Modeling the dengue fever transmission in a periodic environment

Modelando la transmisión de la fiebre del dengue en un entorno periódico

JULIÁN ALEJANDRO OLARTE*, ANIBAL MUÑOZ

Universidad del Quindío, Armenia, Colombia

ABSTRACT. A mathematical model for dengue fever transmission is analyzed, which incorporates relevant biological and ecological factors: vertical transmission and seasonality in the interaction between the vector (Aedes aegypti females) and the host (human). The existence and uniqueness of a positive disease-free periodic solution is proved; the global stability of the disease-free solution and the effect of periodic migrations of mosquitoes carrying the virus on the transmission of dengue are analyzed utilizing the mathematical definition of the Basic Reproductive Number in periodic environments; finally, it is numerically corroborated with the help of the Basic Reproductive Number that dengue cannot invade the disease-free state if it is less than one and can invade if it is greater than one, however, in both threshold conditions when vertical transmission occurs, the number of infected people and carrier vectors rises, representing a mechanism for the persistence of dengue cases in a community throughout a natural year.

Key words and phrases. Dengue, seasonality, Aedes aegypti, vertical transmission, Basic Reproductive Number.

2020 Mathematics Subject Classification. 37B55, 37C27, 34K60.

RESUMEN. Se propone un modelo matemático para la transmisión de la fiebre del dengue que incorpora factores biológicos y ecológicos relevantes: transmisión vertical y estacionalidad en la interacción entre el vector (Aedes aegypti) y el hospedero (humano). Se demuestra la existencia y unicidad de una solución periódica positiva libre de la enfermedad; la estabilidad global de la solución libre de la enfermedad y el efecto de las inmigraciones periódicas de mosquitos portadores del virus en la transmisión del dengue se analizan mediante la definición matemática del Número Reproductivo Básico en ambientes...
períódicos; finalmente, se corrobora numéricamente con ayuda del Número Reproductivo Básico que el dengue no puede invadir el estado libre de la enfermedad si es menor que uno y puede invadir si es mayor que uno, sin embargo, cuando en ambas condiciones de umbral ocurre transmisión vertical, se eleva el número de personas infectadas y vectores portadores, representando un mecanismo para la persistencia de casos de dengue en una comunidad a lo largo de un año natural.

**Palabras y frases clave.** Dengue, estacionalidad, *Aedes aegypti*, transmisión vertical, Número Reproductivo Básico.

1. Introduction

Dengue fever (DENV) is the arthropod-transmitted disease with the highest morbi-mortality in the world, also one of the most frequent causes of hospitalization and significant interruption of income potential in endemic areas (an estimated 390 million people become infected every year, 500,000 people suffering from severe dengue require hospitalization and 2.5% die), it affects the tropical and subtropical countries of Asia, the Pacific Islands, the Caribbean islands, Africa and Central and South America [11]. There are macrofactors to explain the increase of DENV on a global scale: climatics (global warming) and social, such as the increase in world population, the tendency to disorderly urbanization, international travel and poverty expressed in problems of housing, education, water supply, solid waste collection and others, as well as the lack of effective national and international programs against this disease and its vector; currently, vector control is the predominant strategy to prevent the spread of DENV because there are no effective, economical or tetravalent vaccine and treatment for the disease [43].

DENV belongs to the family *Flaviviridae* and there are four variants (serotypes) formally recognized: DEN-1, DEN-2, DEN-3 and DEN-4 [48], but in October 2013 a possible fifth sylvatic serotype (DENV-5) has been detected during screening of viral samples taken from a 37 year old farmer admitted in hospital in Sarawak state of Malaysia in the year 2007 [45]; the infection by a serotype 1 to 4 confers permanent immunity against this serotype and only for a few months against the rest of the serotypes; if a person is infected by one of the four serotypes, they will never be infected by the same serotype (homologous immunity), but lose immunity to the other three serotypes (heterologous immunity) in approximately 12 weeks and then becomes more susceptible to developing dengue hemorrhagic fever [22]. The primary vector of DENV is *Aedes aegypti* and the secondary vector is *Aedes albopictus*, both can feed at any time during the day and acquires the virus through the bite to a sick person during his period of viremia, which goes from a day before the onset of fever to an average of 5 or 6 days after the start of the same, being able to reach up to 9-10 days exceptionally [25].
Climate variables such as temperature, humidity and rainfall significantly influence the mosquito development and several studies suggest that entomological parameters are temperature sensitive as the dengue fever normally occurs in tropical and subtropical regions [41]; the high temperature increases the lifespan of mosquitoes and shortens the extrinsic incubation period of the dengue virus, increasing the number of infected mosquitoes, the rainfall provides places for eggs and for larva development [26]. Vertical (or transovarial) transmission (VT) of dengue virus in *Ae. aegypti* and *Ae. albopictus* also could explain the persistence over inter-epidemic periods in endemic areas [4] and is well documented under both experimental [44, 36] and field [5, 39] conditions.

Esteva and Vargas [18] studied the impact of VT on dengue disease dynamics, assuming that a proportion of vector recruitment occurred in the infectious class; the authors found that VT to increase dramatically the endemic proportion of infectious vectors, which could favour the persistence of the virus in areas with low human densities, conversely to mechanical transmission, which had a weak impact. Similar conclusions to Esteva and Vargas [18] were drawn by Coutinhoa et al. [12] from a structured model accounting for a pre-adult stage with periodic maturation rates and assuming that a proportion of the eggs laid by infected mosquitoes were vertically infected; the authors identified VT as a possible explanation of dengue overwintering and explained, using a time-dependent threshold condition, the delay observed between the peaks in vector density and in dengue cases.

A natural and important problem associated with epidemic models is to estimate whether an infection can invade and persist in a population, a threshold value used for this is the basic reproduction number (BRN), epidemiologically defined as the average number of secondary cases produced by an infected individual introduced into a fully susceptible population during their period of infectivity [16]. Diekmann et al., van den Driessche and Watmough [16, 56, 57] presented a general approach for the calculus of the BRN for compartmental disease transmission models, but with constant environment parameters. It is known that periodic fluctuations are common in the evolution of vector-borne diseases; periodic changes in the birth rates, mortality and contact of the populations are evident in ecosystems and many models in the literature inappropriately assume that the vector population is constant over time [2]. In the past twenty years, many authors have extended the definition of the BRN to periodic environments, we highlight authors like Bacaër and Guernaoui (2006), Thieme (2009), Bacaër (2011), Inaba (2012), Bacaër and Ait Dads (2012), Wang and Zhao (2017) [8, 55, 6, 35, 7, 59].

This paper is organized as follows: in section 2, the mathematical formulation and methodological approach of the proposed model is discussed; in section 3, existence of a dengue-free periodic solution is established; in section 4, we show that the dynamical properties of the model are completely determined by the BRN; in section 5, the BRN is derived numerically by solving a matrix
eigenvalue problem and computational simulations are presented to illustrate the analytical findings; finally, general conclusions are drawn in section 6.

Notation and concepts

Throughout the text the omission of \((t)\) from expressions that depend on time is allowed, the superscript \(\top\) indicates transposition of a matrix, \(\Re(\lambda)\) is the real part of \(\lambda\), \(\rho(A)\) denotes the spectral radius of a matrix \(A\), \(I_n\) is the \(n \times n\) identity matrix and \(O_n\) is the \(n \times n\) null matrix.

For a non-negative piecewise continuous \(\omega\)-periodic function \(L : \mathbb{R}_+ = [0, \infty) \to \mathbb{R}\), let

\[
L^n = \max_{t \in [0, \omega)} L(t), \quad L'^{l} = \min_{t \in [0, \omega)} L(t), \quad L^\infty = \limsup_{t \to \infty} L(t), \quad L_{\infty} = \liminf_{t \to \infty} L(t).
\]

The long-term average of \(L\) on the interval \([T, t + T]\) is defined by

\[
\langle L(t) \rangle = \lim_{T \to \infty} \frac{1}{T} \int_T^{t+T} L(\tau) d\tau.
\]

Let \(A\) be a square matrix. We say that \(A\) is cooperative if all its off-diagonal elements are non-negative and we say that \(A\) is irreducible if it cannot be placed into block upper-triangular form by simultaneous row/column permutations.

2. Model formulation

We consider a model of a dengue serotype that circulated in some community due to the interaction between the population of humans and the population of \textit{Aedes aegypti}, whose assumptions are:

1. Preserving some resemblance regarding the symptomatology of the disease in the host, we use the following nomenclature:
   - susceptible population/non-carrier population, subscript \(S\), comprising those individuals capable of catching the disease;
   - non-infectious carrier population, subscript \(E\), comprising those mosquitoes temporarily unable of transmitting the disease;
   - symptomatic population/infectious carrier population, subscript \(I\), comprising those individuals capable of transmitting the disease; and
   - recovered or immune population, subscript \(R\), including those individuals who acquire permanent immunity against infection.

2. All vector population measures refer to densities of female mosquitoes.

3. Humans are the main host of the virus and there are no alternative hosts available as blood sources.
(4) Disease-induced death in humans or in vectors is not considered.

(5) Carrier vectors probably transmit the virus throughout the life-span.

(6) The intrinsic rate of growth of the human population is constant.

(7) The variation birth rates of adult mosquitoes is strongly influenced by environmental factors such as temperature, humidity and rainfall and, therefore, is seasonally forced.

(8) Both populations are uniform and mixes homogeneously.

(9) Humans move from symptomatic state to immune state at constant rate $r$ and mosquitoes move from non-carrier state to carrier states at constant rate $r$ and $c$, where $1/r$ and $1/c$ are the average survival periods in their immediately preceding states.

(10) Because of vertical transmission, a fraction $g$ of newly emerged adult mosquitoes contributes to the non-infectious carrier class and the complementary fraction, $1 - g$, contributes to the non-carrier class.

**Model parameters:**

- $m$: natural mortality rate of adult mosquitoes.
- $h$: natural mortality rate of humans.
- $r$: human recovery rate.
- $b$: per head contact rate of adult female mosquitoes on humans, namely, the average number of bites per mosquito per day.
- $p$: probability of transmission of an infectious carrier mosquito by bite on a susceptible human.
- $q$: probability of transmission from a symptomatic human to mosquito.
- $c$: transfer rate of mosquitoes from non-infectious carrier to infectious carrier.
- $g$: rate of female mosquitoes hatched from viable eggs carrying the dengue serotype.
- $\Delta(t)$: mosquito recruitment rate (by birth and immigration) at time $t$.
- $H(t)$: total number of people in the community at time $t$. 

*Revista Colombiana de Matemáticas*
Dengue infection with one serotype is confirmed to produce lifelong immunity to that type, but only short-time protection against the other serotypes, which will increase risk of severe disease from secondary infection if a person previously exposed to serotype DENV-1 contracts serotype DENV-2 or -3, or if someone previously exposed to DENV-3 acquires DENV-2 [27]. Considering the structure of the SIR epidemic model, our epidemic model divides the human population into a susceptible, exposed and infectious classes. The population of the vector is described by a susceptible-exposed-infectious model, in which the intrinsic incubation period has a relatively low impact on the dynamics of transmission of dengue as shown in [47], and therefore is not represented. Conversely, the extrinsic incubation period is not ignored when studying the process of transmission of dengue because it covers a large part of the mosquito’s life expectancy [19, 52]. Even more, in many regions, the incidence of dengue fluctuates seasonally with few or no infections recorded in unfavorable periods and there is evidence that VT within the mosquito population allows the virus to persist at this time, so it is important include seasonal parameters and VT to explain intra-annual fluctuations of dengue epidemics [12].

Transmission dynamics is interpreted according to the compartment diagram in Figure 1.

With all the previous considerations, the transmission process is governed by the system of differential equations (1), where the cross infection between humans and vectors is modeled by the principle of mass-action normalized with the total population of humans, that is, the transmission of the virus occurs only when there is effective contact, which corresponds to an encounter between a susceptible individual (respectively non-carrier) with a symptomatic (respectively infectious carrier) with a transmission rate that includes, among
other aspects, the probability that the virus is transmitted

\[
\begin{align*}
\dot{H}_S &= hH - \frac{qb}{H}M_sH_S - hH_S \\
\dot{H}_I &= \frac{qb}{H}M_sH_I - (h + r)H_I \\
\dot{H}_R &= rH_I - hH_R \\
\dot{M}_S &= (1 - g)\Delta(t) - \frac{pb}{H}H_I M_s - m M_S \\
\dot{M}_E &= g\Delta(t) + \frac{pb}{H}H_I M_s - (c + m) M_E \\
\dot{M}_I &= c M_E - m M_I.
\end{align*}
\]

(1)

with initial conditions: \(H_s(0) = H_{s0} > 0, H_i(0) = H_{i0} > 0, H_e(0) = H_{e0} > 0, M_s(0) = M_{s0} > 0, M_i(0) = M_{i0} > 0, M_e(0) = M_{e0} > 0\) and \(H = H_s(t) + H_i(t) + H_e(t)\); the parameters verify that \(m, h, r, b, c > 0\) and \((p, q, g) \in [0, 1]^3\); the mosquito immigration rate, \(\Delta(t)\), is a non-constant continuous periodic function with period \(\omega\). The region of states of epidemiological interest is:

\[
\Pi = \left\{ x = [H_s H_i H_r M_s M_e M_i]^T \in \mathbb{R}_+^6 : 1 \leq H_s + H_i + H_r = H = \text{const} \wedge 0 \leq M_s + M_e + M_i \leq \frac{\Delta^u}{m} \right\}
\]

Proposition 2.1. The set (2), \(\Pi\), is forward invariant under the dynamic system (1).

Proof. Let

\[
\begin{bmatrix}
H_s & H_i & H_r & M_s & M_e & M_i
\end{bmatrix}^T = \begin{bmatrix} x_1 & x_2 & x_3 & x_4 & x_5 & x_6 \end{bmatrix}.
\]

Clearly for \(s_1 = -(qb + h)\), \(s_2 = -(h + r)\), and \(s_3 = -h\), it is possible to get \(\dot{x}_i \geq s_i x_i\) for \(x_i(0) \geq 0\), \(i = 1, 2, 3\). Analogously, for \(s_4 = -(pb + m)\), \(s_5 = -(c + ml)\), and \(s_6 = -m^2\), we have \(\dot{x}_i \geq s_i x_i\) for \(x_i(0) \geq 0\), \(i = 4, 5, 6\). Let \(W_i(t)\) be the solution of the scalar differential equation \(\dot{W}_i = s_i W_i\), \(W_i(0) = x_i(0)\), whose unique global solution is \(W_i(t) = x_i(0)e^{s_i t}\). By the standard comparison Lemma (see, for example, Lemma 3.4 in [38]), \(x_i(t) \geq x_i(0)e^{s_i t} \geq 0\) for all \(t \geq 0\). Now, the total number of hosts, \(H = H(t) = H_s(t) + H_i(t) + H_e(t)\), remains invariant over time since \(\dot{H} = 0\) for all \(t \geq 0\); the total number of vectors, \(M = M(t) = M_s(t) + M_e(t) + M_i(t)\), satisfies

\[
\dot{M} \leq \Delta^u - mM.
\]

(2)

Let \(W(t)\) be the solution of the differential equation \(\dot{W} = \Delta^u - mW\) and \(W(0) = M(0)\), then

\[
W(t) = \left( W(0) - \frac{\Delta^u}{m} \right) \exp(-mt) + \frac{\Delta^u}{m}.
\]
Note that $W$ is continuously strictly increasing over $\mathbb{R}_+$ if $W(0) < \Delta^u/m$. Consequently, by the comparison lemma, the solution of (2) is defined for all $t \geq 0$ and satisfies

$$M \leq \left( M(0) - \frac{\Delta^u}{m} \right) \exp\left(-mt\right) + \frac{\Delta^u}{m}$$

if $W(0) < \Delta^u/m$. Therefore, the invariance of $\Pi$ is a direct consequence of the non-negativity of solutions, $H$ constant and $M^\infty = \Delta^u/m$. □✓

The solution $x(t)$ with initial condition in (2) of the system of differential equations (1) is non-negative and uniformly bounded on $\mathbb{R}_+$. Moreover, the right-hand sides of these equations are differentiable with respect to $H_S$, $H_I$, $H_R$, $M_S$, $M_E$ and $M_I$ with continuous derivatives, therefore system (1) provides a unique maximal solution that remains in $\Pi$ for all $t \geq 0$ [38].

3. The dengue-free solution - DFS

As the dynamic system is non-autonomous by means of the mosquito recruitment rate, it has no steady states. This means that the right-hand sides of these equations in (1) equaled to zero, can not be satisfied simultaneously as $\Delta$ is time dependent function and all other variables are assumed to be constant at equilibrium, but still there is a free-disease solution ($g \equiv 0$): if $H_I(t) = M_E(t) = M_I(t) = 0$ for all $t \in \mathbb{R}_+$, the differential equation of non-carrier mosquitoes becomes

$$\frac{d}{dt} M_S(t) = \Delta(t) - mM_S(t); \quad M_S(t_0) > 0, \forall t_0 \geq 0. \quad (3)$$

Below are several lemmas that will be helpful in proving the main results.

Lemma 3.1. If $w : [0, \infty) \rightarrow \mathbb{R}$ is a periodic function of period $\omega$ and $t$ is any real number, then

$$\langle w(t) \rangle = \frac{1}{\omega} \int_0^\omega w(t) dt. \quad (4)$$

**Proof.** Let $T \in [k\omega, (k+1)\omega)$, $k = 0, 1, 2, \ldots$, then $T = k\omega + \rho$ where $\rho \in [0, \omega)$. Furthermore,

$$\int_t^{t+T} w(\tau) d\tau = \int_t^{t+k\omega} w(\tau) d\tau + \int_{t+k\omega}^{t+k\omega+\rho} w(\tau) d\tau = k \int_0^\omega w(\tau + t) d\tau + \int_0^\rho w(\tau + t) d\tau.$$

Dividing by $T = k\omega + \rho$ and taking the limit $k \to \infty$, we get

$$\langle w(t) \rangle = \frac{1}{\omega} \int_t^{t+\omega} w(\tau) d\tau.$$
Differentiating with respect to $t$ it follows that $\langle w(t) \rangle$ is constant $\forall t \in \mathbb{R}$, then just set $t = 0$.

Consider the following linear differential equation
\[ \frac{d}{dt} S(t) = \left( m - \Delta(t) S(t) \right) S(t), \quad S(t_0) = \frac{1}{M_S(t_0)}. \tag{5} \]

Lemma 3.2 implies that $M_S(t)$ is bounded by positive constants. For continuous periodic rates $\Delta(t)$ and $m(t)$ of period $\omega$, it follows from the theory of linear differential equations ([30], Theorem 1.1, p. 408) that a $\omega$-periodic solution exists if and only if (3) has at least one bounded solution. In the next lemma, we show that (3) has a unique globally attractive periodic solution.

**Lemma 3.2.** The linear differential equations (3) and (5) are equivalent, and there exist constants $N^l > 0$ and $N^u > 0$ such that $N^l \leq M_S(t) \leq N^u$ and $1/N^u \leq S(t) \leq 1/N^l$ for all $t \in \mathbb{R}_+$.

**Proof.** It is easy to verify that the change of variable $M_S(t) = 1/N_S(t)$ transforms equation (3) into equation (5). Let $\Delta^l \leq \Delta(t) \leq \Delta^u$, where $\Delta^u > \Delta^l > 0$. Notice that $\frac{d}{dt} N_S(t) \leq \left( m - \Delta^l N_S(t) \right) N_S(t)$, then $\frac{d}{dt} N_S(t) < 0$ if $N_S(t) > m/\Delta^l$ and hence, for all $t \in \mathbb{R}_+$, $N_S(t) \leq \max\{N_S(0), m/\Delta^l\} = 1/N^l > 0$.

To prove that $N_S(t)$ is bounded below by a positive constant, choose $\sigma > 0$ such that $N_S(0) \geq \sigma$, and
\[ m - \sigma \langle \Delta(t) \rangle - \sigma^2 = \rho_0 > 0. \tag{6} \]

Suppose $N_S(t)$ is not bounded below, then for each $0 < \theta < \sigma$, there exists an interval $[\tau_1, \tau_2]$ such that $N_S(\tau_1) = \sigma$, $N_S(\tau_2) = \theta$, and $N_S(t) < \sigma$ for $t \in [\tau_1, \tau_2]$.

Now notice that $\frac{d}{dt} N_S(t) \geq \left( m - \sigma \Delta^u \right) N_S(t)$ for $t \in [\tau_1, \tau_2]$. If $m - \sigma \Delta^u \geq 0$ it follows that $\theta = N_S(\tau_2) \geq N_S(t) \geq N_S(\tau_1) = \sigma$ for $t \in [\tau_1, \tau_2]$, therefore, the concordant inequality must be
\[ m - \sigma \Delta^u = -\rho_1 < 0. \]

In this case $N_S(t) \geq \sigma e^{\rho_1(t-\tau_1)}$ for $t \in [\tau_1, \tau_2]$. At $t = \tau_2$, $\theta \geq \sigma e^{\rho_1(\tau_2-\tau_1)}$, or equivalently
\[ \ln \left( \frac{\sigma}{\theta} \right)^{1/\rho_1} \leq \tau_2 - \tau_1. \tag{7} \]

Since $\theta$ can be chosen arbitrarily close to zero, Lemma 3.1 and inequality (7) imply that $T = \tau_2 - \tau_1$ can be sufficiently large so that
\[ \frac{1}{T} \int_{\tau}^{\tau+T} \Delta(\tau) d\tau < \langle \Delta(t) \rangle + \sigma. \tag{8} \]
For \( \theta \) chosen sufficiently small such that inequality (8) holds on the interval \( \tau_1 \leq t \leq \tau_2 \), \( \frac{d}{dt} N_S(t) \geq (m - \sigma \Delta(t)) N_S(t) \) for \( t \in [\tau_1, \tau_2] \). Thus,

\[
N_S(\tau_2) \geq N_S(\tau_1) \exp \left( \int_{\tau_1}^{\tau_2} (m - \sigma \Delta(t)) \, dt \right) > N_S(\tau_1) e^{\rho_0 \tau}.
\]

But because of the choice of \( \sigma \) in equation (6), the preceding inequality leads again to the contradiction, \( \theta > \sigma \). It only remains to conclude that \( N_S(t) \geq \min\{N_S(0), m/\Delta^u\} = 1/N^u > 0 \) for all \( t \in \mathbb{R}_+ \). Boundedness of \( N_S(t) \) implies that \( M_S(t) \) is bounded by positive constants \( N^l \leq M_S(t) \leq N^u \).

In the next lemma, we show that (3) has a unique globally attractive periodic solution.

**Lemma 3.3.** The solution \( M_S(t) \) of the initial scalar value problem (3) converges uniformly to a unique periodic solution \( \overline{M}_S(t) \) with period \( \omega \).

**Proof.** The initial value problem (3) has the solution

\[
M_S(t) = M_S(t_0) e^{-\omega(t-t_0)} + e^{-\omega(t-t_0)} \int_{t_0}^{t} e^{\omega t} \Delta(t) \, dt.
\]

A recursive relationship between the average number of non-carriers at \( t_k = t_0 + k\omega \) is given by

\[
M_{k+1} = M_S(t_{k+1}) = M_k e^{-\omega \Delta} + e^{-\omega} \int_{t_k}^{t_{k+1}} e^{\omega \Delta} \, d\zeta.
\]

Taking the change of variable \( \eta = \zeta - k\omega \) and because \( \Delta(\zeta) \) is a periodic function, then

\[
M_{k+1} = M_k e^{-\omega \Delta} + e^{-\omega} \int_{t_0}^{t_0+\omega} e^{\omega \eta} \Delta(\eta) \, d\eta.
\]

This defines a mapping \( S \) such that \( S(M_k) = M_{k+1} \). If \( M_{k_1} \) and \( M_{k_2} \) are different values of \( M_k \), then

\[
|S(M_{k_1}) - S(M_{k_2})| \leq e^{-\omega \Delta} |M_{k_1} - M_{k_2}|.
\]

So, \( S \) is a contraction mapping and in virtue of the Banach fixed point theorem [10] has a unique fixed point \( M_S^*(t_{k^*}) \) such that \( S(M_S(t_{k^*+1})) = S(M_S(t_{k^*})) = M_S(t_{k^*}) \), or equivalently, \( S(M_S(t_0 + k^* \omega)) = M_S(t_0 + (k^* + 1) \omega) \). This fixed point can be found for any solution \( M_S \) of the differential equation with arbitrary initial time \( t_0^* \). The fixed point has the form:

\[
M_S(t_0^*) = \left( \frac{1}{e^{\omega \Delta} - 1} \right) \int_{t_0^*}^{t_0^*+\omega} \exp \left( \int_{t_0^*}^{t_0^*+\omega} m(\tau) \, d\tau \right) \Delta(\eta) \, d\eta.
\]
This fixed point is a continuously differentiable function with respect to $t^*_0$ and leads to defining the function

$$
\overline{M}_S(t) = \frac{1}{e^{\omega m} - 1} \int_t^{t+\omega} e^{(\eta-t)m} \Delta(\eta) d\eta,
$$

which satisfies the property:

$$
\overline{M}_S(t + \omega) = \frac{1}{e^{\omega m} - 1} \int_{t+\omega}^{t+2\omega} e^{(\eta-(t+\omega))m} \Delta(\eta) d\eta = \int_{t+\omega}^{t+\omega+\omega} e^{(\eta-(t+\omega))m} \Delta(\eta) d\eta = \overline{M}_S(t).
$$

Hence $\overline{M}_S$ is periodic with period $\omega$, or what is the same $\overline{M}_S(t) = \overline{M}_S(t + k\omega)$ ($k \in \mathbb{Z}_+)$.

Applying the substitution $\zeta = \eta + k\omega$ we arrive to:

$$
\overline{M}_S(t) = \frac{1}{e^{\omega m} - 1} \int_t^{t+(k+1)\omega} e^{(\zeta-k\omega-t)m} \Delta(\zeta - k\omega) d\zeta
$$

$$
= \frac{1}{e^{\omega m} - 1} \int_t^{t+(k+1)\omega-k\omega} e^{((\zeta+k\omega)-k\omega-t)m} \Delta((\zeta+k\omega) - k\omega) d\zeta
$$

$$
= \frac{1}{2e^{\omega m/2}(e^{\omega m/2} - e^{-\omega m/2})/2} \int_t^{t+\omega} e^{(\zeta-t)m} \Delta(\zeta) d\zeta
$$

$$
= \frac{1}{2e^{\omega m/2} \sinh (\omega m/2)} \int_t^{t+\omega} e^{(\zeta-t)m} \Delta(\zeta) d\zeta
$$

$$
= \frac{1}{2} \csc h \left( \frac{\omega}{2} m \right) \exp \left( -\frac{\omega}{2} m \right) \int_t^{t+\omega} e^{(\zeta-t)m} \Delta(\zeta) d\zeta
$$

What follows is to prove that all the solutions of (3) converges uniformly to the periodic solution (12) and $\overline{M}_S(t)$ is unique. The derivative of $N(t) = |M_S(t) - \overline{M}_S(t)|$ is

$$
\frac{d}{dt} N(t) = \text{sgn} \left( M_S(t) - \overline{M}_S(t) \right) \left( \Delta(t) - mM_S(t) - \Delta(t) - m\overline{M}_S(t) \right) = -mN(t).
$$

The problem $\frac{d}{dt} N(t) = -mN(t)$ with $N(t_0) = |M_S(t_0) - \overline{M}_S(t_0)|$ has solution

$$
N(t) = N(t_0)e^{-(t-t_0)m} \Rightarrow N(t + k\omega) \leq N(t_0)e^{-k\omega m}.
$$

Having $|M_S(t + k\omega) - \overline{M}_S(t)| \leq N(t_0)e^{-k\omega m}$ for $t \in [0, \omega)$, it means that $M_S(t + k\omega)$ converges uniformly to the periodic solution $\overline{M}_S(t)$ as $k \to \infty$ for $t \in [0, \omega)$. 
There cannot be two periodic solutions: the right member of equation (5) is continuous in $\mathbb{R}_+$ and locally Lipschitzian with respect to $M_S$ in $[N^I, N^u] \subset \mathbb{R}_+$, with Lipschitz constant $m > 0$, that is
\[
\left| \sum_{i=1}^{2} (-1)^i \left[ \Delta(t) - m M_{S,i}(t) \right] \right| \leq m |M_{S,2}(t) - M_{S,1}(t)|
\]
for all $(t, M_{S,i}) \in [t_0, \infty) \times [N^I, N^u]$, then there exists a unique solution passing through $(t_0, M_S(t_0))$ ([38], Theorem 3.3, p. 94). □✓

**Proposition 3.4.** System (1) have a unique continuously differentiable dengue-free periodic solution of period $\omega$, given by
\[
x^0 = [\overline{H}_S(t) \ 0 \ 0 \ 0 \ \overline{M}_S(t) \ 0 \ 0]^T,
\]
where $\overline{H}_S(t) = H(0)$ and $\overline{M}_S(t) = \frac{1}{2} \text{csch} \left( \frac{\omega}{2m} \right) \exp \left( -\frac{\omega}{2m} \right) \int_{t}^{t+\omega} e^{(\zeta-t)m} \Delta(\zeta) d\zeta.$

And any DFS to (1) approaches this one as time becomes large.

**Proof.** By Lemmas 3.2 and 3.3, the Cauchy problem (3) admits a unique positive periodic solution (12), which is globally attractive. Since $H'_R(t) = -h H_R(t)$ then $H_R(t) = 0$ is an equilibrium solution for the recovered population, moreover $H_s(t) = H - H_i(t) - H_R(t) = H(0)$ at equilibrium. As dengue is originally absent, it is not spreading in the ecosystem, so the condition is that $g \equiv 0$. Notice that any solution of (3) ultimately lies in (2):
\[
\int_{t_0}^{t} e^{(\zeta-t_0)m} \Delta(\zeta) d\zeta \leq \int_{t_0}^{t} e^{(\zeta-t_0)m} \Delta u d\zeta = \frac{\Delta u}{m} e^{(t-t_0)m} - \frac{\Delta u}{m^r}.
\]
and substituting this into (9),
\[
M_S(t) < \frac{\Delta u}{m} e^{-(t-t_0)m} + e^{-(t-t_0)m} \left( \frac{\Delta u}{m} e^{(t-t_0)m} - \frac{\Delta u}{m^r} \right) = \frac{\Delta u}{m^r}.
\]
Thus system (1) admits a unique DF periodic solution given by (13). □✓

### 4. Threshold dynamics

#### 4.1. Basic Reproductive Number

Utilizing a notation similar to that in [58], we sort the compartments so that the first three compartments correspond to infected individuals. Let
\[
x^T = [H_i \ M_i \ M_s \ H_s \ M_b \ H_b] = [x_1 \ x_2 \ x_3 \ x_4 \ x_5 \ x_6]
\]
and define
- $F_i$: the rate of new infections in compartment $i$.
- $V_i^+$: the rate of transfer individuals into compartment $i$ by others means.
- $V_i^-$: the rate of transfer individuals out of compartment $i$.

System (1) can be written in the form:

$$\frac{d\mathbf{x}}{dt} = F(t, \mathbf{x}) - V(t, \mathbf{x}) = f(t, \mathbf{x}),$$

where

$$F(t, \mathbf{x}) = [F_1(t, \mathbf{x}) F_2(t, \mathbf{x}) \cdots F_6(t, \mathbf{x})]^\top = \begin{bmatrix} \frac{qb}{H} \mathbb{P}_4 0 \frac{pb}{H} \mathbb{P}_1 0 0 0 \end{bmatrix}^\top,$$

$$V(t, \mathbf{x}) = \begin{bmatrix} V_1(t, \mathbf{x}) & V_2(t, \mathbf{x}) & \ldots & V_6(t, \mathbf{x}) \end{bmatrix} = V^-(t, \mathbf{x}) - V^+(t, \mathbf{x}),$$

$$V^-(t, \mathbf{x}) = \begin{bmatrix} (h + r)\mathbb{P}_1 \\ m\mathbb{P}_2 \\ (c + m)\mathbb{P}_3 \\ \frac{qb}{H}\mathbb{P}_4 + h\mathbb{P}_4 \\ \frac{pb}{H}\mathbb{P}_5 + m\mathbb{P}_5 \\ h\mathbb{P}_6 \end{bmatrix},$$

$$V^+(t, \mathbf{x}) = \begin{bmatrix} 0 \\ c\mathbb{P}_3 \\ g\Delta(t) \\ hH \\ (1 - g)\Delta(t) \\ r\mathbb{P}_1 \end{bmatrix}$$

and

$$f(t, \mathbf{x}) = \begin{bmatrix} f_1(t, \mathbf{x}) \\ f_2(t, \mathbf{x}) \\ f_3(t, \mathbf{x}) \\ f_4(t, \mathbf{x}) \\ f_5(t, \mathbf{x}) \\ f_6(t, \mathbf{x}) \end{bmatrix}.$$ 

Linearizing system (1) around the disease free solution (13), we obtain the partial derivative matrices

$$\mathbf{T}(t) = \left[ \frac{\partial F_i(t, \mathbf{x}^0)}{\partial \mathbb{P}_j} \right]_{1 \leq i, j \leq 3} = \begin{bmatrix} 0 & qb & 0 \\ 0 & 0 & 0 \\ \frac{pb\mathbb{X}_6(t)}{H} & 0 & 0 \end{bmatrix}$$

and

$$\mathbf{V}(t) = \left[ \frac{\partial V_i(t, \mathbf{x}^0)}{\partial \mathbb{P}_j} \right]_{1 \leq i, j \leq 3} = \begin{bmatrix} h + r & 0 & 0 \\ 0 & m & -c \\ 0 & 0 & c + m \end{bmatrix}.$$ 

For a compartmental epidemiological model based on an autonomous system, the BRN is defined as the expected number of secondary cases produced by a typical infected individual during its entire infectious period in a population consisting of susceptibles only [57], and it is determined by the spectral radius of the next-generation matrix (which is independent of time). Multiple researchers have investigated the rich nonlinear effects caused by periodically varying rates in epidemic models to the point of generalizing the definition of BRN for non-autonomous systems as mentioned in section 1. Particularly, Wang and Zhao in [58] extended the work of [57] for a large class of epidemic models in periodic
environments. They established the next infection operator $\mathcal{L} : C_\omega \rightarrow C_\omega$ given by

$$\text{(16)} (\mathcal{L}\phi)(t) = \int_0^\infty Y(t, t-s)F(t-s)\phi(t-s)ds$$

where $C_\omega$ be the ordered Banach space of all $\omega$-periodic functions $\phi : \mathbb{R} \rightarrow \mathbb{R}^3$, which is equipped with the maximum norm and the positive cone $C_\omega^+ = \{ \phi \in C_\omega : \phi(t) \geq 0, \forall t \in \mathbb{R} \}$. $\phi(s) \in C_\omega$ represents the initial distribution of infectious individuals in this periodic environment, and $Y(t, s)$ is the evolution operator of the linear $\omega$-periodic system:

$$\text{(17)} \frac{dz}{dt} = -\nabla(t)z,$$

which means the $3 \times 3$ matrix $Y$ satisfies

$$\frac{dY(t, s)}{dt} = -\nabla(t)Y(t, s), \quad Y(s, s) = I_3$$

for each $t \geq s$, $s \in \mathbb{R}$. Interpretation: $\mathcal{L}\phi$ is the distribution of accumulative new infections at time $t$ produced by all those infected individuals $\phi(s)$ introduced before $t$, with kernel $K(t, s) = Y(t, t-s)F(t-s)$; the coefficient $K_{ij}(t, s)$ in row $i$ and column $j$ represents the expected number of individuals in compartment $I_i$ that one individual in compartment $I_j$ generates at the beginning of an epidemic per unit time at time $t$ if it has been in compartment $I_j$ for $s$ units of time, with $I_1 = H, I_2 = M$ and $I_3 = M_i$ [7].

Let $r_0 > 0$, $r_0$ is an eigenvalue of $L$ if there is a non-negative eigenfunction $\psi(t) \in C_\omega$ such that

$$\mathcal{L}\psi = r_0\psi.$$

Therefore, the basic reproduction number is defined as

$$R_0 := \rho(\mathcal{L}),$$

the spectral radius of $\mathcal{L}$.

### 4.2. Dengue extinction

Following the setting of [58] for non-autonomous compartmental epidemic models, we verify the following assumptions that show again that the model is well-posed and makes biological sense.

(A1) For $1 \leq i \leq n$, the functions $F_i(t, \mathbf{x})$, $V_i^+(t, \mathbf{x})$ and $V_i^-(t, \mathbf{x})$ are nonnegative and continuous on $\mathbb{R} \times \mathbb{R}^n$ and continuously differential with respect to $\mathbf{x}$.
(A2) There is a real number $\omega > 0$ such that for each $1 \leq i \leq n$, the functions are $\omega$-periodic in $t$. (This is new for periodic models.)

(A3) If $\pi_i = 0$ then $V_i^{-} = 0$. In particular, we define that $X_s$ is a disease-free subspace, so that if $x \in X_s$, then $V_i^{-} = 0$ for $i = 1, \ldots, \tilde{m}$ ($\tilde{m}$ is the number of compartments of infected and carrier individuals.)

(A4) $F_i = 0$ for $i > \tilde{m}$.

(A5) If $x \in X_s$, then $F_i = 0$ and $V_i^{+} = 0$ for $i = 1, \ldots, \tilde{m}$.

(A6) Define an $(n - \tilde{m}) \times (n - \tilde{m})$ matrix $\tilde{M}(t) = \left[ \frac{\partial f_i(t, x^0)}{\partial x_j} \right]_{\tilde{m}+1 \leq i,j \leq n}$ and let $\Phi_{\tilde{M}}(t)$ be the monodromy matrix of the linear $\omega$-periodic system $dy/dt = \tilde{M}(t)y$, we then have that $\rho(\Phi_{\tilde{M}}(\omega)) < 1$.

(A7) $\rho(\Phi_{\omega}^{-1}(\omega)) < 1$, where $\Phi_{\omega}^{-1}(t)$ is the monodromy matrix of (17).

With $\tilde{m} = 3$ and $n = 6$, it is simple to check the assumptions (A1)-(A5) from observation of the vectors $F$ and $V$ in (14). Now it remains to verify conditions (A6) and (A7).

We know that (1) has the disease-free periodic solution (13), so to verify assumption (A6), we define

$$\tilde{M}(t) = \left[ \frac{\partial f_i(t, x^0)}{\partial x_j} \right]_{4 \leq i,j \leq 6} = \left[ \begin{array}{ccc} -\frac{q b}{H} x_2 - h & 0 & 0 \\ 0 & -\frac{p b}{H} x_1 - m & 0 \\ 0 & 0 & -h \end{array} \right]_{x = x^0} \quad (19)$$

and solving the system $dy/dt = \tilde{M}(t)y$ yields the principal fundamental matrix:

$$\Phi_{\tilde{M}}(t) = \left[ \begin{array}{ccc} e^{-ht} & 0 & 0 \\ 0 & e^{-mt} & 0 \\ 0 & 0 & e^{-ht} \end{array} \right].$$

Clearly $\Phi_{\tilde{M}}^{-1}(0) = I_3$, and the monodromy matrix is the principal fundamental matrix evaluated at the period ($\Phi_{\tilde{M}}(\omega)$), thus (A6) is satisfied.

From matrices (15) and the evolution operator of the linear system (17), that is

$$\frac{dY(t, s)}{dt} = \left[ \begin{array}{ccc} -(h + r) & 0 & 0 \\ 0 & -m & c \\ 0 & 0 & -(c + m) \end{array} \right] Y(t, s), \ Y(s, s) = I_3 \quad (20)$$

Revista Colombiana de Matemáticas
for each \( t \geq s \) (\( s \in \mathbb{R} \)), then (A7) above must be verified. The eigenvalues of \(-V(t)\) are

\[
s_0 = -(h + r), \quad s_1 = -m \quad \text{and} \quad s_2 = -(c + m).
\]

A triplet of corresponding eigenvectors is

\[
\begin{bmatrix}
1 \\
0 \\
0
\end{bmatrix}, \quad \begin{bmatrix}
0 \\
1 \\
0
\end{bmatrix}, \quad \begin{bmatrix}
0 \\
0 \\
1
\end{bmatrix}.
\]

We create the matrices

\[
P = \begin{bmatrix}
1 & 0 & 0 \\
0 & 1 & -1 \\
0 & 0 & 1
\end{bmatrix} \quad \text{and} \quad P^{-1} = \begin{bmatrix}
1 & 0 & 0 \\
0 & 1 & 1 \\
0 & 0 & 1
\end{bmatrix}.
\]

Under the coordinate transformation \( \eta = P^{-1}z \), we obtain the uncoupled linear system

\[
\dot{\eta} = -P^{-1}V(t)P\eta = \begin{bmatrix}
s_0 & 0 & 0 \\
0 & s_1 & 0 \\
0 & 0 & s_2
\end{bmatrix} \eta
\]

whose general solution is given by

\[
\eta(t) = \text{diag} \left[ e^{s_0 t}, \quad e^{s_1 t}, \quad e^{s_2 t} \right] d,
\]

where \( d \) is a constant vector. Since \( z = P\eta \) and \( d = P^{-1}c \), it follows that (17) has a fundamental matrix:

\[
\Psi(t) = P \text{diag} \left[ e^{s_0 t}, \quad e^{s_1 t}, \quad e^{s_2 t} \right] P^{-1} = \begin{bmatrix}
e^{s_0 t} & 0 & 0 \\
0 & e^{s_1 t} & e^{s_1 t} - e^{s_2 t} \\
0 & 0 & e^{s_2 t}
\end{bmatrix}
\]

with inverse

\[
\Psi^{-1}(t) = \begin{bmatrix}
e^{-s_0 t} & 0 & 0 \\
0 & e^{-s_1 t} & e^{-s_1 t} - e^{-s_2 t} \\
0 & 0 & e^{-s_2 t}
\end{bmatrix},
\]

but \( \Psi(t) \) is not a principal fundamental matrix at \( t = s \). Using that

\[
\Psi(t)\Psi^{-1}(s) = \begin{bmatrix}
e^{s_0(t-s)} & 0 & 0 \\
0 & e^{s_1(t-s)} - e^{s_2(t-s)} & 0 \\
0 & 0 & e^{s_2(t-s)}
\end{bmatrix}
\]

Volumen 55, Número 1, Año 2021
is also a fundamental matrix which satisfies $\Psi(s)\Psi(s)^{-1} = I_3$, we define the evolution operator by

$$Y(t, s) = \begin{bmatrix} e^{s_0(t-s)} & 0 & 0 \\ 0 & e^{s_1(t-s)} - e^{s_2(t-s)} & 0 \\ 0 & 0 & e^{s_2(t-s)} \end{bmatrix}.$$  

The monodromy matrix $\Phi_{-V}(t)$ of the system (17) equals $Y(t, 0)$, $\forall t \geq 0$. So one need only consider the monodromy matrix evaluated at the period; the roots of the equation $\det [\Phi_{-V}(\omega) - \lambda I] = 0$ are $\lambda_j = e^{s_j \omega}$, $(j = 1, 2, 3)$. Since $\Re(s_j) < 0$ for all $j$, the spectral radius becomes $\max_j \{|\exp(s_j \omega)|\} < 1$ and clearly (A7) is true.

Furthermore, in order to characterize $R_0$ for periodic systems, we consider the following linear $\omega$-periodic system

$$\dot{w} = \left(\frac{1}{\lambda} F(t) - V(t)\right)w(t) \quad t \in \mathbb{R}^+, \lambda \in (0, \infty). \quad (21)$$

Let $W(t, s, \lambda), t \geq s, s \in \mathbb{R}$, be the evolution operator of the system (21) on $\mathbb{R}^3$. It is clear that

$$W(t, 0, 1) = \Phi_{-V}(t), \quad \forall t \geq 0.$$  

We need other results to analyze the stability of the disease-free solution in this section.

**Lemma 4.1** (Lemma 2.1 in [58]). Assume that (A1)-(A7) hold.

(i) $\rho(W(\omega, 0, \lambda)) = 1$ has a positive solution $\lambda_0$, then $\lambda_0$ is an eigenvalue of $\mathcal{L}$, and hence $R_0 > 0$.

(ii) If $R_0 > 0$, then $\lambda = R_0$ is the unique solution of $\rho(W(\omega, 0, \lambda)) = 1$.

(iii) If $R_0 = 0$ if and only if $\rho(W(\omega, 0, \lambda)) < 1$, for all $\lambda > 0$.

**Lemma 4.2.**

$$\max \left\{ (M_S - \overline{M}_S)^\infty, (H_S - \overline{H}(0))^\infty \right\} \leq 0.$$  

**Proof.** The differential equation of non-carrier mosquitoes implies that

$$\frac{d}{dt} M_S(t) = (1 - f)\Delta - \frac{p}{H}H_1 M_S - m M_S \leq (1 - f)\Delta(t) - m M_S(t).$$

Revista Colombiana de Matemáticas
Since \( \mathbf{x}^0 = [H \ 0 \ 0 \ 0 \ \overline{M}_S(t) \ 0 \ 0] \) is a solution of the system (1), then
\[
\frac{d}{dt} \overline{M}_S(t) = (1 - f) \Delta - m \overline{M}_S.
\]
Therefore,
\[
\frac{d}{dt} (M_S(t) - \overline{M}_S(t)) \leq -m (M_S(t) - \overline{M}_S(t)).
\]
Integrating this inequality over \([t_0, t]\), then
\[
M_S(t_n) - \overline{M}_S(t_n) \leq (M_S(0) - \overline{M}_S(0)) e^{-mt_n}.
\]
Now, by applying the fluctuation lemma [34], there is a sequence \( \{t_n\} \) such that \( t_n \to \infty \) and \( (M_S(t_n) - \overline{M}_S(t_n)) \to (M_S(t) - \overline{M}_S(t)) \) as \( n \to \infty \). Letting \( n \to \infty \) and knowing that \( (M_S - \overline{M}_S)(t) \) is bounded for all \( t \geq 0 \) because it is a solution of equation (3), it follows that
\[
(M_S - \overline{M}_S) \to 0.
\]
Arguing as previously, we deduce that
\[
\frac{d}{dt} (H_S(t) - \overline{H}_S(0)) \leq -h (H_S(t) - \overline{H}_S(0))
\]
and \( (H_S - \overline{H}_S(0)) \to 0 \). Lemma 4.2 now follows straightforwardly. \( \Box \)

**Lemma 4.3** (Lemma 2.1 in [46]). Let \( A(t) \) be a continuous, cooperative, irreducible and \( \omega \)-periodic matrix function, let \( \Phi_{A(t)} \) be the fundamental matrix solution of
\[
\dot{x} = A(t)x
\]
and let \( \overline{p} = \frac{1}{\omega} \ln (\rho (A(\omega))) \), then there exists a positive \( \omega \)-periodic function \( \mathbf{v}(t) \) such that \( e^{\overline{p}t} \mathbf{v}(t) \) is a solution of (22).

**Proposition 4.4** ([58]). Assume that (A1)-(A7) hold.

(i) \( R_0 = 1 \iff \rho (\Phi_{F - V}) = 1 \).

(ii) \( R_0 < 1 \iff \rho (\Phi_{F - V}) < 1 \).

(iii) \( R_0 > 1 \iff \rho (\Phi_{F - V}) > 1 \).

**Proof.** (i) If \( R_0 = 1 \), then from Lemma 4.5(ii), we have \( \rho(W(\omega,0,1)) = \rho(\Phi_{F - V}(\omega)) = 1 \). Otherwise, if \( \rho(\Phi_{F - V}(\omega)) = 1 \), then Lemma 4.5 (i) and (ii) imply that \( R_0 = 1 \).
\((ii) (a) \) Assume that \( R_0 > 1 \). Since \( R_0 \) is positive and the linear operator \( \mathcal{L} \) is compact and positive, then thanks to Krein-Rutman theorem (see, e.g., [32], Theorem 7.1), \( R_0 \) is an eigenvalue of \( \mathcal{L} \) with a positive eigenfunction \( y \) in \( C_\omega \). Thus, for some \( t_0 \in [0, \omega] \), \( y(t_0) > 0 \) and we have
\[
\dot{y} = (\mathcal{F}(t) - \mathcal{V}(t))y + \left( \frac{1}{R_0} - 1 \right) \mathcal{F}(t)y \tag{23}
\]
with \( \mathcal{F}y(t) \neq 0 \) for all \( t \in \mathbb{R} \). Moreover, by applying the constant-variation formula to equation (23), we obtain
\[
y(t_0) - W(t_0 + \omega, t_0, 1) = \mathcal{K}
\]
with
\[
\mathcal{K} = \left( \frac{1}{R_0} - 1 \right) \int_{t_0}^{t_0+\omega} W(t_0 + \omega, s, 1) \mathcal{F}(s)y(s)ds.
\]
Note that if the matrix \( \mathcal{V}(t) \) is irreducible, then \( W(t_0 + \omega, s, 1) \) is strongly positive for each \( t > s \), \( s \in \mathbb{R} \) and \( \mathcal{K} \ll 0 \) if \( R_0 > 1 \). Hence,
\[
-\dot{y}(t_0) + W(t_0 + \omega, t_0, 1) = -\mathcal{K} \gg 0 \text{ in } \mathbb{R}^4.
\]
Since \( -\dot{y}(t_0) \ll 0 \), then from ([32], Theorem 7.3) \( \rho(W(t_0 + \omega, t_0, 1)) = \rho(\Phi_{\mathcal{T}_-\mathcal{V}}(\omega)) > 1 \).

\((b)\) Assume that \( \rho(\Phi_{\mathcal{T}_-\mathcal{V}}(\omega)) > 1 \). Thus we have \( \rho(W(\omega, t_0, 1)) = \rho(\Phi_{\mathcal{T}_-\mathcal{V}}(\omega)) > 1 \) and from Lemma 4.5(iii) we get \( R_0 > 0 \), and of course (23) is still valid. Hence, if \( R_0 \in (0, 1) \) and in the case where \( \mathcal{V} \) is irreducible, it follows that \( W(t_0 + \omega, t_0, 1) = \rho(\Phi_{\mathcal{T}_-\mathcal{V}}(\omega)) < 1 \) that leads a contradiction, so \( R_0 > 1 \).

\((iii)\) is a consequence of the conclusions (i) and (ii) above.

\(\Box\)

**Proposition 4.5.** Let \( R_0 \) be defined as (18), then the disease free periodic solution (3.4) is locally asymptotically stable if \( R_0 < 1 \), and unstable if \( R_0 > 1 \).

**Proof.** Let \( \mathcal{J}(t) \) be the Jacobian matrix of (1) evaluated at \( x^0 \). Then, we have
\[
\mathcal{J}(t) = \begin{bmatrix} \mathcal{F}(t) - \mathcal{V}(t) & O_3 \\ J_3(t) & J_4(t) \end{bmatrix}
\]
where \( \mathcal{F} \) and \( \mathcal{V} \) are the matrices defined in (15), \( J_i(t) = \tilde{M}(t) \) is the matrix defined in (19), and
\[
J_3(t) = \begin{bmatrix} 0 & -\frac{q_b}{\mathcal{H}} \pi_4 & 0 \\ \frac{p_b}{\mathcal{H}} \pi_5 & 0 & 0 \\ \frac{r}{\mathcal{H}} & 0 & 0 \end{bmatrix} \bigg|_{x=x^0}
\]
In view of assumption (A6), \( \rho(\Phi_{j_1}(\omega)) < 1 \), so that the stability of the DFS depends on the eigenvalues of \( \Phi_{F - V}(\omega) \); if \( \rho(\Phi_{F - V}(\omega)) < 1 \) then \( x^0 \) is stable, but, if \( \rho(\Phi_{F - V}(\omega)) > 1 \) then \( x^0 \) is unstable [51]. Thus, thanks to Proposition 4.5, \( x^0 \) is locally asymptotically stable if \( R_0 < 1 \) and unstable if \( R_0 > 1 \). □✓

We are now in conditions to state a result about global stability of the DFS.

**Proposition 4.6.** The DFS (3.4) of system (1) is globally asymptotically stable if \( R_0 < 1 \).

**Proof.** By Proposition 4.5, if \( R_0 < 1 \) then \( x^0 = [H \ 0 \ 0 \ 0 \ \overline{M}_S(t) \ 0 \ 0]^\top \) is locally asymptotically stable, so it is sufficient prove that \( x^0 \) attracts all non-negative solutions \( x(t) \) of (1). On the other hand, given \( \epsilon > 0 \) and by Lemma 4.2, we have

\[
(M_S - \overline{M}_S)^\infty = \lim_{t \to \infty} \sup_{\tau \geq t} (M_S(\tau) - \overline{M}_S(\tau)) = L \leq 0,
\]

then there exists a \( \mathcal{N} > 0 \) such that for all \( \tau_3 > \mathcal{N} \)

\[
-\epsilon < \sup_{\tau \geq \tau_3} (M_S(t) - \overline{M}_S(t)) - L < \epsilon,
\]

this implies that \( \sup_{\tau \geq \tau_3} (M_S(t) - \overline{M}_S(t)) < L + \epsilon \leq \epsilon \). Then, from definition of supremum we have \( M_S(t) \leq \overline{M}_S(t) + \epsilon \) for all \( t \geq \tau_3 \). Therefore, by the second, fifth and sixth equations in (1) we have

\[
\begin{align*}
\dot{H}_i &\leq q b M_1 - (h + r) H_i, \\
\dot{M}_i &= c M_E - m M_i, \\
\dot{M}_E &\leq \frac{p b}{H} H_i (\overline{M}_S + \epsilon) - (c + m) M_E,
\end{align*}
\]

Let

\[
M_1(t) = \begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ \frac{p b}{H} & 0 & 0 \end{bmatrix}.
\]

Consider the perturbed subsystem

\[
\begin{align*}
\dot{w}_1 &= q b w_2 - (h + r) w_1, \\
\dot{w}_2 &= c w_3 - m w_2, \\
\dot{w}_3 &= \frac{p b}{H} w_1 (\overline{M}_S + \epsilon) - (c + m) w_3,
\end{align*}
\]
or, in matrix language,
\[
\begin{bmatrix}
\dot{w}_1 \\
\dot{w}_2 \\
\dot{w}_3
\end{bmatrix}
= \begin{bmatrix}
\mathbf{F}(t) - \mathbf{V}(t) + \epsilon M_1(t)
\end{bmatrix}
\begin{bmatrix}
w_1 \\
w_2 \\
w_3
\end{bmatrix}
\]
with \( \mathbf{F} \) and \( \mathbf{V} \) defined in (15). Matrix \( \mathbf{F} - \mathbf{V} + \epsilon M_1(t) \) is \( \omega \)-periodic, cooperative, irreducible and continuous. By Lemma 4.3, for all \( s > 0 \), the function \( se^{\mathbf{F}(t-\tau_0)}v(t-\tau_0) \) is also a solution of system (25) with initial condition \( sv(0) \) at \( t = \tau_0 \).

Choose a \( \tilde{t} > t_1 \) and \( \bar{s} > 0 \) such that \( \left[ \mathbf{H}(\tilde{t}) \quad M_\epsilon(\tilde{t}) \quad M_1(\tilde{t}) \right]^T \leq \bar{s}v(0) \), then from (24)

\[
\frac{d}{dt} \mathbf{y} = \frac{d}{dt} \left[ \mathbf{H} \quad M_\epsilon \quad M_1 \right]^T \leq (\mathbf{F} - \mathbf{V}) \mathbf{y} + \epsilon M_1 \mathbf{y}
\]
and applying comparison principle ([33], Theorem B.1), we have \( \mathbf{y} \leq \bar{s} e^{\mathbf{F}(t-\tilde{t})}v(t-\tilde{t}) \) for all \( t \geq \tilde{t} \).

From Proposition 4.5 we conclude that \( \rho \left( \Phi_{\mathbf{F} - \mathbf{V}} \right) < 1 \) if and only if \( R_0 < 1 \). By the continuity of the spectrum for matrices ([37], Section II.5.8), there exists a \( \epsilon > 0 \) small enough such that \( \rho \left( \Phi_{\mathbf{F} - \mathbf{V} + \epsilon M_1(t)} \right) < 1 \), consequently \( \bar{p} < 0 \). So, utilizing positivity of solutions and comparison theorem [40]:

\[
0 \leq \lim_{t \to \infty} H_i(t) \leq \lim_{t \to \infty} s_1 e^{\mathbf{F}(t-\tilde{t})}v_1(t-\tilde{t}) = 0.
\]

Similarly for \( M_\epsilon(t) \) and \( M_1(t) \):

\[
\lim_{t \to \infty} M_\epsilon(t) = 0, \quad \lim_{t \to \infty} M_1(t) = 0.
\]

We need prove that \( H_S(t) \) approaches to \( H(0) \). At infection free solution \( H_R(t) = 0 \), where \( H_R \) satisfies the equation

\[
\frac{d}{dt} \left( H_R - \overline{H}_u \right) = r H_i - h \left( H_R - \overline{H}_u \right).
\]

Due to (26) and given \( \epsilon_1 > 0 \) we can find a \( \tau_4 > 0 \) such that \( H_I < \epsilon_1 \) for \( t > \tau_4 \), then

\[
\frac{d}{dt} H_R \leq r\epsilon_1 - h H_R.
\]

Multiplying in both sides by \( e^{ht} \) and integrating this inequality over \([\tau_4, t]\) we get

\[
H_R(t) \leq H_R(\tau_4)e^{-h(t-\tau_4)} + \frac{r\epsilon_1}{h} \left( 1 - e^{-h(t-\tau_4)} \right)
\]
and $H_{\infty}^R \leq \frac{r\epsilon_1}{h}$. As $\epsilon_1$ is arbitrarily small then $H_{\infty}^R \leq 0$. For $\epsilon_2 > 0$, we can find a $\tau_5 > 0$ such that $H_{R}(t) \leq \epsilon_2/2$ for $t \geq \tau_5$. Also, from (26), we can find a $\tau_4 > 0$ with $H_1 < \epsilon_2/2$ for $t > \tau_4$. Let $t > \tau_6 = \max\{\tau_4, \tau_5\}$, we have

$$H_{S}(t) = H(0) - H_{I}(t) - H_{R}(t) \geq H(0) - \epsilon_2,$$

or equivalently $H_{S}(t) - H(0) \geq -\epsilon_2$ with $\epsilon_2$ arbitrarily small, and this implies that $(H_5 - H(0))_{\infty} \geq 0$. We conclude by comparison and utilizing Lemma 4.2 that $0 \geq (H_5 - H(0))_{\infty} \geq (H_5 - H(0))_{\infty} \geq 0$, and so $\lim_{t \to \infty} H_{S}(t) = H_{S}(0)$.

Finally, since $M(t)$ (total size of mosquito population) is a solution of equation (3) we conclude that $\lim_{t \to +\infty} (M(t) - M_{S}(t)) = 0$ and

$$M_{S}(t) - \overline{M}_{S}(t) = M(t) - \overline{M}_{S}(t) - M_{E}(t) - M_{I}(t) \to 0$$

as $t \to \infty$, or equivalently $\lim_{t \to \infty} M_{S}(t) = \overline{M}_{S}(t)$. Therefore the DFS is globally attractive.

5. Numerical computation of $R_0$ and simulations

In this section we present a numerical algorithm and provide some numerical simulations to illustrate the results obtained in the propositions. A efficient numerical method for calculating $R_0$ for compartmental epidemiological models based on the fundamental formula (16) was propoused by Posny and Wang [50]. Assuming that the kernel of $L$ is a periodic function of $t$ of period $\omega$, they discretize the integral operator using the trapezoidal rule. Some characteristic details of the method are the following:

Operator (16) and $\omega$-periodicity of $\phi$ implies

$$(L\phi)(t) = \int_{0}^{\omega} U(t,s)\phi(t-s)ds,$$

where

$$U(t,s) = \sum_{j=0}^{\infty} Y(t,s-j\omega)F(t-s-j\omega) = \left(\sum_{j=0}^{\infty} Y(t,t-s-j\omega)\right)F(t-s).$$

According to standard theory of linear periodic systems [28], there exists a $C > 0$ and $k > 0$ such that

$$\|Y(t,s)\| \leq Ce^{-k(t-s)}, \forall t \geq s \text{ with } s \in \mathbb{R}.$$ 

It then follows that

$$\|Y(t,t-s-j\omega)F(t-s)\| \leq C\|F(t-s)\|e^{-k(s+j\omega)}, \forall t \in \mathbb{R}, s \geq 0 \text{ and } j = 0,1,2,\ldots$$
Hence, $U(t, s)$ may approximate by a finite sum:

$$U(t, s) \approx \left( \sum_{j=0}^{d} Y(s, t - s - j\omega) \right) \mathcal{F}(t - s)$$

for some integer $d > 0$, owing to the exponential decay of the terms in the summation. In this model

$$U(t, s) = \sum_{j=0}^{\infty} \left[ \begin{array}{c} 0 \\ q b \exp (s_0 (s + \omega j)) - q b \exp (s_2 (s + j\omega)) \frac{M_S(t - s)}{H} \\ \cdot \\ \cdot \\ \cdot \\ p b \exp (s_2 (s + j\omega)) \frac{M_S(t - s)}{H} \\ \cdot \\ \cdot \\ \cdot \\ 0 \\ 0 \end{array} \right]$$

The numerical method transforms the integral operator eigenvalue problem into a matrix eigenvalue problem of the form

$$\omega_n \hat{A} \hat{\phi} = \lambda \hat{\phi},$$

where $n$ is the number of nodes that uniformly partition the interval $[0, \omega]$, $\hat{\phi} = [\phi(t_0) \phi(t_1) \cdots \phi(t_{n-1})]^T$ is a $(n\tilde{m}) \times 1$ vector, and

$$\hat{A} = \left[ \hat{A}_{ij} = \frac{1}{2} (1 + \text{sgn}(i - j)) U(t_{i-1}, t_{i-1}) + \frac{1}{2} (1 - \text{sgn}(i - j)) U(t_{i-1}, t_{i+i-1}) \right]_{1 \leq i,j \leq n}$$

is a $(n\tilde{m}) \times (n\tilde{m})$ matrix. Hence,

$$R_0 \approx \frac{\omega}{n} \rho(\hat{A})$$

**Remark 5.1.** Let us understand qualitatively the term $\frac{M_S(t - s)}{H}$, $t \geq s$, in matrix (28). This expresses seasonal variations of the so-called “vector density”, defined as average number of vectors (female mosquitoes) per one human host [20]. Due to the cyclical pattern of mosquito population density, in winter the vector density drops to very low levels, below the $R_0 = 1$ threshold for transmission; followed by winter and preceding summer, the vector density begins to increase until it reaches a critical level at which the threshold crosses $R_0 = 1$, and transmission begins. Control campaigns have been mainly interested in reducing this important ratio (through larval control measures and elimination of breeding sites), in order to set vector densities below the threshold of epidemic transmission.

**Remark 5.2.** It is possible to show that $R_0$ for the periodic environment converges to the standard basic reproduction number for the time-averaged non-autonomous epidemic system, that is, the one in which the parameters in system (1) are replaced by their long-time averages.

Lemma 3.2 implies that

$$N_l \leq \liminf_{t \to \infty} M_S(t) \leq \limsup_{t \to \infty} M_S(t) \leq N_u,$$
where we can choose

\[ N_l = \left( \frac{\Delta}{m} \right)_\infty \quad \text{and} \quad N_u = \left( \frac{\Delta}{m} \right)_\infty. \]

Since the time-averaged non-autonomous epidemic system has a free-dengue equilibrium point, \( \tilde{x}_0 = [H \quad 0 \quad 0 \quad \bar{M}_s \quad 0 \quad 0]^T \), one has in that model that \( N_u = N_l \), and then \( M_s = \langle \Delta(t) \rangle / m \).

Furthermore, using the formula of the geometric series, the matrix function (28) converges to

\[
U(t, s) = \sum_{j=0}^{\infty} \begin{bmatrix}
0 & qb \exp \left( s_0 (s + \omega j) \right) & 0 \\

\frac{pb \exp \left( s_1 (s + j \omega) \right) - \exp \left( s_2 (s + j \omega) \right)}{H} & 0 & 0 \\

\frac{pb \exp \left( s_2 (s + j \omega) \right)}{H} & 0 & 0
\end{bmatrix}
\]

\[
= \begin{bmatrix}
0 & \frac{qb \sum_{j=0}^{\infty} \exp \left( s_0 (s + \omega j) \right)}{H} & 0 \\

\frac{pb \bar{M}_s (t-s)}{H} \sum_{j=0}^{\infty} \exp \left( s_1 (s + j \omega) \right) - \exp \left( s_2 (s + j \omega) \right) & 0 & 0 \\

\frac{pb \bar{M}_s (t-s)}{H} \sum_{j=0}^{\infty} \exp \left( s_2 (s + j \omega) \right) & 0 & 0
\end{bmatrix}
\]

\[
= \begin{bmatrix}
0 & \frac{qb \exp \left( s_0 s \right)}{1 - \exp^\omega (\omega s_0)} & 0 \\

\frac{pb \bar{M}_s (t-s)}{H} \left( \frac{\exp \left( s_1 s \right) - \exp \left( s_2 s \right)}{1 - \exp^\omega (\omega s_1)} \right) & 0 & 0 \\

\frac{pb \bar{M}_s (t-s)}{H} \left( \frac{\exp \left( s_2 s \right)}{1 - \exp^\omega (\omega s_2)} \right) & 0 & 0
\end{bmatrix}
\]

(31)

Under positivity of the matrix function \( U(t, s) \), there exists a unique real number \( R_0 \) such that there exists a nonnegative, nonzero, continuous and \( \omega \)-periodic vector function \( \mathbf{v}(t) \in C_\omega \), such that

\[
\int_0^\omega U(t, s) \mathbf{v}(t-s) ds = R_0 \mathbf{v}(t).
\]

Notice that if \( \bar{M}_s(t) \) is a constant \( \bar{M}_s \), then \( U(t, s) \) does not depend on \( t \), i.e. \( U(t-s) = U(s) \). In this case, considering a constant function \( \mathbf{v}(t) \) equal to a nonnegative eigenvector of the nonnegative matrix \( \int_0^\omega U(s) ds \), we see that \( R_0 \) is the spectral radius of this matrix, which is generally called the next-generation...
matrix ([15], p. 74). More precisely, we get

\[
R_0 = \left| \left( \int_0^\omega \left( \frac{qb \exp(s_0 s)}{1 - \exp(\omega s_0)} \right) ds \right) \int_0^\omega \left( \frac{p b M_S}{H} \left( \frac{\exp(s_1 s)}{1 - \exp(\omega s_1)} - \frac{\exp(s_2 s)}{1 - \exp(\omega s_2)} \right) \right) \right|^{1/2}
\]

\[
= \left| \left( \frac{qb \exp(s_0 s)}{s_0 (1 - \exp(\omega s_0))} \right) \left( \frac{p b M_S}{H} \left( \frac{\exp(s_1 s)}{s_1 (1 - \exp(\omega s_1))} - \frac{\exp(s_2 s)}{s_2 (1 - \exp(\omega s_2))} \right) \right) \right|^{1/2}
\]

\[
= \left| \left( - \frac{qb}{h + r} \right) \left( \frac{p b M_S}{H} \left( \frac{1}{m} - \frac{1}{c + m} \right) \right) \right|^{1/2}
\]

\[
= \sqrt{R_H R_M},
\]

where

\[
R_H = \frac{b p}{(h + r)H}
\]

(32)

and

\[
R_M = \frac{b q c}{m (c + m)} \frac{\langle \Delta(t) \rangle}{m}.
\]

(33)

To avoid misunderstanding, let us recall that some authors call \( R_0 \) what appears here as \( R_2^0 \). This point is discussed briefly in ([31], sec. 2.1). Our definition of \( R_0 \) is consistent with that given for autonomous compartmental epidemic models.

**Remark 5.3.** The factor (32) is the number of humans infected by a carrier vector during its period of portability of the dengue virus strain in a population of only susceptibles, and the factor (33) is the number of vectors that become carriers by biting an infected human in the vector population, all of them being non-carriers. Suppose that an infectious mosquito is introduced into a completely susceptible, non-carrier population of humans and mosquitoes, respectively. This infectious carrier mosquito bites an average number of \( (qb/m) \) \( \langle \Delta(t) \rangle/m \) susceptible humans during the infectious period of the vector; \( q b/m \) is the number of bites per mosquito. Afterwards, these humans are bitten on average by \( pb/(h + r)H \) non-carrier mosquitoes during the infectious period \( 1/(h + r) \). Finally, the probability that these non-infectious carrier mosquitoes survive the extrinsic incubation period and become infectious carrier mosquitoes is given by \( c/(c + m) \). The introduction of an infectious human follows a similar interpretation. Therefore, \( R_2^0 = R_H R_M \) is the average
number of secondary infected humans/mosquitoes produced by a typical infectious human or mosquito introduced into a dengue-free human and mosquito ecosystem, and determines whether dengue disappears or persists in the population [16].

The ordinary differential equations (1) are integrated utilizing MATLAB’s inbuilt routine ode45, considering the parameter values and initial conditions provided in Table 1.

Table 1. Parameters and initial data described in the model and their ranges of possible values.

| Parameter | Value | Range | Source | Dimensions |
|-----------|-------|-------|--------|------------|
| m         | See text | [1/20, 1/4] | [21, 17] day^{-1} | |
| r         | 1/7   | [0.07, 0.25] | [42, 29] day^{-1} | |
| b         | 1/3   | [0.3, 1]   | [49, 23] day^{-1} | |
| p         | 0.51  | [0.5, 1]   | [42, 54] Dimensionless | |
| q         | 0.40  | [0.1, 1]   | [3, 9] Dimensionless | |
| c         | 0.10  | [0.08, 0.13] | [42] day^{-1} | |
| H0        | 90008 | 0     | 200704 | 599467 | 0 | 33 | 5.0 x 10^{-7} |
| IC1       | 90005 | 4     | 200703 | 599468 | 32 | 0  | 1.0 x 10^{-4} |
| IC2       | 90010 | 3     | 200703 | 599469 | 0  | 31 | 1.5 x 10^{-4} |
| IC3       | 90013 | 3     | 200696 | 599470 | 30 | 0  | 2.0 x 10^{-4} |

For all numerical simulations, seasonality is represented through a periodic vector recruitment rate with a yearly period [53]:

\[ \Delta(t) = \delta \left( 1 + \varepsilon \cos(\omega t + \psi) \right) \tag{34} \]

where \( \omega = \frac{2\pi}{365} \) corresponds to a period of one year, \( \delta \) represents the average vector recruitment rate, \( \psi \) denote the phase shift and \( \varepsilon \) defines the amplitude of the seasonal variations (degree of periodic forcing). To ensure the positivity of \( \Delta(t) \), it is required that \( 0 < \varepsilon < 1 \).

We set \( d = 100 \) and \( n = 1000 \) in the numerical evaluation of the next infection operator, generate a \( 3n \times 3n \) matrix \( \hat{A} \) in the form of (29), determine its spectral radius and get a reasonable approximation of \( R_0 \) by (30). In figure 2, we plot \( R_0 \) when the parameter \( m \) is variable and the other parameters remain fixed. Consistent with the biological interpretation of \( R_0 \), \( m \) is inversely proportional to \( m \), the graph of \( R_0 \) versus \( m \) is seen as a convex equilateral hyperbola in the first quadrant. In particular, \( R_0 \approx 0.8545 \) if \( m = 1/10 \), \( R_0 \approx 1 \) if \( m \approx 1/11.382 \) and \( R_0 \approx 1.3345 \) if \( m = 1/14.5 \). Thus, whenever the mortality rate is smaller than \( 1/11.382 \) mosquitoes per day, dengue persists in the community.
The numerical results in figures 3 and 4 show us four solutions of the system (1) when $R_0 < 1$ and $R_0 > 1$, respectively. The planes on the left-hand side in each figure illustrate the evolution of disease states in humans and mosquitoes during a calendar year, while in the planes on the right-hand side we have extended the time to more than a year in the simulations to numerically demonstrate propositions 3.4 and 4.6. When $R_0 < 1$, the effect of seasonal variation can be described as follows: in the human population, outbreaks occur in less than two weeks, shortly after the disease is introduced into the community, then the number of infected people decreases almost exponentially and the disease disappears, similar to the behavior observed in mosquito populations carriers; meanwhile, the rate of variation in both the susceptible population (which grows) and the recovered population (which decreases) is slow, being necessary to extend the time scale to appreciate an asymptotic behavior of the trajectories of these population; in relation to the densities of mosquito populations that make transmission possible, they are higher in the first season of the year that covers the first three months with the highest incidence of dengue in humans, but after this season their sizes become too small to sustain outbreaks of dengue. See Figure 3a-b.

When $R_0 > 1$, the effect of seasonal variation can be described as follows: the highest annual daily number of dengue cases occurs around 200 days, and after the peak and before 500 days, a daily case is not exceeded; the average number of susceptible people grows monotonously from the beginning of the outbreak, until several years later growth stops and over time oscillations between 100 thousand and 250 thousand continue; the average number of people recovered decreases monotonically from the beginning of the outbreak, until several years later stops continuously decreasing and describes oscillations between 150 thousand and 200 thousand. We notice in an annual cycle that human outbreaks are in phase with the abundance of carrier mosquitoes, exhibit a lag with the bottleneck of non-carrier mosquitoes, and transmission is unfavorable when the non-carrier vector population begins to decline. See Figure 4a-b.
The numerical results in Figure 5 show solutions of the initial value problem (1) if $R_0 < 1$ and $R_0 > 1$, respectively, when $g$ varies. Eventually, the percent increase in VT generates the reemergence of dengue and flocks of non-infectious carrier mosquitoes in the environment, even in a situation of extinction of the disease ($R_0 < 1$), which implies endemicity despite seasonal variations (see Subfigure 5a). If $R_0 > 1$, the increase in the average number of viable female eggs infected vertically induces a decrease in the densities of mosquitoes and humans without the virus within their organism and magnifies the levels of non-carrier mosquitoes; successively lower values of $g$ produce a delay between progressively higher peaks in cases of dengue and the periods of the epidemic outbreak are shortened (see Subfigure 5a).

6. Conclusion

This model for dengue is based on a deterministic non-autonomous compartmental model of dengue transmission that incorporate transovarial transmission and a seasonal recruitment rate of mosquito population depending explicitly on the time variable and defined by a periodic function, in order to emulate these diverse seasonal oscillations in vector density and dengue overwintering. Utilizing the standard methodology applicable to non-autonomous epidemiological models we were able to determine well-posedness, existence of a dengue-free periodic solution and the basic reproduction number associated to the model.

It was further shown that the disease-free solution is globally asymptotically stable if the basic reproduction number of the model is less than one. The epidemiological implication of this result is that the disease can be effectively controlled if the control strategies implemented in the community can bring (and maintain) the reproduction number to a value less than one. In other words, this result shows that bringing (and maintaining) the reproduction number to a value less than one is necessary and sufficient for the effective control of the disease in the community. Simulation analysis was performed about endemic and epidemic dynamics, confirming that the disease completely dies out if $R_0 < 1$ and persists if $R_0 > 1$.

Likewise, the horizontal transmission of dengue between humans and mosquitoes is a determining factor in the epidemiology of this disease, but it has also been shown that *Aedes aegypti* is capable of transmitting the dengue virus to the progeny after it has been invaded by the virus [60, 24], suggesting that vertical transmission is an important mechanism of sustained virus circulation in vector populations during adverse periods for horizontal transmission. The consequence of the vertical transmission process is that carrier vectors continue to emerge (during favorable habitat conditions) even when there are no infected hosts, since the infected eggs can survive the dry season and re-emerge as carrier adult mosquitoes. This fact was corroborated numerically, where the incidence of vertical transmission suddenly increases the endemic amount of infectious
vectors and humans, which favors the persistence of the virus in the areas with enough breeding-sites and permanent pupal productivity.

Figure 3. Trajectories when $R_0 < 1$, $g = 0$ and initial conditions: IC1, IC2, IC3, and IC4 (see Table 1). The solutions converges to the dengue-free solution $\ast$. 

Revista Colombiana de Matemáticas
Figure 4. Trajectories when $R_0 > 1$, $g = 0$ and initial conditions: IC1, IC2, IC3, IC4 (see Table 1). The long-term behaviors illustrate that the disease is endemic.
Figure 5. Solution curves of the system (1) subject to IC1 (see Table 1) when $R_0 \neq 1$ with $g = 0.0 \square, g = 5.0 \times 10^{-7}$, $g = 1.0 \times 10^{-6}$, $g = 1.5 \times 10^{-6}$ and $g = 2.0 \times 10^{-6}$ .
Acknowledgment. The authors would like to thank the anonymous reviewer for his (her) careful reading of our manuscript and for his (her) comments and suggestions which improved the presentation of the results.

References

[1] B. Adams and M. Boots, How important is vertical transmission in mosquitoes for the persistence of dengue? Insights from a mathematical model, Epidemics (2010).

[2] S. Altizer, A. Dobson, and P. Hosseini et al, Seasonality and the dynamics of infectious diseases, Ecology letters (2006).

[3] M. Andraud, N. Hens, C. Marais, and P. Beutels, Dynamic epidemiological models for dengue transmission: a systematic review of structural approaches, PloS one (2012).

[4] B. Angel and V. Joshi, Distribution and seasonality of vertically transmitted dengue viruses in Aedes mosquitoes in arid and semi-arid areas of rajasthan, india, Journal of vector borne diseases (2008).

[5] N. Arunachalam, S. C. Tewari, and V. Thenmozhi et al, Natural vertical transmission of dengue viruses by Aedes aegypti in Chennai, Tamil Nadu, India, Indian Journal of Medical Research (2008).

[6] N. Bacaër, Genealogy with seasonality, the basic reproduction number, and the influenza pandemic, Journal of Mathematical Biology (2011).

[7] N. Bacaër and E. H. Ait Dads, On the biological interpretation of a definition for the parameter R₀ in periodic population models, Journal of mathematical biology (2012).

[8] N. Bacaër and S. Guernaoui, The epidemic threshold of vector-borne diseases with seasonality, Journal of mathematical biology (2006).

[9] L. M. Bartley, C. A. Donnelly, and G. P. Garnett, The seasonal pattern of dengue in endemic areas: mathematical models of mechanisms, Transactions of the royal society of tropical medicine and hygiene (2002).

[10] T. A. Burton, Stability and periodic solutions of ordinary and functional differential equations, Academic Press, New York, 1985.

[11] M. Canals, C. González, and A. Canals et al, Dinámica epidemiológica del dengue en Isla de Pascua, Revista chilena de infectología 29 (2012), 388–394.
[12] F. A. B. Coutinhoa, M. N. Burattinia, and L. F. Lopez et al, *Threshold conditions for a non-autonomous epidemic system describing the population dynamics of dengue*, Bulletin of mathematical biology (2006).

[13] Departamento Administrativo Nacional de Estadística, *Proyecciones demográficas por municipio basadas en el censo del año 2005*, Colombia, DANE, 2010, Retrieved from https://www.dane.gov.co/index.php/estadisticas-por-tema/demografia-y-poblacion/proyecciones-de-poblacion.

[14] , *Proyecciones nacionales y departamentales de población 2005-2020*, Colombia, DANE, 2010, Retrieved from https://www.dane.gov.co/index.php/estadisticas-por-tema/demografia-y-poblacion/proyecciones-de-poblacion.

[15] O. Diekmann and J. A. P. Heesterbeek, *Mathematical epidemiology of infectious diseases: model building, analysis and interpretation (Vol. 5)*, John Wiley & Sons, Chichester, 2000.

[16] O. Diekmann, J. A. P. Heesterbeek, and M. G. Roberts, *The construction of next-generation matrices for compartmental epidemic models*, Journal of the Royal Society Interface (2010).

[17] L. Esteva and C. Vargas, *Analysis of a dengue disease transmission model*, Mathematical biosciences (1998).

[18] , *Influence of vertical and mechanical transmission on the dynamics of dengue disease*, Mathematical biosciences (2000).

[19] E. Quispe et al, *Ciclo biológico y Tabla de Vida de Aedes aegypti, en laboratorio: Trujillo (Perú), 2014*, Revista REBIOLEST (2015).

[20] J. Liu-Helmersson et al., *Climate change and Aedes vectors: 21st century projections for dengue transmission in Europe*, EBioMedicine (2016).

[21] Z. Feng and J. X. Velasco-Hernández, *Competitive exclusion in a vector-host model for the dengue fever*, Journal of mathematical biology (1997).

[22] H. O. Florentino, D. R. Cantane, and F. L. Santos et al, *Multiobjective genetic algorithm applied to dengue control*, Mathematical biosciences 258 (2014), 77–84.

[23] D. A. Focks, R. J. Brenner, J. Hayes, and E. Daniels, *Transmission thresholds for dengue in terms of Aedes aegypti pupae per person with discussion of their utility in source reduction efforts*, American Journal of Tropical Medical Hygiene (2014).
[24] F. Fouque, R. Carinci, P. Gaborit, J. Issaly, D. J. Bicout, and P. Sabatier, *Aedes aegypti survival and dengue transmission patterns in French Guiana*, Journal of Vector Ecology (2006).

[25] V. Frantchez, R. Fornelli, and G. P. Sartori et al, *Dengue en adultos: diagnóstico, tratamiento y abordaje de situaciones especiales*, Revista Médica del Uruguay 32 (2016), 43–51.

[26] K. L. Gage, T. R. Burkot, and R. J. Eisen et al, *Climate and vectorborne diseases*, American journal of preventive medicine (2008).

[27] M. G. Guzman, S. B. Halstead, and H. Artsob et al, *Dengue: a continuing global threat*, Nature Reviews Microbiology (2010).

[28] J. K. Hale, *Ordinary Differential Equations*, Malabar, FL, USA: Robert E. Krieger Publishing Company, Inc, 1980.

[29] S. B. Halstead, *Dengue*, The lancet (2007).

[30] P. Hartman, *Ordinary Differential Equations*, John Wiley and Sons, Inc., New York, 1964.

[31] J. A. P. Heesterbeek and M. G. Roberts, *Threshold quantities for infectious diseases in periodic environments*, Journal of biological systems (1995).

[32] P. Hess, *Periodic-parabolic boundary value problems and positivity*, Pitman Res. Notes Math. Ser., vol. 247, Longman Scientific and Technical, Harlow, 1991.

[33] ———, *H. L. Smith and P. Waltman*, The theory of the chemostat: dynamics of microbial competition (Vol. 13), Cambridge university press, 1995.

[34] W. M. Hirsch, H. Hanisch, and J. P. Gabriel, *Differential equation models of some parasitic infections: methods for the study of asymptotic behavior*, Communications on Pure and Applied Mathematics (1985).

[35] H. Inaba, *On a new perspective of the basic reproduction number in heterogeneous environments*, Journal of mathematical biology (2012).

[36] V. Joshi, D. T. Mourya, and R. C. Sharma, *Persistence of dengue-3 virus through transovarial transmission passage in successive generations of Aedes aegypti mosquitoes*, The American journal of tropical medicine and hygiene (2002).

[37] T. Kato, *Perturbation theory for linear operators (Vol. 132)*, Springer Science & Business Media, 2013.
[38] H. K. Khalil and J. W. Grizzle, *Nonlinear systems (Vol. 3)*, Upper Saddle River, NJ: Prentice hall, 2002.

[39] C. Y. Kow, L. L. Koon, and P. F. Yin, *Detection of dengue viruses in field caught male Aedes aegypti and Aedes albopictus (Diptera: Culicidae) in Singapore by type-specific PCR*, Journal of medical entomology (2001).

[40] V. Lakshmikantham, S. Leela, and A. A. Martynyuk, *Stability analysis of nonlinear systems*, Marcel Dekker Inc., New York, Basel, 1989.

[41] J. Liu-Helmersson, H. Stenlund, and A. Wilder-Smith et al, *Vectorial capacity of Aedes aegypti: effects of temperature and implications for global dengue epidemic potential*, Addison-Wesley Publishing Company, PLoS one 9 (2014), e89783.

[42] D. P. Lizarralde-Bejarano, S. Arboleda-Sánchez, and M. E. Puerta-Yepes, *Understanding epidemics from mathematical models: Details of the 2010 dengue epidemic in Bello (Antioquia, Colombia)*, Applied Mathematical Modelling (2017).

[43] E. Martínez, *Dengue*, Estudos avançados 22 (2008), 33–52.

[44] C. J. Mitchell and B. R. Miller, *Vertical transmission of dengue viruses by strains of Aedes albopictus recently introduced into brazil*, Journal of the American Mosquito Control Association (1990).

[45] M. S. Mustafa, V. Rasotgi, and S. Jain et al, *Discovery of fifth serotype of dengue virus (DENV-5): A new public health dilemma in dengue control*, Medical Journal Armed Forces India 71 (2015), 67–70.

[46] Y. Nakata and T. Kuniya, *Global dynamics of a class of SEIRS epidemic models in a periodic environment*, J. Math. Anal. Appl. (2010).

[47] E. A. Newton and P. Reiter, *A model of the transmission of dengue fever with an evaluation of the impact of ultra-low volume (ULV) insecticide applications on dengue epidemics*, The American Journal of Tropical Medicine and Hygiene (1992).

[48] World Health Organization, *Dengue hemorrhagic fever: diagnosis, treatment, prevention and control*, 2nd edition, Geneva, Switzerland: WHO, 1997.

[49] S. T. R. D. Pinho, C. P. Ferreira, L. Esteva, F. R. Barreto, V. C. Morato e Silva, and M. G. L. Teixeira, *Modelling the dynamics of dengue real epidemics*, Philosophical Transactions of the Royal Society A: Mathematical, Physical and Engineering Sciences (2010).
[50] D. Posny and J. Wang, Computing the basic reproductive numbers for epidemiological models in nonhomogeneous environments, Applied Mathematics and Computation (2014).

[51] J. P. Tian and J. Wang, Some results in Floquet theory, with application to periodic epidemic models, Applicable Analysis (2015).

[52] J. G. Rigau-Pérez, G. G. Clark, and D. J. Gubler et al, Dengue and dengue haemorrhagic fever, The Lancet (1998).

[53] H. S. Rodrigues, M. T. T. Monteiro, and D. F. Torres, Seasonality effects on dengue: basic reproduction number, sensitivity analysis and optimal control, Mathematical Methods in the Applied Sciences (2016).

[54] T. Sardar, S. Rana, and J. Chattopadhyay, A mathematical model of dengue transmission with memory, Communications in Nonlinear Science and Numerical Simulation (2015).

[55] H. R. Thieme, Spectral bound and reproduction number for infinite-dimensional population structure and time heterogeneity, SIAM Journal on Applied Mathematics (2009).

[56] P. van den Driessche, Reproduction numbers of infectious disease models, Infectious Disease Modelling (2017).

[57] P. van den Driessche and J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, Mathematical biosciences (2002).

[58] W. Wang and X. Q. Zhao, Threshold dynamics for compartmental epidemic models in periodic environments, Journal of Dynamics and Differential Equations (2008).

[59] X. Wang and X. Q. Zhao, Dynamics of a time-delayed Lyme disease model with seasonality, SIAM Journal on Applied Dynamical Systems (2017).

[60] L. Wasinpiyamongkol, S. Thongrungkiat, and N. Jirakanjanakit et al, Susceptibility and transovarial transmission of dengue virus in Aedes aegypti: a preliminary study of morphological variations, Southeast Asian journal of tropical medicine and public health (2003).

(Recibido en diciembre de 2020. Aceptado en marzo de 2021)
Grupo de Modelación Matemática en Epidemiología (GMME)
Universidad del Quindío
Facultad de Ciencias Básicas y Tecnologías
Carrera 15 Calle 12 Norte
Armenia (Quindío), Colombia

e-mail: jaolarteg@uqvirtual.edu.co
e-mail: anibalml@hotmail.com