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Research article

Dynamical analysis and Hopf bifurcation of an SEIR epidemic model with nonlinear incidence and time-delayed behavioral response

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Abstract: In this paper, an SEIR epidemic dynamical model incorporating a fear effect, a nonlinear incidence rate, and a time-delayed behavioral response was established. The basic reproduction number R_0 was derived using the next-generation matrix method. Theoretical analysis demonstrates that if $R_0 < 1$, the disease-free equilibrium is globally asymptotically stable; if $R_0 > 1$, there exists a unique endemic equilibrium, which is also globally asymptotically stable. By performing stability analysis, the critical time delay threshold τ_0 for triggering a Hopf bifurcation was derived. Moreover theoretical analysis and numerical simulations consistently showed that when the time delay exceeds this threshold, the endemic equilibrium loses its stability and a stable limit cycle is generated, leading to periodic epidemic outbreaks. Furthermore, sensitivity analysis revealed an inverse relationship between fear coefficient α and the critical time delay threshold τ_0.

Keywords: SEIR model; delay differential equation; fear effect; behavioral response; Hopf bifurcation

1. Introduction

Mathematical modeling of infectious diseases acts as a critical tool for elucidating the evolutionary dynamics of pathogens and for the formulation of effective prevention and control strategies. Since the seminal introduction of the classic SIR compartmental model by Kermack and McKendrick [1] in 1927, this field has undergone nearly a century of profound development. Following the establishment of the threshold theory for basic reproduction number R_0 by Hethcote [2], the research paradigm has progressively transitioned from fundamental compartmental frameworks to models incorporating more sophisticated and realistic biological characteristics.

As research has progressed, scholars have recognized that the traditional bilinear mass-action law frequently lacks the capacity to precisely replicate the complexities of real-world transmission processes. Consequently, research focus has shifted toward exploring the dynamical behaviors of SEIR models featuring nonlinear incidence rates and finite treatment capacities [3, 4]. Such nonlinear characteristics frequently lead to rich dynamical phenomena; for instance, Han et al. [5] established the global stability of diffusive SEIR models under nonlinear incidence rates, while Tipsri et al. [6] further elucidated the impact of time delays on the stability of nonlinear SEIR systems.

In exploring the interplay between human social behavior and epidemic transmission, Cui et al. [7] pioneered the use of an exponentially decreasing function μe^{-mI} to characterize the public alertness induced by media coverage. Their work demonstrated that media influence can effectively modulate contact rates and trigger Hopf bifurcations. Subsequently, Funk et al. [8] and Kiss et al. [9] further elucidated the pivotal role of information flow in mitigating epidemic peaks from the perspective of crisis awareness dissemination. Additionally, from the perspective of long-term control, the role of continuous vaccination and demographic turnover has been established as a critical factor in determining the threshold for disease eradication [10]. However, a critical limitation in many behavioral models, such as the seminal work by Cui et al. [7], is the assumption that public response to epidemic information is instantaneous. In reality, an inherent lag exists between the generation of infection data and the subsequent collective change in human behavior. For instance, while Song and Xiao [11] highlighted that delayed media effects can precipitate "multi-peak" phenomena, and others have explored time delays in viral evolution [12] and non-local transmission [13], a research gap remains. Specifically, most delayed models utilize monotonic incidence rates, failing to capture the non-monotonic dynamics that arise from intense fear. Despite the general delay analysis provided by Tipsri et al. [6], the intricate coupling between fear-driven exponential behavioral feedback and explicit decision delays has yet to be systematically characterized.

In this paper, we develop an SEIR model incorporating a non-monotonic contact rate function $\beta_0 e^{-\alpha I(t-\tau)}$ to account for behavioral changes during an epidemic. We derive an analytical expression for the critical time-delay threshold τ_0 and prove the transversality condition for a Hopf bifurcation. Sensitivity analysis reveals an inverse relationship between fear coefficient α and the delay threshold τ_0, indicating that a stronger behavioral response can reduce the system's tolerance for information lags. These results provide a mathematical basis for public health strategies, suggesting that enhancing awareness and accelerating information dissemination are necessary to maintain epidemic stability and prevent periodic fluctuations.

The rest of this paper is organized as follows. In Section 2, we formulate the SEIR epidemic model with time-delayed behavioral response and define the biological meanings of parameters. In Section 3, we investigate the basic properties of the model, including the positivity and boundedness of solutions, and derive the basic reproduction number R_0. In Section 4, the local and global stability of the disease-free equilibrium and the endemic equilibrium are analyzed. In Section 5, we focus on the Hopf bifurcation analysis, where the critical time delay threshold τ_0 is determined. Numerical simulations are performed in Section 6 to validate the theoretical findings. Finally, In Section 7, we conclude the paper.

2. Model formulation

In this section, an SEIR epidemic dynamical model incorporating a fear effect, a nonlinear incidence rate, and a time-delayed behavioral response is formulated. Considering the risk-adaptive behavior of humans in response to an epidemic, the total population $N(t)$ is partitioned into four disjoint compartments: susceptible (S), exposed (E), infected (I), and recovered (R). At any time t, the total population is defined as $N(t) = S(t) + E(t) + I(t) + R(t)$.

Based on the dynamical mechanism of infectious disease transmission, we establish the following SEIR model with a nonlinear incidence rate and time-delayed behavioral response:

$$\begin{cases} \frac{dS}{dt} = \Lambda - \beta(I(t-\tau))S(t)I(t) - \mu S(t), \\ \frac{dE}{dt} = \beta(I(t-\tau))S(t)I(t) - (\sigma+\mu)E(t), \\ \frac{dI}{dt} = \sigma E(t) - (\gamma+\mu)I(t), \\ \frac{dR}{dt} = \gamma I(t) - \mu R(t), \end{cases} \tag{2.1}$$

where $\beta(I(t-\tau))$ represents the prevalence-dependent contact rate, reflecting the influence of delayed information feedback. To quantitatively characterize the behavioral response mechanism triggered by the fear effect, we adopt the following exponentially decreasing function form

$$\beta(I) = \beta_0 \exp(-\alpha I), \quad \alpha \geq 0, \beta_0 > 0,$$

and this choice characterizes a non-monotonic incidence rate, accounting for the scenario where intense public fear significantly suppresses transmission during severe outbreaks.

All parameters in the model are assumed to be positive constants, and their biological meanings are summarized in Table 1.

Table 1. Biological meanings of the parameters in model (2.1).

Parameter	Biological Meaning
Λ	Constant recruitment rate of the population
β_0	Baseline contact rate without behavioral changes
α	Intensity of behavioral response
μ	Natural death rate of the population
σ	Rate of transition from exposed (E) to infected (I)
γ	Recovery rate of infected individuals
τ	Time delay in behavioral response

To analyze the dynamical properties of the system, we make the following hypotheses based on the biological background:

(H1): Each compartment has the same constant birth and natural death rate μ, and the system is at a total population equilibrium.

(H2): Infected individuals gain permanent immunity after recovery and do not return to the susceptible compartment.

(H3): The contact rate β is influenced by the historical scale of infection, with a fixed time delay $\tau \geq 0$ between information generation and the implementation of protective behaviors.

Since the evolution of the recovered compartment R in system (2.1) does not appear in the first three equations, and R is decoupled from the dynamics of S, E, and I, we focus our analysis on the following reduced subsystem (2.2):

$$\begin{cases} \frac{dS}{dt} = \Lambda - \beta(I(t-\tau))S(t)I(t) - \mu S(t), \\ \frac{dE}{dt} = \beta(I(t-\tau))S(t)I(t) - (\sigma+\mu)E(t), \\ \frac{dI}{dt} = \sigma E(t) - (\gamma+\mu)I(t), \end{cases} \tag{2.2}$$

for simplicity, we denote $I_\tau = I(t-\tau)$ in the following discussion when no confusion arises.

Considering the time-delay characteristics of the system, we define its state space as the Banach space of continuous functions $C = C([-\tau, 0], \mathbb{R}^3)$. The supremum norm on C is defined as

$$\|\phi\| = \sup_{-\tau \leq \theta \leq 0} |\phi(\theta)|, \quad \forall \phi \in C.$$

Let $C_+ = C([-\tau, 0], \mathbb{R}^3_+)$ be the positive cone of C. In view of the biological significance of the epidemic model, the initial conditions for system (2.2) are defined as

$$S(\theta) = \phi_1(\theta), \quad E(\theta) = \phi_2(\theta), \quad I(\theta) = \phi_3(\theta), \quad \forall \theta \in [-\tau, 0],$$

where the initial function $\phi(\theta) = (\phi_1(\theta), \phi_2(\theta), \phi_3(\theta))^T \in C_+$ satisfies $\phi_i(\theta) \geq 0$ $(i = 1, 2, 3)$ and $\phi_i(0) > 0$.

3. Basic properties of the model

In this section, we prove the existence, uniqueness, non-negativity, and boundedness of the solutions for system (2.2) and derive the expression for the basic reproduction number R_0.

Theorem 3.1. *For any initial condition $\phi \in C_+$, system* (2.2) *has a unique solution $z(t) = (S(t), E(t), I(t))^T$ on $t \in [0, +\infty)$. Furthermore, this solution remains in $\mathbb{R}^3_+$ and is uniformly bounded. Specifically, the set C_+ is a positively invariant set for system* (2.2).

Proof. System (2.2) can be regarded as a functional differential equation on the Banach space $C = C([-\tau, 0], \mathbb{R}^3)$. For any $t \geq 0$, let

$$x(t) = (S(t), E(t), I(t))^T$$

and define the mapping $f : C_+ \to \mathbb{R}^3$ as follows:

$$f(\phi) = \begin{pmatrix} f_1(\phi) \\ f_2(\phi) \\ f_3(\phi) \end{pmatrix} = \begin{pmatrix} \Lambda - \beta_0 e^{-\alpha\phi_3(-\tau)}\phi_1(0)\phi_3(0) - \mu\phi_1(0) \\ \beta_0 e^{-\alpha\phi_3(-\tau)}\phi_1(0)\phi_3(0) - (\sigma+\mu)\phi_2(0) \\ \sigma\phi_2(0) - (\gamma+\mu)\phi_3(0) \end{pmatrix},$$

where $\phi = (\phi_1, \phi_2, \phi_3)^T \in C_+$. Based on the fundamental theory of delay differential equations [14], if the operator $f(\phi)$ satisfies the local Lipschitz condition on any bounded open set in C, then there exists a unique local solution for the given initial condition.

For the nonlinear term $g(\phi) = e^{-\alpha\phi_3(-\tau)}\phi_1(0)\phi_3(0)$ in f, its partial derivatives with respect to each component are

$$\begin{aligned}\frac{\partial g}{\partial \phi_1(0)} &= e^{-\alpha\phi_3(-\tau)}\phi_3(0),\\ \frac{\partial g}{\partial \phi_3(0)} &= e^{-\alpha\phi_3(-\tau)}\phi_1(0),\\ \frac{\partial g}{\partial \phi_3(-\tau)} &= -\alpha e^{-\alpha\phi_3(-\tau)}\phi_1(0)\phi_3(0).\end{aligned}$$

Since exponential and linear functions, as well as their products, are continuous and bounded on any bounded region, for any bounded subset $U \subset C$, there exists a constant $L_U > 0$ such that

$$\|f(\phi) - f(\psi)\| \le L_U\|\phi - \psi\|_C, \quad \forall \phi, \psi \in U,$$

where $\|\cdot\|_C$ is the supremum norm on C. Thus, f is locally Lipschitz.

Since system (2.2) is a functional differential equation with the quasi-monotone property on C_+, according to Theorem 3.4 in Smith [15], the non-negativity of the solution starting from C_+ is inherently guaranteed for all $t \ge 0$. This implies that C_+ is a positively invariant set for system (2.2).

The initial function $\phi = (\phi_1, \phi_2, \phi_3)^T$ belongs to the Banach space $C = C([-\tau, 0], \mathbb{R}^3)$. Since ϕ is continuous on the compact set $[-\tau, 0]$, $W(0) = \phi_1(0) + \phi_2(0) + \phi_3(0)$ is a finite positive constant. Define $W(t) = S(t) + E(t) + I(t)$. Summing the three equations of system (2.2) yields

$$\begin{aligned}\frac{dW(t)}{dt} &= \Lambda - \mu S(t) - \mu E(t) - (\gamma + \mu)I(t)\\ &= \Lambda - \mu W(t) - \gamma I(t).\end{aligned}$$

Since $I(t) \ge 0$, we obtain the following differential inequality

$$\frac{dW(t)}{dt} \le \Lambda - \mu W(t).$$

Solving this linear inequality gives

$$W(t) \le W(0)e^{-\mu t} + \frac{\Lambda}{\mu}(1 - e^{-\mu t}) = \frac{\Lambda}{\mu} + (W(0) - \frac{\Lambda}{\mu})e^{-\mu t}.$$

It follows that $W(t) \le \max\{W(0), \Lambda/\mu\}$. According to the comparison principle, we have

$$\limsup_{t\to+\infty} W(t) \le \frac{\Lambda}{\mu}.$$

This indicates that all solutions eventually enter and remain in the region

$$\Omega = \left\{(S, E, I) \in \mathbb{R}^3_+ \mid S + E + I \le \frac{\Lambda}{\mu} + \epsilon\right\}, \quad (\forall \epsilon > 0).$$

Particularly, if $W(0) \le \Lambda/\mu$, then $W(t) \le \Lambda/\mu$ for all $t > 0$. Thus, the region $\Omega = \{(S, E, I) \in \mathbb{R}^3_+ \mid S + E + I \le \Lambda/\mu\}$ is a positively invariant attracting set for the system. This completes the proof.

Acting as a vital threshold parameter for disease persistence, R_0 quantifies the expected secondary cases originating from a representative infected individual over its complete duration of infectivity within a naive population. Following the next-generation matrix method proposed by van den Driessche and Watmough [16], the basic reproduction number for system (2.2) is derived as follows.

Theorem 3.2. *The basic reproduction number of system* (2.2) *is given by*

$$R_0 = \rho(FV^{-1}) = \frac{\beta_0 \Lambda \sigma}{\mu(\sigma+\mu)(\gamma+\mu)}. \tag{3.1}$$

Proof. System (2.2) always possesses a disease-free equilibrium $E_0 = (\Lambda/\mu, 0, 0)$. Let $x = (E, I)^T$ represent the infected compartments. The subsystem for the infected individuals can be written as:

$$\frac{dx}{dt} = \mathcal{F}(x) - \mathcal{V}(x),$$

where the new infection term $\mathcal{F}$ and the transition term $\mathcal{V}$ are given by

$$\mathcal{F} = \begin{pmatrix} \beta(I_\tau) S I \\ 0 \end{pmatrix}, \quad \mathcal{V} = \begin{pmatrix} (\sigma+\mu)E \\ -\sigma E + (\gamma+\mu)I \end{pmatrix}.$$

The Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ at the disease-free equilibrium E_0 are

$$F = \frac{\partial \mathcal{F}}{\partial x}\Big|_{E_0} = \begin{pmatrix} 0 & \beta_0 \frac{\Lambda}{\mu} \\ 0 & 0 \end{pmatrix}, \quad V = \frac{\partial \mathcal{V}}{\partial x}\Big|_{E_0} = \begin{pmatrix} \sigma+\mu & 0 \\ -\sigma & \gamma+\mu \end{pmatrix}.$$

The inverse of matrix V is

$$V^{-1} = \frac{1}{(\sigma+\mu)(\gamma+\mu)} \begin{pmatrix} \gamma+\mu & 0 \\ \sigma & \sigma+\mu \end{pmatrix}.$$

Thus, the next-generation matrix $K = FV^{-1}$ is obtained as

$$K = \begin{pmatrix} \frac{\beta_0 \Lambda \sigma}{\mu(\sigma+\mu)(\gamma+\mu)} & \frac{\beta_0 \Lambda}{\mu(\gamma+\mu)} \\ 0 & 0 \end{pmatrix}.$$

The basic reproduction number R_0 is the spectral radius of matrix K, which leads to

$$R_0 = \rho(K) = \frac{\beta_0 \Lambda \sigma}{\mu(\sigma+\mu)(\gamma+\mu)}.$$

This completes the proof.

Theorem 3.3. *When $R_0 > 1$, the system* (2.2) *has a unique endemic equilibrium $E^* = (S^*, E^*, I^*)$.*

Proof. The equilibrium points of system (2.2) satisfy the following equations:

$$\begin{cases} \Lambda - \beta(I^*) S^* I^* - \mu S^* = 0, \\ \beta(I^*) S^* I^* - (\sigma+\mu) E^* = 0, \\ \sigma E^* - (\gamma+\mu) I^* = 0. \end{cases} \tag{3.2}$$

From the last two equations of (3.2), we can express E^* and S^* in terms of I^*

$$E^* = \frac{\gamma+\mu}{\sigma}I^*, \quad S^* = \frac{(\sigma+\mu)(\gamma+\mu)}{\sigma\beta(I^*)}.$$

Substituting these into the first equation of (3.2), we obtain

$$\Lambda - (\sigma+\mu)\frac{\gamma+\mu}{\sigma}I^* - \mu\frac{(\sigma+\mu)(\gamma+\mu)}{\sigma\beta_0 e^{-\alpha I^*}} = 0.$$

Rearranging the terms, we get an equation for I^*

$$\Lambda = \frac{\mu(\sigma+\mu)(\gamma+\mu)}{\sigma\beta_0}e^{\alpha I^*} + \frac{(\sigma+\mu)(\gamma+\mu)}{\sigma}I^*.$$

To prove the existence and uniqueness of a positive root I^*, we consider the biologically feasible range $I \in [0, \Lambda/\mu]$ and define the following auxiliary function:

$$H(I) = \frac{\mu(\sigma+\mu)(\gamma+\mu)}{\sigma\beta_0}e^{\alpha I} + \frac{(\sigma+\mu)(\gamma+\mu)}{\sigma}I - \Lambda, \quad I \in [0, \Lambda/\mu].$$

It is clear that $H(I)$ is a continuous function of I. Calculating $H(0)$, we have

$$\begin{aligned} H(0) &= \frac{\mu(\sigma+\mu)(\gamma+\mu)}{\sigma\beta_0} - \Lambda \\ &= \frac{\Lambda}{R_0} - \Lambda = \Lambda\left(\frac{1}{R_0} - 1\right). \end{aligned}$$

Since $R_0 > 1$, it follows that $H(0) < 0$. Furthermore, it can be verified that for $I = \Lambda/\mu$, $H(\Lambda/\mu) > 0$ holds. According to the Intermediate Value Theorem, there exists at least one positive root $I^* \in (0, \Lambda/\mu)$ such that $H(I^*) = 0$.

Next, we check the derivative of $H(I)$:

$$H'(I) = \frac{\alpha\mu(\sigma+\mu)(\gamma+\mu)}{\sigma\beta_0}e^{\alpha I} + \frac{(\sigma+\mu)(\gamma+\mu)}{\sigma}.$$

Since all parameters are positive, $H'(I) > 0$ for all $I \in [0, \Lambda/\mu]$, which means $H(I)$ is strictly monotonically increasing. Therefore, the positive endemic equilibrium $E^* = (S^*, E^*, I^*)$ is unique. This completes the proof.

4. Stability analysis

In this section, we analyze the local and global stability of the disease-free equilibrium E_0 and the endemic equilibrium E^*.

Theorem 4.1. *Regarding the stability of the disease-free equilibrium E_0:*

(a) *If $R_0 < 1$, E_0 is locally asymptotically stable;*
(b) *If $R_0 > 1$, E_0 is unstable.*

Proof. The Jacobian matrix of system (2.2) at the disease-free equilibrium $E_0 = (\Lambda/\mu, 0, 0)$ is given by

$$J(E_0) = \begin{pmatrix} -\mu & 0 & -\beta_0 \frac{\Lambda}{\mu} \\ 0 & -(\sigma+\mu) & \beta_0 \frac{\Lambda}{\mu} \\ 0 & \sigma & -(\gamma+\mu) \end{pmatrix}.$$

The corresponding characteristic equation is $|\lambda I - J(E_0)| = 0$, which yields

$$(\lambda+\mu)\left[(\lambda+\sigma+\mu)(\lambda+\gamma+\mu) - \sigma\beta_0\frac{\Lambda}{\mu}\right] = 0.$$

Thus, the system has one negative eigenvalue $\lambda_1 = -\mu < 0$. The other two eigenvalues are determined by the following quadratic equation

$$\lambda^2 + (\sigma+\gamma+2\mu)\lambda + (\sigma+\mu)(\gamma+\mu) - \frac{\sigma\beta_0\Lambda}{\mu} = 0. \tag{4.1}$$

Let this equation be $\lambda^2 + a_1\lambda + a_0 = 0$. Due to the positivity of all biological parameters (σ, γ, μ), it is evident that $a_1 = \sigma+\gamma+2\mu > 0$ holds inherently. The sign of constant term a_0 remains to be examined:

$$a_0 = (\sigma+\mu)(\gamma+\mu) - \frac{\sigma\beta_0\Lambda}{\mu}.$$

Based on the stability conditions of the Routh-Hurwitz theorem [17], the eigenvalues of the quadratic characteristic equation possess strictly negative real components provided that the coefficients satisfy $a_1 > 0$ and $a_0 > 0$. We need to examine only the sign of a_0:

$$\begin{aligned} a_0 > 0 &\iff (\sigma+\mu)(\gamma+\mu) > \frac{\sigma\beta_0\Lambda}{\mu} \\ &\iff 1 > \frac{\sigma\beta_0\Lambda}{\mu(\sigma+\mu)(\gamma+\mu)} = R_0 \\ &\iff R_0 < 1. \end{aligned}$$

Therefore, if $R_0 < 1$, all eigenvalues have negative real parts, and E_0 is locally asymptotically stable. Conversely, if $R_0 > 1$, then $a_0 < 0$, implying at least one positive real root exists; thus, E_0 is unstable.

Theorem 4.2. *If $R_0 \leq 1$, the disease-free equilibrium $E_0 = (\Lambda/\mu, 0, 0)$ of system (2.2) is globally asymptotically stable in Ω.*

Proof. Since the recovery compartment R does not affect the evolution of other variables and its own stability is guaranteed by $I(t)$, it is sufficient to construct a Lyapunov function in the three-dimensional state space of the sub-system (2.2). We construct the following Lyapunov function:

$$L(t) = \sigma E(t) + (\sigma+\mu)I(t).$$

Thus, $L(E, I)$ is continuously differentiable in Ω. $L = 0$ if and only if $E = I = 0$, and $L > 0$ for other points in Ω. Thus, L is a positive definite Lyapunov function with respect to E_0.

The derivative of $L(t)$ along the solutions of system (2.2) is

$$\dot{L}(t) = \sigma\frac{dE}{dt} + (\sigma+\mu)\frac{dI}{dt}.$$

Substituting the equations for dE/dt and dI/dt from (2.2) gives

$$\dot{L}(t) = \sigma[\beta_0 e^{-\alpha(I_\tau)}S(t)I(t) - (\sigma+\mu)E(t)] + (\sigma+\mu)[\sigma E(t) - (\gamma+\mu)I(t)].$$

Simplifying the expression gives

$$\dot{L}(t) = \sigma\beta_0 e^{-\alpha(I_\tau)}S(t)I(t) - (\sigma+\mu)(\gamma+\mu)I(t).$$

In the invariant set Ω, $I \geq 0$ and $\alpha \geq 0$, so $e^{-\alpha(I_\tau)} \leq 1$. According to the definition of the invariant set Ω, it holds that $S(t) \leq S(t) + E(t) + I(t) \leq \Lambda/\mu$ for any $(S, E, I) \in \Omega$. Thus,

$$\begin{aligned}\dot{L}(t) &\leq \sigma\beta_0\frac{\Lambda}{\mu}I(t) - (\sigma+\mu)(\gamma+\mu)I(t)\\ &= (\sigma+\mu)(\gamma+\mu)\left[\frac{\sigma\beta_0\Lambda}{\mu(\sigma+\mu)(\gamma+\mu)} - 1\right]I(t)\\ &= (\sigma+\mu)(\gamma+\mu)[R_0 - 1]I(t).\end{aligned}$$

If $R_0 \leq 1$, then $\dot{L}(t) \leq 0$. Let $M = \{(S, E, I) \in \Omega \mid \dot{L}(t) = 0\}$. From the expression of $\dot{L}(t)$, it is clear that $\dot{L}(t) = 0$ implies $I(t) = 0$. Substituting $I(t) = 0$ into the third equation of system (2.2), we obtain $\sigma E(t) = 0$, which implies $E(t) = 0$. Consequently, the dynamics of system (2.2) on the set M reduces to the following ordinary differential equation:

$$\frac{dS(t)}{dt} = \Lambda - \mu S(t).$$

This linear equation has a general solution $S(t) = \frac{\Lambda}{\mu} + (S(0) - \frac{\Lambda}{\mu})e^{-\mu t}$, which implies that $S(t) \to \Lambda/\mu$ as $t \to \infty$.

Therefore, any solution $(S(t), E(t), I(t))$ starting in M remains in M if and only if $E(t) = 0$ and $I(t) = 0$, and all such solutions converge to the point $E_0 = (\Lambda/\mu, 0, 0)$. According to LaSalle's Invariance Principle [18], the disease-free equilibrium E_0 is globally asymptotically stable in Ω when $R_0 \leq 1$.

Theorem 4.3. *Assume $R_0 > 1$. If the intensity of behavioral response satisfies the condition $\alpha \leq 1/I^*$, then for $\tau = 0$, the unique endemic equilibrium $E^* = (S^*, E^*, I^*)$ of system (2.2) is globally asymptotically stable in Ω.*

Proof. When $\tau = 0$, system (2.2) reduces to a system of ordinary differential equations (ODEs), where $I(t-\tau) = I(t)$. For simplicity, let $\beta(I) = \beta_0 e^{-\alpha I}$. We construct a Lyapunov function of the following form

$$L(t) = \left(S - S^* - S^*\ln\frac{S}{S^*}\right) + \left(E - E^* - E^*\ln\frac{E}{E^*}\right) + \frac{\sigma+\mu}{\sigma}\left(I - I^* - I^*\ln\frac{I}{I^*}\right).$$

Thus, $L(S, E, I)$ is continuously differentiable on the interior of Ω. The unique minimum of L is reached at E^*, where $L(S^*, E^*, I^*) = 0$. For any other point in $\Omega \setminus \{E^*\}$, $L > 0$. Thus, L is a positive definite Lyapunov function with respect to E^*.

The derivative of $L(t)$ along the solutions of the system when $\tau = 0$ is

$$\dot{L}(t) = \left(1 - \frac{S^*}{S}\right)\dot{S} + \left(1 - \frac{E^*}{E}\right)\dot{E} + \frac{\sigma + \mu}{\sigma}\left(1 - \frac{I^*}{I}\right)\dot{I}.$$

Substituting the equations of system (2.2) and the equilibrium relations

$$\Lambda = \beta(I^*)S^*I^* + \mu S^*, \quad \sigma + \mu = \frac{\beta(I^*)S^*I^*}{E^*}, \quad \gamma + \mu = \frac{\sigma E^*}{I^*},$$

we obtain

$$\dot{L} = \mu S^*\left(2 - \frac{S}{S^*} - \frac{S^*}{S}\right) + \beta(I^*)S^*I^*\left[3 - \frac{S^*}{S} - \frac{\beta(I)SIE^*}{\beta(I^*)S^*I^*E} - \frac{I^*E}{IE^*} + \left(\frac{\beta(I)I}{\beta(I^*)I^*} - \frac{I}{I^*}\right)\right].$$

To facilitate sign analysis, we introduce the auxiliary function $g(x) = x - 1 - \ln x \geq 0$ for all $x > 0$. By adding and subtracting logarithmic terms, we can rewrite $\dot{L}$ as

$$\begin{aligned}\dot{L} = &-\mu\frac{(S - S^*)^2}{S} - \beta(I^*)S^*I^*\left[g\left(\frac{S^*}{S}\right) + g\left(\frac{\beta(I)SIE^*}{\beta(I^*)S^*I^*E}\right) + g\left(\frac{I^*E}{IE^*}\right)\right] \\ &+ \beta(I^*)S^*I^* \cdot W(I),\end{aligned} \tag{4.2}$$

where the term $W(I)$ is defined as

$$W(I) = \frac{\beta(I)I}{\beta(I^*)I^*} - \frac{I}{I^*} + \ln\left(\frac{I}{I^*}\right) - \ln\left(\frac{\beta(I)I}{\beta(I^*)I^*}\right). \tag{4.3}$$

Substituting $\beta(I) = \beta_0 e^{-\alpha I}$ and letting $f(I) = \beta(I)I = \beta_0 I e^{-\alpha I}$, we have

$$W(I) = \frac{f(I)}{f(I^*)} - \frac{I}{I^*} + \ln\left(\frac{I}{I^*}\right) - \ln\left(\frac{f(I)}{f(I^*)}\right). \tag{4.4}$$

It is easy to verify that $W(I^*) = 0$. Taking the first derivative of $W(I)$ with respect to I, we get

$$W'(I) = \frac{f'(I)}{f(I^*)} - \frac{1}{I^*} + \frac{1}{I} - \frac{f'(I)}{f(I)}, \tag{4.5}$$

where $f'(I) = \beta_0 e^{-\alpha I}(1 - \alpha I)$. Since $\frac{f'(I)}{f(I)} = \frac{1}{I} - \alpha$, the expression for $W'(I)$ can be simplified as

$$W'(I) = \frac{f'(I)}{f(I^*)} - \left(\frac{1}{I^*} - \alpha\right) = \frac{f'(I) - f'(I^*)}{f(I^*)}. \tag{4.6}$$

To establish that I^* is the global maximum point, we examine the monotonicity of $f'(I)$. First, we consider $I \in (0, 1/\alpha]$. In this interval, $f''(I) = \beta_0\alpha e^{-\alpha I}(\alpha I - 2) < 0$, which means $f'(I)$ is strictly decreasing. Since $I^* \leq 1/\alpha$, for any $0 < I < I^*$, we have $f'(I) > f'(I^*)$, implying $W'(I) > 0$. For $I^* < I \leq 1/\alpha$, we have $f'(I) < f'(I^*)$, implying $W'(I) < 0$.

Second, we consider $I > 1/\alpha$. In this range, the term $(1 - \alpha I)$ is negative, so $f'(I) < 0$. However, since $I^* \leq 1/\alpha$, we have $f'(I^*) \geq 0$. This ensures that $f'(I) < f'(I^*)$ remains true for all $I > 1/\alpha$, which means $W'(I) < 0$ throughout this region. Therefore, $W(I)$ increases on $(0, I^*)$ and decreases on $(I^*, \Lambda/\mu]$, reaching its unique global maximum at $I = I^*$. Since $W(I^*) = 0$, it follows that $W(I) \leq 0$ for all $I > 0$.

Combining this with the fact that $g(x) \geq 0$, we conclude that $\dot{L} \leq 0$ holds within the invariant set Ω. The equality $\dot{L} = 0$ occurs if and only if $(S, E, I) = (S^*, E^*, I^*)$. According to LaSalle's Invariance Principle [18], the endemic equilibrium E^* is globally asymptotically stable in Ω when $R_0 > 1$ and $\tau = 0$.

5. Hopf bifurcation analysis

In this section, we choose the behavioral response time delay τ as the bifurcation parameter to study the stability of the endemic equilibrium E^* and the existence of a Hopf bifurcation when $\tau > 0$.

Theorem 5.1. *Suppose $R_0 > 1$. The stability of system (2.2) at E^* satisfies the following:*

(a) *There exists a critical time delay threshold $\tau_0 > 0$. When $\tau \in [0, \tau_0)$, E^* is locally asymptotically stable.*
(b) *When $\tau = \tau_0$, the system (2.2) undergoes a Hopf bifurcation at E^*, meaning the equilibrium loses stability and a periodic solution is generated.*
(c) *When $\tau > \tau_0$, E^* is unstable, and a stable limit cycle exists near E^*.*

Proof. Let $x(t) = S(t) - S^*$, $y(t) = E(t) - E^*$, and $z(t) = I(t) - I^*$. The nonlinear incidence function is $F(S, I, I_\tau) = \beta(I_\tau)SI$. Its Taylor expansion at E^* is

$$F \approx F^* + \frac{\partial F}{\partial S}\Big|_{E^*}(S - S^*) + \frac{\partial F}{\partial I}\Big|_{E^*}(I - I^*) + \frac{\partial F}{\partial I_\tau}\Big|_{E^*}(I_\tau - I^*),$$

where the partial derivatives are

$$p = \frac{\partial F}{\partial S}\Big|_{E^*} = \beta(I^*)I^*, \quad q = \frac{\partial F}{\partial I}\Big|_{E^*} = \beta(I^*)S^*, \quad k = -\frac{\partial F}{\partial I_\tau}\Big|_{E^*} = \alpha\beta(I^*)S^*I^* > 0.$$

The linearized system of (2.2) at E^* is

$$\frac{dX}{dt} = A_0X(t) + A_1X(t - \tau),$$

where $X(t) = (x(t), y(t), z(t))^T$, and the matrices A_0 and A_1 are

$$A_0 = \begin{pmatrix} -(p+\mu) & 0 & -q \\ p & -(\sigma+\mu) & q \\ 0 & \sigma & -(\gamma+\mu) \end{pmatrix}, \quad A_1 = \begin{pmatrix} 0 & 0 & k \\ 0 & 0 & -k \\ 0 & 0 & 0 \end{pmatrix}.$$

The characteristic equation is $\det(\lambda I - A_0 - A_1e^{-\lambda\tau}) = 0$, which simplifies to

$$P(\lambda) + Q(\lambda)e^{-\lambda\tau} = 0, \tag{5.1}$$

where

$$P(\lambda) = \lambda^3 + b_2\lambda^2 + b_1\lambda + b_0,$$

and

$$Q(\lambda) = \sigma k(\lambda + \mu).$$

Here,

$$b_2 = p + \sigma + \gamma + 3\mu,$$

$$b_1 = (p + \mu)(\sigma + \gamma + 2\mu),$$

and

$$b_0 = p(\sigma + \mu)(\gamma + \mu).$$

We search for purely imaginary roots $\lambda = \pm i\omega$ ($\omega > 0$). Substituting $\lambda = i\omega$ into (5.1) and separating real and imaginary parts gives

$$\begin{cases} P_R(\omega) + Q_R(\omega)\cos(\omega\tau) + Q_I(\omega)\sin(\omega\tau) = 0, \\ P_I(\omega) + Q_I(\omega)\cos(\omega\tau) - Q_R(\omega)\sin(\omega\tau) = 0. \end{cases}$$

Squaring and adding these equations to eliminate τ, we obtain the equation for ω:

$$|P(i\omega)|^2 = |Q(i\omega)|^2.$$

Let $u = \omega^2$. This yields a cubic equation $G(u) = 0$, where

$$G(u) = u^3 + (b_2^2 - 2b_1)u^2 + (b_1^2 - 2b_0b_2 - \sigma^2k^2)u + (b_0^2 - \sigma^2k^2\mu^2) = 0. \tag{5.2}$$

If $b_0^2 - \sigma^2k^2\mu^2 < 0$, then $G(u) = 0$ has at least one positive real root u_0. The threshold frequency is denoted by $\omega_0 = \sqrt{u_0}$. The corresponding time delay threshold τ_k is

$$\tau_k = \frac{1}{\omega_0}\left[\pi - \arg\left(\frac{P(i\omega_0)}{Q(i\omega_0)}\right) + 2k\pi\right], \quad k = 0, 1, 2, \ldots$$

The Hopf bifurcation threshold [16] is the smallest positive value $\tau_0 = \min\{\tau_k\}$. Finally, we verify the transversality condition. By differentiating (5.1) with respect to τ, we find

$$Re\left(\frac{d\lambda}{d\tau}\right)^{-1}\Big|_{\tau=\tau_0} = \frac{G'(u_0)}{|Q(i\omega_0)|^2} > 0.$$

Since $G'(u_0) > 0$, the eigenvalues cross the imaginary axis from left to right as τ increases, inducing a Hopf bifurcation.

Remark 5.2. Regarding the conditions in Theorem 5.1, we provide two clarifications:

(i) The condition $b_0^2 - \sigma^2k^2\mu^2 < 0$ ensures the existence of the critical frequency ω_0. If $b_0^2 - \sigma^2k^2\mu^2 \geq 0$, then according to Descartes' Rule of Signs, the cubic equation $G(u) = 0$ has no positive real roots. In this case, the characteristic equation possesses no purely imaginary roots for any $\tau > 0$, meaning the endemic equilibrium E^* remains locally asymptotically stable for all time delays, and no Hopf bifurcation occurs.

(ii) Regarding the potential for stability switches, as the transversality condition $Re(d\lambda/d\tau)|_{\tau=\tau_0} > 0$ holds, the eigenvalues always cross the imaginary axis from left to right as τ increases. According to the general theory of functional differential equations [14], once the stability of E^* is lost at the first threshold τ_0, it cannot be regained as τ increases further. Therefore, no stability switches occur in this model, and E^* remains unstable for all $\tau > \tau_0$.

6. Numerical simulation

In this section, to explore the impact of time-delayed behavioral responses on epidemic evolution, the numerical simulations and Hopf bifurcation are performed.

Considering that the model primarily explores respiratory infectious diseases with information delay, the time unit is set to "days". To highlight the dynamical instability caused by time delay, a set of representative parameter values is tabulated in Table 2.

Table 2. Parameter values used in the numerical simulation.

Parameter	Physical/Biological Meaning	Value	Unit	Source/Remark
Λ	Constant recruitment rate of population	0.5	person·day^{-1}	[2] Standardized
μ	Natural death rate	0.04	day^{-1}	[2] Standardized
σ	Transformation rate from E to I	0.5	day^{-1}	Latent period: 2 days
γ	Recovery rate of infected individuals	0.3	day^{-1}	Infectious period: 3.3 days
β_0	Baseline transmission rate	0.2	person·day^{-1}	Medium intensity
α	Fear coefficient	12.0	I^{-1}	Assumed for sensitivity
τ	Behavioral response time delay	Variable	day	Bifurcation parameter

Based on Theorem 3.2 and the parameters in Table 2, the basic reproduction number is calculated as

$$R_0 = \frac{\beta_0 \Lambda \sigma}{\mu(\sigma + \mu)(\gamma + \mu)} \approx 6.81 > 1.$$

This represents a scenario of medium-intensity epidemic transmission. According to Theorem 3.3, there exists a unique endemic equilibrium $E^* \approx (11.1, 0.10, 0.15)$.

By solving the characteristic equation $G(u) = 0$ using MATLAB, we find the unique positive root $u_0 \approx 0.0213$, which leads to the critical frequency $\omega_0 \approx 0.4615$. Substituting ω_0 into the time delay formula gives the critical threshold:

$$\tau_0 \approx 12.75 \text{ days}.$$

Furthermore, the transversality condition $Re(d\lambda/d\tau)^{-1}|_{\tau=\tau_0} \approx 0.13 > 0$ is satisfied.

To observe the dynamical behavior near the threshold τ_0, we choose different values of τ:

(i) When $\tau = 3 < \tau_0$, Figure 1 shows that the number of infected individuals initially oscillates and then converges to the stable endemic equilibrium E^*. Figure 2 depicts the corresponding spiral convergence in three-dimensional phase space.

(ii) When $\tau = 20 > \tau_0$, Figure 1 shows persistent, equal-amplitude periodic oscillations. Figure 2 confirms that the equilibrium E^* has lost its stability and the trajectory is attracted to a stable limit cycle.

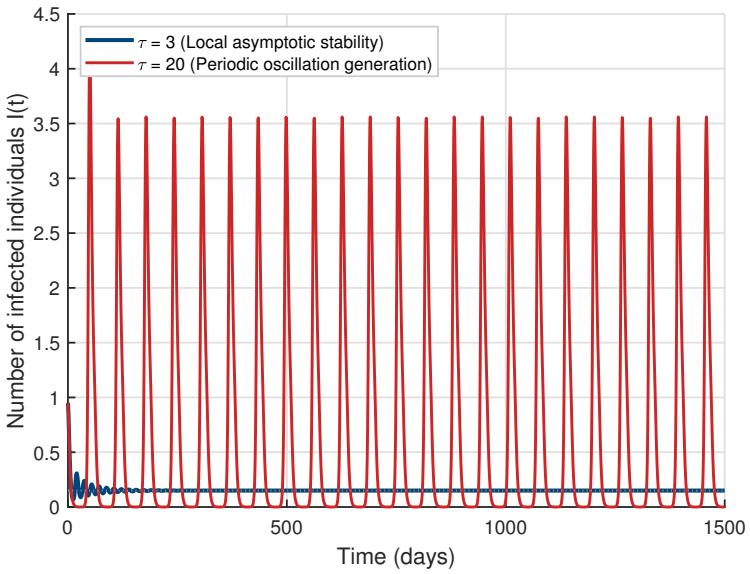

Figure 1. Epidemic evolution comparison with time delays of diverse behavioral responses.

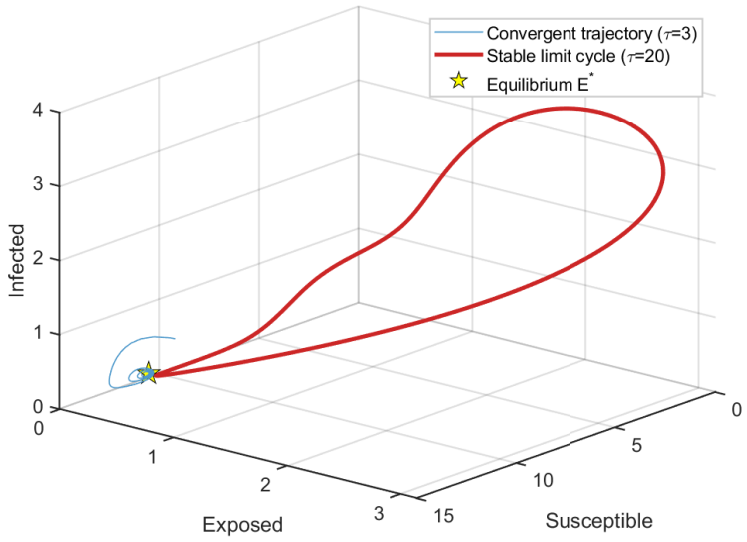

Figure 2. Comparison of three-dimensional phase space trajectories.

Last, Figure 3 exhibits the sensitivity of critical delay τ_0 to fear coefficient α. This shows that τ_0 is a monotonically decreasing function of α, implying that in populations with higher sensitivity to epidemic information, the system's tolerance for information delay decreases, making the epidemic more prone to oscillatory outbreaks.

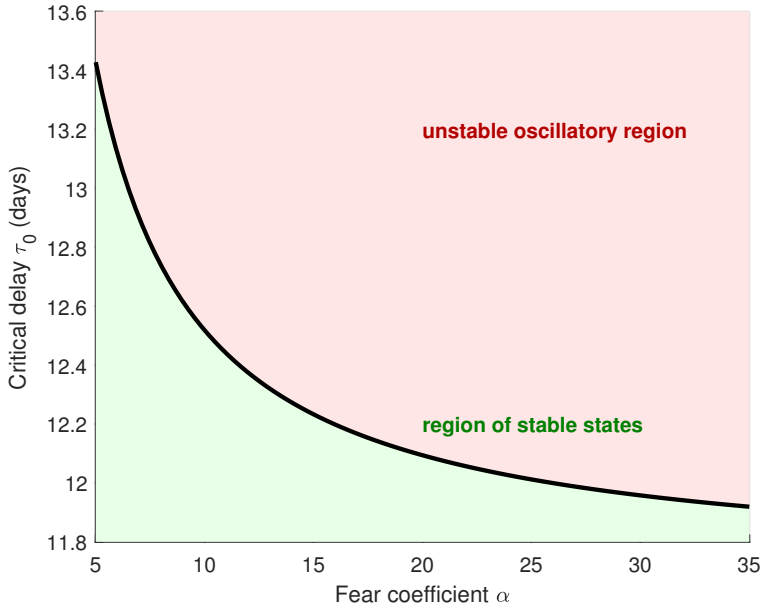

Figure 3. Influence of fear coefficient on system stability threshold.

7. Conclusions

In this paper, we establish an SEIR model incorporating fear effects and time-delayed behavioral responses. Our theoretical and numerical analyses reveal that while fear effects can reduce the peak of an epidemic, the inherent delay in information feedback and collective decision-making can trigger periodic rebounds. To ensure a stable and controlled epidemic situation, public health departments should not only augment public awareness but also expedite information dissemination to minimize decision-making lags.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

The authors declare there are no conflicts of interest.

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