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Complex Dynamics of a host–parasite model with both horizontal and vertical transmissions in a spatial heterogeneous environment

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ABSTRACT

In this paper, we investigate the dynamical outcomes of a host–parasite model incorporating both horizontal and vertical transmissions in a spatial heterogeneous environment analytically and numerically. Our study provides valuable insights in two aspects: Mathematically, we propose three threshold parameters, the demographic reproduction number R_d, the horizontal transmission reproduction number R_0^h and the vertical transmission reproduction number R_0^v, to identify the conditions that lead to disease-free dynamics, or susceptible-free dynamics, or endemic dynamics. Epidemiologically, we find that both host population movements and spatial heterogeneity strongly affect the disease dynamics of our proposed epidemic model: (1) the larger random mobility can result in 100% infection prevalence; and (2) the heterogeneity tends to enhance the persistence of the infected hosts with uninfected ones. As a consequence, our work suggests that, in order to control the invasion of the parasite, different preventive measures can be implemented in different regions.

1. Introduction

The interplays between parasites and their hosts constitute biological processes of major ecological importance that is ubiquitous in nature [1]. It is now widely believed that diseases and parasites are responsible for a number of extinctions of important species on islands and large land masses. As a result, both ecologists and mathematicians acknowledge the importance of disease and parasites in affecting population dynamics of endangered species [2–8].

Recently, an interesting topic in the study of parasite diseases is to understand how parasites regulate host populations by using host–parasite epidemiological models. In modeling a host–parasite system, a

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parasite can be transmitted horizontally or vertically or both. Horizontal transmission is the transmission of infections between members of the same species; and vertical transmission (sometimes called as mother-to-child transmission) is the transmission of a bacteria, viruses, or in rare cases, parasites transmitted directly from the mother to an embryo, fetus, or baby during pregnancy or childbirth. A wide range of pathogens are transmitted by a combination of horizontal and vertical transmission: among these are microsporidians [9], helminths [10], Feline immunodeficiency virus and Feline leukemia virus [11], Human T-cell leukemia virus [12], human immunodeficiency virus [13], human papilloma virus [14], and hepatitis B/C viruses [15,16], etc.

There are a fair amount of mathematical models on studying the host–parasite interactions with mixed vertical and horizontal transmissions [17–20]. Especially, Stewart et al. [21] found that with horizontal transmission, virulence will increase as a byproduct of a trade-off; with vertical transmission, virulence will decrease and allow parasite transmission to more host offspring. Kang and Castillo-Chavez [22] found that under "small" horizontal transmission rates, the model may result in susceptible-free dynamics and with an inefficient reproductive infectious class the model may lead to disease-driven extinction scenarios.

On the other hand, it is well-known that spatial heterogeneity plays an important role in the dynamics of host–parasite interactions [23]. Reaction–diffusion equations have been a useful modeling tool to incorporate the spatial movement of hosts into epidemic models by assuming some types of host random movements [24,25]. There are rich literature on the investigation of the roles of spatial diffusion and spatial heterogeneity in the transmission and control of diseases [26–36].

Based on the discussions above, especially following the insightful work of [7,18,19,22], we will study the following reaction–diffusion host–parasite epidemiological model incorporating a horizontally and vertically transmitted disease in a spatial heterogeneity of environment:

$$\begin{cases} \partial_t S - d_S \Delta S = rS\left(1 - a(S+I)\right) - \mu S - \beta(x) SI, & x \in \Omega,\ t > 0, \\ \partial_t I - d_I \Delta I = r\rho I\left(1 - a(S+I)\right) - (\mu+\delta) I + \beta(x) SI, & x \in \Omega,\ t > 0, \\ \partial_{\mathbf{n}} S = \partial_{\mathbf{n}} I = 0, & x \in \partial\Omega,\ t > 0, \\ S(x,0) = \phi_1(x) \geq 0,\ I(x,0) = \phi_2(x) \geq 0, & x \in \Omega, \end{cases} \tag{1}$$

where $S := S(x,t)$ and $I := I(x,t)$ are susceptible (uninfected) hosts and infected hosts, respectively. The parameters r, a, μ, ρ, δ are all positive constants. r is the birth rate of $S(x,t)$, $\rho r > 0$ the birth rate of $I(x,t)$ and $\rho \in [0,1]$ describes the reduced reproductive ability of $I(x,t)$: $\rho = 0$ means that $I(x,t)$ loses their reproducing ability, in this case, there is no vertical transmission; while $\rho = 1$ indicates that there is no reduction in the reproductive fitness. $1/a$ is the carrying capacity, μ the natural mortality of the hosts, and δ the parasite-induced death rate. The spatial heterogeneity is taken into account via the assumption that the transmission rate $\beta(x)$ is spatially dependent. Specifically, we require that $\beta(\cdot) \in C^1(\bar{\Omega})$ and $\beta(x) \neq 0$. As a note, the so-called homogeneous case refers to the case when β is spatially independent, i.e., $\beta(x)$ is a constant. d_S, d_I are the diffusion coefficients of $S(x,t)$ and $I(x,t)$, respectively, and Δ the Laplacian operator. $\Omega \subset \mathbb{R}^n$ has a smooth boundary $\partial\Omega$ and $\mathbf{n}$ is the outward unit normal vector on $\partial\Omega$. And zero-flux conditions reflect the situation where the population cannot move across the boundary of the domain. We also require that the initial values $\phi_1(x), \phi_2(x) \in C(\bar{\Omega})$.

The main focus of this paper is to study the effect of spatial heterogeneity on the extinction and persistence of infectious disease, and therefore, we will concentrate on the following steady-state system:

$$\begin{cases} -d_S \Delta S = rS\left(1 - a(S+I)\right) - \mu S - \beta(x) SI, & x \in \Omega, \\ -d_I \Delta I = r\rho I\left(1 - a(S+I)\right) - (\mu+\delta) I + \beta(x) SI, & x \in \Omega, \\ \partial_{\mathbf{n}} S = \partial_{\mathbf{n}} I = 0, & x \in \partial\Omega. \end{cases} \tag{2}$$

And the concept of the stability, such as local asymptotical stability and global asymptotical stability, in the rest of this paper, is understood in the sense of the classical textbook (Section 5.1, pages 98–101) [37] and [32,38].