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*Research article*

## **A mathematical model of TB transmission dynamics incorporating delays in funding and treatment programs**

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**Abstract:** Tuberculosis (TB) remains a persistent global health concern, particularly in high-burden countries where delays in funding allocation and treatment initiation hinder control efforts. In this study, we introduced a delay-differential TB transmission model that incorporated two critical delays: The funding response delay ( $\tau_1$ ) and the treatment initiation delay ( $\tau_2$ ). The model was calibrated using South African TB incidence data from 2000–2023 to assess how these delays influence disease dynamics and system stability. Analytical results revealed that short delays lead the system toward a stable endemic equilibrium, while exceeding critical thresholds triggers a Hopf bifurcation, resulting in oscillatory or recurrent outbreaks. The treatment delay ( $\tau_2$ ) exhibited a stronger destabilizing impact than the funding delay ( $\tau_1$ ), underscoring the importance of timely diagnosis and treatment initiation in maintaining control. Sensitivity analysis identified prevention efficiency ( $\alpha$ ), treatment rate ( $\gamma$ ), and funding responsiveness ( $\omega$ ) as key parameters in suppressing transmission, whereas prolonged delays and higher depletion rates of prevention and funding programs elevated disease prevalence. From a policy perspective, the findings highlight that administrative bottlenecks and slow institutional responses can unintentionally amplify TB transmission, even under strong intervention programs. Maintaining steady funding flows, improving program responsiveness, and minimizing diagnostic delays are therefore essential for sustainable TB reduction. Overall, the proposed model offers a comprehensive framework for understanding how time delays influence TB dynamics and provides practical insights to guide responsive, data-driven strategies for effective TB control and elimination.

**Keywords:** tuberculosis; delay differential equations; Hopf bifurcation, stability analysis; treatment initiation delay; funding response delay

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## 1. Introduction

Tuberculosis (TB) remains one of the most severe global health challenges, causing substantial morbidity and mortality, particularly in low- and middle-income countries (LMICs). According to the World Health Organization (WHO), TB continues to be a leading infectious disease killer, with an estimated 10.7 million new cases and about 1.23 million deaths reported annually [1, 2]. Following three years of COVID-19 dominance, TB re-emerged in 2024 as the leading cause of death from a single infectious agent, including approximately 150,000 deaths among people living with HIV [2]. Despite sustained control efforts, TB transmission remains a significant public-health challenge, largely driven by delayed diagnosis, incomplete treatment, and the increasing burden of multidrug-resistant TB (MDR-TB), with only 34,000 individuals affected by MDR/RR-TB receiving adequate care [2]. Furthermore, TB continues to be the leading cause of death among individuals with HIV and a significant contributor to antimicrobial resistance. Effective prevention and control demand comprehensive strategies encompassing early detection, treatment adherence, vaccination, and public-awareness initiatives. However, the success of these interventions relies heavily on sustainable financial resources, particularly in resource-limited settings where domestic health funding is insufficient and dependence on external support remains high [3].

TB is an infectious disease caused by *Mycobacterium tuberculosis*, a rod-shaped, non-motile, and obligate aerobic bacterium that has affected humans for thousands of years [4, 5]. Transmission primarily occurs through inhalation of airborne droplets released by individuals with active pulmonary TB when they cough, speak, or sneeze [6]. The pathogen thrives in oxygen-rich environments, particularly in the upper lobes of the lungs [7, 8]. Following infection, the host immune system can often contain the bacteria without eradicating them completely, leading to latent tuberculosis infection (LTBI), a non-contagious and asymptomatic state [9]. Reactivation may occur when the immune system is compromised, progressing to active disease [10, 11]. Clinical manifestations typically include a persistent cough, chest pain, hemoptysis, fatigue, weight loss, fever, and night sweats [4, 12]. Importantly, TB is not transmitted through casual contact such as handshakes or sharing food and utensils [6]. Because the infection spreads via airborne particles, its transmission risk is particularly high in crowded or poorly ventilated spaces, emphasizing the importance of early diagnosis, effective therapy, and strong public health interventions [13].

The global burden of TB is disproportionately concentrated in LMICs, where delays in treatment initiation and inconsistent financial support exacerbate the epidemic's impact. Sustained and timely funding is critical for maintaining progress toward TB elimination. However, WHO reports reveal that reductions in TB financing, particularly in LMICs dependent on external aid, have disrupted essential services such as diagnostic testing, drug supply, and surveillance, threatening millions of lives [14]. Effective TB prevention relies on well-funded, coordinated interventions, including early detection and treatment of latent TB infection (LTBI), adherence support through directly observed therapy (DOT), and robust infection control practices in healthcare and community settings [11, 15]. Complementary measures such as public education, Bacille Calmette Guérin (BCG) vaccination in high-burden areas, and improved access to care further strengthen TB prevention efforts [13, 16].

Adequate and consistent financing remains the cornerstone of effective TB control. Many LMICs rely heavily on international funding sources to sustain prevention, diagnosis, treatment, and research programs [17, 18]. The Global Fund to Fight AIDS, TB and Malaria has invested approximately

US\$9.9 billion in TB programs as of June 2024, accounting for nearly 76% of global external TB financing [19]. Global TB efforts have saved an estimated 79 million lives since 2000 [2], yet achieving the 2023 UN High-Level Meeting targets for incidence and mortality reduction by 2027 will require around US\$22 billion annually [2]. Persistent funding gaps and donor volatility continue to threaten these gains, leading to reduced case detection, treatment interruptions, and increased multidrug-resistant TB (MDR-TB) transmission [18].

Mathematical modeling has become an indispensable tool for understanding the complex dynamics of TB transmission and for guiding the design of effective intervention strategies. Through simulation of disease progression and health system responses, such models provide valuable insights into how variations in treatment access, prevention efforts, and resource allocation can influence epidemic outcomes [20]. Dynamic compartmental frameworks, in particular, enable researchers to assess intervention scenarios under resource-constrained conditions commonly seen in low- and middle-income countries. Infectious-disease modeling studies have further demonstrated the usefulness of mathematical frameworks for investigating intervention strategies, stability properties, reproduction numbers, and parameter sensitivity [21–23]. Researchers have focused on modeling TB-related funding programs [24], social determinants of healthcare access [25], emerging technologies and global control strategies [26], and the critical timing of treatment initiation and adherence [27]. Delays in treatment initiation remain a major driver of continued transmission and poor outcomes [28], while funding interruptions and slow resource mobilization have been shown to hinder TB control progress in many high-burden regions [29]. Other modeling studies have examined the impact of vaccination coverage and healthcare access [30], age-specific latency and reactivation [31], diagnostic sequencing and detection efficiency [32, 33], co-infection with diabetes [34], and optimal control strategies [35, 36]. Collectively, these efforts demonstrate the power and flexibility of mathematical models in addressing the multifaceted challenges of TB prevention and control.

Although mathematical models have advanced the understanding of TB dynamics, most overlook the temporal delays that occur between disease progression, funding responses, and treatment initiation. Frameworks often assume that financial resources and treatment programs respond instantly to changes in TB burden, neglecting the bureaucratic and behavioral delays that characterize real-world control efforts [37]. Delays in funding allocation, program implementation, healthcare-seeking behavior, diagnosis, and treatment initiation create temporal memory in TB dynamics, causing current transmission and control outcomes to depend on past as well as present conditions. These delayed responses can prolong infectiousness, delay the impact of interventions, and lead to transient or recurrent epidemic patterns. Few researchers have examined how delayed funding allocation and treatment initiation jointly influence epidemic outcomes. In this study, we address this gap by developing a delay differential model that simultaneously captures programmatic and individual-level delays, providing a more realistic representation of TB control processes and offering insights into how response timing affects the effectiveness of prevention and treatment strategies.

This study extends our earlier TB transmission model in [24], which assumed immediate responses in intervention and resource allocation, by introducing a delay differential equation (DDE) framework with two explicit delays: a funding and implementation delay, and a treatment initiation delay. By enabling past epidemic states to influence current transmission and control dynamics, the model

enables the analysis of critical delay thresholds, stability switches, and delay-induced Hopf bifurcations. Consequently, it provides a new dynamical framework for quantifying how delays in funding and treatment responses affect TB control and may induce oscillatory epidemic behavior.

The remainder of this paper is structured as follows: In Section 2, we formulate the model and outline the underlying assumptions. In Section 3, we present the analytical results, including the basic reproduction number, equilibrium analysis, stability, and bifurcation. In Section 4, we provide numerical simulations, focusing on sensitivity analysis, identifiability, model fitting, and policy insights. In Section 5, we conclude the study.

## 2. Description of the model

To investigate the dynamics of TB and assess the impact of prevention and control strategies, we construct a deterministic compartmental model that divides the human population into six epidemiological states at time  $t$ : susceptible individuals  $S(t)$ , slow-progressing latents  $L_s(t)$ , fast-progressing latents  $L_f(t)$ , infectious individuals  $I(t)$ , those under treatment  $H(t)$ , and recovered individuals  $R(t)$ . The total population is therefore expressed as

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

In addition to these epidemiological states, the framework incorporates two intervention-related variables: The prevention program intensity index  $P(t)$  and funding availability  $F(t)$ . These variables represent external control mechanisms rather than population compartments and influence the transmission and treatment dynamics over time.

A distinctive feature of the model is the incorporation of two time delays that capture realistic TB control processes. The funding-response delay,  $\tau_1$ , represents administrative and institutional lags in adjusting financial resources and implementing control measures following changes in TB burden [14, 38]. The treatment-initiation delay,  $\tau_2$ , represents delays between infectiousness or symptom onset and the start of treatment due to behavioral, financial, healthcare-access, and diagnostic factors [39]. Consequently,  $\tau_1$  reflects health-system response delays, while  $\tau_2$  captures delays at the individual treatment level.

Recruitment into the susceptible class occurs at a constant rate of  $\Pi$ . The model assumes that susceptible individuals  $S(t)$  acquire infection through contact with infectious individuals  $I(t)$ , entering either the slow-progressing latent class  $L_s(t)$  with probability  $q$  or the fast-progressing class  $L_f(t)$  with probability  $1 - q$ , depending on host immunity and risk factors. Exogenous reinfection may transfer individuals from  $L_s$  to  $L_f$  at rate  $\eta$ . Latent individuals progress to active TB at rates  $\rho_1$  and  $\rho_2$ , respectively. Infectious individuals  $I(t)$  transmit the disease and enter treatment at the delayed rate  $\gamma e^{-(\mu+\delta)\tau_2} I(t - \tau_2)$ , where  $e^{-(\mu+\delta)\tau_2}$  represents the probability of surviving the treatment delay  $\tau_2$ . Thus, only infectious individuals who survive the delay contribute to the treatment class [37, 40].

Those in the treatment class  $H(t)$  recover at rate  $\theta$ , and recovered individuals  $R(t)$  may relapse to the infectious class at rate  $k$ . All individuals are subject to natural mortality  $\mu$ , while additional TB-related deaths occur among infectious individuals at rate  $\delta$ .

The prevention program variable  $P(t)$  is interpreted as a non-negative prevention intensity index representing the overall level of implementation of preventive interventions, such as vaccination, case finding, health education, and other TB prevention activities. This variable evolves in response to

funding availability  $F(t)$ , which depends on baseline contributions and disease burden. Specifically, funding grows through two components: A constant baseline inflow  $F_0$  and an additional contribution proportional to the infectious population at a delayed time  $t - \tau_1$ , denoted by  $\omega I(t - \tau_1)$ . Funding naturally declines at rate  $\mu_f$ , reflecting the depletion of resources or withdrawal of donor support.

The prevention intensity index increases with available funding according to

$$\alpha(F) = \alpha_0 + \alpha F,$$

where  $\alpha_0$  is the baseline rate of increase and  $\alpha$  represents the marginal effect of increased funding. The prevention intensity declines at rate  $\mu_p$  but maintains a baseline level  $P_0$ , representing a minimum level of prevention effort in the absence of strong financial support.

The funding-prevention subsystem captures the dependence of disease-prevention interventions on available financial resources. Consistent with resource-dependent epidemiological models, increased funding enhances prevention coverage and reduces disease transmission. The model structure is adapted from our previous work [24], which directly linked funding availability to prevention coverage. In this study, the framework is extended by incorporating time delays into the disease-control process, providing a more realistic representation of TB dynamics while retaining the underlying resource-dependent structure. The force of infection that determines the risk of infection is given by,

$$\lambda_i(t) = \frac{\beta I}{1 + \epsilon P},$$

where  $\beta$  is the transmission coefficient and  $\epsilon \geq 0$  represents the prevention program effectiveness coefficient and measures the strength of the protective effect of prevention efforts on TB transmission. The term  $\frac{1}{1 + \epsilon P}$  represents the protective effect of prevention, such that increasing the prevention effort index  $P(t)$  monotonically reduces the effective force of infection. Larger values of  $\epsilon$  indicate greater effectiveness of prevention efforts [24, 41]. The rational functional form captures saturation and diminishing marginal benefits at high prevention intensity while reducing to the classical bilinear incidence when  $\epsilon = 0$ .

Bringing these components together, the model is described by the following system of delay differential equations:

$$\left. \begin{aligned}
 \frac{dS}{dt} &= \Pi - \mu S - \lambda_i S, \\
 \frac{dL_s}{dt} &= q\lambda_i S - \eta\lambda_i L_s - d_0 L_s, \\
 \frac{dL_f}{dt} &= (1-q)\lambda_i S + \eta\lambda_i L_s - d_1 L_f, \\
 \frac{dI}{dt} &= \rho_1 L_s + \rho_2 L_f + kR - d_2 I - \gamma e^{-d_2 \tau_2} I(t - \tau_2), \\
 \frac{dH}{dt} &= \gamma e^{-d_2 \tau_2} I(t - \tau_2) - d_3 H, \\
 \frac{dR}{dt} &= \theta H - d_4 R, \\
 \frac{dF}{dt} &= F_0 + \omega I(t - \tau_1) - \mu_f F, \\
 \frac{dP}{dt} &= P_0 + \alpha(F) - \mu_p P,
 \end{aligned} \right\} \quad (2.1)$$

where

$$d_0 = \mu + \rho_1, \quad d_1 = \mu + \rho_2, \quad d_2 = \mu + \delta, \quad d_3 = \mu + \theta, \quad \text{and} \quad d_4 = \mu + k.$$

The system is complemented by the following initial history conditions:

$$\begin{aligned}
 S(\varphi) &= \Psi_1(\varphi), & L_s(\varphi) &= \Psi_2(\varphi), & L_f(\varphi) &= \Psi_3(\varphi), & I(\varphi) &= \Psi_4(\varphi), \\
 H(\varphi) &= \Psi_5(\varphi), & R(\varphi) &= \Psi_6(\varphi), & P(\varphi) &= \Psi_7(\varphi), & F(\varphi) &= \Psi_8(\varphi),
 \end{aligned}$$

where  $\tau = \max\{\tau_1, \tau_2\}$  and  $\Psi_i \in C([- \tau, 0], \mathbb{R}_+)$ ,  $i = 1, \dots, 8$ , so that each  $\Psi_i$  is a continuous scalar-valued history function defined on  $[- \tau, 0]$ . These functions satisfy  $\Psi_i(\varphi) \geq 0$ ,  $\varphi \in [- \tau, 0]$ ,  $i = 1, \dots, 8$ , with strictly positive values at the initial time, that is  $\Psi_i(0) > 0$ ,  $i = 1, \dots, 8$ . Equivalently, the vector-valued history function  $\Psi(\varphi) = (\Psi_1(\varphi), \Psi_2(\varphi), \dots, \Psi_8(\varphi))^T$  belongs to  $C([- \tau, 0], \mathbb{R}_+^8)$ . Figure 1 illustrates the schematic diagram of the model, highlighting the epidemiological transitions and the influence of prevention and funding mechanisms. Parameter definitions and their biological interpretations are summarized in Table 1, emphasizing the roles of financial and behavioral delays in shaping TB transmission dynamics and intervention outcomes.

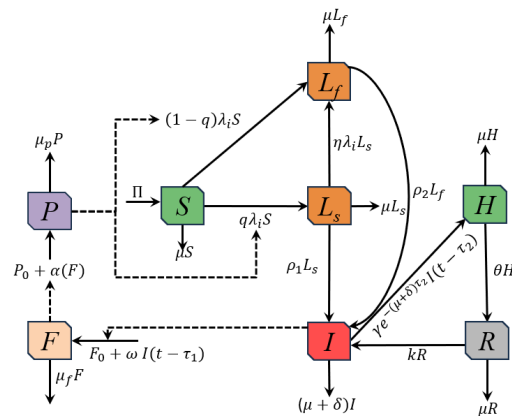


**Figure 1.** Schematic representation of the TB transmission model. The human population is divided into six epidemiological compartments: susceptible ( $S$ ), slow-progressing latent ( $L_s$ ), fast-progressing latent ( $L_f$ ), infectious ( $I$ ), under treatment ( $H$ ), and recovered ( $R$ ). Two intervention-related variables, funding level ( $F$ ) and prevention program coverage ( $P$ ), influence transmission, treatment, and expansion of preventive measures.

**Table 1.** Description of model parameters. In this study, time is measured in years and funding is measured in US dollars (USD).

| Parameter  | Description                                             | Units                                       |
|------------|---------------------------------------------------------|---------------------------------------------|
| $\Pi$      | Recruitment rate into the susceptible population        | individual <sup>-1</sup> year <sup>-1</sup> |
| $\beta$    | Transmission rate of TB                                 | individual <sup>-1</sup> year <sup>-1</sup> |
| $\epsilon$ | Prevention program effectiveness coefficient            | program <sup>-1</sup>                       |
| $q$        | Proportion of newly infected entering slow latent class | dimensionless                               |
| $\eta$     | Level of exogenous reinfection                          | dimensionless                               |
| $\rho_1$   | Progression rate from $L_s$ to active TB                | year <sup>-1</sup>                          |
| $\rho_2$   | Progression rate from $L_f$ to active TB                | year <sup>-1</sup>                          |
| $\gamma$   | Treatment initiation rate                               | year <sup>-1</sup>                          |
| $\theta$   | Recovery rate from treatment                            | year <sup>-1</sup>                          |
| $k$        | Relapse rate from recovered to infectious class         | year <sup>-1</sup>                          |
| $\mu$      | Natural death rate                                      | year <sup>-1</sup>                          |
| $\delta$   | TB-induced death rate                                   | year <sup>-1</sup>                          |
| $\omega$   | Rate at which funding increases due to disease burden   | USD/(individual · year)                     |
| $\mu_f$    | Rate of funding usage                                   | year <sup>-1</sup>                          |
| $F_0$      | Baseline inflow of funding                              | USD/year                                    |
| $\alpha_0$ | Baseline rate of prevention program growth              | program <sup>-1</sup>                       |
| $\alpha$   | Additional prevention program growth per unit funding   | program/(USD · year)                        |
| $\mu_p$    | Prevention program declining rate                       | year <sup>-1</sup>                          |
| $P_0$      | Baseline prevention program inflow                      | program <sup>-1</sup>                       |
| $\tau_1$   | Funding response delay                                  | years                                       |
| $\tau_2$   | Treatment initiation delay                              | years                                       |

### 3. Model analysis

In this section, we establish that all solutions of system (2.1) remain non-negative and bounded for  $t > 0$ . This ensures that the model retains biological validity, since population variables cannot take negative values, and remain confined within the feasible region  $\Omega$ .

#### 3.1. Positivity of solutions

In this subsection, we establish that the solutions of the system remain strictly positive for all  $t \geq 0$ , assuming that the initial conditions are positive. This property is crucial to ensure the biological feasibility of the model.

##### Lemma 1.

*Let the model parameters be nonnegative and suppose the initial conditions satisfies*

$$S(0), L_s(0), L_f(0), I(0), H(0), R(0), F(0), P(0) > 0.$$

*Then the solution of the system exists for all  $t \geq 0$  and*

$$S(t), L_s(t), L_f(t), I(t), H(t), R(t), F(t), P(t) > 0, \quad \text{for every } t \geq 0.$$

*Proof.*

Define, for each  $t \geq 0$ ,

$$L(t) = \min\{S(t), L_s(t), L_f(t), I(t), H(t), R(t), F(t), P(t)\}.$$

By the hypothesis on the initial conditions, we have  $L(0) > 0$ . We will show that  $L(t) > 0$  for all  $t \geq 0$ .

Assume, to obtain a contradiction, that there exists  $t_0 > 0$ , such that

$$L(t_0) = 0 \quad \text{and} \quad L(t) > 0 \quad \text{for all } t \in [0, t_0).$$

At time  $t_0$ , the minimum is attained by at least one of the state variables; hence, one of the following must hold:

$$S(t_0) = 0, L_s(t_0) = 0, L_f(t_0) = 0, I(t_0) = 0, H(t_0) = 0, R(t_0) = 0, F(t_0) = 0, P(t_0) = 0.$$

We treat the case  $S(t_0) = 0$ ; the other cases are analogous.

On the interval  $[0, t_0)$ , we have  $S(t) > 0$ , and the equation for  $S$  yields

$$\frac{dS}{dt} = \Pi - \mu S - \lambda_i S \geq -(\mu + \lambda_i)S, \quad \text{for all } t \in [0, t_0).$$

Integrating this differential inequality from 0 to  $t_0$  gives

$$0 = S(t_0) \geq S(0) \exp\left(-\mu t_0 - \int_0^{t_0} \lambda_i(\varpi) d\varpi\right) > 0,$$

which is a contradiction. Therefore, no such  $t_0$  exists.

Hence,

$$S(t), L_s(t), L_f(t), I(t), H(t), R(t), F(t), P(t) > 0 \quad \text{for all } t \geq 0.$$

□

### 3.2. Invariant region

We first characterize the biologically feasible region in which system trajectories remain. Define

$$\Omega = \left\{ \chi \in \mathbb{R}_+^6 : 0 < N(t) \leq \frac{\Pi}{\mu} \right\}, \quad \text{where } \chi = (S, L_s, L_f, I, H, R).$$

The auxiliary variables  $P(t)$ , representing the prevention effort index, and  $F(t)$ , representing funding availability, are not demographic compartments and therefore do not contribute directly to the total population size. Their non-negativity and boundedness are instead established from their respective governing equations.

**Lemma 2.** For system (2.1), let  $\tau = \max\{\tau_1, \tau_2\}$ . Assume that the initial functions defined on  $[-\tau, 0]$  are nonnegative and satisfy

$$N(\varphi) \leq \frac{\Pi}{\mu}, \quad P(\varphi) \geq 0, \quad F(\varphi) \geq 0, \quad \varphi \in [-\tau, 0].$$

Then, for all  $t \geq 0$ ,

$$0 < N(t) \leq \frac{\Pi}{\mu}, \quad 0 \leq F(t) \leq \frac{F_0 + \frac{\omega\Pi}{\mu}}{\mu_f}, \quad 0 \leq P(t) \leq \frac{P_0 + \alpha_0 + \alpha F_{\max}}{\mu_p},$$

where

$$F_{\max} = \frac{F_0 + \frac{\omega\Pi}{\mu}}{\mu_f}.$$

Consequently, the trajectories of system (2.1) remain in the feasible region  $\Omega$ .

*Proof.* Summing the human compartments gives

$$\frac{dN}{dt} = \Pi - \mu N - \delta I \leq \Pi - \mu N.$$

The comparison equation  $y'(t) = \Pi - \mu y(t)$  with  $y(0) = \max\{N(0), \frac{\Pi}{\mu}\}$  yields  $N(t) \leq y(t)$  for all  $t \geq 0$ . Since  $y(t)$  decreases monotonically to  $\frac{\Pi}{\mu}$ , we obtain

$$0 < N(t) \leq \max\left\{N(0), \frac{\Pi}{\mu}\right\}, \quad \limsup_{t \rightarrow \infty} N(t) \leq \frac{\Pi}{\mu}.$$

Because each compartment is nonnegative, it follows that  $0 \leq I(t) \leq N(t)$ , and hence

$$0 \leq I(t - \tau_1) \leq \frac{\Pi}{\mu}, \quad \forall t \geq 0.$$

Consider the delayed funding dynamics given by

$$\frac{dF}{dt} = F_0 + \omega I(t - \tau_1) - \mu_f F.$$

Using the bound on  $I(t - \tau_1)$ , we have

$$\frac{dF}{dt} \leq F_0 + \frac{\omega\Pi}{\mu} - \mu_f F.$$

Comparison with the associated linear ODE gives

$$F(t) \leq \frac{F_0 + \frac{\omega\Pi}{\mu}}{\mu_f} + \left( F(0) - \frac{F_0 + \frac{\omega\Pi}{\mu}}{\mu_f} \right) e^{-\mu_f t},$$

so that

$$0 \leq F(t) \leq \frac{F_0 + \frac{\omega\Pi}{\mu}}{\mu_f}, \quad \forall t \geq 0.$$

Next, consider the prevention program dynamics

$$\frac{dP}{dt} = P_0 + \alpha(F(t)) - \mu_p P,$$

where

$$\alpha(F(t)) = \alpha_0 + \alpha F(t).$$

Since  $F(t) \leq F_{\max}$ , it follows that

$$\frac{dP}{dt} \leq P_0 + \alpha_0 + \alpha F_{\max} - \mu_p P.$$

Comparison with the linear equation

$$z'(t) = P_0 + \alpha_0 + \alpha F_{\max} - \mu_p z(t)$$

gives

$$P(t) \leq \frac{P_0 + \alpha_0 + \alpha F_{\max}}{\mu_p} - \left( P(0) - \frac{P_0 + \alpha_0 + \alpha F_{\max}}{\mu_p} \right) e^{-\mu_p t}.$$

Consequently,

$$0 \leq P(t) \leq P_{\max}, \quad P_{\max} = \frac{P_0 + \alpha_0 + \alpha F_{\max}}{\mu_p}, \quad t \geq 0.$$

Thus, all state variables

$$S(t), L_s(t), L_f(t), I(t), H(t), R(t), F(t), P(t)$$

remain nonnegative and bounded for all  $t \geq 0$ . Hence, the trajectories of system (2.1) remain in the feasible region  $\Omega$ .  $\square$

### 3.3. Model equilibrium points

To determine the equilibrium states of system (2.1), we evaluate the model at steady state. At equilibrium, all state variables are constant in time, so the delayed terms satisfy  $X(t - \tau_i) = X(t) = X^*$  for each compartment. Consequently, the equilibrium equations do not depend on the delays  $\tau_1$  and  $\tau_2$  [42].

### 3.3.1. Disease-free equilibrium (DFE)

To establish the DFE of system (2.1), we assume that in the absence of infection, all disease-associated compartments vanish. Accordingly, we set

$$L_s = L_f = I = H = R = 0.$$

Substituting these values into the model equations and equating the right-hand sides to zero, we solve for the remaining compartments at steady state. This yields

$$S^* = \frac{\Pi}{\mu}, \quad F^* = \frac{F_0}{\mu_f}, \quad P^* = \frac{\mu_f(P_0 + \alpha_0) + \alpha F_0}{\mu_f \mu_p}.$$

Thus, the disease-free equilibrium of the model is expressed as

$$E_0 = \left( \frac{\Pi}{\mu}, 0, 0, 0, 0, 0, \frac{F_0}{\mu_f}, \frac{\mu_f(P_0 + \alpha_0) + \alpha F_0}{\mu_f \mu_p} \right). \quad (3.1)$$

### 3.3.2. The basic reproduction number

In epidemiological modeling, the basic reproduction number, denoted by  $\mathcal{R}_0$ , represents the average number of secondary infections generated by a single infectious individual introduced into a completely susceptible population. It serves as a key threshold parameter for determining whether an infectious disease can invade and persist within a community [43].

For the model system (2.1), let  $\tilde{\gamma} = \gamma e^{-d_2 \tau_2}$  denote the survival-adjusted treatment rate. The basic reproduction number is given by

$$\mathcal{R}_0 = \frac{\beta \Pi (q \rho_1 d_1 + (1 - q) \rho_2 d_0)}{d_0 d_1 \mu (1 - \xi_1) (\tilde{\gamma} + d_2)} \Theta_0, \quad (3.2)$$

where

$$\xi_1 = \frac{\tilde{\gamma} \theta k}{d_3 d_4 (\tilde{\gamma} + d_2)}, \quad \text{and} \quad \Theta_0 = \frac{\mu_f \mu_p}{\mu_f ((\alpha_0 + P_0) \epsilon + \mu_p) + \alpha \epsilon F_0}.$$

The detailed derivation of  $\mathcal{R}_0$  is provided in Appendix A.

**Remark 1.** It is important to note that although the basic reproduction number  $\mathcal{R}_0$  is expressed in terms of  $\tilde{\gamma} = \gamma e^{-d_2 \tau_2}$ , the value of  $\tilde{\gamma}$  is determined at the disease-free equilibrium where  $\tau_2$  is treated as a fixed system parameter. Therefore,  $\mathcal{R}_0$  depends parametrically on  $\tau_2$  through  $\tilde{\gamma}$ , but is otherwise a threshold constant for given parameter values. Moreover, the threshold does not shift with  $\tau_1$ , since the funding delay enters only the funding subsystem and not the infection generation matrix. Their destabilizing influence manifests only around the endemic equilibrium, where delays may alter stability properties and potentially induce oscillatory behavior through Hopf bifurcation.

The factor  $\Theta_0$ , referred to as the funding prevention efficiency factor, captures the combined effect of funding dynamics and prevention programs on disease transmission. Specifically, it reflects how baseline funding, the efficiency of translating funding into prevention, prevention effectiveness, and the decay rates of funding and prevention coverage jointly influence the effective transmission potential.

In particular, smaller values of  $\Theta_0$  correspond to more efficient prevention systems and are associated with a reduction in the basic reproduction number  $\mathcal{R}_0$ . Consequently, strategies that increase baseline funding, improve conversion efficiency, enhance prevention effectiveness, or reduce prevention decay act to lower  $\Theta_0$ , thereby decreasing  $\mathcal{R}_0$  and strengthening the prospects for tuberculosis control.

### 3.3.3. Local stability of the DFE

#### Theorem 1.

- (1) If  $(d_3^2 - \tilde{\gamma}^2) > 0$ ,  $a_1 > 0$ ,  $a_2 > 0$ , and  $a_3 > 0$ , then the disease-free equilibrium  $E_0$  is locally asymptotically stable whenever  $\tau_1 \geq 0$  and for admissible values of  $\tau_2 \geq 0$ , such that no characteristic roots cross the imaginary axis, provided that  $\mathcal{R}_0 < 1$ .
- (2) The disease-free equilibrium  $E_0$  is unstable whenever  $\mathcal{R}_0 > 1$ .

**Remark 2.** The delay  $\tau_1$  does not affect the local stability of the disease-free equilibrium since it is absent from the linearized infection subsystem. Although the treatment delay  $\tau_2$  appears in the characteristic equation through exponential terms, Theorem 1 shows that no stability switching occurs; hence, the disease-free equilibrium remains locally asymptotically stable whenever  $\mathcal{R}_0 < 1$ .

*Proof.* A complete proof is given in Appendix B.

### 3.3.4. Endemic equilibrium

By solving the last two equations of the system (2.1), we obtain

$$F^* = \frac{I^*\omega + F_0}{\mu_f} \quad \text{and} \quad P^* = \frac{(\alpha_0 + P_0)\mu_f + \alpha(I^*\omega + F_0)}{\mu_f\mu_p}.$$

Likewise, solving the fifth, sixth, first, second, and third equation of system (2.1) for  $H^*$ ,  $R^*$ ,  $S^*$ ,  $L_s^*$ , and  $L_f^*$ , respectively, in terms of  $I^*$  gives

$$\begin{aligned} H^* &= \frac{\tilde{\gamma}I^*}{d_3}, \quad R^* = \frac{\gamma\theta I^*}{d_3d_4}, \quad S^* = \frac{\Pi(\mu_f((\alpha_0 + P_0)\epsilon + \mu_p) + \alpha\epsilon(I^*\omega + F_0))}{B_1^*}, \\ L_s^* &= \frac{\beta I^*\mu_f\mu_p\Pi q(\mu_f((\alpha_0 + P_0)\epsilon + \mu_p) + \alpha\epsilon(I^*\omega + F_0))}{B_1^*B_2^*}, \\ L_f^* &= \frac{\beta I^*\mu_f\mu_p\Pi(\beta\eta I^*\mu_f\mu_p + d_0(1 - q)(\mu_f((\alpha_0 + P_0)\epsilon + \mu_p) + \alpha\epsilon(I^*\omega + F_0)))}{d_1B_1^*B_2^*}, \end{aligned}$$

where,

$$\begin{aligned} \tilde{\gamma} &= \gamma e^{-d_2\tau_2}, \\ B_1^* &= \mu_f((\alpha_0 + P_0)\mu\epsilon + \mu_p(\beta I^* + \mu)) + \alpha\mu\epsilon(I^*\omega + F_0), \\ B_2^* &= \beta\eta I^*\mu_f\mu_p + d_0(\mu_f((\alpha_0 + P_0)\epsilon + \mu_p) + \alpha\epsilon(I^*\omega + F_0)). \end{aligned}$$

By substituting the values of  $L_s^*$ ,  $L_f^*$ , and  $R^*$  into the fourth equation of the system (2.1) and expressing it in terms of  $I^*$ , we obtain the following third-order polynomial equation for  $I^*$ :

$$I^* (\Phi_2 I^{*2} + \Phi_1 I^* + \Phi_0) = 0, \quad (3.3)$$

where

$$\begin{aligned} \Phi_2 &= -(d_0 d_1 d_3 d_4 (1 - \xi_2) (1 - \xi_1) (\tilde{\gamma} + d_2) (\alpha \mu \omega \epsilon + \beta \mu_1 \mu_2) (\beta \eta \mu_f \mu_p + \alpha d_0 \omega \epsilon)), \\ \Phi_1 &= ((d_0 - \eta \mu) (\beta \mu_f \mu_p (B_4 d_1 + \beta d_3 d_4 \eta \mu_f \mu_p \Pi \rho_2) + d_0 (\alpha \beta d_3 d_4 \mu_f \mu_p \Pi (1 - q) \rho_2 \omega \epsilon - B_3))), \\ \Phi_0 &= B_5 d_0 (1 - \xi_2) (\mu_f ((\alpha_0 + P_0) \epsilon + \mu_p) + \alpha \epsilon F_0) (\mathcal{R}_0 - 1), \\ B_3 &= d_1 d_3 d_4 (1 - \xi_1) (\tilde{\gamma} + d_2) (2 \alpha \mu \omega \epsilon + \beta \mu_f \mu_p) (\mu_f ((\alpha_0 + P_0) \epsilon + \mu_p) + \alpha \epsilon F_0), \\ B_4 &= d_3 d_4 \eta \mu \mu_f (1 - \xi_1) (\tilde{\gamma} + d_2) ((\alpha_0 + P_0) \epsilon + \mu_p) + \alpha \epsilon (d_3 d_4 (\Pi q \rho_1 \omega - \eta \mu (\tilde{\gamma} + d_2) F_0) + \tilde{\gamma} \eta \theta k \mu F_0), \\ B_5 &= d_1 d_0 d_3 d_4 \mu (1 - \xi_1) (\tilde{\gamma} + d_2) (\mu_f ((\alpha_0 + P_0) \epsilon + \mu_p) + \alpha \epsilon F_0), \\ \xi_2 &= \frac{\eta \mu}{d_0}. \end{aligned}$$

Solving (3.3) gives  $I^* = 0$ , which corresponds to the disease-free equilibrium. The endemic equilibrium is obtained by solving the following quadratic equation in  $I^*$ :

$$\Phi_2 I^{*2} + \Phi_1 I^* + \Phi_0 = 0. \quad (3.4)$$

Table 2 below shows various possibilities for the existence of the endemic equilibrium. Here,  $I^*$  denotes the number of positive roots.

**Table 2.** Shows the number of possible positive solutions for  $I^*$ .

|       | $\Phi_2 < 0$                     |                                  |                                  |                                  |
|-------|----------------------------------|----------------------------------|----------------------------------|----------------------------------|
|       | $\Phi_1 > 0$                     |                                  | $\Phi_1 < 0$                     |                                  |
|       | $\Phi_0 > 0 (\mathcal{R}_0 > 1)$ | $\Phi_0 < 0 (\mathcal{R}_0 < 1)$ | $\Phi_0 > 0 (\mathcal{R}_0 > 1)$ | $\Phi_0 < 0 (\mathcal{R}_0 < 1)$ |
| $I^*$ | 1                                | 2                                | 1                                | 0                                |

Here, we summarize the results on the existence of the endemic equilibrium of system (2.1) in Theorem (2).

**Theorem 2.** System (2.1) has

- (1) A unique endemic equilibrium  $E^* = (S^*, L_s^*, L_f^*, I^*, H^*, R^*, F^*, P^*)$  if and only if  $\Phi_2 < 0$ , and  $\Phi_0 > 0$ ,  $\mathcal{R}_0 > 1$ , or when  $\Phi_1^2 + 4\Phi_2\Phi_0 = 0$ .
- (2) Two endemic equilibria

$$E_1^* = (S_1^*, L_{s1}^*, L_{f1}^*, I_1^*, H_1^*, R_1^*, F_1^*, P_1^*),$$

and

$$E_2^* = (S_2^*, L_{s2}^*, L_{f2}^*, I_2^*, H_2^*, R_2^*, F_2^*, P_2^*),$$

when  $\Phi_2 < 0$ ,  $\Phi_1 > 0$  and  $\Phi_0 < 0$ ,  $\mathcal{R}_0 < 1$ , where

$$I^* = \left( \frac{-\Phi_1 \pm \sqrt{\Phi_1^2 + 4\Phi_2\Phi_0}}{2\Phi_2} \right).$$

No endemic equilibrium otherwise.

As established in Theorem 2, a unique endemic equilibrium exists whenever  $\mathcal{R}_0 > 1$ . Moreover, the model admits the possibility of backward bifurcation, see [44, 45] for more detailed synopses.

It is important to note that, in the case of two delays, the equilibrium conditions are unaffected, since delays vanish at steady state (i.e.,  $I(t - \tau_j) = I^*$  for  $j = 1, 2$ ). Therefore, the occurrence of backward bifurcation is still governed by the discriminant of Eq (3.4). Setting this discriminant equal to zero yields the critical value of the reproduction number, denoted by  $\mathcal{R}_0^c$ , which is given as

$$\mathcal{R}_0^c = 1 - \frac{\Phi_1^2}{4B_5d_0(1 - \xi_2)(\mu_f((\alpha_0 + P_0)\epsilon + \mu_p) + \alpha\epsilon F_0)\Phi_2}.$$

Thus, backward bifurcation occurs whenever  $\mathcal{R}_0^c < \mathcal{R}_0 < 1$ . While the delays do not affect the existence of equilibria, they play a crucial role in determining their local stability and may further give rise to oscillatory behavior through Hopf bifurcation.

### 3.3.5. Stability of the endemic equilibrium ( $E^*$ ) and local Hopf bifurcation

In this subsection, we analyze the stability of the endemic equilibrium  $E^*$  and establish the occurrence of a local Hopf bifurcation in system (2.1). A Hopf bifurcation occurs when a pair of complex conjugate eigenvalues of the Jacobian matrix crosses the imaginary axis as system parameters vary, leading to a transition from a stable equilibrium to sustained oscillations in the form of a periodic solution [46, 47]. In epidemiological models, such a bifurcation implies that the disease prevalence may exhibit recurrent outbreaks or oscillatory dynamics rather than converging to a steady state. It is important to note that the Hopf bifurcations considered in this study are delay-induced, in the sense that the emergence of oscillatory dynamics is triggered by the presence of time delays in the system.

Let  $E^* = (S^*, L_s^*, L_f^*, I^*, H^*, R^*, F^*, P^*)$  denote the endemic equilibrium of system (2.1). To investigate its stability, we linearize system (2.1) about  $E^*$ .

Defining the state vector  $\mathbf{x} = (S, L_s, L_f, I, H, R, F, P)^\top \in \mathbb{R}^8$ , the linearized system with two discrete delays  $\tau_1, \tau_2 \geq 0$  can be expressed as

$$\frac{d\mathbf{x}}{dt} = A_0\mathbf{x}(t) + A_1\mathbf{x}(t - \tau_1) + A_2\mathbf{x}(t - \tau_2), \quad (3.5)$$

where  $A_0, A_1, A_2 \in \mathbb{R}^{8 \times 8}$  are the Jacobian matrices evaluated at  $E^*$ . Here,  $A_0$  captures the instantaneous dynamics, while  $A_1$  and  $A_2$  account for the effects of the delays  $\tau_1$  and  $\tau_2$ , respectively.

The Jacobian matrix  $A_0$  is given by

$$A_0 = \begin{pmatrix} C_1 & 0 & 0 & C_2 & 0 & 0 & 0 & C_3 \\ C_4 & C_5 & 0 & C_6 & 0 & 0 & 0 & C_7 \\ C_8 & C_9 & -d_1 & C_{10} & 0 & 0 & 0 & C_{11} \\ 0 & \rho_1 & \rho_2 & -d_2 & 0 & k & 0 & 0 \\ 0 & 0 & 0 & \tilde{\gamma} & -d_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \theta & -d_4 & 0 & 0 \\ 0 & 0 & 0 & \omega & 0 & 0 & -\mu_1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \alpha & -\mu_2 \end{pmatrix},$$

where

$$\begin{aligned} C_1 &= -\mu - \frac{\beta I^*}{1 + \epsilon P^*}, & C_2 &= \frac{\beta S^*}{1 + \epsilon P^*}, & C_3 &= -\frac{\beta I^* S^* \epsilon}{(1 + \epsilon P^*)^2}, \\ C_4 &= \frac{q\beta I^*}{1 + \epsilon P^*}, & C_5 &= -\eta \frac{\beta I^*}{1 + \epsilon P^*} - d_0, & C_6 &= \frac{q\beta S^*}{1 + \epsilon P^*} - \frac{\eta\beta L_s^*}{1 + \epsilon P^*}, \\ C_7 &= \frac{\eta\epsilon\beta L_s^* I^*}{(1 + \epsilon P^*)^2} - \frac{q\beta\epsilon I^* S^*}{(1 + \epsilon P^*)^2}, & C_8 &= \frac{(1 - q)\beta I^*}{1 + \epsilon P^*}, & C_9 &= \frac{\eta\beta I^*}{1 + \epsilon P^*}, \\ C_{10} &= \frac{(1 - q)\beta S^*}{1 + \epsilon P^*} + \frac{\eta\beta L_s^*}{1 + \epsilon P^*}, & C_{11} &= -\frac{\eta\epsilon\beta L_s^* I^*}{(1 + \epsilon P^*)^2} - \frac{(1 - q)\beta\epsilon I^* S^*}{(1 + \epsilon P^*)^2}. \end{aligned}$$

The transcendental equation associated with the delayed system is

$$\det(A_0 + A_1 e^{-\hat{\lambda}\tau_1} + A_2 e^{-\hat{\lambda}\tau_2} - \hat{\lambda}I_8) = 0,$$

where  $I_8$  is the  $8 \times 8$  identity matrix and  $\hat{\lambda} \in \mathbb{C}$  denotes the eigenvalues. This can be equivalently written as

$$\begin{aligned} p(\hat{\lambda}) &= \det(\hat{\lambda}I_8 - A_0) - \omega C_{FI}(\hat{\lambda})e^{-\hat{\lambda}\tau_1} + \tilde{\gamma}(C_{II}(\hat{\lambda}) - C_{HI}(\hat{\lambda}))e^{-\hat{\lambda}\tau_2} \\ &= \det(\hat{\lambda}I_8 - A_0) + M(\hat{\lambda})e^{-\hat{\lambda}\tau_1} + Q(\hat{\lambda})e^{-\hat{\lambda}\tau_2}, \end{aligned}$$

where  $C_{FI}(\hat{\lambda})$ ,  $C_{II}(\hat{\lambda})$ , and  $C_{HI}(\hat{\lambda})$  denote the relevant cofactors, and  $M(\hat{\lambda})$  and  $Q(\hat{\lambda})$  represent polynomial functions of  $\hat{\lambda}$  derived from these cofactors.

All symbolic computations in this paper are carried out using MATLAB's Symbolic Math Toolbox. Specifically, the Jacobian matrices  $A_0$ ,  $A_1$ , and  $A_2$  are constructed symbolically, and the characteristic determinant  $\det(A_0 + A_1 e^{-\hat{\lambda}\tau_1} + A_2 e^{-\hat{\lambda}\tau_2} - \hat{\lambda}I_8)$  is expanded using MATLAB's `det`, `collect`, and `simplify` functions. The polynomial coefficients  $J_i$ ,  $M_i$ , and  $N_i$  are then extracted by matching powers of  $\hat{\lambda}$  and grouping delay-exponential terms via the `coeffs` command. No purely numerical root-finding is used at this stage; the reduction to Eq (3.6) is a symbolic manipulation. All subsequent simulations of system (2.1) are performed numerically using MATLAB's `dde23` solver, which handles constant-delay differential equations directly.

Therefore, by using MATLAB, the simplified transcendental equation will be given by

$$\begin{aligned}
p(\hat{\lambda}, \tau_1, \tau_2) = & \hat{\lambda}^8 + J_7 \hat{\lambda}^7 + J_6 \hat{\lambda}^6 + J_5 \hat{\lambda}^5 + J_4 \hat{\lambda}^4 + J_3 \hat{\lambda}^3 + J_2 \hat{\lambda}^2 + J_1 \hat{\lambda} + J_0 \\
& + (M_4 \hat{\lambda}^4 + M_3 \hat{\lambda}^3 + M_2 \hat{\lambda}^2 + M_1 \hat{\lambda} + M_0) e^{-\hat{\lambda} \tau_1} \\
& + (N_7 \hat{\lambda}^7 + N_6 \hat{\lambda}^6 + N_5 \hat{\lambda}^5 + N_4 \hat{\lambda}^4 + N_3 \hat{\lambda}^3 + N_2 \hat{\lambda}^2 + N_1 \hat{\lambda} + N_0) e^{-\hat{\lambda} \tau_2} = 0, \quad (3.6)
\end{aligned}$$

where  $J_7, \dots, J_0, M_4, \dots, M_0$ , and  $N_7, \dots, N_0$  are listed in Appendix C. The next step is to analyze how the roots of  $p(\hat{\lambda}, \tau_1, \tau_2) = 0$  are distributed.

**Case 1:**  $\tau_1 = \tau_2 = 0$

When  $\tau_1 = \tau_2 = 0$ , Eq (3.6) reduces to an eighth-degree polynomial

$$\begin{aligned}
p(\hat{\lambda}) = & \hat{\lambda}^8 + (J_7 + N_7) \hat{\lambda}^7 + (J_6 + N_6) \hat{\lambda}^6 + (J_5 + N_5) \hat{\lambda}^5 \\
& + (J_4 + M_4 + N_4) \hat{\lambda}^4 + (J_3 + M_3 + N_3) \hat{\lambda}^3 \\
& + (J_2 + M_2 + N_2) \hat{\lambda}^2 + (J_1 + M_1 + N_1) \hat{\lambda} + (J_0 + M_0 + N_0) = 0. \quad (3.7)
\end{aligned}$$

To determine the stability of the system, we apply the Routh–Hurwitz criterion. Define the coefficients

$$b_8 = 1, \quad b_7 = J_7 + N_7, \quad b_6 = J_6 + N_6, \quad b_5 = J_5 + N_5, \quad b_4 = J_4 + M_4 + N_4,$$

$$b_3 = J_3 + M_3 + N_3, \quad b_2 = J_2 + M_2 + N_2, \quad b_1 = J_1 + M_1 + N_1, \quad b_0 = J_0 + M_0 + N_0.$$

Let  $H_n$  denote the Hurwitz matrix of order  $n$  corresponding to the polynomial (3.7). Its general form is

$$H_n = \begin{bmatrix} v_1 & v_3 & v_5 & \cdots & 0 & 0 \\ v_2 & v_4 & v_6 & \cdots & 0 & 0 \\ 0 & v_1 & v_3 & \cdots & 0 & 0 \\ 0 & v_2 & v_4 & \cdots & 0 & 0 \\ \vdots & \vdots & \vdots & \ddots & \vdots & \vdots \\ 0 & 0 & 0 & \cdots & v_{n-1} & v_{n-3} \\ 0 & 0 & 0 & \cdots & v_n & v_{n-2} \end{bmatrix}, \quad \text{with } v_j = \begin{cases} b_j, & 0 \leq j \leq 8, \\ 0, & j < 0 \text{ or } j > 8. \end{cases}$$

The polynomial (3.7) is Hurwitz stable, that is, all its roots have negative real parts, if and only if all principal Hurwitz determinants are positive

$$|H_1| > 0, |H_2| > 0, \dots, |H_8| > 0. \quad (3.8)$$

These conditions imply, in particular, that the coefficients of the characteristic polynomial are positive,

$$b_i > 0, \quad i = 0, 1, \dots, 8. \quad (3.9)$$

However, since Eq (3.7) is of degree eight, positivity of the coefficients alone is necessary but not sufficient for stability. Therefore, the local asymptotic stability of the equilibrium  $E^*$  is established through the Hurwitz conditions (3.8).

In the following, we investigate the stability of system (3.6) when delays are incorporated in the patterns.

**Case 2:**  $\tau_1 > 0$  and  $\tau_2 = 0$

*Proof.* See Appendix D for the proof of Theorem 3

**Theorem 3.** Let  $\tau_1 > 0$ ,  $\tau_2 = 0$ , and suppose that conditions (3.8) and (3.9) hold. If  $\Delta_8 < 0$ , then the equilibrium point  $E^*$  of system (2.1) is locally asymptotically stable for  $\tau_1 < \tau_1^*$ . Moreover, if  $g(z^*) \neq 0$ , then system (2.1) undergoes a Hopf bifurcation at  $E^*$  when  $\tau_1 = \tau_1^*$ .

**Case 3:**  $\tau_1 = 0$  and  $\tau_2 > 0$

*Proof.* See Appendix E for the proof of Theorem 4

**Theorem 4.** Let  $\tau_2 > 0$  and  $\tau_1 = 0$ , and assume conditions (3.8) and (3.9) hold. If  $\Gamma_8 < 0$ , then the equilibrium  $E^*$  is locally asymptotically stable for  $\tau_2 < \tau_2^*$ . Moreover, system (2.1) undergoes a Hopf bifurcation at  $\tau_2 = \tau_2^*$ , giving rise to a limit cycle, where

$$\tau_2^* = \frac{1}{z^*} \arccos \left( -\frac{\zeta_1^* \zeta_3^* + z^* \zeta_2^* \zeta_4^*}{(z^*)^2 (\zeta_2^*)^2 + (\zeta_1^*)^2} \right).$$

**Case 4:**  $\tau_1 = \tau_2 = \tau$ .

*Proof.* See Appendix F for the proof of Theorem 5.

**Theorem 5.** Let  $\tau_1 = \tau_2 = \tau$ , and assume conditions (3.8) and (3.9) are satisfied. If  $\Gamma_{16} < 0$ , then the equilibrium  $E^*$  is locally asymptotically stable for  $\tau < \tau^*$ . Moreover, system (2.1) undergoes a Hopf bifurcation at  $\tau = \tau^*$ , resulting in a limit cycle, where

$$\tau^* = \frac{1}{z^*} \arccos \left( -\frac{\zeta_5^* \zeta_7^* + \zeta_6^* \zeta_8^*}{(\zeta_5^*)^2 + (\zeta_6^*)^2} \right).$$

### Direction and stability of the Hopf bifurcation

In Subsection 3.3.5, it was shown that system (2.1) admits a family of periodic orbits that emerge from the equilibrium point  $E^*$  as the delay parameters  $\tau_1$  and  $\tau_2$  cross their respective critical thresholds. In what follows, we apply the center manifold reduction together with the normal form approach [46] to rigorously analyze the nature of the Hopf bifurcation occurring at  $\tau_1^*$ . In particular, this analysis enables us to determine both the direction of bifurcation and the stability of the bifurcating periodic solutions. For the remainder of this section, we restrict attention to the case  $\tau_2^* < \tau_1^*$ , with  $\tau_2^* \in (0, \tau_{2j})$ .

**Remark 3.** A Hopf bifurcation occurs when a periodic orbit emerges or vanishes due to a local change in the stability of a steady state. Depending on the stability of the bifurcating solutions, the bifurcation is classified as supercritical if the resulting periodic solutions are stable, or subcritical if they are unstable. Parameter  $\mu_1$  dictates the direction of the bifurcation: Specifically,  $\mu_1 > 0$  ( $\mu_1 < 0$ ) corresponds to a supercritical (subcritical) Hopf bifurcation. For a more detailed discussion of these properties, including their derivation, see Appendix G.

**Theorem 6.** System (2.1) undergoes a Hopf bifurcation at  $E^*$  when  $\tau_1$  crosses its critical value  $\tau_1^*$ . If  $\text{Re}\{c_1(0)\} < 0$  (resp.  $\text{Re}\{c_1(0)\} > 0$ ), the bifurcation is supercritical (resp. subcritical). Moreover, the periodic solutions arising from the Hopf bifurcation are orbitally asymptotically stable on the center manifold when  $\text{Re}\{c_1(0)\} < 0$ , and unstable when  $\text{Re}\{c_1(0)\} > 0$ .

## 4. Numerical simulation

### 4.1. Numerical solution method

The delay differential system (2.1) was numerically solved using MATLAB's dde23 solver with adaptive time stepping, employing relative and absolute tolerances of  $10^{-6}$  and  $10^{-8}$ , respectively, and

a maximum step size of 0.1 years. Model fitting was performed over the 2000–2023 TB incidence data period using the parameter-dependent history function described in Section 2, ensuring consistency between the initial conditions and observed data. The same numerical settings were used for parameter estimation, delay analysis, and bifurcation simulations. As the model describes only temporal dynamics, spatial discretization, grid size, and boundary conditions were not applicable.

#### 4.2. Sensitivity analysis

The forward normalized sensitivity index (FNSI) was used to assess the influence of the model parameters on the infectious population. This index quantified the proportional change in a model output resulting from a proportional change in a single parameter, with all other parameters held constant. Positive sensitivity values indicate that increasing a parameter increases the outcome, whereas negative values reflect an inverse effect. The magnitude of the index measures the strength of this influence: Values below 0.1 indicate negligible impact, values between 0.1 and 0.5 indicate moderate sensitivity, and values exceeding 0.5 indicate strong sensitivity and a significant influence on the system dynamics. For a model output  $Y$  and a parameter  $p$ , the FNSI is defined by

$$S_p^Y = \frac{p}{Y} \frac{\partial Y}{\partial p}.$$

In this study, each parameter was perturbed symmetrically by  $\pm 20\%$  around its baseline value, while all other parameters were held constant. The resulting changes in the infectious population were used to compute the FNSI. Numerically, the FNSI was approximated using the symmetric finite-difference formula

$$S_p^Y \approx \frac{p}{Y} \frac{Y(p + 0.2p) - Y(p - 0.2p)}{0.4p}. \quad (4.1)$$

This analysis identifies the most influential biological and epidemiological parameters governing disease persistence and control, thereby improving understanding of model robustness and informing parameter prioritization for accurate estimation and targeted intervention strategies [48]. The FNSI values of the infectious population with respect to the model parameters are presented in Table 3.

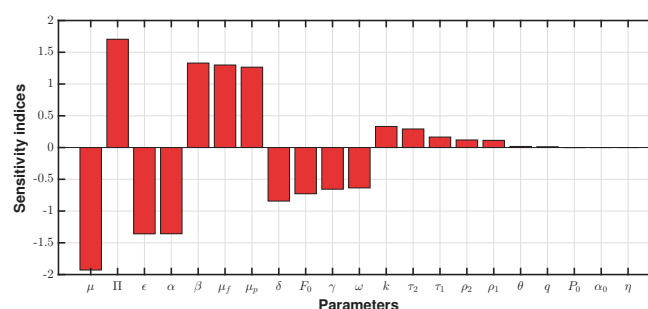


**Figure 2.** The FNSI values quantify the influence of key model parameters on the infection population, with positive indices indicating parameters that amplify infection levels and negative indices indicating those that suppress them. The magnitude of each index reflects the strength of its effect, where values close to zero denote weak sensitivity and larger values signify stronger influence on model dynamics.

**Table 3.** Forward normalized sensitivity indices of the infectious population with respect to the model parameters.

| Parameter  | FNSI   | Parameter  | FNSI   |
|------------|--------|------------|--------|
| $\mu$      | -1.91  | $\Pi$      | 1.70   |
| $\epsilon$ | -1.34  | $\alpha$   | -1.34  |
| $\beta$    | 1.32   | $\mu_f$    | 1.28   |
| $\mu_p$    | 1.26   | $\delta$   | -0.83  |
| $F_0$      | -0.71  | $\gamma$   | -0.65  |
| $\omega$   | -0.62  | $k$        | 0.32   |
| $\tau_2$   | 0.28   | $\tau_1$   | 0.15   |
| $\rho_2$   | 0.11   | $\rho_1$   | 0.10   |
| $\theta$   | -0.012 | $q$        | 0.011  |
| $P_0$      | -0.008 | $\alpha_0$ | -0.007 |
| $\eta$     | 0.007  |            |        |

Figure 2 presents the sensitivity of model parameters with respect to the infectious population. Parameters related to funding growth, prevention implementation and effectiveness, and treatment rates show strong negative correlations, highlighting the critical role of sustained financial investment and effective prevention and treatment strategies in reducing TB prevalence. Natural mortality, disease-induced death, and baseline funding also exhibit negative associations, indicating that improved population health and stable funding further support disease suppression. In contrast, higher removal rates of funding and prevention programs are positively correlated with infection levels, underscoring the risk of premature withdrawal or mismanagement of resources. Transmission-related parameters, including the effective contact rate and recruitment of susceptibles, also increase infection levels, consistent with classical epidemiological theory. Additionally, relapse, progression rates, and delays in treatment initiation and financial response positively influence infection burden, emphasizing the importance of timely intervention and uninterrupted programmatic support for sustained TB control.

#### 4.3. Practical parameter identifiability

In mathematical epidemiological modeling, it is important to determine whether model parameters can be reliably distinguished from one another. Two related but distinct concepts arise in this context: structural identifiability and practical identifiability. Structural identifiability is a theoretical property of the model equations that assumes ideal, noise-free observations and depends only on the model structure. In contrast, practical identifiability reflects the ability to estimate parameters under finite, and possibly imperfect, observational conditions, and depends on the parameter values, model outputs, and numerical properties of the system.

In this study, identifiability was assessed using collinearity indices derived from the sensitivity matrix, computed via the `collin` function in R. This approach was therefore aligned with practical (numerical) identifiability rather than formal structural identifiability. Specifically, the method evaluated the extent to which parameter sensitivities exhibit near-linear dependence, which would indicate that variations in one parameter can be compensated by others, thereby hindering reliable

estimation. In particular, no observational dataset was used as an output variable in the collinearity analysis. The identifiability assessment was based solely on the sensitivity functions generated by the model through the `collin` procedure, evaluated at the nominal parameter values. Therefore, the analysis examined numerical dependencies among parameter sensitivities rather than identifiability with respect to a specific measured quantity such as annual TB incidence.

Following the framework of [49], the degree of collinearity for a subset of parameters is measured by the index

$$C_i = \frac{1}{\sqrt{\min(\text{Ev}(\hat{Y}^\top \hat{Y}))}},$$

where  $\hat{Y}$  is the normalized sensitivity matrix with entries

$$\hat{Y}_{ij} = \frac{Y_{ij}}{\sqrt{\sum_j Y_{ij}^2}}.$$

A value  $C_i = 1$  indicates complete independence among parameter sensitivities, whereas large values indicate strong dependence. In practice, values of  $C_i$  greater than approximately 10–15 indicate high collinearity and may suggest poor practical identifiability [50]. In this study, we adopted the more conservative threshold of  $C_i = 15$  to distinguish acceptable from high collinearity.

In this study, we investigated the identifiability of parameters associated with the infectious compartment  $I$ . The analysis considered the full parameter set:  $\Pi, \beta, \epsilon, q, \eta, \rho_1, \rho_2, \gamma, \theta, k, \mu, \delta, \omega, \mu_f, F_0, \alpha_0, \alpha, \mu_p$ , and  $P_0$ . As shown in Figure 3, the collinearity index increases with the number of parameters included, indicating stronger interdependencies in larger parameter sets. Using the `collin` function, we systematically evaluated parameter combinations to identify subsets with acceptable levels of collinearity.

Results in Figure 3 and Table 4 show that several eighteen-parameter combinations yield collinearity indices below the adopted threshold of 15. Among these, the subsets with  $C_i = 8.1$  and  $C_i = 8.7$  exhibit low collinearity. The selection of the parameter subset was therefore based not only on the magnitude of the collinearity index but also on the parameters retained for subsequent estimation. In particular, some candidate subsets exclude either the baseline funding parameter  $F_0$  or the parameter  $\omega$ , which represents the rate at which funding increases in response to disease burden. Since  $\omega$  is important for describing the adaptive funding response to disease burden, we retained this parameter for joint estimation. Accordingly, we selected the eighteen-parameter subset

$$\{1, 1, 1, 1, 1, 1, 1, 1, 1, 1, 1, 1, 1, 1, 0, 1, 1, 1, 1\}, \quad (N = 18, C_i = 8.1),$$

which excludes  $F_0$  while retaining  $\omega$ . This subset satisfies the adopted collinearity criterion and provides a suitable parameter set for subsequent joint estimation.

Therefore, we selected  $\Pi, \beta, \epsilon, q, \eta, \rho_1, \rho_2, \gamma, \theta, k, \mu, \delta, \omega, \mu_f, \alpha_0, \alpha, \mu_p$ , and  $P_0$  as the practically identifiable subset for joint estimation, while the remaining parameters were handled separately. This approach improves the robustness of parameter estimation by focusing on parameter combinations with reduced numerical interdependence.

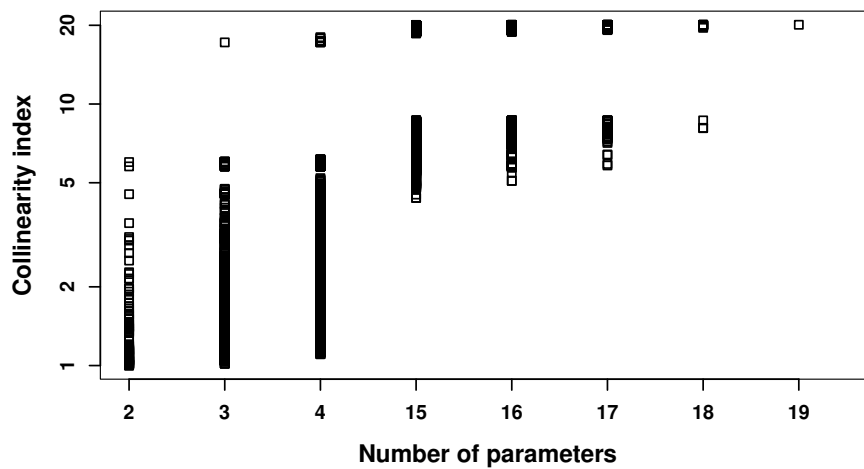


**Figure 3.** Collinearity analysis of TB model parameters. The plot displays the collinearity index for groups of parameters in the TB transmission model. In this study, values below 15 were considered to indicate acceptable collinearity, whereas values above 15 indicate stronger parameter dependence that may affect practical identifiability

**Table 4.** Parameter subsets and their collinearity indices for the TB model. A value of 1 indicates inclusion of the parameter in the subset; 0 indicates exclusion.

| $\Pi$ | $\mu$ | $\beta$ | $\epsilon$ | $q$ | $\eta$ | $\rho_1$ | $\rho_2$ | $\theta$ | $k$ | $\delta$ | $\gamma$ | $\omega$ | $\mu_f$ | $F_0$ | $\alpha_0$ | $\alpha$ | $\mu_p$ | $P_0$ | $N$ | collinearity |
|-------|-------|---------|------------|-----|--------|----------|----------|----------|-----|----------|----------|----------|---------|-------|------------|----------|---------|-------|-----|--------------|
| 1     | 1     | 1       | 1          | 1   | 1      | 1        | 1        | 1        | 1   | 1        | 1        | 1        | 1       | 1     | 1          | 1        | 1       | 0     | 18  | 20.0         |
| 1     | 1     | 1       | 1          | 1   | 1      | 1        | 1        | 1        | 1   | 1        | 1        | 1        | 1       | 1     | 1          | 1        | 0       | 1     | 18  | 20.0         |
| 1     | 1     | 1       | 1          | 1   | 1      | 1        | 1        | 1        | 1   | 1        | 1        | 1        | 1       | 1     | 1          | 0        | 1       | 1     | 18  | 20.1         |
| 1     | 1     | 1       | 1          | 1   | 1      | 1        | 1        | 1        | 1   | 1        | 1        | 1        | 1       | 1     | 0          | 1        | 1       | 1     | 18  | 20.0         |
| 1     | 1     | 1       | 1          | 1   | 1      | 1        | 1        | 1        | 1   | 1        | 1        | 1        | 1       | 0     | 1          | 1        | 1       | 1     | 18  | 8.1          |
| 1     | 1     | 1       | 1          | 1   | 1      | 1        | 1        | 1        | 1   | 1        | 1        | 1        | 0       | 1     | 1          | 1        | 1       | 1     | 18  | 8.7          |
| 1     | 1     | 1       | 1          | 1   | 1      | 1        | 1        | 1        | 1   | 1        | 1        | 0        | 1       | 1     | 1          | 1        | 1       | 1     | 18  | 8.1          |
| 1     | 1     | 1       | 1          | 1   | 1      | 1        | 1        | 1        | 1   | 1        | 0        | 1        | 1       | 1     | 1          | 1        | 1       | 1     | 18  | 20.0         |
| 1     | 1     | 1       | 1          | 1   | 1      | 1        | 1        | 1        | 1   | 0        | 1        | 1        | 1       | 1     | 1          | 1        | 1       | 1     | 18  | 20.1         |
| 1     | 1     | 1       | 1          | 1   | 1      | 1        | 1        | 1        | 0   | 1        | 1        | 1        | 1       | 1     | 1          | 1        | 1       | 1     | 18  | 20.0         |
| 1     | 1     | 1       | 1          | 1   | 1      | 1        | 0        | 1        | 1   | 1        | 1        | 1        | 1       | 1     | 1          | 1        | 1       | 1     | 18  | 20.1         |
| 1     | 1     | 1       | 1          | 1   | 1      | 1        | 1        | 0        | 1   | 1        | 1        | 1        | 1       | 1     | 1          | 1        | 1       | 1     | 18  | 19.6         |
| 1     | 1     | 1       | 1          | 1   | 1      | 0        | 1        | 1        | 1   | 1        | 1        | 1        | 1       | 1     | 1          | 1        | 1       | 1     | 18  | 20.0         |
| 1     | 1     | 1       | 1          | 0   | 1      | 1        | 1        | 1        | 1   | 1        | 1        | 1        | 1       | 1     | 1          | 1        | 1       | 1     | 18  | 20.0         |
| 1     | 1     | 1       | 0          | 1   | 1      | 1        | 1        | 1        | 1   | 1        | 1        | 1        | 1       | 1     | 1          | 1        | 1       | 1     | 18  | 19.8         |
| 1     | 1     | 0       | 1          | 1   | 1      | 1        | 1        | 1        | 1   | 1        | 1        | 1        | 1       | 1     | 1          | 1        | 1       | 1     | 18  | 20.1         |
| 1     | 0     | 1       | 1          | 1   | 1      | 1        | 1        | 1        | 1   | 1        | 1        | 1        | 1       | 1     | 1          | 1        | 1       | 1     | 18  | 19.8         |
| 0     | 1     | 1       | 1          | 1   | 1      | 1        | 1        | 1        | 1   | 1        | 1        | 1        | 1       | 1     | 1          | 1        | 1       | 1     | 18  | 20.0         |

#### 4.4. Model fitting and parameter estimation

To accurately capture the dynamics of TB transmission in South Africa, the model was calibrated using annual incidence data spanning 2000 to 2023. TB incidence and population estimates for all age groups were obtained from the World Bank [51,52], enabling for a comprehensive representation of the disease burden. The World Bank TB incidence data were reported as estimated incidence rates per 100,000 population. To obtain the corresponding annual number of TB incident cases used for model calibration, the incidence rate for each year was converted using

$$\mathcal{J}_t = \left( \frac{R_t}{100000} \right) N_t,$$

where  $N_t$  denotes the total population in year  $t$  and  $R_t$  is the reported TB incidence rate per 100,000 population. The resulting  $\mathcal{J}_t$  represents the estimated number of incident TB cases in year  $t$ .

The model, formulated as a system of delay differential equations (DDEs) to account for temporal lags in treatment initiation and financial response, was solved numerically using MATLAB's `dde23` solver, which is suitable for handling such delay systems. Parameter estimation was performed by minimizing the discrepancy between the model-predicted and observed TB incidence using MATLAB's `fminsearch` routine, which implements the Nelder-Mead simplex algorithm. For the `fminsearch` optimization, the maximum number of function evaluations was set to  $4 \times 10^4$ , the maximum number of iterations to 2000, and the function and parameter tolerances were set to  $10^{-6}$ .

The model was fitted to observed annual TB incidence using the incidence flow into the infectious compartment, defined as

$$\mathcal{J}(t) = \rho_1 L_s(t) + \rho_2 L_f(t) + kR(t),$$

where new active TB cases arise from the slow- and fast-progressing latent classes, respectively, while relapse or reactivation cases originate from the recovered class. Accordingly, the fitted model output at observation time  $t_j$  is  $\mathcal{J}(t_j)$ .

The delay parameters  $\tau_1$  and  $\tau_2$  were fixed during parameter estimation because the available annual TB incidence data were insufficient for their reliable estimation. Baseline values of  $\tau_1 = 0.25$  years and  $\tau_2 = 0.08$  years were specified a priori to represent plausible funding-response and treatment-initiation delays, respectively, consistent with reported TB diagnosis and treatment delays in high-burden settings [28,39]. The influence of these delays on disease dynamics was subsequently explored through numerical simulations and Hopf bifurcation analyses over wider delay ranges.

The parameter starting values and bounds were specified a priori based on biological plausibility, model structure, and numerical considerations, and were used to constrain the optimization to feasible regions. The starting values and corresponding lower and upper bounds are reported in Table 5. The initial history was prescribed using the parameter-dependent history function, with  $S_0 = 5 \times 10^6$ ,  $L_s(0) = 5 \times 10^4$ ,  $L_f(0) = 2 \times 10^4$ ,  $I_0 = 10^4$ ,  $H_0 = 5 \times 10^3$ ,  $R_0 = 10^4$ ,  $F_0 = 1200$ , and  $P_0 = 1$ . These initial values were selected to provide a feasible initial population distribution among the epidemiological compartments and were used consistently throughout the numerical simulations.

The fitted model outputs, shown in Figure 4, were compared visually with the observed TB incidence, and the final parameter values from the estimation procedure are summarized in Table 5.

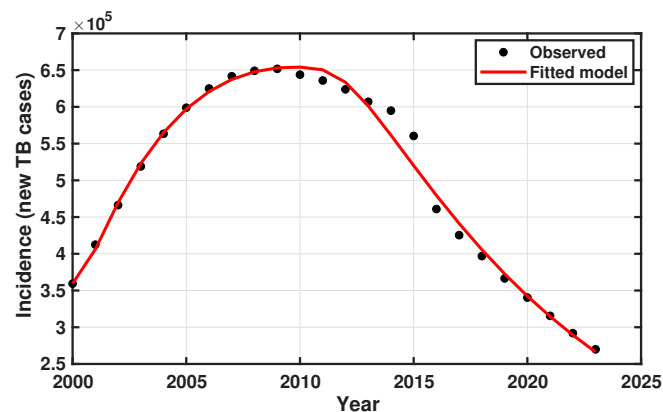


**Figure 4.** Model fit of the simulated TB incidence against the observed data from 2000–2023. The red curve represents the model-predicted incidence based on the estimated parameters, while the black circles denote the reported incidence cases. The goodness of fit was assessed using the residual sum of squares (RSS), root mean squared error (RMSE), mean absolute error (MAE), and coefficient of determination ( $R^2$ ), with values of  $\text{RSS} = 4.041 \times 10^9$ ,  $\text{RMSE} = 12975.77$ ,  $\text{MAE} = 8359.12$ , and  $R^2 = 0.9897$ .

**Table 5.** Starting values, parameter bounds, and final parameter values used for model calibration.

| Parameter  | Starting value       | Lower bound          | Upper bound          | Final value          | Source  |
|------------|----------------------|----------------------|----------------------|----------------------|---------|
| $\Pi$      | $2.0 \times 10^5$    | $1.0 \times 10^5$    | $5.0 \times 10^5$    | $4.1652 \times 10^5$ | Fitted  |
| $\mu$      | 0.0143               | 0.005                | 0.03                 | 0.0215               | Fitted  |
| $\beta$    | $1.0 \times 10^{-6}$ | $1.0 \times 10^{-8}$ | $1.0 \times 10^{-4}$ | $5.0 \times 10^{-5}$ | Fitted  |
| $\epsilon$ | 0.25                 | 0.001                | 3.0                  | 2                    | Fitted  |
| $q$        | 0.65                 | 0.40                 | 0.90                 | 0.46                 | Fitted  |
| $\eta$     | 0.12                 | 0.001                | 0.50                 | 0.046584             | Fitted  |
| $\rho_1$   | 0.02                 | 0.005                | 0.06                 | 0.01                 | Fitted  |
| $\rho_2$   | 0.04                 | 0.01                 | 0.1                  | 0.08                 | Fitted  |
| $\theta$   | 0.12                 | 0.02                 | 0.30                 | 0.24517              | Fitted  |
| $k$        | 0.015                | 0.001                | 0.05                 | 0.05                 | Fitted  |
| $\delta$   | 0.02                 | 0.001                | 0.08                 | 0.005                | Fitted  |
| $\gamma$   | 0.02                 | 0.005                | 0.08                 | 0.005                | Fitted  |
| $\omega$   | 0.01                 | 0.0001               | 0.10                 | 0.05                 | Fitted  |
| $\mu_f$    | 0.04                 | 0.005                | 0.50                 | 0.2972               | Fitted  |
| $\alpha_0$ | 0.03                 | 0.001                | 0.80                 | 0.5                  | Fitted  |
| $\alpha$   | $1.0 \times 10^{-5}$ | $1.0 \times 10^{-8}$ | $1.0 \times 10^{-2}$ | 0.0027               | Fitted  |
| $\mu_p$    | 0.02                 | 0.005                | 0.2                  | 0.08                 | Fitted  |
| $P_0$      | 0.5                  | 0.01                 | 5.0                  | 0.9                  | Fitted  |
| $F_0$      | —                    | —                    | —                    | 1200                 | Assumed |

The estimated parameter values in Table 5 are epidemiologically plausible and consistent with typical TB modeling ranges. The small transmission rate, realistic demographic and disease progression parameters, and meaningful intervention effects indicate that the calibrated model preserves biologically reasonable dynamics while accurately reproducing observed TB incidence trends.

#### 4.5. Time-delay induced dynamics and Hopf bifurcation analysis

Time delays are fundamental in shaping the dynamical behavior of TB transmission, influencing the stability of equilibria and the emergence of oscillatory patterns. To investigate these effects, we analyzed system (2.1) by treating the delays as bifurcation parameters under various scenarios. Specifically, we considered the funding delay ( $\tau_1$ ), treatment delay ( $\tau_2$ ), and their combined influence on the infected population.

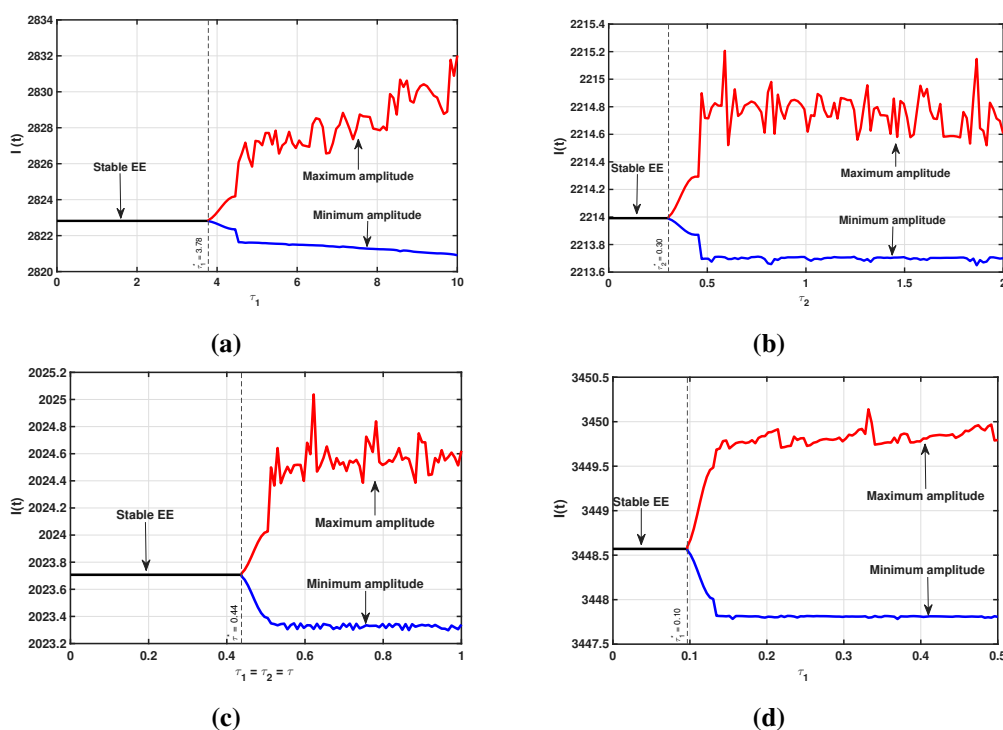


**Figure 5.** Bifurcation diagrams of system (2.1) illustrating the effect of time delays on the infected population. (a) Variation with  $\tau_1$  when  $\tau_2 = 0$ . (b) Variation with  $\tau_2$  when  $\tau_1 = 0$ . (c) Dynamics when  $\tau_1 = \tau_2 = \tau$ . (d) Variation with  $\tau_1$  for  $\tau_2 = 0.082 < \tau_2^*$ .

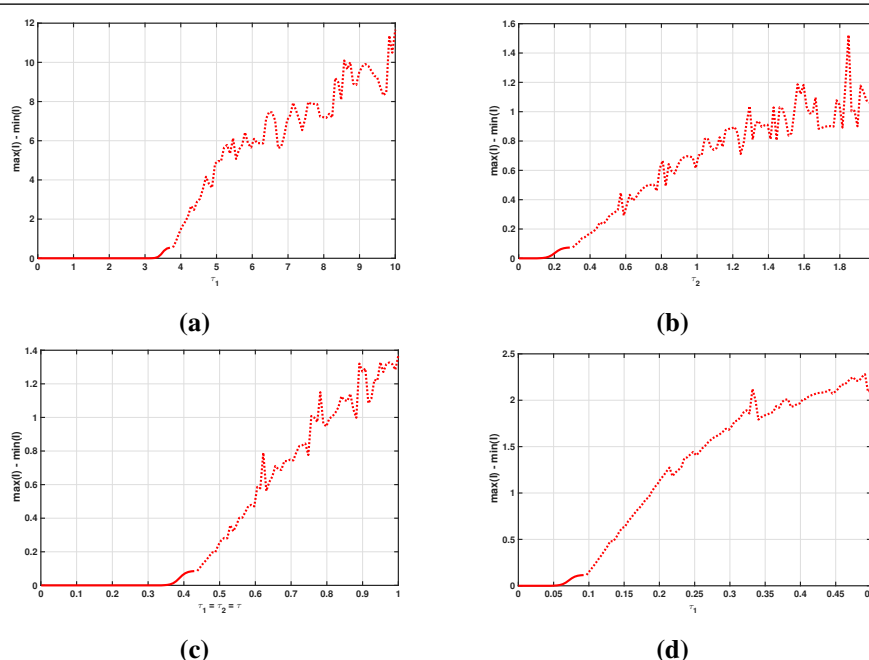


**Figure 6.** Global Hopf branches of system (2.1) with respect to the time delays, illustrating the transition from stable equilibria to oscillatory dynamics. (a) Branches for  $\tau_1 = \tau_1^* = 3.78$  with  $\tau_2 = 0$ , showing the onset of oscillations as  $\tau_1$  crosses its critical value. (b) Branches for  $\tau_2 = \tau_2^* = 0.3$  with  $\tau_1 = 0$ , highlighting the Hopf bifurcation due to  $\tau_2$ . (c) Branches for  $\tau = \tau^* = 0.44$  with  $\tau_1 = \tau_2 = \tau$ , representing the coupled effect of identical delays on the system's dynamics. (d) Branches for  $\tau_1 = \tau_1^* = 0.1$  and  $\tau_2 = 0.082 < \tau_2^*$ , showing how a subcritical  $\tau_2$  affects the emergence of oscillations.

Figure 5 illustrates the variation of the maximum and minimum amplitudes of the infected population as the delay parameters  $\tau_1$  and  $\tau_2$  increase. It is observed that before the critical threshold, the system remains at a stable endemic equilibrium. Once the delay exceeds the critical value, a Hopf bifurcation occurs, giving rise to sustained oscillations characterized by increasing maximum amplitude and decreasing minimum amplitude.

The small irregularities observed in the amplitude curves as the delay parameters vary do not indicate any loss of continuity of the limit cycle with respect to  $\tau_1$  or  $\tau_2$ . Instead, they arise from numerical and transient effects near the Hopf bifurcation threshold. In this regime, convergence to the periodic orbit becomes slow and highly sensitive to parameter perturbations, so that finite-time simulations and discretization may introduce apparent fluctuations in the computed extrema.

From the theoretical standpoint, classical Hopf bifurcation theory for delay differential equations [53] guarantees that the family of periodic solutions emerging from the bifurcation point depends continuously (indeed smoothly under standard non-degeneracy conditions) on the delay parameter in a neighborhood of the critical value. Therefore, the underlying limit cycle varies continuously with respect to  $\tau_1$  and  $\tau_2$ , and no discontinuity occurs. The observed non-smoothness is purely a numerical artifact associated with transient dynamics near the bifurcation point.

Figure 5(a)–(d) illustrate different scenarios: Variation of  $\tau_1$  with  $\tau_2 = 0$ , variation of  $\tau_2$  with  $\tau_1 = 0$ , identical delays ( $\tau_1 = \tau_2 = \tau$ ), and a subcritical combination of delays ( $\tau_1 > 0$ ,  $\tau_2 < \tau_2^*$ ), highlighting

how distinct delay configurations impact the onset of oscillatory behavior.

To gain a deeper understanding of the system's global dynamics, we extend this analysis to the global Hopf bifurcation behavior. By systematically tracking the branches of periodic solutions, Figure 6 demonstrates the transition from stable endemic equilibria to sustained oscillations as the delays cross their critical thresholds. The panels capture independent and coupled effects of  $\tau_1$  and  $\tau_2$ , illustrating how delay-induced feedback shapes the amplitude and persistence of oscillations. This comprehensive analysis provides valuable insights into the interplay between delays and their critical role in generating complex dynamical regimes in TB transmission.

#### 4.6. Qualitative effects of the treatment initiation delay

The treatment initiation delay  $\tau_2$  affects disease dynamics through the survival-adjusted treatment rate

$$\tilde{\gamma}(\tau_2) = \gamma e^{-d_2 \tau_2},$$

which decreases as  $\tau_2$  increases. Consequently, infectious individuals remain untreated for longer, leading to a higher infectious burden.

Although the model structure remains unchanged, the characteristic equation coefficients and the delay-dependent terms are influenced by  $\tau_2$  through  $\tilde{\gamma}(\tau_2)$ . As a result, variations in  $\tau_2$  can modify the endemic equilibrium level, convergence rate, and stability properties. To examine these effects,  $\tau_2$  was varied over

$$\tau_2 \in \{0.01, 0.15, 0.25, 0.3, 0.5, 2, 2.5\},$$

while all other parameters were held at their baseline values. The resulting trajectories of the infectious population  $I(t)$  are shown in Figure 7. For small delays, the infectious population converges to a stable endemic equilibrium. As  $\tau_2$  increases, the equilibrium infectious burden rises, and convergence becomes progressively weaker, reflecting the reduction in the effective treatment rate.

As  $\tau_2$  approaches the critical value  $\tau_2^*$  identified from the Hopf bifurcation analysis in Figure 5(b), oscillatory behavior becomes more pronounced, and the endemic equilibrium loses stability. At  $\tau_2 = \tau_2^*$ , a pair of complex conjugate eigenvalues crosses the imaginary axis, satisfying the Hopf bifurcation condition. Thus, treatment initiation delays can induce qualitative changes in stability rather than only quantitative changes in disease burden.

For delays exceeding the critical value, the endemic equilibrium becomes unstable, and the numerical trajectories display increasingly pronounced oscillations in the infectious population. These results indicate that sufficiently long treatment initiation delays can substantially alter epidemic dynamics.

Overall, the analysis shows that  $\tau_2$  has quantitative and qualitative effects on disease dynamics. Shorter treatment initiation delays are associated with lower infectious burden and stable convergence to the endemic equilibrium, whereas increasing  $\tau_2$  weakens treatment effectiveness and can destabilize the endemic equilibrium. This underscores the importance of timely treatment initiation for reducing transmission and maintaining stable disease dynamics.

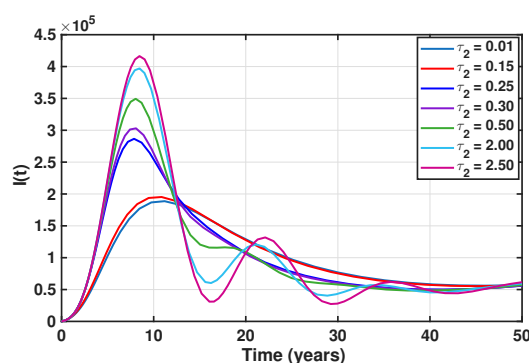


**Figure 7.** Time-series trajectories of the infectious population  $I(t)$  for different treatment initiation delays  $\tau_2$ , with  $\tau_1 = 0$ . The trajectories illustrate the progressive change in the epidemic dynamics as  $\tau_2$  increases, including the increasingly pronounced oscillatory behavior near the critical delay  $\tau_2^*$ .

#### 4.7. Sensitivity of critical delays (Hopf threshold) $\tau_1^*$ and $\tau_2^*$ to model parameters

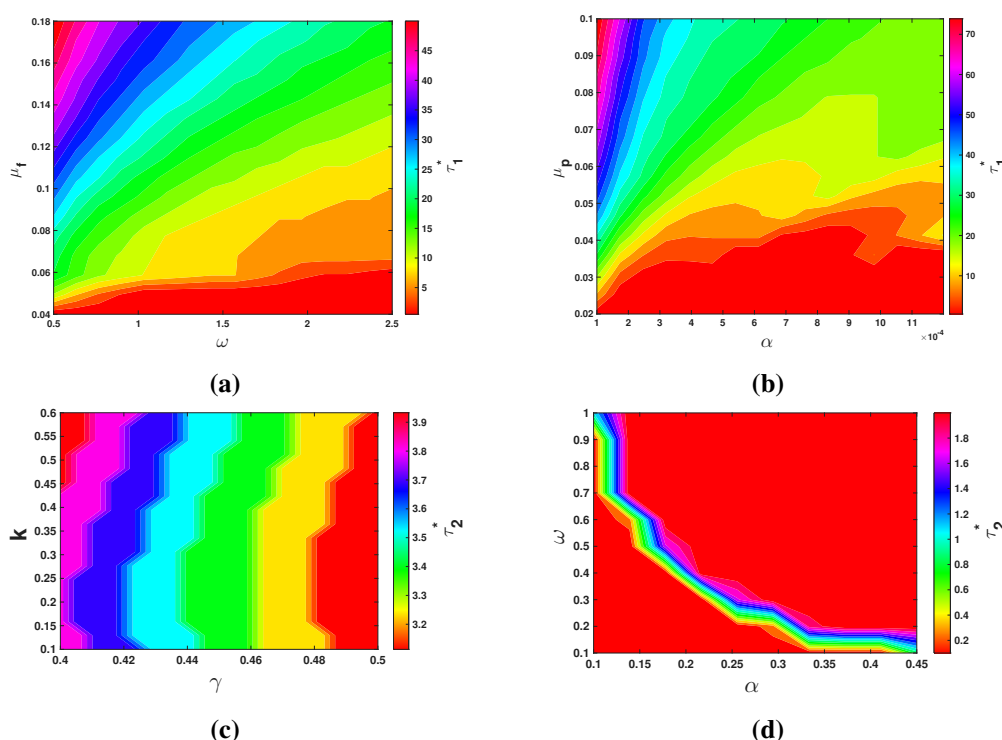


**Figure 8.** Contour plots showing the dependence of the critical delays  $\tau_1^*$  and  $\tau_2^*$  on key epidemiological and intervention parameters. (a)  $\tau_1^*$  versus funding usage rate ( $\mu_f$ ) and funding responsiveness ( $\omega$ ). (b)  $\tau_1^*$  versus prevention program decay rate ( $\mu_p$ ) and prevention program impact ( $\alpha$ ). (c)  $\tau_2^*$  versus relapse rate ( $k$ ) and treatment rate ( $\gamma$ ). (d)  $\tau_2^*$  versus funding responsiveness ( $\omega$ ) and prevention program impact ( $\alpha$ ).

Understanding how model parameters influence the onset of oscillatory dynamics is essential for designing effective TB control strategies. In this section, a sensitivity analysis of the critical delays  $\tau_1^*$  and  $\tau_2^*$  was conducted with respect to key epidemiological and intervention parameters. The analysis explored how variations in funding, prevention effectiveness, treatment rate, and relapse affect system stability. By examining the contour plots of  $\tau_1^*$  and  $\tau_2^*$  across parameter combinations, we identify the factors that most strongly enhance or weaken the system's ability to tolerate delays, providing valuable insights into the mechanisms that drive stability in TB transmission dynamics.

Figure 8 presents four contour plots illustrating the sensitivity of the critical delays,  $\tau_1^*$  and  $\tau_2^*$ , to variations in key epidemiological and intervention factors within the TB model. Figure 8(a) reveals that when the availability of financial resources responds more effectively to disease burden, the system exhibits greater stability, enabling longer critical delays, whereas rapid depletion of available resources reduces the delay threshold and destabilizes the system. Figure 8(b) shows that improved effectiveness and persistence of prevention programs enhance system stability by extending the critical delay, indicating the importance of sustained preventive interventions. In Figure 8(c), the results demonstrate that faster initiation of treatment strengthens system resilience, while increased relapse tendencies weaken stability by shortening the allowable delay. Finally, Figure 8(d) highlights that both responsive funding and strong preventive measures jointly enhance stability, leading to higher critical delay thresholds. Collectively, these findings indicate that stability is reinforced by effective prevention, adaptive funding, and timely treatment, whereas rapid resource depletion, weak prevention, and high relapse diminish the system's capacity to tolerate delays. Overall, the results emphasize that sustained prevention, flexible funding, and prompt treatment initiation are vital for maintaining long-term stability and optimizing TB control strategies.

The bifurcation analysis yields epidemiologically meaningful delay thresholds. For treatment initiation delay with  $\tau_1 = 0$ , the critical value is  $\tau_2^* = 0.3$  years (about 3.6 months), which aligns with reported TB diagnosis and treatment delays of 1 to 4 months in high-burden sub-Saharan African settings [28, 39]. For funding response delay with  $\tau_2 = 0$ , the critical threshold  $\tau_1^* = 3.78$  years represents the maximum delay in funding response that preserves system stability and is consistent with the multi-year funding and implementation cycles of donor-supported TB programs [54]. When both delays occur simultaneously ( $\tau_1 = \tau_2 = \tau^* = 0.44$  years), stability is lost more rapidly, demonstrating the compounded destabilizing effects of concurrent funding and treatment delays. These thresholds are epidemiologically plausible and underscore the need to minimize clinical and administrative delays to prevent oscillatory TB dynamics.

#### 4.8. Projection of the infectious population and funding dynamics

In this subsection, we examined the simulated trajectories of the infectious population ( $I$ ) and the financial resource variable ( $F$ ) obtained from the baseline delay-differential model. The analysis explored how the introduction of time delays in financial response ( $\tau_1 = 0.25$ ) and treatment initiation ( $\tau_2 = 0.08$ ) alters the evolution of disease transmission and resource allocation over time.

Figure 9 shows that the infectious population ( $I$ ) and funding variable ( $F$ ) initially increase, reaching a peak before gradually declining over time. The observed transient peak reflects the delayed response in funding allocation and treatment initiation, which temporarily enables the infection and resource accumulation to grow before corrective effects take place. As time progresses, the system stabilizes

toward a lower level, indicating that while delays do not induce sustained oscillations in this scenario, they significantly influence the transient behavior and peak burden of the disease. Overall, the results highlight the importance of timely intervention in reducing peak infection levels and improving the efficiency of funding responses.

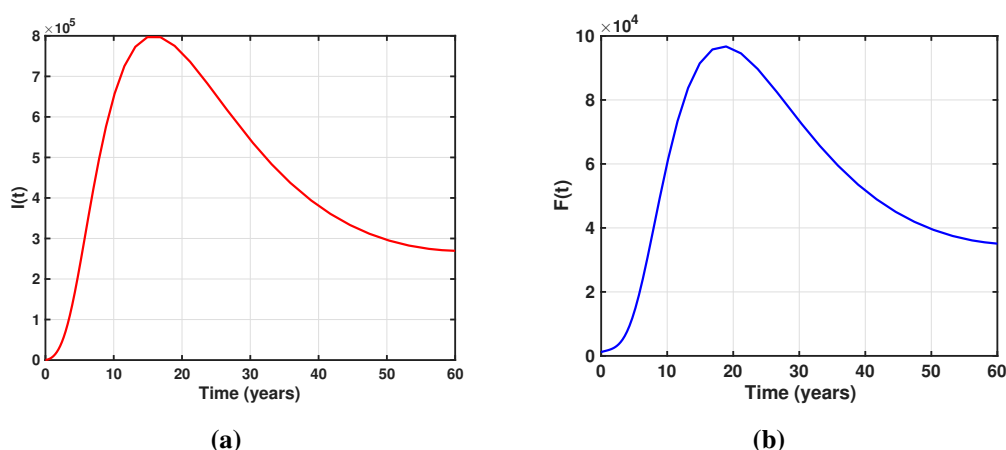


**Figure 9.** Model projection showing the temporal evolution of the infectious population ( $I$ ) and financial resources ( $F$ ) under feedback delays in funding response ( $\tau_1 = 0.25$ ) and treatment initiation ( $\tau_2 = 0.08$ ). The results exhibit an initial rise followed by a pronounced peak and a gradual decline toward a lower long-term level, highlighting the impact of delays on transient dynamics and system adjustment.

#### 4.9. Comparative effects of financial and treatment delays on the infectious population

In this subsection, we investigated the comparative influence of the financial response delay ( $\tau_1$ ) and the treatment initiation delay ( $\tau_2$ ) on the infectious population. In Figure 10(a),  $\tau_1$  is varied while  $\tau_2$  is kept constant to assess how delayed financial mobilization affects disease progression. Conversely, Figure 10(b) explores the effect of increasing  $\tau_2$  while maintaining a fixed  $\tau_1$ , capturing the consequences of slower treatment initiation. Together, these analyses reveal how both delays contribute to the persistence and intensity of infection, emphasizing the importance of timely interventions in controlling disease spread.

The results presented in Figure 10 demonstrate that increasing either of the delay parameters amplifies the infectious population and prolongs the epidemic period. As shown in Figure 10(a), longer financial response delays ( $\tau_1$ ) lead to higher infection peaks, indicating that slow mobilization of funds weakens the immediate response capacity. In Figure 10(b), increasing the treatment initiation delay ( $\tau_2$ ) produces a similar but more direct effect on infection levels, as delayed access to treatment allows infectious individuals to remain in circulation for extended periods. These findings highlight that minimizing both financial and treatment delays is crucial for effective TB control and the timely reduction of disease burden.

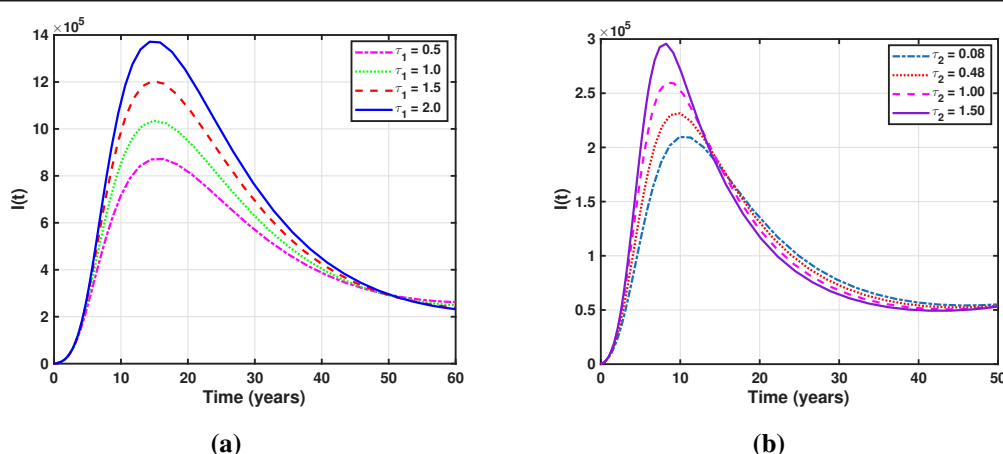


**Figure 10.** Comparative influence of financial response delay ( $\tau_1$ ) (a) and treatment delay ( $\tau_2$ ) (b) on the infectious population. Both analyzes highlight that longer delays intensify infection peaks and prolong disease persistence, underscoring the importance of prompt control actions.

#### 4.10. Impact of delays and control parameters on the infectious population

In this subsection, we examined how temporal delays and control measures jointly influence the infectious population. Heatmap analyzes were used to illustrate how variations in the delays and control parameters affect the peak number of infectious individuals. These results highlight the interplay between intervention timing and effectiveness in shaping disease dynamics.

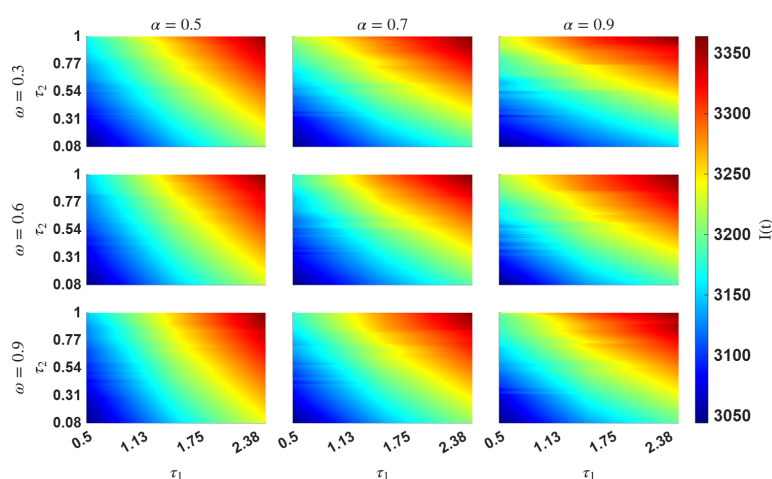


**Figure 11.** Heatmap grid showing the maximum infectious population ( $I$ ) as a function of the delays  $\tau_1$  and  $\tau_2$  across values of the control parameters  $\alpha$  and  $\omega$ . Each tile represents the peak  $I$  for a specific combination of  $\alpha$  and  $\omega$ , illustrating how variations in intervention timing and strength interact to influence disease dynamics.

Figure 11 reveals that the peak infectious population is highly sensitive to the temporal delays and

the strength of control measures. Longer delays in financial response ( $\tau_1$ ) and treatment initiation ( $\tau_2$ ) generally lead to higher peak infection levels, reflecting the detrimental effect of slow intervention. Conversely, higher values of the control parameters ( $\alpha$  and  $\omega$ ) substantially reduce the peak number of infectious individuals, indicating that stronger prevention efforts and faster funding mobilization can effectively mitigate the outbreak. Overall, the figure highlights a clear trade-off between delay duration and intervention intensity, emphasizing that timely responses combined with adequate control measures are crucial for minimizing disease burden.

#### 4.11. Effect of financial and preventive decay parameters in the presence of delayed interventions

In this subsection, we explored how variations in the financial and preventive decay parameters influence the progression of the infectious population when response delays are present. Specifically, the decay rates  $\mu_f$  and  $\mu_p$  determine how quickly financial support and preventive measures diminish over time, while the delays  $\tau_1$  and  $\tau_2$  represent the lag in financial and treatment responses, respectively. Understanding the combined influence of these parameters is essential, as rapid decay in resources or extended delays can weaken control efforts and amplify disease transmission dynamics.

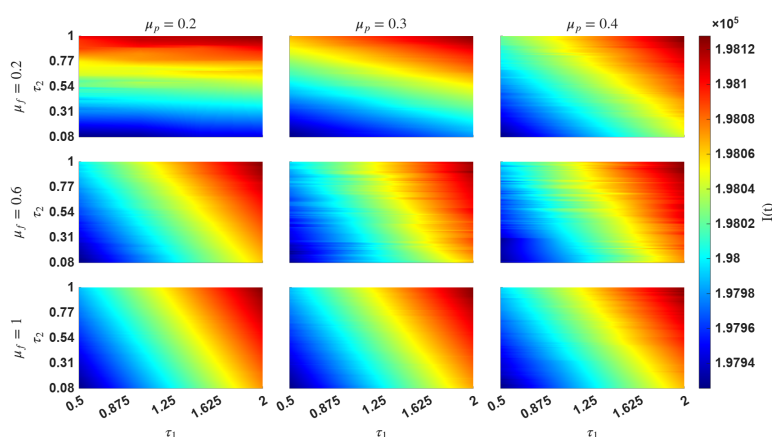


**Figure 12.** Heatmap illustrating the influence of the financial decay rate ( $\mu_f$ ) and preventive decay rate ( $\mu_p$ ) on the infectious population under varying delay conditions ( $\tau_1$ ,  $\tau_2$ ). The results show that higher  $\mu_f$  values combined with shorter delays correspond to lower infection levels, highlighting the importance of rapid financial responsiveness and sustained preventive efforts in mitigating disease spread.

The heatmap in Figure 12 illustrates the combined influence of the financial and preventive decay rates ( $\mu_f$ ,  $\mu_p$ ) under varying delay conditions ( $\tau_1$ ,  $\tau_2$ ). The results show that when the decay rates increase moderately, the infectious population experiences a slight increase, likely due to more efficient short-term use of available resources. However, as  $\mu_f$  and  $\mu_p$  continue to rise, representing a faster depletion of financial and preventive capacities, infection levels begin to increase, especially in the presence of longer response delays. This pattern highlights that excessive loss of resources or delayed interventions weakens control effectiveness, allowing the infection to rebound. Overall, the results underscore the importance of maintaining stable funding and prevention efforts, while minimizing

financial and treatment delays, to achieve sustained epidemic suppression.

## 5. Discussion and conclusions

In this study, we developed a delay-differential TB transmission model that integrates prevention efforts, treatment processes, and funding dynamics to enhance understanding of TB control mechanisms. By incorporating two key time delays, the funding response delay ( $\tau_1$ ) and the treatment initiation delay ( $\tau_2$ ), the model captures realistic temporal gaps often present in public health systems. Calibration using South African TB incidence data (2000–2023) showed strong agreement between model predictions and observed trends, confirming that the model accurately reflects the real-world behavior of TB transmission and control.

The analysis demonstrated that both time delays critically influence system stability. When delays are short, the TB burden converges to a stable endemic equilibrium. However, when either delay exceeds a critical threshold, the system experiences a Hopf bifurcation, leading to sustained oscillations that resemble periodic TB outbreaks. The treatment delay ( $\tau_2$ ) had a more destabilizing effect than the funding delay ( $\tau_1$ ), highlighting the importance of rapid diagnosis and timely treatment initiation for effective TB control. These findings emphasize that responsiveness in treatment delivery can substantially improve epidemic stability.

Sensitivity analysis further revealed that prevention efficiency ( $\alpha$ ), treatment rate ( $\gamma$ ), and funding responsiveness ( $\omega$ ) are the most influential parameters in reducing infection levels. Higher values of these parameters suppressed disease transmission, while larger depletion rates of prevention and funding programs ( $\mu_p, \mu_f$ ) or longer delays increased infection levels. This illustrates that enhancing the speed and consistency of prevention and treatment activities is as critical as expanding their coverage.

From a policy perspective, the model highlights that slow institutional responses and administrative bottlenecks can unintentionally amplify TB transmission. The transition from a stable equilibrium to oscillatory dynamics demonstrates how fragile TB control can become when health systems are slow to adapt. Maintaining consistent funding flows, improving program responsiveness, and shortening diagnostic delays should therefore be prioritized in high-burden regions. In addition, strengthening monitoring and evaluation systems can help detect early signs of delay-induced instability and guide timely corrective actions.

Despite its strengths, the model has several limitations. It assumes homogeneous mixing and does not account for spatial heterogeneity in TB transmission and intervention coverage, although spatially structured models may better capture regional variation in disease dynamics and healthcare access [55,56]. In addition, some parameters were estimated from aggregated annual data, which may limit precision and the representation of short-term fluctuations. The funding-response and treatment-initiation delays were treated as fixed constants despite likely variability across individuals, regions, and time periods. Furthermore, the deterministic framework does not capture stochastic effects or external disruptions that may influence TB transmission, healthcare access, and funding mechanisms.

In future work, researchers should incorporate spatial structure, population heterogeneity, and stochastic effects to better represent TB transmission dynamics. Adaptive or distributed delay formulations could more realistically capture variability in treatment-seeking, diagnosis, treatment initiation, and funding responses. Additional extensions may include socioeconomic and behavioral

factors, as well as optimal control and economic analyzes, to identify cost-effective intervention strategies and support geographically targeted, data-driven TB control policies in high-burden settings.

### Conflict of interest

The authors declare that there is no conflict of interest concerning the publication of this article.

### Data availability statement

The data used in the manuscript can be obtained from (<https://data.worldbank.org/indicator/SP.POP.TOTL?locations=ZA>) and (<https://data.worldbank.org/indicator/SH.TBS.INCD?end=2023&locations=ZA&start=2000>). In addition, the MATLAB codes and supporting materials will be made available at [https://github.com/veremachele/TB-Delayed\\_model\\_funding\\_dynamics](https://github.com/veremachele/TB-Delayed_model_funding_dynamics).

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### Authors' contributions

All authors contributed equally to the writing of this paper. All authors read and approved the final manuscript.

### Use of AI tools declaration

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

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## Appendix

### A. Derivation of the basic reproduction number $\mathcal{R}_0$

In this appendix, we present the detailed derivation of the basic reproduction number  $\mathcal{R}_0$  for system (2.1). In particular, the derivation is based on the next-generation matrix approach, and the

final expression is consistent with Eq (3.2) in the main text.

To compute  $\mathcal{R}_0$  for system (2.1), we employ the widely used next-generation matrix method, initially formalized by Diekmann et al. [57] and later generalized by van den Driessche and Watmough [58]. Evaluated at the disease-free equilibrium (DFE), given in Eq (3.1), the next-generation matrix is expressed as

$$\mathcal{FV}^{-1} = \left[ \frac{\partial \mathcal{F}_i(E_0)}{\partial x_j} \right] \times \left[ \frac{\partial \mathcal{V}_i(E_0)}{\partial x_j} \right]^{-1},$$

where  $\mathcal{F}_i$  represents the rate of appearance of new infections in compartment  $i$ , and  $\mathcal{V}_i$  denotes the net transfer of individuals into and out of compartment  $i$  through all other mechanisms. The spectral radius of this matrix,  $\rho(\mathcal{FV}^{-1})$ , defines  $\mathcal{R}_0$ .

To construct the matrices  $\mathcal{F}$  and  $\mathcal{V}$ , we focus on the infection-related (epidemiological) compartments of system (2.1), namely  $L_s, L_f, I, H$ , and  $R$ . In contrast, the variables  $F$  and  $P$  do not represent infected states; therefore, following the framework in [58], they are evaluated at the disease-free equilibrium and treated as auxiliary parameters in the analysis.

Under these considerations, the infection-related subsystem is given by

$$\left. \begin{aligned} \frac{dL_s}{dt} &= q\lambda_i S - \eta\lambda_i L_s - d_0 L_s, \\ \frac{dL_f}{dt} &= (1-q)\lambda_i S + \eta\lambda_i L_s - d_1 L_f, \\ \frac{dI}{dt} &= \rho_1 L_s + \rho_2 L_f + kR - d_2 I - \gamma e^{-d_2 \tau_2} I(t - \tau_2), \\ \frac{dH}{dt} &= \gamma e^{-d_2 \tau_2} I(t - \tau_2) - d_3 H, \\ \frac{dR}{dt} &= \theta H - d_4 R. \end{aligned} \right\} \quad (\text{A.1})$$

Based on this subsystem, the matrices representing new infection terms and transition terms, denoted respectively by  $\mathcal{F}$  and  $\mathcal{V}$ , are constructed as follows:

$$\mathcal{F} = \begin{pmatrix} 0 & 0 & \frac{\beta\mu_f\mu_p\Pi q}{\alpha\mu\epsilon F_0 + \mu\mu_f((\alpha_0 + P_0)\epsilon + \mu_p)} & 0 & 0 \\ 0 & 0 & \frac{\beta\mu_f\mu_p\Pi(1-q)}{\alpha\mu\epsilon F_0 + \mu\mu_f((\alpha_0 + P_0)\epsilon + \mu_p)} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \quad \text{and} \quad \mathcal{V} = \begin{pmatrix} d_0 & 0 & 0 & 0 & 0 \\ 0 & d_1 & 0 & 0 & 0 \\ -\rho_1 & -\rho_2 & d_2 + \gamma e^{-d_2 \tau_2} & 0 & -k \\ 0 & 0 & -\gamma e^{-d_2 \tau_2} & d_3 & 0 \\ 0 & 0 & 0 & -\theta & d_4 \end{pmatrix}.$$

Therefore, the inverse of  $\mathcal{V}$  is given by

$$\mathcal{V}^{-1} = \begin{pmatrix} \frac{1}{d_0} & 0 & 0 & 0 & 0 \\ 0 & \frac{1}{d_1} & 0 & 0 & 0 \\ \frac{d_3 d_4 \rho_1}{d_0 \Delta} & \frac{d_3 d_4 \rho_2}{d_1 \Delta} & \frac{d_3 d_4}{\Delta} & \frac{\theta k}{\Delta} & \frac{d_3 k}{\Delta} \\ \frac{\tilde{\gamma} d_4 \rho_1}{d_0 \Delta} & \frac{\tilde{\gamma} d_4 \rho_2}{d_1 \Delta} & \frac{\tilde{\gamma} d_4}{\Delta} & \frac{d_4(\tilde{\gamma} + d_2)}{\Delta} & \frac{\tilde{\gamma} k}{\Delta} \\ \frac{\tilde{\gamma} \theta \rho_1}{d_0 \Delta} & \frac{\tilde{\gamma} \theta \rho_2}{d_1 \Delta} & \frac{\tilde{\gamma} \theta}{\Delta} & \frac{\theta(\tilde{\gamma} + d_2)}{\Delta} & \frac{d_3(\tilde{\gamma} + d_2)}{\Delta} \end{pmatrix},$$

where

$$\Delta = d_3 d_4 (\tilde{\gamma} + d_2) - \tilde{\gamma} \theta k \quad \text{and} \quad \tilde{\gamma} = \gamma e^{-d_2 \tau_2}.$$

Next, we compute the product  $\mathcal{FV}^{-1}$ , given by

$$\mathcal{FV}^{-1} = \begin{pmatrix} 0 & 0 & \frac{\beta \mu_f \mu_p \Pi q}{\alpha \mu \epsilon F_0 + \mu \mu_f ((\alpha_0 + P_0) \epsilon + \mu_p)} & 0 & 0 \\ 0 & 0 & \frac{\beta \mu_f \mu_p \Pi (1-q)}{\alpha \mu \epsilon F_0 + \mu \mu_f ((\alpha_0 + P_0) \epsilon + \mu_p)} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \times \begin{pmatrix} \frac{1}{d_0} & 0 & 0 & 0 & 0 \\ 0 & \frac{1}{d_1} & 0 & 0 & 0 \\ \frac{d_3 d_4 \rho_1}{d_0 \Delta} & \frac{d_3 d_4 \rho_2}{d_1 \Delta} & \frac{d_3 d_4}{\Delta} & \frac{\theta k}{\Delta} & \frac{d_3 k}{\Delta} \\ \frac{\tilde{\gamma} d_4 \rho_1}{d_0 \Delta} & \frac{\tilde{\gamma} d_4 \rho_2}{d_1 \Delta} & \frac{\tilde{\gamma} d_4}{\Delta} & \frac{d_4(\tilde{\gamma} + d_2)}{\Delta} & \frac{\tilde{\gamma} k}{\Delta} \\ \frac{\tilde{\gamma} \theta \rho_1}{d_0 \Delta} & \frac{\tilde{\gamma} \theta \rho_2}{d_1 \Delta} & \frac{\tilde{\gamma} \theta}{\Delta} & \frac{\theta(\tilde{\gamma} + d_2)}{\Delta} & \frac{d_3(\tilde{\gamma} + d_2)}{\Delta} \end{pmatrix}.$$

By computing the eigenvalues of  $\mathcal{FV}^{-1}$ , we obtain

$$\left( 0, 0, 0, 0, \frac{\beta \mu_f \mu_p \Pi (q \rho_1 d_1 + (1-q) \rho_2 d_0)}{d_0 d_1 \mu (1 - \xi_1) (\tilde{\gamma} + d_2) (\mu_f ((\alpha_0 + P_0) \epsilon + \mu_p) + \alpha \epsilon F_0)} \right).$$

Therefore, the basic reproduction number is given by the dominant eigenvalue of  $\mathcal{FV}^{-1}$ , namely

$$\mathcal{R}_0 = \frac{\beta \mu_f \mu_p \Pi (q \rho_1 d_1 + (1-q) \rho_2 d_0)}{d_0 d_1 \mu (1 - \xi_1) (\tilde{\gamma} + d_2) (\mu_f ((\alpha_0 + P_0) \epsilon + \mu_p) + \alpha \epsilon F_0)}.$$

This expression can be rewritten as

$$\mathcal{R}_0 = \frac{\beta \Pi (q \rho_1 d_1 + (1-q) \rho_2 d_0)}{d_0 d_1 \mu (1 - \xi_1) (\tilde{\gamma} + d_2)} \Theta_0,$$

where

$$\xi_1 = \frac{\tilde{\gamma} \theta k}{d_3 d_4 (\tilde{\gamma} + d_2)}, \quad \text{and} \quad \Theta_0 = \frac{\mu_f \mu_p}{\mu_f ((\alpha_0 + P_0) \epsilon + \mu_p) + \alpha \epsilon F_0}.$$

## B. Proof of Theorem 1

In this section, we prove the local asymptotic stability of the disease-free equilibrium by reducing the linearized system to an infection-driven subsystem, deriving the characteristic equation with delays, and analyzing its roots using factorization, Descartes' rule of signs, and the Routh-Hurwitz criterion.

*Proof.* To analyze the local stability of the disease-free equilibrium, we focus on the infection-related compartments ( $L_s$ ,  $L_f$ ,  $I$ ,  $H$ ), since the non-infectious classes ( $R$ ,  $F$ ,  $P$ ) yield only negative eigenvalues at the DFE and thus do not affect the force of infection. Hence, the characteristic equation is obtained from the reduced  $4 \times 4$  system, which fully captures the transmission dynamics.

Let  $\tilde{\gamma} = \gamma e^{-d_2 \tau_2}$  and  $\mathbf{x} = (L_s, L_f, I, H)^\top \in \mathbb{R}^4$  denote the state vector of infection-related compartments. The linearized system with discrete delays  $\tau_1, \tau_2 \geq 0$  can be written as

$$\frac{d\mathbf{x}}{dt} = W_0 \mathbf{x}(t) + W_1 \mathbf{x}(t - \tau_1) + W_2 \mathbf{x}(t - \tau_2),$$

where  $W_0, W_1, W_2 \in \mathbb{R}^{4 \times 4}$  are given by

$$W_0 = \begin{pmatrix} -d_0 & 0 & \frac{\beta \mu_f \mu_p \Pi q}{\alpha \mu \epsilon F_0 + \mu \mu_f ((\alpha_0 + P_0) \epsilon + \mu_p)} & 0 \\ 0 & -d_1 & \frac{\beta \mu_f \mu_p \Pi (1 - q)}{\alpha \mu \epsilon F_0 + \mu \mu_f ((\alpha_0 + P_0) \epsilon + \mu_p)} & 0 \\ \rho_1 & \rho_2 & -d_2 - \tilde{\gamma} & 0 \\ 0 & 0 & \tilde{\gamma} & -d_3 \end{pmatrix},$$

$$W_1 = \mathbf{0}_{4 \times 4}, \quad W_2 = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & -\tilde{\gamma} & 0 \\ 0 & 0 & \tilde{\gamma} & 0 \end{pmatrix}.$$

The corresponding characteristic equation is

$$\det(W_0 + W_1 e^{-\hat{\lambda} \tau_1} + W_2 e^{-\hat{\lambda} \tau_2} - \hat{\lambda} I_4) = 0, \quad (\text{B.1})$$

where  $I_4$  is the  $4 \times 4$  identity matrix and  $\hat{\lambda} \in \mathbb{C}$  denotes the eigenvalues.

Equation (B.1) leads to the following characteristic polynomial

$$p(\hat{\lambda}) = (\hat{\lambda} + d_3 - \tilde{\gamma} e^{-\hat{\lambda} \tau_2}) (\hat{\lambda}^3 + \psi_1 \hat{\lambda}^2 + \psi_2 \hat{\lambda} + \psi_3 + (\tilde{\gamma} \hat{\lambda}^2 + \psi_4 \hat{\lambda} + \psi_5) e^{-\hat{\lambda} \tau_2}) = 0, \quad (\text{B.2})$$

with coefficients

$$\begin{aligned} \psi_1 &= \tilde{\gamma} + d_0 + d_1 + d_2, \\ \psi_2 &= d_1(\tilde{\gamma} + d_2) + d_0(\tilde{\gamma} + d_1 + d_2) - (Q_1 + Q_2), \\ \psi_3 &= \tilde{\gamma} d_0 d_1 + d_0 d_1 d_2 - (d_0 Q_1 + d_1 Q_2), \\ \psi_4 &= \tilde{\gamma}(d_0 + d_1), \quad \psi_5 = d_0 d_1, \end{aligned}$$

where

$$Q_1 = \frac{\beta \mu_f \mu_p \Pi (1 - q) \rho_2}{\alpha \mu \epsilon F_0 + \mu \mu_f ((\alpha_0 + P_0) \epsilon + \mu_p)},$$

$$Q_2 = \frac{\beta \mu_f \mu_p \Pi q \rho_1}{\alpha \mu \epsilon F_0 + \mu \mu_f ((\alpha_0 + P_0) \epsilon + \mu_p)}.$$

The eigenvalues are obtained from the two factors of (B.2):

$$\hat{\lambda} + d_3 - \tilde{\gamma}e^{-\hat{\lambda}\tau_2} = 0,$$

and

$$\hat{\lambda}^3 + \psi_1\hat{\lambda}^2 + \psi_2\hat{\lambda} + \psi_3 + (\tilde{\gamma}\hat{\lambda}^2 + \psi_4\hat{\lambda} + \psi_5)e^{-\hat{\lambda}\tau_2} = 0.$$

### Case 1: Analysis of the first factor.

Let  $\hat{\lambda} = iz$  ( $z > 0$ ). Substituting into the first factor and separating real and imaginary parts yields

$$d_3 = \tilde{\gamma} \cos(z\tau_2), \quad z = -\tilde{\gamma} \sin(z\tau_2).$$

Adding the squares gives

$$z^2 + (d_3^2 - \tilde{\gamma}^2) = 0.$$

By Descartes' rule of signs, if

$$d_3^2 - \tilde{\gamma}^2 > 0,$$

then the above equation admits no positive real root, and hence all roots of the first factor have negative real parts.

### Case 2: Analysis of the second factor.

For  $\hat{\lambda} = iz$ , separating real and imaginary parts leads to the system

$$\begin{aligned} -\psi_1 z^2 + \psi_3 &= -\psi_5 \cos(z\tau_2) + \tilde{\gamma} z^2 \cos(z\tau_2) - \psi_5 \sin(z\tau_2), \\ -z^3 + \psi_2 z &= -\psi_5 z \cos(z\tau_2) + \psi_5 \sin(z\tau_2) - \tilde{\gamma} z^2 \sin(z\tau_2). \end{aligned}$$

Eliminating trigonometric terms yields the polynomial

$$z^6 + (\psi_1^2 - 2\psi_2 - \tilde{\gamma}^2)z^4 + (\psi_2^2 + 2\psi_5\tilde{\gamma} - 2\psi_1\psi_3 - \psi_5)z^2 + (\psi_3^2 - \psi_5^2) = 0.$$

Setting  $n^* = z^2$ , this reduces to

$$g(n^*) = n^{*3} + a_1 n^{*2} + a_2 n^* + a_3 = 0,$$

with  $a_1 = \psi_1^2 - 2\psi_2 - \tilde{\gamma}^2$ ,  $a_2 = \psi_2^2 + 2\psi_5\tilde{\gamma} - 2\psi_1\psi_3 - \psi_5$ , and  $a_3 = \psi_3^2 - \psi_5^2$ .

By Descartes' rule of signs, if  $a_1 > 0$ ,  $a_2 > 0$ ,  $a_3 > 0$ , then no positive real root exists. Moreover, applying the Routh-Hurwitz criterion confirms that all roots of  $g(n^*)$  have negative real parts [59]. Thus, the eigenvalues associated with this factor also lie in the left half-plane.

### Case 3: When $\tau_2 = 0$ . Equation (B.2) reduces to

$$p(\hat{\lambda}) = (\hat{\lambda} + d_3 - \gamma)(\hat{\lambda}^3 + (\psi_1 + \gamma)\hat{\lambda}^2 + (\psi_2 + \psi_4)\hat{\lambda} + (\psi_3 + \psi_5)).$$

Since

$$\psi_3 + \psi_5 = d_0 d_1 (1 + (\gamma + d_2)(1 - \mathcal{R}_0)),$$

if  $\mathcal{R}_0 < 1$ , Descartes' rule of signs ensures that all roots have negative real parts, implying that  $E_0$  is locally asymptotically stable.

Finally, we observe that

$$\lim_{\hat{\lambda} \rightarrow -\infty} p(\hat{\lambda}) = +\infty, \quad p(0) = d_0 d_1 (1 + (\gamma + d_2)(1 - \mathcal{R}_0)).$$

When  $\mathcal{R}_0 > 1$ , we have  $p(0) < 0$ , and thus  $p(\hat{\lambda}) = 0$  admits a positive real root.

Hence,  $E_0$  is unstable when  $\mathcal{R}_0 > 1$ . □

### C. Characteristic polynomial coefficients

In this appendix, we present the coefficients of the characteristic polynomial associated with Eq (3.6). These coefficients are expressed in terms of the model parameters and are essential for analyzing the stability properties of the equilibrium points.

$$J_7 = d_1 - C_5 - C_1 + d_2 + d_3 + d_4 + \mu_1 + \mu_2,$$

$$J_6 = (d_1 + d_2 + d_3 + d_4)(d_1 + d_2 + d_3 + d_4 + \mu_1 + \mu_2) + \mu_1\mu_2 \\ - (C_1 + C_5)(d_1 + d_2 + d_3 + d_4 + \mu_1 + \mu_2) - C_6\rho_1 - C_{10}\rho_2 + C_1C_5,$$

$$J_5 = C_1C_5(d_1 + d_2 + d_3 + d_4 + \mu_1 + \mu_2) - C_1(d_1d_2 + d_1d_3 + d_1d_4 + d_2d_3 + d_2d_4 + d_3d_4) \\ - C_5(d_1d_2 + d_1d_3 + d_1d_4 + d_2d_3 + d_2d_4 + d_3d_4) - (C_1 + C_5)(d_1 + d_2 + d_3 + d_4)(\mu_1 + \mu_2) \\ - (C_1 + C_5)\mu_1\mu_2 - C_2C_4\rho_1 + C_1C_6\rho_1 - C_6(d_1 + d_3 + d_4 + \mu_1 + \mu_2)\rho_1 - C_2C_8\rho_2 \\ + (C_1 + C_5)C_{10}\rho_2 - C_{10}(d_3 + d_4 + \mu_1 + \mu_2)\rho_2 - C_6C_9\rho_2 + d_1d_2d_3 + d_1d_2d_4 + d_1d_3d_4 + d_2d_3d_4 \\ + (d_1d_2 + d_1d_3 + d_1d_4 + d_2d_3 + d_2d_4 + d_3d_4)(\mu_1 + \mu_2) + (d_1 + d_2 + d_3 + d_4)\mu_1\mu_2 - \tilde{\gamma}k\theta,$$

$$J_4 = \mu_1\mu_2(d_1d_2 + d_1d_3 + d_1d_4 + d_2d_3 + d_2d_4 + d_3d_4) - \tilde{\gamma}k\theta(d_1 + \mu_1 + \mu_2) \\ - C_2C_4C_9\rho_2 + C_2C_5C_8\rho_2 - C_1C_5C_{10}\rho_2 + C_1C_6C_9\rho_2 + C_1C_5(d_1d_2 + d_1d_3 + d_1d_4 + d_2d_3 \\ + d_2d_4 + d_3d_4 + d_1\mu_1 + d_1\mu_2 + d_2\mu_1 + d_2\mu_2 + d_3\mu_1 + d_3\mu_2 + d_4\mu_1 + d_4\mu_2 + \mu_1\mu_2) \\ - C_2C_4(d_1 + d_3 + d_4 + \mu_1 + \mu_2)\rho_1 + C_1C_6(d_1 + d_3 + d_4 + \mu_1 + \mu_2)\rho_1 \\ - C_2C_8(d_3 + d_4 + \mu_1 + \mu_2)\rho_2 + (C_1 + C_5)C_{10}(d_3 + d_4 + \mu_1 + \mu_2)\rho_2 \\ - C_6C_9(d_3 + d_4 + \mu_1 + \mu_2)\rho_2 - C_1(d_1d_2d_3 + d_1d_2d_4 + d_1d_3d_4 + d_2d_3d_4 + d_1d_2\mu_1 \\ + d_1d_2\mu_2 + d_1d_3\mu_1 + d_1d_3\mu_2 + d_1d_4\mu_1 + d_1d_4\mu_2 + d_2d_3\mu_1 + d_2d_3\mu_2 + d_2d_4\mu_1 + d_2d_4\mu_2 \\ + d_3d_4\mu_1 + d_3d_4\mu_2 + d_1\mu_1\mu_2 + d_2\mu_1\mu_2 + d_3\mu_1\mu_2 + d_4\mu_1\mu_2) - C_5(d_1d_2d_3 + d_1d_2d_4 + d_1d_3d_4 \\ + d_2d_3d_4 + d_1d_2\mu_1 + d_1d_2\mu_2 + d_1d_3\mu_1 + d_1d_3\mu_2 + d_1d_4\mu_1 + d_1d_4\mu_2 + d_2d_3\mu_1 + d_2d_3\mu_2 \\ + d_2d_4\mu_1 + d_2d_4\mu_2 + d_3d_4\mu_1 + d_3d_4\mu_2 + d_1\mu_1\mu_2 + d_2\mu_1\mu_2 + d_3\mu_1\mu_2 + d_4\mu_1\mu_2) \\ - C_6(d_1d_3 + d_1d_4 + d_3d_4 + d_1\mu_1 + d_1\mu_2 + d_3\mu_1 + d_3\mu_2 + d_4\mu_1 + d_4\mu_2 + \mu_1\mu_2)\rho_1 \\ - C_{10}(d_3d_4 + d_3\mu_1 + d_3\mu_2 + d_4\mu_1 + d_4\mu_2 + \mu_1\mu_2)\rho_2 \\ - C_7\alpha\omega\rho_1 - C_{11}\alpha\omega\rho_2 + (C_1 + C_5)\tilde{\gamma}k\theta + d_1d_2d_3d_4 - C_6\mu_1\mu_2\rho_1 - C_{10}\mu_1\mu_2\rho_2 \\ + (d_1d_2d_3 + d_1d_2d_4 + d_1d_3d_4 + d_2d_3d_4)(\mu_1 + \mu_2),$$

$$J_3 = (\mu_1 + \mu_2)(C_2C_5C_8\rho_2 - C_2C_4C_9\rho_2 - C_1C_5C_{10}\rho_2 + C_1C_6C_9\rho_2) \\ + C_1C_5(d_1d_2d_3 + d_1d_2d_4 + d_1d_3d_4 + d_2d_3d_4 + d_1d_2\mu_1 + d_1d_2\mu_2 + d_1d_3\mu_1 + d_1d_3\mu_2 \\ + d_1d_4\mu_1 + d_1d_4\mu_2 + d_2d_3\mu_1 + d_2d_3\mu_2 + d_2d_4\mu_1 + d_2d_4\mu_2 + d_3d_4\mu_1 + d_3d_4\mu_2 + d_1\mu_1\mu_2 \\ + d_2\mu_1\mu_2 + d_3\mu_1\mu_2 + d_4\mu_1\mu_2) \\ + (C_1C_6 - C_2C_4)(d_1d_3\rho_1 + d_1d_4\rho_1 + d_3d_4\rho_1 + d_1\mu_1\rho_1 + d_1\mu_2\rho_1 + d_3\mu_1\rho_1 + d_3\mu_2\rho_1 \\ + d_4\mu_1\rho_1 + d_4\mu_2\rho_1 + \mu_1\mu_2\rho_1) \\ + (C_1C_{10} + C_5C_{10} - C_2C_8 - C_6C_9)(d_3d_4\rho_2 + d_3\mu_1\rho_2 + d_3\mu_2\rho_2 + d_4\mu_1\rho_2 + d_4\mu_2\rho_2 + \mu_1\mu_2\rho_2) \\ + (C_1C_7 - C_3C_4)\alpha\omega\rho_1 + (C_1C_{11} + C_5C_{11} - C_3C_8 - C_7C_9)\alpha\omega\rho_2$$

$$\begin{aligned}
& -C_1C_5\tilde{\gamma}k\theta + (C_1 + C_5)d_1\tilde{\gamma}k\theta + (d_1d_2d_3d_4)(\mu_1 + \mu_2) - (d_1\tilde{\gamma}k\mu_1\theta + d_1\tilde{\gamma}k\mu_2\theta + \tilde{\gamma}k\mu_1\mu_2\theta) \\
& -C_6(d_1d_3d_4 + d_1\mu_1\mu_2 + d_3\mu_1\mu_2 + d_4\mu_1\mu_2)\rho_1 \\
& -C_{10}(d_3d_4 + d_3\mu_1 + d_3\mu_2 + d_4\mu_1 + d_4\mu_2 + \mu_1\mu_2)\rho_2,
\end{aligned}$$

$$\begin{aligned}
J_2 = & (C_2C_5C_8 - C_2C_4C_9 - C_1C_5C_{10} + C_1C_6C_9)d_3d_4\rho_2 \\
& + (C_2C_5C_8 - C_2C_4C_9 - C_1C_5C_{10} + C_1C_6C_9)(d_3 + d_4)(\mu_1 + \mu_2)\rho_2 \\
& + (C_1C_5)[d_1d_2d_3d_4 + d_1d_2d_3\mu_1 + d_1d_2d_3\mu_2 + d_1d_2d_4\mu_1 + d_1d_2d_4\mu_2 + d_1d_3d_4\mu_1 + d_1d_3d_4\mu_2 \\
& + d_2d_3d_4\mu_1 + d_2d_3d_4\mu_2 + d_1d_2\mu_1\mu_2 + d_1d_3\mu_1\mu_2 + d_1d_4\mu_1\mu_2 \\
& + d_2d_3\mu_1\mu_2 + d_2d_4\mu_1\mu_2 + d_3d_4\mu_1\mu_2] + (C_1C_6 - C_2C_4)[d_1d_3d_4\rho_1 + d_1d_3\mu_1\rho_1 \\
& + d_1d_3\mu_2\rho_1 + d_1d_4\mu_1\rho_1 + d_1d_4\mu_2\rho_1 + d_3d_4\mu_1\rho_1 + d_3d_4\mu_2\rho_1 \\
& + d_1\mu_1\mu_2\rho_1 + d_3\mu_1\mu_2\rho_1 + d_4\mu_1\mu_2\rho_1] \\
& + (C_1C_{10} + C_5C_{10} - C_2C_8 - C_6C_9)[d_3d_4\rho_2 + d_3\mu_1\rho_2 + d_3\mu_2\rho_2 + d_4\mu_1\rho_2 + d_4\mu_2\rho_2 + \mu_1\mu_2\rho_2] \\
& + (C_1C_7 - C_3C_4)\alpha d_1\omega\rho_1 + (C_1C_7 - C_3C_4)\alpha d_3\omega\rho_1 + (C_1C_7 - C_3C_4)\alpha d_4\omega\rho_1 \\
& + (C_1C_{11} + C_5C_{11} - C_3C_8 - C_7C_9)\alpha(d_3 + d_4)\omega\rho_2 \\
& - C_1C_5d_1\tilde{\gamma}k\theta + (C_1 + C_5)d_1\tilde{\gamma}k(\mu_1 + \mu_2)\theta + (C_1 + C_5)\tilde{\gamma}k\mu_1\mu_2\theta \\
& + d_1d_2d_3d_4(\mu_1 + \mu_2) + d_1d_2d_3d_4\mu_1\mu_2 - d_1\tilde{\gamma}k\mu_1\mu_2\theta,
\end{aligned}$$

$$\begin{aligned}
J_1 = & (C_1 - C_1C_5 + C_5)d_1\tilde{\gamma}k\mu_1\mu_2\theta - (C_1 + C_5 + C_6)d_1d_2d_3d_4\mu_1\mu_2 - C_7\alpha d_1d_3d_4\omega\rho_1 \\
& - C_6d_1d_3d_4\mu_1\mu_2\rho_1 + (C_1C_7 - C_3C_4)\alpha(d_1d_3 + d_1d_4 + d_3d_4)\omega\rho_1 \\
& + (-C_3C_4C_9 + C_3C_5C_8 - C_1C_5C_{11} + C_1C_7C_9)\alpha(d_3 + d_4)\omega\rho_2 \\
& + (-C_2C_4C_9 + C_2C_5C_8 - C_1C_5C_{10} + C_1C_6C_9)d_3d_4(\mu_1 + \mu_2)\rho_2 \\
& + (-C_2C_4C_9 + C_2C_5C_8 - C_1C_5C_{10} + C_1C_6C_9)(d_3 + d_4)\mu_1\mu_2\rho_2 \\
& + (C_1C_5)[d_1d_2d_3d_4(\mu_1 + \mu_2) + d_1d_2d_3\mu_1\mu_2 + d_1d_2d_4\mu_1\mu_2 + d_1d_3d_4\mu_1\mu_2 + d_2d_3d_4\mu_1\mu_2] \\
& + (C_1C_{10} + C_5C_{10} - C_2C_8 - C_6C_9)d_3d_4\mu_1\mu_2\rho_2 - C_1C_5d_1\tilde{\gamma}k(\mu_1 + \mu_2)\theta,
\end{aligned}$$

$$\begin{aligned}
J_0 = & \alpha\omega[(C_3C_5C_8 - C_3C_4C_9 - C_1C_5C_{11} + C_1C_7C_9)d_3d_4\rho_2 + (-C_3C_4 + C_1C_7)d_1d_3d_4\rho_1] \\
& + d_3d_4\mu_1\mu_2(-C_2C_4C_9 + C_2C_5C_8 - C_1C_5C_{10} + C_1C_6C_9)\rho_2 \\
& + d_1d_3d_4\mu_1\mu_2(-C_2C_4 + C_1C_6)\rho_1 + C_1C_5d_1d_2d_3d_4\mu_1\mu_2 - C_1C_5d_1\tilde{\gamma}k\mu_1\mu_2\theta,
\end{aligned}$$

$$M_4 = -\alpha\omega(C_7\rho_1 + C_{11}\rho_2),$$

$$\begin{aligned}
M_3 = & -\alpha\omega[(C_3C_4 - C_1C_7 + C_7)\rho_1 + (C_3C_8 - C_1C_{11} - C_5C_{11} + C_7C_9 + C_{11})\rho_2 \\
& + C_7(d_1 + d_3 + d_4)\rho_1 + C_{11}(d_3 + d_4)\rho_2],
\end{aligned}$$

$$\begin{aligned}
M_2 = & -\alpha\omega[(C_3C_4C_9 - C_3C_5C_8 + C_1C_5C_{11} - C_1C_7C_9)\rho_2 \\
& + (C_3C_4d_1 - C_1C_7d_1 + C_3C_4d_3 - C_1C_7d_3 + C_3C_4d_4 - C_1C_7d_4)\rho_1 \\
& + (C_3C_8d_3 - C_1C_{11}d_3 + C_3C_8d_4 - C_1C_{11}d_4 - C_5C_{11}d_3 + C_7C_9d_3 - C_5C_{11}d_4 + C_7C_9d_4)\rho_2 \\
& + C_7(d_1d_3 + d_1d_4 + d_3d_4)\rho_1 + C_{11}d_3d_4\rho_2],
\end{aligned}$$

$$M_1 = -\alpha d_3\omega[C_3C_4C_9\rho_2 - C_3C_5C_8\rho_2 + C_1C_5C_{11}\rho_2 - C_1C_7C_9\rho_2 + C_3C_4d_1\rho_1 - C_1C_7d_1\rho_1$$

$$\begin{aligned}
& + C_3 C_4 d_4 \rho_1 - C_1 C_7 d_4 \rho_1 + C_3 C_8 d_4 \rho_2 - C_1 C_{11} d_4 \rho_2 - C_5 C_{11} d_4 \rho_2 + C_7 C_9 d_4 \rho_2 + C_7 d_1 d_4 \rho_1 \Big] \\
& - \alpha d_4 \omega \Big[ C_3 C_4 C_9 \rho_2 - C_3 C_5 C_8 \rho_2 + C_1 C_5 C_{11} \rho_2 - C_1 C_7 C_9 \rho_2 d + C_3 C_4 d_1 \rho_1 - C_1 C_7 d_1 \rho_1 \Big], \\
M_0 = & -\alpha d_3 d_4 \omega \Big( C_3 C_4 C_9 \rho_2 - C_3 C_5 C_8 \rho_2 + C_1 C_5 C_{11} \rho_2 - C_1 C_7 C_9 \rho_2 + C_3 C_4 d_1 \rho_1 - C_1 C_7 d_1 \rho_1 \Big),
\end{aligned}$$

$$N_7 = \tilde{\gamma},$$

$$N_6 = \tilde{\gamma} (d_1 - C_5 - C_1 + d_3 + d_4 + \mu_1 + \mu_2),$$

$$\begin{aligned}
N_5 = & \tilde{\gamma} \Big( d_1 d_3 - C_1 (d_1 + d_3 + d_4 + \mu_1 + \mu_2) - k\theta + \mu_1 (d_1 + d_3 + d_4) - C_5 (d_1 - C_1 + d_3 + d_4 + \mu_1 + \mu_2) \\
& + \mu_2 (d_1 + d_3 + d_4 + \mu_1) + d_4 (d_1 + d_3) \Big),
\end{aligned}$$

$$\begin{aligned}
N_4 = & \tilde{\gamma} \Big[ \mu_1 (d_1 d_3 + d_4 (d_1 + d_3)) - C_5 (d_1 d_3 - C_1 (d_1 + d_3 + d_4 + \mu_1 + \mu_2) + \mu_1 (d_1 + d_3 + d_4) \\
& + \mu_2 (d_1 + d_3 + d_4 + \mu_1) + d_4 (d_1 + d_3)) - C_1 (d_1 d_3 \\
& + \mu_1 (d_1 + d_3 + d_4) + \mu_2 (d_1 + d_3 + d_4 + \mu_1) + d_4 (d_1 + d_3)) \\
& + \mu_2 (d_1 d_3 + \mu_1 (d_1 + d_3 + d_4) + d_4 (d_1 + d_3)) - d_1 k\theta - k\mu_1 \theta - k\mu_2 \theta + C_1 k\theta + C_5 k\theta + d_1 d_3 d_4 \Big],
\end{aligned}$$

$$\begin{aligned}
N_3 = & \tilde{\gamma} \Big[ \mu_2 (\mu_1 (d_1 d_3 + d_4 (d_1 + d_3)) + d_1 d_3 d_4) + C_1 (d_1 k\theta + k\mu_1 \theta + k\mu_2 \theta) \\
& - C_1 (\mu_1 (d_1 d_3 + d_4 (d_1 + d_3)) + \mu_2 (d_1 d_3 + \mu_1 (d_1 + d_3 + d_4) + d_4 (d_1 + d_3)) + d_1 d_3 d_4) \\
& - \mu_2 (d_1 k\theta + k\mu_1 \theta) + C_5 (d_1 k\theta - C_1 k\theta + k\mu_1 \theta + k\mu_2 \theta) \\
& - C_5 (\mu_1 (d_1 d_3 + d_4 (d_1 + d_3)) - C_1 (d_1 d_3 + \mu_1 (d_1 + d_3 + d_4) + \mu_2 (d_1 + d_3 + d_4 + \mu_1) \\
& + d_4 (d_1 + d_3)) + \mu_2 (d_1 d_3 + \mu_1 (d_1 + d_3 + d_4) + d_4 (d_1 + d_3)) + d_1 d_3 d_4) + d_1 d_3 d_4 \mu_1 - d_1 k\mu_1 \theta \Big],
\end{aligned}$$

$$\begin{aligned}
N_2 = & \tilde{\gamma} \Big[ C_5 (\mu_2 (d_1 k\theta + k\mu_1 \theta) - C_1 (d_1 k\theta + k\mu_1 \theta + k\mu_2 \theta) + d_1 k\mu_1 \theta) \\
& - C_5 (\mu_2 (\mu_1 (d_1 d_3 + d_4 (d_1 + d_3)) + d_1 d_3 d_4) - C_1 (\mu_1 (d_1 d_3 + d_4 (d_1 + d_3)) + \mu_2 (d_1 d_3 \\
& + \mu_1 (d_1 + d_3 + d_4) + d_4 (d_1 + d_3)) + d_1 d_3 d_4) + d_1 d_3 d_4 \mu_1) \\
& - C_1 (\mu_2 (\mu_1 (d_1 d_3 + d_4 (d_1 + d_3)) + d_1 d_3 d_4) + d_1 d_3 d_4 \mu_1) \\
& + C_1 (\mu_2 (d_1 k\theta + k\mu_1 \theta) + d_1 k\mu_1 \theta) + d_1 d_3 d_4 \mu_1 \mu_2 - d_1 k\mu_1 \mu_2 \theta \Big],
\end{aligned}$$

$$\begin{aligned}
N_1 = & \tilde{\gamma} \Big[ C_5 (C_1 (\mu_2 (\mu_1 (d_1 d_3 + d_4 (d_1 + d_3)) + d_1 d_3 d_4) + d_1 d_3 d_4 \mu_1) - d_1 d_3 d_4 \mu_1 \mu_2) \\
& - C_5 (C_1 (\mu_2 (d_1 k\theta + k\mu_1 \theta) + d_1 k\mu_1 \theta) - d_1 k\mu_1 \mu_2 \theta) - C_1 d_1 d_3 d_4 \mu_1 \mu_2 + C_1 d_1 k\mu_1 \mu_2 \theta \Big],
\end{aligned}$$

$$N_0 = C_1 C_5 d_1 \tilde{\gamma} \mu_1 \mu_2 (d_3 d_4 - k\theta).$$

## D. Proof of Theorem 3

In this appendix, we present a detailed derivation of the stability properties and Hopf bifurcation conditions associated with Eq (3.6). We focus on the case  $\tau_2 = 0$  and systematically analyze the resulting characteristic equation to determine the existence of purely imaginary roots and the corresponding critical delay values.

By setting  $\tau_2 = 0$ , Eq (3.6) reduces to:

$$p(\hat{\lambda}, \tau_1) = \hat{\lambda}^8 + m_7 \hat{\lambda}^7 + m_6 \hat{\lambda}^6 + m_5 \hat{\lambda}^5 + m_4 \hat{\lambda}^4 + m_3 \hat{\lambda}^3 + m_2 \hat{\lambda}^2 + m_1 \hat{\lambda} + m_0 + (M_4 \hat{\lambda}^4 + M_3 \hat{\lambda}^3 + M_2 \hat{\lambda}^2 + M_1 \hat{\lambda} + M_0) e^{-\hat{\lambda} \tau_1} = 0, \quad (\text{D.1})$$

where

$$m_7 = J_7 + N_7, \quad m_6 = J_6 + N_6, \quad m_5 = J_5 + N_5, \quad m_4 = J_4 + N_4, \\ m_3 = J_3 + N_3, \quad m_2 = J_2 + N_2, \quad m_1 = J_1 + N_1, \quad m_0 = J_0 + N_0.$$

Consider purely imaginary roots  $\lambda = \pm iz, z > 0$ , of Eq (D.1). By substituting  $\lambda = iz$  and separating into real and imaginary parts, we obtain the following systems on  $z$

$$z^8 - m_6 z^6 + m_4 z^4 - m_2 z^2 + m_0 = -(M_4 z^4 - M_2 z^2 + M_0) \cos(\tau_1 z) - z (M_1 - M_3 z^2) \sin(\tau_1 z), \\ -m_7 z^7 + m_5 z^5 - m_3 z^3 + m_1 z = -z \cos(\tau_1 z) (M_1 - M_3 z^2) + \sin(\tau_1 z) (M_4 z^4 - M_2 z^2 + M_0). \quad (\text{D.2})$$

Eliminating trigonometric terms, Eq (D.2) yields the polynomial

$$p(z) = z^{16} + \Delta_1 z^{14} + \Delta_2 z^{12} + \Delta_3 z^{10} + \Delta_4 z^8 + \Delta_5 z^6 + \Delta_6 z^4 + \Delta_7 z^2 + \Delta_8 = 0, \quad (\text{D.3})$$

where,

$$\Delta_1 = m_7^2 - 2m_6, \quad \Delta_2 = m_6^2 + 2m_4 - 2m_5 m_7, \quad \Delta_3 = m_5^2 - 2m_2 + 2m_3 m_7 - 2m_4 m_6, \\ \Delta_4 = -M_4^2 + m_4^2 + 2m_0 - 2m_1 m_7 + 2m_2 m_6 - 2m_3 m_5, \\ \Delta_5 = -M_3^2 + m_3^2 - 2m_0 m_6 + 2m_1 m_5 - 2m_2 m_4 + 2M_2 M_4, \\ \Delta_6 = -M_2^2 + m_2^2 + 2m_0 m_4 - 2m_1 m_3 - 2M_0 M_4 + 2M_1 M_3, \\ \Delta_7 = -M_1^2 + m_1^2 - 2m_0 m_2 + 2M_0 M_2, \quad \Delta_8 = m_0^2 - M_0^2.$$

Put  $w = z^2$  in Eq (D.3), then the polynomial will reduce to

$$p(w) = w^8 + \Delta_1 w^7 + \Delta_2 w^6 + \Delta_3 w^5 + \Delta_4 w^4 + \Delta_5 w^3 + \Delta_6 w^2 + \Delta_7 w + \Delta_8 = 0. \quad (\text{D.4})$$

According to the results in [60, 61], we can deduce the conditions for the existence of positive roots of Eq (D.4). When  $\Delta_8 < 0$ , it follows that  $p(0) = \Delta_8 < 0$ , while  $\lim_{w \rightarrow +\infty} p(w) = +\infty$ . By continuity, this guarantees that (D.4) admits at least one positive real root.

Moreover, suppose that Eq (D.4) has eight distinct positive roots, denoted by  $w_1, w_2, \dots, w_8$ . Since Eq (D.4) is obtained by substituting  $z^2 = w$ , each positive root  $w_i$  of (D.4) corresponds to a positive root of (D.3) of the form

$$z_i = \sqrt{w_i}, \quad i = 1, 2, \dots, 8.$$

Thus, Eq (D.3) also possesses exactly eight positive roots. The corresponding critical value of time delay is found by solving (D.2) for  $\tau_1$ . Thus,

$$\cos z \tau_1 = \frac{-M_0 B_6 + M_2 B_7 - M_4 B_8 + M_1 B_9 - M_3 (-m_7 z^{10} + m_5 z^8 - m_3 z^6 + m_1 z^4)}{M_0^2 - 2 M_0 M_2 z^2 + 2 M_0 M_4 z^4 + M_1^2 z^2 - 2 M_1 M_3 z^4 + B_{10}},$$

where

$$\begin{aligned} B_6 &= (z^8 - m_6 z^6 + m_4 z^4 - m_2 z^2 + m_0), \\ B_7 &= (z^{10} - m_6 z^8 + m_4 z^6 - m_2 z^4 + m_0 z^2), \\ B_8 &= (z^{12} - m_6 z^{10} + m_4 z^8 - m_2 z^6 + m_0 z^4), \\ B_9 &= (-m_7 z^8 + m_5 z^6 - m_3 z^4 + m_1 z^2), \\ B_{10} &= M_2^2 z^4 - 2 M_2 M_4 z^6 + M_3^2 z^6 + M_4^2 z^8. \end{aligned}$$

Therefore,  $\tau_{1,i}^{(j)} = \frac{1}{z_i} \arccos \left( \frac{-M_0 B_6 + M_2 B_7 - M_4 B_8 + M_1 B_9 - M_3 (-m_7 z_i^{10} + m_5 z_i^8 - m_3 z_i^6 + m_1 z_i^4)}{M_0^2 - 2 M_0 M_2 z_i^2 + 2 M_0 M_4 z_i^4 + M_1^2 z_i^2 - 2 M_1 M_3 z_i^4 + B_{10}} \right) + \frac{2\pi j}{z_i},$

where  $i = 1, 2, \dots, 8$ ,  $j = 0, 1, 2, 3, \dots$ . Consequently,  $\hat{\lambda} = \pm i z_i$  is a pair of pure imaginary roots of Eq (D.1) with  $\tau_1 = \tau_{1,i}^{(j)}$ . Define

$$\tau_1^* = \tau_{1,i_0}^{(0)} = \min_{i \in \{1, 2, 3, \dots, 8\}} \{ \tau_{1,i}^{(0)} \}, \quad z^* = z_{i_0}.$$

To verify the transversality condition for the Hopf bifurcation, we first differentiate the characteristic equation (D.1) with respect to delay parameter  $\tau_1$ . This yields the derivative of a characteristic root  $\hat{\lambda}$  as

$$\frac{d\hat{\lambda}}{d\tau_1} = \frac{\hat{\lambda} e^{-\hat{\lambda}\tau_1} (M_4 \hat{\lambda}^4 + M_3 \hat{\lambda}^3 + M_2 \hat{\lambda}^2 + M_1 \hat{\lambda} + M_0)}{B_{11} + e^{-\hat{\lambda}\tau_1} B_{12} + 8\hat{\lambda}^7 - \tau_1 e^{-\hat{\lambda}\tau_1} (M_4 \hat{\lambda}^4 + M_3 \hat{\lambda}^3 + M_2 \hat{\lambda}^2 + M_1 \hat{\lambda} + M_0)},$$

where

$$\begin{aligned} B_{11} &= m_1 + 2\hat{\lambda}m_2 + 3\hat{\lambda}^2m_3 + 4\hat{\lambda}^3m_4 + 5\hat{\lambda}^4m_5 + 6\hat{\lambda}^5m_6 + 7\hat{\lambda}^6m_7, \\ B_{12} &= (4M_4\hat{\lambda}^3 + 3M_3\hat{\lambda}^2 + 2M_2\hat{\lambda} + M_1). \end{aligned}$$

Following standard practice, we consider the reciprocal derivative

$$\left( \frac{d\hat{\lambda}}{d\tau_1} \right)^{-1} = \frac{B_{11} + e^{-\hat{\lambda}\tau_1} B_{12} + 8\hat{\lambda}^7}{\hat{\lambda} e^{-\hat{\lambda}\tau_1} (M_4 \hat{\lambda}^4 + M_3 \hat{\lambda}^3 + M_2 \hat{\lambda}^2 + M_1 \hat{\lambda} + M_0)} - \frac{\tau_1}{\hat{\lambda}}.$$

Eliminating the exponential via the characteristic equation (D.1)

$$e^{-\hat{\lambda}\tau_1} = -\frac{\hat{\lambda}^8 + m_7\hat{\lambda}^7 + m_6\hat{\lambda}^6 + m_5\hat{\lambda}^5 + m_4\hat{\lambda}^4 + m_3\hat{\lambda}^3 + m_2\hat{\lambda}^2 + m_1\hat{\lambda} + m_0}{M_4\hat{\lambda}^4 + M_3\hat{\lambda}^3 + M_2\hat{\lambda}^2 + M_1\hat{\lambda} + M_0},$$

we obtain a purely algebraic expression for the reciprocal derivative

$$\left( \frac{d\hat{\lambda}}{d\tau_1} \right)^{-1} = \frac{-(B_{11} + 8\hat{\lambda}^7)}{\hat{\lambda} B_{13}} + \frac{B_{12}}{\hat{\lambda} B_{14}} - \frac{\tau_1}{\hat{\lambda}},$$

where

$$\begin{aligned} B_{13} &= (\hat{\lambda}^8 + m_7\hat{\lambda}^7 + m_6\hat{\lambda}^6 + m_5\hat{\lambda}^5 + m_4\hat{\lambda}^4 + m_3\hat{\lambda}^3 + m_2\hat{\lambda}^2 + m_1\hat{\lambda} + m_0) \\ B_{14} &= (M_4\hat{\lambda}^4 + M_3\hat{\lambda}^3 + M_2\hat{\lambda}^2 + M_1\hat{\lambda} + M_0). \end{aligned}$$

For the Hopf bifurcation, we substitute  $\hat{\lambda} = iz$ , take the real part, and collect powers of  $z^2$

$$\operatorname{Re} \left[ \left( \frac{d\hat{\lambda}}{d\tau_1} \right)^{-1} \right]_{\lambda=iz} = \frac{g(z)}{f(z)},$$

where

$$g(z) = (-4M_4 + M_3\tau_1 + M_4m_7\tau_1)z^{11} + G_1z^9 + G_2z^7 + G_3z^5 + G_4z^3 + G_5z,$$

$$f(z) = G_6z^3 - z(M_4z^4 - M_2z^2 + M_0)(z^8 - m_6z^6 + m_4z^4 - m_2z^2 + m_0),$$

with

$$G_1 = 6M_2 + 4M_3m_7 + 2M_4m_6 - M_1\tau_1 - M_2m_7\tau_1 - M_3m_6\tau_1 - M_4m_5\tau_1,$$

$$G_2 = -8M_0 - 6M_1m_7 - 4M_2m_6 - 2M_3m_5 + M_0m_7\tau_1 + M_1m_6\tau_1 + M_2m_5\tau_1 + M_3m_4\tau_1 + M_4m_3\tau_1,$$

$$G_3 = 6M_0m_6 + 4M_1m_5 + 2M_2m_4 - 2M_4m_2 - M_0m_5\tau_1 - M_1m_4\tau_1 - M_2m_3\tau_1 - M_3m_2\tau_1 - M_4m_1\tau_1,$$

$$G_4 = -4M_0m_4 - 2M_1m_3 + 2M_3m_1 + 4M_4m_0 + M_0m_3\tau_1 + M_1m_2\tau_1 + M_2m_1\tau_1 + M_3m_0\tau_1,$$

$$G_5 = 2M_0m_2 - 2M_2m_0 - M_0m_1\tau_1 - M_1m_0\tau_1,$$

$$G_6 = (M_1 - M_3z^2)(-m_7z^6 + m_5z^4 - m_3z^2 + m_1).$$

Finally, the transversality condition can be expressed in terms of the sign of the real part of the reciprocal:

$$\operatorname{sign} \frac{d \operatorname{Re}(\hat{\lambda})}{d\tau_1} \bigg|_{\tau_1=\tau_1^*} = \operatorname{sign} \operatorname{Re} \left[ \left( \frac{d\hat{\lambda}}{d\tau_1} \right)^{-1} \right] \bigg|_{\tau_1=\tau_1^*} = \operatorname{sign} [g(z^*)].$$

Hence, the transversality condition is satisfied if

$$\operatorname{sign} [g(z^*)] \neq 0.$$

The analysis above, together with the Hopf bifurcation theorem [46], yields Theorem 3.

## E. Proof of Theorem 4

This appendix presents the detailed derivation of the stability and delay-induced dynamics associated with Eq (3.6), under the case  $\tau_1 = 0$ . The analysis proceeds by reducing the characteristic equation, determining conditions for purely imaginary roots, and deriving the associated polynomial whose roots govern the stability behavior.

By setting  $\tau_1 = 0$ , the transcendental equation (3.6) simplifies to:

$$\begin{aligned} p(\hat{\lambda}, \tau_1, \tau_2) &= \hat{\lambda}^8 + J_7\hat{\lambda}^7 + J_6\hat{\lambda}^6 + J_5\hat{\lambda}^5 + J_4\hat{\lambda}^4 + J_3\hat{\lambda}^3 + J_2\hat{\lambda}^2 + J_1\hat{\lambda} + J_0 \\ &+ (N_7\hat{\lambda}^7 + N_6\hat{\lambda}^6 + N_5\hat{\lambda}^5 + N_4\hat{\lambda}^4 + N_3\hat{\lambda}^3 + N_2\hat{\lambda}^2 + N_1\hat{\lambda} + N_0)e^{-\hat{\lambda}\tau_2} = 0. \end{aligned} \quad (\text{E.1})$$

Considering purely imaginary roots  $\hat{\lambda} = \pm iz$  ( $z > 0$ ), and separating real and imaginary parts leads to:

$$\begin{aligned} J_0 - J_2z^2 + J_4z^4 - J_6z^6 + z^8 + \zeta_1 \cos(\tau_2 z) + z\zeta_2 \sin(\tau_2 z) &= 0, \\ J_1z - J_3z^3 + J_5z^5 - J_7z^7 - \zeta_1 \sin(\tau_2 z) + z\zeta_2 \cos(\tau_2 z) &= 0, \end{aligned} \quad (\text{E.2})$$

where

$$\zeta_1 = -N_6 z^6 + N_4 z^4 - N_2 z^2 + N_0, \quad \zeta_2 = -N_7 z^6 + N_5 z^4 - N_3 z^2 + N_1.$$

Eliminating the trigonometric terms from (E.2) gives the 16th-degree polynomial:

$$p(z) = z^{16} + \Gamma_1 z^{14} + \Gamma_2 z^{12} + \Gamma_3 z^{10} + \Gamma_4 z^8 + \Gamma_5 z^6 + \Gamma_6 z^4 + \Gamma_7 z^2 + \Gamma_8 = 0, \quad (\text{E.3})$$

with coefficients

$$\begin{aligned} \Gamma_1 &= J_7^2 - N_7^2 - 2J_6, \Gamma_2 = J_6^2 - N_6^2 + 2J_4 - 2J_5 J_7 + 2N_5 N_7, \\ \Gamma_3 &= J_5^2 - N_5^2 - 2J_2 + 2J_3 J_7 - 2J_4 J_6 - 2N_3 N_7 + 2N_4 N_6, \\ \Gamma_4 &= J_4^2 - N_4^2 + 2J_0 - 2J_1 J_7 + 2J_2 J_6 - 2J_3 J_5 + 2N_1 N_7 - 2N_2 N_6 + 2N_3 N_5, \\ \Gamma_5 &= J_3^2 - N_3^2 - 2J_0 J_6 + 2J_1 J_5 - 2J_2 J_4 + 2N_0 N_6 - 2N_1 N_5 + 2N_2 N_4, \\ \Gamma_6 &= J_2^2 - N_2^2 + 2J_0 J_4 - 2J_1 J_3 - 2N_0 N_4 + 2N_1 N_3, \\ \Gamma_7 &= J_1^2 - N_1^2 - 2J_0 J_2 + 2N_0 N_2, \Gamma_8 = J_0^2 - N_0^2. \end{aligned}$$

Setting  $w = z^2$  reduces (E.3) to an 8th-degree polynomial:

$$p(w) = w^8 + \Gamma_1 w^7 + \Gamma_2 w^6 + \Gamma_3 w^5 + \Gamma_4 w^4 + \Gamma_5 w^3 + \Gamma_6 w^2 + \Gamma_7 w + \Gamma_8 = 0. \quad (\text{E.4})$$

Equation (E.2) can also be written as:

$$\begin{aligned} \zeta_3^* + \zeta_1^* \cos(\tau_2 z^*) + z^* \zeta_2^* \sin(\tau_2 z^*) &= 0, \\ \zeta_4^* - \zeta_1^* \sin(\tau_2 z^*) + z^* \zeta_2^* \cos(\tau_2 z^*) &= 0, \end{aligned}$$

with

$$\begin{aligned} \zeta_3^* &= J_0 - J_2(z^*)^2 + J_4(z^*)^4 - J_6(z^*)^6 + (z^*)^8, \quad \zeta_4^* = J_1 z^* - J_3(z^*)^3 + J_5(z^*)^5 - J_7(z^*)^7, \\ \zeta_1^* &= -N_6(z^*)^6 + N_4(z^*)^4 - N_2(z^*)^2 + N_0, \quad \zeta_2^* = -N_7(z^*)^6 + N_5(z^*)^4 - N_3(z^*)^2 + N_1. \end{aligned}$$

Following an analogous discussion to Case 2 ( $\tau_1 > 0$ ,  $\tau_2 = 0$ ), we obtain Theorem 4.

## F. Proof of Theorem 5

In this appendix, we present the stability and Hopf bifurcation analysis associated with Eq (3.6) under the case  $\tau_1 = \tau_2 = \tau$ . The analysis proceeds by reducing the characteristic equation, deriving conditions for purely imaginary roots, and obtaining the corresponding polynomial whose roots determine the stability behavior of the system.

By setting  $\tau_1 = \tau_2 = \tau$ , the transcendental characteristic equation (3.6) reduces to

$$\begin{aligned} p(\hat{\lambda}, \tau) &= \hat{\lambda}^8 + J_7 \hat{\lambda}^7 + J_6 \hat{\lambda}^6 + J_5 \hat{\lambda}^5 + J_4 \hat{\lambda}^4 + J_3 \hat{\lambda}^3 + J_2 \hat{\lambda}^2 + J_1 \hat{\lambda} + J_0 \\ &\quad + (N_7 \hat{\lambda}^7 + N_6 \hat{\lambda}^6 + N_5 \hat{\lambda}^5 + (M_4 + N_4) \hat{\lambda}^4 \\ &\quad + (M_3 + N_3) \hat{\lambda}^3 + (M_2 + N_2) \hat{\lambda}^2 + (M_1 + N_1) \hat{\lambda} + (M_0 + N_0)) e^{-\hat{\lambda} \tau} = 0. \end{aligned}$$

Considering purely imaginary roots  $\hat{\lambda} = \pm iz$  ( $z > 0$ ) and separating the real and imaginary components yields:

$$\begin{aligned}\zeta_7 + \zeta_5 \cos(\tau z) + \zeta_6 \sin(\tau z) &= 0, \\ \zeta_8 + \zeta_6 \cos(\tau z) - \zeta_5 \sin(\tau z) &= 0,\end{aligned}\tag{F.1}$$

where

$$\begin{aligned}\zeta_5 &= (M_0 + N_0) - (M_2 + N_2)z^2 + (M_4 + N_4)z^4 - N_6z^6, \\ \zeta_6 &= (M_1 + N_1)z - (M_3 + N_3)z^3 + N_5z^5 - N_7z^7, \\ \zeta_7 &= J_0 - J_2z^2 + J_4z^4 - J_6z^6 + z^8, \\ \zeta_8 &= J_1z - J_3z^3 + J_5z^5 - J_7z^7.\end{aligned}$$

Eliminating the trigonometric terms from (F.1) leads to the following 16th-degree polynomial in  $z$ :

$$P(z) = z^{16} + \Gamma_9 z^{14} + \Gamma_{10} z^{12} + \Gamma_{11} z^{10} + \Gamma_{12} z^8 + \Gamma_{13} z^6 + \Gamma_{14} z^4 + \Gamma_{15} z^2 + \Gamma_{16},$$

with coefficients

$$\begin{aligned}\Gamma_9 &= J_7^2 - N_7^2 - 2J_6, \\ \Gamma_{10} &= J_6^2 - N_6^2 + 2J_4 - 2J_5J_7 + 2N_5N_7, \\ \Gamma_{11} &= J_5^2 - N_5^2 - 2J_2 + 2J_3J_7 - 2J_4J_6 - 2M_3N_7 + 2M_4N_6 - 2N_3N_7 + 2N_4N_6, \\ \Gamma_{12} &= J_4^2 - M_4^2 - 2M_4N_4 - N_4^2 + 2J_0 - 2J_1J_7 + 2J_2J_6 - 2J_3J_5 + 2M_1N_7 - 2M_2N_6 + 2M_3N_5 \\ &\quad + 2N_1N_7 - 2N_2N_6 + 2N_3N_5, \\ \Gamma_{13} &= J_3^2 - M_3^2 - 2M_3N_3 - N_3^2 - 2J_0J_6 + 2J_1J_5 - 2J_2J_4 + 2M_2M_4 + 2M_0N_6 - 2M_1N_5 + 2M_2N_4 \\ &\quad + 2M_4N_2 + 2N_0N_6 - 2N_1N_5 + 2N_2N_4, \\ \Gamma_{14} &= J_2^2 - M_2^2 - 2M_2N_2 - N_2^2 + 2J_0J_4 - 2J_1J_3 - 2M_0M_4 + 2M_1M_3 - 2M_0N_4 + 2M_1N_3 \\ &\quad + 2M_3N_1 - 2M_4N_0 - 2N_0N_4 + 2N_1N_3, \\ \Gamma_{15} &= J_1^2 - M_1^2 - 2M_1N_1 - N_1^2 - 2J_0J_2 + 2M_0M_2 + 2M_0N_2 + 2M_2N_0 + 2N_0N_2, \\ \Gamma_{16} &= J_0^2 - M_0^2 - 2M_0N_0 - N_0^2.\end{aligned}$$

Similarly, Eq (F.1) can be expressed as

$$\begin{aligned}\zeta_7^* + \zeta_5^* \cos(\tau z^*) + \zeta_6^* \sin(\tau z^*) &= 0, \\ \zeta_8^* + \zeta_6^* \cos(\tau z^*) - \zeta_5^* \sin(\tau z^*) &= 0,\end{aligned}$$

where

$$\begin{aligned}\zeta_5^* &= (M_0 + N_0) - (M_2 + N_2)(z^*)^2 + (M_4 + N_4)(z^*)^4 - N_6(z^*)^6, \\ \zeta_6^* &= (M_1 + N_1)z^* - (M_3 + N_3)(z^*)^3 + N_5(z^*)^5 - N_7(z^*)^7, \\ \zeta_7^* &= J_0 - J_2(z^*)^2 + J_4(z^*)^4 - J_6(z^*)^6 + (z^*)^8, \\ \zeta_8^* &= J_1z^* - J_3(z^*)^3 + J_5(z^*)^5 - J_7(z^*)^7.\end{aligned}$$

Following a similar argument as in Case 2, we obtain Theorem 5.

## G. Proof of Theorem 6

In this section, we establish the Hopf bifurcation at the critical delay  $\tau_1^*$  by reformulating the system as a functional differential equation, applying center manifold theory, and deriving the associated normal form coefficients that determine the direction, stability, and period of the bifurcating periodic solutions.

Define

$$\begin{aligned}\phi_1 &= S - S^*, & \phi_2 &= L_s - L_s^*, & \phi_3 &= L_f - L_f^*, & \phi_4 &= I - I^*, \\ \phi_5 &= H - H^*, & \phi_6 &= R - R^*, & \phi_7 &= F - F^*, & \phi_8 &= P - P^*.\end{aligned}$$

Let  $\tau_1 = \varrho + \tau_1^*$  with  $\varrho \in \mathbb{R}$ . By normalizing the delay  $\tau_1$  through the time-scaling  $t \mapsto \frac{t}{\tau_1}$ , system (2.1) can be rewritten in the functional differential form

$$\dot{\phi}(t) = L_{\varrho}(\phi_t) + \Upsilon(\varrho, \phi_t), \quad (\text{G.1})$$

where  $L_{\varrho}: C \rightarrow \mathbb{R}^8$  and  $\Upsilon: \mathbb{R} \times C \rightarrow \mathbb{R}^8$  are given by

$$L_{\varrho}\phi = (\tau_1^* + \varrho) \left[ K_0\phi(0) + K_{\tau_2}\phi\left(-\frac{\tau_2}{\tau_1^*}\right) + K_{\tau_1}\phi(-1) \right],$$

and

$$\Upsilon(\varrho, \phi) = \begin{pmatrix} \beta_{11} \phi_1(0)\phi_4(0) + \beta_{12} \phi_1(0)\phi_4(-1) + \beta_{13} \phi_4(0)\phi_4(-1) \\ \beta_{21} \phi_1(0)\phi_4(0) + \beta_{22} \phi_1(0)\phi_4(-1) + \beta_{23} \phi_4(0)\phi_4(-1) + \beta_{24} \phi_2(0)\phi_4(0) + \beta_{25} \phi_2(0)\phi_4(-1) \\ \beta_{31} \phi_1(0)\phi_4(0) + \beta_{32} \phi_1(0)\phi_4(-1) + \beta_{33} \phi_4(0)\phi_4(-1) + \beta_{34} \phi_2(0)\phi_4(0) + \beta_{35} \phi_2(0)\phi_4(-1) \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix},$$

with

$$K_0 = \begin{pmatrix} C_1 & 0 & 0 & C_2 & 0 & 0 & 0 & C_3 \\ C_4 & C_5 & 0 & C_6 & 0 & 0 & 0 & C_7 \\ C_8 & C_9 & -d_1 & C_{10} & 0 & 0 & 0 & C_{11} \\ 0 & \rho_1 & \rho_2 & -d_2 & 0 & k & 0 & 0 \\ 0 & 0 & 0 & 0 & -d_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \theta & -d_4 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\mu_f & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \alpha & -\mu_p \end{pmatrix},$$

$$K_{\tau_1} = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \omega & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \quad K_{\tau_2} = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\tilde{\gamma} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \tilde{\gamma} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

and

$$\begin{aligned} \beta_{11} &= -\frac{\beta}{1 + \epsilon P^*}, & \beta_{12} &= \frac{\beta \epsilon I^*}{(1 + \epsilon P^*)^2} c, & \beta_{13} &= \frac{S^* \beta \epsilon}{(1 + \epsilon P^*)^2} c, \\ \beta_{21} &= q \frac{\beta}{1 + \epsilon P^*}, & \beta_{22} &= -q \frac{\beta \epsilon I^*}{(1 + \epsilon P^*)^2} c, & \beta_{23} &= -\frac{\beta \epsilon}{(1 + \epsilon P^*)^2} (q S^* - \eta L_s^*) c, \\ \beta_{24} &= -\eta \frac{\beta}{1 + \epsilon P^*}, & \beta_{25} &= \eta \frac{\beta \epsilon I^*}{(1 + \epsilon P^*)^2} c, \\ \beta_{31} &= (1 - q) \frac{\beta}{1 + \epsilon P^*}, & \beta_{32} &= -(1 - q) \frac{\beta \epsilon I^*}{(1 + \epsilon P^*)^2} c, & \beta_{33} &= -\frac{\beta \epsilon}{(1 + \epsilon P^*)^2} ((1 - q) S^* + \eta L_s^*) c, \\ \beta_{34} &= \eta \frac{\beta}{1 + \epsilon P^*}, & \beta_{35} &= -\eta \frac{\beta \epsilon I^*}{(1 + \epsilon P^*)^2} c. \end{aligned}$$

According to the Riesz representation theorem [62], there exists a function  $\Theta(\varepsilon, \varrho)$ , whose components are of bounded variation for  $\varepsilon \in [-1, 0]$ , such that

$$L_{\varrho} \phi = \int_{-1}^0 d\Theta(\varepsilon, \varrho) \phi(\varepsilon).$$

We may choose

$$\Theta(\varepsilon, \varrho) = \begin{cases} (\tau_1^* + \varrho)(K_0 + K_{\tau_2} + K_{\tau_1}), & \varepsilon = 0, \\ (\tau_1^* + \varrho)(K_{\tau_2} + K_{\tau_1}), & \varepsilon \in \left[-\frac{\tau_2^*}{\tau_1^*}, 0\right], \\ (\tau_1^* + \varrho)(K_{\tau_1}), & \varepsilon \in \left[-1, -\frac{\tau_2^*}{\tau_1^*}\right], \\ 0, & \varrho = -1, \end{cases}$$

with

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

For  $\phi \in C([-1, 0], \mathbb{R}^8)$ , define

$$A(\varrho)\phi = \begin{cases} \frac{d\phi(\varepsilon)}{d\varepsilon}, & -1 \leq \varepsilon < 0, \\ \int_{-1}^0 d\Theta(\varepsilon, \varrho) \phi(\varepsilon), & \varepsilon = 0, \end{cases}$$

and

$$B(\varrho)\phi = \begin{cases} 0, & -1 \leq \varepsilon < 0, \\ \Upsilon(\varrho, \varepsilon), & \varepsilon = 0. \end{cases}$$

Thus, system (G.1) is equivalent to

$$\dot{\phi}(t) = A(\varrho)\phi(t) + B(\varrho)\phi(t). \quad (\text{G.2})$$

For  $\vartheta \in C^1([0, 1], (\mathbb{R}^8)^*)$ , the adjoint operator  $A^*$  of  $A(0)$  is given by

$$A^*(\vartheta) = \begin{cases} -\frac{d\vartheta(s)}{ds}, & 0 < s \leq 1, \\ \int_{-1}^0 d\Theta^T(s, 0) \vartheta(-s), & s = 0. \end{cases}$$

Finally, for  $\phi \in C([-1, 0], \mathbb{R}^8)$  and  $\vartheta \in C^1([0, 1], (\mathbb{R}^8)^*)$ , the bilinear inner form is defined as

$$\langle \vartheta(s), \phi(s) \rangle = \bar{\vartheta}(0) \phi(0) - \int_{\varepsilon=-1}^0 \int_{\nu_1=0}^{\varepsilon} \bar{\vartheta}(\nu_1 - \varepsilon) d\Theta(\varepsilon) \phi(\nu_1) d\nu_1, \quad (\text{G.3})$$

where  $\Theta(\varepsilon) = \Theta(\varepsilon, 0)$ .

From Subsection 3.3.5, the eigenvalues of  $A(0)$  are identified as  $\pm i\tau_1^* z_0^*$ . Consequently, the same pair  $\pm i\tau_1^* z_0^*$  serves as the eigenvalues of the adjoint operator  $A^*(0)$ . Define the corresponding eigenvectors

as  $\phi(\varepsilon) = \begin{bmatrix} 1 & \phi_2 & \phi_3 & \phi_4 & \phi_5 & \phi_6 & \phi_7 & \phi_8 \end{bmatrix}^T e^{i\tau_1^* z_0^* \varepsilon}$ ,

and  $\phi^*(s) = D \begin{bmatrix} 1 & \phi_2^* & \phi_3^* & \phi_4^* & \phi_5^* & \phi_6^* & \phi_7^* & \phi_8^* \end{bmatrix}^T e^{i\tau_1^* z_0^* s}$ .

These represent the eigenvectors of  $A(0)$  and  $A^*(0)$  associated with the eigenvalues  $+i\tau_1^* z_0^*$  and  $-i\tau_1^* z_0^*$ , respectively. Therefore, we obtain

$$\begin{aligned} \phi_2 &= \frac{C_4 + C_6 \phi_4 - C_7 \phi_8}{iz_0^* - C_5}, & \phi_3 &= \frac{C_{10} \phi_4 + C_{11} \phi_8 + C_8 + C_9 \phi_2}{iz_0^* + d_1}, \\ \phi_4 &= \frac{iz_0^* - C_1}{C_2 + C_3 \frac{-\alpha \omega e^{-iz_0^* \tau_1^*}}{(iz_0^* + \mu_p)(iz_0^* + \mu_f)}}, & \phi_5 &= \frac{\tilde{\gamma} e^{iz_0^* \tau_2^*}}{iz_0^* + d_3} \phi_4, \\ \phi_6 &= \frac{\theta \tilde{\gamma} e^{-iz_0^* \tau_2^*}}{(iz_0^* + d_4)(iz_0^* + d_3)} \phi_4, & \phi_7 &= \frac{\omega e^{-iz_0^* \tau_1^*}}{iz_0^* + \mu_f} \phi_4, \\ \phi_8 &= \frac{-\alpha \omega e^{-iz_0^* \tau_1^*}}{(iz_0^* + \mu_p)(iz_0^* + \mu_f)} \phi_4, \end{aligned}$$

$$\begin{aligned}\phi_2^* &= \Xi_3 \phi_4^*, & \phi_3^* &= \Xi_2 \phi_4^*, & \phi_4^* &= \frac{-C_2}{\Xi_7}, \\ \phi_5^* &= \Xi_4 \phi_4^*, & \phi_6^* &= \Xi_1 \phi_4^*, & \phi_7^* &= \Xi_6 \phi_4^*, & \phi_8^* &= \Xi_5 \phi_4^*,\end{aligned}$$

where

$$\begin{aligned}\Xi_1 &= \frac{-k}{iz_0^* - d_4}, & \Xi_2 &= \frac{-\rho_2}{iz_0^* - d_1}, & \Xi_3 &= \frac{-C_9 \Xi_2 - \rho_1}{iz_0^* + C_5}, \\ \Xi_4 &= \frac{-\theta \Xi_1}{iz_0^* - d_3}, & \Xi_5 &= \frac{-C_3 - (C_7 \Xi_3 + C_{11} \Xi_2)}{iz_0^* - \mu_p}, \\ \Xi_6 &= \frac{-\alpha \Xi_5}{iz_0^* - \mu_f}, & \Xi_7 &= C_6 \Xi_3 + C_{10} \Xi_2 + (iz_0^* - d_2) + \omega e^{iz_0^* \tau_1^*} \Xi_6 - \tilde{\gamma} e^{iz_0^* \tau_2^*} + \tilde{\gamma} e^{iz_0^* \tau_2^*} \Xi_4.\end{aligned}$$

To show that  $\langle \phi^*, \bar{\phi} \rangle = 0$  and  $\langle \phi^*, \phi \rangle = 1$ , we need to compute the value of  $\bar{D}$ . In view of (G.3), we may choose

$$\bar{D} = \left[ 1 + \sum_{i=2}^8 \phi_i^* \phi_i + \omega \phi_7^* \phi_4 e^{-iz_0^* \tau_1^*} + (-\tilde{\gamma} \phi_4^* + \tilde{\gamma} \phi_5^*) \phi_4 e^{-iz_0^* \tau_2^*} \right]^{-1}.$$

Using the same notation as in [46], we compute the coordinates that describe the center manifold  $C_0$  at  $\varrho = 0$ . Let  $\phi_t$  be the solution of equation G.1 when  $\varrho = 0$ . Define

$$u(t) = \langle \phi^*, \phi_t \rangle, \quad W(t, \varepsilon) = \phi_t(\varepsilon) - 2 \operatorname{Re}\{u(t)\phi(\varepsilon)\}.$$

On the center manifold  $C_0$ , we have

$$W(t, \varepsilon) = W(u(t), \bar{u}(t), \varepsilon),$$

where

$$W(u(t), \bar{u}(t), \varepsilon) = W(u, \bar{u}) = W_{20}(\varepsilon) \frac{u^2}{2} + W_{11}(\varepsilon) u \bar{u} + W_{02}(\varepsilon) \frac{\bar{u}^2}{2} + W_{30}(\varepsilon) \frac{u^3}{2} + \dots$$

Here,  $u$  and  $\bar{u}$  serve as local coordinates on the center manifold  $C_0$  along the directions of  $\phi$  and  $\bar{\phi}^*$ . Note that  $W$  is real whenever  $\phi_t$  is real; hence, we focus only on real solutions. For solutions  $\phi_t \in C_0$  of (G.1), with  $\varrho = 0$ ,

$$\dot{u}(t) = i\tau_1^* z_0^* u + \bar{\phi}^*(0) \Upsilon(0, W(u, \bar{u}, \varepsilon)) + 2 \operatorname{Re}\{u(t)\phi(\varepsilon)\} = i\tau_1^* z_0^* u + g(u, \bar{u}),$$

where

$$g(u, \bar{u}) = g_{20} \frac{u^2}{2} + g_{11} u \bar{u} + g_{02} \frac{\bar{u}^2}{2} + g_{21} \frac{u^2 \bar{u}}{2} + \dots, \quad (\text{G.4})$$

from (G.1), we have

$$\begin{aligned}\phi_t &= W(t, \varepsilon) + 2 \operatorname{Re}\{u(t)\phi(\varepsilon)\} \\ &= W_{20}(\varepsilon) \frac{u^2}{2} + W_{11}(\varepsilon) u \bar{u} + W_{02}(\varepsilon) \frac{\bar{u}^2}{2} + (1, \phi_2) e^{i\tau_1^* z_0^* \varepsilon} u + (1, \bar{\phi}_2) e^{-i\tau_1^* z_0^* \varepsilon} \bar{u} + \dots\end{aligned}$$

Therefore, we obtain

$$g(u, \bar{u}) = \bar{\phi}^*(0) \Upsilon(0, \phi_t).$$

By carrying out some algebra and comparing the coefficients in (G.4), we find

$$\begin{aligned} g_{20} &= 2\bar{D}\tau_1^* \left( H_{20}^{(1)}(0) + \phi_2^* H_{20}^{(2)}(0) + \phi_3^* H_{20}^{(3)}(0) \right), \\ g_{11} &= \bar{D}\tau_1^* \left( H_{11}^{(1)}(0) + \phi_2^* H_{11}^{(2)}(0) + \phi_3^* H_{11}^{(3)}(0) \right), \\ g_{02} &= D \left( H_{02}^{(1)}(0) + \phi_2^* H_{02}^{(2)}(0) + \phi_3^* H_{02}^{(3)}(0) \right), \\ g_{21} &= D \left( H_{21}^{(1)}(0) + \phi_2^* H_{21}^{(2)}(0) + \phi_3^* H_{21}^{(3)}(0) \right), \end{aligned}$$

where,

$$\begin{aligned} H_{20}^{(1)}(0) &= \beta_{11} \phi_4(0) + \beta_{12} \phi_4 \left( -\frac{\tau_2}{\tau_1^*} \right) + \beta_{13} \phi_4(0) \phi_4 \left( -\frac{\tau_2}{\tau_1^*} \right), \\ H_{20}^{(2)}(0) &= \beta_{21} \phi_4(0) + \beta_{22} \phi_4 \left( -\frac{\tau_2}{\tau_1^*} \right) + \beta_{23} \phi_4(0) \phi_4 \left( -\frac{\tau_2}{\tau_1^*} \right) \\ &\quad + \beta_{24} \phi_2(0) \phi_4(0) + \beta_{25} \phi_2(0) \phi_4 \left( -\frac{\tau_2}{\tau_1^*} \right), \\ H_{20}^{(3)}(0) &= \beta_{31} \phi_4(0) + \beta_{32} \phi_4 \left( -\frac{\tau_2}{\tau_1^*} \right) + \beta_{33} \phi_4(0) \phi_4 \left( -\frac{\tau_2}{\tau_1^*} \right) \\ &\quad + \beta_{34} \phi_2(0) \phi_4(0) + \beta_{35} \phi_2(0) \phi_4 \left( -\frac{\tau_2}{\tau_1^*} \right), \\ H_{11}^{(1)}(0) &= \beta_{11}(\bar{\phi}_4 + \phi_4) + \beta_{12}(\bar{\phi}_4 + \phi_4)e^{-iz_0^* \tau_2 / \tau_1^*} \\ &\quad + \beta_{13}(\phi_4 \bar{\phi}_4 + \bar{\phi}_4 \phi_4)e^{-iz_0^* \tau_2 / \tau_1^*}, \\ H_{11}^{(2)}(0) &= \beta_{21}(\bar{\phi}_4 + \phi_4) + \beta_{22}(\bar{\phi}_4 + \phi_4)e^{-iz_0^* \tau_2 / \tau_1^*} \\ &\quad + \beta_{23}(\phi_4 \bar{\phi}_4 + \bar{\phi}_4 \phi_4)e^{-iz_0^* \tau_2 / \tau_1^*} \\ &\quad + \beta_{24}(\phi_2 \bar{\phi}_4 + \bar{\phi}_2 \phi_4) + \beta_{25}(\phi_2 \bar{\phi}_4 + \bar{\phi}_2 \phi_4)e^{-iz_0^* \tau_2 / \tau_1^*}, \\ H_{11}^{(3)}(0) &= \beta_{31}(\bar{\phi}_4 + \phi_4) + \beta_{32}(\bar{\phi}_4 + \phi_4)e^{-iz_0^* \tau_2 / \tau_1^*} \\ &\quad + \beta_{33}(\phi_4 \bar{\phi}_4 + \bar{\phi}_4 \phi_4)e^{-iz_0^* \tau_2 / \tau_1^*} \\ &\quad + \beta_{34}(\phi_2 \bar{\phi}_4 + \bar{\phi}_2 \phi_4) + \beta_{35}(\phi_2 \bar{\phi}_4 + \bar{\phi}_2 \phi_4)e^{-iz_0^* \tau_2 / \tau_1^*}, \\ H_{02}^{(1)}(0) &= \beta_{11} \bar{\phi}_4(0) + \beta_{12} \bar{\phi}_4 \left( -\frac{\tau_2}{\tau_1^*} \right) + \beta_{13} \bar{\phi}_4(0) \bar{\phi}_4 \left( -\frac{\tau_2}{\tau_1^*} \right), \\ H_{02}^{(2)}(0) &= \beta_{21} \bar{\phi}_4(0) + \beta_{22} \bar{\phi}_4 \left( -\frac{\tau_2}{\tau_1^*} \right) + \beta_{23} \bar{\phi}_4(0) \bar{\phi}_4 \left( -\frac{\tau_2}{\tau_1^*} \right) \\ &\quad + \beta_{24} \bar{\phi}_2(0) \bar{\phi}_4(0) + \beta_{25} \bar{\phi}_2(0) \bar{\phi}_4 \left( -\frac{\tau_2}{\tau_1^*} \right), \\ H_{02}^{(3)}(0) &= \beta_{31} \bar{\phi}_4(0) + \beta_{32} \bar{\phi}_4 \left( -\frac{\tau_2}{\tau_1^*} \right) + \beta_{33} \bar{\phi}_4(0) \bar{\phi}_4 \left( -\frac{\tau_2}{\tau_1^*} \right) \\ &\quad + \beta_{34} \bar{\phi}_2(0) \bar{\phi}_4(0) + \beta_{35} \bar{\phi}_2(0) \bar{\phi}_4 \left( -\frac{\tau_2}{\tau_1^*} \right). \end{aligned}$$

$$H_{21}^{(1)}(0) = 2\beta_{11} W_{11}^{(4)}(0) + 2\beta_{12} W_{11}^{(4)}\left(-\frac{\tau_2}{\tau_1^*}\right) + 2\beta_{13} \phi_4(0) W_{11}^{(4)}\left(-\frac{\tau_2}{\tau_1^*}\right) \\ + \beta_{11} W_{20}^{(1)}(0) \bar{\phi}_4(0) + \beta_{12} W_{20}^{(1)}(0) \bar{\phi}_4\left(-\frac{\tau_2}{\tau_1^*}\right) + \beta_{13} W_{20}^{(4)}(0) \bar{\phi}_4\left(-\frac{\tau_2}{\tau_1^*}\right),$$

$$H_{21}^{(2)}(0) = 2\beta_{21} W_{11}^{(4)}(0) + 2\beta_{22} W_{11}^{(4)}\left(-\frac{\tau_2}{\tau_1^*}\right) + 2\beta_{23} \phi_4(0) W_{11}^{(4)}\left(-\frac{\tau_2}{\tau_1^*}\right) \\ + 2\beta_{24} \phi_2(0) W_{11}^{(4)}(0) + 2\beta_{25} \phi_2(0) W_{11}^{(4)}\left(-\frac{\tau_2}{\tau_1^*}\right) \\ + \beta_{21} W_{20}^{(1)}(0) \bar{\phi}_4(0) + \beta_{22} W_{20}^{(1)}(0) \bar{\phi}_4\left(-\frac{\tau_2}{\tau_1^*}\right) + \beta_{23} W_{20}^{(4)}(0) \bar{\phi}_4\left(-\frac{\tau_2}{\tau_1^*}\right) \\ + \beta_{24} W_{20}^{(2)}(0) \bar{\phi}_4(0) + \beta_{25} W_{20}^{(2)}(0) \bar{\phi}_4\left(-\frac{\tau_2}{\tau_1^*}\right),$$

$$H_{21}^{(3)}(0) = 2\beta_{31} W_{11}^{(4)}(0) + 2\beta_{32} W_{11}^{(4)}\left(-\frac{\tau_2}{\tau_1^*}\right) + 2\beta_{33} \phi_4(0) W_{11}^{(4)}\left(-\frac{\tau_2}{\tau_1^*}\right) \\ + 2\beta_{34} \phi_2(0) W_{11}^{(4)}(0) + 2\beta_{35} \phi_2(0) W_{11}^{(4)}\left(-\frac{\tau_2}{\tau_1^*}\right) \\ + \beta_{31} W_{20}^{(1)}(0) \bar{\phi}_4(0) + \beta_{32} W_{20}^{(1)}(0) \bar{\phi}_4\left(-\frac{\tau_2}{\tau_1^*}\right) + \beta_{33} W_{20}^{(4)}(0) \bar{\phi}_4\left(-\frac{\tau_2}{\tau_1^*}\right) \\ + \beta_{34} W_{20}^{(2)}(0) \bar{\phi}_4(0) + \beta_{35} W_{20}^{(2)}(0) \bar{\phi}_4\left(-\frac{\tau_2}{\tau_1^*}\right),$$

with,

$$W_{20}(\varepsilon) = \frac{ig_{20}\phi(0)}{\tau_1^*z_0^*} e^{i\tau_1^*z_0^*\varepsilon} + \frac{i\bar{g}_{02}\bar{\phi}(0)}{3\tau_1^*z_0^*} e^{-i\tau_1^*z_0^*\varepsilon} + E_1 e^{2i\tau_1^*z_0^*\varepsilon},$$

$$W_{11}(\varepsilon) = -\frac{ig_{11}\phi(0)}{\tau_1^*z_0^*} e^{i\tau_1^*z_0^*\varepsilon} + \frac{i\bar{g}_{11}\bar{\phi}(0)}{\tau_1^*z_0^*} e^{-i\tau_1^*z_0^*\varepsilon} + E_2.$$

The values of  $E_1$  and  $E_2$  are determined by the following equations:

$$E_1 = 2 \begin{pmatrix} C_1^* & 0 & 0 & -C_2 & 0 & 0 & 0 & -C_3 \\ -C_4 & C_5^* & 0 & -C_6 & 0 & 0 & 0 & -C_7 \\ -C_8 & -C_9 & d_1^* & -C_{10} & 0 & 0 & 0 & -C_{11} \\ 0 & -\rho_1 & -\rho_2 & d_2^* & 0 & -k & 0 & 0 \\ 0 & 0 & 0 & 0 & d_3^* & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\theta & d_4^* & 0 & 0 \\ 0 & 0 & 0 & \omega^* & 0 & 0 & \mu_f^* & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\alpha & \mu_p^* \end{pmatrix}^{-1} \begin{pmatrix} H_{20}^{(1)}(0) \\ H_{20}^{(2)}(0) \\ H_{20}^{(3)}(0) \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix},$$

$$E_2 = - \begin{pmatrix} C_1 & 0 & 0 & C_2 & 0 & 0 & 0 & C_3 \\ C_4 & C_5 & 0 & C_6 & 0 & 0 & 0 & C_7 \\ C_8 & C_9 & -d_1 & C_{10} & 0 & 0 & 0 & C_{11} \\ 0 & \rho_1 & \rho_2 & -d_2 + \tilde{\gamma} & 0 & k & 0 & 0 \\ 0 & 0 & 0 & -\tilde{\gamma} & -d_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \theta & -d_4 & 0 & 0 \\ 0 & 0 & 0 & \omega & 0 & 0 & -\mu_f & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \alpha & -\mu_p \end{pmatrix}^{-1} \begin{pmatrix} H_{11}^{(1)}(0) \\ H_{11}^{(2)}(0) \\ H_{11}^{(3)}(0) \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix},$$

where

$$\begin{aligned} C_1^* &= 2i^* z_0^* - C_1, & C_5^* &= 2i^* z_0^* - C_5, \\ d_1^* &= 2i^* z_0^* + d_1, & d_2^* &= 2i^* z_0^* + d_2 + \tilde{\gamma} e^{-2i^* z_0^* \tau_2^*}, \\ d_3^* &= 2i^* z_0^* + d_3 - \tilde{\gamma} e^{-2i^* z_0^* \tau_2^*}, & d_4^* &= 2i^* z_0^* + d_4, \\ \omega^* &= -\omega e^{-2i^* z_0^* \tau_1^*}, & \mu_f^* &= 2i^* z_0^* + \mu_f, \\ \mu_p^* &= 2i^* z_0^* + \mu_p. \end{aligned}$$

Therefore, the following values can be determined

$$\begin{aligned} c_1(0) &= \frac{i}{2\tau_1^* z_0^*} \left( g_{20} g_{11} - 2|g_{11}|^2 - \frac{|g_{02}|^2}{3} \right) + \frac{g_{21}}{2}, \\ \alpha_1 &= 2 \operatorname{Re}\{c_1(0)\}, \\ \mu_1 &= -\frac{\operatorname{Re}\{c_1(0)\}}{\operatorname{Re}[\hat{\lambda}'(\tau_1^*)]}, \\ T_1 &= -\frac{\operatorname{Im}\{c_1(0)\} + \mu_1 \operatorname{Im}\{\hat{\lambda}'(\tau_1^*)\}}{\tau_1^* z_0^*}. \end{aligned}$$

At the critical delay  $\tau_1^*$ , the Hopf bifurcation generates periodic solutions whose properties are fully determined by  $\mu_1$ ,  $\alpha_1$ , and  $T_1$ . Specifically,  $\mu_1$  determines the direction of the bifurcation:  $\mu_1 > 0$  ( $\mu_1 < 0$ ) indicates a supercritical (subcritical) bifurcation. The stability of these bifurcating solutions is dictated by  $\alpha_1$ , with  $\alpha_1 < 0$  ( $\alpha_1 > 0$ ) implying stable (unstable) oscillations. Finally,  $T_1$  controls the period of the bifurcating solution:  $T_1 > 0$  ( $T_1 < 0$ ) governs an increasing (decreasing) oscillation period. These findings are consistent with the framework of Hassard et al. [46].

Therefore, the analysis results are provided in Theorem 6.



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