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A model for coupling within-host and between-host dynamics in an infectious disease

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Abstract Studies on the modeling of the coupled dynamics of infectious diseases at both the population level (the epidemic process or between-host dynamics) and at the cell level (the early viremia or within-host dynamics) are scarce. Most of them deal with these two processes separately by postulating assumptions that render them decoupled.

In this work, we present a new model that allows the two dynamic processes to explicitly depend on each other. It is shown that new properties can emerge from the coupled system and more complex dynamics may be expected.

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1 Introduction

For infectious diseases, such as HIV infection, two key processes play important roles in the study of the host-parasite interaction. One is the epidemic process that involves disease transmission between hosts, and the other is the immunological process related to the virus-cell interaction at the level of an individual host. Although an increasing number of mathematical models have been developed to study the transmission dynamics of these diseases, most of them treat these two processes separately. Viral dynamic models (e.g., Anderson and May [1], De Boer and Perelson [4], Nowak and May [9, 10], Perelson et al. [11], Perelson and Nelson [12], Regoes et al. [14], Wodarz [16]) consider the within-host dynamics independent of the interaction at the population level), whereas epidemic models of population dynamics (e.g., Anderson and May [1], Thieme [15], and references therein) consider the interaction between susceptible and infected hosts without an explicit link to the viral dynamics within hosts (by implicitly assuming that all infectious hosts have the same constant viral load, and hence the same infectivity). When the two processes are decoupled, the mathematical models are in general easier to analyze. However, there remain questions that can only be addressed by using models which explicitly