Bifurcation thresholds and optimal control in transmission dynamics of arboviral diseases

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Abstract In this paper, we derive and analyse a model for the control of arboviral diseases which takes into account an imperfect vaccine combined with some other control measures already studied in the literature. We begin by analysing the basic model without control. We prove the existence of two disease-free equilibrium points and the possible existence of up to two endemic equilibrium points (where the disease persists in the population). We show the existence of a transcritical bifurcation and a possible saddle-node bifurcation and explicitly derive threshold conditions for both, including defining the basic reproduction number, $R_0$, which provides whether the disease can persist in the population or not. The epidemiological consequence of saddle-node bifurcation is that the classical requirement of having the reproduction number less than unity, while necessary, is no longer sufficient for disease elimination from the population. It is further shown that in the absence of disease-induced death, the model does not exhibit this phenomenon. The model is extended by reformulating...
the model as an optimal control problem, with the use of five time dependent controls, to assess the impact of vaccination combined with treatment, individual protection and two vector control strategies (killing adult vectors and reduction of eggs and larvae). By using optimal control theory, we establish conditions under which the spread of disease can be stopped, and we examine the impact of combined control tools on the transmission dynamic of disease. The Pontryagin’s maximum principle is used to characterize the optimal control. Numerical simulations and efficiency analysis show that, vaccination combined with other control mechanisms, would reduce the spread of the disease appreciably.

**Keywords** Arboviral diseases · Bifurcation · Optimal control · Pontryagin’s maximum principle (PMP) · Efficiency analysis

**Mathematics Subject Classification** 37N25 · 34C23 · 49J15 · 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 (Chippaux 2003; Karabatsos 1985; Gubler 2001). Examples of those kinds of diseases are Dengue, Yellow fever, Saint Louis fever, Encephalitis, West Nile fever and Chikungunya. A wide range of arboviral diseases are transmitted by mosquito bites and constitute a public health emergency of international concern. For example, 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 9–16 years (Sanofi Pasteur 2013; World Health Organization 2013, 2009).

The dynamics of arboviral diseases like Dengue or Chikungunya are influenced by many factors such as human and mosquito behaviours. The virus itself [multiple serotypes of dengue virus (World Health Organization 2013, 2009), and multiple strains of chikungunya virus (Djamila 2011; Parola et al. 2006)], as well as the environment, affects directly or indirectly all the present mechanisms of control (Brasseur 2011; Carvalho et al. 2015). Indeed, in the absence of conditions which favour the development of their larvae, eggs of certain Aedes mosquitoes (Aedes albopictus, for example) enter in diapause, allowing the eggs to hatch even after 2 years (Paupy et al. 2009; Sota and Mogi 1992). Taking the case of Aedes mosquitoes for example, the main control method used in many countries continues to be space spraying of insecticide for adult mosquito control. This strategy must be repeated constantly, its cost is high, and its effectiveness is limited. Also, A. aegypti, for example, prefers to rest inside houses, so truck or aerial insecticide spraying simply does not reach mosquitoes resting in hidden places such as cupboards (Parks and Lloyd 2004).

The different types of control mechanisms put in place to reduce the proliferation of vectors responsible for the transmission of pathogens such as arboviruses are listed below.
(i) Biological control or “biocontrol” is the use of natural enemies to manage vector populations: introduction of parasites, pathogens and predators to target vectors. For example, effective biocontrol agents include predatory fish that feed on mosquito larvae such as mosquitofish (*Gambusia affinis*) and some cyprinids (carps and minnows) and killifish. Tilapia also consume mosquito larvae (*Alcaraz and García-Berthou 2007*). As biological control does not cause chemical pollution, it is considered as a better method for mosquito control by many people. However, there are limitations on employing biological agents for mosquito control. The agent introduced usually has to be substantial in number for giving appreciable effect.

(ii) Mechanical control consist of the environmental sanitation measures to reduce mosquito breeding sites, such as the physical management of water containers (e.g. mosquito-proof covers for water storage containers, polystyrene beads in water tanks), better designed and reliable water supplies, and recycling of solid waste such as discarded tyres, bottles, and cans (*Parks and Lloyd 2004; Dumont and Chiroleu 2010*).

(iii) Chemical methods (*Parks and Lloyd 2004; Dumont and Chiroleu 2010*):
- chemical methods against the mosquito’s aquatic stages for use in water containers (larviciding—killing of larvae),
- chemical methods directed against adult mosquitoes, such as insecticide space sprays or residual applications (adulticiding—killing of adult mosquitoes).

(iv) Personal protection consist of the use of repellents, vaporizers, mosquito coils, and insecticide treated screens, curtains, and bed nets (for daytime use against *Aedes*) (*Parks and Lloyd 2004*).

The main problem encountered in the implementation of some of these control mechanisms is the preservation of the ecological systems. For example, in the “biocontrol” mechanism, direct introduction of tilapia and mosquitofish into ecosystems around the world have had disastrous consequences (*Alcaraz and García-Berthou 2007*). Also, the chemical methods can not be applied in continuous times. Some chemical product like *Deltamethrin* seems to be effective only during a couple of hours (*Dumont and Chiroleu 2010; Bosc et al. 2006; Rodrigues 2012*). So its use over a long period and continuously, leads to strong resistance of the wild populations of *Aedes aegypti* (*Darriet et al. 2007*), for example.

For all the diseases mentioned above, only yellow fever has a licensed vaccine. Nevertheless, considerable efforts are made to obtain vaccines for other diseases. In the case of dengue, for example, tests carried out in Asia and Latin America, have shown that the future dengue vaccine will have a efficacy between 30.2 and 77.7%, and this, depending on the serotype (*Sabchareon et al. 2012; Villar et al. 2015*). Also, the future dengue vaccine will have an overall efficacy of 60.8% against all forms of the disease in children and adolescents aged 9–16 years who received three doses of the vaccine (*Sanofi Pasteur 2014*).

As the future vaccines (e.g., dengue vaccine) will be imperfect, it is therefore necessary to combine such vaccines with some control mechanisms cited above (*Abboubakar et al. 2015, 2016b; Nishiura 2006*), to find the best sufficient combi-
nation, which permit to decrease the expansion of these kind of diseases in human communities.

A number of studies have been conducted to study host-vector models for arboviral diseases transmission (Abboubakar et al. 2015, 2016b; Aldila et al. 2013; Antonio and Yoneyama 2001; Blayneh et al. 2010; Cannon and Galiffa 2012; Coutinho et al. 2006; Cruz-Pacheco et al. 2009; Derouich and Boutayeb 2006; Dumont and Chiroleu 2010; Esteva and Vargas 1998, 1999; Feng and Velasco-Hernandez 1997; Garba et al. 2008; Maidana and Yang 2011; Moulay et al. 2011, 2012; Poletti et al. 2011; Rodrigues et al. 2014). Some of these works have been conducted to explore optimal control theory for arboviral disease models (see Aldila et al. 2013; Blayneh et al. 2010; Moulay et al. 2012; Rodrigues et al. 2014; Dias and Wanner 2015).

Aldila et al. (2013) derive an optimal control problem for a host-vector Dengue transmission model, in which treatments with mosquito repellent are given to adults and children and those who undergo treatment are classified in treated compartments. The only control considered by the authors is the treatment of people with clinical signs of the disease. Blayneh et al. (2010) consider a deterministic model for the transmission dynamics of West Nile virus (WNV) in the mosquito-bird-human zoonotic cycle. They use two control functions, one for mosquito-reduction strategies and the other for personal (human) protection, and redefining the demographic parameters as density-dependent rates. Moulay et al. (2012) derive optimal prevention (individual protection), vector control (Larvae reduction) and treatment strategies used during the Chikungunya Réunion Island epidemic in 2006. Rodrigues et al. (2014) derive the optimal control efforts for vaccination in order to prevent the spread of a Dengue disease using a system of ordinary differential equations (ODEs) for the host and vector populations. Recently, Dias and Wanner (2015) analyse the Dengue vector control problem in a multiobjective optimization approach, in which the intention is to minimize both social and economic costs, using a dynamic mathematical model representing the mosquitoes’ population. This multiobjective optimization approach consists in finding optimal alternated step-size control policies combining chemical (via application of insecticides) and biological control (via insertion of sterile males produced by irradiation).

None of the above mentioned models (Aldila et al. 2013; Blayneh et al. 2010; Moulay et al. 2012; Rodrigues et al. 2014; Dias and Wanner 2015) takes into account the combination of optimal control mechanisms such as vaccination, individual protection, treatment and vector control strategies. In our effort, we investigate such optimal strategies for vaccination combined with individual protection, treatment and two vector controls (adulticiding—killing of adult vectors, and larviciding—killing eggs and larvae), using two systems of ODEs which consist of a complete stage structured model Eggs–Larvae–Pupae for the vectors, and a SEI/SEIR type model for the vector/host population. This provides a new different mathematical perspective to the subject. Furthermore, a efficiency analysis is performed here in order to evaluate the control combination that is most effective in the design of optimal strategies.

We start with the formulation of a model without control which is a modified version of the previous models developed in Abboubakar et al. (2015, 2016b). We compute the net reproductive number $N$, as well as the basic reproduction number, $R_0$, and investigate the existence and stability of equilibria. We prove that the trivial
equilibrium is globally asymptotically stable whenever $N < 1$. When $N > 1$ and $R_0 < 1$, we prove that the system exhibits the backward bifurcation phenomenon. The implication of this occurrence is that the classical epidemiological requirement for effective eradication of the disease, $R_0 < 1$, is no longer sufficient, even though necessary. We show the existence of a transcritical bifurcation and a possible saddle-node bifurcation and explicitly derive threshold conditions for both.

Then, we formulate an optimal control model by adding five control functions: three for human (vaccination, protection against mosquitoes bites and treatment), and two for mosquito-reduction strategies (the use of adulticide to kill adult vectors, and the use of larvicide to increase the mortality rate of eggs and larvae). By using optimal control theory, we establish conditions under which the spread of disease can be stopped and examine the impact of a possible combination of vaccination, treatment, individual protection and vector control strategies on the disease transmission. The Pontryagin’s maximum principle is used to characterize the optimal control. Numerical simulations and efficiency analysis are performed to determine the best combination, in terms of efficacy.

The rest of the paper is organized as follows. In Sect. 2, we present the basic transmission model and carry out some analysis by determining important thresholds such as the net reproductive number $N$ and the basic reproduction number $R_0$, and different equilibria of the model. We then demonstrate the stability of equilibria and derive the threshold conditions for saddle-node bifurcation. We present the optimal control problem and its mathematical analysis in Sect. 3. Section 4 is devoted to numerical simulations and efficiency analysis. A conclusion completes the paper.

2 The basic model and its analysis

The model we propose here is based on the modelling approach given in Abboubakar et al. (2015, 2016b). It is assumed that the human and vector populations are divided into compartments described by time-dependent state variables. The compartments in which the populations are divided are the following ones:

(i) For humans, we consider a SEIR model: Susceptible (denoted by $S_h$), infected human in latent stage ($E_h$), infectious ($I_h$) and resistant or immune ($R_h$) which includes naturally-immune individuals. The recruitment in human population is at the constant rate $\Lambda_h$, and newly recruited individuals enter the susceptible compartment $S_h$. Only naive humans to the disease are taken into account in the recruitment. Human individuals die of natural causes at rate $\mu_h$. The human susceptible population is decreased following infection, which can be acquired via effective contact with an exposed or infectious vector at a rate

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

where $a$ is the biting rate per vector; the transmission probability from infected vectors in latent stage $E_v$ (resp. infectious vectors $I_v$) to susceptible human is $\eta_v \beta_{hv}$ (resp. $\beta_{hv}$). The expression of $\lambda_h$ is obtained as follows. The probability that a vector chooses a particular human or other source of blood to bite can be assumed as $1/N_h$. Thus,
a human receives in average $aN_v/N_h$ bites per unit of time. Then, the infection rate per susceptible human is given by $a\beta_v(N_v/N_h)((\eta_v E_v + I_v)/N_v)$. In expression of $\lambda_h$, the modification parameter $0 < \eta_v < 1$ accounts for the assumed reduction in transmissibility of infected vectors in latent stage relative to infectious vectors. It is worth emphasizing that, unlike many of the published modelling studies on dengue transmission dynamics, we assume in this study that infected vectors in latent stage can transmit dengue disease to humans. This is in line with some studies (Abboubakar et al. 2015, 2016b; Eshita et al. 2007; Garba et al. 2008; Wilder-Smith et al. 2004; Yébakima et al. 2004).

Infected humans in latent stage ($E_h$) become infectious ($I_h$) at rate $\gamma_h$. Infectious humans recover at a constant rate, $\sigma$ or die as consequence of infection, at a disease-induced death rate $\delta$. Immune humans retain their immunity for life. We denote the total human population by $N_h$,

$$N_h = S_h + E_h + I_h + R_h.$$  \hspace{1cm} (2)

(ii) Following Moulay et al. (2011), the stage structured model is used to describe the vector population dynamics, which consists of three main stages: eggs (E), larvae (L) and pupae (P). Even if eggs (E) and immature stages (L and P) are both aquatic, it is important to distinguish them because, drying the breeding sites does not kill eggs, but only larvae and pupae. Moreover, chemical interventions on the breeding sites have a great impact on the larval population, but not on the eggs (Moulay et al. 2011). The number of laid eggs is assumed proportional to the number of females. The system of stage structured model of aquatic phase development of vector is given by

\begin{align*}
\dot{E} &= \mu_b \left( 1 - \frac{E}{K_E} \right) (S_v + E_v + I_v) - (s + \mu_E)E \hspace{1cm} \text{(3a)} \\
\dot{L} &= sE \left( 1 - \frac{L}{K_L} \right) - (l + \mu_L)L \hspace{1cm} \text{(3b)} \\
\dot{P} &= lL - (\theta + \mu_P)P. \hspace{1cm} \text{(3c)}
\end{align*}

In Eq. (3), $E$, $L$ and $P$ represent population of eggs, larvae and pupae, respectively. The per capita mortality rate of eggs, larvae and pupae are denoted by $\mu_E$, $\mu_L$ and $\mu_P$ respectively. $\mu_b$ is the intrinsic oviposition rate. $K_E$ and $K_L$ denote the carrying capacity of eggs and larvae, respectively. It has been observed that females of $Aedes$ Mosquitoes, for example, are able to detect the best breeding sites for the egg development, that is to say breeding sites where eggs and then larvae will be able to develop easily. So, we follow Moulay et al. (2011, 2012) and assume that the per capita oviposition rate is also proportional to the number of females and given by $\mu_b \left( 1 - \frac{E}{K_E} \right) (S_v + E_v + I_v)$, which implies that the number of new larvae is given by $sE \left( 1 - \frac{L}{K_L} \right)$ (see Moulay et al. 2011 for other details).

Unlike the authors of Moulay et al. (2011, 2012), we take into account the pupal stage in the development of the vector. On the other hand, we don’t include density dependence for pupae, $P$, because at this transitional stage, they don’t need any nutriment. So, the control mechanisms can not be applied to them.
While predation could be discounted as major causes of larval mortality, intraspecific competition represent a major density dependent source for them, and hence, the effect of crowding could be an important factor in the population dynamics of mosquitoes (Ai et al. 2012). For sake of simplicity, we don’t take into account the effect of density dependence in death rates of eggs and larvae.

With a rate $\theta$, pupae become female adults. Vector individuals die from natural causes at rate $\mu_v$. The vector susceptible population is decreased following infection, which can be acquired via effective contact with an infected in latent stage or infectious human at a rate $\lambda_v = a\beta_v \gamma_v (\eta_h E_h + I_h)$, (4)

where $\eta_h \beta_v$ (respectively $\beta_v E_h$) is the transmission probability from infected humans in latent stage $E_h$ (respectively infectious humans $I_h$) to susceptible vector. As well as in the expression of $\lambda_h$, the modification parameter $0 < \eta_h < 1$ in the expression of $\lambda_v$ accounts for the assumed reduction in transmissibility of infected humans in latent stage relative to infectious humans (Abboubakar et al. 2015, 2016b; Garba et al. 2008). Latent vectors ($E_v$) become infectious ($I_v$) at rate $\gamma_v$. The vector population does not have an immune class, since it is assumed that their infectious period ends with their death (Esteva and Vargas 1999). So, we denote the total adult vector population by $N_v$,

$$N_v = S_v + E_v + I_v.$$ (5)

Therefore, our basic arboviral disease model reads as

\[
\begin{align*}
\dot{S}_h &= \Lambda_h - (\lambda_h + \mu_h)S_h \\
\dot{E}_h &= \lambda_h S_h - (\mu_h + \gamma_h)E_h \\
\dot{I}_h &= \gamma_h E_h - [\mu_h + \delta + \sigma] I_h \\
\dot{R}_h &= \sigma I_h - \mu_h R_h \\
\dot{S}_v &= \theta P - \lambda_v S_v - \mu_v S_v \\
\dot{E}_v &= \lambda_v S_v - (\mu_v + \gamma_v) E_v \\
\dot{I}_v &= \gamma_v E_v - (\mu_v) I_v \\
\dot{E} &= \mu_b \left( 1 - \frac{E}{K_E} \right) N_v - (s + \mu_E) E \\
\dot{L} &= s E \left( 1 - \frac{L}{K_L} \right) - (l + \mu_L) L \\
\dot{P} &= l L - (\theta + \mu_P) P
\end{align*}
\]

where the upper dot denotes the time derivative and $\lambda_h$ and $\lambda_v$ are given by (1) and (4), respectively. A schematic of the model is shown in Fig. 1. The states and parameters are all positive and are described in Tables 1 and 2, respectively.

Remark 1 (i) It is important to note that, in the case of arboviral diseases like Chikungunya and Yellow fever, the infected humans and vectors in latent stages do not play any role in the infection process. In this case $\eta_h = \eta_v = 0$. 

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Fig. 1  Schematic of the vector-borne epidemic model with development stage of vectors

Table 1  The state variables of model (6)

| Human variables | Description | Aquatic vectors | Description | Adult vectors | Description |
|-----------------|-------------|-----------------|-------------|--------------|-------------|
| $S_h$           | Susceptible | $E$             | Eggs        | $S_v$        | Susceptible |
| $E_h$           | Infected in | $L$             | Larvae      | $E_v$        | Infected in |
|                 | latent stage|                 |             | latent stage |             |
| $I_h$           | Infectious  | $P$             | Pupae       | $I_v$        | Infectious  |
| $R_h$           | Resistant (immune) |          |             |              |             |

(ii) The model (6) is the same that we studied in a previous work (see Abboubakar et al. 2016b, model 18, page 9) to show that the backward bifurcation is caused by the disease-induced death in human. In this previous work, we have shown that backward bifurcation is possible in the model without vaccination. In the present work, we give a sufficient and necessary condition, as well as the explicit expressions of the thresholds which govern this phenomenon.

**Remark 2** Most mathematical models of transmission dynamics of infectious diseases like vector-borne diseases, assume that humans and vectors die at constant rates. However, others factors like density (Blayneh et al. 2009, 2010; Chitnis et al. 2008; Freedman 1979; Ngwa and Shu 2000) and age (Bellan 2010; Gurtin and Mac-Camy 1974, 1979; Stukalin et al. 2013) increase the death rate of the populations involved. For example, using a simple mathematical model, Bellan (2010), shows that the common assumption of a constant mortality hazard (age independent) has led most vector-borne disease models to overestimate the efficiency of vector control. For sake
Table 2  Description and baseline values/range of parameters of model (6)

| Parameter | Description | Baseline value/range | Sources |
|-----------|-------------|----------------------|---------|
| $\Lambda_h$ | Recruitment rate of humans | 2.5 day$^{-1}$ | Garba et al. (2008) |
| $\mu_h$ | Natural mortality rate in humans | $\frac{1}{(67 \times 365)}$ day$^{-1}$ | Garba et al. (2008) |
| $a$ | Average number of bites | 1 day$^{-1}$ | Aldila et al. (2013); Garba et al. (2008) |
| $\beta_{hv}$ | Probability of transmission of Infection from an infected vector To a susceptible human | 0.1, 0.75 day$^{-1}$ | Aldila et al. (2013), Garba et al. (2008) |
| $\gamma_h$ | Progression rate from $E_h$ to $I_h$ | $\left[\frac{1}{15}, \frac{1}{3}\right]$ day$^{-1}$ | Dumont and Chiroleu (2010), Scott and Morrison (2010) |
| $\delta$ | Disease-induced death rate | $10^{-3}$day$^{-1}$ | Garba et al. (2008) |
| $\sigma$ | Recovery rate for humans | 0.1428 day$^{-1}$ | Aldila et al. (2013), Garba et al. (2008) |
| $\eta_h, \eta_v$ | Modifications parameter | [0, 1) | Garba et al. (2008) |
| $\mu_v$ | Natural mortality rate of vectors | $\left[\frac{1}{30}, \frac{1}{14}\right]$ day$^{-1}$ | Aldila et al. (2013), Garba et al. (2008) |
| $\gamma_v$ | Progression rate from $E_v$ to $I_v$ | $\left[\frac{1}{21}, \frac{1}{2}\right]$ day$^{-1}$ | Dumont and Chiroleu (2010), Scott and Morrison (2010) |
| $\beta_{vh}$ | Probability of transmission of Infection from an infected human To a susceptible vector | 0.1, 0.75 day$^{-1}$ | Aldila et al. (2013), Garba et al. (2008) |
| $\theta$ | Maturation rate from pupae to adult | 0.08 day$^{-1}$ | Dumont and Chiroleu (2010), Moulay et al. (2011), Moulay et al. (2012) |
| $\mu_b$ | Number of eggs at each deposit | 6 day$^{-1}$ | Dumont and Chiroleu (2010), Moulay et al. (2011), Moulay et al. (2012) |
| $K_E$ | Carrying capacity for eggs | $10^3, 10^6$ | Aldila et al. (2013), Moulay et al. (2011) |
| $K_L$ | Carrying capacity for larvae | $5 \times 10^2, 5 \times 10^5$ | Aldila et al. (2013), Moulay et al. (2011) |
### Table 2 continued

| Parameter | Description                  | Baseline value/range | Sources                      |
|-----------|------------------------------|----------------------|------------------------------|
| $\mu_E$   | Eggs death rate              | 0.2 or 0.4           | Moulay et al. (2012)         |
| $\mu_L$   | Larvae death rate            | 0.2 or 0.4           | Moulay et al. (2012)         |
| $\mu_P$   | Pupae death rate             | 0.4                  | Abboubakar et al. (2016b)    |
| $s$       | Transfer rate from eggs to larvae | 0.7 day$^{-1}$   | Moulay et al. (2012)         |
| $l$       | Transfer rate from larvae to pupae | 0.5 day$^{-1}$   | Moulay et al. (2011)         |

The baseline values refer to dengue fever transmission.
of simplicity, we assume in this work that mortality death rates (of human and vector populations) is density-independent and age-independent. A possible modification of our model in which we will take into account density-dependant or/and age dependant death rates in human and vector populations, will be the subject of future work, to assess the sensitivity of the assumption of exponential age distribution in humans and vectors, and thus predict the level of control which will be needed.

2.1 Basic properties and equilibria

For easier readability, we introduce the following quantities,

\[ k_1 := \mu_h; \quad k_3 := \mu_h + \gamma_h; \quad k_4 := \mu_h + \delta + \sigma; \quad k_5 := s + \mu_E; \quad k_6 := l + \mu_L; \]
\[ k_7 := \theta + \mu_P; \quad k_8 := \mu_v; \quad k_9 := \mu_v + \gamma_v; \quad k_{10} = \eta_h k_4 + \gamma_h; \]
\[ k_{11} = \eta_v k_8 + \gamma_v, \]

and (the positive quantity), \( k_2 = k_3 k_4 - \delta \gamma_h = \mu_h k_4 + \gamma_h (\mu_h + \sigma). \)

Then, the basic model (6) becomes

\[
\begin{align*}
\dot{S}_h &= \Lambda_h - (\lambda_h + k_1) S_h \quad (8a) \\
\dot{E}_h &= \lambda_h S_h - k_3 E_h \quad (8b) \\
\dot{I}_h &= \gamma_h E_h - k_4 I_h \quad (8c) \\
\dot{R}_h &= \sigma I_h - k_1 R_h \quad (8d) \\
\dot{S}_v &= \theta P - \lambda_v S_v - k_8 S_v \quad (8e) \\
\dot{E}_v &= \lambda_v S_v - k_9 E_v \quad (8f) \\
\dot{I}_v &= \gamma_v E_v - k_8 I_v \quad (8g) \\
\dot{E} &= \mu_b \left(1 - \frac{E}{K_E}\right) N_v - \sigma E \quad (8h) \\
\dot{L} &= s E \left(1 - \frac{L}{K_L}\right) - \sigma L \quad (8i) \\
\dot{P} &= l L - k_7 P \quad (8j)
\end{align*}
\]

The rates of change of the total populations of humans (2) and adult vectors (5) for the basic arboviral model (8) are,

\[
\begin{align*}
\dot{N}_h &= \Lambda_h - k_1 N_h - \delta I_h, \quad (8k) \\
\dot{N}_v &= \theta P - k_8 N_v \quad (8l)
\end{align*}
\]
Therefore, by standard arguments (see Abboubakar et al. 2015, 2016b; Moulay et al. 2011) it follows that the feasible region for model (8) is

\[
\mathcal{D} = \{ (S_h, E_h, I_h, R_h, S_v, E_v, I_v, E, L, P) \in \mathbb{R}^{10}_+ : N_h \leq \Lambda_h/k_1; E \leq K_E; L \leq K_L;\]

\[
P \leq \frac{l K_L}{k_7}; N_v \leq \frac{\theta l K_L}{k_7 k_8}\}
\]

where \(\mathbb{R}^{10}_+\) represents the nonnegative orthant of \(\mathbb{R}^{10}\).

The model is epidemiologically (the state variables have a valid physical interpretation) and mathematically (the system of equations has a unique solution that is bounded and exists for all time) well-posed in the region \(\mathcal{D}\).

We begin by focus on the vector population dynamics. Adding the equations for adult vectors \((S_v, E_v, I_v)\) in (8) together, with the last three equations in (8) leads to the following closed system:

\[
\begin{align*}
\dot{E} &= \mu_b \left(1 - \frac{E}{K_E}\right) N_v - k_5 E, \\
\dot{L} &= s E \left(1 - \frac{L}{K_L}\right) - k_6 L, \\
\dot{P} &= l L - k_7 P, \\
\dot{N}_v &= \theta P - k_8 N_v.
\end{align*}
\]

(9)

Since the state variables of the system (9) are non-negative for all time \(t\) (and all its parameters are positive), the system (9) can be studied in the invariant region:

\[
\Omega = \{(E, L, P, N_v) \in \mathbb{R}^4 : E, L, P, N_v \geq 0\},
\]

where the system is mathematically and biologically well-posed.

Define the net reproductive number (Abboubakar et al. 2016b; Cushing 1998; Moulay et al. 2011), given by

\[
\mathcal{N} = \frac{\mu_b \theta l s}{k_5 k_6 k_7 k_8}.
\]

\(\mathcal{N}\) measures the average expected number of new adult female offsprings produced by a single female vector during its life time. It can be interpreted as the product of the fraction of eggs that survived and hatched into larvae \(s/k_5 = s/(s + \mu_E)\), the fraction of larvae that survived and progressed into pupae \(l/k_6 = l/(l + \mu_L)\), the fraction of pupae that survived to become adult female mosquitoes \(\mu_b \theta /k_7 = \mu_b \theta / (\theta + \mu_P)\) and the average lifespan of female adult mosquitoes \(1/k_8 = 1/\mu_v\).

**Lemma 1** *The trivial equilibrium point \(E_0 = (0, 0, 0, 0)\) of (9) is globally asymptotically stable in \(\mathbb{R}^4_+\) if \(\mathcal{N} \leq 1\), and unstable otherwise.*
Proof. See appendix A.

In the following, we consider that the net reproductive number $\mathcal{N}$ is greater than unity. The system (9) has a non-trivial equilibrium, denoted by $E_1 = (E^*, L^*, P^*, N_v^0)$, where

$$
N_v^0 = \frac{K_E K_L k_5 k_6 (\mathcal{N} - 1)}{\mu_b (K_E s + k_6 K_L)},
$$

$$
P^* = \frac{K_E K_L k_5 k_6 k_8 (\mathcal{N} - 1)}{\mu_b \theta (K_E s + k_6 K_L)}.
$$

We have the following result (see Appendix B).

Lemma 2. If $\mathcal{N} > 1$, then the unique positive equilibrium of system (9), $E_1 = (E^*, L^*, P^*, N_v^0)$, is globally asymptotically stable in $\mathbb{R}_+^4 \setminus \{0\mathbb{R}_+^4\}$.

2.1.1 Disease-free equilibria and stability analysis

Without disease in both populations (i.e $\lambda_h = \lambda_v = 0$ or $E_h = I_h = E_v = I_v = 0$), the basic model (8) has two disease-free equilibria (equilibria without disease) given by $Q_1 = (N_h^0, 0, 0, 0, 0, 0, 0, 0, 0, 0)$ which corresponds to the trivial equilibrium (a human population free of vectors and free of disease), and $Q_2 = (N_h^0, 0, 0, 0, N_v^0, 0, 0, E^*, L^*, P^*)$ which correspond to the biological disease-free equilibrium, where $N_h^0 = \frac{\Lambda_h}{\mu_h}$ and $N_v^0, E^*, L^*$ and $P^*$, are given in (11).

Define the basic reproductive number (Diekmann and Heesterbeek 2000; Driessche and Watmough 2002)

$$
\mathcal{R}_0 = \sqrt{\frac{a^2 \beta_v \beta_v k_{10} k_{11} N^0_v}{k_3 k_4 k_8 k_9 N_h^0}}.
$$

We note that,

$$
\mathcal{R}_0 = \sqrt{K_v h K_h v},
$$

where,

$$
K_v h = (K^E_v h + K^I_v h)
$$

$$
= (a) (\beta_v h) \left( \frac{N^0_v}{N_h^0} \right) \left( \frac{1}{k_3} \right) \left[ (\eta_v h) + (\gamma h) \right]
$$

$$
= \frac{a \beta_v h (\gamma h + k_4 \eta_v h) N^0_v}{k_3 k_4 N_h^0}
$$

is the expected number of vectors that one human infects through his/her latent/infectious life time, in the “virgin” situation. Near the DFE, $Q_2$, it is equal to the sum of the number of vector infections generated by infected human in latent stage $K^E_v h$, and

---

1 The expression “biological disease-free equilibrium” refers to the equilibrium without disease, and which is realistic (Dumont and Chiroleu 2010).
the number of vector infections generated by an infectious human $K_{v h}^I$. $K_{v h}^E$ is given by the product of the infection rate of infected humans in latent stage ($a \beta_{v h} \eta_h N_0^v / N_0^h$) and the average duration in the latent ($E_h$) class $(1/k_3)$. $K_{v h}^I$ is given by the product of the infection rate of infectious humans ($a \beta_{v h} N_0^v / N_0^h$), the probability that an infected human survives the latent stage and move to the infectious stage ($\gamma_h / (\mu_h + \gamma_h)$) and the average duration in the infectious stage $(1/(\mu_h + \delta + \sigma))$.

Analogously, we have

$$K_{h v} = K_{h v}^E + K_{h v}^I = (a) (\beta_{h v}) \left( \frac{1}{k_2} \right) \left( \eta_v + \frac{\gamma_v}{k_8} \right) = \frac{a \beta_{h v} (\gamma_v + k_8 \eta_v)}{k_8 k_9} := \frac{a \beta_{h v} k_{11}}{k_8 k_9},$$

which is the expected number of humans that one vector infects throughout its infectious life time. Near the DFE, $Q_2$, it is equal to the sum of the number of human infections generated by an infected vector in latent stage, $K_{h v}^E$, and the number of human infections generated by an infectious vector, $K_{h v}^I$. $K_{h v}^E$ is given by the product of the infection rate of infectious vectors in latent stage ($a \beta_{h v} \eta_v$) and the average duration in the latent ($E_v$) class $(1/(\mu_v + \gamma_v))$. $K_{v h}^I$ is given by the product of the infection rate of infectious vectors ($a \beta_{h v}$), the probability that an infected vector survives the latent stage and moves to the infectious stage ($\gamma_v / k_9$) and the average duration in the infectious stage $(1/k_8)$.

The basic reproduction number is equal to the geometric mean of $K_{v h}$ and $K_{h v}$ because infection from human to human goes through one generation of vectors (Abboubakar et al. 2016a; Lord et al. 1996).

The local asymptotic stability result of equilibria $Q_1$ and $Q_2$ is given in the following.

**Theorem 1** (i) if $N \leq 1$, the trivial equilibrium $Q_1$ is locally asymptotically stable in $D$;

(ii) if $N > 1$, the trivial equilibrium is unstable and the disease-free equilibrium $Q_2$ is locally asymptotically stable in $D$ whenever $R_0 < 1$.

**Proof** See appendix C.

The epidemiological implication of item (ii) in Theorem 1 is that, in general, when the basic reproduction number, $R_0$ is less than unity, a small influx of infectious vectors into the community would not generate large outbreaks, and the disease dies out in time (since the DFE is locally asymptotically stable) (Abboubakar et al. 2016b; Cruz-Pacheco et al. 2005; Diekmann and Heesterbeek 2000; Garba et al. 2008; Driessche and Watmough 2002). However, we will show in the Sect. 2.1.2 that the disease may still persist even when $R_0 < 1$. 

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

**Theorem 2** If $N \leq 1$, then $Q_1$ is globally asymptotically stable in $D$.

**Proof** See Appendix D. \(\square\)

### 2.1.2 Endemic equilibria and backward bifurcation

For $N > 1$, it follows from Lemma 2 that $E^*, L^*$ and $P^*$ are the components of any biologically meaningful equilibrium of (8), and the rest of the components, of an endemic equilibrium of (8), are the components of the equilibria of the following limiting system:

\[
\begin{align*}
\dot{S}_h &= \Lambda_h - (\lambda_h + k_1)S_h, \\
\dot{E}_h &= \lambda_h S_h - k_3 E_h, \\
\dot{I}_h &= \gamma_h E_h - k_4 I_h, \\
\dot{R}_h &= \sigma I_h - k_1 R_h, \\
\dot{S}_v &= k_8 N_0^v - \lambda_v S_v - k_8 S_v, \\
\dot{E}_v &= \lambda_v S_v - k_9 E_v, \\
\dot{I}_v &= \gamma_v E_v - k_8 I_v,
\end{align*}
\]

which is defined on

\[
\mathcal{O} = \left\{ (S_h, E_h, I_h, R_h, S_v, E_v, I_v) \in \mathbb{R}_+^7 : N_h \leq \frac{\Lambda_h}{\mu_h}, N_v \leq N_0^v \right\}.
\]

where $N_0^v$ is given by (11).

First we introduce:

\[
\begin{align*}
\psi &= k_{10\alpha} \mu_h \beta_{vh} - \delta \gamma_h k_8, \\
R_c &= \sqrt{2k_8k_2 + k_{10\alpha} \mu_h \beta_{vh}} k_3k_4k_8, \\
R_{1b} &= \frac{1}{k_3k_4} \left( \sqrt{1,k_8} - \sqrt{k_{10\alpha} \mu_h \beta_{vh} + k_2k_8} \right), \\
R_{2b} &= \frac{1}{k_3k_4} \left( \sqrt{1,k_8} + \sqrt{k_{10\alpha} \mu_h \beta_{vh} + k_2k_8} \right).
\end{align*}
\]

Note that (as shown in the proof of Theorem 3), when $R_c < R_0 < 1$, we have $\psi \leq 0$ and correspondingly, $R_{1b}$ and $R_{2b}$ are real. In the particular case when $\psi = 0$, we have $R_{1b} = R_{2b}$, which we label by,

\[
\tilde{R} = R_{1b}|_{\psi=0} = R_{2b}|_{\psi=0}.
\]
Theorem 3 Let us consider the following inequalities:

\[ \mathcal{R}_c < \mathcal{R}_0 < \min(1, \mathcal{R}_{1b}), \]  
\[ \max(\mathcal{R}_c, \mathcal{R}_{2b}) < \mathcal{R}_0 < 1. \]  

The number of endemic equilibrium points of the basic arboviral disease model (8) depends on \( \mathcal{R}_0 \) as follows:

(i) For \( \mathcal{R}_0 > 1 \), the system has a unique endemic equilibrium point.

(ii) For \( \mathcal{R}_0 = 1 \), the system has

(a) A unique endemic equilibrium point if \( \mathcal{R}_c < 1 \).

(b) No endemic equilibrium points otherwise.

(iii) For \( \mathcal{R}_0 < 1 \), the system has

(a) Two endemic equilibrium points if either inequality (18a) or (18b) is satisfied.

(b) A unique endemic equilibrium point if \( \mathcal{R}_c < \mathcal{R}_0 \) and either \( \mathcal{R}_0 = \mathcal{R}_{1b} \) or \( \mathcal{R}_0 = \mathcal{R}_{2b} \).

(c) No endemic equilibrium points otherwise.

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

\[ Q^{**} = (S_h^*, V_h^*, E_h^*, I_h^*, R_h^*, S_v^*, E_v^*, I_v^*), \]

we have to look for the solution of the algebraic system of equations obtained by equating the right sides of system (13) to zero.

Solving the equations in the system (13) in terms of \( \lambda_h^* \) and \( \lambda_v^* \), gives

\[ S_h^* = \frac{\Lambda_h}{k_1 + \lambda_h^*}, \quad E_h^* = \frac{\lambda_h^* S_h^*}{k_3}, \quad I_h^* = \frac{\gamma_h \lambda_h^* S_h^*}{k_3 k_4}, \quad R_h^* = \frac{\sigma \gamma_h \lambda_h^* S_h^*}{k_1 k_3 k_4}, \]  

(19)

and

\[ S_v^* = \frac{\mu_v N_v^0}{(\lambda_v^* + k_8)}, \quad E_v^* = \frac{\mu_v N_v^0 \lambda_v^*}{k_9 (\lambda_v^* + k_8)}, \quad I_v^* = \frac{\gamma_v \mu_v N_v^0 \lambda_v^*}{k_8 k_9 (\lambda_v^* + k_8)}. \]  

(20)

Substituting (19) and (20) into the expression of \( \lambda_h^* \) and \( \lambda_v^* \) and simplifying, lead the non-zero equilibria of the limited system (13) satisfy the quadratic equation

\[ d_2(\lambda_h^*)^2 + d_1 \lambda_h^* + d_0 = 0, \]  

(21)

where \( d_i, i = 0, 1, 2 \), are given by

\[ d_2 = k_2 k_8 k_9 \Lambda_h (k_{10} a \mu_h \beta_v + k_2 k_8), \]  
\[ d_1 = k_2^2 k_2^2 k_9 k_9 \Lambda_h \mu_h \left[ \mathcal{R}_c^2 - \mathcal{R}_0^2 \right], \]  
\[ d_0 = k_2^2 k_2^2 k_9 k_9 \Lambda_h \mu_h^2 \left( 1 - \mathcal{R}_0^2 \right). \]  

(22a)

(22b)

(22c)

and \( \mathcal{R}_0 \) and \( \mathcal{R}_c \) are given by (12) and (15), respectively.
This equation may be simply analysed through the Descartes’ rule of signs. First of all, note that $d_2$ is positive. Therefore the following cases are possible:

1. There is a unique endemic equilibrium if $d_0 < 0$;
2. There is a unique endemic equilibrium if

\[(d_1 < 0 \text{ and } d_0 = 0) \text{ or } (d_1 < 0 \text{ and } d_0 > 0 \text{ and } d_1^2 - 4d_2d_0 = 0);\]

(23)

3. There are two endemic equilibria if

\[d_1 < 0 \text{ and } d_0 > 0 \text{ and } d_1^2 - 4d_2d_0 > 0;\]

(24)

4. There are no endemic equilibria otherwise.

We observe that $d_0 < 0$ is equivalent to $\mathcal{R}_0 > 1$, so statement (i) of Theorem 3 is equivalent to point (a).

When $\mathcal{R}_0 = 1$, $d_0 = 0$. We observe that $d_1 < 0$ is equivalent to $\mathcal{R}_c < \mathcal{R}_0$. Therefore, when $\mathcal{R}_0 = 1$ and $\mathcal{R}_c < 1$, $d_0 = 0$ and $d_1 < 0$, so statement (ii) $a$) of Theorem 3 follows from statement (b) above. Since the condition $d_0 = 0$ does not appear elsewhere in statements (a), (b), or (c) above, statement (ii) $b$) of Theorem 3 follows from statement (d) above.

When $\mathcal{R}_0 < 1$, $d_0 > 0$, and when $\mathcal{R}_c < \mathcal{R}_0, d_1 < 0$. We also note that for $\mathcal{R}_0 < 1$, when $d_1 < 0$, $\psi \leq 0$ because $\psi > 0$ is equivalent to $d_1 > 0$. Indeed,

\[\psi > 0 \iff k_{10}a\mu_h \beta_{vh} > \delta \gamma_h k_8\]
\[\iff k_{10}a\mu_h \beta_{vh} + 2k_8k_2 - k_3k_4k_8\mathcal{R}_0^2 > \delta \gamma_h k_8 + 2k_8k_2 - k_3k_4k_8\mathcal{R}_0^2\]
\[\iff k_{10}a\mu_h \beta_{vh} + 2k_8k_2 - k_3k_4k_8\mathcal{R}_0^2 > k_8k_2 + k_8k_3k_4(1 - \mathcal{R}_0^2)\]
\[\iff k_3k_4k_8k_9\Lambda_h \mu_h \left[ k_{10}a\mu_h \beta_{vh} + 2k_8k_2 - k_3k_4k_8\mathcal{R}_0^2 \right]\]
\[> k_3k_4k_8k_9\Lambda_h \mu_h \left[ k_8k_2 + k_8k_3k_4(1 - \mathcal{R}_0^2) \right] > 0, \text{ since } \mathcal{R}_0 < 1.\]

(25)

Consequently, we show that $d_1^2 - 4d_2d_0 = 0$ is equivalent to,

\[\rho_2\mathcal{R}_0^4 + \rho_1\mathcal{R}_0^2 + \rho_0 = 0,\]

(26)

where

\[\rho_2 = k_3^4k_4^4k_8^2k_9^2\Lambda_h^2\mu_h^2,\]

(27a)

\[\rho_1 = -2k_3^2k_4^3k_8k_9^2\Lambda_h^2\mu_h^2 \left[ \delta \gamma_h (k_{10}a\mu_h \beta_{vh} + k_2k_8) - k_2(k_{10}a\mu_h \beta_{vh} - k_8\delta \gamma_h) \right],\]

(27b)

\[\rho_0 = k_3^2k_4^2k_8^2k_9k_{10}a^2\Lambda_h^2\mu_h^4\beta_{vh}^2.\]

(27c)
We again use Descartes’ rule of signs to analyse Eq. (26). The discriminant of (26) is \( \Delta_r = \rho_1^2 - 4 \rho_2 \rho_0 \), and can be written
\[
\Delta_r = -16(k_3 k_4 k_8 k_9 \Lambda h \mu h)^4 k_8^2 k_2 \delta \gamma h (k_{10} a \mu h \beta v h + k_2 k_8) \psi.
\]
Since \( \rho_2 > 0 \) and \( \rho_0 > 0 \), Eq. (26) allows real positive solutions if and only if \( \rho_1 < 0 \) and \( \Delta_r \geq 0 \). Now, we express the obtained inequalities in terms of the quantities (14–17). To this aim, we note that \( \Delta_r \geq 0 \) is equivalent to \( \psi \leq 0 \). From the definition of \( \psi \) (14) and \( \rho_1 \) (27b), this implies that \( \rho_1 < 0 \). Therefore, Eq. (26) has exactly two positive solutions given by (16) and (17). Therefore, statement (iii) \( b \) follows from statement (b) above.

Similarly, \( d_1^2 - 4d_2 d_0 > 0 \), with \( d_1 < 0 \) and \( d_0 > 0 \) written in terms of the basic reproduction number, is equivalent to
\[
R_0 < R_{1b} \quad \text{or} \quad R_0 > R_{2b},
\]
so statement (iii) \( a \) follows from statement (c) above.

Finally, statement (iii) \( c \) then follows from statement (d) above. Thus Theorem 3 is established.

**Remark 3** The proposed procedure for the proof of Theorem 3 is applicable only for a quadratic equation. By cons, if the search for equilibrium points, brings us to the resolution of a cubic polynomial, we can referred to the work of Greenhalgh and Griffiths (2009).

It is clear that case (iii) (item (a)) of Theorem 3 indicates the possibility of backward bifurcation (where the locally-asymptotically stable DFE co-exists with a locally asymptotically stable endemic equilibrium when \( R_0 < 1 \)) in the model (8). In a previous work (see Abboubakar et al. 2016b, model 18, page 9), we just showed that the model exhibited the backward bifurcation phenomenon. In the following, we provide not only a sufficient condition, but also the effective thresholds which govern this phenomenon.

### 2.2 The effective threshold for saddle-node bifurcation and region for backward bifurcation

We know from Theorem 3 that a backward bifurcation scenario is possible for model (8). Here, we characterize the critical value in terms of a single parameter, the transmission rate \( \beta_{hv} \), at which the saddle-node bifurcation occurs, i.e. the threshold for the appearance of two endemic equilibria (see Fig. 2), as well as the region in \((\beta_{vh}, \beta_{hv})\)-plane at which this phenomenon occurs (see Fig. 4).

Introducing the quantities,
\[
\bar{\beta}_{hv} = \frac{k_9 N_h^0 (k_{10} a \mu h \beta_{vh} + 2k_2 k_8)}{k_{10} k_{11} a^2 N_v^0 \beta_{vh}},
\]
Bifurcation thresholds and optimal control in transmission… 397

Fig. 2 The backward bifurcation curves for model system (8) in the \((R_0, I_h^*)\) and \((R_0, I_v^*)\) planes. The parameter \(\beta_{hv}\) varied in the range \([0, 0.2390]\) to allow \(R_0\) to vary in the range \([0, 1.5]\). Two endemic equilibrium points coexist for values of \(R_0\) in the range \((0.6764, 1)\) (corresponding to the range \((0.0486, 0.2390)\) of \(\beta_{hv}\)). Solid lines represent stable equilibria and dash lines stand for unstable equilibria.

and,

\[
\beta_{\pm} = \frac{k_9 N_0^h}{k_3 k_4 k_5 k_11 a^2 N_0^v \beta_{hv}} \left[ \sqrt{\delta \gamma_h (k_{10} a \mu_h \beta_{hv} + k_2 k_8) \pm \sqrt{(-k_2 \psi)}} \right]^2,
\]

we have the following result (see appendix E for the proof).

**Theorem 4** Assume that \(\psi < 0\), where \(\psi\) is given by (14). Then the backward bifurcation phenomenon takes place in the basic model (13) if and only if

\[
\bar{\beta}_{hv} < \beta_{hv} < \min(\beta_-, \beta_{hv}^*) \quad \text{or} \quad \max(\bar{\beta}_{hv}, \beta_+) < \beta_{hv} < \beta_{hv}^*,
\]

(31)

where \(\beta_{hv}^* = \frac{k_3 k_4 k_8 k_9 N_0^h}{a^2 \beta_{hv} k_{10} k_{11} N_0^v}\) is obtained by solving \(R_0 = 1\) in term of \(\beta_{hv}\).

The previous result is in line with the observation made by Wangari et al. (2015) (see also Greenhalgh and Griffiths 2009; Rivero-Esquivel et al. 2016 and the references therein) concerning the bifurcation thresholds of epidemiological models.
Let us define the following sets: $A_1 = \{ (\beta_{vh}, \beta_{hv}) \in [0, 1]^2 : \bar{\beta}_{hv} < \beta_{hv} \}$, $A_2 = \{ (\beta_{vh}, \beta_{hv}) \in [0, 1]^2 : \beta_{hv} < \beta_- \}$, $A_3 = \{ (\beta_{vh}, \beta_{hv}) \in [0, 1]^2 : \beta_+ < \beta_{hv} \}$, and $A_4 = \{ (\beta_{vh}, \beta_{hv}) \in [0, 1]^2 : \beta_{hv} < \beta_{hv}^* \}$.

In what follows, we designate by $\bar{A}$ the complement set of $A, C_{fi}(\beta_{vh}), i = 1, 2, 3, 4$, the representative curves of $\beta_{hv} = \bar{\beta} := f_1(\beta_{vh}), \beta_{hv} = \beta_- := f_2(\beta_{vh}), \beta_{hv} = \beta_+ := f_3(\beta_{vh})$ and $\beta_{hv} = \beta_{hv}^* := f_4(\beta_{vh})$, respectively.

Following Rivero-Esquivel et al. (2016), we claim the following result.

**Theorem 5** The number of endemic equilibrium points of the basic arboviral disease model (8) depends of the parameters $\beta_{vh}$ and $\beta_{hv}$ as follows:

1. There is a unique endemic equilibrium if $(\beta_{vh}, \beta_{hv}) \in \bar{A}_4$;
2. There is a unique endemic equilibrium if
   \[(\beta_{vh}, \beta_{hv}) \in A_1 \text{ and } (\beta_{vh}, \beta_{hv}) \in C_{f_4(\beta_{vh})} \text{ or } (\beta_{vh}, \beta_{hv}) \in A_1 \text{ and } (\beta_{vh}, \beta_{hv}) \in A_4 \text{ and } (\beta_{vh}, \beta_{hv}) \in C_{f_2(\beta_{vh})} \cup C_{f_3(\beta_{vh})}; \]
3. There are two endemic equilibria if
   \[(\beta_{vh}, \beta_{hv}) \in A_1 \text{ and } (\beta_{vh}, \beta_{hv}) \in A_4 \text{ and } (\beta_{vh}, \beta_{hv}) \in A_2 \cup A_3; \]
4. There are no endemic equilibria otherwise.

**Proof** The proof of Theorem 5 comes from the expression of conditions (23) and (24) in the proof of Theorem 3, in term of $\beta_{vh}$ and $\beta_{hv}$.

The backward bifurcation phenomenon is illustrated by numerical simulation of the model with the following set of parameter values (it should be noted that these parameters are chosen for illustrative purpose only, and may not necessarily be realistic epidemiologically): $\Lambda_h = 10$, $\beta_{vh} = 0.5$, $\eta_h = 0.78$, $\eta_v = 0.99$, $\delta = 0.1$, $\sigma = 0.01428$, $\gamma_h = 1/14$, $\gamma_v = 1/14$, $K_E = 10^4$, $K_L = K_E/2$, and varying $\beta_{hv}$. All other parameters are as in Table 2. In the particular case $\beta_{hv} = 0.08$, the conditions required by Theorem 3, case (iii), are satisfied. Note, in particular, for this set of parameters, $R_c = 0.5134 < 1$, $\psi = -0.2.3481 \times 10^{-4} < 0$, $R_{1h} = 0.0178 < 1$, $R_{2b} = 0.6764 < 1$, $R_0 = 0.8678 < 1$ (so that $\max(R_c, R_{2b}) < R_0 < 1$). It follows: $d_2 = 1.3470 \times 10^{-9} > 0, d_1 = -1.5555 \times 10^{-12} < 0$ and $d_0 = 3.2076 \times 10^{-17} > 0$, so that $d_1^2 - 4d_2d_0 = 2.2468 \times 10^{-24} > 0$. The resulting two endemic equilibria $Q^{**} = (S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, E_v^*, I_v^*)$, are:

$Q^{**} = (8513, 8508, 84, 29467, 7290, 173, 370)$, which is locally stable and $Q^{**} = (161570, 161470, 30, 10360, 7788, 15, 30)$, which is unstable.

The associated bifurcation diagram is depicted in Fig. 2. This clearly shows the coexistence of two locally-asymptotically stable equilibria when $R_0 < 1$, confirming that the model (8) (or (13)) undergoes the phenomenon of backward bifurcation.
Fig. 3 Solutions of model (13) of the number of infectious humans, $I_h$, for parameter values given in the bifurcation diagram in Fig. 2 with $\beta_{hv} = 0.08$, so $R_0 = 0.8678 < 1$, for two different set of initial conditions. The first set of initial conditions (corresponding to the dotted trajectory) is $S_h = 700, E_h = 220, I_h = 15, R_h = 60, S_v = 3000, E_v = 400, I_v = 120$. The second set of initial conditions (corresponding to the solid trajectory) is $S_h = 70000, E_h = 220, I_h = 15, R_h = 60, S_v = 3000, E_v = 400, I_v = 120$. The solution for initial condition 2 approaches the locally asymptotically stable DFE point, while the solution for initial condition 1 approaches the locally asymptotically stable endemic equilibrium.

Fig. 4 The $(\beta_{vh}, \beta_{hv})$-plane is divided into regions according to the number of positive endemic states. The backward bifurcation region is located between the red curve and the black curve, i.e. the part coloured in violet. We have set $\bar{\beta} := f_1(\beta_{ vh}), \beta_- := f_2(\beta_{ vh}), \beta_+ := f_3(\beta_{ vh})$ and $\beta_{hv}^* := f_4(\beta_{ vh})$ (colour figure online).

The occurrence of the backward bifurcation can be also seen in Fig. 3. Here, $R_0$ is less than the transcritical bifurcation threshold $R_0 = 1$ ($R_0 = 0.8678 < 1$), but the solution of the model (8) can approach either the endemic equilibrium point or the disease-free equilibrium point, depending on the initial condition. In Fig. 4, we determine the region in the $(\beta_{vh}, \beta_{hv})$-plane, in which the backward bifurcation occurs. It then follows that the region in which the backward bifurcation occurs is the region which correspond to $A_1 \cap A_2 \cap A_3 \cap A_4$, i.e. the region located between the red curve and the black curve (the part coloured in violet).
2.3 Non-existence of backward bifurcation in absence of disease-induced death

For the case $\delta = 0$, we have the following result.

**Theorem 6** (i) The model (8) without disease-induced death ($\delta = 0$) has no endemic equilibrium when $R_{0,\delta=0} \leq 1$, and has a unique endemic equilibrium otherwise. (ii) The DFE, $Q_2$, of model (8) without disease-induced death ($\delta = 0$), is globally asymptotically stable (GAS) in $\mathcal{D}$ if $R_{0,\delta=0} < 1$.

**Proof** See appendix F.

**Remark 5** For the sake of brevity we do not discuss the global stability of the endemic equilibrium, when $R_0 > 1$ in detail here. However, we note that if $\delta = 0$ the global asymptotic stability of the unique endemic equilibrium, $Q^{**}$, can be obtained by using the standard Volterra–Goh function (Ai et al. 2012; Goh 1978).

3 A model for optimal control

There are several possible interventions in order to reduce or limit the proliferation of vectors and the explosion of the number of infected humans. In addition of controls used in Moulay et al. (2012), we add vaccination and the control of adult vectors as control variables to reduce or even eradicate the spread of arboviral diseases such as dengue or chikungunya. To this aim, we introduce five time dependent controls as follows:

1. The first control $0 \leq u_1(t) \leq 1$ denotes the rate of susceptible individuals that one decides to vaccinate at time $t$. So we follow Rodrigues et al. (2014) and assume that only susceptible humans receive vaccine, implying that it is possible to distinguish between exposed and susceptible individuals and that the vaccine is effective so that all vaccinated susceptible individuals become immune (Pinho and Nogueira 2017; Rodrigues et al. 2014). It is important to note that the compartment $R_h$ includes those who have acquired natural immunity after infection, and those who have developed immunity through vaccination. Only those who have been vaccinated will lose after a while, their immunity and thus become again susceptible. So, parameter $\omega$ associated to the control $u_1(t)$ represents the waning immunity process (Rodrigues et al. 2014).

2. The second control $0 \leq u_2(t) \leq 1$ represents efforts made to protect human from mosquito bites. It mainly consists to the use of mosquito nets or wearing appropriate clothes (Blayneh et al. 2010; Moulay et al. 2012). Thus we modify the infection term as follows:

$$\lambda^c_h = (1 - u_2(t))\lambda_h, \quad \lambda^c_v = (1 - u_2(t))\lambda_v.$$  \hfill (34)

3. The third control $0 \leq u_3(t) \leq 1$ represents efforts made for treatment. This control consists of all the accompanying measures such as: the patient’s care (use of ambulances to transport patients, isolating infected patients in hospitals), installing an anti-mosquito electric diffuser in the hospital room and the administration of
proper treatment. These accompanying measures must be taken into account when we will estimate the treatment cost. But for now, we focus on the effect of drugs that permit to move a patient from $I_h$ to $R_h$. Depending on the immune response of the host, the efficacy of treatment varies from one person to another, using instance corticosteroids, paracetamol and non-steroidal anti-inflammatory drugs (Moulay et al. 2012). We assume here that people who can benefit from this type of control are those of the $I_h$ compartment. Thus we modify the recovery rate such that 

$$\sigma_c = \sigma_h + \alpha_2 u_3,$$

where $\alpha_2$ is the proportion of effective treatment (Blayneh et al. 2009; Moulay et al. 2012). We take $\alpha_2 = 0.3$ (Abboubakar et al. 2016b; Moulay et al. 2012).

4. The fourth control $0 \leq u_4(t) \leq 1$ represents mosquitoes adulticiding effort with killing efficacy $c_m$. Thus the mosquito natural mortality rate becomes $\mu^c_v = \mu_v + c_m u_4(t)$ (Blayneh et al. 2010), with $0.2 \leq c_m \leq 0.8$ (Abboubakar et al. 2016b; Dumont and Chiroleu 2010).

5. The fifth control $0 \leq u_5(t) \leq 1$ represents the effect of interventions used for the vector control. It mainly consists in the reduction of breeding sites with chemical application methods, for instance using larvicides like BTI (Bacillus Thuringensis Israelensis) which is a biological larvicide, or by introducing larvivore fish. This control focuses on the reduction of the number of eggs and larvae, of any natural or artificial water-filled container (Abboubakar et al. 2016b; Moulay et al. 2012). Thus the eggs and larvae natural mortality rate become $\mu^c_E = \mu_E + \eta_1 u_5(t)$ and $\mu^c_L = \mu_L + \eta_2 u_5(t)$ where $\eta_1 = 0.001$, $\eta_2 = 0.3$, represent eggs and larvae mortality rates induced by chemical intervention (Abboubakar et al. 2016b; Moulay et al. 2012).

Note that $0 \leq u_i \leq 1$, for $i = 1, \ldots, 5$, means that when the control is zero there is no any effort invested (i.e. no control) and when it is one, the maximum control effort is invested.

Remark 6 Although the purpose of this study is to show that the fight against arboviral diseases in general, and particularly dengue, passes by the common implementation of various existing control strategies, it is also important to note that the simultaneous implementation of these various control mechanisms may be very expensive for developing countries. Also, it is important to note that the awareness of local populations is not always effective. In addition to cultural barriers, there are people who are resistant to vaccines, for example. We also note the accessibility of epidemic surveillance zones that sometimes make it difficult the task for health professionals (Hotez et al. 2004; Kane 2015; Thonnon et al. 1999; Togora et al. 2014; Yaméogo et al. 2011).

Therefore, our optimal control model of arboviral diseases reads as

$$\dot{S}_h = \Lambda_h - [(1 - u_2(t))\lambda_h + \mu_h + u_1(t)] S_h + \omega u_1(t) R_h$$

$$\dot{E}_h = (1 - u_2(t))\lambda_h S_h - (\mu_h + \gamma_h) E_h$$

$$\dot{I}_h = \gamma_h E_h - [\mu_h + \delta + \sigma + \alpha_2 u_3(t)] I_h$$

$$\dot{R}_h = (\sigma + \alpha_2 u_3(t)) I_h + u_1 S_h - (\mu_h + \omega u_1) R_h$$

$$\dot{S}_v = \theta P - (1 - u_2(t))\lambda_v S_v - (\mu_v + c_m u_4(t)) S_v$$
\[
\dot{E}_v = (1 - u_2(t))\lambda_v S_v - (\mu_v + \gamma_v + c_m u_4(t))E_v
\]
\[
\dot{I}_v = \gamma_v E_v - (\mu_v + c_m u_4(t))I_v
\]
\[
\dot{E} = \mu_b \left(1 - \frac{E}{K_E}\right) (S_v + E_v + I_v) - (s + \mu_E + \eta_1 u_5(t))E
\]
\[
\dot{L} = sE \left(1 - \frac{L}{K_L}\right) - (l + \mu_L + \eta_2 u_5(t))L
\]
\[
\dot{P} = lL - (\theta + \mu_P)P
\]

(35)

with initial conditions given at \( t = 0 \).

It is worth mentioning that in the absence of anti-arboviral disease control mechanisms (i.e. \( u_i(t) = 0, i = 1, \ldots, 5 \)), the non-autonomous system (35) reduces to the autonomous system (8) when \( \lambda^c_v \) and \( \lambda^c_h \) are replaced by \( \lambda_h \) and \( \lambda_v \), respectively.

For the non-autonomous system (35), the rates of change of the total populations of humans and adult vectors are given, respectively, by

\[
\dot{N}_h = \Lambda_h - \mu_h N_h - \delta I_h \quad \text{and} \quad \dot{N}_v = \theta P - (\mu_v + c_m u_4(t))N_v.
\]

(36)

For bounded Lebesgue measurable controls and non-negative initial conditions, non-negative bounded solutions to the state system exist (Lukes 1982, Theorem 9.2.1).

The objective of control is to minimize the number of cases, i.e., the number of symptomatic humans infected (that is, to reduce sub-population \( I_h \)), while keeping the costs of the control as low as possible.

To achieve this objective we must incorporate the relative costs associated with each policy (control) or combination of policies directed towards controlling the spread of arboviral diseases. We define the objective function as

\[
J(u_1, u_2, u_3, u_4, u_5) = \int_0^{t_f} \left[ D_1 I_h(t) + \sum_{i=1}^{5} B_i u_i^2(t) \right] dt
\]

(37)

and the control set

\[
\Delta = \{(u_1, u_2, u_3, u_4, u_5)|u_i(t) \text{ is Lebesgue measurable on } [0, t_f],
\]

\[
0 \leq u_i(t) \leq 1, i = 1, \ldots, 5\}.
\]

The first term in the integrand \( J \) represent benefit of \( I_h \) populations. The quantity \( D_1 \) represents the weight constant of symptomatic humans. Positive constants \( B_i, i = 1, \ldots, 5 \), are costs for vaccination, individual protection (human), treatment and vector control effort respectively, which regularize the optimal control. Although the Quadratic State Independent costs (QSI) (Kassa and Hove-Musekwa 2014) do not depend on the number of people on whom we have to apply the control mechanisms, we turn our choice on this type of controls for the simple reason that they are the most used (see Adams et al. 2004; Blayneh et al. 2009, 2010; Buonomo 2011, 2015; Djombe Njankou and Nyabadza 2016; Jung et al. 2002; Moulay et al. 2012; Rodrigues et al. 2014; Schättler and Ledzewicz 2012; Dias and Wanner 2015; Yusuf and Benyah
2012; Zaman et al. 2008) and make the problem convex and thus guarantee that a unique solution exists. See also Kassa and Hove-Musekwa (2014) for other cost function types.

We solve the problem using optimal control theory.

**Theorem 7** Let \( X = (S_h, E_h, I_h, R_h, S_v, E_v, I_v, E, L, P)^t \). The following set

\[
D_1 = \left\{ X \in \mathbb{R}_+^{10} : N_h \leq \frac{\Lambda_h}{\mu_h}; E \leq K_E; L \leq K_L; P \leq \frac{lK_L}{k_7}; N_v \leq \frac{0lK_L}{k_7k_8} \right\}
\]

is positively invariant under system (35).

**Proof** On the one hand, we can easily see that it is possible to get,

\[
\begin{align*}
\dot{S}_h &\geq - (\lambda_h + \mu_h) S_h, \\
\dot{E}_h &\geq - (\mu_h + \gamma_h) E_h, \\
\dot{I}_h &\geq - (\mu_h + \delta + \sigma) I_h, \\
\dot{R}_h &\geq - \mu_h R_h, \\
\dot{E} &\geq - \left( \frac{\mu_b}{K_E} + s + \mu_E + \eta_1 \right) E, \\
\dot{L} &\geq - \left( \frac{s}{K_L} + l + \mu_L + \eta_2 \right) L, \\
\dot{P} &\geq - (\theta + \mu_p + \eta_3) P, \\
\dot{S}_v &\geq - (\lambda_v + \mu_v) S_v, \\
\dot{E}_v &\geq - (\mu_v + \gamma_v) E_v, \\
\dot{I}_v &\geq - \mu_v I_v,
\end{align*}
\]

for \((S_h(0), E_h(0), I_h(0), R_h(0), E(0), A(0), P(0), S_v(0), E_v(0), I_v(0)) \geq 0\). Thus, solutions with initial value in \( \Omega \) remain nonnegative for all \( t \geq 0 \). On the other hand, we have

\[
\begin{align*}
\dot{N}_h &\leq \Lambda_h - \mu_h N_h, \quad \text{(39a)} \\
\dot{N}_v &\leq \theta P - \mu_v N_v, \quad \text{(39b)} \\
\dot{E} &\leq \mu_b \left( 1 - \frac{E}{K_E} \right) (S_v + E_v + I_v) - (s + \mu_E) E, \quad \text{(39c)} \\
\dot{L} &\leq sE \left( 1 - \frac{L}{K_L} \right) - (l + \mu_L) L, \quad \text{(39d)} \\
\dot{P} &\leq lL - (\theta + \mu_p) P. \quad \text{(39e)}
\end{align*}
\]

The right hand side of the inequalities (39) correspond to the transmission model without control, and it is easy to show that solutions remain in \( D_1 \). Then using Gronwall’s inequality, we deduce that solutions of (35) are bounded.
3.1 Existence of an optimal control

The existence of an optimal control can be obtained by using a result of Fleming and Rishel (1975, Theorem 4.1., page 68).

**Theorem 8** Consider the control problem with system (35).

There exists \( u^\star = (u_1^\star, u_2^\star, u_3^\star, u_4^\star, u_5^\star) \) such that

\[
\min_{(u_1, u_2, u_3, u_4, u_5)\in \Delta} J(u_1, u_2, u_3, u_4, u_5) = J(u_1^\star, u_2^\star, u_3^\star, u_4^\star, u_5^\star)
\]

**Proof** To use an existence result, Theorem III.4.1 from Fleming and Rishel (1975, Theorem 4.1., page 68) we must check if the following properties are satisfied:

1- the set of controls and corresponding state variables is non empty;
2- the control set \( \Delta \) is convex and closed;
3- the right hand side of the state system is bounded by a linear function in the state and control;
4- the integrand of the objective functional is convex;
5- there exist constants \( c_1 > 0, c_2 > 0 \), and \( \beta > 1 \) such that the integrand of the objective functional is bounded below by \( c_1 \left( \sum_{i=1}^{5} |u_i|^2 \right)^{\frac{\beta}{2}} - c_2 \).

In order to verify these properties, we use a result from Lukes (1982, Theorem 9.2.1) to give the existence of solutions for the state system (35) with bounded coefficients, which gives condition 1. Since by definition, the control set \( \Delta \) is bounded, then condition 2 is satisfied. The right hand side of the state system (35) satisfies condition 3 since the state solutions are bounded. The integrand of our objective functional is clearly convex on \( \Delta \), which gives condition 4. There are \( c_1 > 0, c_2 > 0 \), and \( \beta > 1 \) satisfying

\[
D_1 I_h + \sum_{i=1}^{5} B_i u_i^2 \geq c_1 \left( \sum_{i=1}^{5} |u_i|^2 \right)^{\frac{\beta}{2}} - c_2,
\]

because the states variables are bounded. Thus condition 5 is satisfied. We conclude that there exists an optimal control \( u^\star = (u_1^\star, u_2^\star, u_3^\star, u_4^\star, u_5^\star) \) that minimizes the objective functional \( J(u_1, u_2, u_3, u_4, u_5) \).

\[\Box\]

3.2 Characterization of an optimal control

The necessary conditions that an optimal control must satisfy come from the Pontryagin’s maximum principle (PMP) (Pontryagin et al. 1962). This principle converts (35–37) into a problem of minimizing point wise a Hamiltonian \( \mathbb{H} \), with respect to \( (u_1, u_2, u_3, u_4, u_5) \):

\[
\mathbb{H} = D_1 I_h + \sum_{i=1}^{5} B_i u_i^2 + \lambda_S \{ \Lambda_h - [(1 - u_2)\lambda_h + \mu_h + u_1] S_h + \omega u_1 R_h \}
\]

\[
+ \lambda_E \{ (1 - u_2)\lambda_h S_h - (\mu_h + \gamma_h) E_h \}
\]

\[
+ \lambda_{I_h} \{ \gamma_h E_h - [\mu_h + \delta + \sigma + \alpha_2 u_3] I_h \}
\]

\[
+ \lambda_{R_h} \{ (\sigma + \alpha_2 u_3) I_h + u_1 S_h - (\mu_h + \omega u_1) R_h \}
\]
where the $\lambda_i, i = S_h, E_h, I_h, R_h, S_v, E_v, I_v, E, L, P$ are the adjoint variables or co-state variables. Applying Pontryagin’s maximum principle (Pontryagin et al. 1962), we obtain the following result.

**Theorem 9** Given an optimal control $u^* = (u_1^*, u_2^*, u_3^*, u_4^*, u_5^*)$ and solutions $(S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, E_v^*, I_v^*, A^*, P^*)$ of the corresponding state system (35), there exist adjoint variables $\Pi = (\lambda_{S_h}, \lambda_{E_h}, \lambda_{I_h}, \lambda_{R_h}, \lambda_{S_v}, \lambda_{E_v}, \lambda_{I_v}, \lambda_E, \lambda_L, \lambda_P)$ satisfying:

\[
\frac{d\lambda_{S_h}}{dt} = \mu_h \lambda_{S_h} + u_1 (\lambda_{S_h} - \lambda_{R_h}) + (1 - u_2) \lambda_h \left(1 - \frac{S_h}{N_h}\right) (\lambda_{S_h} - \lambda_{E_h}) + (1 - u_2) \frac{S_v \lambda_v}{N_h} (\lambda_{E_v} - \lambda_{S_v})
\]

\[
\frac{d\lambda_{E_h}}{dt} = \mu_h \lambda_{E_h} + \gamma_h (\lambda_{E_h} - \lambda_{I_h}) + (1 - u_2) \frac{S_h \lambda_h}{N_h} (\lambda_{E_h} - \lambda_{S_h}) + (1 - u_2) \frac{S_v}{N_h} (a \beta_v h \eta_v - \lambda_v) (\lambda_{S_v} - \lambda_{E_v})
\]

\[
\frac{d\lambda_{I_h}}{dt} = -D_1 + [\mu_h + \delta] \lambda_{I_h} + (\sigma + \alpha v u_3) (\lambda_{I_h} - \lambda_{R_h}) + (1 - u_2) \frac{S_h \lambda_h}{N_h} (\lambda_{E_h} - \lambda_{S_h}) + (1 - u_2) \frac{S_v}{N_h} (a \beta_v h \eta_v - \lambda_v) (\lambda_{S_v} - \lambda_{E_v})
\]

\[
\frac{d\lambda_{R_h}}{dt} = \mu_h \lambda_{R_h} + \omega u_1 (\lambda_{R_h} - \lambda_{S_h}) + (1 - u_2) \frac{S_h \lambda_h}{N_h} (\lambda_{E_h} - \lambda_{S_h}) + (1 - u_2) \frac{S_v \lambda_v}{N_h} (\lambda_{E_v} - \lambda_{S_v})
\]

\[
\frac{d\lambda_{S_v}}{dt} = (\mu_v + c_m u_4) \lambda_{S_v} + (1 - u_2) \lambda_v (\lambda_{S_v} - \lambda_{E_v}) - \mu_b \left(1 - \frac{E}{K_E}\right) \lambda_E
\]

\[
\frac{d\lambda_{E_v}}{dt} = (\mu_v + c_m u_4) \lambda_{E_v} + \gamma_v (\lambda_{E_v} - \lambda_{I_v}) + a \eta_v \beta_v h v (1 - u_2) (\lambda_{S_h} - \lambda_{E_h}) \frac{S_h}{N_h} - \mu_b \left(1 - \frac{E}{K_E}\right) \lambda_E
\]
\[
\frac{d\lambda_{E}}{dt} = \left(\frac{\mu_{b}}{K_{E}}N_{v} + s + \mu_{E} + \eta_{1}u_{5}\right)\lambda_{E} - s \left(1 - \frac{L}{K_{L}}\right)\lambda_{L}
\]
(48)
\[
\frac{d\lambda_{L}}{dt} = \left[\frac{s}{K_{L}}E + \mu_{L} + l + \eta_{2}u_{5}\right]\lambda_{L} - l\lambda_{P}
\]
(49)
\[
\frac{d\lambda_{P}}{dt} = (\mu_{P} + \theta)\lambda_{P} - \theta\lambda_{S_{v}}
\]
(50)

and the transversality conditions
\[
\lambda_{i}^{*}(t_f) = 0, \quad i = 1, \ldots, 10.
\]
(51)

Furthermore,
\[
u_{1}^{*} = \min \left\{ 1, \max \left( 0, \frac{(S_{h} - \omega R_{h})(\lambda_{S_{h}} - \lambda_{R_{h}})}{2B_{1}} \right) \right\},
\]
\[
u_{2}^{*} = \min \left\{ 1, \max \left( 0, \frac{\lambda_{h}S_{h}(\lambda_{E_{h}} - \lambda_{S_{h}}) + \lambda_{v}S_{v}(\lambda_{E_{v}} - \lambda_{S_{v}})}{2B_{2}} \right) \right\},
\]
\[
u_{3}^{*} = \min \left\{ 1, \max \left( 0, \frac{\alpha_{2}(\lambda_{I_{h}} - \lambda_{R_{h}})I_{h}}{2B_{3}} \right) \right\},
\]
\[
u_{4}^{*} = \min \left\{ 1, \max \left( 0, \frac{cm[S_{h}\lambda_{S_{h}} + E_{v}\lambda_{E_{v}} + I_{v}\lambda_{I_{v}}]}{2B_{4}} \right) \right\},
\]
\[
u_{5}^{*} = \min \left\{ 1, \max \left( 0, \frac{\eta_{1}E\lambda_{E} + \eta_{2}L\lambda_{L}}{2B_{5}} \right) \right\},
\]
(52)

Proof The differential equations governing the adjoint variables are obtained by differentiation of the Hamiltonian function, evaluated at the optimal control. Then the adjoint system can be written as
\[
\frac{d\lambda_{S_{h}}}{dt} = -\frac{\partial H}{\partial S_{h}}, \quad \frac{d\lambda_{E_{h}}}{dt} = -\frac{\partial H}{\partial E_{h}}, \quad \frac{d\lambda_{I_{h}}}{dt} = -\frac{\partial H}{\partial I_{h}}, \quad \frac{d\lambda_{R_{h}}}{dt} = -\frac{\partial H}{\partial R_{h}},
\]
\[
\frac{d\lambda_{S_{v}}}{dt} = -\frac{\partial H}{\partial S_{v}}, \quad \frac{d\lambda_{E_{v}}}{dt} = -\frac{\partial H}{\partial E_{v}}, \quad \frac{d\lambda_{I_{v}}}{dt} = -\frac{\partial H}{\partial I_{v}},
\]
\[
\frac{d\lambda_{E}}{dt} = -\frac{\partial H}{\partial E}, \quad \frac{d\lambda_{L}}{dt} = -\frac{\partial H}{\partial L}, \quad \frac{d\lambda_{P}}{dt} = -\frac{\partial H}{\partial P},
\]

with zero final time conditions (transversality).

To get the characterization of the optimal control given by (52), we follow Lenhart and Workman (2007), Rodrigues et al. (2014) and solve the equations on the interior of the control set,
\[
\frac{\partial H}{\partial u_{i}} = 0, \quad i = 1, \ldots, 5.
\]
Table 3 Parameter values used in the numerical simulations of the optimal control model

| Parameter | Value       | Parameter | Value       | Parameter | Value       | Parameter | Value       |
|-----------|-------------|-----------|-------------|-----------|-------------|-----------|-------------|
| $\mu_v$   | $1/30$      | $l$       | 0.5         | $\alpha_2$| 0.3         | $\gamma_h$| $1/17$      |
| $a$       | 1           | $\mu_E$  | 0.2         | $\mu_h$  | $1/67+6365$| $\gamma_v$| $1/10$      |
| $A_h$     | 2.5         | $\mu_b$  | 6           | $\theta$ | 0.08        | $\mu_P$  | 0.4         |
| $\beta_{hv}$ | 0.75       | $\omega$ | 0.05        | $\mu_L$  | 0.4         | $\delta$ | $10^{-3}$   |
| $K_E$     | 10,000      | $s$      | 0.7         | $\eta_1$ | 0.001       | $\eta_2$ | 0.3         |
| $K_L$     | 5000        | $\eta_h$ | 0.35        | $c_m$    | 0.2         |           |             |

Using the bounds on the controls, we obtain the desired characterization. This ends the proof.

4 Numerical simulations and discussion

The simulations were carried out using the values of Table 3. We use an iterative scheme to solve the optimality system. We first solve the state equations (35) with a guess for the controls over the simulated time using fourth order Runge–Kutta scheme. Then, we use the current iterations solutions of the state equation to solve the adjoint Eqs. (41–50) by a backward fourth order Runge–Kutta scheme. Finally, we update the controls by using a convex combination of the previous controls and the value from the characterizations (52) (see e.g. Buonomo 2011; Lenhart and Workman 2007; Moulay et al. 2012; Okosun et al. 2011; Zaman et al. 2008).

In the formulation of our optimal control model (35), we have introduced new variables such as $\omega, \alpha_2, c_m, \eta_1$ and $\eta_2$. The parameter $\omega$ related to the waning immunity is taken in Rodrigues et al. (2014) ($0 \leq \omega \leq 0.5$). Since the treatment is symptomatic and its efficiency largely depends on the immune response of the host, it is more realistic to take $\alpha_2$, the recovery rate induced by treatment, less than unity ($0.1 \leq \alpha_2 \leq 0.8$). So, we follow Moulay et al. (2012), and assumed that $\alpha_2 = 0.3$. Nowadays, Deltamethrin is the most used insecticide for impregnation of bed nets, because it is a highly effective compound on mosquitoes, and this, at of very low doses. However, its use over a long period and continuously, leads to strong resistance of the wild populations of Aedes aegypti, for example Abboubakar et al. (2016b), Dushoff et al. (1998), Darriet et al. (2007). To be more realistic, we will consider that the mortality of the mosquitoes after spraying varied between 20 and 80% Abboubakar et al. 2016b; Dumont and Chiroleu 2010). In this work we use $c_m = 0.2$ (Moulay et al. 2012). The duration of a conventional larvicide Bti (Bacillus thuringiensis var. israelensis) strongly depend on several factors like water quality, exposure, and even the type of breeding sites. Also, eggs of certain populations of vectors such as Aedes albopictus, come into prolonged hibernation when conditions in the breading sites are not conducive to their good growth. So, we assumed that $\eta_1 = 0.001$ and $\eta_2 = 0.3$ (Abboubakar et al. 2016b; Moulay et al. 2012). Table 3 gives the complete list of parameter values using in the numerical simulations.
It is important to note that the values attributed to various parameters in the objective function $J \ (37)$ are only for illustration. Indeed, it is almost impossible for us to say, for example, how many vaccinations per unit of time can be given (and the cost of individual vaccination), the price of taking into hospital care of a patient, how do we get the bednets where they are needed. Nevertheless, with regard to protective measures such as the use of Long lasting insecticide-treated bed nets (LLIN) and Indoor residual spraying (IRS), we refer to the work of Stuckey et al. (2014) (see Table 4). Regarding the price of dengue vaccine Dengvaxia®, we refer to the French newspaper Le Figaro, in which the heads of the laboratory SANOFI estimated at 20 Euro ($\approx 21.18$US) the price of a dose of this vaccine to the Phillipines (lefigaro, Dengue: vaccination de masse aux Philippines. 2 The above information is summarized in Table 4. Table 5 gives the initials conditions of state variables. Table 6 gives the different strategies evaluated in this work. We simulated the system (35) in a period of 400 days ($t_f = 400$ days). Figure 5 shows the optimal control profiles.

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2 http://www.lefigaro.fr/flash-eco/2016/04/04/97002-20160404FILWWW00165-dengue-campagne-de-vaccination-de-masse-aux-philippines.php, Updated on 04/04/2016 at 16:00 Published on 04/04/2016 at 15:39. Accessed 2017-02-24.
4.1 The effect of control on the occurrence of backward bifurcation

In Sects. 2.1 and 2.1.2, we have concluded that the backward bifurcation phenomenon occurs in our basic model (3) (Theorems 3 and 4). However, we claimed that the disease induced death rate, $\delta$, is the principal cause of this phenomenon (see also Abboubakar et al. 2016b). Since the decrease of the disease-induced death rate, $\delta$, is related to the control $u_3(t)$ (the care of patients and the use of effective treatment), it is important to know if the backward bifurcation interferes too, when we apply this control. To this aim, we plot, with the same parameter values and the same initial conditions, as in Figs. 2 and 3, the number of infectious humans. The result is displayed in Fig. 6. Although treatment allows reducing the number of infectious humans, it follows from Fig. 6, that the use of optimal treatment can not be sufficient to remove the backward bifurcation phenomenon (magenta dotted line in Fig. 6). But, if we combine treatment with optimal vaccination, the backward bifurcation disappears (solid line in Fig. 6).

4.2 The effect of control in infectious humans and vector populations

As the purpose of our study is to seek the best combination linking vaccination to other control mechanism, we will just determine the best strategy among the strategies listed
Fig. 6 Simulation results of optimal control model (35) showing the effect of using optimal treatment, \( u_3(t) \), on the occurrence of the backward bifurcation phenomenon. The set of parameter values is \( \Lambda_h = 10, \beta_{vh} = 0.5, \eta_h = 0.78, \eta_v = 0.99, \delta = 0.1, \sigma = 0.01428, \gamma_h = 1/14, \gamma_v = 1/14, K_E = 10^4, \)
\( K_L = K_E/2, \beta_{hv} = 0.08, \omega = 0, D_1 = 1 \) and the other values of parameter are in Tables 3 and 4

in Table 6. Therefore, we will distinguished the six control strategies listed in Table 6, as follows:

(i) \( Z_1: \) Individual protection combined with treatment, adulticide and larvicide Here, we combine optimal individual protection, \( u_2 \), with, optimal treatment, \( u_3 \), optimal usage of adulticide, \( u_4 \) and optimal usage of larvicide, \( u_5 \), in one strategy to minimise the objective function \( J (37) \), while the control related to human vaccination is set to zero (\( u_1 = 0 \)). The result is displays on Fig. 7. It follows that the control strategy resulted in a decrease in the number of infected humans (\( I_h \)) while an increase is observed in the number of infected humans (\( I_h \)) in strategy without control. The use of this combination have also a great impact on the decreasing total vector population (\( N_v \)), as well as aquatic vector populations (\( E \) and \( L \)).

(ii) \( Z_2: \) Vaccination combined with individual protection, treatment and adulticide With this strategy, only the combination of the control \( u_1 \) on vaccination, the control \( u_2 \) on individual protection, the control \( u_3 \) on treatment and the control \( u_4 \) on adulticide, is used to minimise the objective function \( J (37) \), while the control \( u_5 \) is set to zero. On Fig. 8, we observed that the control strategy resulted in a decrease in the number of infected humans (\( I_h \)) while an increase is observed in the number of infected humans (\( I_h \)) in strategy without control. The use of this combination have also a great impact on the decreasing total vector population (\( N_v \)), as well as aquatic vector populations (\( E \) and \( L \)).

(iii) \( Z_3: \) Vaccination combined with individual protection, treatment and larvicide With this strategy, only the combination of the control \( u_1 \) on vaccination, the control \( u_2 \) on individual protection, the control \( u_3 \) on treatment and the control \( u_5 \) on larvicide, is used to minimise the objective function \( J (37) \), while the control \( u_4 \) is set to zero. On Fig. 9, we observed that the control strategy resulted in a decrease in the number of infected humans (\( I_h \)) while an increase is observed in the number of infected humans (\( I_h \)) in strategy without control. The use of this combination have a small impact on the decreasing total vector population (\( N_v \)), as well as aquatic vectors (\( E \) and \( L \)).
Fig. 7  Simulation results of optimal control model (35) showing the effect of using optimal individual protection combined with treatment, adulticide and larvicidal ($u_2 \neq 0, u_3 \neq 0, u_4 \neq 0, u_5 \neq 0$)

(iv) $Z_4$: Vaccination combined with treatment, adulticide and larvicidal

With this strategy, only the combination of the control $u_1$ on vaccination, the control $u_3$ on treatment, the control $u_4$ on adulticide and the control $u_5$ on larvicidal, is used to minimise the objective function $J$ (37), while the control $u_2$ is set to zero. On Fig. 10, we observed that the control strategy resulted in a decrease in the number of infected humans ($I_h$) while an increase is observed in the number of infected humans ($I_h$) in strategy without control. The use of this combination have a considerable impact on the decreasing total vector population ($N_v$), as well as aquatic vector populations ($E$ and $L$).

(v) $Z_5$: Vaccination combined with individual protection, adulticide and larvicidal

With this strategy, only the combination of the control $u_1$ on vaccination, the control $u_2$ on individual protection, the control $u_4$ on adulticide and the control $u_5$ on larvicidal, is used to minimise the objective function $J$ (37), while the control $u_4$ is set to zero. On Fig. 11, we observed that the control strategy resulted in a decrease in the number of
infected humans \((I_h)\) while an increase is observed in the number of infected humans \((I_h)\) in strategy without control. The use of this combination have a great impact on the decreasing total vector population \((N_v)\), as well as aquatic vector populations \((E\) and \(L\)).

Figures 7, 8, 10 and 11 have the same shape, which it is difficult to say which is the best control strategy. In the following, we perform an efficiency analysis to determine the best strategy in terms of efficiency.

4.3 Efficiency analysis

To compare different control strategies listed above (see Table 6), we perform an efficiency analysis which will allows us to determine the best control strategy (Carvalho
et al. 2015; Dumont and Chiroleu 2010; Yang and Ferreira 2008). In line with Carvalho et al. (2015), Dumont and Chiroleu (2010) and Yang and Ferreira (2008), we compare the effects of possible different strategies on the reduction of the number of cases (infectious humans \( I_h \)) following infection by an arboviral disease, by the introduction of the efficiency index, designated by \( F \). To this aim, we define the variable \( A \) as the area comprised between the curve of the symptomatic infectious human \( (I_h) \) population size, for instance, and the time axis during the period of time from \( 0 \) to \( t_f \), as

\[
A = \int_{0}^{t_f} I_h(t) \, dt, 
\]

which measures the cumulated number of infectious human during the time interval \( [0, t_f] \) (Carvalho et al. 2015; Dumont and Chiroleu 2010; Yang and Ferreira 2008). Hence the efficiency index, \( F \), be can defined by
Fig. 10  Simulation results of optimal control model (35) showing the effect of using optimal vaccination combined with treatment, adulticide and larvicide ($u_1 \neq 0$, $u_3 \neq 0$, $u_4 \neq 0$, $u_5 \neq 0$)

Table 7  Effectiveness index

| Strategies | $\mathcal{A}_h^c$ | $F$ (%) | Strategies | $\mathcal{A}_h^c$ | $F$ (%) |
|------------|-------------------|--------|------------|-------------------|--------|
| No control | 13,561            | 0      | Z3         | 2990              | 77.951 |
| Z1         | 1844              | 86.402 | Z4         | 1433              | 89.433 |
| Z2         | 801               | 94.093 | Z5         | 2064              | 84.78  |

$$F = \left( 1 - \frac{\mathcal{A}_h^c}{\mathcal{A}_h^{(0)}} \right) \times 100,$$  \hspace{1cm} (54)

where $\mathcal{A}_h^c$ and $\mathcal{A}_h^{(0)}$ are the cumulated number of infectious human with and without the different control mechanisms, respectively. So, It follows that the best strategy will be
the one whom efficiency index will be the biggest (Carvalho et al. 2015; Dumont and Chiroleu 2010; Yang and Ferreira 2008).

Using the above simulation results, we obtain the Table of effectiveness index (Table 7).

From Table 7, it follows that the combination which permit to reduce the number of cases, is the vaccination combined with individual protection, treatment and adulticide, i.e $Z_2$, following by $Z_4$, the optimal vaccination combined with treatment, adulticide and larvicide.

5 Conclusion

In this paper, we derived and analysed a model for the control of arboviral diseases with non linear form of infection and complete stage structured model for vectors, and
which we take into account a vaccination with waning immunity, treatment, individual protection and vector control strategies (use of adulticides to reduce population of adult vectors, use of larvicides to reduce eggs and larvae).

We have begun by focus on the vector population dynamics. We calculated the net reproductive number $N$ and proved the global asymptotic stability of the two equilibrium points of the corresponding model, using Lyapunov methods. Indeed, we have shown that if the net reproductive number, $N \leq 1$, the trivial equilibrium, $E_0$, is globally asymptotically stable. And when $N > 1$, the trivial equilibrium is unstable and, the non-trivial equilibrium $E_1$, is globally asymptotically stable. Then, we computed the basic reproduction number of the basic model (the complete model without control), $R_0$, and investigate the existence and stability of equilibria. The stability analysis revealed that for $N \leq 1$, the trivial equilibrium, $Q_1$, is globally asymptotically stable. When $N > 1$ and $R_0 < 1$, the disease-free equilibrium, $Q_2$, is locally asymptotically stable. We have found that the model exhibits backward bifurcation. The epidemiological implication of this phenomenon is that for effective eradication and control of diseases, $R_0$ should be less than a critical values less than one. For the backward bifurcation phenomenon, we have explicitly derived threshold conditions in term of the the basic reproduction number and in term of the transmission rate, $\beta_{hv}$. Then, we determined the region in the $(\beta_{vh}, \beta_{hv})$-plane on which these phenomenon occurs. We have proved, that the disease-induced death is the principal cause of the occurrence of backward bifurcation phenomenon in the basic model.

We then considered five time dependent controls as a way out, to ensure a considerably decreasing of the spread of the disease. We performed optimal control analysis of the model. In this light, we addressed the optimal control by deriving and analysing the conditions for optimal eradication of the disease and in a situation where eradication is impossible, reduce considerably the spread of the disease.

We perform numerical studies to validate our qualitative results. The effect of control efforts on the occurrence of backward bifurcation phenomenon is then investigated. We concluded, via numerical simulations, that the only use of optimal treatment, is not sufficient to remove this phenomenon. But, if we add some other control like vaccination, the backward bifurcation disappears. Finally, we assess the impact of different combination of control on the reduction of symptomatic infectious humans, the total number of adult vectors, eggs and larvae. From these numerical simulations and efficiency analysis, we concluded that the fight against arboviral diseases passes through the simultaneous implementation of various existing control mechanisms.

However, this conclusion must be taken with caution because of the uncertainties around the parameter values and to the budget/resource limitation. In addition, the utilization of a vaccine of small efficacy could have a negative impact on the health of the population. The results of clinical trials, in which involved more than 40,000 volunteers, did have not raised uncertainties about the impact of the vaccine in human populations (Le Monde Economie 2015). Also, in developing countries, the implementation of these controls is confronted to some constraints such as financial and material resources limited, sociological and cultural barriers that sometimes make difficult the task of health workers in vaccination campaigns, and treatment, access to areas of epidemics.
Besides being good means of protection against diseases, vaccines also are, generally, less expensive than setting up all the mechanisms which permit to treat individuals presenting different symptoms of certain diseases. Regarding the vaccine against dengue on sale in some emerging countries (Mexico, Brazil, Venezuela, Philippines), some questions still remain unanswered. Indeed, the fact that sequential infections with different strains can cause severe forms of the disease must be taken into account. Also, it is not currently known if a vaccinated individual, and for who the efficacy for a given strain is small, can develop a severe form of the disease when coming into contact with such an other strain. Although its efficiency is higher in children 9–16 years (two thirds are immune) and in individuals who have already been infected, the dengue vaccine increased hospitalizations among children less than 9 years (Hadinegoro et al. 2015; World Health Organization 2016).

Therefore, pending the completion of Phase III trials on the efficacy of the vaccine against dengue (Dengvaxia®), and therefore its acceptance by public health organizations such as the World Health Organisation (WHO) and the Centre for Disease Control (CDC), it is important to focus on other control mechanisms. That is why it is better for developing countries to increase their efforts to implement existing prevention mechanisms which already done their evidence in several countries, such as personal protection, education campaigns, as well as vector control mechanisms. It is also important to note that the implementation of vector control strategies should not be the only problem of health officials. Indeed, it has been shown that the involvement of health personnel in the destruction of vector breeding sites, as well as in the spraying of homes, despite having contributed to a considerable decline of transmission of chikungunya virus, is accompanied in a long term, by a decreased alertness of local populations (Dumont and Thuilliez 2016).

All simulated intervention combinations in this work, can be considered cost-effective in the context of available resources for health in countries affected by arboviruses. These results have the potential to help managers control programs against arbovirus infections in high endemicity countries by modifying the implementation of current interventions, or by adding new control mechanisms.

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A Proof of Lemma 1

Consider the Lyapunov function

\[ V(N_v, E, L, P) = \frac{\mu b s}{k_5 k_8} N_v + \frac{s}{k_5} E + L + \frac{\mu b s \theta l}{k_5 k_6 k_7 k_8} P. \]
with Lyapunov derivative,

\[
\frac{dV}{dt} = \frac{\mu_b s}{k_5 k_8} \dot{N}_v + \frac{s}{k_5} \dot{E} + \dot{L} + \frac{\mu_b s \theta}{k_5 k_7 k_8} \dot{P} \\
= \frac{\mu_b s}{k_5 k_8} (\theta - k_8 N_v) + \frac{s}{k_5} (\mu_b \left(1 - \frac{E}{K_E}\right) N_v - k_5 E) + sE \left(1 - \frac{L}{K_L}\right) \\
- k_6 L + \frac{\mu_b s \theta}{k_5 k_7 k_8} (\theta - k_7 P) \\
= -\frac{s}{k_5 K_E} E N_v - \frac{s}{K_L} E L + k_6 (N - 1) L.
\]

It follows, for \(N < 1\), that \(\frac{dV}{dt} \leq 0\). Thus, from Lyapunov theory, we deduce that \(E_0 = (0, 0, 0, 0)\) is GAS in \(\Omega\), if and only if \(N \leq 1\).

**B Proof of Lemma 2**

Setting \(Y = X - E_1\) with \(X = (E, L, P, N_v)^t\), we can rewrite (9) in the following manner

\[
\frac{dY}{dt} = B(Y) Y. 
\]  

where

\[
B(Y) = \begin{pmatrix}
-(k_5 + \frac{\mu_b}{K_E} N_0^0) & 0 & 0 & \mu_b \left(1 - \frac{E}{K_E}\right) \\
&s \left(1 - \frac{L}{K_L}\right) & -(k_6 + \frac{s}{K_L} E^* ) & 0 & 0 \\
0 & l & -k_7 & 0 \\
0 & 0 & \theta & -k_8
\end{pmatrix}
\]

It is clear that \(Y = (0, 0, 0, 0)^t\) is the only equilibrium. Then it suffices to consider the following Lyapunov function \(\dot{L}(Y) = \langle g, Y \rangle\) were \(g = \left(\frac{k_8}{\mu_b}, \frac{l \theta K_L}{k_7 (k_6 K_L + s E^*)}, \frac{\theta}{k_7}, 1\right)\). Straightforward computations lead that

\[\dot{L}(Y) = \langle g, \dot{Y} \rangle \overset{\text{def}}{=} \langle g, B(Y) Y \rangle \]

\[= -k_8 \frac{E}{K_E} Y_4 - \frac{s l \theta K_L}{k_7 (k_6 K_L + s E^*) K_L} Y_1.
\]

We have \(\dot{L}(Y) < 0\) if \(N > 1\) and \(\dot{L}(Y) = 0\) if \(Y_i = 0, i = 1, 2, \ldots, 4\) (i.e \(E = E^*, L = L^*, P = P^*\) and \(N_v = N_v^0\)). Moreover, the maximal invariant set contained in \(\mathcal{L}|\dot{L}(Y) = 0\) is \(\{0, 0, 0, 0\}^t\). Thus, from Lyapunov theory, we deduce that \((0, 0, 0, 0)^t\) and thus, \(E_1\), is GAS if and only if \(N > 1\).
C Proof of Theorem 1

The Jacobian matrix of \( f = (\dot{S}_h, \dot{E}_h, \dot{I}_h, \dot{R}_h, \dot{S}_v, \dot{E}_v, \dot{I}_v, \dot{E}, \dot{L}, \dot{P})^T \) at the trivial equilibrium is given by

\[
D_f(Q_1) = \begin{pmatrix}
-k_1 & 0 & 0 & 0 & -a_{hv} & \nu_v & -a_{hv} & 0 & 0 & 0 \\
0 & -k_3 & 0 & 0 & a_{hv} & \nu_v & a_{hv} & 0 & 0 & 0 \\
0 & \gamma_v & -k_4 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & \sigma & -k_5 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & -k_8 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & \mu_v & -k_9 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & \mu_v & \mu_v & \mu_v & -k_5 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & s & -k_6 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & l & -k_7 \\
\end{pmatrix}
\]  
(56)

The characteristic polynomial of \( D_f(Q_1) \) is given by:

\[
P(\lambda) = (\lambda - k_1)^2(\lambda - k_3)(\lambda - k_4)(\lambda - k_9)(\lambda - k_8)\phi_1(\lambda)
\]

where \( \phi_1(\lambda) = \lambda^4 + A_1\lambda^3 + A_2\lambda^2 + A_3\lambda + A_4 \), with \( A_1 = \mu_v + k_7 + k_6 + k_5 \), \( A_2 = (k_7 + k_6 + k_5)\mu_v + (k_6 + k_5)k_7 + k_5k_6 \), \( A_3 = ((k_6 + k_5)k_7 + k_5k_6)\mu_v + k_5k_6k_7 \).

The roots of \( P(\lambda) \) are \( \lambda_1 = \lambda_2 = -k_1, \lambda_3 = -k_3, \lambda_4 = -k_4, \lambda_5 = -k_9, \lambda_6 = -k_9 \), and the others roots are the roots of \( \phi_1(\lambda) \). Since \( N < 1 \), it is clear that all coefficients of \( \phi_1(\lambda) \) are always positive. Now we just have to verify that the Routh–Hurwitz criterion holds for polynomial \( \phi_2(\lambda) \). To this aim, setting \( H_1 = A_1, H_2 = \begin{vmatrix} A_1 & 1 \\ A_3 & A_2 \end{vmatrix} \), \( H_3 = \begin{vmatrix} A_1 & 0 \\ A_3 & A_2 & A_1 \end{vmatrix} \), \( H_4 = \begin{vmatrix} A_1 & 0 & 0 \\ A_3 & A_2 & A_1 \\ 0 & A_4 & A_3 \end{vmatrix} \) = \( A_4H_3 \). The Routh–Hurwitz criterion of stability of the trivial equilibrium \( E^0 \) is given by

\[
\begin{cases}
H_1 > 0 \\
H_2 > 0 \\
H_3 > 0 \\
H_4 > 0 \\
\end{cases} \Leftrightarrow \begin{cases}
H_1 > 0 \\
H_2 > 0 \\
H_3 > 0 \\
A_4 > 0 \\
\end{cases}
\]  
(57)

We have \( H_1 = A_1 > 0, \)

\[
H_2 = A_1A_2 - A_3 \\
= (k_7 + k_6 + k_5)\mu_v (\mu_v + k_7 + k_6 + k_5) + (k_6 + k_5) \left[ k_7^2 + (k_6 + k_5)k_7 + k_5k_6 \right],
\]

\[
H_3 = A_1A_2A_3 - A_1^2A_4 - A_3^2
\]
\[
\begin{align*}
&= \left( (k_6 + k_5) k_7^2 + \left( k_6^2 + 2k_5 k_6 + k_5^2 \right) k_7 + k_5 k_6^2 + k_5^2 k_6 \right) \mu_v^3 \\
&+ \left( \mu_b l s \theta + (k_6 + k_5) k_7^2 + \left( 2k_5^2 + 4k_5 k_6 + 2k_5^3 \right) k_7 \right) \\
&+ \left( k_6^3 + 4k_5 k_6^2 + 4k_6^2 k_5 + k_6^3 \right) k_7 + k_5 k_6^2 + 2k_5^2 k_6 + k_5^3 k_6 \mu_v^2 \\
&+ \left( 2k_7 + 2k_6 + 2k_5 \right) \mu_b l s \theta + \left( k_6^2 + 2k_5 k_6 + k_5^2 \right) k_7^3 \\
&+ \left( k_6^3 + 4k_5 k_6^2 + 4k_6^2 k_5 + k_6^3 \right) k_7^2 \\
&+ \left( 2k_5 k_6^3 + 4k_6^2 k_5^2 + 2k_6^3 k_5 \right) k_7 + k_5^2 k_6^3 + k_5^3 k_6 \mu_v \\
&+ \left( k_7^2 + (2k_6 + 2k_5) k_7 + k_6^2 + 2k_5 k_6 + k_5^2 \right) \mu_b l s \theta + \left( k_5 k_6^2 + k_5^2 k_6 \right) k_7^3 \\
&+ \left( k_5 k_6^2 + 2k_6^2 k_5 + k_6^3 k_5 \right) k_7^2 + \left( k_5^2 k_6^3 + k_5^3 k_6 \right) k_7 \\
&> 0.
\end{align*}
\]

We always have \( H_1 > 0, H_2 > 0, H_3 > 0 \) and \( H_4 > 0 \) if \( \mathcal{N} < 1 \). Thus, the trivial equilibrium \( Q_1 \) is locally asymptotically stable whenever \( \mathcal{N} < 1 \).

We assume the net reproductive number \( \mathcal{N} > 1 \). Following the procedure and the notation in Driessche and Watmough (2002), we may obtain the basic reproduction number \( R_0 \) as the dominant eigenvalue of the next-generation matrix (Diekmann and Heesterbeek 2000; Driessche and Watmough 2002). Observe that model (8) has four infected populations, namely \( E_h, I_h, E_v \) and \( I_v \). It follows that the matrices \( F \) and \( V \) defined in Driessche and Watmough (2002), which take into account the new infection terms and remaining transfer terms, respectively, are given by

\[
F = \begin{pmatrix}
0 & 0 & \beta_{hv} \eta_v & \beta_{hv} \\
0 & 0 & \beta_{vh} \eta_v & 0 \\
N_v^0 & \beta_{vh} N_v^0 & 0 & 0 \\
0 & 0 & N_h^0 & 0
\end{pmatrix}
\quad \text{and} \quad
V = \begin{pmatrix}
k_3 & 0 & 0 & 0 \\
\gamma_h & k_4 & 0 & 0 \\
0 & 0 & k_9 & 0 \\
0 & 0 & -\gamma_v & k_8
\end{pmatrix}
\]

The dominant eigenvalue of the next-generation matrix \( FV^{-1} \) is given by (12). The local stability of the disease-free equilibrium \( Q_2 \) is a direct consequence of Theorem 2 in Driessche and Watmough (2002) and showing that the model equations satisfy the five assumptions (A1–A5) in Driessche and Watmough (2002). This ends the proof.

**D Proof of Theorem 2**

Setting \( Y = X - Q_1 \) with \( X = (S_h, E_h, I_h, R_h, S_v, E_v, I_v, E, L, P)' \),

\[
A_{88} = \left( k_5 + \mu_b \frac{S_v + E_v + I_v}{K_E} \right), \quad \text{and} \quad A_{99} = \left( k_6 + s \frac{E}{K_L} \right).
\]

We can rewrite (8) in the following manner

\[
\frac{dY}{dt} = B(Y)Y.
\]
where \( \mathcal{B}(Y) = \begin{pmatrix} \mathcal{B}_1 & \mathcal{B}_2 \\ \mathcal{B}_3 & \mathcal{B}_4 \end{pmatrix} \), with

\[
\mathcal{B}_1 = \begin{pmatrix} -(\lambda_h + \mu_h) & 0 & 0 & 0 & 0 \\ \lambda_h & -k_3 & 0 & 0 & 0 \\ 0 & \gamma_h & -k_4 & 0 & 0 \\ 0 & 0 & \sigma & -\mu_h & 0 \\ 0 & 0 & 0 & 0 & -(\lambda_v + \mu_v) \end{pmatrix}, 
\mathcal{B}_3 = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix},
\mathcal{B}_2 = \begin{pmatrix} -\frac{a\beta_{hv}\eta_h S_h^0}{N_h} & -\frac{a\beta_{hv}S_h^0}{N_h} & 0 & 0 & 0 \\ \frac{a\beta_{hv}S_h^0}{N_h} & \frac{a\beta_{hv}\eta_h S_h^0}{N_h} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad \mathcal{B}_4 = \begin{pmatrix} -k_9 & 0 & 0 & 0 & 0 \\ \gamma_v & -\mu_v & 0 & 0 & 0 \\ \mu_b & \mu_b & -A_{88} & 0 & 0 \\ 0 & 0 & s & -A_{99} & 0 \\ 0 & 0 & 0 & l & -k_7 \end{pmatrix}.
\]

It is clear that \( Y = (0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0)^T \) is the only equilibrium. Then it suffices to consider the following Lyapunov function \( \mathcal{L}(Y) = < g, Y > \) where

\[
g = \left( 1, 1, 1, 1, 1, 1, 1, \frac{k_8}{\mu_b}, \frac{k_5k_8}{\mu_b s}, \frac{k_5k_6k_8}{\mu_b s l} \right).
\]

Straightforward computations lead that

\[
\dot{\mathcal{L}}(Y) \equiv < g, \dot{Y} > \equiv \dot{g} \mathcal{B}(Y) Y >
= -\mu_h Y_1 - \mu_h Y_2 - (\mu_h + \delta) Y_3 - \mu_h Y_4 - \frac{k_8}{K_E} (Y_5 + Y_6 + Y_7) - \frac{k_5k_8}{\mu_b K_L} Y_8 Y_9
+ \theta \left( 1 - \frac{1}{\mathcal{N}} \right) Y_{10}.
\]

We have \( \dot{\mathcal{L}}(Y) < 0 \) if \( \mathcal{N} \leq 1 \) and \( \dot{\mathcal{L}}(Y) = 0 \) if \( Y_i = 0, i = 1, 2, \ldots, 10 \) (i.e. \( S_h = S_h^0 \) and \( E_h = I_h = R_h = S_v = E_v = I_v = E = L = P = 0 \)). Moreover, the maximal invariant set contained in \( \{ \mathcal{L}(Y) = 0 \} \) is \( \{ (0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0)^T \} \). Thus, from Lyapunov theory, we deduce that \( (0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0)^T \) and thus, \( Q_1 \), is GAS if and only if \( \mathcal{N} \leq 1 \).

**E Proof of Theorem 4**

We follow the approach given in Safan et al. (2013). At this aim, note that equation (21) may be written as

\[
F(\beta_{hv}, \lambda_h) := d_2(\lambda_h^+)^2 + d_1\lambda_h^+ + d_0 = 0, \quad (58)
\]

where \( d_2, d_1 \) and \( d_0 \) are the same coefficients as in (21). Thus, the positive endemic equilibria of model (13) are obtained by solving (58) for positive \( \lambda_h^+ \) and substituting the results into (19). Clearly, the coefficient \( d_2 \), of (58), is always positive while \( d_1 \)
and $d_0$ may change sign. Therefore, there is a single endemic equilibrium if and only if $d_0 < 0$, which correspond to $R_0 > 1$. There are two endemic equilibria if and only if $d_0 > 0$, $d_1 < 0$ and $d_2^2 - 4d_2d_0 > 0$.

Now, first remember that $d_0 > 0$ (i.e. $R_0 < 1$) is equivalent to $\beta_{hv} < \beta^*_h$. Then, inequality $d_1 < 0$, is equivalent to

$$\beta_{hv} > \bar{\beta}$$

(59)

where $\bar{\beta}$ is given by (29).

Finally, equation $d_2^2 - 4d_2d_0 = 0$, in terms of $\beta_{hv}$, is equivalent to

$$\alpha_2\beta_{hv}^2 + \alpha_1\beta_{hv} + \alpha_0 = 0,$$

(60)

where $\alpha_2 = k_3^2 k_4 k_8 k_9 k_{10} k_{11} a^2 \mu^4_h (N_v^0)^2 (\beta_{vh})^2$, $\alpha_0 = k_3^2 k_4 k_8 k_9 k_{10} a^2 \mu^6_h (N_v^0)^2 (\beta_{vh})^2$ and $\alpha_1 = -2k_3 k_4 k_8 k_9 k_{10} k_{11} a^2 \mu^4_h N_h^0 N_v^0 \beta_{vh} (\delta \gamma_h (k_{10} a \mu_h \beta_{vh} + k_2 k_8) - k_2 \psi)$.

Now we compute the discriminant $\Delta := \alpha_2^2 - 4\alpha_2\alpha_0$, to obtain:

$$\Delta = -16(k_3 k_4 k_8 k_9 k_{10} k_{11} a^2 \mu^4_h N_h^0 N_v^0 \beta_{vh})^2 \delta \gamma_h (k_{10} a \mu_h \beta_{vh} + k_2 k_8) k_2 \psi.$$

Equation (60) admits a real solution if and only if $\Delta \geq 0$. This condition is equivalent to

$$\psi := k_{10} a \mu_h \beta_{vh} - \delta \gamma_h k_8 \leq 0$$

(61)

Under condition (61), we conclude that $\alpha_1 < 0$. Thus, Eq. (60) admits exactly two positive solutions which are given by

$$\beta_{\pm} = \frac{-\alpha_1 \pm \sqrt{\Delta}}{2\alpha_2} = \frac{k_9 N_h^0}{k_3 k_4 k_{10} k_{11} a^2 N_v^0 \beta_{vh}} \left[ \sqrt{\delta \gamma_h (k_{10} a \mu_h \beta_{vh} + k_2 k_8) \pm \sqrt{(-k_2 \psi)}} \right]$$

Thus, condition $d_2^2 - 4d_2d_0 > 0$ written in the terms of $\beta_{hv}$ is equivalent to

$$\beta_{hv} < \beta_- \quad \text{or} \quad \beta_{hv} > \beta_+.$$ 

and the inequalities (31) then follow.

F Proof of Theorem 6

Considering the model (8) without disease-induced death in human, and applying the same procedure as in proof of Theorem 3, we obtain that the non-zero equilibria of the basic model (8) satisfies the linear equation

$$(s K_E + k_5 K_L) (p_1 \lambda^*_h + p_0) = 0,$$
where \( p_1 = \mu_h \Lambda_h k_9 (k_2 \alpha \mu_h \beta_{vh} + k_3 k_8 (\mu_h + \sigma)) \) and \( p_0 = -\mu_h k_3 k_4 k_8 k_9 \mu_h \Lambda_h \left( \mathcal{R}^2_{0,\delta=0} - 1 \right) \).

Clearly, \( p_1 > 0 \) and \( p_0 \geq 0 \) whenever \( \mathcal{R}_{0,\delta=0} \leq 1 \), so that \( \lambda_h^* = -\frac{p_0}{p_1} \leq 0 \).

Therefore, the model (8) without disease-induced death in human, has no endemic equilibrium whenever \( \mathcal{R}_{0,\delta=0} \leq 1 \). The above result suggests the impossibility of backward bifurcation in the model (8) without disease-induced death, since no endemic equilibrium exists when \( \mathcal{R}_{0,\delta=0} < 1 \) (and backward bifurcation requires the presence of at least two endemic equilibria when \( \mathcal{R}_{0,\delta=0} < 1 \)) (Garba et al. 2008; Sharomi et al. 2007).

To completely rule out backward bifurcation in model (8), we use the direct Lyapunov method to prove the global stability of the DFE.

Define the positively-invariant and attracting region

\[
D_1 = \left\{ (S_h, E_h, I_h, R_h, S_v, E_v, I_v, E, L, P) \in D : S_h \leq N^0_h, S_v \leq N^0_v \right\}.
\]

Consider the Lyapunov function

\[
\mathcal{G} = q_1 E_h + q_2 I_h + q_3 E_v + q_4 I_v,
\]

where

\[
q_1 = \frac{1}{k_3}; \quad q_3 = \frac{\tau_1 S^0_h k_{11}}{k_3 k_8}, \quad q_2 = \frac{\tau_1 S^0_h k_{11} \xi_2 S^0_v}{k_3 k_8}, \quad q_4 = \frac{\tau_1 S^0_h}{k_3 k_8},
\]

and we have set \( \tau_1 = \frac{\mu_h \beta_{vh}}{\Lambda_h} \) and \( \tau_2 = \frac{\mu_h \beta_{vh}}{\Lambda_h} \). The derivative of \( \mathcal{G} \) is given by

\[
\dot{\mathcal{G}} = q_1 \dot{E}_h + q_2 \dot{I}_h + q_3 \dot{E}_v + q_4 \dot{I}_v
\]

\[
= q_1 (\lambda_h S_h - k_3 E_h) + q_2 (\gamma_h E_h - k_4 I_h) + q_3 (\lambda_v S_v - k_9 E_v) + q_4 (\gamma_v E_v - k_8 I_v)
\]

\[
= q_1 \tau_1 S_h (\eta_v E_v + I_v) - q_3 k_9 E_v + q_4 \gamma_v E_v - q_4 k_8 I_v + q_3 \tau_2 S_v (\eta_h E_h + I_h)
\]

\[
- q_1 k_3 E_h + q_2 \gamma_h E_h - q_2 k_4 I_h
\]

\[
= (q_1 \tau_1 S_h \eta_v + q_4 \gamma_v - q_3 k_9) E_v + (q_1 \tau_1 S_h - q_4 k_8) I_v
\]

\[
+ (q_3 \tau_2 S_v \eta_h + q_2 \gamma_h - q_1 k_3) E_h + (q_3 \tau_2 S_v - q_2 k_4) I_h
\]

\[
\leq (q_1 \tau_1 S^0_h \eta_v + q_4 \gamma_v - q_3 k_9) E_v + (q_1 \tau_1 S^0_h - q_4 k_8) I_v
\]

\[
+ (q_3 \tau_2 S^0_v \eta_h + q_2 \gamma_h - q_1 k_3) E_h + (q_3 \tau_2 S^0_v - q_2 k_4) I_h,
\]

since \( S_h \leq S^0_h, S_v \leq S^0_v \) in \( D_1 \).

Replacing \( q_i, i = 1, \ldots, 4 \), by their value gives after straightforward simplifications

\[
\dot{\mathcal{G}} \leq \left( \mathcal{R}^2_{0,\delta=0} - 1 \right) E_h.
\]
We have $\dot{G} \leq 0$ if $R_0, \delta = 0 \leq 1$, with $\dot{G} = 0$ if $R_0, \delta = 0 = 1$ or $E_h = 0$. Whenever $E_h = 0$, we also 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 fifth equation of Eq. (8) with $\delta = 0$ gives $S_h(t) \to S^0_h = N^0_h$, $R_h(t) \to 0$, and $S_v(t) \to S^0_v = N^0_v$ as $t \to \infty$. Thus

$$ [S_h(t), E_h(t), I_h(t), R_h(t), S_v(t), E_v(t), I_v(t), E(t), L(t), P(t)] \to (N^0_h, 0, 0, 0, N^0_v, 0, 0, E^*, L^*, P^*) \text{ as } t \to \infty. $$

It follows from the LaSalle’s invariance principle (Hale 1969; LaSalle 1968, 1976) that every solution of (8) (when $R_0, \delta = 0 \leq 1$), with initial conditions in $D_1$ converges to $Q_2$, as $t \to \infty$. Hence, the DFE, $Q_2$, of model (8) without disease-induced death, is GAS in $D_1$ if $R_0, \delta = 0 \leq 1$.

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