Physica A 514 (2019) 729–740

Contents lists available at ScienceDirect

Physica A

journal homepage: www.elsevier.com/locate/physa

Transmission dynamics of Zika virus with spatial structure—A case study in Rio de Janeiro, Brazil

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HIGHLIGHTS

- A Zika virus (ZIKV) model with spatial heterogeneity is developed.
- The threshold dynamics of the ZIKV model are given.
- The spatial distribution and final size of the ZIKV are strongly dependent on the location when ZIKV appears in a region.
- It is useful to understand the influences of spatial heterogeneity on the dynamics of ZIKV.

ARTICLE INFO

Article history:
Received 26 July 2018
Received in revised form 3 September 2018
Available online xxxx

Keywords:
Zika virus (ZIKV)
Spatial heterogeneity
Basic reproduction number
Infection-free equilibrium
Uniformly persistent

ABSTRACT

In this paper, we investigate the effect of the spatial heterogeneity on the extinction and persistence of the Zika virus (ZIKV) further. We define the basic reproduction number $\mathcal{R}_0$ and prove that $\mathcal{R}_0$ can be used to govern the threshold dynamics of the ZIKV: if $\mathcal{R}_0 < 1$, the unique disease-free equilibrium is globally asymptotic stable and the ZIKV will die out, while if $\mathcal{R}_0 > 1$, there is at least one endemic equilibrium and the ZIKV will persist uniformly. Via numerical simulations, we show the evolution of the spatial distribution of infected people and find that the final size of the infected people is 25,717, which agrees nearly with the real weekly reported case data 25,400 from November 1, 2015 to April 10, 2016 in Rio de Janeiro, Brazil.

1. Introduction

Zika virus (ZIKV) spreads mostly by the bite of an infected *Aedes* species mosquito, and can be passed from a pregnant woman to her fetus [1]. In 2016, the World Health Organization reported that local circulation of the ZIKV has been reported by 72 countries and territories, and the most common symptoms of ZIKV are headache, muscle and joint pain, mild fever, rash, and inflammation of the underside of the eyelid [2]. Unfortunately, there is no vaccine or medicine for ZIKV [1]. Therefore, the ongoing ZIKV epidemic poses a major global public health emergency [3–12].

It is well-known that the mathematical model has become a crucial tool in analyzing the prevention and control measures for infectious diseases qualitatively and quantitatively. The research results are helpful to predict the developing tendency of infectious diseases, to determine the key factors of the spread of infectious disease and to seek the optimum strategies of preventing and controlling the spread of infectious diseases [13–19]. There are an amount of studies show that spatial diffusion and environmental heterogeneity can decide the speed and pattern of the spread of infectious diseases and therefore, it is essential to investigate the role of diffusion in the transmission and control of diseases in a heterogeneous environment [20–37].

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https://doi.org/10.1016/j.physa.2018.09.100
0378-4371/

Table 1
Biological interpretations for parameters in model (1.1).

Parameter	Definition
$\Lambda(X)$	Geographic density of the background population of uninfected and susceptible hosts in Ω
$\lambda(X)$	The loss rate of the infected host population (due to recovery or other removal)
$\beta(X)$	The breeding rate of the vector population
$\mu(X)$	The loss rate of the vector population due to environmental crowding
$\sigma_1(X)$	The transmission rate of infected hosts
$\sigma_2(X)$	The transmission rate of vectors
$\delta_1(X)$	The diffusion rate of the infected hosts
$\delta_2(X)$	The diffusion rate of the vectors

In an effort to understand how spatial heterogeneity of the vector and host populations influence the dynamics of the outbreak of ZIKV in Rio de Janeiro, Brazil, Fitzgibbon, Morgan and Webb [12] formulated the following criss-cross partial differential equations model:

$$\begin{cases} \partial_t H_i = \nabla\cdot(\delta_1(X)\nabla H_i) - \lambda(X)H_i + \sigma_1(X)\Lambda(X)V_i, & X\in\Omega,\ t>0,\\ \partial_t V_u = \nabla\cdot(\delta_2(X)\nabla V_u) + \beta(X)(V_u+V_i) - \sigma_2(X)H_iV_u - \mu(X)(V_u+V_i)V_u, & X\in\Omega,\ t>0,\\ \partial_t V_i = \nabla\cdot(\delta_2(X)\nabla V_i) + \sigma_2(X)H_iV_u - \mu(X)(V_u+V_i)V_i, & X\in\Omega,\ t>0,\\ \partial_{\mathbf{n}} H_i = \partial_{\mathbf{n}} V_u = \partial_{\mathbf{n}} V_i = 0, & X\in\partial\Omega, t>0,\\ H_i(X,0) = H_{i0}(X)\geq 0,\ V_u(X,0) = V_{u0}(X)\geq 0,\ V_i(X,0) = V_{i0}(X)\geq 0, & X\in\Omega, \end{cases} \tag{1.1}$$

where $H_i(X,t)$, $V_u(X,t)$, $V_i(X,t)$ are the densities of infected hosts, uninfected vectors and the infected vectors at time t at $X\in\Omega$, respectively. And Ω is a bounded domain in $\mathbb{R}^2$ with smooth boundary $\partial\Omega$. The space-dependent parameters of the model (1.1) are summarized in Table 1.

In [12], the authors proved that model (1.1) is mathematically well-posed, and compared its properties with an analogous ordinary differential equations model in the spatially independent case. And furthermore, the authors studied numerically the following model

$$\begin{cases} \partial_t H_i = \nabla\cdot(\delta_1(X)\nabla H_i) - \lambda(X)H_i + \sigma_1(X)\Lambda(X)V_i, & X\in\Omega,\ t>0,\\ \partial_t V_u = \nabla\cdot(\delta_2(X)\nabla V_u) + \beta(X)(V_u+V_i) - \sigma_2(X)H_iV_u - \mu(X)(V_u+V_i)V_u & \\ \qquad -\mu_1(X)V_u, & X\in\Omega,\ t>0,\\ \partial_t V_i = \nabla\cdot(\delta_2(X)\nabla V_i) + \sigma_2(X)H_iV_u - \mu(X)(V_u+V_i)V_i - \mu_1(X)V_i, & X\in\Omega,\ t>0,\\ \partial_{\mathbf{n}} H_i = \partial_{\mathbf{n}} V_u = \partial_{\mathbf{n}} V_i = 0, & X\in\partial\Omega, t>0,\\ H_i(X,0) = H_{i0}(X)\geq 0,\ V_u(X,0) = V_{u0}(X)\geq 0,\ V_i(X,0) = V_{i0}(X)\geq 0, & X\in\Omega, \end{cases} \tag{1.2}$$

where $\mu_1(X)$ is a time-independent vector loss term. They found that the spatial distribution and final size of the 2015–2016 Zika outbreak in Rio de Janeiro Municipality are strongly dependent on the location and magnitude of local outbreaks at the beginning of the season. But the authors paid little attentions to the dynamics of model (1.1) except well-posedness (Theorem in Appendix [12]). And in a new study [38], the authors give rich dynamical analysis of model (1.1), involving extinction and persistence of ZIKV.

In this paper, we will focus on the effect of spatial heterogeneous environment on the extinction and persistence of the ZIKV model (1.1).

The rest of this article is organized as follows: In Section 2, we give some notations and preliminaries. In Section 3, we accomplish the proofs of our main results. Section 4 focuses on the disease dynamics of model (1.1) via numerical simulations. And in the last section, we provide a brief discussion and the summary of our main results.

2. Preliminaries

In this section, we begin with some basic properties of model (1.1). In what follows, we assume that all of the parameters $\lambda(X)$, $\sigma_1(X)$, $\sigma_2(X)$, $\delta_1(X)$, $\delta_2(x)$, $\Lambda(X)$, $\beta(X)$ and $\mu(X)$ of model (1.1) are continuous, strictly positive and uniformly bounded on $\bar{\Omega}$. The following notations will be used:

$$\|f\|_\infty := \max_{X\in\bar{\Omega}} f(X),\ \hat{f} := \min_{X\in\bar{\Omega}} f(X).$$

Let $\mathbb{X} := C(\bar{\Omega},\mathbb{R}^3)$ be the Banach space with the supremum norm such that

$$\|\phi\|_{\mathbb{X}} := \sup_{X\in\bar{\Omega}} \|\phi(X)\| = \sup_{X\in\bar{\Omega}} \sqrt{|\phi_1(X)|^2+|\phi_2(X)|^2+|\phi_3(X)|^2},\ \phi=(\phi_1,\phi_2,\phi_3)^{\mathbf{T}}\in\mathbb{X},$$

where $\mathbf{T}$ denotes the transpose of the vector. Define $\mathbb{X}^+ := C(\bar{\Omega},\mathbb{R}^3_+)$, then $(\mathbb{X},\mathbb{X}^+)$ is a strongly ordered spaces.