A MATHEMATICAL MODEL OF VERTICALLY TRANSMITTED VECTOR DISEASES

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ABSTRACT. A mathematical model of vector-borne infectious diseases is presented, which takes into account the local interactions between reservoirs and vectors, as well as the transmission from vectors to dilution hosts. In the model, vectors possess the ability to keep the virus within their own population through vertical transmission. The existence and the stability of disease free and endemic equilibria, together with the existence of backward bifurcation, are discussed.

1. Introduction

Vector-borne diseases are infectious diseases caused by pathogens which are transmitted by insects, bacteria and protozoa (called vectors), infected by biological agents (anthropoids). Malaria, dengue, yellow fever, St Louis Encephalitis and West Nile Fever (WNF) are examples of such vector-borne diseases whose vectors are mosquitoes. These infectious diseases can be transmitted to two types of host populations: reservoir (birds) and accidental (humans and horses).

In these diseases, the primary cycle (endemic) of the virus is characterized by mosquito-bird-mosquito transmission: adult mosquitoes become infected by biting viremic birds (reservoir). Once the virus is ingested, it spreads within the mosquito organism and is subsequently transmitted to the host vertebrate. The secondary epidemic cycle manifests itself when accidental hosts, such as humans or horses, enter the transmission cycle and are affected by the infection. The virus is not transmitted from person to person or from horse to horse via mosquito bites due to the low level of virus concentration in the blood.

In addition to the horizontal transmission cycle (vector-host-vector), some species of mosquitoes transmit the pathogen to their offspring (vertical transmission). Indeed, even in the absence of infected hosts, the disease is transmitted by the adult mosquitoes to eggs that survive the dry season and evolve as adult and infectious mosquitoes. Vertical transmission is observed, for example, in dengue virus transmitting mosquitoes such as Aedes aegypti, Aedes albopitus, the Culex species and other mosquito-borne flavivirus.

In recent years, various epidemic models have been proposed to describe and control the spread of infectious diseases such as malaria, dengue and WNF [see Wonham *et al.* (2004),

Bowman *et al.* (2005), Cruz-Pacheco *et al.* (2005), Maidana and Yang (2008a), Maidana and Yang (2008b), Cruz-Pacheco *et al.* (2009), Lashari and Zaman (2011), Maidana and Yang (2011), Chitnis *et al.* (2013), Asmaidi *et al.* (2014), Chen *et al.* (2016), and Blayneh (2017) and related references]. Models differ in the description of the interaction between vectors and host populations. Furthermore some authors incorporate the vertical transmission, others analyse the interaction of different species of birds with vectors, while others examine the effect of seasonality on disease transmission. These models describe the interaction between birds and mosquitoes and only a few studies have explored the transmission of WNF between birds, mosquitoes and humans [see, for example, Bowman *et al.* (2005) and Chen *et al.* (2016)].

In many mathematical models, the aquatic stage of the vector population consisting of eggs, larvae and pupae is not included in the transmission of the virus. First in Wonham *et al.* (2004) the classical SIR model for malaria transmission is extended to a model describing WNF cross-infection between birds and mosquitoes, including the larval stage of vector population. In order to incorporate the vertical transmission of the virus, in our model we insert the aquatic stage with its epidemiological classes. We also include the exposed class for the adult stage, accidental hosts, the exposed class in host populations, together with vertical transmission in vector populations. In this way, horizontal and vertical transmission of vector-borne diseases are investigated. Our model extends the work by Chen et al. proposed in Chen *et al.* (2016).

This paper is organized as follows. In Section 2 we derive a mathematical model for vector-borne epidemics, taking into account the interaction between birds and mosquitoes, mosquitoes and humans, as well as the transmission from adult mosquitoes and eggs. In Section 3 a linear stability analysis around the steady states is investigated both analytically and numerically together with the possible existence of a backward bifurcation. Finally, some concluding remarks are made in Section 4.

2. Mathematical Model

The populations involved in transmission are the vector population (mosquitoes) divided into aquatic (eggs, larvae, pupae) and adult stages. The reservoir host population are birds while humans constitute the dilution host population. We also assume that only adult mosquitoes are able to spread the disease.

Due to the short duration of the aquatic phase, there is no incubation period and no healing from the disease, so exposed and removed classes for the aquatic stage are not considered. For the same reason, the removed class for the adult stage is also not examined. While, for host populations, we take into account all the epidemiological classes of the SEIR model (susceptibles, exposed, infectives and removed).

Let $N_A(t) = S_A(t) + I_A(t)$ and $N_M(t) = S_M(t) + E_M(t) + I_M(t)$ be the total number of mosquitoes in the aquatic stage and adult stage, $N_B(t) = S_B(t) + E_B(t) + I_B(t) + R_B(t)$ and $N_H(t) = S_H(t) + E_H(t) + I_H(t) + R_H(t)$ the total number of birds and humans, respectively.

The host populations (birds and humans) have constant recruitment rates, γ_B and γ_H, and they decrease by natural and disease-induced death rates, d_B, d_H, δ_B and δ_H, respectively. The vector population increases through logistic growth, with r the intrinsic oviposition rate and γ the intrinsic maturation rate from aquatic stage to adult stage. The carrying

capacity in the aquatic stage, k_A, is defined as the available amount of breeding sites, while the carrying capacity in the adult stage, k_M, takes into account the fact that the mosquitoes cannot survive at high altitudes or temperatures. Therefore, the per-capita oviposition rate is given by $r(1 - \frac{N_A}{k_A})$ and the per-capita growth rate in the adult stage is $\gamma(1 - \frac{N_M}{k_M})$.

The vector populations (aquatic and adult stages) decrease by natural death rates, d_A and d_M, by the predation rate, m_A, and by chemical control rates, μ_A and μ_M, respectively. The infectious mosquitoes can transmit the virus to host populations and, via vertical transmission, to their eggs, but they can only be infected by viremic birds.

We assume that the mosquito biting rate to host populations is constant and we accept the validity of the 'frequency dependent law' for the infection force. The cross-infection between birds or humans and mosquitoes is modeled as $a_B \beta_{MB} \frac{I_M}{N_B} S_B$ or $a_H \beta_{MH} \frac{I_M}{N_H} S_H$, where a_B and a_H describe per capita biting rate of mosquitoes on birds and humans, respectively. The probabilities of transmission from mosquitoes to birds and humans are denoted by β_{MB} and β_{MH}, respectively. Similarly, the infection of mosquitoes through biting infectious birds is described by $a_B \beta_{BM} \frac{I_B}{N_B} S_M$, where β_{BM} is the probability of transmission from birds to mosquitoes.

To incorporate the vertical transmission, we assume that a fraction of newborns q, with $0 < q < 1$, is already infectious at birth. For this reason, the rate of newborns in the S_A class is $r(1 - \frac{N_A}{k_A})(S_M + E_M + (1-q)I_M)$, while the rate of newborns in I_A class is $r(1 - \frac{N_A}{k_A})q I_M$. Instead, the rate of newborns in the S_M class is $\gamma(1 - \frac{N_M}{k_M})S_A$ and similarly the rate of newborns in I_M class is given by $\gamma(1 - \frac{N_M}{k_M})I_A$. Adult mosquitoes, birds and humans shift from the exposed class to the infectious class with rates τ_M, τ_B and τ_H, respectively. Furthermore, infectious birds and infectious humans migrate into the corresponding removed class with rates ω_B and ω_H, respectively.

Under these assumptions, our model is described by the following system of thirteen ordinary differential equations

$$
\begin{cases}
\dot{S}_A = r\left(1 - \frac{N_A}{k_A}\right)\left[S_M + E_M + (1-q)I_M\right] - \alpha_A S_A, \\[4pt]
\dot{I}_A = r\left(1 - \frac{N_A}{k_A}\right)qI_M - \alpha_A I_A, \\[4pt]
\dot{S}_M = \gamma\left(1 - \frac{N_M}{k_M}\right)S_A - a_B\beta_{BM}\frac{I_B}{N_B}S_M - \alpha_M S_M, \\[4pt]
\dot{E}_M = a_B\beta_{BM}\frac{I_B}{N_B}S_M - \nu_M E_M, \\[4pt]
\dot{I}_M = \gamma\left(1 - \frac{N_M}{k_M}\right)I_A + \tau_M E_M - \alpha_M I_M, \\[4pt]
\dot{S}_B = \gamma_B - a_B\beta_{MB}\frac{I_M}{N_B}S_B - d_B S_B, \\[4pt]
\dot{E}_B = a_B\beta_{MB}\frac{I_M}{N_B}S_B - \alpha_B E_B, \\[4pt]
\dot{I}_B = \tau_B E_B - \nu_B I_B, \\[4pt]
\dot{R}_B = \omega_B I_B - d_B R_B, \\[4pt]
\dot{S}_H = \gamma_H - a_H\beta_{MH}\frac{I_M}{N_H}S_H - d_H S_H, \\[4pt]
\dot{E}_H = a_H\beta_{MH}\frac{I_M}{N_H}S_H - \alpha_H E_H, \\[4pt]
\dot{I}_H = \tau_H E_H - \nu_H I_H, \\[4pt]
\dot{R}_H = \omega_H I_H - d_H R_H,
\end{cases}
\tag{1}
$$

where, for the sake of simplicity, in (1) we set:

$$
\begin{aligned}
&\alpha_A = \gamma + d_A + m_A + \mu_A, \quad \alpha_M = d_M + \mu_M, \quad \nu_M = d_M + \mu_M + \tau_M, \\
&\nu_B = d_B + \delta_B + \omega_B, \qquad \alpha_B = d_B + \tau_B, \quad \nu_H = d_H + \delta_H + \omega_H, \quad \alpha_H = d_H + \tau_H.
\end{aligned}
\tag{2}
$$

Adding the appropriate equations in (1) it is possible to see that the growth of the whole populations of aquatic mosquitoes, adult mosquitoes, birds and humans satisfy the following equations

$$
\begin{cases}
\dot{N}_A = r\left(1 - \frac{N_A}{k_A}\right)N_M - \alpha_A N_A, \\[4pt]
\dot{N}_M = \gamma\left(1 - \frac{N_M}{k_M}\right)N_A - \alpha_M N_M, \\[4pt]
\dot{N}_B = \gamma_B - d_B N_B - \delta_B I_B, \\[4pt]
\dot{N}_H = \gamma_H - d_H N_H - \delta_H I_H.
\end{cases}
\tag{3}
$$

Thus, all total populations may vary in time. In particular, in the absence of disease, the population size N_B and N_H converge to the equilibrium value $\frac{\gamma_B}{d_B}$ and $\frac{\gamma_H}{d_H}$, respectively. Furthermore, from (3)$_{3,4}$, it follows that $limsup_{t\to\infty}N_B(t) \leq \frac{\gamma_B}{d_B}$ and $limsup_{t\to\infty}N_H(t) \leq \frac{\gamma_H}{d_H}$.

Since the first nine equations are independent of the other four, we can define the two vectors

$$
\mathbf{X}_1(t) = (S_A(t), I_A(t), S_M(t), E_M(t), I_M(t), S_B(t), E_B(t), I_B(t), R_B(t))^T
\tag{4}
$$

$$\mathbf{X}_2(t) = (S_H(t), E_H(t), I_H(t), R_H(t))^T \tag{5}$$

so system (1) can be recast in the following compact form:

$$\begin{cases} \dot{\mathbf{X}}_1 = \mathbf{F}_1(\mathbf{X}_1), \\ \dot{\mathbf{X}}_2 = \mathbf{F}_2(\mathbf{X}_1, \mathbf{X}_2), \end{cases} \tag{6}$$

where $\mathbf{F}_1$ and $\mathbf{F}_2$ can be easily deduced from system (1).

Now, denoting with $\mathbb{R}^n_+$ the positive orthant in $\mathbb{R}^n$ and with $\partial\mathbb{R}^n_+$ its boundary, the model $(6)_1$ is epidemiologically well-posed in the domain

$$D_1 = \{(S_A, I_A, S_M, E_M, I_M, S_B, E_B, I_B, R_B) \in \mathbb{R}^9_+ : S_A + I_A \le k_A, \ S_M + E_M + I_M \le k_M,$$

$$\frac{\gamma_B}{d_B + \delta_B} \le S_B + E_B + I_B + R_B \le \frac{\gamma_B}{d_B}\}, \tag{7}$$

while system $(6)_2$ is epidemiologically well-posed in the domain

$$D_2 = \{(S_H, E_H, I_H, R_H) \in \mathbb{R}^4_+ : \frac{\gamma_H}{d_H + \delta_H} \le S_H + E_H + I_H + R_H \le \frac{\gamma_H}{d_H}\}; \tag{8}$$

so the whole system (1) is biologically well-defined in $D = D_1 \times D_2$.

3. Mathematical Analysis

In this section, we determine the equilibria permitted by our model and study local stability.

3.1. Existence, unicity and boundedness of solutions.

Theorem 1. *For any initial condition which lies in D, system (1) has a unique solution that exists and remains in D, $\forall t \ge 0$. Furthermore, the compact D is a positively invariant set, which attracts all positive orbits in $\mathbb{R}^{13}_+$.*

Proof. . For all initial conditions belonging to D, the function $\mathbf{F} = (\mathbf{F}_1, \mathbf{F}_2)^T$ is locally lipschitzian in $\mathbf{X}(t) = (\mathbf{X}_1(t), \mathbf{X}_2(t))^T$, then the Cauchy-Lipschitz theorem ensures that system (1) admits a unique local solution.

Furthermore, from $(3)_{3,4}$ by applying the standard comparison theorem, it follows $N_B(t) \le \frac{\gamma_B}{d_B} \ \forall t > 0$ and $N_H(t) \le \frac{\gamma_H}{d_H} \ \forall t > 0$, if initially $N_B(0) \le \frac{\gamma_B}{d_B}$ and $N_H(0) \le \frac{\gamma_H}{d_H}$.

Then, D is positively invariant and all solutions of (1) are non-negative and bounded. $\square$

3.2. Disease Free Equilibrium. Basic Reproduction Number.

Theorem 2. *Between all possible equilibria, model $(6)_1$ admits the two equilibrium points with no disease in the population on $D_1 \cap \partial\mathbb{R}^9_+$*

$$\boldsymbol{E}_0 = (0,0,0,0,0,\frac{\gamma_B}{d_B},0,0,0)^T, \ \boldsymbol{E}_1 = (A_0,0,M_0,0,0,\frac{\gamma_B}{d_B},0,0,0)^T \tag{9}$$

which are the trivial and non-trivial one, where

$$A_0 = \frac{M_0 k_M \alpha_M}{\gamma(k_M - M_0)}, \quad M_0 = \frac{k_A k_M(r\gamma - \alpha_M \alpha_A)}{r(\gamma k_A + k_M \alpha_M)}. \tag{10}$$

The equilibrium $\mathbf{E}_1$ exists if the condition $r\gamma > \alpha_A \alpha_M$ holds.

Theorem 3. *The whole model (1) admits the following trivial and non trivial disease free equilibrium points*

$$\boldsymbol{P}_0 = (0,0,0,0,0,\frac{\gamma_B}{d_B},0,0,0,\frac{\gamma_H}{d_H},0,0,0)^T, \; \boldsymbol{P}_1 = (A_0,0,M_0,0,0,\frac{\gamma_B}{d_B},0,0,0,\frac{\gamma_H}{d_H},0,0,0)^T$$

(11)

on $D \cap \partial \mathbb{R}^{13}_+$.

It follows directly by substituting the equilibria (9) of $(6)_1$ into $(6)_2$.

In the following, we consider the more biologically realistic equilibrium $\mathbf{P}_1$. Its character is related to the basic reproduction number R_0 which is defined as the average number of new cases of an infection caused by an infected individual, in a population consisting of susceptibles only and where the disease is vertically transmitted.

Following Diekmann and Heesterbeek (2000) and Van den Driessche and Watmough (2002) and linearizing system (1) around $\mathbf{P}_1$, it is possible to compute the transmission matrix F, describing the production of new infections, and the transition matrix V, representing changes in state (including removal by death or the acquisition of immunity). The spectral radius ρ of the matrix $\mathbf{K}_L = FV^{-1}$ is the basic reproduction number

$$R_0 = \frac{1}{2}\left(q + \sqrt{q^2 + \frac{4M_0 a_B^2 d_B \beta_{BM} \beta_{MB} \tau_B \tau_M}{\gamma_B \alpha_M \alpha_B \nu_M \nu_B}}\right) = \frac{1}{2}\left(R_{vt} + \sqrt{R_{vt}^2 + 4R_{ht}}\right),$$

(12)

where

$$R_{vt} = q, \quad R_{ht} = R_H R_V = \frac{a_B^2 d_B \beta_{BM} \tau_B}{\alpha_B \nu_B} \frac{M_0 \beta_{MB} \tau_M}{\alpha_M \nu_M}.$$

(13)

We observe that R_0 consists of two contributions: the first is due to vertical transmission, whereas the second is caused by horizontal transmission. In particular, R_{ht} is the product of the number of new infections in the host population and the number of new infections in the vector population. As we expected, R_0 contains only terms related to the populations that spread and transmit the disease and not terms related to the human population that, in this model, constitutes the accidental host population.

The following local stability result about $\mathbf{P}_1$ holds (Van den Driessche and Watmough 2002):

Theorem 4. *The disease-free equilibrium point, $\boldsymbol{P}_1$ of the model (1), is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$, where R_0 is defined by (12).*

Theorem 4 shows that in the case $R_0 < 1$ the disease could be eliminated for a small initial value.

3.3. Endemic Equilibrium States. Bifurcation Analysis.

3.3.1. *Existence of endemic equilibria.* In this part we study the dynamic of system (1) as the threshold R_0 changes. We start calculating the endemic equilibrium points, which are solutions of the algebraic system

$$\begin{cases} \mathbf{F}_1(\mathbf{X}_1) = 0, \\ \mathbf{F}_2(\mathbf{X}_1, \mathbf{X}_2) = 0. \end{cases}$$

(14)

Firstly, from $(14)_1$ we deduce

$$I_A = \frac{q a_B d_B A_0 \beta_{BM} \tau_M I_B}{(1-q)\alpha_M \nu_M (\gamma_B - \delta_B I_B) + I_B a_B d_B \beta_{BM}((1-q)\alpha_M + \tau_M)},$$

$$E_M = \frac{(1-q) a_B d_B M_0 \alpha_M \beta_{BM} I_B}{(1-q)\alpha_M \nu_M (\gamma_B - \delta_B I_B) + I_B a_B d_B \beta_{BM}((1-q)\alpha_M + \tau_M)},$$

$$I_M = \frac{a_B d_B M_0 \beta_{BM} \tau_M I_B}{(1-q)\alpha_M \nu_M (\gamma_B - \delta_B I_B) + I_B a_B d_B \beta_{BM}((1-q)\alpha_M + \tau_M)}, \qquad (15)$$

$$S_A = A_0 - I_A, \quad S_M = M_0 - E_M - I_M, \quad S_B = \frac{\tau_B \gamma_B - \alpha_B \nu_B I_B}{\tau_B d_B}, \quad E_B = \frac{\nu_B}{\tau_B} I_B, \quad R_B = \frac{\omega_B}{d_B} I_B$$

while I_B is the positive solution of the following equation

$$f(I_B) = a_b I_B^2 + b_b I_B + c_b = 0, \qquad (16)$$

where

$$a_b = \alpha_B \delta_B \nu_B [a_B \beta_{BM} d_B((1-q)\alpha_M + \tau_M) - (1-q)\alpha_M \nu_M \delta_B],$$

$$b_b = 2(1-q)\alpha_B \alpha_M \gamma_B \delta_B \nu_B \nu_M - a_B d_B \alpha_B \beta_{BM} \gamma_B \nu_B((1-q)\alpha_M + \tau_M) + \qquad (17)$$

$$- a_B^2 d_B \beta_{BM} \beta_{MB} \tau_M \alpha_B \nu_B M_0,$$

$$c_b = -\gamma_B \alpha_M \alpha_B \nu_M \nu_B \gamma_B (1-q)(1-R_0).$$

Existence of endemic equilibrium requires that the roots of (16) are real and positive. Moreover, since S_B must be positive, we also have to impose the further condition $I_B < I_B^{**} = \frac{\tau_B \gamma_B}{\alpha_B \nu_B}$.

Let us denote with Δ_b the discriminant of (16). Solving $\Delta_b = 0$ in terms of R_0 we obtain the critical value

$$R_0^c = 1 + \frac{b_b^2}{4r(1-q)a_b \alpha_B \nu_B \gamma_B^2 \alpha_M \nu_M (\gamma k_A + k_M \alpha_M)}, \qquad (18)$$

so the following relations are verified:

$$\Delta_b < 0 \iff R_0 < R_0^c, \quad \Delta_b = 0 \iff R_0 = R_0^c, \quad \Delta_b > 0 \iff R_0 > R_0^c. \qquad (19)$$

The study of the solutions of (16) implies the following result.

Theorem 5. (1) *Let $a_b = 0$. Equation (16) is a linear equation with a unique solution $I_B = -\frac{c_b}{b_b}$. Then the system $(6)_1$ has a unique endemic equilibrium when $R_0 > 1$ and $b_b < 0$ and has no endemic equilibrium when $R_0 \leq 1$.*

    (2) *Let $a_b \neq 0$.*

        a) System (6_1) has a unique endemic equilibrium whenever $R_0 > 1$

        b) System (6_1) has a unique endemic equilibrium whenever $R_0 = 1$, $a_b < 0$ and $b_B > 0$

        c) System (6_1) has two endemic equilibria $\boldsymbol{E}_i$ when $R_0^c < R_0 < 1$, $a_b < 0$ and $b_b > 0$

        d) System (6_1) has a unique endemic equilibrium of multiplicity 2 when $R_0 = R_0^c$, $a_b < 0$ and $b_b > 0$

        e) System (6_1) has no endemic equilibria whenever $R_0 < R_0^c$ or whenever $R_0 \leq 1$ and $a_b > 0$

Proof. . Evaluating $f(I_B)$ at $I_B = I_B^{**}$ we have $f(I_B^{**}) < 0$.

(1) Let $a_b = 0$. This case happens when the disease-induced death rate $\delta_B = 0$ or $a_B \beta_{BM} d_B((1-q)\alpha_M + \tau_M) - (1-q)\alpha_M \nu_M \delta_B = 0$. Equation (16) has one positive solution $I_B = -\frac{c_b}{b_b}$ if $b_b < 0$. If $R_0 < 1$, then $f(0) < 0$. In this case there are no positive real solutions in the interval $[0, I_B^{**}]$. For $R_0 = 1$, from (15) we find the non-trivial DFE $\mathbf{E}_1$.

(2) Let $a_b \neq 0$.
    If $R_0 > 1$ than $f(0) > 0$. In this case there is always a unique positive real solution in the interval $[0, I_B^{**}]$. This solution is $I_B = \frac{-b_b + \sqrt{\Delta_b}}{2a_b}$ if $a_b > 0$ and $I_B = \frac{-b_b - \sqrt{\Delta_b}}{2a_b}$ if $a_b < 0$. If $R_0 = 1$ there is a positive solution $I_B = -\frac{b_b}{a_b}$ if and only if $a_b < 0$ and $b_b > 0$ (otherwise there are no positive solutions in the interval $[0, I_B^{**}]$). If $R_0 < 1$ then $f(0) < 0$. When $a_b > 0$ equation (16) has no positive solutions in the interval $[0, I_B^{**}]$. When $a_b < 0$, $b_b > 0$ and $\Delta_b > 0$ there are two positive real solutions $I_B^1 = \frac{-b_b - \sqrt{\Delta_b}}{2a_b}$ and $I_B^2 = \frac{-b_b + \sqrt{\Delta_b}}{2a_b}$ of equation (16). These solutions coalesce if $\Delta_b = 0$.

$\square$

To each endemic equilibrium state for the system $(6)_1$, solution of the equation $(14)_1$, there is a corresponding equilibrium state for system $(6)_2$. Known I_M^i from Theorem 5 and solving equation $(14)_2$ in terms of I_H, we obtain the following relations

$$S_H = \frac{\tau_H \gamma_H - \alpha_H \nu_H I_H}{\tau_H d_H}, \quad E_H = \frac{\nu_H}{\tau_H} I_H, \quad R_H = \frac{\omega_H}{d_H} I_H, \tag{20}$$

in which the endemic value I_H is the positive solution of

$$g(I_H) = a_h I_H^2 + b_h I_H + c_h = 0 \tag{21}$$

where

$$\begin{aligned}
a_h &= \alpha_H \delta_H \nu_H, \\
b_h &= -(\alpha_H \gamma_H \nu_H + I_M^i a_H \beta_{MH} \alpha_H \nu_H), \\
c_h &= a_H \beta_{MH} \tau_H \gamma_H I_M^i.
\end{aligned} \tag{22}$$

We set $\Delta_h = b_h^2 - 4a_h c_h > 0$ which ensures that the solutions of the equation (21) are real. Since $S_H > 0$, then $I_H < I_H^{**} = \frac{\tau_H \gamma_H}{\alpha_H \nu_H}$. Because of $a_h > 0$ and $c_h > 0$, equation (21) has a unique positive solution $I_H = \frac{-b_h - \sqrt{\Delta_h}}{2a_h}$ in the interval $[0, I_H^{**}]$.

Now, denoting as $\mathbf{P}^* = (S_A^*, I_A^*, S_M^*, E_M^*, I_M^*, S_B^*, E_B^*, I_B^*, R_B^*, S_H^*, E_H^*, I_H^*, R_H^*)$ a generic endemic equilibrium point of system (1), we evaluate its Jacobian matrix

$$J(\mathbf{P}_*) = \begin{pmatrix} A_{11} & 0 \\ A_{21} & A_{22} \end{pmatrix}, \tag{23}$$

where A_{11} and A_{22} are the Jacobian matrices of the two partial systems in (6) evaluated at $\mathbf{E}^* = (S_A^*, I_A^*, S_M^*, E_M^*, I_M^*, S_B^*, E_B^*, I_B^*, R_B^*)$ and $\mathbf{U}^* = (S_H^*, E_H^*, I_H^*, R_H^*)$ respectively, while A_{21} is a matrix that takes into account the interactions between the two partial systems.

Applying the Routh-Hurwith criterion to the matrix

$$
A_{22} = \begin{pmatrix}
-d_H - \frac{a_H \beta_{MH} I_M^* (E_H^* + I_H^* + R_H^*)}{(N_H^*)^2} & \frac{a_H \beta_{MH} I_M^* S_H^*}{(N_H^*)^2} & \frac{a_H \beta_{MH} I_M^* S_H^*}{(N_H^*)^2} & \frac{a_H \beta_{MH} I_M^* S_H^*}{(N_H^*)^2} \\
\frac{a_H \beta_{MH} I_M^* (E_H^* + I_H^* + R_H^*)}{(N_H^*)^2} & -\alpha_H - \frac{a_H \beta_{MH} I_M^* S_H^*}{(N_H^*)^2} & -\frac{a_H \beta_{MH} I_M^* S_H^*}{(N_H^*)^2} & -\frac{a_H \beta_{MH} I_M^* S_H^*}{(N_H^*)^2} \\
0 & \tau_H & -\nu_H & 0 \\
0 & 0 & \omega_H & -d_H
\end{pmatrix},
$$

(24)

we proved that all eigenvalues of A_{22} have negative real parts. Following this, the stability of the equilibrium $\mathbf{P}^*$ depends only on the eigenvalues of the matrix A_{11}. This means that the system $(6)_1$, which describes the primary transmission of disease between birds and mosquitoes, adult mosquitoes and the aquatic stage, determines the stability of any arbitrary equilibrium point of the whole system (1). To conclude this Section, we remark that the stability of the endemic equilibria is determined by the eigenvalues of the matrix A_{11}. However, since it is difficult to determine their signs analytically, in Section 3.4 we will examine the possible dynamical behaviors for the whole system (1) by means of numerical simulations.

3.3.2. *Backward Bifurcation Analysis*.

In this section we use analytical and numerical techniques to assess the directions of bifurcations. Theorem 5 shows the possibility of backward bifurcation in model (1) when $R_0^c < R_0 < 1$, $a_b < 0$ and $b_b > 0$. Backward bifurcation is a phenomenon where two endemic equilibria, one stable and the other unstable co-exist along with the disease-free equilibrium for $R_0 < 1$. The existence of a backward bifurcation indicates that reduction of the epidemiology threshold, R_0, below unity is simply not a sufficient condition for disease control. We establish that once the epidemiology threshold R_0 is reduced below a critical value R_0^c, under some conditions, the disease could be eliminated for any initial size.

To do so, we chose as the bifurcation parameter the transmission probability from mosquitoes to birds β_{MB}^*, obtained by solving for β_{MB} from $R_0 = 1$:

$$
\beta_{MB}^* = \frac{(1-q)\gamma_B \alpha_B \alpha_M \nu_B \nu_M}{a_B^2 d_B M_0 \beta_{BM} \tau_B \tau_M}
$$

(25)

Let $\mathbf{J}^*$ be the Jacobian matrix of the system $(6)_1$ evaluated at the DFE $\mathbf{E}_1$ and at the bifurcation value β_{MB}^*. It has a simple eigenvalue with zero real part and the other eigenvalues with negative real parts, so we can use the 'center manifold theory'(Diekmann and Heesterbeek 2000) to analyze the dynamics of the model (1) near the criticality $\beta_{MB} = \beta_{MB}^*$ and apply a theorem proved by Castillo-Chavez and Song (2004) that states the conditions for the existence of backward bifurcation. Let $\mathbf{v} \equiv (v_i)$ and $\mathbf{w} \equiv (w_i)$ be the left and right eigenvectors corresponding to the zero eigenvalue of the Jacobian matrix $\mathbf{J}^*$, respectively,

$$
\begin{aligned}
& v_1 = 0, && v_2 = \frac{I_M \gamma \alpha_M (r k_M + \alpha_A k_A)}{r \alpha_A (\gamma k_A + \alpha_M k_M)}, && v_3 = 0, \\
& v_4 = \frac{\tau_M I_M}{\nu_M}, && v_5 = I_M, && v_6 = 0, \\
& v_7 = \frac{a_B d_B \beta_{BM} \tau_B \tau_M M_0}{\alpha_B \gamma_B \nu_B \nu_M}, && v_8 = \frac{a_B d_B \beta_{BM} \tau_M M_0}{\gamma_B \nu_B \nu_M}, && v_9 = 0,
\end{aligned}
$$

(26)

$$w_1 = -I_A, \qquad w_2 = I_A, \qquad w_3 = -\frac{\gamma(rk_M+\alpha_A k_A)([(1-q)\alpha_M+\tau_M]I_A}{qr\tau_M(\gamma k_A+\alpha_M k_M)},$$

$$w_4 = \frac{\alpha_M\gamma(rk_M+\alpha_A k_A)I_A}{qr\tau_M(\gamma k_A+\alpha_M k_M)}, \quad w_5 = \frac{\gamma(rk_M+\alpha_A k_A)I_A}{qr(\gamma k_A+\alpha_M k_M)}, \quad w_6 = -\frac{\alpha_B\alpha_M\gamma_B v_B v_M(1-q)I_A}{qa_B d_B^2 A_0 \beta_{BM}\tau_B\tau_M},$$

$$w_7 = \frac{\alpha_B\gamma_B v_B v_M(1-q)I_A}{qa_B d_B A_0 \beta_{BM}\tau_B\tau_M}, \quad w_8 = \frac{\alpha_B\gamma_B v_M(1-q)I_A}{qa_B d_B A_0 \beta_{BM}\tau_M}, \quad w_9 = \frac{\alpha_B\gamma_B v_M(1-q)\omega_B I_A}{qa_B d_B^2 A_0 \beta_{BM}\tau_M}.$$
$$(27)$$

Let $f_k(\mathbf{X}_1,\phi)$ be the k-th component of vector $\mathbf{F}_1$, x_k the k-th component of vector state $\mathbf{X}_1$ and $\phi = \beta_{MB} - \beta_{MB}^*$. The model (1) exhibits a backward bifurcation at $R_0 = 1$ ($\phi = 0$) if the following coefficients (Castillo-Chavez and Song 2004)

$$a_1 = \sum_{k,i,j=1}^9 v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(\mathbf{E}_1,0) = B_1 - B_2,$$
$$a_2 = \sum_{k,i=1}^9 v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(\mathbf{E}_1,0) = v_7 w_5 a_B,$$
$$(28)$$

are positive, where

$$B_1 = -2v_2 w_1 w_5 \frac{qr}{k_A} - 2v_5 w_2 w_3 \frac{\gamma}{k_M} > 0,$$

$$B_2 = 2v_2 w_2 w_5 \frac{qr}{k_A} - 2v_4 w_3 w_8 \frac{a_B\beta_{BM}d_B}{\gamma_B} + 2v_4 w_8 \frac{a_B\beta_{BM}d_B^2 M_0}{\gamma_B^2}(w_6+w_7+w_8+w_9)+ \qquad (29)$$

$$+2v_5 w_2 \frac{\gamma}{k_M}(w_4+w_5) + 2v_7 w_5 \frac{a_B\beta_{MB}^* d_B}{\gamma_B}(w_7+w_8+w_9) > 0.$$

The coefficient a_2 is always positive, but for the other coefficient we have to impose the further condition

$$B_1 > B_2. \qquad (30)$$

Thus, the following result is established.

Theorem 6. *Model (1) exhibits a backward bifurcation at $R_0 = 1$ ($\beta_{MB} = \beta_{MB}^*$) whenever the inequality (30) holds. If the reversed inequality holds, then the bifurcation at $R_0 = 1$ is forward.*

Thus the backward bifurcation scenario involve the existence of a subcritical transcritical bifurcation at $R_0 = R_0^c < 1$. The qualitative bifurcation diagrams describing two types of bifurcation al $R_0 = 1$ are depicted in Figure 1.

3.4. Numerical Simulations. In the subsequent discussion observations are made on the nature of the stability of each fixed point through numerical simulation. These simulations are made for both primary and secondary transmission cycle.

The case $R_0 < 1$. System (1) has a disease-free equilibrium $\mathbf{P}_1$. We show three numerical simulations corresponding to different initial data sets with $R_0 = 0.854753$. Figure 2 illustrates that each solution is close to the DFE. The occurrence of the backward bifurcation can be also seen in Figure 3, where R_0 is less than the transcritical bifurcation threshold $R_0 = 1$, but the solution of the system $(6)_1$ can approach either the endemic equilibrium point or the DFE point, depending on initial condition values.

The case $R_0 > 1$. System (1) has a endemic point when $R_0 > 1$ which can be shown by simulation. The endemic value is obtained using the parameter values listed in Figure 1-(b) with $a_B = 3.9$. So we find an endemic equilibrium point $\mathbf{P}^* = (S_A^*, I_A^*, S_M^*, E_M^*, I_M^*, S_B^*, E_B^*, I_B^*, R_B^*, S_H^*, E_H^*, I_H^*, R_H^*)$ with $R_0 = 6.31514$. If we consider the Jacobian matrix of model (1), evaluated in $\mathbf{P}^*$, all eigenvalues have a negative real part, so we have the local linear stability

of the endemic equilibrium point. Figure 4 shows the behavior of each population in the existing state of the disease. We see that, after an initial oscillating trend, each solution riches its endemic value.

4. Conclusions

An autonomous differential equation system for infectious diseases dynamics, which incorporates vertical transmission and logistic growth for vector population is considered. We derived an explicit formula for the basic reproductive number R_0, investigated the stability of DFE and the existence of an endemic equilibrium state. A detailed analysis of the model, based on the use of center manifold theory, shows the presence of the phenomenon of backward bifurcation, where two stable equilibria co-exist, when the associated basic reproduction number is less than unity. We compiled three parameter ranges: one representing the case $R_0 < 1$, one representing the case $R_0 > 1$ and finally a suitable set of parameters for to exhibit backward bifurcation. The presence of vertical transmission makes the model more realistic, as it takes into account that some eggs may already be infected at birth. The introduction of carrier capacity in the vector population places a limit on the number of existing mosquitoes: a lesser spread of the virus is evident and a reduction in the time necessary to reach equilibrium.
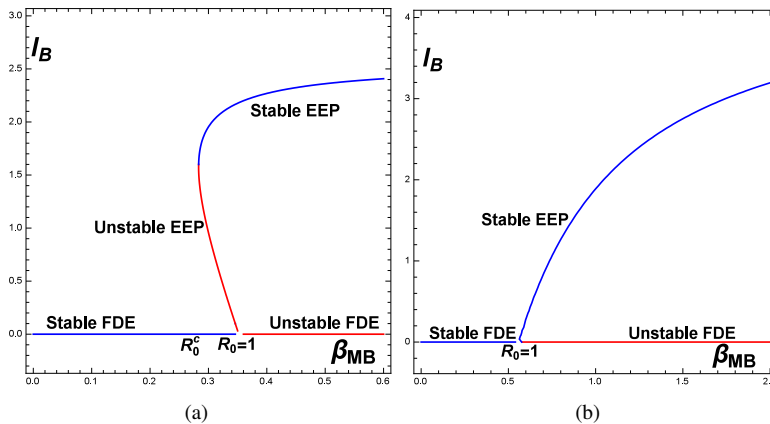
FIGURE 1. Bifurcation diagram showing the equilibrium value of the Infective Birds as the Reproduction Number is varied. (a) Backward bifurcation. Suitable set of parameters value for the exhibition of backward bifurcation d_A=0.056, k_A=1000, $r = 0.5$, q=0.2, μ_A=0, m_A=0, d_M=0.0286, $k_M = 500$, γ=0.2, $\tau_M = 0.143$, μ_M=0, d_B=0.003, γ_B=3.1, β_{BM}=0.9, δ_B=0.9, τ_B=0.6, ω_B=0.3, a_B=0.5. Two endemic equilibrium points coexist for value of $R_0 \in (0.908433, 1)$. (b) Forward bifurcation corresponding to the parameters value d_A=0.056, k_A=100, $r = 0.1$, q=0.1, μ_A=0, m_A=0, d_M=0.0286, k_M=25, γ=0.2, $\tau_M = 0.143$, μ_M=0, d_B=0.02, γ_B=2.1, β_{BM}=0.4, δ_B=0.1, τ_B=0.2, ω_B=0.3, a_B=0.5, d_H=9 × 10^{-4}, γ_H=5 × 10^{-2}, β_{MH}=0.5, δ_H=0.001, τ_H=0.25, ω_H=0.143, a_H=0.143.

$S_M(t), S_B(t), S_H(t)$
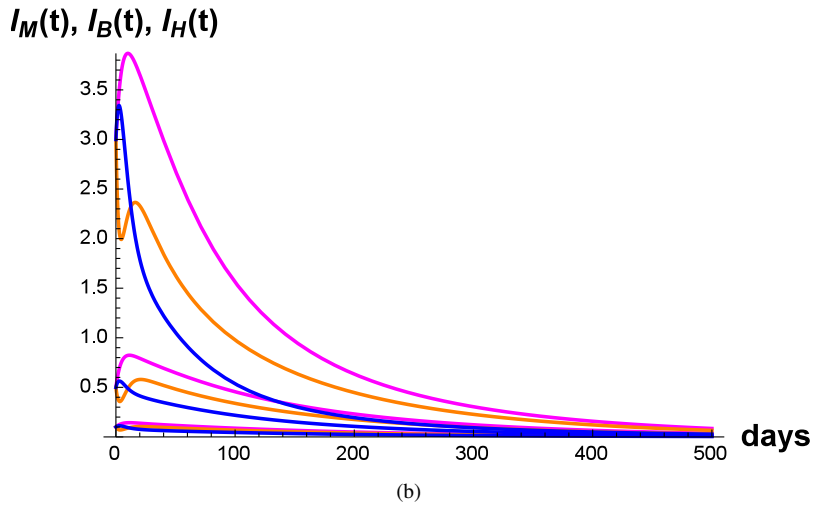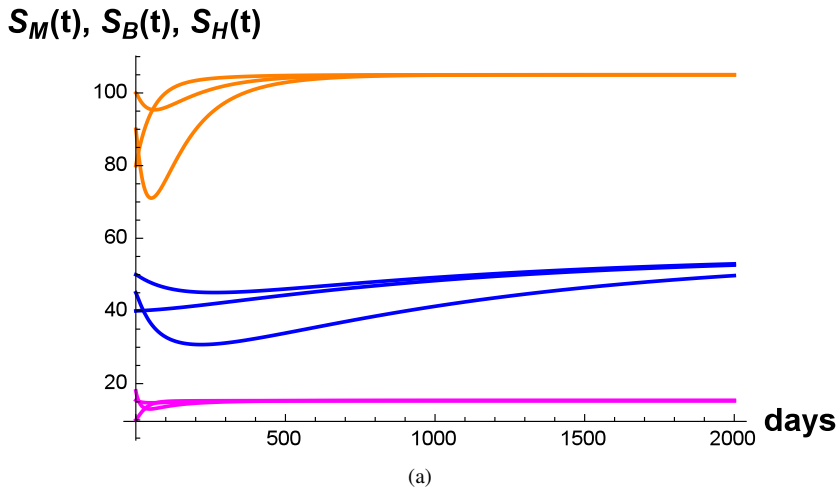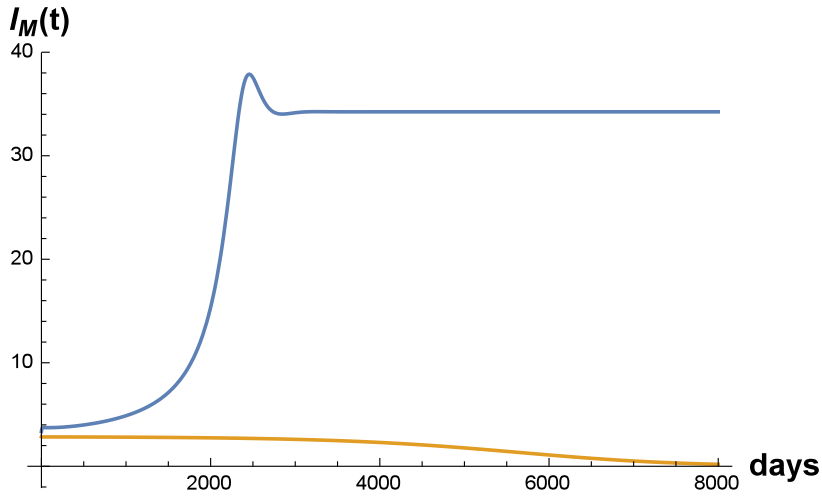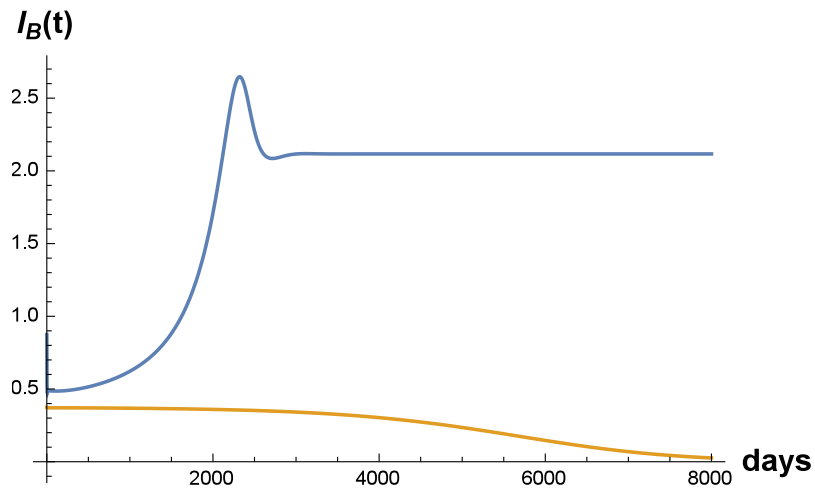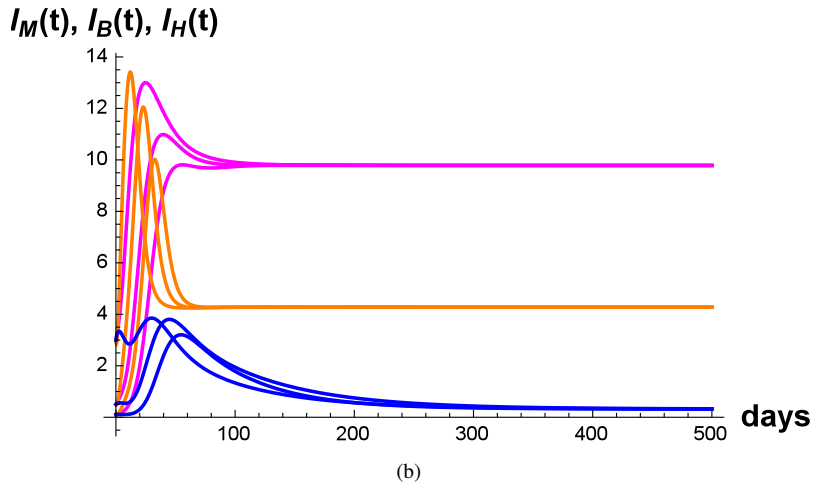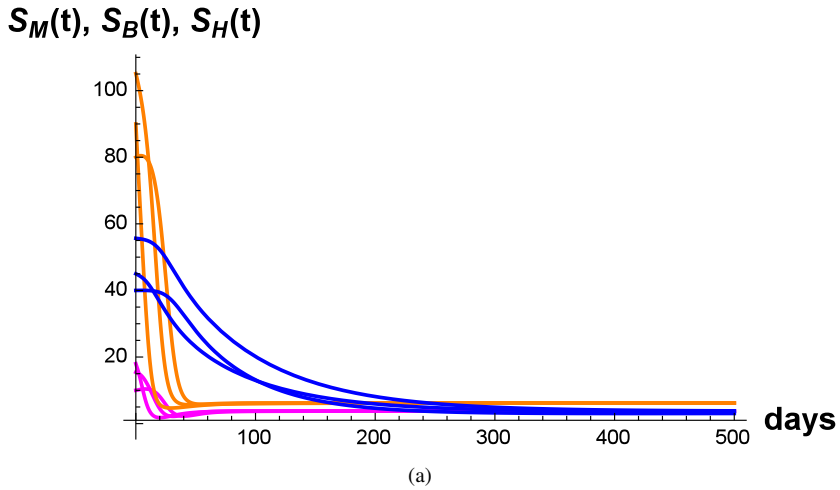

(a)

$I_M(t), I_B(t), I_H(t)$

(b)

FIGURE 2. Evolution over time of the susceptible (a) and infective (b) classes of mosquito (magenta line), bird (orange line) and human (blue line) populations, corresponding to different initial condition values and parameter values listed in Fig.1-(b) with $\beta_{MB} = 0.7$. (a)-(b) show that system (1) has only one disease-free equilibrium $\mathbf{P}_1 = (5, 65771, 0, 15, 3257, 0, 0, 105, 0, 0, 0, 55.5556, 0, 0, 0)$ and it is locally asymptotically stable.

(a)

(b)

FIGURE 3. Solutions regarding model $(6)_1$ of the number of infectious adult mosquitoes, I_M, and the number of infectious birds, I_B, for parameter values given in the bifurcation diagram (Fig. 1-(a)), with $\beta_{MB} = 0.33$, so $R_0 = 0.971781 < 1$, for two different sets of initial conditions. (a)-(b) show that system $(6)_1$ has the bistable equilibria: the DFE $\mathbf{E}_1$ and an endemic equilibrium, and the other endemic equilibrium is unstable.

$S_M(t)$, $S_B(t)$, $S_H(t)$

(a)

$I_M(t)$, $I_B(t)$, $I_H(t)$

(b)

FIGURE 4. Evolution over time of susceptible (a) and infective (b) classes of mosquito (magenta line), bird (orange line) and human (blue line) populations, corresponding to different initial data. We use the parameter values of Fig.1-(b) with $a_B = 3.9$ and $\beta_{MB} = 0.7$. (a)-(b) show that system (1) has a DFE $\mathbf{P}_1$, which is unstable, and an endemic equilibrium $\mathbf{P}^*$, which is LAS.

Acknowledgments

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