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Competitive exclusion in an infection-age structured model with environmental transmission

Maia Martcheva [a,*], Xue-Zhi Li [b]

[a] Department of Mathematics, University of Florida, 358 Little Hall, PO Box 118105, Gainesville, FL 32611–8105, United States
[b] Department of Mathematics, Xinyang Normal University, Xinyang 464000, China

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ABSTRACT

It has been shown in the past that for the most basic multi-strain ordinary differential equation (ODE) model of SIR-type a competitive exclusion principle holds. The competitive exclusion principle means that the strain with the largest reproduction number persists but eliminates all other strains with suboptimal reproduction numbers. In this paper, we extend the competitive exclusion principle to a multi-strain age-since-infection structured model of SIR/SI-type. We also include environmental transmission for each of the pathogens. The model describes well transmission of avian influenza or cholera. Using a Lyapunov functional, we are able to establish global stability of the disease-free equilibrium if all reproduction numbers are smaller or equal to one. If $\mathcal{R}_j$, the reproduction number of strain j is larger than one, then a single-strain equilibrium, corresponding to strain j exists. This single strain equilibrium is locally stable whenever $\mathcal{R}_j > 1$ and $\mathcal{R}_j$ is the unique maximal reproduction number. If $\mathcal{R}_1 > 1$ is the maximal reproduction number, using a Lyapunov functional, we establish that the corresponding single-strain equilibrium $\mathcal{E}_1$ is globally stable. That is, strain one eliminates all other strains, independently of their reproduction numbers as long as they are smaller than $\mathcal{R}_1$.

1. Introduction

The competitive exclusion principle is a fundamental result in ecology, postulated first by Gause [5]. It states that two species competing for the same resource cannot coexist indefinitely if all other ecological conditions are the same. If one of the species has even a small advantage over the other, that species will dominate and exclude the less advantageous one. Competitive exclusion in ecology is predicted by a number of mathematical models of Lotka–Volterra or chemostat type (see [7] and the references therein). These models have extended the principle to n species. The extended version states that n complete competitors cannot coexist on a single resource. Only the species that can persist on the smallest amount of the resource remain dominant, the others are eliminated.

The competitive exclusion principle has been recast through mathematical models in the context of epidemiology. The first article that derives the epidemiological description of that principle does that through ordinary differential equations (ODEs) [4]. In epidemiology, the competitive exclusion principle states that if multiple strains circulate in the population only the strain with the largest reproduction number persists, the strains with suboptimal reproduction numbers are eliminated. The goal of this article is to extend this principle to age-since-infection structured multi-strain models. The proof of a competitive exclusion principle is based on a proof of global stability of the single-strain equilibrium. In the past, however, most results on age-since-infection models were local and often demonstrated that the age-since-infection structure can

* Corresponding author.
E-mail addresses: maia@ufl.edu, maia@math.ufl.edu (M. Martcheva), xzli66@126.com (X.-Z. Li).

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destabilize the endemic equilibrium and oscillations are possible [22,13,14]. More recently it has been shown that simple age-since-infection structured models with mass action incidence can have a locally stable endemic equilibrium. This has paved the way to establishing global stability of the endemic equilibrium [11,12,21]. We draw on these results to derive the competitive exclusion principle for infinite dimensional systems.

The persistence and the pandemic threat of avian influenza as well as the very publicized cholera outbreak in Haiti have increased the awareness of diseases which transmit both directly and environmentally. Many recent articles have been devoted to indirectly transmitted diseases [23,16,8,24,1]. Few of these articles, however, investigate the role of the environment on the competition of pathogens. Questions of competition in the context of both direct and indirect transmission have been discussed in [3,17]. Responding to these interests, we include both direct and indirect transmission in our age-since-infection structured model. Indirect transmission has been modeled predominantly via bilinear incidence or saturating in the free virus incidence. Our model uses bilinear incidence for the environmental transmission. It appears that if the indirect transmission incidence is saturating, coexistence may occur and complete competitive exclusion cannot be established [10].

Our model was inspired by the model introduced in [3]. Compared to the model in [3] our model includes age-since-infection structure which turns our system into a PDE system. Furthermore, the authors of [3] consider saturating force of infection for environmental transmission, while we consider linear force of infection. Our results are focused on establishing rigorously the local and global stability of a single-strain equilibrium, while [3] derives invasion conditions and studies the interplay between a directly transmitted and both directly and indirectly transmitted strain.

In the next section we introduce a multi-strain, age-since-infection structured, SIR/SI model with direct and environmental transmission. By assuming that the shedding rate into the environment is zero, the model can be used for a number of directly transmitted diseases (such as some childhood diseases). It is also suitable for modeling directly and indirectly transmitted diseases such as cholera, influenza A, and hantavirus. In Section 2 we also introduce the reproduction numbers of each strain $\mathcal{R}_j$ for $j = 1, \ldots, n$ as well as the disease reproduction number. Section 3 is devoted to the endemic equilibria and their local stability. We find that there is a unique disease-free equilibrium and one single-strain equilibrium corresponding to each strain. If $\mathcal{R}_j < 1$ for $j = 1, \ldots, n$, then the disease-free equilibrium is locally stable. If $\mathcal{R}_j > 1$ for some j and $\mathcal{R}_j$ is the unique maximal reproduction number, then the single-strain equilibrium corresponding to strain j is locally asymptotically stable. In Section 4 we construct a Lyapunov functional to show the global stability of the disease-free equilibrium. Section 5 is devoted to the principle of competitive exclusion. We assume without loss of generality that strain one has the maximal reproduction number and $\mathcal{R}_1 > 1$. Under that assumption, we show uniform strong persistence of strain one when the remaining strains become extinct. In Section 6 we again use a Lyapunov functional to derive the global stability of the strain one equilibrium, thus establishing complete competitive exclusion.

2. A multi-strain model with environmental transmission

In this section we introduce a model of disease that is both directly and environmentally transmitted. We assume that the pathogen causing the disease is represented by multiple strains. A good example of such a disease is influenza A. To introduce the model, we denote by $S(t)$ the number of susceptible individuals. We structure the infected individuals by the age-since-infection a. Let $i_j(a, t)$ be the density of individuals infected by strain j. Furthermore, let $R(t)$ be the number of recovered individuals. Finally, let $V_j(t)$ be the number of virions of strain j in the environment. The model takes the form [3]

$$\begin{cases} \dfrac{dS}{dt} = \Lambda - \displaystyle\sum_{j=1}^{n} S \int_0^\infty \beta_j(a) i_j(a,t)\,da - S \sum_{j=1}^{n} \rho_j V_j - \mu S, \\ \dfrac{\partial i_j}{\partial t} + \dfrac{\partial i_j}{\partial a} = -(\mu + \nu_j(a)) i_j(a,t), \\ i_j(0,t) = S \displaystyle\int_0^\infty \beta_j(a) i_j(a,t)\,da + \rho_j S V_j, \quad j = 1, \ldots, n, \\ \dfrac{dR}{dt} = \displaystyle\sum_{j=1}^{n} \int_0^\infty \nu_j(a) i_j(a,t)\,da - \mu R(t), \\ \dfrac{dV_j}{dt} = \displaystyle\int_0^\infty \eta_j(a) i_j(a,t)\,da - \delta_j V_j. \end{cases} \tag{2.1}$$

In model (2.1) Λ is the birth/recruitment rate, $\beta_j(a)$ is the time-since-infection structured transmission rate of strain j, $\nu_j(a)$ is the duration of infectiousness/recovery rate, ρ_j is the transmission rate from the environmental contamination, $\eta_j(a)$ is the age specific shedding rate of individuals infected with strain j, δ_j is the clearance rate of the virus strain j from the environment, and μ is the natural death rate.

To understand the model, notice that susceptible individuals are recruited at a rate Λ. Susceptible individuals can become infected with strain j either through a direct contact with an infected individual with strain j or through coming into contact with viral particles of strain j that are in the environment. Infection through direct contact with infected individuals can happen through contact with individuals of any age-since-infection at a specific age-specific transmission rate. As a