



Global hopf bifurcation of a delayed equation describing the lag effect of media impact on the spread of infectious disease

Pengfei Song¹ · Yannì Xiao¹

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Abstract We proposed a delay differential model, associated with the response time for individuals to the current infection, to examine the media impact on the transmission dynamics of infectious diseases. We investigated the global bifurcation by considering the delay as a bifurcation parameter and examined the onset and termination of Hopf bifurcations from a positive equilibrium. Numerical studies to identify ranges of parameters for coexisting multiple periodic solutions are guided by the bifurcation analysis and the Matlab package DDE-BIFTOOL developed by Engelborghs et al. Further, we parameterized the proposed model on the basis of the 2009 A/H1N1 pandemic influenza data in Shaanxi province, China, and estimated the basic reproduction number to be 1.79 [95% CI (1.77–1.80)] and the time delay to be 2.94 days [95% CI (2.56–3.24)]. Our main results indicated that media impact with time delay significantly influenced the transmission dynamics of infectious diseases.

Keywords Media impact · Time delay · Global Hopf bifurcation

Mathematics Subject Classification 34K18 · 65L03 · 92B05

1 Introduction

In recent years, emerging infectious diseases such as 2003 severe acute respiratory syndrome (SARS), the recent H1N1, Dengue, have become a worldwide problem,

✉ Yannì Xiao
yxiao@mail.xjtu.edu.cn

Pengfei Song
song921012@stu.xjtu.edu.cn

¹ Department of Applied Mathematics, School of Mathematics and Statistics, Xi'an Jiaotong University, Xi'an 710049, People's Republic of China

threatening the public health and the stability of the whole world. When an emerging disease starts to spread, it is essential for individuals to recognize the infectivity, virulence and fatality of the infectious disease. Once knowing about the diseases such as the transmission routes and interventions, individuals will remain on high alert and may choose not to go to crowded areas and thus avoid unnecessary contact with infected individuals. Moreover, infected individuals will choose to cancel their trips and stay in home or hospital being treated, therefore new infection will apparently decrease due to reduction of contact rates (Cai and Li 2009; Tang et al. 2010). In particular, in the 21st century media coverage has exerted crucial influence on affecting spread and outbreaks of infectious disease, such as the 2003 SARS and 2009 H1N1 influenza epidemic. There is much evidence showing that media coverage plays a significant role in quick releasing important information, influencing individuals' behaviour and therefore helping contain the spread of the infectious diseases (Liu et al. 2007; Cannon 2008; Cui et al. 2008; Li and Cui 2009; Tchuente et al. 2011; Sun et al. 2011; Wang and Xiao 2014; Xiao et al. 2015).

Modelling the media impact on disease spread shares tremendous popularity in these years. Liu et al. (2007) described the media coverage impact on the transmission dynamics by including a decreasing factor $\beta e^{-a_1 E - a_2 I - a_3 H}$ in the transmission coefficient, where E , I , and H are the numbers of reported exposed, infectious, and hospitalized individuals, respectively. Cannon (2008) formulated a compartment model with incidence rate $\mu e^{-mI} SI$ with $m > 0$ reflecting the impact of media coverage on transmission dynamics. They have shown the existence of Hopf bifurcation when $m > 0$ is sufficiently small. Li and Cui (2009) formulated an SIS (susceptible–infective–susceptible) epidemic model with incidence rate $(\beta_1 - \beta_2 \frac{I}{m+I}) \frac{SI}{N}$ and used $\beta_2 \frac{I}{m+I}$ to reflect the reduced amount of contact rate due to media coverage. Recently, Xiao et al. (2015) formulated the media function depending on both the case number and its rate of change and obtained that media impact switches on and off in a highly nonlinear fashion with the greatest effect during the early stage of the outbreak. Yan et al. (2015) presented a novel methodology through using cross-correlation analysis and embedding a media function and the dynamics of the number of news reports into classical SEIR model, showed that combining statistical analysis with a mathematical model was beneficial for analyzing media impacts. Moreover, other forms, such as $(\mu_1 - \mu_2 f(I)) \frac{SI}{S+I}$, have been proposed to describe the media-induced incidence rate (see details in Tchuente et al. (2011), Cannon (2008), Sun et al. (2011)).

A common assumption in these media functions for the models (Liu et al. 2007; Cannon 2008; Cui et al. 2008; Li and Cui 2009; Tchuente et al. 2011; Sun et al. 2011; Wang and Xiao 2014; Xiao et al. 2015) is that media impact on transmission dynamics is instantaneous, that is, the number of infected individuals at time t timely affects the transmission coefficient at time t and leads to a reduction in incidence rate. However, this is not how the thing looks like. Mostly, impact of media coverage on transmission dynamics has time lag, describing both the time duration for individuals' response to the reported infection and the reported delay. Hence, it is more reasonable to include $I(t - \tau)$ rather than $I(t)$ in the media function in the incidence rate. Consequently, the delay differential equations provide a natural description of such systems with media impact. Functional differential equations appear in many domains of applied sciences,

and theoretical study on functional differential equations were given in Wu (1998). How the delay embedded media impact affects the transmission dynamics remains unclear, and therefore falls within the scope of this study.

Our main purpose is then to modify the known models in order to describe the individuals's response to infection due to media coverage by introducing a delayed media function. We shall analyze the global dynamics of the proposed system in order to examine how the refined media function influences the global dynamics of disease transmission and address the effect of media coverage on disease transmission. Further, we estimate the parameters of the novel model on the basis of the 2009 H1N1 pandemic influenza data in Shaanxi province of China, utilizes a stochastic simulation method to estimate the basic reproduction number R_0 , the response time for individuals to the current infection τ , and their confidence intervals.

The rest of this paper is organized as follows. Section 2 collects some preliminary results of the well-posedness of the delay differential system and the existence of equilibria. In Sect. 3, we focus on the local Hopf bifurcation of the positive equilibrium. The global onset and termination of Hopf bifurcations is validated in Sect. 4. Moreover, a case study and numerical results are shown in Sect. 5. Finally, concluding remarks are given in Sect. 6.

2 Preliminaries

The population is divided into three groups: susceptible (S), infected (I) and recovered (R). Media coverage and fast information flow induce a profound psychological impact on the public, a reduction in the incidence rate is represented by $e^{-\alpha I(t-\tau)}$. Here time delay τ denotes response time for individuals to the current infection. Thus the model equations are as follows.

$$\begin{cases} \dot{S} = \Lambda - \beta e^{-\alpha I(t-\tau)} SI - dS, \\ \dot{I} = \beta e^{-\alpha I(t-\tau)} SI - (d + \gamma)I, \\ \dot{R} = \gamma I - \mu R. \end{cases} \quad (1)$$

where Λ stands for the rate of flow into the population, d is the natural death rate, β denotes the baseline transmission rate, and γ represents the recovery rate. All parameters are nonnegative constants. Note that the recovered class R is decoupled with the first two equations in (1), then we only need to focus on the first two equations in the rest of this work,

$$\begin{cases} \dot{S} = \Lambda - \beta e^{-\alpha I(t-\tau)} SI - dS, \\ \dot{I} = \beta e^{-\alpha I(t-\tau)} SI - (d + \gamma)I. \end{cases} \quad (2)$$

In this section, we initially give some preliminary results of system (2).

For any $\tau > 0$, let $C := C([- \tau, 0], R)$ be Banach space of continuous functions on $[- \tau, 0]$ with the norm defined as $\|\phi\| = \sup_{-\tau \leq \theta \leq 0} |\phi(\theta)|$. Denote $C_+ = C([- \tau, 0], R_+)$. Given biological background, we choose initial values from $C_+ \times C_+$. For the initial value with the form $\Phi(\theta) = (\phi_1(\theta), \phi_2(\theta))$, $\Phi(\theta) \in C_+ \times C_+$, $\phi_i(0) > 0$, $i = 1, 2$, it can be shown that the system admits a unique solution X_t and $X_t \in R_+^2$ for any $t > 0$.

Proposition 1 For initial value with the form $\Phi(\theta) = (\phi_1(\theta), \phi_2(\theta))$, $\Phi(\theta) \in C_+ \times C_+$, $\phi_i(0) > 0, i = 1, 2$, the system (2) admits a unique solution X_t and X_t is nonnegative and bounded.

Proof It follows from Hale (1971) and Hale and Lunel (2013) that the existence and uniqueness of solutions to system (2) hold true.

For the initial value with the form $\Phi(\theta) = (\phi_1(\theta), \phi_2(\theta))$, $\Phi(\theta) \in C_+ \times C_+$, $\phi_i(0) > 0, i = 1, 2$, it follows from system (2) that

$$I(t) = I(0)e^{\int_0^t (\beta e^{-\alpha I(\theta-\tau)} S(\theta) - (d+\gamma)) d\theta} \geq 0, \text{ for } t \geq 0.$$

Thus the solution $I(t)$ remains nonnegative for $t \geq 0$. □

To verify that $S(t) > 0$ for $t > 0$, we assume the contrary. Let $t_1 > 0$ be the first time such that $S(t_1) = 0$, then by the first equation of (2) we have $S'(t_1) = \Lambda > 0$, and therefore, $S(t) < 0$ for $t \in (t_1 - \epsilon, t_1)$ where $\epsilon > 0$ is sufficiently small. This contradicts $S(t) > 0$ for $t \in [0, t_1)$. We conclude that $S(t) > 0$ for $t > 0$.

Besides, it follows from system (2) that

$$(S(t) + I(t))' \leq \Lambda - d(S(t) + I(t)) - \gamma I(t).$$

Then we have

$$\limsup_{t \rightarrow \infty} (S(t) + I(t)) \leq \frac{\Lambda}{d}.$$

Summarizing above-mentioned discussion, we can get the conclusion and complete the proof.

It is easy to calculate the disease-free equilibrium $E_0 = (\Lambda/d, 0)$, and the endemic equilibrium

$$E_1 = (S_*, I_*) = \left(\frac{d + \gamma}{\alpha d} \text{Lambert } W \left(\frac{d\alpha}{\beta} e^{\frac{\alpha\Lambda}{d+\gamma}} \right), \frac{\Lambda - dS_*}{d + \gamma} \right), \tag{3}$$

where *Lambert W*(.) is a Lambert W function, defined to be the multivalued inverse of the function $\omega \rightarrow \omega e^\omega$ (Corless et al. 1996).

The basic reproduction number is denoted as

$$R_0 = \frac{\Lambda\beta}{d(d + \gamma)}. \tag{4}$$

In view of Wang and Xiao (2014), we obtain the following proposition.

Proposition 2 Let $\tau = 0$. The disease-free equilibrium E_0 is globally asymptotically stable if $R_0 \leq 1$, while the endemic equilibrium E_1 is feasible and globally asymptotically stable if $R_0 > 1$.

Proof When $\tau = 0$, main conclusions for $R_0 > 1$ or $R_0 < 1$ have been carefully proved by Wang and Xiao (2014). Here, we only need to prove that the disease-free equilibrium E_0 is globally asymptotically stable if $R_0 = 1$.

Let $y = S - S_*$. Rewriting system (2) yields,

$$\begin{cases} \dot{y} = -\beta e^{-\alpha I(t)} y I - dy - \beta e^{-\alpha I(t)} S_* I, \\ \dot{I} = \beta e^{-\alpha I(t)} (y + S_*) I - (d + \gamma) I, \end{cases} \tag{5}$$

where $(y, I) \in D = \{(y, I) | y \geq -S_*, I \geq 0, y + I \leq 0\}$.

Now we validate the global asymptotically stability of zero point in D by using the Lyapunov function $V(y, I) = \frac{y^2}{2} + S_* I$. It is easy to obtain

$$\frac{dV}{dt} = -dy^2 - \beta e^{-\alpha I(t)} I y^2 + S_* I (d + \gamma) (R_0 e^{-\alpha I(t)} - 1) \leq 0.$$

Besides, when $R_0 = 1$, $\frac{dV}{dt} = 0$ only if $y = I = 0$. Thus, we complete the proof. \square

3 Dynamics of delayed equations

3.1 Stability of disease-free equilibrium

If $R_0 \leq 1$, the system (2) possesses only the disease-free equilibrium. We have the subsequent conclusion.

Theorem 1 *The disease-free equilibrium E_0 of system (2) is globally asymptotically stable if $R_0 \leq 1$ and unstable if $R_0 > 1$.*

Proof Let $y = S - S_*$. Rewriting system (2) yields,

$$\begin{cases} \dot{y} = -\beta e^{-\alpha I(t-\tau)} y I - dy - \beta e^{-\alpha I(t-\tau)} S_* I, \\ \dot{I} = \beta e^{-\alpha I(t-\tau)} (y + S_*) I - (d + \gamma) I, \end{cases} \tag{6}$$

where $(y, I) \in D = \{(y, I) | y \geq -S_*, I \geq 0, y + I \leq 0\}$.

Now we validate the global asymptotically stability of zero point in D by using the Lyapunov functional $L = \frac{y_t(0)^2}{2} + S_* I_t(0)$. It is easy to obtain

$$\frac{dL}{dt} = -dy^2 - \beta e^{-\alpha I(t-\tau)} I y^2 + S_* I (d + \gamma) (R_0 e^{-\alpha I(t-\tau)} - 1) \leq 0.$$

Besides, when $R_0 < 1$, $\frac{dL}{dt} = 0$ only if $y = I = 0$. When $R_0 = 1$, $\frac{dL}{dt} = 0$ yields $y = 0$ and $y = 0$ yields $I = 0$. Thus, we complete the proof. \square

The Jacobian matrix concerned with the linearization of system (2) at E_0 is,

$$M_{E_0} = \begin{pmatrix} -d - \lambda & -\frac{\beta \Delta}{d} \\ 0 & \frac{\beta \Delta}{d} - (d + \gamma) - \lambda \end{pmatrix}. \tag{7}$$

Therefore the characteristic equation of E_0 gives $(\lambda + d)(\lambda + (d + \gamma)(1 - R_0)) = 0$. In view of the characteristic equation, it's easy to verify that the disease-free equilibrium E_0 of system (2) is unstable if $R_0 > 1$.

3.2 Stability and Hopf bifurcation of the endemic equilibrium

If $R_0 > 1$, in addition to the disease-free equilibrium, the system (2) possesses an endemic equilibrium E_1 . The Jacobian matrix concerned with the liberalization of system (2) at E_1 is,

$$M_{E_1} = \begin{pmatrix} -\frac{\Lambda}{S_*} - \lambda - (d + \gamma) + \alpha e^{-\lambda\tau} (d + \gamma) I_* & \\ \frac{(d + \gamma) I_*}{S_*} & -\alpha e^{-\lambda\tau} (d + \gamma) I_* - \lambda \end{pmatrix}. \quad (8)$$

Thus, the characteristic equation of E_1 gives

$$\lambda^2 + a\lambda + b(\lambda + d)e^{-\lambda\tau} + c = 0, \quad (9)$$

where $a = \frac{\Lambda}{S_*}$, $b = \alpha(d + \gamma)I_*$, $c = \frac{(d + \gamma)^2 I_*}{S_*}$. It is easy to get that the endemic equilibrium E_1 is locally asymptotically stable for $\tau = 0$. Further, E_1 is globally asymptotically stable for $\tau = 0$ and see details in paper Wang and Xiao (2014).

We now set delay τ as the bifurcation parameter to investigate whether Hopf bifurcation can occur or not. The stability of E_1 may change if a pair of purely imaginary roots $\lambda = \pm i\omega$, $\omega > 0$ arises.

By substituting $\lambda = i\omega$ into (9) and separating the real and imaginary part, we obtain

$$\begin{aligned} -\omega^2 + b\omega \sin \omega\tau + bd \cos \omega\tau + c &= 0, \\ a\omega + b\omega \cos \omega\tau - bd \sin \omega\tau &= 0. \end{aligned} \quad (10)$$

Rewriting the equations yields

$$\omega^4 + p\omega^2 + q = 0, \quad (11)$$

with

$$p =: a^2 - b^2 - 2c, \quad q =: c^2 - b^2 d^2. \quad (12)$$

Whether Eq. (11) has one or two positive roots yields the following two cases:

Case 1: (H1) $q < 0$, i.e., $\alpha d e^{\frac{\alpha\Lambda}{d+\gamma}} - 1 / \beta < 1$.

By using Lambert W function (Corless et al. 1996), we easily know that $q < 0$ is equivalent to

$$\alpha < \frac{d + \gamma}{\Lambda} \text{LambertW}(eR_0).$$

In such case the Eq. (11) has one root,

$$\omega_0 = \sqrt{\frac{-2(a^2 - b^2 - 2c) + \sqrt{(a^2 - b^2 - 2c)^2 - 4(c^2 - b^2d^2)}}{2}}. \tag{13}$$

(10) yields that $\cos(\omega\tau) = \frac{b\omega_0^2(d - \frac{A}{s_*}) - cbd}{(b\omega_0)^2 + (bd)^2} < 0$. Therefore,

$$\tau_k = \frac{1}{\omega_0}((2k + 1)\pi - \arcsin A), \quad A = \frac{(\omega_0^2 - c)b\omega_0 + abd\omega_0}{(b\omega_0)^2 + (bd)^2}, \tag{14}$$

with $k = 0, 1, 2, 3, \dots$

Case 2: (H2) $q > 0, p < 0, p^2 - 4q > 0$.

In such scenario the Eq. (11) has two roots,

$$\omega_1 = \sqrt{\frac{-2(a^2 - b^2 - 2c) + \sqrt{(a^2 - b^2 - 2c)^2 - 4(c^2 - b^2d^2)}}{2}},$$

$$\omega_2 = \sqrt{\frac{-2(a^2 - b^2 - 2c) - \sqrt{(a^2 - b^2 - 2c)^2 - 4(c^2 - b^2d^2)}}{2}}.$$

Solving Eq. (10) for τ yields

$$\tau_k^{1,2} = \frac{1}{\omega_{1,2}}((2k + 1)\pi - \arcsin A_{1,2}), \quad A_{1,2} = \frac{(\omega_{1,2}^2 - c)b\omega_{1,2} - abd\omega_{1,2}}{(b\omega_{1,2})^2 + (bd)^2}.$$

In order to validate the transversion condition for existence of local Hopf bifurcation, we need to identify the sign of $Re(\frac{d\lambda}{d\tau})|_{\tau=\tau_k}$ with $Re(x)$ denoting the real part of x and $k = 0, 1, 2, \dots$. For simplicity, we analyze the sign of $Re(\frac{d\lambda}{d\tau})^{-1}|_{\tau=\tau_k}$.

Proposition 3 (i) For Case 1, that is, conditions (H1) holds, we have

$$Re\left(\frac{d\lambda}{d\tau}\right)^{-1} |_{\tau=\tau_k} > 0.$$

(ii) For Case 2, that is, conditions (H2) holds, we have

$$Re\left(\frac{d\lambda}{d\tau}\right)^{-1} |_{\tau=\tau_k^1} > 0 > Re\left(\frac{d\lambda}{d\tau}\right)^{-1} |_{\tau=\tau_k^2}.$$

Proof (i) Differentiating both sides of the characteristic Eq. (9) with respect to τ yields

$$\frac{d\lambda}{d\tau} = \frac{e^{-\lambda\tau}\lambda b(\lambda + d)}{2\lambda + a + be^{-\lambda\tau} - \tau e^{-\lambda\tau} b(\lambda + d)}.$$

Replacing $b(\lambda + d)e^{-\lambda\tau}$ with $-(a^2 + b\lambda + c)$, we have

$$\begin{aligned} \operatorname{Re} \left(\frac{d\lambda}{d\tau} \right)^{-1} \Big|_{\tau=\tau_k} &= \operatorname{Re} \left[\frac{2i\omega_0 + a}{i(\omega_0^3 - c\omega_0) + a\omega_0^2} \right] + \operatorname{Re} \left[\frac{1}{i\omega(i\omega + d)} \right] - \operatorname{Re} \left[\frac{\tau}{i\omega} \right] \\ &= \frac{a^2 - b^2 + 2(\omega_0^2 - c)}{b^2(\omega_0^2 + d^2)} \\ &= \frac{\omega_0^4 + b^2d^2 - c^2}{b^2\omega_0^2(\omega_0^2 + d^2)}. \end{aligned} \tag{15}$$

Therefore we obtain $\operatorname{Re} \left(\frac{d\lambda}{d\tau} \right)^{-1} \Big|_{\tau=\tau_k} > 0$. Then the transversion condition holds at $\tau = \tau_k, k = 0, 1, 2, 3, \dots$

- (ii) For condition (H2), it is complicated to determine $\operatorname{Re} \left(\frac{d\lambda}{d\tau} \right)^{-1} \Big|_{\tau=\tau_k^1}$ or $\operatorname{Re} \left(\frac{d\lambda}{d\tau} \right)^{-1} \Big|_{\tau=\tau_k^2}$. However, we have the following formula

$$\begin{aligned} \operatorname{Re} \left(\frac{d\lambda}{d\tau} \right)^{-1} \Big|_{\tau=\tau_k^1} \cdot \operatorname{Re} \left(\frac{d\lambda}{d\tau} \right)^{-1} \Big|_{\tau=\tau_k^2} &= \frac{a^2 - b^2 + 2(\omega_1^2 - c)}{b^2(\omega_1^2 + d^2)} \frac{a^2 - b^2 + 2(\omega_2^2 - c)}{b^2(\omega_2^2 + d^2)} \\ &= \frac{(p + 2\omega_1^2)(p + 2\omega_2^2)}{b^4(\omega_1^2 + d^2)(\omega_2^2 + d^2)} \\ &= \frac{p^2 + 2(\omega_1^2 + \omega_2^2)p + 4\omega_1^2\omega_2^2}{b^4(\omega_1^2 + d^2)(\omega_2^2 + d^2)} \\ &= \frac{4q - p^2}{b^4(\omega_1^2 + d^2)(\omega_2^2 + d^2)} < 0. \end{aligned} \tag{16}$$

Furthermore, we note that $\operatorname{Re} \left(\frac{d\lambda}{d\tau} \right)^{-1} \Big|_{\tau=\tau_k^{1,2}} = \frac{p+2\omega_{1,2}^2}{b^2(\omega_{1,2}^2+d^2)}$. Since $\omega_1 > \omega_2$, we obtain,

$$\operatorname{Re} \left(\frac{d\lambda}{d\tau} \right)^{-1} \Big|_{\tau=\tau_k^1} > 0 > \operatorname{Re} \left(\frac{d\lambda}{d\tau} \right)^{-1} \Big|_{\tau=\tau_k^2}.$$

Then the transversion condition holds for $\lambda = i\omega_1, \tau = \tau_k^1$, doesn't hold for $\lambda = i\omega_2, \tau = \tau_k^2, k = 0, 1, 2, 3, \dots$ Applying Proposition 3 and Collary 2.4 of Ruan and Wei (2003) yields the spectral results. □

- Proposition 4** (i) For condition (H1), the characteristic equation (9) has a pair of simple purely imaginary roots $\pm i\omega_0$ at $\tau_k, k = 0, 1, 2, \dots$. Furthermore, if $\tau \in [0, \tau_0)$, all roots of Eq. (9) have negative real parts; if $\tau = \tau_0$, all roots of Eq. (9) except for $\pm i\omega_0$ have negative real parts; if $\tau \in (\tau_k, \tau_{k+1}]$, Eq. (9) has $2(k + 1)$ positive roots.
- (ii) For condition (H2), the characteristic equation (9) has two pairs of simple purely imaginary roots $\pm i\omega_1, \pm i\omega_2$ at $\tau_k^1, \tau_k^2, k = 0, 1, 2, \dots$, respectively. Furthermore, if $\tau \in [0, \tau_0)$, all roots of Eq. (9) have non-positive real parts, if $\tau \in (\tau_k, \tau_{k+1})$, Eq. (9) has $2(k + 1)$ positive roots.

(iii) *If neither of conditions (H1–H2) holds, all roots of Eq. (9) have negative real parts.*

In fact, the expressions of ω_1 and τ_k^1 in Case 2 are the same as ω_0 and τ_k in Case 1, respectively. For convenience, we use the same notation ω_0, τ_k in the following part of this study. In summary, for Case 1 and Case 2, transversion condition holds at the endemic equilibrium when $\tau = \tau_k, k = 0, 1, 2, \dots$. Applying the Hopf bifurcation theorem for delay differential equations (Hale and Lunel 2013; Hassard et al. 1981) yields system (2) undergoes Hopf bifurcations at the endemic equilibrium along a sequence of τ value $\tau_k, k = 0, 1, 2, \dots$. Proposition (4) immediately leads to stability properties of the endemic equilibrium of system (2).

Theorem 2 *Let $R_0 > 1$ and $\tau > 0$ be satisfied.*

- (i) *If condition (H1) or (H2) holds, the endemic equilibrium is asymptotically stable for $\tau \in [0, \tau_0)$ and unstable for $\tau > \tau_0$. Besides, system (2) undergoes Hopf bifurcation at the endemic equilibrium when $\tau = \tau_k, k = 0, 1, 2, \dots$*
- (ii) *If neither of conditions (H1–H2) holds, the endemic equilibrium is locally asymptotically stable.*

4 Global Hopf bifurcation

In this section, we explore the continuation and termination of the local Hopf bifurcation emanating from $\tau_k, k = 1, 2, 3, \dots$ by using global Hopf bifurcation theorem of functional differential equation (Erbe et al. 1992; Wu 1998). Let $X = C([-1, 0], R^2)$, $z(t) = (z_1(t), z_2(t)) = (S(\tau t), I(\tau t))$. Rewriting system (2) as a functional differential equation yields

$$z'(t) = F(z_t, \tau, T), \quad (t, \tau, T) \in R \times R \times R_+, \tag{17}$$

where $z_t(\theta) = z(t + \theta), \theta \in [-1, 0], z_t \in X$ and

$$F(z_t, \tau, T) = \begin{cases} \tau \Lambda - \tau \beta e^{-\alpha z_2(t-1)} z_1(t) z_2(t) - \tau d z_1(t) \\ \tau \beta e^{-\alpha z_2(t-1)} z_1(t) z_2(t) - \tau (d + \gamma) z_2(t). \end{cases} \tag{18}$$

The subspace of X contains all constant function from $[-1, 0]$ to R^2 . The restricted function of F can be given in the following form,

$$\begin{aligned} \tilde{F} &:= F|_{R_+^2 \times R \times R_+} \longrightarrow R, \\ \tilde{F}(z, \tau, T) &= \begin{cases} \tau \Lambda - \tau \beta e^{-\alpha z_2} z_1 z_2 - \tau d z_1 \\ \tau \beta e^{-\alpha z_2} z_1 z_2 - \tau (d + \gamma) z_2. \end{cases} \end{aligned} \tag{19}$$

It's obvious that \tilde{F} is twice continuously differentiable. Thus the assumption (A1) in global hopf bifurcation theorem (Wu 1998) is corroborated.

We denote the set of stationary solutions of (19) by

$$N(F) = \{(\tilde{y}, \tilde{\tau}, \tilde{T}) : \tilde{F}(\tilde{z}, \tilde{\tau}, \tilde{T}) = 0\}.$$

For any $(\tilde{z}, \tilde{\tau}, \tilde{T}) \in N(F)$, $DF(\tilde{z}, \tilde{\tau}, \tilde{T})(e^{-\lambda} Id) \neq 0$, which implies the assumption (A2) is validated.

For any stationary solution, the characteristic matrix is

$$\begin{aligned} \Delta_{(\tilde{z}, \tilde{\tau}, \tilde{T})}(\lambda) &= \lambda Id - DF(\tilde{z}, \tilde{\tau}, \tilde{T})(e^{-\lambda} Id) \\ &= \begin{pmatrix} \tau\beta e^{-\alpha\tilde{z}_2}\tilde{z}_2 + \tau d + \lambda & \tau\beta e^{-\alpha\tilde{z}_2}\tilde{z}_1 - \tau\alpha e^{-\lambda}\beta e^{-\alpha\tilde{z}_2}\tilde{z}_1\tilde{z}_2 \\ -\tau\beta e^{-\alpha\tilde{z}_2}\tilde{z}_2 & -\tau\beta e^{-\alpha\tilde{z}_2}\tilde{z}_1 + \tau\alpha e^{-\lambda}\beta e^{-\alpha\tilde{z}_2}\tilde{z}_1\tilde{z}_2 + \tau(d + \gamma) + \lambda \end{pmatrix}, \end{aligned} \tag{20}$$

and the characteristic equation of the stationary solution gives

$$\det(\Delta_{(\tilde{z}, \tilde{\tau}, \tilde{T})}(\lambda)) = \lambda^2 + \tau\tilde{a}\lambda + \tau\tilde{b}(\lambda + \tau\tilde{d})e^{-\lambda} + \tau^2\tilde{c} = 0, \tag{21}$$

where $\tilde{a} = \frac{\Delta}{\tilde{z}_1}$, $\tilde{b} = \alpha(d + \gamma)\tilde{z}_2$, $\tilde{c} = \frac{(d+\gamma)^2\tilde{z}_2}{\tilde{z}_1}$. Therefore it can be easily obtained that the assumption (A3) holds.

In view of Wu (1998), if $\det(\Delta_{(\tilde{z}, \tilde{\tau}, \tilde{T})}(im\frac{2\pi}{T})) = 0$ for some integer m , we call this stationary solution $(\tilde{z}, \tilde{\tau}, \tilde{T}) \in N(F)$ a center. Moreover, if it is the only center in some neighborhood of $(\tilde{z}, \tilde{\tau}, \tilde{T})$ and it has finitely purely imaginary characteristic values of the form $im\frac{2\pi}{T}$, where m is an integer, we call this center is isolated. Let $J(\tilde{z}, \tilde{\tau}, \tilde{T})$ denote the set of all such positive integers m .

From Wu (1998), we know that $((S_*, I_*), \tau_n, \frac{2\pi}{\omega_0\tau_n})$ for any integer $n \geq 0$ is an isolated center, where τ_n and ω_0 are defined in (13) and (14), respectively. Moreover, it has only one purely imaginary eigenvalue of the form $im\frac{2\pi}{T}$ and the only interger $m = 1$. Note that the crossing number in Wu (1998) satisfies

$$\gamma_1 \left((S_*, I_*), \tau_n, \frac{2\pi}{\omega_0\tau_n} \right) = -1. \tag{22}$$

Thus the assumption (A4) in Wu (1998) holds.

Let $\Sigma(F) = Cl\{(z, \tau, T) : z \text{ is a nontrivial } T\text{-periodic solution of system(2)}\} \subset X \times R_+ \times R_+$ with $n = 1, 2, \dots, X = C([-\tau, 0], R^2)$, and $C((S_*, I_*), \tau_n, \frac{2\pi}{\omega_0\tau_n})$ denotes the connected component of $((S_*, I_*), \tau_n, \frac{2\pi}{\omega_0\tau_n})$ in $\Sigma(F)$. The global Hopf theorem implies that either of the subsequent two assertions holds,

- (i) $C((S_*, I_*), \tau_n, \frac{2\pi}{\omega_0\tau_n})$ is unbounded,
- (ii) $C((S_*, I_*), \tau_n, \frac{2\pi}{\omega_0\tau_n})$ is bounded, $C((S_*, I_*), \tau_n, \frac{2\pi}{\omega_0\tau_n}) \cap N(F)$ is finite and for all $m = 1, 2, 3, \dots$, we have

$$\sum_{(z, \tau, T) \in C((S_*, I_*), \tau_n, \frac{2\pi}{\omega_0\tau_n}) \cap N(F)} \gamma_m(z, \tau, T) = 0,$$

where $\gamma_m(z, \tau, T)$ is the m th crossing number of (z, τ, T) if $m \in J(z, \tau, T)$, otherwise, $\gamma_m(z, \tau, T) = 0$.

Proposition 4 reveals for each $n = 1, 2, \dots, (z, \tau, T) \in C((S_*, I_*), \tau_n, \frac{2\pi}{\omega_0\tau_n})$,

$$J(z, \tau, T) = \{1\}, \tag{23}$$

$$\sum_{(z, \tau, T) \in C((S_*, I_*), \tau_n, \frac{2\pi}{\omega_0\tau_n}) \cap N(F)} \gamma_m(z, \tau, T) = \gamma_1(z, \tau, T) = -1 < 0. \tag{24}$$

Therefore, for all $n = 1, 2, \dots$, assertion (i) holds, which means the connected component of $((S_*, I_*), \tau_n, \frac{2\pi}{\omega_n\tau_n})$ in $\Sigma(F)$, $C((S_*, I_*), \tau_n, \frac{2\pi}{\omega_0\tau_n})$ is unbounded. If its projection onto z – space and T – space are bounded, then the projection on τ – space is unbounded. Therefore we reap the final results of global Hopf bifurcation branches. The following two lemmas help confirm the boundedness of projection of $C((S_*, I_*), \tau_n, \frac{2\pi}{\omega_0\tau_n})$ onto z – space and T – space.

Lemma 1 For the initial value with the form $\Phi(\theta) = (\phi_1(\theta), \phi_2(\theta))$, $\Phi(\theta) \in C_+$, $\phi_i(0) > 0, i = 1, 2$, all periodic solutions of system (17) is uniformly bounded.

Proof For the initial value with the form $\Phi(\theta) = (\phi_1(\theta), \phi_2(\theta))$, $\Phi(\theta) \in C_+$, $\phi_i(0) > 0, i = 1, 2$, it can be shown that the system admits a unique solution X_t and $X_t \in R_+^2$ for any $t > 0$. Then we obtain the lower bound.

Moreover, it follows from 2 that

$$(S(t) + I(t))' \leq \Lambda - d(S(t) + I(t)) - \gamma I(t),$$

which implies

$$\limsup_{t \rightarrow \infty} (z_1(t) + z_2(t)) \leq \frac{\Lambda}{d}.$$

Thus, we obtain an upper bound. This completes the proof. □

Note that lemma 1 ensures that the projection of $C((S_*, I_*), \tau_n, \frac{2\pi}{\omega_0\tau_n})$ onto z – space is bounded. It follows from (13) and (14) that $\tau_k\omega_0 = 2k\pi + \arccos A, k = 1, 2, \dots$. Hence, we have

$$\frac{1}{k + 1} < \frac{2\pi}{\tau_0\omega_k} < 1. \tag{25}$$

If we exclude the existence of periodic solution of period 1, then system (17) has no periodic solutions of period $\frac{1}{k}$ for any positive integer k . Thus, the projection of $C((S_*, I_*), \tau_k, \frac{2\pi}{\omega_0\tau_k})$ onto T – space is bounded.

Lemma 2 Suppose that condition (H1) or (H2) holds, then the system (17) has no periodic solutions of period 1.

Proof Assume that $y(t) = (y_1(t), y_2(t))$ is a periodic solution of system (17) with period 1, then $y(t) = (y_1(t), y_2(t))$ is a periodic solution of the following ordinary differential equations,

$$\begin{cases} y_1(t)' = \tau \Lambda - \tau \beta e^{-\alpha y_2(t)} y_1(t) y_2(t) - d y_1(t), \\ y_2(t)' = \tau \beta e^{-\alpha y_2(t)} y_1(t) y_2(t) - \tau(d + \gamma) y_2(t). \end{cases} \tag{26}$$

However, by simply analyzing the above system we know that the unique positive equilibrium is globally asymptotically stable and no periodic solution occurs [referring to proposition (2)]. This completes the proof. \square

Theorem 3 Assume that $R_0 > 1$ and condition (H1) or (H2) holds, then for any $\tau > \tau_1$ system (17) has at least one nontrivial periodic solution.

Proof It follows from Eq. (19) that hypothesis (A1), (A2) and (A3) in Wu (1998) hold. Moreover, Eqs. (22) and (23) imply conditions (A4) and (A5) hold.

In view of (2), we know for each $n = 1, 2, \dots, (z, \tau, T) \in C((S_*, I_*), \tau_n, \frac{2\pi}{\omega_0 \tau_n})$, and

$$\sum_{(z, \tau, T) \in C((S_*, I_*), \tau_n, \frac{2\pi}{\omega_n \tau_n}) \cap N(F)} \gamma_m(z, \tau, T) = \gamma_1(z, \tau, T) = -1 < 0. \tag{27}$$

Thus, for all $n = 1, 2, \dots$, assertion (i) holds, which means the connected component of $((S_*, I_*), \tau_n, \frac{2\pi}{\omega_n \tau_n})$ in $\Sigma(F)$, $C((S_*, I_*), \tau_n, \frac{2\pi}{\omega_0 \tau_n})$ is unbounded.

It follows Lemma 2 that the projection of $C((S_*, I_*), \tau_k, \frac{2\pi}{\omega_0 \tau_k}), k = 1, 2, 3, \dots$ onto $T - space$ is bounded. Moreover, (13) and (14) indicate $\tau_k \omega_0 = 2k\pi + \arccos A$ is satisfied. Hence

$$\frac{1}{k + 1} < \frac{2\pi}{\tau_0 \omega_k} < 1. \tag{28}$$

From Lemma 2, we can exclude the existence of periodic solution of period τ , thus the projection of $C((S_*, I_*), \tau_k, \frac{2\pi}{\omega_0 \tau_k}), k = 1, 2, 3, \dots$, onto $T - space$ is bounded. This completes the proof.

We now use numerical simulations to demonstrate our theoretical results. We initially fix parameters as $\Lambda = d = 0.2, \beta = 1, \gamma = 0.1(\text{day}^{-1}), \alpha = 3$. It is easy to verify that $R_0 = 10/3 > 1$ and condition (H1) : $c^2 - b^2 d^2 = -0.019 < 0$ holds. It follows from (13) and (14) that $\tau_0 = 11.758, \tau_1 = 43.901, \tau_2 = 76.045$ theoretically. Figure 1 shows the tendency of each eigenvalue with the increasing time delay τ , which illustrates the curves hit the line of $Re(x) = 0$ at $\tau \approx 11.758$ for the first time, $\tau \approx 43.901$ the second time, $\tau \approx 76.045$ third time, respectively. Figure 2 shows that the endemic equilibrium is asymptotically stable for $\tau = 10.758 < \tau_0$ (shown in Fig. 2a, b) and the bifurcated periodic solution is feasible for $\tau = 12.758 > \tau_0 = 11.758$ (shown in Fig. 2c, d). By using DDE-BIFTOOL (Engelborghs et al. 2002), we can depict the the global Hopf branches of periodic solution originating from Hopf bifurcation points. Figure 3 shows the global

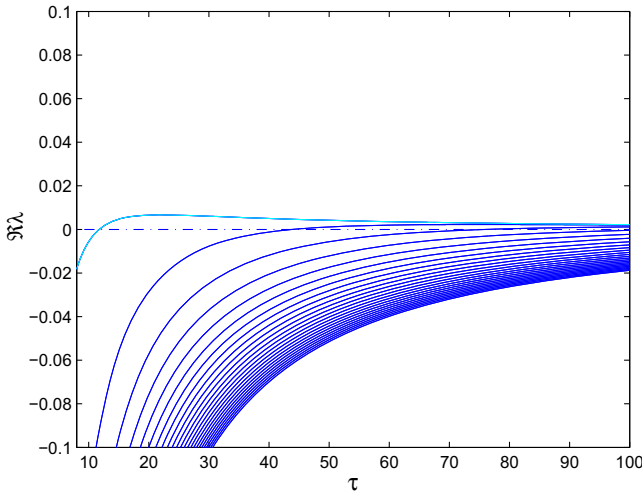


Fig. 1 Each curve represents the tendency of each eigenvalue with increasing delay τ . Three curves hit $Re(\lambda) = 0$ at $\tau \approx 11.758(\tau_0)$, $43.901(\tau_1)$, $76.045(\tau_2)$ respectively

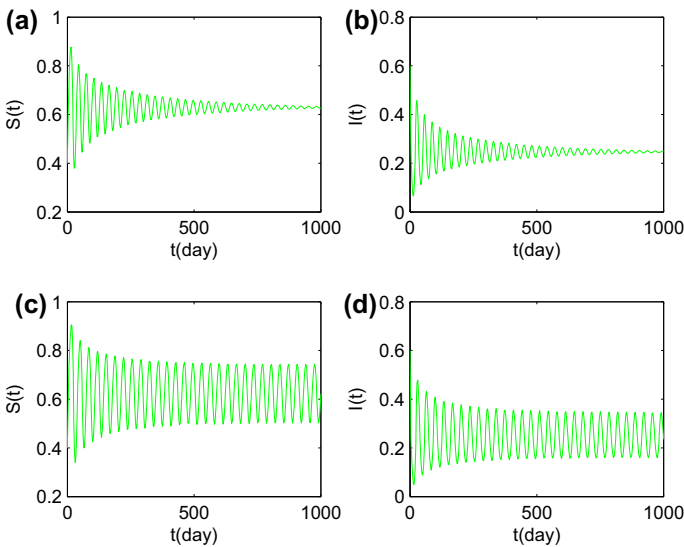


Fig. 2 Solutions of system (2) for $\tau = 10.758 < \tau_0 = 11.758$ (a, b) and for $\tau = 12.758 > \tau_0 = 11.758$ (c, d)

Hopf branches of periodic solution emanating from τ_0, τ_1, τ_2 . When $\tau_0 < \tau < \tau_1$, system(2) have only one periodic solution originating from τ_0 . As τ increases and satisfies $\tau_1 < \tau < \tau_2$, periodic solutions originating from τ_0, τ_1 coexist. As τ further increases and satisfies $\tau_2 < \tau < \tau_3$, we obtain three periodic solutions originating from τ_0, τ_1, τ_2 respectively. This result also implies that the Hopf branch emanating from τ_0 can continue in a wide range. Further, we plotted the bifurcation diagram using the delay as the bifurcation parameter (showed in Fig. 4), and obtained the periodic

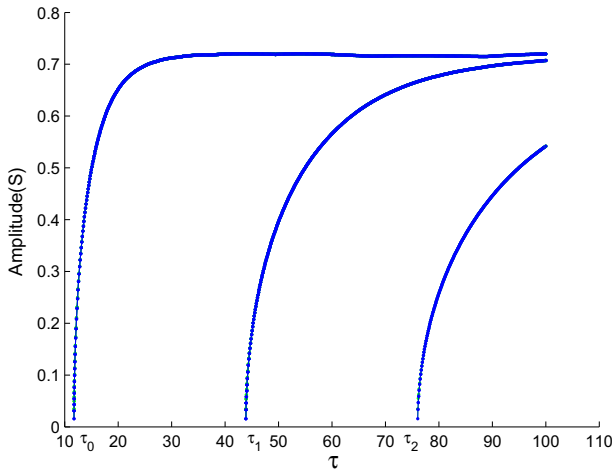


Fig. 3 Global Hopf branches of τ_0, τ_1, τ_2

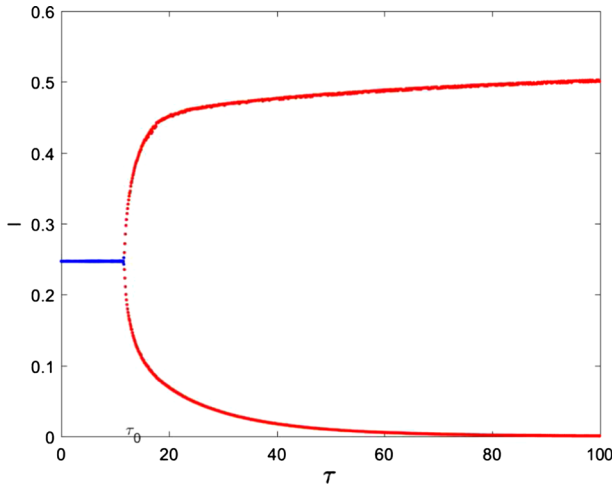


Fig. 4 Bifurcation diagram describing the dynamics of system (2) as the delay τ increases

solution bifurcated from τ_0 is stable whereas the periodic solutions bifurcated from τ_1, τ_2 are unstable. □

5 A case study

In this section, we estimate the parameters of model (2) on the basis of the 2009 H1N1 pandemic influenza data in Shaanxi province of China from September 3rd to October 12th by using nonlinear least-square method. Moreover, a stochastic simulation method was utilized to estimate the basic reproduction number R_0 , the response time for individuals to the current infection τ (day), and their confidence intervals.

As shown in previous work, media coverage significantly delayed the epidemics peak and decreased the intensity of the outbreak (Xiao et al. 2015; Yan et al. 2015; Tang et al. 2010; Liu et al. 2007; Cannon 2008). Moreover, Yan et al. (2015) revealed the importance of the response of individuals to the media reports, with behaviour changes being more important in emerging infectious control than the substantial media attention given to unexpected events and reports. Besides, by conducting cross-correlation analysis between the number of new hospital notifications and the average number of daily news, Yan et al. (2015) noted a specific lag of 4 days between the average number of daily news items and the number of daily hospital notifications, which revealed the lag effect of media impact on H1N1 transmission. However, the lag effect of media coverage impact hadn't been taken into consideration in mathematical modelling (Yan et al. 2015). In this section, we explore the lag effect of media coverage impact from the perspective of mathematical models.

5.1 Parameter estimation and model fitting

We obtained data on laboratory-confirmed cases of the A/H1N1 influenza pandemic in the Shaanxi province of China (shown in Fig. 5a) from the Provinces Public Health Information System, which are actually the numbers of hospital notifications since almost every diagnosed case has been hospitalized in early September Tang et al. (2010). Note that daily number of hospital notifications was reported separately every two or three days, and no data were available during the weekends (Fig. 5a). In order to deal with this irregular data, the cubic spline interpolation method on the surveillance data was used while we estimated the parameters and fitted the model (Fig. 5b). Since in our model (2) the individuals in the infected compartment may include both isolated (or hospitalized) and un-isolated individuals, we have to multiply the number of infected individuals $I(t)$ with the rate of isolation $\delta = 0.4$ (Tang et al. 2010) when fitting our model to the data on hospital notifications. The demographic effects are not considered

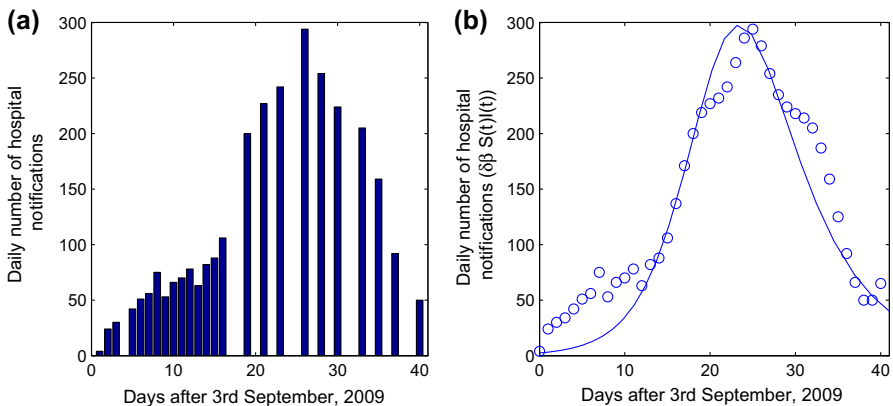


Fig. 5 The numbers of cases of A/H1N1 flu reported for the Shaanxi Province. **a** Daily number of hospital notifications for the Shaanxi province from September 3rd to October 12th 2009; **b** goodness of fit for (a)

Table 1 Parameter values of model (2)

Parameter	Description	Value	Mean	Std
S_0	Number of initial susceptible individuals	18167 (LS)	18906	499.8
I_0	Number of initial infected individuals	10 (fixed)	–	–
Λ	Birth rate of the susceptible population	0	–	–
d	Natural death rate	0	–	–
β	Contact transmission rate	0.6056 (LS)	0.5946	0.0118
α	Weight of media effect sensitive to number of infected population	1 (LS)	0.9898	0.1041
γ	Recovery rate (day^{-1})	0.33 (LS)	0.3324	0.0135
τ	The response time for individuals to the current infection (day)	2.92 (LS)	2.9371	0.1954

in the following discussion because of the short epidemic time scale in comparison to the demographic time scale, that is, we set $\Lambda = 0$, $d = 0$ here.

To estimate the parameters of model (2), we utilized the nonlinear least-square method (NLES) in matlab to fit the daily number sets of hospital notifications for the Shaanxi province from September 3rd to October 12th 2009 which correspond to the model solution time series $\delta\beta S(t)I(t)$, as shown in Fig. 5b. Note that when $\alpha = 0$ i.e. media effect haven't been exploited, model (2) becomes an ordinary differential system. While solving the delayed differential equations (2), we used the solution of the ordinary differential system ($\alpha = 0$ in model (2)) as the initial functions. The estimated parameter values are listed in Table 1.

5.2 Basic reproduction number R_0 and the response time for individuals to the current infection τ

To calculate the confidence intervals of the basic reproduction number R_0 and the response time for individuals to the current infection τ , we utilized a stochastic simulation method. To start with, we generated 200 samples of the daily number of hospital notifications from a Poisson process as the counting process was a Poisson process. Secondly, NLES method was used to fit model (2) and consequently we obtained 200 groups of values for these estimated values (S_0 , β , α , γ , τ), which were used to calculate the confidence interval of R_0 and τ . Finally, we fitted the frequency histograms of all of the estimated values by using normal distributions. The mean values and standard deviations are shown in Table 1.

The basic reproduction number R_0 , which is defined as the number of newly infected individuals produced by a single infected individual in a totally susceptible population during the infectious period, can be explicitly calculated as $R_0 = \frac{\beta}{\gamma}$. Based on the above-mentioned parameter estimations, we then calculated the mean values of R_0

and τ are 1.7913 days (95% confidence interval is 1.7663–1.8014), 2.9371 days (95% confidence interval is 2.5612–3.2350 days), respectively.

6 Conclusion and discussion

Media communications have a great impact on individual behaviour changes, therefore significantly impact on the spread and outbreak of infectious disease. In recent years, models associated with the impact of media coverage on disease spread show a tremendous popularity. However, these models ignore the lag effect of media impact on the spread of infectious disease. Thus, a model of the lag effect of media impact is analyzed and discussed in this paper. The results obtained here could be beneficial for accurately assessing the effect of the media coverage in the control and treatment of infective diseases.

In this paper, we have proposed and analyzed a functional differential system induced by the lag effect of media impact. We should point out that the global Hopf bifurcation of a two dimensional delayed system is explored here. To start with, we address the existence of local Hopf bifurcation of the positive equilibrium. The properties of the LambertW function (Corless et al. 1996) is utilized while solving and representing the positive equilibrium. The results show that when the basic reproduction number R_0 is smaller than 1, the disease-free equilibrium E_0 of system (2) is globally asymptotically stable which means the disease will go to extinction. When the basic reproduction number R_0 is greater than 1, there exists an endemic equilibrium. Besides, if condition (H1) or (H2) holds, the endemic equilibrium is asymptotically stable for $\tau \in [0, \tau_0)$ and unstable for $\tau > \tau_0$. System (2) undergoes Hopf bifurcation at the endemic equilibrium when $\tau = \tau_k, k = 0, 1, 2, \dots$. If neither of conditions (H1–H2) hold, the endemic equilibrium is asymptotically stable. Besides, we validate the global onset and termination of Hopf bifurcations by employing a global Hopf bifurcation theorem (Wu 1998; Qu et al. 2010; Wei and Li 2005; Shu et al. 2014). From the bifurcation analysis, we know that if $R_0 > 1$ and condition (H1) or (H2) holds, system (2) has at least one nontrivial periodic solution for any $\tau > \tau_1$. We should point out that we haven't excluded the existence of periodic solutions with period 2 or 4 of system (17) under the condition (H1) or (H2). Therefore, we can't assert that system (2) has at least one nontrivial periodic solution for any $\tau > \tau_0$ theoretically. It will be the future work. However, DDE-BIFTOOL developed by Engelborghs et al. (Engelborghs et al. 2001, 2002) implies that the Hopf branch emanating from τ_0 can continue in a wide range.

Further, we conduct a case study based on the 2009 H1N1 pandemic influenza data in Shaanxi province of China. From the literature Yan et al. (2015), a specific lag of 4 days about the media impact on transmission of H1N1 pandemic was noted by conducting cross-correlation analysis between the number of new hospital notifications and the average number of daily news. However, the lag effect of media coverage impact hadn't been taken into consideration while proposing mathematical models in Yan et al. (2015). Therefore, the specific lag 4 days couldn't be explained from the perspective of mathematical models. In this paper, we calculate the response time for individuals to the current infection τ and its confidence interval by fitting the

daily number of hospital notifications during the 2009 H1N1 pandemic influenza in Shaanxi province of China to our proposed model (2). We estimate the parameters of model (2) by using nonlinear least-square method (in Table 1). Then, by utilizing a stochastic simulation method we obtain the mean value of the basic reproduction number R_0 1.7913 (95% confidence interval is 1.7663–1.8014) and the response time for individuals to the current infection 2.9371 (95% confidence interval is 2.5612–3.2350). The mean value and confidence interval of the basic reproduction number R_0 here have little differences from those (1.794, % CI 1.3858–1.9091) in (Yan et al. 2015). Besides, the mean value of the lag of media impact calculated by modelling simulation here is similar to the lags in (Yan et al. 2015) which was explored by cross-correlation analysis between the number of new hospital notifications and the average number of daily news, with small differences associated with the differences in methodology.

In conclusion, we have proposed a delay differential model associated with the lag effect of media coverage impact on the transmission and outbreak of infectious diseases. Local and global Hopf bifurcation of a two dimensional functional differential system are explored theoretically and numerically in this paper. Further, we estimate the parameters of the novel model on the basis of the 2009 H1N1 pandemic influenza data in Shaanxi province of China, calculate the basic reproduction number R_0 , the response time for individuals to the current infection τ , and their confidence intervals.

An assumption in this paper is that the impact of media coverage on the transmission of the infectious disease occurs as soon as the disease emerges and remains during the whole process of the diseases spreading. However, this is not really the case. Mostly, at the initial stages of an emerging infectious disease, both the general individuals and public mass media are unaware of the disease. Media reports, information processing, and individuals alerted responses to the information can only arise as the number of infected individuals reaches and exceeds a certain level. Thus a piecewise system with delay should be analyzed. It would be the following work.

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