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Comparison between Chikungunya and Dengue viruses transmission based on a mathematical model

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Chikungunya and Dengue viruses are transmitted by mosquitoes of genus *Aedes*. Based on a mathematical model dealing with arboviruses transmission that encompasses human and mosquito populations, the risks of dengue and chikungunya infections are compared. By the fact that chikungunyia virus attains high viral load earlier than dengue virus in both humans and mosquitoes, the potential risk of chikungunya could be higher than the dengue infection. The risk of arboviruses infections is assessed by the reproduction number R, which is obtained by the next generation matrix method and Routh–Hurwitz criteria.

Keywords: Reproduction number; partial reproduction numbers; stability of equilibrium points; next generation matrix; Routh–Hurwitz criteria.

Mathematics Subject Classification 2010: 92B05, 97M10, 62P10

1. Introduction

Dengue, a *Flavivirus*, and Chikungunya, an *Alphavirus*, are transmitted by arthropod of the genus *Aedes*, and are prevalent in different parts of the world. Especially dengue constitutes one of the major public health problems in many tropical and subtropical regions of the world where *Aedes aegypti* (*A. aegypti*) and other appropriate mosquito vectors are present [6]. Chikungunya is a re-emerging virus in Asia and caused outbreaks in Italy [11], Indian Ocean Islands [9] and Caribbean regions [8].

Dengue occurs in an urban transmission cycle encompassing mosquitoes and humans, and due to reintroduction of *A. aegypti* into urban environments, dengue incidence has risen dramatically. There are four major serotypes of dengue and infection with one does not confer immunity to another. *A. albopictus* can be an

important secondary vector. Both mosquito species are diurnal, biting mostly in the morning and evening rather than at night. Dengue infection exhibits relatively mild to severe flu-like symptoms, although in rare cases, hemorrhagic fever can result. Chikungunya is an arbovirus first identified in 1953, which is transmitted by *A. aegypti* and *A. albopictus*. Chikungunya has a low death rate, but often causes disease with symptoms similar to dengue fever accented by severe arthritis-type pain. *A. albopictus* has been considered to be a less competent vector for chikungunya, but it is starting to play a more prominent role in part due to the virus mutation.

Chikungunya virus transmission is compared with relatively well-understood Dengue transmission, by comparing the natural history of infection. Rudolph *et al.* [8] compared the onset of symptoms among individuals infected with Chikungunya and Dengue viruses. They observed that symptoms of both diseases initiate at quasi same time, but almost all Chikungunya infected persons will manifest the symptoms during next one day, while among Dengue infected persons, the symptoms appear distributed in a prolonged period of time [8]. A mathematical model is proposed to compare the potential risk of both infections taking into account this characteristic. The risk is evaluated by computing the reproduction number denoted by R, which is obtained by the next generation matrix method [10] and applying Routh–Hurwitz criteria [5].

The paper is structured as follows: In Sec. 2, model for arboviruses transmission is presented, and the equilibrium points and the stability of disease-free equilibrium (DFE) point are presented in Sec. 3. Section 4 presents the comparison between Chikungunya and Dengue infections, and conclusion is given in Sec. 5.

2. Model for Arboviruses Transmission

Arboviruses circulate due to the interaction between human and mosquito populations in urban areas. Abiotic conditions influence strongly the size of mosquito populations [15], which are not considered here. With respect to dengue, infection with a unique serotype is being considered in the modeling.

2.1. *Variables and parameters of mosquito population*

The life cycle of *A. aegypti* encompasses an aquatic phase (eggs, larva and pupa) followed by winged (adult) form [17]. The number of eggs, which do not constitute a state variable (see [12] for a model including this compartment), is determined by the oviposition rate $\varphi(M) = \phi_m M$, where ϕ_m is the per capita oviposition rate and M, the number of female mosquitoes at time t. Defining L as the number of larvae (female) at time t, the effective larvae production rate is given by $qf[1 - L/(DC)]\phi_m M$, where q and f are the fractions of eggs that are hatching to larva and that will originate female mosquitoes, respectively, and DC is the total (carrying) capacity of the breeding sites. The constant parameter D represents the magnitude

Table 1. Summary of mosquito population parameters and respective mean values. The values of entomological parameters correspond to 26°C. The values for mosquitoes controlling parameters are arbitrary and can vary (*).

Symbol	Meaning	Unit	Value
f	fraction of eggs originating female mosquitoes	—	0.5
q	fraction of eggs hatching to larva stage	—	0.5
D	magnitude of breeding sites	—	10^6
C	demographic depending breeding sites	—	1
ϕ_m	intrinsic oviposition rate per female mosquito	day^{-1}	7.2980
σ_l	per capita transition rate from larva to pupa	day^{-1}	0.1699
σ_p	per capita transition rate from pupa to mosquito	day^{-1}	0.4705
μ_l	per capita mortality rate of larva	day^{-1}	0.0862
μ_p	per capita mortality rate of pupa	day^{-1}	0.0660
μ_m	per capita mortality rate of mosquito	day^{-1}	0.0319
ρ_l	mortality rate induced by larvicide	day^{-1}	0.01*
ρ_m	mortality rate induced by insecticide	day^{-1}	0.01*
k_c	mechanical reduction in carrying capacity	—	0.1*

of the breeding sites, while C carries on the abiotic conditions and demography of humans which can vary with time. The number of larvae decreases according to change of larvae to pupae and death, described, respectively, by the changing σ_l and the mortality μ_l rates. The number of pupae in time t, denoted by P, increases with change of larvae to pupae (σ_l) and decreases according to transformation of pupae to adult mosquitoes and death, described, respectively, by the emerging σ_p and the mortality μ_p rates. Finally, the number of female mosquitoes increases according to the emerging of pupae (σ_p) and decreases according to the mortality rate μ_f.

In order to control the mosquito population, chemical and mechanical controls are considered. Larvicide and insecticide induce additional mortality rates denoted by ρ_l and ρ_m, respectively. Mechanical control reduces the carrying capacity in a fraction k_c, with $0 \leq k_c < 1$. As we will show in analysis of the model below, the controlling parameters ρ_l, ρ_m and k_c are handled in order to obtain thresholds of eradication of mosquito population.

The summary of parameters regarded to the mosquito population and respective values are given in Table 1. The values of entomological parameters correspond to 26°C [17]. The controlling parameters ρ_l, ρ_m and k_c are unknown, and arbitrary values are allowed.

2.2. *Hypotheses of arboviruses transmission*

With respect to arboviruses transmission, after an intrinsic incubation period, the onset of symptoms in humans depends on the arboviruses. Based on the cumulative distribution of time of onset of symptoms, the infectious classes are classified as stage 1 or 2. Let the symptoms be dependent on the viral load. Based on Rudolph *et al.* [8], infectious stage 1 corresponds to the period of time elapsed

from the beginning of the onset of symptoms until reaching an asymptote, and stage 2 comprises the period of time since the reaching of asymptote until the recovering from the disease (see Sec. 4 for details). In other words, infectious stage 1 can be understood as being on average less transmissible than stage 2. Hence, the human population is divided into five compartments according to the natural history of the disease: S, E, I_1, I_2 and Z, which are the numbers at time t of, respectively, susceptible, exposed, infectious at stage 1, infectious at stage 2 and recovered individuals, with $S+E+I_1+I_2+Z = N$, where N is the size of the human population.

Among mosquitoes, after an extrinsic incubation period, female mosquitoes become infectious, but with low viral load. After a period of time, these mosquitoes harbor more viral load, becoming more transmissible. Then, the infectious mosquitoes are classified as infectious at stage 1 (less infectious) and more infectious at stage 2 [3]. Hence, the female mosquito population is divided into four compartments: M_s, M_e, M_1 and M_2, which are the numbers of mosquitoes at time t of, respectively, susceptible, exposed, infectious at stage 1 and infectious at stage 2. The size of mosquito population is given by $M = M_s + M_e + M_1 + M_2$.

Arboviruses transmission is sustained by the flows among human and mosquito compartments according to the epidemics cycle presented above. Susceptible humans are infected during the blood meal by infectious mosquitoes, with a force of infection (or per capita incidence) being denoted by B_h. The exposed persons are, then, transferred to a first stage infectious class by rate γ_h, where $1/\gamma_h$ is the intrinsic incubation period. These infectious persons progress to second stage of infectious class at rate σ_h^1, from which class they progress to recovered (immune) class at rate σ_h. Neither loss of immunity nor induced mortality due to the disease (a unique serotype of dengue) are considered. With respect to the vector, the susceptible mosquitoes are infected at a force of infection B_m. These exposed mosquitoes are transferred to first stage of infectious class at a rate γ_m, where $1/\gamma_m$ is the extrinsic incubation period. These mosquitoes progress to second stage of infectious class at rate σ_m, and remain infective until death.

The forces of infection B_h and B_m depend on the frequency of bites on humans by mosquitoes and the number of infective populations [15]. The rate at which one mosquito bites one human is directly proportional to the average biting rate of mosquitoes (intrinsic behavior of mosquitoes), and inversely proportional to the number of humans (one specific person having the chance of being bitten by one mosquito is reduced in a large population). Assuming that infectious classes at stage 2 are more infective and denoting the maximum capacities as β_h and β_m (the dimensionless transmission coefficients), for classes at stage 1, these capacities are then decreased by factors k_h and k_m, with $0 \leq (k_h, k_m) \leq 1$. Hence, B_h and B_m are written as $B_h = (k_h M_1 + M_2)\beta_h \phi_m / N$ and $B_m = (k_m I_1 + I_2)\beta_m \phi_m / N$. Note that β_h and β_m are the per capita transmission coefficients, but, when the population

Table 2. Summary of arboviruses transmission coefficients, incubation rates, and human population parameters. The assigned values are common for arboviruses.

Symbol	Meaning	Unit	Value
β_m	transmission coefficient to female mosquitoes	—	0.01
β_h	transmission coefficient to humans	—	0.01
k_m	decreased infectivity in mosquitoes	—	0.5
k_h	decreased infectivity in humans	—	0.5
γ_m	per capita extrinsic incubation rate	day^{-1}	0.167
γ_h	per capita intrinsic incubation rate	day^{-1}	0.357
ϕ_h	per capita natality rate of human	day^{-1}	7.310×10^{-5}
μ_h	per capita mortality rate of human	day^{-1}	3.805×10^{-5}
N_0	human population size	—	10^6

sizes N and M do not vary with time, the total transmission coefficients are $\hat{\beta}_h$ and $\hat{\beta}_m$, where $\hat{\beta}_h = \beta_h M$ and $\hat{\beta}_m = \beta_m N$.

In the modeling, the caught up of infectious humans by public health agents is considered. These infectious individuals are then considered "removed" from the cycle of transmission, and treated if necessary. (This type of control is not perfect because the infectious individuals in general do not obey all orientations provided by physicians in order to avoid being bitten by mosquitoes.) Let the infectious stages 1 and 2 be under caught up rates ρ_h^1 and ρ_h, respectively. In the analysis of the model, the controlling parameters ρ_h^1 and ρ_h will be considered in order to obtain thresholds of eradication of dengue propagation.

The summary of parameters regarded to the arboviruses transmission and respective values are given in Tables 2 and 3. In Table 2, common parameters to dengue and chikungunya are presented with arbitrary values for transmission coefficients β_h and β_m. The parameters regarding to the vital dynamics of human population correspond to Campinas City, Brazil [15]. In Table 3, the per capita infectious rate of humans and mosquitoes at stages 1 and 2, plus the caught up rate of infectious humans at stages 1 and 2 are presented for dengue and chikungunya (see below for details regarding to values associated to these parameters). The superscripts d and c stand for dengue and chikungunya.

2.3. *Dynamics of arboviruses transmission*

Let the size of human population at time t, denoted by N, obeys the Malthus law. Hence, it varies according to the difference between natality and mortality rates denoted by ϕ_h and μ_h, respectively, that is,

$$\frac{d}{dt}N = (\phi_h - \mu_h)N. \tag{2.1}$$

Taking into account this equation, the equations for the fractions of humans result, for instance for the fraction of susceptibles $s = S/N$, in

$$\frac{d}{dt}s = \frac{d}{dt}\frac{S}{N} = \frac{1}{N}\frac{d}{dt}S - (\phi_h - \mu_h)s$$

Table 3. Summary of human population parameters for dengue (superscript d) and chikungunya (superscript c). The assigned values for infectious human caught up parameters are arbitrary and can vary (*).

Symbol	Meaning	Unit	Value
σ_m^d	per capita infectious rate of mosquitoes at stage 1	day^{-1}	0.167
σ_h^{1d}	per capita infectious rate of humans at stage 1	day^{-1}	0.139
σ_h^d	per capita infectious rate of humans at stage 2	day^{-1}	∞
ρ_h^{1d}	caught up rate of infectious humans at stage 1	day^{-1}	0.1^*
ρ_h^d	caught up rate of infectious humans at stage 2	day^{-1}	0^*
σ_m^c	per capita infectious rate of mosquitoes at stage 1	day^{-1}	0.25
σ_h^{1c}	per capita infectious rate of humans at stage 1	day^{-1}	5.0
σ_h^c	per capita infectious rate of humans at stage 2	day^{-1}	0.143
ρ_h^{1c}	caught up rate of infectious humans at stage 1	day^{-1}	0^*
ρ_h^c	caught up rate of infectious humans at stage 2	day^{-1}	0.1^*

and so on for $e = E/N$ and $i = I/N$. For mosquito population, the numbers L, P, M_s, M_e, M_1 and M_2 are divided by the magnitude D, and lower cases correspond to densities. For instance, $l = L/D$ is the density of larvae with respect to magnitude D.

Based on the foregoing descriptions of the model parameters and variables, arboviruses transmission is described by two systems of differential equations. The arboviruses transmission among mosquitoes is described by

$$\begin{cases} \dfrac{d}{dt}l = qf\phi_m m\left[1 - \dfrac{l}{(1-k_c)\,C}\right] - (\sigma_l + \rho_l + \mu_l)l, \\ \dfrac{d}{dt}p = \sigma_l l - (\sigma_p + \mu_p)p, \\ \dfrac{d}{dt}m_s = \sigma_p p - [(k_m i_1 + i_2)\beta_m\phi_m + \rho_m + \mu_m]m_s, \\ \dfrac{d}{dt}m_e = (k_m i_1 + i_2)\beta_m\phi_m m_s - (\gamma_m + \rho_m + \mu_m)m_e, \\ \dfrac{d}{dt}m_1 = \gamma_m m_e - (\sigma_m + \rho_m + \mu_m)m_1, \\ \dfrac{d}{dt}m_2 = \sigma_m m_1 - (\rho_m + \mu_m)m_2, \end{cases} \tag{2.2}$$

while for humans, by

$$\begin{cases} \dfrac{d}{dt}s = \phi_h - \left[(k_h m_1 + m_2)\beta_h\phi_m\dfrac{D}{N} + \phi_h\right]s, \\ \dfrac{d}{dt}e = (k_h m_1 + m_2)\beta_h\phi_m\dfrac{D}{N}s - (\gamma_h + \phi_h)e, \\ \dfrac{d}{dt}i_1 = \gamma_h e - \left(\sigma_h^1 + \rho_h^1 + \phi_h\right)i_1, \\ \dfrac{d}{dt}i_2 = \sigma_h^1 i_1 - (\sigma_h + \rho_h + \phi_h)i_2, \end{cases} \tag{2.3}$$

where the decoupled fraction of immune humans is given by $z = 1 - s - e - i_1 - i_2$ (for simplicity, caught up individuals go to the recovered compartment).

3. Analysis of the Model

When the human population N varies with time, that is, $\phi_h \neq \mu_h$, then Eqs. (2.2) and (2.3) are non-autonomous. Hence, it is assumed that $\phi_h = \mu_h$, and steady states of the model are determined, and the stability of these points is assessed.

3.1. *Equilibrium points*

There are three equilibrium points: absence of mosquito population, trivial and non-trivial.

3.1.1. *Absence of mosquito population*

The equilibrium point P^{ab}, which corresponds to the absence of mosquito population in a community, is given by

$$P^{\text{ab}} = \big(l^* = 0, p^* = 0, m_s^* = 0, m_e^* = 0, m_1^* = 0, m_2^* = 0, s^* = 1, e^* = 0, i_1^* = 0, i_2^* = 0\big).$$

This steady state describes a community before the colonization by mosquitoes, or a community that eliminated the mosquitoes.

3.1.2. *Trivial equilibrium point*

Let human and mosquito populations in interaction be analyzed, but arboviruses are not circulating. The trivial equilibrium point P^0, or DFE, is given by

$$P^0 = \big(l^*, p^*, m_s^* = m^*, m_e^* = 0, m_1^* = 0, m_2^* = 0, s^* = 1, e^* = 0, i_1^* = 0, i_2^* = 0\big),$$

where l^*, p^* and m^* are given by

$$\begin{cases} l^* = (1 - k_c)C\left(1 - \dfrac{1}{Q}\right), \\ p^* = \dfrac{\sigma_l}{\sigma_p + \mu_p}(1 - k_c)C\left(1 - \dfrac{1}{Q}\right), \\ m^* = \dfrac{\sigma_p}{\rho_m + \mu_m}\dfrac{\sigma_l}{\sigma_p + \mu_p}(1 - k_c)\,C\left(1 - \dfrac{1}{Q}\right) \end{cases} \tag{3.1}$$

with the offspring number Q being given by

$$Q = \frac{\sigma_l}{\sigma_l + \rho_l + \mu_l} \frac{\sigma_p}{\sigma_p + \mu_p} \frac{qf\phi_m}{\rho_m + \mu_m}. \tag{3.2}$$

Note that mosquito population exists if $Q > 1$. Otherwise, mosquito population is eliminated, and there is only human population, which is described by equilibrium P^{ab}.

When control mechanisms are removed ($\rho_l = 0$ and $\rho_m = 0$), we have the basic offspring number Q_0, which is given by

$$Q_0 = \frac{\sigma_l}{\sigma_l + \mu_l} \frac{\sigma_p}{\sigma_p + \mu_p} \frac{qf\phi_m}{\mu_m}. \tag{3.3}$$

This number Q_0 is interpreted as follows. One (female) egg must hatch (with probability q) and survive successively the larva (with probability $\sigma_l/(\sigma_l+\mu_l)$) and pupa (with probability $\sigma_p/(\sigma_p + \mu_p)$) phases, and then emerges as a female adult. This female mosquito lays on average $f\phi/\mu_m$ (female) eggs during her entire lifespan. Hence, Q_0 is the average number of female mosquitoes generated by a single female mosquito.

Suppose that $Q_0 > 1$. The reproduction number Q can be rewritten as

$$Q = \frac{\sigma_l + \mu_l}{\sigma_l + \rho_l + \mu_l} \frac{\mu_m}{\rho_m + \mu_m} Q_0$$

and controlling parameters ρ_l and ρ_m decrease the basic reproduction number Q_0 (the third controlling parameter k_c decreases the number of mosquitoes). When $Q < 1$, the mosquito population goes to extinction. Hence, at $Q = 1$, we have a threshold (or critical) condition to eradicate mosquitoes. For instance, for a fixed ρ_l, the critical value for ρ_m, denoted by ρ_m^t, is

$$\rho_m^t = \mu_m \left(\frac{\sigma_l + \mu_l}{\sigma_l + \rho_l + \mu_l} Q_0 - 1 \right)$$

and mosquito population is eradicated whenever $\rho_m \geq \rho_m^t$. Similarly, the critical value ρ_l^t for ρ_l can be obtained.

3.1.3. *Non-trivial equilibrium point*

The non-trivial equilibrium point P^*, or endemic equilibrium, is given by

$$P^* = \left(l^*, p^*, m_s^*, m_e^*, m_1^*, m_2^*, s^*, e^*, i_1^*, i_2^*\right),$$

where l^*, p^* and $m^* = m_s^* + m_e^* + m_1^* + m_2^*$ are given by Eq. (3.1), and other coordinates are, as a function of i_2^* or m_2^*,

$$\begin{cases} m_s^* = \dfrac{\sigma_h^1}{\big[k_m(\sigma_h+\rho_h+\mu_h)+\sigma_h^1\big]\beta_m\phi_m i_2^* + \sigma_h^1(\rho_m+\mu_m)}(\rho_m+\mu_m)m^*, \\ m_e^* = \dfrac{1}{\gamma_m+\rho_m+\mu_m}\dfrac{\big[k_m(\sigma_h+\rho_h+\mu_h)+\sigma_h^1\big]\beta_m\phi_m i_2^*}{\big[k_m(\sigma_h+\rho_h+\mu_h)+\sigma_h^1\big]\beta_m\phi_m i_2^* + \sigma_h^1(\rho_m+\mu_m)} \\ \qquad \times(\rho_m+\mu_m)m^*, \\ m_1^* = \dfrac{1}{\sigma_m+\rho_m+\mu_m}\dfrac{\gamma_m}{\gamma_m+\rho_m+\mu_m} \\ \qquad \times \dfrac{\big[k_m(\sigma_h+\rho_h+\mu_h)+\sigma_h^1\big]\beta_m\phi_m i_2^*}{\big[k_m(\sigma_h+\rho_h+\mu_h)+\sigma_h^1\big]\beta_m\phi_m i_2^* + \sigma_h^1(\rho_m+\mu_m)}(\rho_m+\mu_m)m^*, \\ s^* = \dfrac{\sigma_m\mu_h}{\big[k_h(\rho_m+\mu_m)+\sigma_m\big]\beta_h\dfrac{D}{N}\phi_m m_2^* + \sigma_m\mu_h}, \\ e^* = \dfrac{\mu_h}{\gamma_h+\mu_h}\dfrac{[k_h(\rho_m+\mu_m)+\sigma_m]\beta_h\dfrac{D}{N}\phi_m m_2^*}{\big[k_h(\rho_m+\mu_m)+\sigma_m\big]\beta_h\dfrac{D}{N}\phi_m m_2^* + \sigma_m\mu_h}, \\ i_1^* = \dfrac{\gamma_h}{\sigma_h^1+\rho_h^1+\mu_h}\dfrac{\mu_h}{\gamma_h+\mu_h}\dfrac{[k_h(\rho_m+\mu_m)+\sigma_m]\beta_h\dfrac{D}{N}\phi_m m_2^*}{[k_h(\rho_m+\mu_m)+\sigma_m]\beta_h\dfrac{D}{N}\phi_m m_2^* + \sigma_m\mu_h}, \end{cases} \tag{3.4}$$

where m_2^* and i_2^* are given by

$$\begin{cases} m_2^* = \dfrac{\sigma_m}{\sigma_m+\rho_m+\mu_m}\dfrac{\gamma_m}{\gamma_m+\rho_m+\mu_m} \\ \qquad \times \dfrac{\big[k_m(\sigma_h+\rho_h+\mu_h)+\sigma_h^1\big]\beta_m\phi_m i_2^*}{\big[k_m(\sigma_h+\rho_h+\mu_h)+\sigma_h^1\big]\beta_m\phi_m i_2^* + \sigma_h^1(\rho_m+\mu_m)}m^*, \\ i_2^* = \dfrac{\sigma_h^1\gamma_h\mu_h(\rho_m+\mu_m)(R-1)}{\big(\sigma_h^1+\rho_h^1+\mu_h\big)(\rho_m+\mu_m)(\sigma_h+\rho_h+\mu_h)(\gamma_h+\mu_h)R} \\ \qquad + \gamma_h\mu_h\big[k_m(\sigma_h+\rho_h+\mu_h)+\sigma_h^1\big]\beta_m\phi_m \end{cases}$$

with the reproduction number R being given by

$$R = R^h R^m, \tag{3.5}$$

where the partial reproduction numbers of arboviruses in humans and mosquitoes R^h and R^m are given by

$$\begin{cases} R^h = \left(\dfrac{k_h}{\sigma_m+\rho_m+\mu_m} + \dfrac{\sigma_m}{\sigma_m+\rho_m+\mu_m}\dfrac{1}{\rho_m+\mu_m}\right)\beta_h\phi_m\dfrac{\gamma_h}{\gamma_h+\mu_h}, \\ R^m = \left(\dfrac{k_m}{\sigma_h^1+\rho_h^1+\mu_h} + \dfrac{\sigma_h^1}{\sigma_h^1+\rho_h^1+\mu_h}\dfrac{1}{\sigma_h+\rho_h+\mu_h}\right) \\ \qquad \times \beta_m\dfrac{D}{N}m^*\phi_m\dfrac{\gamma_m}{\gamma_m+\rho_m+\mu_m}. \end{cases} \tag{3.6}$$

Note that this endemic equilibrium is biologically feasible and is unique for $R > 1$ and $Q > 1$, the latter condition resulting from $m^* > 0$.

Removing the control mechanisms ($\rho_m = 0$, $\rho_l = 0$ and $k_c = 0$ for mosquito population, and $\rho_h = 0$ and $\rho_h^1 = 0$ for human population), the basic reproduction number R_0 is given by

$$R_0 = R_0^h R_0^m.$$

Biological interpretation of the basic reproduction number R_0 follows, defining

$$\begin{cases} R_{01}^h = \dfrac{k_h\beta_h\phi_m}{\sigma_m+\mu_m}\dfrac{\gamma_h}{\gamma_h+\mu_h}, \\ R_{02}^h = \dfrac{\sigma_m}{\sigma_m+\mu_m}\dfrac{\beta_h\phi_m}{\mu_m}\dfrac{\gamma_h}{\gamma_h+\mu_h}, \\ R_{01}^m = \dfrac{k_m\beta_m\dfrac{D}{N}m^*\phi_m}{\sigma_h^1+\mu_h}\dfrac{\gamma_m}{\gamma_m+\mu_m}, \\ R_{02}^m = \dfrac{\sigma_h^1}{\sigma_h^1+\mu_h}\dfrac{\beta_m\dfrac{D}{N}m^*\phi_m}{\sigma_h+\mu_h}\dfrac{\gamma_m}{\gamma_m+\mu_m}. \end{cases}$$

R_{01}^h is the average number of infectious humans (infected and become infectious after elapsing the intrinsic incubation period) produced by one infectious mosquito during the period of time it stays at first stage of infection; and R_{02}^h is the average number of infectious humans produced by one infectious mosquito during the period of time spent at second stage of infection, after surviving the first stage of infection. Hence, $R_0^h = R_{01}^h + R_{02}^h$ is the secondary infectious humans originated from one infectious mosquito during the time spent in both stages of infectious states. Similarly, $R_0^m = R_{01}^m + R_{02}^m$ is the secondary infectious mosquitoes originated from one infectious human during the time spent in both stages of infectious states. Therefore, R_0 gives the average number of secondary infectious humans (or mosquitoes) produced by one primary infectious human (or mosquito) introduced in completely susceptible populations of humans and mosquitoes.

Suppose that $R_0 > 1$. Arboviruses can be controlled by two ways. First, controlling efforts are targeted to mosquitoes, in order to diminish Q to below unity (see foregoing section). The second way is catching up and "isolating" infectious humans, which is assessed by controlling parameters ρ_h and ρ_h^1. Similarly to mosquito population control, critical caught up rates can be obtained. For instance, the critical value for ρ_h, denoted by ρ_h^t, can be obtained solving $R^h R^m = 1$, from Eq. (3.5), fixing values for all other parameters. Hence, for $\rho_h \geq \rho_h^t$, the eradication of arboviruses infection is achieved.

Summarizing, the conditions for the existence of above three equilibrium points are

(1) Absence of mosquito population P^{ab} — Always exists.
(2) Free of arboviruses infection P^0 — It exists if $Q > 1$.
(3) Endemic arboviruses infection P^* — It exists if $Q > 1$ and $R > 1$.

3.2. *Stability analysis*

The stability analysis is performed with details for the trivial equilibrium point. In this section, Eqs. (2.2) and (2.3) are reordered as

$$(m_e, m_1, m_2, e, i_1, i_2, l, p, m_s, s),$$

in order to evaluate the stability of the DFE.

The Jacobian matrix evaluated at the DFE, named $J_0 = J(P^0)$, results in

$$J_0 = \begin{bmatrix} F & 0 \\ J_2 & J_1 \end{bmatrix}.$$

The matrix F is given by $F = F_1 - V$, where the disease transmission matrix F_1 is

$$F_1 = \begin{bmatrix} 0 & 0 & 0 & 0 & k_m\beta_m\phi_m m^* & \beta_m\phi_m m^* \\ \gamma_m & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma_m & 0 & 0 & 0 & 0 \\ 0 & k_h\beta_h\dfrac{D}{N}\phi_m & \beta_h\dfrac{D}{N}\phi_m & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma_h & 0 & 0 \\ 0 & 0 & 0 & 0 & \sigma_h^1 & 0 \end{bmatrix} \tag{3.7}$$

and the diagonal transition matrix V is

$$V = \begin{bmatrix} \gamma_m + \rho_m + \mu_m & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma_m + \rho_m + \mu_m & 0 & 0 & 0 & 0 \\ 0 & 0 & \rho_m + \mu_m & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma_h + \mu_h & 0 & 0 \\ 0 & 0 & 0 & 0 & \sigma_h^1 + \rho_h^1 + \mu_h & 0 \\ 0 & 0 & 0 & 0 & 0 & \sigma_h + \rho_h + \mu_h \end{bmatrix}. \tag{3.8}$$

The matrix J_1 is

$$J_1 = \begin{bmatrix} M & 0 \\ 0 & H \end{bmatrix}$$

with the matrices M and H being given by

$$M = \begin{bmatrix} -(\sigma_l + \rho_l + \mu_l)Q & 0 & qf\phi_m \dfrac{1}{Q} \\ \sigma_l & -(\sigma_p + \mu_p) & 0 \\ 0 & \sigma_p & -(\rho_m + \mu_m) \end{bmatrix}$$

and

$$H = [-\mu_h],$$

a unitary matrix. Finally, matrix J_2 is

$$J_2 = \begin{bmatrix} qf\phi_m \dfrac{1}{Q} & qf\phi_m \dfrac{1}{Q} & qf\phi_m \dfrac{1}{Q} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -k_m\beta_m\phi_m m^* & -\beta_m\phi_m m^* \\ 0 & -k_h\beta_h \dfrac{D}{N}\phi_m & -\beta_h \dfrac{D}{N}\phi_m & 0 & 0 & 0 \end{bmatrix}.$$

Note that matrix F encompasses the disease transmission parameters, while matrices M and H, encompass the vital dynamics parameters of mosquito and human populations. The vital dynamics of human population is Malthusian with constant population, hence H is a 1×1 matrix.

The local stability of DFE is assessed by the eigenvalues of the characteristic equation $\det(J - \lambda I) = 0$, where

$$\det(J - \lambda I) \equiv \det(F - \lambda I)\det(M - \lambda I)\det(H - \lambda I).$$

The eigenvalue corresponding to vital dynamics matrix of humans H is $\lambda_1 = -\mu_h$.

The characteristic equation corresponding to vital dynamics matrix of mosquitoes M is

$$\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0,$$

where the coefficients are

$$\begin{cases} a_2 = (\sigma_l + \rho_l + \mu_l)Q + (\sigma_p + \mu_p) + (\rho_m + \mu_m), \\ a_1 = (\sigma_l + \rho_l + \mu_l)(\sigma_p + \mu_p + \mu_f)Q + (\sigma_p + \mu_p)(\rho_m + \mu_m), \\ a_0 = (\sigma_l + \rho_l + \mu_l)(\sigma_p + \mu_p)(\rho_m + \mu_m)(Q - 1) \end{cases}$$

with Q being given by Eq. (3.2). The difference $a_2a_1 - a_0$ can be evaluated, resulting in

$$a_2a_1 - a_0 = a_1[(\sigma_l + \rho_l + \mu_l)Q + (\sigma_p + \mu_p)] + \frac{qf\phi}{Q}\sigma_l\sigma_p + [(\sigma_l + \rho_l + \mu_l)Q + (\sigma_p + \mu_p)](\rho_m + \mu_m)^2 > 0.$$

Hence, the eigenvalues $\lambda_{2,3,4}$ have negative real part since all the Routh–Hurwitz criteria (for a third degree polynomial they are $a_0 > 0$, $a_2 > 0$ and $a_2a_1 > a_0$) are satisfied when $Q > 1$, which is the condition for the existence of mosquito population.

The last matrix F must be analyzed to establish the stability of DFE. Two methods are applied in order to achieve this goal.

3.2.1. *Routh–Hurwitz criteria*

The characteristic polynomial corresponding to arbovirus transmission matrix F is

$$\begin{aligned}\Lambda(\lambda) = {} & [(\gamma_m + \rho_m + \mu_m) + \lambda][(\sigma_m + \rho_m + \mu_m) + \lambda][(\rho_m + \mu_m) + \lambda] \\ & \times [(\gamma_h + \mu_h) + \lambda]\left[\left(\sigma_h^1 + \rho_h^1 + \mu_h\right) + \lambda\right][(\sigma_h + \rho_h + \mu_h) + \lambda] \\ & - \gamma_m\gamma_h\left\{k_m\beta_m\phi_m m^*[(\sigma_h + \rho_h + \mu_h) + \lambda] + \beta_m\phi_m m^*\sigma_h^1\right\} \\ & \times \left\{k_h\beta_m\frac{D}{N}\phi_m[(\rho_m + \mu_m) + \lambda] + \beta_m\frac{D}{N}\phi_m m^*\sigma_m\right\},\end{aligned}$$

which is a 6th degree polynomial, with the independent term, obtained from $\Lambda_0 = \Lambda(0) = \det(F)$, being given by

$$\Lambda_0 = \theta(1 - R),$$

where $\theta > 0$ is given by

$$\theta = (\gamma_m + \rho_m + \mu_m)(\sigma_m + \rho_m + \mu_m)(\rho_m + \mu_m)(\gamma_h + \mu_h) \times \left(\sigma_h^1 + \rho_h^1 + \mu_h\right)(\sigma_h + \rho_h + \mu_h)$$

and R is given by Eq. (3.5). All Routh–Hurwitz criteria corresponding to 6th degree polynomial that assure negative real part for all eigenvalues are extremely complicated. (For instance, seven Routh–Hurwitz's conditions corresponding to a 5th degree polynomial are presented in [5].) Hence, applying the conjecture proposed by Leite *et al.* [4], the independent term is $\Lambda_0 > 0$ when $R < 1$, and all Routh–Hurwitz criteria should be satisfied, resulting that all eigenvalues $\lambda_{5,\ldots,10}$ have negative real part.

Therefore, the trivial equilibrium P^0 is locally asymptotically stable for $Q > 1$ and $R < 1$. At $R = 1$, there is a forward bifurcation, from P^0 to P^*.

3.2.2. *Next generation matrix method*

Instead of evoking the conjecture in [4], the threshold parameter R can be obtained from the characteristic equation corresponding to the next generation matrix.

First, defining vectors f and v as

$$f = \begin{pmatrix} (k_m i_1 + i_2)\beta_m \phi_m m_s \\ \gamma_m m_e \\ \sigma_m m_1 \\ (k_h m_1 + m_2)\beta_h \phi_m \dfrac{D}{N} s \\ \\ \gamma_h e \\ \sigma_h^1 i_1 \end{pmatrix} \quad \text{and} \quad v = \begin{pmatrix} (\gamma_m + \rho_m + \mu_m)m_e \\ (\sigma_m + \rho_m + \mu_m)m_1 \\ (\rho_m + \mu_m)m_2 \\ (\gamma_h + \phi_h)e \\ \left(\sigma_h^1 + \rho_h^1 + \phi_h\right)i_1 \\ (\sigma_h + \rho_h + \phi_h)i_2 \end{pmatrix},$$

and differentiating with respect to the variables $(m_e, m_1, m_2, e, i_1, i_2)$, the matrices F_1 and V are obtained, which are given by Eqs. (3.7) and (3.8), respectively. Being V diagonal, the next generation matrix $F_1 V^{-1}$ is easily evaluated, resulting in

$$F_1 V^{-1} = \begin{bmatrix} 0 & 0 & 0 & 0 & \dfrac{k_m \beta_m \phi_m m^*}{\sigma_h^1 + \rho_h^1 + \mu_h} & \dfrac{\beta_m \phi_m m^*}{\sigma_h + \rho_h + \mu_h} \\ \dfrac{\gamma_m}{\gamma_m + \rho_m + \mu_m} & 0 & 0 & 0 & 0 & 0 \\ 0 & \dfrac{\sigma_m}{\sigma_m + \rho_m + \mu_m} & 0 & 0 & 0 & 0 \\ 0 & \dfrac{k_h \beta_h \dfrac{D}{N} \phi_m}{\sigma_m + \rho_m + \mu_m} & \dfrac{\beta_h \dfrac{D}{N} \phi_m}{\rho_m + \mu_m} & 0 & 0 & 0 \\ 0 & 0 & 0 & \dfrac{\gamma_h}{\gamma_h + \mu_h} & 0 & 0 \\ 0 & 0 & 0 & 0 & \dfrac{\sigma_h^1}{\sigma_h^1 + \rho_h^1 + \mu_h} & \sigma_h + \rho_h + \mu_h \end{bmatrix}.$$

The characteristic equation corresponding to $F_1 V^{-1}$ is

$$\lambda^6 - R_{11}\lambda^2 - (R_{12} + R_{21})\lambda - R_{22} = 0, \tag{3.9}$$

where the coefficients are given by

$$\begin{cases} R_{11} = R_1^h R_1^m, & R_{12} = R_1^h R_2^m, \\ R_{21} = R_2^h R_1^m \quad \text{and} & R_{22} = R_2^h R_2^m \end{cases}$$

with the partial terms R_i^j and R_i^j, with $i = 1{,}2$ and $j = h$, m, being given by Eq. (3.6). By applying the conjecture proposed in [13] and proved in [16], the threshold is obtained as

$$R = R_{11} + R_{12} + R_{21} + R_{22} = R^h R^m, \tag{3.10}$$

which is the reproduction number given by Eq. (3.5).

However, there is a second way of constructing vectors f and v as

$$f = \begin{pmatrix} (k_m i_1 + i_2)\beta_m \phi_m m_s \\ 0 \\ 0 \\ (k_h m_1 + m_2)\beta_h \phi_m \dfrac{D}{N} s \\ 0 \\ 0 \end{pmatrix} \quad \text{and} \quad v = \begin{pmatrix} (\gamma_m + \rho_m + \mu_m) m_e \\ -\gamma_m m_e + (\sigma_m + \rho_m + \mu_m) m_1 \\ -\sigma_m m_1 + (\rho_m + \mu_m) m_2 \\ (\gamma_h + \phi_h) e \\ -\gamma_h e + \left(\sigma_h^1 + \rho_h^1 + \phi_h\right) i_1 \\ -\sigma_h^1 i_1 + \left(\sigma_h + \rho_h + \phi_h\right) i_2 \end{pmatrix},$$

and differentiating with respect to the variables $(m_e, m_1, m_2, e, i_1, i_2)$, the matrices F_1 and V are given by

$$F_1 = \begin{bmatrix} 0 & 0 & 0 & 0 & k_m \beta_m \phi_m m^* & \beta_m \phi_m m^* \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & k_h \beta_h \dfrac{D}{N} \phi_m & \beta_h \dfrac{D}{N} \phi_m & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

and

$$V = \begin{bmatrix} \gamma_m + \rho_m + \mu_m & 0 & 0 & 0 & 0 & 0 \\ -\gamma_m & \sigma_m + \rho_m + \mu_m & 0 & 0 & 0 & 0 \\ 0 & -\sigma_m & \rho_m + \mu_m & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma_h + \mu_h & 0 & 0 \\ 0 & 0 & 0 & -\gamma_h & \sigma_h^1 + \rho_h^1 + \mu_h & 0 \\ 0 & 0 & 0 & 0 & -\sigma_h^1 & \sigma_h + \rho_h + \mu_h \end{bmatrix}.$$

Evaluating the next generation matrix $F_1 V^{-1}$, the corresponding characteristic equation is

$$\lambda^6 - R^h R^m \lambda^4 = 0, \tag{3.11}$$

and applying the conjecture proposed in [13], the threshold is $R^h R^m$, which is the same obtained in the previous construction of vectors f and v, given by Eq. (3.10). It is worth mentioning that there are other ways of constructing vector f, but the sum of the coefficients of the characteristic equation corresponding to FV^{-1} always results in the threshold $R^h R^m$. Additionally, when above two special cases of constructing vectors f and v result in the same threshold, then there is not a second threshold [14].

Some authors define the spectral radius as the basic reproduction number, which is the reproduction number letting zero to the controlling parameters (see, for instance, [2, 7, 10]). According to their definition, the spectral radius corresponding to Eq. (3.9) does not have analytical expression, but the spectral radius of

Eq. (3.11) is $\rho(F_1V^{-1}) = \sqrt{R^hR^m}$. The conjecture presented in [13] says that the spectral radius is not the basic reproduction number, but it is the geometric mean of the partial reproduction numbers.

With respect to the equilibrium point P^{ab}, the local stability is determined by the previous matrices F_1 (letting $m^* = 0$), V and H, but matrix M is given by

$$M = \begin{bmatrix} -(\sigma_l + \rho_l + \mu_l) & 0 & qf\phi_m \\ \sigma_l & -(\sigma_p + \mu_p) & 0 \\ 0 & \sigma_p & -(\rho_m + \mu_m) \end{bmatrix},$$

which has corresponding eigenvalues with negative real part if $Q < 1$.

Summarizing, the conditions for the stability of the equilibrium points are

(1) Absence of mosquito population P^{ab} — Always exists, but is stable if $Q < 1$.
(2) Free of arboviruses infection P^0 — It exists if $Q > 1$, and is stable if $Q > 1$ and $R < 1$.
(3) Endemic arboviruses infection P^* — It exists and is stable (not shown here) if $Q > 1$ and $R > 1$.

4. Comparing Dengue and Chikungunya Infections

Common values for dengue and chikungunya infections were presented in Tables 1 and 2. In Table 2, it was assumed that the intrinsic and extrinsic incubation rates were equal for both infections, or, $\gamma_h = \gamma_h^d = \gamma_h^c$ and $\gamma_m = \gamma_m^d = \gamma_m^c$.

Values assigned in Table 3 are now explained. Infectious rates of humans at stages 1 and 2 for dengue and chikungunya are obtained from Rudolph *et al.* [8], assuming that symptoms correspond to harboring high viral load. From Fig. 2 in [8], the period of time from the inoculation of virus to the recovery of infection in humans is 10 days, and the infectious rates of humans can be calculated as follows: (1) the period of time corresponding to the increasing phase of the curve is assumed to be stage 1, and (2) the asymptote (horizontal line), to stage 2. Hence, for dengue, the periods are (in days) $(\sigma_h^1)^{-1} = 7.2$ and $\sigma_h^{-1} = 0$ (in this case, the infectious humans in stage 1 go directly to the recovered class without passing through stage 2). In terms of rates (the inverse of periods), $\sigma_h^1 = 0.139$ and $\sigma_h = \infty$ (in days^{-1}). For chikungunya, the periods are (in days) $(\sigma_h^1)^{-1} = 0.2$ and $\sigma_h^{-1} = 7$, or, in terms of rates, $\sigma_h^1 = 5$ and $\sigma_h = 0.143$ (in days^{-1}) (see Table 3). For dengue (superscript d) and chikungunya (superscript c), the infectious rates are such that $\sigma_h^{1c} > \sigma_h^c > \sigma_h^{1d}$.

The controlling parameters ρ_h^{1d} and ρ_h^{1c} are the caught up rates of infectious individuals at stage 1 for dengue and chikungunya, and ρ_h^c is caught up rate of infectious individuals at stage 2 for chikungunya ($\rho_h^d = 0$, since stage 2 does not exist for dengue). Since the infectious period at stage 1 for chikungunya is 4.8 h (see Table 3), it is assumed that $\rho_h^{1c} = 0$ (infectious humans at stage 1 are hardly

Table 4. The reproduction number R, using values given in Tables 1, 2 and 3.

Reproduction number	Dengue	Chikungunya
R^h	1.567	1.617
R^m	0.375	0.755
$R = R^h R^m$	0.587	1.220

diagnosed due to the short period of time at this stage). Other controlling parameter values are arbitrary, aiming only the illustrative purpose.

With respect to mosquito population, it is assumed that infectious period related to chikungunya is higher than dengue infection. The reason behind this is the quick increase of viral load in humans, which could occur in mosquitoes. Hence, it is assumed that the infectious rate of mosquitoes at stage 1 (mosquitoes do not mount immune response, and they stay at stage 2 during lifespan) is $\sigma_m^{-1} = 6$ days for dengue, or, $\sigma_m = 0.167$ days^{-1}. For chikungunya, it is assumed that $\sigma_m^{-1} = 4$ days, or, $\sigma_m = 0.25$ days^{-1} (see Table 3). The infectious rates at stage 1 among mosquitoes are such that $\sigma_m^c > \sigma_m^d$, and the extrinsic incubation rate was assumed to be the same ($\gamma_m = \gamma_m^d = \gamma_m^c$).

Based on values given in Table 1, the estimations for Q and m^* are 24.38 and 3.07, obtained from Eqs. (3.2) and (3.1). Table 4 gives the reproduction number R, based on values given in Tables 1, 2 and 3. Note that the reproduction number for chikungunya is almost two times higher than dengue, which is due to two times higher partial reproduction number of mosquitoes R^m. Illustrative values allowed to controlling parameters resulted in the eradication of dengue ($R < 1$), but chikungunya remains endemic.

Let the difference in the reproduction number R for dengue and chikungunya transmission be assessed. To facilitate the comparison, it is assumed that the transmission coefficients β_m and β_h are equal, as well as the reduced fractions of transmission k_m and k_h. These oversimplifications, with the previous assumption that γ_m and γ_h are equal for both dengue and chikungunya, allow to determine easily the difference between the partial reproduction numbers due to humans R^h and mosquitoes R^m, given by Eq. (3.6). The reproduction number R is denoted as $R_d = R_d^m R_d^h$ for dengue, and $R_c = R_c^m R_c^h$ for chikungunya.

4.1. *Difference in R^m*

By the fact that $\sigma_h^d = \infty$ for dengue, the partial reproduction number for dengue due to mosquitoes R_d^m is given by

$$R_d^m = \left(\frac{k_m}{\sigma_h^{1d} + \rho_h^{1d} + \mu_h}\right) \beta_m \frac{D}{N} m^* \phi_m \frac{\gamma_m}{\gamma_m + \rho_m + \mu_m}.$$

For chikungunya, R_c^m is given by

$$R_c^m = \left(\frac{k_m}{\sigma_h^{1c} + \mu_h} + \frac{\sigma_h^{1c}}{\sigma_h^{1c} + \mu_h} \frac{1}{\sigma_h^c + \rho_h^c + \mu_h}\right) \beta_m \frac{D}{N} m^* \phi_m \frac{\gamma_m}{\gamma_m + \rho_m + \mu_m}.$$

The controlling parameter ρ_h^{1d} is the caught up rate of infectious individuals at stage 1 for dengue, and ρ_h^c is caught up rate of infectious individuals at stage 2 for chikungunya (remembering that $\rho_h^d = 0$ and $\rho_h^{1c} = 0$).

The difference $\Delta^m = R_c^m - R_d^m$ is given by

$$\Delta^m = \frac{\sigma_h^{1c}\left(1 - \dfrac{k_m}{\hat{k}_m}\right)}{\left(\sigma_h^{1c} + \mu_h\right)\left(\sigma_h^c + \rho_h^c + \mu_h\right)} \beta_m \frac{D}{N} m^* \phi_m \frac{\gamma_m}{\gamma_m + \rho_m + \mu_m}, \tag{4.1}$$

where the critical value for k_m, denoted by $\hat{k}_m$, is defined by

$$\hat{k}_m = \frac{\sigma_h^{1c}\left(\sigma_h^{1d} + \rho_h^{1d} + \mu_h\right)}{\left(\sigma_h^c + \rho_h^c + \mu_h\right)\left(\hat{\rho}_h^{1d} - \rho_h^{1d}\right)}, \tag{4.2}$$

with $\hat{\rho}_h^{1d}$ being the critical value for ρ_h^{1d}, defined by

$$\hat{\rho}_h^{1d} = \sigma_h^{1c} - \sigma_h^{1d}, \tag{4.3}$$

which is always positive due to $\sigma_h^{1c} > \sigma_h^{1d}$.

Let the sign of Δ^m be determined. (1) If $\rho_h^{1d} > \hat{\rho}_h^{1d}$, that is, there is a well-organized heath system that recognizes the dengue infected persons and provides efficient orientations in order to avoid dengue transmission, then $\hat{k}_m < 0$, leading to $\Delta^m > 0$, and yielding $R_c^m > R_d^m$. (2) If $\rho_h^{1d} < \hat{\rho}_h^{1d}$, then $\hat{k}_m > 0$. In this case, $k_m > \hat{k}_m$ (remember that $0 \le k_m \le 1$) is satisfied whenever

$$\rho_h^c > \hat{\rho}_h^c \equiv \hat{\sigma}_h^c \left(1 - \frac{\sigma_h^c}{\hat{\sigma}_h^c}\right), \tag{4.4}$$

where $\hat{\rho}_h^c$ is defined as the critical value for ρ_h^c, and the critical value for σ_h^c, denoted by $\hat{\sigma}_h^c$, is defined by

$$\hat{\sigma}_h^c = \frac{\left(\sigma_h^{1c} + k_m \mu_h\right)\left(\sigma_h^{1d} + \rho_h^{1d}\right) + (1 - k_m)\sigma_h^{1c}\mu_h}{k_m\left(\hat{\rho}_h^{1d} - \rho_h^{1d}\right)}. \tag{4.5}$$

Three cases arise: (2.a) If $\sigma_h^c > \hat{\sigma}_h^c$, it is true that $\hat{\rho}_h^c < 0$, then $k_m > \hat{k}_m$, leading to $\Delta^m < 0$, resulting in $R_c^m < R_d^m$; (2.b) If $\sigma_h^c < \hat{\sigma}_h^c$ and $\rho_h^c > \hat{\rho}_h^c$, it is true that $k_m > \hat{k}_m$, leading to $\Delta^m < 0$, resulting in $R_c^m < R_d^m$; and (2.c) If $\sigma_h^c < \hat{\sigma}_h^c$ and $\rho_h^c < \hat{\rho}_h^c$, it is true that $k_m < \hat{k}_m$, leading to $\Delta^m > 0$, resulting in $R_c^m > R_d^m$. Note that higher values for σ_h^c mean lower time spending at the infectious stage 2.

Summarizing, $R_c^m > R_d^m$ is true for: (1) $\rho_h^{1d} > \hat{\rho}_h^{1d}$, and (2) $\rho_h^{1d} < \hat{\rho}_h^{1d}$, $\sigma_h^c < \hat{\sigma}_h^c$ and $\rho_h^c < \hat{\rho}_h^c$. Case (1) means higher rate of caught up of dengue infectious individuals at stage 1 (there are no dengue infectious individuals at stage 2), while case (2) means lower rate of dengue caught up together with lower rates of transition and caught up of chikungunya infectious individuals at stage 2.

4.2. *Difference in R^h*

The infectious rates at stage 1 among mosquitoes are such that $\sigma_m^c > \sigma_m^d$ (see Table 3).

The partial reproduction number for dengue due to humans R_d^h is given by

$$R_d^h = \left(\frac{k_h}{\sigma_m^d + \rho_m + \mu_m} + \frac{\sigma_m^d}{\sigma_m^d + \rho_m + \mu_m}\frac{1}{\rho_m + \mu_m}\right)\beta_h\phi_m\frac{\gamma_h}{\gamma_h + \mu_h},$$

and for chikungunya, R_c^h is obtained by substituting σ_m^d by σ_m^c, that is,

$$R_c^h = \left(\frac{k_h}{\sigma_m^c + \rho_m + \mu_m} + \frac{\sigma_m^c}{\sigma_m^c + \rho_m + \mu_m}\frac{1}{\rho_m + \mu_m}\right)\beta_h\phi_m\frac{\gamma_h}{\gamma_h + \mu_h}.$$

The difference $\Delta^h = R_c^h - R_d^h$ is given by

$$\Delta^h = \frac{(1-k_h)\left(\sigma_m^c - \sigma_m^d\right)}{\left(\sigma_m^c + \rho_m + \mu_m\right)\left(\sigma_m^d + \rho_m + \mu_m\right)}\beta_h\phi_m\frac{\gamma_h}{\gamma_h + \mu_h} > 0. \tag{4.6}$$

Hence, the partial reproduction number for chikungunya due to humans R_c^h is always higher than the partial number corresponding to dengue R_d^h.

4.3. *Comparison*

All previous results were obtained assuming that both dengue and chikungunya are equally transmissible, that is, $\beta_m = \beta_m^d = \beta_m^c$ and $\beta_h = \beta_h^d = \beta_h^c$. As a simple comparison between both infections, the differences Δ^m and Δ^h were determined, given by Eqs. (4.1) and (4.6).

The difference Δ^h is always positive, because $\sigma_m^c > \sigma_m^d$, in other words, infectious mosquitoes with chikungunya virus at stage 1 enter to stage 2 faster than mosquitoes infected with dengue virus. Hence, the risk of susceptible humans being infected by infectious mosquitoes is higher for chikungunya infection ($R_c^h > R_d^h$).

However, Δ^m can change signal. The risk of susceptible mosquitoes being infected by infectious humans is higher for chikungunya infection ($R_c^m > R_d^m$) when

(1) $\rho_h^{1d} > \hat{\rho}_h^{1d}$, that is, the rate of caught up of infectious humans with dengue virus at stage 1 (there is no stage 2 for dengue) is very high,
(2) $\rho_h^{1d} < \hat{\rho}_h^{1d}$, then additional conditions $\sigma_h^c < \hat{\sigma}_h^c$ and $\rho_h^c < \hat{\rho}_h^c$ must be satisfied, which are low infectious rate of humans with chikungunya at stage 2 and low caught up rate of humans infected with chikungunya at stage 2, respectively.

Based on values given in Tables 1, 2 and 3, the critical values $\hat{k}_m$, $\hat{\rho}_h^{1d}$, $\hat{\rho}_h^c$ and $\hat{\sigma}_h^c$ are, respectively, 1.033, 4.861, 0.359 and 0.502 (in years^{-1}, except $\hat{k}_m$, dimensionless), using Eqs. (4.2), (4.3), (4.4) and (4.5), respectively, for three last critical values. To determine the signal of Δ^m, three conditions stated above are evaluated. From $\hat{\rho}_h^{1d} = 4.861 > 0.1 = \rho_h^{1d}$, this corresponds to case (2). From (2), conditions $\hat{\sigma}_h^c = 0.502 > 0.142 = \sigma_h^c$ and $\hat{\rho}_h^c = 0.359 > 0.1 = \rho_h^c$ establish that $\Delta^m > 0$, implying that $\Delta = \Delta^h\Delta^m > 0$. Note that in the absence of control

Table 5. The basic reproduction number R_0, using values given in Tables 1, 2 and 3 by letting zero to all controlling parameters.

Basic reproduction number	Dengue	Chikungunya
R_0^h	2.104	2.158
R_0^m	0.998	1.969
$R_0 = R_0^h R_0^m$	2.101	4.248

(letting zero to all controlling parameters), the critical values $\hat{k}_m$, $\hat{\rho}_h^c$ and $\hat{\sigma}_h^c$ are decreased ($\hat{\rho}_h^{1d}$ does not change) and become, respectively, 0.999, 0.143 and 0.286, but result in $\Delta > 0$. Hence, chikungunya is more transmissible than dengue.

The critical value $\hat{\rho}_h^{1d}$ is very higher (4.861 years^{-1}), showing that the case (2) must prevail. Hence, when conditions in case (2) are not satisfied, then dengue transmission has higher reproduction number than chikungunya. In other words, if the controlling parameter ρ_h^c is increased above its critical value $\hat{\rho}_h^c$, then the epidemics of chikungunya can be controlled at least at lower incidence than dengue.

Finally, the beginning of dengue and chikungunya infections can be assessed, assuming that control mechanisms are not applied. Hence, letting zero to all controlling parameters in Table 1, Q_0 and m_0^* are 33.28 and 4.53. Based on values given in Tables 1, 2 and 3, by letting zero to all controlling parameters, the basic reproduction number R_0 is given in Table 5. The basic reproduction numbers R_0 for dengue and chikungunya are almost four times the corresponding reproduction numbers R (see Table 4).

Dynamical trajectories of the non-autonomous system of Eqs. (2.2) and (2.3), and Eq. (2.1) for humans, are obtained numerically considering the initial conditions, at $t = 0$, given by

$$\Big(l(0) = l^*, p(0) = p^*, m_s(0) = m^*, m_e(0) = 0, m_1(0) = 0, m_2(0) = 0,$$

$$s(0) = 1 - \frac{1}{N_0}, e(0) = 0, i_1(0) = \frac{1}{N_0}, i_2(0) = 0, N(0) = N_0\Big),$$

where N_0 is given in Table 2, which corresponds to the introduction of one infectious human at stage 1 in a community of size 10^6 which is free of arboviruses. Simulations (not shown here) showed that dengue epidemics begin after elapsing 400 days and infectious individuals reach maximum value after 750 days since the introduction of one case. For chikungunya epidemics, the epidemics initiate after 200 days, and infectious individuals reach maximum value after 340 days. Moreover, the peak of infectious humans with chikungunya is four times that found for dengue.

5. Conclusion

Chikungunya and dengue viruses infections were studied by a mathematical model considering two stages for infectious populations of mosquitoes and humans. In order to calculate the infectious rates of humans at stages 1 and 2, the onset of symptoms was assumed to be proportional to virus load [8].

The model was analyzed determining the equilibrium points and their stability. Specially with respect to the DFE, the analytical verification of all Routh–Hurwitz criteria is unfeasible, which was the reason for applying the conjecture proposed in [4] to obtain the reproduction number R. However, the next generation matrix method, applying the conjecture proposed in [13], provided the same reproduction number R. Hence, next generation matrix method or Routh–Hurwitz criteria must be chosen aiming the easy calculation of the (basic) reproduction number.

Assuming equal transmission coefficients β_m and β_h for chikungunya and dengue infections, and considering the entomological and disease progression-related parameters given in Tables 1, 2 and 3, the analysis of the model resulted in a higher risk for chikungunya (see Tables 4 and 5) and the outbreak occurs quickly and more intense than dengue. The rate of virus replication in human and mosquito populations plays an important role: how quickly infected individuals (both humans and mosquitoes) attain the infectious class at stage 2 is determinant to the course of epidemics. Therefore, the caught up of these infectious individuals by public health agents, which is strongly dependent on the skills of physicians to diagnosing dengue and particularly chikungunya, is crucial to control epidemics.

Transmission coefficients β_m and β_h were assumed equal to chikungunya and dengue infections. Different values for these parameters must be considered due to different species of mosquitoes and viruses are being considered, which is left to further work, as well as the controlling of arboviruses infections (controlling parameter values allowed here had only illustrative purpose).

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