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Backward Bifurcation and Control in Transmission Dynamics of Arboviral Diseases

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Abstract

In this paper, we derive and analyse a compartmental model for the control of arboviral diseases which takes into account an imperfect vaccine combined with individual protection and some vector control strategies already studied in the literature. After the formulation of the model, a qualitative study based on stability analysis 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 reproduction number, R_0 , is less than unity. Using Lyapunov function theory, we prove that the trivial equilibrium is globally asymptotically stable; When the disease-induced death is not considered, or/and, when the standard incidence is replaced by the mass action incidence, the backward bifurcation does not occur. Under a certain condition, we establish the global asymptotic stability of the disease-free equilibrium of the full model. Through sensitivity analysis, we determine the relative importance of model parameters for disease transmission. Numerical simulations show that the combination of several control mechanisms would significantly reduce the spread of the disease, if we maintain the level of each control high, and this, over a long period.

Keywords: Compartmental model, Arboviral diseases, Vaccination, Vector control strategies, Stability, Backward bifurcation, Sensitivity analysis.

AMS Subject Classification (2010): 34D20, 34D23, 37N25, 92D30.

1 Introduction

Arboviral diseases are affections transmitted by hematophagous arthropods. There are currently 534 viruses registered in the International Catalog of Arboviruses and 25% of them have caused documented illness in human populations [1, 2, 3]. Examples of those 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 [4, 5, 6]. The dynamics of arboviral diseases like Dengue or Chikungunya are influenced by many factors such as human and mosquito behaviour, the virus itself, as well as the environment which directly or indirectly affects all the present mechanisms of control.

For all mentioned diseases, only yellow fever has a licensed vaccine. Nevertheless, considerable efforts are made to obtain the vaccines for other diseases. In the case of Dengue for

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38 example, the scientists of French laboratory SANOFI have conducted different trials in Latin
39 America and Asia. Thus, a tetravalent vaccine could be quickly set up in the coming months.
40 But in any case, it is clear that this vaccine will be imperfect. However, the trials in Latin
41 America have shown that vaccine efficacy was 64.7%. Serotype-specific vaccine efficacy was
42 50.3% for serotype 1, 42.3% for serotype 2, 74.0% for serotype 3, and 77.7% for serotype 4 [7].
43 The trials in Asia have shown that efficacy was 30.2%, and differed by serotype [8].

44 Host-vector models for arboviral diseases transmission were proposed in [9, 10, 11, 12, 13,
45 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26] with the focus on the construction of the basic
46 reproductive ratio and related stability analysis of the disease free and endemic equilibria. Some
47 of these works in the literature focus on modeling the spread of arboviral diseases and its control
48 using some mechanism of control like imperfect vaccines [19, 20, 26] and other control tools like
49 individual protection and vector control strategies [9, 10, 15, 21, 23, 24].

50 In [15], Dumont and Chiroleu proposed a compartmental model to study the impact of vector
51 control methods used to contain or stop the epidemic of Chikungunya of 2006 in Réunion island.
52 Moulay et al. [23] study an optimal control based on protection and vector control strategies
53 to fight against Chikungunya. In [20], Rodrigues et al. simulate an hypothetical vaccine as
54 an extra protection to the human population against epidemics of Dengue, using the optimal
55 control. In these models [15, 20, 23],

- 56 (i) the population is constant,
- 57 (ii) the disease-induced death in humans is not considered,
- 58 (iii) the complete stage progression of development of vectors is not considered,
- 59 (iv) none of the above mentioned models takes into account the combination of the mechanisms
60 of control already studied in the literature, such as vaccination, individual protection and
61 vector control strategies (destruction of breeding site, eggs and larvae reduction).

62 The aim of this work is to propose and study a arboviral disease control model which
63 takes into account human immigration, disease-induced mortality in human communities, the
64 complete stage structured model for vectors and a combination of human vaccination, individual
65 protection and vector control strategies to fight against the spread of these kind of diseases.

66 We start with the formulation of the constant control model, which is an extension of the
67 previous model developed in [26]. We include the complete stage progression of development
68 of vectors, the waning vaccine, and four other continuous controls (individual protection, using
69 adulticides, the mechanical control, Eggs and larvae reduction). We compute the net repro-
70 ductive number \mathcal{N} , as well as the basic reproduction number, \mathcal{R}_0 , and investigate the existence
71 and stability of equilibria. We prove that the trivial equilibrium is globally asymptotically
72 stable whenever $\mathcal{N} < 1$. When $\mathcal{N} > 1$ and $\mathcal{R}_0 < 1$, we prove that the system exhibit the
73 backward bifurcation phenomenon. The implication of this occurrence is that the classical epi-
74 demiological requirement for effective eradication of the disease, $\mathcal{R}_0 < 1$, is no longer sufficient,
75 even though necessary. However considering two situations: the model without vaccination
76 and the model with mass incidence rates, we prove that the disease-induced death and the
77 standard incidence functions, respectively, are the main causes of the occurrence of backward
78 bifurcation. We found that the disease-free equilibrium is globally asymptotically stable un-
79 der certain condition. Through local and global sensitivity analysis, we determine the relative
80 importance parameters of the model on the disease transmission. By using the pulse control
81 technique in numerical simulations, we evaluate the impact of different controls combinations
82 on the decrease of the spread of these diseases.

83 The paper is organized as follows. In Section 2 we present the transmission model and
 84 in Section 3 we carry out some analysis by determining important thresholds such as the net
 85 reproductive number \mathcal{N} and the basic reproduction number \mathcal{R}_0 , and different equilibria of the
 86 model. We then demonstrate the stability of equilibria and carry out bifurcation analysis. In
 87 section 4, both local and global sensitivity analysis are used to assess the important parameters
 88 in the spread of the diseases. Section 5 is devoted to numerical simulations and discussion. A
 89 conclusion rounds up the paper.

90 2 The formulation of the model

91 The model we propose here is an extension of the previous model studied in [26], and is based
 92 on the modelling approach given in [15, 16, 17, 18, 19, 23, 24]. It is assumed that the human and
 93 vector populations are divided into compartments described by time-dependent state variables.
 94 The compartments in which the populations are divided are the following ones:

95 –For humans, we consider susceptible (denoted by S_h), vaccinated (V_h), exposed (E_h),
 96 infectious (I_h) and resistant or immune (R_h); So that, $N_h = S_h + V_h + E_h + I_h + R_h$. Following
 97 Garba et al. [19] and Rodrigues et al. [20], we assume that the immunity, obtained by the
 98 vaccination process, is temporary. So, the immunity has the waning rate ω . The recruitment
 99 in human population is at the constant rate Λ_h , and newly recruited individuals enter the
 100 susceptible compartment S_h . Are concerned by recruitment people that are totally naive from
 101 the disease. Each individual human compartment goes out from the dynamics at natural
 102 mortality rates μ_h . The human susceptible population is decreased following infection, which
 103 can be acquired via effective contact with an exposed or infectious vector at a rate $\lambda_h =$
 104 $\frac{a\beta_{hv}(\eta_v E_v + I_v)}{N_h}$ [19] where a is the biting rate per susceptible vector, β_{hv} is the transmission
 105 probability from an infected vector (E_v or I_v) to a susceptible human (S_h). The probability
 106 that a vector chooses a particular human or other source of blood to bite can be assumed as $\frac{1}{N_h}$.

107 Thus, a human receives in average $a\frac{N_v}{N_h}$ bites per unit of times. Then, the infection rate per
 108 susceptible human is given $a\beta_{hv}\frac{N_v}{N_h}\frac{(\eta_v E_v + I_v)}{N_v}$. In expression of λ_h , the modification parameter
 109 $0 < \eta_v < 1$ accounts for the assumed reduction in transmissibility of exposed mosquitoes relative
 110 to infectious mosquitoes [19] (see the references therein for the specific sources). Latent humans
 111 (E_h) become infectious (I_h) at rate γ_h . Infectious humans recover at a constant rate, σ or dies
 112 as consequence of infection, at a disease-induced death rate δ . Immune humans retain their
 113 immunity for life.

114 – Following [23], the stage structured model is used to describe the vector population dy-
 115 namics, which consists of three main stages: embryonic (E), larvae (L) and pupae (P). Even if
 116 eggs (E) and immature stages (L and P) are both aquatic, it is important to dissociate them
 117 because, for control point of view, drying the breeding sites does not kill eggs, but only larvae
 118 and pupae. Moreover, chemical interventions on the breeding sites has impact on the larvae
 119 population, but not on the eggs [23]. The number of laid eggs is assumed proportional to the
 120 number of females. The system of stage structured model of aquatic phase development of

121 vector is given by (see [23] for details)

$$\begin{cases} \dot{E} &= \mu_b \left(1 - \frac{E}{\Gamma_E}\right) (S_v + E_v + I_v) - (s + \mu_E)E \\ \dot{L} &= sE \left(1 - \frac{L}{\Gamma_L}\right) - (l + \mu_L)L \\ \dot{P} &= lL - (\theta + \mu_P)P \end{cases}$$

122 Unlike the authors of [23], we take into account the pupal stage in the development of the
 123 vector. This is justified by the fact that they do not feed during this transitional stage of
 124 development, as they transform from larvae to adults. So, the control mechanisms can not be
 125 applied to them.

126 A rate, θ , of pupae become female Adults. Each individual vector compartment goes out
 127 from the dynamics at natural mortality rates μ_v . The vector susceptible population is decreased
 128 following infection, which can be acquired via effective contact with an exposed or infectious
 129 human at a rate $\lambda_v = \frac{a\beta_{vh}(\eta_h E_h + I_h)}{N_h}$ [19] where β_{vh} is the probability of transmission of
 130 infection from an infectious human (E_h or I_h) to a susceptible vector (S_v). Latent vectors
 131 (E_v) become infectious (I_v) at rate γ_v . The modification parameter $0 \leq \eta_h < 1$ accounts for
 132 the relative infectiousness of exposed humans in relation to infectious humans. Here too, it is
 133 assumed that susceptible mosquitoes can acquire infection from exposed humans [12, 19, 26].
 134 The vector population does not have an immune class, since it is assumed that their infectious
 135 period ends with their death [17].

136 Then, we add new terms in the model to assess the different control tools studied:

137 (i) α_1 represents the efforts made to protect human from mosquitoes bites. It mainly consists
 138 to the use of mosquito nets or wearing appropriate clothing [24]. Thus we modify the
 139 infection term as follows:

$$\lambda_h^c = (1 - \alpha_1)\lambda_h, \quad \text{and} \quad \lambda_v^c = (1 - \alpha_1)\lambda_v, \quad \text{with} \quad 0 \leq \alpha_1 < 1; \quad (1)$$

140 (ii) η_1 and η_2 are eggs and larvae mortality rates induced by chemical intervention respectively,

141 (iii) c_m is the additional mortality rate due to the adulticide,

142 (iv) α_2 is the parameter associated with the efficacy of the mechanical control.

143 The above assumptions lead to the following non-linear system of ordinary differential equations

$$\begin{cases} \dot{S}_h &= \Lambda_h + \omega V_h - (\lambda_h^c + \xi + \mu_h) S_h \\ \dot{V}_h &= \xi S_h - [(1 - \epsilon)\lambda_h^c + \omega + \mu_h] V_h \\ \dot{E}_h &= \lambda_h^c [S_h + (1 - \epsilon)V_h] - (\mu_h + \gamma_h) E_h \\ \dot{I}_h &= \gamma_h E_h - (\mu_h + \delta + \sigma) I_h \\ \dot{R}_h &= \sigma I_h - \mu_h R_h \\ \dot{S}_v &= \theta P - \lambda_v^c S_v - (\mu_v + c_m) S_v \\ \dot{E}_v &= \lambda_v^c S_v - (\mu_v + \gamma_v + c_m) E_v \\ \dot{I}_v &= \gamma_v E_v - (\mu_v + c_m) I_v \\ \dot{E} &= \mu_b \left(1 - \frac{E}{\alpha_2 \Gamma_E}\right) (S_v + E_v + I_v) - (s + \mu_E + \eta_1) E \\ \dot{L} &= sE \left(1 - \frac{L}{\alpha_2 \Gamma_L}\right) - (l + \mu_L + \eta_2) L \\ \dot{P} &= lL - (\theta + \mu_P) P \end{cases} \quad (2)$$

144 It is important to note that no intervention measure is performed to kill the pupae for two
 145 reasons: the first reason is the fact that at this stage, no food is absorbed by the insect, so it
 146 is impossible to make her ingest a toxic substance; the second reason is the fact that products
 147 soluble in water deposits by contact are not selective mosquito nymphs and act on all the
 148 wildlife of the cottage.

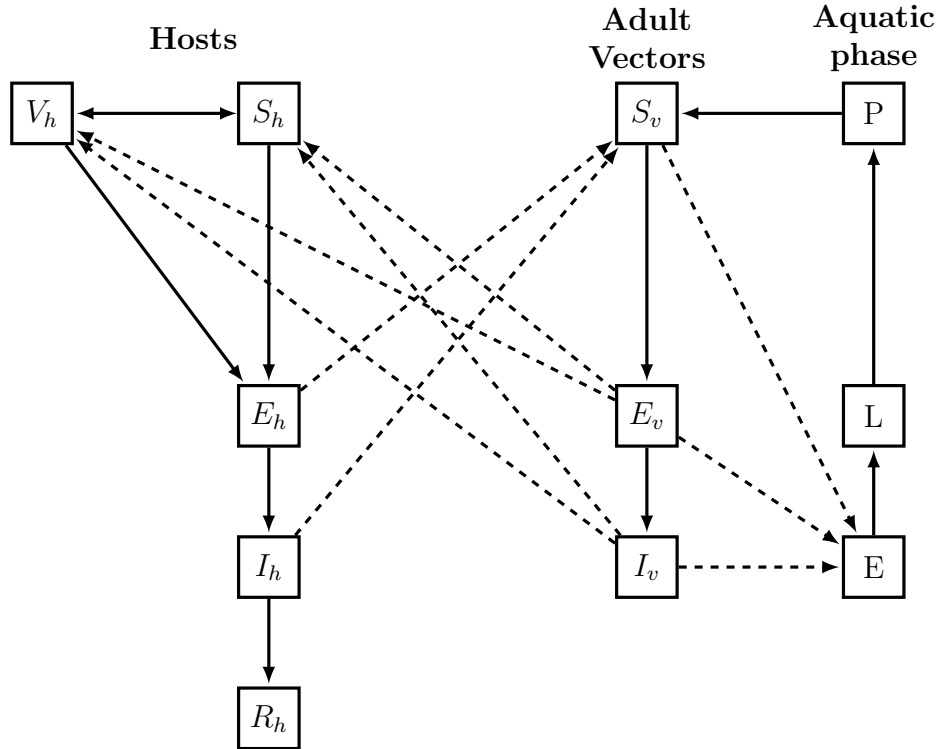


Figure 1: A compartmental model for the transmission dynamics of vector-borne diseases, which takes into account a waning vaccine and the complete phase of aquatic development of vector.

149 The description of state variables and parameters of model (2) are given in Tables 1–3.

Table 1: The state variables of model (2).

Humans		Vectors	
S_h :	Susceptible	E :	Eggs
V_h :	Vaccines	L :	Larvae
E_h :	Infected in latent stage	P :	Pupae
I_h :	Infectious	S_v :	Susceptible
R_h :	Resistant (immune)	E_v :	Infected in latent stage
		I_v :	Infectious

Table 2: Description and baseline values/range of parameters of model (2).

Par.	Description	Baseline value/range	Sources
Λ_h	Recruitment rate of humans	2.5 day ⁻¹	[19]
μ_h	Natural mortality rate in humans	$\frac{day^{-1}}{(67 \times 365)}$	[19]
ξ	Vaccine coverage	Variable	
ω	Vaccine waning rate	Variable	
ϵ	The vaccine efficacy	Variable	
a	Average number of bites	1 day ⁻¹	[9, 19]
β_{hv}	Probability of transmission of infection from an infectious human to a susceptible vector	0.1, 0.75 day ⁻¹	[9, 19]
γ_h	Progression rate from E_h to I_h	$[\frac{1}{15}, \frac{1}{3}]$ day ⁻¹	[15, 27]
δ	Disease-induced death rate	10 ⁻³ day ⁻¹	[19]
σ	Recovery rate for humans	0.1428 day ⁻¹	[9, 19]
η_h, η_v	Modifications parameter	[0, 1)	[19]
μ_v	Natural mortality rate of vectors	$[\frac{1}{30}, \frac{1}{14}]$ day ⁻¹	[9, 19]
γ_v	Progression rate from E_v to I_v	$[\frac{1}{21}, \frac{1}{2}]$ day ⁻¹	[15, 27]
β_{vh}	Probability of transmission of infection from an infectious vector to a susceptible human	0.1, 0.75 day ⁻¹	[9, 19]
θ	Maturation rate from pupae to adult	0.08 day ⁻¹	[15, 23, 24]
μ_b	Number of eggs at each deposit	6 day ⁻¹	[15, 23, 24]
Γ_E	Carrying capacity for eggs	10 ³ , 10 ⁶	[9, 23]
Γ_L	Carrying capacity for larvae	5 × 10 ² , 5 × 10 ⁵	[9, 23]
μ_E	Eggs death rate	0.2 or 0.4	[24]
μ_L	Larvae death rate	0.2 or 0.4	[24]
μ_P	Pupae death rate	0.4	Assumed

Table 3: Description and baseline values/range of parameters of model (2).

Par.	Description	Baseline value/range	Sources
s	Transfer rate from eggs to larvae	0.7 day ⁻¹	[24]
l	Transfer rate from larvae to pupae	0.5 day ⁻¹	[23]
η_1, η_2	Eggs and larvae mortality rates induced by chemical intervention	0.001, 0.3	[24]
α_1	Human protection rate	[0, 1)	
α_2	Efficacy of the mechanical control	(0, 1]	[15]
c_m	Adulticide killing rate	[0, 0.8]	[15]

2.1 Well posedness of the model

We now show that the system (2) is mathematically well defined and biologically feasible. We write

$$\begin{aligned} k_1 &:= \xi + \mu_h; & k_2 &:= \omega + \mu_h; & k_3 &:= \mu_h + \gamma_h; & k_4 &:= \mu_h + \delta + \sigma; \\ k_5 &:= s + \mu_E + \eta_1; & k_6 &:= l + \mu_L + \eta_2; & k_7 &:= \theta + \mu_P; & k_8 &:= \mu_v + c_m; \\ k_9 &:= \mu_v + \gamma_v + c_m; & K_E &:= \alpha_2 \Gamma_E; & K_L &:= \alpha_2 \Gamma_L; & \pi &:= 1 - \epsilon, \tau = a(1 - \alpha_1). \end{aligned} \quad (3)$$

System (2) can be rewritten in the following way

$$\frac{dX}{dt} = \mathbb{A}(X)X + F \quad (4)$$

with $X = (S_h, V_h, E_h, I_h, R_h, S_v, E_v, I_v, E, L, P)^T$, $\mathbb{A}(X) = \begin{pmatrix} A_1(X) & 0 \\ 0 & A_4(X) \end{pmatrix}$

with

$$A_1(X) = \begin{pmatrix} -\lambda_h^c - k_1 & \omega & 0 & 0 & 0 \\ \xi & -\pi \lambda_h^c - k_2 & 0 & 0 & 0 \\ \lambda_h & \pi \lambda_h & -k_3 & 0 & 0 \\ 0 & 0 & \gamma_h & -k_4 & 0 \\ 0 & 0 & 0 & \sigma & -\mu_h \end{pmatrix}$$

and

$$A_2(X) = \begin{pmatrix} -(\lambda_v^c + k_8) & 0 & 0 & 0 & 0 & \theta \\ \lambda_v & -k_9 & 0 & 0 & 0 & 0 \\ 0 & \gamma_v & -k_8 & 0 & 0 & 0 \\ A_{96} & A_{96} & A_{96} & -A_{97} & 0 & 0 \\ 0 & 0 & 0 & A_{109} & -A_{10} & 0 \\ 0 & 0 & 0 & 0 & l & -k_7 \end{pmatrix}$$

where $A_{96} = \mu_b \left(1 - \frac{E}{K_E}\right)$, $A_{97} = \left(\frac{\mu_b N_v}{K_E} + k_5\right)$, $A_{109} = s \left(1 - \frac{L}{K_L}\right)$ and $A_{10} = \frac{sE}{K_L} + k_6$;

and $F = (\Lambda_h, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0)^T$.

Note that $\mathbb{A}(X)$ is a Metzler matrix, i.e. a matrix such that off diagonal terms are non negative [28, 29], for all $X \in \mathbb{R}_+^{11}$. Thus, using the fact that $F \geq 0$, system (4) is positively invariant in \mathbb{R}_+^{11} , which means that any trajectory of the system starting from an initial state in the positive orthant \mathbb{R}_+^{11} , remains forever in \mathbb{R}_+^{11} . The right-hand side is Lipschitz continuous: there exists a unique maximal solution.

By adding the first four equations of model system (2), it follows that

$$\dot{N}_h(t) = \Lambda_h - \mu_h N_h - \delta I_h \leq \Lambda_h - \mu_h N_h$$

So that

$$0 \leq N_h(t) \leq \frac{\Lambda_h}{\mu_h} + \left(N_h(0) - \frac{\Lambda_h}{\mu_h}\right) e^{-\mu_h t}$$

Thus, at $t \rightarrow \infty$, $0 \leq N_h(t) \leq \frac{\Lambda_h}{\mu_h}$.

By adding the equations in S_v , E_v and I_v of system (2), it follows that

$$\dot{N}_v(t) = \theta P - \mu_v N_v$$

So that

$$0 \leq N_v(t) = \frac{\theta P}{\mu_v} + \left(N_v(0) - \frac{\theta P}{\mu_v}\right) e^{-\mu_v t}$$

Thus, at $t \rightarrow \infty$, $0 \leq N_v(t) \leq \frac{\theta l K_L}{\mu_v k_7}$ since $P \leq \frac{l K_L}{k_7}$.

Therefore, all feasible solutions of model system (2) enter the region:

$$\mathcal{D} = \left\{ (S_h, V_h, E_h, I_h, R_h, S_v, E_v, I_v, E, L, P) \in \mathbb{R}_+^{11} : N_h \leq \frac{\Lambda_h}{\mu_h}; E \leq K_E; L \leq K_L; P \leq \frac{l K_L}{k_7}; N_v \leq \frac{\theta l K_L}{k_7 k_8} \right\}$$

3 Mathematical analysis

In this section, we focus on the qualitative analysis of our model. We determine the different equilibrium points which are governed by two epidemiological thresholds, the net reproductive number denoted by \mathcal{N} and the basic reproduction number denoted by \mathcal{R}_0 . Then, dependent of the values of these thresholds, we study the stability (local as well as global) of the different equilibrium points, and also we test the occurrence of the backward bifurcation phenomenon. These stability analysis are also done for the different corresponding models, to determine the causes of the occurrence of backward bifurcation phenomenon.

3.1 The disease-free equilibria and its stability

In the absence of disease in the both population (human and Adult vector), i.e. $\lambda_h^c = \lambda_v^c = 0$ (or $E_h = I_h = E_v = I_v = 0$), we obtain two equilibria without disease: the trivial equilibrium (equilibrium without vector and disease) $\mathcal{E}_0 = (S_h^0, V_h^0, 0, 0, 0, 0, 0, 0, 0, 0, 0)$ and the disease-free equilibrium (equilibrium with vector and without disease) $\mathcal{E}_1 = (S_h^0, V_h^0, 0, 0, 0, N_v^0, 0, 0, E, L, P)$ with

$$\begin{aligned} S_h^0 &= \frac{\Lambda_h k_2}{\mu_h (k_2 + \xi)}, & V_h^0 &= \frac{\xi \Lambda_h}{\mu_h (k_2 + \xi)}, & N_v^0 &= \frac{K_E K_L k_5 k_6 (\mathcal{N} - 1)}{\mu_b (K_E S + k_6 K_L)}, \\ P &= \frac{K_E K_L k_5 k_6 k_8 (\mathcal{N} - 1)}{\mu_b \theta (K_E S + k_6 K_L)}, & L &= \frac{K_E K_L k_5 k_6 k_7 k_8 (\mathcal{N} - 1)}{\mu_b \theta l (K_E S + k_6 K_L)}, \\ E &= \frac{K_E K_L k_5 k_6 k_7 k_8 (\mathcal{N} - 1)}{s (\mu_b l K_L \theta + k_5 k_7 k_8 K_E)}. \end{aligned} \quad (5)$$

where \mathcal{N} is the net reproductive number [23, 30, 31] given by

$$\mathcal{N} = \frac{\mu_b \theta l s}{k_5 k_6 k_7 k_8} \quad (6)$$

3.1.1 Local stability of disease-free equilibria

The local asymptotic stability result of equilibria \mathcal{E}_0 and \mathcal{E}_1 is given in the following.

Theorem 1. Define the basic reproductive number [32, 33]

$$R_0 = \sqrt{\frac{a^2 (1 - \alpha_1)^2 \beta_{hv} \beta_{vh} \mu_h k_5 k_6 (\gamma_h + k_4 \eta_h) (\gamma_v + k_8 \eta_v) (\pi \xi + k_2) \alpha_2 \Gamma_E \Gamma_L (\mathcal{N} - 1)}{k_3 k_4 k_8 k_9 \mu_b \Lambda_h (\xi + k_2) (k_6 \Gamma_L + s \Gamma_E)}} \quad (7)$$

Then,

- (i) if $\mathcal{N} \leq 1$, the trivial equilibrium \mathcal{E}_0 is locally asymptotically stable in \mathcal{D} ;
- (ii) if $\mathcal{N} > 1$, the trivial equilibrium is unstable and the disease-free equilibrium \mathcal{E}_1 is locally asymptotically stable in \mathcal{D} whenever $\mathcal{R}_0 < 1$.

188 *Proof.* See [B](#). □

189 The basic reproduction number of a disease is the average number of secondary cases that
 190 one infectious individual produces during his infectious period in a totally susceptible pop-
 191 ulation. The epidemiological implication of [Theorem 1](#) is that, in general, when the basic
 192 reproduction number, \mathcal{R}_0 is less than unity, a small influx of infectious vectors into the com-
 193 munity would not generate large outbreaks, and the disease dies out in time (since the DFE is
 194 LAS) [[19](#), [32](#), [33](#), [34](#)]. However, we show in the subsection [3.2](#) that the disease may still persist
 195 even when $\mathcal{R}_0 < 1$.

196 3.1.2 Global stability of the trivial equilibrium

197 The global stability of the trivial equilibrium is given by the following result:

198 **Theorem 2.** *If $\mathcal{N} \leq 1$, then \mathcal{E}_0 is globally asymptotically stable on \mathcal{D} .*

199 *Proof.* To prove the global asymptotic stability of the trivial disease-free equilibrium \mathcal{E}_0 , we
 200 use the direct Lyapunov method. To this aim, we set $Y = X - TE$ with
 201 $X = (S_h, V_h, E_h, I_h, R_h, S_v, E_v, I_v, E, L, P)^T$ and rewrite [\(2\)](#) in the following manner

$$\frac{dY}{dt} = \mathcal{B}(Y)Y.$$

The global asymptotic stability of \mathcal{E}_0 is achieved by considering the following Lyapunov function $\mathcal{L}(Y) = \langle g, Y \rangle$ where

$$g = \left(1, 1, 1, 1, 1, 1, 1, 1, \frac{k_8}{\mu_b}, \frac{k_5 k_8}{\mu_b s}, \frac{k_5 k_6 k_8}{\mu_b s l} \right).$$

202 See [C](#) for the details. □

203 3.1.3 Global stability of the disease-free equilibrium

204 We now turn to the global stability of the disease-free equilibrium \mathcal{E}_1 . we prove that the disease-
 205 free equilibrium \mathcal{E}_1 is globally asymptotically stable under a certain threshold condition. To
 206 this aim, we use a result obtained by Kamgang and Sallet [[35](#)], which is an extension of some
 207 results given in [[33](#)]. Using the property of DFE, it is possible to rewrite [\(2\)](#) in the following
 208 manner

$$\begin{cases} \dot{X}_S = \mathcal{A}_1(X)(X_S - X_{DFE}) + \mathcal{A}_{12}(X)X_I \\ \dot{X}_I = \mathcal{A}_2(X)X_I \end{cases} \quad (8)$$

where X_S is the vector representing the state of different compartments of non transmitting in-
 dividuals $(S_h, V_h, R_h, S_v, E, L, P)$ and the vector X_I represents the state of compartments of dif-
 ferent transmitting individuals (E_h, I_h, E_v, I_v) . Here, we have $X_S = (S_h, V_h, R_h, S_v, E, L, P)^T$,
 $X_I = (E_h, I_h, E_v, I_v)^T$, $X = (X_S, X_I)$ and $X_{DFE} := \mathcal{E}_1 = (S_h^0, V_h^0, 0, 0, 0, N_v^0, 0, 0, E, L, P)^T$,

$$\mathcal{A}_1(X) = \begin{pmatrix} \mathcal{A}_1^{(1)} & \mathcal{A}_1^{(2)} \\ \mathcal{A}_1^{(3)} & \mathcal{A}_1^{(4)} \end{pmatrix},$$

$$\begin{aligned}
\text{with } \mathcal{A}_1^{(1)}(X) &= \begin{pmatrix} -(\lambda_h^c + k_1) & \omega & 0 & 0 \\ \xi & -(\pi\lambda_h^c + k_2) & 0 & 0 \\ 0 & 0 & -\mu_h & 0 \\ 0 & 0 & 0 & -(\lambda_v^c + k_8) \end{pmatrix}, \\
\mathcal{A}_1^{(2)}(X) &= \begin{pmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & \theta \end{pmatrix}, \quad \mathcal{A}_1^{(3)}(X) = \begin{pmatrix} 0 & 0 & 0 & \mu_b \left(1 - \frac{E}{K_E}\right) \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \\
\mathcal{A}_1^{(4)}(X) &= \begin{pmatrix} -\left(k_5 + \mu_b \frac{S_v^0}{K_E}\right) & 0 & 0 \\ s \left(1 - \frac{L}{K_L}\right) & -\left(k_6 + \frac{sE^*}{K_L}\right) & 0 \\ 0 & l & -k_7 \end{pmatrix}, \\
\mathcal{A}_{12}(X) &= \begin{pmatrix} 0 & 0 & -\frac{\tau\beta_{hv}\eta_v S_h^0}{N_h} & -\frac{\tau\beta_{hv} S_h^0}{N_h} & 0 & 0 & 0 \\ 0 & 0 & -\frac{\tau\beta_{hv}\eta_v \pi V_h^0}{N_h} & -\frac{\tau\beta_{hv} \pi V_h^0}{N_h} & 0 & 0 & 0 \\ 0 & \sigma & 0 & 0 & 0 & 0 & 0 \\ -\frac{\tau\beta_{vh}\eta_h S_v^0}{N_h} & -\frac{\tau\beta_{vh} S_v^0}{N_h} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \mu_b \left(1 - \frac{E}{K_E}\right) & \mu_b \left(1 - \frac{E}{K_E}\right) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \\
\mathcal{A}_2(X) &= \begin{pmatrix} -k_3 & 0 & \frac{\tau\beta_{hv}\eta_v(S_h + \pi V_h)}{N_h} & \frac{\tau\beta_{hv}(S_h + \pi V_h)}{N_h} \\ \frac{\gamma_h}{N_h} & -k_4 & 0 & 0 \\ \frac{\tau\beta_{vh}\eta_h S_v}{N_h} & \frac{\tau\beta_{vh} S_v}{N_h} & -k_9 & 0 \\ 0 & 0 & \gamma_v & -k_8 \end{pmatrix}.
\end{aligned}$$

209 A direct computation shows that the eigenvalues of $\mathcal{A}_1(X)$ have negative real parts. Thus the
210 system $\dot{X}_S = \mathcal{A}_1(X)(X_S - X_{DFE})$ is globally asymptotically stable at X_{DFE} . Note also that
211 $\mathcal{A}_2(X)$ is a Metzler matrix.

212 We now consider the bounded set \mathcal{G} :

$$\begin{aligned}
\mathcal{G} &= \left\{ (S_h, V_h, E_h, I_h, R_h, S_v, E_v, I_v, E, L, P) \in \mathbb{R}^{11} : S_h \leq N_h, V_h \leq N_h, E_h \leq N_h, \right. \\
&\quad I_h \leq N_h, R_h \leq N_h, \bar{N}_h = \Lambda_h / (\mu_h + \delta) \leq N_h \leq N_h^0 = \Lambda_h / \mu_h; \\
&\quad \left. E \leq K_E; L \leq K_L; P \leq \frac{lK_L}{k_7}; N_v \leq \frac{\theta l K_L}{k_7 k_8} \right\}
\end{aligned}$$

213 Let us recall the following theorem [35] (See [35] for a proof in a more general setting).

214 **Theorem 3.** *Let $\mathcal{G} \subset \mathcal{U} = \mathbb{R}^7 \times \mathbb{R}^4$. The system (2) is of class C^1 , defined on \mathcal{U} . If*

215 (1) \mathcal{G} is positively invariant relative to (8).

216 (2) The system $\dot{X}_S = \mathcal{A}_1(X)(X_S - X_{DFE})$ is Globally asymptotically stable at X_{DFE} .

217 (3) For any $x \in \mathcal{G}$, the matrix $\mathcal{A}_2(x)$ is Metzler irreducible.

218 (4) There exists a matrix $\bar{\mathcal{A}}_2$, which is an upper bound of the set

219 $\mathcal{M} = \{\mathcal{A}_2(x) \in \mathcal{M}_4(\mathbb{R}) : x \in \mathcal{G}\}$ with the property that if $\mathcal{A}_2 \in \mathcal{M}$, for any $\bar{x} \in \mathcal{G}$, such
220 that $\mathcal{A}_2(\bar{x}) = \bar{\mathcal{A}}_2$, then $\bar{x} \in \mathbb{R}^7 \times \{0\}$.

221 (5) The stability modulus of $\bar{\mathcal{A}}_2$, $\alpha(\mathcal{A}_2) = \max_{\lambda \in \text{sp}(\mathcal{A}_2)} \text{Re}(\lambda)$ satisfied $\alpha(\mathcal{A}_2) \leq 0$.

222 Then the DFE is GAS in \mathcal{G} .

For our model system (2), conditions (1–3) of the theorem 3 are satisfied. An upper bound of the set of matrices \mathcal{M} , which is the matrix $\bar{\mathcal{A}}_2$ is given by

$$\bar{\mathcal{A}}_2 = \begin{pmatrix} -k_3 & 0 & \frac{\tau\beta_{hv}\eta_v(S_h^0 + \pi V_h^0)}{\bar{N}_h} & \frac{\tau\beta_{hv}(S_h^0 + \pi V_h^0)}{\bar{N}_h} \\ \gamma_h & -k_4 & 0 & 0 \\ \frac{\tau\beta_{vh}\eta_h S_v^0}{\bar{N}_h} & \frac{\tau\beta_{vh} S_v^0}{\bar{N}_h} & -k_9 & 0 \\ 0 & 0 & \gamma_v & -k_8 \end{pmatrix},$$

223 where $\bar{N}_h = \frac{\Lambda_h}{(\mu_h + \delta)}$.

224 To check condition (5) in theorem 3, we will use the useful lemma [35] in A. To this aim,
225 let

$$226 \quad A = \begin{pmatrix} -k_3 & 0 \\ \gamma_h & -k_4 \end{pmatrix}, \quad B = \begin{pmatrix} \frac{\tau\beta_{hv}\eta_v(S_h^0 + \pi V_h^0)}{\bar{N}_h} & \frac{\tau\beta_{hv}(S_h^0 + \pi V_h^0)}{\bar{N}_h} \\ 0 & 0 \end{pmatrix},$$

$$227 \quad C = \begin{pmatrix} \frac{\tau\beta_{vh}\eta_h S_v^0}{\bar{N}_h} & \frac{\tau\beta_{vh} S_v^0}{\bar{N}_h} \\ 0 & 0 \end{pmatrix}, \quad D = \begin{pmatrix} -k_9 & 0 \\ \gamma_v & -k_8 \end{pmatrix}.$$

228 Clearly, A is a stable Metzler matrix. Then, after some computations, we obtain $D - CA^{-1}B$
229 is a stable Metzler matrix if and only if

$$\mathcal{R}_c < 1 \tag{9}$$

230 where

$$\mathcal{R}_c = \sqrt{\frac{a^2(1 - \alpha_1)^2\beta_{hv}\beta_{vh}k_5k_6(\gamma_h + k_4\eta_h)(\gamma_v + k_8\eta_v)K_E K_L(k_2 + \pi\xi)(\mathcal{N} - 1)(\mu_h + \delta)^2}{k_3k_4k_8k_9\mu_b(k_2 + \xi)(k_6K_L + K_{ES})\Lambda_h \mu_h}}. \tag{10}$$

231

232 We claim the following result

233 **Theorem 4.** *If $\mathcal{N} > 1$ and $\mathcal{R}_0 < \mathcal{R}_c < 1$, then the disease-free equilibrium \mathcal{E}_1 is globally*
234 *asymptotically stable in \mathcal{G} .*

235 **Remark 1.** *From (10), we have $\mathcal{R}_c = \frac{(\mu_h + \delta)}{\mu_h} \mathcal{R}_0 > \mathcal{R}_0$, showing that \mathcal{R}_c is not necessarily*
236 *an optimal threshold parameter.*

237 **Remark 2.** *Note that in the absence of disease-induced death, i.e. $\delta = 0$, we have $\mathcal{R}_c = \mathcal{R}_0$.*
238 *This suggests that the disease-induced death may be a cause of the occurrence of the backward*
239 *bifurcation phenomenon.*

240 **Remark 3.** *The above results show that if, at any time, through appropriate interventions (eg*
241 *the destruction of breeding sites, massive spraying, personal protection, vaccination,...), we are*
242 *able to reduce \mathcal{N} or \mathcal{R}_0 and \mathcal{R}_c less than 1 for a sufficiently long period, then, the disease may*
243 *disappear [15].*

244 Theorem 4 means that for $\mathcal{R}_0 < \mathcal{R}_c < 1$, the DFE is the unique equilibrium (no co-existence
 245 with an endemic equilibrium). If $\mathcal{R}_c \leq R_0 \leq 1$, then it is possible to have co-existence with
 246 endemic equilibria and thus, the occurrence of backward bifurcation phenomenon.

247 The backward bifurcation phenomenon, in epidemiological systems, indicate the possibility
 248 of existence of at least one endemic equilibrium when \mathcal{R}_0 is less than unity. Thus, the classical
 249 requirement of $\mathcal{R}_0 < 1$ is, although necessary, no longer sufficient for disease elimination [19,
 250 36, 37, 38]. In some epidemiological models, it has been shown that the backward bifurcation
 251 phenomenon is caused by factors such as non-linear incidence (the infection force), disease-
 252 induced death and vaccine (perfect or not) [19, 38, 39, 40, 41, 42]. To confirm whether or not
 253 the backward bifurcation phenomenon occurs in this case, one could use the approach developed
 254 in [33, 41, 43], which is based on the general centre manifold theorem [44]. We will explore this
 255 method in the next section.

256 3.2 Endemic equilibria and bifurcation analysis

257 3.2.1 Existence of endemic equilibria

258 We turn now to the existence of endemic equilibria. Let us introduce the following quantity
 259 $R_1 = R_0^2|_{\delta=0}$. We prove the following result

260 **Theorem 5.** *We assume that $\mathcal{N} > 1$, then*

261 (i) *In the absence of disease-induced death in human population ($\delta = 0$), model system (2)*
 262 *have*

263 1. *an unique endemic equilibrium whenever $R_1 > 1$.*

264 2. *no endemic equilibrium otherwise.*

265 (ii) *In presence of disease-induced death in human population ($\delta > 0$), model system (2) could*
 266 *have*

267 3. *at least one endemic equilibrium whenever $\mathcal{R}_0 > 1$.*

268 4. *zero, one or more than one endemic equilibrium whenever $\mathcal{R}_0 < 1$.*

269 *Proof.* See D. □

270 Note that case 4 of Theorem 5 indicate the possibility of existence of at least one endemic
 271 equilibrium for $\mathcal{R}_0 < 1$ and hence the potential occurrence of a backward bifurcation phe-
 272 nomenon.

273 3.2.2 Backward bifurcation analysis

274 In the following, we use the centre manifold theory [21, 33, 41, 43] to explore the possibility of
 275 backward bifurcation in (2). To do so, a bifurcation parameter β_{hv}^* is chosen, by solving for β_{hv}
 276 from $\mathcal{R}_0 = 1$, giving

$$\beta_{hv}^* = \frac{k_3 k_4 k_8 k_9 \mu_b \Lambda_h (\xi + k_2) (k_6 K_L + s K_E)}{a^2 (1 - \alpha_1)^2 \beta_{vh} \mu_h k_5 k_6 (\gamma_h + k_4 \eta_h) (\gamma_v + k_8 \eta_v) (\pi \xi + k_2) K_E K_L (\mathcal{N} - 1)}. \quad (11)$$

277 Let $J_{\beta_{hv}^*}$ denotes the Jacobian of the system (2) evaluated at the DFE (\mathcal{E}_1) and with $\beta_{hv} = \beta_{hv}^*$.
 278 Thus,

$$J_{\beta_{hv}^*} = \begin{pmatrix} J_1 & J_2 \\ J_3 & J_4 \end{pmatrix}, \quad (12)$$

279 where

$$280 \quad J_1 = \begin{pmatrix} -k_1 & \omega & 0 & 0 & 0 \\ \xi & -k_2 & 0 & 0 & 0 \\ 0 & 0 & -k_3 & 0 & 0 \\ 0 & 0 & \gamma_h & -k_4 & 0 \\ 0 & 0 & 0 & \sigma & -\mu_h \end{pmatrix}, \quad J_4 = \begin{pmatrix} -k_8 & 0 & 0 & 0 & 0 & \theta \\ 0 & -k_9 & 0 & 0 & 0 & 0 \\ 0 & \gamma_v & -k_8 & 0 & 0 & 0 \\ K_1 & K_1 & K_1 & -K_2 & 0 & 0 \\ 0 & 0 & 0 & K_3 & -K_4 & 0 \\ 0 & 0 & 0 & 0 & l & -k_7 \end{pmatrix}.$$

$$281 \quad J_2 = \begin{pmatrix} 0 & -\frac{\tau\beta_{hv}^*\eta_v S_h^0}{N_h^0} & -\frac{\tau\beta_{hv}^* S_h^0}{N_h^0} & 0 & 0 & 0 \\ 0 & -\frac{\tau\beta_{hv}^*\pi\eta_v V_h^0}{N_h^0} & -\frac{\tau\beta_{hv}^*\pi V_h^0}{N_h^0} & 0 & 0 & 0 \\ 0 & \frac{\tau\beta_{hv}^*\eta_v H^0}{N_h^0} & \frac{\tau\beta_{hv}^* H^0}{N_h^0} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \quad J_3 = \begin{pmatrix} 0 & 0 & -\frac{\tau\beta_{vh}\eta_h S_v^0}{N_h^0} & -\frac{\tau\beta_{vh} S_v^0}{N_h^0} & 0 \\ 0 & 0 & \frac{\tau\beta_{vh}\eta_h S_v^0}{N_h^0} & \frac{\tau\beta_{vh} S_v^0}{N_h^0} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

282 with $H^0 = S_h^0 + \pi V_h^0$, $K_1 = \mu_b \left(1 - \frac{E^*}{K_E}\right)$, $K_2 = k_5 + \frac{\mu_b}{K_E} S_v^0$, $K_3 = s \left(1 - \frac{L^*}{K_L}\right)$, and
 283 $K_4 = \left(k_6 + \frac{sE^*}{K_L}\right)$.

284 Note that the system (2), with $\beta_{hv} = \beta_{hv}^*$, has a hyperbolic equilibrium point (i.e., the
 285 linearised system (2) has a simple eigenvalue with zero real part and all other eigenvalues have
 286 negative real part). Hence, the centre manifold theory [44, 45] can be used to analyse the
 287 dynamics of the model (2) near $\beta_{hv} = \beta_{hv}^*$. The technique in Castillo-Chavez and Song (2004)
 288 [43] entails finding the left and right eigenvectors of the linearised system above as follows.

The left eigenvector components of $J_{\beta_{hv}^*}$, which correspond to the uninfected states are zero
 (see Lemma 3 in [33]). Thus a non-zero components correspond to the infected states. It follows
 that the matrix $J_{\beta_{hv}^*}$ has a left eigenvector given by $\mathbf{v} = (v_1, v_2, \dots, v_{11})$, where

$$v_1 = v_2 = v_5 = v_6 = v_9 = v_{10} = v_{11} = 0; \quad v_3 = \frac{k_8 N_h^0}{a(1-\alpha_1)\beta_{hv}^* H^0} v_8;$$

$$v_4 = \frac{a(1-\alpha_1)\beta_{vh} S_v^0 (\eta_v k_8 + \gamma_v)}{k_4 k_9 N_h^0} v_8, \quad v_7 = \frac{(\eta_v k_8 + \gamma_v)}{k_9} v_8, \quad v_8 = v_8 > 0.$$

The system (2) has a right eigenvector given by $\mathbf{w} = (w_1, w_2, \dots, w_{11})^T$, where

$$w_{11} > 0, \quad w_8 > 0,$$

$$w_{10} = \frac{k_7}{l} w_{11}, \quad w_9 = \frac{K_1 \theta}{k_5 k_8} w_{11}, \quad w_7 = \frac{k_8}{\gamma_v} w_8, \quad w_6 = \frac{\theta}{k_8} w_{11} - \frac{k_9}{\gamma_v} w_8,$$

$$w_5 = \frac{\gamma_h \sigma k_8 k_9 N_h^0}{a(1-\alpha_1)\beta_{vh}\mu_h \gamma_v S_v^0 (\eta_h k_4 + \gamma_h)} w_8, \quad w_4 = \frac{\mu_h}{\sigma} w_5, \quad w_3 = \frac{k_4}{\gamma_h} w_4,$$

$$w_2 = -\frac{a(1-\alpha_1)\beta_{hv}^* (\eta_v k_8 + \gamma_v)}{\gamma_v N_h^0 (k_1 k_2 - \xi \omega)} (\xi S_h^0 + k_1 V_h^0) w_8,$$

$$w_1 = \frac{\omega}{k_1} w_2 - \frac{a(1-\alpha_1)\beta_{hv}^* S_h^0}{k_1 N_h^0} (\eta_v w_7 + w_8).$$

289 Theorem 4.1 in Castillo-Chavez and Song [43] is then applied to establish the existence of
 290 backward bifurcation in (2). To apply such a theorem, it is convenient to let f_k represent the
 291 right-hand side of the k^{th} equation of the system (2) and let x_k be the state variables whose
 292 derivative is given by the k^{th} equation for $k = 1, \dots, 11$. The local bifurcation analysis near
 293 the bifurcation point ($\beta_{hv} = \beta_{hv}^*$) is then determined by the signs of two associated constants,
 294 denoted by \mathcal{A}_1 and \mathcal{A}_2 , defined by

$$\mathcal{A}_1 = \sum_{k,i,j=1}^n v_k w_i w_j \frac{\partial^2 f_k(0,0)}{\partial x_i \partial x_j} \quad \text{and} \quad \mathcal{A}_2 = \sum_{k,i=1}^n v_k w_i \frac{\partial^2 f_k(0,0)}{\partial x_i \partial \phi} \quad (13)$$

295 with $\phi = \beta_{hv} - \beta_{hv}^*$. It is important to note that in $f_k(0, 0)$, the first zero corresponds to the
 296 disease-free equilibrium, \mathcal{E}_1 , for the system (2). Since $\beta_{hv} = \beta_{hv}^*$ is the bifurcation parameter,
 297 it follows from $\phi = \beta_{hv} - \beta_{hv}^*$ that $\phi = 0$ when $\beta_{hv} = \beta_{hv}^*$ which is the second component in
 298 $f_k(0, 0)$.

It follows then, after some algebraic manipulations, that

$$\mathcal{A}_1 = \Gamma_1 - \Gamma_2$$

299 with

$$\begin{aligned} \Gamma_1 &= \frac{a(1 - \alpha_1)\beta_{hv}^*(2V_h^0 w_1 + \pi S_h^0 w_2)}{(N_h^0)^2} (\eta_v w_7 + w_8) v_3 \\ &\quad + \frac{a(1 - \alpha_1)\beta_{vh} S_v^0}{N_h^0} \left[(\eta_h w_3 + w_4) \frac{1}{S_v^0} + \left(\eta_h w_3 + \frac{1}{S_v^0} w_4 \right) \right] w_6 v_7, \\ \Gamma_2 &= 2 \frac{a(1 - \alpha_1)\beta_{vh} S_v^0}{(N_h^0)^2} \left(\sum_{i=1}^5 w_i \right) (\eta_h w_3 + w_4) v_7 \\ &\quad + \frac{a(1 - \alpha_1)\beta_{hv}^*(S_h^0 + \pi V_h^0)(N_h^0 + 1)}{(N_h^0)^2} \left(\sum_{i=3}^5 w_i \right) (\eta_v w_7 + w_8) v_3, \end{aligned}$$

and

$$\mathcal{A}_2 = \frac{a(S_h^0 + \pi V_h^0)}{N_h^0} (\eta_v w_7 + w_8) v_3$$

301 Hence, the coefficient $\mathcal{A}_1 > 0$ if and only if

$$\Gamma_1 > \Gamma_2 \tag{14}$$

302 Note that the coefficient \mathcal{A}_2 is automatically positive. Thus, using Theorem 4.1 in [43], the
 303 following result is established.

304 **Theorem 6.** *The model (2) exhibits a backward bifurcation at $\mathcal{R}_0 = 1$ whenever the inequality*
 305 *(14) holds. If the reversed inequality holds, then the bifurcation at $\mathcal{R}_0 = 1$ is forward.*

306 The associated bifurcation diagrams are depicted in Figures 2 and 3. Parameter values used
 307 in figure 2 correspond to those in Table 4, except $\Lambda_h = 10$, $\epsilon = 1$, $\beta_{vh} = 0.8$, $\eta_h = 1$, $\eta_v = 1$,
 308 $\sigma = 0.01428$, $\delta = 1$, $\alpha_1 = 0.001$, $\alpha_2 = 1$, $c_m = 0.0001$, $\Gamma_E = 10^5$, $\Gamma_L = 50000$. In this case the
 309 conditions required by Theorem 6, are satisfied: $\mathcal{A}_1 = 0.0114 > 0$ and $\mathcal{A}_2 = 1.1393 > 0$.

310 Parameter values used in figure 3 correspond to those in Table 4, except $\Lambda_h = 10$, $\beta_{vh} = 0.8$,
 311 $\eta_h = \eta_v = 0 = \delta = c_m = \alpha_1 = 0$, $\alpha_2 = 1$, $\Gamma_E = 10^5$, $\Gamma_L = 50000$. We also have $\mathcal{A}_1 = -2.4223 <$
 312 0 and $\mathcal{A}_2 = 0.8333 > 0$.

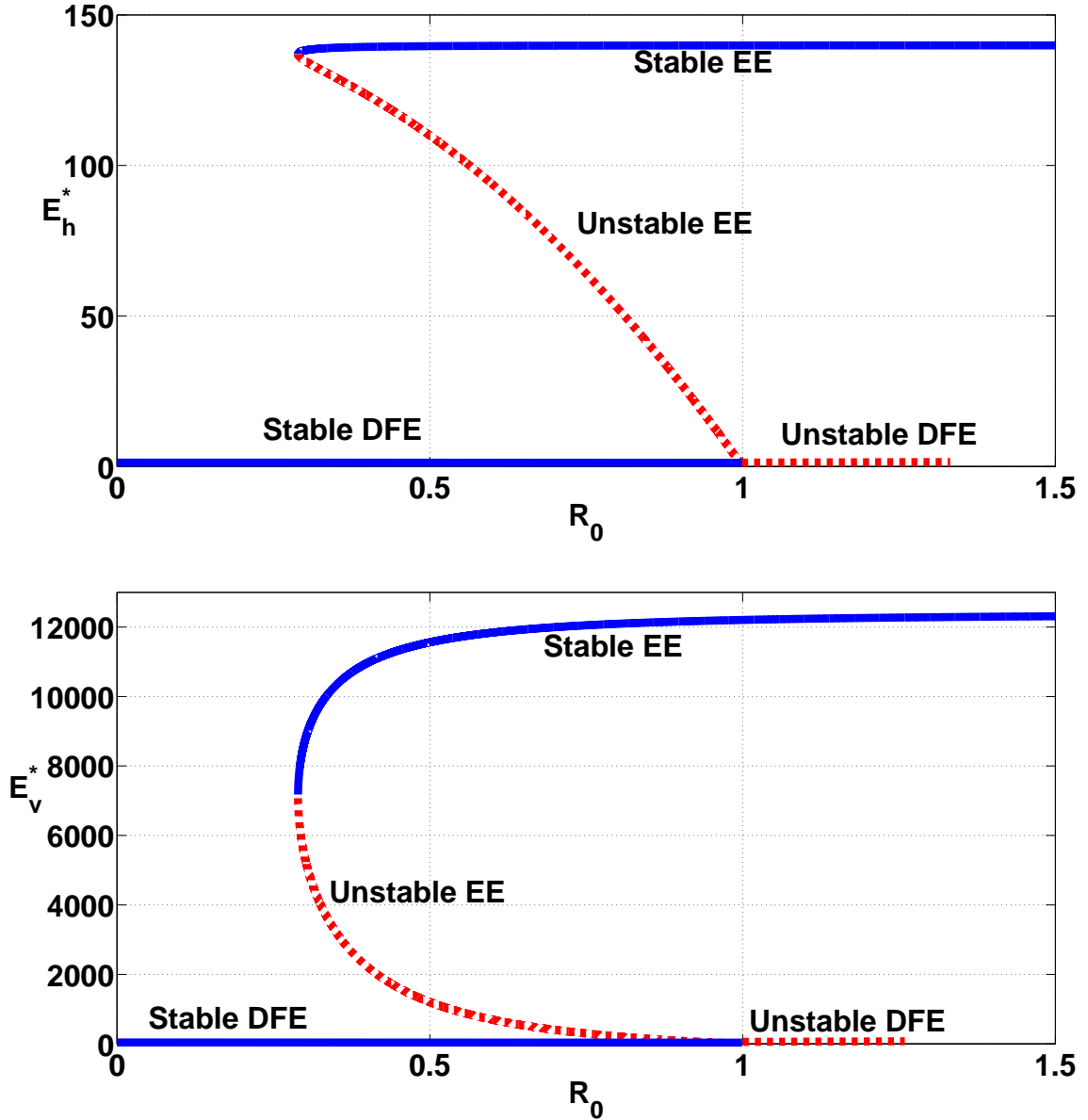


Figure 2: The backward bifurcation curves for model system (15) in the (\mathcal{R}_0, E_h^*) , and (\mathcal{R}_0, E_v^*) planes. The parameter β_{hv} is varied in the range $[0, 0.2810]$ to allow \mathcal{R}_0 to vary in the range $[0, 1.5]$. Two endemic equilibrium points coexist for values of \mathcal{R}_0 in the range $(0.2894, 1)$ (corresponding to the range $(0.0105, 0.1249)$ of β_{hv}). The notation EE and DFE stand for endemic equilibrium and disease-free equilibrium, respectively. Solid line represent stable equilibria and dash line stands for unstable equilibria.

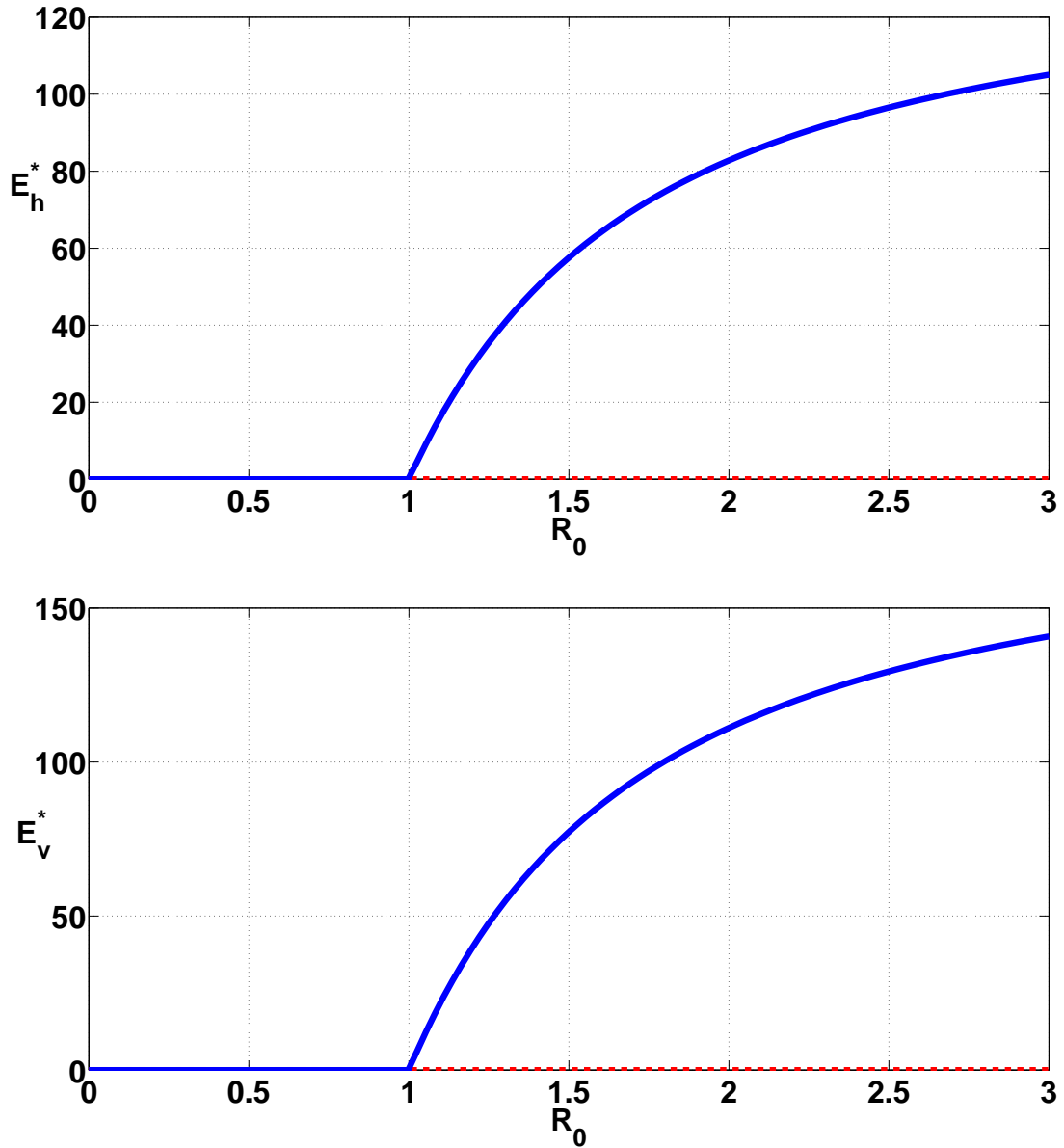


Figure 3: The forward bifurcation curves for model system (15) in the (\mathcal{R}_0, E_h^*) , and (\mathcal{R}_0, E_v^*) planes. Solid line represent stable equilibria and dash line stands for unstable disease-free equilibrium.

313 The occurrence of the backward bifurcation can be also seen in Figure 4. Here, \mathcal{R}_0 is less
 314 than the transcritical bifurcation threshold ($\mathcal{R}_0 = 0.29 < 1$), but the solution of the model 2 can
 315 approach either the endemic equilibrium point or the disease-free equilibrium point, depending
 316 on the initial condition.

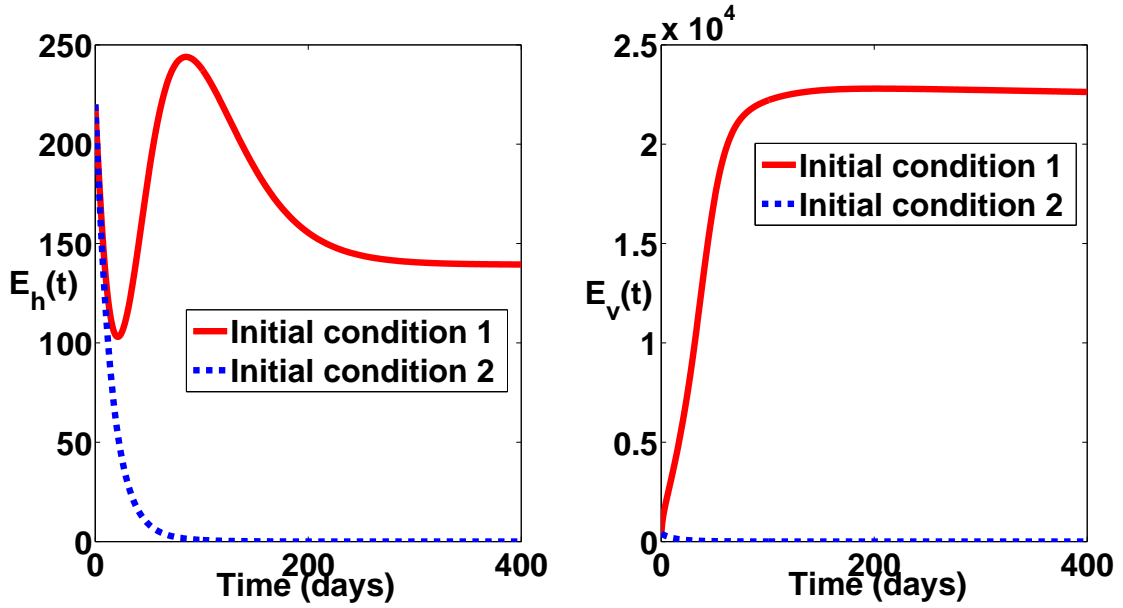


Figure 4: Solutions of model (2) of the number of infected humans, E_h , and the number of infected vectors, E_v , for parameter values given in the bifurcation diagram in Figure 2 with $\beta_{hv} = 0.0105$. So $\mathcal{R}_0 = 0.29 < 1$, for two different set of initial conditions. The first set of initial conditions (corresponding to the solid trajectory) is $S_h = 700$, $V_h = 10$, $E_h = 220$, $I_h = 100$, $R_h = 60$, $S_v = 3000$, $E_v = 400$, $I_v = 120$, $E = 10000$, $L = 5000$ and $P = 3000$. The second set of initial conditions (corresponding to the dotted trajectory) is $S_h = 489100$, $V_h = 10$, $E_h = 220$, $I_h = 100$, $R_h = 60$, $S_v = 3000$, $E_v = 400$, $I_v = 120$, $E = 10000$, $L = 5000$ and $P = 3000$. The solution for initial condition 1 approaches the locally asymptotically stable endemic equilibrium point, while the solution for initial condition 2 approaches the locally asymptotically stable DFE.

317 From theorem 5, item (i), it follows that the disease-induced death in human (δ) may be a
 318 cause of the occurrence of backward bifurcation phenomenon. In the following, we show that
 319 the backward bifurcation phenomenon is caused by the disease-induced death in human and/or
 320 the standard incidence functions (λ_h^c and λ_v^c).

321 3.3 The different causes of the backward bifurcation

322 To determine the different causes of the backward bifurcation phenomenon, we will consider, in
 323 this section, two variants of the model (2): the corresponding model without vaccination and
 324 the corresponding model with mass action incidence.

3.3.1 Analysis of the model without vaccination

The model without vaccination is given by

$$\begin{cases} \dot{S}_h &= \Lambda_h - (\lambda_h^c + \mu_h) S_h \\ \dot{E}_h &= \lambda_h^c S_h - (\mu_h + \gamma_h) E_h \\ \dot{I}_h &= \gamma_h E_h - (\mu_h + \delta + \sigma) I_h \\ \dot{R}_h &= \sigma I_h - \mu_h R_h \\ \dot{S}_v &= \theta P - \lambda_v^c S_v - (\mu_v + c_m) S_v \\ \dot{E}_v &= \lambda_v^c S_v - (\mu_v + \gamma_v + c_m) E_v \\ \dot{I}_v &= \gamma_v E_v - (\mu_v + c_m) I_v \\ \dot{E} &= \mu_b \left(1 - \frac{E}{\alpha_2 \Gamma_E}\right) (S_v + E_v + I_v) - (s + \mu_E + \eta_1) E \\ \dot{L} &= sE \left(1 - \frac{L}{\alpha_2 \Gamma_L}\right) - (l + \mu_L + \eta_2) L \\ \dot{P} &= lL - (\theta + \mu_P) P \end{cases} \quad (15)$$

where λ_h^c and λ_v^c are given at section 2. Model system (15) is defined in the positively-invariant set

$$\mathcal{D}_1 = \left\{ (S_h, E_h, I_h, R_h, S_v, E_v, I_v, E, L, P) \in \mathbb{R}^{10} : N_h \leq \Lambda_h / \mu_h; E \leq K_E; L \leq K_L; P \leq \frac{lK_L}{k_7}; N_v \leq \frac{\theta l K_L}{k_7 k_8} \right\}.$$

Without loss of generality, we assume that $\mathcal{N} > 1$. The corresponding disease-free equilibria of model (15) are given by $\mathcal{E}_0^{nv} = (N_h^0, 0, 0, 0, 0, 0, 0, 0, 0, 0)$ and

$\mathcal{E}_1^{nv} = (N_h^0, 0, 0, 0, N_v^0, 0, 0, E, L, P)$ with $N_h^0 = \frac{\Lambda_h}{\mu_h}$ and N_v^0, E, L and P are the same, given by (5). The associated next generation matrices, F_1 and V_1 , are, respectively, given by

$$F_1 = \begin{pmatrix} 0 & 0 & \tau \beta_{hv} \eta_v & \tau \beta_{hv} \\ 0 & 0 & 0 & 0 \\ \frac{\tau \beta_{vh} \eta_v N_v^0}{N_h^0} & \frac{\tau \beta_{vh} N_v^0}{N_h^0} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \text{ and } V_1 = \begin{pmatrix} k_3 & 0 & 0 & 0 \\ -\gamma_h & k_4 & 0 & 0 \\ 0 & 0 & k_9 & 0 \\ 0 & 0 & -\gamma_v & k_8 \end{pmatrix}.$$

It follows that the associated reproduction number for the model without vaccination, denoted by $\mathcal{R}_{nv} = \rho(F_1 V_1^{-1})$, is given by

$$\mathcal{R}_{nv} = \sqrt{\frac{a^2 (1 - \alpha_1)^2 \beta_{hv} \beta_{vh} (\gamma_h + k_4 \eta_h) (\gamma_v + k_8 \eta_v) N_v^0}{k_3 k_4 k_8 k_9 N_h^0}}. \quad (16)$$

Using Theorem 2 of [33], we establish the following result:

Theorem 7. *Assumed that $\mathcal{N} > 1$. For basic arboviral model without vaccination, given by (15), the corresponding disease-free equilibrium is LAS if $\mathcal{R}_{nv} < 1$, and unstable if $\mathcal{R}_{nv} > 1$.*

Existence of endemic equilibria Here, the existence of endemic equilibria of the model (15) will be explored. Let us set the following coefficients

$$\begin{aligned} \mathcal{R}_c &= \sqrt{\frac{\{2k_8(k_3 k_4 - \delta \gamma_h) + (\eta_h k_4 + \gamma_h) a \mu_h (1 - \alpha_1) \beta_{vh}\}}{k_3 k_4 k_8}}, \\ d_2 &= -k_9 \mu_b \Lambda_h (s K_E + k_6 K_L) (k_3 k_4 - \delta \gamma_h) ((\eta_h k_4 + \gamma_h) a \mu_h (1 - \alpha_1) \beta_{vh} \\ &\quad + (k_3 k_4 - \delta \gamma_h) k_8) < 0, \\ d_1 &= k_3^2 k_4^2 k_8 k_9 (s K_E + k_6 K_L) \mu_b \Lambda_h \mu_h (\mathcal{R}_{nv}^2 - R_c^2), \\ d_0 &= k_3^2 k_4^2 k_8 k_9 (s K_E + k_6 K_L) \mu_b \Lambda_h \mu_h^2 (\mathcal{R}_{nv}^2 - 1). \end{aligned} \quad (17)$$

341 We claim the following:

342 **Theorem 8.** *The arboviral diseases model without vaccination (15) has:*

343 (i) *a unique endemic equilibrium if $d_0 > 1 \Leftrightarrow \mathcal{R}_{nv} > 1$;*

344 (ii) *a unique endemic equilibrium if $d_1 > 0$, and $d_0 = 0$ or $d_1^2 - 4d_2d_0 = 0$;*

345 (iii) *two endemic equilibria if $d_0 < 0$ (i.e. $\mathcal{R}_{nv} < 1$), $d_1 > 0$ (i.e. $\mathcal{R}_{nv} > \mathcal{R}_c$) and $d_1^2 - 4d_2d_0 > 0$;*

346 (iv) *no endemic equilibrium if $d_0 < 0$ (i.e. $\mathcal{R}_{nv} < 1$) and $\delta = 0$.*

347 (v) *no endemic equilibrium otherwise.*

348 *Proof.* Solving the equations in the model (15) in terms of $\lambda_h^{c,*}$ and $\lambda_v^{c,*}$, gives

$$S_h^* = \frac{\Lambda_h}{\mu_h + \lambda_h^{c,*}}, \quad E_h^* = \frac{\lambda_h^{c,*} S_h^*}{k_3}, \quad I_h^* = \frac{\gamma_h \lambda_h^{c,*} S_h^*}{k_3 k_4}, \quad R_h^* = \frac{\sigma \gamma_h \lambda_h^{c,*} S_h^*}{\mu_h k_3 k_4}, \quad (18)$$

349 and

$$\begin{aligned} S_v^* &= \frac{\theta P}{(\lambda_v^{c,*} + k_8)}, & E_v^* &= \frac{\theta P \lambda_v^{c,*}}{k_9 (\lambda_v^{c,*} + k_8)}, & I_v^* &= \frac{\gamma_v \theta P \lambda_v^{c,*}}{k_8 k_9 (\lambda_v^{c,*} + k_8)}, \\ E &= \frac{\mu_b \theta K_E P}{(k_5 k_8 K_E + \mu_b \theta P)}, & L &= \frac{\mu_b \theta s K_E K_L P}{k_6 K_L (k_5 k_8 K_E + \mu_b \theta P) + s \mu_b \theta K_E P}, \end{aligned} \quad (19)$$

350 Substituting (18) and (19) into the expression of λ_h^* and λ_v^* and simplifying, shows that the
351 nonzero equilibria of the model without vaccination satisfy the quadratic equation

$$d_2 (\lambda_h^{c,*})^2 + d_1 \lambda_h^{c,*} + d_0 = 0 \quad (20)$$

352 where d_i , $i = 0, 1, 2$, are given by (17).

353 Clearly, $d_2 < 0$ and $d_0 > 0$ (resp. $d_0 < 0$) if $\mathcal{R}_{nv} > 1$ (resp. $\mathcal{R}_{nv} < 1$). Thus Theorem 8 is
354 established. \square

355 It is clear that cases (ii) and (iii) of theorem 8 indicates the possibility of backward bi-
356 furcation (where the locally-asymptotically stable DFE co-exists with a locally-asymptotically
357 stable endemic equilibrium when $\mathcal{R}_{nv} < 1$) in the model without vaccination (15).

358 This is illustrated by simulating the model with the following set of parameter values (it
359 should be stated that these parameters are chosen for illustrative purpose only, and may not
360 necessarily be realistic epidemiologically): $\Lambda_h = 5$, $\beta_{hv} = 0.03$, $\eta_h = \eta_v = 1$, $\delta = 1$, $\sigma = 0.01$,
361 $c_m = 0.1$, $\beta_{vh} = 0.4$, $\alpha_1 = 0.7$ and $\alpha_2 = 0.5$. All other parameters are as in Table 4. With this
362 set of parameters, $\mathcal{R}_c = 0.0216 < 1$, $\mathcal{R}_{nv} = 0.2725 < 1$ (so that $\mathcal{R}_c < \mathcal{R}_{nv} < 1$). It follows:
363 $d_2 = -0.0263 < 0$, $d_1 = 4.8763 \times 10^{-4}$ and $d_0 = -3.5031 \times 10^{-7}$, so that $d_1^2 - 4d_2d_0 = 2.0093 \times$
364 $10^{-7} > 0$. The resulting two endemic equilibria $\mathcal{E}^{nv} = (S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, E_v^*, I_v^*, E, L, P)$, are:

365 $\mathcal{E}_1^{nv} = (281, 70, 5, 1207, 5739, 182, 44, 22180, 10201, 9977)$ which is locally stable and

366 $\mathcal{E}_2^{nv} = (6333, 67, 4, 1147, 5936, 37, 2, 22180, 10201, 9977)$ which is unstable.

367 The associated bifurcation diagram is depicted in figure 5. This clearly shows the co-
368 existence of two locally-asymptotically stable equilibria when $\mathcal{R}_{nv} < 1$, confirming that the
369 model without vaccination (15) undergoes the phenomenon of backward bifurcation too.

370 Thus, the following result is established.

371 **Lemma 1.** *The model without vaccination (15) undergoes backward bifurcation when Case (iii)*
372 *of Theorem 8 holds.*

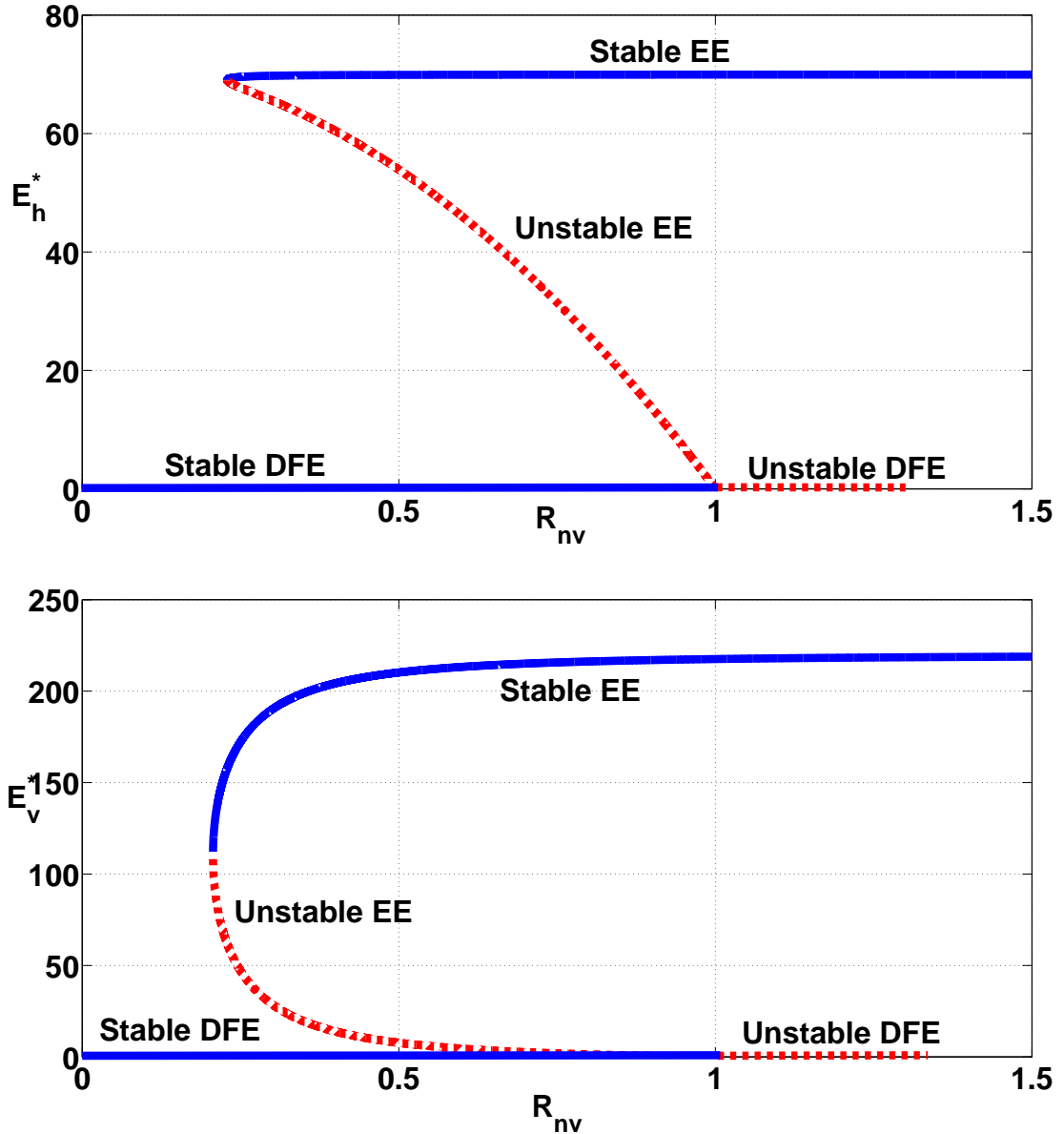


Figure 5: The backward bifurcation curves for model system (15) in the $(\mathcal{R}_{nv}, E_h^*)$, and $(\mathcal{R}_{nv}, E_v^*)$ planes. The parameter β_{hv} is varied in the range $[0, 0.9090]$ to allow \mathcal{R}_0 to vary in the range $[0, 1.5]$. Two endemic equilibrium points coexist for values of \mathcal{R}_0 in the range $(0.2286, 1)$ (corresponding to the range $(0.0211, 0.4040)$ of β_{hv}). The notation EE and DFE stand for endemic equilibrium and disease free equilibrium, respectively. Solid line represent stable equilibria and dash line stands for unstable equilibria.

373 **Non-existence of endemic equilibria for $\mathcal{R}_{nv} < 1$ and $\delta = 0$** In this case, we have the
 374 following result.

375 **Lemma 2.** *The model (15) without disease-induced death ($\delta = 0$) has no endemic equilibrium*
 376 *when $\mathcal{R}_{nv, \delta=0} \leq 1$, and has a unique endemic equilibrium otherwise.*

377 *Proof.* Considering the model (15) without disease-induced death in human, and applying the
 378 same procedure, we obtain that the non-zero equilibria of the model without vaccination satisfy

379 the linear equation

$$p_1 \lambda_h^{c,*} + p_0 = 0,$$

380 where $p_1 = k_9 k_{10} K_{12} a \mu_b \Lambda_h \mu_h (1 - \alpha_1) \beta_{vh} + k_3 (\mu_h + \sigma) k_8 k_9 K_{12} \mu_b \Lambda_h$ and
 381 $p_0 = -\mu_h k_3 k_4 k_8 k_9 K_{12} \mu_b \Lambda_h (\mathcal{R}_{nv, \delta=0}^2 - 1)$.

382 Clearly, $p_1 > 0$ and $p_0 \geq 0$ whenever $R_{nv, \delta=0} \leq 1$, so that $\lambda_h^{c,*} = -\frac{p_0}{p_1} \leq 0$. Therefore,
 383 the model (15) without disease-induced death in human, has no endemic equilibrium whenever
 384 $\mathcal{R}_{nv, \delta=0}^2 \leq 1$. \square

385 The above result suggests the impossibility of backward bifurcation in the model (15) with-
 386 out disease-induced death, since no endemic equilibrium exists when $\mathcal{R}_{nv, \delta=0} < 1$ (and backward
 387 bifurcation requires the presence of at least two endemic equilibria when $\mathcal{R}_{nv, \delta=0} < 1$) [19, 38].
 388 To completely rule out backward bifurcation in model (15), we use the direct Lyapunov method
 389 to prove the global stability of the DFE.

390 **Global stability of the DFE of (15) for $\delta = 0$** Define the positively-invariant and attract-
 391 ing region

$$\mathcal{D}_2 = \{(S_h, E_h, I_h, R_h, S_v, E_v, I_v, E, L, P) \in \mathcal{D}_1 : S_h \leq N_h^0; S_v \leq N_v^0\}$$

392 We claim the following result.

393 **Theorem 9.** *The DFE, \mathcal{E}_1^{nv} , of model (15) without disease-induced death ($\delta = 0$), is globally*
 394 *asymptotically stable (GAS) in \mathcal{D}_2 if $\mathcal{R}_{nv, \delta=0} < 1$.*

395 *Proof.* Consider the Lyapunov function

$$\mathcal{G} = q_1 E_h + q_2 I_h + q_3 E_v + q_4 I_v.$$

396 where

$$q_1 = \frac{1}{k_3}; q_3 = \frac{\zeta_1 S_h^0 (\gamma_v + k_8 \eta_v)}{k_3 k_8 k_9}, q_2 = \frac{\zeta_1 S_h^0 (\gamma_v + k_8 \eta_v) \zeta_2 S_v^0}{k_3 k_8 k_4 k_9}, q_4 = \frac{\zeta_1 S_h^0}{k_3 k_8}.$$

397 and we have set $\zeta_1 = \frac{\tau \mu_h \beta_{hv}}{\Lambda_h}$ and $\zeta_2 = \frac{\tau \mu_h \beta_{vh}}{\Lambda_h}$. The derivative of \mathcal{G} is given by

$$\begin{aligned} \dot{\mathcal{G}} &= q_1 \dot{E}_h + q_2 \dot{I}_h + q_3 \dot{E}_v + q_4 \dot{I}_v \\ &= q_1 (\lambda_h^c S_h - k_3 E_h) + q_2 (\gamma_h E_h - k_4 I_h) + q_3 (\lambda_v^c S_v - k_9 E_v) + q_4 (\gamma_v E_v - k_8 I_v) \\ &= q_1 \zeta_1 S_h (\eta_v E_v + I_v) - q_3 k_9 E_v + q_4 \gamma_v E_v - q_4 k_8 I_v \\ &\quad + q_3 \zeta_2 S_v (\eta_h E_h + I_h) - q_1 k_3 E_h + q_2 \gamma_h E_h - q_2 k_4 I_h \\ &= (q_1 \zeta_1 S_h \eta_v + q_4 \gamma_v - q_3 k_9) E_v + (q_1 \zeta_1 S_h - q_4 k_8) I_v \\ &\quad + (q_3 \zeta_2 S_v \eta_h + q_2 \gamma_h - q_1 k_3) E_h + (q_3 \zeta_2 S_v - q_2 k_4) I_h \\ &\leq (q_1 \zeta_1 S_h^0 \eta_v + q_4 \gamma_v - q_3 k_9) E_v + (q_1 \zeta_1 S_h^0 - q_4 k_8) I_v \\ &\quad + (q_3 \zeta_2 S_v^0 \eta_h + q_2 \gamma_h - q_1 k_3) E_h + (q_3 \zeta_2 S_v^0 - q_2 k_4) I_h, \text{ since } S_h \leq S_h^0, S_v \leq S_v^0 \end{aligned}$$

398 Replacing q_i , $i = 1, \dots, 4$, by their value gives after straightforward simplifications

$$\dot{\mathcal{G}} \leq (\mathcal{R}_{nv, \delta=0}^2 - 1) E_h$$

399 We have $\dot{\mathcal{G}} \leq 0$ if $R_{nv, \delta=0} \leq 1$, with $\dot{\mathcal{G}} = 0$ if $\mathcal{R}_{nv, \delta=0} = 1$ or $E_h = 0$. Whenever $E_h = 0$, we also
 400 have $I_h = 0$, $E_v = 0$ and $I_v = 0$. Substituting $E_h = I_h = E_v = I_v = 0$ in the first, fourth and

401 fifth equation of Eq. (15) with $\delta = 0$ gives $S_h(t) \rightarrow S_h^0 = N_h^0$, $R_h(t) \rightarrow 0$, and $S_v(t) \rightarrow S_v^0 = N_v^0$
 402 as $t \rightarrow \infty$. Thus

$$[S_h(t), E_h(t), I_h(t), R_h(t), S_v(t), E_v(t), I_v(t), E(t), L(t), P(t)] \rightarrow (N_h^0, 0, 0, 0, N_v^0, 0, 0, E, L, P)$$

as $t \rightarrow \infty$.

403 It follows from the LaSalle's invariance principle [46, 47, 48] that every solution of (15) (when
 404 $\mathcal{R}_{nv, \delta=0} \leq 1$), with initial conditions in \mathcal{D}_2 converges to \mathcal{E}_1^{nv} , as $t \rightarrow \infty$. Hence, the DFE, \mathcal{E}_1^{nv} ,
 405 of model (15) without disease-induced death, is GAS in \mathcal{D}_2 if $\mathcal{R}_{nv, \delta=0} \leq 1$. \square

406 3.3.2 Analysis of the model with mass action incidence

407 Consider the model (2) with mass action incidence. Thus, the associated forces of infection, λ_h^c
 408 and λ_v^c , respectively, reduce to

$$\lambda_{mh} = C_h(\eta_v E_v + I_v) \quad \text{and} \quad \lambda_{mv} = C_v(\eta_h E_h + I_h), \quad (21)$$

where, $C_h = a(1 - \alpha_1)\beta_{hv}$ and $C_v = a(1 - \alpha_1)\beta_{vh}$. The resulting model (mass action model),
 obtained by using (21) in (2), has the same disease-free equilibria given by (5). Without lost
 of generality, we consider that $\mathcal{N} > 1$. The associated next generation matrices, F_m and V_m
 are given by

$$F_m = \begin{pmatrix} 0 & 0 & C_h \eta_v H^0 & C_h H^0 \\ 0 & 0 & 0 & 0 \\ C_v \eta_v S_v^0 & C_v S_v^0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad V_m = \begin{pmatrix} k_3 & 0 & 0 & 0 \\ -\gamma_h & k_4 & 0 & 0 \\ 0 & 0 & k_9 & 0 \\ 0 & 0 & -\gamma_v & k_8 \end{pmatrix},$$

409 where $H^0 = S_h^0 + \pi V_h^0$. It follows that the associated reproduction number for the mass action
 410 model, denoted by $\mathcal{R}_{0,m} = \rho(F_m V_m^{-1})$, is given by

$$\mathcal{R}_{0,m} = \sqrt{\mathcal{R}_{hv}^m \mathcal{R}_{vh}^m}, \quad (22)$$

411 where

$$412 \mathcal{R}_{hv}^m = \left(\frac{C_h \Lambda_h (\gamma_h + k_4 \eta_h) (\pi \xi + k_2)}{\mu_h k_3 k_4 (\xi + k_2)} \right) \quad \text{and} \quad \mathcal{R}_{vh}^m = \left(\frac{C_v (\gamma_v + k_8 \eta_v) \theta P}{k_8^2 k_9} \right).$$

413 Using Theorem 2 of [33], the following result is established:

414 **Theorem 10.** *Assume that $\mathcal{N} > 1$. For the arboviral disease model with mass action incidence,*
 415 *given by (2) with (21), the DFE, \mathcal{E}_1 , is LAS if $\mathcal{R}_{0,m} < 1$, and unstable if $\mathcal{R}_{0,m} > 1$*

416 **Existence of endemic equilibria** Solving the equations in the model (15) in terms of λ_{mh}^*
 417 and λ_{mv}^* , gives

$$S_{mh}^* = \frac{\Lambda_h (\pi \lambda_{mh}^{c,*} + k_2)}{\lambda_{mh}^{c,*} (k_2 + \pi (k_1 + \lambda_{mh}^{c,*})) + k_1 k_2 - \omega \xi}, \quad V_{mh}^* = \frac{\xi S_{mh}^*}{k_2 + \pi \lambda_{mh}^{c,*}}, \quad (23)$$

$$E_{mh}^* = \frac{\lambda_{mh}^{c,*} S_{mh}^*}{k_3}, \quad I_{mh}^* = \frac{\gamma_h \lambda_{mh}^{c,*} S_{mh}^*}{k_3 k_4}, \quad R_{mh}^* = \frac{\sigma \gamma_h \lambda_{mh}^{c,*} S_{mh}^*}{\mu_h k_3 k_4},$$

418 and

$$S_{mv}^* = \frac{\theta P}{(\lambda_{mv}^{c,*} + k_8)}, \quad E_{mv}^* = \frac{\theta P \lambda_{mv}^{c,*}}{k_9 (\lambda_{mv}^{c,*} + k_8)}, \quad I_{mv}^* = \frac{\gamma_v \theta P \lambda_{mv}^{c,*}}{k_8 k_9 (\lambda_{mv}^{c,*} + k_8)}. \quad (24)$$

419 Substituting (23) and (24) into the expression of λ_{mh}^* and λ_{mv}^* and simplifying, shows that the
 420 nonzero equilibria of the model without vaccination satisfy the quadratic equation

$$e_2(\lambda_{mh}^{c,*})^2 + e_1\lambda_{mh}^{c,*} + e_0 = 0, \quad (25)$$

421 where e_i , $i = 0, 1, 2$, are given by

$$\begin{aligned} e_2 &= k_8 k_9 \pi [(\gamma_h + k_4 \eta_h) C_v \Lambda_h + k_3 k_4 k_8] \\ e_1 &= \frac{k_3 k_4 k_8^2 k_9 \kappa \pi}{(\pi \xi + k_2)} (\mathcal{R}_{cm}^2 - \mathcal{R}_{0,m}^2), \\ e_0 &= k_3 k_4 k_8^2 k_9 \kappa (1 - \mathcal{R}_{0,m}^2), \end{aligned}$$

with $\kappa = k_1 k_2 - \xi \omega > 0$ and

$$\mathcal{R}_{cm} = \sqrt{\frac{[(\gamma_h + k_4 \eta_h) (\pi \xi + k_2) \Lambda_h C_v + (k_1 \pi + k_2) k_3 k_4 k_8] (\pi \xi + k_2)}{k_3 k_4 k_8 \kappa \pi}}.$$

422 e_2 is always positive and e_0 is positive (resp. negative) whenever $R_{0,m}$ is less (resp. greater) than
 423 unity. Thus, the mass action model admits only one endemic equilibria whenever $\mathcal{R}_{0,m} > 1$.

424 Now, we consider the case $\mathcal{R}_{0,m} < 1$. The occurrence of backward bifurcation phenomenon
 425 depend of the sign of coefficient e_1 . The coefficient e_1 is always positive if and only if $R_{0,m} <$
 426 \mathcal{R}_{cm} . It follows that the disease-free equilibrium is the unique equilibrium when $\mathcal{N} > 1$ and
 427 $\mathcal{R}_{cm} < 1$. Now if $\mathcal{R}_{cm} < \mathcal{R}_{0,m} < 1$, then in addition to the DFE \mathcal{E}_1 , there exists two endemic
 428 equilibria whenever $\Delta_m = e_1^2 - 4e_2e_0 > 0$. However, $\mathcal{R}_{cm} < \mathcal{R}_{0,m} < 1 \Rightarrow \mathcal{R}_{cm} < 1 \Leftrightarrow$
 429 $\beta_{vh} < -\frac{k_3 k_4 k_8 (\xi \omega \pi + k_1 \pi^2 \xi + k_2 (\pi \xi + k_2))}{a(1 - \alpha_1) (\gamma_h + k_4 \eta_h) (\pi \xi + k_2) (\pi \xi + k_2) \Lambda_h} < 0$. Since all parameter of model are non-
 430 negative, we conclude that the condition $\mathcal{R}_{cm} < \mathcal{R}_{0,m} < 1$ does not hold. And thus, the model
 431 with mass-action incidence does not admit endemic equilibria for $\mathcal{R}_{cm} < 1$.

432 **Global stability of the DFE for the model with mass action incidence** Since the DFE
 433 of the model with mass action incidence is the unique equilibrium whenever the corresponding
 434 basic reproduction number $R_{0,m}$ is less than unity, it remains to show that the DFE is gas. To
 435 this aim, we use the direct Lyapunov method. Let us define the following positive constants:

$$p_1 = \frac{1}{k_3}, p_2 = \frac{C_h H^0 (\eta_v k_8 + \gamma_v) C_v S_v^0}{k_8 k_9 k_3 k_4}, p_3 = p_1 C_h H^0 \frac{(\eta_v k_8 + \gamma_v)}{k_8 k_9}, p_4 = \frac{C_h H^0}{k_3 k_8}.$$

436 Consider the Lyapunov function

$$\mathcal{L} = p_1 E_h + p_2 I_h + p_3 E_v + p_4 I_v.$$

437 The derivative of \mathcal{L} is given by

$$\begin{aligned} \dot{\mathcal{L}} &= p_1 \dot{E}_h + p_2 \dot{I}_h + p_3 \dot{E}_v + p_4 \dot{I}_v \\ &= (p_1 C_h \eta_v H + p_4 \gamma_v - p_3 k_9) E_v + (p_1 C_h H - p_4 k_8) I_v \\ &\quad + (p_3 C_v \eta_h S_v + p_2 \gamma_h - p_1 k_3) E_h + (p_3 C_v S_v - p_2 k_4) I_h \end{aligned}$$

438 Replacing p_i , $i = 1, \dots, 4$, by their respective term, and using the fact that $H = (S_h + \pi V_h) \leq$
 439 $H^0 = (S_h^0 + \pi V_h^0)$ and $S_v \leq N_v^0$ in

$$\mathcal{D}_3 = \left\{ (S_h, V_h, E_h, I_h, R_h, S_v, E_v, I_v, E, L, P) \in \mathcal{D} : N_h \leq \frac{\Lambda_h}{\mu_h}, S_v \leq N_v^0 = \theta P, E \leq K_E, L \leq K_L, P \leq \frac{l K_L}{k_7 k_8} \right\},$$

440 we obtain $\dot{\mathcal{L}} \leq (R_{0,m}^2 - 1) E_h$.

441 We have $\dot{\mathcal{L}} \leq 0$ if $R_{0,m} \leq 1$, with $\dot{\mathcal{L}} = 0$ if $\mathcal{R}_{0,m} = 1$ or $E_h = 0$. Whenever $E_h = 0$, we also
 442 have $I_h = 0$, $E_v = 0$ and $I_v = 0$. Substituting $E_h = I_h = E_v = I_v = 0$ in the first, fourth and
 443 fifth equation of Eq. (15) with mass action incidence gives $S_h(t) \rightarrow S_h^0$, $V_h(t) \rightarrow V_h^0$, $R_h(t) \rightarrow 0$,
 444 and $S_v(t) \rightarrow S_v^0 = N_v^0$ as $t \rightarrow \infty$. Thus

$$\begin{aligned} & [S_h(t), V_h(t), E_h(t), I_h(t), R_h(t), S_v(t), E_v(t), I_v(t), E(t), L(t), P(t)] \\ & \rightarrow (S_h^0, V_h^0, 0, 0, 0, N_v^0, 0, 0, E, L, P) \quad \text{as } t \rightarrow \infty. \end{aligned}$$

445 It follows from the LaSalle's invariance principle [46, 47, 48], that every solution of (2) with
 446 mass action incidence, with initial conditions in \mathcal{D}_3 converges to the DFE, as $t \rightarrow \infty$. Hence,
 447 the DFE, \mathcal{E}_1 , of the model with mass action incidence, is GAS in \mathcal{D}_3 if $\mathcal{R}_{0,m} \leq 1$.

448 Thus, we claim the following result.

449 **Theorem 11.** *The DFE, \mathcal{E}_1 , of the model (2) with mass action incidence, is globally asymp-*
 450 *totically stable (GAS) in \mathcal{D}_3 if $\mathcal{R}_{0,m} < 1$.*

451 Thus, the substitution of standard incidence with mass action incidence in the arboviral
 452 model (2) removes the backward bifurcation phenomenon of the model. It should be mentioned
 453 that a similar situation was reported by Garba *et al.* in [19] and by Sharomi *et al.* in [38].

454 We summarize the previous analysis of Subsection 3.3 as follows:

455 **Lemma 3.** *The main causes of occurrence of backward bifurcation phenomenon in models (2)*
 456 *and (15) are the disease-induced death and the standard incidence rates.*

457 4 Sensitivity analysis

458 As shown in the previous sections, model (2) may admit single or multiple steady states accord-
 459 ing to the value of the basic reproduction number \mathcal{R}_0 . In turn, \mathcal{R}_0 depends on the parameters
 460 of the model. The various uncertainties encountered in data collection and the estimated values
 461 leads us to evaluate the robustness of the model predictions with the parameter values and,
 462 in particular, to estimate the effect on \mathcal{R}_0 of varying single parameters. To this aim, we use
 463 sensitivity analysis and calculate the sensitivity indices of \mathcal{R}_0 to the parameters in the model
 464 using both local and global methods.

465 4.1 Local sensitivity analysis

466 The local sensitivity analysis, based on the *normalised sensitivity index* of \mathcal{R}_0 (see [49]), is given
 467 by

$$S_\Psi = \frac{\Psi}{\mathcal{R}_0} \frac{\partial \mathcal{R}_0}{\partial \Psi}$$

468 where Ψ denotes the generic parameter of (2).

469 This index indicates how sensitive \mathcal{R}_0 is to changes of parameter Ψ . Clearly, a positive (resp.
 470 negative) index indicates that an increase in the parameter value results in an increase (resp.
 471 decrease) in the \mathcal{R}_0 value [49].

472 For instance, the computation of the sensitivity index of \mathcal{R}_0 with respect to a is given by

$$S_a = \frac{a}{\mathcal{R}_0} \frac{\partial \mathcal{R}_0}{\partial a} = 1 > 0.$$

Table 4: Parameter values using to compute the sensitivity indices of \mathcal{R}_0 .

Parameter	value	Parameter	value	Parameter	value
c_m	0.01	s	0.7	β_{vh}	0.75
μ_b	6	η_2	0.3	Γ_E	10000
μ_P	0.4	μ_E	0.2	Γ_L	5000
θ	0.08	ϵ	0.61	α_2	0.5
l	0.5	Λ_h	2.5	μ_h	$\frac{1}{67*365}$
a	1	β_{hv}	0.75	η_v	0.35
μ_v	$\frac{1}{30}$	μ_L	0.4	σ	0.1428
γ_h	$\frac{1}{14}$	η_h	0.35	γ_v	$\frac{1}{21}$
ξ	0.5	ω	0.05	η_1	0.001
δ	0.001	α_1	0.2		

Table 5: Sensitivity indices of \mathcal{R}_0 to parameters of model (2), evaluated at the baseline parameter values given in Table 4.

Parameter	Index	Parameter	Index	Parameter	Index
a	+1	σ	-0.2911	ξ	-0.0566
μ_v	-0.9190	c_m	-0.2757	ω	+0.0565
ϵ	-0.6223	α_1	-0.25	μ_E	-0.0171
s	+0.5172	η_h	+0.2067	δ	-0.0020
Λ_h	-0.5	γ_h	-0.2064	η_1	-0.0000858
$\beta_{hv}, \beta_{vh}, \Gamma_E, \Gamma_L, \alpha_2$	+0.5	η_v	+0.1207		
μ_h	+0.4996	γ_v	+0.1174		
μ_P	-0.4810	μ_L	-0.1026		
θ	+0.4810	μ_b	+0.0772		
l	+0.4489	η_2	-0.0770		

473 This shows that \mathcal{R}_0 is an increasing function of a and the parameter a has an influence on the
474 spread of disease.

475 We tabulate the indices of the remaining parameters in Table 2 using parameter values on
476 Table 4. The results, displayed in Table 5 and Figure 7a. The parameters are arranged from
477 most sensitive to least. The model system (2) is most sensitive to a , the average number of
478 mosquitoes bites, followed by μ_v , ϵ , s , Λ_h , β_{hv} , β_{vh} , Γ_E , Γ_L and α_2 . It is important to note
479 that increasing (decreasing) a by 10% increases (decreases) \mathcal{R}_0 by 10%. However, increasing
480 (decreasing) the parameters μ_v by 10% decreases (increases) \mathcal{R}_0 by 9.190%. The same reasoning
481 can be done for other parameters.

482 4.2 Uncertainty and global sensitivity analysis

483 Local sensitivity analysis assesses the effects of individual parameters at particular points in
484 parameter space without taking into account of the combined variability resulting from con-
485 sidering all input parameters simultaneously. Here, we perform a global sensitivity analysis to
486 examine the model's response to parameter variation within a wider range in the parameter
487 space.

488 Following the approach by Marino *et al.* [50] and Wu *et al.* [51], partial rank correlation

489 coefficients (PRCC) between the basic reproduction number \mathcal{R}_0 and each parameter are derived
 490 from 5,000 runs of the Latin hypercube sampling (LHS) method [52]. The parameters are
 491 assumed to be random variables with uniform distributions with its mean value listed in Table 4.

492 With these 5,000 runs of LHS, the derived distribution of \mathcal{R}_0 is given in Figure 6. This
 493 sampling shows that the mean of \mathcal{R}_0 is 2.0642 and the standard deviation is 2.6865. The
 494 probability that $\mathcal{R}_0 > 1$ is 54.86%. This implies that for the mean of parameter values given
 495 in Table 4, we may be confident that the model predicts a endemic state.

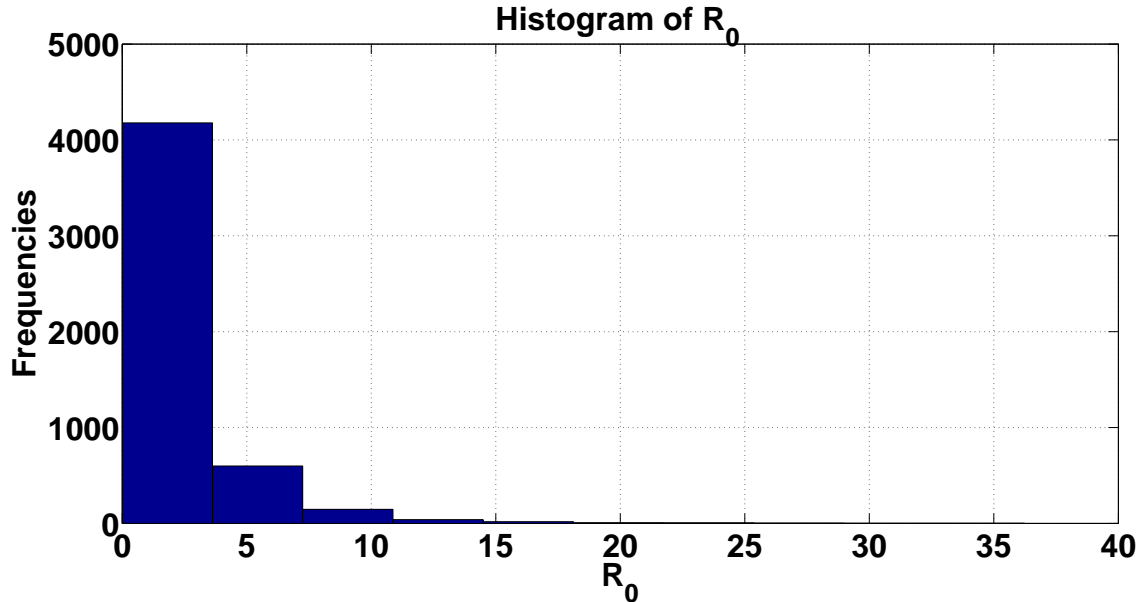


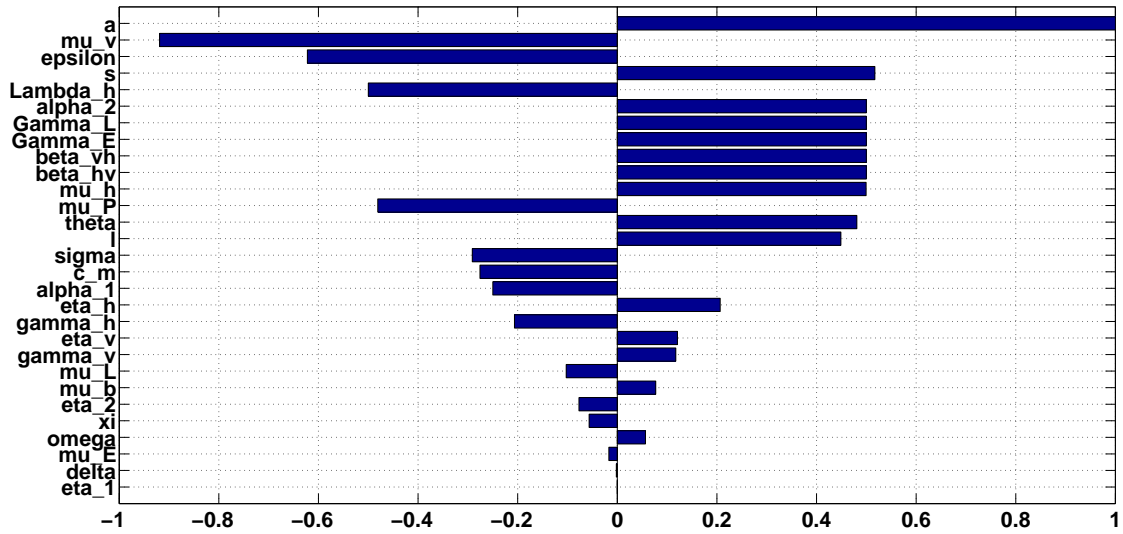
Figure 6: Sampling distribution of \mathcal{R}_0 from 5,000 runs of Latin hypercube sampling. The mean of \mathcal{R}_0 is 2.0642 and the standard deviation is 2.6865. Furthermore, $\mathbb{P}(R_0 > 1) = 0.5486$.

496 We now use sensitivity analysis to analyse the influence of each parameter on the basic
 497 reproductive number. From the previously sampled parameter values, we compute the PRCC
 498 between \mathcal{R}_0 and each parameter of model (2). The parameters with large PRCC values (> 0.5 or
 499 < -0.5) statistically have the most influence [51]. The results, displayed in Table 6 and Figure
 500 7 (b), show that the parameters α_1 , the human protection rate, has the highest influence on
 501 \mathcal{R}_0 . This suggests that individual protection may potentially be the most effective strategy to
 502 reduce \mathcal{R}_0 . The other parameter with an important effect are α_2 , β_{hv} , β_{vh} and θ .

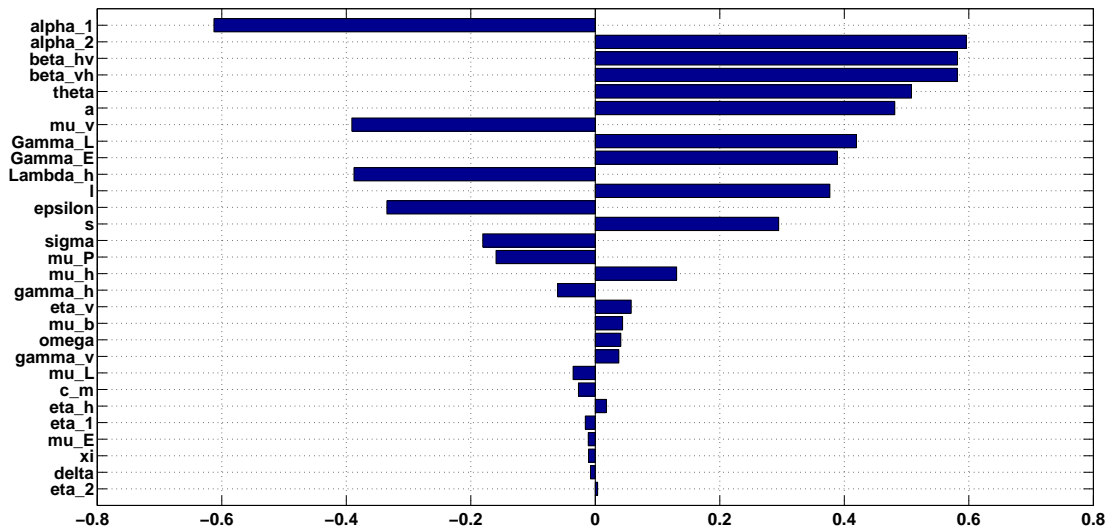
503 We note that the order of the most important parameters for \mathcal{R}_0 from the local sensitivity
 504 analysis does not match that of the global sensitivity analysis, showing that the local results
 505 are not robust.

Table 6: Partial Rank Correlation Coefficients between \mathcal{R}_0 and each parameters of model (2).

Para- meter	Correlation Coefficients	Para- meter	Correlation Coefficients	Para meter	Correlation Coefficients
α_1	-0.6125	l	0.3767	γ_v	0.0378
α_2	0.5960	ϵ	-0.3348	μ_L	-0.0357
β_{hv}	0.5817	s	0.2945	c_m	-0.0271
β_{vh}	0.5815	σ	-0.1808	η_h	0.0178
θ	0.5078	μ_P	-0.1594	η_1	-0.0161
a	0.4810	μ_h	0.1306	μ_E	-0.0113
μ_v	-0.3911	γ_h	-0.0605	ξ	-0.0109
Γ_L	0.4195	η_v	0.0578	δ	-0.0077
Γ_E	0.3888	μ_b	0.0439	η_2	0.0037
Λ_h	-0.3876	ω	0.0410		



(a) Local sensitivity indices for \mathcal{R}_0



(b) Partial rank correlation coefficients for \mathcal{R}_0

Figure 7: Local (a) and global (b) sensitivity indices for \mathcal{R}_0 against model parameters show that the local sensitivity results are not robust: the order of the most important parameters for \mathcal{R}_0 from the local sensitivity analysis not match those from the global sensitivity analysis.

506 5 Numerical simulations and discussions

507 In the previous model [26], we have shown that the use of a vaccine with efficacy of about 60%,
 508 was to be accompanied by other control measurements such as means of personal protection
 509 (Information in relation to the damage caused by these diseases, spanning wearing clothes
 510 during hours of vector activity, use of repellents), vector control (combining the use of adulticide
 511 to kill adult vectors, chemical control with use of larvicide to kill the eggs and larvae, and
 512 mechanical control to reduce the number of breeding sites at least near inhabited areas) [15].
 513 Here, we investigate and compare numerical results, with the different scenario. We use the

514 following initial state variables $S_h(0) = 700$, $V_h(0) = 10$, $E_h(0) = 220$, $I_h(0) = 100$, $R_h(0) = 60$,
515 $S_v(0) = 3000$, $E_v(0) = 400$, $I_v(0) = 120$, $E(0) = 10000$, $L = 5000$, $P = 3000$.

516 **5.1 Strategy A: Vaccination combined with individual protection** 517 **only**

518 In this strategy, we consider the model (2) without vector control. we set $\alpha_2 = 1$ and $c_m = \eta_1 =$
519 $\eta_2 = 0$ and vary the parameter related to individual protection, namely α_1 , between 0 and 0.8.
520 The values of other parameters are given in Table 4. Figure 8 shows that the increase of the
521 individual protection level, permit to reduce the total number of infected humans, and the total
522 number of infected vectors, but has no impact on the populations of eggs and larvae. However,
523 from this figure, it is clear that, this reduction is significant if the level of protection must turn
524 around 80% at least, and this, over a long period. Thus, continuous education campaigns of
525 people, on how to protect themselves individually, are important in the fight against the spread
526 of arboviral diseases.

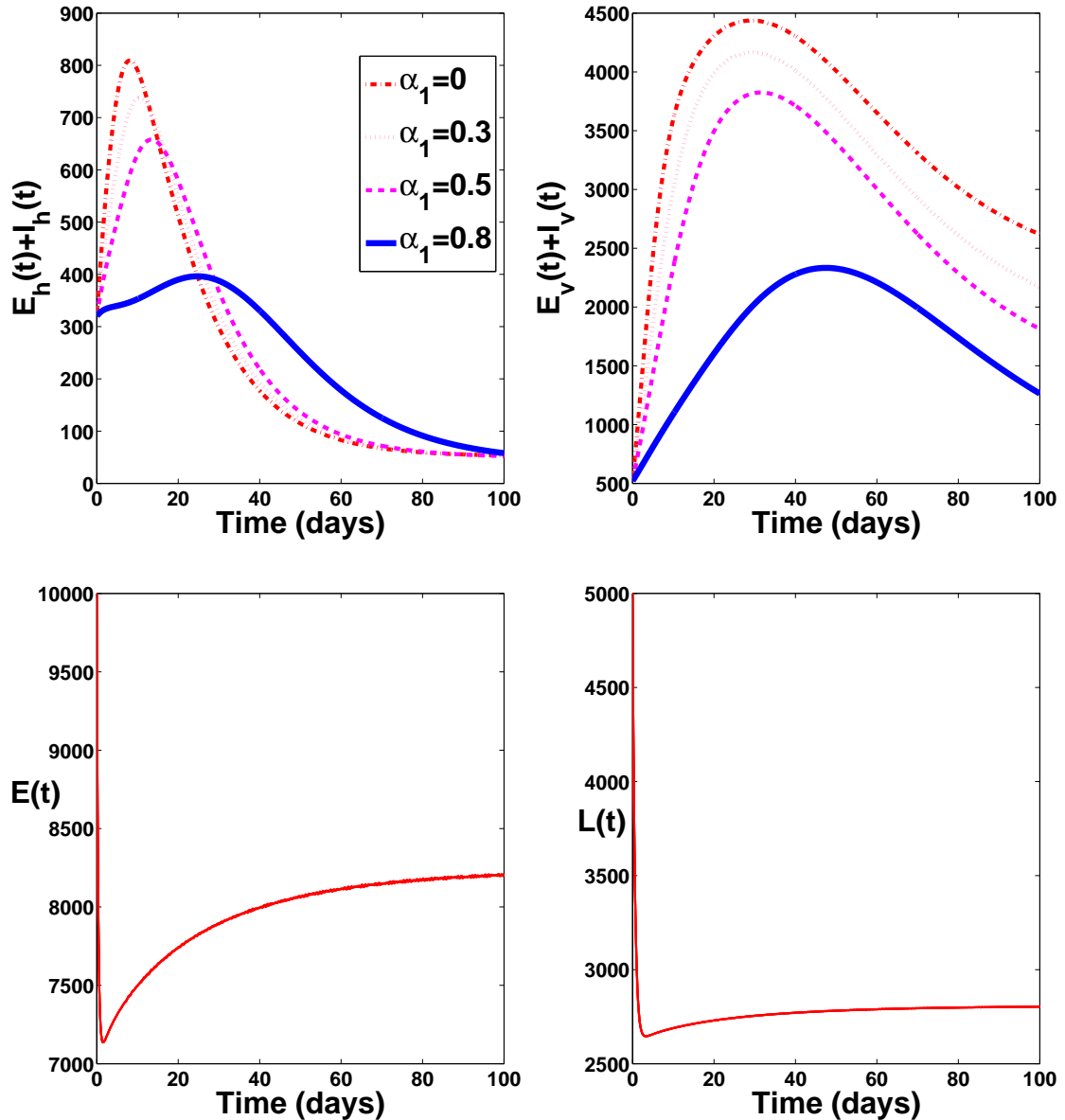


Figure 8: Simulations results showing how the total number of infected humans and the total number of infected vectors decrease when the individual protection increase. All others parameters values are in Table 4.

5.2 Strategy B: Vaccination combined with adulticide

Nowadays, Deltamethrin is the most used insecticide for impregnation of bednets, because it is a highly effective compound on mosquitoes at of very low doses [53]. However, when sprayed in an open environment, Deltamethrin seems to be effective only during a couple of hours [15, 54, 55]. Also, its use over a long period and continuously, leads to strong resistance of the wild populations of *Aedes aegypti*, for example [53]. The mortality of the mosquitoes after spraying varied between 20% and 80%. To be more realistic, we will consider the technique called "pulse control" (the control is not continuous in time order is effective only one day every T days) [15]. To this aims, we consider that spraying is carried out once a week, and this, for 100 days. We set $\alpha_1 = \eta_1 = \eta_2 = 0$ and $\alpha_2 = 1$.

537 Simulation result on figure 9 show that a mortality rate induced by the use of larvicide,
 538 c_m , greater than 60% has a significant impact on the decrease of the total number of infected
 539 humans and vectors, and on the decrease of eggs and larvae.

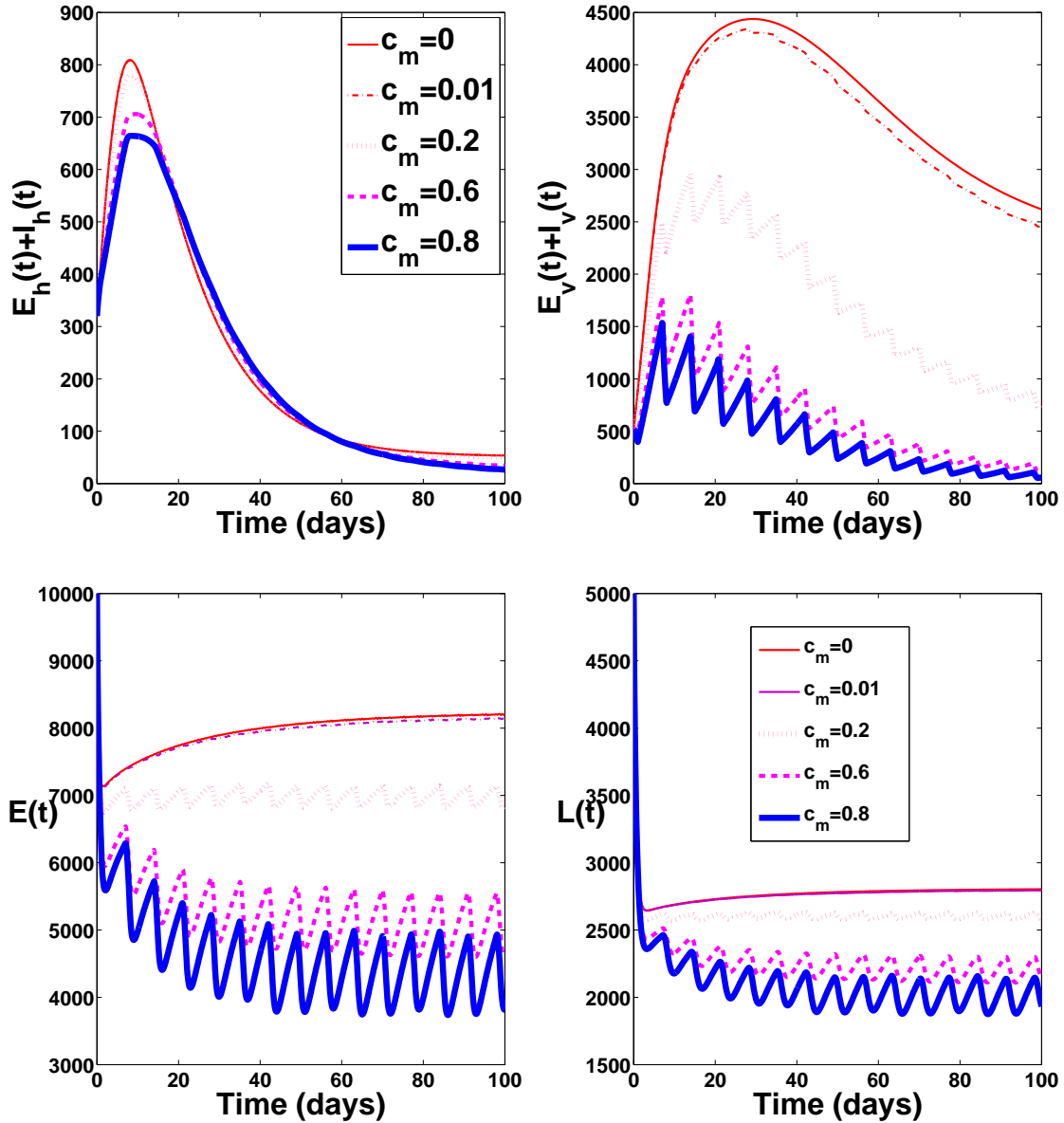


Figure 9: Simulations results showing how the total number of infected vectors, eggs and larvae populations decrease when the adulticide control parameter c_m increase. All others parameters values are in Table 4.

540 5.3 Strategy C: Vaccination combined with larvicide

541 Since the efficacy and the duration of a larvicide (*Bti*=*Bacillus thuringiensis var. israelensis*)
 542 strongly depend on several factors like water quality, exposure, and even the type of breeding
 543 sites, we thus consider, to be more realistic, that the duration can vary between a couple of
 544 days and two weeks [15, 56]. We consider that the larvicide spraying happens once every 15
 545 days, and this, on a period of 100 days. We set $\alpha_1 = c_m = 0$ and $\alpha_2 = 1$.

546 The figure 10 shows that the use of larvicide has no significant impact on the decrease of
 547 total number of infected humans and vectors, as well as on the number of eggs and larvae. This
 548 can be justified by the fact that the use of conventional larvicides requires certain constraints
 549 on their use: they can not be used continuously, their duration of action decreases with time. In
 550 addition, eggs of certain populations of vectors such as *Aedes albopictus*, come into prolonged
 551 hibernation when conditions in the breeding sites are not conducive to their good growth (this
 552 is justified by the control rate value $\eta_1 = 0.001$). Also, the pupae do not consume anything,
 553 until reaching the mature stage.

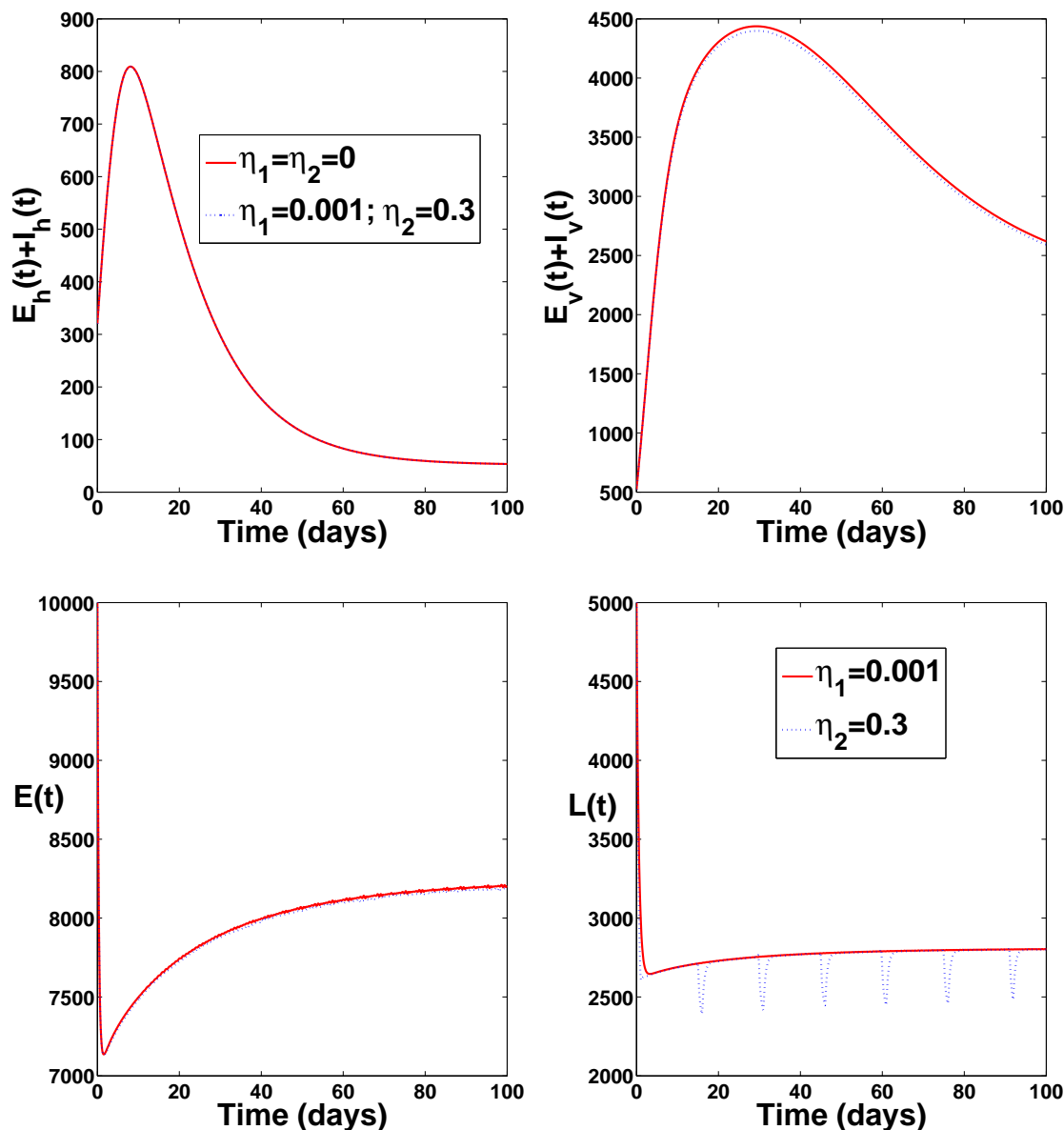


Figure 10: Simulations results showing how the total number of infected humans, the total number of infected vectors, and the eggs and larvae populations decrease with the larvicide control associated parameters η_1 and η_2 . All others parameters values are in Table 4.

554 **5.4 Strategy D: Vaccination combined with mechanical control**

555 The effectiveness of this type of control depends largely on awareness campaigns of local people
 556 in the sense that, to reduce the proliferation of vectors, people should always destroy, and
 557 systematically, potential breeding sites. Thus, we consider that this type of control can be
 558 achieved by local populations, and this, every daily. We set $\alpha_1 = c_m = 0 = \eta_1 = \eta_2$.

559 The figure 11 shows that this type of control is appropriate in the fight against the prolif-
 560 eration of vectors. This can only be possible by that by the multiplication of local populations
 561 awareness campaigns.

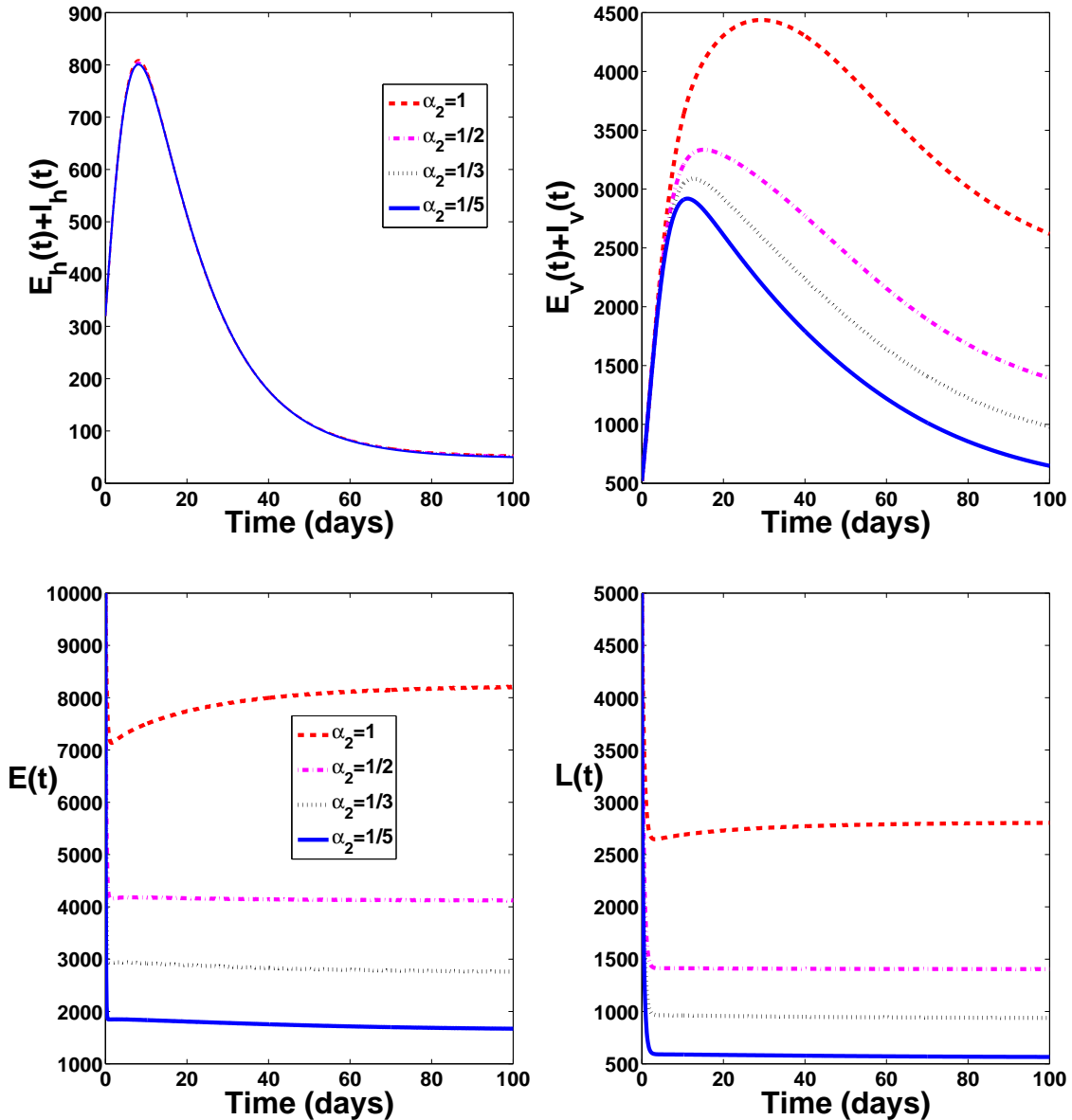


Figure 11: Simulations results showing how the total number of infected vectors, eggs and larvae populations decrease with the mechanical control associated parameter α_2 . All others parameters values are in Table 4.

562 **5.5 Strategy E: Combining vaccination, individual protection and**
 563 **adulticide**

564 In this strategy, we consider the model (2) without larvicide and mechanical control. we set
 565 $\alpha_2 = 1$ and $\eta_1 = \eta_2 = 0$ and vary the parameter related to individual protection and the
 566 use of adulticide, namely α_1 and c_m , respectively, between 0 and 0.8. The values of other
 567 parameters are given in Table 4. Figure 12 shows that the use of the combination of these
 568 controls decreases significantly the total number of infected humans, infected vectors as well as
 569 the number of eggs and larvae, when its associated rates, namely α_1 and c_m , are greater than
 570 0.3 and 0.2, respectively.

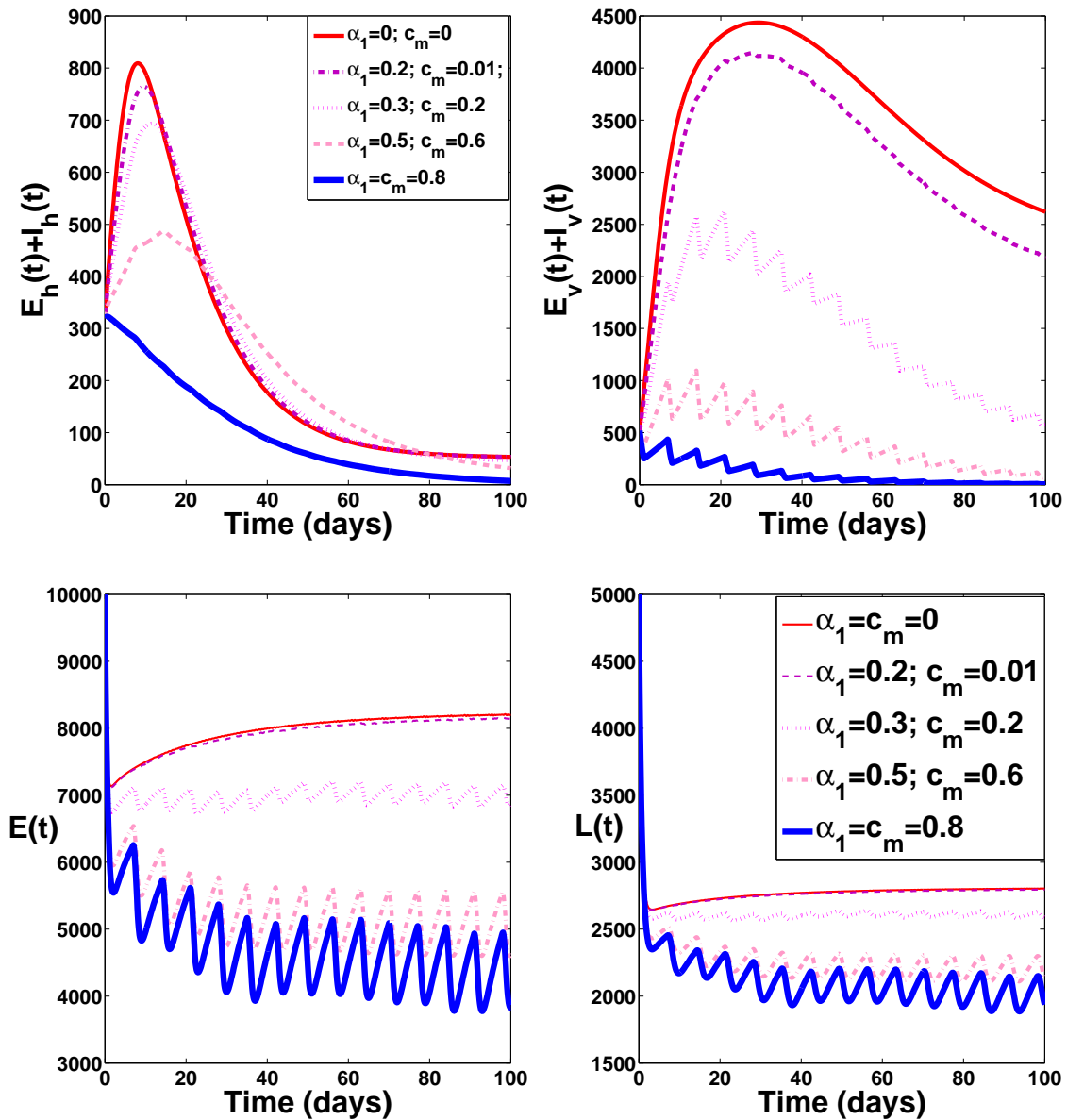


Figure 12: Simulations results showing the advantage that we have to combine vaccination, individual protection and adulticide.

571 **5.6 Strategy F: Combining vaccination, individual protection and**
 572 **mechanical control**

573 Like for strategy E, the simulations results on figure 13 show that the combined use of these
 574 three types of controls has a positive impact in the vector control.

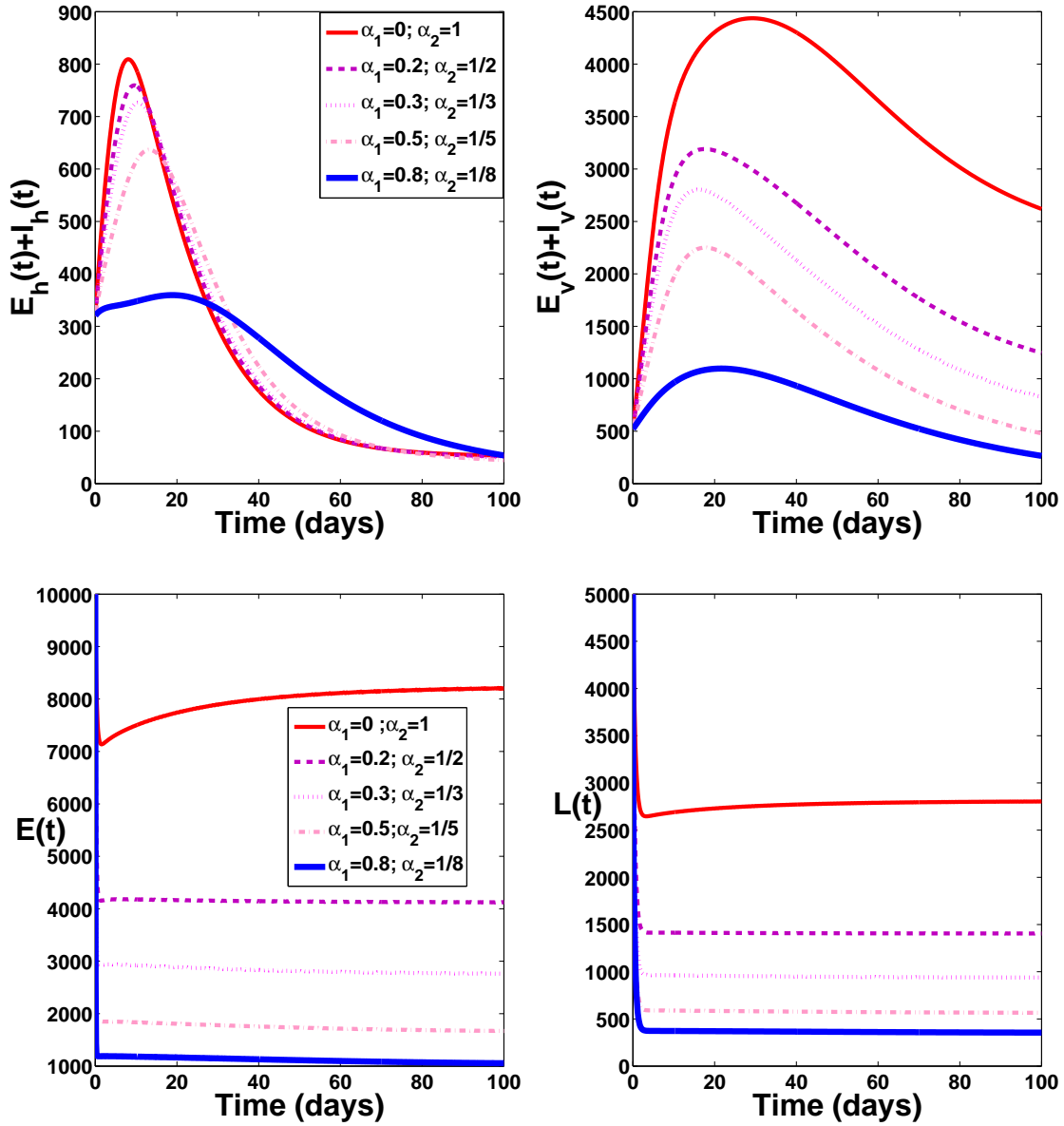


Figure 13: Simulations results showing the advantage that we have to combine vaccination, individual protection and mechanical control.

575 **6 Conclusion**

576 In this paper, we derived and analysed a deterministic model for the transmission of arboviral
 577 diseases with non linear form of infection and complete stage structured model for vectors,
 578 which takes into account a vaccination with waning immunity, individual protection and vector
 579 control strategies.

580 We begin by calculated the net reproductive number \mathcal{N} and the basic reproduction number,
581 \mathcal{R}_0 , and investigated the existence and stability of equilibria. The stability analysis reveals that
582 for $\mathcal{N} \leq 1$, the trivial equilibrium is globally asymptotically stable. When $\mathcal{N} > 1$ and $\mathcal{R}_0 <$
583 1 , the disease-free equilibrium is locally asymptotically stable. Under certain condition, the
584 disease-free equilibrium is also globally asymptotically stable. We found that the model exhibits
585 backward bifurcation. The epidemiological implication of this phenomenon is that for effective
586 eradication and control of diseases, \mathcal{R}_0 should be less than a critical values less than one. Thus,
587 we proved, that the disease-induced death is the principal cause of the backward bifurcation
588 phenomenon, in the full model and the corresponding model without vaccination. However, the
589 substitution of standard incidence with mass action incidence removes the backward bifurcation
590 phenomenon.

591 We proved that the model admits at least one endemic equilibrium, and only one endemic
592 equilibrium point in the model without disease-induced death, and in the model with mass
593 action incidences, whenever the basic reproduction number is great than unity.

594 Using parameters value of Chikungunya and Dengue fever, we calculated the sensitivity
595 indices of the basic reproduction number, \mathcal{R}_0 , to the parameters in the model using both
596 local and global methods. Local sensitivity analysis showed that the model system is most
597 sensitive to a , the average number of mosquitoes bites, followed by μ_v , the natural mortality
598 rate of vectors. Considering that all input parameters vary simultaneously, we use the Latin
599 Hypercube Sampling (LHS) to estimate statistically the mean value of the basic reproduction
600 number. The result showed that the model is in an endemic state, since the mean of \mathcal{R}_0 is
601 2.0642, which is greater than unity. Then, using global sensitivity analysis, we computed the
602 Partial Rank Correlation Coefficients between \mathcal{R}_0 and each parameter of the model. Unlike
603 the local sensitivity analysis, the global analysis showed that the parameters α_1 , the human
604 protection rate, has the highest influence on \mathcal{R}_0 . The other parameter with an important
605 effect are α_2 , the efficacy of the mechanical control, β_{hv} , the probability of transmission of
606 infection from an infectious vector to a susceptible human, β_{vh} , the probability of transmission
607 of infection from an infectious human to a susceptible vector, and θ , the maturation rate from
608 pupae to adult vectors. This showed that the order of the most important parameters for \mathcal{R}_0
609 from the local sensitivity analysis not match those from the global sensitivity analysis. So, the
610 local sensitivity results are not robust.

611 To assess the impact of combination of different controls, we conduct several simulations,
612 using the called "pulse control" technique. According to the numerical results, we conclude that
613 the use of an imperfect vaccine with low efficiency combined with high individual protection and
614 good vector control strategy (reduction of breeding sites by local populations action, chemical
615 action using adulticides and larvicides), can effectively reduce the transmission of the pathogen
616 and the proliferation of vector populations. However, due to lack of resources to implement these
617 control mechanisms, developing countries should focus on the education of the local populations.
618 Because, unlike diseases such as malaria whose breeding sites of *Anopheles* mosquitoes are
619 known, those of arboviruses (old tires, flower pots, vases and other hollow...) are smaller and
620 unknown for many local populations, which favour the development of vectors.

621 Thus, pending the development of a high efficacy vaccine and long-acting, individual pro-
622 tection and the various vector control methods are effective ways to combat the arboviruses, for
623 developing countries. In addition, the realization of the combination of these controls may be
624 too expensive, because it means that, for constant controls, we must keep them at levels high,
625 and this, for a long time. Thus it is important to know what happens when, instead of the
626 constant controls, we use time dependent controls, in optimal control theory. This represents
627 a perspective of this work.

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A Useful result.

We use the following result, which is the characterization of Metzler matrices, to compute the threshold \mathcal{R}_c , obtained at Eq. (10).

Lemma 4 ([35]). *Let M be a square Metzler matrix written in block form $\begin{pmatrix} A & B \\ C & D \end{pmatrix}$ with A and D square matrices. M is Metzler stable if and only if matrices A and $D - CA^{-1}B$ are Metzler stable.*

B Proof of Theorem 1.

The Jacobian matrix of f at the Trivial equilibrium is given by

$$Df(\mathcal{E}_0) = \begin{pmatrix} Df_1 & Df_2 \\ Df_3 & Df_4 \end{pmatrix}. \quad (26)$$

where

$$Df_1 = \begin{pmatrix} -k_1 & \omega & 0 & 0 & 0 & 0 \\ \xi & -k_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & -k_3 & 0 & 0 & 0 \\ 0 & 0 & \gamma_h & -k_4 & 0 & 0 \\ 0 & 0 & 0 & \sigma & -\mu_h & 0 \\ 0 & 0 & 0 & 0 & 0 & -k_8 \end{pmatrix}, \quad Df_3 = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \mu_b \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

$$Df_2 = \begin{pmatrix} -\frac{a(1-\alpha_1)\beta_{hv}\eta_v S_h^0}{N_h^0} & -\frac{a(1-\alpha_1)\beta_{hv} S_h^0}{N_h^0} & 0 & 0 & 0 \\ -\frac{a(1-\alpha_1)\beta_{hv}\pi\eta_v V_h^0}{N_h^0} & -\frac{a(1-\alpha_1)\beta_{hv}\pi V_h^0}{N_h^0} & 0 & 0 & 0 \\ \frac{a(1-\alpha_1)\beta_{hv}\eta_v H^0}{N_h^0} & \frac{a(1-\alpha_1)\beta_{hv} H^0}{N_h^0} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \theta \end{pmatrix},$$

$$Df_4 = \begin{pmatrix} -k_9 & 0 & 0 & 0 & 0 \\ \gamma_v & -k_8 & 0 & 0 & 0 \\ \mu_b & \mu_b & -k_5 & 0 & 0 \\ 0 & 0 & s & -k_6 & 0 \\ 0 & 0 & 0 & l & -k_7 \end{pmatrix}, \quad \text{and } H^0 = S_h^0 + \pi V_h^0.$$

The characteristic polynomial of $Df(\mathcal{E}_0)$ is given by:

$$P(\lambda) = -(\lambda + k_3)(\lambda + k_4)(\lambda + k_8)(\lambda + k_9)(\lambda + \mu_h)\phi_1(\lambda)\phi_2(\lambda)$$

where

$\phi_1(\lambda) = \lambda^2 + (k_2 + k_1)\lambda + \mu_h(k_2 + \xi)$ and $\phi_2(\lambda) = \lambda^4 + A_1\lambda^3 + A_2\lambda^2 + A_3\lambda + A_4$. we have set

$$A_1 = k_5 + k_6 + k_7 + k_8, \quad A_2 = k_8(k_5 + k_6 + k_7) + k_7(k_5 + k_6) + k_5k_6, \\ A_3 = k_5k_6k_7 + k_8(k_5k_6 + k_7(k_5 + k_6)), \quad A_4 = k_5k_6k_7k_8(1 - \mathcal{N}).$$

646 The roots of $P(\lambda)$ are $\lambda_1 = -\mu_h$, $\lambda_1 = -k_1$, $\lambda_2 = -k_3$, $\lambda_3 = -k_4$, $\lambda_4 = -k_8$, $\lambda_4 = -k_9$, and
647 the others roots are the roots of $\phi_1(\lambda)$ and $\phi_2(\lambda)$. The real part of roots of $\phi_1(\lambda)$ are negative.
648 Since $\mathcal{N} < 1$, it is clear that all coefficients of $\phi_2(\lambda)$ are always positive. Now we just have
649 to verify that the Routh–Hurwitz criterion holds for polynomial $\phi_2(\lambda)$. To this aim, setting

$$650 \quad H_1 = A_1, \quad H_2 = \begin{vmatrix} A_1 & 1 \\ A_3 & A_2 \end{vmatrix}, \quad H_3 = \begin{vmatrix} A_1 & 1 & 0 \\ A_3 & A_2 & A_1 \\ 0 & A_4 & A_3 \end{vmatrix}, \quad H_4 = \begin{vmatrix} A_1 & 1 & 0 & 0 \\ A_3 & A_2 & A_1 & 1 \\ 0 & A_4 & A_3 & A_2 \\ 0 & 0 & 0 & A_4 \end{vmatrix} = A_4 H_3.$$

651 The Routh–Hurwitz criterion of stability of the trivial equilibrium \mathcal{E}^0 is given by

$$\begin{cases} H_1 > 0 \\ H_2 > 0 \\ H_3 > 0 \\ H_4 > 0 \end{cases} \Leftrightarrow \begin{cases} H_1 > 0 \\ H_2 > 0 \\ H_3 > 0 \\ A_4 > 0 \end{cases} \quad (27)$$

652 We have $H_1 = A_1 = k_5 + k_6 + k_7 + k_8 > 0$,

$$\begin{aligned} H_2 &= A_1 A_2 - A_3 \\ &= (k_7 + k_6 + k_5) k_8^2 + (k_7^2 + (2k_6 + 2k_5) k_7 + k_6^2 + 2k_5 k_6 + k_5^2) k_8 \\ &\quad + (k_6 + k_5) k_7^2 + (k_6^2 + 2k_5 k_6 + k_5^2) k_7 + k_5 k_6^2 + k_5^2 k_6 \end{aligned}$$

653

$$\begin{aligned} H_3 &= A_1 A_2 A_3 - A_1^2 A_4 - A_3^2 \\ &= (k_6 + k_5) (k_7^2 + (k_6 + k_5) k_7 + k_5 k_6) k_8^3 \\ &\quad + (\mu_b l s \theta + (k_6 + k_5) k_7^3 + 2(k_6 + k_5)^2 k_7^2 + (k_6^3 + 4k_5 k_6^2 + 4k_5^2 k_6 + k_5^3) k_7 + k_5 k_6^3 + 2k_5^2 k_6^2 + k_5^3 k_6) k_8^2 \\ &\quad + [(2k_7 + 2k_6 + 2k_5) \mu_b l s \theta + (k_6^2 + 2k_5 k_6 + k_5^2) k_7^3 + (k_6^3 + 4k_5 k_6^2 + 4k_5^2 k_6 + k_5^3) k_7^2 \\ &\quad + (2k_5 k_6^3 + 4k_5^2 k_6^2 + 2k_5^3 k_6) k_7 + k_5^2 k_6^3 + k_5^3 k_6^2] k_8 + (k_7^2 + (2k_6 + 2k_5) k_7 + k_6^2 + 2k_5 k_6 + k_5^2) \mu_b l s \theta \\ &\quad + (k_5 k_6^2 + k_5^2 k_6) k_7^3 + (k_5 k_6^3 + 2k_5^2 k_6^2 + k_5^3 k_6) k_7^2 + (k_5^2 k_6^3 + k_5^3 k_6^2) k_7 \end{aligned}$$

654 We always have $H_1 > 0$, $H_2 > 0$, $H_3 > 0$ and $H_4 > 0$ if $\mathcal{N} < 1$. Thus, the trivial equilibrium
655 \mathcal{E}_0 is locally asymptotically stable whenever $\mathcal{N} < 1$.

656 We assume the net reproductive number $\mathcal{N} > 1$. Following the procedure and the notation
657 in [33], we may obtain the basic reproduction number \mathcal{R}_0 as the dominant eigenvalue of the
658 *next-generation matrix* [32, 33]. Observe that model (2) has four infected populations, namely
659 E_h, I_h, E_v, I_v . It follows that the matrices F and V defined in [33], which take into account
660 the new infection terms and remaining transfer terms, respectively, are given by

$$661 \quad F = \begin{pmatrix} 0 & 0 & \frac{\tau \beta_{hv} \eta_v H^0}{N_h^0} & \frac{\tau \beta_{hv} H^0}{N_h^0} \\ 0 & 0 & 0 & 0 \\ \frac{\tau \beta_{vh} \eta_v S_v^0}{N_h^0} & \frac{\tau \beta_{vh} S_v^0}{N_h^0} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad \text{and } V = \begin{pmatrix} k_3 & 0 & 0 & 0 \\ -\gamma_h & k_4 & 0 & 0 \\ 0 & 0 & k_9 & 0 \\ 0 & 0 & -\gamma_v & k_8 \end{pmatrix}, \quad \text{with}$$

662 $\tau = a(1 - \alpha_1)$. The dominant eigenvalue of the next-generation matrix FV^{-1} is given by (7).
663 The local stability of the disease-free equilibrium \mathcal{E}_1 is a direct consequence of Theorem 2 of
664 [33]. This ends the proof.

665 C Proof of Theorem 2.

666 Setting $Y = X - TE$ with $X = (S_h, V_h, E_h, I_h, R_h, S_v, E_v, I_v, E, L, P)^T$, $H^0 = (S_h^0 + \pi V_h^0)$,
667 $A_{99} = \left(k_5 + \mu_b \frac{S_v + E_v + I_v}{K_E} \right)$, and $A_{10} = \left(k_6 + s \frac{E}{K_L} \right)$. we can rewrite (2) in the following

$$\frac{dY}{dt} = \mathcal{B}(Y)Y \quad (28)$$

669 where $\mathcal{B}(Y) = \begin{pmatrix} A(Y) & B(Y) \\ C(Y) & D(Y) \end{pmatrix}$, with

$$670 \quad A(Y) = \begin{pmatrix} -(\lambda_h^c + k_1) & \omega & 0 & 0 & 0 & 0 \\ \xi & -(\pi\lambda_h^c + k_2) & 0 & 0 & 0 & 0 \\ \lambda_h^c & \pi\lambda_h^c & -k_3 & 0 & 0 & 0 \\ 0 & 0 & \gamma_h & -k_4 & 0 & 0 \\ 0 & 0 & 0 & \sigma & -\mu_h & 0 \end{pmatrix},$$

$$671 \quad B(Y) = \begin{pmatrix} -\frac{a(1-\alpha_1)\beta_{hv}\eta_v S_h^0}{N_h} & -\frac{a(1-\alpha_1)\beta_{hv} S_h^0}{N_h} & 0 & 0 & 0 \\ \frac{a(1-\alpha_1)\beta_{hv}\eta_v \pi V_h^0}{N_h} & \frac{a(1-\alpha_1)\beta_{hv}\pi V_h^0}{N_h} & 0 & 0 & 0 \\ \frac{a(1-\alpha_1)\beta_{hv}\eta_v H^0}{N_h} & \frac{a(1-\alpha_1)\beta_{hv} H^0}{N_h} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

$$672 \quad C(Y) = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & -(\lambda_v^c + k_8) \\ 0 & 0 & 0 & 0 & 0 & \lambda_v^c \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \mu_b \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \quad D(Y) = \begin{pmatrix} 0 & 0 & 0 & 0 & \theta \\ -k_9 & 0 & 0 & 0 & 0 \\ \gamma_v & -k_8 & 0 & 0 & 0 \\ \mu_b & \mu_b & -A_{99} & 0 & 0 \\ 0 & 0 & s & -A_{10} & 0 \\ 0 & 0 & 0 & l & -k_7 \end{pmatrix}.$$

673 It is clear that $Y = (0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0)$ is the only equilibrium. Then it suffices to con-
 674 sider the following Lyapunov function $\mathcal{L}(Y) = \langle g, Y \rangle$ were $g = \left(1, 1, 1, 1, 1, 1, 1, \frac{k_8}{\mu_b}, \frac{k_5 k_8}{\mu_b s}, \frac{k_5 k_6 k_8}{\mu_b s l}\right)$.
 675 Straightforward computations lead that

$$\begin{aligned} \dot{\mathcal{L}}(Y) &= \langle g, \dot{Y} \rangle \stackrel{\text{def}}{=} \langle g, \mathcal{B}(Y)Y \rangle \\ &= -\mu_h Y_1 - \mu_h Y_2 - \mu_h Y_3 - (\mu_h + \delta) Y_4 - \mu_h Y_5 \\ &\quad - \frac{k_8}{K_E} (Y_6 + Y_7 + Y_8) - \frac{k_5 k_8}{\mu_b K_L} Y_9 Y_{10} + \theta \left(1 - \frac{1}{\mathcal{N}}\right) Y_{11} \end{aligned}$$

676 We have $\dot{\mathcal{L}}(Y) < 0$ if $\mathcal{N} \leq 1$ and $\dot{\mathcal{L}}(Y) = 0$ if $Y_i = 0$, $i = 1, 2, \dots, 11$ (i.e $S_h = S_h^0$, $V_h = V_h^0$
 677 and $E_h = I_h = R_h = S_v = E_v = I_v = E = L = P = 0$). Moreover, the maximal invariant
 678 set contained in $\{\mathcal{L} | \dot{\mathcal{L}}(Y) = 0\}$ is $(0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0)$. Thus, from Lyapunov theory, we
 679 deduce that $(0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0)$ and thus, \mathcal{E}_0 , is GAS if and only if $\mathcal{N} \leq 1$.

680 D Proof of Theorem 5.

681 In order to determine the existence of endemic equilibria, i. e. equilibria with all positive
 682 components, say

$$\mathcal{E}^{**} = (S_h^*, V_h^*, E_h^*, I_h^*, R_h^*, S_v^*, E_v^*, I_v^*, E, L, P),$$

683 we have to look for the solution of the algebraic system of equations obtained by equating the
 684 right sides of system (2) to zero. In this way we consider two case:

685 **(i) Special case: Absence of disease-induced death in human** ($\delta = 0$) Note that in the
686 absence of disease-induced death in human population, we have $N_h^* = N_h^0 = \Lambda_h / \mu_h$. Let

$$\lambda_h^{c,*} = \frac{a(1 - \alpha_1)\beta_{hv}(\eta_v E_v^* + I_v^*)}{N_h^*}, \quad \lambda_v^{c,*} = \frac{a(1 - \alpha_1)\beta_{vh}(\eta_h E_h^* + I_h^*)}{N_h^*} \quad (29)$$

687 be the forces of infection of humans and vectors at steady state, respectively. Solving the
688 equations in (2) at steady state gives

$$\begin{aligned} S_h^* &= \frac{\Lambda_h(\pi\lambda_h^{c,*} + k_2)}{\mu_h(k_2 + \xi) + \lambda_h^{c,*}(\pi\lambda_h^{c,*} + \pi k_1 + k_2)}, & V_h^* &= \frac{\xi S_h^*}{(\pi\lambda_h^{c,*} + k_2)}, \\ E_h^* &= \frac{\lambda_h^{c,*}(S_h^* + \pi V_h^*)}{k_3}, & I_h^* &= \frac{\gamma_h \lambda_h^{c,*}(S_h^* + \pi V_h^*)}{k_3 k_4}, & R_h^* &= \frac{\sigma \gamma_h \lambda_h^{c,*}(S_h^* + \pi V_h^*)}{\mu_h k_3 k_4}, \end{aligned} \quad (30)$$

689 and

$$\begin{aligned} S_v^* &= \frac{\theta P}{(\lambda_v^{c,*} + k_8)}, & E_v^* &= \frac{\theta P \lambda_v^{c,*}}{k_9(\lambda_v^{c,*} + k_8)}, & I_v^* &= \frac{\gamma_v \theta P \lambda_v^{c,*}}{k_8 k_9(\lambda_v^{c,*} + k_8)}, \\ E &= \frac{\mu_b \theta K_E P}{(k_5 k_8 K_E + \mu_b \theta P)}, & L &= \frac{\mu_b \theta s K_E K_L P}{k_6 K_L (k_5 k_8 K_E + \mu_b \theta P) + s \mu_b \theta K_E P}, \end{aligned} \quad (31)$$

690 where P is solution of the following equation

$$f(P) = -k_7 P [\mu_b \theta (s K_E + k_6 K_L) P + k_5 k_6 k_8 K_E K_L (\mathcal{N} - 1)] = 0 \quad (32)$$

691 A direct resolution of the above equation give $P = 0$ or $P = \frac{k_5 k_6 k_8 K_E K_L (\mathcal{N} - 1)}{\mu_b \theta (s K_E + k_6 K_L)}$.

692 Note that $P = 0$ corresponds to the trivial equilibrium \mathcal{E}_0 . Now we consider $P > 0$ i.e.
693 $\mathcal{N} > 1$. Replacing (30) and (31) in (29) give

$$\lambda_h^{c,*} = \frac{a(1 - \alpha_1)\beta_{hv}\mu_h}{\Lambda_h} \left(\eta_v \frac{\theta P \lambda_v^*}{k_9(\lambda_v^* + k_8)} + \frac{\gamma_v \theta P \lambda_v^*}{k_8 k_9(\lambda_v^* + k_8)} \right) \quad (33)$$

694

$$\lambda_v^{c,*} = \frac{a(1 - \alpha_1)\beta_{vh}\mu_h}{\Lambda_h} \left(\eta_h \frac{\lambda_h^*(S_h^* + \pi V_h^*)}{k_3} + \frac{\gamma_h \lambda_h^*(S_h^* + \pi V_h^*)}{k_3 k_4} \right) \quad (34)$$

695 Substituting (34) in (33) give

$$(k_6 K_L + s K_E) \lambda_h^* [a_2 (\lambda_h^*)^2 + a_1 \lambda_h^* + a_0] = 0 \quad (35)$$

696 where a_2 , a_1 and a_0 are given by

$$\begin{aligned} R_b &= \frac{(\pi\xi + k_2)}{\pi(\xi + k_2)} \left(\frac{(k_1\pi + k_2)}{\mu_h} + \frac{a(1 - \alpha_1)\beta_{vh}(\gamma_h + k_4\eta_h)(\pi\xi + k_2)}{k_3 k_4 k_8} \right), \\ a_2 &= (a(1 - \alpha_1)\beta_{vh}\mu_h(\gamma_h + k_4\eta_h) + k_3 k_4 k_8) k_9 \mu_b \Lambda_h \pi, \\ a_1 &= \frac{k_3 k_4 k_8 k_9 \mu_b \Lambda_h (\xi + k_2) \mu_h \pi}{(\pi\xi + k_2)} (R_b - R_1), \\ a_0 &= \mu_h k_3 k_4 k_8 k_9 \mu_b \Lambda_h (\xi + k_2) (1 - R_1). \end{aligned} \quad (36)$$

697 The trivial solution $\lambda_h^* = 0$ of (35) corresponds to the disease-free equilibrium \mathcal{E}_1 . Now, we
698 just look the equilibria when $\lambda_h^* > 0$. Note that coefficient a_2 is always positive and a_0 is less
699 (resp. greater) than unity if and only if $R_1 > 1$ (resp. $R_1 < 1$). Thus model system (2),
700 in absence of disease-induced death in human population ($\delta = 0$), admits only one endemic
701 equilibrium whenever $\mathcal{R}_0 > 1$. Since the sign of coefficient a_1 depend of the value of parameter,

702 we investigate the possibility of occurrence of backward bifurcation phenomenon when $\mathcal{R}_0 < 1$.
 703 Furthermore, consider the inequality

$$R_1 \leq R_b. \quad (37)$$

704 Since a_2 is always positive and a_0 is always positive whenever $\mathcal{R}_0 < 1$, then, the occurrence
 705 of backward bifurcation phenomenon depend of the sign of coefficient a_1 . The coefficient a_1 is
 706 always positive if and only if condition (37) holds (i.e $R_1 < R_b$). It follows that the disease-free
 707 equilibrium is the unique equilibrium when $\mathcal{N} > 1$ and $\mathcal{R}_0 < 1$. Now if $R_b < R_1 < 1$, then in
 708 addition to the DFE \mathcal{E}_1 , there exists two endemic equilibria whenever $\Delta = a_1^2 - 4a_2a_0 > 0$. How-
 709 ever, $R_b < R_1 < 1 \Rightarrow R_b < 1 \Leftrightarrow \beta_{vh} < -\frac{[\pi^2\xi^2 + (\mu_h\pi^2 + (2\omega + \mu_h)\pi)\xi + (\omega + \mu_h)^2]k_3k_4k_8}{a(1 - \alpha_1)\mu_h(\pi\xi + k_2)^2(\gamma_h + k_4\eta_h)} <$
 710 0. Since all parameter of model (2) are nonnegative, we conclude that the condition $R_b < R_1 < 1$
 711 does not hold. And thus, the backward bifurcation never occurs in the absence of disease-
 712 induced death in human.

713 (ii) Presence of disease induced death in human ($\delta \neq 0$)

714 In this case, we have $N_h^* = \frac{\Lambda_h - \delta I_h^*}{\mu_h}$. Applying the same procedure as case (i), we obtain that
 715 λ_h^* at steady state is solution of the following equation

$$f(\lambda_h^*) = \lambda_h^* [c_4(\lambda_h^*)^4 + c_3(\lambda_h^*)^3 + c_2(\lambda_h^*)^2 + c_1\lambda_h^* + c_0] = 0, \quad (38)$$

716 where

$$c_4 = -\pi^2 k_9 K_{12} \mu_b \Lambda_h (k_3 k_4 - \delta \gamma_h) (k_{10} a \mu_h (1 - \alpha_1) \beta_{vh} + k_8 (k_3 k_4 - \delta \gamma_h)),$$

717

$$\begin{aligned} c_3 = & \pi(k_3 k_4 k_5 k_6 k_{10} k_{11} a^2 \mu_h^2 (1 - \alpha_1)^2 \beta_{hv} n \pi \beta_{vh} K_E K_L + 2k_9 k_{10} K_{12} a \mu_b \delta \Lambda_h \mu_h \gamma_h \pi (1 - \alpha_1) \beta_{vh} \xi \\ & - k_3 k_4 k_9 k_{10} K_{12} a \mu_b \Lambda_h \mu_h \pi (1 - \alpha_1) \beta_{vh} \xi - 2k_8 k_9 K_{12} \mu_b \delta^2 \Lambda_h \gamma_h^2 \pi \xi + 2k_3 k_4 k_8 k_9 K_{12} \mu_b \delta \Lambda_h \gamma_h \pi \xi \\ & - k_1 k_3 k_4 k_9 k_{10} K_{12} a \mu_b \Lambda_h \mu_h \pi (1 - \alpha_1) \beta_{vh} + 2k_2 k_9 k_{10} K_{12} a \mu_b \delta \Lambda_h \mu_h \gamma_h (1 - \alpha_1) \beta_{vh} \\ & - 2k_2 k_3 k_4 k_9 k_{10} K_{12} a \mu_b \Lambda_h \mu_h (1 - \alpha_1) \beta_{vh} + 2k_1 k_3 k_4 k_8 k_9 K_{12} \mu_b \delta \Lambda_h \gamma_h \pi - 2k_1 k_3^2 k_4^2 k_8 k_9 K_{12} \mu_b \Lambda_h \pi \\ & - 2k_2 k_8 k_9 K_{12} \mu_b \delta^2 \Lambda_h \gamma_h^2 + 4k_2 k_3 k_4 k_8 k_9 K_{12} \mu_b \delta \Lambda_h \gamma_h - 2k_2 k_3^2 k_4^2 k_8 k_9 K_{12} \mu_b \Lambda_h), \end{aligned}$$

718

$$\begin{aligned} c_2 = & k_3 k_4 k_5 k_6 k_{10} k_{11} a^2 \mu_h^2 (1 - \alpha_1)^2 \beta_{hv} n \pi^2 \beta_{vh} \xi K_E K_L \\ & + k_1 k_3 k_4 k_5 k_6 k_{10} k_{11} a^2 \mu_h^2 (1 - \alpha_1)^2 \beta_{hv} n \pi^2 \beta_{vh} K_E K_L \\ & + 2k_2 k_3 k_4 k_5 k_6 k_{10} k_{11} a^2 \mu_h^2 (1 - \alpha_1)^2 \beta_{hv} n \pi \beta_{vh} K_E K_L + k_9 k_{10} K_{12} a \mu_b \delta \Lambda_h \mu_h \gamma_h \pi^2 (1 - \alpha_1) \beta_{vh} \xi^2 \\ & - k_8 k_9 K_{12} \mu_b \delta^2 \Lambda_h \gamma_h^2 \pi^2 \xi^2 - k_1 k_3 k_4 k_9 k_{10} K_{12} a \mu_b \Lambda_h \mu_h \pi^2 (1 - \alpha_1) \beta_{vh} \xi \\ & + k_3 k_4 k_9 k_{10} K_{12} a \mu_b \Lambda_h \mu_h \omega \pi (1 - \alpha_1) \beta_{vh} \xi + 2k_2 k_9 k_{10} K_{12} a \mu_b \delta \Lambda_h \mu_h \gamma_h \pi (1 - \alpha_1) \beta_{vh} \xi \\ & - k_2 k_3 k_4 k_9 k_{10} K_{12} a \mu_b \Lambda_h \mu_h \pi (1 - \alpha_1) \beta_{vh} \xi + 2k_1 k_3 k_4 k_8 k_9 K_{12} \mu_b \delta \Lambda_h \gamma_h \pi^2 \xi \\ & - 2k_3 k_4 k_8 k_9 K_{12} \mu_b \delta \Lambda_h \gamma_h \omega \pi \xi + 2k_3^2 k_4^2 k_8 k_9 K_{12} \mu_b \Lambda_h \omega \pi \xi - 2k_2 k_8 k_9 K_{12} \mu_b \delta^2 \Lambda_h \gamma_h^2 \pi \xi \\ & + 2k_2 k_3 k_4 k_8 k_9 K_{12} \mu_b \delta \Lambda_h \gamma_h \pi \xi - 2k_1 k_2 k_3 k_4 k_9 k_{10} K_{12} a \mu_b \Lambda_h \mu_h (1 - \alpha_1) \pi \beta_{vh} \\ & + k_2^2 k_9 k_{10} K_{12} a \mu_b \delta \Lambda_h \mu_h \gamma_h (1 - \alpha_1) \beta_{vh} - k_2^2 k_3 k_4 k_9 k_{10} K_{12} a \mu_b \Lambda_h \mu_h (1 - \alpha_1) \beta_{vh} \\ & - k_1^2 k_3^2 k_4^2 k_8 k_9 K_{12} \mu_b \Lambda_h \pi^2 + 4k_1 k_2 k_3 k_4 k_8 k_9 K_{12} \mu_b \delta \Lambda_h \gamma_h \pi - 4k_1 k_2 k_3^2 k_4^2 k_8 k_9 K_{12} \mu_b \Lambda_h \pi \\ & - k_2^2 k_8 k_9 K_{12} \mu_b \delta^2 \Lambda_h \gamma_h^2 + 2k_2^2 k_3 k_4 k_8 k_9 K_{12} \mu_b \delta \Lambda_h \gamma_h - k_2^2 k_3^2 k_4^2 k_8 k_9 K_{12} \mu_b \Lambda_h, \end{aligned}$$

719

$$\begin{aligned} c_1 = & ((k_1 k_3 k_4 k_5 k_6 k_{10} k_{11} a^2 \mu_h^2 (1 - \alpha_1) \beta_{hv} n \pi^2 + k_3 k_4 k_5 k_6 k_{10} k_{11} a^2 \mu_h^2 (1 - \alpha_1)^2 \beta_{hv} n (k_2 - \omega) \pi) \beta_{vh} \xi \\ & + (2k_1 k_2 k_3 k_4 k_5 k_6 k_{10} k_{11} a^2 \mu_h^2 (1 - \alpha_1) \beta_{hv} n \pi + k_2^2 k_3 k_4 k_5 k_6 k_{10} k_{11} a^2 \mu_h^2 (1 - \alpha_1) \beta_{hv} n) (1 - \alpha_1) \beta_{vh}) K_E K_L \\ & + (k_3 k_4 k_9 k_{10} K_{12} a \mu_b \Lambda_h \mu_h \omega \pi (1 - \alpha_1) \beta_{vh} - 2k_3 k_4 k_8 k_9 K_{12} \mu_b \delta \Lambda_h \gamma_h \omega \pi) \xi^2 \\ & + ((k_2 k_3 k_4 k_9 k_{10} K_{12} a \mu_b \Lambda_h \mu_h \omega - k_1 k_2 k_3 k_4 k_9 k_{10} K_{12} a \mu_b \Lambda_h \mu_h \pi) (1 - \alpha_1) \beta_{vh} \\ & + (2k_1 k_3^2 k_4^2 k_8 k_9 K_{12} \mu_b \Lambda_h \omega + 2k_1 k_2 k_3 k_4 k_8 k_9 K_{12} \mu_b \delta \Lambda_h \gamma_h) \pi \\ & + (2k_2 k_3^2 k_4^2 k_8 k_9 K_{12} \mu_b \Lambda_h - 2k_2 k_3 k_4 k_8 k_9 K_{12} \mu_b \delta \Lambda_h \gamma_h) \omega) \xi \\ & - k_1 k_2^2 k_3 k_4 k_9 k_{10} K_{12} a \mu_b \Lambda_h \mu_h (1 - \alpha_1) \beta_{vh} - 2k_1^2 k_2 k_3^2 k_4^2 k_8 k_9 K_{12} \mu_b \Lambda_h \pi \\ & + 2k_1 k_2^2 k_3 k_4 k_8 k_9 K_{12} \mu_b \delta \Lambda_h \gamma_h - 2k_1 k_2^2 k_3^2 k_4^2 k_8 k_9 K_{12} \mu_b \Lambda_h, \end{aligned}$$

$$c_0 = k_3^2 k_4^2 k_8 k_9 K_{12} \mu_b \Lambda_h \mu_h^2 (k_2 + \xi)^2 (R_0^2 - 1),$$

721 with $k_{10} = \gamma_h + \eta_h k_4$, $k_{11} = \gamma_v + \eta_v k_8$, $K_{12} = (sK_E + k_6 K_L)$ and $n = \mathcal{N} - 1$. Notes that c_4 is
 722 always negative and c_0 is positive (resp. negative) if \mathcal{R}_0 is greater (resp. less) than the unity.
 723 It follows, depending of the sign of coefficients c_3 , c_2 and c_1 , that the model system (2) admits
 724 at least one endemic equilibrium whenever $\mathcal{R}_0 > 1$ and the phenomenon of backward (resp.
 725 forward) bifurcation can occur when $\mathcal{R}_0 < 1$ (resp. $\mathcal{R}_0 > 1$). This ends the proof.

726 References

- 727 [1] A. Chippaux, Généralités sur arbovirus et arboviroses overview of arbovirus and arbovi-
 728 rosis, *Med. Maladies Infect.* 33 (2003) 377–384.
- 729 [2] N. Karabatsos, International Catalogue of Arboviruses, including certain other viruses of
 730 vertebrates, American Society of Tropical Medicine and Hygiene, 2001 update.
- 731 [3] D. J. Gubler, Human arbovirus infections worldwide, *Ann. N. Y. Acad. Sci.* 951 (2001)
 732 13–24.
- 733 [4] SANOFI PASTEUR, Dengue vaccine, a priority for global health.
- 734 [5] World Health Organization, Dengue and severe dengue, Fact sheet n.117.
- 735 [6] World Health Organization, Dengue and dengue haemorrhagic fever, Fact sheet n.117.
- 736 [7] L. Villar, M. D. G. H. Dayan, *al*, Efficacy of a tetravalent dengue vaccine in children in
 737 latin america, *The New England Journal of Medicine* 372 (2).
- 738 [8] A. Sabchareon, D. Wallace, C. Sirivichayakul, *et al.*, Protective efficacy of the recombi-
 739 nant, live-attenuated, cyd tetravalent dengue vaccine in thai schoolchildren: a randomised,
 740 controlled phase 2b trial, *Lancet* 380 (2012) 1559–67.
- 741 [9] Dipo Aldila, Thomas Götz, Edy Soewono, An optimal control problem arising from a
 742 dengue disease transmission model, *Mathematical Biosciences* 242 (2013) 9–16.
- 743 [10] M. Antonio, T. Yoneyama, Optimal and sub-optimal control in dengue epidemics, *Optim.*
 744 *Control Appl. Methods* 22 63.
- 745 [11] J. R. Cannon, D. J. Galiffa, An epidemiology model suggested by yellow fever, *Math.*
 746 *Methods Appl. Sci.* 35 (2012) 196–206.
- 747 [12] F. A. B. Coutinho, M. N. Burattini, L. F. Lopez, E. Massad, Threshold conditions for a
 748 non-autonomous epidemic system describing the population dynamics of dengue, *Bulletin*
 749 *of Mathematical Biology* 68 (2006) 2263–2282.
- 750 [13] G. Cruz-Pacheco, L. Esteva, C. Vargas, Seasonality and outbreaks in west nile virus infec-
 751 tion, *Bull. Math. Biol.* 71 (2009) 1378–1393.
- 752 [14] M. Derouich, A. Boutayeb, Dengue fever: mathematical modelling and computer simula-
 753 tion, *Applied Mathematics and Computation* 177 2 (2006) 528–544.

- 754 [15] Y. Dumont, F. Chiroleu, Vector control for the chikungunya disease, *Math. Biosci. Eng.* 7
755 (2010) 313–345.
- 756 [16] L. Esteva, C. Vargas, Analysis of a dengue disease transmission model, *Math. Biosci.* 150
757 (1998) 131–151.
- 758 [17] L. Esteva, C. Vargas, A model for dengue disease with variable human population, *J.*
759 *Math. Biol.* 38 (1999) 220–240.
- 760 [18] Z. Feng, V. Velasco-Hernandez, Competitive exclusion in a vector–host model for the dengue
761 fever, *J. Math. Biol.* 35 (1997) 523–544.
- 762 [19] S. M. Garba, A. B. Gumel, M. R. A. Bakar, Backward bifurcations in dengue transmission
763 dynamics, *Math. Biosci.* 215 (2008) 11–25.
- 764 [20] H. S. Rodrigues, M. T. T. Monteiro, D. F. M. Torres, Vaccination models and optimal
765 control strategies to dengue, *Mathematical Biosciences* 247 (2014) 1–12.
- 766 [21] K. W. Blayneha, A. B. Gumel, S. Lenhart, T. Clayton, Backward bifurcation and optimal
767 control in transmission dynamics of west nile virus, *Bulletin of Mathematical Biology* 72
768 (2010) 1006–1028. doi:10.1007/s11538-009-9480-0.
- 769 [22] N. A. Maidana, H. M. Yang, Dynamic of west nile virus transmission considering several
770 coexisting avian populations, *Math. Comput. Modelling* 53 (2011) 1247–1260.
- 771 [23] D. Moulay, M. A. Aziz-Alaoui, M. Cadivel, The chikungunya disease: Modeling, vector
772 and transmission global dynamics, *Math. Biosci.* 229 (2011) 50–63.
- 773 [24] D. Moulay, M. A. Aziz-Alaoui, K. Hee-Dae, Optimal control of chikungunya disease: larvae
774 reduction, treatment and prevention, *Mathematical Biosciences and Engineering* 9 (2).
- 775 [25] P. Poletti, G. Messeri, M. Ajelli, R. Vallorani, C. Rizzo, S. Merler, Transmission potential
776 of chikungunya virus and control measures: the case of italy, *PLoS One* 6 (e18860).
- 777 [26] Hamadjam Abboubakar, Jean C. Kamgang, Léontine N. Nkamba, Daniel Tieudjo, Lucas
778 Emini, Modeling the dynamics of arboviral diseases with vaccination perspective, *Biomath*
779 4 (2015).
- 780 [27] T. W. Scott, A. C. Morrison, Vector dynamics and transmission of dengue virus: impli-
781 cations for dengue surveillance and prevention strategies: vector dynamics and dengue
782 prevention, *Current Topics in Microbiology and Immunology* 338 (2010) 115–128.
- 783 [28] A. Berman, R. J. Plemmons, *Nonnegative matrices in the mathematical sciences*, 1994.
- 784 [29] J. A. Jacquez, *Qualitative theory of compartmental systems*, *SIAM Rev.* 35 (1993) 43–79.
- 785 [30] J. M. Cushing, *An Introduction to Structured Population Dynamics*, SIAM, Philadelphia,
786 1998.
- 787 [31] J. M. Cushing, Z. Yicang, The net reproductive value and stability in matrix population
788 models, *Nat. Resour. Model.* 8 (1994) 297–333.
- 789 [32] O. Diekmann, J. A. P. Heesterbeek, *Mathematical Epidemiology of Infectious Diseases.*
790 *Model building, analysis and interpretation*, John Wiley & Sons, Chichester, 2000.

- 791 [33] P. van den Driessche, J. Watmough, Reproduction numbers and the sub-threshold endemic
792 equilibria for compartmental models of disease transmission, *Math. Biosci.* 180 (2002) 29–
793 48.
- 794 [34] G. Cruz-Pacheco, L. Esteva, J. A. Montañño-Hirose, C. Vargas, Modelling the dynamics of
795 west nile virus, *Bulletin of Mathematical Biology* 67 (2005) 1157–1172.
- 796 [35] J. C. Kamgang, G. Sallet, Computation of threshold conditions for epidemiological models
797 and global stability of the disease-free equilibrium (dfe), *Mathematical Biosciences* 213
798 (2008) 1–12.
- 799 [36] J. Arino, C. C. McCluskey, P. van den Driessche, Global results for an epidemic model with
800 vaccination that exhibits backward bifurcation, *SIAM Journal on Applied Mathematics* 64
801 (2003) 260–276.
- 802 [37] F. Brauer, Backward bifurcations in simple vaccination models, *J. Math. Anal. Appl.* 298
803 (2004) 418–431.
- 804 [38] O. Sharomi, C. Podder, A. Gumel, E. Elbasha, J. Watmough, Role of incidence function in
805 vaccine-induced backward bifurcation in some hiv models, *Mathematical Biosciences* 210
806 (2007) 436–463.
- 807 [39] B. Buonomo, D. Lacitignola, On the backward bifurcation of a vaccination model with
808 nonlinear incidence, *Nonlinear Analysis: Modelling and Control* 16 (1) (2011) 30–46.
- 809 [40] B. Buonomo, A note on the direction of the transcritical bifurcation in epidemic models,
810 *Nonlinear Analysis: Modelling and Control* 20 (2015) 38–55.
- 811 [41] J. Dushoff, W. Huang, C. Castillo-Chavez, Backward bifurcations and catastrophe in sim-
812 ple models of fatal diseases, *J. Math. Biol.* 36 (1998) 227–248.
- 813 [42] M. Safan, M. Kretzschmar, K. P. Hadeler, Vaccination based control of infections in sirs
814 models with reinfection: special reference to pertussis, *J. Math. Biol.* 67 (2013) 1083–1110.
- 815 [43] C. Castillo-Chavez, B. Song, Dynamical models of tuberculosis and their applications,
816 *Math. Biosci. Eng.* 1 (2004) 361–404.
- 817 [44] J. Guckenheimer, P. Holmes, *Dynamical Systems and Bifurcations of Vector Fields, Non-*
818 *linear Oscillations*, 1983.
- 819 [45] J. Carr, *Applications of Centre Manifold Theory*, Springer, New York, 1981.
- 820 [46] J. K. Hale, *Ordinary Differential Equations*, John Wiley and Sons, 1969.
- 821 [47] J. P. LaSalle, Stability theory for ordinary differential equations, *J. Differ. Equ.* (1968)
822 57–65.
- 823 [48] J. P. LaSalle, *The stability of dynamical systems*, Society for Industrial and Applied Math-
824 ematics, 1976.
- 825 [49] N. Chitnis, J. M. Hyman, J. M. Cushing, Determining important parameters in the spread
826 of malaria through the sensitivity analysis of a mathematical model, *Bull. Math. Biol.* 70
827 (2008) 1272–1296.

- 828 [50] S. Marino, I. B. Hogue, C. J. Ray, , D. E. Kirschner, A methodology for performing global
829 uncertainty and sensitivity analysis in systems biology, *Journal of Theoretical Biology* 254
830 (2008) 178–196.
- 831 [51] J. Wu, R. Dhingra, M. Gambhir, J. V. Remais, Sensitivity analysis of infectious disease
832 models: methods, advances and their application, *Journal of the Royal Society Interface*
833 10.
- 834 [52] M. Stein, Large sample properties of simulations using latin hypercube sampling, *Techno-*
835 *metrics* 29 (1987) 143–151.
- 836 [53] F. Darriet, S. Marcombe, V. Corbel, Insecticides larvicides et adulticides alternatifs pour
837 les opérations de démoustication en france, synthèse bibliographique, IRD.
- 838 [54] P. Bosc, V. Boulet, M. Echaubard, M. L. Corre, S. Quilici, J. P. Quod, J. Rochat,
839 S. Ribes, M. Salamolard, E. Thybaud, Premier Bilan sur les Impacts des Traitements
840 Anti-moustiques, dans le cadre de la lutte Contre le Chikungunya, sur les Espèces et les
841 Milieux de l’île de la Réunion (Juin 2006).
- 842 [55] Helena Sofia Ferreira Rodrigues, Optimal control and numerical optimization applied to
843 epidemiological models, Ph.D. thesis, Universidade de Aveiro Departamento de Matemática
844 (2012).
- 845 [56] S. Licciardi, personal communication.