



Research article

Dynamical analysis and bifurcation study of an epidemic model with media coverage and time delay

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Abstract: During infectious disease outbreaks, media dissemination, and time delays critically influence the transmission dynamics. To capture the coupled interactions among media influence, time delay, and hospitalization resources, we propose a delayed *susceptible–exposed–infected–hospitalized–recovered–media* (SEIHRM) epidemic model incorporating media feedback and systematically investigate its dynamic behavior both theoretically and numerically. The model is shown to be well-posed in terms of the existence, uniqueness, positivity, and boundedness of solutions. The basic reproduction number R_0 is derived, thereby determining the stability of the disease-free equilibria and serving as a threshold for the existence of endemic equilibria. Bifurcation analysis with the time delay τ as the bifurcation parameter reveals that once τ exceeds a critical threshold τ_0 , a Hopf bifurcation emerges, giving rise to sustained periodic oscillations and thereby recurrent epidemic outbreaks. Numerical simulations substantiate the theoretical results and reveal the distribution of stable equilibria, periodic oscillations, and unstable dynamics across parameter regimes. The results demonstrate that the coupling between media dissemination and time delay can drive complex oscillatory dynamics via delayed feedback, providing a mechanistic explanation for both endemic persistence and recurrent epidemic outbreaks. This study offers a novel theoretical perspective on information-feedback-driven epidemic dynamics and provides a quantitative basis for delay-based targeted intervention strategies.

Keywords: time delay; Hopf bifurcation; media influence; periodic oscillations; infectious disease model

1. Introduction

Despite significant advances in global healthcare systems over recent decades, infectious diseases remain a persistent threat to human health and social stability, causing substantial morbidity and mortality worldwide. Globalization and population mobility have intensified the spread of infectious diseases, resulting in a broader transmission range and enhanced dynamic complexity [1, 2]. Epidemics' persistence is driven not only by pathogens' mutability and cross-species transmission but also by socioeconomic disparities, uneven healthcare resource distribution, and heterogeneity in public behavioral responses. Furthermore, regional differences in public health capacity and risk perception amplify the spatiotemporal heterogeneity of disease spread, thereby complicating epidemic control efforts. Historical outbreaks—including cholera pandemics [3], the 1918 influenza pandemic [4], SARS [5, 6], H1N1 influenza [7], and COVID-19 [8, 9]—underscore that disease transmission arises from complex interactions among biological, social, and behavioral factors. In parallel with these epidemiological challenges, the rapid development of information technology has positioned media dissemination as a key driver of public risk perception and behavior. Media coverage can enhance compliance with public health measures; however, factors such as information overload or misinformation may also induce irrational responses, thereby significantly influencing the epidemic dynamics and the effectiveness of interventions [10].

Given the complex transmission mechanisms of infectious diseases and the diverse factors affecting interventions, using mathematical modeling to elucidate an epidemic's evolution patterns and evaluate the effectiveness of control strategies constitutes a core approach and a research hotspot in epidemiological dynamics. The effective control of infectious disease outbreaks depends on a thorough understanding of the disease transmission dynamics. Therefore, constructing mathematical models that capture disease dynamics is of fundamental importance. Infectious disease-related dynamic models, through reasonable assumptions concerning the population structure and the characterization of interactions between key parameters and state variables, can systematically describe epidemic patterns, predict disease trajectories, and underpin the development of effective control strategies. As an essential tool for analyzing complex biological systems, mathematical modeling is central to understanding epidemics' transmission mechanisms [11], specifically in the quantitative assessment of the effects of interventions such as quarantine and vaccination. Increasingly, with the rapid development of information technology, the influence of public risk perception and behavioral responses on disease transmission has become pronounced. This has made the incorporation of behavioral changes and information diffusion into dynamic modeling frameworks an important research direction [12].

Among the various external factors that influence public epidemic prevention behaviors, media communication plays a key regulatory role. Through channels such as the internet, television, and radio, media can disseminate epidemic-related information to a wide audience at low cost, alter public risk perception and protective behaviors, and directly affect the rate of disease transmission, making it an indispensable tool for infectious disease prevention and control [13, 14]. Existing modeling approaches to media effects are mainly divided into two categories: One introduces media modulation terms into the incidence rate to suppress infection transmission [15–17]; the other incorporates population stratification based on awareness or protective behavior levels to describe the impact of behavioral heterogeneity on disease dynamics [18–20]. In recent years, several researchers have

begun to integrate media coverage effects with optimal control theory. Diagne et al. constructed an epidemic model incorporating treatment measures and media coverage within an optimal control framework, examined the effects of different contact rate functions on disease transmission, and derived time-varying optimal control strategies for treatment and vaccination [21]. Jiang et al., from the perspective of vaccination strategy optimization, proposed a two-stage intervention strategy for brucellosis, using discontinuous adjustments of the vaccination rates to address sudden outbreaks, thereby extending the design paradigm of vaccination strategies beyond the conventional optimal control framework [22]. These studies provide new theoretical tools for the joint optimization of media coverage and intervention strategies.

When the level of media reporting increases, its most direct and pronounced effect is manifested through the modulation of the disease transmission rate. Consequently, a substantial body of research has focused on incorporating media effects into transmission functions. In the existing literature, media influence is typically modeled by introducing nonlinear modifying terms to reduce the effective contact rate. For instance, in Ref. [23], the term $\frac{\beta_2 I}{m+I}$ is used to characterize the reduction in the contact rate induced by media intervention, leading to a transmission function of the form $\frac{\beta_1 - \beta_2 I}{m+I}$ to characterize the effective transmission process under media awareness. Subsequent studies have extended this framework by incorporating the psychological impact of both infected and hospitalized individuals, introducing an exponential decay factor that yields a transmission rate of the form $\beta_0 = \beta e^{-\alpha_1 E - \alpha_2 I - \alpha_3 H}$, thereby capturing the suppressive effects of multi source information on the disease's spread [24].

Moreover, some studies adopt functional forms such as $e^{-M(I, \frac{dI}{dt})}$ to capture behavioral responses induced by epidemic-related information, where $M(I, \frac{dI}{dt}) = \max\{0, p_1 I + p_2 \frac{dI}{dt}\}$, thereby revealing the regulatory role of dynamic information feedback in modulating transmission dynamics [25]. In addition, media effects have also been modeled as $f(M, p) = e^{pM}$, where the parameter p denotes the strength of media influence and is typically assumed to depend on the cumulative intensity of information [26]. On the other hand, some studies move beyond treating media effects as mere modifiers of the transmission rate and instead model media reporting as an independent dynamical variable. By introducing additional compartments, these approaches capture the coupling between media dynamics and population dynamics [27–29].

However, the influence of media coverage is typically not instantaneous. A significant time lag often exists between information release, public reception, and subsequent behavioral responses. Such delays are ubiquitous in biological systems, particularly in infectious disease transmission, arising from intrinsic processes as well as extrinsic mechanisms such as media dissemination and behavioral feedback [30–32]. Incorporating time delays improves the realism of epidemic models and enables them to capture complex dynamics, including stability loss, periodic oscillations, bifurcations, and even chaotic behavior [33, 34]. The effects of time delays have been extensively investigated. Wang et al. [35] and Zhu et al. [36] analyzed the global dynamics of multi-delay systems, while Barman and Mishra [37] and Zhou et al. [38] highlighted the role of delays in shaping stability and bifurcation structures, particularly in vaccination assessments. In addition, Liu et al. [39] developed a delayed *susceptible–antibody–sensitive–infected–media–aware* (SAIM) model to capture media-influenced vaccination willingness and evaluate its impact on COVID-19 control. Overall, these studies demonstrate that incorporating time delays is essential for understanding the coupled dynamics of information spread and disease transmission, and for improving epidemic intervention strategies.

Meanwhile, research on time-delay effects has gradually expanded from single-model frameworks

to more complex transmission settings. Wang et al. investigated the spread of infectious diseases on time-varying networks with time delays, established an averaging theorem to approximate the time-varying system by an autonomous differential system, and derived the critical delay threshold for the onset of Hopf bifurcation, thereby revealing the driving role of delays in the system's phase transitions within time-varying network environments [33]. Han et al., focusing on spatial heterogeneity, developed a two-community *susceptible–infected–recovered* (SIR) model with asymmetric coupling, and uncovered the non-monotonic regulation mechanism of asymmetric population movements on the basic reproduction number, deepening the understanding of transmission dynamics in multi-community coupled structures [40]. These studies indicate that the coupling of delay effects with network structure and spatial heterogeneity has become an important frontier in epidemic modeling.

In summary, although existing studies have made important extensions to epidemic models from various perspectives, including optimal control, vaccination strategy optimization, network delays, and spatial heterogeneity, the coupling mechanism between media effects and information delays (namely, how an epidemic's prevalence forms a closed-loop regulation through media feedback and how information delays drive periodic oscillations from within the system) still lacks a systematic dynamic characterization in the current literature. In particular, the complex dynamic behaviors arising from the interplay between media dynamics and hospitalization/isolation mechanisms remain to be further investigated. The core contributions of this paper are as follows: We construct an dynamic epidemic model that incorporates media coverage and time-delay effects, and introduce a hospitalized compartment to characterize the impact of isolation measures on the transmission process. On this basis, we systematically analyze the basic reproduction number and the stability of equilibria, and further investigate the Hopf bifurcation behavior driven jointly by nonlinearity and time delay, in order to more accurately reveal the evolution patterns of epidemics and provide theoretical foundations for prevention and control strategies.

The rest of this paper is structured as follows. Section 2 presents the formulation of the model and related definitions. Section 3 establishes the existence, uniqueness, and boundedness of solutions. It also derives R_0 and investigates the stability properties of the model. Section 4 examines the bifurcation behavior exhibited by the model. In Section 5, we carry out numerical simulations to support and validate the theoretical findings. Finally, Section 6 summarizes the main findings of this study.

2. Model formulation

Hethcote [41] presented a comprehensive account of the classical *susceptible–exposed–infected–recovered* (SEIR) framework in his seminal review, where the dynamics are governed by the following model of ordinary differential equations:

$$\begin{aligned} \frac{dS(t)}{dt} &= \mu N - \mu S - \frac{\beta S I}{N}, & \frac{dE(t)}{dt} &= \frac{\beta S I}{N} - (\mu + \sigma)E, \\ \frac{dI(t)}{dt} &= \sigma E - (\mu + \gamma)I, & \frac{dR(t)}{dt} &= \gamma I - \mu R. \end{aligned} \quad (2.1)$$

Building upon the model in Eq (2.1), we take the following aspects into account:

- The function $\beta_1 - \beta_2 \frac{M(t)}{1+kM(t)}$ is introduced to describe the reduction in the transmission rate induced by media coverage while accounting for its saturation effect. This prevents the unrealistic scenario

of unbounded media influence and thereby yields a more faithful representation of the underlying transmission dynamics.

- A time-delay term is incorporated to describe the delayed response to media reporting, reflecting the time lag between information dissemination and behavioral response.
- We assume a uniform mortality risk across all population subgroups.
- Using Hopf bifurcation theory, the local dynamical behavior and the properties of the Hopf bifurcation near the endemic equilibrium (EE) are systematically investigated. The results show that, with the time delay τ as the bifurcation parameter, the model transitions from a stable equilibrium to periodic oscillations once τ crosses a critical threshold (see Figures 8–12). This result highlights the crucial role of time delay in inducing complex dynamical behaviors and provides new theoretical insights into oscillatory phenomena in epidemic dynamics.

Motivated by the above considerations, we propose a time-delayed epidemic model that incorporates a media coverage compartment $M(t)$, where the delayed infectious population $I(t - \tau)$ serves as the information source driving media-triggered behavioral responses. The full model of governing equations is given by

$$\left\{ \begin{array}{l} \frac{dS(t)}{dt} = \Pi - \left(\beta_1 - \beta_2 \frac{M(t)}{1 + kM(t)} \right) S(t)I(t) + \delta R(t) - \mu S(t), \\ \frac{dE(t)}{dt} = \left(\beta_1 - \beta_2 \frac{M(t)}{1 + kM(t)} \right) S(t)I(t) - \eta_e E(t) - \mu E(t), \\ \frac{dI(t)}{dt} = \eta_e E(t) - (\mu_1 + r_1 + \sigma + \mu) I(t), \\ \frac{dH(t)}{dt} = \sigma I(t) - (\mu_2 + r_2 + \mu) H(t), \\ \frac{dR(t)}{dt} = r_1 I(t) + r_2 H(t) - (\delta + \mu) R(t), \\ \frac{dM(t)}{dt} = \xi_1 \eta_e E(t) + \xi_1 I(t - \tau) - \xi_2 M(t). \end{array} \right. \quad (2.2)$$

Figure 1 depicts the principal transmission pathways characterized by the model, whereas Table 1 provides the biological interpretations of the associated parameters. The initial conditions for the state variables are given as follows:

$$S(0) = S_0, E(0) = E_0, I(0) = I_0, H(0) = H_0, R(0) = R_0, M(0) = M_0. \quad (2.3)$$

The transmission dynamics described by the model in Eq (2.2) are illustrated in Figure 1. The susceptible population $S(t)$ is recruited into the system at a rate Π and transitions to the exposed class $E(t)$ through effective contact with infectious individuals $I(t)$, governed by the incidence function $\left(\beta_1 - \beta_2 \frac{M(t)}{1 + kM(t)} \right) S(t)I(t)$. This incidence formulation incorporates the suppressive effect of media coverage $M(t)$ on disease transmission. The exposed compartment $E(t)$ consists of individuals who are infected but not yet capable of transmitting the disease. These individuals transition to the infectious class at rate η_e , after which they may either enter hospitalization at a rate σ or recover directly at a rate r_1 . Hospitalized individuals $H(t)$ may further recover at a rate r_2 , while disease-induced

mortality occurs in the infectious and hospitalized classes at the rates μ_1 and μ_2 , respectively. Recovered individuals $R(t)$ may lose immunity and return to the susceptible class at a rate δ , while all compartments are subject to natural death at a rate μ . The media variable $M(t)$ evolves in response to the current number of infected individuals and, in turn, suppresses disease transmission by reducing the effective contact rate.

In most compartmental epidemic models that incorporate a media coverage compartment, the variable $M(t)$ is used to quantify the intensity of media coverage and the level of public attention to disease-related information. Specifically, as prevalent infectious cases grow, both the volume of media reporting and public concern tend to rise correspondingly, leading to an increase in $M(t)$. This trend characterizes a positive feedback mechanism through which the epidemic's prevalence drives media responsiveness. To capture this regulatory effect, we formulate the following effective transmission rate function:

$$\beta(M) = \beta_1 - \beta_2 \frac{M(t)}{1 + kM(t)},$$

here $\beta_1 > 0$ denotes the baseline effective transmission rate between susceptible and infectious individuals in the absence of media interventions, $\beta_2 > 0$ is the reduction coefficient of effective exposure induced by media coverage, and $k > 0$ is the saturation parameter that governs the diminishing marginal effect of media-induced suppression. To guarantee the biological and mathematical validity of the model under extreme media intensity, we impose the constraint $\beta_1 k > \beta_2$. Compared with the standard incidence function, this function exhibits the following limiting behaviors: As $M \rightarrow 0$, it reduces to the baseline transmission rate β_1 ; as $M \rightarrow +\infty$, it converges to $\beta_1 - \beta_2/k$, indicating that media regulation of the transmission rate possesses an inherent upper bound at saturation and does not completely halt the disease's spread. This formulation enables a more reasonable characterization of the nonlinear regulatory impact of media coverage on transmission rates across distinct epidemic phases.

We incorporate $M(t)$ as an independent state variable into the dynamical framework to characterize the feedback regulatory mechanism of media response on the epidemic's evolution. The evolution of $M(t)$ is driven by two sources of information input. The first is the instantaneous contribution from the latent reservoir $\xi_1 \eta_e E(t)$: Although latent individuals have not yet developed clinical symptoms, their population size, as a leading indicator of the epidemic's progression, still provides real-time information input through channels such as epidemic bulletins. The second is the public risk perception and behavioral feedback driven by the delayed infectious cases $\xi_1 I(t - \tau)$, where the delay term τ primarily describes the response lag of media coverage, reflecting the time delay between the dissemination of external information and its actual impact on population behavior. In addition, media-related public attention naturally decays over time, which is modeled by the linear decay term $-\xi_2 M(t)$. Accordingly, the dynamical equation governing $M(t)$ is formulated as follows:

$$\frac{dM(t)}{dt} = \xi_1 \eta_e E(t) + \xi_1 I(t - \tau) - \xi_2 M(t),$$

where $\xi_1 > 0$ denotes the follow-up rate of media coverage, characterizing the responsiveness of the media system to epidemic information; $\xi_2 > 0$ denotes the decay rate of media coverage, reflecting the natural decline of public attention over time; and $\eta_e > 0$ denotes the transfer rate from the exposed class to the infectious class. Furthermore, the incorporation of the delay term in the formulation is grounded in the following modeling assumptions: (i) The primary information sources for media

coverage and public risk perception are symptomatic infectious individuals who have been confirmed by the public health surveillance system, rather than asymptomatic exposed individuals; hence, the delay τ is applied exclusively to the $I(t)$ compartment; (ii) although exposed individuals $E(t)$ do not directly trigger delayed feedback, their population size, serving as a leading indicator of the epidemic's progression, still provides real-time information input to the media system through the instantaneous term $\xi_1\eta_e E(t)$, thereby retaining their role in media dynamics within the model.

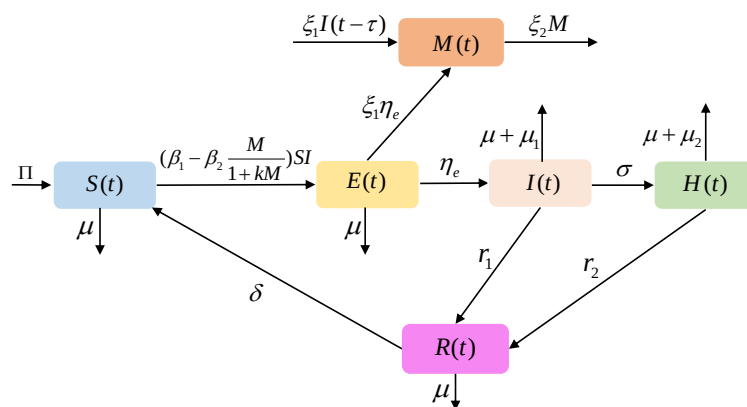


Figure 1. Flow diagram of the model in Eq (2.2).

Table 1. Description of the parameter values used in the model in Eq (2.2). All remaining parameter values are assigned solely for illustrative numerical simulations.

Parameters	Definition	Values/range	Reference
Π	Population recruitment rate	Varied	–
β_1	Effective contact rate between susceptible and infected individuals	0.0069	[42]
β_2	Reduction coefficient of effective exposure due to media coverage	0.0039	Assumed
k	Saturation rate parameter (media-related psychological saturation, half-saturation constant)	0.52	[43]
δ	Transition rate of recovered individuals returning to the susceptible class	0.25	[43]
σ	Rate at which infectious individuals are isolated and transferred to the hospitalized class	0.3	[43]
μ	Natural mortality rate	0.005	[44]
η_e	Transition rate from exposed individuals to infectious individuals	0.024	Assumed
μ_1	Disease-induced mortality rate of infected individuals	0.02	[45]
μ_2	Disease-induced mortality rate of hospitalized individuals	0.01	[45]
r_1	Recovery rate of infectious individuals	0.0015	Assumed
r_2	Recovery rate of hospitalized individuals	0.34	[45]
ξ_1	Rate of behavioral response to media reports	0.02	Assumed
ξ_2	Fading rate of media reports	0.01	Assumed

3. Dynamic analysis of model in Eq (2.2)

To gain a deeper insight into the transmission mechanism of model in Eq (2.2), we perform a systematic dynamic analysis in this section. We rigorously establish the existence, uniqueness, non-negativity, and boundedness of the solutions, thereby guaranteeing the biological relevance of the model.

3.1. Existence and uniqueness of solutions

Theorem 1. *Let constant delay $\tau > 0$. For any non-negative continuous initial function*

$$\phi \in C([-\tau], \mathbb{R}_+^6),$$

the retarded delay differential model in Eq (2.2) admits a unique global solution $X(t; \phi)$ defined on $[0, +\infty)$.

Proof. Step 1: Basic space and history notation

Denote the Banach space of continuous vector-valued functions on the following delay interval:

$$C = C([-\tau, 0], \mathbb{R}^6),$$

equipped with the supremum norm

$$\|\phi\|_C = \sup_{s \in [-\tau, 0]} \max_{1 \leq i \leq 6} |\phi_i(s)|, \quad \forall \phi \in C.$$

For any trajectory $X : [-\tau, T] \rightarrow \mathbb{R}^6$ satisfying $X(s) = \phi(s)$, $s \in [-\tau, 0]$, its history segment at time $t \geq 0$ is defined as

$$X_t(s) = X(t + s), \quad s \in [-\tau, 0].$$

The right-hand side vector field $G = (G_1, G_2, G_3, G_4, G_5, G_6) : \mathbb{R}_+^6 \rightarrow \mathbb{R}^6$ of the model in Eq (2.2):

$$G_1(X) = \Pi - \left(\beta_1 - \frac{\beta_2 M}{1 + kM} \right) S I + \delta R - \mu S,$$

$$G_2(X) = \left(\beta_1 - \frac{\beta_2 M}{1 + kM} \right) S I - \eta_e E - \mu E,$$

$$G_3(X) = \eta_e E - (\mu_1 + r_1 + \sigma + \mu) I,$$

$$G_4(X) = \sigma I - (\mu_2 + r_2 + \mu) H,$$

$$G_5(X) = r_1 I + r_2 H - (\delta + \mu) R,$$

$$G_6(X_t) = \xi_1 \eta_e E(t) + \xi_1 I_t(0) - \xi_2 M(t).$$

Define the integral solution operator $F : C \rightarrow C$ as follows:

$$(F\phi)(t) = \phi(0) + \int_0^t G(X_\phi(s)) ds, \quad t \in [0, T],$$

where X_ϕ stands for the trajectory generated by the initial segment ϕ .

Step 2: Local Lipschitz property of G on bounded state sets

Let $U > 0$ be an arbitrary finite constant whose choice is independent of the model parameters and can be taken to be arbitrarily large. We define the bounded region

$$\Omega_U = \{(S, E, I, H, R, M) \in \mathbb{R}_+^6 \mid \max\{|S|, |E|, |I|, |H|, |R|, |M|\} \leq U\}.$$

Take two history functions $\phi, \psi \in C$, such that $X_\phi(s), X_\psi(s) \in \Omega_U$ for all $s \in [0, T]$. We write

$$Y = X_\phi(s) = (S, E, I, H, R, M), \quad Z = X_\psi(s) = (S_1, E_1, I_1, H_1, R_1, M_1).$$

Compute the components' absolute differences as follows:

$$|G_1(Y) - G_1(Z)| \leq \mu|S - S_1| + \beta_1|SI - S_1I_1| + \beta_2 \left| \frac{MSI}{1 + kM} - \frac{M_1S_1I_1}{1 + kM_1} \right| + \delta|R - R_1|,$$

$$|G_2(Y) - G_2(Z)| \leq \beta_1|SI - S_1I_1| + \beta_2 \left| \frac{MSI}{1 + kM} - \frac{M_1S_1I_1}{1 + kM_1} \right| + (\eta_e + \mu)|E - E_1|,$$

$$|G_3(Y) - G_3(Z)| \leq \eta_e|E - E_1| + (\mu_1 + r_1 + \sigma + \mu)|I - I_1|,$$

$$|G_4(Y) - G_4(Z)| \leq \sigma|I - I_1| + (\mu_2 + r_2 + \mu)|H - H_1|,$$

$$|G_5(Y) - G_5(Z)| \leq r_1|I - I_1| + r_2|H - H_1| + (\delta + \mu)|R - R_1|,$$

$$|G_6(Y_t) - G_6(Z_t)| \leq \xi_1\eta_e|E - E_1| + \xi_1\|I_t - I_t\|_C + \xi_2|M - M_1|.$$

For all $Y, Z \in \Omega_U$, we have $|SI - S_1I_1| \leq U\|Y - Z\|_{\mathbb{R}^6}$. There is a positive Lipschitz constant

$$B = \max\{\mu + 2\beta_1U + 2\beta_2U, 2\eta_e + \mu + \xi_1\eta_e, \mu_1 + r_1 + \sigma + \mu, \mu_2 + r_2 + \mu, 2\delta + \mu, \xi_1 + \xi_2\},$$

satisfying

$$\|G(Y) - G(Z)\|_{\mathbb{R}^6} \leq B\|Y - Z\|_{\mathbb{R}^6}.$$

This uniform Lipschitz bound holds uniformly for all $s \in [0, T]$. Then for the operator F , we have:

$$\|F\phi - F\psi\|_C \leq \sup_{t \in [0, T]} \int_0^t B\|X_\phi(s) - X_\psi(s)\|_C ds \leq BT \cdot \|\phi - \psi\|_C.$$

Step 3: Local unique solution via Banach's fixed-point theorem

Choose a sufficiently small $T_0 > 0$ such that $BT_0 < 1$. Then F becomes a strict contraction mapping on the Banach space C . By the Banach's fixed-point theorem, there is a unique fixed point $X_\phi \in C$, which corresponds to the unique local solution of model in Eq (2.2) defined on $[0, T_0]$.

Step 4: Global extension to $[0, +\infty)$

Let $[0, T_{\max})$ denote the maximal existence interval of the local solution derived above. Theorem 2 will prove all population compartments satisfy the following uniform global bounds:

$$N(t) = S(t) + E(t) + I(t) + R(t) \leq \frac{\Pi}{\mu}, \quad 0 < M(t) \leq \frac{\Pi\xi_1}{\mu\xi_2}, \quad \forall t \in [0, T_{\max}).$$

All state variables are uniformly bounded on $[0, T_{\max})$, which excludes finite-time blow-up. Thus $T_{\max} = +\infty$. We extend the local unique solution iteratively on successive intervals $[T_0, 2T_0], [2T_0, 3T_0], \dots$, and finally obtain the unique global solution on $[0, +\infty)$.

Remark 1. The constant U introduced in the proof of Theorem 1 is a temporary auxiliary upper bound only used to construct the bounded state subset Ω_U and to verify the local Lipschitz property of vector field G . This constant can be chosen as any sufficiently large finite positive number and does not need to be preset by the model parameters. The Lipschitz constant B depends on U as well as the model parameters $\beta_1, \beta_2, k, \mu, \xi_1, \xi_2$, which indicates that U merely serves as an intermediate analytical tool rather than the genuine global upper bound of population variables. Explicit uniform global bounds $N(t) \leq \Pi/\mu$ and $M(t) \leq \xi_1 \Pi/(\mu \xi_2)$ for all state variables will be derived independently in Theorem 2. Combined with the continuation theory for functional differential equations, the global boundedness result in Theorem 2 rules out finite-time blow-up, so the unique local solution can be extended to $[0, +\infty)$. The global existence and uniqueness of the system's solutions are jointly guaranteed by Theorems 1 and 2. The whole proof strictly follows the theoretical framework of functional differential equations proposed in earlier work [46, 47].

3.2. Non-negativity and boundedness of solutions

To guarantee that the model is both biologically meaningful and mathematically well-posed, one must verify that its solutions remain non-negative and bounded. These properties guarantee that all state variables remain within biologically meaningful ranges throughout the evolution of the model. To this end, we introduce the positively invariant set $\Omega^+ = \{(S, E, I, H, R, M) \in \Omega \mid S > 0, E > 0, I > 0, H > 0, R > 0, M > 0\}$, which characterizes the set of solutions satisfying the non-negativity constraint.

Theorem 2. If the initial conditions satisfy Eq (2.3), then the solution of the model in Eq (2.2) is confined to $\mathbb{R}_+^6$ for all time, indicating that the system is positively invariant. Moreover, all solutions eventually enter and remain in a bounded region Ω , where $\Omega^* \subset \Omega$ is defined as follows:

$$\Omega^* = \left\{ (S, E, I, H, R, M) \in \mathbb{R}_+^6 : S + E + I + H + R \leq \frac{\Pi}{\mu}, 0 < M \leq \frac{\Pi \xi_1}{\mu \xi_2} \right\}.$$

Proof. The right-hand side of the model in Eq (2.2) defines a continuously differentiable vector field in $\mathbb{R}_+^6$. Thus, given any non-negative initial condition, the model in Eq (2.2) possesses a unique solution.

From the model in Eq (2.2), we have

$$\begin{aligned} \left. \frac{dS(t)}{dt} \right|_{S=0, E, I, H, R, M \geq 0} &= \Pi + \delta R(t) \geq 0, \\ \left. \frac{dE(t)}{dt} \right|_{E=0, S, I, H, R, M \geq 0} &= \left(\beta_1 - \beta_2 \frac{M(t)}{1 + kM(t)} \right) S(t)I(t) \geq 0, \\ \left. \frac{dI(t)}{dt} \right|_{I=0, S, E, H, R, M \geq 0} &= \eta_e E(t) \geq 0, \\ \left. \frac{dH(t)}{dt} \right|_{H=0, S, E, I, R, M \geq 0} &= \sigma I(t) \geq 0, \\ \left. \frac{dR(t)}{dt} \right|_{R=0, S, E, I, H, M \geq 0} &= r_1 I(t) + r_2 H(t) \geq 0, \end{aligned}$$

$$\left. \frac{dM(t)}{dt} \right|_{M=0, S, E, I, H, R \geq 0} = \xi_1 \eta_e E(t) + \xi_1 I(t - \tau) \geq 0.$$

This implies that, on the boundary of the non-negative orthant, the vector field is inward pointing. Consequently, all solution trajectories remain in the non-negative region; that is, $\mathbb{R}_+^6$ is positively invariant. In particular, for any $t > 0$, the unique solution of the model in Eq (2.2) with the initial condition $(S_0, E_0, I_0, H_0, R_0, M_0) \in \mathbb{R}_+^6$ remains non-negative.

To analyze the boundedness of the model in Eq (2.2), we define the following variables:

$$N(t) = S(t) + E(t) + I(t) + H(t) + R(t).$$

Differentiating the above expression yields

$$\begin{aligned} \frac{dN(t)}{dt} &= \frac{dS(t)}{dt} + \frac{dE(t)}{dt} + \frac{dI(t)}{dt} + \frac{dH(t)}{dt} + \frac{dR(t)}{dt}, \\ &= \Pi - \mu(S(t) + E(t) + I(t) + H(t) + R(t)) - \mu_1(I(t) + H(t)), \\ &= \Pi - \mu N(t) - \mu_1(I(t) + H(t)). \end{aligned} \quad (3.1)$$

Since $\mu_1 > 0$ and $I(t) + H(t) \geq 0$ for all $t \geq 0$, Eq (3.1) becomes

$$\frac{dN(t)}{dt} \leq \Pi - \mu N(t), \quad (3.2)$$

integrating Eq (3.2) yields

$$N(t) = \frac{\Pi}{\mu} + \left(N(0) - \frac{\Pi}{\mu}\right)e^{-\mu t} \geq 0,$$

where $N(0)$ is the total population size at $t = 0$. Hence, for $t > 0$, we have

$$\limsup_{t \rightarrow \infty} N(t) \leq \frac{\Pi}{\mu}.$$

To further investigate the boundedness of $M(t)$, the last equation of model in Eq (2.2) yields

$$\frac{dM(t)}{dt} = \xi_1 \eta_e E(t) + \xi_1 I(t - \tau) - \xi_2 M(t) \leq \xi_1 \frac{\Pi}{\mu} - \xi_2 M,$$

which implies

$$\limsup_{t \rightarrow \infty} M(t) \leq \frac{\Pi \xi_1}{\mu \xi_2}.$$

In summary, all trajectories of the model in Eq (2.2) are uniformly bounded and eventually enter the region

$$\Omega^* = \left\{ (S, E, I, H, R, M) \in \mathbb{R}_+^6 : S + E + I + H + R \leq \frac{\Pi}{\mu}, 0 < M \leq \frac{\Pi \xi_1}{\mu \xi_2} \right\}.$$

Thus completing the proof of Theorem 2.

3.3. Existence of equilibria

The dynamical behavior of the model in Eq (2.2) is governed by its equilibrium structure, which comprises the disease-free equilibrium (DFE) and the EE, corresponding to the extinction and persistence of the disease, respectively. By imposing the steady-state condition, namely that the time derivatives of all state variables vanish, one obtains the constraints that characterize the equilibria of the model. Under this condition, the behavior of the model in Eq (2.2) at equilibrium can be equivalently described by the following algebraic relations, which describes the distribution and qualitative properties of its equilibrium solutions.

$$\begin{cases} \Pi - \left(\beta_1 - \beta_2 \frac{M(t)}{1 + kM(t)} \right) S(t)I(t) + \delta R(t) - \mu S(t) = 0, \\ \left(\beta_1 - \beta_2 \frac{M(t)}{1 + kM(t)} \right) S(t)I(t) - \eta_e E(t) - \mu E(t) = 0, \\ \eta_e E(t) - (\mu_1 + r_1 + \sigma + \mu) I(t) = 0, \\ \sigma I(t) - (\mu_2 + r_2 + \mu) H(t) = 0, \\ r_1 I(t) + r_2 H(t) - (\delta + \mu) R(t) = 0, \\ \xi_1 \eta_e E(t) + \xi_1 I(t - \tau) - \xi_2 M(t) = 0. \end{cases} \quad (3.3)$$

The model in Eq (2.2) always possesses a DFE, denoted by $\epsilon_0 = (\frac{\Pi}{\mu}, 0, 0, 0, 0, 0)$. Substituting the DFE gives the following equation:

$$\Pi - \mu S(t) = 0.$$

Consequently, for the equilibrium $\epsilon_* = (S^*, E^*, I^*, H^*, R^*, M^*)$, each component obeys the following system of equations:

$$\begin{aligned} S^* &= \frac{(\eta_e + \mu)(\mu_1 + r_1 + \sigma + \mu)(\xi_2 + k\xi_1(1 + \mu_1 + r_1 + \sigma + \mu)I^*)}{\eta_e \beta_1 \xi_2 + \eta_e \xi_1(\beta_1 k - \beta_2)(1 + \mu_1 + r_1 + \sigma + \mu)I^*}, \\ E^* &= \frac{(\mu_1 + r_1 + \sigma + \mu)I^*}{\eta_e}, \\ H^* &= \frac{\sigma I^*}{\mu_2 + r_2 + \mu}, \\ R^* &= \frac{(r_1 \mu_2 + r_1 r_2 + r_1 \mu + r_2 \sigma)I^*}{(\delta + \mu)(\mu_2 + r_2 + \mu)}, \\ M^* &= \frac{\xi_1(1 + \mu_1 + r_1 + \sigma + \mu)I^*}{\xi_2}, \end{aligned}$$

and I^* can be expressed in the following form

$$AI^3 + BI^2 + CI + D = 0, \quad (3.4)$$

where

$$\begin{aligned} A &= a_1 a_2 k (\xi_1)^2 (a_3 + \mu k) (1 + a_1)^2 (a_4 + a_5) + \eta_e a_3 k (\xi_1)^2 (1 + a_1)^2, \\ B &= a_1 a_2 \xi_1 \xi_2 (1 + a_1) (a_4 + a_5) (a_3 + \beta_1 k + 2\mu k) + a_6 (r_2 \sigma - \delta r_1 a_5) (a_3 + k\beta_1) \\ &\quad - \Pi \eta_e a_3 k (\xi_1)^2 (1 + a_1)^2 (a_4 + a_5), \\ C &= a_1 a_2 (\xi_2)^2 (\beta_1 + \mu) (a_4 + a_5) + \eta_e \beta_1 (\xi_2)^2 (r_2 \sigma \delta r_1 a_5) - \Pi a_6 (a_4 + a_5) (a_3 + k\beta_1), \\ D &= -\Pi \eta_e \beta_1 (\xi_2)^2 (a_4 + a_5). \end{aligned}$$

3.4. Basic reproduction number

The basic reproduction number R_0 is defined as the average number of secondary cases generated by a single infectious individual over the course of the infectious period, assuming a fully susceptible population [48]. This quantity serves as an essential threshold characterizing the epidemic's transmission capability: When $R_0 > 1$, the infection can spread persistently within the population and lead to sustained transmission; in contrast, when $R_0 < 1$, the transmission chain cannot be sustained and the disease will gradually decline and eventually die out. To quantitatively evaluate this threshold, the next-generation matrix method [49] is used to systematically derive R_0 . Let

$$\phi = \begin{bmatrix} \beta_1 - \beta_2 \frac{M}{1+kM} S I \\ 0 \\ 0 \end{bmatrix}, \quad \psi = \begin{bmatrix} (\eta_e + \mu) E \\ (\mu_1 + r_1 + \sigma + \mu) I - \eta_e E \\ (\mu_2 + r_2 + \mu) H - \sigma I \end{bmatrix}.$$

For the model in Eq (2.2), the DFE is given by $\epsilon_0 = \left(\frac{\Pi}{\mu}, 0, 0, 0, 0, 0\right)$, which corresponds to the state where all infection-related compartments vanish, indicating that the disease is completely absent from the population. At this equilibrium, the Jacobian matrix derived from the functions φ and ψ is given by

$$F = \begin{bmatrix} 0 & \beta_1 \frac{\Pi}{\mu} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \quad V = \begin{bmatrix} \eta_e + \mu & 0 & 0 \\ -\eta_e & \mu_1 + r_1 + \sigma + \mu & 0 \\ 0 & -\sigma & \mu_2 + r_2 + \mu \end{bmatrix},$$

the R_0 for the model in Eq (2.2) is given by

$$FV^{-1} = \begin{bmatrix} \frac{\Pi \beta_1 \eta_e}{\mu(\eta_e + \mu)(\mu_1 + r_1 + \sigma + \mu)} & \frac{\Pi \beta_1}{\mu(\mu_1 + r_1 + \sigma + \mu)} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix},$$

and thus,

$$R_0 = \frac{\Pi \beta_1 \eta_e}{\mu(\eta_e + \mu)(\mu_1 + r_1 + \sigma + \mu)}.$$

The R_0 acts as a key threshold parameter characterizing the transmission intensity of the disease in the model in Eq (2.2). When $R_0 < 1$, the infection cannot be sustained and eventually dies out; when $R_0 > 1$, the disease may persist in the population and lead to an endemic state. It is worth noting that the epidemic's dynamics are not solely determined by R_0 , but are also closely related to the intrinsic model structure and external intervention factors. To this end, by fixing $\eta_e, \mu_1, r_1, \sigma$, we focus on evaluating the effects of the natural mortality rate, recruitment rate, and the effective contact

rate between susceptible and infectious individuals on R_0 . The simulation outcomes are illustrated in Figure 2.

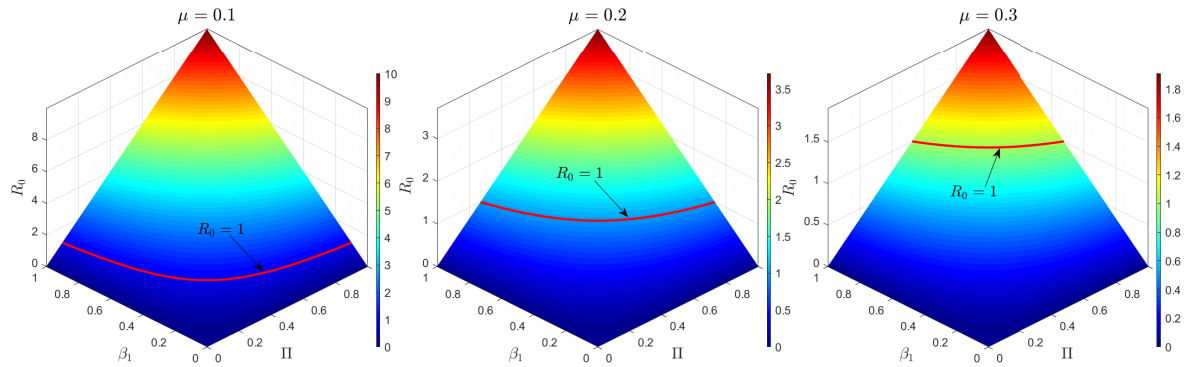


Figure 2. Under the fixed parameters $\eta_e = 0.4$, $\mu_1 = 0.2$, $r_1 = 0.2$, and $\sigma = 0.3$, we examine the effects of Π and β_1 on the R_0 .

The analysis reveals that the R_0 increases significantly with the parameters Π and β_1 . Further numerical analysis shows that, under the threshold condition $R_0 = 1$, the corresponding critical values of Π and β_1 increase simultaneously, highlighting their dominant roles in determining the transmission threshold of the disease. In contrast, an increase in the parameter μ suppresses disease transmission by shortening the average lifespan of susceptible individuals, thereby reducing the effective window of exposure to the infection. Consequently, reducing the population recruitment rate Π (e.g., by limiting population mobility) and decreasing the effective contact rate β_1 (e.g., through isolation measures) constitute key strategies for regulating R_0 , enabling effective control of the disease's spread without significantly compromising the population's viability.

It is noteworthy that the expression for the basic reproduction number R_0 does not explicitly contain any media-related parameters. This is because R_0 characterizes the initial transmission potential of the disease in a completely susceptible population, whereas media coverage, as a responsive feedback mechanism, can only be triggered once the epidemic has reached a certain scale and thus cannot affect the transmission threshold before the disease becomes established. However, this does not imply that media effects are unimportant; as demonstrated by the bifurcation analysis in this paper, media feedback can significantly influence the stability and long-term dynamical behavior of the system at the EE by modulating the effective transmission rate.

3.5. Stability of the equilibria

Local stability analysis reveals the asymptotic properties of nonlinear dynamic systems. In this section, we investigate the stability of the DFE and EE to characterize the dynamic behavior of the model in the neighborhood of these steady states.

Theorem 3. When $R_0 < 1$, the DFE ϵ_0 of the model in Eq (2.2) is locally asymptotically stable; when $R_0 > 1$, ϵ_0 loses its stability (see Figure 3).

Proof. The Jacobian matrix evaluated at ϵ_0 is given by

$$J(\epsilon_0) = \begin{bmatrix} -\mu & 0 & -\frac{\beta_1 \Pi}{\mu} & 0 & \delta & 0 \\ 0 & -(\eta_e + \mu) & \frac{\beta_1 \Pi}{\mu} & 0 & 0 & 0 \\ 0 & \eta_e & -(\mu_1 + r_1 + \sigma + \mu) & 0 & 0 & 0 \\ 0 & 0 & \sigma & -(\mu_2 + r_2 + \mu) & 0 & 0 \\ 0 & 0 & r_1 & r_2 & -(\delta + \mu) & 0 \\ 0 & \xi_1 \eta_e & \xi_1 e^{-\lambda \tau} & 0 & 0 & -\xi_2 \end{bmatrix}.$$

The associated characteristic equation takes the form

$$\det(J(\epsilon_0) - \lambda I) = 0,$$

where I stands for the identity matrix. This yields

$$(\lambda + \mu)(\lambda + \xi_2)(\lambda + \delta + \mu)[\lambda^2 + (\mu_1 + r_1 + \sigma + 2\mu + \eta_e)\lambda + (\eta_e + \mu)(\mu_1 + r_1 + \sigma + \mu)(1 - R_0)] = 0,$$

at the DFE ϵ_0 , the model in Eq (2.2) admits four negative real eigenvalues, namely $\lambda_1 = -\mu$, $\lambda_2 = -\xi_2$, $\lambda_3 = -\delta - \mu$, and $\lambda_4 = -\mu_2 - r_2 - \mu$, with the remaining eigenvalues determined by the following characteristic equation:

$$\lambda^2 + (\mu_1 + r_1 + \sigma + 2\mu + \eta_e)\lambda + (\eta_e + \mu)(\mu_1 + r_1 + \sigma + \mu)(1 - R_0) = 0. \quad (3.5)$$

An analysis of Eq (3.5) reveals that when $R_0 < 1$, the equilibrium ϵ_0 is locally asymptotically stable according to the Routh–Hurwitz criterion; conversely, when $R_0 > 1$, Vieta's formulas imply that the characteristic equation possesses at least one eigenvalue with a positive real part, and hence ϵ_0 is unstable for the model in Eq (2.2).

Remark 2. From Theorem 3, it follows that when $R_0 < 1$, the model approaches the disease-free state in a neighborhood of the DFE ϵ_0 , indicating that the infection will die out naturally without any additional control interventions.

Theorem 4. If $R_0 > 1$, and $v_0, v_1, v_2, v_3, v_4, v_5 > 0$ with Eqs (3.8)–(3.11) being satisfied, then the EE ϵ_* is locally asymptotically stable, as illustrated in Figure 4.

Proof. The Jacobian at ϵ_* is

$$J(\epsilon_*) = \begin{bmatrix} \Phi_{11} & \Phi_{12} \\ \Phi_{21} & \Phi_{22} \end{bmatrix},$$

where

$$\Phi_{11} = \begin{bmatrix} -(\beta_1 - \beta_2 \frac{M^*}{1+kM^*} I^* + \mu) & 0 & -(\beta_1 - \beta_2 \frac{M^*}{1+kM^*} S^*) \\ (\beta_1 - \beta_2 \frac{M^*}{1+kM^*} I^* & -(\eta_e + \mu) & (\beta_1 - \beta_2 \frac{M^*}{1+kM^*} S^*) \\ 0 & \eta_e & -(\mu + r_1 + \sigma + \mu) \end{bmatrix},$$

$$\Phi_{12} = \begin{bmatrix} 0 & \delta & \frac{\beta_2 S^* I^*}{(1+kM^*)^2} \\ 0 & 0 & -\frac{\beta_2 S^* I^*}{(1+kM^*)^2} \\ 0 & 0 & 0 \end{bmatrix},$$

$$\Phi_{21} = \begin{bmatrix} 0 & 0 & \sigma \\ 0 & 0 & r_1 \\ 0 & \xi_1 \eta_e & \eta_e e^{-\lambda \tau} \end{bmatrix}, \quad \Phi_{22} = \begin{bmatrix} -(\mu_2 + r_2 + \mu) & 0 & 0 \\ r_2 & -(\delta + \mu) & 0 \\ 0 & 0 & -\xi_2 \end{bmatrix}.$$

where $\theta_1 = \beta_1 - \beta_2 \frac{M^*}{1+kM^*} I^*$, $\theta_2 = \frac{\beta_2 S^* I^*}{(1+kM^*)^2}$. From the equation $\det(J(\epsilon_*) - \lambda I)$, the corresponding characteristic equation can be written as

$$(b_3 \lambda^3 + b_2 \lambda^2 + b_1 \lambda + b_0) e^{-\lambda \tau} + \lambda^6 + a_5 \lambda^5 + a_4 \lambda^4 + a_3 \lambda^3 + a_2 \lambda^2 + a_1 \lambda + a_0 = 0, \quad (3.6)$$

where

$$b_3 = \theta_2 \eta_e \xi_1,$$

$$b_2 = \theta_2 \eta_e \xi_1 (Q_1 + Q_4 + Q_5) + \theta_1 \theta_2 \eta_e \xi_1,$$

$$b_1 = \theta_2 \eta_e \xi_1 (Q_1 Q_4 + Q_4 Q_5 + Q_1 Q_5) + \theta_1 \theta_2 \eta_e \xi_1 (Q_4 + Q_5),$$

$$b_0 = \theta_2 \eta_e \xi_1 Q_1 Q_4 Q_5 + \theta_1 \theta_2 \eta_e \xi_1 Q_4 Q_5,$$

$$a_5 = \xi_2 + Q_1 + Q_2 + Q_3 + Q_4 + Q_5,$$

$$a_4 = Q_4 (\xi_2 + Q_1 + Q_2 + Q_3 + Q_5) + (\xi_2 + Q_1 + Q_5) (Q_2 + Q_3) \\ + Q_2 Q_3 - \eta_e \theta_1 S^* + \xi_2 Q_1 + \xi_2 Q_5 + Q_1 Q_5 + \theta_2 \eta_e \xi_1,$$

$$a_3 = Q_4 ((\xi_2 + Q_1 + Q_5) (Q_2 + Q_3) + Q_2 Q_3 - \eta_e \theta_1 S^* + \xi_2 Q_1 + \xi_2 Q_5 + Q_1 Q_5 + \theta_2 \eta_e \xi_1) \\ + Q_2 \eta_e \xi_1 (Q_1 + Q_3 + Q_5) + ((Q_2 Q_3 - \eta_e \theta_1 S^*) (\xi_2 + Q_1 + Q_5) + \xi_2 Q_5 + Q_1 Q_5 (1 + \xi_2)) \\ + (\theta_1^2 \eta_e S^* + \theta_1 \xi_1 \eta_e Q_2),$$

$$a_2 = Q_4 (Q_2 \eta_e \xi_1 (Q_1 + Q_3 + Q_5) + ((Q_2 Q_3 - \eta_e \theta_1 S^*) (\xi_2 + Q_1 + Q_5) + \xi_2 Q_5 + Q_1 Q_5 (1 + \xi_2)) \\ + (\theta_1^2 \eta_e S^* + \theta_1 \xi_1 \eta_e Q_2)) + \theta_2 \eta_e \xi_1 (Q_1 Q_3 + Q_3 Q_5 + Q_1 Q_5) \\ + (Q_2 Q_3 - \eta_e \theta_1 S^*) (\xi_2 Q_1 + \xi_2 Q_5 + Q_1 Q_5) + (Q_2 + Q_3) (Q_1 Q_5 \xi_2) \\ - \theta_1 \eta_e \delta r_1 + (\theta_1^2 \eta_e (\xi_2 + Q_5) + \theta_1 \xi_1 \eta_e Q_2 (Q_3 + Q_4)),$$

$$a_1 = Q_4 (Q_2 \eta_e \xi_1 (Q_1 Q_3 + Q_3 Q_5 + Q_1 Q_5) + (Q_2 Q_3 - \eta_e \theta_1 S^*) (\xi_2 Q_1 + \xi_2 Q_5 + Q_1 Q_5) \\ + (Q_2 + Q_3) (Q_1 Q_5 \xi_2) - \theta_1 \eta_e \delta r_1 + (\theta_1^2 \eta_e S^* (\xi_2 + Q_5) + \theta_1 \xi_1 \eta_e Q_2 (Q_3 + Q_4))) \\ + \theta_2 \eta_e \xi_1 Q_1 Q_3 Q_5 + Q_1 Q_5 \xi_2 (Q_1 Q_3 - \eta_e \theta_1 S^*) - \theta_1 \eta_e \delta r_1 \xi_2 + \theta_1^2 \eta_e S^* \xi_2 Q_5 \\ + \theta_1 \xi_1 \eta_e Q_1 Q_3 Q_5 + \theta_1 \eta_e \delta \sigma r_2,$$

$$a_0 = Q_4 (\theta_2 \eta_e \xi_1 Q_1 Q_3 Q_5 + Q_1 Q_5 \xi_2 (Q_1 Q_3 - \eta_e \theta_1 S^*) - \theta_1 \eta_e \delta r_1 \xi_2 + \theta_1^2 \eta_e S^* \xi_2 Q_5 \\ + \theta_1 \xi_1 \eta_e Q_2 Q_3 Q_5) + \theta_1 \eta_e \delta \sigma r_2 \xi_2,$$

$$Q_1 = \theta_1 + \mu, \quad Q_2 = \eta_e + \mu, \quad Q_3 = \mu_1 + r_1 + \sigma + \mu, \quad Q_4 = \mu_2 + r_2 + \mu.$$

Assume that when $\tau = 0$, Eq (3.6) transforms into

$$\lambda^6 + v_5 \lambda^5 + v_4 \lambda^4 + v_3 \lambda^3 + v_2 \lambda^2 + v_1 \lambda + v_0 = 0, \quad (3.7)$$

where $v_5 = a_5, v_4 = a_4, v_3 = a_3 + b_3, v_2 = a_2 + b_2, v_1 = a_1 + b_1, v_0 = a_0 + b_0$. Applying the Routh–Hurwitz criterion [50, 51], we derive the following expression:

$$\Delta_1 = v_5 = a_5 = \xi_2 + Q_1 + Q_2 + Q_3 + Q_4 + Q_5 > 0, \quad (3.8)$$

$$\Delta_2 = \begin{vmatrix} v_5 & v_3 \\ 1 & v_4 \end{vmatrix} = v_5 v_4 - v_3 > 0, \quad \Delta_3 = \begin{vmatrix} v_5 & v_3 & v_1 \\ 1 & v_4 & v_2 \\ 0 & v_5 & v_3 \end{vmatrix} > 0, \quad (3.9)$$

$$\Delta_4 = \begin{vmatrix} v_5 & v_3 & v_1 & 0 \\ 1 & v_4 & v_2 & v_0 \\ 0 & v_5 & v_3 & v_1 \\ 0 & 1 & v_4 & v_2 \end{vmatrix} > 0, \quad \Delta_5 = \begin{vmatrix} v_5 & v_3 & v_1 & 0 & 0 \\ 1 & v_4 & v_2 & v_0 & 0 \\ 0 & v_5 & v_3 & v_1 & 0 \\ 0 & 1 & v_4 & v_2 & v_0 \\ 0 & 0 & v_5 & v_3 & v_1 \end{vmatrix} > 0, \quad (3.10)$$

$$\Delta_6 = \begin{vmatrix} v_5 & v_3 & v_1 & 0 & 0 & 0 \\ 1 & v_4 & v_2 & v_0 & 0 & 0 \\ 0 & v_5 & v_3 & v_1 & 0 & 0 \\ 0 & 1 & v_4 & v_2 & v_0 & 0 \\ 0 & 0 & v_5 & v_3 & v_1 & 0 \\ 0 & 0 & 1 & v_4 & v_2 & v_0 \end{vmatrix} > 0. \quad (3.11)$$

Therefore, under the conditions $v_i > 0$ ($i = 0, 1, 2, 3, 4, 5$) and with Eqs (3.8)–(3.11) being satisfied, the EE ϵ_* of the model in Eq (2.2) is locally asymptotically stable, and the theorem is proved.

Remark 3. According to Theorem 4, when $R_0 > 1$, the trajectories of the model in Eq (2.2) ultimately approach the vicinity of the EE ϵ_* and remain stable around it. This suggests that, without intervention, the disease persists and eventually stabilizes at an endemic state.

To corroborate the theoretical results and examine the dynamic behavior of the model in Eq (2.2), we perform numerical simulations for various values of the parameter k , as illustrated in Figures 3 and 4.

As shown in Figure 3, when $R_0 < 1$, all infection-related compartments decay. Specifically, $E(t)$, $I(t)$, and $H(t)$ converge to zero, while the media variable $M(t)$ also vanishes. Meanwhile, the recovered population $R(t)$ stabilizes at a positive constant after a transient adjustment, and the susceptible population $S(t)$ increases monotonically to a constant level. These numerical observations confirm that the solutions' trajectories converge to the DFE ϵ_0 , indicating that the disease dies out under this regime, which is consistent with the theoretical predictions.

In contrast, Figure 4 illustrates the asymptotic dynamics when $R_0 > 1$. It can be observed that E , I , and H initially exhibit pronounced transient growth with damped oscillations, and subsequently approach strictly positive steady states. Likewise, R and M converge to nonzero equilibrium levels. Meanwhile, S decreases rapidly in the early stage, followed by a gradual rebound toward a lower endemic level. These dynamical features indicate that the system ultimately approaches the EE ϵ_* , implying the long-term persistence of the disease.

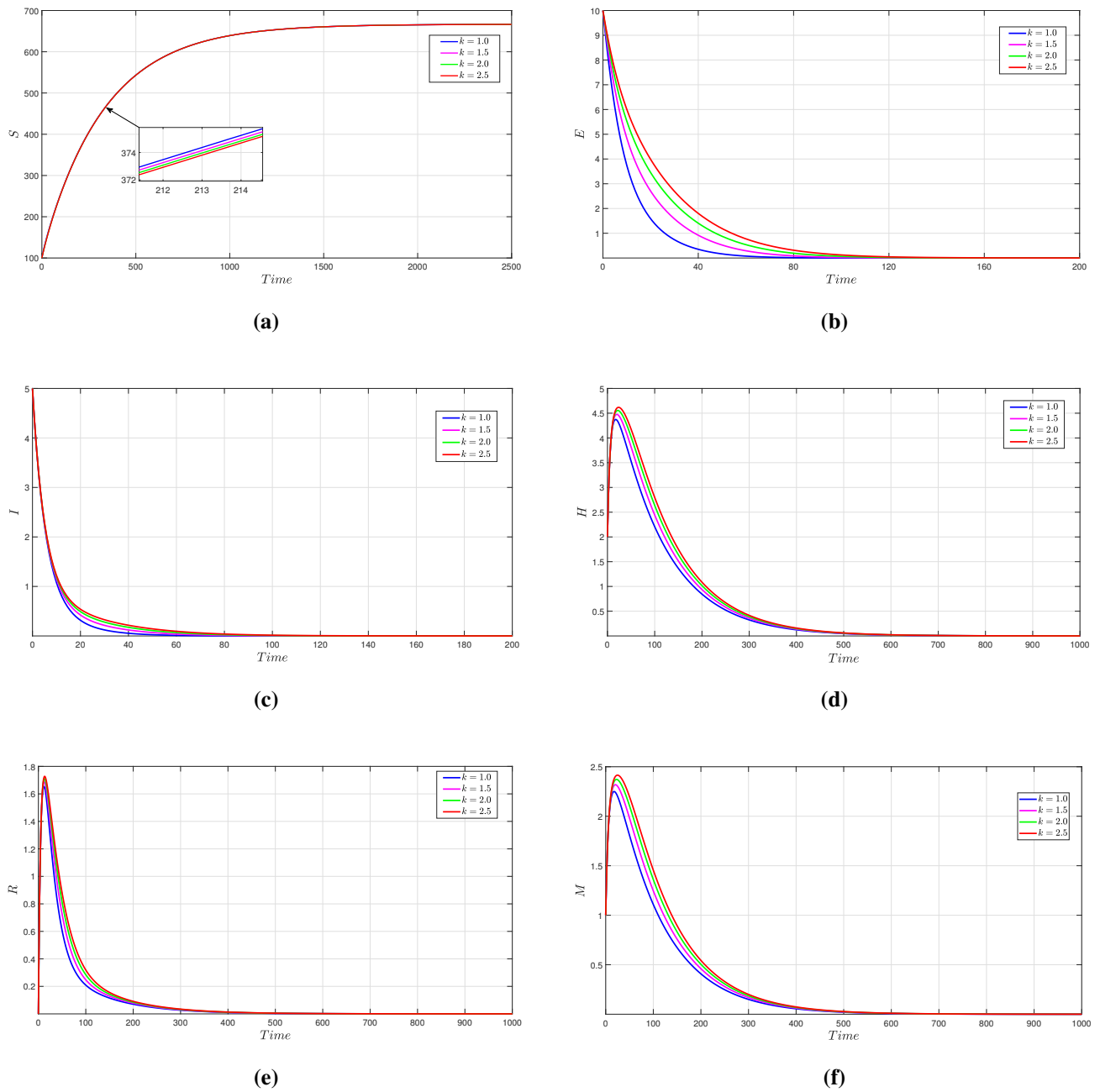


Figure 3. Numerical simulations of the model in Eq (2.2) for different values of the saturation parameter k when the basic reproduction number satisfies $R_0 < 1$. Parameter values: $\Pi = 2$, $\beta_1 = 0.00025$, $\beta_2 = 0.0033$, $\eta_e = 0.02$, $\mu_1 = 0.02$, $\mu_2 = 0.003$, $r_1 = 0.085$, $r_2 = 0.0036$, $\sigma = 0.1$, $\mu = 0.003$, $\xi_1 = 0.05$, $\xi_2 = 0.01$, $\delta = 0.05$, and $\tau = 12$. (a) Changes in the infected population $S(t)$. (b) Changes in the infected population $E(t)$. (c) Changes in the infected population $I(t)$. (d) Changes in the infected population $H(t)$. (e) Changes in the infected population $R(t)$. (f) Changes in the infected population $M(t)$.

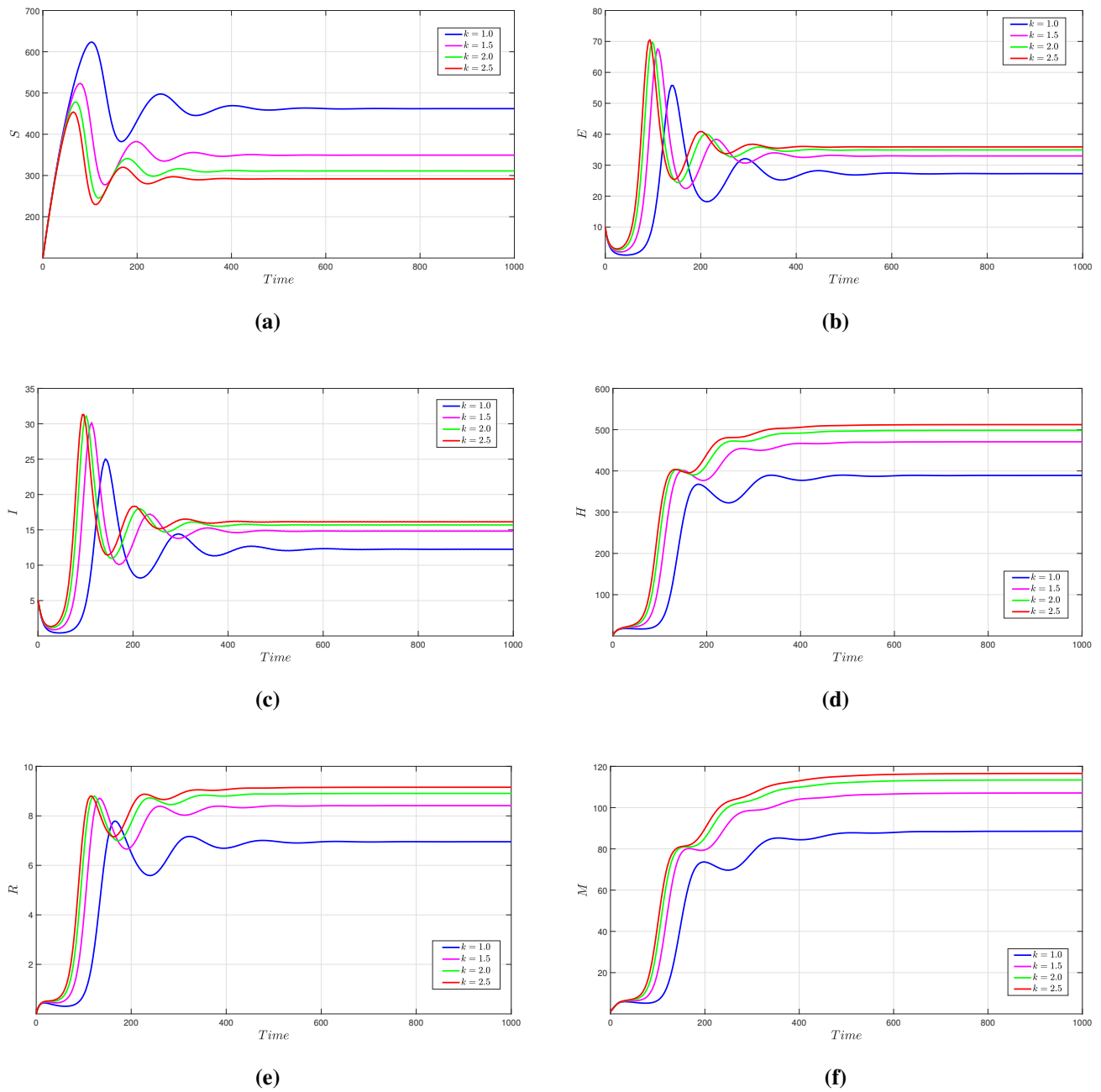


Figure 4. Numerical simulations of the model in Eq (2.2) for different values of the saturation parameter k when the basic reproduction number satisfies $R_0 > 1$. Parameter values: $\Pi = 10$, $\beta_1 = 0.002$, $\beta_2 = 0.001$, $\eta_e = 0.2$, $\mu_1 = 0.02$, $\mu_2 = 0.002$, $r_1 = 0.015$, $r_2 = 0.0006$, $\sigma = 0.04$, $\mu = 0.01$, $\xi_1 = 0.05$, $\xi_2 = 0.01$, $\delta = 0.05$, and $\tau = 12$. (a) Changes in the infected population $S(t)$. (b) Changes in the infected population $E(t)$. (c) Changes in the infected population $I(t)$. (d) Changes in the infected population $H(t)$. (e) Changes in the infected population $R(t)$. (f) Changes in the infected population $M(t)$.

Furthermore, a comparative analysis under different values of k reveals that this parameter exerts a significant influence on the transient dynamics of the model. As k increases, the amplitude of oscillations in all state variables is markedly reduced, and convergence toward equilibrium becomes faster and smoother, reflecting a stronger damping effect and improved stabilization. However, the steady-state levels remain essentially unchanged, suggesting that k primarily affects the convergence rate rather than the equilibrium structure itself. This finding underscores the important role of media-related effects (or saturation mechanisms) in regulating the transmission dynamics.

Overall, the simulation results show strong consistency with the analytical findings and further elucidate the transient behavior and parameter sensitivity of the model under different epidemiological regimes, thereby supporting the validity and robustness of the proposed model.

4. Bifurcation analysis

Bifurcation analysis plays a central role in epidemiological dynamics, revealing that small perturbations in the model parameters can induce qualitative changes in the model's behavior, including the loss of stability of the DFE and the emergence of an EE. Motivated by this insight, we perform a systematic bifurcation analysis of the model in Eq (2.2) to characterize its epidemic threshold and dynamical regimes. In this subsection, we investigate the criteria for the occurrence of a local Hopf bifurcation at the EE $\epsilon_* = (S^*, E^*, I^*, H^*, R^*, M^*)$ of model in Eq (2.2).

At the EE ϵ_* , the characteristic equation of the model in of Eq (2.2) takes the form Eq (3.6), namely

$$(b_3\lambda^3 + b_2\lambda^2 + b_1\lambda + b_0)e^{-\lambda\tau} + \lambda^6 + a_5\lambda^5 + a_4\lambda^4 + a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0,$$

where the coefficients a_i and b_i are specified in Theorem 4. Under the analytical framework established therein, if the conditions $v_i > 0$ ($i = 0, \dots, 5$) and if Eqs (3.8)–(3.11) are satisfied, then the EE ϵ_* of the model in Eq (2.2) is locally asymptotically stable.

For $\tau > 0$, suppose that the characteristic equation possesses a pair of purely imaginary roots $\lambda = i\omega$ with $\omega > 0$. Substitution into the characteristic equation, followed by separation of the real and imaginary components, yields

$$\begin{cases} P_3(\omega) = P_1(\omega) \cos(\omega\tau) + P_2(\omega) \sin(\omega\tau), \\ P_6(\omega) = P_5(\omega) \cos(\omega\tau) + P_4(\omega) \sin(\omega\tau), \end{cases} \quad (4.1)$$

where

$$\begin{aligned} P_1(\omega) &= b_2\omega^2 - b_0, & P_2(\omega) &= b_1\omega - b_3\omega^3, & P_3(\omega) &= -\omega^6 + a_4\omega^4 - a_2\omega^2 + a_0, \\ P_4(\omega) &= b_0 - b_2\omega^2, & P_5(\omega) &= b_3\omega^3 - b_1\omega, & P_6(\omega) &= a_5\omega^5 - a_3\omega^3 + a_1\omega. \end{aligned}$$

From Eq (4.1), we have

$$\cos(\omega\tau) = \frac{m_8\omega^8 + m_6\omega^6 + m_4\omega^4 + m_2\omega^2 - m_0}{n_6\omega^6 + n_4\omega^4 + n_2\omega^2 + n_0}, \quad (4.2)$$

$$\sin(\omega\tau) = \frac{e_9\omega^9 + e_7\omega^7 + e_5\omega^5 + e_3\omega^3 - e_1\omega}{h_6\omega^6 + h_4\omega^4 + h_2\omega^2 + h_0}. \quad (4.3)$$

From $\sin^2(\omega\tau) + \cos^2(\omega\tau) = 1$, we obtain

$$d_{16}\omega^{30} + d_{15}\omega^{28} + d_{14}\omega^{26} + \dots + d_2\omega^4 + d_1\omega^2 + d_0 = 0. \quad (4.4)$$

The coefficients m_i ($i = 0, 1, \dots, 8$), n_i ($i = 0, 1, \dots, 6$), e_i ($i = 1, \dots, 9$), and h_i ($i = 0, 1, \dots, 6$) appearing in Eqs (4.2)–(4.4) are functions of the model parameters. Their explicit expressions are lengthy and algebraically involved; for the sake of readability and to maintain the analytical flow, these expressions are fully provided in Appendix.

To facilitate the subsequent analysis, suppose that Eq (4.2) admits a positive real root ω_0 , which implies that the characteristic Eq (3.6) has a pair of purely imaginary eigenvalues $\pm i\omega_0$. Accordingly, the bifurcation threshold τ corresponding to the stability switch is uniquely determined by ω_0 and can be expressed as follows:

$$\tau_0 = \frac{\arccos\left(\frac{m_8\omega^8 + m_6\omega^6 + m_4\omega^4 + m_2\omega^2 - m_0}{n_6\omega^6 + n_4\omega^4 + n_2\omega^2 + n_0}\right)}{\omega_0}. \quad (4.5)$$

Thus, by differentiating the characteristic Eq (3.6) with respect to τ , we obtain

$$\left[\frac{d\lambda}{d\tau}\right]^{-1} = \frac{Q(\lambda)}{V(\lambda)} - \frac{\tau}{\lambda}, \quad (4.6)$$

where

$$Q_\lambda = 3b_3\lambda^2 + 2b_2\lambda + b_1 + (6\lambda^5 + 5a_5\lambda^4 + 4a_4\lambda^3 + 3a_3\lambda^2 + 2a_2\lambda + a_1)e^{\lambda\tau},$$

$$V_\lambda = b_3\lambda^4 + b_2\lambda^3 + b_1\lambda^2 + b_0\lambda.$$

We define

$$\operatorname{Re}\left[\left(\frac{d\lambda}{d\tau}\right)^{-1}\right]_{\tau=\tau_0} = \frac{\operatorname{Im}(Q)\operatorname{Im}(V) + \operatorname{Re}(V)\operatorname{Re}(Q)}{\operatorname{Im}(V)^2 + \operatorname{Re}(V)^2}.$$

Provided that $\operatorname{Im}(Q)\operatorname{Im}(V) + \operatorname{Re}(V)\operatorname{Re}(Q) \neq 0$, the transversality condition $\operatorname{Re}\left[\left(\frac{d\lambda}{d\tau}\right)^{-1}\right]_{\tau=\tau_0} \neq 0$ is satisfied. Combining this with the preceding analysis and the classical Hopf bifurcation theory [52], we obtain the following result.

Theorem 5. Assume that the transversality condition $\operatorname{Re}\left[\left(\frac{d\lambda}{d\tau}\right)^{-1}\right]_{\tau=\tau_0} \neq 0$ holds. Let $\epsilon_* = (S^*, E^*, I^*, H^*, R^*, M^*)$ denote the EE of the model in Eq (2.2). Then the following statements hold:

- (i) If $\tau \in [0, \tau_0)$, then ϵ_* is locally asymptotically stable;
- (ii) If $\tau = \tau_0$, then the model in Eq (2.2) undergoes a Hopf bifurcation at ϵ_* , and the transversality condition is satisfied.

Here, the critical delay τ_0 is determined by Eq (4.5).

5. Numerical simulation

In this section, numerical simulations are performed to corroborate and complement the dynamical properties of the model in Eq (2.2). First, the influences of pivotal parameters on the system's evolution are systematically investigated, with particular emphasis on their regulatory effects on the dynamics of susceptible and infection-related populations. Second, bifurcation analysis is used as a fundamental tool to characterize qualitative transitions in the dynamical structure induced by parameter variations, thereby revealing the emergence of periodic orbits, limit cycles, and more intricate behaviors such as chaos [53]. The criteria for the occurrence of a Hopf bifurcation have been rigorously derived in the preceding theoretical analysis.

Given these results, MATLAB R2017b is used to provide numerical visualizations, offering an intuitive illustration of the system's dynamics before and after the bifurcation. By constructing the corresponding Hopf bifurcation diagrams, the transition from steady states to periodic oscillatory solutions is clearly captured, demonstrating a high level of consistency between the theoretical predictions and numerical simulations. These results not only further support the validity of the analytical findings, but also provide strong numerical evidence for the parameter-driven oscillatory mechanisms inherent in the model. We set the initial compartment populations $S_0 = 100$, $E_0 = 10$, $I_0 = 5$, $H_0 = 2$, $R_0 = 0$, and $M_0 = 1$ to capture the epidemic's dynamics at the early stage of disease emergence. Every initial configuration complies with fundamental epidemiological rules, and robustness analysis confirms that all biologically admissible initial states yield identical long-run equilibrium properties of the system.

Case 1: Dynamical behaviors of the model in Eq (2.2) under varying hospitalization and isolation rates.

As shown in Figure 5, for all parameter values, the system's solutions converge to the unique EE, implying the global asymptotic stability of this equilibrium. Further analysis indicates that the parameter σ critically modulates the epidemic dynamics: As σ increases, the growth rate during the outbreak phase is markedly elevated; the peak levels of the latent (E), infectious (I), and hospitalized (H) compartments increase significantly; and the susceptible (S) compartment exhibits a more pronounced decline.

At the EE, σ is negatively correlated with the steady-state level S^* , but positively correlated with E^* , I^* , H^* , R^* , and M^* . This indicates that σ directly governs the long-term prevalence of the disease, as well as the associated healthcare and mortality burdens. These findings suggest that reducing σ constitutes a key intervention strategy for mitigating disease transmission and alleviating the system's burden, thereby providing a rigorous theoretical basis for the design of effective epidemic control measures.

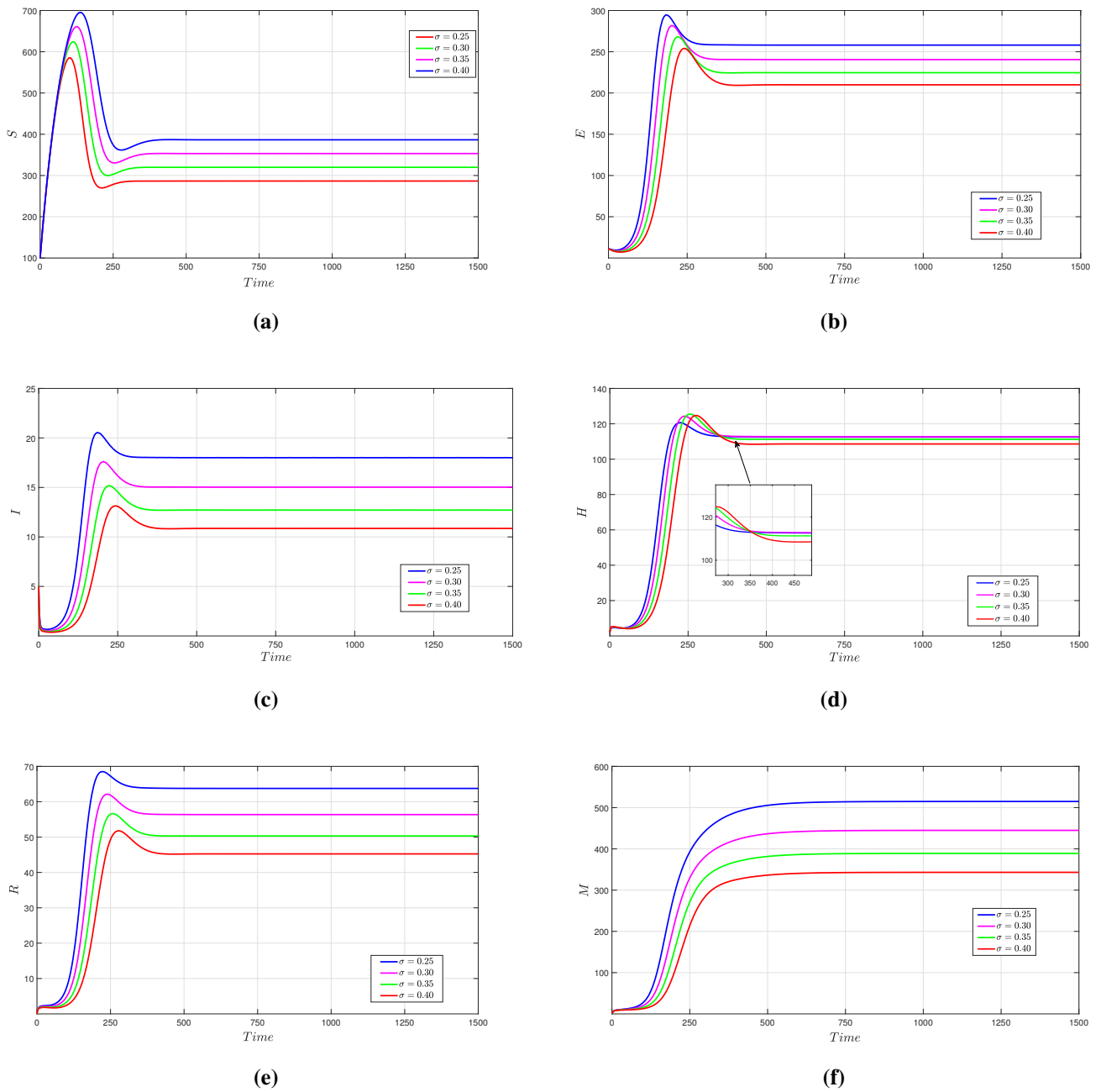


Figure 5. Numerical simulations of the model in Eq (2.2) for different values of the saturation parameter k when the basic reproduction number satisfies $R_0 > 1$. Parameter values: $\Pi = 10$, $\beta_1 = 0.0026$, $\beta_2 = 0.0012$, $\eta_e = 0.03$, $\mu_1 = 0.02$, $\mu_2 = 0.02$, $r_1 = 0.15$, $r_2 = 0.01$, $\mu = 0.01$, $k = 2$, $\xi_1 = 0.2$, $\xi_2 = 0.01$, $\delta = 0.05$, and $\tau = 12$. (a) Changes in the infected population $S(t)$. (b) Changes in the infected population $E(t)$. (c) Changes in the infected population $I(t)$. (d) Changes in the infected population $H(t)$. (e) Changes in the infected population $R(t)$. (f) Changes in the infected population $M(t)$.

Case 2: Dynamic behavior analysis of the model in Eq (2.2) under different time delays

Figure 6 demonstrates that, for $R_0 < 1$, the time delay τ exerts a pronounced regulatory influence on the transient dynamics of the model. As τ increases, the susceptible population $S(t)$ exhibits progressively stronger oscillations with deeper declines (Figure 6(a)); the exposed class $E(t)$ shows a substantial increase in peak magnitude accompanied by a delayed peak occurrence (Figure 6(b)); both the infected population $I(t)$ and hospitalized individuals $H(t)$ display synchronized amplification of the peak sizes together with delayed outbreak timing (Figure 6(c),(d)); the recovered class $R(t)$ fluctuates relatively mildly (Figure 6(e)) but exhibits a clear phase-lagged response relative to the infectious compartments; and the media coverage variable $M(t)$ exhibits the most prominent oscillatory amplification, with its amplitude increasing notably with τ (Figure 6(f)). Overall, larger time delays lead to more pronounced transient oscillations and substantially prolonged adjustment periods, highlighting the strong modulatory role of delay-induced memory effects on the system's transient dynamics.

Furthermore, all numerical trajectories converge to the same EE under different values of τ , indicating that time delay does not alter the global asymptotic stability of the model but primarily reshapes its transient evolution. The compartmental variables exhibit a clear time-ordered coupling along the transmission chain, with the media coverage variable playing a key driving role in the emergence of oscillatory behavior. Therefore, when $R_0 < 1$, although time delay does not affect the long-term epidemiological outcome, it significantly influences the timing and magnitude of the epidemic's peaks; larger delays intensify transient oscillations and prolong the convergence process, thereby increasing the overall uncertainty in the system's dynamic evolution.

The numerical simulations in Figure 7 demonstrate that, when $R_0 > 1$, time delay τ can significantly alter the transient dynamics of the model. As τ increases, $S(t)$, $E(t)$, $I(t)$, $H(t)$, $R(t)$, and $M(t)$ all exhibit progressively enhanced oscillation amplitudes and elongated periods during the transient phase. Specifically, $E(t)$, $I(t)$, and $H(t)$ exhibit significantly elevated peak magnitudes accompanied by progressively delayed occurrence times, whereas $S(t)$ displays more pronounced periodic oscillations (Figure 7(a)). $R(t)$ shows a clear phase-lagged response (Figure 7(e)); the media coverage variable $M(t)$ exhibits the most pronounced oscillatory behavior, maintaining a strong coupling with the infectious-related compartments (Figure 7(f)). Overall, all compartments exhibit a well-defined temporal structure along the transmission chain. Despite these transient oscillations, all trajectories converge to the same equilibrium (DFE or EE depending on the parameter values), suggesting that time delay does not affect the global asymptotic stability when $R_0 > 1$.

Further analysis reveals that as the time delay increases, the model gradually loses stability at the EE and transitions into sustained periodic oscillations, indicating that the time delay induces a Hopf bifurcation through the introduction of a delayed feedback mechanism, thereby altering the long-term dynamic behavior of the model. This result highlights the critical role of the time delay in regulating the epidemic's peak magnitude, oscillation intensity, and periodic structure, suggesting that under the condition $R_0 > 1$, the time delay not only influences transient dynamics but may also drive disease transmission to exhibit persistent periodic fluctuations.

In summary, R_0 determines the epidemic threshold and the final outcome, whereas time delay primarily modulates the evolutionary pathway of the system. When $R_0 < 1$, the delay influences only the transient dynamics by amplifying oscillatory amplitudes and prolonging the convergence process, without affecting the global asymptotic stability. When $R_0 > 1$, however, increasing τ may gradually

destabilize the EE and trigger a Hopf bifurcation, leading to sustained periodic oscillations. Thus, time delay serves as a bifurcation parameter in high-transmission regimes, fundamentally reshaping the long-term dynamical structure of the model.

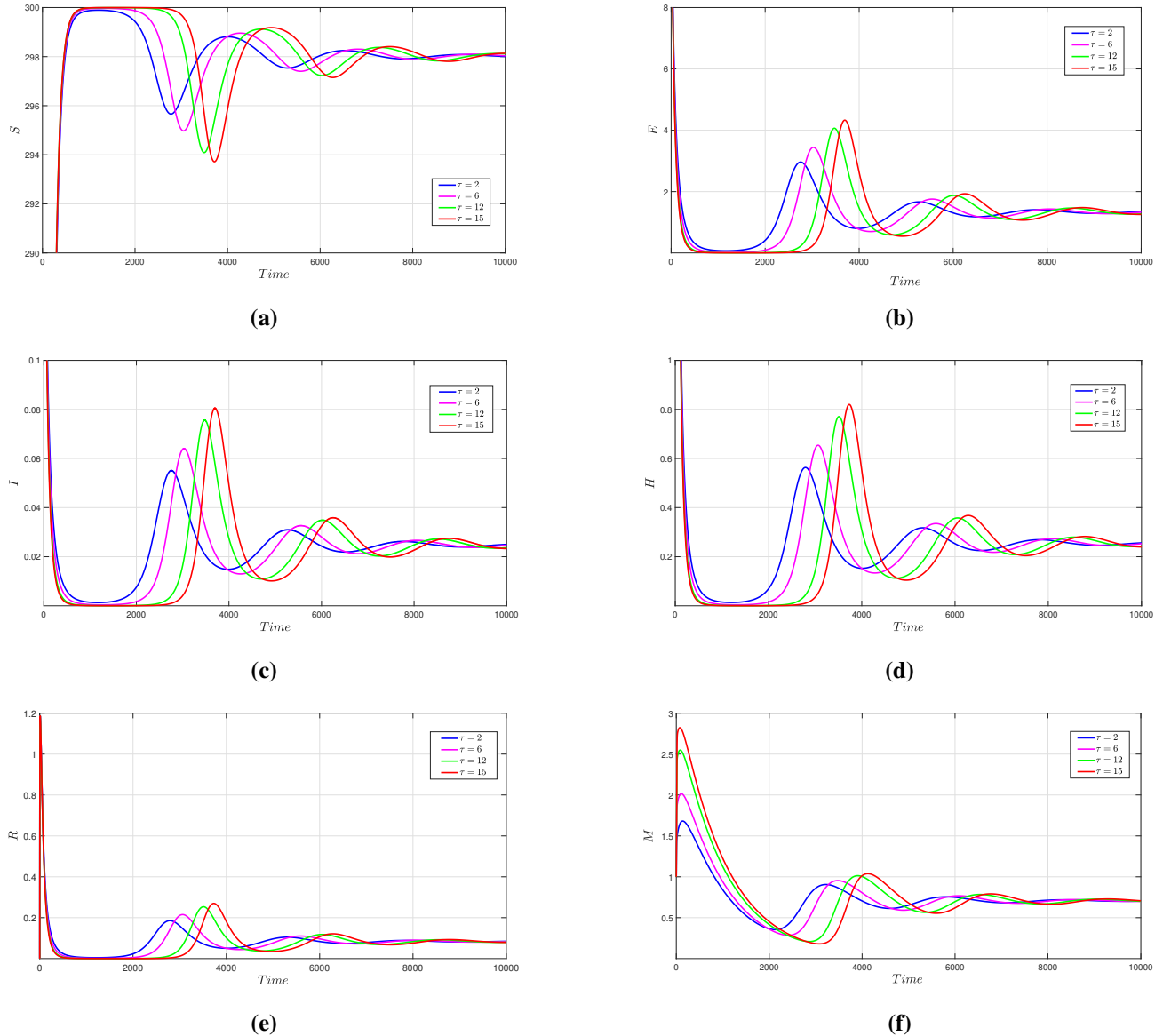


Figure 6. Numerical simulations of the model in Eq (2.2) for different values of the saturation parameter k when the basic reproduction number satisfies $R_0 < 1$. Parameter values: $\Pi = 2$, $\beta_1 = 0.00025$, $\beta_2 = 0.0033$, $\eta_e = 0.02$, $\mu_1 = 0.02$, $\mu_2 = 0.003$, $r_1 = 0.085$, $r_2 = 0.0036$, $\sigma = 0.1$, $\mu = 0.003$, $\xi_1 = 0.05$, $\xi_2 = 0.01$, $\delta = 0.05$, and time delay $\tau = 12$. (a) $S(t)$; (b) $E(t)$; (c) $I(t)$; (d) $H(t)$; (e) $R(t)$; (f) $M(t)$.

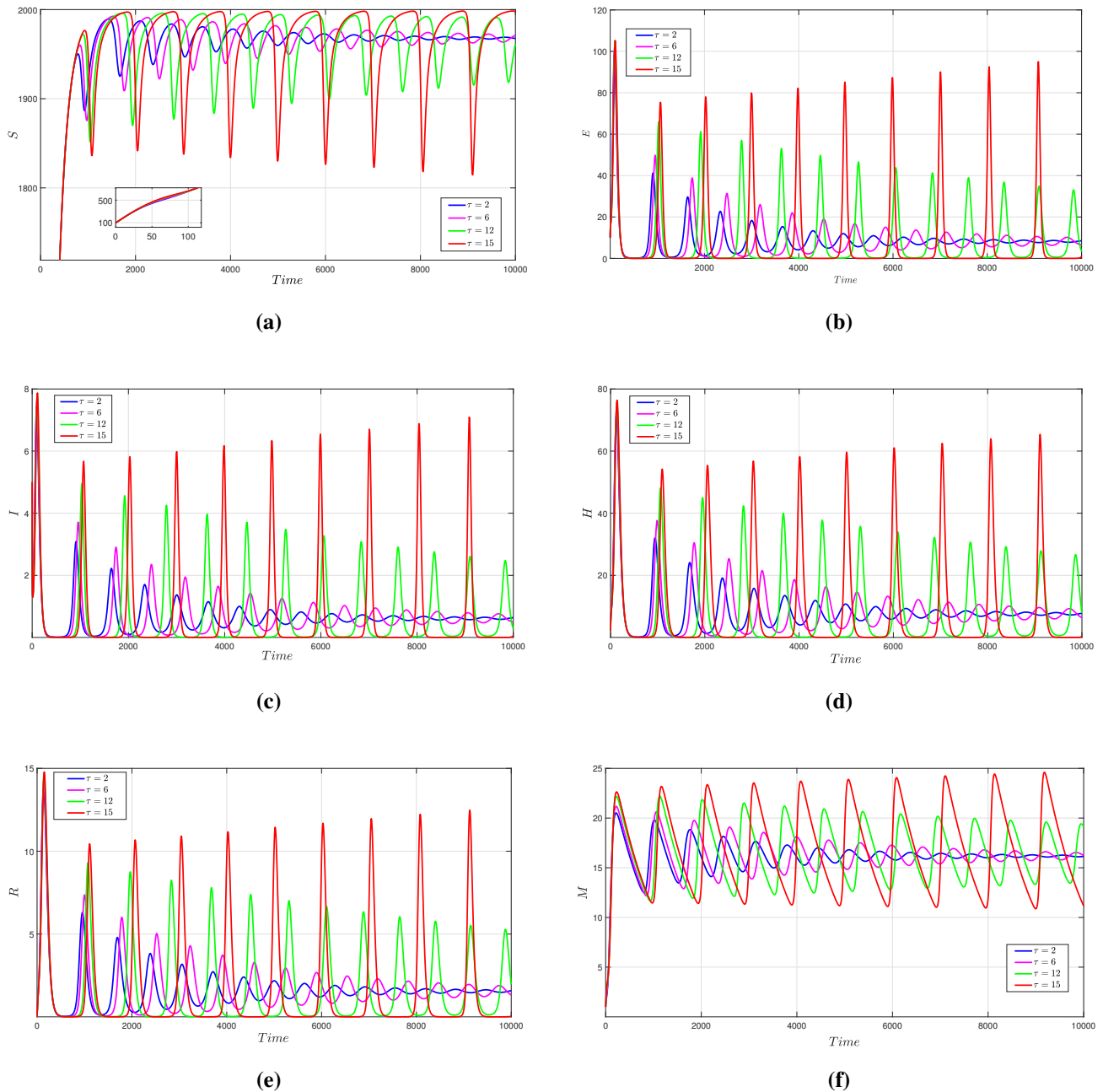


Figure 7. Numerical simulations of the model in Eq (2.2) for different values of the time delay τ when the basic reproduction number satisfies $R_0 > 1$. Parameter values: $\Pi = 10$, $\beta_1 = 0.0069$, $\beta_2 = 0.0039$, $\eta_e = 0.024$, $\mu_1 = 0.0002$, $\mu_2 = 0.0092$, $r_1 = 0.015$, $r_2 = 0.01$, $\sigma = 0.3$, $\mu = 0.005$, $k = 0.52$, $\xi_1 = 0.02$, $\xi_2 = 0.001$, and $\delta = 0.05$. (a) $S(t)$; (b) $E(t)$; (c) $I(t)$; (d) $H(t)$; (e) $R(t)$; (f) $M(t)$.

Case 3: Periodic oscillatory dynamics of the model in Eq (2.2) under different time delays.

Through systematic parameter regulation of the model in Eq (2.2) combined with numerical simulations, we further reveal how time delay simultaneously regulates both the steady-state behavior and periodic responses of the model. This conclusion is supported by qualitative and quantitative analyses of the numerical results. This study not only enriches the theoretical framework of infectious disease dynamics, but also contributes to the accurate modeling of disease transmission and provides a theoretical foundation for the development of effective control interventions.

For the model in Eq (2.2), increasing the time delay τ induces a pronounced modification in the dynamic regime of the model. When $\tau = 13$, the EE remains asymptotically stable (see Figure 8), and all state variables converge to it in a spiral manner (see Figures 11(a) and 12(a)).

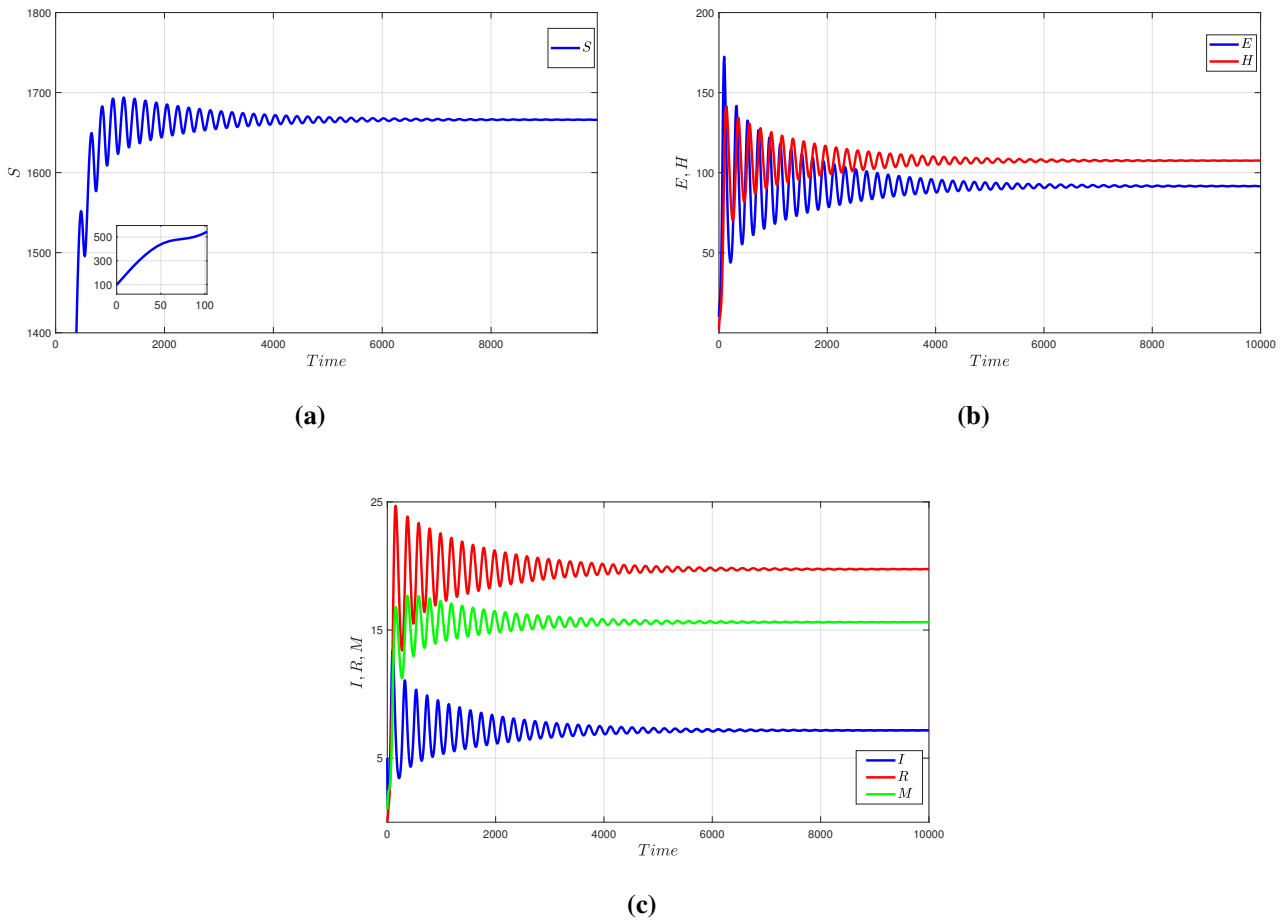


Figure 8. For the model in Eq (2.2), the time-series profiles obtained with $\tau = 13 < \tau_0$ under the parameter set $\Pi = 10$, $\beta_1 = 0.0069$, $\beta_2 = 0.0039$, $\eta_e = 0.024$, $\mu_1 = 0.02$, $\mu_2 = 0.01$, $r_1 = 0.0015$, $r_2 = 0.34$, $\sigma = 0.3$, $\mu = 0.005$, $k = 0.52$, $\xi_1 = 0.02$, $\xi_2 = 0.01$, and $\delta = 0.05$ are illustrated. (a) Susceptible population $S(t)$. (b) Exposed population $E(t)$ and hospitalized population $H(t)$. (c) Infected population $I(t)$, recovered population $R(t)$, and media coverage variable $M(t)$.

As τ is further increased, the equilibrium gradually loses its stability, and the attractor of the model evolves from a stable equilibrium point into a limit cycle (see Figure 9). During this process, the

emerging limit cycle exhibits varying oscillation amplitudes as the parameter varies. When τ increases from 13 to 14.5, approaching the critical value τ_0 , the model undergoes a Hopf bifurcation, and stability shifts from the equilibrium to a periodic orbit (see Figures 11(b) and 12(b)). Further analysis shows that in a neighborhood of τ_0 , the equilibrium loses stability and sustained periodic oscillations arise, eventually converging to a stable limit cycle.

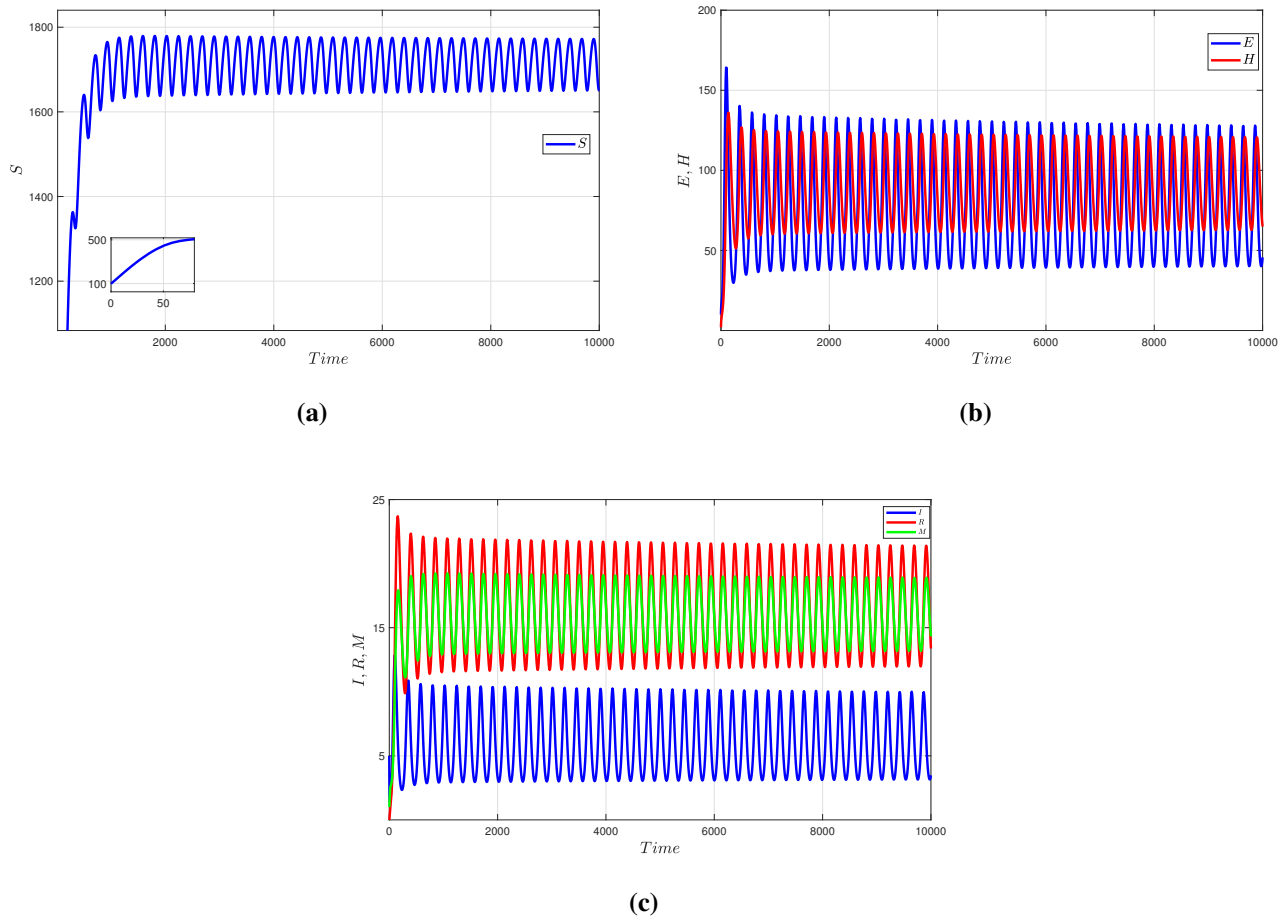


Figure 9. Dynamical behavior of the time series of the model in (2.2) at $\tau = 14.5 = \tau_0$, while all other parameters are the same as those in Figure 8. (a) Susceptible population $S(t)$; (b) exposed population $E(t)$ and hospitalized population $H(t)$; (c) infected population $I(t)$, recovered population $R(t)$, and media coverage variable $M(t)$.

When $\tau = 16$ (i.e., $\tau > \tau_0$), the oscillation amplitude increases further, and the equilibrium completely loses stability (see Figure 10). In this regime, the system's trajectories gradually diverge from the equilibrium over time (see Figures 11(c) and 12(c)), indicating the loss of stability.

This Hopf bifurcation phenomenon plays a significant role in infectious disease dynamics. The resulting sustained periodic oscillations suggest that the disease may exhibit recurrent outbreaks. The three groups of numerical results provide strong evidence for the critical influence of Hopf bifurcation in driving the emergence of periodic transmission patterns. In particular, the time delay not only serves as a key control parameter governing dynamic bifurcations in the model but also directly determines the oscillatory patterns of the disease's spread. From an epidemiological perspective, these findings

highlight the importance of implementing timely and effective intervention strategies, especially near epidemic peaks, to mitigate the oscillatory propagation effects induced by time delay, thereby achieving effective control of disease transmission.

Even slight variations in these quantities can drive the model in Eq (2.2) from stable periodic oscillations to more intricate dynamical regimes, thereby reflecting the inherent nonlinearity and complexity of infectious disease transmission. From a disease control perspective, it is essential to account for the combined effects of nonlinear interactions, behavioral feedback, and environmental influences, which provide a theoretical basis for improving the foresight and effectiveness of public health intervention strategies.

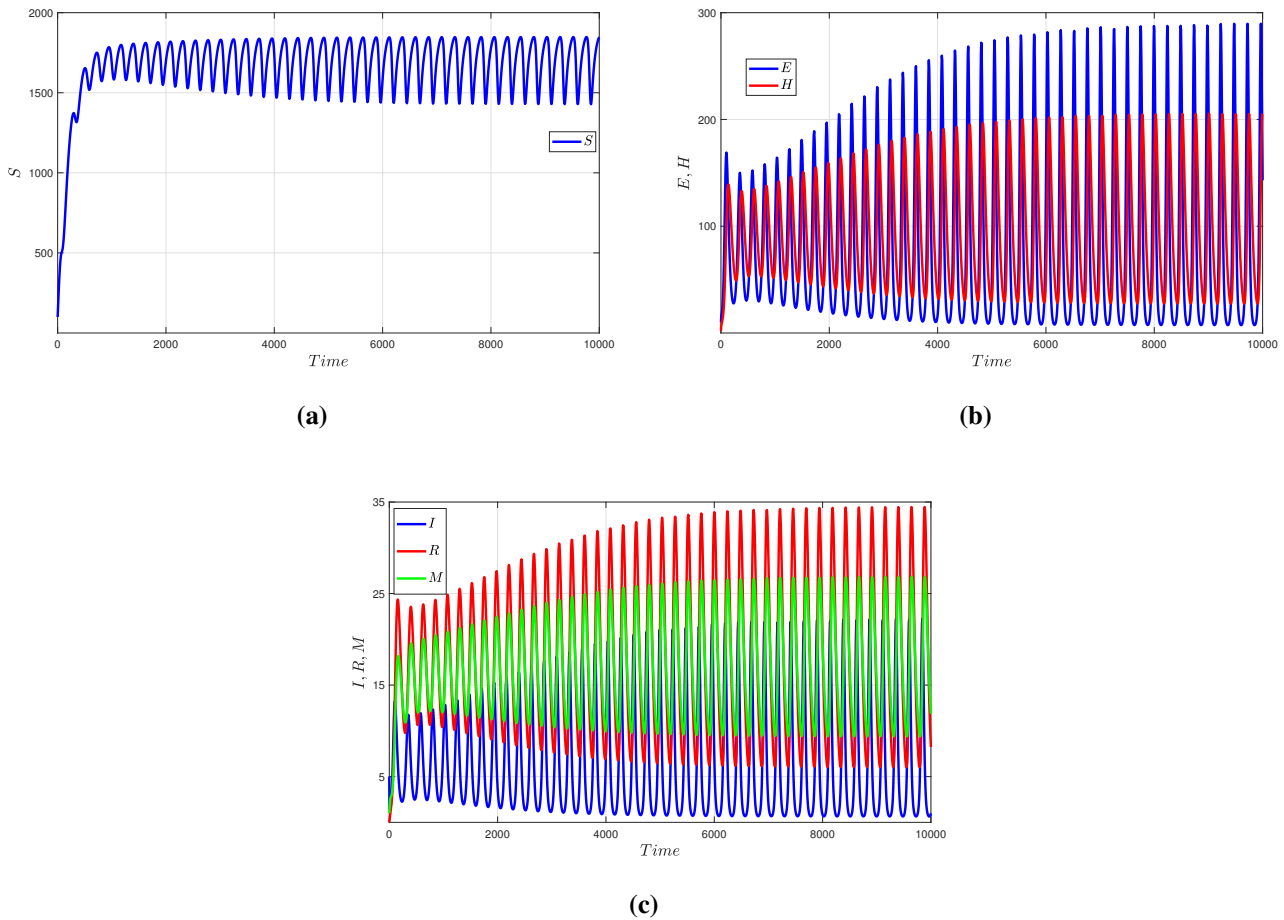


Figure 10. Dynamical behavior of the time series of the model (2.2) at $\tau = 16 < \tau_0$, while all other parameters are the same as those in Figure 8. (a) Susceptible population $S(t)$; (b) exposed population $E(t)$ and hospitalized population $H(t)$; (c) infected population $I(t)$, recovered population $R(t)$, and media coverage variable $M(t)$.

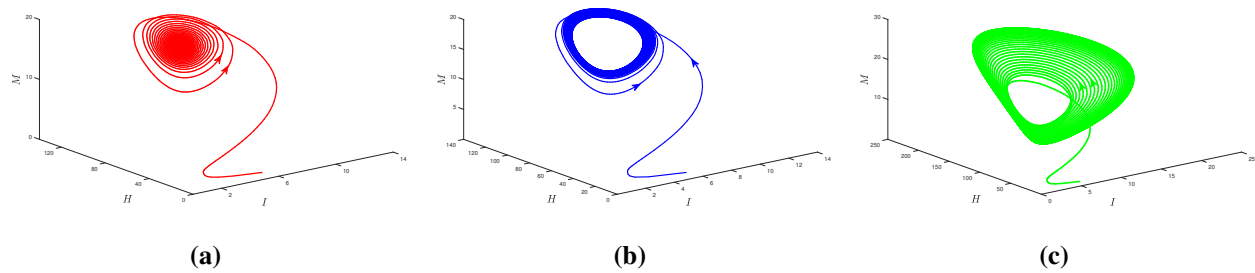


Figure 11. Attractor images of the state variables, infectious individuals I , hospitalized individuals H , and media coverage M , in the model in Eq (2.2). The parameter values are the same as those in Figure 8. (a) Time delay $\tau = 13$ (red line); (b) time delay $\tau = 14.5$ (blue line); (c) time delay $\tau = 16$ (green line).

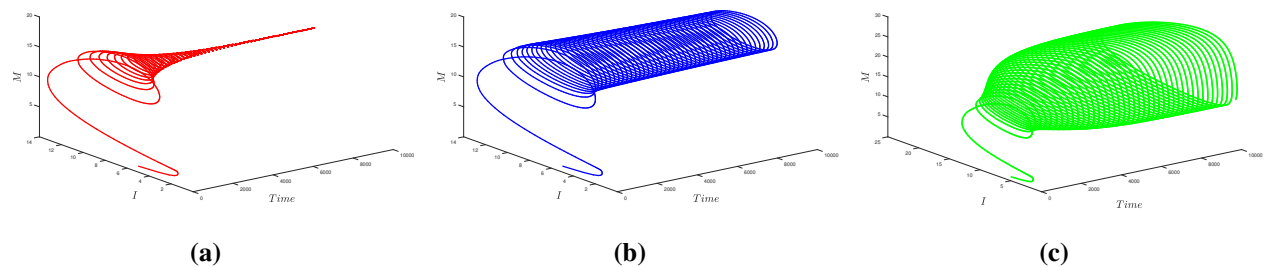


Figure 12. The projection of the state variables of the model in Eq (2.2) in the I – M plane over time. The parameter values are the same as those in Figure 8. (a) Time delay $\tau = 13$ (red line); (b) time delay $\tau = 14.5$ (blue line); (c) time delay $\tau = 16$ (green line).

Case 4: Hopf bifurcation diagram analysis with τ as the bifurcation parameter.

To provide a comprehensive characterization of the dynamics of the proposed delayed epidemic model, Figure 13 depicts the system's trajectories in the SI , SH , and IM phase planes over the interval $\tau \in [0, 20]$, where the time delay τ is treated as an additional dimension. The figure reveals a Hopf bifurcation phenomenon: For $\tau < \tau_0$, the trajectories approach the stable the EE ϵ_* , whereas for $\tau > \tau_0$, EE ϵ_* becomes unstable and the trajectories evolve toward a stable limit cycle, indicating a transition from steady-state behavior to sustained periodic oscillations.

As illustrated in Figure 14(a), when $\tau < \tau_0$, the maximum and minimum values of the susceptible population S coincide, implying convergence of the model to a stable equilibrium, namely the EE, which represents a steady transmission state. As τ approaches the critical value τ_0 , the upper branch increases rapidly while the lower branch gradually declines, signaling the emergence of a Hopf bifurcation. For $\tau > \tau_0$, the equilibrium becomes unstable, and the solution's trajectories spread into a periodic oscillatory pattern (indicated by the purple scattered points), with the oscillation amplitude increasing markedly as τ continues to grow. From a biological perspective, this suggests that time delay exerts a pronounced regulatory effect on the susceptible population, driving a transition from a single steady level to sustained oscillations, thereby reflecting a disruption of the dynamic balance underlying the transmission process.

As shown in Figure 14(b), the response of the infected population I to the time delay parameter τ exhibits pronounced nonlinear characteristics. When $\tau < \tau_0$, the infected population stabilizes at a low endemic level, indicating that the system resides in a controlled transmission regime. As τ approaches the critical value τ_0 , the system progressively loses stability, and a Hopf bifurcation arises, leading to a transition of I from a steady state to periodic oscillations. With further increases in τ , the trajectories of I display sustained oscillations and even quasi-periodic fluctuations, suggesting recurrent outbreak dynamics in the transmission process. These results underscore the pivotal role of time delay in shaping the dynamics of the infected population and identify it as a key mechanism underlying the periodic resurgence of epidemics.

As shown in Figure 14(c), when $\tau < \tau_0$, the media variable M converges to a stable equilibrium, indicating that information dissemination is well-coupled with the epidemic's dynamics, and the model remains in a state of stable regulation. When τ exceeds the critical threshold, M loses stability and exhibits sustained oscillations, with the oscillation amplitude increasing significantly as τ further increases. This result indicates that time delay not only disrupts the influence of infected individuals on media coverage, but also induces oscillatory instability at the system level through the feedback regulation of the recovery/removal process mediated by M . Acting as a delayed coupler, M further amplifies the nonlinear response of the recovery process through its dynamic oscillations, ultimately driving the overall model into long-term periodic behavior. These results reveal the mechanism by which time delay indirectly reshapes an intervention's effectiveness through the media feedback pathway, emphasizing that information-induced behavioral rhythm changes must be accounted for when modeling recovery and immune processes.

In summary, the model exhibits a critical threshold τ_0 : When $\tau < \tau_0$, all state variables converge to a stable equilibrium, and disease transmission remains in a stable endemic state; when $\tau > \tau_0$, the model undergoes a Hopf bifurcation, the equilibrium loses its stability, and S , I , and M sequentially transition to periodic oscillations and even complex fluctuations. Among these variables, the media variable M acts as a “delayed coupler”, whose oscillations amplify the nonlinear responses of the recovery and immune processes through feedback mechanisms, ultimately driving the model into long-term periodic behavior. These results reveal the key mechanism by which time delay reshapes the epidemic's rhythms through information delays, providing a mathematical basis for real-time feedback-based intervention strategies.

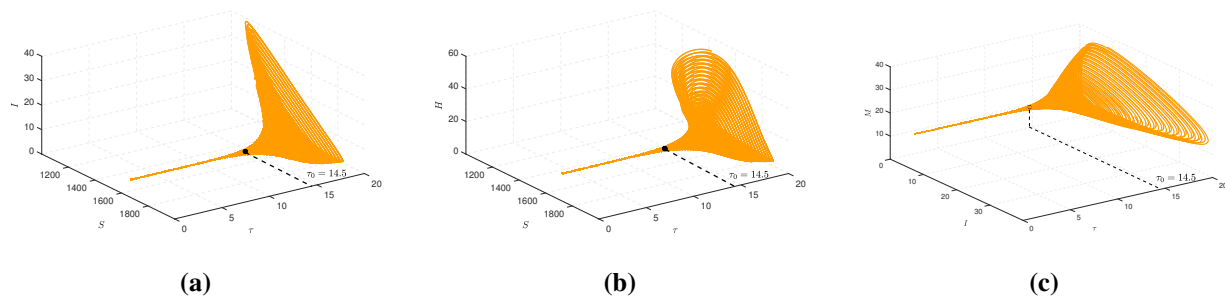


Figure 13. Three-dimensional bifurcation diagrams for the susceptible population S , the infected population I , the hospitalized population H , and the media perception variable M with the time delay τ as the bifurcation parameter. All remaining parameter values are consistent with those adopted in Figure 8. (a) (S, τ, I) bifurcation surface; (b) (S, τ, H) bifurcation surface; (c) (I, τ, M) bifurcation surface.

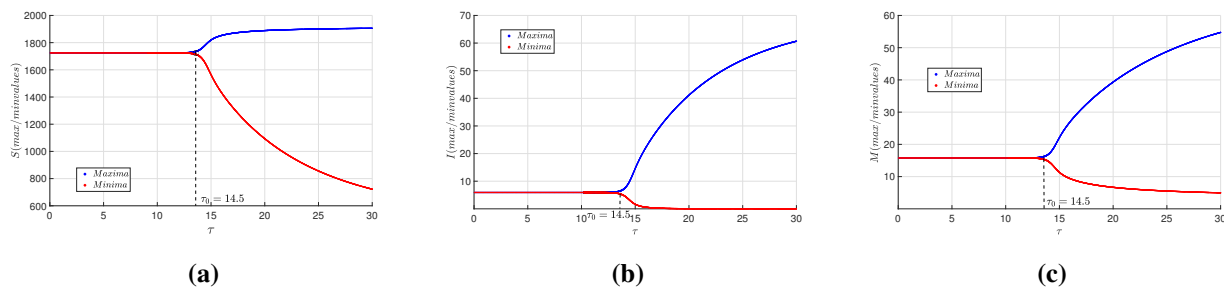


Figure 14. Hopf bifurcation diagrams of S , I , and M with τ as the bifurcation parameter.

6. Conclusions

Infectious diseases continue to exert substantial pressure on public health systems worldwide. Notably, in the early phases of emerging and re-emerging infections, the transmission dynamics are characterized by pronounced uncertainty. In this context, media reporting and delays in information dissemination can markedly influence individuals' behavior and consequently reshape the course of the disease's spread. Conversely, inadequate media coverage or delayed public responses may further accelerate the epidemic's progression, and in severe cases, outbreaks can rapidly escalate into large-scale epidemics. Therefore, it is essential to systematically investigate epidemic models incorporating both media effects and time-delay mechanisms in order to achieve reliable prediction and effective control.

In this work, an *SEIHRM* model is formulated to characterize the effects of media reporting and time delay on disease transmission. Extending the classical *SEIR* framework, a media-related variable $M(t)$ and a hospitalized compartment $H(t)$ are incorporated to represent the intensity of information dissemination and the regulatory influence of isolation measures, respectively. In addition, a time-delay term is included to capture the intrinsic lag between the spread of information and behavioral response. This modeling framework offers a more realistic representation of the dynamic interplay between media feedback and an epidemic's evolution, thereby enabling a systematic investigation of the joint influence of media coverage, isolation strategies, and time-delay effects on infectious disease dynamics.

First, we conduct a comprehensive analysis of the dynamical properties of solutions to the model in Eq (2.2), establishing the existence, uniqueness, positivity, and boundedness of the solutions, which guarantees that the model is biologically well-posed. Subsequently, we investigate the existence and stability of equilibria of the model in Eq (2.2), and derive the R_0 via the next-generation matrix approach. It follows that the disease dies out when $R_0 < 1$, whereas an endemic state persists when $R_0 > 1$. The numerical results (see Figure 2) further indicate that R_0 increases with Π and β_1 , but decreases with the natural death rate μ . In particular, Π and β_1 are key determinants of the epidemic threshold. These findings suggest that reducing population inflows and transmission intensity may serve as effective strategies for controlling R_0 .

We begin by examining the local stability properties of both the DFE and EE of the model in Eq (2.2), and subsequently consider τ as the bifurcation parameter to explore its influence on the system's dynamics. The results reveal that disease transmission is governed not only by the intrinsic biological characteristics of the pathogen (e.g., R_0), but also by feedback mechanisms associated with media reporting, public behavioral responses, and the healthcare system's capacity. The nonlinear interaction among these factors determines the long-term evolution of the model. Numerical simulations under varying values of τ (see Figures 8–12) demonstrate that τ acts as a critical parameter regulating the bifurcation structure of the model. As τ varies, the model evolves across different dynamical regimes, including a stable equilibrium and periodic oscillatory states, and undergoes a Hopf bifurcation in the vicinity of a critical value τ_0 , giving rise to oscillatory epidemic dynamics. These results indicate that, in epidemic models incorporating delayed media effects, time delay can fundamentally reshape the temporal patterns of disease transmission, inducing oscillatory propagation and phase-dependent outbreak dynamics. The proposed mechanism effectively captures the lag between information reception and behavioral response, highlighting the importance of prompt and accurate information dissemination for effective epidemic control.

Furthermore, numerical experiments are conducted to corroborate the theoretical findings. The results demonstrate that, under varying values of k , the model exhibits distinct outcomes corresponding to the disease's extinction or persistence (see Figures 3 and 4), in agreement with the analytical predictions. In addition, Figure 5 illustrates that increasing the hospital-based isolation rate σ significantly reduces the levels of E , I , and R , highlighting the effectiveness of isolation measures in suppressing disease transmission. Moreover, the results presented in Figures 6 and 7 indicate that R_0 determines the epidemic threshold and asymptotic system behavior, whereas the time delay τ primarily regulates the dynamic evolution. Specifically, when $R_0 < 1$, increasing τ amplifies transient oscillations and slows convergence without affecting the asymptotic stability. In contrast, for $R_0 > 1$, the delay can trigger a Hopf bifurcation, leading the system from a stable equilibrium into sustained periodic oscillations, thereby modifying its long-term dynamics.

Finally, the results from Figure 13 together with the bifurcation analysis of all state variables indicate that the time delay τ is a key parameter governing the dynamic transition of the model. When $\tau < \tau_0$, the model converges to a stable EE, where the maximum and minimum values of all state variables coincide, indicating a steady endemic state. As τ exceeds τ_0 , the system loses stability and undergoes a Hopf bifurcation. The trajectory transitions from a stable EE to a stable limit cycle, indicating the emergence of periodic oscillations. With a further increase in τ , all state variables exhibit oscillations of increasing amplitude. The infectious population I shows particularly strong oscillations. Meanwhile, the media variable M amplifies the nonlinear response through feedback

effects, further enhancing the oscillatory dynamics. These results collectively demonstrate that time delay, by introducing feedback, fundamentally alters the system's stability and acts as a key mechanism inducing periodic epidemic dynamics.

It should be noted that, in this model, the discrete delay τ is introduced only in the infectious compartment $I(t)$, without incorporating a corresponding structure in the exposed compartment $E(t)$, which constitutes a simplification of the actual media reporting system. In reality, the information sources driving public risk perception are not limited to confirmed cases; exposed individuals can also influence behavioral decisions through various channels. Nevertheless, from a modeling perspective, this simplification represents a trade-off between tractability and mechanistic realism, since introducing delays in both the E and I compartments would lead to a high-dimensional delay system, rendering stability and bifurcation analyses extremely challenging. To preserve tractability, we concentrate the delay in $I(t)$, while retaining the instantaneous input from the latent reservoir through $\xi_1 \eta_e E(t)$, thereby accommodating the essential features of media feedback. Numerical simulations demonstrate that our qualitative conclusions remain robust within a biologically reasonable range of delays, indicating that this simplification is justified and does not compromise the core contributions of this work. Future work may introduce a distributed delay framework to better capture the cumulative effects of multi-source delays.

In addition, the present model assumes a homogeneous recovery and waning immunity process for all recovered individuals, regardless of whether they recovered from a mild infection or after hospitalization. In reality, the duration of protective immunity may differ substantially between these two groups. By aggregating them into a single compartment R with a unified waning rate δ , we have adopted a simplified representation of the immune response. This simplification is justifiable, given that our primary objective is to elucidate the dynamic role of media-induced delayed feedback, rather than to capture the fine-scale immunological heterogeneity. Future work may further incorporate a distributed delay framework to more accurately capture the cumulative effects of multi-source delays, while seeking a more balanced modeling strategy between tractability and biological realism.

Use of AI tools declaration

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

Conflict of interest

The authors declare there is no conflict of interest.

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Author contributions

Chan Ma: Study conceptualization, methodological development, formal analysis, investigation, and preparation of the original manuscript, as well as manuscript review and editing. Yun Zhao: Coordination of the project, conceptual design, investigation, model validation, and manuscript review and editing. Xuewen Tan: Methodological support, investigation, and manuscript review and editing. Wenjie Shi: Project administration, formal analysis, visualization.

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Appendix

Coefficient expressions for the bifurcation analysis

1. Coefficients in Eqs (4.2) and (4.3)

The coefficients appearing in Eqs (4.2) and (4.3) are given as follows:

$$\begin{aligned}
 m_8 &= a_5 b_3 - b_2, \\
 m_6 &= a_4 b_2 - a_5 b_1 - a_3 b_3 + b_0, \\
 m_4 &= a_3 b_1 + a_1 b_3 - a_4 b_0 - a_2 b_2, \\
 m_2 &= a_2 b_0 + a_0 b_2 - a_1 b_1, \\
 m_0 &= -a_0 b_0, \\
 n_6 &= -b_3^2, \\
 n_4 &= 2b_1 b_3 - b_2^2, \\
 n_2 &= 2b_0 b_2 - b_1^2, \\
 n_0 &= -b_0^2, \\
 e_9 &= b_3, \\
 e_7 &= a_3 b_2 - a_4 b_3 - b_1, \\
 e_5 &= a_2 b_3 + a_4 b_1 - a_3 b_2 - a_5 b_0, \\
 e_3 &= a_1 b_2 + a_3 b_0 - a_2 b_1 - a_0 b_3, \\
 e_1 &= a_0 b_1 + a_1 b_0, \\
 h_6 &= -b_3^2, \\
 h_4 &= a_1 b_3 - b_2^2, \\
 h_2 &= a_0 b_0 - b_1^2, \\
 h_0 &= -b_0^2.
 \end{aligned}$$

2. Coefficients of the polynomial equation

The coefficients d_i ($i = 0, 1, \dots, 16$) in Eq (4.4) are given by the following expressions:

$$\begin{aligned}
 d_{15} &= n_6^2 e_9^2, \\
 d_{14} &= 2n_6^2 e_7 e_9 + 2n_4 n_6 e_9^2 + h_6^2 m_8^2, \\
 d_{13} &= n_6^2 (2e_5 e_9 - e_7^2) + 4n_4 n_6 e_7 e_9 + e_9^2 (2n_2 n_6 + n_4^2) + 2h_4 h_6 m_8^2 + 2h_6^2 m_6 m_8, \\
 d_{12} &= n_6^2 (2e_3 e_9 + 2e_5 e_7 - h_6^2) + 2n_4 n_6 (2e_5 e_9 - e_7^2) + 2e_7 e_9 (2n_2 n_6 + n_4^2) + 2e_9^2 (n_0 n_6 + n_2 n_4) \\
 &\quad + 2h_2 h_6 m_8^2 + h_4^2 m_8^2 + 4h_4 h_6 m_6 m_8 + 2h_6^2 m_4 m_8 + h_6^2 m_6^2, \\
 d_{11} &= 2h_0 h_6 m_8^2 + h_2 h_4 + 4h_2 h_6 m_6 m_8 + 2h_4 h_6 m_4 m_8 + 2h_4 h_6 m_6^2 + 2h_6^2 (m_2 m_8 + m_4 m_6) \\
 &\quad + n_6^2 (2e_7 e_9 + e e_9 + e_5^2 - 2h_4 h_6) + 2n_4 n_6 (2e_3 e_9 + 2e_5 e_7 - h_6^2) + e_9^2 (2n_0 n_4 + n_2^2) \\
 &\quad + 4e_7 e_9 (n_0 n_6 + n_2 n_4) + (2n_2 n_6 + n_4^2) (2e_5 e_9 - e_7^2), \\
 d_{10} &= n_6^2 (2e_3 e_5 + 2ee_7 - 2h_2 h_6 - h_4^2) + 2n_4 n_6 (2e_7 e_9 + 2ee_9 + e_5^2 - 2h_4 h_6) \\
 &\quad + 2(n_2 n_6 + n_4^2) (2e_3 e_9 + 2e_5 e_7 - h_6^2) + 2(n_0 n_6 + n_2 n_4) (2e_5 e_9 - e_7^2) \\
 &\quad + 2e_7 e_9 (2n_0 n_4 + n_2^2) + 2n_0 n_2 e_9^2 + 2h_0 h_4 m_8^2 + h_2^2 m_8^2 + 4m_6 m_8 (h_0 h_6 + h_2 h_4) \\
 &\quad + 2h_2 h_6 + h_4^2 (2m_4 m_8 + m_6^2) + 4h_4 h_6 (m_2 m_8 + m_4 m_6) + h_6^2 (2m_2 m_6 - 2m_0 m_8 + m_4^2), \\
 d_9 &= 2h_0 h_2 m_8^2 + 2m_6 m_8 (2h_0 h_4 + h_2^2) + 2(h_0 h_6 + h_2 h_4) (2m_4 m_8 + m_6^2) + 4n_0 n_2 e_7 e_9 \\
 &\quad + 2(2h_2 h_6 + h_4^2) (m_2 m_8 + m_4 m_6) + 2h_4 h_6 (2m_2 m_6 - 2m_0 m_8 + m_4^2) \\
 &\quad + n_6^2 (2ee_5 + e_3^2 - 2h_0 h_6 - h_2 h_4) + n_6^2 e_9^2 + 2n_4 n_6 (2e_3 e_5 + 2ee_7 - 2h_2 h_6 - h_4^2) \\
 &\quad + (2n_2 n_6 + n_4^2) (2e_7 e_9 + 2ee_9 + e_5^2 - 2h_4 h_6) + 2(n_0 n_6 + n_2 n_4) (2e_3 e_9 + 2e_5 e_7 - h_6^2) \\
 &\quad + (2n_0 n_4 - n_2^2) (2e_5 e_9 - e_7^2) + 2h_6^2 (m_2 m_4 - m_0 m_6), \\
 d_8 &= n_6^{12} (2ee_3 - 2h_0 h_4 - h_2^2) + 2n_4 n_6 (2ee_5 + e_3^2 - 2h_0 h_6 - h_2 h_4) + 4h_4 h_6 (m_2 m_4 - m_0 m_6) \\
 &\quad + 2(n_0 n_6 + n_2 n_4) (2e_7 e_9 + 2ee_9 + e_5^2 - 2h_4 h_6) + (2n_0 n_4 + n_2^2) (2e_3 e_9 + 2e_5 e_7 - h_6^2) \\
 &\quad + 2n_0^2 e_7 e_9 + h_6^2 (m_2^2 - 2m_0 m_4) + (2n_2 n_6 + n_4^2) (2e_3 e_5 + 2ee_7 - 2h_2 h_6 - h_4^2) \\
 &\quad + h_0^2 m_8^2 + (2h_0 h_4 + h_2^2) (2m_4 m_8 + m_6^2) + 4(h_0 h_6 + h_2 h_4) (m_2 m_8 + m_4 m_6) \\
 &\quad + (2h_2 h_6 + h_4^2) (2m_2 m_6 - 2m_0 m_8 + m_4^2) + 2n_0 n_2 (2e_5 e_9 - e_7^2) + 4h_0 h_2 m_6 m_8, \\
 d_7 &= 2h_0^2 m_6 m_8 + 2h_0 h_2 (2m_4 m_8 + m_6^2) + 2(2h_0 h_4 + h_2^2) (m_2 m_8 + m_4 m_6) \\
 &\quad + 2(2h_2 h_6 + h_4^2) (m_2 m_4 - m_0 m_6) + 2(h_0 h_6 + h_2 h_4) (2m_2 m_8 - 2m_0 m_8 + m_4^2) \\
 &\quad + 2h_4 h_6 (m_2^2 - 2m_0 m_4) - 2h_6^2 m_0 m_2 + n_6^2 (e^2 - 2h_0 h_2) + 2n_4 n_6 (2ee_3 - 2h_0 h_4 - h_2^2) \\
 &\quad + (2n_2 n_6 + n_4^2) (2ee_5 + e_5^2 - 2h_0 h_6 - h_2 h_4) + 2(n_0 n_6 + n_2 n_4) (2e_3 e_5 + 2ee_7 - 2h_2 h_6 - h_4^2) \\
 &\quad + (2n_0 n_4 + n_2^2) (2e_7 e_9 + 2ee_9 + e_5^2 - 2h_4 h_6) + 2n_0 n_2 (2e_3 e_9 + 2e_5 e_7 - h_6^2) + n_0^2 (2e_5 e_9 - e_7^2), \\
 d_6 &= -n_6^2 h_0^2 + 2n_4 n_6 (e^2 - 2h_0 h_2) + (2n_2 n_6 + n_4^2) (2ee_3 - 2h_0 h_4 - h_2^2) \\
 &\quad + 2(n_0 n_6 + n_2 n_4) (2ee_5 + e_3^2 - 2h_0 h_6 - h_2 h_4) + (2n_0 n_4 + n_2^2) (2e_3 e_5 + 2ee_7 - 2h_2 h_6 - h_4^2)
 \end{aligned}$$

$$\begin{aligned}
& + 2n_0n_2(2e_7e_9 + 2ee_9 + e_5^2 - 2h_4h_6) + n_0^2(2e_3e_9 + 2e_5e_7 - h_6^2) \\
& + h_0^2(2m_4m_8 + m_6^2) + 4h_0h_2(m_2m_8 + m_4m_6) + (2h_0h_4 + h_2^2)(2m_2m_6 - 2m_0m_8 + m_4^2) \\
& + 4(h_0h_6 + h_2h_4)(m_2m_4 - m_0m_6) + (2h_2h_6 + h_4^2)(m_2^2 - 2m_0m_4) - 4h_4h_6m_0m_2 + h_6^2m_0^2, \\
d_5 = & 2h_0^2(m_2m_8 + m_4m_6) + 2h_0h_2(2m_2m_6 - 2m_0m_8 + m_4^2) + 2(2h_0h_4 + h_2^2)(m_2m_4 - m_0m_6) \\
& + 2(h_0h_6 + h_2h_4)(m_2^2 - 2m_0m_4) - 2m_0m_2(2h_2h_6 + h_4^2) + 2h_4h_6m_0^2 \\
& - 2n_4n_6h_0^2 + (2n_2n_6 + n_4^2)(e^2 - 2h_0h_2) + 2(n_0n_6 + n_2n_4)(2ee_3 - 2h_0h_4 - h_2^2) \\
& + (2n_0n_4 + n_2^2)(2ee_5 + e_3^2 - 2h_0h_6 - h_2h_4) + 2n_0n_2(2e_3e_5 + 2ee_7 - 2h_2h_6 - h_4^2) \\
& + n_0^2(2e_7e_9 + 2ee_9 + e_5^2 - 2h_4h_6), \\
d_4 = & h_0^2(2m_2m_6 - 2m_0m_8 + m_4^2) + 4h_0h_2(m_2m_4 - m_0m_6) + (2h_0h_4 + h_2^2)(m_2^2 - 2m_0m_4) \\
& - 4m_0m_2(h_0h_6 + h_2h_4) + m_0^2(2h_2h_6 + h_4^2) - h_0^2(2n_2n_6 + n_4^2) \\
& + 2(n_0n_6 + n_2n_4)(e^2 - 2h_0h_2) + (2n_0n_4 + n_2^2)(2ee_3 - 2h_0h_4 - h_2^2) \\
& + 2n_0n_2(2ee_5 + e_3^2 - 2h_0h_6 - h_2h_4) + n_2^2(2e_3e_5 + 2ee_7 - 2h_2h_6 - h_4^2), \\
d_3 = & 2h_0^2(m_2m_4 - m_0m_6) + 2h_0h_2(m_2^2 - 2m_0m_4) - 2m_0m_2(2h_0h_4 + h_2^2) + 2m_0^2(h_0h_6 + h_2h_4) \\
& - 2h_0^2(n_0n_6 + n_2n_4) + (2n_0n_4 + n_2^2)(e^2 - 2h_0h_2) + 2n_0n_2(2ee_3 - 2h_0h_4 - h_2^2) \\
& + n_0^2(2ee_5 + e_3^2 - 2h_0h_6 - h_2h_4), \\
d_2 = & h_0^2(m_2^2 - 2m_0m_4) - 4h_0h_2m_0m_2 + m_0^2(2h_0h_4 + h_2^2) - 2h_0^2(2n_0n_4 + n_2^2) \\
& + 2n_0n_2(e^2 - 2h_0h_2) + n_0^{12}(2ee_3 - 2h_0h_4 - h_2^2), \\
d_1 = & 2h_0h_2m_0^2 - 2h_0^2m_0m_2 - 2n_0n_2h_0^2 + n_0^2(e^2 - 2h_0h_2), \\
d_0 = & h_0^2m_0^2 - n_0^2h_0^2.
\end{aligned}$$



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