Spatial patterns in a discrete-time SIS patch model

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Received: 1 December 2007 / Published online: 12 June 2008
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Abstract How do spatial heterogeneity, habitat connectivity, and different movement rates among subpopulations combine to influence the observed spatial patterns of an infectious disease? To find out, we formulated and analyzed a discrete-time SIS patch model. Patch differences in local disease transmission and recovery rates characterize whether patches are low-risk or high-risk, and these differences collectively determine whether the spatial domain, or habitat, is low-risk or high-risk. In low-risk habitats, the disease persists only when the mobility of infected individuals lies below some threshold value, but for high-risk habitats, the disease always persists. When the disease does persist, then there exists an endemic equilibrium (EE) which is unique and positive everywhere. This EE tends to a spatially inhomogeneous disease-free equilibrium (DFE) as the mobility of susceptible individuals tends to zero. The limiting DFE is nonempty on all low-risk patches and it is empty on at least one high-risk patch. Sufficient conditions for the limiting DFE to be empty on other high-risk patches are given in terms of disease transmission and recovery rates, habitat connectivity, and the infected movement rate. These conditions are also illustrated using numerical examples.

Keywords Spatial heterogeneity · Dispersal · Habitat connectivity · Basic reproduction number · Disease-free equilibrium · Endemic equilibrium

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1 Introduction

Recently, the influence of spatial heterogeneity on infectious disease outcomes has been explored for spatially discrete habitats using continuous-time patch models [7, 15, 21] and for spatially continuously habitats using both reaction–diffusion models [22, 27, 28] and integro-differential equation models [25]. One of the main drawbacks of any deterministic continuous-time model is that the multiple processes which combine to influence population dynamics (such as growth, infection, and dispersal) must operate together at all times. However, discrete-time models suffer no such limitation, and it is natural when using them to break time steps up into distinct stages so that each process occurs separately. Such a modeling approach has proved successful in studying the dynamics of many spatially structured populations (e.g., [9, 18, 31, 32]), including those subject to the influence of infectious diseases [5, 10, 11].

In this paper, we model infection and movement among \( n \) patches over discrete time intervals. We assume that susceptible individuals (\( S \)) become infected (and infective, \( I \)) but do not develop immunity. Eventually, any individual will recover, and upon doing so they immediately become susceptible again. Thus, there are two compartments (susceptible and infected) in each patch and the dynamics of this population is described by a system of \( 4n \) difference equations which together constitute our discrete-time SIS epidemic model. We do not consider the effects of population growth and other changes in demography, such as disease-related deaths. In each time step, we assume that infection and recovery occur first (stage 1) and that individuals subsequently disperse among the \( n \) patches in the habitat (stage 2). In the first stage, individuals within a patch either contract the disease or recover from it based solely on local conditions within the patch. This produces an intermediate number of susceptible and infected individuals within each patch, who are then free to move between patches according to susceptible and infected dispersal matrices. These matrices need not have the same structure so long as every patch is accessible from every other patch. Characterizing different patches as low-risk or high-risk, and the habitat as a whole as low-risk or high-risk, allows us to determine various conditions under which the disease can invade or persist, and to describe the different equilibrium patterns that can arise.

In this investigation, we are particularly interested in studying equilibrium patterns of the discrete-time epidemic patch model as the susceptible dispersal rate diminishes to zero. This study therefore complements our prior research on a pair of related continuous-time epidemic models: one a patch model [1] and the other a reaction–diffusion model [2]. Here, we show that the discrete-time formulation of the disease model can exhibit dynamics and equilibrium patterns that are quite similar to those that can occur for its two continuous-time cousins.

When \( A = (a_{jk}) \) is a matrix and \( u = (u_j) \) is a vector, we will use the notation \((Au)_j\) to denote the \( j \)th entry of the vector \( Au \), i.e., \((Au)_j = \sum_k a_{jk}u_k\). Let \( 1_n \) denote the vector \([1, 1, \ldots, 1] \) of \( n \) ones and \( I_n \) the \( n \times n \) identity matrix.