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Modelling the *Wolbachia* Strains for Dengue Fever Virus Control in the Presence of Seasonal Fluctuation*

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Abstract Consider that infection with *Wolbachiacan* limit a mosquito's ability to transmit Dengue fever virus through its saliva, a mathematical model describing the transmission of Dengue fever between vector mosquitoes and human, incorporating *Wolbachia*-carrying mosquito population and seasonal fluctuation, is proposed. Firstly, the stability and bifurcation of this model are investigated exactly in the case where seasonality can be neglected. Further, the basic reproductive number $\mathcal{R}_0^s$ for this model with seasonal variation is obtained, that is, if $\mathcal{R}_0^s$ is less than unity the disease is extinct and $\mathcal{R}_0^s$ is greater than unity the disease is uniformly persistent. Finally, numerical simulations verify the theoretical results. Theoretical results suggest that, compared with the mosquito reduction strategies (such as the elimination of mosquito breeding sites, killing of adult mosquitoes by spraying), introducing *Wolbachia* strains is as effectual to fight against the transmission of Dengue virus.

Keywords Dengue fever, *Wolbachia*, Seasonal fluctuation, Stability and sensitivity analysis, Extinction and persistence.

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1. Introduction

Dengue fever is a viral disease mainly prevalent in tropical and subtropical regions of the world, which is transmitted by the bite of an *Aedes* mosquito infected with Dengue virus. It is estimated that approximately 1.5 billion people are at risk, and may be 50-100 million individuals are affected by Dengue fever virus each year [23]. Currently, there are still no specific antiviral therapy or vaccines available to combat Dengue fever [6], so the method of controlling the vector population is still a main measure to prevent the transmission of Dengue fever virus. It is well-known that traditional measures such as the use of insecticides to reduce the mosquito population tend to be very expensive, unsustainable, environmentally undesirable, which may indeed lead to insecticide resistance [6].

Experiments and field trials have demonstrated that the intracellular bacterium

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Wolbachia is a maternally transmitted endosymbiotic bacterium that is estimated to infect as much as 65% of insect species and have been surveyed that infect about 28% of mosquito species [9, 22]. It lives in the testes and ovaries of hosts and interferes with the reproductive mechanisms, inducting a variety of mosquito phenotypes such as those with cytoplasmic incompatibility (CI), parthenogenesis, feminization of genetic males and so on. The effects of CI causes embryos from females uninfected with *Wolbachia* to die when they are mated with infected males. Whereas, infected females are not affected in this manner [11]. In mosquitoes vectors, *Wolbachia* induced CI and matrilinear inheritance may have opposite effects, such as population extinction, coexistence or all of the uninfected population may be replaced by infected insects. Some *Wolbachia* cannot be only successfully spread within mosquito populations through CI, but also prevent mosquito host to replicate and spread Dengue fever virus [10, 20].

Recently, McMeniman et al. [13] have proposed a *Wolbachia* infection in an *Aedes aegypti* population through microinjecting bacteria into the mosquito embryos and reducing the transmission of Dengue fever virus from mosquitoes to humans. New analysis of that data shows that there are usually two ways in which *Wolbachia*-infected mosquitoes may be inferior Dengue vectors, one reduces adult survival sufficiently may result in very few infected mosquitoes reaching the infectious stage [18], another limits a mosquito's ability to transmit Dengue fever virus through its saliva [10]. Taking these means into account, there are many mathematical models (including discrete-time and continuous-time models) are investigated for the effects of *Wolbachia* infection (see, e.g., [2,16,25] and the references therein).

On the other hand, for infectious diseases spreading by vectors, the seasonality is apparent on the varying contact rate over the years [3]. It becomes natural to model these diseases as periodically forced nonlinear systems [8]. The seasonality is often seen as the main factor responsible for periodic epidemic cycles, and different kinds of seasonality sources are analyzed. For example, varying transmission rates [4], the volatility of birth rates [12], vaccination program [14] and so on. Particularly, the increased intensity and frequency of El Nio in the past few years, which leads to frequent outbreaks and quick spread of insect-borne infectious diseases around the world. Therefore, how to control and eliminate vector-borne diseases should be one of worldwide public health problems.

Based on the above discussion, we propose a mathematical model of the transmission dynamics of Dengue fever in vector mosquitoes and human population in this paper, where *Wolbachia*-carrying mosquito population and seasonal fluctuation are introduced. The main purpose is to discuss the effects of *Wolbachia* and seasonal change for the control and elimination of Dengue fever virus. The rest of the paper is outlined as follows: a basic mathematical model with *Wolbachia* and seasonal fluctuation for Dengue virus transmission dynamics and some preliminaries are proposed in Section 2. We consider the existence and stability of equilibria in Section 3. In Section 5, we investigate the extinction and uniform persistence of Dengue fever in the presence of seasonal fluctuation. Section 6 contains numerical simulations for theoretical results and biological conclusions.

2. Model formulation and preliminaries

We set up a mathematical model to study how introducing *Wolbachia* into a mosquito population might affect the transmission of Dengue fever virus. In it,

the model comprises human population, wild mosquito population and *Wolbachia*-carrying mosquito population. The human population is namely divided into three subpopulations including Susceptible S_h, Infectious I_h and Recovered R_h; the wild mosquito population is divided into two subpopulations of susceptible S_m and Infectious I_m; the *Wolbachia*-carrying mosquito population is also divided into Susceptible S_w and Infectious I_w. Table 1 provides glossaries of all of the variables and parameters used. Therefore, the model is guided by the following system of differential equations.

Table 1. Parameter definitions, possible value for model (2.1)

Param.	Description	Value	Source
$\beta_{hm}(t)$	Infected rate from infectious humans to wild mosquitoes	unknown	–
$\beta_{mh}(t)$	Infected rate from infectious wild mosquitoes to humans	unknown	–
$\beta_{hw}(t)$	Infected rate from infectious humans to *Wolbachia*-carrying mosquitoes	unknown	–
$\beta_{wh}(t)$	Infected rate from infectious *Wolbachia*-carrying mosquitoes to humans	unknown	–
$1/\mu_h$	Average host life expectancy (year)	72	[19]
γ_h	Dengue recovery rate in human (day^{-1})	$[0.0713, 0.3333]$	[19]
$\mu_m(t)$	Mosquito natural mortality rate (day^{-1})	$[0.05, 0.25]$	[19]
$\mu_w(t)$	*Wolbachia*-carrying mosquito natural mortality rate (day^{-1})	$[0.016, 0.425]$	[2]
$\Lambda_m(t)$	Birth rate of mosquitoes	unknown	–
$\Lambda_w(t)$	Birth rate of *Wolbachia*-carrying mosquitoes	unknown	–

$$\begin{cases} \dfrac{\mathrm{d}S_h(t)}{\mathrm{d}t} = \mu_h N_h(t) - \left(\beta_{mh}(t)\dfrac{I_m(t)}{N_h(t)} + \beta_{wh}(t)\dfrac{I_w(t)}{N_h(t)} + \mu_h\right) S_h(t), \\ \dfrac{\mathrm{d}I_h(t)}{\mathrm{d}t} = \left(\beta_{mh}(t)I_m(t) + \beta_{wh}(t)I_w(t)\right)\dfrac{S_h(t)}{N_h(t)} - (\gamma_h + \mu_h)I_h(t), \\ \dfrac{\mathrm{d}S_m(t)}{\mathrm{d}t} = \Lambda_m(t) - \left(\beta_{hm}(t)\dfrac{I_h(t)}{N_h(t)} + \mu_m(t)\right) S_m(t), \\ \dfrac{\mathrm{d}I_m(t)}{\mathrm{d}t} = \beta_{hm}(t)\dfrac{I_h(t)}{N_h(t)} S_m(t) - \mu_m(t)I_m(t), \\ \dfrac{\mathrm{d}S_w(t)}{\mathrm{d}t} = \Lambda_w(t) - \left(\beta_{hw}(t)\dfrac{I_h(t)}{N_h(t)} + \mu_w(t)\right) S_w(t), \\ \dfrac{\mathrm{d}I_w(t)}{\mathrm{d}t} = \beta_{hw}(t)\dfrac{I_h(t)}{N_h(t)} S_w(t) - \mu_w(t)I_w(t), \end{cases} \tag{2.1}$$

with the equation

$$\frac{\mathrm{d}R_h(t)}{\mathrm{d}t} = \gamma_h I_h(t) - \mu_h R_h(t),$$

where $N_h(t)$=$S_h(t)$+$I_h(t)$+$R_h(t)$, $N_m(t)$=$S_m(t)$+$I_m(t)$, $N_w(t)$=$S_w(t)$+$I_w(t)$ and $\beta_{mh}(t)$, $\beta_{wh}(t)$, $\beta_{hm}(t)$, $\beta_{hw}(t)$, $\mu_m(t)$, $\mu_w(t)$ are all positive T-periodic functions.

Let $\mathbb{R}^n_+ := \{(x_1, x_2, \cdots, x_n) : x_i \geq 0, i = 1, 2, \cdots, n\}$. According to the biological background of model (2.1), we only need to discuss the dynamical behaviors in

$\mathbb{R}^7_+$. First of all, on the positivity of solution of model (2.1), we have the following Lemma 2.1 and the proof is obvious.

Lemma 2.1. *The solution of model* (2.1) *with nonnegative initial values is nonnegative and bounded for all* $t > 0$.

By adding the first one to the third equation of model (2.1), it follows that $\mathrm{d}N_h(t)/\mathrm{d}t = 0$. Therefore, the total numbers of human population $N_h(t) := N_h$ is constant, respectively. Due the $R_h(t)$ has no influence on other variables of model (2.1). Thus, we only need to consider the dynamics of model (2.1) for full system on region Ω, where

$$\Omega := \Big\{(S_h(t), I_h(t), S_m(t), I_m(t), S_w(t), I_w(t)) \in \mathbb{R}^6_+ : S_h(t) + I_h(t) \le N_h,$$
$$S_m(t) + I_m(t) \le \Lambda^u_m/\mu^l_m,\ S_w(t) + I_w(t) \le \Lambda^u_w/\mu^l_w\Big\}$$

and $\Lambda^u_i = \sup_{t\in[0,\infty)} \Lambda_i(t)$, $\Lambda^l_i = \inf_{t\in[0,\infty)} \Lambda_i(t)$, $i = m,\ w$.

Let $(\mathbb{R}^n, \mathbb{R}^n_+)$ be the standard ordered n-dimensional Euclidean space with a norm $\|\cdot\|$. For $u, v \in \mathbb{R}^n$, we write $u \ge v$ if $u - v \in \mathbb{R}^n_+$, $u > v$ if $u - v \in \mathbb{R}^n_+\backslash\{0\}$, and $u \gg v$ if $u - v \in \mathrm{Int}(\mathbb{R}^n_+)$. Let $A(t)$ be a continuous, cooperative, irreducible and T-periodic $n \times n$ matrix function, $\Phi_A(t)$ be the fundamental solution matrix of the linear ordinary differential equation

$$\frac{\mathrm{d}x}{\mathrm{d}t} = A(t)x \tag{2.2}$$

and $\rho(\Phi_A(T))$ be the spectral radius of $\Phi_A(T)$. By the Perron-Frobenius theorem, $\rho(\Phi_A(T))$ is the principal eigenvalue of $\Phi_A(T)$ in sense that it is simple and admits an eigenvector $v^* \gg 0$. The following result is useful for our theoretical results.

Lemma 2.2 (See [24], Lemma 2.1)**.** *Let* $p = \ln \rho(\Phi_A(T))/T$, *then there is a positive,* T*-periodic function* $v(t)$ *such that* $e^{pt}v(t)$ *is a solution of equation* (2.2).

3. Stability analysis in the absence of seasonality

In this section, we investigate a particular case of model (2.1). That is, the incidence rates $\beta_{mh}(t) := \beta_{mh}$, $\beta_{wh}(t) := \beta_{wh}$, $\beta_{hm}(t) := \beta_{hm}$, $\beta_{hw}(t) := \beta_{hw}$, $\mu_m(t) := \mu_m$, $\mu_w(t) := \mu_w$, $\Lambda_m(t) := \Lambda_m$ and $\Lambda_w(t) := \Lambda_w$ are constants, respectively. Now, we define the basic reproduction number of model (2.1) without seasonal fluctuation as follows:

$$\mathcal{R}_0 = \frac{\beta_{mh}\beta_{hm}\Lambda_m}{\mu^2_m(\mu_h + \gamma_h)N_h} + \frac{\beta_{wh}\beta_{hw}\Lambda_w}{\mu^2_w(\mu_h + \gamma_h)N_h} := \mathcal{R}_{01} + \mathcal{R}_{02}.$$

For model (2.1) without seasonal fluctuation, we get the disease-free equilibrium $E_0(N_h,\ 0,\ \Lambda_m/\mu_m,\ 0,\ \Lambda_w/\mu_w)$ and endemic equilibrium $E^*(S^*_h, I^*_h, S^*_m, I^*_m, S^*_w, I^*_w)$,

$$I^*_m = \frac{\beta_{hm}\Lambda_m I^*_h}{\mu_m(\beta_{hm}I^*_h + \mu_m N_h)}, I^*_w = \frac{\beta_{hw}\Lambda_w I^*_h}{\mu_w(\beta_{hw}I^*_h + \mu_w N_h)},$$
$$S^*_h = \frac{a}{b}, S^*_m = \frac{\lambda_m}{\mu_m} - I^*_m, S^*_w = \frac{\lambda_w}{\mu_w} - I^*_w,$$

and

$$\begin{aligned} a =&\mu_h N_h^2(\mu_h N_h + \beta_{hm} I_h^*)(\mu_w N_h + \beta_{hw} I_h^*), \\ b =&\mu_h N_h(\mu_m N_h + \beta_{hm} I_h^*)(\mu_w N_h + \beta_{hw} I_h^*) + \beta_{mh}\beta_{hm}\Lambda_m \mu_m^{-1} I_h^*(\mu_w N_h \\ &+ \beta_{hw} I_h^*) + \beta_{wh}\beta_{hw}\Lambda_w \mu_w^{-1} I_h^*(\mu_m N_h + \beta_{hm} I_h^*), \end{aligned}$$

and I_h^* is received by the solutions I_h of following equation

$$AI_h^2 + BI_h + C = 0, \tag{3.1}$$

where

$$\begin{aligned} A =&\beta_{hm}\beta_{hw}(\mu_h + \gamma_h)(\mu_h N_h + \beta_{mh}\Lambda_m \mu_m^{-1} + \beta_{wh}\Lambda_w \mu_w^{-1}), \\ B =&N_h\big\{\mu_m \beta_{hw}\mu_h N_h(1 - \mathcal{R}_{01}) + \mu_w \beta_{hm}\mu_h N_h(1 - \mathcal{R}_{02}) + \mu_m \beta_{hw}\beta_{wh}\Lambda_w \mu_w^{-1} \\ &+ \mu_w \beta_{hm}\beta_{mh}\Lambda_m \mu_m^{-1}\big\} \\ C =&\mu_h \mu_m \mu_w(\mu_h + \gamma_h)N_h^3\left(1 - \mathcal{R}_{01} - \mathcal{R}_{02}\right). \end{aligned}$$

It is obvious that $A > 0$ for the meaningful values of model parameters. Noting that $C < 0$ is equal to $\mathcal{R}_0 > 1$, which is a necessary condition to equation (3.1) has a unique positive root. Obviously, if $B > 0$ and $C > 0$, that is, $\mathcal{R}_0 < 1$, there is no positive root of equation (3.1); if $B < 0$ and $B^2 - 4AC > 0$, there are two positive roots of equation (3.1).

In summary, we have the following Theorem 3.1.

Theorem 3.1. *The disease-free equilibrium of model* (2.1) *without seasonal fluctuation always exists. Furthermore, the following statements are valid:*

(*i*) *if* $C < 0$, *then model* (2.1) *without seasonal fluctuation has a unique endemic equilibrium;*

(*ii*) *if* $B < 0$ *and* $B^2 - 4AC > 0$, *then model* (2.1) *without seasonal fluctuation has two endemic equilibria;*

(*iii*) *if* $B > 0$ *and* $C \geq 0$, *then model* (2.1) *without seasonal fluctuation has no endemic equilibrium.*

On the local asymptotical stability of the disease-free equilibrium E_0 of model (2.1) without seasonal fluctuation, we have Theorem 3.2.

Theorem 3.2. *If* $\mathcal{R}_0 < 1$ *and* $H > 0$, *where*

$$\begin{aligned} H =&(\mu_w + \mu_m + \mu_h + \gamma_h)[\mu_w(\mu_h + \gamma_h)(1 - \mathcal{R}_{02}) + \mu_m(\mu_h + \gamma_h)(1 - \mathcal{R}_{01})] \\ &- \mu_w \mu_m(\mu_h + \gamma_h)(1 - \mathcal{R}_0), \end{aligned}$$

then E_0 *is locally asymptotically stable; if* $\mathcal{R}_0 > 1$, *then* E_0 *is unstable.*

Proof. To investigate the stability of E_0, we linearise model (2.1) without seasonal

fluctuation about E_0 and the corresponding Jacobian matrix is given as follows:

$$J = \begin{pmatrix} -\mu_h & 0 & 0 & -\beta_{mh} & 0 & -\beta_{wh} \\ 0 & -(\mu_h+\gamma_h) & 0 & \beta_{mh} & 0 & \beta_{wh} \\ 0 & -\beta_{hm}\frac{\Lambda_m}{\mu_m N_h} & -\mu_m & 0 & 0 & 0 \\ 0 & \beta_{hm}\frac{\Lambda_m}{\mu_m N_h} & 0 & -\mu_m & 0 & 0 \\ 0 & -\beta_{hw}\frac{\Lambda_w}{\mu_w N_h} & 0 & 0 & -\mu_w & 0 \\ 0 & \beta_{hw}\frac{\Lambda_w}{\mu_w N_h} & 0 & 0 & 0 & -\mu_w \end{pmatrix}.$$

From Jacobian matrix J above we get the characteristic equation about E_0

$$\begin{aligned}(\lambda+\mu_h)(\lambda+\mu_m)(\lambda+\mu_w)\big\{\lambda^3+(\mu_w+\mu_m+\mu_h+\gamma_h)\lambda^2+[\mu_w(\mu_h+\gamma_h)(1\\ -\mathcal{R}_{01})+\mu_m(\mu_h+\gamma_h)(1-\mathcal{R}_{02})]\lambda+\mu_w\mu_m(\mu_h+\gamma_h)(1-\mathcal{R}_0)\big\}=0.\end{aligned} \tag{3.2}$$

According to the Routh-Hurwize conditions, if $H>0$ holds, then all eigenvalues of equation (3.2) are non-positive. Accordingly, if $\mathcal{R}_0<1$ and $H>0$, all eigenvalues of equation (3.2) have negative real parts. In addition, if $\mathcal{R}_0>1$, at least one of eigenvalues of equation (3.2) has positive real part. This completes the proof. □

As for the endemic equilibria, it is difficult to determine the stability of endemic equilibria. So we concentrate on the existence of backward bifurcation, which is important in the control of Dengue virus just as it is in the control of epidemics in general. Through the investigations above we know that, model (2.1) without seasonal fluctuation have two positive roots when $\mathcal{R}_0^c<\mathcal{R}_0<1$; and has no positive root when $\mathcal{R}_0<\mathcal{R}_0^c$, where

$$\mathcal{R}_0^c := 1-\frac{B^2}{4AC},$$

and can be obtained from $B^2-4AC=0$, which is the threshold condition for the existences of positive roots. Then we have the following conclusion.

Theorem 3.3. *Model* (2.1) *without seasonal fluctuation exhibits a backward bifurcation when* $\mathcal{R}_0^c<\mathcal{R}_0<1$ *if* $B<0$.

4. The threshold condition of model (2.1) with seasonal fluctuation

In this section, we show the existence of the disease-free periodic solution of model (2.1). To find this solution of model (2.1), we consider the following subsystem

$$\begin{cases} \dfrac{\mathrm{d}S_h(t)}{\mathrm{d}t} = \mu_h N_h - \mu_h S_h(t), \\ \dfrac{\mathrm{d}S_m(t)}{\mathrm{d}t} = \Lambda_m(t) - \mu_m(t) S_m(t), \\ \dfrac{\mathrm{d}S_w(t)}{\mathrm{d}t} = \Lambda_w(t) - \mu_w(t) S_w(t). \end{cases} \tag{4.1}$$

Obviously, (4.1) admits a unique positive periodic solution $(\widetilde{S}_h(t),\widetilde{S}_m(t),\widetilde{S}_w(t)) = (N_h,\widetilde{S}_m(t),\widetilde{S}_w(t))$, which is globally attractive in $\mathbb{R}^3_+$. That is to say, model (2.1) admits a unique disease-free periodic solution $(N_h,0,\widetilde{S}_m(t),0,\widetilde{S}_w(t),0)$.

Now, we calculate the basic reproduction number of model (2.1) according to the recent results from Reference [1, 21]. Let

$$F(t) = \begin{pmatrix} 0 & \beta_{mh}(t) & \beta_{wh}(t) \\ \beta_{hm}(t)\frac{\widetilde{S}_m(t)}{N_h} & 0 & 0 \\ \beta_{hw}(t)\frac{\widetilde{S}_w(t)}{N_h} & 0 & 0 \end{pmatrix}, \qquad V(t) = \begin{pmatrix} \mu_h + \gamma_h & 0 & 0 \\ 0 & \mu_m(t) & 0 \\ 0 & 0 & \mu_w(t) \end{pmatrix},$$

and assume that $Y(t,s)$, $t \geq s$, is the evolution operator of the linear periodic model $\mathrm{d}y(t)/\mathrm{d}t = -V(t)y(t)$. That is, for each $s \in \mathbb{R}$, the 3×3 matrix $Y(t,s)$ satisfies

$$\frac{\mathrm{d}Y(t,s)}{\mathrm{d}t} = -V(t)Y(t,s), \qquad \text{for all} \quad t \geq s, \qquad Y(s,s) = I$$

where I is 3×3 identity matrix.

Let C_T be the ordered Banach space of all T-periodic functions from $\mathbb{R}$ to $\mathbb{R}^3$, which is equipped with the maximum norm $\|\cdot\|$ and the positive cone $C_T^+ := \{\phi \in C_T : \phi(t) \geq 0, t \in \mathbb{R}\}$. Suppose $\phi(s) \in C_T$ is the initial distribution of infectious individuals in this periodic environment, then $F(s)\phi(s)$ is the rate of new infections produced by the infected individuals which is introduced at time s, and $Y(t,s)F(s)\phi(s)$ represents the distribution of those infected individuals who were newly infected at time s and remain in the infected compartments at time t for $t \geq s$. Hence,

$$\Psi(t) := \int_{-\infty}^{t} Y(t,s)F(s)\phi(s)\mathrm{d}s = \int_{0}^{\infty} Y(t,t-a)F(t-a)\phi(t-a)\mathrm{d}a$$

is the distribution of accumulative new infections at time t produced by all those infected individuals $\phi(s)$ introduced before t.

We define a linear operator $\mathcal{L} : C_T \to C_T$ as follows

$$(\mathcal{L}\phi)(t) = \int_{0}^{\infty} Y(t,t-a)F(t-a)\phi(t-a)\mathrm{d}a \qquad \text{for all } t \in \mathbb{R},\ \phi \in C_T.$$

Then, the basic reproduction number is defined as $\mathcal{R}_0^s := \rho(\mathcal{L})$, the spectral radius of $\mathcal{L}$.

Let $W(t,s,\lambda)$ $(t \geq s,\ s \in \mathbb{R})$ be the evolution operator of the following linear T-periodic model

$$\frac{\mathrm{d}w}{\mathrm{d}t} = \left[-V(t) + \frac{F(t)}{\lambda}\right] w, \qquad t \in \mathbb{R}.$$

Clearly, $\Phi_{F-V}(t) = W(t,0,1)$, for all $t \geq 0$, and for each $\lambda \in (0,\infty)$, the matrix $-V(t) + F(t)/\lambda$ is cooperative. Then, it follows that the linear operator $W(t,s,\lambda)$ is positive in $\mathbb{R}^3$ for each $t \geq s$, $s \in \mathbb{R}$, and $\rho(W(T,0,\lambda))$ is continuous and nonincreasing for $\lambda \in (0,\infty)$, $\lim_{\lambda\to\infty} \rho(W(T,0,\lambda)) < 1$. It is easy to verify that model (2.1) satisfies assumptions (A_1)-(A_7) in Reference [21].

Next, we show that $\mathcal{R}_0^s$ serves as a threshold value, when $\mathcal{R}_0^s < 1$, there is a unique globally asymptotically stable disease-free periodic solution, and when $\mathcal{R}_0^s > 1$, there is at least one positive periodic solution and the disease is persistent in the population.

Theorem 4.1. *If $\mathcal{R}_0^s < 1$, the disease-free periodic solution $(N_h, 0, \widetilde{S}_m(t), 0, \widetilde{S}_w(t), 0)$ of model (2.1) is globally asymptotically stable with respect to the interior of Ω. If $\mathcal{R}_0^s > 1$, it is unstable.*

Proof. By Theorem 2.2 in Reference [21], and if $\mathcal{R}_0^s > 1$; $(N_h, 0, \widetilde{S}_m(t), 0, \widetilde{S}_w(t), 0)$ is unstable and if $\mathcal{R}_0^s < 1$. Then, $(N_h, 0, \widetilde{S}_m(t), 0, \widetilde{S}_w(t), 0)$ is locally stable. Hence, it is sufficient to prove that the solution is globally attractive for $\mathcal{R}_0^s < 1$.

Suppose that $(S_h(t), I_h(t), S_m(t), I_m(t), S_w(t), I_w(t))$ is a nonnegative solution of model (2.1) in Ω, we have

$$\begin{cases} \dfrac{\mathrm{d}S_h(t)}{\mathrm{d}t} \le \mu_h N_h - \mu_h S_h(t), \\ \dfrac{\mathrm{d}S_m(t)}{\mathrm{d}t} \le \Lambda_m(t) - \mu_m S_m(t), \\ \dfrac{\mathrm{d}S_w(t)}{\mathrm{d}t} \le \Lambda_w(t) - \mu_w S_w(t). \end{cases}$$

The comparison theorem of differential equation implies that for any $\epsilon > 0$, there is a $t_1 > 0$ such that

$$S_h(t) \le N_h + \epsilon, \quad S_m(t) \le \widetilde{S}_m(t) + \epsilon, \quad S_w(t) \le \widetilde{S}_m(t) + \epsilon, \qquad \text{for all } t \ge t_1.$$

Consider an auxiliary system

$$\begin{cases} \dfrac{\mathrm{d}\widehat{I}_h(t)}{\mathrm{d}t} = (\beta_{mh}(t)\widehat{I}_m(t) + \beta_{wh}(t)\widehat{I}_w(t))\dfrac{N_h + \epsilon}{N_h} - (\gamma_h + \mu_h)\widehat{I}_h(t), \\ \dfrac{\mathrm{d}\widehat{I}_m(t)}{\mathrm{d}t} = \beta_{hm}(t)\widehat{I}_h(t)\dfrac{\widetilde{S}_m(t) + \epsilon}{N_h} - \mu_m(t)\widehat{I}_m(t), \\ \dfrac{\mathrm{d}\widehat{I}_w(t)}{\mathrm{d}t} = \beta_{hw}(t)\widehat{I}_h(t)\dfrac{\widetilde{S}_m(t) + \epsilon}{N_h} - \mu_w(t)\widehat{I}_w(t), \end{cases} \tag{4.2}$$

which is equivalent to

$$\begin{pmatrix} \frac{\mathrm{d}\widehat{I}_h(t)}{\mathrm{d}t} \\ \frac{\mathrm{d}\widehat{I}_m(t)}{\mathrm{d}t} \\ \frac{\mathrm{d}\widehat{I}_w(t)}{\mathrm{d}t} \end{pmatrix} = (F(t) - V(t) + \epsilon M(t)) \begin{pmatrix} \widehat{I}_h(t) \\ \widehat{I}_m(t) \\ \widehat{I}_w(t) \end{pmatrix}, M(t) = \begin{pmatrix} 0 & \frac{\beta_{mh}(t)}{N_h} & \frac{\beta_{wh}(t)}{N_h} \\ \frac{\beta_{hm}(t)}{N_h} & 0 & 0 \\ \frac{\beta_{hw}(t)}{N_h} & 0 & 0 \end{pmatrix}.$$

It follows from Lemma 2.2 that there is a positive T-periodic function $v_1(t)$ such that $e^{p_1 t}v_1(t)$ is a solution of (4.2), where $p_1 = \ln \rho(\Phi_{F-V+\epsilon M}(T))/T$. Choose $t_2 > t_1$ and a real number $\alpha_1 > 0$ such that $(I_h(t_2), I_m(t_2), I_w(t_2))^T \le \alpha_1 v_1(0)$. By the comparison theorem of differential equation we get $(I_h(t), I_m(t), I_w(t))^T \le \alpha_1 v_1(t - t_2)e^{p_1(t-t_2)}$, for all $t \ge t_2$.

Applying Theorem 2.2 in Reference [21], $\mathcal{R}_0^s < 1$ if and only if $\rho(\Phi_{F-V}(T)) < 1$. By the continuity of the spectrum for matrices (more details can be found in Reference [7], Section II. 5.8), we choose $\epsilon > 0$ small enough such that $\rho(\Phi_{F-V+\epsilon M}(T)) < 1$. Then, it follows the comparison theorem that $(I_h(t), I_m(t), I_w(t))^T \to (0, 0, 0)$ as $t \to \infty$. Moreover, by the theory of asymptotically periodic semiflows (see Reference [26], Section 3.2), we obtain $\lim_{t\to\infty}(S_h(t) - N_h) = \lim_{t\to\infty}(S_m(t) - \widetilde{S}_m(t)) = \lim_{t\to\infty}(S_w(t) - \widetilde{S}_w(t)) = 0$. Hence, $(N_h, 0, \widetilde{S}_m(t), 0, \widetilde{S}_w(t), 0)$ is globally attractive. The proof is completes. □

Theorem 4.2. *If $\mathcal{R}_0^s > 1$, the disease is uniform persistent. That is, there is an $\eta > 0$ such that any solution $(S_h(t), I_h(t), S_m(t), I_m(t), S_w(t), I_w(t))$ of model (2.1) with positive initial value satisfies $\liminf_{t\to\infty} I_j(t) \geq \eta$, $j = h, m, w$. Further, model (2.1) admits at least one positive periodic solution.*

Proof. Let $\mathcal{P} : \Omega \to \Omega$ be the Poincaré map associated with model (2.1), that is $\mathcal{P}(X^0) = \phi(T, X^0)$, $X^0 \in \Omega$, where $\phi(t, X^0)$ is the solution of model (2.1) with $\phi(0, X^0) = X^0$. We define

$$\Omega_0 := \{(S_h, I_h, S_m, I_m, S_w, I_w) \in \Omega : I_h > 0, I_m > 0, I_w > 0\}, \qquad \partial\Omega_0 := \Omega\backslash\Omega_0.$$

It is easy to see that Ω_0 is positively invariant, and $\mathcal{P}$ is point dissipative from the analysis for model (2.1) in Section 2. Set $M_\partial = \{(S_h^0, I_h^0, S_m^0, I_m^0, S_w^0, I_w^0) \in \partial\Omega_0 : \mathcal{P}^k(S_h^0, I_h^0, S_m^0, I_m^0, S_w^0, I_w^0) \in \partial\Omega_0, k \geq 0\}$. To use the theory of uniform persistence developed in Reference [24], we now show that

$$M_\partial = \{(S_h, 0, S_m, 0, S_w, 0) : S_h, S_m, S_w \geq 0\}. \tag{4.3}$$

Obviously, $\{(S_h, 0, S_m, 0, S_w, 0) : S_i \geq 0, i = h, m, w\} \subseteq M_\partial$. For any $(S_h^0, I_h^0, S_m^0, I_m^0, S_w^0, I_w^0) \in \partial\Omega_0\backslash\{(S_h, 0, S_m, 0, S_w, 0) : S_i \geq 0, i = h, m, w\}$, there are following six situations should we discuss

$$\begin{aligned}
&(i)\ I_h^0 = 0, I_m^0 > 0, I_w^0 > 0; && (ii)\ I_m^0 = 0, I_h^0 > 0, I_w^0 > 0;\\
&(iii)\ I_w^0 = 0, I_h^0 > 0, I_m^0 > 0; && (iv)\ I_h^0 = I_m^0 = 0, I_w^0 > 0;\\
&(v)\ I_h^0 = I_w^0 = 0, I_m^0 > 0; && (vi)\ I_m^0 = I_w^0 = 0, I_h^0 > 0.
\end{aligned}$$

Here, we merely prove (i) and (iv), other situations are similar.

First, if (i) is valid, then $I_m(t) > 0$, $I_w(t) > 0$ and $S_h(t), S_m(t), S_w(t) > 0$ for any $t > 0$. From the second equation of model (2.1), we have

$$I_h(t) = \left[I_h^0 + \int_0^t (\beta_{mh}(s)I_m(s) + \beta_{wh}(s)I_w(s))\frac{S_h(s)}{N_h}e^{(\mu_h+\gamma_h)s}\mathrm{d}s\right] e^{-(\mu_h+\gamma_h)t} > 0$$

for all $t > 0$. So, situation (i) is not in M_∂.

Next, if (iv) is valid, then $I_w(t) > 0$ and $S_h(t) > 0, S_m(t) > 0, S_w(t) > 0$ for any $t > 0$. From the second and third equations of model (2.1), we have

$$I_h(t) \geq \left(I_h^0 + \int_0^t \beta_{wh}(s)I_w(s)\frac{S_h(s)}{N_h}e^{(\mu_h+\gamma_h)s}\mathrm{d}s\right) e^{-(\mu_h+\gamma_h)t} > 0, \text{ for all } t > 0;$$

and

$$I_m(t) = \left(I_m^0 + \int_0^t \beta_{hm}(s)S_m(s)\frac{I_h(s)}{N_h}e^{\int_0^s \mu_m(\tau)\mathrm{d}\tau}\mathrm{d}s\right) e^{-\int_0^t \mu_m(s)\mathrm{d}s} > 0, \text{ for all } t > 0.$$

Then, it follows that $(S_h(t), I_h(t), S_m(t), I_m(t), S_w(t), I_w(t)) \notin \partial\Omega_0$ for $0 < t \ll 1$. Thus, the positive invariance of Ω_0 implies (4.3). Clearly, there are two fixed points of P in M_∂, which are $M_0(0, 0, 0, 0, 0, 0)$ and $M_1(N_h, 0, N_m^*, 0, 0, 0)$.

Now, we prove that P is uniformly persistent with respect to $(\Omega_0, \partial\Omega_0)$. By Theorem 2.2 in Reference [21], we have that $\mathcal{R}_0^s > 1$ if and only if $\rho(\Phi_{F-V}(T)) > 1$.

Then we choose $\varepsilon > 0$ small enough that $\rho(\Phi_{F-V-\varepsilon M}(T)) > 1$. Note that a perturbed system of model (4.1)

$$\begin{cases} \dfrac{\mathrm{d}S_h^\alpha(t)}{\mathrm{d}t} = \mu_h N_h - \left[\dfrac{\alpha}{N_h}(\beta_{mh}(t) + \beta_{wh}(t)) + \mu_h\right] S_h^\alpha(t), \\ \dfrac{\mathrm{d}S_m^\alpha(t)}{\mathrm{d}t} = \Lambda_m(t) - \left(\beta_{hm}(t)\dfrac{\alpha}{N_h} + \mu_m(t)\right) S_m^\alpha(t), \\ \dfrac{\mathrm{d}S_w^\alpha(t)}{\mathrm{d}t} = \Lambda_w(t) - \left(\beta_{hw}(t)\dfrac{\alpha}{N_h} + \mu_w(t)\right) S_w^\alpha(t), \end{cases} \tag{4.4}$$

has globally uniformly attractive positive T-periodic solution $(S_h^\alpha(t), S_m^\alpha(t), S_w^\alpha(t))$. By the continuity of solutions of ordinary differential equation with to parameter α, for above ε, there exists a $\alpha_0 \in (0, \alpha)$ such that

$$S_h^\alpha(t) > N_h - \varepsilon, \quad S_m^\alpha(t) > \widetilde{S}_m(t) - \varepsilon, \quad S_w^\alpha(t) > \widetilde{S}_w(t) - \varepsilon, \qquad \text{for all } t \in [0, T].$$

By the continuity of solutions with respect to the initial values, for above give constant ε, there is a $\delta > 0$ such that for any $X^0 = (S_h^0, I_h^0, S_m^0, I_m^0, S_w^0, I_w^0) \in \Omega_0$ with $\|X^0 - M_i\| \le \delta$, we have $\|\phi(t, X^0) - \phi(t, M_i)\| < \varepsilon$, for all $t \in [0, T]$, $i = 0, 1$. We now claim that

$$\limsup_{m\to\infty} \|\mathcal{P}^k(X^0) - M_i\| \ge \varepsilon, \quad \text{for all } X^0 \in \Omega_0. \tag{4.5}$$

Suppose, by contradiction, that

$$\limsup_{k\to\infty} \left\|\mathcal{P}^k(X^0) - M_i\right\| < \varepsilon, \text{ for all } X^0 \in \Omega_0.$$

Without loss of generality, we can assume that $\|\mathcal{P}^k(X^0) - M_i\| < \varepsilon$, for all $k \ge 0$. Then, we have $\|\phi(t, \mathcal{P}^k(X^0)) - \phi(t, M_i)\| < \alpha$, for all $k \ge 0$ and for all $t \in [0, T]$. For any $t \ge 0$, let $t = mT + t_1$, where $t_1 \in [0, T)$ and $m = [t/T]$ is the greatest integer less than or equal to t/T. Then, we get, for all $t \ge 0$

$$\left\|\phi(t, \mathcal{P}^k(X^0)) - \phi(t, M_i)\right\| = \left\|\phi(t_1, \mathcal{P}^k(X^0)) - \phi(t_1, M_i)\right\| < \varepsilon.$$

Let $(S_h(t), I_h(t), S_m(t), I_m(t), S_w(t), I_w(t)) = \phi(t, (S_h^0, I_h^0, S_m^0, I_m^0, S_w^0, I_w^0))$. Then, it follows that $0 \le S_h(t), I_h(t), S_m(t), I_m(t), S_w(t), I_w(t) \le \varepsilon$, for all $t \ge 0$. Then, for $t \ge 0$, we have

$$\begin{cases} \dfrac{\mathrm{d}S_h(t)}{\mathrm{d}t} \ge \mu_h N_h - \left[\dfrac{\varepsilon}{N_h}(\beta_{mh}(t) + \beta_{wh}(t)) + \mu_h\right] S_h(t), \\ \dfrac{\mathrm{d}S_m(t)}{\mathrm{d}t} \ge \Lambda_m(t) - \left(\beta_{hm}(t)\dfrac{\varepsilon}{N_h} + \mu_m(t)\right) S_m(t), \\ \dfrac{\mathrm{d}S_w(t)}{\mathrm{d}t} \ge \Lambda_w(t) - \left(\beta_{hw}(t)\dfrac{\varepsilon}{N_h} + \mu_w(t)\right) S_w(t). \end{cases}$$

From the comparison theorem of ordinary differential equation, one get $S_h(t) > S_h^\varepsilon(t)$, $S_m(t) > S_m^\varepsilon(t)$ and $S_w(t) > S_w^\varepsilon(t)$ for all $t \ge 0$, where $S_h^\varepsilon(t)$, $S_m^\varepsilon(t)$ and $S_w^\varepsilon(t)$ is the solution of model (4.4) with respect to parameter ε satisfying the initial condition $S_h^\varepsilon(0) = S_h(0)$, $S_m^\varepsilon(0) = S_m(0)$ and $S_w^\varepsilon(0) = S_w(0)$ respectively. Therefore, there is $\widehat{t} > 0$ such that

$$S_h(t) > N_h - \varepsilon, \quad S_m(t) > \widetilde{S}_m(t) - \varepsilon, \quad S_w(t) > \widetilde{S}_w(t) - \varepsilon, \qquad \text{for all } t \ge \widehat{t}.$$

As a consequence, for $t \geq \widehat{t}$, it yields that

$$\begin{cases} \dfrac{\mathrm{d}I_h(t)}{\mathrm{d}t} \geq (\beta_{mh}(t)I_m(t) + \beta_{wh}(t)I_w(t))\dfrac{N_h - \varepsilon}{N_h} - (\gamma_h + \mu_h)I_h(t), \\ \dfrac{\mathrm{d}I_m(t)}{\mathrm{d}t} \geq \beta_{hm}(t)I_h(t)\dfrac{\widetilde{S}_m(t) - \varepsilon}{N_h} - \mu_m(t)I_m(t), \\ \dfrac{\mathrm{d}I_w(t)}{\mathrm{d}t} \geq \beta_{hw}(t)I_h(t)\dfrac{\widetilde{S}_w(t) - \varepsilon}{N_h} - \mu_w(t)I_w(t). \end{cases}$$

Consider the following auxiliary system

$$\begin{cases} \dfrac{\mathrm{d}\widetilde{I}_h(t)}{\mathrm{d}t} = (\beta_{mh}(t)\widetilde{I}_m(t) + \beta_{wh}(t)\widetilde{I}_w(t))\dfrac{N_h - \varepsilon}{N_h} - (\gamma_h + \mu_h)\widetilde{I}_h(t), \\ \dfrac{\mathrm{d}\widetilde{I}_m(t)}{\mathrm{d}t} = \beta_{hm}(t)\widetilde{I}_h(t)\dfrac{\widetilde{S}_m(t) - \varepsilon}{N_h} - \mu_m(t)\widetilde{I}_m(t), \\ \dfrac{\mathrm{d}\widetilde{I}_w(t)}{\mathrm{d}t} = \beta_{hw}(t)\widetilde{I}_h(t)\dfrac{\widetilde{S}_w(t) - \varepsilon}{N_h} - \mu_w(t)\widetilde{I}_w(t). \end{cases} \tag{4.6}$$

It follows from Lemma 2.2 that there is a positive T-periodic function $v_2(t)$ such that $e^{p_2 t}v_2(t)$ is a solution of (4.6), where $p_2 = \ln \rho(\Phi_{F-V-\varepsilon M}(T))/T$. There is $\bar{t} \geq \widehat{t}$ and a small number $\alpha_2 > 0$ such that $(I_h(\bar{t}), I_m(\bar{t}), I_w(\bar{t}))^T \geq \alpha_2 v_2(0)$. By the comparison theorem of differential equation we get $(I_h(t), I_m(t), I_w(t))^T \geq \alpha_2 v_2(t - \bar{t})e^{p_2(t-\bar{t})}$, for all $t \geq \bar{t}$. Now we have that $\rho(\Phi_{F-V-\varepsilon M}(T)) > 1$ and thus $p_2 > 0$, which implies that $I_i(t) \to \infty$ as $t \to \infty$, $i = h, m, w$. This leads to a contradiction. Then, the above claim (4.5) show that M_0 and M_1 are isolated invariant sets in Ω.

Let

$$W^s(M_i) := \left\{ M_i^0 : \mathcal{P}^k(M_i^0) \to M_i, m \to \infty \right\},$$

then $W^s(M_i) \cap \Omega_0 = \emptyset$, $i = 0, 1$. Every orbit in M_∂ converges to M_0 or M_1, and M_0 and M_1 are acyclic in M_∂. By the acyclicity theorem on uniform persistence for maps (see Reference [26], Theorem 1.3.1 and Remark 1.3.1), it follows that $\mathcal{P}$ is uniformly persistent with respect to $(\Omega_0, \partial\Omega_0)$. Thus, Reference [26] (see Reference Theorem 1.3.1) implies the uniform persistence of the solutions of model (2.1) with respect to $(\Omega_0, \partial\Omega_0)$. That is, there is a $\epsilon > 0$ such that any solution $(S_h(t), I_h(t), S_m(t), I_m(t), S_w(t), I_w(t))$ of model (2.1) with initial values $(S_h^0, I_h^0, S_m^0, I_m^0, S_w^0, I_w^0) \in \Omega_0$ satisfies $\liminf_{t\to\infty} I_i(t) \geq \epsilon, i = h, m, w$. Moreover, by Theorem 1.3.6 in Reference [26], $\mathcal{P}$ has a fixed point $(S_h^*(0), I_h^*(0), S_m^*(0), I_m^*(0), S_w^*(0), I_w^*(0)) \in \Omega_0$. Consequently, $(S_h^*(t), I_h^*(t), S_m^*(t), I_m^*(t), S_w^*(t), I_w^*(t))$ is a positive T-periodic solution of model (2.1). This is complete the proof. □

5. Numerical simulation and discussion

In this paper, we proposed a model which controlling Dengue fever transmission by *Wolbachia* with seasonal fluctuation, incorporating the interaction of individual population, wild mosquito population and *Wolbachia*-carrying mosquito population. To verify these theoretical conclusions, in this section, we perform some numerical simulations to illustrate the main theoretical results using the Runge-Kutta method in the software MATLAB. For verifying the theoretical results without seasonal fluctuation, we fix basic model parameters as $N_h = 2.0 \times 10^5$, $\Lambda_m = 1.50 \times 10^4$, $\Lambda_w = 1.2 \times 10^4$, $\gamma_h = 1/7$, $\mu_h = 1/(72 \times 365)$, $\mu_m = 1/20$ and $\mu_w = 1/20$.

Firstly, we choose $\beta_{mh} = \beta_{hm} = 3.475 \times 10^{-2}$ and $\beta_{wh} = \beta_{hw} = 1.215 \times 10^{-3}$, which is easy to calculate that $\mathcal{R}_0 \approx 0.2538 < 1$. Therefore, from the conclusion of Theorem 3.2, it follows that the disease-free equilibrium is asymptotically stable. Namely, all the infectious classes are decreasing to zero eventually for any initial value. The plots in Figure 1(a) and 1(b) verified this conclusion.

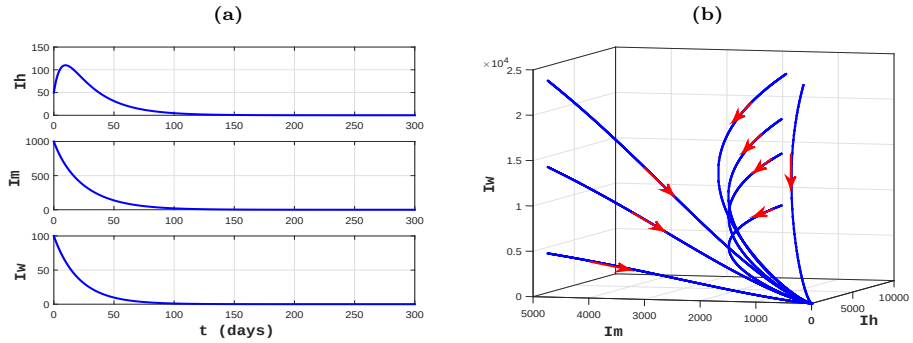

Figure 1. The stability of the disease-free equilibrium of model (2.1) without seasonal fluctuation, where $\mathcal{R}_0 \approx 0.2538 < 1$.

However, we choose $\beta_{mh} = \beta_{hm} = 8.475 \times 10^{-2}$, and other parameters are fixed as above. For these parameter values we get, $\mathcal{R}_0 \approx 1.5082 > 1$. That is, the disease-free equilibrium loses its stability and the disease is an outbreak, which is shown in Figure 2(a). In addition, numerical simulations are also carried out with a variety of initial conditions to check the influence of the initial infectious population sizes on the infectious population $I_H(t)$. To do so, we choose the initial values of infectious humans and infectious wild mosquitoes are $(I_h^0, I_m^0) = (50, 1000)$, $(100, 1000)$, $(50, 2000)$ and $(100, 2000)$ respectively. The plots in Figure 2(b) show that the infectious humans have obvious explosion in early phase if the initial value of infectious mosquitoes is relatively large. The numerical simulations also show that the size of infectious human is more sensitive to the size of infectious mosquitoes than the size of infectious humans. Therefore, reducing the population size of infectious mosquitoes (that is, the size of mosquitoes) is a useful method to control the transmission of the disease in the early days.

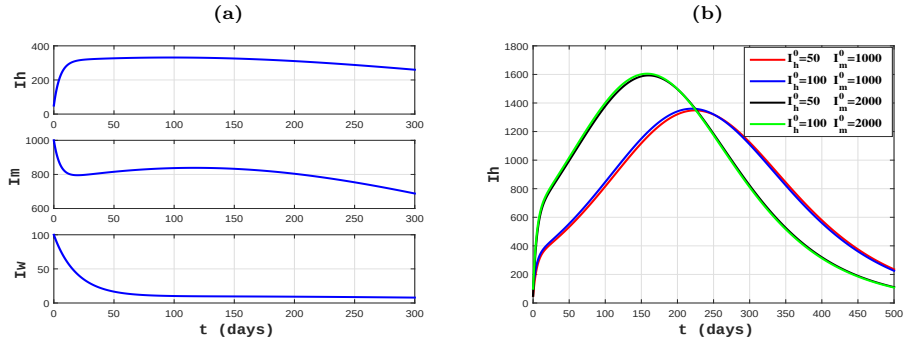

Figure 2. The dynamics behaviors of model (2.1) without seasonal fluctuation, where $\mathcal{R}_0 \approx 1.5082 > 1$: (a) the instability of disease-free equilibrium; (b) the sensitive of the initial values of infectious populations.

Next, we consider the sensitivity of threshold value $\mathcal{R}_0$. To better visualize the

sensitivity of $\mathcal{R}_0$, we choose $\beta_{mh} = \beta_{hm} = 3.475 \times 10^{-2}$, 5.475×10^{-2}, 7.475×10^{-2}, 8.475×10^{-2}, 9.475×10^{-2} and $\beta_{wh} = \beta_{hw} = 0.1 \times \beta_{mh}$ respectively. The plots in Figure 3(a) show that there will be a sharp outbreak of the number of infected human population with the increase of β_{mh} and β_{hm}. Therefore, reducing the infected rate from infectious humans to wild mosquitoes or wild mosquitoes to infectious humans (by using window screens, long-sleeved clothes and insecticide treated materials) is important and effective for controlling the disease and preventing large local outbreaks. Further, if we fixed $\beta_{mh} = \beta_{hm} = 7.475 \times 10^{-2}$ and choose $\beta_{wh} = \beta_{hw}$ are 0.1, 0.3, 0.5, 0.7 and 0.9 times to β_{mh} respectively. The plots in Figure 3(b) show that, as the values β_{wh} and β_{hw} increases, the number of infected human population significant increases and decremented. Therefore, the effectiveness of *Wolbachia* strains is of more importance to control the spread of Dengue virus.

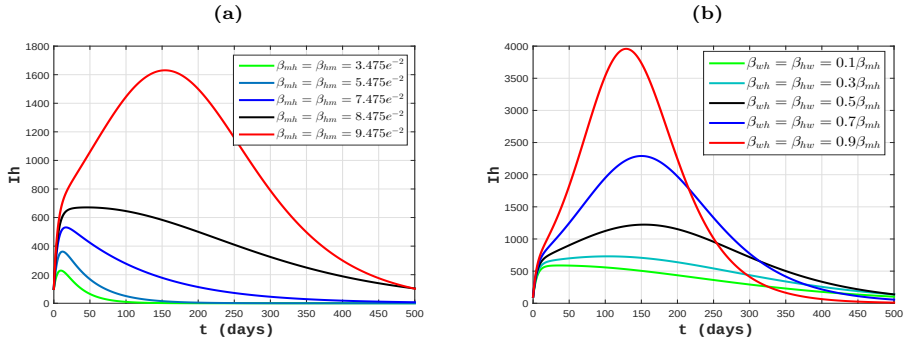

Figure 3. The sensitivity of threshold value $\mathcal{R}_0$: (a) the sensitivity of infected rate from infectious humans to wild mosquitoes; (b) the sensitivity of infected rate from infectious humans to *Wolbachia*-carrying mosquitoes.

Finally, for verifying the theoretical results of model (2.1) with seasonal fluctuation, we choose $\beta = 9.475 \times 10^{-2}$, $\beta_{mh}(t) = \beta_{hm}(t) = \beta + k\beta \sin(2\pi ft + \phi)$ and $\beta_{wh}(t) = \beta_{hw}(t) = 0.1\beta + k\beta \sin(2\pi ft + \phi)$, where $f = 1/30$, $k = 0.5$ and $\phi = 1$. The number of infectious humans has a frequency instability which are observed via numerical simulation as the seasonal fluctuation. Definitely, the intensity oscillation directly depends on the intensity of seasonal fluctuation, which is also consistent with the actual situation. This is shown in Figure 4(a). Further, if the background of model (2.1) is not considered, we choose $\beta_{mh}(t) = \beta_{hm}(t) = (0.375 + \alpha_1 sin(2\pi ft + \phi))$, $\beta_{wh}(t) = \beta_{hw}(t) = (0.1875 + \alpha_2 sin(2\pi ft + \phi))$, where $f = 1/30$, $\alpha_1 = 0.075$, $\alpha_2 = 0.01875$, $\phi = 0$, and $\mu_m = \mu_w = 1/15$, $\gamma_h = 1/100$. The plots in Figure 4(b) shown that model (2.1) admits different positive periodic solutions for different initial values. Numerical simulations imply that vector-born epidemic models with seasonal fluctuation have complex dynamical behaviors, which will be the problems we need to study further.

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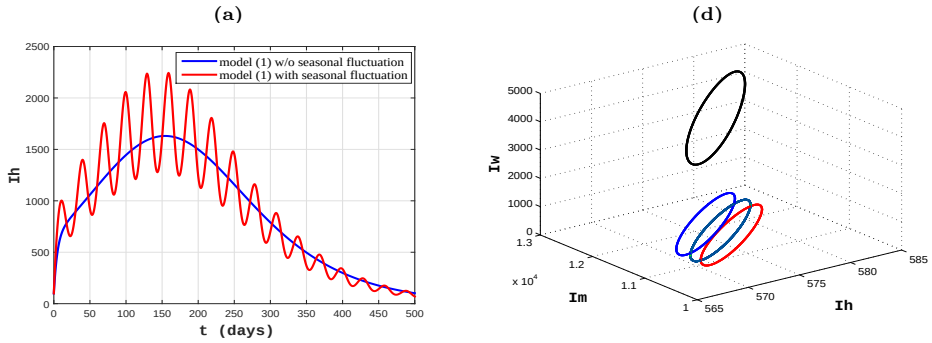

Figure 4. The complex dynamical behaviors of model (2.1) with seasonal fluctuation.

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