible ones to the new pathogen strain.

Pathogen mutation is modeled by a computational "bitstring model", proposed by Girvan et al., in which different pathogen strains are represented by bitstrings that can mutate [18]. The bitstrings are regarded as an abstract representation of a pathogen's genetic code. Immunity depends explicitly on the history of strains with which the individual has been infected. Individuals are immune to infection by pathogens that are highly similar to the pathogens with which they have been previously infected. Here, similarity between two strains is measured by hamming distance. Hamming distance between two bitstrings is simply the number of bits that differ between them. For example, the hamming distance between 0110001 and 0100011 is 2. If the length of each bitstring is l, the possible number of bitstrings is 2^l. Each individual keeps the record of all the strains with which it has been infected. We refer to the history as the memory repertoire of an individual. After immunity duration τ_R, the record of the corresponding strain is erased.

At the beginning, we randomly choose an individual as the infected one with the bitstring $000\ldots000$ and all others have never been infected (modeling a deliberate release of the mutable virus). Afterwards, at any time step, each infected individual can infect its neighbors. The susceptibility of a contacted individual (susceptible