



# **An age-structured model for Tilapia Lake Virus transmission in freshwater with vertical and horizontal transmission**

Cyrille Kenne, René Dorville, Gisèle Mophou, Pascal Zongo

## **► To cite this version:**

Cyrille Kenne, René Dorville, Gisèle Mophou, Pascal Zongo. An age-structured model for Tilapia Lake Virus transmission in freshwater with vertical and horizontal transmission. *Bulletin of Mathematical Biology*, In press. <hal-03266563>

**HAL Id: hal-03266563**

**<https://hal.science/hal-03266563v1>**

Submitted on 21 Jun 2021

**HAL** is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.



HAL Authorization

# An age-structured model for Tilapia Lake Virus transmission in freshwater with vertical and horizontal transmission

Cyrille Kenne\*      René Dorville†      Gisèle Mophou‡      Pascal Zongo§

May 14, 2021

## Abstract

This paper proposes a mathematical model for Tilapia Lake Virus (TiLV) transmission in wild and farmed tilapias within freshwater. This model takes into account two routes of transmission: vertical and horizontal. This latter route integrates both the direct and indirect transmission. We define an explicit formula for the reproductive number  $\mathcal{R}_0$  and show by means of the Fatou's Lemma that the disease free equilibrium is globally asymptotically stable when,  $\mathcal{R}_0 < 1$ . Furthermore, we find an explicit formula of the endemic equilibria and study its local stability as well as the uniform persistence of the disease when  $\mathcal{R}_0 > 1$ . Finally, a numerical scheme to solve the model is developed and some parameters of the model are estimated based on biological data. The numerical results illustrate the role of routes of transmission on the epidemic evolution.

Mathematics Subject Classification. 35K57, 35C07, 92D30

**Key-words :** Population dynamics, tilapia lake virus (TiLV), basic reproduction number, stability, persistence.

## 1 Introduction

### 1.1 Biological motivations

Tilapia is considered as an inexpensive primary source of protein for the majority of developing countries and contributes significantly to global food security through its production of among five million metric tons yearly [2]. However, instead of some various diseases reported early, there is an outbreak of a new disease that is reported as the major cause of mortality in tilapia fisheries.

The Tilapia Lake Virus (TiLV) is a newly discovered virus of tilapia. The infection has been reported in either farmed and wild tilapia populations in fourteen countries across the world (Africa, Asia and South America) [18]. Natural outbreaks of TiLV reportedly resulted in 20 – 90% mortality [5, 13]. In [18, 31], the authors reported that the TiLV is most important in tilapia fry, fingerling and juveniles although viral infections were also evidenced in sub-adult and adult fish. In general, clinical signs and high mortality rates were associated with fish weighing 1 – 50g. Most dead occurred within two weeks after the first dead were found. It is also reported that fish that survived massive die-offs rarely showed clinical signs, suggesting the development of specific immunity against the virus [35]. As TiLV has been horizontally transmitted through cohabitation, disease transmission is likely with movement of live aquatic animals [14]. Disease has been associated with transfer between ponds and thus may be associated with stress [11, 15]. Infected populations of fish, both farmed and wild, are the only established reservoirs of infection. Up to date, the original source of TiLV is not known. No other risk factors (temperature, salinity, etc.) have been identified as potential risk factors up to date. Recently, Yamkasem et al. [39] and T. Dong et al. [12]

---

\*Laboratoire LAMIA, Université des Antilles, Campus Fouillole, 97159 Pointe-à-Pitre Guadeloupe (FWI)- University of Buea, Department of Mathematics, Buea, Cameroon (email :kenne853@gmail.com).

†Laboratoire L3MA, DSI et IUT, Université des Antilles, Schoelcher, Martinique (email :rene.dorville@orange.fr).

‡Laboratoire LAMIA, Université des Antilles, Campus Fouillole, 97159 Pointe-à-Pitre Guadeloupe (FWI)- Laboratoire MAINEGE, Université Ouaga 3S, 06 BP 10347 Ouagadougou 06, Burkina Faso (email :gisele.mophou@univ-antilles.fr).

§Laboratoire L3MA, DSI et IUT, Université des Antilles, Schoelcher, Martinique (email :pascal.zongo@gmail.com).

showed that the transmission can be also vertical from the infected fish to their reproductive organs, eggs, sperm and to their fingerling. Currently, no published methods have been shown to be effective in limiting the impact of an outbreak on an infected farm and there is no reported vaccine.

## 1.2 Mathematical model

Though the TiLV transmission has been considered by the authors cited above, there is only one mathematical model for the spread of this disease to the best of our knowledge. In [40], the authors considered a deterministic susceptible-infectious-mortality (SIM) model to derive key disease information appraised with published TiLV induced cumulative mortality data. They used the Hill model to describe the relationship between tilapia mortality and TiLV exposure.

The main goal of this paper is to take profit of the numerous information on the routes of TiLV transmission to develop a model as close as possible to reality, i.e. considering the different attributes of the infection depending on the time since the infection: transmission rate, mortality rate, birth rate, shedding rate as well as duration of infectious period. Indeed, the infectivity of TiLV-positive fish is high for a relatively short time after becoming infectious, decreases and completely vanish thereafter [10, 14, 27, 37], this suggests that in the spread of the TiLV, the effective transmission depends greatly on the infection age (day post challenge [14, 27, 37], day post infection [10]). This means that the rate of movement out of the infectious class (trought recovery or dead) is better captured as a function of time since infection, rather than a constant value. Moreover it has been showed recently that tilapia develop a immune response to TiLV see for instance [10, 27, 37]. This allows us to better choose a model in which the population is divided into members who may become infected and members who can not become infected, either because they are already infected or because they are immune, that is the underlying idea of infection age models [6]. By choosing all parameters constant in the age-structured model developed here, we obtain a non age-structured model. Therefore, age of infection epidemic models can be viewed as a generalization of compartments models with an arbitrary number of compartments and transfer of some members within these compartments.

In this paper, we formulate a partial differential model with age of infection traducing the spread of the TiLV in a given population. The model incorporates the vertical, the horizontal direct and indirect transmission. To do so, let  $S(t)$  be the density of susceptible fish at time  $t$ ,  $i(a, t)$  the density of fish infected at time  $t$  with respect to age of infection  $a$ . To take into account the indirect transmission of the virus through water, we introduce a new compartment  $V(t)$ , that measures pathogen concentration in a water source at time  $t$ . Susceptible becomes exposed either through direct contact with infected or through contact with contaminated water. The term  $\Lambda_0$  denotes the total recruitment of fish in the population. The constant  $\sigma^{ind}$  denotes the transmission rate parameter for water-to-fish contact (horizontal indirect transmission). The natural mortality is denoted by  $\mu$  while the disease related mortality  $\delta(a)$  depending of the age of infection. We denote by  $b(a)$  the birth rate of an infected fish depending on its age of infection  $a$ . Let  $\pi(a)$  be the proportion of offspring born in the infected class, then the term  $\int_0^\infty \pi(a)b(a)i(a, t)da$  denotes the flux of infected offspring born into the infected class  $i$  at time  $t$ . The term  $\beta(a)i(a, t)$  represents the flux of pathogen shed by infected fish with respect to the age of their infection at time  $t$ . The flux of shed pathogen a time  $t$  is modeled by the term  $\int_0^\infty \beta(a)i(a, t)da$ , we assume that a fraction  $p \in [0, 1]$  of this flux, is directly ingested by fish at time  $t$  by direct contact (from fish to fish) with a rate  $\sigma^{dir}$  and the remaining  $1 - p$  is shed in water. The flux of newly infected fish corresponds to the boundary condition for  $i$  at age  $a = 0$ . The mortality rate of the virus is denoted by  $\mu_V$ . Following all the above assumptions, the model reads as follow:

$$\begin{cases} \frac{dS}{dt} = \Lambda_0 - \int_0^\infty \pi(a)b(a)i(a, t)da - J(t)S(t) - \mu S(t), \\ \frac{\partial i}{\partial t} + \frac{\partial i}{\partial a} = -\eta(a)i, \\ \frac{dV}{dt} = (1 - p) \int_0^\infty \beta(a)i(a, t)da - \mu_V V, \end{cases} \quad (1.1)$$

where

$$J(t) = \sigma^{dir} p \int_0^\infty \beta(a)i(a, t)da + \sigma^{ind} V(t), \quad \text{and} \quad \eta(a) = \mu + \delta(a).$$

The system (1.1) is together with boundary conditions

$$i(0, t) = J(t)S(t) + \int_0^\infty \pi(a)b(a)i(a, t)da, \quad (1.2)$$

and initial conditions

$$S(0) = S_0 \in \mathbb{R}_+, i(\cdot, 0) = i_0(\cdot) \in L_+^1(0, \infty), V(0) = V_0 \in \mathbb{R}_+, \quad (1.3)$$

where  $L_+^1(0, \infty)$  is the space of functions of  $L^1(0, \infty)$  which are positive. Many types of diseases transmission with age of infection have been widely concerned by researchers [4, 7, 9, 20, 23, 26]. In particular in [7], the authors addressed the dynamics of an age-of-infection cholera model for which they incorporated both the direct and indirect transmission. They proved the global stability of the equilibria by constructing a suitable Lyapunov functional. In the same considerations J. Yang et al. [20] studied the global stability of an age-structured model describing the transmission dynamics of cholera. After establishing the dynamical properties of the model by using a Lyapunov function, they extended the global results obtained for multi-stage models to the general continuous age model. None of the above models take into account two routes of transmission (vertical and horizontal) by integrating both the direct and indirect transmission.

We aim to study the behaviour of the system (1.1)-(1.3). In order to do so, we structure the paper as follows. In Section 2, we prove the well posedness of system (1.1)-(1.3). The Section 3 is devoted to the computation of the basic reproduction number, the disease-free equilibrium and its stability. We prove the local stability of the endemic equilibrium in Section 4. The uniform persistence of the disease is discussed in Section 5. In Section 6, we perform some numerical simulations. Finally, a conclusion is given in Section 7.

### Assumption 1.1

We assume that the following conditions are satisfied

- (i)  $\Lambda_0, \mu, \mu_V, \sigma^{dir}, \sigma^{ind} > 0$ ;
- (ii) The functions  $\delta(\cdot) \in L_+^\infty(0, \infty)$  and  $\delta(a) > \delta_0$  for a.e  $a \in (0, \infty)$  and for some  $\delta_0 > 0$ ;
- (iii) The functions  $\beta(\cdot), b(\cdot), \pi(\cdot)$  are positive, bounded and uniformly continuous on  $(0, \infty)$ . And there exist two positive constants  $\pi_0$  and  $b_0$  such that  $b(a) \geq b_0$  and  $\pi(a) \geq \pi_0$  for a.e  $a \in (0, \infty)$ .

Here,  $L_+^\infty(0, \infty)$  is space of functions of  $L^\infty(0, \infty)$  which are positive.

Let us introduce the following function

$$\mathcal{F}(a) = \exp\left(-\int_0^a \eta(s)ds\right). \quad (1.4)$$

Then, it is clear that

$$\mathcal{F} \text{ is a decreasing function, } \mathcal{F}(0) = 1 \text{ and } \mathcal{F}(a) \leq 1. \quad (1.5)$$

## 2 Well posedness of the system (1.1)-(1.3)

To show that the system (1.1)-(1.3) is well posed, we argue as in [25, 26]. Let  $\mathbb{X}$  be the space defined as

$$\mathbb{X} = \mathbb{R} \times L^1(0, \infty) \times \mathbb{R}.$$

We define on  $\mathbb{X}$  the norm, for  $\varphi = (\varphi_1, \varphi_2, \varphi_3)^T \in \mathbb{X}$ ,

$$\|\varphi\|_{\mathbb{X}} = |\varphi_1| + \int_0^\infty |\varphi_2(a)|da + |\varphi_3|.$$

It is clear that  $(\mathbb{X}, \|\cdot\|_{\mathbb{X}})$  is a Banach space. Now, to take the boundary condition into account, we extend the state space by setting  $\mathcal{X} = \mathbb{X} \times \mathbb{R}$ , and we denote by  $\mathcal{X}_+$  the positive cone of  $\mathcal{X}$ . Let also  $\mathcal{X}^\circ = \mathbb{X} \times \{0\}$ . For  $u = (u_1, u_2, u_3)^T \in \mathbb{X}$ , let  $v = (u, 0)^T \in \mathcal{X}^\circ$  and we define the linear operator  $A : D(A) \subset \mathcal{X} \rightarrow \mathcal{X}$  by

$$Av = \begin{pmatrix} -\mu u_1 \\ -\left(\frac{\partial}{\partial a} + \eta(a)\right) u_2 \\ -\mu_V u_3 \\ -u_2(0) \end{pmatrix},$$

with

$$D(A) = \{v \in \mathcal{X}_+^\circ : u_2(\cdot) \in W^{1,1}(0, +\infty)\}.$$

Then  $\overline{D(A)} = \mathcal{X}^\circ$  is not dense in  $\mathcal{X}$ . Let

$$F_1(v) = \Lambda_0 - \int_0^\infty \pi(a)b(a)u_2(a)da - \left[ \sigma^{dir} p \int_0^\infty \beta(a)u_2(a)da + \sigma^{ind} u_3 \right] u_1,$$

$$F_4(v) = \int_0^\infty \pi(a)b(a)u_2(a)da + \left[ \sigma^{dir} p \int_0^\infty \beta(a)u_2(a)da + \sigma^{ind} u_3 \right] u_1,$$

and

$$F_3(v) = (1-p) \int_0^\infty \beta(a)u_2(a)da.$$

We consider  $F : \overline{D(A)} \rightarrow \mathcal{X}$  the nonlinear map defined by

$$F(v) = \begin{pmatrix} F_1(v) \\ 0 \\ F_3(v) \\ F_4(v) \end{pmatrix}.$$

Set  $\mathcal{X}_0 = \overline{D(A)}$  and  $\mathcal{X}_0^+ = \overline{D(A)} \cap \mathbb{X}_+$ . Then, the system (1.1)-(1.3) rewrites as the following abstract Cauchy problem

$$\frac{dv(t)}{dt} = Av(t) + F(v(t)), \quad \text{for } t \geq 0, \quad \text{with } v(0) = v_0 \in \overline{D(A)}. \quad (2.1)$$

In general, the differential equation (2.1) may not have a strong solution. Thus, we solve it in integrated form:

$$v(t) = v_0 + A \int_0^t v(s)ds + \int_0^t F(v(s))ds. \quad (2.2)$$

A continuous solution to (2.1) is called an integral solution to (2.2).

**Theorem 2.1** *The system of equations (1.1)-(1.3) represented by the integral equation (2.2) has a unique continuous solution with values in  $\mathcal{X}_0^+$ . Moreover the map  $\Psi : [0, \infty) \times \mathcal{X}_0^+ \rightarrow \mathcal{X}_0^+$  defined by  $\Psi(t, x) = v(t)$  is a continuous semiflow. That is the map  $\Psi$  is continuous and  $\Psi(t, \Psi(s, \cdot)) = \Psi(t+s, \cdot)$  and  $\Psi(0, \cdot)$  is the identity map.*

**Proof.** We argue as in [26]. First, we notice that  $F$  is Lipschitz continuous on bounded sets. It is standard to prove that the operator  $(A, D(A))$  is a Hille-Yosida operator and  $A$  is resolvent positive see for e.g [25, 26]. Next, we show that for all  $t \geq 0$  and  $v \in \mathcal{X}_0^+$ ,

$$\lim_{h \rightarrow 0^+} \frac{1}{h} \text{dist}(v + hF(v), \mathcal{X}_+) = 0. \quad (2.3)$$

Let  $m > 0$  and  $B(0, m)$  be the ball of  $\mathcal{X}$  centered at 0 and with radius  $m$ . By the expression of  $F$ , there exists a positive constant  $C = C(m, \|\beta\|_{L^\infty(0, \infty)})$  such for any  $v \in \mathcal{X}_0^+ \cap B(0, m)$ ,

$$F(v) + \alpha v \in \mathcal{X}_+.$$

Now, if we let

$$\tilde{F}(v) = F(v) + \alpha v,$$

with  $\alpha$  being chosen such that  $\alpha > C$ . Then,  $\tilde{F}(v) \in \mathcal{X}_+$  and for any positive and sufficiently small  $h$ , we have  $\alpha - \alpha h v \in \mathcal{X}_+$  and  $h\tilde{F}(v) \in \mathcal{X}_+$ . So

$$\lim_{h \rightarrow 0^+} \frac{1}{h} \text{dist}(v + hF(v), \mathcal{X}_+) = \lim_{h \rightarrow 0^+} \frac{1}{h} \text{dist}(\alpha - \alpha h v + h\tilde{F}(v), \mathcal{X}_+) = 0.$$

This complete the proof. ■

In what follows, we prove that the solution of (1.1)-(1.3) is bounded.

**Theorem 2.2** *The solution of system (1.1)-(1.3) is bounded. More precisely we have, for a.e  $t \geq 0$ ,*

$$S(t) + \int_0^\infty i(a, t) da \leq \max \left\{ S_0 + \int_0^\infty i_0(a) da, \frac{\Lambda_0}{\mu} \right\}, \quad V(t) \leq \max \left\{ V_0, \frac{(1-p)\|\beta\|_{L^\infty(0,\infty)} \Lambda_0}{\mu_V \mu} \right\}. \quad (2.4)$$

Moreover the upper bounds are uniforms,

$$\limsup_{t \rightarrow \infty} [S(t) + \int_0^\infty i(a, t) da] \leq \frac{\Lambda_0}{\mu}, \quad \limsup_{t \rightarrow \infty} V(t) \leq \frac{(1-p)\|\beta\|_{L^\infty(0,\infty)} \Lambda_0}{\mu_V \mu}. \quad (2.5)$$

**Proof.** Integrating the second equation of (1.1) on  $(0, \infty)$  with respect to  $a$ , and combining with the first equation of (1.1) yields

$$S + I \leq \Lambda_0 - \mu(S + I) = (S_0 + I_0)e^{-\mu t} + \frac{\Lambda_0}{\mu}(1 - e^{-\mu t}),$$

where  $I(t) := \int_0^\infty i(a, t) da$ . from which we deduce the first inequality of (2.4). Moreover,

$$\limsup_{t \rightarrow \infty} [S(t) + I(t)] \leq \frac{\Lambda_0}{\mu},$$

and then, from the third equation of (1.1), we have

$$\begin{aligned} \frac{dV}{dt} &\leq (1-p)\|\beta\|_{L^\infty(0,\infty)} I(t) - \mu_V V \\ &\leq (1-p)\|\beta\|_{L^\infty(0,\infty)} \frac{\Lambda_0}{\mu} - \mu_V V. \end{aligned}$$

Hence

$$V(t) \leq V_0 e^{-\mu_V t} + \frac{(1-p)\|\beta\|_{L^\infty(0,\infty)} \Lambda_0}{\mu_V \mu} (1 - e^{-\mu_V t}),$$

from which we deduce the second inequality of (2.4). Furthermore,

$$\limsup_{t \rightarrow \infty} V(t) \leq \frac{(1-p)\|\beta\|_{L^\infty(0,\infty)} \Lambda_0}{\mu_V \mu}.$$

This complete the proof. ■

**Remark 1** *We have shown in Theorem 2.2 that the set  $\mathcal{D}$  defined as*

$$\mathcal{D} = \left\{ (S, i, V); S(t) + \int_0^\infty i(a, t) da \leq \frac{\Lambda_0}{\mu}, \quad V(t) \leq \frac{(1-p)\|\beta\|_{L^\infty(0,\infty)} \Lambda_0}{\mu_V \mu} \right\}, \quad (2.6)$$

*is positively invariant for system (1.1).*

The next section concerns the basic reproduction number, the existence and the stability of the disease-free equilibrium.

### 3 Basic reproduction number

Anticipating our future needs, we start this section by the following definition

**Definition 3.1** We define the basic reproduction number of system (1.1)-(1.3) by the following formula:

$$\mathcal{R}_0 = \mathcal{R}_0^{Hdir} + \mathcal{R}_0^{Hind} + \mathcal{R}_0^V \quad (3.1)$$

where

$$\mathcal{R}_0^{Hdir} = \frac{\Lambda_0}{\mu} \sigma^{dir} p \int_0^\infty \beta(a) \mathcal{F}(a) da, \quad (3.2a)$$

$$\mathcal{R}_0^{Hind} = \frac{\Lambda_0}{\mu} \frac{\sigma^{ind}(1-p)}{\mu_V} \int_0^\infty \beta(a) \mathcal{F}(a) da, \quad (3.2b)$$

$$\mathcal{R}_0^V = \int_0^\infty \pi(a) b(a) \mathcal{F}(a) da, \quad (3.2c)$$

where  $\mathcal{F}(a)$  is given by (1.4).

$\mathcal{R}_0$  depicts the expected number of secondary infections resulting from a single primary infection into an otherwise susceptible population. The term  $\mathcal{R}_0^{Hdir}$  is the average number of secondary infections produced by one infective individual during its infectious period by horizontal direct transmission while  $\mathcal{R}_0^{Hind}$  represents the average number of secondary infections produced by one infective individual during its infectious period by horizontal indirect transmission.  $\mathcal{R}_0^V$  is the average number of secondary infections produced by one infective individual during its infectious period by vertical transmission.

**Theorem 3.1** Let  $\mathcal{R}_0$  be defined by (3.1). Then, system (1.1)-(1.3) always has a disease-free equilibrium given by  $E_0 = (S^0, 0, 0)$ , where  $S^0 = \frac{\Lambda_0}{\mu}$ . Moreover the disease-free equilibrium  $E_0$  is locally asymptotically stable if  $\mathcal{R}_0 < 1$  while unstable if  $\mathcal{R}_0 > 1$ .

**Proof.** It is clear that  $E_0$  exists. Now let  $S(t) = S^0 + S_1(t)$ ,  $i(a, t) = i_1(a, t)$  and  $V(t) = V_1(t)$ . Linearizing the system (1.1)-(1.3) about  $E_0$ , we obtain the following system

$$\begin{cases} \frac{dS_1}{dt} = - \int_0^\infty \pi(a) b(a) i_1(a, t) da - \left[ \sigma^{dir} p \int_0^\infty \beta(a) i_1(a, t) da + \sigma^{ind} V_1 \right] S^0 - \mu S_1(t), \\ \frac{\partial i_1}{\partial t} + \frac{\partial i_1}{\partial a} = -\eta(a) i_1, \\ \frac{dV_1}{dt} = (1-p) \int_0^\infty \beta(a) i_1(a, t) da - \mu_V V_1, \end{cases} \quad (3.3)$$

together with boundary condition

$$i_1(0, t) = \left[ \sigma^{dir} p \int_0^\infty \beta(a) i_1(a, t) da + \sigma^{ind} V_1 \right] S^0 + \int_0^\infty \pi(a) b(a) i_1(a, t) da. \quad (3.4)$$

We look for solutions on the form  $S_1(t) = x e^{rt}$ ,  $i_1(a, t) = y(a) e^{rt}$  and  $V_1(t) = z e^{rt}$ , with  $r$  a real number. Thus we consider the following problem

$$\begin{cases} rx = - \int_0^\infty \pi(a) b(a) y(a) da - \left[ \sigma^{dir} p \int_0^\infty \beta(a) y(a) da + \sigma^{ind} z \right] S^0 - \mu x, \\ ry(a) + \frac{dy(a)}{da} = -\eta(a) y(a), \\ rz = (1-p) \int_0^\infty \beta(a) y(a) da - \mu_V z, \\ y(0) = \left[ \sigma^{dir} p \int_0^\infty \beta(a) y(a) da + \sigma^{ind} z \right] S^0 + \int_0^\infty \pi(a) b(a) y(a) da. \end{cases} \quad (3.5)$$

Solving the second equation of (3.5) lead us to

$$y(a) = y(0)e^{-\int_0^a (r+\eta(s))ds}. \quad (3.6)$$

Moreover the third equation of (3.5) gives

$$z = \frac{(1-p)}{r+\mu_V} y(0) \int_0^\infty \beta(a)e^{-ar} \mathcal{F}(a) da, \quad (3.7)$$

with  $\mathcal{F}(a)$  given by (1.4).

Taking (3.6) and (3.7) into account, the fourth equation of (3.5) lead us to

$$y(0) = y(0) \left[ \sigma^{dir} p + \frac{\sigma^{ind}(1-p)}{r+\mu_V} \right] S^0 \int_0^\infty \beta(a)e^{-ar} \mathcal{F}(a) da + y(0) \int_0^\infty \pi(a)b(a)e^{-ar} \mathcal{F}(a) da.$$

That is

$$1 = \left[ \sigma^{dir} p + \frac{\sigma^{ind}(1-p)}{r+\mu_V} \right] S^0 \int_0^\infty \beta(a)e^{-ar} \mathcal{F}(a) da + \int_0^\infty \pi(a)b(a)e^{-ar} \mathcal{F}(a) da. \quad (3.8)$$

Define a function

$$H(r) = \left[ \sigma^{dir} p + \frac{\sigma^{ind}(1-p)}{r+\mu_V} \right] S^0 \int_0^\infty \beta(a)e^{-ar} \mathcal{F}(a) da + \int_0^\infty \pi(a)b(a)e^{-ar} \mathcal{F}(a) da. \quad (3.9)$$

Then,  $H(0) = \mathcal{R}_0$ . Moreover it is easy to see that,  $H$  is a continuously differentiable satisfying

$$\lim_{r \rightarrow +\infty} H(r) = 0, \quad \lim_{r \rightarrow -\infty} H(r) = +\infty, \quad \text{and} \quad H'(r) < 0.$$

Therefore  $H$  is a decreasing function. Hence, any real solution of equation (3.8) is negative if  $\mathcal{R}_0 < 1$ , and positive if  $\mathcal{R}_0 > 1$ . Thus, if  $\mathcal{R}_0 > 1$  the infection-free equilibrium is unstable. Next, we show that equation (3.8) has no complex solutions with nonnegative real part if  $\mathcal{R}_0 < 1$ . To do this, let  $r = x + iy$ , with  $x, y \in \mathbb{R}$  be a solution of equation (3.8). Now define

$$\mathcal{G}(a) = (\sigma^{dir} p S^0 \beta(a) + \pi(a)b(a)) \mathcal{F}(a), \quad (3.10)$$

and

$$\mathcal{H}(a) = \sigma^{ind}(1-p) S^0 \beta(a) \mathcal{F}(a). \quad (3.11)$$

So

$$H(r) = \int_0^\infty \mathcal{G}(a)e^{-ar} da + \frac{1}{r+\mu_V} \int_0^\infty \mathcal{H}(a)e^{-ar} da.$$

We argue by contradiction by assuming that  $x \geq 0$ . Then,

$$\begin{aligned} |H(r)| &= \left| \int_0^\infty \mathcal{G}(a)e^{-ar} da + \int_0^\infty \mathcal{H}(a)e^{-ar} da \right| \\ &\leq \int_0^\infty |e^{-a(x+iy)}| |\mathcal{G}(a)| da + \frac{1}{\sqrt{(x+\mu_V)^2 + y^2}} \int_0^\infty |e^{-a(x+iy)}| |\mathcal{H}(a)| da \\ &\leq \int_0^\infty e^{-ax} \mathcal{G}(a) da + \frac{1}{x+\mu_V} \int_0^\infty e^{-ax} \mathcal{H}(a) da \\ &= |H(x)| \leq \mathcal{R}_0 < 1. \end{aligned}$$

That is  $|H(r)| < 1$ , which is a contradiction. Thus,  $x < 0$  and every solution of (3.8) has a negative real part. Therefore, if  $\mathcal{R}_0 < 1$ , the disease-free equilibrium  $E_0$  is locally asymptotically stable and is unstable if  $\mathcal{R}_0 > 1$ . This complete the proof. ■

Next, we prove the global stability of the disease-free equilibrium.



**Theorem 3.2** *Under the assumptions of Theorem 3.1, the disease-free equilibrium  $E_0$  of system (1.1)-(1.3) is globally asymptotically stable if  $\mathcal{R}_0 < 1$ .*

**Proof.** From Theorem 3.1, we know that the disease-free equilibrium  $E_0$  is locally asymptotically stable when  $\mathcal{R}_0 < 1$ . It suffices to show that  $E_0$  is a global attractor. By integrating the equation of (1.1) in  $i$  along the characteristic line  $t - a = c$ , we obtain

$$i(a, t) = \begin{cases} B(t - a)\mathcal{F}(a) & \text{if } 0 \leq a < t, \\ i_0(a - t)e^{-\int_{a-t}^a \eta(s)ds} & \text{if } a > t \geq 0, \end{cases} \quad (3.12)$$

where  $B(t) = i(0, t)$ . Let  $m = \limsup_{t \rightarrow \infty} B(t)$ . Also by integrating the third equation of (1.1), we obtain

$$V(t) = V_0 e^{-\mu_V t} + (1 - p) \int_0^t \int_0^\infty e^{-\mu_V(t-s)} \beta(a) i(a, s) dads. \quad (3.13)$$

Now considering (1.2), (3.12) and (3.13), we write

$$\begin{aligned} B(t) &= S(t) \left[ \sigma^{dir} p \int_0^t \beta(a) B(t - a) \mathcal{F}(a) da + \sigma^{dir} p \int_t^\infty \beta(a) i(a, t) da \right] \\ &\quad + S(t) \left[ \sigma^{ind} V_0 e^{-\mu_V t} + \sigma^{ind} (1 - p) \int_0^t \int_0^\infty e^{-\mu_V(t-s)} \beta(a) i(a, s) dads \right] \\ &\quad + \int_0^t \pi(a) b(a) B(t - a) \mathcal{F}(a) da + \int_t^\infty \pi(a) b(a) i(a, t) da. \end{aligned} \quad (3.14)$$

Notice that by a change of variable, we have

$$\begin{aligned} \int_0^t \int_0^\infty e^{-\mu_V(t-s)} \beta(a) i(a, s) dads &= \int_0^t \int_0^t e^{-\mu_V(t-s)} \beta(a) B(s - a) \mathcal{F}(a) dads \\ &\quad + \int_0^t \int_t^\infty e^{-\mu_V(t-s)} \beta(a) i(a, s) dads \\ &= \int_0^t \int_0^t e^{-\mu_V s} \beta(a) B(t - s - a) \mathcal{F}(a) dads \\ &\quad + \int_0^t \int_t^\infty e^{-\mu_V(t-s)} \beta(a) i(a, s) dads. \end{aligned}$$

Thus (3.14) gives

$$\begin{aligned} B(t) &= S(t) \left[ \sigma^{dir} p \int_0^t \beta(a) B(t - a) \mathcal{F}(a) da + \sigma^{dir} p \int_t^\infty \beta(a) i(a, t) da \right] \\ &\quad + S(t) \left[ \sigma^{ind} V_0 e^{-\mu_V t} + \sigma^{ind} (1 - p) \int_0^t \int_0^t e^{-\mu_V s} \beta(a) B(t - s - a) \mathcal{F}(a) dads \right] \\ &\quad + \sigma^{ind} (1 - p) S(t) \int_0^t \int_t^\infty e^{-\mu_V(t-s)} \beta(a) i(a, s) dads + \int_0^t \pi(a) b(a) B(t - a) \mathcal{F}(a) da \\ &\quad + \int_t^\infty \pi(a) b(a) i(a, t) da. \end{aligned}$$

Taking the lim sup when  $t \rightarrow \infty$  on both sides of this latter equality and thanks to Fatou's Lemma and the relation (2.2), we obtain

$$\begin{aligned}
m &\leq \frac{\Lambda_0}{\mu} \left[ \sigma^{dir} p m \int_0^\infty \beta(a) \mathcal{F}(a) da + \sigma^{ind} (1-p) m \int_0^\infty \int_0^\infty e^{-\mu_V s} \beta(a) \mathcal{F}(a) da ds \right] \\
&\quad + m \int_0^\infty \pi(a) b(a) \mathcal{F}(a) da \\
&= \frac{\Lambda_0}{\mu} \left[ \sigma^{dir} p m \int_0^\infty \beta(a) \mathcal{F}(a) da + \frac{\sigma^{ind} (1-p)}{\mu_V} m \int_0^\infty \beta(a) \mathcal{F}(a) da \right] \\
&\quad + m \int_0^\infty \pi(a) b(a) \mathcal{F}(a) da \\
&= m \mathcal{R}_0.
\end{aligned}$$

So  $m(1 - \mathcal{R}_0) \leq 0$ . Since  $\mathcal{R}_0 < 1$ , then  $m = 0$ . This implies that

$$\lim_{t \rightarrow \infty} i(a, t) = 0 \quad \text{and} \quad \lim_{t \rightarrow \infty} V(t) = 0.$$

From the first equation in (1.1), we have  $\lim_{t \rightarrow \infty} S(t) = \frac{\Lambda_0}{\mu}$ . So the disease free equilibrium is a global attractor. This complete the proof. ■

## 4 Endemic equilibria and their stability

**Lemma 4.1** (*Existence of an endemic equilibrium*) Let  $\mathcal{R}_0$  and  $\mathcal{R}_0^V$  be given respectively by (3.1) and (3.2c). If  $\mathcal{R}_0 > 1$ , then there exists a positive endemic equilibrium  $P^* = (S^*, i^*(a), V^*)$ , when  $\mathcal{R}_0^V < 1$  where

$$S^* = \frac{(1 - \mathcal{R}_0^V) \mu_V}{(\sigma^{dir} p \mu_V + \sigma^{ind} (1-p)) K}, \quad i^*(a) = (\mathcal{R}_0 - 1) \frac{\mu \mu_V \mathcal{F}(a)}{(\sigma^{dir} p \mu_V + \sigma^{ind} (1-p)) K}, \quad (4.1)$$

and

$$V^* = (\mathcal{R}_0 - 1) \frac{\mu(1-p)}{(\sigma^{dir} p \mu_V + \sigma^{ind} (1-p))}, \quad (4.2)$$

with

$$K = \int_0^\infty \beta(a) \mathcal{F}(a) da.$$

**Remark 2** Note that the biologically meaningful interpretation of the condition  $\mathcal{R}_0^V < 1$  is the following: the vertical transmission alone is almost insufficient to sustain epidemic growth.

**Proof.** Let  $(S^*, i^*(a), V^*)$  represent any arbitrary endemic equilibrium of the model (1.1)-(1.3). Then it satisfies the following equations

$$\begin{cases}
\Lambda_0 - \int_0^\infty \pi(a) b(a) i^*(a) da - \left[ \sigma^{dir} p \int_0^\infty \beta(a) i^*(a) da + \sigma^{ind} V^* \right] S^* - \mu S^* = 0, \\
\frac{di^*(a)}{da} = -\eta(a) i^*(a), \\
(1-p) \int_0^\infty \beta(a) i^*(a) da - \mu_V V^* = 0, \\
i^*(0) = \left[ \sigma^{dir} p \int_0^\infty \beta(a) i^*(a) da + \sigma^{ind} V^* \right] S^* + \int_0^\infty \pi(a) b(a) i^*(a) da.
\end{cases} \quad (4.3)$$

The solution of the second equation of (4.3) is given by

$$i^*(a) = i^*(0) \mathcal{F}(a). \quad (4.4)$$

Taking (4.4) into account, we obtain from the third equation of (4.3)

$$V^* = \frac{1-p}{\mu_V} i^*(0) K. \quad (4.5)$$

Hence the fourth equation gives

$$i^*(0) = i^*(0) K \left( \sigma^{dir} p + \frac{\sigma^{ind}(1-p)}{\mu_V} \right) S^* + i^*(0) \mathcal{R}_0^V,$$

so

$$1 = K \left( \sigma^{dir} p + \frac{\sigma^{ind}(1-p)}{\mu_V} \right) S^* + \mathcal{R}_0^V. \quad (4.6)$$

That is

$$S^* = \frac{(1 - \mathcal{R}_0^V) \mu_V}{(\sigma^{dir} p \mu_V + \sigma^{ind}(1-p)) K}.$$

Moreover by replacing  $S^*$  by its value in the first equation of (4.3) and taking (4.4) and (4.5) into account, we obtain

$$i^*(0) = (\mathcal{R}_0 - 1) \frac{\mu \mu_V}{(\sigma^{dir} p \mu_V + \sigma^{ind}(1-p)) K}.$$

Hence the second relation of (4.1) holds. Now using this latter relation in (4.5), (4.2) follows. This complete the proof. ■

**Theorem 4.1** (*local stability of the endemic equilibrium*). *If  $\mathcal{R}_0 > 1$ , then the endemic equilibrium is locally asymptotically stable.*

**Proof.** Let  $(S^*, i^*(a), V^*)$  be an equilibrium point of (1.1)-(1.3). We set  $S(t) = S^* + S_1(t)$ ,  $i(a, t) = i^*(a) + i_1(a, t)$  and  $V(t) = V^* + V_1(t)$ . Then  $(S_1, i_1, V_1)$  is solution to the linearized system

$$\begin{cases} \frac{dS_1}{dt} = -J_1(t) S^* - J^* S_1 - \mu S_1(t) - \int_0^\infty \pi(a) b(a) i_1(a, t) da, \\ \frac{\partial i_1}{\partial t} + \frac{\partial i_1}{\partial a} = -\eta(a) i_1, \\ \frac{dV_1}{dt} = (1-p) \int_0^\infty \beta(a) i_1(a, t) da - \mu_V V_1, \end{cases} \quad (4.7)$$

where

$$J_1(t) = \left[ \sigma^{dir} p \int_0^\infty \beta(a) i_1(a, t) da + \sigma^{ind} V_1 \right] \quad \text{and} \quad J^* = \left[ \sigma^{dir} p \int_0^\infty \beta(a) i^*(a) da + \sigma^{ind} V^* \right].$$

And together with boundary condition

$$i_1(0, t) = J_1(t) S^* + J^* S_1 + \int_0^\infty \pi(a) b(a) i_1(a, t) da. \quad (4.8)$$

We are looking solutions on the form  $S_1(t) = x e^{rt}$ ,  $i_1(a, t) = y(a) e^{rt}$  and  $V_1(t) = z e^{rt}$ , with  $r$  a real number. Thus we consider the following problem

$$\begin{cases} rx = - \left[ \sigma^{dir} p \int_0^\infty \beta(a) y(a) da + \sigma^{ind} z \right] S^* - \left[ \sigma^{dir} p \int_0^\infty \beta(a) i^*(a) da + \sigma^{ind} V^* \right] x - \mu x - \int_0^\infty \pi(a) b(a) y(a) da, \\ ry(a) + \frac{dy(a)}{da} = -\eta(a) y(a), \\ rz = (1-p) \int_0^\infty \beta(a) y(a) da - \mu_V z, \\ y(0) = \left[ \sigma^{dir} p \int_0^\infty \beta(a) y(a) da + \sigma^{ind} z \right] S^* + \left[ \sigma^{dir} p \int_0^\infty \beta(a) i^*(a) da + \sigma^{ind} V^* \right] x + \int_0^\infty \pi(a) b(a) y(a) da. \end{cases} \quad (4.9)$$

Solving the second equation of (4.9) lead us to

$$y(a) = y(0)e^{-\int_0^a (r+\eta(s))ds},$$

which by using the expression of  $\mathcal{F}(a)$  given by (1.4) gives

$$y(a) = y(0)e^{-ar}\mathcal{F}(a). \quad (4.10)$$

Moreover the third equation of (4.9) gives

$$z = \frac{(1-p)}{r+\mu_V}y(0)\int_0^\infty \beta(a)e^{-ar}\mathcal{F}(a)da. \quad (4.11)$$

Taking (4.10) and (4.11) into account, the fourth equation of (4.9) lead us to

$$\begin{aligned} y(0) = y(0) \left[ \sigma^{dir}p + \frac{\sigma^{ind}(1-p)}{r+\mu_V} \right] S^* \int_0^\infty \beta(a)e^{-ar}\mathcal{F}(a)da + \left[ \sigma^{dir}p \int_0^\infty \beta(a)i^*(a)da + \sigma^{ind}V^* \right] x \\ + y(0) \int_0^\infty \pi(a)b(a)e^{-ar}\mathcal{F}(a)da. \end{aligned} \quad (4.12)$$

Moreover, combining the first and the fourth equations of (4.9), we obtain

$$x = -\frac{y(0)}{r+\mu}.$$

Hence (4.12) becomes

$$1 + \frac{A^*}{r+\mu} = \left[ \sigma^{dir}p + \frac{\sigma^{ind}(1-p)}{r+\mu_V} \right] S^* \int_0^\infty \beta(a)e^{-ar}\mathcal{F}(a)da + \int_0^\infty \pi(a)b(a)e^{-ar}\mathcal{F}(a)da, \quad (4.13)$$

where  $A^* = \left[ \sigma^{dir}p \int_0^\infty \beta(a)i^*(a)da + \sigma^{ind}V^* \right]$ . Next, for  $r$  being a complex number with a nonnegative real part, it is easy to see that

$$\left| 1 + \frac{A^*}{r+\mu} \right| = \left| \frac{r+\mu+A^*}{r+\mu} \right| > 1. \quad (4.14)$$

Moreover, we can write

$$\begin{aligned} \left| \left[ \sigma^{dir}p + \frac{\sigma^{ind}(1-p)}{r+\mu_V} \right] S^* \int_0^\infty \beta(a)e^{-ar}\mathcal{F}(a)da + \int_0^\infty \pi(a)b(a)e^{-ar}\mathcal{F}(a)da \right| \\ \leq \left[ \sigma^{dir}p + \frac{\sigma^{ind}(1-p)}{\mu_V} \right] S^* \int_0^\infty \beta(a)\mathcal{F}(a)da + \int_0^\infty \pi(a)b(a)\mathcal{F}(a)da = 1. \end{aligned} \quad (4.15)$$

Combining (4.14) and (4.15), we obtain a contradiction. So the real part of  $r$  is strictly negative. Hence, the endemic equilibrium is locally asymptotically stable according to [8, 26]. This complete the proof. ■

## 5 Uniform persistence of the disease

Let us introduce the following sets

$$\mathcal{M} = \left\{ \varphi(a) \in L_+^1(0, \infty); \exists t \geq 0 : \int_0^\infty \beta(a+t)\varphi(a)da > 0 \quad \text{and} \quad \int_0^\infty \pi(a+t)b(a+t)\varphi(a)da > 0 \right\},$$

$$\mathcal{D}_0 = \mathbb{R}_+ \times \mathcal{M} \times \mathbb{R}_+, \quad \text{and} \quad X_0 = \mathcal{D} \cap \mathcal{D}_0,$$

where  $\mathcal{D}$  is given by (2.6).

In this section we aim to prove the following

**Theorem 5.1** (*Strongly uniform persistence*) Assume that  $\mathcal{R}_0 > 1$ , then there exists a constant  $\rho > 0$  (independent of initial conditions) such that any solution  $(S, i, V)$  of (1.1)-(1.3) with  $(S_0, i_0, V_0) \in \mathbb{R}_+ \times L_+^1(0, \infty) \times \mathbb{R}_+$  satisfies

$$\liminf_{t \rightarrow \infty} \left( \int_0^\infty i(a, t) da + V(t) \right) > \rho.$$

To prove this theorem, we state and prove Lemmas 5.1 and 5.2. We will denote by  $\|\cdot\|_\infty$ , the  $L^\infty$ -norm in a suitable space.

**Lemma 5.1** (*Weakly uniform persistence*) Assume that  $\mathcal{R}_0 > 1$ , then there exist a constant  $\gamma > 0$  such that any solution  $(S, i, V)$  of (1.1)-(1.3) with  $(S_0, i_0, V_0) \in \mathbb{R}_+ \times L_+^1(0, \infty) \times \mathbb{R}_+$  satisfies

$$\limsup_{t \rightarrow \infty} \left( \int_0^\infty i(a, t) da + V(t) \right) > \gamma.$$

**Proof.** We argue by contradiction, that is we assume that for every  $\varepsilon > 0$ , we have

$$\limsup_{t \rightarrow \infty} \left( \int_0^\infty i(a, t) da + V(t) \right) < \varepsilon. \quad (5.1)$$

Without loss of generality we choose  $\varepsilon$  such that  $0 < \varepsilon < \frac{\Lambda_0}{2\|b\|_\infty}$ . Hence there exists  $t_1 > 0$  such that for all  $t > t_1$ ,

$$\int_0^\infty i(a, t) da + V(t) < \varepsilon. \quad (5.2)$$

$\pi$  being a probability, it follows from the first equation in (1.1) that,

$$\frac{dS(t)}{dt} \geq \Lambda_0 - \varepsilon\|b\|_\infty - [\sigma^{dir}p\|\beta\|_\infty + \sigma^{ind}]\varepsilon S(t) - \mu S(t).$$

Therefore,

$$\limsup_{t \rightarrow \infty} S(t) \geq \liminf_{t \rightarrow \infty} S(t) \geq \frac{\Lambda_0 - \varepsilon\|b\|_\infty}{\mu + [\sigma^{dir}p\|\beta\|_\infty + \sigma^{ind}]\varepsilon}.$$

Thus

$$\limsup_{t \rightarrow \infty} S(t) \geq \frac{\Lambda_0 - \varepsilon\|b\|_\infty}{\mu + m\varepsilon}, \quad (5.3)$$

with  $m = \sigma^{dir}p\|\beta\|_\infty + \sigma^{ind}$ .

We let  $B(t) = i(0, t)$  and using the inequality (5.3), we obtain

$$B(t) \geq \frac{\sigma^{dir}(\Lambda_0 - \varepsilon\|b\|_\infty)}{\mu + m\varepsilon} \int_0^\infty \beta(a)i(a, t)da + \frac{\sigma^{ind}(\Lambda_0 - \varepsilon\|b\|_\infty)}{\mu + m\varepsilon} V(t) + \int_0^\infty \pi(a)b(a)i(a, t)da. \quad (5.4)$$

Using the expression of  $i$  given by (3.12) in (5.4), we obtain

$$B(t) \geq \frac{\sigma^{dir}(\Lambda_0 - \varepsilon\|b\|_\infty)}{\mu + m\varepsilon} \int_0^t \beta(a)B(t-a)\mathcal{F}(a)da + \frac{\sigma^{ind}(\Lambda_0 - \varepsilon\|b\|_\infty)}{\mu + m\varepsilon} V(t) + \int_0^t \pi(a)b(a)B(t-a)\mathcal{F}(a)da \quad (5.5)$$

Using again the expression of  $i$  given by (3.12) in the third equation of (1.1), we have that

$$\frac{dV(t)}{dt} \geq (1-p) \int_0^t \beta(a)B(t-a)\mathcal{F}(a)da - \mu_V V(t). \quad (5.6)$$

Let  $\widehat{B}(\lambda)$  be the Laplace transform of  $B(t)$  and  $\widehat{V}(\lambda)$  be the Laplace transform of  $V(t)$ . Furthermore, we set

$$\widehat{K}_1(\lambda) = \int_0^\infty \beta(a)\mathcal{F}(a)e^{-\lambda a}da \quad \text{and} \quad \widehat{K}_2(\lambda) = \int_0^\infty \pi(a)\beta(a)\mathcal{F}(a)e^{-\lambda a}da. \quad (5.7)$$

Then by applying the Laplace transform in the both sides of (5.5) and (5.6), we respectively obtain

$$\widehat{B}(\lambda) \geq \frac{\sigma^{dir}(\Lambda_0 - \varepsilon\|b\|_\infty)}{\mu + m\varepsilon} \widehat{K}_1(\lambda) \widehat{B}(\lambda) + \frac{\sigma^{ind}(\Lambda_0 - \varepsilon\|b\|_\infty)}{\mu + m\varepsilon} \widehat{V}(\lambda) + \widehat{K}_2(\lambda) \widehat{B}(\lambda), \quad (5.8)$$

and

$$\lambda \widehat{V}(\lambda) - V_0 \geq (1 - p) \widehat{K}_1(\lambda) \widehat{B}(\lambda) - \mu_V \widehat{V}(\lambda). \quad (5.9)$$

Hence by combining (5.8) and (5.9), we are led to

$$\widehat{B}(\lambda) \geq \left\{ \frac{\Lambda_0 - \varepsilon\|b\|_\infty}{\mu + m\varepsilon} \left[ \sigma^{dir} p + \frac{\sigma^{ind}(1 - p)}{\lambda + \mu_V} \right] \widehat{K}_1(\lambda) + \widehat{K}_2(\lambda) \right\} \widehat{B}(\lambda) + \frac{\sigma^{ind}(\Lambda_0 - \varepsilon\|b\|_\infty) V_0}{(\mu + m\varepsilon)(\lambda + \mu_V)}. \quad (5.10)$$

Notice that (5.10) holds for a given  $\varepsilon \approx 0$  and for any  $\lambda > 0$ . By assuming that  $\lambda \approx 0$ , we have

$$\left\{ \frac{\Lambda_0 - \varepsilon\|b\|_\infty}{\mu + m\varepsilon} \left[ \sigma^{dir} p + \frac{\sigma^{ind}(1 - p)}{\lambda + \mu_V} \right] \widehat{K}_1(\lambda) + \widehat{K}_2(\lambda) \right\} = \mathcal{R}_0 > 1.$$

Then (5.10) can be rewritten as

$$\widehat{B}(\lambda) \geq \widehat{B}(\lambda) + \frac{\Lambda_0 \sigma^{ind} V_0}{2\mu\mu_V}.$$

Which is impossible since  $\frac{\Lambda_0 \sigma^{ind} V_0}{2\mu\mu_V} > 0$ . Hence there is a constant  $\gamma > 0$  such that any solution  $(S, i, V)$  of (1.1)-(1.3) with  $(S_0, i_0, V_0) \in \mathbb{R}_+ \times L_+^1(0, \infty) \times \mathbb{R}_+$  satisfies

$$\limsup_{t \rightarrow \infty} \left( \int_0^\infty i(a, t) da + V(t) \right) > \gamma. \quad (5.11)$$

Moreover every solution  $(S, i, V, B)$  of (1.1)-(1.3) is bounded below. Indeed using (5.4) and (5.11), we are lead to

$$\begin{aligned} B(t) &\geq \frac{\sigma^{dir}(\Lambda_0 - \varepsilon\|b\|_\infty)}{\mu + m\varepsilon} \int_0^\infty \beta(a) i(a, t) da + \frac{\sigma^{ind}(\Lambda_0 - \varepsilon\|b\|_\infty)}{\mu + m\varepsilon} V(t) + \int_0^\infty \pi(a) b(a) i(a, t) da \\ &\geq \frac{\sigma^{dir} \beta_0 (\Lambda_0 - \varepsilon\|b\|_\infty)}{\mu + m\varepsilon} \int_0^\infty i(a, t) da + \frac{\sigma^{ind}(\Lambda_0 - \varepsilon\|b\|_\infty)}{\mu + m\varepsilon} V(t) + \pi_0 b_0 \int_0^\infty i(a, t) da \\ &\geq \left( \frac{\sigma^{dir} \beta_0 (\Lambda_0 - \varepsilon\|b\|_\infty)}{\mu + m\varepsilon} + \pi_0 b_0 \right) \int_0^\infty i(a, t) da + \frac{\sigma^{ind}(\Lambda_0 - \varepsilon\|b\|_\infty)}{\mu + m\varepsilon} V(t) \\ &\geq \min \left\{ \frac{\sigma^{dir} \beta_0 (\Lambda_0 - \varepsilon\|b\|_\infty)}{\mu + m\varepsilon} + \pi_0 b_0, \frac{\sigma^{ind}(\Lambda_0 - \varepsilon\|b\|_\infty)}{\mu + m\varepsilon} \right\} \left( \int_0^\infty i(a, t) da + V(t) \right). \end{aligned}$$

Thus

$$\limsup_{t \rightarrow \infty} B(t) \geq m_\varepsilon \gamma,$$

$$\text{with } m_\varepsilon = \min \left\{ \frac{\sigma^{dir} \beta_0 (\Lambda_0 - \varepsilon\|b\|_\infty)}{\mu + m\varepsilon} + \pi_0 b_0, \frac{\sigma^{ind}(\Lambda_0 - \varepsilon\|b\|_\infty)}{\mu + m\varepsilon} \right\}.$$

On the other hand, this implies

$$\begin{aligned} \limsup_{t \rightarrow \infty} \int_0^\infty \beta(a) i(a, t) da &= \limsup_{t \rightarrow \infty} \int_0^t \beta(a) B(t - a) \mathcal{F}(a) da \\ &\geq m_\varepsilon \gamma \int_0^\infty \beta(a) \mathcal{F}(a) da. \end{aligned}$$

Hence using the third equation in (1.1), we obtain

$$\frac{dV(t)}{dt} \geq (1-p)m_\varepsilon\gamma \int_0^\infty \beta(a)\mathcal{F}(a)da - \mu_V.$$

So

$$\limsup_{t \rightarrow \infty} V(t) \geq \frac{(1-p)m_\varepsilon\gamma}{\mu_V} \int_0^\infty \beta(a)\mathcal{F}(a)da.$$

This complete the proof. ■

The next result shows that system (1.1)-(1.3) has a global compact attractor.

**Lemma 5.2** *Assume that  $\mathcal{R}_0 > 1$ , then there exists a compact subset  $\mathcal{M}_0$  of  $X_0$  which is a global attractor for the solution semiflow  $\Psi$  of system (1.1)-(1.3) in  $X_0$ .*

**Proof.** Set

$$\Psi(t, S_0, i_0(\cdot), V_0) = (S(t), i(\cdot, t), V(t)),$$

$\Psi : [0, \infty) \times X_0 \rightarrow X_0$  with  $\Psi(t, \Psi(s, -)) = \Psi(t+s, -)$  for all  $t, s \geq 0$ , and  $\Psi(0, -)$  being the identity map. We aim to show that  $\Psi$  satisfies the assumptions Lemma 3.2.3 and Theorem 3.4.6 in [16]. To do so, split the solution semiflow  $\Psi$  in two components  $\Psi(t, x_0) = \widehat{\Psi}(t, x_0) + \widetilde{\Psi}(t, x_0)$  such that  $\widehat{\Psi}(t, x_0) \rightarrow 0$  as  $t \rightarrow \infty$  for every  $x_0 \in X_0$ , and for any fixed  $t$ , and any bounded set  $\mathcal{B}$  in  $X_0$ , the set  $\{\widetilde{\Psi}(t, x_0) : x_0 \in \mathcal{B}\}$  is precompact. The two summands are defined as follows

$$\widehat{\Psi}(t, S_0, i_0, V_0) = (0, \widehat{i}(\cdot, t), 0)$$

$$\widetilde{\Psi}(t, S_0, i_0, V_0) = (S(t), \widetilde{i}(\cdot, t), V(t)).$$

Notice that  $S(t)$  and  $V(t)$  satisfy the system (1.1)-(1.3) with  $i(a, t) = \widehat{i}(a, t) + \widetilde{i}(a, t)$ . The function  $\widehat{i}(a, t)$  is solution to

$$\begin{cases} \frac{\partial \widehat{i}}{\partial t} + \frac{\partial \widehat{i}}{\partial a} = -\eta(a)\widehat{i}, \\ \widehat{i}(0, t) = 0, \\ \widehat{i}(a, 0) = i_0(a), \end{cases} \quad (5.12)$$

and  $\widetilde{i}(a, t)$  is solution to

$$\begin{cases} \frac{\partial \widetilde{i}}{\partial t} + \frac{\partial \widetilde{i}}{\partial a} = -\eta(a)\widetilde{i}, \\ \widetilde{i}(0, t) = S(t) (\sigma^{dir} p \int_0^\infty \beta(a)\widetilde{i}(a, t)da + \sigma^{ind} V(t)) + \int_0^\infty \pi(a)b(a)\widetilde{i}(a, t)da, \\ \widetilde{i}(a, 0) = 0. \end{cases} \quad (5.13)$$

It is clear that  $\widehat{i}$  and  $\widetilde{i}$  are nonnegative. We set  $v(t) = \int_0^\infty \widehat{i}(a, t)da$ . Then

$$\begin{aligned} v'(t) &= \int_0^\infty \frac{\partial \widehat{i}}{\partial t}(a, t)da \\ &= \int_0^\infty \left( -\eta(a)\widehat{i}(a, t) - \frac{\partial \widehat{i}}{\partial a}(a, t) \right) da \\ &= - \int_0^\infty \eta(a)\widehat{i}(a, t)da \\ &\leq -\mu v(t). \end{aligned}$$

So  $v(t) \leq \left( \int_0^\infty i_0(a) da \right) e^{-\mu t}$ , and  $\lim_{t \rightarrow \infty} v(t) = 0$ . Thus  $\widehat{\Psi}(t, x_0) \rightarrow 0$  as  $t \rightarrow \infty$ , for every  $x_0 \in X_0$ . Now, it remains to show that the set  $\{\widehat{\Psi}(t, x_0) : x_0 \in X_0, t \text{ fixed}\}$  is precompact. We use the Fréchet-Kolmogorov Theorem (see [42]). In fact, first we have the family  $\{\Psi(t, x_0) : x_0 \in X_0, t \text{ fixed}\} \subset X_0$ . Notice that  $X_0$  is bounded. Therefore  $\{\Psi(t, x_0)\}$  is bounded for different values of initial conditions in  $X_0$ . On the other hand, from (5.13), it is easy to show that  $\widetilde{i}(a, t) = 0$  for  $a > t$ . hence the third condition of the Fréchet-Kolmogorov Theorem is satisfied. Finally to show that the second condition is fulfilled, we have to bound by a constant the  $L^1$ -norm of  $\frac{\partial \widetilde{i}}{\partial a}$ . In fact from (5.13), we have

$$\widetilde{i}(a, t) = \begin{cases} \widetilde{B}(t-a)\mathcal{F}(a) & \text{if } 0 \leq a < t, \\ 0 & \text{if } a > t \geq 0, \end{cases} \quad (5.14)$$

where

$$\widetilde{B}(t) = S(t) \left( \sigma^{dir} p \int_0^\infty \beta(a) \widetilde{B}(t-a) \mathcal{F}(a) da + \sigma^{ind} V(t) \right) + \int_0^\infty \pi(a) b(a) \widetilde{B}(t-a) \mathcal{F}(a) da. \quad (5.15)$$

Notice that for  $x_0 \in X_0$ ,  $\widetilde{B}(t)$  is bounded. In fact, from Theorem 2.2, we have that  $S(t)$  and  $V(t)$  are bounded. Thus from (5.15), there exist two positive constants  $C_1$  and  $C_2$ , which may depend of the bound of parameters and solutions such that

$$\widetilde{B}(t) \leq C_1 \int_0^t \widetilde{B}(t-a) da + C_2. \quad (5.16)$$

Moreover by differentiating  $\widetilde{B}(t)$ , it is easy to see that, there exist positive constants  $C_3$  and  $C_4$  depending of the bound of parameters as well as bound of the solutions such that

$$|\widetilde{B}'(t)| \leq C_3 \int_0^t |\widetilde{B}'(t-a)| da + C_4. \quad (5.17)$$

Using Gronwall's inequality, we have

$$\widetilde{B}(t) \leq C_2 e^{C_1 t} \quad \text{and} \quad \widetilde{B}'(t) \leq C_4 e^{C_2 t}. \quad (5.18)$$

Then from (5.14), we can write

$$\frac{\partial \widetilde{i}}{\partial a}(a, t) = \begin{cases} -\widetilde{B}'(t-a)\mathcal{F}(a) + \widetilde{B}(t-a)\mathcal{F}'(a) & \text{if } 0 \leq a < t, \\ 0 & \text{if } a > t \geq 0. \end{cases}$$

So

$$\begin{aligned} \int_0^\infty \left| \frac{\partial \widetilde{i}}{\partial a}(a, t) \right| da &\leq \int_0^\infty |\widetilde{B}'(t-a)| \mathcal{F}(a) da + \int_0^\infty \widetilde{B}(t-a) |\mathcal{F}'(a)| da \\ &\leq C_4 e^{C_2 t} \int_0^\infty \mathcal{F}(a) da + C_2 e^{C_1 t} \int_0^\infty |\mathcal{F}'(a)| da < C, \quad \text{for } t \text{ fixed.} \end{aligned}$$

Finally since

$$\int_0^\infty |\widetilde{i}(a+h, t) - \widetilde{i}(a, t)| da \leq \|\partial \widetilde{i} / \partial a\| |h| < C|h|. \quad (5.19)$$

Therefore, it follows that the integral (5.19) can be made arbitrary small in the family of functions. Thus all requirements of the Fréchet-Kolmogorov Theorem are satisfied. This complete the proof. ■

Now we are about to prove the Theorem 5.1

**Proof.** (Proof of Theorem) We apply Theorem 2.6 in [38]. We consider the solution semiflow  $\Psi$  on  $X_0$ . Let us consider the function

$$\phi : X_0 \rightarrow \mathbb{R}_+$$



as follows

$$\phi(\Psi(t, x_0)) = \int_0^\infty i(a, t) da + V(t). \quad (5.20)$$

Lemma 5.1 shows that the semiflow is uniformly weakly  $\phi$ -persistent. Lemma 5.2 shows that the solution semiflow has a global attractor  $\mathcal{M}_0$ . Since the solution semiflow is non negative for all times  $t \in \mathbb{R}_+$ , we have that for any  $r$  with  $t > r$  and using (5.6)

$$\int_0^\infty i(a, t) da + V(t) \geq V(r) e^{-\mu_V(t-r)}.$$

Therefore  $\int_0^\infty i(a, t) da + V(t) > 0$  for all  $t > r$  provided  $V(r) > 0$ . Thus from Theorem 2.6 in [38], it follows that the solution semiflow is uniformly strongly  $\phi$ -persistent. Hence there exists a constant  $\rho$  such that

$$\liminf_{t \rightarrow \infty} \phi(\Psi(t, x_0)) \geq \rho.$$

This complete the proof. ■

## 6 Numerical experiments

In this section, we present some numerical simulations to illustrate our theoretical results.

### 6.1 Model parameters

The list of the parameters of our model as well as their values are summarized in the table 1.

	Description	Dimension	Values	Sources
$\Lambda_0$	Recruitment rate	fish.day <sup>-1</sup>	274	fixed
$b(a)$	birth rate of an infected fish	day <sup>-1</sup>	$\frac{4}{365}$	[28]
$\theta$	Normalization param. for shedding rate	copies.day <sup>-3</sup> .fish <sup>-1</sup>	$10^4$	[43, 39]
$\tau_1$	Mean duration of the latency period	day	2	[10, 14, 37]
$\tau_2$	Mean duration of the infectious period	day	13	[37]
$\mu$	Natural mortality rate	day <sup>-1</sup>	$\frac{1}{365}$	[21, 41]
$\mu_V$	Mortality rate of virus	day <sup>-1</sup>	0.5	[29, 32]
$\delta_0$	Normalization param. for death rate	day <sup>-3</sup>	$1.63 \times 10^{-2}$	[14]
$a_1$	Mean delay between infection and onset of mortality	day	2	[10, 14]
$a_2$	Mean duration of disease related mortality	day	7	[10, 14]
$\sigma^{dir}$	Horizontal direct transmission rate	copies <sup>-1</sup>	$1.48 \times 10^{-9}$	estimated
$\sigma^{ind}$	Horizontal indirect transmission rate	copies <sup>-1</sup> .day <sup>-1</sup>	$0.75 \times 10^{-11}$	estimated
$\varepsilon$	Normalization param.	fish <sup>-1</sup>	$4 \times 10^{-5}$	[28]
$\pi_0$	Normalization param.	day <sup>-2</sup>	$\frac{1}{86}$	[12]
$\pi_1$	Mean delay between infection and laying being affected	day	2	[14, 39]
$\pi_2$	Mean duration of laying being affected	day	13	[14, 37]
$p$	Probability of disease transmission due to direct contact	none	0.01	fixed

Table 1: Baseline values of the model parameters.

Value of parameter  $\mu$  : following [21, 41], the natural mortality rate in tilapia is around  $1.14 \pm 0.36$  year<sup>-1</sup>, hence we take  $\mu = \frac{1}{365}$  day<sup>-1</sup>.

Value of parameter  $\Lambda_0$ : the recruitment is defined as the number of new young fish that enter a population in a given year, its value is very difficult to predict. Inter annual fluctuations often appear random or even chaotic, and may be superimposed on decadal trends and periods of high or low recruitment. Recruitment levels often are

poorly correlated with adult stock abundance, are vaguely related to fishing effort on adults and, in most cases, are unpredictable. This led fishery scientists to commonly refer to a deficient knowledge of causes of recruitment variability as the “recruitment problem” [17] (or the “central problem of fish population dynamics” [3]). This problem is also due to the difficulty to predict the number of fish larvae in one season that will survive and become juvenile fish in the next season. Since fish produce huge volumes of larvae, but the volumes are very variable and mortality is high, making good predictions difficult [3]. To test the routes of transmission in scenario (i), the recruitment rate was fixed to  $274 \text{ fish.day}^{-1}$ . In that case, the total size of fish converges towards the carrying capacity  $\Lambda_0/\mu = 10^5$  fish at the disease-free equilibrium. To simulate the longer term forecasting of epidemic in Scenario (ii), the recruitment rate was assumed to depend on the water temperature.

Value of  $b(\cdot)$ : A work conducted by Myers et al. [28], established that at low population sizes, the maximum annual reproductive rate for any species examined in their study is typically between 1 and 7 ( $\frac{1}{365}$  and  $\frac{7}{365} \text{ day}^{-1}$ ). They also find that this number relatively constant within species, has a relatively little variation among species. According to the fact that the disease duration is very small, we choose  $b(a)$  independent of the age of infection  $a$  and we estimate  $b(a) = \frac{4}{365} \text{ day}^{-1}$ , so that the birth rate  $b(a)$  lies between the values given above as in [28].

Values of  $\beta(\cdot)$ : we choose the shedding rate of infected fish with respect to age of infection  $a$  to be a function of the form

$$\beta(a) = \begin{cases} \theta(\tau_1 - a)(a - (\tau_1 + \tau_2)) & \text{if } a \in [\tau_1, \tau_1 + \tau_2], \\ 0 & \text{otherwise,} \end{cases} \quad (6.1)$$

where  $\tau_1$  (resp.  $\tau_2$ ) is the mean duration of the latency (resp. infectious) period and  $\theta$  is set to  $10^4 \text{ copies.day}^{-3} \cdot \text{fish}^{-1}$ , estimated from [39, 43]. According to [10, 14, 33, 36], the clinical signs appeared within 3 - 7 days post infection, but since under natural conditions, infected fish without clinical signs might transmit the virus to susceptible fish [31], hence  $\tau_1 = 2$  days. Moreover from [37], the level of viral load peaks at day 3 post infection and then gradually declines until its absence at day 15 post infection, Hence  $\tau_2 = 13$  days.

Values of  $\delta(\cdot)$ : we let

$$\delta(a) = \begin{cases} \delta_0(a_1 - a)(a - (a_1 + a_2)) & \text{if } a \in [a_1, a_1 + a_2], \\ 0 & \text{otherwise,} \end{cases} \quad (6.2)$$

where  $a_1$  (resp.  $a_2$ ) is the mean delayed time between infection and the onset of mass mortality (resp. the mean duration of mass mortality) and  $\delta_0$  is set to  $1.63 \times 10^{-2} \text{ day}^{-3}$ , so that as in [14], the maximal value of  $\delta$  equals **0.2**. Following [10, 14], the onset of mortality occurs from 1 - 5 days post infection and lasted until 9 - 11 days post infection, hence  $a_1 = 2$  days,  $a_2 = 7$  days.

Values of  $\pi(\cdot)$ : we consider  $\pi(a)$  on the form

$$\pi(a) = \begin{cases} \pi_0(\pi_1 - a)(a - (\pi_1 + \pi_2)) & \text{if } a \in [\pi_1, \pi_1 + \pi_2], \\ 0 & \text{otherwise,} \end{cases} \quad (6.3)$$

Here,  $\pi_0$  is a normalization parameter,  $\pi_1$  is the mean delay between the infection and the laying being affected,  $\pi_2$  is the mean duration of the laying being affected. At 2 - 9 days post infection, TiLV was detected from fish reproductive organs [12, 37, 39], so  $\pi_1 = 2$  days and we estimate  $\pi_2 = 13$  days. According to [12], 40 - 50% of the fertilized eggs exhibited positive reactivity to TiLV that was observed mainly within the eggs. Hence the normalization rate  $\pi_0$  is set to be  $\pi_0 = \frac{1}{86} \text{ day}^{-2}$ , so that the maximal value of  $\pi$  ( $\approx 49\%$ ), obtained at  $a = 8.5$  days lies between the range 40 - 50%.

Value of parameter  $\mu_V$ : thought the duration of survival of TiLV outside the host has not been determined [19], we estimate the mortality rate of virus using another disease called Koi Herpesvirus (KHV) occurring in carp fisheries and exhibiting almost the same behavior and mass mortality [29] as the TiLV. According to [29, 32], KHV lost infectivity within 2 - 3 days in natural environmental water. Hence we estimate  $\mu_V = 0.5 \text{ day}^{-1}$ .

Value of parameter  $p$ : concerning  $p$ , we may have two cases:

**Case 1:**  $p \gg (1 - p)$  This situation may occurs in high stock density fisheries, where the number of fish in pond is very high, and so the frequency of direct contacts between fish may also be high. Though excessive stocking density in fish culture operations has a negative influence on fish survival [1] and higher density of fish results in increased mortality rates, elevated viral loads and reduced body condition compared with fish in low density [40], this case is not recommended.

**Case 2:**  $(1 - p) \gg p$  This situation is the most common, there will be only few contacts between fish and the excretion of infectious fish will tend to go in the water. So only a very small quantity might be directly ingested by some susceptible fish. In that case the disease will be mainly transmitted indirectly through water. For the simulations, we assume that 1% of excreted pathogens by infectious fish is directly ingested by the susceptible fish, then  $p = 0.01$ .

Values of parameters  $\sigma^{dir}$  and  $\sigma^{ind}$ : the Horizontal direct transmission rate  $\sigma^{dir}$  was estimated to  $1.48 \times 10^{-9}$  copies $^{-1}$  and the Horizontal indirect transmission rate  $\sigma^{ind}$  to  $0.75 \times 10^{-11}$  copies $^{-1}$ .day $^{-1}$ . These values should be estimated by fitting the model with the experimental data. But unfortunately we have not data to estimate these parameters. Here, the values of  $\sigma^{dir}$  and  $\sigma^{ind}$  were calibrated in order to have a reproductive number  $\mathcal{R}_0^{Hdir}$  or  $\mathcal{R}_0^{Hind}$  very close to that estimated in [40], that was  $\mathcal{R}_0 = 2.60 \pm 0.16$ .

## 6.2 Numerical simulation

The numerical scheme is detailed in Appendix A and was implemented using Matlab (www.matlab.org). Because, we have not sufficient data on the evolution over day of the infected Tilapia in the literature to validate our model, we will simply explore the behaviour of our model with scenarios: (i) role of routes of transmission, (ii) longer term forecasting of epidemic spreading when the recruitment rate depending on the temperature. To simplify the graphical representation, we show the following quantities

$$E(t) = \int_0^{\tau_1} i(a, t) da, \quad I(t) = \int_{\tau_1}^{\tau_1 + \tau_2} i(a, t) da, \quad \text{and} \quad R(t) = \int_{\tau_1 + \tau_2}^{A_{max}} i(a, t) da.$$

### (i) role of routes of transmission

To test the role of routes of transmission, we use the values of parameters in Table 1 except for the transmission rates  $\sigma^{ind}$  and  $\sigma^{dir}$ .

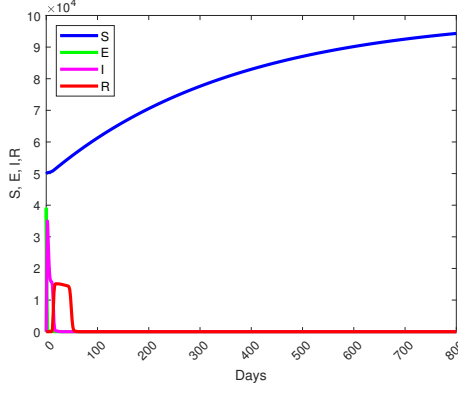
- To test the role of vertical transmission, we set  $\sigma^{dir} = \sigma^{ind} = 0$ . The initial conditions were fixed as follows:  $S_0 = 5 \times 10^4$ ,  $E(0) = 4 \times 10^4$ ,  $I(0) = 0$ ,  $R(0) = 0$ ,  $V(0) = 0$ ,  $\mathcal{R}_0 = \mathcal{R}_0^V = 0.0226$  and  $\mathcal{R}_0^{Hdir} = \mathcal{R}_0^{Hind} = 0$ . The Figures 1(a)-(b) show the evolution over days of the infected Tilapia and the pathogens. Since  $\mathcal{R}_0 < 1$ , one can see that the vertical transmission alone is almost insufficient to sustain epidemic growth, and epidemic will die out over time.
- To test the role of horizontal direct transmission, we set  $\sigma^{ind} = b(.) = 0$ . The initial conditions were fixed as follows:  $S_0 = 5 \times 10^5$ ,  $E(0) = 1000$ ,  $I(0) = 0$ ,  $R(0) = 0$ ,  $V(0) = 0$ ,  $\mathcal{R}_0 = \mathcal{R}_0^{Hdir} = 2.6158$  and  $\mathcal{R}_0^{Hind} = \mathcal{R}_0^V = 0$ . The Figure 1(c)-(d) show the evolution over day of the infected Tilapia and the pathogens when we only consider the horizontal direct transmission. Since  $\mathcal{R}_0 > 1$ , we can see that the system converges towards an endemic equilibrium and the disease becomes endemics. This confirm the theoretical results stated.
- To test the role of horizontal indirect transmission, we set  $\sigma^{dir} = b(.) = 0$ . The initial conditions were fixed as follows:  $S_0 = 5 \times 10^4$ ,  $E(0) = 4 \times 10^4$ ,  $I(0) = 0$ ,  $R(0) = 0$ ,  $V(0) = 10^6$ ,  $\mathcal{R}_0 = \mathcal{R}_0^{Hind} = 2.6246$  and  $\mathcal{R}_0^{Hdir} = \mathcal{R}_0^V = 0$ . The Figure 1(e)-(f) show the evolution over day of the infected Tilapia and the pathogens when we only consider the horizontal indirect transmission. As above,  $\mathcal{R}_0 > 1$  and the model converges towards an endemic equilibrium.

### (ii) Longer term forecasting of epidemic spreading

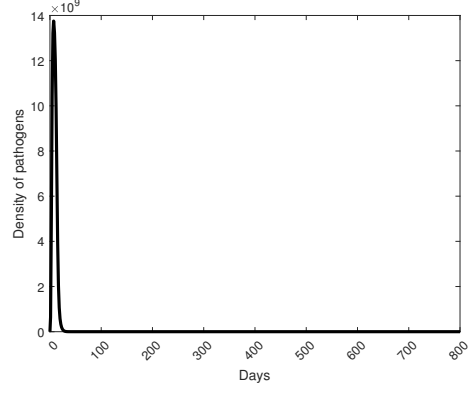
Firstly, the simulation was achieved with constants parameters in Table 1. The figures 2(a),(c) and (e) show the longer term forecasting of the infected Tilapia and the pathogens.

Secondly, the simulation was performed with constants parameters in Table 1 except for the recruitment rate  $\Lambda_0$  which varies with the water temperature. It is chosen on the form  $\Lambda(t) = T(t)\Lambda_0/T_0$ , where  $\Lambda_0 = 274$  and  $T_0$  is the average annual temperature  $T(t)$  given in [30]:

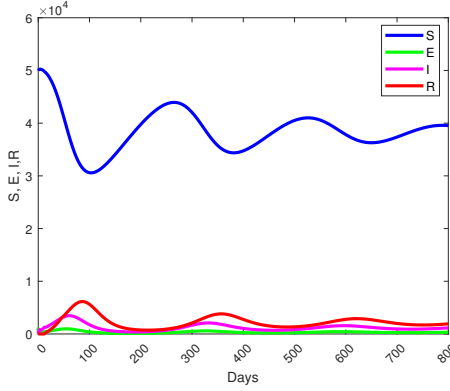
$$T(t) = T_0 - d \cos\left(\frac{2\pi t}{365}\right),$$



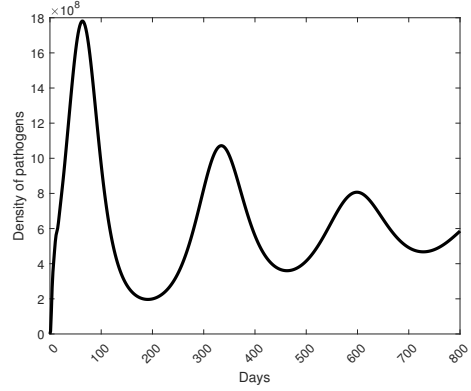
(a) Only vertical transmission



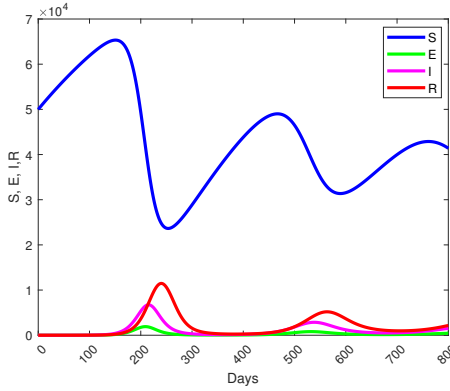
(b) Only vertical transmission



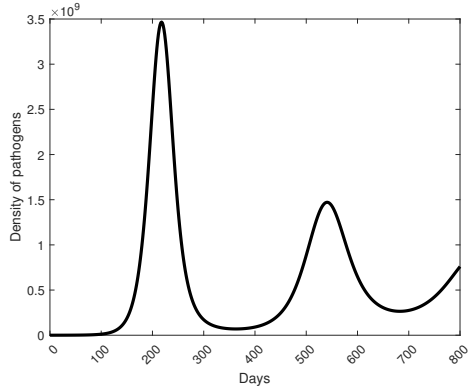
(c) Only horizontal direct transmission



(d) Only horizontal direct transmission



(e) Only horizontal indirect transmission



(f) Only horizontal indirect transmission

Figure 1: Results of simulations achieved with parameters in Table 1. (a) Only vertical transmission:  $\sigma^{dir} = \sigma^{ind} = 0$ , the initial conditions are  $S_0 = 5 \times 10^4$ ,  $E(0) = 4 \times 10^4$ ,  $I(0) = 0$ ,  $R(0) = 0$ ,  $V(0) = 0$ ,  $\mathcal{R}_0 = \mathcal{R}_0^V = 0.0226$  and  $\mathcal{R}_0^{Hdir} = \mathcal{R}_0^{Hind} = 0$ , (b) Only horizontal direct transmission:  $\sigma^{ind} = b(\cdot) = 0$ , the initial conditions are  $S_0 = 5 \times 10^5$ ,  $E(0) = 1000$ ,  $I(0) = 0$ ,  $R(0) = 0$ ,  $V(0) = 0$ ,  $\mathcal{R}_0 = \mathcal{R}_0^{Hdir} = 2.6158$  and  $\mathcal{R}_0^{Hind} = \mathcal{R}_0^V = 0$ , (c) Only horizontal indirect transmission:  $\sigma^{dir} = b(\cdot) = 0$ , the initial conditions are  $S_0 = 5 \times 10^4$ ,  $E(0) = 4 \times 10^4$ ,  $I(0) = 0$ ,  $R(0) = 0$ ,  $V(0) = 10^6$ ,  $\mathcal{R}_0 = \mathcal{R}_0^{Hind} = 2.6246$  and  $\mathcal{R}_0^{Hdir} = \mathcal{R}_0^V = 0$ .

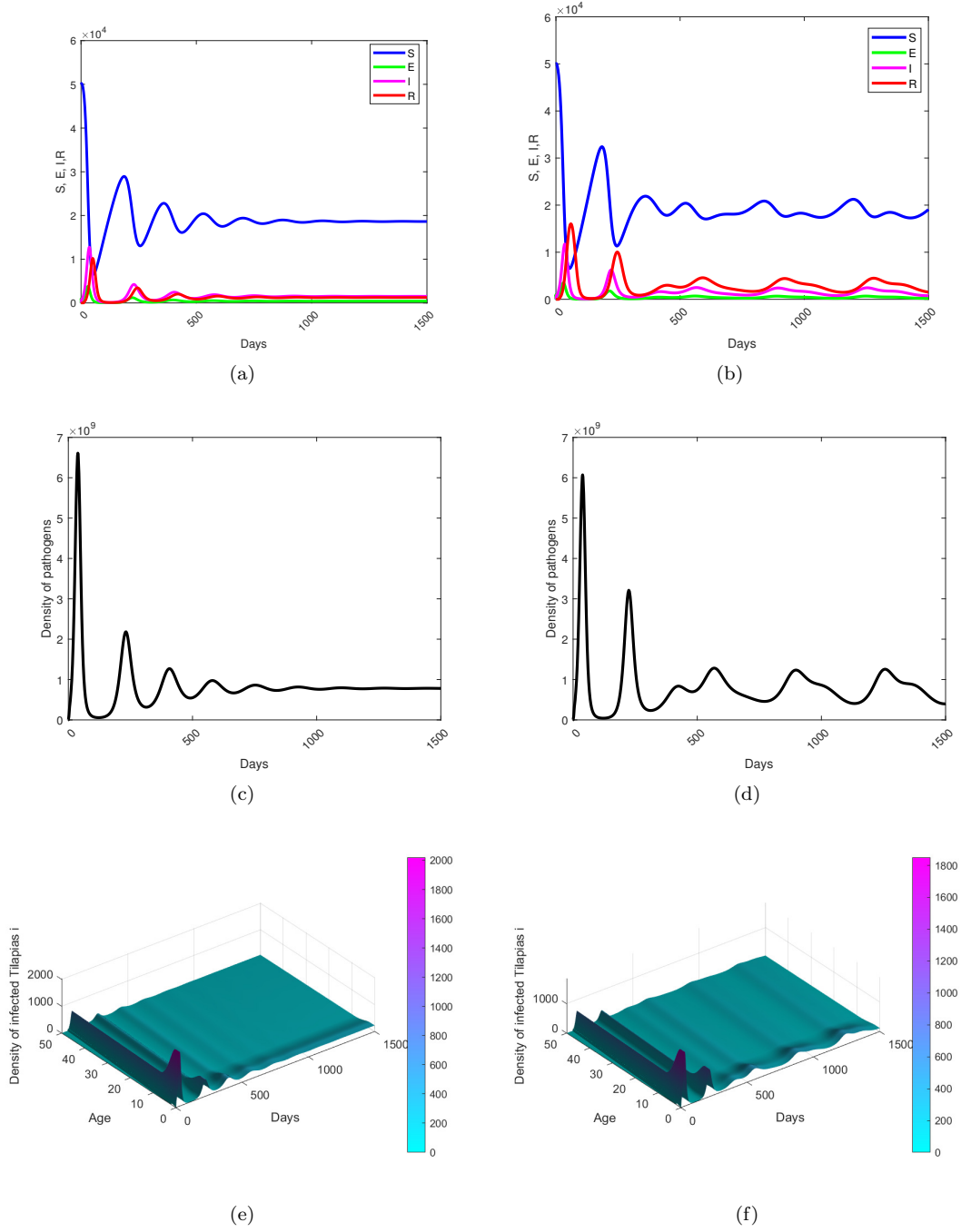


Figure 2: longer term forecasting of epidemic spreading. The simulations were achieved with parameters in Table 1 excepted the recruitment rate which depends on temperature. (a),(c) and (e) when the recruitment rate is fixed to  $\lambda_0 = 274$ . (b), (d) and (e) when the recruitment rate is depending on the temperature. The initial conditions are  $S_0 = 5 \times 10^4$ ,  $E(0) = 1000$ ,  $I(0) = 0$ ,  $R(0) = 0$ . The calculation of the basic reproduction number at the beginning of the outbreak with parameters in Table 1 gives  $\mathcal{R}_0 = 5.2630$  where  $\mathcal{R}_0^{H_{dir}} = 2.6158$ ,  $\mathcal{R}_0^{H_{ind}} = 2.6246$ ,  $\mathcal{R}_0^V = 0.0226$ .

with  $d$  representing the amplitude of the cosine function. Since the tilapia is a warm-water fish, its growth rate in optimal temperatures is in the range of  $24 - 32^\circ C$ . We choose  $T_0 = 28^\circ C$  and  $d = 8$ . The value of the basic reproduction number was computed at the beginning of the outbreak with constants parameters in Table 1. We find  $\mathcal{R}_0 = 5.2630$  where  $\mathcal{R}_0^{Hdir} = 2.6158$ ,  $\mathcal{R}_0^{Hind} = 2.6246$ ,  $\mathcal{R}_0^V = 0.0226$ . Since  $\Lambda(t)$  depends on time  $t \geq 0$ ,  $\mathcal{R}_0$  will depend on time and is called the net reproduction number.

The figures 2(b), (d) and (e) show the longer term forecasting of epidemic spreading when the recruitment rate is depending on the temperature.

## 7 Discussion and Conclusion

This work concerns a new model of TiLV occurring in a given tilapia's population. This model include both direct (fish-to-fish close contact) and indirect (through infected water) horizontal transmission and vertical transmission (fish to eggs). After proving the well posedness of our system, we compute the basic reproduction number and perform the stability analysis of the equilibria. We begin to show that the disease free equilibrium is globally asymptotically stable when,  $\mathcal{R}_0 < 1$ , while unstable when  $\mathcal{R}_0 > 1$ . Next, we compute the endemic equilibria and study its local stability when  $\mathcal{R}_0 > 1$ . Unfortunately under this latter condition, we were unable to prove mathematically the global stability of this equilibria. Nevertheless, we discuss the uniform persistence of the disease when  $\mathcal{R}_0 > 1$ . This means that the infectious individuals survive above a certain number for any initial infection number. Finally, we perform some numerical simulations to confirm our theoretical analysis.

While exploring the different scenarios of the TiLV transmission, we find that the vertical transmission alone is unable to sustain the disease. This could be explained by the relatively high cumulative mortality rate up to 100% in 5 – 10 days infected tilapia fry [39]. Furthermore, it appears that  $\mathcal{R}_0^V$  is very low with our estimated parameters. It could be higher, always less than 1 but very close to 1 if the birth rate of an infected fish  $b(a)$  were high. This may be due to the relatively short infectiousness period and no evidence of potential vertical transmission after the infectious period. Interestingly, when considering only the horizontal indirect transmission, the basic reproductive number ( $\mathcal{R}_0^{Hind} = 2.6158$ ) is close to that estimated in [40], that was  $\mathcal{R}_0 = 2.60 \pm 0.16$ , implicating that the epidemic of TiLV was spreading within tilapia population via cohabitation route and the incidence was increasing [40]. Furthermore, it appears that the horizontal direct and indirect transmissions are the main severe modes of transmission of the TiLV confirming that TiLV infection is mainly transmitted through cohabitation by waterborne route [14] and that the control of live fish movement must be regulated nationally across different regions as well as internationally across country borders in order to gradually reduce the burden of disease [34].

When the initial dose of environment infection  $V(0)$  is fixed to  $10^6$  copies, we observe that the kinetics of infection is lower compared to the case where 1000 exposed fish were introduced in the population. By comparing the figures 1(c)-(d) and 1(e)-(f), one can see that the direct or indirect horizontal transmission influences the kinetics of infection and the level of endemicity of infection. As late as 210 days post environmental infection with a dose,  $V(0) = 10^6$  copies, the peak of the first wave of indirect infection is reached (see Fig 1(b)-(c)). While when we consider only the direct transmission, the peak of the first wave of infection is reached about 50 days post infection, see Fig 1(d)-(e). From other simulations not shown in this paper, we have observed a peak of the first wave of infection at about 50-60 days post infection when the dose of environment infection is high ( $V(0) = 5 \times 10^9$  copies). By combining the three routes of infection, the figures 2 shows the longer term forecasting of epidemic spreading. More precisely, the figures 2(a),(c) and (e) show the forecasting when the recruitment rate is not depending on the temperature. While the figures 2(b),(d) and (f) show the prediction of epidemic when the recruitment rate varies when the water temperature varies with the seasons. This consideration arises from the fact that the tilapia is a warm-water fish and thus it is difficult to predict the number of fish larvae in one season that will survive and become juvenile fish in the next season provided that fish produce huge volumes of larvae, but the volumes are very variable and mortality is high, making good predictions difficult [3]. Hence the dependence on seasonality and other periodic changes of environmental conditions are important indexes in studying dynamics of TiLV infection and should be included in our future work. The Figure 2 shows the uniform persistence of the disease when  $\mathcal{R}_0 > 1$  confirming the theoretical results.

When looking at the figures, there are some oscillations on the patterns, this may be due to Hopf bifurcation occurring sometimes in age-structured models (see Liu et al. [22], Magal and Ruan [24]). Moreover the existence of

those oscillations demonstrates that age-structured models have more dynamic possibilities than the unstructured models [22].

Our model could be further improve, by including the space. Indeed, in a freshwater, fish wander randomly in their living space, in which different locations may have heterogeneous geographic situations and thus different contact fashions. So the transmission rates  $\sigma^{dir}$  and  $\sigma^{ind}$  may depend on the spatial position. This spatial factor may significantly impact the TiLV infectious transmission and dynamics. Thought the TiLV was recently discovered [14, 15], the amount of data available to estimate parameter values was limited, and our parameter estimates could be better if we have more data to estimate. Moreover, the recent studies showed that tilapia develop protective immunity including a humoral response following exposure to tilapia lake virus and upon re-infection, an increased antibody response occurred within 7 – 14 days, demonstrating that tilapia that survive TiLV infections develop humoral memory [37]. These studies suggest that tilapia mount antibody responses against TiLV that supports protective immunity to subsequent TiLV disease. We expect that with more biological data, our model developed here could be significantly improved further by also considering the immune status of fish, in order to potentially predict the impact of control strategies such as vaccination.

## Funding

This work was funded by a grant from the African Institute for Mathematical Sciences, [www.nexteinstein.org](http://www.nexteinstein.org), with financial support from the Government of Canada, provided through Global Affairs Canada, [www.international.gc.ca](http://www.international.gc.ca), and the International Development Research Centre, [www.idrc.ca](http://www.idrc.ca).

## Acknowledgments

The authors are very grateful to the editors and the anonymous referees for their valuable comments and suggestions, which helped us to improve the presentation of this work significantly.

## A Numerical method for solving model (1.1)-(1.3)

To compute the numerical solution, we use the forward/backward finite difference method for time and age to discretize equations (1.1)-(1.3) with the time interval being replaced by  $(0, T)$ , with  $T > 0$ . It is natural to make the following assumptions:

- $\beta(0) = 0$ ,
- There is  $A > 0$  such that  $\beta(a) = 0$ , for all  $a \geq A$ .

The time interval  $(0, T)$  is partitioned into subintervals  $(t_j, t_{j+1})$  with a time step  $\delta t = t_{j+1} - t_j$ ; for  $j = 0, 1, 2, \dots, N$ . The age interval  $(0, A)$  is partitioned into subintervals  $(a_k, a_{k+1})$  with an age step  $\delta a = a_{k+1} - a_k$ ; for  $k = 0, 1, 2, \dots, M$ . We set  $S_j = S(t_j)$ ,  $i_j^k = i(a_k, t_j)$ ,  $V_j = V(t_j)$ ,  $b^k = b(a_k)$ ,  $\pi^k = \pi(a_k)$ ,  $\beta^k = \beta(a_k)$  and  $\eta^k = \eta(a_k)$ . Next, we approximate the integrals  $\int_0^\infty \beta(a)i(a, t)da$  and  $\int_0^\infty \pi(a)b(a)i(a, t)da$  using the trapezoidal rule. That is

$$\int_0^\infty \beta(a)i(a, t)da \approx \delta a \sum_{k=1}^{M-1} \beta_k i(a_k, t),$$

and

$$\int_0^\infty \pi(a)i(a, t)da \approx \delta a \sum_{k=1}^{M-1} \pi^k i(a_k, t).$$

Hence the discrete form of the model (1.1)-(1.3) is given by

$$\frac{S_{j+1} - S_j}{\delta t} = \Lambda - \delta a \Lambda \sum_{k=1}^{M-1} \pi^k i_j^k - J_j S_j - \mu S_j, \quad (\text{A.1})$$

$$\frac{i_{j+1}^k - i_j^k}{\delta t} + \frac{i_j^k - i_j^{k-1}}{\delta a} = -\eta^k i_j^k, \quad (\text{A.2})$$

$$\frac{V_{j+1} - V_j}{\delta t} = (1-p)\delta a \sum_{k=1}^{M-1} \beta^k i_j^k - \mu_V V_j, \quad (\text{A.3})$$

$$i_j^0 = J_j S_j + \delta a \Lambda \sum_{k=1}^{M-1} \pi^k i_j^k, \quad (\text{A.4})$$

$$i_0^k = i_0(a_k). \quad (\text{A.5})$$

Where

$$J_j = \sigma^{dir} p \delta a \sum_{k=1}^{M-1} \beta^k i_j^k + \sigma^{ind} V_j. \quad (\text{A.6})$$

After some algebraic manipulation, equations (A.1)-(A.3) can be rewritten as

$$S_{j+1} = (1 - \delta t J_j - \mu \delta t) S_j + \Lambda \delta t - \delta a \delta t \Lambda \sum_{k=1}^{M-1} \pi^k i_j^k, \quad (\text{A.7})$$

$$i_{j+1}^k = \left(1 - \frac{\delta t}{\delta a} - \delta t \eta^k\right) i_j^k + \frac{\delta t}{\delta a} i_j^{k-1}, \quad (\text{A.8})$$

$$V_{j+1} = (1 - \mu_V \delta t) V_j + (1-p)\delta t \delta a \sum_{k=1}^{M-1} \beta^k i_j^k, \quad (\text{A.9})$$

## References

- [1] Ivan Abaho, Thaddeus Zaabwe, Andrew Izaara, Howard N Kasigwa, Norman Mushabe, Steven Byenkya, Mujibu Nkambo, Sylvester D Baguma, David LN Hafashimana, and Jackson Efitre. Effect of stocking density on growth and survival of nile tilapia (*oreochromis niloticus*, linnaeus 1758) under cage culture in lake albert, uganda. *International Journal of Fisheries and Aquaculture*, 12(2):26–35, 2020.
- [2] Eran Bacharach, Nischay Mishra, Thomas Briesse, Michael C Zody, Japhette Esther Kembou Tsofack, Rachel Zamostiano, Asaf Berkowitz, James Ng, Adam Nitido, André Corvelo, et al. Characterization of a novel orthomyxo-like virus causing mass die-offs of tilapia. *MBio*, 7(2), 2016.
- [3] ANDREW Bakun. Comparative studies and the recruitment problem: searching for generalizations. *CalCOFI Rep*, 26:30–40, 1985.
- [4] C. Beaumont, J.-B. Burie, A. Ducrot, and P. Zongo. Propagation of salmonella withinan industrial hen house. *SIAM Journal of Applied Mathematics*, 72(4):1113 – 1148, 2012.
- [5] B.K. Behera, P.K. Pradhan, T.R. Swaminathan, N. Sood, Prasenjit Paria, Abhishek Das, D.K. Verma, R. Kumar, M.K. Yadav, A.K. Dev, P.K. Parida, B.K. Das, K.K. Lal, and J.K. Jena. Emergence of tilapia lake virus associated with mortalities of farmed nile tilapia *oreochromis niloticus* (linnaeus 1758) in india. *Aquaculture*, 484:168 – 174, 2018.
- [6] Fred Brauer. Age of infection in epidemiology models. *Electronic Journal of Differential Equations (EJDE)[electronic only]*, 2005:29–37, 2005.



- [7] Fred Brauer, Zhisheng Shuai, and P van den Driessche. Dynamics of an age-of-infection cholera model. *Mathematical Biosciences and Engineering*, 10(5-6):1335–1349, 2013.
- [8] Liming Cai, Maia Martcheva, and Xue-Zhi Li. Epidemic models with age of infection, indirect transmission and incomplete treatment. *Discrete and Continuous Dynamical Systems-B*, 18(9):22 – 39, 2013.
- [9] Yongli Cai, Yun Kang, Malay Banerjee, and Weiming Wang. Complex dynamics of a host-parasite model with both horizontal and vertical transmissions in a spatial heterogeneous environment. *Nonlinear Analysis: Real World Applications*, 40:444 – 465, 2018.
- [10] Nguyen Dinh-Hung, Pattiya Sangpo, Thanapong Kruangkum, Pattanapon Kayansamruaj, Tilladit Rung-ruangkijkrai, Saengchan Senapin, Channarong Rodkhum, and Ha Thanh Dong. Dissecting the localization of tilapia tilapia virus in the brain of the experimentally infected Nile tilapia, *Oreochromis niloticus* (L.). *Journal of Fish Diseases*, 2021.
- [11] H.T. Dong, G.A. Ataguba, P. Khunrae, T. Rattanarojpong, and S. Senapin. Evidence of tilv infection in tilapia hatcheries from 2012 to 2017 reveals probable global spread of the disease. *Aquaculture*, 479:579 – 583, 2017.
- [12] H.T. Dong, S. Senapin, W. Gangnonngiw, V.V. Nguyen, C. Rodkhum, P.P. Debnath, J. Delamare-Deboutteville, and C.V. Mohan. Experimental infection reveals transmission of tilapia lake virus (tilv) from tilapia broodstock to their reproductive organs and fertilized eggs. *Aquaculture*, 515:734541, 2020.
- [13] H.T. Dong, S. Siriroob, W. Meemetta, W. Santimanawong, W. Gangnonngiw, N. Pirarat, P. Khunrae, T. Rattanarojpong, R. Vanichviriyakit, and S. Senapin. Emergence of tilapia lake virus in Thailand and an alternative semi-nested rt-pcr for detection. *Aquaculture*, 476:111 – 118, 2017.
- [14] Marina Eyngor, Rachel Zamostiano, J. E. Kembou Tsofack, Asaf Berkowitz, Hillel Bercovier, Simon Tinman, Menachem Lev, Avshalom Hurvitz, Marco Galeotti, Eran Bacharach, and Avi Eldar. Identification of a novel RNA virus lethal to tilapia. *Journal of Clinical Microbiology*, 52(12):4137 – 4146, 2014.
- [15] H W Ferguson, R Kabuusu, S Beltran, E Reyes, J A Lince, and J del Pozo. Syncytial hepatitis of farmed tilapia, *Oreochromis niloticus* (L.): a case report. *Journal of Fish Diseases*, 37(6):583–589, 2014.
- [16] Jack K Hale. *Asymptotic behavior of dissipative systems*. Number 25. American Mathematical Soc., 2010.
- [17] Edward D Houde. Recruitment variability. *Fish reproductive biology*, pages 98–187, 2016.
- [18] M.D. Jansen, H.T. Dong, and C.V. Mohan. Tilapia lake virus: a threat to the global tilapia industry? *Reviews in Aquaculture*, 11(3):725–739, 2019.
- [19] Mona Dverdal Jansen, Ha Thanh Dong, and Chadag Vishnumurthy Mohan. Tilapia lake virus: a threat to the global tilapia industry? *Reviews in Aquaculture*, 11(3):725–739, 2019.
- [20] Yang Jianxin, Qiu Zhipeng, and Li Xuezhi. Global stability of an age-structured cholera model. *Mathematical Biosciences and Engineering*, 11(3):641–665, 2014.
- [21] Samuel KK Amponsah, Berchie Asiedu, and Pierre Failler. Population parameters of *Oreochromis niloticus* (L) from a semi-open lagoon (Sakumo II), Ghana and its implications on management. *Egyptian Journal of Aquatic Biology and Fisheries*, 24(2):195–207, 2020.
- [22] Zhihua Liu, Pierre Magal, and Shigui Ruan. Oscillations in age-structured models of consumer-resource mutualisms. *Disc. Cont. Dyna. Systems-B*, 21:537–555, 2016.
- [23] P Magal, CC McCluskey, and GF Webb. Lyapunov functional and global asymptotic stability for an infection-age model. *Applicable Analysis*, 89(7):1109 – 1140, 2010.
- [24] Pierre Magal and Shigui Ruan. *Center manifolds for semilinear equations with non-dense domain and applications to Hopf bifurcation in age structured models*. American Mathematical Soc., 2009.

- [25] Pierre Magal and Shigui Ruan. *Theory and applications of abstract semilinear Cauchy problems*. Springer, 2018.
- [26] Maia Martcheva and Horst R Thieme. Progression age enhanced backward bifurcation in an epidemic model with super-infection. *Journal of Mathematical Biology*, 46(5):385 – 424, 2003.
- [27] Kizito K Mugimba, Mustapha Lamkhannat, Saurabh Dubey, Stephen Mutoloki, Hetron M Munangandu, and Øystein Evensen. Tilapia lake virus downplays innate immune responses during early stage of infection in Nile tilapia (*Oreochromis niloticus*). *Scientific reports*, 10(1):1–12, 2020.
- [28] Ransom A Myers, Keith G Bowen, and Nicholas J Barrowman. Maximum reproductive rate of fish at low population sizes. *Canadian Journal of Fisheries and Aquatic Sciences*, 56(12):2404–2419, 1999.
- [29] D Pokorova, T Vesely, V Piackova, S Reschova, J Hulova, et al. Current knowledge on koi herpesvirus (khv): a review. *Vet. Med. Czech*, 50(2005):4, 2005.
- [30] Matthew A. Rittenhouse, Crawford W. Revie, and Amy Hurford. A model for sea lice (*Lepeophtheirus salmonis*) dynamics in a seasonally changing environment. *Epidemics*, 16:8 – 16, 2016.
- [31] S. Senapin, K.U. Shyam, W.i Meemetta, T. Rattanarojpong, and H. T. Dong. Inapparent infection cases of tilapia lake virus (tlv) in farmed tilapia. *Aquaculture*, 487:51 – 55, 2018.
- [32] Tomoko Shimizu, Natsuko Yoshida, Hisae Kasai, and Mamoru Yoshimizu. Survival of koi herpesvirus (khv) in environmental water. *Fish Pathology*, 41(4):153–157, 2006.
- [33] W. Surachetpong, T. Janetanakit, P. Tattiyapong N. Nonthabenjawan, P. Sirikanchana, and A. Amonsin. Outbreaks of tilapia lake virus infection, Thailand, 2015–2016. *Emerging Infectious Diseases*, 23:1031–1033, 2017.
- [34] Win Surachetpong, Sri Rajiv Kumar Roy, and Pamela Nicholson. Tilapia lake virus: The story so far. *Journal of Fish Diseases*, 43(10):1115–1132, 2020.
- [35] Win Surachetpong, Nutthawan Nonthabenjawan Taveesak Janetanakit, Puntanat Tattiyapong, Kwanraewee Sirikanchana, and Alongkorn Amonsin. Outbreaks of tilapia lake virus infection, Thailand, 2015–2016. *Research Letter*, 23(6):1031–1033, 2017.
- [36] Puntanat Tattiyapong, Worawan Dachavichitlead, and Win Surachetpong. Experimental infection of tilapia lake virus (tlv) in Nile tilapia (*Oreochromis niloticus*) and red tilapia (*Oreochromis spp.*). *Veterinary Microbiology*, 207:170 – 177, 2017.
- [37] Puntanat Tattiyapong, Worawan Dechavichitlead, Thomas B Waltzek, and Win Surachetpong. Tilapia develop protective immunity including a humoral response following exposure to tilapia lake virus. *Fish & Shellfish Immunology*, 106:666–674, 2020.
- [38] Horst R. Thieme. Uniform persistence and permanence for non-autonomous semiflows in population biology. *Mathematical Biosciences*, 166(2):173 – 201, 2000.
- [39] Jidapa Yamkasem, Puntanat Tattiyapong, Attapon Kamlangdee, and Win Surachetpong. Evidence of potential vertical transmission of tilapia lake virus. *Journal of fish diseases*, 42(9):1293–1300, 2019.
- [40] Ying-Fei Yang, Tien-Hsuan Lu, Hsing-Chieh Lin, Chi-Yun Chen, and C. Liao. Assessing the population transmission dynamics of tilapia lake virus in farmed tilapia. *Journal of fish diseases*, 41:1439–1448, 2018.
- [41] Edwine Yongo and Nicholas Outa. Growth and population parameters of Nile tilapia, *Oreochromis niloticus* (L.) in the open waters of Lake Victoria, Kenya. *Lakes & Reservoirs: Research & Management*, 21(4):375–379, 2016.
- [42] Kosaku Yosida. *Functional analysis*, volume 123. Springer, 1988.

- [43] Weiwei Zeng, Yingying Wang, Xiaoyu Chen, Qing Wang, Sven M Bergmann, Ying Yang, Yahui Wang, Bo Li, Yuefeng Lv, Hua Li, et al. Potency and efficacy of vp20-based vaccine against tilapia lake virus using different prime-boost vaccination regimens in tilapia. *Aquaculture*, page 736654, 2021.