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Backward bifurcation and control in transmission dynamics of arboviral diseases

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ABSTRACT

In this paper, we derive and analyze a compartmental model for the control of arboviral diseases which takes into account an imperfect vaccine combined with individual protection and some vector control strategies already studied in the literature. After the formulation of the model, a qualitative study based on stability analysis and bifurcation theory reveals that the phenomenon of backward bifurcation may occur. The stable disease-free equilibrium of the model coexists with a stable endemic equilibrium when the reproduction number, $\mathcal{R}_0$, is less than unity. Using Lyapunov function theory, we prove that the trivial equilibrium is globally asymptotically stable. When the disease–induced death is not considered, or/and, when the standard incidence is replaced by the mass action incidence, the backward bifurcation does not occur. Under a certain condition, we establish the global asymptotic stability of the disease–free equilibrium of the principal model. Through sensitivity analysis, we determine the relative importance of model parameters for disease transmission. Numerical simulations show that the combination of several control mechanisms would significantly reduce the spread of the disease, if we maintain the level of each control high, and this, over a long period.

1. Introduction

Arboviral diseases are affections transmitted by hematophagous arthropods. There are currently 534 viruses registered in the International Catalog of Arboviruses and 25% of them have caused documented illness in human populations [1–3]. 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. According to World Health Organisation (WHO), Dengue, caused by any of four closely-related virus serotypes (DEN-1-4) of the genus Flavivirus, causes 50–100 million infections worldwide every year, and the majority of patients worldwide are children aged 9 to 16 years [4–6]. The dynamics of arboviral diseases like Dengue or Chikungunya are influenced by many factors such as human and mosquito behaviors. The virus itself (multiple serotypes of dengue virus [5,6], and multiple strains of chikungunya virus [7,8]), as well as the environment directly or indirectly affects all the present mechanisms of control [9,10].

For all mentioned diseases, only yellow fever has a licensed vaccine. Nonetheless, considerable efforts are made to obtain the vaccines for other diseases. In the case of Dengue for example, the scientists of French laboratory SANOFI have conducted different tries in Latin America and Asia. Thus, a tetravalent vaccine could be quickly set up in the coming months. The tries in Latin America have shown that vaccine efficacy was 64.7%. Serotype–specific vaccine efficacy was 50.3% for serotype 1, 42.3% for serotype 2, 74.0% for serotype 3, and 77.7% for serotype 4 [11]. The tries in Asia have shown that efficacy was 30.2%, and differed by serotype [12]. In any case, it is clear that this vaccine will be imperfect.

Host-vector models for arboviral diseases transmission were proposed in [13–30] with the focus on the construction of the

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basic reproductive ratio and related stability analysis of the disease free and endemic equilibria. Some of these works in the literature focus on modeling the spread of arboviral diseases and its control using some mechanism of control like imperfect vaccines [23,24,30] and other control tools like individual protection and vector control strategies [13,14,19,25,27,28].

In [19], Dumont and Chiroleu proposed a compartmental model to study the impact of vector control methods used to contain or stop the epidemic of Chikungunya of 2006 in Réunion island. Moulay et al. [27] studied an optimal control based on protection and vector control strategies to fight against Chikungunya. In [24], Rodrigues et al. simulate an hypothetical vaccine as an extra protection to the human population against epidemics of Dengue, using the optimal control theory. In those models [19,24,27],

(i) the population is constant (no population migration),
(ii) the disease-induced death in humans is not considered,
(iii) the complete stage progression of development of vectors is not considered,
(iv) none of the above mentioned models takes into account the combination of the mechanisms of control already studied in the literature, such as vaccination, individual protection and vector control strategies (destruction of breeding site, eggs and larvae reduction).

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

We start with the formulation of a constant control model, which is an extension of the previous model developed in [30]. We include the complete stage progression of development of vectors, the waning vaccine, and four others controls (individual protection, the use of adulticides, destruction of breeding site, and reduction of eggs and larvae through chemical interventions). We compute the net reproductive number $\mathcal{N}$, as well as the basic reproduction number, $\mathcal{R}_0$, and investigate the existence and stability of equilibria. We prove that the trivial equilibrium is globally asymptotically stable whenever $\mathcal{N} < 1$. When $\mathcal{N} > 1$ and $\mathcal{R}_0 < 1$, we prove that the system exhibit the backward bifurcation phenomenon. The implication of this occurrence is that the classical epidemiological requirement for effective eradication of the disease, $\mathcal{R}_0 < 1$, is no longer sufficient, even though necessary. However, considering two situations: the model without vaccination and the model with mass incidence rates, we prove that the disease–induced death and the standard incidence functions, respectively, are the main causes of the occurrence of backward bifurcation. We find that the disease–free equilibrium is globally asymptotically stable under certain threshold condition. Through local and global sensitivity analysis, we determine the relative importance parameters of the model on the disease transmission. By using the pulse control technique (the control is not continuous in time order is effective only one day every T days [19]) in numerical simulations, we evaluate the impact of different control combinations on the decrease of the spread of these diseases.

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

2. The formulation of the model

The model we propose here is an extension of the previous model studied in [30], and is based on the modeling approach given in [19–23,27,28]. 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:

–For humans, we consider susceptible (denoted by S_h), vaccinated (V_h), exposed (E_h), infectious (I_h) and resistant or immune (R_h). So that, $N_h = S_h + V_h + E_h + I_h + R_h$. Following Garba et al. [23] and Rodrigues et al. [24], we assume that the immunity, obtained by the vaccination process, is temporary. So, we denote by ω, the waning rate of vaccine. The recruitment in human population is at the constant rate Λ_h, and newly recruited individuals enter the susceptible compartment S_h. Only naive humans to the disease are taken into account in the recruitment. Each individual human compartment goes out from the dynamics at natural mortality rates μ_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

$$\lambda_h = \frac{a\beta_{hv}(\eta_v E_v + I_v)}{N_h} \text{ [23]},$$

where a is the biting rate per susceptible vector, β_{hv} is the transmission probability from an infected vector (E_v or I_v) to a susceptible human (S_h). The expression of λ_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 times. Then, the infection rate per susceptible human is given by $a\beta_{hv}(N_v/N_h)((\eta_v E_v + I_v)/N_v)$. In expression of λ_h, the modification parameter $0 < \eta_v < 1$ accounts for the assumed reduction in transmissibility of exposed mosquitoes relative to infectious mosquitoes [23,30] (see the references therein for the specific sources). Latent humans (E_h) become infectious (I_h) at rate γ_h. Infectious humans recover at a constant rate, σ or dies as consequence of infection, at a disease-induced death rate δ. After infection, immune humans retain their immunity for life.

– Following [27], the stage structured model is used to describe the vector population dynamics, which consists of three main stages: embryonic (E), larvae (L) and pupae (P). Even if eggs (E) and immature stages (L and P) are all aquatic, it is important to dissociate them because, for the control point of view, drying the breeding sites does not kill eggs, but only larvae and pupae. Moreover, chemical interventions on the breeding sites has impact on the larvae population, but not on the eggs [27]. 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 (see [27] for details)

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

Unlike the authors of [27], we take into account the pupal stage in the development of the vector. This is justified by the fact that they do not feed during this transitional stage of development, as they transform from larvae to adults [10,31]. So, the control mechanisms cannot be applied to them.

With a rate θ, pupae become female Adults. Each individual vector compartment goes out from the dynamics at natural mortality rates μ_v. The vector susceptible population is decreased following infection, which can be acquired via effective contact with