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A dengue epidemic model with the impact of media influence

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Abstract. In this paper, we establish an $S_hI_hA_hS_vI_vW$ model to investigate the impact of media communication on the transmission mechanism of dengue fever. Firstly, the basic reproduction number $\mathcal{R}_0$ of the model is obtained by using the method of the next generation matrix. It shows that disease-free equilibrium is globally asymptotically stable when $\mathcal{R}_0 < 1$; the disease is uniformly persistent when $\mathcal{R}_0 > 1$. Secondly, we select dengue fever case data from Guangdong Province from 2006 to 2019 for numerical simulations and predict its development trend. Finally, we conduct parameter sensitivity analysis, and the results show that increasing media publicity can to some extent reduce the number of patients.

§1 Introduction

Dengue fever is a mosquito-borne disease caused by four different serotypes of viruses, mainly transmitted by Aedes aegypti and Aedes albopictus [3, 26, 4]. After recovering from a specific dengue virus infection, patients can gain lifelong immunity against that specific virus serotype. However, this immunity can only provide partial and temporary protection from the infection of the other three serotypes of the virus [6]. In recent years, dengue fever, as a typical vector infectious disease, has frequently spread in Southeast Asia and around the world, especially in tropical climate countries, causing economic losses and inconvenience to people's lives. In 2022, the World Health Organization listed it as one of the ten potentially threatening diseases in 2019, and the current outbreaks of dengue fever in many countries confirm this result [30, 5]. This disease is considered the deadliest vector-borne epidemic after malaria. It is a self-limiting febrile disease, with symptoms ranging from asymptomatic to severe. Dengue fever symptoms may occur approximately 4-10 days after mosquito bites. Common symptoms are similar to influenza, including fever, nausea, muscle soreness, etc. Although there have been

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some suggested advances in the treatment of dengue fever virus, there is currently no specific drug available on the market [15].

The severity of dengue fever and various insect-borne diseases has prompted many researchers to use mathematical models to study the spread and control of diseases. For example, Fischer and Halstead established a mathematical model for the transmission mechanism of dengue fever in 1970 [13]. Subsequently, various dengue fever models were established and studied. Esteva and Vargas proposed a dengue fever SIR-SI model in 1998 [10]. They later enhanced this model by incorporating variable population size. Then, using competitive systems theory, additivity matrix, and central manifold theory, the global stability of the system equilibrium point was proved [11]. Afterwards, Massawe et al. studied an SITR (susceptibility, infection, treatment, rehabilitation) and ASI (aquatic, susceptibility, infection) epidemic models to describe the interaction between human and dengue mosquito populations [21]. Yang et al. considered the impact of pulse killing strategy on dengue infectious diseases [25]. In addition to the above models, Li et al. established a dengue fever type with both latent infection and vertical transmission and studied its dynamic behavior [20]. Altaf et al. published a mathematical model for dengue fever transmission and hospitalization to describe the dynamics of infection [1]. Driss proposed and analyzed a generalized stochastic dengue fever model that includes both mild and significant environmental disturbances [7].

The aforementioned literature examined the impact of population changes, vertical transmission, vaccination, disease incubation, patient isolation, pulse killing of media, and random factors on dengue disease. However, the influence of media on infectious diseases cannot be ignored nowadays. Many researchers have introduced different forms of media impact factor analysis to examine the impact of media coverage on infectious diseases [16, 24, 17, 18, 12, 23]. Liu, et al. [16] discussed an EIH model influenced by media, where the media influence factor function is $f(E,I,H)=e^{-a_1E-a_2I-a_3H}$, where E,I and H represent susceptible, infected and hospitalized individuals, respectively. Xiao et al. used a nonlinear function $f(I)=\frac{k}{(1+aI^2)}$ to represent the media influence factor function [24]. Besides, $f(I)=\frac{k}{S+aI}$ was used to represent the changes in people's behavior as the ratio of infected and susceptible individuals increases [17]. They concluded that when the basic reproduction number $\mathcal{R}_0$ is less than 1, the equilibrium state of disease elimination is globally asymptotically stable; When $\mathcal{R}_0$ is greater than 1, the equilibrium state of endemic diseases is globally asymptotically stable. Liu et al. described the saturation of the influence factor m using a nonsmooth function $f(x)=\begin{cases} e^{-mI} & 0<I\leq I_c; \\ e^{-mI_c} & I>I_c. \end{cases}$ This reflects the influence of media coverage on effective contact rate, ultimately reaching saturation value [18]. Reference [12] introduced a novel SEIS epidemic model with the impact of media. It applied an exponential function $e^{-\alpha M}$ in which α determines how effectively the disease-related messages can affect the transmission rate. They derived the occurrence of forward, backward, and Hopf bifurcations. The results showed that media can serve as a good indicator for controlling the occurrence and spread of the epidemic. In addition, Sun et al. employed a nonlinear function $\beta_i(I_i)=a_i-b_if_i(I_i)$ to represent the effective contact rate of

infectious diseases under the media influence [23]. These articles show that while media influence does not play a significant role in determining the occurrence of a disease outbreak, it exerts a substantial impact on the extent of infectious disease transmission. The media is an important channel for information dissemination, and it can greatly influence the behavior and attitudes of the public. For instance, platforms like Weibo and public accounts can report the number of infected and deceased cases, symptoms, and preventive measures for diseases. This can raise awareness among susceptible individuals to take proactive measures such as purchasing mosquito nets and insecticides to reduce disease transmission. Without media influence, many unaware individuals may become infected due to a lack of disease information. In conclusion, media coverage significantly reduces infection rates and is instrumental in disease control. Therefore, it is meaningful to study the dengue fever model under the influence of media.

In this paper, we introduce the influence factor of media coverage and are inspired by relevant literature. In reality, when an infected individual is reported in a particular region, media coverage alerts susceptible individuals to potential dangers, prompting them to adopt appropriate preventive measures, thus decreasing the contact rate between vectors and hosts. Consequently, employing the same nonlinear function as in [21], we define the effective contact rate function as $\theta - \alpha g(I_h)$. Here, θ denotes the mosquito bite rate, α signifies the maximum reduction effect of media coverage on effective contact rates. The greater the number of reported infected individuals, the less susceptible individuals are likely to come into contact with mosquitoes. It is assumed that media coverage increases with the number of infected individuals, hence $g'(I_h) \geq 0$. Furthermore, given the limited capacity to contact infected individuals, $\lim\limits_{t\to\infty} g(I_h) = 1$, and we must have $g(0) = 0$. Media coverage cannot entirely prevent transmission,$\theta > \alpha$. In short, the functional $g(I_h)$ satisfies

$$g(0) = 0,\ g'(I_h) \geq 0,\ \lim_{t\to\infty} g(I_h) = 1$$

A dengue fever infectious disease model with conscious compartments is established. For this model, we mainly study the promotional plans executed by the media and the impact of controlling vector populations through insecticides.

§2 Mathematical Modeling

According to the transmission mechanism of dengue fever, The model is divided into 6 compartments. $S_h(t)$ represents susceptible humans at time t. $I_h(t)$ represents infected humans and $A_h(t)$ represents aware humans at time t. Therefore, the total human population $N_h(t) = S_h(t) + I_h(t) + A_h(t)$. $S_v(t)$ represents susceptible vectors and $I_v(t)$ represents infected vectors at time t. Then the total vector population $N_v(t) = S_v(t) + I_v(t)$. $W(t)$ represents the number of messages that infected humans provide about epidemic disease at time t. It follows that the transmission process of dengue fever is described by the following nonlinear ordinary differential

equations

$$\begin{cases} \dfrac{\mathrm{d}S_h}{\mathrm{d}t} = & C_h - (\theta - \alpha g(I_h))\beta_1 S_h I_v - \beta_2 W S_h + \delta A_h - \mu_h S_h, \\ \dfrac{\mathrm{d}I_h}{\mathrm{d}t} = & (\theta - \alpha g(I_h))\beta_1 S_h I_v - (\alpha_1 + \mu_h) I_h, \\ \dfrac{\mathrm{d}A_h}{\mathrm{d}t} = & \beta_2 W S_h - \delta A_h - \mu_h A_h, \\ \dfrac{\mathrm{d}S_v}{\mathrm{d}t} = & C_v - (\theta - \alpha g(I_h))\beta_3 S_v I_h - (\mu_1 + \mu_v) S_v, \\ \dfrac{\mathrm{d}I_v}{\mathrm{d}t} = & (\theta - \alpha g(I_h))\beta_3 S_v I_h - (\mu_1 + \mu_v) I_v, \\ \dfrac{\mathrm{d}W}{\mathrm{d}t} = & \Lambda + \sigma_1 I_h - \sigma_2 W. \end{cases} \tag{2.1}$$

Here θ is the number of mosquito bites per unit time per human. β_1 symbolizes the transmission rate of getting infection from mosquitoes. Thus, the rate at which a susceptible host contracts an infection from mosquitoes is denoted as $\theta\beta_1$. Meanwhile, β_3 represents the transmission rate of infection from humans to mosquitoes. Then $\theta\beta_3$ is the rate at which a mosquito gets infection from humans. The biological interpretation of all parameters is listed in Table 1.

It is easy to know that the solution of the model (2.1) is non-negative when the initial data are non-negative. From biological considerations, we study (2.1) in the closed set

$$\Gamma = \Big\{(S_h, I_h, A_h, S_v, I_v, W) \in \mathbb{R}^6_+ : \quad S_h + I_h + A_h \le \tfrac{C_h}{\mu_h}, S_v + I_v \le \tfrac{C_v}{\mu_1 + \mu_v}, \\ W \le \tfrac{\sigma_1 C_h + \Lambda\mu_h}{\sigma_2 \mu_h}\Big\}.$$

Table 1. Descriptions of the parameters in model (2.1).

Parameters	Description
C_h	The input rate of humans
C_v	The input rate of mosquitoes
θ	Mosquito bite rate, in other, the average number of bites per mosquito per day
β_1	Transmission rate of dengue from infected mosquito to human
β_3	Transmission rate of dengue from human to mosquito
β_2	Rate of propagation of awareness
δ	Rate at which individuals lose awareness due to memory fading
μ_h	Natural death rate for humans
μ_v	Natural death rate for mosquitoes
α_1	Disease death rate of infected humans
σ_2	Rate at which messages become outdated
σ_1	Rate at which infected individuals send information about the epidemic disease
α	The maximum value of the reduction in the contact rate
μ_1	Pesticide control for mosquito population
Λ	Value of baseline number of programs maintained in a system

§3 Threshold Dynamics

In this section, we consider the existence and stability of the disease-free equilibrium.

To study the dynamic properties of a system, the first step is to determine the equilibrium point. This section will discuss the existence and stability of the disease-free equilibrium of the

system (2.1). System (2.1) always has a unique disease-free and stable $E_0(S_h^0, 0, A_h^0, S_v^0, 0, W^0)$, where

$$S_h^0 = \frac{C_h(\delta+\mu_h)}{\mu_h\beta_2 W^0+\mu_h(\delta+\mu_h)}, \quad A_h^0 = \frac{\beta_2 W^0 C_h}{(\mu_h\beta_2 W^0+\mu_h(\delta+\mu_h))},$$
$$S_v^0 = \frac{C_v}{\mu_1+\mu_v}, \qquad\qquad W^0 = \frac{\Lambda}{\sigma_2}.$$

Using the next generation matrix method [9], we derive the expression of the basic reproduction number $\mathcal{R}_0$. Here we have the following matrix of new infection $\mathcal{F}(x)$, and the matrix of transfer $\mathcal{V}(x)$. Let $x = (I_h, A_h, I_v, W)^{\mathrm{T}}$, then $\mathcal{F}(x)$ and $\mathcal{V}(x)$ are given by

$$\mathcal{F}(x) = \begin{pmatrix} (\theta-\alpha g(I_h))\beta_1 S_h I_v \\ 0 \\ (\theta-\alpha g(I_h))\beta_3 S_v I_h \\ 0 \end{pmatrix}, \ \mathcal{V}(x) = \begin{pmatrix} (\alpha_1+\mu_h)I_h \\ \delta A_h + \mu_h A_h - \beta_2 W S_h \\ (\mu_1+\mu_v)I_v \\ \sigma_2 W - \Lambda - \sigma_1 I_h \end{pmatrix}.$$

The Jacobi matrices of $\mathcal{F}(x)$ and $\mathcal{V}(x)$ at the disease-free equilibrium E_0 respectively are,

$$\mathcal{DF}(E_0) = \begin{pmatrix} 0 & 0 & \theta\beta_1 S_h^0 & 0 \\ 0 & 0 & 0 & 0 \\ \theta\beta_3 S_v^0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \ \mathcal{DV}(E_0) = \begin{pmatrix} \alpha_1+\mu_h & 0 & 0 & 0 \\ 0 & \delta+\mu_h & 0 & -\beta_2 S_h^0 \\ 0 & 0 & \mu_1+\mu_v & 0 \\ -\sigma_1 & 0 & 0 & \sigma_2 \end{pmatrix}$$

and

$$\mathcal{DV}(E_0)^{-1} = \begin{pmatrix} \frac{1}{\alpha_1+\mu_h} & 0 & 0 & 0 \\ \frac{\beta_2\sigma_1 S_h^0}{(\alpha_1+\mu_h)(\delta+\mu_h)\sigma_2} & \frac{1}{\delta+\mu_h} & 0 & \frac{\beta_2 S_h^0}{(\delta+\mu_h)\sigma_2} \\ 0 & 0 & \frac{1}{\mu_1+\mu_v} & 0 \\ \frac{\sigma_1}{\sigma_2(\alpha+\mu_h)} & 0 & 0 & \frac{1}{\sigma_2} \end{pmatrix}.$$

Therefore, the basic reproduction number $\mathcal{R}_0$ is

$$\mathcal{R}_0 = \rho(\mathcal{DF}(E_0)\mathcal{DV}(E_0)^{-1}) = \max\{|\lambda|\,; \lambda \in \sigma(\mathcal{DF}(E_0)\mathcal{DV}(E_0)^{-1})\},$$

where $\rho(\cdot)$ and $\sigma(\cdot)$ denote the spectral radius and the set of eigenvalues of a matrix, respectively. Thus, the basic reproduction number denoted by $\mathcal{R}_0$ is given by

$$\mathcal{R}_0 = \sqrt{\frac{\theta^2\beta_1\beta_3 S_h^0 S_v^0}{(\mu_1+\mu_v)(\alpha_1+\mu_h)}}.$$

Theorem 1 *The disease-free equilibrium E_0 of the system (2.1) is locally asymptotically stable when $\mathcal{R}_0 < 1$, and is unstable when $\mathcal{R}_0 > 1$.*

Proof. The Jacobi matrix of system (2.1) at the disease-free equilibrium E_0 is

$$J_0 = \begin{pmatrix} -\beta_2 W^0 - \mu_h & 0 & \delta & 0 & -\theta\beta_1 S_h^0 & -\beta_2 S_h^0 \\ 0 & -(\alpha_1+\mu_h) & 0 & 0 & \theta\beta_1 S_h^0 & 0 \\ \beta_2 W^0 & 0 & \delta-\mu_h & 0 & 0 & 0 \\ 0 & -\theta\beta_3 S_v^0 & 0 & -(\mu_1+\mu_v) & 0 & 0 \\ 0 & \theta\beta_3 S_v^0 & 0 & 0 & -(\mu_1+\mu_v) & 0 \\ 0 & \sigma_1 & 0 & 0 & 0 & -\sigma_2 \end{pmatrix}.$$

The characteristic equation of the system (2.1) at the disease-free equilibrium E_0 is

$$\begin{aligned}&(\lambda+\sigma_2)(\lambda+(\mu_1+\mu_v))[(\lambda+\alpha_1+\mu_h)(\lambda+\mu_1+\mu_v)-\theta^2\beta_1\beta_3S_h^0S_v^0]\\&[(\lambda+\beta_2W^0+\mu_h)(\lambda+\delta+\mu_h)-\beta_2W^0\delta]=0.\end{aligned}\tag{3.1}$$

Thus, the two eigenvalues of (3.1) are $\lambda_1=-\sigma_2$, $\lambda_2=-(\mu_1+\mu_v)$ and the others are determined by

$$\lambda^2+a_1\lambda+a_2=0,\tag{3.2}$$

$$\lambda^2+b_1\lambda+b_2=0.\tag{3.3}$$

Here

$$a_1=(\alpha+\mu_h+\mu_1+\mu_v),\ a_2=(\alpha_1+\mu_h)(\mu_1+\mu_v)-\theta^2\beta_1\beta_3S_h^0S_v^0$$

and

$$b_1=(\beta_2W^0+2\mu_h+\delta),\ b_2=\beta_2W^0\mu_h+\mu_h\delta+\mu_h^2.$$

Obviously, $a_1>0$, $b_1>0$, $b_2>0$. When $\mathcal{R}_0<1$, we can get $(\alpha_1+\mu_h)(\mu_1+\mu_v)>\theta^2\beta_1\beta_3S_h^0S_v^0$. Thus $a_2>0$. According to Routh-Hurwitz criteria, all the eigenvalues of equations (3.2) and (3.3) have negative real parts. Therefore, the disease-free equilibrium E_0 of the system (2.1) is locally asymptotically stable when $\mathcal{R}_0<1$. When $\mathcal{R}_0>1$, the disease-free equilibrium E_0 of system (2.1) is unstable. This completes the proof of Theorem 1.

Theorem 2 *The disease-free equilibrium E_0 is globally attractive for system (2.1) when $R_0<1$.*

Proof. From the first, third and fourth equations of system (2.1), we have

$$\begin{cases}S_h' &\le C_h-\beta_2WS_h-\mu_hS_h+\delta A_h,\\ A_h' &\le \beta_2WS_h-(\mu_h+\delta)A_h,\\ S_v' &\le C_v-(\mu_1+\mu_v)S_v.\end{cases}\tag{3.4}$$

For any $\varepsilon>0$, by the comparison principle, there exists a positive number $T_1=T_1(\varepsilon)$ such that the inequality

$$S_v(t)\le S_v^0+\varepsilon$$

are true for all $t\ge T_1$.

Consider an auxiliary system as follows

$$\begin{cases}\widehat{S}_h'(t) &=C_h-\beta_2W\widehat{S}_h-\mu_h\widehat{S}_h+\delta\widehat{A}_h,\\ \widehat{A}_h'(t) &=\beta_2W\widehat{S}_h-(\mu_h+\delta)\widehat{A}_h.\end{cases}\tag{3.5}$$

Write $\widehat{D}_h=\widehat{S}_h+\widehat{A}_h$. By (3.5), we can get $\widehat{D}_h'(t)=C_h-\mu_h\widehat{D}_h$. Then $\lim\limits_{t\to\infty}\widehat{D}_h(t)=\dfrac{C_h}{\mu_h}$. From the sixth equation of system (2.1), we have $W'(t)\ge\Lambda-\sigma_2W$, thus $\liminf\limits_{t\to\infty}W(t)\ge\dfrac{\Lambda}{\sigma_2}$, Substituting $\widehat{D}_h$ into the first equations of system (3.5) yields

$$\widehat{S}_h'(t)\le C_h-\frac{\beta_2\Lambda}{\sigma_2}\widehat{S}_h-\mu_h\widehat{S}_h+\delta(\widehat{D}_h-\widehat{S}_h).$$

Consider the following auxiliary system

$$\widetilde{S}_h'=C_h+\delta\widehat{D}_h-(\frac{\beta_2\Lambda}{\sigma_2}+\mu_h+\delta)\widetilde{S}_h.\tag{3.6}$$

Multiplying by $e^{(\frac{\beta_2\Lambda}{\sigma_2}+\mu_h+\delta)t}$ on both sides of (3.6), we have

$$(e^{(\frac{\beta_2\Lambda}{\sigma_2}+\mu_h+\delta)t}\widetilde{S}_h)'=e^{(\frac{\beta_2\Lambda}{\sigma_2}+\mu_h+\delta)t}(C_h+\delta\widehat{D}_h).$$

Thus

$$\widetilde{S}_h(t) = \frac{\int_0^t e^{(\frac{\beta_2\Lambda}{\sigma_2}+\mu_h+\delta)s}(C_h+\delta\widehat{D}_h)\mathrm{d}s + \widetilde{S}_h(0)}{e^{(\frac{\beta_2\Lambda}{\sigma_2}+\mu_h+\delta)t}},$$

and

$$\begin{aligned}\lim_{t\to\infty}\widetilde{S}_h(t) &= \lim_{t\to\infty}\frac{e^{(\frac{\beta_2\Lambda}{\sigma_2}+\mu_h+\delta)t}(C_h+\delta\widehat{D}_h)}{e^{(\frac{\beta_2\Lambda}{\sigma_2}+\mu_h+\delta)t}(\frac{\beta_2\Lambda}{\sigma_2}+\mu_h+\delta)}\\ &= \lim_{t\to\infty}\frac{C_h+\delta\widehat{D}_h}{\frac{\beta_2\Lambda}{\sigma_2}+\mu_h+\delta}\\ &= \frac{C_h(\delta+\mu_h)}{\frac{\beta_2\Lambda}{\sigma_2}\mu_h+\mu_h(\delta+\mu_h)}.\end{aligned}$$

Therefore $\widetilde{S}_h \to S_h^0$ as $t\to\infty$. By the comparison principle, $S_h(t)\le\widehat{S}_h(t)\le\widetilde{S}_h(t)$ as $S_h(t_0)=\widehat{S}_h(t_0)=\widetilde{S}_h(t_0)$. So we have $\limsup_{t\to\infty} S_h(t)\le S_h^0$. It follows that for any $\varepsilon>0$ there exists a positive number $T_2=T_2(\varepsilon)$ such that the inequality

$$S_h(t)\le S_h^0+\varepsilon$$

are true for all $t\ge T_2$.

When $\mathcal{R}_0<1$, we have $\theta^2\beta_1\beta_3S_h^0S_v^0<(\mu_1+\mu_v)(\alpha_1+\mu_h)$. Thus, we can choose three numbers $\varepsilon>0$, $r_1>0$ and $r_2>0$ such that

$$\frac{\theta\beta_1(S_h^0+\varepsilon)}{\mu_1+\mu_v}<\frac{r_2}{r_1}<\frac{\alpha_1+\mu_h}{\theta\beta_3(S_v^0+\varepsilon)}.$$

Then we define a Lyapunov function $V(t)=r_1I_h+r_2I_v$. It is obvious that $V(t)\ge0$, and the equality holds if and only if $I_h(t)=I_v(t)=0$. Solving the derivative of $V(t)$ along system (2.1) gives

$$\begin{aligned}\frac{\mathrm{d}V}{\mathrm{d}t} &= r_1((\theta-\alpha g(I_h))\beta_1S_hI_v-(\alpha_1+\mu_h)I_h)+r_2((\theta-\alpha g(I_h))\beta_3S_vI_h-(\mu_1+\mu_v)I_v)\\ &\le r_1\theta\beta_1(S_h^0+\varepsilon)I_v-r_1(\alpha_1+\mu_h)I_h+r_2\theta\beta_3(S_v^0+\varepsilon)I_h-r_2(\mu_1+\mu_v)I_v\\ &\le -[r_2(\mu_1+\mu_v)-r_1\theta\beta_1(S_h^0+\varepsilon)]I_v-[r_1(\alpha_1+\mu_h)-r_2\theta\beta_3(S_v^0+\varepsilon)]I_h\\ &\le -\eta V(t),\end{aligned}$$

for all $t\ge\max\{T_1,T_2\}$, where

$$\eta=\min\left\{\frac{r_2(\mu_1+\mu_v)-r_1\theta\beta_1(S_h^0+\varepsilon)}{r_2},\ \frac{r_1(\alpha+\mu_h)-r_2\theta\beta_3(S_v^0+\varepsilon)}{r_1}\right\}.$$

Hence $V(t)\to0$ as $t\to\infty$, the inequality $V'(t)\le0$ is valid as $\mathcal{R}_0<1$.

Let $\Omega=\left\{(S_h,I_h,A_h,S_v,I_v,W)\in\Gamma\middle|\frac{\mathrm{d}V}{\mathrm{d}t}=0\right\}$. On Ω, system (2.1) reduces to the system below

$$\begin{cases}\dfrac{\mathrm{d}S_h}{\mathrm{d}t} = C_h-\beta_2WS_h+\delta A_h-\mu_hS_h,\\ \dfrac{\mathrm{d}A_h}{\mathrm{d}t} = \beta_2WS_h-\delta A_h-\mu_hA_h,\\ \dfrac{\mathrm{d}S_v}{\mathrm{d}t} = C_v-(\mu_1+\mu_v)S_v,\\ \dfrac{\mathrm{d}W}{\mathrm{d}t} = \Lambda-\sigma_2W.\end{cases}\tag{3.7}$$

Obviously, from the third and the fourth equations of system (3.7), we have $S_v \to S_v^0$, $W \to W^0$, as $t \to \infty$. By the first and the second equations of system (3.7), we can get

$$S_h'(t) + A_h'(t) = C_h - \mu_h(S_h + A_h),$$

so $\lim\limits_{t\to\infty} S_h(t) + A_h(t) = \dfrac{C_h}{\mu_h}$. Then by the second equation of system(3.7), we have

$$\begin{aligned} \frac{\mathrm{d}A_h}{\mathrm{d}t} &= \beta_2 W(t)(S_h(t) + A_h(t) - A_h(t)) - (\mu_h + \delta)A_h(t) \\ &= \beta_2 W(t)(S_h(t) + A_h(t)) - (\beta_2 W(t) + \mu_h + \delta)A_h(t). \end{aligned} \tag{3.8}$$

Multipiying by $e^{\int_0^t(\beta_2 W(s)+\mu_h+\delta)\mathrm{d}s}$ on both sides of (3.8), we get

$$(e^{\int_0^t(\beta_2 W(s)+\mu_h+\delta)\mathrm{d}s}A_h(t))' = e^{\int_0^t(\beta_2 W(s)+\mu_h+\delta)\mathrm{d}s}\beta_2 W(t)(S_h(t) + A_h(t)).$$

By integrating on both sides, we have

$$A_h(t) = \frac{A_h(0) + \int_0^t e^{\int_0^\tau(\beta_2 W(s)+\mu_h+\delta)\mathrm{d}s}\beta_2 W(\tau)(S_h(\tau) + A_h(\tau))\mathrm{d}\tau}{e^{\int_0^t(\beta_2 W(s)+\mu_h+\delta)\mathrm{d}s}}.$$

Using L'Hospital's rule, we can get $A_h(t) \to A_h^0$ as $t \to \infty$.

By the LaSalle invariance principle [2], the disease-free equilibrium E^0 is globally attractive in Γ. Combining the stability of E^0, we deduce that E^0 is globally asymptotically stable in Γ. The proof is completed.

§4 Permanence of Disease

In this section, we investigate the permanence of the disease for system (2.1).

Theorem 3 *Given any solution$(S_h(t), I_h(t), A_h(t), S_v(t), I_v(t), W(t))$ of (2.1) with initial values $S_h(0) \geq 0$, $I_h(0) > 0$, $A_h(0) \geq 0$, $S_v(0) \geq 0$, $I_v(0) > 0$, $W(0) \geq 0$. Then the system has at least one positive equilibrium $E^* = (S_h^*, I_h^*, A_h^*, S_v^*, I_v^*, W^*)$ and exists an $\varepsilon > 0$ such that*

$$\liminf_{t\to\infty} I_h(t) \geq \varepsilon, \ \liminf_{t\to\infty} I_v(t) \geq \varepsilon.$$

as $\mathcal{R}_0 > 1$.

Proof. Define

$$\begin{aligned} \mathcal{X} &= \{(S_h, I_h, A_h, S_v, I_v, W) \in \mathbb{R}^6 | S_h \geq 0,\ I_h \geq 0,\ A_h \geq 0,\ S_v \geq 0,\ I_v \geq 0,\ W \geq 0\}, \\ \mathcal{X}_0 &= \{(S_h, I_h, A_h, S_v, I_v, W) \in \mathcal{X} | I_h > 0,\ I_v > 0\}, \\ \partial\mathcal{X}_0 &= \mathcal{X} \setminus \mathcal{X}_0. \end{aligned}$$

Next, we demonstrate that system (2.1) is uniformly persistent with regard to $(\mathcal{X}_0, \partial\mathcal{X}_0)$ [27]. It is evident that $\mathcal{X}$ and $\mathcal{X}_0$ are positively invariant in relation to (2.1).

Let

$$\begin{aligned} M_\partial = \{(S_h(0), I_h(0), A_h(0), S_v(0), I_v(0), W(0)) : \ & (S_h(t), I_h(t), A_h(t), \\ & S_v(t), I_v(t), W(t)) \in \partial\mathcal{X}_0, t \geq 0\}. \end{aligned}$$

Next we claim that

$$M_\partial = \{(S_h, 0, A_h, S_v, 0, W) | S_h \geq 0, A_h \geq 0, S_v \geq 0, W \geq 0\}. \tag{4.1}$$

Obviously,

$$\{(S_h, 0, A_h, S_v, 0, W) | S_h \geq 0, A_h \geq 0, S_v \geq 0, W \geq 0\} \subseteq M_\partial.$$

Then it only needs to prove

$$M_\partial \subseteq \{(S_h, 0, A_h, S_v, 0, W) | S_h \geq 0, A_h \geq 0, S_v \geq 0, W \geq 0\}.$$

Suppose that $(S_h(0), I_h(0), A_h(0), S_v(0), I_v(0), W(0)) \in M_\partial$. It suffices to show $I_h(t) = 0$ and $I_v(t) = 0$ for any $t \geq 0$. If it is not true, then exists a $t_0 \geq 0$ that makes one of the following true:

$$\text{(i)} \ \ I_h(t_0) > 0, \quad \text{(ii)} \ \ I_v(t_0) > 0.$$

If (i) holds, then solving the model (2.1) yields

$$I_h(t) \geq I_h(t_0) e^{-(\alpha_1+\mu_h)(t-t_0)} > 0, \forall t \geq t_0.$$

From the fourth equation of system (2.1), we have

$$S_v' = C_v - [(\theta - \alpha g(I_h))\beta_3 I_h + (\mu_1 + \mu_v)]S_v. \tag{4.2}$$

Multiplying by $e^{\int_0^t [(\theta-\alpha g(I_h))\beta_3 I_h + (\mu_1+\mu_v)]ds}$ on both sides of (4.2), we can get $S_v(t) > 0$ for all $t > 0$. From the fifth equation of system (2.1), we have

$$I_v'(t) \geq (\theta - \alpha)\beta_3 S_v I_h - (\mu_1 + \mu_v) I_v. \tag{4.3}$$

Multiplying by the integral factor $e^{(\mu_1+\mu_v)t}$ and integrating from t_0 to t on both sides of (4.3), we can get

$$e^{(\mu_1+\mu_v)t} I_v(t) = e^{(\mu_1+\mu_v)t_0} I_v(t_0) + \int_{t_0}^{t} e^{(\mu_1+\mu_v)\tau}(\theta - \alpha)\beta_3 S_v(\tau) I_h(\tau) d\tau.$$

Therefore, $I_v(t) > 0$ for all $t > t_0$. Hence, $(S_h(t), I_h(t), A_h(t), S_v(t), I_v(t), W(t)) \notin \partial\mathcal{X}_0$ for all $t > t_0$, This is contradiction with $(S_h(0), I_h(0), A_h(0), S_v(0), I_v(0), W(0)) \in M_\partial$. This proves (4.1). If (ii) is true, we also obtain a similar contradiction.

Let

$$\begin{aligned} \Phi = \ & \bigcup \{\omega(S_h(0), I_h(0), A_h(0), S_v(0), I_v(0), W(0)) : \\ & (S_h(0), I_h(0), A_h(0), S_v(0), I_v(0), W(0)) \in M_\partial\}, \end{aligned}$$

where $\omega(S_h(0), I_h(0), A_h(0), S_v(0), I_v(0), W(0))$ is the omega limit set of solutions to (2.1) starting in $(S_h(0), I_h(0), A_h(0), S_v(0), I_v(0), W(0))$. Limiting (2.1) on M_∂ gets

$$\begin{cases} \dfrac{\mathrm{d}S_h}{\mathrm{d}t} = & C_h - \beta_2 W S_h + \delta A_h - \mu_h S_h, \\ \dfrac{\mathrm{d}A_h}{\mathrm{d}t} = & \beta_2 W S_h - \delta A_h - \mu_h A_h, \\ \dfrac{\mathrm{d}S_v}{\mathrm{d}t} = & C_v - (\mu_1 + \mu_v) S_v, \\ \dfrac{\mathrm{d}W}{\mathrm{d}t} = & \Lambda - \sigma_2 W. \end{cases} \tag{4.4}$$

The existence of a unique equilibrium $(S_h^0, A_h^0, S_v^0, W^0)$ in system (4.4) is easily verified. Therefore $E_0(S_h^0, 0, A_h^0, S_v^0, 0, W^0)$ is the unique equilibrium of (2.1) in M_∂. The globally asymptotically stable state of $(S_h^0, A_h^0, S_v^0, W^0)$ can also be easily verified. Consequently, we have $\Phi = \{E_0\}$. And the singleton E_0 is a covering of Φ, which is isolated and is acyclic. Lastly, if we demonstrate that E_0 is a weak repeller for $\mathcal{X}_0$, the proof will be completed, i.e.

$$\limsup_{t\to\infty} \mathrm{d}(\Psi(t), E_0) > 0,$$

where $\Psi(t) = (S_h(t), I_h(t), A_h(t), S_v(t), I_v(t), W(t))$ is a solution with a initial value in $\mathcal{X}_0$. By

the proof of [28, Theorem 4], we need only to prove $M^s(E_0)\cap\mathcal{X}_0=\emptyset$ where $M^s(E_0)$ is the stable manifold of E_0. If that is not true, then there is a solution $(S_h(t),I_h(t),A_h(t),S_v(t),I_v(t),W(t))$ in $\mathcal{X}_0$, such that

$$\begin{aligned}&\lim_{t\to\infty}S_h(t)=S_h^0,\quad \lim_{t\to\infty}I_h(t)=0,\quad \lim_{t\to\infty}A_h(t)=A_h^0,\\&\lim_{t\to\infty}S_v(t)=S_v^0,\quad \lim_{t\to\infty}I_v(t)=0,\quad \lim_{t\to\infty}W(t)=W^0.\end{aligned}\tag{4.5}$$

The inequality $\mathcal{R}_0>1$ is equal to $\theta^2\beta_1\beta_3S_h^0S_v^0>(\mu_1+\mu_v)(\alpha_1+\mu_h)$. Thus, we can choose $\varepsilon>0,\ \rho_1>0$ and $\rho_2>0$ so

$$\frac{\alpha_1+\mu_h}{(\theta-\alpha g(\varepsilon))\beta_3(S_v^0-\varepsilon)}<\frac{\rho_2}{\rho_1}<\frac{(\theta-\alpha g(I_h))\beta_1(S_h^0-\varepsilon)}{\mu_1+\mu_v}.$$

For the above-mentioned $\varepsilon>0$, by (4.5), there exists a $T>0$ such that

$$S_h^0-\varepsilon<S_h(t)<S_h^0+\varepsilon,\ 0\le I_h(t)<\varepsilon,\ S_v^0-\varepsilon<S_v(t)<S_v^0+\varepsilon$$

for all $t\ge T$.

Let $L(t)=\rho_1I_h(t)+\rho_2I_v(t)$. The derivative of $V(t)$ along the solution $(S_h(t),I_h(t),A(t),S_v(t),I_v(t),W(t))$ is given by

$$\begin{aligned}L'(t)=&\ \rho_1((\theta-\alpha g(I_h))\beta_1S_hI_v-(\alpha_1+\mu_h)I_h)+\rho_2((\theta-\alpha g(I_h))\beta_3S_vI_h-(\mu_1+\mu_v)I_v)\\\ge&\ \rho_1(\theta-\alpha g(\varepsilon))\beta_1(S_h^0-\varepsilon)I_v-\rho_1(\alpha_1+\mu_h)I_h\\&+\rho_2(\theta-\alpha g(\varepsilon))\beta_3(S_v^0-\varepsilon)I_h-\rho_2(\mu_1+\mu_v)I_v\\=&\ [\rho_2(\theta-\alpha g(\varepsilon))\beta_3(S_v^0-\varepsilon)-\rho_1(\alpha_1+\mu_h)]I_h\\&+[\rho_1(\theta-\alpha g(\varepsilon))\beta_1(S_h^0-\varepsilon)-\rho_2(\mu_1+\mu_v)]I_v\\\ge&\ \rho L(t)\end{aligned}$$

for all $t\ge t$, where

$$\rho=$$

$$\min\left\{\frac{\rho_2(\theta-\alpha g(\varepsilon))\beta_3(S_v^0-\varepsilon)-\rho_1(\alpha_1+\mu_h)}{\rho_1},\frac{[\rho_1(\theta-\alpha g(\varepsilon))\beta_1(S_h^0-\varepsilon)-\rho_2(\mu_1+\mu_v)}{\rho_2}\right\}>0.$$

Hence $L(t)\to\infty$ as $t\to\infty$, which contradicts to the boundedness of $L(t)$. It implies the uniform persistence stated in the theorem.

From[29, Theorem 2.4], it can be concluded that system (2.1) has at least one equilibrium point $E^*(S_h^*,I_h^*,A_h^*,S_v^*,I_v^*,W^*)\in X_0$. In fact, from $S_h^*>0$, E^* is a positive equilibrium point of the system. This completes the proof of Theorem 3.

Remark From Theorem 2 and 3, we know that $\mathcal{R}_0$ plays a very important role in the infectious disease system. It characterizes whether an epidemic will break out. When $\mathcal{R}_0<1$, the disease will go to extinction; when $\mathcal{R}_0>1$ the disease will develop endemic diseases. These results indicate that the basic reproduction number can be used to control the spread of dengue fever disease.

§5 Numerical Simulations

In this section, based on the real dengue fever data from the Public Health Science Data Center(see Table 2 and Fig.1) [31], model (2.1) is used to numerically simulate the cumulative

number of dengue fever patients in Guangdong Province from 2006 to 2019, predict the number of dengue fever patients from 2020 to 2023, and perform sensitivity analysis on some parameters. Finally, a strategy for controlling dengue fever is proposed based on theoretical and numerical simulation results.

Table 2. Number of newly diagnosed dengue fever patients in Guangdong Province.

Year	2006	2007	2008	2009	2010	2011	2012
Number	1010	397	87	19	139	49	474
Year	2013	2014	2015	2016	2017	2018	2019
Number	2894	45189	1683	544	1662	3315	6045

At the beginning of 2006, the population of Guangdong Province was approximately 91940000 [32]. So it is assumed that $S(0) = 91940000$. The birth rate of Guangdong Province in 2006 was 0.01178, and the natural mortality rate was 0.00449 [33], So $C_h = 91940000 \times 0.01178 \approx 1083053$. Firstly, we select $g(I_h) = \frac{I_h}{m+I_h}$, and since there was a sudden increase in dengue fever patients in Guangdong Province in 2014, we use the least square and piecewise fitting system to fit the cumulative number of dengue fever patients from 2006 to 2019 (See Fig.2 and Fig.3). In order to obtain the parameter values of model (2.1) (See Table **??** for details). We use the least squares method (LMS) to fit the cumulative number of dengue fever patients from 2006 to 2019 (See Fig.2 and Fig.3). The parameter values of model (2.1) are listed in Table **??** and Table 4.

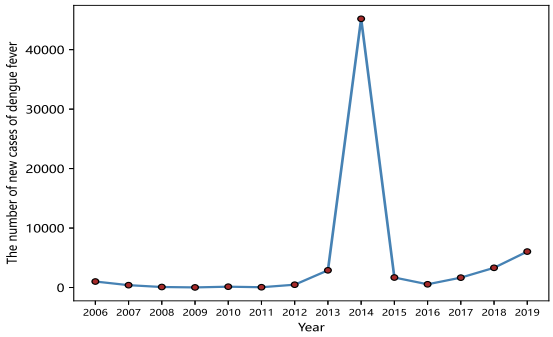

Fig 1. Number of newly diagnosed dengue fever patients in Guangdong Province from 2006 to 2019.

In 2020, the World Health Organization formulated a global strategy for the prevention and control of dengue fever based on the latest progress in the epidemic situation. The strategic goal is to reduce the mortality and incidence rate of dengue fever by at least 50% and 20% respectively by 2020. Human intervention will have a great impact on the number of cases, so this paper only predicts the number of dengue patients in Guangdong Province before 2023. Predicted cumulative number of dengue fever patients is in Fig.4. As can be seen from the

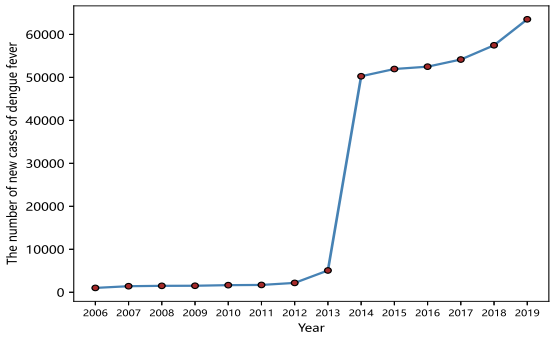

Fig 2. Accumulated number of dengue fever patients in Guangdong Province from 2006 to 2019.

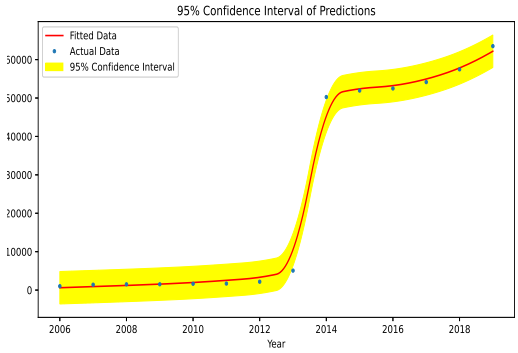

Fig 3. Comparison between the cumulative number of dengue fever patients from 2006 to 2019 and the fitting data of the model.

graph, if control measures are not strengthened, the cumulative number of dengue patients in Guangdong Province will continue to increase in the next few years.

Table 3. Description of some model parameters and their values from 2006 to 2013.

Description	Symbol	Value	Source
Mosquito bite rate	θ	$8.60646880 \times 10^{-3}$	Estimated
Transmission probability of dengue from infected mosquito to human	β_1	$3.64162027 \times 10^{-4}$	Estimated
Transmission probability of dengue from human to mosquito	β_3	$1.27207340 \times 10^{-7}$	Estimated
Rate of propagation of awareness	β_2	$4.76870717 \times 10^{-9}$	Estimated
Rate at which individuals loose awareness due to memory fading	δ	$6.88161409 \times 10^{-2}$	Estimated
Rate at which infected individuals send information about the epidemic disease	σ_1	$5.78289772 \times 10-2$	Estimated
Pesticide control for mosquito population	μ_1	0.00001	Estimated
Human psychological response rate to infectious diseases	m	$1.81443925 \times 10^{-6}$	Estimated
The maximum value of the reduction in the contact rate	α	$3.37180955 \times 10^{-2}$	Estimated
Rate at which message become outdated	σ_2	$1.98188640 \times 10^{-1}$	Estimated
Recruitment rate for humans	C_h	1083053	[32]
Natural death rate for humans	μ_h	0.00449	[32]
Natural death rate for mosquitoes	μ_v	0.0132	[19]
Disease death rate of infected humans	α_1	0.00005	Estimated
Value of baseline number of programs maintained in system	Λ	5	[14]
birth rate of mosquitoes	C_v	750000	[10]

The partial rank correlation coefficient (PRCC) is used to analyze the impact of the main

Table 4. Description of some model parameters and their values from 2014 to 2019.

Description	Symbol	Value	Source
Mosquito bite rate	θ	$8.83450552 \times 10^{-3}$	Estimated
Transmission probability of dengue from infected mosquito to human	β_1	$1.80087588 \times 10^{-3}$	Estimated
Transmission probability of dengue from human to mosquito	β_3	$2.24424674 \times 10^{-6}$	Estimated
Rate of propagation of awareness	β_2	$9.99999935 \times 10^{-1}$	Estimated
Rate at which individuals loose awareness due to memory fading	δ	$1.05494515 \times 10^{-2}$	Estimated
The maximum value of the reduction in the contact rate	α	$1.23679432 \times 10^{-9}$	Estimated
Rate at which message become outdated	σ_2	$1.05494575 \times 10^{-2}$	Estimated

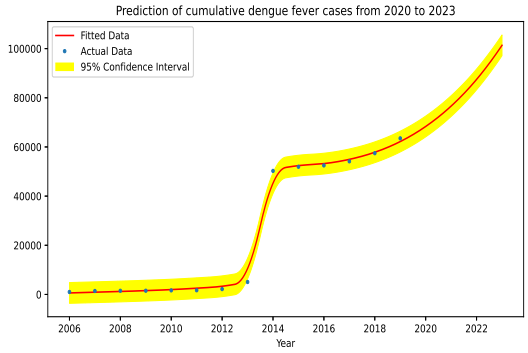

Fig 4. Prediction of the cumulative number of dengue fever patients from 2020 to 2023.

parameters in model (2.1) on $\mathcal{R}_0$. Based on the results of the parameters in Table 3, we choose a normal distribution for the main parameters. According to the LHS (Latin hypercube sampling) matrix, the sensitivity between the output variable $\mathcal{R}_0$ and the input parameter $\beta_2, \delta, \mu_1, \theta, \beta_1, \beta_3$ is evaluated by using PRCC. A positive PRCC value indicates a positive correlation between the parameter and $\mathcal{R}_0$, while a negative value indicates a negative correlation with R_0. From the graph, it can be seen that the parameters with positive impacts on $\mathcal{R}_0$ are $\theta, \beta_1, \beta_3, \delta$, and the parameters with negative impacts are β_2, μ_1. So this result indicates that controlling these parameters is more effective in preventing the further development of dengue fever. For example, it is possible to reduce the contact rate between susceptible individuals and mosquitoes, use some insecticides to increase the mortality rate of mosquitoes, and improve the transmission rate of awareness programs to reduce the basic reproduction rate, thereby controlling the spread of diseases.

In the following, we select the parameter estimation results from Table 3 and consider the impact of parameters θ and α on the number of dengue fever patients (see Fig.6 and Fig.7). From the graph, it can be seen that increasing the contact rate between mosquitoes and susceptible individuals will increase the number of dengue fever patients, while increasing media publicity will reduce the number of dengue fever patients. Therefore in today's era of advanced information, a large number of media promotion methods can be used to deepen the understanding of diseases among susceptible individuals and take appropriate preventive measures as soon as possible.

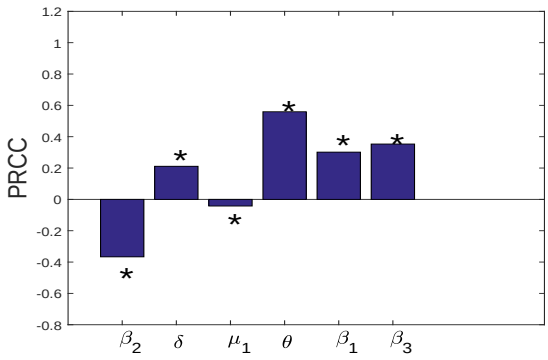

Fig 5. The influence of partial correlation coefficients.

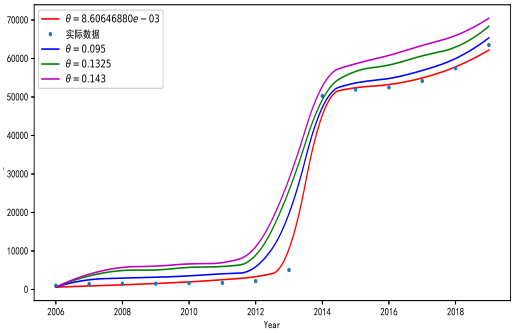

Fig 6. The impact of increasing θ on the number of dengue fever patients.

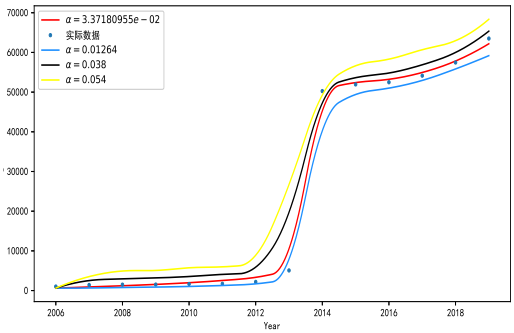

Fig 7. The impact of α on the number of dengue fever patients.

§6 Discussion

Dengue fever is a global infectious disease, and there is currently no specific antiviral treatment drug or vaccine available [8]. China is facing an increasing threat of imported dengue

fever cases due to the relaxation of COVID-19 prevention measures and resumption of domestic and international exchanges [22]. Therefore, controlling dengue fever remains one of the major global public health issues. In order to study the dynamic transmission behavior of dengue fever, we first proposed and studied a $S_hI_hA_hS_vI_vW$ model with media influence, and then defined the basic reproduction number $\mathcal{R}_0$ to determine the extinction and persistence of the disease. If $\mathcal{R}_0 < 1$, the disease-free equilibrium point is globally attractive, and the disease becomes extinct. If $\mathcal{R}_0 > 1$, the disease becomes endemic in the population. From the viewpoint of theoretical results, the basic reproduction number $\mathcal{R}_0$ can act as a threshold value that determines whether the disease will go to extinction or not.

Based on the dengue fever data released by Guangdong Province from 2006 to 2019, we conduct numerical simulations and predict the number of dengue fever patients. The results show that the number of dengue fever patients will continue to increase in a short time if the control measures are not strengthened. Sensitivity analysis shows that positive media promotion can quickly help susceptible populations understand the pathogenesis of diseases, generate awareness of proactive prevention, and reduce contact rates with mosquitoes. So we can report on disease outbreaks, transmission routes, and prevention measures through traditional news media such as television, radio, and newspapers. Government departments and health organizations can also publish authoritative health information and guide public behavior through official websites, announcements, and promotional materials. These media communication strategies can quickly disseminate key information and increase public awareness of diseases.

In the model, we only consider the impact of positive media coverage on infectious diseases. In fact, negative media coverage and incorrect information dissemination may also occur, which can easily cause public panic. In future work, we can consider these factors in the model and explore how to strike a balance between informing the public and avoiding panic or incorrect information.

Declarations

Conflict of interest The authors declare no conflict of interest.

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