Modeling the Dynamics of Arboviral Diseases with Vaccination Perspective

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Abstract—In this paper, we propose a model of transmission of arboviruses, which takes into account a future vaccination strategy in human population. A qualitative analysis based on stability and bifurcation theory reveals that the phenomenon of backward bifurcation may occur; the stable disease-free equilibrium of the model coexists with a stable endemic equilibrium when the associated reproduction number, $R_0$, is less than unity. We show that the backward bifurcation phenomenon is caused by the arbovirus induced mortality. Using the direct Lyapunov method, we prove the global stability of the trivial equilibrium. Through a global sensitivity analysis, we determine the relative importance of model parameters for disease transmission. Simulation results using a nonstandard qualitatively stable numerical scheme are provided to illustrate the impact of vaccination strategy in human communities.

Keywords—Mathematical model; Arboviral disease; Vaccination; Stability; Backward bifurcation; Sensitivity analysis; Nonstandard numerical scheme.

I. INTRODUCTION

Arboviral diseases are affections transmitted by hematophagous arthropods. There are currently 534 viruses registered in the International Catalogue of Arboviruses and 25% of them have caused documented illness in humans [20], [49], [42]. Examples of these kinds of diseases are dengue, yellow fever, Saint Louis fever, encephalitis, West Nile Fever and chikungunya. A wide range of arbovirus diseases are transmitted by mosquito bites and constitute a public health emergency of international concern. According to WHO, dengue, caused by any of four closely-related virus serotypes (DEN-1-4) of the genus Flavivirus, causes 50–100 million infections worldwide every year, and the majority of patients worldwide are children aged 9 to 16 years [72], [84], [86]. The dynamics of arboviral diseases like dengue or chikungunya are influenced by many factors such as humans, the mosquito vector, the virus itself, as well as the environment which affects all the present mechanisms of control directly.
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or indirectly.

For all mentioned diseases, only yellow fever has a licensed vaccine. However, some works are underway for development of a vaccine for dengue \cite{10, 11, 33, 50, 73, 85}, Japanese encephalitis \cite{73}, and Chikungunya \cite{53, 54, 55, 46}. Moreover, the existence of different strains of dengue virus, for example, makes the development of an efficient vaccine a challenge for scientists. However, according to the French newspaper Le Figaro, the SANOFI laboratory hopes to market in the second half of 2015, the first vaccine against dengue fever, with an overall efficacy of 61% vaccine among young people aged 9 to 16 years and the rate of protection against severe dengue 95.5% \cite{39}. Therefore, it is important to assess the potential impact of such vaccines in human communities.

As part of the necessary multi-disciplinary research approach, mathematical models have been extensively used to provide a framework for understanding arboviral diseases transmission and control strategies of the infection spread in the host population. In the literature, considerable works can be found on the mathematical modeling of specific arboviral diseases, like West Nile Fever, yellow fever, dengue and chikungunya, see e.g. \cite{2, 17, 24, 30, 35, 36, 38, 40, 56, 60, 61, 64, 68, 79}. Although these models highlight the means to fight against these arbovirus, few papers deal with models that consider vaccination \cite{40, 68, 79}.

In this paper, we formulate a model, described by differential equations, in which we include two aspects: vaccination in the human population and the aquatic stage in the vectors population. We perform a qualitative analysis of the model, based on stability and bifurcation theory. In particular, we use an approach based on the center manifold theory \cite{19, 31, 43} to evaluate the occurrence of a transcritical backward bifurcation and, as a consequence, the presence of multiple endemic equilibria when the basic reproduction number $R_0$ is less than unity. Under the point of view of disease control, the occurrence of backward bifurcation has relevant implications for disease control because the classical threshold condition $R_0 < 1$, is no longer sufficient to ensure the elimination of the disease from the population.

The global stability of the trivial equilibrium and the disease–free equilibrium (the equilibrium without disease in both populations), whenever the associated thresholds (the net reproductive number $N$ and the basic reproduction number $R_0$) are less than unity, is derived through the use of Lyapunov stability theory and LaSalle’s invariant set theorem, and the approach of Kamgang and Sallet \cite{48}, respectively.

Through global sensitivity analysis, we determine the relative importance of model parameters for disease transmission. The analysis of the model is completed by the construction of a nonstandard numerical scheme which is qualitatively stable.

The rest of this paper is organized as follows. In Section II, we develop the mathematical model and give the invariant set in which the model is defined. In Section III, we compute two thresholds: the net reproductive number $N$ and the basic reproduction number $R_0$. Depending of the values of these thresholds, we identify two disease–free equilibria: the trivial equilibrium which corresponds to the extinction of vectors, when $N \leq 1$, and the disease-free equilibrium (DFE) when $N > 1$ and $R_0 < 1$. The results concerning the local and global stability of these two equilibria are also given. The section IV is devoted to the existence of endemic equilibria and bifurcation analysis. Vaccine impact is evaluated in Section V. Uncertainty and sensitivity analysis and the construction of a stable numerical scheme, are made in sections VI and VII, respectively. A conclusion completes the paper.

II. MODEL FORMULATION, INVARIANT REGION.

In this section we describe the mathematical model that we shall study in this paper. The formulation is mostly inspired, with some exceptions, by the models proposed in \cite{30, 40, 68, 80}. We assume that the human and vector populations
are divided into compartments described by time-dependent state variables. This said, the compartments in which the populations are divided are the following ones:

–For humans, we consider susceptible (denoted by $S_h$), vaccinated ($V_h$), exposed ($E_h$), infectious ($I_h$) and resistant or immune ($R_h$). Humans susceptible population is recruited at a constant rates $\Lambda_h$. The human susceptible population decreased following infection, which can be acquired via effective contact with an exposed or infectious vector at a rate $\lambda_h$ (the incidence rate of infection for humans), given by

$$\lambda_h = a\beta_{hv} \frac{N_v}{N_h + m} \frac{(\eta_v E_v + I_v)}{N_v} = \beta_{hv} \frac{(\eta_v E_v + I_v)}{N_h + m},$$

where $m$ denote the alternatively sources of blood [11, 80], $a$ is the biting rate per susceptible vector, $\beta_{hv}$ denotes the probability of transmission of infection from an infectious vector ($E_v$ or $I_v$) to a susceptible human ($S_h$ or $V_h$). We obtain the expression of $\lambda_h$ as follows: the probability that a vector chooses a particular human or other source of blood to bite can be assumed as $\frac{1}{N_h + m}$. Thus, a human receives in average $a\frac{N_v}{N_h + m}$ bites per unit of times. Then, the infection rate per susceptible human is given $a\beta_{hv} \frac{N_v}{N_h + m} \frac{(\eta_v E_v + I_v)}{N_v}$. In expression (1), the modification parameter $0 < \eta_v < 1$ accounts for the assumed reduction in transmissibility of exposed mosquitoes relative to infectious mosquitoes. It is worth emphasizing that, unlike many of the published modelling studies on dengue transmission dynamics, we assume in this study that exposed vectors can transmit dengue disease to humans. This is in line with some studies (see, for instance [34], [40], [87], [90]). However, it is significant to note that, in the case of Chikungunya for example, the exposed vectors do not play any role in the infectious process, in this case $\eta_v = 0$.

The vaccinated population is generated by vaccination of susceptible humans at a rate $\xi$. Further, it is expected that any future dengue vaccine would be imperfect [40], [68]. Thus, vaccinated individuals acquire infection at a rate $(1 - \epsilon)\lambda_h$ where $\epsilon$ is the vaccine efficacy. Exposed humans develop clinical symptoms of disease, and move to the infectious class at rate $\gamma_h$. Infectious humans may die as consequence of the infection, at a disease–induced death rate $\delta$, or recover at a rate $\sigma$. It is assumed that individuals successfully acquire lifelong immunity against the virus.

–Vectors population is classified into four compartments: susceptible ($S_v$), exposed ($E_v$), infectious ($I_v$) and aquatic ($A_v$). The aquatic state includes the eggs, larvae, and pupae. The vector population does not have an immune class, since it is assumed that their infectious period ends with their death. The population of vectors consists essentially of females which reached adulthood. A susceptible vector is generated by the adulthood females at rate $\theta$. The susceptible vector population decreased following infection, which can be acquired via effective contact with an exposed or infectious human at a rate $\lambda_v$ (the incidence rate of infection for vectors), given by

$$\lambda_v = a\beta_{vh} \frac{N_v}{N_h + m} \frac{(\eta_h E_h + I_h)}{N_v} = \beta_{vh} \frac{(\eta_h E_h + I_h)}{N_h + m},$$

where $\beta_{vh}$ is the probability of transmission of infection from an infectious human ($E_h$ or $I_h$) to a susceptible vector ($S_v$), where the modification parameter $0 \leq \eta_h < 1$ accounts for the relative infectiousness of exposed humans in relation to infectious humans. Here too, it is assumed that susceptible mosquitoes can acquire infection from exposed humans [23], [40]. Exposed vectors move to the infectious class with the rate $\gamma_v$. As in the case of the outbreak of Chikungunya on Réunion Island, it has been shown that lifespan of the infected mosquitoes is almost halved. This particular feature of infected mosquitoes therefore influences the dynamics of the disease [32], [30]. Thus, following Dumont and coworkers [29], [30], we assume in this work that the lifespan of a vector depends on its epidemiological status. So the average lifespan for susceptible mosquitoes is
1/µ_v days, the average lifespan of the exposed mosquitoes is 1/µ_1 days and the average adult lifespan for infected vector is 1/µ_2. Thus, we have 1/µ_2 ≤ 1/µ_1 ≤ 1/µ_v (with equality for other arboviral diseases). We do not consider vertical transmission in this work, so only susceptible humans are recruited, and vectors are assumed to be born susceptible.

We are now in position to write the model (the meaning of the state variables and parameters are summarized in Table I and Table II).

In model (3) the upper dot denotes the time derivative. K denote the carrying capacity of breeding sites. The parameter K is highly dependent on some factors such as (the location, temperature, season). In this work, we follow Dumont and Chiolero [30], and consider, in our sensitive analysis, that K depend of the location, thus K = χN_h, where χ is a positive integer which represent the location and N_h the human population size. For example, in the year 2005 at Saint-Denis and Saint-Pierre in Réunion island, χ = 2) [30]. µ_b represent the number of eggs at each deposit per capita and µ_A is the natural mortality of larvae.

We set π = 1 − ϵ, k_1 = µ_h + ξ, k_2 = µ_h + γ_h, k_3 = µ_h + δ + σ, k_4 = µ_1 + γ_v, k_6 = µ_A + θ.

Let N_h the total human population and N_v the total of adult vectors, i.e. N_h = S_h + V_h + E_h + I_h + R_h and N_v = S_v + E_v + I_v. System (3) can be rewritten in the following way

\[
\frac{dX}{dt} = A(X)X + F \tag{4}
\]

with X = (S_h, V_h, E_h, I_h, R_h, A_v, S_v, E_v, I_v)^T, A(X) = (A_ij)_{1≤i,j≤9} were were A_{1.1} = −(λ_h + k_1), A_{2.1} = ξ, A_{2.2} = −(πλ_h + µ_h), A_{3.1} = λ_h, A_{3.2} = πλ_h, A_{3.3} = −k_2, A_{4.3} = γ_h, A_{4.4} = −k_3, A_{5.4} = σ, A_{5.5} = −µ_h, A_{6.7} = A_{6.8} = A_{6.9} = µ_b, A_{7.6} = θ, A_{7.7} = −(λ_v + µ_v), A_{8.7} = λ_v, A_{8.8} = −k_4, A_{8.9} = γ_v, A_{9.9} = −µ_2, A_{6.6} = −(k_6 + µ_b S_v + E_v + I_v) K and the other entries of A(X) are equal to zero; and F = (A_h, 0, 0, 0, 0, 0, 0, 0, 0)^T.

Note that A(X) is a Metzler matrix, i.e. a matrix such off diagonal terms are non negative [8], [47], for all X ∈ R^9_+. Thus, using the fact that F ≥ 0, system (4) is positively invariant in R^9_+.

### Table I: The State Variables of Model [3].

<table>
<thead>
<tr>
<th>Humans</th>
<th>Vectors</th>
</tr>
</thead>
<tbody>
<tr>
<td>S_h: Susceptible</td>
<td>A_v: Aquatic</td>
</tr>
<tr>
<td>V_h: Vaccinated</td>
<td>S_v: Susceptible</td>
</tr>
<tr>
<td>E_h: Infected</td>
<td>E_v: Exposed</td>
</tr>
<tr>
<td>I_h: Infectious</td>
<td>I_v: Infectious</td>
</tr>
<tr>
<td>R_h: Resistant (immune)</td>
<td></td>
</tr>
</tbody>
</table>

### Table II: Description of Parameters of Model [3].

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Λ_h</td>
<td>Recruitment rate of humans</td>
</tr>
<tr>
<td>ξ</td>
<td>Vaccine coverage</td>
</tr>
<tr>
<td>ϵ</td>
<td>The vaccine efficacy</td>
</tr>
<tr>
<td>η_h, η_v</td>
<td>Modification parameters</td>
</tr>
<tr>
<td>β_h</td>
<td>Probability of transmission of infection from an infectious vector to a susceptible vector</td>
</tr>
<tr>
<td>γ_h</td>
<td>Progression rate from Exposure to Infected</td>
</tr>
<tr>
<td>µ_h</td>
<td>Natural mortality rate in humans</td>
</tr>
<tr>
<td>µ_v</td>
<td>Natural mortality rate of susceptible vectors</td>
</tr>
<tr>
<td>µ_A</td>
<td>Natural mortality of larval</td>
</tr>
<tr>
<td>µ_v^{-1}</td>
<td>Average lifespan of exposed mosquitoes</td>
</tr>
<tr>
<td>µ_v^{-1}</td>
<td>Average lifespan of infected mosquitoes</td>
</tr>
<tr>
<td>ρ</td>
<td>Maturation rate from larvae to adult</td>
</tr>
<tr>
<td>δ</td>
<td>Disease–induced death rate in humans</td>
</tr>
<tr>
<td>σ</td>
<td>Recovery rate for humans</td>
</tr>
<tr>
<td>a</td>
<td>Average number of bites</td>
</tr>
<tr>
<td>m</td>
<td>Number of alternative source of blood</td>
</tr>
<tr>
<td>K</td>
<td>Capacity of breeding sites</td>
</tr>
<tr>
<td>µ_b</td>
<td>Number of eggs at each deposit per capita</td>
</tr>
</tbody>
</table>
which means that any trajectory of the system starting from an initial state in the positive orthant \( R^9_{>0} \), remains forever in \( R^9_{>0} \). The right-hand side is Lipschitz continuous: there exists an unique maximal solution.

On the other hand, from the first four equations of model system (3), it follows that
\[
\dot{N}_h(t) = \Lambda_h - \mu_h N_h - \delta I_h \leq \Lambda_h - \mu_h N_h. \tag{5}
\]
So that
\[
0 \leq \dot{N}_h(t) \leq \frac{\Lambda_h}{\mu_h} + \left( N_h(0) - \frac{\Lambda_h}{\mu_h} \right) e^{-\mu_h t}. \tag{6}
\]

Thus, at \( t \to \infty \), \( 0 \leq \dot{N}_h(t) \leq \frac{\Lambda_h}{\mu_h} \).

From the last three equations of model system (3), it also follows that
\[
\dot{N}_v(t) = \theta A_v - \mu_v S_v - \mu_1 E_v - \mu_2 I_v
\leq \theta A_v - \mu_v N_v. \tag{7}
\]
So that
\[
0 \leq \dot{N}_v(t) \leq \frac{\theta A_v}{\mu_v} + \left( N_v(0) - \frac{\theta A_v}{\mu_v} \right) e^{-\mu_v t}. \tag{8}
\]

Thus, at \( t \to \infty \), \( 0 \leq \dot{N}_v(t) \leq \frac{\theta K}{\mu_v} \) since \( A_v \leq K \). Therefore, all feasible solutions of model system (3) enter the region:
\[
D = \{(S_h, V_h, E_h, I_h, R_h, A_v, S_v, E_v, I_v) \in R^9 : \\
S_h + V_h + E_h + I_h + R_h \leq \frac{\Lambda_h}{\mu_h}; A_v \leq K; \\
S_v + E_v + I_v \leq \theta K/\mu_v \}. 
\]

III. THE DISEASE–FREE EQUILIBRIA AND STABILITY ANALYSIS

Now let \( \mathcal{N} \) the net reproductive number \([25]\) given by
\[
\mathcal{N} = \frac{\mu_h \theta}{\mu_v (\theta + \mu_A)}. \tag{9}
\]
We prove the following result

**Proposition 3.1:** a) If \( \mathcal{N} \leq 1 \), then, system (3) has only one trivial disease–free equilibrium \( TE := P_0 = \left( \frac{\Lambda_h}{k_1}, \frac{\xi \Lambda_h}{\mu_h k_1}, 0, 0, 0, 0, 0, 0, 0, 0 \right) \).

b) If \( \mathcal{N} > 1 \), then, system (3) has a Disease–Free Equilibrium \( P_1 = (S_h^0, V_h^0, 0, 0, 0, A_v^0, S_v^0, 0, 0) \), with
\[
S_h^0 = \frac{\Lambda_h}{k_1}, \quad V_h^0 = \frac{\xi \Lambda_h}{\mu_h k_1}, \quad A_v^0 = K \left( 1 - \frac{1}{\mathcal{N}} \right), \\
S_v^0 = \frac{\theta}{\mu_v} K \left( 1 - \frac{1}{\mathcal{N}} \right).
\]

**Proof:** See Appendix A. \( \blacksquare \)

In Proposition 3.1, we have shown that system (3) have at least two equilibria depending of the value of threshold \( \mathcal{N} \) and the basic reproduction number \( R_0 \). Precisely, we have proved that when \( \mathcal{N} \leq 1 \), model system (3) admits only one equilibrium called trivial equilibrium \( (TE := P_0) \). When \( \mathcal{N} > 1 \), model system (3) admits additionally the disease–free equilibrium \( (DFE := P_1) \). We prove, in the following, that the trivial equilibrium is locally and globally asymptotically stable whenever \( \mathcal{N} \leq 1 \). Then, we prove that the trivial equilibrium is unstable when \( \mathcal{N} > 1 \), and the disease–free equilibrium is locally asymptotically stable whenever \( R_0 < 1 \). Using Kamgang and Sallet approach \([48]\), a necessary condition for the global stability of the disease–free equilibrium is also given.

A. Local stability analysis

The local stability of the trivial equilibrium and the disease–free equilibrium is given in the following result:

**Proposition 3.2:** a) If \( \mathcal{N} \leq 1 \), then the trivial equilibrium \( TE \) is locally asymptotically stable.

b) If \( \mathcal{N} > 1 \), then the trivial equilibrium is unstable and the Disease Free Equilibrium \( P_1 \) is locally asymptotically stable if \( R_0 < 1 \) and unstable if \( R_0 > 1 \), where \( R_0 \) is the basic reproduction number \([26], [82] \), given by
\[
R_0^2 = \frac{\beta_{hv} \beta_{vh} K \theta (\pi \xi + \mu_h) (k_3 \eta_h + \gamma_h)}{(\mu_h + \xi) (\mu_h + \gamma_h) (\mu_h + \delta + \sigma)} \\
\times \frac{\Lambda_h \mu_h (\mu_2 \eta_v + \gamma_v)}{\mu_v \mu_2 (\Lambda_h + m \mu_h)^2 (\mu_1 + \gamma_v)} \left( 1 - \frac{1}{\mathcal{N}} \right). \tag{10}
\]

**Proof:** See appendix B. \( \blacksquare \)
B. Global stability analysis

1) Global asymptotic stability of the trivial equilibrium $TE := P_0$.

Proposition 3.3: If $N \leq 1$, then $TE := P_0$ is globally asymptotically stable on $\mathcal{D}$.

Proof: See Appendix C.

2) Global asymptotic stability of the disease–free equilibrium: Following [30], we prove that the disease–free equilibrium $DFE := P_1$ is globally asymptotically stable under a certain threshold condition. To this aim, we use a result obtained by Kamgang and Sallet [48], which is an extension of some results given in [82]. Using the property of DFE, it is possible to rewrite (3) in the following manner

$$\begin{align*}
X_S &= A_1(X)(X_S - X_{DFE}) + A_{12}(X)X_I \\
X_I &= A_2(X)X_I
\end{align*}$$

where $X_S$ is the vector representing the state of different compartments of non transmitting individuals $(S_h, V_h, R_h, A_v, S_v)$ and the vector $X_I$ represents the state of compartments of different transmitting individuals $(E_h, I_h, E_v, I_v)$. Here, we have $X_S = (S_h, V_h, R_h, A_v, S_v)^T$, $X_I = (E_h, I_h, E_v, I_v)^T$, $X = (X_S, X_I)$ and $X_{DFE} = (S_h^0, V_h^0, 0, 0, A_v^0, S_v^0, 0, 0)^T$, with

$$A_1(X) = \begin{pmatrix}
-k_1 & 0 & 0 & 0 & 0 \\
\xi & -\mu_h & 0 & 0 & 0 \\
0 & 0 & -\mu_h & 0 & 0 \\
0 & 0 & 0 & -K_6 & K_7 \\
0 & 0 & 0 & \theta & -\mu_v
\end{pmatrix},$$

$$A_{12}(X) = \begin{pmatrix}
0 & 0 & -a_{13} & -a_{14} & 0 \\
0 & 0 & -a_{23} & -a_{24} & 0 \\
0 & \sigma & 0 & 0 & 0 \\
0 & 0 & \kappa & \kappa & 0 \\
-a_{41} & -a_{42} & 0 & 0 & 0
\end{pmatrix},$$

$$A_2(X) = \begin{pmatrix}
-k_2 & 0 & b_{13} & b_{14} \\
\gamma_h & -k_3 & 0 & 0 \\
b_{31} & b_{32} & -k_4 & 0 \\
0 & 0 & \gamma_v & -\mu_2
\end{pmatrix},$$

with $K_6 = \frac{k_6 + \mu_b S_v^0}{K}$, $K_7 = \mu_b \frac{(1 - A_v K)}{K}$, $a_{13} = \frac{\beta_{hv} V_h}{N_h + m}$, $a_{14} = \frac{\beta_{hv} V_h}{N_h + m}$, $a_{23} = \frac{\pi \beta_{hv} V_h}{N_h + m}$, $a_{24} = \frac{\pi \beta_{hv} V_h}{N_h + m}$, $a_{41} = \frac{\beta_{hv} S_v}{N_h + m}$, $a_{42} = \frac{\beta_{hv} S_v}{N_h + m}$, $b_{13} = \frac{\beta_{hv} S_v}{N_h + m}$, $b_{14} = \frac{\beta_{hv} H}{N_h + m}$, $b_{31} = \frac{\beta_{hv} S_v}{N_h + m}$, $b_{32} = \frac{\beta_{hv} S_v}{N_h + m}$, $\kappa = \mu_b \left(1 - \frac{A_v}{K}\right)$ and $H = (S_h + \pi V_h)$.

A direct computation shows that the eigenvalues of $A_1(X)$ are real and negative. Thus the system $X_S = A_1(X)(X_S - X_{DFE})$ is globally asymptotically stable at $X_{DFE}$. Note also that $A_2(X)$ is a Metzler matrix, i.e. a matrix such that off diagonal terms are non negative [8], [47].

Following $D$, we now consider the bounded set $G$:

$$G = \{(S_h, V_h, E_h, I_h, R_h, A_v, S_v, E_v, I_v) \in \mathbb{R}^9 : S_h \leq N_h, V_h \leq N_h, E_h \leq N_h, I_h \leq N_h, R_h \leq N_h, N_h = \frac{\lambda_h}{(\mu_h + \delta)} \leq N_h \leq N_h^0 = \frac{\lambda_h}{\mu_h}; A_v \leq K; S_v + E_v + I_v \leq \theta K / \mu_v\}.$$

Let us recall the following theorem [48]

Theorem 3.1: Let $G \subset U = \mathbb{R}^5 \times \mathbb{R}^4$. The system (11) is of class $C^1$, defined on $U$. If

1) $G$ is positively invariant relative to (11).
2) The system $X_S = A_1(X)(X_S - X_{DFE})$ is globally asymptotically stable at $X_{DFE}$.
3) For any $x \in G$, the matrix $A_2(X)$ is Metzler irreducible.
4) There exists a matrix $\bar{A}_2$, which is an upper bound of the set $M = \{A_2(x) \in M_4(\mathbb{R}) : x \in G\}$ with the property that if $A_2 \in M$, for any $\bar{x} \in G$, such that $A_2(\bar{x}) = \bar{A}_2$, then $\bar{x} \in \mathbb{R}^5 \times \{0\}$.
5) The stability modulus of $\bar{A}_2$,

$$\alpha(\bar{A}_2) = \max_{\lambda \in sp(\bar{A}_2)} \Re(\lambda) \text{ satisfied } \alpha(\bar{A}_2) \leq 0.$$

Then the DFE is GAS in $G$. (See [48] for a proof).

Let us now verify the assumptions of the previous theorem: it is obvious that conditions (1–3) of the theorem are satisfied. An upper bound of the set of matrices $M$, which is the matrix $A_2$ is given
Metzler stability, by induction, to the stability of matrix is equivalent to
\[ A = D \]
Some computations, we obtain
\[ A \text{ stable is that the elements on the diagonal are } D \text{ stable.} \]
Metzler stable matrices if and only if
\[ CA \text{ matrices. } M \text{ is Metzler stable if and only if written in block form a characterization of Metzler stable matrices:}\]
Use the following useful lemma [48] which is the
\[ \text{Lemma 3.1: Let } M \text{ be a square Metzler matrix written in block form } \left( \begin{array}{cc} A & B \\ C & D \end{array} \right) \text{ with } A \text{ and } D \text{ square matrices. } M \text{ is Metzler stable if and only if matrices } A \text{ and } D - CA^{-1}B \text{ are Metzler stable.}\]
A necessary condition for a Metzler matrix to be stable is that the elements on the diagonal are negative. Note also that \( A \) is a Metzler stable matrix is equivalent to \( A \) is invertible and \( -A^{-1} \geq 0 \). Lemma 3.1 allows to reduce the problem of Metzler stability, by induction, to the stability of \( 2 \times 2 \) Metzler matrices [48]. In our case, we have
\[ A = \begin{pmatrix} -k_2 & 0 \\ \gamma_h & -k_3 \end{pmatrix}, \]
\[ B = \begin{pmatrix} \beta_{hv}(\bar{S}_h + \pi \bar{V}_h) & \beta_{hv}(\bar{S}_h + \pi \bar{V}_h) \\ N_h + m & N_h + m \end{pmatrix}, \]
\[ C = \begin{pmatrix} \beta_{hv}\eta_h \bar{S}_v & \beta_{hv}\eta_h \bar{S}_v \\ N_h + m & N_h + m \end{pmatrix}, \text{ and } \]
\[ D = \begin{pmatrix} -k_4 & 0 \\ \gamma_v & -\mu_2 \end{pmatrix}. \]
Clearly, \( A \) is a stable Metzler matrix. Then, after some computations, we obtain \( D - CA^{-1}B \) is a stable Metzler matrix if and only if
\[ R_G^2 \leq 1 \quad (12) \]
where
\[ R_G^2 = \frac{\beta_{hv}\beta_v K \theta A_h(\eta_v \mu_2 + \gamma_v)(k_3 \eta_h + \gamma_h)}{\mu_v \mu_2 \mu_h k_1 k_2 k_3 k_4} \times \frac{(\mu_2 + \pi \xi)(\mu_h + \delta)^2}{(A_h + \mu_1 + \mu_2)^2} \]
Thus we claim the following result
\[ \text{Theorem 3.2: If } N > 1 \text{ and } R_G^2 \leq 1, \text{ then the disease–free equilibrium } P_1 \text{ is globally asymptotically stable in } G. \]
\[ \text{Remark 3.1: Note that } R_G^2 = R_0^2 \frac{(\mu_h + \delta)^2(A_h + m(\mu_h + \delta))^2}{(\mu_v^2(\mu_h + \delta))^2} \left( \frac{N}{N-1} \right) \]
and condition (12) is equivalent to
\[ R_G^2 \leq \left( \frac{N-1}{N} \right) \frac{\mu_v^2}{(\mu_h + \delta)^2} (A_h + m(\mu_h + \delta))^2 \]
In absence of disease–induced death in human \( \delta = 0 \), inequality (13) becomes
\[ R_G^2 \leq \left( \frac{N-1}{N} \right) < 1. \quad (14) \]
This shows that with the establishment of an effective treatment, it is possible to have \( R_0 \) and \( R_G \) less than 1.
\[ \text{Theorem 3.2} \] means that for \( R_0 < R_G < 1 \), the DFE is the unique equilibrium (no co-existence with an endemic equilibrium). If \( R_0 \in [R_G, 1] \), then it is possible to have co-existence with endemic equilibrium. To confirm whether or not the backward bifurcation phenomenon occurs in this case, one could use the approach developed in [19], [31], [82], which is based on the general center manifold theory [43].

IV. THE ENDEMIC EQUILIBRIA AND BIFURCATION ANALYSIS
A. Existence of endemic equilibria
We now turn to study the existence of an endemic equilibrium of model system (5). Let \( R_0 \) the basic reproduction number [26], [82] given by Eq. (10).
we claim the following
\[ \text{Proposition 4.1: Let } N > 1 \text{ and } \mu_v \leq \mu_1 \leq \mu_2. \]
Then
(i) There exists at least one endemic equilibrium whenever $R_0 > 1$.

(ii) The backward bifurcation phenomenon may occur when $R_0 \leq 1$.

(iii) The disease–induced death is responsible of the backward bifurcation phenomenon.

(iv) In the absence of the disease–induced death ($\delta = 0$ and $\mu_v = \mu_1 = \mu_2$), system (4) have a unique endemic equilibrium whenever $R_0 > 1$, and the backward bifurcation phenomenon not occurs whenever $R_0 \leq 1$ (See remark 4.1).

**Proof:** See appendix D.

The backward bifurcation phenomenon, in epidemiological systems, indicate the possibility of existence of at least one endemic equilibrium when $R_0$ is less than unity. Thus, the classical requirement of $R_0 < 1$ is, although necessary, no longer sufficient for disease elimination [6, 14, 40, 75]. In some epidemiological models, it has been shown that the phenomenon of backward bifurcation is caused by factors such as nonlinear incidence (the infection force), disease–induced death or imperfect vaccine [15, 16, 31, 40, 70, 75].

It is important to note that case (i) of Proposition 4.1 suggests the possibility of a pithcfork (Forward) bifurcation when $R_0 = 1$. Also, case (iv) of Proposition 4.1 suggests that the principal cause of occurrence of backward bifurcation phenomenon is the disease-induced death in both humans and vectors.

In the following remark, we prove that, in absence of disease–induced death in both populations, the disease–free equilibrium is the unique equilibrium whenever $\mathcal{N}_h > 1$ and $R_0|_{\delta=0, \mu_v=\mu_1=\mu_2} < 1$. Using the direct Lyapunov method, we prove the global asymptotic stability of the disease–free equilibrium whenever $R_0|_{\delta=0, \mu_v=\mu_1=\mu_2} < 1$.

**Remark 4.1:** Assumed that $\mathcal{N}_h > 1$.

Let $k_7 = \Lambda_h + m\mu_h$, $k_8 = \pi\xi + \mu_h$, $k_{11} = k_3\eta h + \gamma h$ and $\mathcal{R}_1 = R_0|_{\delta=0, \mu_v=\mu_1=\mu_2}$. In the absence of disease-induced death, i.e, $\delta = 0$ and $\mu_v = \mu_1 = \mu_2$, Eq. (44) (see appendix D) becomes

$$
\lambda_h\left[B_{02}(\lambda_h)^2 + B_{01}\lambda_h + B_{00}\right] = 0 \quad (15)
$$

with $B_{02} = k_2k_3k_2^2\pi\mu_v + \beta_{ih}k_7k_{11}\Lambda_h\mu_h\pi > 0$, $B_{00} = k_1k_2k_3k_2^2\mu_h\mu_v (1 - \mathcal{R}_1^2)$ and $B_{01} = k_1k_2k_3k_2^2\mu_h\mu_v (1 - \mu_h\mathcal{R}_1^2) + k_2k_3k_2^2\mu_h\mu_v + \beta_{ih}k_7k_{11}\Lambda_h\mu_h\mu_v.$

Equation (15) have only one positive solution whenever $\mathcal{R}_1 > 1$. If $\mathcal{R}_1 \leq 1$, then coefficients $B_{00}, B_{01}, B_{02}$ are always positive, and the disease-free equilibrium is the unique equilibrium. From this we conclude that the disease–induced mortality is the possible cause for the occurrence of multiple endemic equilibria below the classical threshold $\mathcal{R}_1 = 1$.

The global stability of the disease–free equilibrium may be achieved by Lyapunov function [37, 40].

$$
\mathcal{V} = \sum_{i=1}^4 g_i I_i \quad \text{where} \quad I_i = (E_h, I_h, E_v, I_v) \quad \text{and} \quad g_i, \ i = 1, \ldots, 4 \quad \text{are positive weights given by} \quad g_1 = 1;
$$

$$
g_2 = \frac{(k_3\eta h + \gamma h)}{k_2}, \quad g_3 = \frac{\beta_{ih}\gamma h}{(S_h + \pi V_h)^2},
$$

$$
g_4 = \frac{\beta_{hv} \gamma h}{\mu_2(N_h^0 + m)}.
$$

Along the solutions of (3) we have:

$$
\dot{\mathcal{V}} = \sum_{i=1}^4 g_i \dot{I}_i = g_1 \dot{E}_h + g_2 \dot{I}_h + g_3 \dot{E}_v + g_4 \dot{I}_v
$$

$$
= g_1 \left[\lambda_h [S_h + (1 - \epsilon)V_h] - (\mu_h + \gamma_h)E_h\right] + g_2 \left[\gamma_h E_h - (\mu_h + \delta + \sigma)I_h\right] + g_3 \left[\lambda_v S_v - (\mu_v + \gamma_v)E_v\right] + g_4 (\gamma_v E_v - \mu_2 I_v)
$$

$$
= \left(g_1 \beta_{hv} \eta v [S_h + \pi V_h] + g_4 \gamma v - g_3 k_4\right) E_v + \left(g_1 \beta_{hv} [S_h + \pi V_h] - g_4 \mu_2\right) I_v + \left(g_3 \beta_{hv} S_v + g_2 \gamma v - g_1 k_2\right) E_h + \left(g_3 \beta_{hv} S_v + g_2 \gamma v - g_1 k_2\right) E_h
$$

After replacing the constants $g_i$, $i = 1, \ldots, 4$ by their value, and using the fact that $S_h \leq S_h^0$, $V_h \leq V_h^0$, $A_v \leq A_v^0$, and $S_v \leq S_v^0$ in

$$
\mathcal{D}_1 = \{(S_h, V_h, E_h, I_h, A_v, S_v, E_v, I_v) \in \mathcal{D} : S_h \leq S_h^0, V_h \leq V_h^0, A_v \leq A_v^0, S_v \leq S_v^0\},
$$
it follows that
\[
\dot{y} \leq \left( g_1 \beta_{hv} \left[ S_0^0 + \pi V_0^0 \right] + g_4 \gamma_v - g_3 k_4 \right) E_v \\
= \frac{k_2 k_3 k_4 (N_0^0 + m)}{\beta_{vh} S_0^0 (k_3 \eta_h + \gamma_h)} \left( R_1^2 - 1 \right) E_v
\]

We have \( \dot{y} \leq 0 \) if \( R_1 \leq 1 \), with \( \dot{y} = 0 \) if \( R_1 = 1 \) or \( E_v = 0 \). Whenever \( E_v = 0 \), we also have \( E_h = 0 \), \( I_h = 0 \), and \( I_v = 0 \). Substituting \( E_h = I_h = E_v = I_v = 0 \) in the first, second, fifth, sixth, and seventh equations of Eq. (3) with \( \delta_1 = 0 \) gives \( S_h(t) \to S_h^0 \), \( V_h(t) \to V_h^0 \), \( R_h(t) \to 0 \), \( A_v(t) \to A_v^0 \), \( S_v(t) \to S_v^0 \) as \( t \to \infty \). Thus

\[
[S_h(t), V_h(t), E_h(t), I_h(t), R_h(t), A_v(t), S_v(t), E_v(t), I_v(t)] \to (S_h^0, V_h^0, 0, 0, 0, A_v^0, S_v^0, 0, 0) \text{ as } t \to \infty.
\]

It follows from the LaSalle’s invariance principle that every solution of (3) (when \( R_1 \leq 1 \)), with initial conditions in \( D_1 \) converges to \( P_1 \), as \( t \to \infty \). Hence, the DFE \( (P_1) \), of model (3), is GAS in \( D_1 \) if \( R_1 \leq 1 \).

B. Bifurcation analysis

Previous Analysis state that multiple endemic equilibria may occur although \( R_0 < 1 \). In order to better investigate the variation of model’s prediction as \( R_0 \) varied, we perform a bifurcation analysis at the criticality, i.e. at the Disease–free Equilibrium \( DFE := P_1 \) and \( R_0 = 1 \). On one hand, this will provide a rigorous proof that the occurrence of multiple endemic equilibria comes from a backward bifurcation. On the other hand, we will get also information on the stability of equilibria near the criticality. In particular, on the stability of the endemic equilibrium points emerging from the criticality. We study the center manifold near the criticality by using the approach developed in [19], [31], [82], which is based on the general center manifold theory [43]. In summary, this approach establishes that the normal form representing the dynamics of the system on the center manifold is given by

\[
\dot{u} = a^* u^2 + b^* w u,
\]

where, \( u \) represent a real function of real variable,

\[
a^* = \frac{v}{2} \cdot D_{xx} f(x_0, w) w^2 \equiv \sum_{k,i,j=1}^n v_k w_i w_j \frac{\partial^2 f_k(0, 0)}{\partial x_i \partial x_j}
\]

and

\[
b^* = v \cdot D_{xw} f(x_0, w) w \equiv \sum_{k,i=1}^n v_k w_i \frac{\partial^2 f_k(0, 0)}{\partial x_i \partial w}.
\]

Note that the symbol \( w \) denotes a bifurcation parameter to be chosen, \( f_i \)’s denotes the right hand side of system [3], \( x \) denotes the state vector, \( x_0 \) the Disease–free Equilibrium \( P_1 \); \( v \) and \( w \) denote the left and right eigenvectors, respectively, corresponding to the null eigenvalue of the Jacobian matrix of system [3] evaluated at the criticality.

In order to apply this approach, let us choose \( \beta_{hv} \) as bifurcation parameter. From \( R_0 = 1 \) we get the critical value

\[
\beta_{hv}^* = \frac{\mu_v \mu_2 k_1 k_2 k_3 k_4 (\Lambda_h + m \mu_h)^2 \left( \frac{N}{N-1} \right)}{\beta_{vh} \Lambda_h \mu_h \mu_k K \theta (\pi \xi + \mu_h)(\mu_2 \eta_v + \gamma_v) [\eta_v k_3 + \gamma_h]}
\]

Note also that in \( f_k(0, 0) \), the first zero corresponds to the disease–free equilibrium, \( P_1 \), for the system (3). Since \( \beta_{hv} = \beta_{hv}^* \) is the bifurcation parameter, it follows from \( w = \beta_{hv} - \beta_{hv}^* \) that \( w = 0 \) when \( \beta_{hv} = \beta_{hv}^* \) which is the second component in \( f_k(0, 0) \).

The Jacobian matrix of the system (3) evaluated at the disease–free equilibrium \( P_1 \) with \( \beta_{hv} = \beta_{hv}^* \) is given by

\[
J(P_1) = \begin{pmatrix}
-k_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & -\mu_h & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & -k_2 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & \gamma_h & -k_3 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & \sigma & -\mu_h & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & -\beta_{vh} \eta_v S_0^0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & \beta_{vh} \eta_v S_0^0 & \beta_{vh} S_0^0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & \beta_{vh} \eta_v S_0^0 & \beta_{vh} S_0^0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0
\end{pmatrix}
\]
where we have set $H = N_h^0 + m$, $K_1 = \frac{\mu_b\theta}{\mu_v}$ and $K_2 = \frac{k_5\mu_v}{\theta}$.

The eigenvalues of the Jacobian matrix $J(P_1)$ are $\lambda_1 = -\mu_h$, $\lambda_2 = -k_1$, and the other eigenvalues are the eigenvalue of the following matrix

$$J = \begin{pmatrix}
-k_2 & 0 & 0 & 0 & \frac{\beta_v\eta\eta_0}{H} & \frac{\beta_v\eta\eta_0}{H} \\
\gamma_h & -k_3 & 0 & 0 & \frac{H}{H} & \frac{H}{H} \\
-k_4 & 0 & k_2 & K_2 & K_2 & K_2 \\
0 & -\frac{\beta_v\eta_0\eta}{H} & -\frac{\beta_v\eta_0\eta}{H} & \theta & -\mu_v & 0 & 0 \\
0 & -k_4 & 0 & \frac{H}{H} & 0 & 0 & \gamma_v & -\mu_2
\end{pmatrix}.$$

The characteristic polynomial of $J$ is given by

$$f(\lambda) = \lambda^6 + a_5\lambda^5 + a_4\lambda^4 + a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0$$

with

$$a_0 = k_1k_2k_3k_4k_7\mu_b\mu_v(k_5\mu_v - \mu_b\theta)(1 - R_0^2).$$

The other coefficients $a_5$, $a_4$, $a_3$, $a_2$, and $a_1$ are obtained after computations on Maxima software [58, 89]. Since at the criticality, we have $R_0 = 1$, then $a_0 = 0$, thus equation (18) becomes

$$f(\lambda) = \lambda^6 + a_5\lambda^5 + a_4\lambda^4 + a_3\lambda^3 + a_2\lambda^2 + a_1\lambda.$$

Then, the Jacobian $J(P_1)$ of the linearized system has a simple zero eigenvalue and therefore $P_1$ is a nonhyperbolic equilibrium for $R_0 = 1$. In order to get the coefficients (16) and (17), we need to calculate the right and the left eigenvectors corresponding to the zero eigenvalue.

The right eigenvector of $J(P_1)$ is given by

$$w = (w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8, w_9)^T$$

where

$$w_1 = -\frac{\beta^*_v\theta k_9\mu_h}{k_1^2}\gamma_v k_7 w_9 < 0,$$

$$w_2 = -\frac{\lambda_1\Lambda_h k_9(\mu_h + k_1\pi)}{k_1k_2k_7\gamma_v} w_9 < 0,$$

$$w_3 = \frac{\beta^*_v\Lambda_h k_0(\mu_h + k_1\pi)}{k_1k_2k_7\gamma_v} w_9 > 0,$$

$$w_4 = \frac{\beta^*_v\Lambda_h\sigma\gamma_v k_9(\mu_h + k_1\pi)}{k_1k_2k_7\gamma_v} w_9 > 0,$$

$$w_5 = \frac{\beta^*_v\Lambda_h\gamma_v k_0(\mu_h + k_1\pi)}{k_1k_2k_7\gamma_v} w_9 > 0,$$

$$w_6 = \frac{\beta^*_v\Lambda_h\gamma_v k_9(\mu_h + k_1\pi)}{k_1k_2k_7\gamma_v} w_9 > 0,$$

Similarly, $J(P_1)$ has a left eigenvector

$$v = (v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8, v_9)$$

where

$$v_1 = v_2 = v_5 = v_6 = v_7 = 0, v_3 = \frac{\mu_v k_1 k_7}{\beta_v k_3 k_7} v_9,$$

$$v_4 = \frac{\beta_v k_2 k_3 k_7 \mu_v}{\mu_v k_4 k_7} (1 - \frac{1}{N}) \eta_v w_3 + w_4 < 0,$$

$$v_5 = \frac{\beta_v k_2 k_3 k_7 \mu_v}{\mu_v k_4 k_7} (1 - \frac{1}{N}) \eta_v w_3 + w_4 > 0,$$

$$v_6 = \frac{k_v\gamma_v}{k_4} (w_7 + w_8 + w_9) \text{ and } w_9 > 0.$$

a) Computation of $a^*$: Using the non–zero components of $v$ and the associated non–zero partial derivatives of $f$ (at the DFE $P_1$), for system (3), we obtain

$$a^* = \frac{1}{2} v_3 \sum_{i,j=1}^9 w_i w_j \frac{\partial^2 f_3(0,0)}{\partial x_i \partial x_j} + \frac{1}{2} v_8 \sum_{i,j=1}^9 w_i w_j \frac{\partial^2 f_3(0,0)}{\partial x_i \partial x_j}.$$

We finally obtain (See the details in appendix E)

$$a^* = \phi_1 - \phi_2.$$
where
\[ \phi_1 = \frac{1}{2} v_3 \left\{ \frac{\beta_{vh} \mu_h}{k_1 (\Lambda_h + \mu_h m)^2} [(\xi \Lambda_h + m \mu_h)(w_1 + \pi w_2) \times (w_3 \pi \nu + w_9 + \eta_v + 1) - 2 \Lambda_h (\mu_h + \pi \xi)(w_3 + w_4 + w_5)(w_5 \pi \nu + w_9)] \right\} \]
\[ - \frac{1}{2} v_8 \frac{\beta_{vh} \mu_h K \theta}{\mu_v(\Lambda_h + \mu_h m)^2} \left( 1 - \frac{1}{N} \right) w_5 (\eta_v w_3 + w_4) \]
\[ + \frac{1}{2} v_8 \frac{\beta_{vh} \mu_h}{(\Lambda_h + \mu_h m)} (\eta_v w_3 + w_4) w_7 \]
\[ - \frac{1}{2} v_8 \frac{\beta_{vh} \mu_h^2 K \theta}{\mu_v(\Lambda_h + \mu_h m)^2} \left( 1 - \frac{1}{N} \right) \times [2(\eta_v + 1)w_3 w_4 + 2(\eta_v w_3 + w_2)] < 0 \]

and
\[ \phi_2 = \frac{1}{2} v_8 \frac{\beta_{vh} \mu_h^2 K \theta}{\mu_v(\Lambda_h + \mu_h m)^2} \left( 1 - \frac{1}{N} \right) \times [(\eta_v w_3 + w_4)(w_1 + w_2)] < 0 \]

b) Computation of \( b^* \):
\[ b^* = v_3 \frac{\Lambda_h (\mu_h + \pi \xi)}{k_1 (\Lambda_h + \mu_h m)} (\eta_v w_8 + w_9) > 0. \]

Since \( b^* > 0 \) according to the sign of \( w_i, v_i \), for \( i \in \{1, \ldots, 9\} \), we conclude that the backward bifurcation phenomenon may occurs if \( a^* > 0 \).

We can summarize the results obtained above in the following theorem:

**Theorem 4.1:** If \( a^* > 0 \), then model (3) exhibits backward bifurcation at \( R_0 = 1 \). If the reversed inequality holds, then the bifurcation at \( R_0 = 1 \) is forward.

This is illustrated by simulating the model with different set of parameter values (it should be stated that these parameters are chosen for illustrative purpose only, and may not necessarily be realistic epidemiologically):

—Using the parameters values in Table II except \( \mu_v = \mu_1 = \mu_2 = 1/14, \Lambda_h = 100, \epsilon = 0.80, \xi = 0.475, \delta = 0.6, \beta_{hv} = 4.0385, \beta_{vch} = 100 \) and \( K = 1000 \) such that \( R_0 = 1 \) and \( a^* = 2.3665 \times 10^{-4} > 0 \), the numerical resolution of equation (44) (see appendix A) gives the following solution: \( \lambda_{1h}^* = 0, \lambda_{2h}^* = 0.0114, \lambda_{3h}^* = 8.5310, \) and \( \lambda_{4h}^* = -0.0111 \); The first solution \( \lambda_{1h}^* = 0 \) corresponds to the disease free equilibrium. The second, and third solution, \( \lambda_{2h}^* = 0.0083, \lambda_{3h}^* = 10.9412, \) correspond to endemic equilibria; \( \lambda_{2h}^* = 0.0083 \) correspond to unstable endemic equilibrium and \( \lambda_{3h}^* = 10.9412 \) corresponds to the stable endemic equilibrium. The fourth and fifth solution \( \lambda_{4h}^* = -0.0080 \) and \( \lambda_{5h}^* = -0.0001 \) are not biologically meaningful.

—Using the parameters values in Table II except \( \mu_v = \mu_1 = \mu_2 = 1/14, \Lambda_h = 100, \epsilon = 0.80, \xi = 0.475, \delta = 0.6, \beta_{hv} = 4.0188 \) and \( K = 1000 \) such that \( R_0 = 1 \), equation (44) admit only one solution \( \lambda_{h}^* = 0 \) which corresponds to the disease–free equilibrium. In this case, the backward bifurcation phenomenon does not occurs.

—Choosing \( \beta_{hv} = 10 \) and \( K = 1000 \) such that \( R_0 = 1.630976 > 1 \) and \( a^* = -1.8011 < 0 \), equation (44) admit only one positive solution given by \( \lambda_{1h}^* = 0.0001 \), which correspond to the
endemic equilibria when the basic reproduction number, \( R_0 \), is greater than 1.

To conclude, depending to the values of parameters of model (3), the phenomenon of backward bifurcation may occurs when the classical basic reproduction number \( R_0 \) is less than unity.

V. THRESHOLD ANALYSIS AND VACCINE IMPACT

Since a future dengue vaccine, for example, is expected to be imperfect, it is instructive to determine whether or not its widespread use in a community will be benefic (or not) [10], [40], [68]. Now, consider the following model (model 3 without vaccination).

\[
\begin{align*}
\dot{S}_h &= \Lambda_h - \lambda_h S_h - \mu_h S_h \\
\dot{E}_h &= \lambda_h S_h - (\mu_h + \gamma_h) E_h \\
\dot{I}_h &= \gamma_h E_h - (\mu_h + \delta + \sigma) I_h \\
\dot{R}_h &= \sigma I_h - \mu_h R_h \\
\dot{A}_v &= \mu_b \left( 1 - \frac{A_v}{K} \right) (S_v + E_v + I_v) - (\theta + \mu_A) A_v \\
\dot{S}_v &= \theta A_v - \lambda_v S_v - \mu_v S_v \\
\dot{E}_v &= \lambda_v S_v - (\mu_1 + \gamma_v) E_v \\
\dot{I}_v &= \gamma_v E_v - \mu_2 I_v \\
\end{align*}
\]  

(19)

with \( \lambda_h \) and \( \lambda_v \), defined at (1) and (2), respectively. Following procedure in [26], [82], the basic reproduction number \( R_0 \) of model (19), \( R_s \), is given by

\[
R_s^2 = \frac{\beta_{hv} \beta_{vh} K \theta (k_3 \eta_h + \gamma_h) (\gamma_v + \eta_v \mu_2)}{\mu_v \mu_2 (\mu_h + \gamma_h) (\mu_h + \delta + \sigma) (\mu_1 + \gamma_v)} \\
	imes \frac{\Lambda_h \mu_h}{(\Lambda_h + m_m u_h)^2} \left( 1 - \frac{1}{N} \right)
\]  

(20)

So we deduce that

\[
R_{vac} := R_0 = R_s \sqrt{\frac{\pi \xi + \mu_h}{\mu_h + \xi}}
\]  

(21)

From Eq. (21), it follows that, in the absence of vaccination \( (\xi = 0) \) or when the vaccine efficacy is very low \( (\epsilon \rightarrow 0) \), we have \( R_{vac} = R_s \). However, when humans vaccination comes to play, the basic reproductive number is reduced by a factor of

\[
\sqrt{\frac{\pi \xi + \mu_h}{\mu_h + \xi}} < 1.
\]

Since increasing vaccination efforts results in decreasing the magnitude of arboviruses infection, humans vaccination can contribute to control the spread of arboviral diseases. In the following, we use the set of parameters values given in Table III, which refer to Dengue and Chikungunya. Figs. 3–5 show several simulations, by varying the vaccine efficacy and the percentage of population that is vaccinated. Figure 5 shows simulations with different proportions of susceptible human vaccinated, using an imperfect vaccine, with a level of efficacy of 60%. Both total number of infected humans and infected vectors reache a peak after 25 days approximatively. However, when \( \epsilon = 60\% \), the variation of vaccine coverage parameter have not a great impact in the number of infected humans and vectors. Figure 4 illustrates the effect of vaccine efficacy in the reduction of the total number of infected humans and vectors. It is clear that the effectiveness of the vaccine has a great impact when \( \epsilon \geq 90\% \). Thus, it is suitable to add to vaccination (when \( \epsilon < 90\% \)) another control, such as, treatment of infected individuals, personal protection, and vector control strategies to stop the spread of arboviral diseases. Figure 5 shows the representation of the basic reproduction number \( R_0 \) plotted as function of the vaccine efficacy parameter \( \epsilon \) and the proportion.
of susceptible population vaccinated \( \xi \). The use of a vaccine with level of efficacy greater than 90% approximatively, dramatically decrease the basic reproduction number, when the proportion of susceptible humans vaccinated are greater than 85%. We observe the same result at Figure 6. Thus, the use of a vaccine with a high level of efficacy and a wide vaccine coverage has an impact on stopping the spread of the disease. However, if the vaccine efficacy is not high, it is important to add another control strategies. Our sensitive analysis in later section will further support this result.

VI. SENSITIVITY ANALYSIS

To determine the best way to fight against arboviruses, it is necessary to know the relative importance of the various factors responsible for their transmission in both the human population than in the vector population, as well as effective means to fight these diseases. The transmission of the disease is directly related to \( R_0 \), and the prevalence of the disease is directly related to the infected states, especially for sizes of \( E_h(t) \), \( I_h(t) \), \( E_v(t) \) and \( I_v(t) \). These variables are relevant to

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Baseline value</th>
<th>Sources</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \Lambda_h )</td>
<td>2.5 day(^{-1} )</td>
<td>[40]</td>
</tr>
<tr>
<td>( \xi )</td>
<td>variable</td>
<td></td>
</tr>
<tr>
<td>( \epsilon )</td>
<td>Variable</td>
<td></td>
</tr>
<tr>
<td>( \eta_h, \eta_v )</td>
<td>0.35</td>
<td>Assumed</td>
</tr>
<tr>
<td>( \beta_h, \beta_v )</td>
<td>0.75 day(^{-1} )</td>
<td>[40]</td>
</tr>
<tr>
<td>( \gamma_h )</td>
<td>1/3 day(^{-1} )</td>
<td>[30]</td>
</tr>
<tr>
<td>( \gamma_v )</td>
<td>1/2 day(^{-1} )</td>
<td>[30]</td>
</tr>
<tr>
<td>( \mu_h )</td>
<td>((71 \times 365))</td>
<td>[68]</td>
</tr>
<tr>
<td>( \mu_v )</td>
<td>((1/14)) day(^{-1} )</td>
<td>[40]</td>
</tr>
<tr>
<td>( \mu_{A} )</td>
<td>1/5 day(^{-1} )</td>
<td>[30]</td>
</tr>
<tr>
<td>( \mu_{I_h} )</td>
<td>10 days</td>
<td>[30]</td>
</tr>
<tr>
<td>( \mu_{I_v} )</td>
<td>5 days</td>
<td>[30]</td>
</tr>
<tr>
<td>( \theta )</td>
<td>0.08 day(^{-1} )</td>
<td>[30], [68]</td>
</tr>
<tr>
<td>( \delta )</td>
<td>10(^{-1} ) day(^{-1} )</td>
<td>[40]</td>
</tr>
<tr>
<td>( \sigma )</td>
<td>0.1428 day(^{-1} )</td>
<td>[2], [40]</td>
</tr>
<tr>
<td>( a )</td>
<td>1 day(^{-1} )</td>
<td>[40], [61]</td>
</tr>
<tr>
<td>( n )</td>
<td>100</td>
<td>Assumed</td>
</tr>
<tr>
<td>( K )</td>
<td>(2 \times 5000)</td>
<td>Assumed</td>
</tr>
<tr>
<td>( \mu_b )</td>
<td>6 day(^{-1} )</td>
<td>[68], [60], [61]</td>
</tr>
</tbody>
</table>

Fig. 3. Total number of infected humans and vectors varying the proportion of susceptible humans vaccinated \( \xi = (0.05; 0.25; 0.5; 0.75; 1) \) with a vaccine simulating 60% of effectiveness (i.e. \( \epsilon = 0.60 \) or \( \pi = 1 - \epsilon = 0.4 \)).

Fig. 4. Infected humans and Vector varying the efficacy level of the vaccine \( \epsilon = (0.25; 0.50; 0.80; 0.90; 1) \) and considering that 85% of susceptible humans is vaccinated.
Fig. 5. The basic reproduction number $R_0$ plotted as function of the vaccine efficacy parameter $\epsilon$ and the proportion of susceptible population vaccinated $\xi$. The set of parameter values is given in Table III.

Fig. 6. Time profile of total number of infected human and vector without vaccination and with vaccination.

the individuals (humans and vectors) who have some life stage of arboviruses in their bodies. The number of infectious humans, $I_h$, is especially important because it represents the people who may be clinically ill, and is directly related to the total number of arboviral deaths \[22\]. We will perform a global sensitivity analysis.

A. Mean values of parameters and initial values of variables

Since we focus in this article, to a general model of arboviral diseases, we will, in this sec-

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean Value</th>
<th>Initial Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>$S_h$:</td>
<td>1000</td>
<td>$A_v$:</td>
</tr>
<tr>
<td>$V_h$:</td>
<td>0</td>
<td>500</td>
</tr>
<tr>
<td>$E_h$:</td>
<td>20</td>
<td>$E_v$:</td>
</tr>
<tr>
<td>$I_h$:</td>
<td>10</td>
<td>40</td>
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<tr>
<td>$R_h$:</td>
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TABLE IV

<table>
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<th>Parameter</th>
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</thead>
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<td>$\xi$</td>
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<tr>
<td>$\epsilon$</td>
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</tr>
<tr>
<td>$\eta_h, \eta_v$</td>
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</tr>
<tr>
<td>$\beta_{hv}$</td>
<td>$[0.375, 1]$</td>
</tr>
<tr>
<td>$\beta_{vh}$</td>
<td>$[0.375, 1]$</td>
</tr>
<tr>
<td>$\gamma_h$</td>
<td>$[1/12, 1/2]$</td>
</tr>
<tr>
<td>$\gamma_v$</td>
<td>$[1/21, 1/2]$</td>
</tr>
<tr>
<td>$\mu_h$</td>
<td>$\frac{1}{78 \times 365}$</td>
</tr>
<tr>
<td>$\mu_v$</td>
<td>$\frac{1}{21,1/10}$</td>
</tr>
<tr>
<td>$\mu_1$</td>
<td>$[1/10, 1/4]$</td>
</tr>
<tr>
<td>$\mu_2$</td>
<td>$[1/21, 1/3]$</td>
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<tr>
<td>$\mu_3$</td>
<td>$[1/21, 1/3]$</td>
</tr>
<tr>
<td>$\theta$</td>
<td>$[0.01, 0.17]$</td>
</tr>
<tr>
<td>$\delta$</td>
<td>$[10^{-5}, 10^{-2}]$</td>
</tr>
<tr>
<td>$\sigma$</td>
<td>$[0.1428, 0.13]$</td>
</tr>
<tr>
<td>$\alpha$</td>
<td>$[1, 3]$</td>
</tr>
<tr>
<td>$\mu$</td>
<td>$[1, 20]$</td>
</tr>
<tr>
<td>$\mu_1$</td>
<td>$[10^{-3}, 10^{-2}]$</td>
</tr>
<tr>
<td>$\mu_2$</td>
<td>$[10^{-1}, 10^{-2}]$</td>
</tr>
<tr>
<td>$\mu_3$</td>
<td>$[10^{-3}, 10^{-2}]$</td>
</tr>
<tr>
<td>$\theta$</td>
<td>$[0.01, 0.17]$</td>
</tr>
<tr>
<td>$\delta$</td>
<td>$[10^{-5}, 10^{-2}]$</td>
</tr>
<tr>
<td>$\sigma$</td>
<td>$[0.1428, 0.13]$</td>
</tr>
<tr>
<td>$\alpha$</td>
<td>$[1, 3]$</td>
</tr>
</tbody>
</table>

TABLE V

INITIAL CONDITIONS.

B. Uncertainty and sensitivity analysis

1) Sensitivity analysis of $R_0$: We study the impact of each parameter of the model on the value of the basic reproduction number $R_0$. Following the approach of Wu and colleagues \[88\], we perform the analysis by calculating the Partial Rank Correlation Coefficients (PRCC) between each parameter of our model and the basic reproduction number, $R_0$. Table IIII roughly estimates
the mean value for each parameter. It is important to notice that, variations of these parameters in our deterministic model lead to uncertainty to model predictions since the basic reproductive number varies with parameters. Due to the absence of data on the distribution function, a uniform distribution is chosen for all parameters. The sets of input parameter values sampled using the Latin Hypercube Sampling (LHS) method were used to run 1,000 simulations.

With these 1,000 runs of Latin Hypercube Sampling, the derived sampling distribution of $R_0$ is shown in Figure 7. From this sampling we get that the mean of $R_0$ is $1.9304$ and the standard deviation is $1.6128$. Hence, statistically we are very confident that model (3) is in an endemic state since $R_0 > 1$.

From the previous sampling we compute the Partial Rank Correlation Coefficients between $R_0$ and each parameter of model (3). The result is displayed in Table VI. According to Boloye Gomero [13], the parameters with large PRCC values ($> 0.5$ or $<-0.5$) as well as corresponding small p-values ($< 0.05$) are most influential in model (3).

Table VI show that the parameter $\epsilon$ have the highest influence on the reproduction number $R_0$. Although $\epsilon$ is the vaccine efficacy. This suggests that the development of a vaccine with high level of efficacy may potentially be an effective strategy to reduce $R_0$. The other parameters with an important effect are $\theta$, $a$, $\Lambda_h$ and $\mu_2$. The parameters which do not have almost any effect on $R_0$ are $\xi$, $\delta$, $\mu_A$, $m$ and $\mu_b$. In particular, the least sensitive parameters is $\mu_b$, the number of eggs at each deposit per capita.

2) Sensitivity analysis of Infected states of model (3): With 1,000 runs of Latin hypercube sampling, we compute the PRCC between infected states $E_h(t)$, $I_h(t)$, $E_v(t)$, and $I_v(t)$ and each parameter of model (3). The result is displayed in Tables VII–X. As in Table VI, the parameters with large PRCC values ($> 0.5$ or $<-0.5$) as well as corresponding small p-values ($< 0.05$) are most influential in model (3).

From Tables VII–X we can observe the following facts:

- For the value of $E_h$, the parameters with more influence are $\theta$, $K$, $a$, $\epsilon$, $\Lambda_h$ and $\mu_2$. The parameters which do not have almost any effect on the variation of $E_h$ are $\mu_h$, $\delta$, $\mu_A$, $m$ and $\mu_b$. In particular, the least sensitive parameters is $\mu_b$, the number of eggs at each deposit per capita;

- For the value of $I_h$, the parameters with more influence are $\Lambda_h$ and $\gamma_h$. The least sensitive pa-

---

**TABLE VI**

PRCC BETWEEN $R_0$ AND EACH PARAMETER.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Correlation Coefficients</th>
<th>P-values</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 $\Lambda_h$</td>
<td>*-0.6067</td>
<td>1.4578E-99</td>
</tr>
<tr>
<td>2 $\xi$</td>
<td>0.0529</td>
<td>0.0977</td>
</tr>
<tr>
<td>3 $\epsilon$</td>
<td>***-0.8043</td>
<td>2.6732E-223</td>
</tr>
<tr>
<td>4 $\eta_h$</td>
<td>0.2879</td>
<td>4.0576E-20</td>
</tr>
<tr>
<td>5 $\beta_{hv}$</td>
<td>0.4354</td>
<td>1.3609E-46</td>
</tr>
<tr>
<td>6 $\gamma_h$</td>
<td>-0.2598</td>
<td>1.4099E-16</td>
</tr>
<tr>
<td>7 $\mu_h$</td>
<td>0.2526</td>
<td>9.9492E-16</td>
</tr>
<tr>
<td>8 $\delta$</td>
<td>-0.0386</td>
<td>0.2274</td>
</tr>
<tr>
<td>9 $\sigma$</td>
<td>-0.3269</td>
<td>7.7785E-26</td>
</tr>
<tr>
<td>10 $\eta_v$</td>
<td>0.2039</td>
<td>1.1635E-10</td>
</tr>
<tr>
<td>11 $\beta_{vh}$</td>
<td>0.4215</td>
<td>1.7130E-43</td>
</tr>
<tr>
<td>12 $\gamma_v$</td>
<td>0.2117</td>
<td>2.1787E-11</td>
</tr>
<tr>
<td>13 $\mu_v$</td>
<td>-0.3029</td>
<td>3.0015E-22</td>
</tr>
<tr>
<td>14 $\mu_A$</td>
<td>-0.0121</td>
<td>0.7049</td>
</tr>
<tr>
<td>15 $\mu_1$</td>
<td>-0.2948</td>
<td>4.2501E-21</td>
</tr>
<tr>
<td>16 $\mu_2$</td>
<td>*-0.5087</td>
<td>1.2669E-65</td>
</tr>
<tr>
<td>17 $\theta$</td>
<td>**0.7626</td>
<td>3.0823E-187</td>
</tr>
<tr>
<td>18 $\alpha$</td>
<td>**0.7134</td>
<td>3.4096E-153</td>
</tr>
<tr>
<td>19 $m$</td>
<td>-0.0436</td>
<td>0.1724</td>
</tr>
<tr>
<td>20 $K$</td>
<td>0.3880</td>
<td>1.4683E-36</td>
</tr>
<tr>
<td>21 $\mu_b$</td>
<td>0.0082</td>
<td>0.7973</td>
</tr>
</tbody>
</table>

---

**Fig. 7.** Sampling distribution of $R_0$ from 1,000 runs of Latin hypercube sampling. The mean of $R_0$ is $1.9304$ and the standard deviation is $1.6128$. 

---


TABLE VII  
PRCC BETWEEN INFECTED HUMANS \( E_h \) AND EACH PARAMETER.  

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Correlation Coefficients ( \varphi )</th>
<th>P-values ( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \Lambda_h )</td>
<td><strong>0.6842</strong></td>
<td>3.2080 ( E )–136</td>
</tr>
<tr>
<td>( \xi )</td>
<td>0.4115</td>
<td>2.4590 ( E )–41</td>
</tr>
<tr>
<td>( \epsilon )</td>
<td>0.7177</td>
<td>6.8762 ( E )–156</td>
</tr>
<tr>
<td>( \eta_h )</td>
<td>–0.2457</td>
<td>6.130E–15</td>
</tr>
<tr>
<td>( \beta_{hv} )</td>
<td>–0.4215</td>
<td>1.7187 ( E )–43</td>
</tr>
<tr>
<td>( \gamma_h )</td>
<td>0.2172</td>
<td>6.2865 ( E )–12</td>
</tr>
<tr>
<td>( \mu_h )</td>
<td>0.0086</td>
<td>0.7879</td>
</tr>
<tr>
<td>( \delta )</td>
<td>–0.0259</td>
<td>0.4176</td>
</tr>
<tr>
<td>( \sigma )</td>
<td>0.3395</td>
<td>7.4246 ( E )–28</td>
</tr>
<tr>
<td>( \eta_v )</td>
<td>–0.2378</td>
<td>4.5858 ( E )–14</td>
</tr>
<tr>
<td>( \beta_{vh} )</td>
<td>–0.4232</td>
<td>7.4972 ( E )–44</td>
</tr>
<tr>
<td>( \gamma_v )</td>
<td>–0.2311</td>
<td>2.4083 ( E )–13</td>
</tr>
<tr>
<td>( \mu_v )</td>
<td>0.2906</td>
<td>1.5881 ( E )–20</td>
</tr>
<tr>
<td>( \mu_A )</td>
<td>0.0210</td>
<td>0.5122</td>
</tr>
<tr>
<td>( \mu_1 )</td>
<td>0.3340</td>
<td>5.8090 ( E )–27</td>
</tr>
<tr>
<td>( \mu_2 )</td>
<td>*0.5747</td>
<td>3.1691 ( E )–87</td>
</tr>
<tr>
<td>( \theta )</td>
<td>***–0.7599</td>
<td>3.7832 ( E )–185</td>
</tr>
<tr>
<td>( \alpha )</td>
<td>***–0.7597</td>
<td>4.9923 ( E )–185</td>
</tr>
<tr>
<td>( \eta )</td>
<td>0.0537</td>
<td>0.0931</td>
</tr>
<tr>
<td>( K )</td>
<td>***–0.7477</td>
<td>4.2124 ( E )–176</td>
</tr>
<tr>
<td>( \mu_b )</td>
<td>–0.0068</td>
<td>0.8328</td>
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</tbody>
</table>

TABLE VIII  
PRCC BETWEEN INFECTIOUS HUMANS \( I_h \) AND EACH PARAMETER.  

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Correlation Coefficients ( \varphi )</th>
<th>P-values ( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \Lambda_h )</td>
<td>***0.8727</td>
<td>9.1342 ( E )–307</td>
</tr>
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<td>( \xi )</td>
<td>0.0078</td>
<td>0.8062</td>
</tr>
<tr>
<td>( \epsilon )</td>
<td>–0.2887</td>
<td>2.8614 ( E )–20</td>
</tr>
<tr>
<td>( \eta_h )</td>
<td>0.0711</td>
<td>0.0261</td>
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<tr>
<td>( \beta_{hv} )</td>
<td>0.0850</td>
<td>0.0078</td>
</tr>
<tr>
<td>( \gamma_h )</td>
<td>***–0.8722</td>
<td>5.9181 ( E )–306</td>
</tr>
<tr>
<td>( \mu_h )</td>
<td>–0.0363</td>
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</tr>
<tr>
<td>( \delta )</td>
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<td>0.1978</td>
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<td>( \sigma )</td>
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<td>0.0965</td>
</tr>
<tr>
<td>( \eta_v )</td>
<td>0.0310</td>
<td>0.3316</td>
</tr>
<tr>
<td>( \beta_{vh} )</td>
<td>0.1297</td>
<td>4.6364 ( E )–5</td>
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<td>( \gamma_v )</td>
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<td>0.5764</td>
</tr>
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<td>( \mu_v )</td>
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</tr>
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<td>( \mu_A )</td>
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<tr>
<td>( \mu_1 )</td>
<td>–0.0580</td>
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<tr>
<td>( \mu_2 )</td>
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<td>0.1855</td>
</tr>
<tr>
<td>( \theta )</td>
<td>0.1312</td>
<td>3.7931 ( E )–5</td>
</tr>
<tr>
<td>( \alpha )</td>
<td>0.1428</td>
<td>2.8933 ( E )–6</td>
</tr>
<tr>
<td>( m )</td>
<td>–0.0017</td>
<td>0.9586</td>
</tr>
<tr>
<td>( K )</td>
<td>0.1783</td>
<td>1.9260 ( E )–8</td>
</tr>
<tr>
<td>( \mu_b )</td>
<td>–0.0054</td>
<td>0.8648</td>
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TABLE IX  
PRCC BETWEEN INFECTED VECTORS \( E_v \) AND EACH PARAMETER.  

<table>
<thead>
<tr>
<th>Parameter</th>
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<th>P-values ( p )</th>
</tr>
</thead>
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<td>–0.0186</td>
<td>0.5603</td>
</tr>
<tr>
<td>( \xi )</td>
<td>–0.0111</td>
<td>0.7280</td>
</tr>
<tr>
<td>( \epsilon )</td>
<td>0.0135</td>
<td>0.6723</td>
</tr>
<tr>
<td>( \eta_h )</td>
<td>–0.1086</td>
<td>6.6203 ( E )–4</td>
</tr>
<tr>
<td>( \beta_{hv} )</td>
<td>–0.0664</td>
<td>0.0375</td>
</tr>
<tr>
<td>( \gamma_h )</td>
<td>0.0560</td>
<td>0.0798</td>
</tr>
<tr>
<td>( \mu_h )</td>
<td>–0.0295</td>
<td>0.3563</td>
</tr>
<tr>
<td>( \delta )</td>
<td>0.0116</td>
<td>0.7170</td>
</tr>
<tr>
<td>( \sigma )</td>
<td>0.0734</td>
<td>0.0215</td>
</tr>
<tr>
<td>( \eta_v )</td>
<td>–0.0273</td>
<td>0.3928</td>
</tr>
<tr>
<td>( \beta_{vh} )</td>
<td>–0.0913</td>
<td>0.0043</td>
</tr>
<tr>
<td>( \gamma_v )</td>
<td>0.0669</td>
<td>0.8282</td>
</tr>
<tr>
<td>( \mu_v )</td>
<td>***–0.5923</td>
<td>7.6830 ( E )–94</td>
</tr>
<tr>
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<td>0.0157</td>
<td>0.6235</td>
</tr>
<tr>
<td>( \mu_1 )</td>
<td>0.0331</td>
<td>0.3006</td>
</tr>
<tr>
<td>( \mu_2 )</td>
<td>0.0043</td>
<td>0.8933</td>
</tr>
<tr>
<td>( \theta )</td>
<td>***–0.9225</td>
<td>0</td>
</tr>
<tr>
<td>( \alpha )</td>
<td>–0.0822</td>
<td>0.0100</td>
</tr>
<tr>
<td>( \mu )</td>
<td>0.0027</td>
<td>0.9324</td>
</tr>
<tr>
<td>( K )</td>
<td>***–0.9199</td>
<td>0</td>
</tr>
<tr>
<td>( \mu_b )</td>
<td>0.1125</td>
<td>4.1594 ( E )–4</td>
</tr>
</tbody>
</table>

TABLE X  
PRCC BETWEEN INFECTIOUS VECTORS \( I_v \) AND EACH PARAMETER.  

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Correlation Coefficients ( \varphi )</th>
<th>P-values ( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \Lambda_h )</td>
<td>0.2254</td>
<td>9.3729 ( E )–13</td>
</tr>
<tr>
<td>( \xi )</td>
<td>–0.0090</td>
<td>0.7785</td>
</tr>
<tr>
<td>( \epsilon )</td>
<td>–0.1228</td>
<td>1.1697 ( E )–4</td>
</tr>
<tr>
<td>( \eta_h )</td>
<td>0.3126</td>
<td>1.1661 ( E )–23</td>
</tr>
<tr>
<td>( \beta_{hv} )</td>
<td>0.0031</td>
<td>0.9216</td>
</tr>
<tr>
<td>( \gamma_h )</td>
<td>–0.3233</td>
<td>2.7921 ( E )–25</td>
</tr>
<tr>
<td>( \mu_v )</td>
<td>0.0381</td>
<td>0.2338</td>
</tr>
<tr>
<td>( \mu_b )</td>
<td>–0.0215</td>
<td>0.5015</td>
</tr>
<tr>
<td>( \sigma )</td>
<td>–0.3869</td>
<td>2.4025 ( E )–36</td>
</tr>
<tr>
<td>( \eta_v )</td>
<td>0.0196</td>
<td>0.5402</td>
</tr>
<tr>
<td>( \beta_{vh} )</td>
<td>0.5584</td>
<td>2.0585 ( E )–109</td>
</tr>
<tr>
<td>( \gamma_v )</td>
<td>–0.6287</td>
<td>6.0859 ( E )–109</td>
</tr>
<tr>
<td>( \mu_v )</td>
<td>–0.4856</td>
<td>4.0722 ( E )–59</td>
</tr>
<tr>
<td>( \mu_A )</td>
<td>0.0294</td>
<td>0.3583</td>
</tr>
<tr>
<td>( \mu_1 )</td>
<td>–0.4380</td>
<td>3.3922 ( E )–47</td>
</tr>
<tr>
<td>( \mu_2 )</td>
<td>–0.0103</td>
<td>0.7470</td>
</tr>
<tr>
<td>( \theta )</td>
<td>**0.8728</td>
<td>7.6088 ( E )–307</td>
</tr>
<tr>
<td>( \alpha )</td>
<td>*0.6011</td>
<td>2.5895 ( E )–97</td>
</tr>
<tr>
<td>( \mu )</td>
<td>–0.0640</td>
<td>0.0451</td>
</tr>
<tr>
<td>( K )</td>
<td>**0.8600</td>
<td>5.9602 ( E )–288</td>
</tr>
<tr>
<td>( \mu_b )</td>
<td>–0.0770</td>
<td>0.0159</td>
</tr>
</tbody>
</table>
rameters is $\mu_v$, the number of eggs at each deposit per capita;

-For the value of $E_v$, the parameters with more influence are the maturation rate from larvae to adult $\theta$, and the capacity of breeding sites $K$. The other parameter is the natural mortality rate of vector $\mu_v$. The least sensitive parameters is $m$, the number of alternative source of blood;

-For the value of $I_v$, the parameters with more influence are $\theta$, $K$, $\gamma_v$, $a$ and $\beta_{vh}$. The least sensitive parameters is $m$, the probability of transmission of infection from an infectious vector to a susceptible human.

Although the model is sensitive to the variations of the vaccine efficacy parameter $\epsilon$, there are other parameters (such as $\theta$, $a$, $K$, $\mu_v$, $\mu_2$) which have a considerable impact on the value of the basic reproduction number $R_0$ and the number of infected individuals. Thus, it is important to take into account other control strategies in the fight against arboviral diseases.

VII. NUMERICAL SIMULATION

In order to illustrate some of the results obtained in the previous sections, we provide here some numerical simulations. We use the nonstandard scheme given by (22). It is important to note that standard numerical methods may fail to preserve the dynamics of continuous models [4], [59], [81]. Generally, compartmental models are solved using standard numerical methods, for example, Euler or Runge Kutta methods included in software package such as Scilab [76] or Matlab [57]. These methods can sometimes lead to spurious behaviours which are not in adequacy with the continuous system properties that they aim to approximate. For example, they may lead to negative solutions, exhibit numerical instabilities, or even converge to the wrong equilibrium for certain values of the time discretization or the model parameters (see [3], [4], [5], [81] for further investigations).

A. A nonstandard scheme for the model (3)

Following [30], system (3) is discretized as follows:

$$ \begin{aligned}
\frac{X^S_{k+1} - X^S_k}{\phi(h)} &= A_1(X^k)X^k_S - X_{DFE}D_{12}(X^k)X^k_S + B_{12}(X^k)X^k_I \\
\frac{X^I_{k+1} - X^I_k}{\phi(h)} &= A_2(X^k_S)X^k_I
\end{aligned} $$

(22)

such that

$$ -D_{12}(X_I)X_S + B_{12}(X)X_I = A_{12}(X)X_I $$

(23)

with

$$ D_{12}(X_I) = \begin{pmatrix} 
\lambda_h & 0 & 0 & 0 & 0 \\
0 & \pi \lambda_h & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & \lambda_v \\
0 & 0 & 0 & 0 & 0 
\end{pmatrix}, $$

and

$$ B_{12}(X) = \begin{pmatrix} 
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & \sigma & 0 & 0 & 0 \\
0 & 0 & \mu_b \left(1 - \frac{A_0}{K}\right) & \mu_b \left(1 - \frac{A_v}{K}\right) & 0 \\
0 & 0 & 0 & 0 & 0 
\end{pmatrix}, $$

which implies that the scheme is consistant with formulation (11).

Rearranging (22), we obtain the foollowing new expression

$$ \begin{aligned}
A_k X^k_{k+1} &= B_k \\
X^k &\geq 0
\end{aligned} $$

(24)

with

$$ A_k = \begin{pmatrix} 
I_5 + \phi(h)D_{12}(X^k_I) & 0 \\
0 & I_5
\end{pmatrix} $$

and

$$ B_k = \begin{pmatrix} 
X^S_k + \phi(h) \left[ A_1(X^k)X^k_S - X_{DFE}D_{12}(X^k)X^k_S + B_{12}(X^k)X^k_I \right] \\
X^I_k \left[ I_4 + \phi(h)A_2(X^k_S) \right]
\end{pmatrix}. $$

where $I_4$ and $I_5$ are the identity matrix of order 4 and 5 respectively. Thus, we claim the following result:
Lemma 7.1: Our non-standard numerical scheme (22) is positively stable, i.e. for \( X^k \geq 0 \) we obtain \( X^{k+1} \geq 0 \), where \( X^k = (S^k_h, V^k_h, E^k_h, I^k_h, B^k_h, A^k_v, S^k_v, E^k_v, I^k_v)^T \).

Proof: We suppose \( X_k \geq 0 \). \( A_k \) is a positive diagonal matrix and thus, \( A_k^{-1} \geq 0 \). \( B_{12} \) is a positive matrix and we also have \( -A_1(X^k)X_{DFE} \geq 0 \). To show that \( B_k \) is a positive matrix, it suffices to choose \( \phi(h) \) such that

\[
I_d + \phi(h)A_1(X) \geq I_d + \phi(h)A_1 \geq 0,
I_d + \phi(h)A_2(X) \geq I_d + \phi(h)A_2 \geq 0
\]

where \( A_1 \) and \( A_2 \) are lower bounds for the sets \( \{ X \in \mathcal{D} | A_1(X) \} \) and \( \{ X \in \mathcal{D} | A_2(X) \} \) respectively. Following [30], to have \( B_k \geq 0 \), it suffices to consider the following time-step function

\[
\phi(h) = \frac{1 - e^{-Qh}}{Q} \quad (25)
\]

with \( Q \geq \max(k_1, k_2, k_3, \mu_h, k_4, k_6, \mu_v, \mu_2) \). We have proved that \( X^k \geq 0 \) implies \( X^{k+1} \geq 0 \). ■

Concerning the equilibria of our numerical scheme, we have the following result

Lemma 7.2: Our non-standard numerical scheme (22) and the continuous model (3) have the same equilibria.

Proof: See appendix F. ■

The stability of the trivial equilibrium is given by the following result

Lemma 7.3: If \( \phi(h) \) has choosen as equation (25), then the trivial equilibrium \( TE := P_0 \) is locally asymptotically stable for our numerical scheme (22) whenever \( N \leq 1 \).

Proof: See appendix G. ■

Now, we also have the following result concerning the stability of the disease-free equilibrium:

Lemma 7.4: If \( \phi(h) \) has choosen as equation (25) and \( R_0 < 1 \), then the disease-free equilibrium \( DFE := P_1 \) is locally asymptotically stable for our numerical scheme (22).

Proof: The proof of Lemma 7.4 follows the proof of Proposition 3.4 in [30]. See also [5] for a proof in a more general setting. ■

VIII. Conclusions

In this paper, we formulated a compartmental model which takes into account a future vaccination strategy in human population, the aquatic development stage of vector and the alternative sources of blood.

The analysis has been performed by means of stability, bifurcation and sensitivity analysis. We have obtained that the disease-induced mortality may be the main cause for the occurrence of the backward bifurcation (see remark 4.1). This means that relatively high values of disease-induced mortality rate may induce stable endemic states also when the basic reproduction number \( R_0 \) is below the classical threshold \( R_0 = 1 \). The stability analysis reveals that for \( N \leq 1 \),
the absence of disease-induced death, the disease–free equilibrium is also globally asymptotically stable. In the absence of disease-induced death, the disease–free equilibrium is also globally asymptotically stable. In
An endemic equilibrium point whenever its associated human and vector populations) have a unique stable. The reduced version of the model \( \mathcal{R} \) (in the absence of disease–induced mortality in both human and vector populations) have a unique endemic equilibrium point whenever its associated reproduction number \( R_1 \) exceeds unity.

Taking as cases study the dengue and chikungunya transmission, we used parameter values from the literature to estimate statistically the basic reproduction number, \( R_0 \), and to perform a global sensitivity analysis on the basic reproduction number and infected states \( (E_h, I_h, E_v, I_v) \). Using Latin Hypercube Sampling, we obtain the mean of \( R_0 \) is 1.9304. Hence, statistically we are very confident that our model \( \mathcal{R} \) is in an endemic state. The global sensitivity analysis reveals that, apart from the parameters related to vaccination, particularly vaccine efficacy, other parameters ( such as parameters related to vector population) also have a great impact on the basic reproduction number \( (R_0) \) and on the number of infected humans and vectors \( (E_h, I_h, E_v, I_v) \).

Numerical simulations of the model \( \mathcal{R} \), using a nonstandard qualitatively stable scheme, show that the use of a vaccine with high level of efficacy has a proponderant role in the reduction of the disease spread. However, since the efficacy of the proposed vaccine for dengue, for example, has been around 60%, it is suitable to combine vaccination with other mechanisms of control.

Also, to be the best control strategy, the vaccination process must verify the following conditions:

(a) The vaccine must be approved by the relevant agencies (such as WHO, CDC), before its use.
(b) The vaccine efficacy should be high, as well as vaccine coverage.
(c) The price of the vaccine must be low for countries affected by the disease.

There are already governments, affected by the diseases, willing to use the vaccine before it is approved, which can have unpredictable consequences, so condition (a) does not hold. Moreover, according to previous analysis and french laboratory SANOFI, the condition (b) does not hold. Thus it is important to know what happens when we combine vaccination with other mechanisms of control already studied in the literature, such as personal protection, chemical interventions and education campaigns \([30], [40], [60], [61], [63], [64], [67], [68], [69]\). This is the perspective of our work.

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APPENDIX

Appendix A: Proof of Proposition 3.1

To find the equilibrium points of our system, we will solve the following system:

\[
\begin{align*}
\Lambda_h - \lambda_h S_h - (\xi + \mu_h)S_h &= 0 \\
\xi S_h - (1 - \epsilon)\lambda_h V_h - \mu_h V_h &= 0 \\
\lambda_h [S_h + (1 - \epsilon)V_h] - (\mu_h + \gamma_h)E_h &= 0 \\
\gamma_h E_h - (\mu_h + \delta + \sigma)I_h &= 0 \\
\sigma I_h - \mu_h R_h &= 0 \\
\mu_b \left(1 - \frac{A_v}{K}\right) (S_v + E_v + I_v) - (\theta + \mu_A)A_v &= 0 \\
\theta A_v - \lambda_v S_v - \mu_v S_v &= 0 \\
\lambda_v S_v - (\mu_1 + \gamma_v)E_v &= 0 \\
\gamma_v E_v - \mu_2 I_v &= 0
\end{align*}
\]

(26)

To this aim, let \( P^* = (S_h^*, E_h^*, I_h^*, R_h^*, A_v^*, S_v^*, E_v^*, I_v^*) \) represents any arbitrary endemic equilibrium of \( \mathcal{R} \). Further, let

\[
\lambda_h^* = \frac{\beta_vh(\eta_h E_v^* + I_v^*)}{(N_h^* + m)} , \quad \lambda_v^* = \frac{\beta_vh(\eta_v E_v^* + I_h^*)}{(N_v^* + m)}
\]

(27)

be the forces of infection of humans and vectors at steady state, respectively. Solving the first five
equations in (26) at steady state gives

\[ S_h^* = \frac{\Lambda_h}{k_1 + \lambda_h^*}, \quad V_h^* = \frac{\xi \Lambda_h}{(k_1 + \lambda_h^*) (\pi \lambda_h^* + \mu_h)}, \]
\[ E_h^* = \frac{k_2 (k_1 + \lambda_h^*) (\pi \lambda_h^* + \mu_h)}{\lambda_h^* (\pi \xi + \mu_h + \pi \lambda_h^*)}, \]
\[ I_h^* = \frac{\gamma_h \lambda_h^* (\pi \xi + \mu_h + \pi \lambda_h^*)}{k_2 k_3 (k_1 + \lambda_h^*) (\pi \lambda_h^* + \mu_h)}, \]
\[ R_h^* = \frac{\sigma \gamma_h \lambda_h^* (\pi \xi + \mu_h + \pi \lambda_h^*)}{\mu_h k_2 k_3 (k_1 + \lambda_h^*) (\pi \lambda_h^* + \mu_h)}. \]

where \( \pi = 1 - \epsilon, k_1 = \mu_h + \xi, k_2 = \mu_h + \gamma_h \) and \( k_3 = \mu_h + \sigma + \delta \). Solving the last three equations in (26) at steady state gives

\[ S_v^* = \frac{\theta A_v^* (\mu_v + \lambda_v^*)}{(\mu_v + \lambda_v^*)}, \quad E_v^* = \frac{\theta A_v^* \lambda_v^*}{k_4 (\mu_v + \lambda_v^*)}, \]
\[ I_v^* = \frac{\gamma_v \theta A_v^* \lambda_v^*}{\mu_2 k_4 (\mu_v + \lambda_v^*)}, \]

where \( k_4 = \mu_1 + \gamma_v \).

Substituting (29) in the sixth equation of (26) gives

\[ A_v^* \left( \frac{\mu_1 \theta}{\mu_2 k_4} \left( 1 - \frac{A_v^*}{K} \right) \left( \mu_2 k_4 + k_5 \lambda_v^* \right) - k_6 \right) = 0 \]

with \( k_5 = \mu_2 + \gamma_v \) and \( k_6 = \theta + \mu_A \).

The trivial solution of (30) is \( A_v^* = 0 \). Substituting this solution in (29) gives \( S_v^* = E_v^* = I_v^* = 0 \). When \( E_v^* = I_v^* = 0 \), we also have \( \lambda_h^* = 0 \), thus \( E_h^* = I_h^* = R_h^* = 0, S_h^* = \frac{\Lambda_h}{k_1} \) and \( V_h^* = \frac{\xi \Lambda_h}{\mu_h k_1} \).

Then we obtain the trivial equilibrium \( T_0^* = \left( \frac{\Lambda_h}{k_1}, \frac{\xi \Lambda_h}{\mu_h k_1}, 0, 0, 0, 0, 0, 0, 0, 0 \right) \).

Now we suppose that \( A_v^* \neq 0 \). The possible solution(s) of (30) is the solution(s) of the following equation

\[ \frac{\mu_1 \theta}{\mu_2 k_4} \left( 1 - \frac{A_v^*}{K} \right) \left( \mu_2 k_4 + k_5 \lambda_v^* \right) - k_6 = 0 \]

The direct resolution of equation (31) gives

\[ A_v^* = K \left( \frac{\mu_2 k_4 \theta k_4 \left( 1 - \frac{1}{\lambda_v^*} \right) + \alpha \lambda_v^*}{\mu_6 (\mu_2 k_4 + k_5 \lambda_v^*)} \right) \]

where \( \lambda_v^* = \frac{\mu_6 \theta}{\mu_v k_6} \) and \( \alpha = \mu_v k_5 - \mu_2 k_4 k_6 \).

Let us first compute the equilibrium without Disease, i.e. \( \lambda_h^* = \lambda_v^* = 0 \) or \( E_h = I_h = E_v = I_v = 0 \). From (32), we obtain

\[ A_v^0 := K \left( 1 - \frac{\mu_v k_6}{\mu_6 \theta} \right) := K \left( 1 - \frac{1}{\lambda_v^*} \right) \]

Thus, the existence of vector is regulated by the threshold \( \lambda_v^* \). If \( \lambda_v^* \leq 1 \), the system (3) correspond to human population of free vectors and the trivial equilibrium in this case is \( P_0 \).

Now we suppose that \( \lambda_v^* > 1 \). From (28) and (29) (with \( \lambda_v^* = 0 \)), we obtain the non trivial equilibrium or the disease–free equilibrium \( P_1 = (S_h^0, V_h^0, 0, 0, 0, A_h^0, S_v^0, 0, 0) \), where

\[ S_h^0 = \frac{\Lambda_h}{k_1}, \quad V_h^0 = \frac{\xi \Lambda_h}{k_1 \mu_h}, \quad A_h^0 = K \left( 1 - \frac{1}{\lambda_v^*} \right), \]
\[ S_v^0 = \frac{\theta A_v^0}{\mu_v}. \]

**Appendix B: Proof of Proposition 3.2**

We consider the Jacobian matrix associated to model (3) at the equilibrium. \( T(E) \).

We have

\[ \mathcal{J}(T(E)) = \begin{pmatrix} -k_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \xi & -\mu_h & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -k_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_h & -k_3 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \sigma & -\mu_h & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -k_6 & 0 & 0 & 0 & 0 \\ -\beta_{hv} \eta_v S_v^0 & 0 & 0 & 0 & 0 & 0 & \beta_{hv} \eta_v S_v^0 & \beta_{hv} S_v^0 & 0 & 0 \\ -\pi \beta_{hv} \eta_v V_h^0 & 0 & 0 & 0 & 0 & 0 & -\pi \beta_{hv} V_h^0 & -\pi \beta_{hv} S_v^0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \mu_h & \mu_v \\ 0 & 0 & 0 & 0 & 0 & 0 & \mu_b & \mu_v & \mu_b \\ 0 & -k_4 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \gamma_v & -\mu_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \]

were \( S_v^0 = S_v^0 + \pi V_v^0 \). The eigenvalues of \( \mathcal{J}(T(E)) \) are given by \( \lambda_1 = \lambda_2 = -\mu_h, \lambda_3 = -k_1, \lambda_4 = -k_2, \lambda_5 = -k_3, \lambda_6, \lambda_7, \lambda_8, \lambda_9 \) are eigenvalues of the submatrix...
We have trivial equilibrium The Routh–Hurwitz criterion of stability of the setting

\[
\mathcal{P}(\lambda) = \lambda^4 + A_1 \lambda^3 + A_2 \lambda^2 + A_3 \lambda + A_4 = 0 \quad (34)
\]

where \( A_1 = \mu_v + \mu_2 + k_4 + k_6 \), \( A_2 = k_6 \mu_v (1 - N) + (k_4 + \mu_2) (\mu_v + k_6) + \mu_2 k_4 \), \( A_3 = k_6 \mu_v (1 - N) (k_4 + \mu_2) + \mu_2 k_4 (\mu_v + k_6) \) and \( A_4 = \mu_2 k_4 (1 - N) \). Thus, it is clear that all coefficients are always positive since \( N < 1 \).

Now we just have to verify that the Routh–Hurwitz criterion holds for polynomial \( \mathcal{P}(\lambda) \). To this aim, setting

\[
H_1 = A_1, \quad H_2 = \begin{bmatrix} A_1 & 1 \\ A_3 & A_2 \end{bmatrix}, \quad H_3 = \begin{bmatrix} A_1 & 1 & 0 \\ A_3 & A_2 & A_1 \end{bmatrix}, \quad H_4 = \begin{bmatrix} A_1 & 1 & 0 & 0 \\ A_3 & A_2 & A_1 & 0 \\ 0 & A_4 & A_3 & A_2 \\ 0 & 0 & 0 & A_4 \end{bmatrix} = A_4 H_3.
\]

The Routh–Hurwitz criterion of stability of the trivial equilibrium \( T_E \) is given by

\[
\begin{cases}
H_1 > 0 \\
H_2 > 0 \\
H_3 > 0 \\
H_4 > 0
\end{cases} \iff \begin{cases}
H_1 > 0 \\
H_2 > 0 \\
H_3 > 0 \\
A_4 > 0
\end{cases} \quad (35)
\]

We have

\[
H_1 = A_1 = \mu_v + \mu_2 + k_4 + k_6 > 0,
\]

\[
H_2 = A_1 A_2 - A_3 = (k_6 + k_4 + \mu_2) \mu_v^2 + \left( \mu_2 k_6 \left( 1 - \frac{\mu_6 \theta}{\mu_2 k_6} \right) + k_6^2 + 2 k_4 k_6 + \mu_2 k_4 + k_4^2 + 2 \mu_2 k_4 + \mu_2^2 \right) \mu_v
\]

\[
+ \mu_2 k_4^2 \left( 1 - \frac{\mu_6 \theta}{\mu_2 k_6} \right) + k_4 k_6^2 + (k_4 + \mu_2)^2 k_6
\]

\[
+ \mu_2 k_4^2 + \mu_2^2 k_4,
\]

\[
H_3 = A_1 A_2 A_3 - A_1^2 A_4 = A_3^2
\]

\[
= (k_4 + \mu_2) (k_6 + \mu_v)
\]

\[
\times \left( k_6 \mu_v (1 - N) + \mu_2 \mu_v + k_6 + \mu_2^2 \right)
\]

\[
\times \left( k_6 \mu_v (1 - N) + k_4 \mu_v + k_4 k_6 + k_4^2 \right).
\]

Using inequality \( 1/\mu_2 \leq 1/\mu_1 \leq 1/\mu_v \), we obtain \( H_2 > 0 \), \( H_3 > 0 \) if \( N < 1 \); \( A_4 > 0 \) if and only if \( N < 1 \). Thus we conclude that the trivial equilibrium is locally asymptotically stable.

Now we assume that \( N > 1 \). Following the procedure and the notation in [82], we may obtain the basic reproduction number \( R_0 \) as the dominant eigenvalue of the next–generation matrix [26], [82]. Observe that model (3) has four infected populations, namely \( E_h, I_h, E_v, I_v \). It follows that the matrices \( F \) and \( V \) defined in [82], which take into account the new infection terms and remaining transfer terms, respectively, are given by

\[
F = \frac{1}{N_h^0 + m} \begin{pmatrix}
\beta_{hv} \eta_v S_0 & \beta_{hv} S_0 \\
0 & 0 & 0 & 0 \\
\beta_{vh} \eta_h S_0 & \beta_{vh} S_0 \\
0 & 0 & 0 & 0
\end{pmatrix},
\]

with \( N_h^0 = \frac{\Lambda_h}{\mu_v} \),

\[
V = \begin{pmatrix}
(\mu_h + \gamma_h) & 0 & 0 & 0 \\
-\gamma_h & (\mu_h + \delta + \sigma) & 0 & 0 \\
0 & 0 & (\mu_v + \gamma_v) & 0 \\
0 & 0 & -\gamma_v & \mu_2
\end{pmatrix},
\]

and the dominant eigenvalue of the next–generation matrix \( FV^{-1} \) is given by Eq. (10).

The local stability of the disease–free equilibrium \( P_t \) is a direct consequence of Theorem 2 of [82]. This ends the proof.

**Appendix C: Proof of Proposition 3.3**

Setting \( Y = X - T_E \) with

\[
X = (S_h, V_h, E_h, I_h, R_h, A_v, S_v, E_v, I_v)^T,
\]

we can rewrite (3) in the following manner

\[
\frac{dY}{dt} = B(Y)Y \quad (36)
\]
where 
\[ B(Y) = \begin{pmatrix}
-\lambda_h - k_1 & 0 & 0 & 0 & 0 \\
\xi & -\pi_l h - \mu_h & 0 & 0 & 0 \\
\lambda_h & \pi_l h & -k_2 & 0 & 0 \\
0 & 0 & \gamma & -k_3 & 0 \\
0 & 0 & 0 & \sigma & -\mu_h \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0
\end{pmatrix} \]

and \( \lambda^*_h \neq 0 \). We assume that \( \mathcal{N} > 1 \).

From the sixth equation of (26), at equilibrium, we have
\[ S^*_v + E^*_v + I^*_v = \frac{Kk_6A^*_v}{\mu_b(K - A^*_v)} \]  
(37)

From the last third equations of (26), at equilibrium, we have
\[ \mu_vS^*_v + \mu_1E^*_v + \mu_2I^*_v = \theta A^*_v \]  
(38)

we will observe the following two cases.

a) Absence of disease–induced death in vector: The absence of disease–induced death in vector is traduce by the relation \( \mu_v = \mu_1 = \mu_2 \), then equation (38) becomes
\[ S^*_v + E^*_v + I^*_v = \theta A^*_v \]  
(39)

Equalling Eqs. (37) and (39) gives like before
\[ A^*_v := K \left( 1 - \frac{\mu_vk_6}{\mu_b\theta} \right) = K \left( 1 - \frac{1}{\mathcal{N}} \right) \]  
(40)

Substituting \( A^*_v \) by \( A^*_v \) in equation (29) gives
\[ S^*_v = \frac{1 - \frac{1}{\mathcal{N}}}{\left( \theta^{-1} \right)} \frac{K\theta}{(\mu_v + \lambda^*_v)} , \\
E^*_v = \frac{1 - \frac{1}{\mathcal{N}}}{\left( \theta^{-1} \right)} \frac{k_3(\mu_v + \lambda^*_v)}{k_7\theta \gamma_v \lambda^*_v} , \\
I^*_v = \frac{1 - \frac{1}{\mathcal{N}}}{\left( \theta^{-1} \right)} \frac{\mu_vk_4}{\mu_b(\mu_v + \lambda^*_v)} . \\
\]  
(41)

Replacing (41) in the expression of \( \lambda^*_h \) gives
\[ \lambda^*_h = \frac{\beta_{hv}(\eta_v E^*_v + I^*_v)}{(N^*_h + m)} = k_{10} \frac{\lambda^*_v}{\theta} \times \\
\left( \frac{\beta_{hv}\mu_hk_2k_3(k_1 + \lambda^*_h)(\pi\lambda^*_h + \mu_h)}{k_2k_3k_7(k_1 + \lambda^*_h)(\pi\lambda^*_h + \mu_h) - \delta\gamma_h\lambda_h\lambda^*_h(k_8 + \pi\lambda^*_h)} \right) \]  
(42)

where \( k_7 = (\Lambda_h + m\mu_h) \), \( k_8 = \pi\xi + \mu_h \), \( k_9 = \mu_2\eta_v + \gamma_v = \eta_m\mu_h + \gamma_v \) and
\[ k_{10} = \frac{\mu_vk_4}{\mu_b(\mu_v + \lambda^*_v)} \left( 1 - \frac{1}{\mathcal{N}} \right) . \]

Replacing (28) in the expression of \( \lambda^*_v \) gives
\[ \lambda^*_v = \\
\left( \frac{\beta_{hv}(\eta_v E^*_v + I^*_v)}{(N^*_h + m)} \right) \times \\
\left( \frac{\beta_{hv}\mu_hk_2k_3(k_1 + \lambda^*_h)(\pi\lambda^*_h + \mu_h) - \delta\gamma_h\lambda_h\lambda^*_h(k_8 + \pi\lambda^*_h)}{k_2k_3k_7(k_1 + \lambda^*_h)(\pi\lambda^*_h + \mu_h) - \delta\gamma_h\lambda_h\lambda^*_h(k_8 + \pi\lambda^*_h)} \right) \]  
(43)

**Appendix D: Proof of Proposition 4.1**

We compute now the endemic equilibrium, i.e. we are looking for an equilibrium such that \( \lambda^*_h \neq 0 \)
where \( k_{11} = k_3 y_h + \gamma_h \).

Substituting (43) in (42) gives the following equation

\[
f(\lambda_n^*) := \lambda_n^* [B_4(\lambda_n^*)^4 + B_3(\lambda_n^*)^3
\quad + B_2(\lambda_n^*)^2 + B_1\lambda_n^* + B_0] = 0 \quad (44)
\]

where

\[
B_4 = \pi^2 [k_7 (\mu_h k_3 + \gamma_h (\mu_h + \sigma)) + \delta \gamma_h m \mu_h]
\times \{\mu_v [k_7 (\mu_h k_3 + \gamma_h (\mu_h + \sigma)) + \delta \gamma_h m \mu_h]
\quad + \beta v_h k_{11} \Lambda_h \mu_h \}
\]

\[
B_3 = 2 \pi X \{k_2 k_3 k_7 \mu_h (1 + \pi) + \delta \gamma_h \Lambda_h \mu_h
\quad + \pi \xi X \} \mu_v + \beta v_h \pi \Lambda_h \mu_h k_{11} \{\pi k_2 k_3 Y
\quad + k_7 [k_8 (k_2 k_3 - 2 \delta \gamma_h) + \mu_h k_2 k_3]\}
\]

\[
B_2 = \mu_v [k_1 k_3 k_7 k_7 - \delta \Lambda_h \gamma_h \pi \xi + X \mu_h]^2
\quad + 2 k_1 k_2 k_3 k_7 \pi \mu_v X
\quad + \beta v_h \Lambda_h \mu_h^2 \pi k_2 k_3 k_{11} \{\pi k_1 k_7
\quad - \beta v_h k_{10} [\pi (k_8 + k_4) + \mu_h] \}
\quad + \beta v_h k_{11} \Lambda_h \mu_h [k_2 k_3 k_7 \pi \mu_h + k_8 X]
\]

\[
B_1 = 2 k_1 k_2 k_3 k_7 \mu_h \mu_v [k_8 X + \pi \mu_h k_2 k_3 k_7]
\quad + k_2 k_3 k_{11} \beta v_h \Lambda_h \mu_h^2 \{k_1 k_7 k_8
\quad - \beta v_h k_{10} (\mu_h k_8 + k_1 \pi (k_8 + \mu_h))\}
\]

with \( X = k_2 k_3 k_7 - \delta \gamma_h \Lambda_h, Y = k_1 k_7 - \beta v_h \mu_v k_{10}; \)

and

\[
B_0 = \mu_v^2 \mu_v k_1^2 k_2^2 k_3^2 \ (1 - R_0^2)
\]

We consider \( \lambda_n^* \neq 0 \), otherwise we recover DFE. The positive endemic equilibria \( P^* = (S^*_h, V^*_h, E^*_h, I^*_h, R^*_h, A^*_v, S^*_v, E^*_v, I^*_v) \) are obtained by solving Eq. (44) for \( \lambda_n^* \). The coefficient \( B_4 \) is always positive and coefficient \( B_0 \) is negative (resp. positive) whenever \( R_0 > 1 \) (resp. \( R_0 < 1 \)). The number of possible nonnegative real roots of the polynomial of Eq. (44) depends on the signs of \( B_3, B_2 \) and \( B_1 \). The various possibilities for the roots of \( f(\lambda_n^*) \) are tabulated in Table XI and XII.

From tables XI and XII, we deduce the following result which gives various possibilities of nonnegative solutions of (44).

**Lemma A.1:** Assume that \( N > 1 \) and \( \mu_v = \mu_1 = \mu_2 \). Then, the arboviral-disease model (3) has a unique endemic equilibrium when Case 1 of Table XI is satisfied and whenever \( R_0 > 1 \).

1. could have more than one endemic equilibrium when Case 2 of Table XI is satisfied whenever \( R_0 > 1 \).

2. could have more than one endemic equilibrium when Case 2, 3 of Table XII are satisfied and whenever \( R_0 < 1 \).

3. has no endemic equilibrium when Case 1 of Table XII is satisfied and whenever \( R_0 < 1 \).

4. has no endemic equilibrium when Case 1 of Table XII is satisfied and whenever \( R_0 < 1 \).

Case 3 of Lemma A.1 suggests that coexistence of the disease–free equilibrium and the endemic equilibrium for the arboviral-disease model (3) is possible, and hence the potential occurrence of the backward bifurcation phenomenon when \( R_0 < 1 \). Also, case 2 of Lemma A.1 suggests the possibility of a pitchfork (Forward) bifurcation when \( R_0 = 1 \).
b) Presence of disease–induced death in vector: Here, we will consider $\mu_v < \mu_1 < \mu_2$ with $\mu_v \neq \mu_2$. Equation (27) becomes

$$
\lambda_h^* = \frac{\beta_{hv}\mu_h \mu_v k_2 k_3 k_{10} (k_1 + \lambda_h^*) (\mu_h + \pi \lambda_h^*)}{k_2 k_3 k_7 (k_1 + \lambda_h^*) (\mu_h + \pi \lambda_h^*) - \delta \gamma_h \Lambda_h \lambda_h^*(k_8 + \pi \lambda_h^*)} \times \left( \frac{\mu_2 \mu_2 \theta k_4 N_1 + \alpha \lambda_v^*}{\mu_2 \theta (\mu_2 k_4 + k_5 \lambda_v^*)} \right) \left( \frac{\lambda_v}{\mu_v + \lambda_v} \right)
$$

(45)

with $k_{12} = \frac{\mu_v k_10}{K}$, $N_1 = \left( 1 - \frac{1}{N} \right)$ and

$$
\lambda_v^* = \frac{\beta_{vh} \mu_h \Lambda_h k_11 \lambda_h^*(k_8 + \pi \lambda_h^*)}{k_2 k_3 k_7 (k_1 + \lambda_h^*) (\mu_h + \pi \lambda_h^*) - \delta \gamma_h \Lambda_h \lambda_h^*(k_8 + \pi \lambda_h^*)}
$$

(46)

Substituting (46) in (45) gives the following equation

$$
\lambda_h^* \sum_{i=0}^{6} C_i (\lambda_h^*)^i = 0
$$

(47)

where $C_0 = k_1 k_2 k_3 k_4 k_5 k_7 \beta_{hv} \mu_h \mu_v \mu_h \lambda_h^* \left( R_0^2 - 1 \right)$ and

$$
C_6 = -\mu_v \pi \theta X \left( \mu_2 k_4 X + \beta_{vh} k_5 k_11 \Lambda_h \mu_h \right) \times \left( \mu_4 X + \beta_{vh} k_11 \Lambda_h \mu_h \right),
$$

with $X = (k_2 k_3 k_7 - \delta \Lambda_h \gamma_h) > 0$. The others coefficients $C_5, C_4, C_3, C_2$, and $C_1$ are obtained after computations on Maxima software. We also obtain the following result which gives various possibilities of solutions of Eq. (47).

**Lemma A.2:** Assume that $N > 1$. Then, the arboviral-disease model (3)

1. could have a unique endemic equilibrium whenever $R_0 > 1$.
2. could have more than one endemic equilibrium whenever $R_0 > 1$.
3. haven’t endemic equilibrium whenever $R_0 < 1$.
4. could have one or more than one endemic equilibrium whenever $R_0 < 1$.

Case 4 of Lemma [A.2] suggests that co-existence of the disease-free equilibrium and endemic equilibrium for the arboviral-disease model (3) is possible, and hence the potential occurrence of a backward bifurcation phenomenon when $R_0 < 1$. Also, case 2 of Lemma [A.2] suggests the possibility of a pitchfork (Forward) bifurcation when $R_0 = 1$.

**Appendix E: Computation of $a^*$ of Theorem 4.1**

$$
a_3^* = \frac{1}{2} v_3 \sum_{i,j=1}^{9} w_i w_j \frac{\partial^2 f_3(0,0)}{\partial x_i \partial x_j} + \frac{1}{2} v_8 \sum_{i,j=1}^{9} w_i w_j \frac{\partial^2 f_8(0,0)}{\partial x_i \partial x_j}.
$$

(48)

Let $a_3^* = \sum_{i,j=1}^{9} w_i w_j \frac{\partial^2 f_3(0,0)}{\partial x_i \partial x_j}$ and $a_8^* = \sum_{i,j=1}^{9} w_i w_j \frac{\partial^2 f_8(0,0)}{\partial x_i \partial x_j}$. After few computations, we obtain

$$
a_3^* = \frac{\beta_{hv} \mu_h (\epsilon \pi \lambda_h + \mu_h)}{k_1(\Lambda_h + m \mu_h)^2} \left( w_1 (\eta_v w_8 + w_9) + \frac{\beta_{hv} \pi \mu_h (\epsilon \lambda_h + m \mu_h)}{k_1(\Lambda_h + m \mu_h)^2} \left( w_2 (\eta_v w_8 + w_9) - \beta_{hv} \mu_h \lambda_h (\mu_h + \pi \xi) \right) \right)
$$

$$
+ \frac{\beta_{hv} \mu_h \lambda_h (\mu_h + \pi \xi)}{k_1(\Lambda_h + m \mu_h)^2} \left( w_4 (\eta_v w_8 + w_9) - \beta_{hv} \mu_h \lambda_h (\mu_h + \pi \xi) \right)
$$

$$
+ \frac{\beta_{hv} \mu_h \lambda_h (\mu_h + \pi \xi)}{k_1(\Lambda_h + m \mu_h)^2} \left( w_5 (\eta_v w_8 + w_9) - \beta_{hv} \mu_h \lambda_h (\mu_h + \pi \xi) \right)
$$

$$
+ \frac{\beta_{hv} \mu_h \lambda_h (\mu_h + \pi \xi)}{k_1(\Lambda_h + m \mu_h)^2} \left( w_6 (\eta_v w_8 + w_9) - \beta_{hv} \mu_h \lambda_h (\mu_h + \pi \xi) \right)
$$

$$
+ \frac{\beta_{hv} \mu_h}{k_1(\Lambda_h + m \mu_h)^2} \left( (\epsilon \lambda_h + m \mu_h) (w_1 + \pi w_2) - \Lambda_h (\mu_h + \pi \xi) \right) \left( w_8 (w_3 + w_4 + w_5) \right)
$$

$$
+ \frac{\beta_{hv} \mu_h}{k_1(\Lambda_h + m \mu_h)^2} \left( (\epsilon \lambda_h + m \mu_h) (w_1 + \pi w_2) - \Lambda_h (\mu_h + \pi \xi) \right) \left( w_9 (w_3 + w_4 + w_5) \right)
$$

$$
= \frac{\beta_{hv} \mu_h}{k_1(\Lambda_h + m \mu_h)^2} \left( (\epsilon \lambda_h + m \mu_h) (w_1 + \pi w_2) - \Lambda_h (\mu_h + \pi \xi) \right) \left( w_3 + w_4 + w_5 \right)
$$

$$
- \Lambda_h (\mu_h + \pi \xi) \left( w_8 (w_3 + w_4 + w_5) \right) \left( \eta_v w_8 + w_9 \right)
$$

$$
+ \frac{\beta_{hv} \mu_h}{k_1(\Lambda_h + m \mu_h)^2} \left( (\epsilon \lambda_h + m \mu_h) (w_1 + \pi w_2) - \Lambda_h (\mu_h + \pi \xi) \right) \left( w_9 (w_3 + w_4 + w_5) \right)
$$

$$
= \frac{\beta_{hv} \mu_h}{k_1(\Lambda_h + m \mu_h)^2} \left( (\epsilon \lambda_h + m \mu_h) (w_1 + \pi w_2) - \Lambda_h (\mu_h + \pi \xi) \right) \left( w_3 + w_4 + w_5 + w_6 \right)
$$

$$
- \Lambda_h (\mu_h + \pi \xi) \left( w_8 (w_3 + w_4 + w_5) \right) \left( \eta_v w_8 + w_9 \right)
$$

$$
- 2 \Lambda_h (\mu_h + \pi \xi) \left( w_3 + w_4 + w_5 + w_6 \right) \left( \eta_v w_8 + w_9 \right) \right),
$$

Using above results, Eq. (48) becomes
\[ a^* = \phi_1 - \phi_2 \]

where
\[ \phi_1 = \frac{1}{2} \sum_{j=1}^{9} \frac{\beta_v h_t^2 K \theta}{\mu_v \Lambda_h + \mu_m m^2} \left( \eta_j w_3 + w_4 \right) \]
\[ \phi_2 = \frac{1}{2} \sum_{j=1}^{9} \frac{\beta_v h_t^2 K \theta}{\mu_v \Lambda_h + \mu_m m^2} \left( \eta_j w_3 + w_4 \right) \]

and
\[ \phi_1 = \frac{1}{2} \sum_{j=1}^{9} \frac{\beta_v h_t^2 K \theta}{\mu_v \Lambda_h + \mu_m m^2} \left( \eta_j w_3 + w_4 \right) \]
\[ \phi_2 = \frac{1}{2} \sum_{j=1}^{9} \frac{\beta_v h_t^2 K \theta}{\mu_v \Lambda_h + \mu_m m^2} \left( \eta_j w_3 + w_4 \right) \]

Using above results, Eq. (48) becomes
\[ a^* = \phi_1 - \phi_2 \]

the disease–free equilibrium (DFE := P_0) are the fixed point of (22). Indeed, rewriting (22) gives
\[ S_h^{k+1} = \frac{\phi(h)A_h + (1 - \phi(h)k_1)S_h^k}{1 + \phi(h)\lambda_h^k} \]
\[ V_h^{k+1} = \frac{\phi(h)S_h^k + (1 - \phi(h)\mu)\lambda_h^k}{1 + \phi(h)\pi V_h^{k+1}} \]
\[ B_h^{k+1} = \frac{(1 - \phi(h)k_2)B_h^k + \phi(h)\lambda_h^k}{(S_h^{k-1} + \pi V_h^{k+1})} \]
\[ I_h^{k+1} = \frac{\phi(h)\gamma_h E_h^k + (1 - \phi(h)k_3)I_h^k}{\phi(h)\gamma_h E_h^k + (1 - \phi(h)\mu)R_h^k} \]
\[ A_v^{k+1} = \frac{\left[ \frac{1}{\phi(h)} \frac{\left( k_6 + \mu_b S_v^k + E_v^k + I_v^k \right)}{K} \right] A_v^k}{\phi(h)\mu(S_v^k + E_v^k + I_v^k)} \]
\[ E_v^{k+1} = \frac{(1 - \phi(h)k_4)E_v^k + \phi(h)\lambda_v^k S_v^{k+1}}{\phi(h)\mu(S_v^k + E_v^k + I_v^k)} \]
\[ I_v^{k+1} = \frac{\phi(h)\gamma_v E_v^k + (1 - \phi(h)\mu_2)I_v^k}{\phi(h)\gamma_v E_v^k + (1 - \phi(h)\mu_2)I_v^k} \]

If \( X^* = (S_h^*, V_h^*, E_h^*, I_h^*, R_h^*, A_v^*, S_v^*, E_v^*, I_v^*)^T \) is an equilibrium of the discrete system (49), then we have after few simplifications
\[ \begin{cases}
\Lambda_h - \lambda_h^* S_h^* - k_1 S_h^* = 0 \\
\xi S_h^* - \pi \lambda_h^* V_h^* - \mu_h V_h^* = 0 \\
\lambda_h^* (S_h^* + \pi V_h^*) - k_2 E_h^* = 0 \\
\gamma_h E_h^* - k_3 I_h^* = 0 \\
\sigma I_h^* - \mu_h R_h^* = 0 \\
\mu_b (S_v^* + E_v^* + I_v^*) \left( 1 - \frac{A_v^k}{K} \right) - k_6 A_v^k = 0 \\
\theta A_v^* - \lambda_v^* S_v^* + \mu_v S_v^* = 0 \\
k_4 E_v^* - \gamma_v E_v^* - \mu_2 I_v^* = 0 \\
\end{cases} \]

which is equivalent to
\[ \begin{cases}
A_1 (X^*) (X_S^* - X_{DFE}) + A_{12} (X^*) X_T^* = 0 \\
A_2 (X^*) X_T^* = 0 \\
\end{cases} \]

where \( A_1, A_{12} \) and \( A_2 \) are given at Equation (11).

Appendix F: Proof of Lemma 7.2

The Kamgang-Sallet approach used for (22) ensures that the trivial equilibrium (TE := P_0) and

Appendix G: Proof of Lemma 7.3

The Jacobian matrix associated with the right-hand side of the numerical scheme (22) at the
tivial equilibrium $TE := P_0$ is given by $J_{TE} = (J_{ij})_{1 \leq i,j \leq 9}$ with

$$
\begin{align*}
J_{1,1} &= 1 - k_1 \phi(h); \\
J_{1,8} &= - \frac{\phi(h) \beta_{hv} \eta_h \Lambda_h \mu_h}{k_1(\Lambda_h + \mu_h m)}; \\
J_{1,9} &= \frac{\phi(h) \beta_{hv} \Lambda_h \mu_h}{k_1(\Lambda_h + \mu_h m)}; \\
J_{2,2} &= 1 - \mu_h \phi(h); \\
J_{2,8} &= - \frac{\phi(h) \pi \beta_{hv} \eta_h \xi \Lambda_h}{k_1(\Lambda_h + \mu_h m)}; \\
J_{2,9} &= \frac{\phi(h) \beta_{hv} \Lambda_h \mu_h}{k_1(\Lambda_h + \mu_h m)}; \\
J_{3,3} &= 1 - k_2 \phi(h); \\
J_{3,8} &= \frac{\phi(h) \beta_{hv} \eta_h \Lambda_h (\mu_h + \pi \xi)}{k_1(\Lambda_h + \mu_h m)}; \\
J_{3,9} &= \frac{\phi(h) \beta_{hv} \Lambda_h (\mu_h + \pi \xi)}{k_1(\Lambda_h + \mu_h m)}; \\
J_{4,4} &= 1 - k_2 \phi(h); \\
J_{4,5} &= 1 - \mu_h \phi(h); \\
J_{5,5} &= 1 - \mu_h \phi(h); \\
J_{6,6} &= 1 - \phi(h) k_6; \\
J_{6,7} &= J_{6,8} = J_{6,9} = \phi(h) \beta_{hv} m; \\
J_{7,6} &= \phi(h) \gamma \mu_v; \\
J_{8,8} &= 1 - \phi(h) k_3; \\
J_{9,9} &= 1 - 2 \phi(h)
\end{align*}
$$

The eigenvalues of $J_{TE}$ are given by $\lambda_1 = \lambda_2 = 1 - \mu_h \phi(h), \lambda_3 = 1 - k_1 \phi(h), \lambda_4 = 1 - k_2 \phi(h), \lambda_5 = 1 - k_3 \phi(h),$ and $\lambda_6, \lambda_7, \lambda_8, \lambda_9$ are eigenvalues of the submatrix

$$
\bar{J} = \begin{pmatrix}
J_{6,6} & \phi(h) \beta_{hv} \eta_h \mu_h & \phi(h) \beta_{hv} \eta_h \mu_h & \phi(h) \beta_{hv} \eta_h \mu_h \\
J_{7,6} & J_{7,7} & 0 & 0 \\
0 & 0 & J_{8,8} & 0 \\
0 & 0 & J_{9,8} & J_{9,9}
\end{pmatrix}
$$

Since $\phi(h) > 0$, it is clear that $|\lambda_i| < 1$, for $i = 1, 2, \ldots, 5$. We need also to show that the characteristic polynomial associated with $\bar{J}$ is Schur polynomials, i.e. polynomials such that all roots $\lambda_i$ verify $|\lambda_i| < 1$.

The characteristic polynomial associated with $\bar{J}$ is given by

$$
P(\lambda) = (\lambda + \mu_2 \phi(h) - 1) (\lambda + k_4 \phi(h) - 1) H(\lambda)
$$

where

$$
H(\lambda) = \lambda^2 + (\phi(h) (\mu_v + k_6) - 2) \lambda \\
+ 1 + \phi(h)^2 (k_6 \mu_v - \mu_h \theta) - \phi(h) (\mu_v + k_6)
$$

The roots of $P(\lambda)$ are $\lambda_6 = 1 - \mu_2 \phi(h), \lambda_7 = 1 - k_4 \phi(h)$ and the others roots are the roots of $H(\lambda)$. Note that $|\lambda_6| < 1$ and $|\lambda_7| < 1$. Now, we need to show that $H(\lambda)$ is a Schur polynomial. To this aim, let $q_1 = (\phi(h) (\mu_v + k_6) - 2)$ and $q_2 = 1 + \phi(h)^2 (k_6 \mu_v - \mu_h \theta) - \phi(h) (\mu_v + k_6)$. Using Lemma 11 in [29], we just show that the following conditions hold:

$$
1 + q_1 + q_2 > 0, \quad 1 - q_1 + q_2 > 0, \quad 1 - q_2 > 0
$$

(52)

We compute $1 + q_1 + q_2 = \phi(h)^2 k_6 \mu_v (1 - \lambda^2)$. Therefore, $1 + q_1 + q_2 > 0$ whenever $\lambda^2 < 1$.

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