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Emerging disease dynamics in a model coupling within-host and between-host systems

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HIGHLIGHTS

- Studied a model coupling within- and between-host disease dynamics.
- Applied the model results to toxoplasmosis.
- The coupled model can generate new disease dynamics which are absent without coupling.
- The appearance of a backward bifurcation makes it challenging for disease control.

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ABSTRACT

Epidemiological models and immunological models have been studied largely independently. However, the two processes (between- and within-host interactions) occur jointly and models that couple the two processes may generate new biological insights. Particularly, the threshold conditions for disease control may be dramatically different when compared with those generated from the epidemiological or immunological models separately. An example is considered in this paper for an environmentally driven infectious disease such as *Toxoplasma gondii*. The model explicitly couples the within-host and between-host dynamics. The within-host sub-system is linked to a contaminated environment E via an additional term $g(E)$ to account for the increase in the parasite load V within a host due to the continuous ingestion of parasites from the contaminated environment. The parasite load V can also affect the rate of environmental contamination, which directly contributes to the infection rate of hosts for the between-host sub-system. When the two sub-systems are considered in isolation, the dynamics are standard and simple. That is, either the infection-free equilibrium is stable or a unique positive equilibrium is stable depending on the relevant reproduction number being less or greater than 1. However, when the two sub-systems are explicitly coupled, the full system exhibits more complex dynamics including backward bifurcations; that is, multiple positive equilibria exist with one of which being stable even if the reproduction number is less than 1. The biological implications of such bifurcations are illustrated using an example concerning the spread and control of toxoplasmosis.

1. Introduction

Two of the important processes in host–parasite interactions and transmission dynamics of infectious diseases are the immunological process associated with the pathogen–cell interactions at the individual level and the epidemiological process concerning the spread of diseases at the population level. Most modeling studies have focused on these two processes separately. Immunological models consider the within-host dynamics independent of the interactions between hosts (e.g., De Leenheer and Smith, 2003; Nowak et al., 1997; Perelson et al., 1993; Perelson and Nelson, 2002; Rong et al., 2007a–c), while epidemiological models consider the between-host transmissions without an explicit link to the within-host dynamics (e.g., standard SI, SIS, SIR, SEIR types of epidemiological models). In the last couple of decades, some attempts have been made to study models that couple these two processes (e.g., Boldin and Diekmann, 2008; Feng et al., 2013, 2012; Gandolfi et al., 2014; Gilchrist and Coombs, 2006; Gilchrist and Sasaki, 2002; Mideo et al., 2008; Qesmi et al., 2014). Findings from

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