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# Analysis of Dengue Disease Transmission Model with General Incidence Functions

Harouna Ouedraogo <sup>1</sup><sup>∗</sup> and Aboudramane Guiro <sup>2</sup>

<sup>1</sup> Department of Mathematics, Joseph Ki-Zerbo University, 03 bp 7021 Ouagadougou 03 Burkina Faso.  $2$  Department of Mathematics, Nazi Boni University, 01 bp 1091 Bobo-Dioulasso 01 Burkina Faso.

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Abstract: In this work, we propose a non-linear system of differential equations that models the dynamics of transmission of dengue fever. Then, we perform a stability analysis of this model. In particular, we prove that when the threshold of the model called the basic reproduction ratio is less than unity, the disease-free equilibrium is globally asymptotically stable. Furthermore, when this value is greater than unity, under suitable conditions, the endemic equilibrium is globally asymptotically stable. Some numerical simulations are provided to illustrate the obtained theoretical results. We also propose a global sensitivity analysis of the basic reproduction ratio.

Keywords: dengue; general incidence function; mathematical analysis; basic reproduction number; Lyapunov function; stability analysis; sensitivity.

Mathematics Subject Classification (2010): 70K75, 93C15, 34D23.

# 1 Introduction

Mathematical modelling and numerical simulation are important decision tools that can be used to study and control human and animal diseases [\[1,](#page-14-1) [2\]](#page-14-2). However, to tackle real situations, the resulting models need to be adapted to each specific disease and its biological characteristics [\[3\]](#page-14-3).

From a general point of view, mathematical models are used to predict the behaviour of a disease in a particular population [\[4,](#page-14-4) [5\]](#page-14-5). In particular, they help to determine if the disease under consideration will be endemic (i.e., it remains active in the population)

<sup>∗</sup> Corresponding author: [mailto:oharounao@gmail.com](mailto: oharounao@gmail.com)

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or not (i.e., it disappears). In this work, we introduce and study a particular mathematical model to estimate the dynamic of the so-called dengue fever disease in a human population.

Dengue disease is a common arboviral disease in tropical regions and the mediterranean. It is transmitted to humans by the bite of *Aedes mosquitoes*. Four serotypes have been recognized, they are denoted by DEN-I, DEN-II, DEN-III, and DEN-IV. These viruses are carried by two kinds of mosquitoes referred to as Aedes aegypti and Aedes albopictus which spread the disease through their bite. However, Aedes aegypti has been the principal vector of dengue virus transmission. Another interesting fact is the shift of patients phenomena when dengue fever previously attacked children of primary school age, but now everybody is vulnerable to the fever [\[6\]](#page-14-6). Dengue viruses can infect only a restricted number of vertebrates but it is an essentially human disease. Infection for any dengue serotype produces permanent immunity to it, but apparently only temporary cross immunity to other serotypes. Therefore, individuals that live in dengue endemic areas can have more than one infection of dengue disease. It is considered that human population growth and the dramatic redistribution of the human population in the urban centers of developing countries have contributed to the introduction and enhancement of dengue fever [\[7\]](#page-14-7).

Mathematical models and methods of non-linear dynamic are used in comparing, planning, implementing, evaluating and optimizing various detection, prevention, therapy and control programmes [\[8,](#page-15-0) [9\]](#page-15-1). Thus, mathematical models are a useful tool to better understand the mechanisms that allow the spread of a dengue epidemic and then to increase the efficiency of the vector control strategy. There are a number of mathematical expert models for dengue fever which involve differential equations. In general, they use compartmental dynamic such as susceptible, infected, removed (SIR) and susceptible, exposed, infected, removed  $(SEIR)$ . In [\[10\]](#page-15-2), the authors formulated stochastic models for dengue in the presence of Wolbachia. This research aims to measure the effectiveness of the Wolbachia intervention to reduce dengue transmission. It determines the proportion of reduction in the basic reproduction number and also the probability of extinction. Putri *et al.* (see [\[11\]](#page-15-3)) proposed the study where the aim is to forecast and analyze the spread of COVID-19 outbreak in Indonesia by applying machine learning and hybrid approaches. Abdelhamid Zaghdani (see [\[9\]](#page-15-1)) formulated a modified  $SEIR$  mathematical model for the coronavirus infected disease-2019 (COVID-19). The author computed the basic reproduction number  $(\mathcal{R}_0)$  and proposed a qualitative analysis of the local and global stability of the equilibrium points.

In this paper, our aim is to study the dengue epidemic model presented and studied in  $[5]$  by J. J. Tewa *et al.* with the law of mass action as the incidence functions. The similar model was presented and studied in [\[12\]](#page-15-4). It appears that the incidence function form is determinative in the study of the model system. Then, changing the form of the incidence function can potentially change the behaviour of the system. In this work, we study a coupling model (Humans and Vectors) with two general incidence functions given by  $f, g$ . From the analysis of the global stability of the equilibrium points, we used the same technique as in Guiro  $et \ al.$  [\[13\]](#page-15-5). We find conditions on the incidence function to get the stability of the model.

This paper is organized as follows. In Section [2,](#page-2-0) we describe the mathematical model which is studied in the paper. In Section [3,](#page-4-0) we give the equilibrium points, the basic reproduction number, we define also a positive invariant and attractive set, which will be used in the studies of the stability of equilibrium points. In Section [4,](#page-6-0) we study the

stability of equilibrium points. Section [6](#page-11-0) contains the numerical result and comments. Section [7](#page-11-1) is devoted to the analysis of global sensitivity of the parameters in the basic reproduction number  $\mathcal{R}_0$ . We end by the conclusion.

## <span id="page-2-0"></span>2 Description of the Model

In this section, we recall the model studied in  $[5]$  by J. J. Tewa *et al.*, which is given with a particular incidence function as follows:

<span id="page-2-1"></span>
$$
\begin{cases}\n\dot{S}_H = \mu_H N_H - \frac{\beta_H b}{N_H + m} S_H I_V - \mu_H S_H, \\
\dot{I}_H = \frac{\beta_H b}{N_H + m} S_H I_V - (\mu_H + \gamma_H) I_H, \\
\dot{R}_H = \gamma_H I_H - \mu_H R_H, \\
\dot{S}_V = A - \frac{\beta_V b}{N_H + m} S_V I_H - \mu_V S_V, \\
\dot{I}_V = \frac{\beta_V b}{N_H + m} S_V I_H - \mu_V I_V.\n\end{cases}
$$
\n(1)

The model above is described as follows: the human and vector populations are divided into classes or states containing susceptible, infective and immune individuals. At time t, there are the susceptible humans  $(S_H)$  and the infectious humans  $(I_H)$ , we assume that the infectious humans recover (or get treated) at a constant rate  $\gamma_H$ ,  $\mu_H + \gamma_H$ is the total exit of the infectious humans,  $R_H$  are the immune humans,  $S_V$  are the susceptible mosquitoes and  $I_V$  are the infectious mosquitoes. The mosquito population does not have an immune class since their infectious period ends with their death. Let  $N_H = S_H + I_H + R_H$  and  $N_V = S_V + I_V$  be, respectively, the total human and vector population at time t. Total death in the mosquito population occurs at a rate  $\mu_V N_V$ , where  $\mu_V$  is the per capita mortality rate of mosquitoes. In this model, it is assumed that the human population has constant size with the birth and death rate constant number equal to  $\mu$ . Also, for the mosquito population, it is assumed a constant recruitment rate A, independent of the actual number of adult mosquitoes. It is admitted that the flow from the susceptible to the infectious class, for each species, depends on the biting rate of the mosquitoes, the transmission probabilities, as well as the number of infectious and susceptible of each species.

Let  $b$  denote the biting rate of mosquitoes, which is the average number of bites per mosquito per day. m denotes the number of alternative hosts available as blood sources, then the probability that a mosquito chooses a human individual as a host is given by  $N_H$  $\frac{N_H}{N_H + m}$ . Thus, it is admitted that a human receives  $b \frac{N_V}{N_H}$  $N_H$  $N_H$  $\frac{N_H}{N_H + m}$  bites per unit of time, and a mosquito takes  $\frac{bN_H}{N_H + m}$  human blood meals per unit of time. Then, the infection rates per susceptible human and susceptible vector are given by

$$
\beta_H b \frac{N_V}{N_H} \frac{N_H}{N_H + m} \frac{I_V}{N_V} = \frac{\beta_H b}{N_H + m} I_V,
$$

$$
\beta_V b \frac{N_H}{N_H + m} \frac{I_H}{N_H} = \frac{\beta_V b}{N_H + m} I_H,
$$

respectively. Here  $\beta_H$  is the transmission probability from a vector to a human and  $\beta_V$ is the transmission probability from a human to a vector.

The aim of our work is to generalize the model [\(1\)](#page-2-1) with the incidence function as the general incidence functions  $f$  and  $g$ . The interaction between the human population and the vector population is given by the following diagram, see Figure [1:](#page-3-0)

<span id="page-3-0"></span>

Figure 1: Transfer diagram for the mathematical model of dengue.

Then, according to Figure [1,](#page-3-0) we have the following system of five differential equations:  $\mathcal{L}$ 

<span id="page-3-1"></span>
$$
\begin{cases}\n\dot{S}_H = \tilde{\Lambda} - f(S_H, I_V) - \mu_H S_H, \\
\dot{I}_H = f(S_H, I_V) - (\mu_H + \gamma_H) I_H, \\
\dot{R}_H = \gamma_H I_H - \mu_H R_H, \\
\dot{S}_V = A - g(S_V, I_H) - \mu_V S_V, \\
\dot{I}_V = g(S_V, I_H) - \mu_V I_V.\n\end{cases}
$$
\n(2)

In the system [\(2\)](#page-3-1), we use the same constant and the same subdivision of the human population and the vector population as described in the system [\(1\)](#page-2-1).

Since  $R_H$  does not appear in the first and second equations of system [\(2\)](#page-3-1), it is sufficient to analyse the behavior of solutions of the following system:

<span id="page-3-2"></span>
$$
\begin{cases}\n\dot{S}_H = \tilde{\Lambda} - f(S_H, I_V) - \mu_H S_H, \\
\dot{I}_H = f(S_H, I_V) - (\mu_H + \gamma_H) I_H, \\
\dot{S}_V = A - g(S_V, I_H) - \mu_V S_V, \\
\dot{I}_V = g(S_V, I_H) - \mu_V I_V.\n\end{cases}
$$
\n(3)

We assume that the functions  $f$  and  $g$  satisfy the following hypotheses:

**H1** f and g are non-negative  $C^1$  functions in the non-negative orthant.

**H2** For all  $(S_H, I_H, R_H, S_V, I_V) \in \mathbb{R}^5_+$ ,  $f(S, 0) = f(0, I) = 0$  and  $g(S, 0) = g(0, I) = 0$ . Also, we denote by  $f_1, g_1$  and  $f_2, g_2$  the partial derivative of f and g with respect to S and I.

**Remark 2.1** f and g are two incidence functions which explain the contact between two species. Therefore, H2 is a natural assumption which means that if there is no infected in the human and vector populations, then the incidence functions are equal to zero. The incidence functions are also equal to zero when there is no susceptible in the human and vector populations.

# <span id="page-4-0"></span>3 Basic Properties and Basic Reproduction number

In this section, we study the basic properties of the solution of system [\(3\)](#page-3-2) and also, we compute the basic reproduction number associated to the system [\(3\)](#page-3-2).

<span id="page-4-1"></span>Proposition 3.1 The positive orthant

$$
\{(S_H, I_H, S_V, I_V) \in \mathbb{R}^4, S_H \ge 0, I_H \ge 0, S_V \ge 0, I_V \ge 0\}
$$

is positively invariant for system [\(3\)](#page-3-2).

To prove Proposition 3.[1,](#page-4-1) we need the following lemma.

**Lemma 3.1** [\[14\]](#page-15-6): Let  $L : \mathbb{R}^n \longrightarrow \mathbb{R}$  be a differentiable function, and let  $a \in \mathbb{R}$ . Let  $X(x)$  be the vector field, and let G be the closed set  $G = \{x \in \mathbb{R}^n : L(x) \le a\}$  such that  $\nabla L(x) \neq 0$  for all  $x \in L^{-1}(a) = \{x \in \mathbb{R}^n, L(x) = a\}$ . If  $\langle X(x), \nabla L(x) \rangle \leq 0$  for all  $x \in L^{-1}(a)$ , then the set G is positively invariant.

**Proof of Proposition [3.1](#page-4-1)** Let  $x = (S_H, I_H, S_V, I_V)$ . Now, we have to prove that  ${S_H \geq 0}$  is positively invariant.

Let  $L(x) = -S_H$ . L is differentiable and  $\nabla L(x) = (-1, 0, 0, 0) \neq 0_{\mathbb{R}^5}$  for all  $x \in$  $L(x)^{-1}(0) = \{x \in \mathbb{R}^4 / L(x) = 0\}.$  The vector field on  $\{S_H = 0\}$  is

$$
X(x) = \begin{pmatrix} \tilde{\Lambda} \\ -(\mu_H + \gamma_H)I_H \\ A - g(S_V, I_H) - \mu_V S_V \\ g(S_V, I_H) - \mu_V I_V \end{pmatrix}.
$$

Then  $\langle X(x), \nabla L(x) \rangle = -\tilde{\Lambda} \langle 0$ . This proves that  $\{S_H \geq 0\}$  is positively invariant. Similarly, we prove that  $\{I_H \geq 0\}$ ,  $\{R_H \geq 0\}$ ,  $\{S_V \geq 0\}$ ,  $\{I_V \geq 0\}$  are positively invariant. Then  $\{(S_H, I_H, S_V, I_V) \in \mathbb{R}^5, S_H \geq 0, I_H \geq 0, S_V \geq 0, I_V \geq 0\}$  is positively invariant for system  $(3)$ . □

Therefore, the model is mathematically well posed and epidemiologically reasonable since all the variables remain non-negative for all  $t > 0$ .

**Proposition 3.2** Let  $(S_H, I_H, S_V, I_V)$  be the solution of system [\(3\)](#page-3-2) with the initial condition  $(S_{0H}, I_{0H}, S_{0V}, I_{0V})$  and the compact set

$$
\mathcal{D} = \left\{ (S_H, I_H, S_V, I_V) \in \mathbb{R}_+^4, W_1 \le N_H + \epsilon, W_2 \le \frac{A}{\mu_V} + \epsilon, for \epsilon > 0 \right\}
$$
(4)

with  $W_1 = S_H + I_H$  and  $W_2 = S_V + I_V$ . Then, under the flow described by [\(3\)](#page-3-2),  $\mathcal{D}$  is a positively invariant set that attracts all solutions in  $\mathbb{R}^4_+$ .

**Proof.** By adding the first two equations of system  $(3)$ , we have

<span id="page-5-0"></span>
$$
\frac{dS_H}{dt} + \frac{dI_H}{dt} = \tilde{\Lambda} - \mu_H S_H - (\mu_H + \gamma_H) I_H,
$$
\n
$$
\frac{d(S_H + I_H)}{dt} \leq \tilde{\Lambda} - \mu_H (S_H + I_H),
$$
\n
$$
\frac{dW_1}{dt} \leq \tilde{\Lambda} - \mu_H W_1,
$$
\n
$$
\frac{dW_1}{dt} + \mu_H W_1 \leq \tilde{\Lambda} + \epsilon.
$$
\n(5)

According to [\[15\]](#page-15-7), from inequation [\(5\)](#page-5-0), we have

$$
W_1(t) \le \frac{\tilde{\Lambda}}{\mu_H} + \frac{\epsilon}{\mu_H} + (W_1(0) - \frac{\tilde{\Lambda}}{\mu_H} - \frac{\epsilon}{\mu_H})e^{-\mu_H t},\tag{6}
$$

where  $W_1(0) = S_{0H} + I_{0H}$ . Thus, when  $t \longrightarrow +\infty$ ,  $W_1(t) \leq \frac{\tilde{\Lambda}}{\tilde{\Lambda}}$  $\frac{\Lambda}{\mu_H} + \frac{\epsilon}{\mu_H}$  $\frac{\epsilon}{\mu_H}$ . Similarly, we prove that  $W_2(t) \leq \frac{A}{\mu}$  $\frac{A}{\mu V} + \frac{\epsilon}{\mu V}$  $\frac{v}{\mu V}$ , where  $W_1(0)$  and  $W_2(0)$  are, respectively, the initial conditions of  $W_1(t)$  and  $W_2(t)$ . Thus, as  $t \longrightarrow \infty$ ,  $0 \leq (W_1(t), W_2(t)) \leq (N_H + \frac{\epsilon}{\epsilon})$  $\frac{\epsilon}{\mu_H}, \frac{A}{\mu_V}$  $\frac{A}{\mu V} + \frac{\epsilon}{\mu V}$  $\frac{c}{\mu V}$  and one can conclude that  $\mathcal D$  is an attractive set.  $\Box$ 

Let  $E = (S_H, I_H, S_V, I_V)$  be an equilibrium point of [\(3\)](#page-3-2). Thus, we have

<span id="page-5-1"></span>
$$
\begin{cases}\n\tilde{\Lambda} - f(S_H, I_V) - \mu_H S_H = 0, \\
f(S_H, I_V) - (\mu_H + \gamma_H)I_H = 0, \\
A - g(S_V, I_H) - \mu_V S_V = 0, \\
g(S_V, I_H) - \mu_V I_V = 0.\n\end{cases}
$$
\n(7)

By adding the first two and the last two equations [\(7\)](#page-5-1), we get

$$
S_H = \frac{\tilde{\Lambda} - (\mu_H + \gamma_H)I_H}{\mu_H} , \ S_V = \frac{A - \mu_V I_V}{\mu_V},
$$

and

$$
E = \left(\frac{\tilde{\Lambda} - (\mu_H + \gamma_H)I_H}{\mu_H}, I_H, \frac{A - \mu_V I_V}{\mu_V}, I_V\right).
$$

Hence, the disease-free equilibrium and the endemic equilibrium of [\(3\)](#page-3-2) are given by

$$
E_0 = (S_H^0, I_H^0, S_V^0, I_V^0) = (\frac{\tilde{\Lambda}}{\mu_H}, 0, \frac{A}{\mu_V}, 0)
$$

and

$$
E^* = (S_H^*, I_H^*, S_V^*, I_V^*) = \left(\frac{\tilde{\Lambda} - (\mu_H + \gamma_H)I_H^*}{\mu_H}, I_H^*, \frac{A - \mu_V I_V^*}{\mu_V}, I_V^*\right).
$$

Here,  $I_H^*$  and  $I_V^*$  are the design infected human and infected mosquito at endemic period.

The reproduction number of model [\(3\)](#page-3-2) is obtained by creating the next generation matrix and funding the maximum eigenvalues of that matrix [\[16\]](#page-15-8). The reproduction number of that model is given by

$$
\mathcal{R}_0 = \sqrt{\frac{f_2(S_H^0, 0)g_2(S_V^0, 0)}{\mu_V(\mu_H + \gamma_H)}}.
$$

**Theorem 3.1** If  $\mathcal{R}_0 > 1$ , then the model [\(3\)](#page-3-2) has only a unique endemic equilibrium  $E^*$ .

**Proof.** Let us define the function  $\psi(I_H, I_V) = (\psi_1(I_H, I_V); \psi_2(I_H, I_V))$ , where

$$
\psi_1(I_H, I_V) = f(S_H^0 - I_H, I_V) - (\mu_H + \gamma_H)I_H
$$

and

$$
\psi_2(I_H, I_V) = g(S_V^0 - I_V, I_H) - \mu_V I_V.
$$

Hence, it follows that any solution of the equation  $\psi = 0$  in the set  $(0, S_H^0) \times (0, S_V^0)$ corresponds to an equilibrium, with  $S_H$ ,  $I_H$ ,  $S_V$ ,  $I_V > 0$ . Since H2 holds, one has  $\psi(0,0) = 0$  and  $\psi(S_H^0, S_V^0) \leq 0$ . Then the sufficient condition for the equation  $\psi = 0$ to have a solution in  $(0, S_H^0) \times (0, S_V^0)$  is that  $\psi$  is increasing at 0. This implies that an endemic equilibrium exits if

<span id="page-6-1"></span>
$$
\nabla \psi(0,0) > 0,\tag{8}
$$

where

$$
\nabla \psi(0,0) = (\nabla \psi_1(0,0), \nabla \psi_2(0,0))
$$
  
= 
$$
(-f_1(H_s^0,0) - \mu_H + \gamma_H + f_2(S_H^0,0), -g_1(S_V^0,0) - \mu_V + g_2(S_s^0,0)).
$$

Note that  $f_1(S_H^0, 0) = g_1(S_V^0, 0) = 0$ . Then inequality [\(8\)](#page-6-1) is equivalent to

$$
f_2(S_H^0, 0) > \mu_H + \gamma_H
$$
, and  $g_2(S_V^0, 0) > \mu_V$ ,

which give

$$
\alpha \gamma f_2(S_H^0, 0) g_2(S_V^0, 0) > (\mu_H + \gamma_H) \mu_V.
$$

That is,

$$
\mathcal{R}_0 = \frac{f_2(S_H^0, 0)g_2(S_V^0, 0)}{(\mu_H + \gamma_H)\mu_V} > 1.
$$

Then system [\(3\)](#page-3-2) has a unique endemic equilibrium given by  $E^* = \left(\frac{\tilde{\Lambda} - (\mu_H + \gamma_H)I_H^*}{\sigma_H}\right)$  $\frac{H^4 + \gamma_H}{\mu_H}$ ,  $I_H^*$ ,  $\frac{A - \mu_V I_V^*}{\mu_V}$  $\frac{\mu_V I_V}{\mu_V}, I_V^*$ ). The proof is completed.  $□$ 

## <span id="page-6-0"></span>4 Stability of Equilibrium

In this section, we analyze the stability of the diseases-free equilibrium  $E_0$  and the endemic equilibrium  $E^*$ . **H3** For all  $(S_H, I_H, S_V, I_V) \in \mathbb{R}^4_+$ ,

$$
f(S_H, I_V) \le f_2(S_H^0, 0)I_V
$$
 and  $g(S_V, I_H) \le g_2(S_V^0, 0)I_H$ .

**H4** 
$$
1 < \frac{f_2(S_H^0, 0)}{\mu_H + \gamma_H}
$$
 and  $1 < \frac{g_2(S_V^0, 0)}{\mu_V}$ .

Remark 4.1 The assumptions H3 and H4 are the technical assumptions which are also used to have the global stability of the diseases-free equilibrium  $E_0$ . Biologically, the assumption H3 allows for the control of the infection speed.

**Theorem 4.1** Assume that **H3** and **H4** hold, then if  $\mathcal{R}_0 \leq 1$ , the diseases-free equilibrium  $E_0$  is globally asymptotically stable on  $D$ .

Proof. Let us consider the candidate Lyapunov function

$$
V = \mu_V I_H + (\mu_H + \gamma_H) I_V.
$$

By differentiating  $V$  with respect to time, we have

$$
\dot{V} = \mu_V \dot{I}_H + (\mu_H + \gamma_H) \dot{I}_V \n= \mu_V f(S_H, I_V) - \mu_V (\mu_H + \gamma_H) I_H + (\mu_H + \gamma_H) g(S_V, I_H) - \mu_V (\mu_H + \gamma_H) I_V \n= \mu_V f(S_H, I_V) + (\mu_H + \gamma_H) g(S_V, I_H) - \mu_V (\mu_H + \gamma_H) (I_H + I_V).
$$

By using the assumption H3, we get

$$
\dot{V} \leq f_2(S_H^0, 0)\mu_V I_V + g_2(S_V^0, 0)(\mu_H + \gamma_H)I_H - \mu_V(\mu_H + \gamma_H)(I_H + I_V).
$$

By adding and subtracting  $f_2(S_H^0, 0)g_2(S_V^0, 0)(I_H + I_V)$  in the inequality above, we have

$$
\dot{V} \leq f_2(S_H^0, 0)g_2(S_V^0, 0)(I_H + I_V) + f_2(S_H^0, 0)I_V[\mu_V - g_2(S_V^0, 0)] +g_2(S_V^0, 0)I_H[(\mu_H + \gamma_H) - f_2(S_H^0, 0)] - \mu_V(\mu_H + \gamma_H)(I_H + I_V).
$$

By using the assumption H4, we obtain

$$
\dot{V} \leq \mu_V(\mu_H + \gamma)(I_H + I_V) \left( \frac{f_2(S_H^0, 0)g_2(S_V^0, 0)}{\mu_V(\mu_H + \gamma)} - 1 \right) \leq \mu_V(\mu_H + \gamma_H)(I_H + I_V)(\mathcal{R}_0^2 - 1).
$$

Since  $\mathcal{R}_0 \leq 1$ , we have  $\dot{V} \leq 0$ , with equality only if  $I_H = 0$  and  $I_V = 0$ . According to LaSalle's extension to Lyapunov method's [\[17\]](#page-15-9), the limit set of each solution is contained in the largest invariant set, for which  $I_H = 0$  and  $I_V = 0$ , which is the singleton  $\{E_0\}$ . Thus, the unique disease-free equilibrium  $E_0$  is globally asymptotically stable on  $\mathcal{D}$ .  $\Box$ 

We assume that the functions  $f$  and  $g$  satisfy the following assumptions: **H5** For all  $(S_H, I_H, S_V, I_V) \in \mathbb{R}^4_+$ ,  $1 \leq \frac{f(S_H, I_V)}{f(S_H, I_V)}$  $\frac{f(S_H, I_V)}{f(S_H, I_V^*)} \leq \frac{I_V}{I_V^*}$  $I_V^*$ and  $1 \leq \frac{g(S_V, I_H)}{g(S_V, I_H)}$  $\frac{g(S_V,I_H)}{g(S_V,I_H^*)} \leq \frac{I_H}{I_H^*}$  $I_H^*$ . **H6** For all  $(S_H, S_V) \in \mathbb{R}^2_+$ ,  $Sign(S_H - S_H^*) = Sign(f(S_H, I_V^*) - f(S_H^*, I_V^*))$ and  $Sign(S_V - S_V^*) = Sign(g(S_V, I_H^*) - g(S_V^*, I_H^*)).$ 

Remark 4.2 The assumptions H5 and H6 are the technical assumptions which are used in the proof of the global stability of the endemic equilibrium.

**Theorem 4.2** When  $\mathcal{R}_0 > 1$ , then the endemic equilibrium  $E^*$  of system [\(3\)](#page-3-2) exists and is globally asymptotically stable on D.

**Proof.** At the endemic equilibrium  $E^*$  and from the system [\(3\)](#page-3-2), we have

<span id="page-7-0"></span>
$$
\begin{cases}\n\tilde{\Lambda} = f(S_H^*, I_V^*) + \mu_H S_H^*, \\
f(S_H^*, I_V^*) = (\mu_H + \gamma_H) I_H^*, \\
A = g(S_V^*, I_H^*) + \mu_V S_V^*, \\
g(S_V^*, I_H^*) = \mu_V I_V^*.\n\end{cases} \tag{9}
$$

Let us define the function h on  $\mathbb{R}_+$  by  $h(x) = x - 1 - \ln x$ . The function h is non-negative for all  $x \in \mathbb{R}_+$ . Let us consider the candidate Lyapunov function U defined by

$$
U(t) = U_H(t) + U_V(t)
$$
 where  $U_H(t) = U_{S_H}(t) + U_{I_H}(t)$  and  $U_V(t) = U_{S_V}(t) + U_{I_V}(t)$ 

with

$$
U_{S_H} = S_H - S_H^* - \int_{S_H^*}^{S_H} \frac{f(S_H^*, I_V^*)}{f(\chi, I_V^*)} d\chi, \ U_{I_H} = I_H^* h\left(\frac{I_H}{I_H^*}\right),
$$
  

$$
U_{S_V} = S_V - S_V^* - \int_{S_V^*}^{S_V} \frac{g(S_V^*, I_H^*)}{g(\chi, I_H^*)} d\chi, \ U_{I_V} = I_V^* h\left(\frac{I_V}{I_V^*}\right).
$$

Now, we have to differentiate the function  $U$  with respect to time.

$$
\dot{U}_{S_H} = \left(1 - \frac{f(S_H^*, I_V^*)}{f(S_H, I_V^*)}\right)\dot{S}_H \n= \left(1 - \frac{f(S_H^*, I_V^*)}{f(S_H, I_V^*)}\right)(\tilde{\Lambda} - f(S_H, I_V) - \mu_H S_H).
$$

By using the first equation of system [\(9\)](#page-7-0), we have

$$
\dot{U}_{S_H} = -\mu_H (S_H - S_H^*) \left( 1 - \frac{f(S_H^*, I_V^*)}{f(S_H, I_V^*)} \right) \n+ f(S_H^*, I_V^*) \left[ 1 - \frac{f(S_H, I_V)}{f(S_H^*, I_V^*)} - \frac{f(S_H^*, I_V^*)}{f(S_H, I_V^*)} + \frac{f(S_H, I_V)}{f(S_H, I_V^*)} \right].
$$

Let us calculate  $\dot{U}_{I_H}$ :

$$
\dot{U}_{I_H} = \left(1 - \frac{I_H^*}{I_H}\right) \dot{I}_H \n= \left(1 - \frac{I_H^*}{I_H}\right) (f(S_H, I_V) - (\mu_H + \gamma_H) I_H^* \frac{I_H}{I_H^*}).
$$

By using the second equation of system [\(9\)](#page-7-0), we get

$$
\dot{U}_{I_H} = \left(1 - \frac{I_H^*}{I_H}\right) (f(S_H, I_V) - f(S_H^*, I_V^*) \frac{I_H}{I_H^*})
$$
\n
$$
= f(S_H^*, I_V^*) \left(1 - \frac{I_H^*}{I_H}\right) \left(\frac{f(S_H, I_V)}{f(S_H^*, I_V^*)} - \frac{I_H}{I_H^*}\right)
$$
\n
$$
= f(S_H^*, I_V^*) \left(\frac{f(S_H, I_V)}{f(S_H^*, I_V^*)} - \frac{I_H}{I_H^*} - \frac{I_H^*}{I_H} \frac{f(S_H, I_V)}{f(S_H^*, I_V^*)} + 1\right).
$$

Let us now evaluate  $\dot{U}_H$ :

$$
\dot{U}_H = \dot{U}_{S_H} + \dot{U}_{I_H}
$$
\n
$$
= -\mu_H (S_H - S_H^*) \left( 1 - \frac{f(S_H^*, I_V^*)}{f(S_H, I_V^*)} \right) + f(S_H^*, I_V^*) Q(S_H, I_V),
$$

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where  $Q(S_H, I_V) = 2 - \frac{f(S_H^*, I_V^*)}{f(S_H - I_W^*)}$  $\frac{f(S^*_H,I^*_V)}{f(S_H,I^*_V)} + \frac{f(S_H,I_V)}{f(S_H,I^*_V)}$  $\frac{f(S_H,I_V)}{f(S_H,I_V^*)}-\frac{I_H}{I_H^*}$  $I_H^*$  $-\frac{I_H^*}{I}$  $I_H$  $f(S_H,I_V)$  $\frac{f(S_H, I_V)}{f(S_H^*, I_V^*)}$ . By adding and subtracting  $1+\ln \frac{f(S_H^*, I_V^*)}{f(S_H^*, I_V^*)}$  $\frac{f(S_H^*, I_V^*)}{f(S_H, I_V^*)} + \ln \frac{f(S_H, I_V)}{f(S_H, I_V^*)} + \ln \frac{I_H}{I_H^*}$ to and from  $Q(S_H, I_V)$ , we get

$$
Q(S_H, I_V) = \left( -\frac{f(S_H^*, I_V^*)}{f(S_H, I_V^*)} + 1 + \ln \frac{f(S_H^*, I_V^*)}{f(S_H, I_V^*)} \right) + \left( -\frac{I_H}{I_H^*} + 1 + \ln \frac{I_H}{I_H^*} \right) + \left( \frac{f(S_H, I_V)}{f(S_H, I_V^*)} - 1 - \ln \frac{f(S_H, I_V)}{f(S_H, I_V^*)} \right) \left( -\frac{I_H^*}{I_H} \frac{f(S_H, I_V)}{f(S_H^*, I_V^*)} + 1 + \ln \frac{I_H^*}{I_H} \frac{f(S_H, I_V)}{f(S_H^*, I_V^*)} \right) = -h \left( \frac{f(S_H^*, I_V^*)}{f(S_H, I_V^*)} \right) - h \left( \frac{I_H}{I_H} \right) + h \left( \frac{f(S_H, I_V)}{f(S_H, I_V^*)} \right) - h \left( \frac{I_H^*}{I_H} \frac{f(S_H, I_V)}{f(S_H^*, I_V^*)} \right).
$$

Let us calculate  $\dot{U}_{S_V}$ :

$$
\dot{U}_{S_V} = \left(1 - \frac{g(S_V^*, I_H^*)}{g(S_V, I_H^*)}\right)\dot{S}_V
$$
\n
$$
= \left(1 - \frac{g(S_V^*, I_H^*)}{g(S_V, I_H^*)}\right)(A - g(S_V, I_H) - \mu_V S_V).
$$

By using the third equation of system [\(9\)](#page-7-0), we obtain

$$
\dot{U}_{S_V} = -\mu_V(S_V - S_V^*) \left( 1 - \frac{g(S_V^*, I_H^*)}{g(S_V, I_H^*)} \right) \n+g(S_V^*, I_H^*) \left( 1 - \frac{g(S_V, I_H)}{g(S_V^*, I_H^*)} - \frac{g(S_V^*, I_H^*)}{g(S_V, I_H^*)} + \frac{g(S_V, I_H)}{g(S_V, I_H^*)} \right).
$$

Let us calculate  $\dot{U}_{I_V}$ :

$$
\dot{U}_{I_V} = \left(1 - \frac{I_V^*}{I_V}\right) \dot{I}_V
$$
\n
$$
= g(S_V^*, I_H^*) \left(1 + \frac{g(S_V, I_H)}{g(S_V^*, I_H^*)} - \frac{I_V}{I_V^*} - \frac{I_V^*}{I_V} \frac{g(S_V, I_H)}{g(S_V^*, I_H^*)}\right).
$$

Let us now evaluate  $\dot{U}_V$ :

$$
\dot{U}_V = \dot{U}_{S_V} + \dot{U}_{I_V}
$$
\n
$$
= -\mu_V (S_V - S_V^*) \left( 1 - \frac{g(S_V^*, I_H^*)}{g(S_V, I_H^*)} \right) + g(S_V^*, I_H^*) \Psi(S_V, I_H),
$$

where  $\Psi(S_V, I_H) = 2 - \frac{g(S_V^*, I_H^*)}{g(S_V^*, I_H^*)}$  $\frac{g(S_V^*, I_H^*)}{g(S_V, I_H^*)} + \frac{g(S_V, I_H)}{g(S_V, I_H^*)}$  $\frac{g(S_V,I_H)}{g(S_V,I_H^*)}-\frac{I_V}{I_V^*}$  $\overline{I_V^*}$  $-\frac{I_V^*}{I}$  $I_V$  $g(S_V, I_H)$  $rac{g(S_V, I_H)}{g(S_V^*, I_H^*)}$ . By adding and subtracting  $1 + \ln \frac{g(S_Y^*, I_H^*)}{g(S_H^*, I_H^*)}$  $g(S_V, I_H^*)$ +  $\ln \frac{g(S_V, I_H)}{g(S_V, I_H^*)}$  $+\ln \frac{I_V}{I_V^*}$ to and from  $\Psi(S_V, I_H)$ , we have

$$
\Psi(S_V, I_H) = \left( -\frac{g(S_V^*, I_H^*)}{g(S_V, I_H^*)} + 1 + \ln \frac{g(S_V^*, I_H^*)}{g(S_V, I_H^*)} \right) + \left( -\frac{I_V}{I_V^*} + 1 + \ln \frac{I_V}{I_V^*} \right) \n+ \left( \frac{g(S_V, I_H)}{g(S_V, I_H^*)} - 1 - \ln \frac{g(S_V, I_H)}{g(S_V, I_H^*)} \right) \n+ \left( -\frac{I_V^*}{I_V} \frac{g(S_V, I_H)}{g(S_V^*, I_H^*)} + 1 + \ln \frac{I_V^*}{I_V} \frac{g(S_V, I_H)}{g(S_V^*, I_H^*)} \right) \n= -h \left( \frac{g(S_V^*, I_H^*)}{g(S_V, I_H^*)} \right) - h \left( \frac{I_V}{I_V^*} \right) + h \left( \frac{g(S_V, I_H)}{g(S_V, I_H^*)} \right) - h \left( \frac{I_V^*}{I_V} \frac{g(S_V, I_H)}{g(S_V^*, I_H^*)} \right)
$$

Let  $\zeta = \max\{f(S_H^*, I_V^*); g(S_V^*, I_H^*)\},\$ 

$$
\dot{U} \leq -\mu_H(S_H - S_H^*) \left( 1 - \frac{f(S_H^*, I_V^*)}{f(S_H, I_V^*)} \right) - \mu_V(S_V - S_V^*) \left( 1 - \frac{g(S_V^*, I_H^*)}{g(S_V, I_H^*)} \right) \n+ \zeta(Q(S_H, I_V) + \Psi(S_V, I_H)).
$$

By using the assumption H5, we have

$$
h\left(\frac{f(S_H, I_V)}{f(S_H, I_V^*)}\right) \le h\left(\frac{I_V}{I_V^*}\right) \text{ and } h\left(\frac{g(S_V, I_H)}{g(S_V, I_H^*)}\right) \le h\left(\frac{I_H}{I_H^*}\right),
$$

thus, from the assumption H6, we can see that  $\dot{U} \leq 0$ . In addition, we can see that U > 0 for all  $S_H, I_H, S_V, I_V \in \mathbb{R}_+$  and  $U = 0$  for  $S_H = S_H^*$ ,  $I_H = I_H^*$ ,  $S_V = S_V^*$  and  $I_V = I_V^*$ . Then the equilibrium state  $E^*$  is the only positively invariant set of the system [\(3\)](#page-3-2) contained in  $\{(S_H, I_H, S_V, I_V) \in \mathbb{R}_+^4; S_H = S_H^*, I_H = I_H^*, S_V = S_V^*$  and  $I_V = I_V^*\}$ and hence, by the asymptotic stability theorem [\[17\]](#page-15-9), the unique endemic equilibrium state  $E^*$  is globally asymptotically stable on  $\mathcal{D}$ .

#### 5 Examples of Incidence Functions

In this section, we give the examples of incidence functions for which the required hypotheses are satisfied.

- 1. Mass action incidence. These incidence functions are defined by  $f(S_H, I_V)$  =  $\alpha_1 S_H I_V$  and  $g(S_V, I_H) = \alpha_2 S_V I_H$ , where  $\alpha_1$  is the positive contact rate between a susceptible human and an infectious mosquito and  $\alpha_2$  designs the positive contact rate between a susceptible mosquito and an infectious human. Then hypotheses  $(H1) - (H6)$  are satisfied and so the global dynamics are determined by the magnitude of the basic reproduction number  $\mathcal{R}_0$ .
- 2. Saturating incidence. Let  $f(S_H, I_V) = S_H \frac{I_V}{1 + \epsilon_0}$  $\frac{1}{1 + c_1 I_V}$  and  $g(S_V, I_H)$  =  $S_V \frac{I_H}{1+\epsilon}$

 $\frac{1}{1+c_2I_H}$ , where  $c_1$  and  $c_2$  are non-negative constant. Then hypotheses  $(H1) - (H6)$  are satisfied and so the global dynamics are determined by the value of  $\mathcal{R}_0$ .

3. **Standard incidence.** These functions are given by  $f(S_H, I_V) = \frac{S_H I_V}{S_H + I_H}$  and  $g(S_V, I_H) = \frac{S_V I_H}{S_V + I_V}$ . Then the assumptions (**H1**) – (**H6**) are satisfied and so the global dynamics are given by the value of the basic reproduction number  $\mathcal{R}_0$ .

.

In the following paragraph, we carry out the numerical simulation taking the mass action law as the incidence function. But we specify that the dynamics remains the same as with the other incidence functions.

# <span id="page-11-0"></span>6 Simulation and Comments

In this section, we carry out the computation work that supports our study. In our simulation, we used the mass action as the incidence functions which are defined by  $f(S_H, I_V) = \alpha_1 S_H I_V$  and  $g(S_V, I_H) = \alpha_2 S_V I_H$ , where  $\alpha_1$  and  $\alpha_2$  are positives constants. We present the graphics which illustrate the evolution of the different classes in two cases: when  $\mathcal{R}_0 \leq 1$  and  $\mathcal{R}_0 > 1$ . The parameter values used in our simulation are:  $\tilde{\Lambda} = 200$ ;  $\mu_H = 0.3; \mu_V = 0.2; \gamma_H = 0.4; \alpha_1 = 0.0005; \alpha_2 = 0.0021; A = 100.$  From these values, we have  $\mathcal{R}_0 = 0.87$ . When we change the values of  $\alpha_1$  and  $\alpha_2$  to  $\alpha_1 = 0.001$  and  $\alpha_2 = 0.21$ , we get  $\mathcal{R}_0 = 12.25$ . The software used for the simulation is scilab.

<span id="page-11-2"></span>

<span id="page-11-3"></span>(a) human population,  $\mathcal{R}_0 < 1$ . (b) human population,  $\mathcal{R}_0 > 1$ .

**Figure 2:** Dynamics of the human population for different magnitudes of  $\mathcal{R}_0$ . Figure [2a](#page-11-2) give the dynamic of susceptible, infectious and remove, in model [\(2\)](#page-3-1). These curves also indicate that the disease tends to disappear. Figure [2b](#page-11-3) presents the dynamic of the same classes, these curves show us that the disease persists in the population.

# <span id="page-11-1"></span>7 Global Sensitivity Analysis for  $\mathcal{R}_0$

In this paragraph, we use the notion of sensitivity analysis to show the importance of different parameters in the basic reproduction number  $\mathcal{R}_0$ . In Subsection [7.1,](#page-12-0) we define the notion of sensitivity of some parameter  $p$  of the model [\(2\)](#page-3-1). Subsection [7.2](#page-12-1) is devoted to the calculations of the analytical expressions of the sensitivity indices of different parameters in the basic reproduction number. In Subsection [7.3,](#page-13-0) we give a numerical representation and comments for different sensitivity indice.

<span id="page-12-2"></span>

<span id="page-12-3"></span>**Figure 3:** Dynamics of the vector population for different values of  $\mathcal{R}_0$ . Figure [3a](#page-12-2) designs the dynamic of susceptible and infectious mosquitoes, in model [\(2\)](#page-3-1). The graphs together also show that the disease tends to disappear. Figure [3b](#page-12-3) shows the dynamic of the vector population, these curves indicate that the disease will persist in the population.

#### <span id="page-12-0"></span>7.1 Definition

Let p be a parameter of the mathematical model  $(2)$ . The parameter p is said to be sensitive if any small alteration of p causes a significant change in the solution. It is worthy to note that the parameter  $p$  is termed to be locally sensitive if the change in the value of the parameter  $p$  influences the output of the model. In the same way, global sensitivity takes into account the overall change in the model output as a result of the change in all parameter values within their respective range [\[18\]](#page-15-10).

In computing the normalized sensitivity index  $(\wp_p^{\mathcal{R}_0})$  for the basic reproduction number  $\mathcal{R}_0$  for each parameter p, we use the relation given by

<span id="page-12-4"></span>
$$
\wp_p^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial p} \times \frac{p}{\mathcal{R}_0}.\tag{10}
$$

# <span id="page-12-1"></span>7.2 Analytic representation of the elasticity

We use the law of mass action as an incidence function. The general incidence functions f and g are defined by the relations  $f(S_H, I_V) = \beta S_H I_V$  and  $g(S_V, I_H) = \epsilon S_V I_H$ . In this case, the expression of the basic reproduction number  $\mathcal{R}_0$  is given by the relation

$$
\mathcal{R}_0 = \sqrt{\frac{\tilde{\beta}\epsilon\tilde{\Lambda}A}{\mu_H\mu_V^2(\mu_H + \gamma_H)}}.
$$

The sensitivity indices of different parameters are given as follows.

Using the principle given by [\(10\)](#page-12-4), we obtain

$$
\wp_{\tilde{\beta}}^{\mathcal{R}_0} = \frac{1}{2}, \ \ \wp_{\epsilon}^{\mathcal{R}_0} = \frac{1}{2}, \ \wp_{\tilde{\Lambda}}^{\mathcal{R}_0} = \frac{1}{2}, \ \ \wp_{A}^{\mathcal{R}_0} = \frac{1}{2},
$$

$$
\wp^{\mathcal{R}_0}_{\gamma_H} = -\frac{1}{2} \frac{\gamma_H}{\mu_H + \gamma_H}, \ \wp^{\mathcal{R}_0}_{\mu_V} = -1, \ \wp^{\mathcal{R}_0}_{\mu_H} = -\frac{2\mu_H + \gamma_H}{2}.
$$

Parameter	Description	Elasticity index
	Humans recruitment rate	0.5
	Positive contact rate between $I_V$ and $S_H$	0.5
$\epsilon$	Positive contact rate between $I_H$ and $S_V$	0.5
A	Mosquito recruitment rate	0.5
$\gamma_H$	Infectious humans who pass in $R_H$	$-0.28$
$\mu_V$	Natural death of mosquito	$-1$
$\mu_H$	Natural death of humans	$-0.5$

Table 1: Parameter description and elasticity value.

#### <span id="page-13-0"></span>7.3 Numerical representation and comments

<span id="page-13-1"></span>In this subsection, we give some numerical representation and comments for different sensitivity indices while the analytical expressions and values are obtained in Subsection [7.2.](#page-12-1) For the numerical representation, we use the R software and the graph is given in Figure [4.](#page-13-1)



Figure 4: Global sensitivity plot.

Parameters with a positive sensitivity index indicate an increase in the transmission of dengue in the population for an increase in these values. On the other hand, parameters with a negative sensitivity index mean that an increase in these values leads to a decrease in the transmission of dengue in the population. For example, the sensitivity index of  $\Lambda$ in  $\mathcal{R}_0$  is 0.5. This implies that an increase of 1% in the value of  $\Lambda$  leads to an increase of 0.5% in the value of  $\mathcal{R}_0$ . The sensitivity indices of  $\tilde{\Lambda}$ ,  $\tilde{\beta}$ ,  $\epsilon$  and A are the same, which means that these parameters have the same impact on the secondary infection rate. In the same way, the elasticity of  $\mu_V$  in  $\mathcal{R}_0$  is  $-1$  meaning that the increase of 1% in the value of mosquito mortality implies the decrease of  $1\%$  in the value of  $\mathcal{R}_0$ . The fact that  $\wp_{\mu}^{\mathcal{R}_0} = -0.28$  means that 1% increase in  $\mu_V$  will produce 0.28% decrease in  $\mathcal{R}_0$ . Also,

the fact that  $\wp_{\gamma_H}^{\mathcal{R}_0} = -0.5$  implies that 1% increase in  $\gamma_H$  will produce 0.5% decrease in the value of the basic reproduction number.

Thus, we find that the parameter  $\mu_V$ , which denotes the mosquito mortality rate, is a good parameter for controlling the dynamics of dengue transmission. As it increases, the basic reproduction number  $\mathcal{R}_0$  decreases more rapidly. However, it is not the only parameter whose growth leads to a decrease in the basic reproduction number.

# 8 Conclusion

In this paper, we have studied the dengue disease transmission model, which includes the human and vector populations with general admission incidence functions. We proved the existence of the equilibrium and its stability. When the value of the basic reproduction number  $\mathcal{R}_0$  is less than unity, the disease-free equilibrium is globally asymptotically stable, in this case the disease will disappear. When  $\mathcal{R}_0 > 1$ , the endemic equilibrium exists and it is globally asymptotically stable, in this case the disease will persist in the population. We used the Lyapunov function to study the stability of our equilibrium points. We have also presented the numerical simulations, and the evolution of our curves corroborate with the theoretical results. We carried out a sensitivity study of the parameters in order to determine the influence of different parameters on the transmission of the disease. We notice that the parameter  $\mu_V$ , which denotes the mortality rate of the mosquitoes, allows to better control the dynamics of dengue disease transmission. In our future work we will integrate the spatial distribution of the disease.

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