Journal of Nonlinear Modeling and Analysis
Volume 4, Number 2, June 2022, 325–351
http://jnma.ca; http://jnma.ijournal.cn
DOI:10.12150/jnma.2022.325

On an Infection-age Structured Epidemic Model with Multiscale*

Yongle Fu[1], Yunfei Lv[1,†] and Yongzhen Pei[1]

Abstract Considering the individual difference, this paper deals with an infection-age structured epidemic model coupling within-host and between-host for environmentally-driven infectious disease. The full system with two time scales, the cellular level and population level, is first separated into the isolated fast and slow systems. For the isolated fast and slow systems, combined with the within-host and between-host reproduction numbers, R_{w0} and R_{b0}, we give the complete global dynamics by using Lyapunov function respectively. Our results indicate that when there is no virus in environment the disease can be not only controlled, but also eliminated. However, when there is always virus in environment the disease is only controlled but not eliminated. Furthermore, the coupled slow system has complex dynamics with multiple positive equilibria and backward bifurcation. The virus contaminated environment plays a critical role on backward bifurcation. When the initial environmental virus is below some threshold the disease will be eliminated, when it is above the threshold the disease will develop an endemic disease. Some numerical simulations are performed to illustrate these results. The age structured model is more general, and this work includes some previous results.

Keywords Age structure, Coupled system, Basic reproduction number, Stability, Backward bifurcation.

MSC(2010) 37N25, 37B25, 92B05.

1. Introduction

There are many viral infectious diseases among human beings. The common viral infectious diseases include 2019-nCoV, influenza, AIDS, rubella, respiratory virus infection, viral hepatitis, etc. These diseases not only cause huge social and economic losses, but also cause great harm to human health. Based on the dynamical mechanisms of the disease transmission, mathematical model can be established to study the properties of the model solution, That is, the threshold conditions which have been widely used to control and predict the current and future epidemic prevalence.

Infection age, which is the time passed since a host was infected, measures the amount of viruses accumulated in an infected host. Some works on HIV/AIDS

†the corresponding author.
Email address: lvyunfei@tiangong.edu.cn (Y. Lv), 1930112004@tiangong.edu.cn (Y. Fu), yzhpei@tiangong.edu.cn (Y. Pei)
[1]School of Mathematical Sciences, Tiangong University, Tianjin 300387, China
*The authors were supported by National Natural Science Foundation of China (Nos. 11871371, 11971023).

and 2019-nCoV, have found that different infection age leads to significant differences in infection rates and mortality caused by diseases. Age structured infection model can be used to describe the individual difference. For instance, the exposed individuals and infected individual have different transmission rates. Recently, infection age has been introduced into epidemic models to study this phenomenon, for example [1, 4, 12, 16, 24, 25]. Nevertheless, the introduction of the infection age brings about changes to the model from ordinary differential equation to first-order partial differential equation (the well-known age structured model), increasing the difficulty of the study.

However, infectious disease dynamics are dominated by many interconnected scales, from complex within-host infection processes to between hosts and environments. Therefore, the other individual difference we take account in this context is that the disease driven by environment has two time scales: the cellular level and the population level. At the cellular level, the virus infection process within the hosts is called the fast system, which usually takes the form of cell-virus interactions, while at the population level, disease transmission between hosts is a slow process, which refers to the transmission of disease among individuals. Therefore, both questions will be raised, and how does the coupling of virus and individual affect the process of disease transmission? What is the impact of infection-age structure on disease dynamics?

The dynamic behavior of the within- and between-host is often considered separately, but it is found that the establishment of the coupled model will have new insights. For Toxoplasma gondii, authors in [3,21] proposed a coupled cell virus model and SI epidemic model, where the virus in polluted environment plays a major and determinant role in transmission of Toxoplasma infectious disease. Backward bifurcation may occur in [3, 21] when the basic reproduction number is less than 1, in which stable disease-free equilibrium and endemic equilibrium can coexist. In such case, the disease can persist and be hard to control. This means the basic reproduction number will not be sufficient to described whether the disease is endemic or not, and the initial values should be paid attention to. Based on the coupling models of [3, 21], authors in [7] considered the disease-induced mortality, obtained the similar dynamic behavior and studied the evolution of virulence. The other works described virus replication and their respective immune responses while disease transmission is represented by the SI model [2], the dynamics of cholera within and between hosts [25] as well as the effects of within-host and population-level dynamics on malaria transmission dynamics [1]. Furthermore, infectious disease models with time-varying parameters and general nonlinear incidence rates have been analyzed in [12]. Authors in [13, 26] considered the impacts of *Wolbachia* on the mosquito-borne diseases in a heterogeneous environment.

The assumptions in the above articles are made on the basis of homogeneity without considering individual differences. That is, the infection age is not taken into account, especially for the coupled within- and between-host model. Authors in [20] derived a stage-structured epidemic model from an age structured model, while did not discuss the age structured model. Recently, the model of [7] has been extended in [14], where the mortality was considered as the function of the infection age. However, it did not consider the infection age of cells in the host. As stated in the pieces of literature [5, 6, 10, 11, 17–19, 27], the infection age of viral diseases is of great important. Therefore, we will introduce the infection age into the coupled within- and between-host models, which is more in line with the biological