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Effect of pulse vaccination on dynamics of dengue with periodic transmission functions

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Abstract

Pulse vaccination is a repeated vaccination policy, which plays a tremendous role in the global fight against communicable diseases in terms of saving medical resources and decreasing the economic burden. In this article, we propose a dynamic model of dengue infection with periodic transmission functions and seasonality in vector population. Furthermore, we introduce a pulse vaccination strategy in the susceptible host population to examine how frequency and intensity of implementation of this strategy affect the dynamics of dengue infection. We successfully obtained the threshold dynamics by defining the basic reproduction number R_0, which is the spectral radius of the next generation operator and governs whether the disease dies out or not. It has been established that the infection-free periodic solution of the proposed impulsive system is globally asymptotically stable if $R_0 < 1$ and is unstable otherwise. Moreover, we found that the dengue infection is uniformly persistent for the proposed system if $R_0 > 1$. Finally, we execute the system numerically to illustrate the piecewise solutions of the proposed system with impulsive vaccination measure and to investigate the influence of different control parameters on the basic reproduction. The finding indicates that a frequent implementation of the vaccination strategy with great intensity and the use of mosquito nets can essentially lead to a decline of new infections.

Keywords: Dengue fever; Periodic transmission; Impulsive vaccination; Asymptotical stability; Uniform persistence; Numerical simulations

1 Introduction

It is evident that vector-borne diseases are induced by pathogenic agents such as helminths, viruses, protozoa, and bacteria. They are transmitted by arthropod vectors, which include bedbugs, biting midges, fleas, lice, kissing bugs, mosquitoes, ticks, sand flies, mites and black flies. It is a fact that dengue infection spreads more rapidly than other vector-borne infections, which causes significant health, economic and social burdens [1, 2]. The symptoms of this viral infection are high fever, red eyes, joints pain, muscles pain, lower back pain, pain behind eyes, nausea, and vomiting, a red flush to the face, severe fatigue, severe weakness and even death [3–8]. The *Aedes* mosquitoes transfer four different serotypes to the host through their bites when they suck the blood of the host. Humans may be infected with dengue virus more than once because it is caused by four different viruses which are carried by female *Aedes* mosquitoes [9]. Dengue fever is rarely fatal, but

more severe forms of dengue infections are DSS and DHF, causing 22,000 deaths annually [10]. Moreover, dengue virus is recognized as the major arbovirus on the globe, and each year produces more than 50 million infections [11, 12].

It is evident that vaccination plays an essential part in eliminating infectious diseases and is used as an effective method to eliminate the infection by reducing the strength of the susceptible group in the community [13–21]. The impact of impulsive vaccination on an epidemic model with regular constant vaccination was studied in [20], where a fraction of the susceptible class was partially transferred to the vaccination class. In [15], the author explored and conceptualized the role of vaccination in the epidemic model and further highlighted the influence of vaccination on the infection. Even though vaccination is the most powerful tool to eradicate and reduce the level of infection, however, most types of it are not fully effective and do not provide complete immunity to the infection. As a result, mathematical models with vaccination strategy provide vital information to decision makers at both global and national levels. Although several epidemic models have been introduced in previous research to conceptualize and explore the dynamics of dengue infection to detect the leading factors that significantly affect the transmission of the infection [22–32], few have focused on modeling the effect of vaccination on dengue dynamics and this falls within the scope of this research.

A common assumption for the infectious model with vaccination strategy is that the immunization activities happen continuously. However, this is not how things look like. Generally, the immunization happens in regular intervals, and consequently, the continuous human's intervention measure is then removed from the system, and replaced by an impulsive perturbation. Thus, mathematical models with impulsive vaccination have been proposed and investigated [33–40]. In [38, 39], the authors inspected the delay epidemic models with the influence of impulsive vaccination, moreover, the stability of the linear impulsive system was investigated in [41]. Note that individuals, who have immunization to one dengue virus due to infection or vaccination, can also be infected by another dengue virus. So the interesting problem is how to design the vaccination regime (i.e., as regards the vaccination intensity and how frequently the vaccination should be implemented) to effectively control the dengue infection.

The target of this work is to initiate a dynamical model for dengue fever with periodic transmission functions and pulse vaccination. The preeminent motivation behind this research is to examine the level of disease after implementation of impulsive vaccination and to investigate how the impulsive policy influences the dynamics of dengue infection. This article is ordered as follows: in Sect. 2 of the article, we introduce the dynamics of dengue fever with periodic transmission functions and impulsive vaccination strategies. In Sect. 3, we reveal that the infection-free periodic solution is GAS for R_0 less than 1 and is unstable in other respects. Furthermore, the uniform persistence of dengue infection has been shown in the proposed impulsive system for $R_0 > 1$. In Sect. 4, we carry out some numerical simulation to elucidate the impulsive system to inspect the feasibility of the impulsive vaccination control strategy. Finally, we present endemic indicator in terms of different parameters of the system numerically.

2 Formulation of the model

To formulate the interaction of vector and human for dengue infection, we indicate the total female vector size by N_v and the total human size by N_h. The total female

vector size is categorized into susceptible (S_v) and infectious (I_v) states while the human population N_h is categorized into susceptible human (S_{h_1}), susceptible human with partial immunity (S_{h_2}), infected (I_h) and recovered (R_h) states. Moreover, it is assumed that negligible mortality is produced by dengue infection and the natural recruitment and death rates of vector and host are supposed to be μ_v and μ_h, respectively.

The flow of dengue infection from an infected mosquito to a susceptible human starts by the bite of infectious mosquitoes when it sucks the blood of a susceptible host. In both populations, the flow of dengue infection from susceptible state to infected one relies on the biting rate of mosquitoes, the number of susceptible and infectious members of both populations, and the transmission probabilities. In this formulation, b is for the per capita bitting rate of vectors and β_{h_1}, β_{h_2} and β_v represent the transmission probabilities from vector to susceptible humans and vice versa. In this formulation, the infection rates per humans and mosquitoes are described by ($\frac{b\beta_{h_1}}{N_h}I_v$), ($\frac{b\beta_{h_2}}{N_h}I_v$) and ($\frac{b\beta_v}{N_h}I_h$), respectively. We assume that a fraction υ of the recovered class lose immunity and become susceptible with less transmission probability.

The differential system that represents the flow of dengue infection with the upper mentioned suppositions is specified as

$$
\begin{cases}
\frac{dS_{h_1}}{dt} = \mu_h N_h - \frac{\beta_{h_1}b}{N_h}S_{h_1}I_v - \mu_h S_{h_1}, \\
\frac{dS_{h_2}}{dt} = -\frac{\beta_{h_2}b}{N_h}S_{h_2}I_v - \mu_h S_{h_2} + \upsilon R_h, \\
\frac{dI_h}{dt} = \frac{\beta_{h_1}b}{N_h}S_{h_1}I_v + \frac{\beta_{h_2}b}{N_h}S_{h_2}I_v - (\mu_h + \gamma_h)I_h, \\
\frac{dR_h}{dt} = \gamma_h I_h - (\upsilon + \mu_h)R_h, \\
\frac{dS_v}{dt} = \mu_v N_v - \frac{\beta_v b}{N_h}S_v I_h - \mu_v S_v, \\
\frac{dI_v}{dt} = \frac{\beta_v b}{N_h}S_v I_h - \mu_v I_v,
\end{cases}
\tag{1}
$$

with appropriate non-negative initial condition, moreover, the total population size of vector and host are

$$
N_v = S_v + I_v, \qquad N_h = S_{h_1} + S_{h_2} + I_h + R_h.
\tag{2}
$$

It is well known that there exist periodic outbreaks of dengue infection and fluctuation occurs in the density of the vector population because of environmental factors such as humidity and temperature. Therefore, we introduce periodic transmission rates and seasonality in the vector population to represent more realistically the phenomena of dengue infection. We denote the periodic transmission rates by $\beta_{h_1}(t)$, $\beta_{h_2}(t)$ and $\beta_v(t)$, and we indicate the periodic recruitment rate and natural death rate of mosquitoes by $A(t)$ and $\mu_v(t)$. Note that here the seasonality in the death rate of mosquitoes is due to the phenology feature in vectors which brings fluctuation in the death rate with time [42]. Further, we introduce an impulsive vaccination mechanism of control and prevention for dengue infection, where vaccination is applied to a fraction q of host population S_{h_1}, these susceptible hosts shift to the recovery class R_h after successful vaccination. Then the system of equations (1) with the above assumptions takes the

form

$$
\left.
\begin{aligned}
\frac{dS_{h_1}}{dt} &= \mu_h N_h - \frac{b\beta_{h_1}(t)}{N_h}S_{h_1}I_v - \mu_h S_{h_1}, \\
\frac{dS_{h_2}}{dt} &= -\frac{b\beta_{h_2}(t)}{N_h}S_{h_2}I_v - \mu_h S_{h_2} + \upsilon R_h, \\
\frac{dI_h}{dt} &= \frac{b\beta_{h_1}(t)}{N_h}S_{h_1}I_v + \frac{b\beta_{h_2}(t)}{N_h}S_{h_2}I_v - (\mu_h + \gamma_h)I_h, \\
\frac{dR_h}{dt} &= \gamma_h I_h - (\upsilon + \mu_h)R_h, \\
\frac{dS_v}{dt} &= A(t) - \frac{b\beta_v(t)}{N_h}S_v I_h - \mu_v(t)S_v, \\
\frac{dI_v}{dt} &= \frac{b\beta_v(t)}{N_h}S_v I_h - \mu_v(t)I_v,
\end{aligned}
\right\} \quad t \ne \mathrm{nT}, \mathrm{n} \in \mathrm{N},
$$

$$
\left.
\begin{aligned}
S_{h_1}(t^+) &= (1-q)S_{h_1}(t), \\
S_{h_2}(t^+) &= S_{h_2}(t), \\
I_h(t^+) &= I_h(t), \\
R_h(t^+) &= R_h(t) + qS_{h_1}(t), \\
S_v(t^+) &= S_v(t), \\
I_v(t^+) &= I_v(t),
\end{aligned}
\right\} \quad t = \mathrm{nT}, \mathrm{n} \in \mathrm{N}. \tag{3}
$$

with non-negative initial condition $S_{h_1}(0) = S_{h_10}$, $S_{h_2}(0) = S_{h_20}$, $I_h(0) = I_{h0}$, $R_h(0) = R_{h0}$, $S_v(0) = S_{v0}$ and $I_v(0) = I_{v0}$. The above impulsive dynamical model (3) implies

$$
\frac{dN_v(t)}{dt} = A(t) - \mu_v(t)N_v(t), \tag{4}
$$

with non-negative initial condition $N_v(0) = N_{v0}$. System (4) has a unique positive T-periodic globally asymptotically stable solution given by

$$
\widetilde{N}_v(t) = \widetilde{N}_v(0)e^{-\int_0^t \mu_v(\rho)\,d\rho} + e^{-\int_0^t \mu_v(\rho)\,d\rho}\int_0^t A(\rho)e^{\int_0^\rho \mu_v(\tau)\,d\tau}\,d\rho,
$$

with

$$
\widetilde{N}_v(0) = \frac{\int_0^T A(\rho)e^{\int_0^\rho \mu_v(\tau)\,d\tau}\,d\rho}{e^{\int_0^T \mu_v(\rho)\,d\rho} - 1}.
$$

In consequence of this, $N_v(t) \to \widetilde{N}_v(t)$ as t tends to infinity. Finally, the system of equations (3) with the assumptions of $\beta_1(t) = \frac{b\beta_{h_1}(t)}{N_h}$, $\beta_2(t) = \frac{b\beta_{h_2}(t)}{N_h}$ and $\beta_3(t) = \frac{b\beta_v(t)}{N_h}$, takes the form

$$
\left.
\begin{aligned}
\frac{dS_{h_1}}{dt} &= \mu_h N_h - \beta_1(t)S_{h_1}I_v - \mu_h S_{h_1}, \\
\frac{dS_{h_2}}{dt} &= -\beta_2(t)S_{h_2}I_v - \mu_h S_{h_2} + \upsilon(N_h - S_{h_1} - S_{h_2} - I_h), \\
\frac{dI_h}{dt} &= \beta_1(t)S_{h_1}I_v + \beta_2(t)S_{h_2}I_v - (\mu_h + \gamma_h)I_h, \\
\frac{dI_v}{dt} &= \beta_3(t)(\widetilde{N}_v(t) - I_v)I_h - \mu_v(t)I_v,
\end{aligned}
\right\} \quad t \ne \mathrm{nT}, \mathrm{n} \in \mathrm{N},
$$

$$
\left.
\begin{aligned}
S_{h_1}(t^+) &= (1-q)S_{h_1}(t), \\
S_{h_2}(t^+) &= S_{h_2}(t), \\
I_h(t^+) &= I_h(t), \\
I_v(t^+) &= I_v(t),
\end{aligned}
\right\} \quad t = \mathrm{nT}, \mathrm{n} \in \mathrm{N}, \tag{5}
$$

with non-negative initial condition $S_{h_1}(0) = S_{h_1 0}$, $S_{h_2}(0) = S_{h_2 0}$, $I_h(0) = I_{h0}$ and $I_v(0) = I_{v0}$.

To analyze the impulsive system (5), we take the general impulsive differential system

$$
\begin{aligned}
\frac{dy(t)}{dt} &= b - dy(t), \quad t \neq nT, n \in N, \\
y(t^+) &= (1 - \phi)y(t), \quad t = nT, n \in N,
\end{aligned}
\tag{6}
$$

where b, d are positive and $0 < \phi < 1$. Here, we present that the impulsive dynamical system (6) admits a positive periodic, and globally asymptotically stable solution.

Lemma 2.1 *The impulsive dynamical system* (6) *has a positive periodic globally asymptotically stable solution*

$$
\widetilde{y}(t) = \frac{b}{d} + \left(y^* - \frac{b}{d} \right) e^{-d(t-nT)}, \quad t \in \left(nT, (n+1)T \right],
$$

where

$$
y^* = \frac{b(1-\phi)(1-e^{-dT})}{d[1-(1-\phi)e^{-dT}]}.
$$

Proof To obtain the required result, we integrate the first equation of the dynamical system (6) at pulses, and we get

$$
\widetilde{y}(t) = \frac{b}{d} + \left(y(nT) - \frac{b}{d} \right) e^{-d(t-nT)}, \quad t \in \left(nT, (n+1)T \right],
\tag{7}
$$

where $y(nT)$ denotes the base value at nT. In the next step, we solve the last equation of the impulsive dynamical system (6) and obtain

$$
y\left((n+1)T\right) = (1-\phi) \left[\frac{b}{d} + \left(y(nT) - \frac{b}{d} \right) e^{-dT} \right] = g\left(y(nT)\right),
\tag{8}
$$

where $g(y) = (1-\phi)[\frac{b}{d} + (y - \frac{b}{d})e^{-dT}]$. Furthermore, it is effortless to show that (8) has a unique positive solution given by $y^* = [\frac{b(1-\phi)(1-e^{-dT})}{d(1-(1-\phi)e^{-dT})}]$, which satisfies the condition $y < g(y) < y^*$, if $0 < y < y^*$ and satisfies the condition $y^* < g(y) < y$ if $y > y^*$. Evidently, y^* is globally asymptotically stable for Eq. (8) by [43]. Consequently, we have a positive periodic globally asymptotically stable solution of the impulsive system (6), given by

$$
\widetilde{y}(t) = \frac{b}{d} + \left(y^* - \frac{b}{d} \right) e^{-d(t-nT)}, \quad t \in \left(nT, (n+1)T \right],
$$

with

$$
y^* = \frac{b(1-\phi)(1-e^{-dT})}{d[1-(1-\phi)e^{-dT}]}.
$$

This completes the proof. $\square$

Next, we present the following concept for the solution of a linear system of the form $y' = G(t)y$. Let $w_1, w_2 \in \mathbb{R}^n$, where $(\mathbb{R}^n, \mathbb{R}^n_+)$ is a standard order n-dimensional Euclidean space and $\| \cdot \|$ is the norm defined on $(\mathbb{R}^n, \mathbb{R}^n_+)$. Further, we take $w_1 \geq w_2$, if $w_1 - w_2 \in \mathbb{R}^n_+$; $w_1 > w_2$, if $w_1 - w_2 \in \mathbb{R}^n_+ \setminus 0$; and $w_1 \gg w_2$, if $w_1 - w_2 \in \text{Int}(\mathbb{R}^n_+)$. Here, we assume a matrix function $G(t)$ of order n, which is irreducible, cooperative, continuous and periodic with a positive period ω. Take the linear system $y' = G(t)y$ having a fundamental matrix solution $\Phi_{G(\cdot)}(t)$ and $r(\Phi_{G(\cdot)}(\omega))$ represents the highest eigenvalue of $\Phi_{G(\cdot)}(\omega)$ in magnitude. Then the Perron–Frobenius theorem implies that $r(\Phi_{G(\cdot)}(\omega))$ is the principal eigenvalue of the solution matrix $\Phi_{G(\cdot)}(\omega)$. The following lemma is introduced for the further analysis of our impulsive system.

Lemma 2.2 ([44, 45]) *Let* $\psi = \frac{1}{\omega} \ln r(\Phi_{G(\cdot)}(\omega))$. *Then the linear system* $y' = G(t)y$ *has a solution* $e^{\psi t}u(t)$, *where* $u(t)$ *is a* ω-*periodic and positive function.*

3 Threshold dynamics

The existence of the disease-free periodic solution of impulsive dynamical model (5) and its stability will be studied in this section of the article. Suppose both populations are free of infections, more specifically there is no infection in host and vector populations, that is, $I_h(t) = I_v(t) = 0$, then the impulsive system (5) becomes

$$
\begin{cases}
\left.\begin{array}{l}
\frac{dS_{h_1}}{dt} = \mu_h N_h - \mu_h S_{h_1}, \\
\frac{dS_{h_2}}{dt} = -\mu_h S_{h_2} + \upsilon(N_h - S_{h_1} - S_{h_2}),
\end{array}\right\} \quad t \neq \text{nT}, n \in \mathbb{N}, \\
\left.\begin{array}{l}
S_{h_1}(t^+) = (1-q)S_{h1}(t), \\
S_{h_2}(t^+) = S_{h_2}(t),
\end{array}\right\} \quad t = \text{nT}, n \in \mathbb{N},
\end{cases}
\tag{9}
$$

with proper non-negative initial conditions. The following result is concerned with the positive periodic solution of the disease-free system (9) and its global asymptotical stability.

Theorem 3.1 *The impulsive dynamical system* (9) *admits a positive periodic, and globally asymptotically stable solution.*

Proof To obtain the result, firstly we take the portion of S_{h_1} of the impulsive system (9), that is

$$
\begin{cases}
\frac{dS_{h_1}}{dt} = \mu_h N_h - \mu_h S_{h_1}, & t \neq \text{nT}, n \in \mathbb{N}, \\
S_{h_1}(t^+) = (1-q)S_{h_1}(t), & t = \text{nT}, n \in \mathbb{N}.
\end{cases}
\tag{10}
$$

Obviously, the impulsive system (10) admits a positive periodic globally asymptotically stable solution by Lemma 2.1, given by

$$
\widetilde{S}_{h_1}(t) = N_h + \left(S_{h_1}^* - N_h\right)e^{-\mu_h(t-\text{nT})}, \quad t \in \left(\text{nT}, (n+1)\text{T}\right],
\tag{11}
$$

with

$$
S_{h_1}^* = \frac{N_h(1-q)(1-e^{-\mu_h \text{T}})}{[1-(1-q)e^{-\mu_h \text{T}}]}.
$$

Secondly, we take the portion of S_{h_2} of the impulsive system (9), that is

$$\begin{cases} \frac{dS_{h_2}(t)}{dt} = \upsilon(N_h - \widetilde{S}_{h_1}(t)) - (\upsilon + \mu_h)S_{h_2}(t), & t \neq \mathrm{n}\mathrm{T}, \mathrm{n} \in \mathrm{N}, \\ S_{h_2}(t^+) = S_{h_2}(t), & t = \mathrm{n}\mathrm{T}, \mathrm{n} \in \mathrm{N}. \end{cases} \tag{12}$$

The above-mentioned system (12) also admits a positive periodic globally asymptotically stable solution, given by

$$\widetilde{S}_{h_2}(t) = e^{-(\upsilon+\mu_h)(t-\mathrm{n}\mathrm{T})}\left[S_{h_2}^* + \int_{\mathrm{n}\mathrm{T}}^{t} \upsilon\big(N_h - \widetilde{S}_{h_1}(\rho)\big)e^{(\upsilon+\mu_h)(t-\mathrm{n}\mathrm{T})}\, d\rho \right],$$
$$t \in \big(\mathrm{n}\mathrm{T}, (\mathrm{n}+1)\mathrm{T}\big], \tag{13}$$

with

$$S_{h_2}^* = \frac{\int_{\mathrm{n}\mathrm{T}}^{(\mathrm{n}+1)\mathrm{T}} \upsilon(N_h - \widetilde{S}_{h_1}(\rho))e^{(\upsilon+\mu_h)\rho}\, d\rho}{e^{(\upsilon+\mu_h)\mathrm{T}} - 1}.$$

As a result, the impulsive system (9) has a positive periodic globally asymptotically stable solution, i.e., $(\widetilde{S}_{h_1}(t), \widetilde{S}_{h_2}(t))$. This completes the proof. □

Theorem 3.1 ensures that the impulsive dynamical system (5) has a unique positive periodic disease-free solution $(\tilde{S}_{h_1}(t), \tilde{S}_{h_2}(t), 0, 0)$ on every impulsive interval. Next, to investigate the stability results of disease-free periodic solution of impulsive system (5), let us take the linear system $y' = G(t)y$, where $G(t)$ is a matrix of order n and assume that $\varPhi_{G(\cdot)}(t)$ is its fundamental solution. Also, the spectral radius of $\varPhi_{G(\cdot)}(\omega)$ is assumed to be $r(\varPhi_{G(\cdot)}(\omega))$, then the local stability result of the disease-free solution can be obtained by taking small-amplitude perturbations of the solutions. After small perturbations, the linearizing system of (5) at the infection-free periodic solution is

$$y'(t) = G(t)y(t), \quad t \neq \mathrm{n}\mathrm{T}, \mathrm{n} \in \mathrm{N},$$
$$y(t^+) = Hy(t), \quad t = \mathrm{n}\mathrm{T}, \mathrm{n} \in \mathrm{N}, \tag{14}$$

in this linear system, we have

$$G(t) = \begin{bmatrix} G_1 & G_2(t) \\ O & \mathrm{F}(t) - \mathrm{V}(t) \end{bmatrix}, \qquad H(t) = \begin{bmatrix} H_1 & 0 \\ 0 & I \end{bmatrix},$$

with

$$G_1 = \begin{bmatrix} -\mu_h & 0 \\ -\upsilon & -(\mu_h + \upsilon) \end{bmatrix}, \qquad G_2(t) = \begin{bmatrix} 0 & -\beta_1(t)\widetilde{S}_{h_1}(t) \\ -\upsilon & -\beta_2(t)\widetilde{S}_{h_2}(t) \end{bmatrix},$$

$$O = \begin{bmatrix} 0 & 0 \\ 0 & 0 \end{bmatrix}, \qquad H_1 = \begin{bmatrix} 1-q & 0 \\ 0 & 1 \end{bmatrix}, \qquad V(t) = \begin{bmatrix} (\mu_h + \gamma_h) & 0 \\ 0 & \mu_v(t) \end{bmatrix}, \quad \text{and}$$

$$\mathrm{F}(t) = \begin{bmatrix} 0 & \beta_1(t)\widetilde{S}_{h_1}(t) + \beta_2(t)\widetilde{S}_{h_2}(t) \\ \beta_3(t)\widetilde{N}_v(t) & 0 \end{bmatrix}.$$

Let $\Phi_G(t) = (\Phi_{ij})_{1 \le i,j \le 2}$ be the fundamental solution of linear system $y'(t) = G(t)y(t)$. Consequently, we have $\Phi'_G(t) = G(t)\Phi_G(t)$ with appropriate initial value $\Phi_G(0) = E_4$. Further computation implies that

$$\Phi_G(t) = \begin{bmatrix} e^{G_1 t} & \Phi_{12}(t) \\ 0 & \Phi_{F(t)-V(t)}(t) \end{bmatrix},$$

then we obtain

$$H\Phi_G(T) = \begin{bmatrix} H_1 e^{G_1 T} & H_1 \Phi_{12}(T) \\ 0 & \Phi_{(F(t)-V(t))}(T) \end{bmatrix}.$$

It can be noticed that $r(H_1 e^{G_1 T}) < 1$. Denote the basic reproduction number

$$R_0 \triangleq r\big(\Phi_{(F-V)(\cdot)(t)}(T)\big),$$

therefore, based on Floquet theory, we obtain the following conclusion.

Theorem 3.2 *The infection-free periodic solution of the impulsive dynamical system* (5) *is locally asymptotically stable if* R_0 *less than* 1, *and is unstable in other cases.*

Note that here R_0 works as a threshold (endemic indicator) for the impulsive system (5) such that, if R_0 is less than 1, then an infected individual generate less than an infected individual and the infection vanishes. In the case that R_0 greater than 1, this implies that an infected individual generates more infected individuals in the community [46].

Theorem 3.3 *The infection-free periodic solution of impulsive dynamical system* (5) *is globally asymptotically stable for* $R_0 < 1$.

Proof To obtain the result, we take the solution $\varphi(t,x) = (S_{h_1}(t), S_{h_2}(t), I_h(t), I_v(t))$ of the impulsive system (5), where $x \in R_+^4$. By the non-negativity of $I_h(t)$ and $I_v(t)$, we have

$$\begin{cases} \frac{dS_{h_1}}{dt} \le \mu_h N_h - \mu_h S_{h_1}, & t \ne nT, n \in N, \\ S_{h_1}(t^+) = (1-q)S_{h_1}(t), & t = nT, n \in N, \end{cases} \tag{15}$$

taking the auxiliary system

$$\begin{cases} \frac{dx_1}{dt} = \mu_h N_h - \mu_h x_1, & t \ne nT, n \in N, \\ x_1(t^+) = (1-q)x_1(t), & t = nT, n \in N. \end{cases} \tag{16}$$

Obviously, the impulsive system (16) admits a positive periodic globally asymptotically stable solution by Lemma 2.1, say $\widetilde{x}_1(t)$, that is, $x_1(t)$ converges to $\widetilde{x}_1(t)$, as t tends to infinity. Further, the comparison theorem of IDEs implies that $S_{h_1}(t) \le x_1(t)$, similarly we can justify that $S_{h_2}(t) \le x_2(t)$. Next, we define the solution $S(t) = (S_{h_1}(t), S_{h_2}(t))^T$ and $x(t) = (x_1(t), x_2(t))^T$ of the infection-free impulsive system (9), with $S(0) = x(0)$. It is clear that the τ-periodic solution is globally asymptotically stable for the infection-free system (9) by Theorem 3.1, say $\widetilde{S}(t) = (\widetilde{S}_{h_1}(t), \widetilde{S}_{h_2}(t))^T$. To be more specific, the solution

$x(t) \to \widetilde{S}(t)$ as t tends to infinity. We fix $\xi > 0$. Then we can find $t_1 > 0$ in such a manner that $x_1(t) \leq \widetilde{S}_{h_1}(t) + \xi$ and $x_2(t) \leq \widetilde{S}_{h_2}(t) + \xi$ for all $t \geq t_1$. Thus, for $t \geq t_1$, we have $S_{h_1}(t) \leq \widetilde{S}_{h_1}(t) + \xi$ and $S_{h_2}(t) \leq \widetilde{S}_{h_2}(t) + \xi$. By system (5), we have

$$\begin{cases} \frac{dI_h}{dt} \leq (\beta_1(t)(\widetilde{S}_{h_1}(t) + \xi) + \beta_2(t)(\widetilde{S}_{h_2}(t) + \xi))I_\nu - (\mu_h + \gamma_h)I_h, \\ \frac{dI_\nu}{dt} \leq \beta_3(t)\widetilde{N}_\nu(t)I_h - \mu_\nu(t)I_\nu. \end{cases} \tag{17}$$

Taking the auxiliary system

$$\begin{cases} \frac{dw_1}{dt} = (\beta_1(t)(\widetilde{S}_{h_1}(t) + \xi) + \beta_2(t)(\widetilde{S}_{h_2}(t) + \xi))w_2 - (\mu_h + \gamma_h)w_1, \\ \frac{dw_2}{dt} = \beta_3(t)\widetilde{N}_\nu(t)w_1 - \mu_\nu(t)w_2, \end{cases} \tag{18}$$

system (18) is converted into

$$\mathrm{w}' = \big(\mathrm{F}(t) - \mathrm{V}(t)\big)\mathrm{w} + \xi\, W(t)\mathrm{w}, \tag{19}$$

where w is a vector of the form $\mathrm{w} = (\mathrm{w}_1, \mathrm{w}_2)^{\mathrm{T}}$ and

$$W(t) = \begin{bmatrix} 0 & \beta_1(t) + \beta_2(t) \\ 0 & 0 \end{bmatrix}.$$

Based on Lemma 2.2, the linear differential system (19) has a solution, that is, $e^{\mu_1 t}u(t)$, where $\mu_1 = \frac{1}{\mathrm{T}}\ln r(\varPhi_{(\mathrm{F-V})(\cdot)(t) + \xi\, W(t)}(\mathrm{T}))$ and the vector $u(t) = (u_1(t), u_2(t))^T$ is a positive T-periodic function. Choose $t_2 > t_1$ and a small number $\alpha > 0$ in such a way that $\mathrm{w}(t_2) \leq \alpha u(0)$. Consequently we obtain

$$\mathrm{w}(t) \leq \alpha e^{\mu_1(t-t_2)}u(t - t_2), \quad t \geq t_2.$$

Applying Theorem B.1 presented in [47], we have

$$\big(I_h(t), I_\nu(t)\big)^T \leq \mathrm{w}(t) \leq \alpha e^{\mu_1(t-t_2)}u(t - t_2), \quad t \geq t_2.$$

While $R_0 < 1$, one can select a small ξ in such a way that $r(\varPhi_{(\mathrm{F}(t)-\mathrm{V}(t))(\cdot) + \xi\, W(t)}(\mathrm{T})) < 1$. Hence, we obtain $\mu_1 < 0$. As a result $\mathrm{w}(t) \to 0$ as t approaches infinity, it shows that $(I_h(t), I_\nu(t))^T \to 0$ as t approaches infinity. As a result, we obtain $\lim_{t \to \infty} S_{h_1}(t) = \widetilde{S}_{h_1}(t)$ and $\lim_{t \to \infty} S_{h_2}(t) = \widetilde{S}_{h_2}(t)$ from the impulsive system (5). Thus, the solution of the impulsive system is globally attractive. As a consequence, the disease-free periodic solution of impulsive system (5) is GAS for $R_0 < 1$. This completes the proof.   □

Remark The stability result presented in Theorem 3.3 exhibits the threshold dynamics and agrees with the epidemiological mechanism, where the infection will vanish if the average number infected by an infective individual in his infectious period is less than 1, but the infection will remain in the community if the average number is greater than 1. Moreover, the stability of the infection-free solution means that the host population converges to the periodic functions given by (11) and (13), while the vector population tends to zero.

Theorem 3.4 *The infection in the impulsive system* (5) *is uniformly persistent if* $R_0 > 1$, *in other words, there is* $\kappa > 0$ *such that* $\lim_{t\to\infty} \inf I_h(t) > \kappa$, $\lim_{t\to\infty} \inf I_v(t) > \kappa$.

Proof To obtain the required result, we will first provide evidence that we can find a constant $\kappa > 0$, in such a way as to make

$$\lim_{t\to\infty} \sup I_h(t) > \kappa, \qquad \lim_{t\to\infty} \sup I_v(t) > \kappa.$$

In conflict with this, we take the assumption as regards time t in such a manner that $I_h(t) < \kappa$ and $I_v(t) < \kappa$, for all $t \geq t_1$. From system (5), we have

$$\begin{cases} \frac{dS_{h_1}}{dt} \geq \mu_h N_h - \beta_1(t)S_{h_1}\kappa - \mu_h S_{h_1}, & t \neq nT, n \in \mathbb{N}, \\ S_{h_1}(t^+) = (1-q)S_{h_1}(t), & t = nT, n \in \mathbb{N}. \end{cases} \quad (20)$$

We take the auxiliary system

$$\begin{aligned} \frac{dx_1}{dt} &= \mu_h N_h - \big(\beta_1(t)\kappa + \mu_h\big)x_1, \quad t \neq nT, n \in \mathbb{N}, \\ x_1\big(t^+\big) &= (1-q)x_1(t), \quad t = nT, n \in \mathbb{N}. \end{aligned} \quad (21)$$

The above-mentioned impulsive system (21) has a positive ω-periodic globally asymptotically stable solution,

$$\widetilde{x}_1(t) = e^{-\int_{nT}^t (\beta_1(\rho)\kappa + \mu_h)\, d\rho}\left(x_1^* + \int_{nT}^t \mu_h N_h e^{\int_{nT}^\rho (\beta_1(\tau)\kappa + \mu_h)\, d\tau}\, d\rho\right), \quad t \in \big(nT, (n+1)T\big],$$

where

$$x_1^* = (1-q)\left[\frac{\int_{nT}^{(n+1)T} \mu_h N_h e^{\int_{nT}^\rho (\beta_1(\tau)k + \mu_h)\, d\tau}\, d\rho}{e^{\int_{nT}^{(n+1)T} (\beta_1(\rho)\kappa + \mu_h)\, d\rho} - (1-q)}\right].$$

In other words, $x_1(t) \to \widetilde{x}_1(t)$, as t tends to infinity. The comparison theorem of IDEs implies that $S_{h_1}(t) \geq x_1(t)$, similarly we can justify that $S_{h_2}(t) \geq x_2(t)$. At the moment $\lim_{\kappa\to 0}(\widetilde{x}_1, \widetilde{x}_2) = (\widetilde{S}_{h_1}, \widetilde{S}_{h_2})$. As a consequence of this, there exists small κ_1 for any $\xi_1 > 0$ such that $\widetilde{x}_1 \geq \widetilde{S}_{h_1} - \xi_1$ and $\widetilde{x}_2 \geq \widetilde{S}_{h_2} - \xi_1$ for $\kappa < \kappa_1$. Using a comparison analysis, there exist $t_2 \geq t_1$ and $\xi_2 > 0$, in such a manner that

$$S_{h_1}(t) \geq x_1(t) \geq \widetilde{x}_1 - \xi_2 \geq \widetilde{S}_{h_1} - \xi_1 - \xi_2$$

and

$$S_{h_2}(t) \geq x_2(t) \geq \widetilde{x}_2 - \xi_2 \geq \widetilde{S}_{h_2} - \xi_1 - \xi_2.$$

Again from the dynamical system (5), we have

$$\begin{aligned} \frac{dI_h}{dt} &\geq \beta_1(t)\big(\tilde{S}_{h_1}(t) - \xi_1 - \xi_2\big)I_v + \beta_2(t)\big(\tilde{S}_{h_2}(t) - \xi_1 - \xi_2\big)I_v - (\mu_h + \gamma_h)I_h, \\ \frac{dI_v}{dt} &\geq \beta_3(t)\big(\widetilde{N}_v(t) - \kappa\big)I_h - \mu_v(t)I_v. \end{aligned} \quad (22)$$

We take the auxiliary system

$$w' = \big(F(t) - V(t)\big)w - M(t)w, \tag{23}$$

where $w = (w_1, w_2)^{\mathrm{T}}$. In the premises of Lemma 2.2, we claim that we can find a T-periodic and positive function $v(t) = (v_1(t), v_2(t))$ so that $e^{\mu_2 t} v(t)$ is a solution of system (23) with $\mu_2 = \frac{1}{\mathrm{T}} \ln r(\Phi_{(F-V)(\cdot)(t)-M(t)}(\mathrm{T}))$. Select $t_3 > t_2$ and an $\eta > 0$ small enough in such a manner that $w(t_2) \geq \eta v(0)$. We get

$$w(t) \geq \eta e^{\mu_2(t-t_2)} v(t - t_2), \quad t \geq t_3.$$

Applying the standard comparison theorem presented in [47], we have

$$\big(I_h(t), I_v(t)\big)^T \geq w(t) \geq \eta e^{\mu_2(t-t_2)} v(t - t_2), \quad t \geq t_3.$$

As $r(\Phi_{(F-V)}(\cdot)(t)(\mathrm{T})) > 1$ and $r(\Phi_{(F-V)}(\cdot)(t) - M(t)(\mathrm{T}))$ is continuous for small ξ_1 and ξ_2, we may choose $\xi_1, \xi_2 > 0$ in such a way that $r(\Phi_{(F-V)(\cdot)-M}(\mathrm{T})) > 1$. As a result we obtain $\mu_2 > 0$. Because of this $w(t)$ converges to infinity as $t \to \infty$, consequently $(I_h(t), I_v(t))^T \to \infty$ as $t \to \infty$, which conflicts the boundedness of I_h and I_v. Thus we obtain the proof of the claim, that is, $\lim_{t\to\infty} \sup I_h(t) \geq \kappa$ and $\lim_{t\to\infty} \sup I_v(t) \geq \kappa$. Furthermore, we have the following two possibilities from the claim:

  (a) $I_h(t) > \kappa$ and $I_v(t) > \kappa$ for large t;

  (b) oscillations of $I_h(t)$ and $I_v(t)$ about κ for large t.

If case (a) is satisfied, then the required proof is obtained. Next, we focus on case (b). As $\lim_{t\to\infty} \sup I_h(t) \geq \kappa$ and $\lim_{t\to\infty} \sup I_v(t) \geq \kappa$, we can find a $t_1 \in (n_1 \mathrm{T}, (n_1 + 1)\mathrm{T}]$ in such a way that $I_h(t_1) \geq \kappa$ and $I_v(t_1) \geq \kappa$. The above discussion implies that there exists $t_2 \in (n_2 \mathrm{T}, (n_2 + 1)\mathrm{T}]$, such that $I_h(t_2) \geq \kappa$ and $I_v(t_2) \geq \kappa$, where $n_2 - n_1 \geq 0$ is finite. We will now observe the solution of the impulsive system (5) in the interval $[t_1, t_2]$:

$$I'_h = \beta_1(t) S_{h_1} I_v + \beta_2(t) S_{h_2} I_v - (\mu_h + \gamma_h) I_h \geq -(\mu_h + \gamma_h) I_h.$$

Consequently, we get

$$I_h(t) \geq I_h(t_1) e^{-(\mu_h+\gamma_h)(t-t_1)} \geq \kappa e^{-(\mu_h+\gamma_h)(t_2-t_1)} \geq \kappa e^{-(\mu_h+\gamma_h)(n_2-n_1)\mathrm{T}}.$$

Moreover,

$$I'_v = \beta_3(t) \big(\widetilde{N}_v(t) - I_v\big) I_h - \mu_v(t) I_v \geq -\mu_v(t) I_v,$$

which gives

$$I_v(t) \geq I_v(t_1) e^{-\int_{t_1}^{t} \mu_v(s)\,ds} \geq \kappa e^{-\int_{t_1}^{t_2} \mu_v(s)\,ds} \geq \kappa e^{-\int_{n_1\mathrm{T}}^{n_2\mathrm{T}} \mu_v(t)\,dt}.$$

Let $\kappa_1 = \min\{\kappa e^{-\int_{n_1\mathrm{T}}^{n_2\mathrm{T}} \mu_v(t)\,dt}, \kappa e^{-(\mu_h+\gamma_h)(n_2-n_1)\mathrm{T}}\}$, then $\kappa_1 > 0$ cannot be infinitely small and $n_2 - n_1 \geq 0$ is finite. We have $I_h(t) \geq \kappa_1 > 0$ and $I_v(t) \geq \kappa_1 > 0$.

Taking the same steps for $t > t_2$, we obtain another non-infinitesimal positive κ_2. As a consequence of this, we get the sequence $\{\kappa_j\}$, where $\kappa_k = \min\{\kappa e^{-\int_{n_k T}^{n_{k+1} T} \mu_v(t)\,dt}$, $\kappa e^{-(\mu_h+\gamma_h)(n_{k+1}-n_k)T}\}$ for $j = 1, 2, \ldots, k, \ldots$ is non-infinitesimal, as the term $n_{k+1} - n_k \geq 0$ is finite. In this case, the solution of system (5) $I_h(t) \geq \kappa_k > 0$ and $I_v(t) \geq \kappa_k > 0$ holds true in the interval $[t_k, t_{k+1}]$ of time, where $t_k \in (n_k T, n_{k+1} T)$, $t_{k+1} \in (n_{k+1} T, (n_{k+1}+1)T]$. Let $\kappa^* = \min_j(\kappa_j) = \kappa_l > 0$, $l \in N$, $\kappa_l \in \{\kappa_j\}$ for $j = 1, 2, \ldots$, hence $I_h(t) \geq \kappa^* > 0$ and $I_v(t) \geq \kappa^* > 0$ for all $t \geq t_1$. This completes the proof. $\qquad\qquad\square$
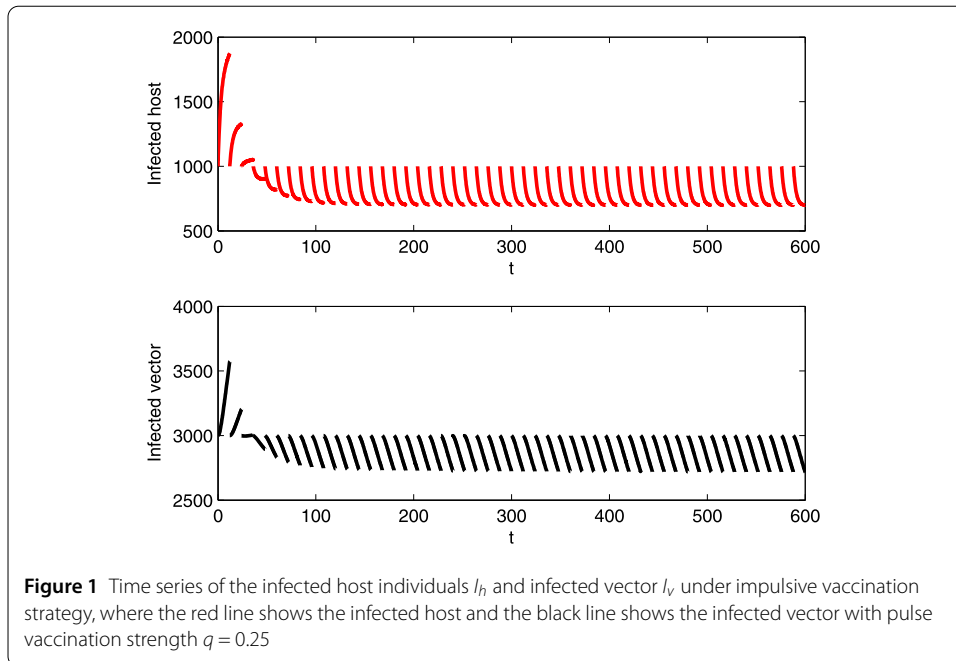
4 Numerical results

In this part of the article, we will analyze and investigate the dengue disease transmission model (5) numerically to conceptualize the transmission pathway of dengue infection in the proposed impulsive system, and to scrutinize the consequences of impulsive vaccination on the transmission dynamics. The periodic transmission probabilities from vector to host and vice versa are taken as $\beta_{h_1}(t) = \alpha_1(1 + \delta_1 \cos(2\pi t))$, $\beta_{h_2}(t) = \alpha_2(1 + \delta_2 \cos(2\pi t))$ and $\beta_v(t) = \alpha_3(1 + \delta_3 \cos(2\pi t))$, respectively.

To demonstrate the influence of pulse vaccination on the transmission of dengue infection numerically, we use the values of parameter for simulation purpose inventoried in Table 1 and select the state variables for simulation purpose as $S_v(0) = 18{,}000$, $I_v(0) = 3000$, $S_{h1}(0) = 30{,}000$, $S_{h2}(0) = 20{,}000$, $I_h(0) = 1000$ and $R_h(0) = 20{,}000$. Furthermore, we choose the values $\alpha_1 = 0.750$, $\alpha_2 = 0.300$, $\alpha_3 = 0.750$, $\delta_1 = 0.05$, $\delta_2 = 0.03$, and $\delta_3 = 0.01$ to conceptualize the dynamical behavior of our impulsive system, moreover, we assumed constant birth rates for simplicity in our simulations. In Fig. 1, we illustrate the moment path of infected host individuals $I_h(t)$ and infected vector individuals $I_v(t)$ with fixed-moment pulse vaccination and the vaccination strength $q = 0.25$, which demonstrates the endemic intensity of dengue infection in both species. To examine the potency of the vaccination rate q, we increase the vaccination rate from 0.25 to 0.75, as shown in Fig. 2. We observe that the vaccination measure has a great influence on the system, which means that pulse vaccination can efficiently control infected individuals and can effectively prevent a dengue outbreak. To be more specific, it can be noticed that after implementing the vaccination strategy, the lower level of infected classes in vector and host decreases. Numerical results predict that the vaccination rate q has a great influence on the infection and can successfully decrease the outbreak.
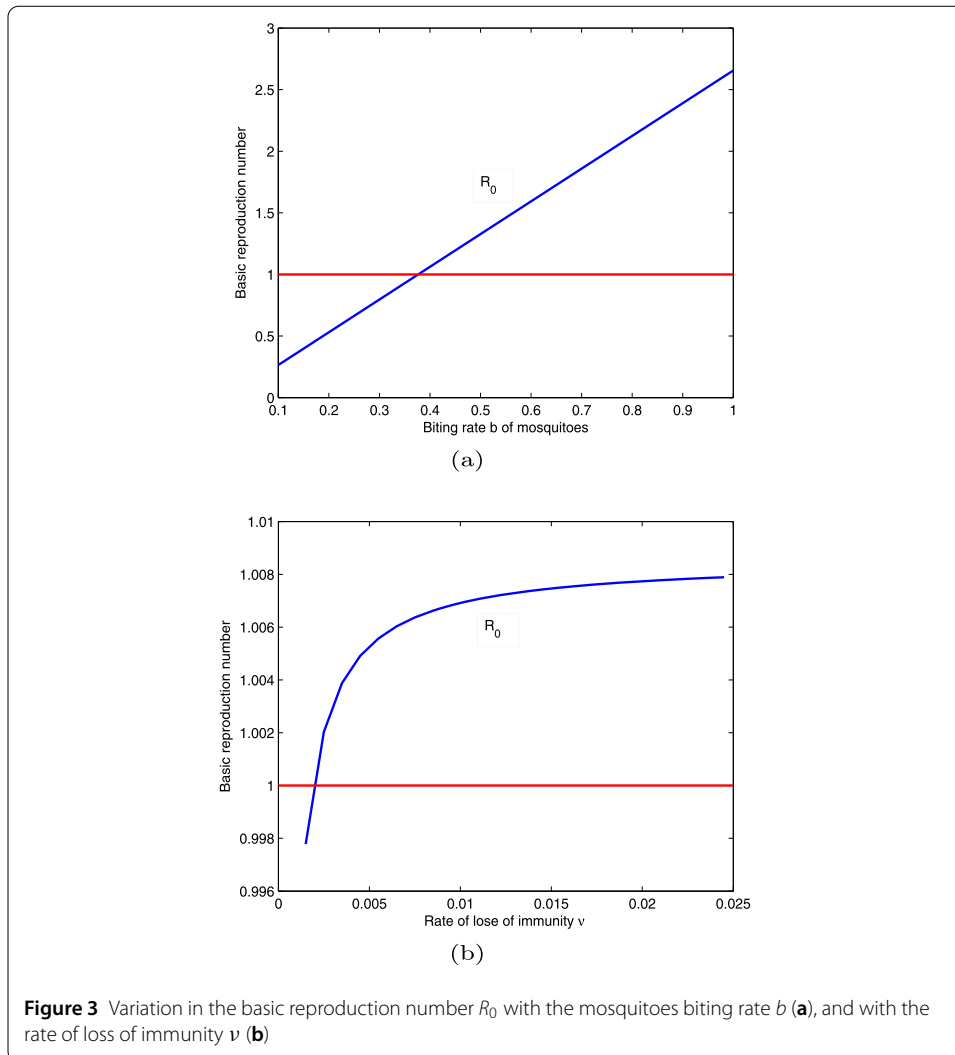
Now we determine the influence of different parameters on the threshold parameter R_0 of system (5). We firstly examine the influence of the biting rate of mosquitoes on the threshold parameter, illustrated in Fig. 3(a). It shows that changing the biting rate b from

Table 1 Parameters values with interpretation used in numerical simulations

Parameter	Interpretation	Values	References
μ_h	Recruitment and natural mortality rate of host	0.000046 & 0.004500	[48, 49]
$\beta_{h_1}(t)$	Transmission rate from mosquitoes to susceptible host	Periodic	[50]
b	Bitting rate of mosquitoes	0.500	[26]
$\beta_{h_2}(t)$	Transmission rate from mosquitoes to susceptible after losing immunity	Periodic	[50]
$\beta_v(t)$	Transmission rate from host to susceptible mosquitoes	Periodic	[50]
υ	Rate of lose of immunity	0.005	Assumed
γ_h	Recovery rate of host	0.3288330	[48, 49]
μ_v	Recruitment and natural mortality rate of vector	Periodic	Assumed

Figure 1 Time series of the infected host individuals I_h and infected vector I_v under impulsive vaccination strategy, where the red line shows the infected host and the black line shows the infected vector with pulse vaccination strength $q = 0.25$

Figure 2 Time series of the infected host individuals I_h and infected vector I_v under impulsive vaccination strategy, where the red line shows infected host and the black line shows infected vector with pulse vaccination strength $q = 0.75$

0.1 to 1 increases the endemic level of the system from 0.26 to 2.34, and consequently leads to an increase in the size of the outbreak and the level of infection, moreover, $R_0 = r(\Phi_{(F-V)(\cdot)}(T)) = 1$ shows that the critical biting rate is $\bar{b} = 0.384$. This indicates that if we control mosquitos such that the biting rate is less than $\bar{b}$, implying that R_0 is less than the unity, then the dengue infection can be successfully controlled. We thus emphasize the importance of using mosquito nets which can effectively result in the bitting rate decline. Figure 3(b) shows the variation in the R_0 with the variation of the losing rate of immunity ν, which implies the rate of loss of immunity ν does not significantly affect the threshold

Figure 3 Variation in the basic reproduction number R_0 with the mosquitoes biting rate b (**a**), and with the rate of loss of immunity ν (**b**)
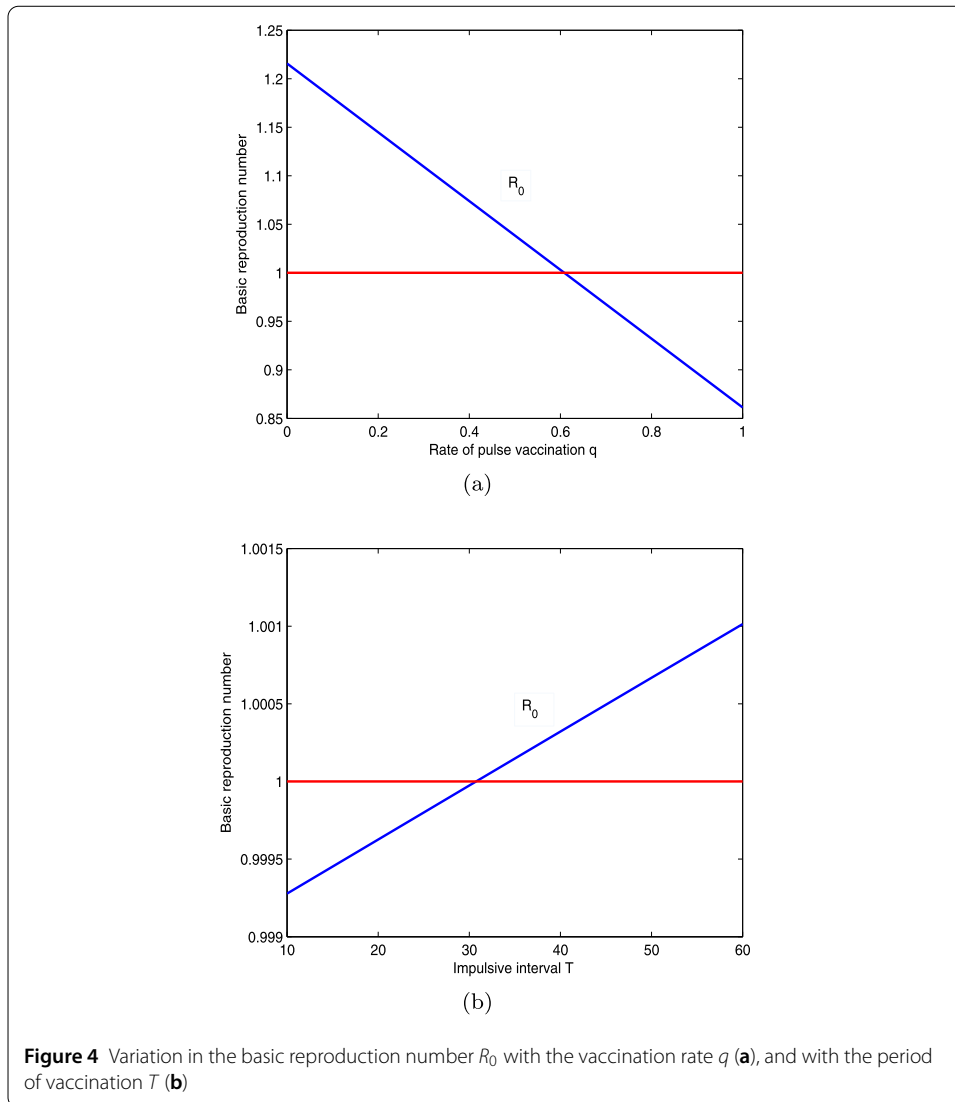
parameter R_0, indicating that the impact of the partial immunization on lowering the new infection is limited, this happens in dengue infection due to the existence of four serotypes.

To examine the influence of impulsive vaccination on new infections we give the variation in threshold parameter R_0 with the intensify q and period of pulse vaccination T, shown in Fig. 4(a) and (b). It says that the higher the vaccination rate is, the less the vaccination period is, and the less the basic reproduction number R_0 is, which implies frequent implementation of the vaccination strategy with great intensity leads to decline of new infections. As more than 50 percent of the world population are in regions with threat of dengue infection and the economic weight of dengue is increasing day by day, pulse vaccination is a beneficial strategy to lower the risk of infection and reduce the economic burden, compared with continuous vaccination strategy in endemic area of dengue fever to control the disease.

5 Conclusion

A pulse vaccination policy is an effective measure to prevent infectious diseases, it has contributed successfully in the elimination of several infectious diseases including poliomyelitis and measles. In [51], the advantages of pulse vaccination have been shown

Figure 4 Variation in the basic reproduction number R_0 with the vaccination rate q (**a**), and with the period of vaccination T (**b**)

against measles through mathematical models. Recently, the periodic repetition of vaccination and pulse vaccination policy has attracted the attention of researchers because of the economic and epidemiological factors mentioned in [52–56]. Thus, in this article, we proposed a dynamical model for the transmission pathway of dengue infection with periodic transmission functions and seasonality in the vector population. Moreover, we introduced the strategy of pulse vaccination in the susceptible host population in order to curb the level of dengue infection. We obtained the basic reproduction number, defined as the spectral radius of an infection operator for the proposed impulsive system, which governs whether the disease dies out or not. It has been proven that the disease-free periodic solution is globally asymptotically stable if R_0 less than unity and is unstable otherwise. We further proved that the dengue infection is uniformly persistent in the community if $R_0 > 1$. Moreover, we numerically demonstrated the influence of pulse vaccination on the infected hosts or infected mosquitos and examined the effect of different input factors on the threshold parameter of the system. The finding indicates that frequent implementation of the vaccination strategy with great intensity leads to a decline of new infections.

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