Modelling and Analysis of Spread Characteristics of Arbovirus Infections

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Abstract: The problem of the definition of a mathematical model for epidemics, where the virus transmission is operated by an infected vector like an insect, is addressed. Through the consideration of a small number of compartments, in order to keep the mathematical analysis affordable, a five dimensional model is proposed and, successively, used for the analysis of the main characteristics of the disease. Closed form expression are then obtained for the equilibria, the stabiliy conditions and the reproduction number. Some numerical results referring to the dengue disease emergency are also presented, firstly to identify the unknown model parameters and then to validate the effectiveness of the model itself. Using such a model, some considerations on the most effective action lines for spidemic containment are discussed.

1 INTRODUCTION

Globalization and climate change make the problem of epidemics spread an emergency to be addressed with coordinated strategies, acting promptly with preventive campaigns, firstly for geographical containment, and then for possible eradication.

An effective approach to the problem makes use of mathematical models for description, analysis, prediction and control the diseases evolution (Di Giamberardino et al., 2019; Attaullah and Sohaib, 2020; Di Giamberardino et al., 2021; Diagne et al., 2021; Ayele et al., 2021; Di Giamberardino and Iacoviello, 2021; Alutto et al., 2024). In particular, arboviruses are diseases (zoonoses) caused by agents (viruses) transmitted from animals (arthropod vectors) to humans, through bites or stings. Among the arboviruses, dengue, yellow fever, chikungunya and Zika viruses are currently an emergency in tropical and subtropical areas, involving almost 4 billion of people; due to this growing threat, the World Health Organization (WHO) launched the Global Arbovirus Initiative on March 31, 2022, proposing a plan to tackle arboviruses threat by means of pandemic prevention, risk analysis, communication and coordination.

For the particular case of the dengue, there has been an increment in the last years, from 505430 cases in 2000 to more than 5 million in 2019, with a spike of 6.5 million cases in 2023 and with about 7300 deaths reported. It is estimated that up to 400 million infections are occurring in the last years, putting approximately half of the world's population at risk, (CDC, 2024). This viral infection is transmitted to humans by the bite of infected mosquitoes, mainly the Aedes aegypti one; the mosquitoes can get the virus when they bite a subject in a symptomatic, presymptomatic or asymptomatic condition, especially when the patients have high fever or with significant viremia.

Generally, individuals infected by dengue do not have symptoms, loosing the viral load after 2 weeks. Symptoms can appear about 1 week after the infection and last at most for a week; they include fever, headache, nausea, rash; severe symptoms include abdominal pain, persistent vomiting with blood, feeling weak also for several weeks. Severe dengue generally occurs with a second infection; the mortality among severely infected individuals is quite high, with even 20% of fatalities.

Up to now, there is no specific treatment for severe dengue and the current therapy generally focuses on treating pain symptoms; the early detection with access to medical care can reduce fatality rates (WHO, 2024a).

The first containment measure is prevention, acting to reduce the possibility of mosquitoes bites: window screens, mosquitoes repellents and use of clothes covering as much of the body as possible. Moreover, mosquito breeding can be prevented by suitable managing water storage and avoiding mosquitoes from ac-

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cessing egg-laying habitats.

Despite dengue diffusion depends on particular environmental and climate conditions, climate change and movement of population are making all the arboviruses a global emergency, reaching regions not involved up to few years ago.

Dengue has been studied in literature especially since the last years of the 20^{th} century, mainly using the typical epidemic modelling considering the peculiarity of the presence of a vector and a host to spread the virus. An effective approach considers two interacting models, one for human population and one for the mosquitoes. For the former, a classical *SEIR* model can be chosen, where *S* stands for the susceptible individuals, *E* for the exposed, that is for the subjects infected but not infectious, *I* for the infected patients, *R* for the part of the population recovered or dead. For the vector population a smaller one, for example a *SEI* model is sufficient. This approach is followed in (Yi et al., 2021), where the estimation and the forecast of dengue outbreak dynamics are performed by means of an ensemble Kalman filter. A similar approach is implemented in (Schaum et al., 2022), with reference to data from Mexico.

As said, a control of dengue spread could be implemented by means of prevention, quarantining severe patients, medical treatments and, in the last years (in Europe only since 2022), also by vaccination. In (Sow et al., 2024) it is shown that the most effective approach is a suitable integration of vaccination and treatment. When contemporary different requirements are considered, especially in presence of resource limitation, optimal control, as confirmed by previous results on different epidemics (Ayele et al., 2021; Zhu et al., 2021; Di Giamberardino and Iacoviello, 2017), seems the framework for effective solutions (Abidemi et al., 2024); the results put in evidence the success of a coordinated application of all the possible prevention actions.

A peculiarity of vector borne disease is the influence of climate factors on the vector presence and diffusion. Favorable climate and environment conditions, as well as prolonged weather anomalies, facilitate virus spread also in regions not used to such epidemics, (Barcellos et al., 2024). This implies the importance of suitably modelling the dynamics taking account spatial and temporal diffusion of the virus; in particular, the periodicity of dengue fever is related to local temperature, humidity and precipitation characteristics, with an associated time-lag, (Xu et al., 2024). An important contribution is given by the study of data time series which, unfortunately, often are not available with lack in completeness and consistency, (Arquam et al., 2020).

In this paper, it is proposed a model in which the human population is described by a *SIR* model where the set of infected patients is split into asymptomatic patients, not aware of having contracted the dengue virus and for which the infection does not produce consequences for their health, and the patients who have severe dengue symptoms. The mosquitoes population is described, in this preliminary study, by means of a *SI* model, since the infection has no effects on the insect life. All the patients can infect an uninfected mosquito; on the other hand, the infected mosquito can spread the virus to susceptible humans. Therefore, the complete model is represented by the two populations with suitable interconnections.

The paper is organized as follows. The mathematical model is introduced and described in Section 2. Section 3 is devoted to the model analysis, including equilibria and stability, with particular emphasis on the relationships with the epidemic spread characteristics. Identification and validation procedures are reported in Section 4, where some considerations on the influence of dynamics characteristics, in term of parameters values, to the qualitative behaviour of the epidemics are reported. A concluding Section 5 ends the paper.

2 THE MATHEMATICAL MODEL

The mathematical model adopted is based on the interactions of two populations dynamics, the human and the mosquitoes, with their natural growth and with their mutual infectious interactions: a mosquito biting an infected human becomes infectious, an infected mosquito which bites a health human infects the person in a light (that is without evident consequences), or dangerous (with serious consequences, even the death) way.

A block scheme is reported in Fig. 1 for a fast visual representation.

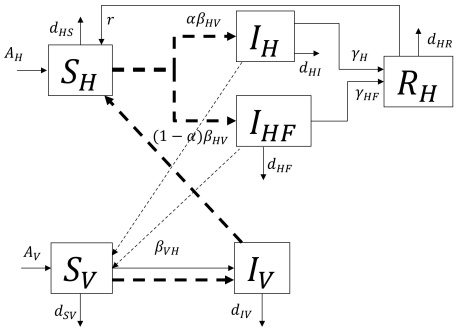

Figure 1: Block diagram of the proposed model.

The differential equations describing the full dynamics can be given by

$$\dot{S}_H = -\beta_{HV} S_H I_V - d_{S_H} S_H + A_H + r R_H \quad (1)$$

$$\dot{I}_H = \alpha\beta_{HV} S_H I_V - \gamma_{I_H} I_H - d_{I_H} I_H \quad (2)$$

$$\dot{I}_{HF} = (1-\alpha)\beta_{HV} S_H I_V - \gamma_{I_{HF}} I_{HF} - d_{I_{HF}} I_{HF} \quad (3)$$

$$\dot{R}_H = \gamma_{I_H} I_H + \gamma_{I_{HF}} I_{HF} - d_{R_H} R_H - r R_H \quad (4)$$

$$\dot{S}_V = a S_V - d_{S_V} S_V - \beta_{VH} S_V I_H - \varphi\beta_{VH} S_V I_{HF} + A_V \quad (5)$$

$$\dot{I}_V = \beta_{VH} S_V I_H + \varphi\beta_{VH} S_V I_{HF} - d_{I_V} I_V \quad (6)$$

The first four state variables, S_H, I_H, I_{HF} and R_H, are referred to the human population, representing the susceptible individuals, S_H, the infected ones with no or negligible symptoms, I_H, the infected ones with symptoms, I_{HF}, and in danger of their life, and the removed, R_H, persons, the ones healed from the infection with or without symptoms.

The remaining two state variables describe the insect vectors dynamics and their infection: the uninfected, S_V, and the infected, I_V, insects.

In the model, β_{HV} denotes the infection transmission rate between a susceptible person S_H and an infected insect I_V. It is a coefficient that includes many factors like the probability to be infected, the infective capability of the insects, the robustness of the individuals with respect to the contact with the virus, and so on. Under this definition, $\beta_{HV} S_H I_V$ is the rate of new infected individuals that move from the class S_H to the classes I_H or I_{HV}, with a fraction $\alpha < 1$ without any symptom and any consequences for his actual and future health, and the remaining fraction $(1-\alpha)$ of fragile individuals for which the consequences can be serious and even fatal.

Symmetrically, β_{VH} represents the infection transmission rate between a susceptible (uninfected) mosquito S_V and an infected human I_H or I_{HV}.

The rate of new infected vectors due to bites to an infected human in I_H is given by $\beta_{VH} S_V I_H$, while, under the hypothesis that a fragile infected individual in I_{HF} is suitable protected and isolated, the rate of new infected vectors due to bites to an infected human in I_H is given by a fraction $\varphi < 1$ of $\beta_{VH} S_V I_{HF}$.

Other therms in the mathematical model are the death rates in each class d_{S_H}, d_{I_H}, $d_{I_{HF}}$, d_{R_H}, d_{S_V}, d_{I_V}, the healing rates γ_{I_H} and $\gamma_{I_{HF}}$, the reproduction rate a of the insects, the new individual incoming in humans (A_H) and mosquito (A_V). Finally, it is considered the possibility that one infected individual, after a sufficiently long time following healing, can be infected again with the time constant $1/r$.

3 MODEL ANALYSIS

An analysis of the qualitative behaviour of the system is reported in this Section. A characterization of the spread and the intensity of the epidemic, as well as the case in which, also in presence of infectious individuals, the epidemic autonomously vanishes is given.

The tools for such an analysis here used are two. Firstly, following the classical dynamical systems approach, the determination of the equilibrium points of the system and the study of their stability are performed; then, according to the usual approach followed in epidemic analysis, the main parameter which describes the dangerousness of a viral disease, the basic reproduction number, is computed from the model. The equivalence of the two approaches is proved.

3.1 Equilibrium Points

In order to compute if and how many equilibrium points the system has, the solution of the nonlinear system

$$-\beta_{HV} S_H I_V - d_{S_H} S_H + A_H + r R_H = 0 \quad (7)$$

$$\alpha\beta_{HV} S_H I_V - m_H I_H = 0 \quad (8)$$

$$(1-\alpha)\beta_{HV} S_H I_V - m_{HV} I_{HF} = 0 \quad (9)$$

$$\gamma_{I_H} I_H + \gamma_{I_{HF}} I_{HF} - m_{R_H} R_H = 0 \quad (10)$$

$$(a - d_{S_V}) S_V - \beta_{VH} S_V I_H - \varphi\beta_{VH} S_V I_{HF} + A_V = 0 \quad (11)$$

$$\beta_{VH} S_V I_H + \varphi\beta_{VH} S_V I_{HF} - d_{I_V} I_V = 0 \quad (12)$$

must be computed, where the positions

$$m_H = \gamma_{I_H} + d_{I_H} \quad (13)$$

$$m_{HV} = \gamma_{I_{HF}} + d_{I_{HF}} \quad (14)$$

$$m_{R_H} = d_{R_H} + r \quad (15)$$

have been used for sake of compactness.

Rewriting equation (11), one can put in evidence the susceptible insects S_V, obtaining

$$-\left(\beta_{VH} I_H + \varphi\beta_{VH} I_{HF} - (a - d_{S_V})\right) S_V + A_V = 0$$

from which it can be written

$$S_V = \frac{A_V}{\left(\beta_{VH} I_H + \varphi\beta_{VH} I_{HF} - (a - d_{S_V})\right)} \quad (16)$$

Moreover, the sum of (11) and (12) gives

$$(a - d_{S_V}) S_V + A_V - d_{I_V} I_V = 0$$

from which the relationship

$$I_V = \frac{(a - d_{S_V})}{d_{I_V}} S_V + \frac{A_V}{d_{I_V}}$$

can be obtained and then, by substitution, the expression

$$I_V = \frac{A_V}{d_{I_V}} \left(\frac{\beta_{VH} I_H + \varphi \beta_{VH} I_{HF}}{(\beta_{VH} I_H + \varphi \beta_{VH} I_{HF} - (a - d_{S_V}))} \right) \quad (17)$$

is given.

Note that a necessary and sufficient condition for existence of the equilibrium is

$$\beta_{VH} I_H + \varphi \beta_{VH} I_{HF} - (\alpha - d_{S_V}) > 0 \quad (18)$$

since all the state values must be non negative.

Looking at the equations of the human population, from (10) it is obtained

$$R_H = \frac{\gamma_{I_H}}{m_{R_H}} I_H + \frac{\gamma_{I_{HF}}}{m_{R_H}} I_{HF} \quad (19)$$

Summing the first three (7), (8) and (9) and making use of (19), the expression

$$\begin{aligned} &-d_{S_H} S_H - \left(d_{I_H} + \frac{d_{R_H} \gamma_{I_H}}{m_{R_H}} \right) I_H \\ &- \left(d_{I_{HF}} + \frac{d_{R_H} \gamma_{I_{HF}}}{m_{R_H}} \right) I_{HF} + A_H = 0 \end{aligned} \quad (20)$$

is obtained, from which it is possible to have the equilibrium value of the susceptible human population as a function of the infected individuals, I_H and I_{HF}:

$$\begin{aligned} S_H = &\frac{A_H}{d_{S_H}} - \left(\frac{d_{I_H} m_{R_H} + d_{R_H} \gamma_{I_H}}{m_{R_H} d_{S_H}} \right) I_H \\ &- \left(\frac{d_{I_{HF}} m_{R_H} + d_{R_H} \gamma_{I_{HF}}}{m_{R_H} d_{S_H}} \right) I_{HF} \end{aligned} \quad (21)$$

This formula can be written in a more compact form, setting

$$\begin{aligned} C_1 &= \frac{d_{I_H} m_{R_H} + d_{R_H} \gamma_{I_H}}{m_{R_H} d_{S_H}} \\ C_2 &= \frac{d_{I_{HF}} m_{R_H} + d_{R_H} \gamma_{I_{HF}}}{m_{R_H} d_{S_H}} \end{aligned}$$

with $C_1 > 0$ and $C_2 > 0$, so getting

$$S_H = \frac{A_H}{d_{S_H}} - C_1 I_H - C_2 I_{HF} \quad (22)$$

Non negativeness of the solution gives rise to the constraint

$$\frac{A_H}{d_{S_H}} > C_1 I_H + C_2 I_{HF} \quad (23)$$

In order to find the expressions of I_H and I_{HF}, it is useful to find their relationship making use of the linear combination of (8) and (9)

$$\begin{aligned} &(1-\alpha)(\alpha \beta_{HV} S_H I_V - m_H I_H) - \\ &\alpha((1-\alpha)\beta_{HV} S_H I_V - m_{HV} I_{HF}) = 0 \end{aligned} \quad (24)$$

which gives the dependency between the two classes of infected individuals

$$(1-\alpha) m_H I_H = \alpha m_{HV} I_{HF} \quad (25)$$

once $\alpha \neq 0$ and $\alpha \neq 1$. The limit cases can be easily studied, since they correspond to the absence of one of the two classes of infected humans.

From one of (8) or (9), due to (25), it is possible to define the equation with respect one of the two variables I_H or I_{HF}. Computing the expression for $S_H I_V$

$$\begin{aligned} S_H I_V = &\frac{A_V}{d_{I_V}} \left(\frac{A_H}{d_{S_H}} - C_1 I_H - C_2 I_{HF} \right) \cdot \\ &\left(\frac{\beta_{VH} I_H + \varphi \beta_{VH} I_{HF}}{(\beta_{VH} I_H + \varphi \beta_{VH} I_{HF} - (a - d_{S_V}))} \right) \end{aligned} \quad (26)$$

and making use of (8), one gets

$$\begin{aligned} &\alpha \beta_{HV} \frac{A_V}{d_{I_V}} \left(\frac{A_H}{d_{S_H}} - C_1 I_H - C_2 I_{HF} \right) \cdot \\ &\left(\frac{\beta_{VH} I_H + \varphi \beta_{VH} I_{HF}}{(\beta_{VH} I_H + \varphi \beta_{VH} I_{HF} - (a - d_{S_V}))} \right) - m_H I_H = 0 \end{aligned} \quad (27)$$

From (25), rewritten as

$$I_{HF} = \frac{(1-\alpha)}{\alpha} \frac{m_H}{m_{HV}} I_H \quad (28)$$

by substitution, the equation in the variable I_H is obtained

$$\begin{aligned} &\alpha \beta_{HV} \frac{A_V}{d_{I_V}} \left(\frac{A_H}{d_{S_H}} - (C_1 + C_2 \frac{(1-\alpha)}{\alpha} \frac{m_H}{m_{HV}}) I_H \right) \cdot \\ &\left(\frac{(\beta_{VH} + \varphi \beta_{VH} \frac{(1-\alpha)}{\alpha} \frac{m_H}{m_{HV}}) I_H}{\left((\beta_{VH} + \varphi \beta_{VH} \frac{(1-\alpha)}{\alpha} \frac{m_H}{m_{HV}}) I_H - (a - d_{S_V}) \right)} \right) \\ &- m_H I_H = 0 \end{aligned} \quad (29)$$

Expanding the computations and setting

$$C_3 = \beta_{VH} + \varphi \beta_{VH} \frac{(1-\alpha)}{\alpha} \frac{m_H}{m_{HV}} \quad (30)$$

$$C_4 = C_1 + C_2 \frac{(1-\alpha)}{\alpha} \frac{m_H}{m_{HV}} \quad (31)$$

where $C_3 > 0$ and $C_4 > 0$, the equation

$$\begin{aligned} &\alpha \beta_{HV} \frac{A_V}{d_{I_V}} \left(\frac{A_H}{d_{S_H}} - C_4 I_H \right) C_3 I_H \\ &- m_H (C_3 I_H - (a - d_{S_V})) I_H = 0 \end{aligned} \quad (32)$$

follows, which, reordered as

$$\begin{aligned} &\left(\alpha \beta_{HV} \frac{A_V}{d_{I_V}} \frac{A_H C_3}{d_{S_H}} + m_H (a - d_{S_V}) \right) I_H - \\ &\left(m_H C_3 + \alpha \beta_{HV} \frac{A_V}{d_{I_V}} C_3 C_4 \right) I_H^2 = 0 \end{aligned} \quad (33)$$

allows to obtain the two solutions

$$I_H = 0 \tag{34}$$

$$I_H = \frac{\left(\alpha\beta_{HV}\frac{A_V}{d_{I_V}}\frac{A_H C_3}{d_{S_H}} + m_H(a - d_{S_V})\right)}{\left(m_H C_3 + \alpha\beta_{HV}\frac{A_V}{d_{I_V}} C_3 C_4\right)} \tag{35}$$

The first one, (34), corresponds to the so called *epidemic free condition*, since, by backward substitutions, it gives

$$P^{e1} = \begin{pmatrix} S_H^{e1} \\ I_H^{e1} \\ I_{HF}^{e1} \\ R_H^{e1} \\ \hline S_V^{e1} \\ I_V^{e1} \end{pmatrix} = \begin{pmatrix} \frac{A_H}{d_{S_H}} \\ 0 \\ 0 \\ 0 \\ \hline \frac{A_V}{d_{S_V} - a} \\ 0 \end{pmatrix} \tag{36}$$

equivalent to the two independent populations, humans and insect vectors, without any infection at all.

From the second solution, renaming I_H in (35) as I_H^{e2}, one has

$$P^{e2} = \begin{pmatrix} S_H^{e2} \\ I_H^{e2} \\ I_{HF}^{e2} \\ R_H^{e2} \\ S_V^{e2} \\ I_V^{e2} \end{pmatrix} = \begin{pmatrix} \frac{A_H}{d_{S_H}} - C_4 I_H^{e2} \\ I_H^{e2} \\ \frac{(1-\alpha)}{\alpha}\frac{m_H}{m_{HV}} I_H^{e2} \\ \left(\frac{\gamma_{I_H}}{m_{R_H}} + \frac{\gamma_{I_{HF}}}{m_{R_H}}\frac{(1-\alpha)}{\alpha}\frac{m_H}{m_{HV}}\right) I_H^{e2} \\ \frac{A_V}{C_3 I_H^{e2} + (d_{S_V} - a)} \\ \frac{A_V}{d_{I_V}}\frac{C_3 I_H^{e2}}{(d_{S_V} - a)} \end{pmatrix} \tag{37}$$

corresponding to the usually denoted *endemic condition*.

Recalling all the existence conditions posed, P^{e1} does exist if and only if $d_{S_V} - a > 0$. If this condition were not fulfilled, the insect population would be characterized by a not feasible exponential growth. As far as P^{e2} is concerned, the existence condition (23) can be rewritten as

$$\frac{A_H}{d_{S_H}} - C_4 I_H^{e2} > 0$$

meaning that in endemic conditions, the number of not aware infected individuals I_H^{e2} satisfies

$$0 < I_H^{e2} < \frac{A_H}{C_4 d_{S_H}} \tag{38}$$

or, more useful for the epidemic consequences, the number of symptomatic individuals I_{HF}^{e2} verifies

$$0 < I_{HF}^{e2} < \frac{(1-\alpha)}{\alpha}\frac{m_H}{m_{HV}}\frac{A_H}{C_4 d_{S_H}}$$

To conclude the analysis, it must be remarked that

$$P^{e2}\big|_{I_H^{e2}=0} = P^{e1} \tag{39}$$

3.2 Stability

Local stability is studied hereinafter. Making use of the indirect method of Lyapunov, the Jacobian matrix describing the local dynamics must be preliminary computed

$$J = \left(\begin{array}{cccc|cc} -\beta_{HV} I_V - d_{S_H} & 0 & 0 & r & 0 & -\beta_{HV} S_H \\ \alpha\beta_{HV} I_V & -m_H & 0 & 0 & 0 & \alpha\beta_{HV} S_H \\ (1-\alpha)\beta_{HV} I_V & 0 & -m_{HV} & 0 & 0 & (1-\alpha)\beta_{HV} S_H \\ 0 & \gamma_{I_H} & \gamma_{I_{HF}} & -m_{R_H} & 0 & 0 \\ 0 & -\beta_{VH} S_V & -\varphi\beta_{VH} S_V & 0 & -(d_{S_V} - a) - \beta_{VH} I_H - \varphi\beta_{VH} I_{HF} & 0 \\ 0 & \beta_{VH} S_V & \varphi\beta_{VH} S_V & 0 & \beta_{VH} I_H + \varphi\beta_{VH} I_{HF} & -d_{I_V} \end{array}\right) \tag{40}$$

Evaluating it in the epidemic free condition P^{e1}, one has the dynamic matrix of the local linear approximation

$$\begin{pmatrix} -d_{S_H} & 0 & 0 & r & 0 & -\beta_{HV} S_H^{e1} \\ 0 & -m_H & 0 & 0 & 0 & \alpha\beta_{HV} S_H^{e1} \\ 0 & 0 & -m_{HV} & 0 & 0 & (1-\alpha)\beta_{HV} S_H^{e1} \\ 0 & \gamma_{I_H} & \gamma_{I_{HF}} & -m_{R_H} & 0 & 0 \\ 0 & -\beta_{VH} S_V^{e1} & -\varphi\beta_{VH} S_V^{e1} & 0 & -(d_{S_V} - a) & 0 \\ 0 & \beta_{VH} S_V^{e1} & \varphi\beta_{VH} S_V^{e1} & 0 & 0 & -d_{I_V} \end{pmatrix} \tag{41}$$

By visual inspection, it is easy to get the three eigenvalues $\lambda_1 = -d_{S_H}$, $\lambda_2 = -m_{R_H}$ and $\lambda_3 = -(d_{S_V} - a)$, real negative by definition or for the existence constraint. The remaining three are the eigenvalues of the reduced matrix

$$J_{1,rid} = \begin{pmatrix} -m_H & 0 & \alpha\beta_{HV} S_H^{e1} \\ 0 & -m_{HV} & (1-\alpha)\beta_{HV} S_H^{e1} \\ \beta_{VH} S_V^{e1} & \varphi\beta_{VH} S_V^{e1} & -d_{I_V} \end{pmatrix} \tag{42}$$

whose characteristic polynomial is

$$p(\lambda) = \lambda^3 + c_2\lambda^2 + c_1\lambda + c_0 = 0 \tag{43}$$

where

$$c_2 = m_H + m_{HV} + d_{I_V} > 0 \tag{44}$$

$$\begin{aligned} c_1 =& m_H m_{HV} + d_{I_V} m_{HV} - (1-\alpha)\beta_{HV} S_H^{e1} \varphi\beta_{VH} S_V^{e1} + \\ & d_{I_V} m_H - \alpha\beta_{HV} S_H^{e1} \beta_{VH} S_V^{e1} \\ =& m_H m_{HV} + d_{I_V} m_{HV} + d_{I_V} m_H - \\ & ((1-\alpha)\varphi + \alpha)\,\beta_{HV}\beta_{VH} S_H^{e1} S_V^{e1} \end{aligned} \tag{45}$$

$$\begin{aligned} c_0 =& m_H m_{HV} d_{I_V} - \\ & (m_{HV}\alpha + m_H\varphi(1-\alpha))\,\beta_{HV}\beta_{VH} S_V^{e1} S_H^{e1} \end{aligned} \tag{46}$$

The Lyapunov criterion is verified if and only if the characteristic polynomial (43) has all zeroes with negative real parts. This can be checked by means of the Routh criterion, which gives the following necessary and sufficient conditions:

$$\begin{aligned} c_2 &> 0 \\ c_1c_2 - c_0 &> 0 \\ c_0 &> 0 \end{aligned}$$

The first one is always verified. The third one is satisfied once

$$\beta_{HV}\beta_{VH}S_V^{e1}S_H^{e1} < \frac{m_H m_{HV} d_{I_V}}{m_{HV}\alpha + m_H\varphi(1-\alpha)} \tag{47}$$

For the second one, the preliminary computation $c_1c_2 - c_0$ is required. One has

$$\begin{aligned} c_1c_2 - c_0 =& c_2(m_H m_{HV} + d_{I_V} m_{HV} + d_{I_V} m_H) - \\ & m_H m_{HV} d_{I_V} - \\ & c_2\left((1-\alpha)\varphi + \alpha\right)\beta_{HV}\beta_{VH}S_H^{e1}S_V^{e1} + \\ & \left(m_{HV}\alpha + m_H\varphi(1-\alpha)\right)\beta_{HV}\beta_{VH}S_V^{e1}S_H^{e1} \end{aligned}$$

bringing to the condition

$$\begin{aligned} &\beta_{HV}\beta_{VH}S_V^{e1}S_H^{e1} \\ &< \frac{c_2(m_H m_{HV} + d_{I_V} m_{HV} + d_{I_V} m_H) - m_H m_{HV} d_{I_V}}{\left(c_2\left((1-\alpha)\varphi + \alpha\right) - \left(m_{HV}\alpha + m_H\varphi(1-\alpha)\right)\right)} \end{aligned} \tag{48}$$

Then, the epidemic free equilibrium is locally asymptotically stable if conditions (47) and (48) are satisfied. Since it is possible to verify that condition (47) implies (48), the only actual condition is (47). In fact, it is possible to prove that

$$\begin{aligned} &\frac{m_H m_{HV} d_{I_V}}{m_{HV}\alpha + m_H\varphi(1-\alpha)} < \\ &\frac{c_2(m_H m_{HV} + d_{I_V} m_{HV} + d_{I_V} m_H) - m_H m_{HV} d_{I_V}}{\left(c_2\left((1-\alpha)\varphi + \alpha\right) - \left(m_{HV}\alpha + m_H\varphi(1-\alpha)\right)\right)} \end{aligned}$$

To do so, rewriting it as

$$\begin{aligned} &\frac{c_2(m_H m_{HV} + d_{I_V} m_{HV} + d_{I_V} m_H) - m_H m_{HV} d_{I_V}}{\left(c_2\left((1-\alpha)\varphi + \alpha\right) - \left(m_{HV}\alpha + m_H\varphi(1-\alpha)\right)\right)} \\ &\qquad - \frac{m_H m_{HV} d_{I_V}}{m_{HV}\alpha + m_H\varphi(1-\alpha)} > 0 \end{aligned}$$

and performing the sum of the two terms, after some easy computations the numerator becomes

$$\begin{aligned} &m_H m_{HV} + d_{I_V} m_{HV} + d_{I_V} m_H)\left(m_{HV}\alpha + m_H\varphi(1-\alpha)\right) \\ &- m_H m_{HV} d_{I_V}\left((1-\alpha)\varphi + \alpha\right) = \\ &m_H m_{HV}\left(m_{HV}\alpha + m_H\varphi(1-\alpha)\right) + d_{I_V} m_{HV}\left(m_{HV}\alpha\right) \\ &+ d_{I_V} m_H\left(m_H\varphi(1-\alpha)\right) > 0 \end{aligned}$$

while it is easy to verify that the denominator is always positive.

It is interesting to evaluate the expression of (37) when condition (47) is satisfied. It is easy to verify preliminary that making use of (47) in (35), one has

$$\begin{aligned} I_H^{e2} =& \frac{\left(\alpha\beta_{HV}\frac{A_V}{d_{I_V}}\frac{A_H}{d_{S_H}}\beta_{VH}\left(1+\varphi\frac{(1-\alpha)}{\alpha}\frac{m_H}{m_{HV}}\right)\right)}{\left(m_H C_3 + \alpha\beta_{HV}\frac{A_V}{d_{I_V}}C_3C_4\right)} \\ &+ \frac{\left(m_H(a-d_{S_V})\right)}{\left(m_H C_3 + \alpha\beta_{HV}\frac{A_V}{d_{I_V}}C_3C_4\right)} < \\ &\frac{\left(\alpha\frac{m_H m_{HV}(d_{S_V}-a)}{m_{HV}\alpha + m_H\varphi(1-\alpha)}\left(1+\varphi\frac{(1-\alpha)}{\alpha}\frac{m_H}{m_{HV}}\right)\right)}{\left(m_H C_3 + \alpha\beta_{HV}\frac{A_V}{d_{I_V}}C_3C_4\right)} \\ &+ \frac{\left(m_H(a-d_{S_V})\right)}{\left(m_H C_3 + \alpha\beta_{HV}\frac{A_V}{d_{I_V}}C_3C_4\right)} = 0 \end{aligned}$$

Then, I_H^{e2} is not an admissible solution and, consequently, the same holds for the remaining components of P^{e2}. Then, it is proved the

Proposition 1: Under the conditions (47) for which the epidemic free equilibrium is locally asymptotically stable, the endemic equilibrium does not exists.

3.3 The Reproduction Numbers

The basic reproduction number R_0 is a useful parameter used in epidemiology to shortly give indication about the spread or the reduction of an epidemic. In a nutshell, it estimates the number of susceptible individuals that the first infected person of the population can infect. Then, it is a non-negative number so that if $R_0 > 1$ the number of infected individuals increases, with a great spread as R_0 increases, while the epidemics tends to vanish if $R_0 < 1$.

The evaluation of the basic reproduction number is here obtained making use of the computation of the next generation matrix on the basis of the model here introduced, according to the approach in (Van Den Driessche, 2017).

The procedure begins with the consideration of the restricted dynamics responsible of the first contagious and transmission. In this case, it is

$$\dot{I}_H = \alpha\beta_{HV}S_H I_V - (\gamma_{I_H} + d_{I_H})I_H \tag{49}$$

$$\dot{I}_{HF} = (1-\alpha)\beta_{HV}S_H I_V - (\gamma_{I_{HF}} + d_{I_{HF}})I_{HF} \tag{50}$$

$$\dot{I}_V = \beta_{VH}S_V I_H + \varphi\beta_{VH}S_V I_{HF} - d_{I_V} I_V \tag{51}$$

that must be rewritten as

$$\begin{pmatrix} \dot{I}_H \\ \dot{I}_{HF} \\ \dot{I}_V \end{pmatrix} = \mathcal{F} - \mathcal{V} \tag{52}$$

separating the first infection terms

$$\mathcal{F} = \begin{pmatrix} \alpha\beta_{HV} S_H I_V \\ (1-\alpha)\beta_{HV} S_H I_V \\ \beta_{VH} S_V I_H + \varphi\beta_{VH} S_V I_{HF} \end{pmatrix} \quad (53)$$

from the first transmission ones

$$-\mathcal{V} = \begin{pmatrix} -m_H I_H \\ -m_{HV} I_{HF} \\ -d_{I_V} I_V \end{pmatrix} \quad (54)$$

A local linearization of both terms in a neighbourhood of the epidemic free condition allows to get the two matrices

$$F = \left.\frac{\partial \mathcal{F}}{\partial I_H, I_{HF}, I_V}\right|_{P^{e1}} = \begin{pmatrix} 0 & 0 & \alpha\beta_{HV} S_H^{e1} \\ 0 & 0 & (1-\alpha)\beta_{HV} S_H^{e1} \\ \beta_{VH} S_V^{e1} & \varphi\beta_{VH} S_V^{e1} & 0 \end{pmatrix} \quad (55)$$

$$V = \left.\frac{\partial \mathcal{V}}{\partial I_H, I_{HF}, I_V}\right|_{P^{e1}} = \begin{pmatrix} m_H & 0 & 0 \\ 0 & m_{HV} & 0 \\ 0 & 0 & d_{I_V} \end{pmatrix} \quad (56)$$

The basic reproduction number is given by the spectral radius of matrix FV^{-1}. Since V is diagonal, its inverse is straightforwardly given and then

$$\sigma\left(FV^{-1}\right) = \sigma \begin{pmatrix} 0 & 0 & \frac{\alpha\beta_{HV} S_H^{e1}}{d_{I_V}} \\ 0 & 0 & \frac{(1-\alpha)\beta_{HV} S_H^{e1}}{d_{I_V}} \\ \frac{\beta_{VH} S_V^{e1}}{m_H} & \frac{\varphi\beta_{VH} S_V^{e1}}{m_{HV}} & 0 \end{pmatrix} \quad (57)$$

Its eigenvalues can be computed as the roots of the characteristic polynomial

$$p(\lambda) = \lambda\left(\lambda^2 - \frac{\beta_{HV}\beta_{VH} S_H^{e1} S_V^{e1}}{d_{I_V}}\left(\frac{\alpha}{m_H} + \frac{\varphi(1-\alpha)}{m_{HV}}\right)\right) \quad (58)$$

and the highest of them is clearly

$$\lambda_{max} = \sqrt{\frac{\beta_{HV}\beta_{VH} S_H^{e1} S_V^{e1}}{d_{I_V}}\left(\frac{\alpha}{m_H} + \frac{\varphi(1-\alpha)}{m_{HV}}\right)} \quad (59)$$

yielding

$$R_0 = \lambda_{max} \quad (60)$$

Recalling the condition on local stability of the epidemic free equilibrium and the meaning of R_0, as usually happens, also for the present model the following result holds:

Proposition 2: The basic reproduction number is lower than 1 if and only if the epidemic free equilibrium point is locally asymptotically stable.

Proof: $R_0 < 1$ iff $R_0^2 < 1$; from (60), it corresponds to

$$\frac{\beta_{HV}\beta_{VH} S_H^{e1} S_V^{e1}}{d_{I_V}}\left(\frac{\alpha}{m_H} + \frac{\varphi(1-\alpha)}{m_{HV}}\right) < 1 \quad (61)$$

that, after some manipulations, assume the expression of condition (47).

In addition to R_0, which gives an indicator of the dangerousness of an epidemic, a second indicator, derived from the basic reproduction number is adopted: it is the current reproduction number and denote the same as R_0 but during the epidemic evolution. It is usually denoted by R_t to put in evidence the dependency on the current time t. Its expression comes from (60) and assumes the form

$$R_t = \sqrt{\frac{\beta_{HV}\beta_{VH} S_H(t) S_V(t)}{d_{I_V}}\left(\frac{\alpha}{m_H} + \frac{\varphi(1-\alpha)}{m_{HV}}\right)} \quad (62)$$

It is interesting to prove that, also in the present case, the current reproduction number in endemic condition is equal to 1. In fact, it corresponds to

$$R_{end} = \sqrt{\frac{\beta_{HV}\beta_{VH} S_H^{e2} S_V^{e2}}{d_{I_V}}\left(\frac{\alpha}{m_H} + \frac{\varphi(1-\alpha)}{m_{HV}}\right)} \quad (63)$$

From (37), one has that

$$S_H^{e2} S_V^{e2} = \left(\frac{A_H}{d_{S_H}} - C_4 I_H^{e2}\right)\frac{A_V}{C_3 I_H^{e2} + (d_{S_V} - \alpha)} \quad (64)$$

Substituting the expression of I^{e2} in (35), after some manipulations, one obtains the expression

$$S_H^{e2} S_V^{e2} = \frac{m_H}{\alpha\beta_{HV}\frac{1}{d_{I_V}} C_3} \quad (65)$$

which, using the explicit form of C_3, can be rewritten as

$$S_H^{e2} S_V^{e2} = \frac{m_H}{\alpha\beta_{HV}\frac{1}{d_{I_V}}\left(\left(1 + \varphi\frac{(1-\alpha)}{\alpha}\frac{m_H}{m_{HV}}\right)\beta_{VH}\right)} = \frac{m_H}{\beta_{HV}\beta_{VH}\frac{m_H}{d_{I_V}}\left(\frac{\alpha}{m_H} + \frac{\varphi(1-\alpha)}{m_{HV}}\right)}$$

This explicit expression, once reported in (63), gives

$$R_{end} = \sqrt{\frac{\frac{\beta_{HV}\beta_{VH} m_H}{\beta_{HV}\beta_{VH}\frac{m_H}{d_{I_V}}\left(\frac{\alpha}{m_H} + \frac{\varphi(1-\alpha)}{m_{HV}}\right)}}{d_{I_V}}\left(\frac{\alpha}{m_H} + \frac{\varphi(1-\alpha)}{m_{HV}}\right)} \quad (66)$$

and, after some easy simplifications, the claimed result

$$R_{end} = 1 \quad (67)$$

is obtained.

4 NUMERICAL RESULTS

In this section, the proposed model is validated considering as reference case the impact of dengue in Brazil between January 1 2014 and January 1 2024, (WHO, 2024b). The choice has been driven by the fact that it is a case in which the epidemics produces a not negligible number of infections and, at the same time, a good surveillance policy is adopted, monitoring the number of severe cases. The time history of such a quantity is reported in Fig. 2; the original data are plotted along with a version smoothed by means of a Gaussian-weighted average of window 3.

Note a sort of periodicity, especially in the second part of the time interval: a seasonality of about 12 months, with a peak of the severe cases occurring every year in April corresponding, more or less, to the last month of the more humid and warmer period.

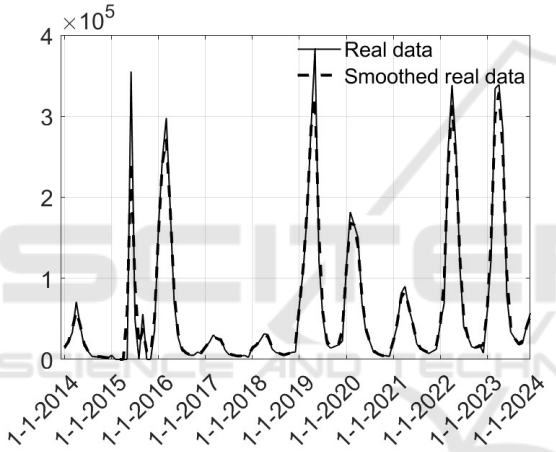

Figure 2: Real cases of dengue epidemics in Brasil; continuous line: original real data, dashed line: smoothed real data by a gaussian filter of window 3.

Some of the model parameters can be deduced from dengue characteristics and herein assumed as averaged quantities. What is known about dengue is that, as said, in most cases, with mild symptoms, a patient is recovered after about 2 weeks, whereas if symptoms occur, usually beginning 4–10 days after the infection, they last for a week. Most infected people can transmit the virus for about 1 week, whereas the presence of viral particles in the blood (viremia) can last up to 12 days. The mortality rate due to severe cases is about 5%, but if not treated it can reach 20%.

Actually, dengue disease can be caused by one of the 4 viruses, Den-1, Den-2, Den-3, Den-4, allowing to get immunization only for the specific family that has infected the patient; here, in order to maintain the model as simple as possible, this distinction is not implemented, favouring an average human population behaviour with respect to infection and introducing the possibility to be reinfected.

As far as the parameters regarding the human population, the values in Table 1 are taken.

Table 1: Values of the parameters of human population used in the numerical simulations.

d_{S_H}	10^{-6}	from statistic data
d_{I_H}	10^{-6}	from statistic data
d_{R_H}	10^{-6}	from statistic data
$d_{I_{HF}}$	$5 \cdot 10^{-6}$	illness statistics
γ_{I_H}	1	one month for recovery for asymptomatic individuals
γ_{HF}	$1/3$	three months for recovery for symptomatic individuals
r	$1/2$	two months of immunity before possible reinfection
A_H	50	population characteristics (see S_H^{el})
α	0.99	only 1% of infected has severe consequences

As far as the parameters regarding the vector population, the values in Table 2 are chosen.

Table 2: Values of the parameters of vector insect population used in the numerical simulations.

d_{S_V}	$1/3$	3 months of life for uninfected insects
d_{I_V}	$1/3$	3 months of life for infected insects
a	$1/4$	4 months as reproduction time
A_V	10^6	population characteristics (see S_V^{el})
φ	0.01	99% of severely infected patients assumed recovered and protected

The two parameters β_{HV} and β_{VH} are, respectively, the transmission rates between healthy humans S_H and the infected vectors I_V and between the healthy vectors S_V and the infected humans, I_H or I_{HF}. They represent the average rate at which an infected individual (mosquito / human) can infect a susceptible one (human / mosquito); they depend mainly on the specificity of the epidemic disease, that is on the probability of infection on contact, on the number of contacts of an infected patients, on the infectious capability of the insects, and on the robustness of the individuals with respect to the contact with the virus.

As already stated in the Introduction, the dengue epidemic is a disease whose seasonality spread is strongly dependent on the reproduction cycle of the vectors; this justifies the periodicity of about 12

months that can be noted in dengue spread in the case study analysed, Fig. 2.

The insect population dynamics here considered is a basic description that do not explicitly include any season dependency and then the periodicity in their life cycle is not present. A more sophisticated model could considers these factors, but in this preliminary study the time changes in the insect effects on the disease are modeled as time dependent values of β_{HV} and β_{VH}. This allows to describe the different stages of dengue spread that increases during the warmer and more humid months, followed by a fast decrease in autumn and winter. Then, they are not prefixed, but dependent on the epidemic evolution. For this reason, they are determined on the basis of the available data, by minimizing the error between the real data I_R and the corresponding output of the model I_{HF}; this quantity is described by means of two terms, the sum of the square of the errors, month by month, and the maximum of the difference of the errors:

$$J(\beta_{HV},\beta_{VH}) = \int_{t_i}^{t_f} w(I_R - I_{HF})^2 + max(I_R - I_{HF}) \quad (68)$$

being w a weight chosen equal to 10^{-3}, used to normalize the two elements of the cost index.

The minimization is obtained in the time interval corresponding to $[t_i \quad t_f]$ to be chosen inside the period in which the real data are available. Two main intervals of 6 months are considered every 12 months: November- April and May-October, corresponding for the Southern Hemisphere to spring-summer and autumn-winter, respectively; every 6 months, a unique value of each of the transmission rates β_{HV} and β_{VH} is estimated.

The results are obtained by applying the optimization algorithm of interior-point, that approximates the Hessian using finite differences.

To avoid data inconsistency, in the following the interval October 1 2020 December 1 2023 is considered.

Results are reported in Table 3; in Figs. 3 and 4 the behaviors of the transmission rates β_{HV} and β_{VH} are shown respectively. Note the high values corresponding to the semester of spring-summer in Brasil and the lower ones in the semester corresponding to autumn and winter.

To show the effectiveness of the identification procedure and of the proposed modelling, the reconstructed state I_{HF}, obtained by using the identified values of the transmission rates, is plotted in Fig.5 with the true evolution I_R referring to the period October 1, 2020 and December 1, 2023: a good correspondence of the values and the general seasonality trend can be observed.

As expected, in the period in which the spread increases the identified transmission rates have the highest values; as mentioned, the expedient adopted allows to highlight the seasonality of the epidemic due to dengue as if the latter depended on the greater or lower contagiousness of mosquitoes and not on their normal reproductive cycle.

The availability of a validated model allows to perform some analysis of the disease characteristics. For example, it is possible to take into account one of the most common parameter, which provides indications on the spread of the infection, the reproduction number, as discussed in Subsection 3.3; in particular,

Table 3: Values of transmission rates β_{HV} and β_{VH}.

Semester	β_{HV} (10^{-6})	β_{VH} (10^{-6})
10/01/20 – 3/1/21	0.211	0.162
04/01/21 – 9/1/21	0.027	0.010
10/01/21 – 3/1/22	0.275	0.195
04/01/22 – 9/1/22	0.010	0.010
10/01/22 – 3/1/23	0.274	0.215
04/01/23 – 9/1/23	0.010	0.010

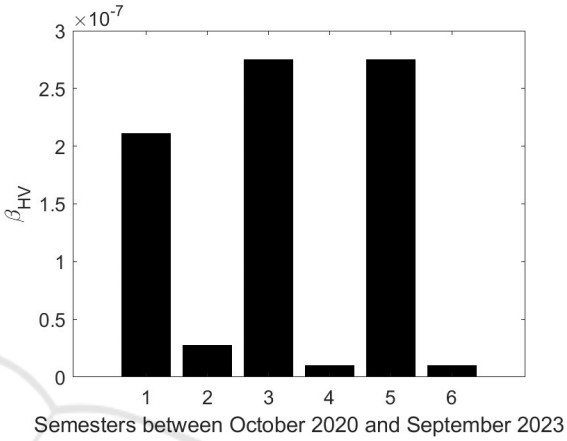

Figure 3: Transmission rate β_{HV} in the 6 semesters starting on October 2020.

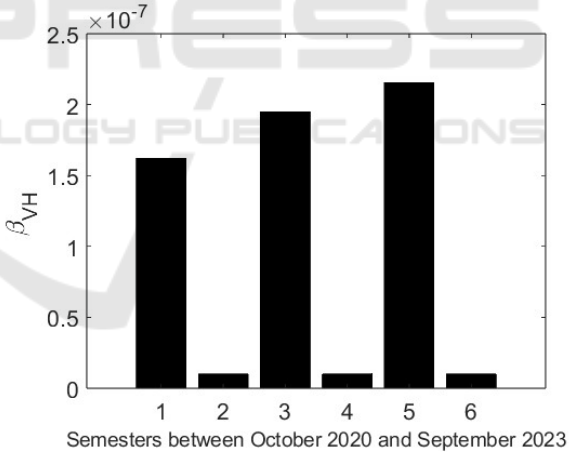

Figure 4: Transmission rate β_{VH} in the 6 semesters starting on October 2020.

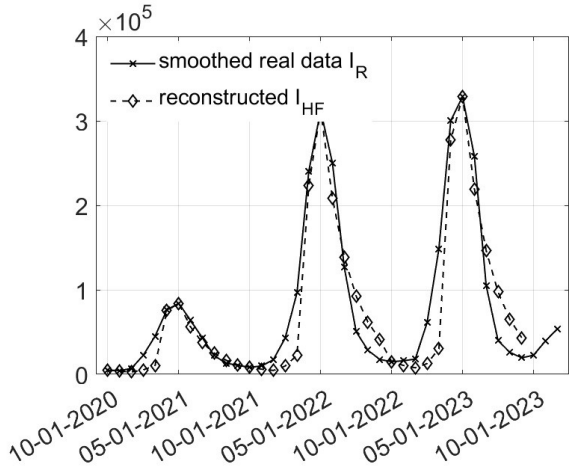

Figure 5: Example of the effectiveness of the reconstruction of the number of severe infected patients by using the identified values of the transmission rates in the period October 2020 - December 2023.

during the epidemic spread, the most meaningful is R_t, whose expression has been computed in Subsection 3.3 and reported in (62). It is now possible to evaluate it by using the monthly values of susceptible individuals and vectors. This implies that a value of R_t can be calculated for each month; the results are reported in Fig. 6. In the months in which the dengue spread increases, the corresponding R_t has a high value, that rapidly decreases in the successive months. The rapid decrease of this indicator depends mainly on the changed environmental conditions for the vectors, ascribed in the present modelling, to the transmission rates.

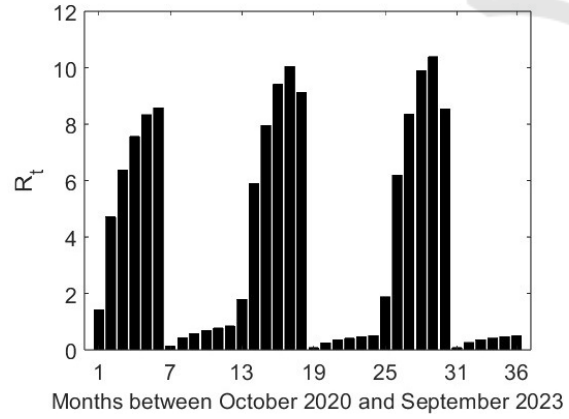

Figure 6: Reproduction number R_t evaluated monthly between October 2020 and September 2023.

It is interesting to study the influence on the basic reproduction number of some parameters, also in view of the application of possible prevention and containment measures. In particular, the most relevant parameters will be varied, keeping the others constant and equal to the values already introduced.

In Fig. 7, note that the basic reproduction number R_0 increases with α, starting, with the chosen values of the other parameters, at $R_0 = 1.3596 > 1$. This implies that a population with a reduced number of patients with severe symptoms cannot by itself decrease the spread: the less are the asymptomatic individuals, the more slow is the epidemic spread, but the infection increases anyway.

Similar consideration can be applied studying the influence of the growth parameter a on R_0; in Fig.8 it can be noted that, as reasonable, R_0 increases with parameter a; also in this case the reduction of the only reproductive capability of mosquitoes is not sufficient to reduce the basic reproduction number.

The dependence of R_0 to γ_{HF} is reported in Fig. 9, where a quite irrelevant contribution of the patients recover parameter to the variation of the epidemic spread is evidenced.

Different considerations can be referred to the death rates of mosquitoes, $d_{SV} = d_{IV}$, Fig. 10. In this case the influence is evident: by increasing this value (for example by suitable disinfestation campaign on the eggs or larval individuals) it is possible to reduce the R_0 up to values lower than 1. In this simulation, keeping the same values for all the other parameters, but increasing $d_{SV} = d_{SV} > 1.46$, it is possible to obtain $R_0 < 1$.

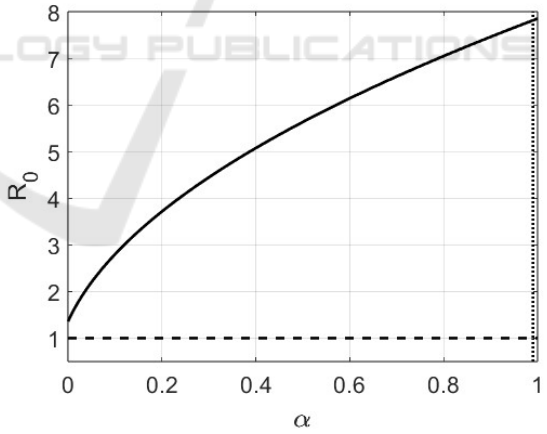

Figure 7: Variation of the basic reproduction number R_0 with respect to α.

5 CONCLUSIONS

Climate changes and increased movements of people and goods are making arboviruses a new epidemic emergency all over the world. Generally, human population does not suffer severe symptoms, but a significant percentage of patients can even die for the

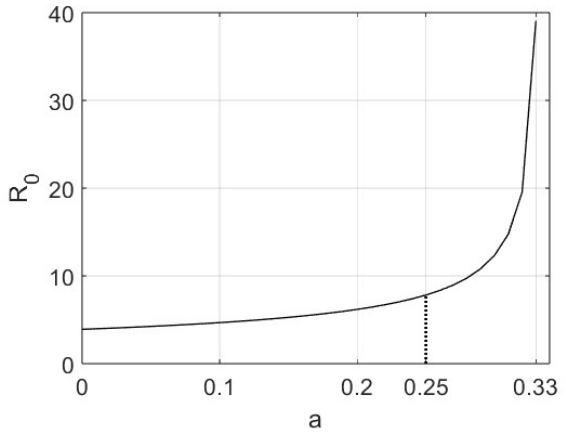

Figure 8: Variation of the basic reproduction number R_0 with respect to a.

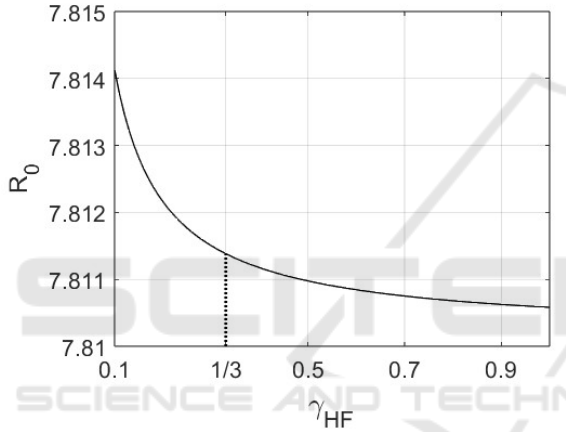

Figure 9: Variation of the basic reproduction number R_0 with respect to γ_{HF}.

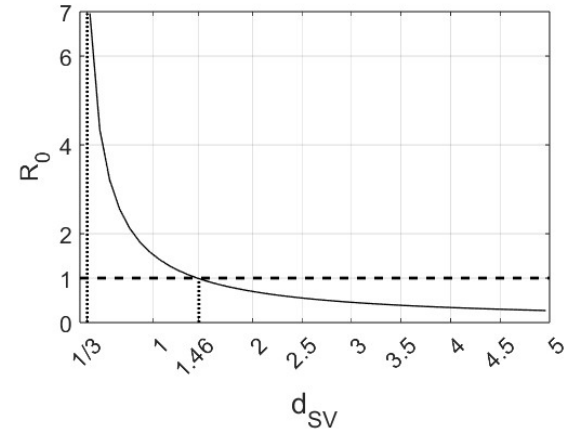

Figure 10: Variation of the basic reproduction number R_0 with respect to d_{SV}.

consequences of such infections, also for the absence of specific medication. The arboviruses have an intrinsic seasonality, due to the environmental requirements for mosquitoes' reproduction and survival. In this paper, making use of compartmental modeling framework, it is proposed and analysed a new model in which human and mosquitoes populations interact, infecting each other; the conditions for the existence of a disease free equilibrium point and the endemic one are determined, along with the basic and current reproduction numbers.

The model is validated by using recent data of dengue disease from Brazil, showing the seasonality aspect of this epidemic and discussing the influence of the model parameters on the disease evolution, also to model future lines of intervention for effective prevention and containment measures.

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