

Dynamics of an epidemic model with relapse and delay*

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Abstract

In this paper, we consider a new epidemiological model with delay and relapse phenomena. Firstly, a basic reproduction number R_0 is identified, which serves as a threshold parameter for the stability of the equilibria of the model. Then, beginning with the delay-free model, the global asymptotic stability of the equilibria is obtained through the construction of suitable Lyapunov functions. For the delay model, the stability of the positive equilibrium and the existence of the local Hopf bifurcation are discussed. Furthermore, the application of the normal form theory and center manifold theorem is used to determine the direction and stability of these Hopf bifurcations. Finally, we shed light on corresponding biological implications from a numerical perspective. It turns out that time delay affects the stability of the positive equilibrium, leading to the occurrence of periodic oscillations and disease recurrence.

Keywords Delay; relapse; stability; persistence; Hopf bifurcation.

1 Introduction

Infectious diseases are illnesses caused by pathogenic microorganisms, such as viruses, bacteria, parasites, and fungi, that can be transmitted from one individual to another through direct or indirect contact, leading to a wide range of symptoms and potentially severe consequences. To gain deeper insights into the dynamics of disease transmission, mathematicians have dedicated their efforts to constructing infectious disease models, enabling insightful predictions of infection spread and meticulous evaluations of the efficacy of control measures. One of the most basic SIR models was posed by Kermack and McKendrick in 1927 [15] and 1932 [16]:

$$\begin{cases} \frac{dS}{dt} = -\beta SI, \\ \frac{dI}{dt} = \beta SI - \gamma I, \\ \frac{dR}{dt} = \gamma I, \end{cases}$$

where S , I , and R are respectively the densities of susceptible, infective, and recovered individuals, β is the transmission coefficient, that is, the average number of effective contacts of an infective individual per unit time, βSI is the total number of individuals infected by infective individuals per unit of time, and γ is the ~~recover rate of infective individuals. rate at which infective individuals recover.~~

It has been well-acknowledged that some diseases, including Herpes simplex virus type 2 and tuberculosis in adults [3, 19, 25], ~~can have the ability to~~ establish latent infections: lie dormant in

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the body for a long period of time and reactivate later to induce relapse. Furthermore, as time elapses, the initial immune response triggered by the initial infection may gradually wane, which can also increase the likelihood of disease recurrence and subsequent reinfection of the individual. To our best knowledge, Tudor [22] firstly investigated the concept of relapse by proposing a compartmental model (later recognized as the SIRS model) incorporating bilinear incidence and constant population size (i.e. $N = S + I + R$, where N is constant). Building upon Tudor's work, Moreira and Wang [18] further extended the model by incorporating an incidence term that depended on the size of the susceptible class. Since then, a lot of SIRS epidemic models described by ordinary differential equations, functional differential equations and reaction diffusion equation with free boundary problem have been proposed and analyzed. We refer van den Driessche et al. [5], van den Driessche and Zou [6], Wang and Shu [27], Xu [28], Yang et al. [32], Vargas-De-León [24], Ding et al. [7] and Zhao et al. [33] to cite a few.

Incidence plays an important role in the modelling of epidemic dynamics. In many epidemic models, besides the bilinear incidence rate βSI , the standard incidence rate $\beta SI/N$ is also frequently used. We refer Castillo-Garsow et al. [2], Wang and Yang [26], Fan [10], et al. Especially, in 2013, Vargas-De-León [24] presented an epidemiological model with relapse and disease-induced death, which was expressed as follows:

$$\begin{cases} \frac{dS}{dt} = \Lambda - \frac{\beta SI}{S + I + R} - \mu S, \\ \frac{dI}{dt} = \frac{\beta SI}{S + I + R} - (\alpha + \gamma + \mu)I + \eta R, \\ \frac{dR}{dt} = \gamma I - (\mu + \eta)R, \end{cases}$$

where Λ is the recruitment rate of susceptibles corresponding to births and immigration, μ is the natural death rate of population, α is the disease-induced death rate, γ describes the rate that the infectious individuals become non-infectious individuals and η denotes the rate that the non-infectious individuals are reverted to the infectious state. By employing suitable combinations of well-known functions, namely common quadratic and Volterra-type functions, as well as a composite Volterra-type function, they successfully constructed the Lyapunov functions and proved the conditions for the global stability of the steady states in epidemiological models with relapse.

Noticing that certain diseases do not confer complete immunity on individuals who have recovered from a previous infection. Instead, they may experience temporary and [limited or no immunity limited/no immunity](#). Based on these considerations, Chen [4] formulated the following SIRS model:

$$\begin{cases} \frac{dS}{dt} = \Lambda - Sf(I) - \mu S + \gamma_1 I + \eta_2 R, \\ \frac{dI}{dt} = Sf(I) - (\mu + \gamma_1 + \gamma_2 + \delta)I + \eta_1 R, \\ \frac{dR}{dt} = \gamma_2 I - (\mu + \eta_1 + \eta_2)R, \end{cases} \quad (1.1)$$

to study the global stability of the disease-free equilibrium and the global stability of the endemic equilibrium. In (1.1), the incidence rate is characterized by a nonlinear function $f(I)$, γ_1 is the rate for infectious individuals recovering with no immunity, γ_2 is the rate for infectious individuals recovering with temporary immunity, η_1 and η_2 are respectively the relapse rate and temporary immunity rate, and δ is the additional death rate for infectious individuals.

Due to time delay plays a critical role in infectious disease models, investigating the ways in which infectious diseases are affected by the time delay has become a hot subject. Time delay in epidemic transmission may have a small impact [17], but sometimes [it can also it is also can](#) significantly alter system dynamics, potentially leading to the occurrence of Hopf bifurcation [1]. For example, in 2004, Greenhalgh et al. [11] studied the Hopf bifurcation in two SIRS density-dependent epidemic models and found that although the Hopf bifurcation was theoretically supported, it did not appear to occur for realistic parameter values. As an extension, Enatsu et

al.[8] investigated the stability of delayed SIR models with a class of nonlinear incidence rates, they showed that the stability of endemic equilibrium would be lost as the length of the delay increases past a critical value. In 2014, Xu [29] considered a delayed SIR model (with $f(S, I) = \beta SI$), he established and established the global stability of the disease-free equilibrium and the endemic equilibrium by using LaSalle invariance principle and suitably constructing Lyapunov functionals. Although enormous efforts have been devoted to the study of models with delay [20, 23, 31] or relapse [9, 30], to our best knowledge, the study of the epidemic model including both delay and relapse remains open.

Motivated by [4] and [24], in this paper, we are concerned with the joint effects of disease relapse, standard incidence and the time delay describing latent period on the global dynamics of infectious diseases. We focus on diseases with shorter courses, thereby neglecting the birth and death of the population. To be precise, we introduce the following delay differential equations:

$$\begin{cases} \frac{dS}{dt} = -\frac{\beta S(t-\tau)I(t-\tau)}{S(t-\tau) + I(t-\tau) + R(t-\tau)} + \eta_2 R, \\ \frac{dI}{dt} = \frac{\beta S(t-\tau)I(t-\tau)}{S(t-\tau) + I(t-\tau) + R(t-\tau)} - \gamma I + \eta_1 R, \\ \frac{dR}{dt} = \gamma I - \eta_1 R - \eta_2 R, \end{cases} \quad (1.2)$$

where β is the disease transmission coefficient, γ is the recovery rate, η_1 is the relapse rate, η_2 is the temporary immunity rate, τ estimates the latent period of the disease.

The primary goal of this paper is to offer a complete mathematical analysis of model (1.2) and establish its global dynamics. In the next section, the basic reproduction number is identified, and the well-posedness of the solution of an epidemic model with relapse is also given. In Section 3, the global stability of equilibria is examined in the absence of delays. The main part is Section 4, where we establish the local stability of the positive equilibrium and the existence of a local Hopf bifurcation. The direction and stability of the Hopf bifurcation are determined using the center manifold theorem and the normal form theory. Finally, in Section 5, we conclude with a brief discussion and provide biological interpretations of our main findings through numerical simulations.

2 Well-posedness of the model (2.1)

In accordance with model (1.2), focusing on the biological relevance, we exclusively examine solutions of the system that adhere to the subsequent initial conditions:

$$\begin{aligned} S(\theta) &= \varphi_1(\theta), \quad I(\theta) = \varphi_2(\theta), \quad R(\theta) = \varphi_3(\theta), \\ \varphi_i(\theta) &\geq 0, \quad \theta \in [-\tau, 0], \quad \varphi_i(0) > 0, \quad i = 1, 2, 3, \end{aligned} \quad (2.1)$$

where $(\varphi_1(\theta), \varphi_2(\theta), \varphi_3(\theta)) \in \mathbf{C}([-\tau, 0], \mathbb{R}_+^3)$, which is the Banach space of continuous functions mapping the interval $[-\tau, 0]$ into \mathbb{R}_+^3 with $\mathbb{R}_+^3 = \{(S, I, R) \mid S \geq 0, I \geq 0, R \geq 0\}$. Denote $\mathbf{C}_\tau^+ = \mathbf{C}([-\tau, 0], \mathbb{R}_+^3)$, for all $\varphi = (\varphi_1, \varphi_2, \varphi_3) \in \mathbf{C}_\tau^+$ and $\sigma \in \mathbb{R}$. It follows from Theorem 3.1 in [21] that model (1.2) has a unique solution $(S, I, R) : [\sigma - \tau, +\infty] \rightarrow \mathbb{R}_+^3$, and for $t \in [\sigma - \tau, \sigma]$, $(S(t), I(t), R(t)) = (\varphi_1(t - \sigma), \varphi_2(t - \sigma), \varphi_3(t - \sigma))$. Hence, \mathbf{C}_τ^+ is a forward invariant set of system (1.2).

Since

$$\frac{d(S + I + R)}{dt} = 0,$$

we have $S + I + R \equiv N$ with $N > 0$ being the constant population size. So, model (1.2) can be

reduced to the following equivalent one:

$$\begin{cases} \frac{dI}{dt} = \frac{\beta}{N}(N - I(t - \tau) - R(t - \tau))I(t - \tau) - \gamma I + \eta_1 R, \\ \frac{dR}{dt} = \gamma I - (\eta_1 + \eta_2)R, \end{cases} \quad (2.2)$$

which still satisfies the initial condition (2.1). To sum up, we have the following results.

Lemma 2.1 *Let $(S(t), I(t), R(t))$ be the solution of model (1.2) with initial conditions (2.1). Then $S(t), I(t), R(t)$ are nonnegative and ultimately bounded for all $t \geq 0$.*

Obviously, all feasible solutions of model (1.2) with (2.1) are bounded and enter the region

$$\Omega = \{(S, I, R) \mid S \geq 0, I \geq 0, R \geq 0, S + I + R = N\},$$

where Ω is an invariant set.

Model (2.2) has a disease-free equilibrium

$$E_0 = (0, 0).$$

By introducing the basic reproduction number

$$R_0 = \frac{\beta(\eta_1 + \eta_2)}{\gamma\eta_2}.$$

Model (2.2) has a unique positive equilibrium $E^* = (I^*, R^*)$ with

$$I^* = \frac{N(\eta_1 + \eta_2)}{\eta_1 + \eta_2 + \gamma} \left(1 - \frac{1}{R_0}\right), \quad R^* = \frac{\gamma N}{\eta_1 + \eta_2 + \gamma} \left(1 - \frac{1}{R_0}\right)$$

under the condition $R_0 > 1$.

In the next section, we will ~~We in the next section~~ explore the global stability of the equilibria of model (2.2) without the time delay.

3 Dynamics of the model (2.2) without delay

This section focuses on the dynamics of ~~the delay-free system the limiting system of~~ (2.2), that is

$$\begin{cases} \frac{dI}{dt} = \frac{\beta}{N}(N - I - R)I - \gamma I + \eta_1 R, \\ \frac{dR}{dt} = \gamma I - (\eta_1 + \eta_2)R. \end{cases} \quad (3.1)$$

To prepare, we introduce the following lemma that plays a key role in proving the global stability of equilibrium states.

Lemma 3.1 (Lasalle Invariance Principle [14]) *Let X^* be an equilibrium point for $X' = F(X)$ and let $L : U \rightarrow \mathbb{R}$ be a Liapunov function for X^* , where U is an open set containing X^* . Let $P \subset U$ be a neighborhood of X^* that is closed and bounded. Suppose that P is positively invariant, $X \in P$ and that there is no entire solution in $P - X^*$ on which L is constant. Then X^* is asymptotically stable, and P is contained in the basin of attraction of X^* .*

Theorem 3.1 *If $R_0 \leq 1$, then E_0 of model (3.1) is globally asymptotically stable.*

Proof. Define Lyapunov function $V_0[I, R]$, functional V_0

$$V_0[I, R] = I + \frac{\eta_1}{\eta_1 + \eta_2} R, \quad (3.2)$$

which is positive definite, and reaches the global minimum at E_0 . Differentiating $V_0[I, R]$ with respect to time t along the solution of model (3.1), we have

$$\begin{aligned} \frac{d}{dt} V_0[I, R]|_{(3.1)} &= \frac{\beta}{N} (N - I - R)I - \gamma I + \eta_1 R + \frac{\gamma \eta_1}{\eta_1 + \eta_2} I - \eta_1 R \\ &= -\frac{\beta}{N} (I + R)I + \beta I - \gamma I + \frac{\gamma \eta_1}{\eta_1 + \eta_2} I \\ &= -\frac{\beta}{N} (I + R)I + \left(\beta - \frac{\gamma \eta_1}{\eta_1 + \eta_2} \right) I \\ &= -\frac{\beta}{N} (I + R)I + \beta \left(1 - \frac{1}{R_0} \right) I. \end{aligned}$$

If $R_0 \leq 1$, then $\frac{d}{dt} V_0[I, R]|_{(3.1)} \leq 0$ holds for any $I, R \geq 0$. In addition, $\frac{d}{dt} V_0[I, R]|_{(3.1)} = 0$ if and only if $I = 0, R = 0$. Therefore, E_0 is the largest invariant set of $\{(I, R) \in \mathbb{R}_+^2 : \frac{d}{dt} V_0[I, R]|_{(3.1)} = 0\}$. It follows from LaSalle invariance principle [14] in Lemma 3.1 that E_0 is global asymptotically stable. This completes the proof. \blacksquare

Regarding the stability of E^* , we have

Theorem 3.2 *If $R_0 > 1$, then the endemic equilibrium E^* of model (3.1) is globally asymptotically stable.*

Proof. Define Lyapunov function $V_1[I, R]$ functional V_1 as follows

$$V_1[I, R] = \frac{N}{\beta} \left(I - I^* - I^* \ln \frac{I}{I^*} \right) + \frac{a}{2} (R - R^*)^2 + \frac{N \eta_1 R^*}{\gamma \beta I^*} (R - R^* - R^* \ln \frac{R}{R^*}), \quad (3.3)$$

where a is a positive number to be determined later. Then $V_1[I, R]$ is positive definite on Ω for all $I, R > 0$. Direct calculation shows that the derivative of $V_1[I, R]$ with respect to time t along solutions of (3.1) is given by

$$\begin{aligned} \frac{d}{dt} V_1[I, R]|_{(3.1)} &= \frac{N}{\beta} \left(\frac{I - I^*}{I} \right) \frac{\beta}{N} I \left\{ -(I - I^*) - (R - R^*) + \frac{N \eta_1}{\beta} \left(\frac{R}{I} - \frac{R^*}{I^*} \right) \right\} \\ &\quad + a(R - R^*) \{ \gamma(I - I^*) - (\eta_1 + \eta_2)(R - R^*) \} \\ &\quad + \frac{N \eta_1 R^*}{\gamma \beta I^*} \frac{R - R^*}{R} [\gamma(I - I^*) - (\eta_1 + \eta_2)(R - R^*)] \\ &= (I - I^*) \{ -(I - I^*) - (R - R^*) \} \\ &\quad + a(R - R^*) \{ \gamma(I - I^*) - (\eta_1 + \eta_2)(R - R^*) \} \\ &\quad + \frac{N \eta_1}{\beta} (I - I^*) \left(\frac{R}{I} - \frac{R^*}{I^*} \right) + \frac{N \eta_1 R^*}{\gamma \beta I^*} \gamma I^* \left(1 - \frac{R^*}{R} \right) \left(\frac{I}{I^*} - \frac{R}{R^*} \right) \\ &= -(I - I^*)^2 - (I - I^*)(R - R^*) + a \gamma (I - I^*)(R - R^*) \\ &\quad - a(\eta_1 + \eta_2)(R - R^*)^2 + \frac{N \eta_1}{\beta} R^* \left(\frac{I}{I^*} - 1 \right) \left(\frac{R I^*}{R^* I} - 1 \right) \\ &\quad + \frac{N \eta_1 R^*}{\gamma \beta I^*} \gamma I^* \left(1 - \frac{R^*}{R} \right) \left(\frac{I}{I^*} - \frac{R}{R^*} \right) \\ &\text{(By choosing } a = \frac{1}{\gamma} \text{ such that } -(I - I^*)(R - R^*) + a \gamma (I - I^*)(R - R^*) = 0) \end{aligned}$$

$$\begin{aligned}
&= -(I - I^*)^2 - \frac{\eta_1 + \eta_2}{\gamma}(R - R^*)^2 + \frac{N\eta_1}{\beta}R^* \left(\frac{I}{I^*} - 1 \right) \left(\frac{RI^*}{R^*I} - 1 \right) \\
&\quad + \frac{N\eta_1 R^*}{\gamma\beta I^*} \gamma I^* \left(1 - \frac{R^*}{R} \right) \left(\frac{I}{I^*} - \frac{R}{R^*} \right) \\
&= -(I - I^*)^2 - \frac{\eta_1 + \eta_2}{\gamma}(R - R^*)^2 + \frac{N\eta_1}{\beta}R^* \left(2 - \frac{(IR^*)^2 + (I^*R)^2}{I^*RIR^*} \right).
\end{aligned}$$

From the basic inequality, $2 - \frac{(IR^*)^2 + (I^*R)^2}{I^*RIR^*} \leq 0$. Therefore, $\frac{d}{dt}V_1[I, R]|_{(3.1)}$ is nonpositive definite on Ω for any $I(t)$ and $R(t)$. In addition, $\frac{d}{dt}V_1[I, R]|_{(3.1)} = 0$ holds if and only if $I(t) = I^*, R(t) = R^*$. From LaSalle invariance principle [14] in Lemma 3.1, E^* is globally asymptotically stable in Ω . This completes the proof. \blacksquare

4 Stability and Hopf bifurcation of model (2.2)

In this section, our focus lies on discussing the stability of the equilibrium and the existence of the Hopf bifurcation for model (2.2) when $\tau > 0$. The bifurcation direction and stability of the model are given by using the normal form theory and the central manifold theorem.

Theorem 4.1 *If $R_0 \leq 1$ and $\tau > 0$, then the diseases-free equilibrium E_0 of model (2.2) is globally asymptotically stable.*

Proof. Define Lyapunov [function functional](#) $V_2[I, R]$ as follows

$$V_2[I, R] = I + \frac{\eta_1}{\eta_1 + \eta_2}R + \beta \int_{t-\tau}^t I(\theta)d\theta - \frac{\beta}{N} \int_{t-\tau}^t (I(\theta) + R(\theta))I(\theta)d\theta, \quad (4.1)$$

which is positive definite. Differentiating V_2 with time t along the solution of model (2.2), we obtain

$$\begin{aligned}
\frac{d}{dt}V_2[I, R]|_{(2.2)} &= \frac{\beta}{N}(N - I_\tau - R_\tau)I_\tau - \gamma I + \eta_1 R + \frac{\gamma\eta_1}{\eta_1 + \eta_2}I - \eta_1 R \\
&\quad + \beta I - \beta I_\tau - \frac{\beta}{N}((I + R)I - (I_\tau + R_\tau)I_\tau) \\
&= \beta I - \frac{\beta}{N}(I + R)I - \frac{\gamma\eta_1}{\eta_1 + \eta_2}I \\
&= -\frac{\beta}{N}(I + R)I + \beta \left(1 - \frac{1}{R_0} \right) I,
\end{aligned} \quad (4.2)$$

where $I_\tau := I(t - \tau), R_\tau := R(t - \tau)$. Obviously, if $R_0 \leq 1$, then $\frac{d}{dt}V_2[I, R]|_{(2.2)} \leq 0$. In addition, $\frac{d}{dt}V_2[I, R]|_{(2.2)} = 0$ if and only if $I(t) = 0$ and $R(t) = 0$. Let Γ be the largest invariant set of $\{(I(t), R(t)) \in \mathbb{R}_+^2 : \frac{d}{dt}V_2[I, R]|_{(2.2)} = 0\}$. Then $\Gamma = \{E_0\}$. According to [LaSalle LaSalle's](#) invariance principle [14] in Lemma 3.1, E_0 is globally asymptotically stable if $R_0 \leq 1$. This completes the proof. \blacksquare

We now study the stability of the endemic equilibrium E^* in the case $\tau > 0$. Consider the linearization of model (2.2) at equilibrium E^* ,

$$x'(t) = A^*x(t) + B^*x(t - \tau), \quad (4.3)$$

where $x(t) = (I(t), R(t))^T$,

$$\begin{aligned}
A^* &= \begin{pmatrix} -\gamma & \eta_1 \\ \gamma & -(\eta_1 + \eta_2) \end{pmatrix}, \\
B^* &= \begin{pmatrix} \beta - \frac{2\beta}{N}I^* - \frac{\beta}{N}R^* & -\frac{\beta}{N}I^* \\ 0 & 0 \end{pmatrix}.
\end{aligned}$$

The characteristic equation of (4.3) is formulated as (Hale [13])

$$\det[\lambda I - A^* - e^{-\lambda\tau} B^*] = 0, \quad (4.4)$$

i.e.,

$$\begin{vmatrix} \lambda + \gamma - e^{-\lambda\tau} \left(\beta - \frac{2\beta}{N} I^* - \frac{\beta}{N} R^* \right) & -\eta_1 + e^{-\lambda\tau} \frac{\beta}{N} I^* \\ -\gamma & \lambda + (\eta_1 + \eta_2) \end{vmatrix} = 0.$$

The eigenvalues of (4.4) at equilibrium E^* are the roots of

$$\lambda^2 + A\lambda + B = e^{-\lambda\tau}(C\lambda + D), \quad (4.5)$$

where

$$\begin{aligned} A &= \gamma + \eta_1 + \eta_2, \quad B = \gamma\eta_2, \\ C &= \beta - \frac{2\beta}{N} I^* - \frac{\beta}{N} R^* = \beta \left(1 - \frac{2}{N} I^* - \frac{1}{N} \frac{\gamma}{\eta_1 + \eta_2} I^* \right) = \beta \left(1 - \frac{2(\eta_1 + \eta_2) + \gamma}{N(\eta_1 + \eta_2)} I^* \right), \\ D &= \left(\beta - \frac{2\beta}{N} I^* - \frac{\beta}{N} R^* \right) (\eta_1 + \eta_2) - \frac{\beta\gamma}{N} I^* = (\eta_1 + \eta_2)C - \frac{\beta\gamma}{N} I^*. \end{aligned}$$

When $\tau = 0$, the characteristic equation (4.5) becomes

$$\lambda^2 + (A - C)\lambda + (B - D) = 0. \quad (4.6)$$

The simple Simple calculation yields that $A - C > 0, B - D > 0$. Thus, both the eigenvalues of the characteristic equation (4.6) have negative real parts. Therefore, the positive equilibrium E^* is locally asymptotically stable at $\tau = 0$.

By Theorem 4.4 of Hal Smith [21], for delay small enough, the characteristic roots of (4.5) are either very near the eigenvalues of (4.6) or have more negative real parts than any of the eigenvalues of (4.6). Hence, when the delay is small, the equilibrium E^* is locally asymptotically stable. When $R_0 > 1$, for any $\tau > 0$, zero is not a root of (4.5). Note that any complex roots to the equations (4.5) appear in pairs, and all roots of (4.5) have negative real parts if $\tau = 0$. Therefore, any root of (4.5) has a negative real part for sufficiently small τ . Assume that there exists $\tau = \hat{\tau}$, such that (4.6) has a pair of purely imaginary roots, denoted by $\lambda = \pm i\omega$, ($\omega > 0$). Substituting $\lambda = i\omega$ into (4.5), we have

$$-\omega^2 + iA\omega + B = (\cos \omega\tau - i \sin \omega\tau)(C\omega i + D).$$

Separating the real and imaginary part, we have

$$\begin{cases} C\omega \sin \omega\tau + D \cos \omega\tau = -\omega^2 + B, \\ C\omega \cos \omega\tau - D \sin \omega\tau = A\omega, \end{cases} \quad (4.7)$$

which leads to

$$\omega^4 + (A^2 - 2B - C^2)\omega^2 + (B^2 - D^2) = 0. \quad (4.8)$$

Let $z = \omega^2$. Then

$$z^2 + pz + q = 0, \quad (4.9)$$

where

$$p = A^2 - 2B - C^2, q = B^2 - D^2.$$

Hurwitz criterion implies that equation (4.9) has no positive roots if $p > 0, q > 0$. Hence, $\hat{\tau}$ doesn't exist, and all solutions of equation (4.9) have a negative real part and E^* is locally asymptotically stable for any $\tau > 0$. Therefore, we have the following theorem.

Theorem 4.2 *If $R_0 > 1$, $p > 0$, and $q > 0$, then the endemic equilibrium E^* of model (2.2) is locally asymptotically stable for any $\tau > 0$.*

Theorem 4.3 *If $R_0 > 1$ and $q < 0$, then there exists a $\hat{\tau} > 0$ such that equation (4.8) has a pair of conjugate purely imaginary roots $\pm i\hat{\omega}$ when $\tau = \hat{\tau}$. Moreover, the endemic equilibrium E^* of model (2.2) is locally asymptotically stable if $\tau < \hat{\tau}$.*

Proof. It is obvious that if $q < 0$, then equation (4.9) has a positive root, denoted by z . Then

$$\tau_n = \frac{1}{\omega} \left[\arccos \frac{(AC - D)\omega^2 + BD}{C^2\omega^2 + D^2} + 2n\pi \right],$$

where $n \in \mathbb{Z}^+$. Let

$$\hat{\tau} = \tau_0.$$

When $\tau = \hat{\tau}$, equation (4.9) has a pair of conjugate purely imaginary roots. Note that all roots of equation (4.9) have negative real part if $\tau = 0$, and all roots of equation (4.9) have negative real part if $\tau < \hat{\tau}$, which follows from the continuous dependence of the solution on τ . Thus, E^* is locally asymptotically stable. ■

We next aim to determine the circumstances under which the equilibrium E^* turns into an unstable one, leading to the occurrence of a Hopf bifurcation. This is equivalent to identifying conditions that ensure the characteristic equation (4.9) possesses a root with a negative real part and a pair of conjugate purely imaginary roots. Furthermore, we will validate the transversal condition necessary for the existence of the Hopf bifurcation.

Theorem 4.4 *Suppose $R_0 > 1$ and $q < 0$. Then there exists a $\hat{\tau} > 0$, such that there is a Hopf bifurcation of model (2.2) from equilibrium E^* as τ passes through the critical value $\hat{\tau}$ if $\tau > \hat{\tau}$.*

Proof. For τ sufficiently close to $\hat{\tau}$, by the implicit function theorem, the eigenvalue of (4.5) can be set as $\lambda(\tau) = \xi(\tau) + i\omega(\tau)$. As τ increases and passes through $\hat{\tau}$, the eigenvalue passes through the imaginary axis, and $\xi(\hat{\tau}) = 0$, $\omega(\hat{\tau}) = \hat{\omega}$. We claim that $\frac{d(\operatorname{Re}\lambda)}{dt} \Big|_{\tau=\hat{\tau}} > 0$. Calculating the derivative of equation (4.9) with respect to τ , we obtain

$$(2\lambda + A) \frac{d\lambda}{d\tau} = C e^{-\lambda\tau} \frac{d\lambda}{d\tau} - (C\lambda + D) e^{-\lambda\tau} (\lambda + \tau \frac{d\lambda}{d\tau}).$$

This gives

$$\frac{d\lambda}{d\tau} = \frac{-\lambda(C\lambda + D)e^{-\lambda\tau}}{2\lambda + A + (\tau(C\lambda + D) - C)e^{-\lambda\tau}},$$

thus

$$\begin{aligned} \left[\frac{d(\operatorname{Re}\lambda)}{d\tau} \Big|_{\tau=\hat{\tau}} \right]^{-1} &= \frac{2\lambda + A + (\tau(C\lambda + D) - C)e^{-\lambda\tau}}{-\lambda(C\lambda + D)e^{-\lambda\tau}} \Big|_{\tau=\hat{\tau}} \\ &= \frac{1}{Z} (A \cos \hat{\omega}\hat{\tau} - 2\omega \sin \hat{\omega}\hat{\tau} + D\hat{\tau} - C + i(A \sin \hat{\omega}\hat{\tau} + 2\hat{\omega} \cos \hat{\omega}\hat{\tau} + C\hat{\omega}\hat{\tau})) \\ &= \frac{1}{Z} (2\hat{\omega}^4 + (A^2 - 2B - C^2)\hat{\omega}^2) \\ &= \frac{1}{Z} (2z_*^2 + (A^2 - 2B - C^2)z_*), \end{aligned} \quad (4.10)$$

where $\hat{\omega}^2 = z_*$ and

$$Z = C^2\hat{\omega}^4 + D^2\hat{\omega}^2 > 0. \quad (4.11)$$

Since

$$z_*^2 + (A^2 - 2B - C^2)z_* + (B^2 - D^2) = 0 \quad (4.12)$$

and $B^2 - D^2 < 0$, we have $2z_*^2 + (A^2 - 2B - C^2)z_* = z_*^2 + [z_*^2 + (A^2 - 2B - C^2)z_*] > 0$. Thus,

$$\frac{d(\operatorname{Re}\lambda)}{dt} \Big|_{\tau=\hat{\tau}} > 0.$$

This completes the proof. ■

Remark 4.1 By Theorem 4.4, we can conclude that if there exist p, q such that $z_*^2 + pz_* + q = 0$ and $\frac{1}{Z}(2z_*^2 + pz_*) > 0$, then periodic solutions are bifurcated near the positive equilibrium E^* .

From Remark 4.1, model (2.2) occurs Hopf bifurcation at the equilibrium $E^*(I^*, R^*)$ when $\tau = \tau_j (j = 0, 1, 2, \dots)$. Next, we shall establish the explicit formulae determining the direction, stability, and period of these periodic solutions bifurcating from equilibrium $E^*(I^*, R^*)$ at these critical values of τ by using the normal theory and the center manifold [12]. Without loss of generality, we denote any one of these critical values $\tau = \tau_n (n = 0, 1, 2, \dots)$ by $\hat{\tau}$, at which model (2.2) has a pair of purely imaginary roots $\pm i\omega$, where the corresponding values of ω are $\hat{\omega}$ when $\hat{\tau} = \tau_n$, and model (2.2) occurs a Hopf bifurcation at $E^*(I^*, R^*)$.

Let $x(t) = I(\tau t) - I^*$, $y(t) = R(\tau t) - R^*$ and $t = s\tau$, denoting s as t . Then model (2.2) can be rewritten as

$$\begin{pmatrix} \dot{x}(t) \\ \dot{y}(t) \end{pmatrix} = \tau A_1 \begin{pmatrix} x(t) \\ y(t) \end{pmatrix} + \tau B_1 \begin{pmatrix} x(t-1) \\ y(t-1) \end{pmatrix} + F(x_t, y_t, \tau), \quad (4.13)$$

where

$$A_1 = \begin{pmatrix} -\gamma & \eta_1 \\ \gamma & -(\eta_1 + \eta_2) \end{pmatrix}, \quad B_1 = \begin{pmatrix} \frac{\beta}{N}(N - I^* - R^*) - \frac{\beta}{N}I^* & -\frac{\beta}{N}I^* \\ 0 & 0 \end{pmatrix},$$

and

$$F = \tau \begin{pmatrix} \frac{\beta}{N}(-x^2(t-1) - x(t-1)y(t-1)) + \frac{\beta}{N}(N - I^* - R^*)I^* - \gamma I^* + \eta_1 R^* \\ \gamma I^* - (\eta_1 + \eta_2)R^* \end{pmatrix}.$$

Let $\hat{\tau}$ be the critical value of τ where model (2.2) undergoes a Hopf bifurcation at E^* . Assume $\tau = \hat{\tau} + h$, then $h = 0$ is the Hopf bifurcation value of model (2.2).

Choose the phase space $C = C([-1, 0], R^2)$. Define $L(h) : C \rightarrow R^2$, model (2.2) be locally expressed as

$$\dot{x}(t) = L(h)\phi + f(h, \phi), \quad (4.14)$$

$$L(h)\phi = (\hat{\tau} + h)A_1\phi(0) + (\hat{\tau} + h)B_1\phi(-1), \phi \in C.$$

From the Riesz representation theorem, there exists a matrix whose components are bounded variation functions $\eta(\theta, h) : [-1, 0] \rightarrow R^2, \theta \in [-1, 0]$, such that the linearized system of (4.14) is

$$L(h)\phi \doteq \int_{-1}^0 d\eta(\theta, h)\phi(\theta), \phi \in C. \quad (4.15)$$

Select $\eta(\theta, h) = (\hat{\tau} + h)A_1\delta(\theta) - (\hat{\tau} + h)B_1\delta(\theta + 1)$, where

$$\delta(\theta) = \begin{cases} 1, & \theta = 0, \\ 0, & \theta \neq 0. \end{cases}$$

For $\phi \in C^1([-1, 0], R^2)$, the corresponding infinitesimal generator $A(h)$ of (4.15) gives

$$A(h)\phi = \begin{cases} \dot{\phi}(\theta), & \theta \in [-1, 0), \\ \int_{-1}^0 d\eta(t, h)\phi(t), & \theta = 0. \end{cases}$$

Define

$$R(h)\phi = \begin{cases} 0, & \theta \in [-1, 0), \\ F(\phi, \hat{\tau} + h), & \theta = 0, \end{cases}$$

then (4.14) is equivalent to the abstract ordinary differential equation

$$\dot{\phi} = A(h)\phi + R(h)\phi. \quad (4.16)$$

Next, we analyze the abstract equation (4.16). For $\varphi \in C^1([0, 1], (C^2)^*)$, where $(C^2)^*$ is a two-dimensional complex row vector space, we define formal adjoint operator of $A(h)$ as

$$A^* \varphi(s) = \begin{cases} -\dot{\varphi}(s), & s \in (0, 1], \\ \int_{-1}^0 \varphi(-t) d\eta(t, 0) = \hat{\tau}\varphi(0)A_1 + \hat{\tau}\varphi(1)B_1, & s = 0. \end{cases}$$

Here, for $\phi \in C([-1, 0], C^2)$ with $\varphi \in C([0, 1], (C^2)^*)$, the bilinear form suitable for a complex vector is

$$\langle \varphi, \phi \rangle = \bar{\varphi}(0)\phi(0) - \int_{-1}^0 \int_0^\theta \bar{\varphi}(\xi - \theta) d\eta(\theta, 0) \phi(\xi) d\xi.$$

Since

$$\begin{aligned} \langle \varphi(s), A(0)\phi(\theta) \rangle &= \bar{\varphi}(0)A(0)\phi(\theta) - \int_{-1}^0 \int_{\xi=0}^\theta \bar{\varphi}(\xi - \theta) d\eta(\theta) A(0)\phi(\xi) d\xi \\ &= \bar{\varphi}(0) \int_{-1}^0 d\eta(\theta)\phi(\theta) - \int_{-1}^0 \int_{\xi=0}^\theta \bar{\varphi}(\xi - \theta) d\eta(\theta) \dot{\phi}(\xi) d\xi \\ &= \bar{\varphi}(0) \int_{-1}^0 d\eta(\theta)\phi(\theta) - \int_{-1}^0 [\bar{\varphi}(\xi - \theta) d\eta(\theta)\phi(\xi)]_{\xi=0}^\theta \\ &\quad + \int_{-1}^0 \int_{\xi=0}^\theta \frac{d\bar{\varphi}(\xi - \theta)}{d\xi} d\eta(\theta)\phi(\xi) d\xi \\ &= \int_{-1}^0 \bar{\varphi}(-\theta) d\eta(\theta)\phi(0) - \int_{-1}^0 \int_{\xi=0}^\theta \left[-\frac{d\bar{\varphi}(\xi - \theta)}{d\xi} \right] d\eta(\theta)\phi(\xi) d\xi \\ &= A^* \bar{\varphi}(0)\phi(0) - \int_{-1}^0 \int_{\xi=0}^\theta A^* \bar{\varphi}(\xi - \theta) d\eta(\theta)\phi(\xi) d\xi \\ &= \langle A^* \varphi(s), \phi(\theta) \rangle, \end{aligned}$$

$A(0)$ and A^* are adjoint operators which satisfies $\langle \varphi, A\phi \rangle = \langle A^* \varphi, \phi \rangle$.

Recalling that $\pm\omega\hat{\tau}$ is a pair of ~~an~~ eigenvalues of $A(0)$, which is also an eigenvalue of A^* , we come to the following conclusion.

Lemma 4.1 $q(\theta) = (1, \alpha_1)^\top e^{i\hat{\omega}\hat{\tau}\theta}$ is the eigenvector of operator A on $i\hat{\omega}\hat{\tau}$, $q^*(s) = \bar{D}(1, \alpha_2) e^{i\hat{\omega}\hat{\tau}s}$ is the eigenvector of operator A^* on $-i\hat{\omega}\hat{\tau}$, and $\langle q^*, q \rangle = 1$, $\langle q^*, \bar{q} \rangle = 0$, where

$$\alpha_1 = \frac{\gamma}{i\hat{\omega} + (\eta_1 + \eta_2)}, \quad \alpha_2 = \frac{\frac{\beta}{N} I^* e^{i\hat{\omega}\hat{\tau}} - \eta_1}{i\hat{\omega} - (\eta_1 + \eta_2)},$$

and

$$D = \frac{1}{1 + \alpha_1 \bar{\alpha}_2 + \hat{\tau} e^{-i\hat{\omega}\hat{\tau}} \left(\frac{\beta}{N} (N - I^* - R^*) - \frac{\beta}{N} I^* - \alpha_1 \frac{\beta}{N} I^* \right)}.$$

Proof. Let $q(\theta) = (1, \alpha_1)^\top e^{i\hat{\omega}\hat{\tau}\theta}$ be an eigenvector of operator A on $i\hat{\omega}\hat{\tau}$, it follows from $Aq(0) = i\hat{\omega}\hat{\tau}q(0)$ that α_1 satisfies the following equation

$$\hat{\tau}A_1 \begin{pmatrix} 1 \\ \alpha_1 \end{pmatrix} + \hat{\tau}B_1 \begin{pmatrix} e^{-i\hat{\omega}\hat{\tau}} \\ \alpha_1 \end{pmatrix} = i\hat{\omega}\hat{\tau} \begin{pmatrix} 1 \\ \alpha_1 \end{pmatrix}.$$

Substituting the A_1, B_1 , we obtain

$$\hat{\tau} \begin{pmatrix} -\gamma & \eta_1 \\ -\gamma & -(\eta_1 + \eta_2) \end{pmatrix} \begin{pmatrix} 1 \\ \alpha_1 \end{pmatrix} + \hat{\tau} \begin{pmatrix} \frac{\beta}{N}(N - I^* - R^*) - \frac{\beta}{N} I^* & -\frac{\beta}{N} I^* \\ 0 & 0 \end{pmatrix} \begin{pmatrix} 1 \\ \alpha_1 \end{pmatrix} = \hat{\tau} \begin{pmatrix} i\hat{\omega} \\ \alpha_1 i\hat{\omega} \end{pmatrix},$$

and hence

$$\alpha_1 = \frac{\gamma}{i\hat{\omega} + (\eta_1 + \eta_2)}.$$

Following a similar procedure, we have

$$\alpha_2 = \frac{\eta_1 - \frac{\beta}{N} I^* e^{-i\hat{\omega}\hat{\tau}}}{\eta_1 + \eta_2 - i\hat{\omega}}.$$

Since

$$q(0) = (1, \alpha_1)^\top, \bar{q}^*(0) = D(1, \alpha_2),$$

we get

$$\begin{aligned} \langle q^*, q \rangle &= \bar{q}^*(0)q(0) - \int_{-1}^0 \int_{\xi=0}^0 \bar{q}^*(\xi - \theta) d\eta(\theta, 0) q(\xi) d\xi \\ &= D(1 + \alpha_1 \bar{\alpha}_2) - \int_{-1}^0 \int_{\xi=0}^0 D(1, \bar{\alpha}_2) e^{-i\hat{\omega}\hat{\tau}(\xi - \theta)} d\eta(\theta, 0) (1, \alpha_1)^\top e^{i\hat{\omega}\hat{\tau}\xi} d\xi \\ &= D(1 + \alpha_1 \bar{\alpha}_2) - D(1, \bar{\alpha}_2) \int_{-1}^0 d\eta(\theta, 0) \theta e^{i\hat{\omega}\hat{\tau}\theta} (1, \alpha_1)^\top \\ &= D \left[1 + \alpha_1 \bar{\alpha}_2 + \hat{\tau} e^{-i\hat{\omega}\hat{\tau}} \left(\frac{\beta}{N} (N - I^* - R^*) - \frac{\beta}{N} I^* - \alpha_1 \frac{\beta}{N} I^* \right) \right]. \end{aligned}$$

The equality $\langle q^*, q \rangle = 1$ requires

$$D = \frac{1}{1 + \alpha_1 \bar{\alpha}_2 + \hat{\tau} e^{-i\hat{\omega}\hat{\tau}} \left(\frac{\beta}{N} (N - I^* - R^*) - \frac{\beta}{N} I^* - \alpha_1 \frac{\beta}{N} I^* \right)}.$$

The proof of $\langle q^*, \bar{q} \rangle = 0$ has been given in Hassard et al. [12], we are not going to repeat it. The proof is completed. \blacksquare

Using the same notations as in [12], we calculate the coordinates to describe the center manifold C_0 at $h = 0$. Let $\mu_t = (\mu_t^{(1)}, \mu_t^{(2)})^\top$ be the solution of (4.14) when $\tau = \hat{\tau}$. Define

$$z(t) = \langle q^*(s), \mu_t(\theta) \rangle$$

and

$$W(t, \theta) = (W^{(1)}(t, \theta), W^{(2)}(t, \theta))^\top = \mu_t(\theta) - 2\text{Re}\{z(t)q(\theta)\}. \quad (4.17)$$

On the center manifold C_0 , one has

$$W(t, \theta) = W(z(t), \bar{z}(t), \theta),$$

where the $W(z, \bar{z}, \theta)$ can be expressed in the form of power series of z and \bar{z} as following,

$$W(z, \bar{z}, \theta) = W_{20}(\theta) \frac{z^2}{2} + W_{11}(\theta) z\bar{z} + W_{02}(\theta) \frac{\bar{z}^2}{2} + W_{30}(\theta) \frac{z^3}{6} + \dots. \quad (4.18)$$

The flow of (4.14) on the [center manifold](#) is determined by the following equation

$$\dot{z}(t) = i\hat{\omega}\hat{\tau}z(t) + \bar{q}^*(0)F(0, W(z, \bar{z}, 0) + 2\text{Re}\{zq(0)\}) \doteq i\hat{\omega}\hat{\tau}z(t) + \bar{q}^*(0)F_0(z, \bar{z}), \quad (4.19)$$

where

$$F_0(z, \bar{z}) = F(0, W(z, \bar{z}, 0) + 2\text{Re}\{zq(0)\}).$$

Therefore, the next aim is to find the coefficients in $W(z, \bar{z}, \theta)$, and the equation restricted to the center manifold. Equation (4.19) can be rewritten as

$$\dot{z}(t) = i\hat{\omega}\hat{\tau}z(t) + g(z, \bar{z}), \quad (4.20)$$

with

$$g(z, \bar{z}) = \bar{q}^*(0)F_0(z, \bar{z}) = g_{20} \frac{z^2}{2} + g_{11} z\bar{z} + g_{02} \frac{\bar{z}^2}{2} + g_{21} \frac{z^2 \bar{z}}{2} + \dots. \quad (4.21)$$

Thus, the normal form restricted on the central manifold can be obtained by figuring out g_{20}, g_{11}, g_{02} and g_{21} . It follows from (4.17) that

$$\mu_t(\theta) = W(t, \theta) + 2\text{Re}\{z(t)q(\theta)\} = W_{20}(\theta)\frac{z^2}{2} + W_{11}(\theta)z\bar{z} + W_{02}(\theta)\frac{\bar{z}^2}{2} + \cdots + z(t)q(\theta) + \bar{z}(t)\bar{q}(\theta).$$

Thus, we obtain

$$\begin{aligned} x(t) &= z + \bar{z} + W_{20}^{(1)}(0)\frac{z^2}{2} + W_{11}^{(1)}(0)z\bar{z} + W_{02}^{(1)}(0)\frac{\bar{z}^2}{2} + \cdots, \\ y(t) &= z\alpha_1 + \bar{z}\bar{\alpha}_1 + W_{20}^{(2)}(0)\frac{z^2}{2} + W_{11}^{(2)}(0)z\bar{z} + W_{02}^{(2)}(0)\frac{\bar{z}^2}{2} + \cdots, \\ x(t-1) &= ze^{-i\hat{w}\hat{\tau}} + \bar{z}e^{i\hat{w}\hat{\tau}} + W_{20}^{(1)}(-1)\frac{z^2}{2} + W_{11}^{(1)}(-1)z\bar{z} + W_{02}^{(1)}(-1)\frac{\bar{z}^2}{2} + \cdots, \\ y(t-1) &= z\alpha_1 e^{-i\hat{w}\hat{\tau}} + \bar{z}\bar{\alpha}_1 e^{i\hat{w}\hat{\tau}} + W_{20}^{(2)}(-1)\frac{z^2}{2} + W_{11}^{(2)}(-1)z\bar{z} + W_{02}^{(2)}(-1)\frac{\bar{z}^2}{2} + \cdots. \end{aligned}$$

According to (4.21), comparing the coefficients of these two expressions directly yields:

$$\begin{aligned} g_{20} &= [-2\frac{\beta}{N}(1 + \alpha_1)e^{-2i\hat{w}\hat{\tau}}]D\hat{\tau}, \quad g_{11} = [-2\frac{\beta}{N}(1 + \alpha_1)]D\hat{\tau}, \quad g_{02} = [-2\frac{\beta}{N}(1 + \alpha_1)e^{2i\hat{w}\hat{\tau}}]D\hat{\tau}, \\ g_{21} &= -\frac{\beta}{N}[2(2 + \alpha_1)e^{-i\hat{w}\hat{\tau}}W_{11}^{(1)}(-1) + (2 + \alpha_1)e^{i\hat{w}\hat{\tau}}W_{20}^{(1)}(-1) + 2e^{-i\hat{w}\hat{\tau}}W_{11}^{(2)}(-1) + 2e^{i\hat{w}\hat{\tau}}W_{20}^{(2)}(-1)]D\hat{\tau}. \end{aligned}$$

To further specify g_{21} , we combine (4.16), (4.17) and (4.19) to reach

$$\begin{aligned} \dot{W} &= \dot{u}_t - 2\text{Re}\{\dot{z}(t)q(\theta)\} \\ &= A(0)W(t, \theta) + R(0)u_t - 2\text{Re}\{\bar{q}^*(0)F_0(z(t), \bar{z}(t))q(\theta)\} \\ &= \begin{cases} A(0)W(t, \theta) - 2\text{Re}\{\bar{q}^*(0)F_0(z(t), \bar{z}(t))q(\theta)\}, & \theta \in [-1, 0), \\ A(0)W(t, \theta) - 2\text{Re}\{\bar{q}^*(0)F_0(z(t), \bar{z}(t))q(0)\} + F_0(z(t), \bar{z}(t)), & \theta = 0, \end{cases} \quad (4.22) \\ &\doteq A(0)W(t, \theta) + H(z(t), \bar{z}(t), \theta), \end{aligned}$$

where

$$H(z, \bar{z}, \theta) = H_{20}(\theta)\frac{z^2}{2} + H_{11}(\theta)z\bar{z} + H_{02}(\theta)\frac{\bar{z}^2}{2} + \cdots. \quad (4.23)$$

Expanding the above series and comparing the coefficients, one has

$$(A - 2i\hat{w}\hat{\tau})W_{20}(\theta) = -H_{20}(\theta), \quad AW_{11}(\theta) = -H_{11}(\theta), \cdots. \quad (4.24)$$

For $\theta \in [-1, 0)$, it follows from \dot{W} that

$$H(z, \bar{z}, \theta) = -(gq(\theta) + \bar{g}\bar{q}(\theta)). \quad (4.25)$$

Comparing the coefficients with (4.23), (4.25) offers

$$H_{20}(\theta) = -(g_{20}q(\theta) + \bar{g}_{02}\bar{q}(\theta)), \quad (4.26)$$

and

$$H_{11}(\theta) = -(g_{11}q(\theta) + \bar{g}_{11}\bar{q}(\theta)). \quad (4.27)$$

From (4.26), and (4.27) and the definition of $A(0)$, we have

$$\dot{W}_{20}(\theta) = 2i\hat{w}\hat{\tau}W_{20}(\theta) + g_{20}q(\theta) + \bar{g}_{02}\bar{q}(\theta).$$

Note that $q(\theta) = q(0)e^{i\hat{\omega}\hat{\tau}\theta}$, it follows from the method of constant variation that

$$W_{20}(\theta) = \frac{ig_{20}}{\omega\hat{\tau}}q(0)e^{i\omega\hat{\tau}\theta} + \frac{i\bar{g}_{02}}{3\omega\hat{\tau}}\bar{q}(0)e^{-i\omega\hat{\tau}\theta} + e^{2i\omega\hat{\tau}\theta}E_1.$$

Similarly, from (4.22), and (4.27) and the definition of $A(0)$, we have

$$W_{11}(\theta) = \frac{ig_{11}}{\omega\hat{\tau}}q(0)e^{i\omega\hat{\tau}\theta} + \frac{i\bar{g}_{11}}{\omega\hat{\tau}}\bar{q}(0)e^{-i\omega\hat{\tau}\theta} + E_2.$$

In what follows, we shall seek appropriate E_1 and E_2 in order to obtain W_{20}, W_{11} , so that we can finally figure g_{21} . By the definition of $A(0)$ and (4.22), we know that

$$\int_{-1}^0 d\eta(\theta)W_{20}(\theta) = 2i\hat{\omega}\hat{\tau}W_{20}(0) - H_{20}(0),$$

and

$$\int_{-1}^0 d\eta(\theta)W_{11}(\theta) = -H_{11}(0).$$

Note that $q(\theta)$ is the eigenvalue of $A(0)$ and the connection E_1 with W_{20} as well as the definition of $A(0)$, we have

$$\begin{aligned} \int_{-1}^0 d\eta(\theta)W_{20}(\theta) &= \frac{ig_{20}}{\hat{\omega}\hat{\tau}} \int_{-1}^0 d\eta(\theta)q(0) + \frac{i\bar{g}_{02}}{3\hat{\omega}\hat{\tau}} \int_{-1}^0 d\eta(\theta)\bar{q}(0) + \int_{-1}^0 d\eta(\theta)e^{2i\hat{\omega}\hat{\tau}\theta}E_1 \\ &= -g_{20}q(0) + \frac{\bar{g}_{02}}{3}\bar{q}(0) + \int_{-1}^0 d\eta(\theta)e^{2i\hat{\omega}\hat{\tau}\theta}E_1. \end{aligned}$$

Hence,

$$-g_{20}q(0) - \bar{g}_{02}\bar{q}(0) + (2i\hat{\omega}\hat{\tau}I - \int_{-1}^0 d\eta(\theta)e^{2i\hat{\omega}\hat{\tau}\theta})E_1 = H_{20}(0).$$

Similarly,

$$-(g_{11}q(0) + \bar{g}_{11}\bar{q}(0)) - \int_{-1}^0 d\eta(\theta)E_2 = H_{11}(0),$$

and hence

$$H(z, \bar{z}, 0) = -(g_{20}q(0) + \bar{g}_{02}\bar{q}(0))\frac{z^2}{2} - (g_{11}q(0) - \bar{g}_{11}\bar{q}(0))z\bar{z} + F_0(z, \bar{z}) + \dots.$$

Thus

$$H_{20}(0) = -(g_{20}q(0) + \bar{g}_{02}\bar{q}(0)) + \hat{\tau} \begin{pmatrix} -\frac{2\beta}{N}(1 + \alpha_1)e^{-2i\hat{\omega}\hat{\tau}} \\ 0 \end{pmatrix}$$

and

$$H_{11}(0) = -g_{11}q(0) - \bar{g}_{11}\bar{q}(0) + \hat{\tau} \begin{pmatrix} -2\frac{\beta}{N}(1 + \alpha_1) \\ 0 \end{pmatrix}.$$

From (4.24) and the definition of A ,

$$\hat{\tau}A_1W_{20}(0) + \hat{\tau}B_1W_{20}(-1) = 2i\hat{\omega}\hat{\tau}W_{20}(0) - H_{20}(0)$$

and

$$\hat{\tau}A_1W_{11}(0) + \hat{\tau}B_1W_{11}(-1) = -H_{11}(0).$$

Therefore, we obtain

$$\begin{aligned} E_1 &= -\frac{1}{\hat{\tau}}(A_1 + B_1^{-2i\hat{\omega}\hat{\tau}} - 2i\hat{\omega}I)^{-1}[H_{20}(0) + 2g_{20}q(0) + \frac{2}{3}\bar{g}_{02}\bar{q}(0) - \frac{i}{\hat{\omega}}g_{20}A_1\bar{q}(0) + \frac{i}{3\hat{\omega}}\bar{g}_{02}A_1\bar{q}(0) \\ &\quad + \frac{i}{\hat{\omega}}g_{20}e^{-i\hat{\omega}\hat{\tau}}B_1q(0) + \frac{i}{3\hat{\omega}}\bar{g}_{02}e^{i\hat{\omega}\hat{\tau}}B\bar{q}(0)], \\ E_2 &= -\frac{1}{\hat{\tau}}(A_1 + B_1)^{-1}(H_{11}(0) + \frac{2}{\hat{\omega}}A_1Im(g_{11}(0)q(0)) + \frac{2}{\hat{\omega}}B_1Im(g_{11}q(0)e^{-i\hat{\omega}\hat{\tau}})), \end{aligned}$$

With g_{21} specified, we can obtain $C_1(0)$, μ_2 , β_2 and T_2 as follows:

$$\begin{aligned} C_1(0) &= \frac{i}{2\hat{\omega}\hat{\tau}}(g_{20}g_{11} - 2|g_{11}|^2 - \frac{|g_{02}|^2}{3}) + \frac{g_{21}}{2}, \\ \mu_2 &= -\frac{Re(C_1(0))}{Re\lambda'(\hat{\tau})}, \\ \beta_2 &= 2Re(C_1(0)), \\ T_2 &= -\frac{ImC_1(0) + \mu_2Im\lambda'(0)}{\hat{\omega}\hat{\tau}}. \end{aligned}$$

Utilizing the results of Hassard et al. [12], we hit the following theorem.

Theorem 4.5 *Suppose that $\tau = \hat{\tau}$ and $\frac{d(Re\lambda)}{dt}|_{\tau=\hat{\tau}} \neq 0$, then*

- (i) *If $\mu_2 > 0$ ($\mu_2 < 0$), then the Hopf bifurcation is supercritical (subcritical).*
- (ii) *If $\beta_2 < 0$ ($\beta_2 > 0$), then the bifurcating periodic solutions are stable (unstable).*
- (iii) *If $T_2 > 0$ ($T_2 < 0$), then the period of the bifurcating periodic solutions increases (decreases).*

5 Discussions

In this paper, we introduce and analyze an epidemic model that incorporates delay and relapse phenomena. We derive threshold dynamics for the model without delay, which is determined by the basic reproduction number $R_0 = \beta(\eta_1 + \eta_2)/\gamma\eta_2$. If $R_0 \leq 1$, then the disease-free equilibrium E_0 of the model (2.2) is globally asymptotically stable. Otherwise, the endemic equilibrium E^* of the model (2.2) is globally asymptotically stable. We also investigate the stability of the positive equilibrium and examine the presence of local Hopf bifurcation. Assuming that $R_0 > 1$ and $q < 0$. We identify a threshold value $\hat{\tau} > 0$ which signifies the occurrence of a Hopf bifurcation within the model (2.2). The bifurcation emanates from the equilibrium E^* , as the parameter τ transgresses the critical value $\hat{\tau}$ provided that $\tau > \hat{\tau}$. By using the principles of normal form theory and the center manifold theorem, we effectively determine both the direction and stability characteristics of these Hopf bifurcations.

To numerically explore our theoretical results and possible biological implications, we set

$$\beta = 0.31, \gamma = 0.6081, \eta_1 = 0.0125, \eta_2 = 0.125, N = 1. \quad (5.1)$$

Hence, $R_0 = 0.5219 < 1$. Figure 1 plots $I(t)$ and $R(t)$ against t when we take $\tau = 0, 15, 30$. Both $I(t)$ and $R(t)$ approach 0, implying the global asymptotically stable of E_0 of model (2.2).

Regarding Theorem 4.2, we set

$$\beta = 0.5, \gamma = 0.3081, \eta_1 = 0.0525, \eta_2 = 0.125, N = 1. \quad (5.2)$$

Then $R_0 = 2.3044 > 1$ and $E^* = (I^*, R^*) = (0.2069, 0.3591)$. Furthermore, the coefficients in (4.9) could be calculated as $p = 0.0091 > 0$ and $q = 0.0013 > 0$. Theoretical results in Theorem 4.2

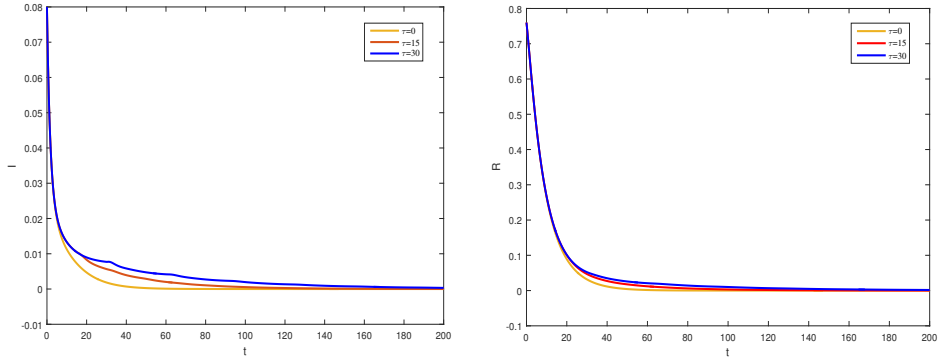


Figure 1: With parameters given in (5.1), three curves of $I(t)$ or $R(t)$ are plotted for $\tau = 0, 15$ and 30 , numerically verifying the results stated in Theorem 4.1.

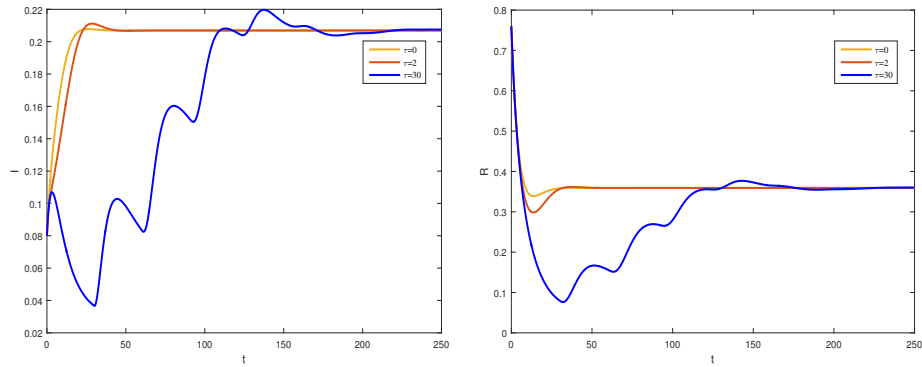


Figure 2: In the case of $\tau = 0, 2, 30$, when $R_0 = 2.643 > 1$, the endemic equilibrium E^* of model (2.2) is stable.

guarantee the local asymptotically stable of E^* . To verify, we in Figure 2 plot the dynamics of I and R by taking $\tau = 0, 2$ and 30 , and we find that $I(t) \rightarrow I^*$ and $R(t) \rightarrow R^*$ as $t \rightarrow \infty$, suggesting the local asymptotically stable of E^* .

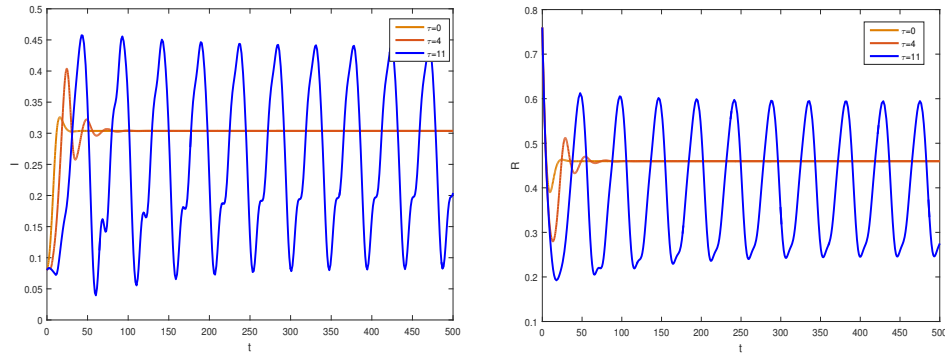


Figure 3: When $\tau = 0, 4, 11$, the stable endemic equilibrium E^* becomes unstable, and periodic solutions appear.

To observe the possible Hopf bifurcation stated in Theorem 4.3 and Theorem 4.4, we choose

$$\beta = 0.8, \gamma = 0.2081, \eta_1 = 0.0125, \eta_2 = 0.125. \quad (5.3)$$

Then $R_0 = 4.2287 > 1$, $E^* = (0.3038, 0.4597)$ and $q = -2.7 \times 10^{-3} < 0$. Figure 3 plots I and R with $\tau = 0, 4$, which numerically verifies the local asymptotically stable of E^* stated in Theorem 4.3. When Figure 3 increases τ to 11, ~~and~~ both I and R show a periodic pattern after a short oscillation. Biologically, our results show that the inclusion of relapse, coupled with substantial time delay, leads to recurrent infections among individuals who haven't yet fully recovered, resulting in oscillations in both the infected and recovered populations due to relapse.

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