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Research article

Epidemic model with time delays and fertility/mortality rates

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Abstract: We developed a delayed SIR (Susceptible-Infected-Recovered) model incorporating infectious/immune periods and demographics (fertility and mortality rates), proving the existence, nonnegativity, and uniqueness of solutions for the system under demographic equilibrium. Analysis confirmed a threshold at $\Re_0 = 1$, with an endemic equilibrium emerging when $\Re_0 > 1$. Crucially, the stability of this endemic state was governed by a critical mortality rate (μ_c). High-mortality populations ($\mu > \mu_c$) exhibited a stable endemic state, whereas low-mortality populations ($\mu < \mu_c$) experienced instability and sustained oscillations. For these low-mortality populations, critical thresholds for the transmission rate (β_c) and disease duration (τ_{1c}) were identified, beyond which destabilization occurred. This demonstrated a fundamental dual dependence of long-term disease dynamics on both demographic (e.g., life expectancy) and epidemiological (e.g., transmission rate, disease duration) parameters. Consequently, public health strategies (like vaccination targets) may need adjustment based on a population's demographic structure, not just its immediate epidemiological characteristics.

Keywords: epidemic-model; time delay; fertility; mortality
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1. Introduction

Mathematical modeling has emerged as an indispensable tool for understanding and mitigating the impacts of epidemics. Its development in epidemiology has been driven by the recurrent emergence of large-scale outbreaks, including HIV (Human Immunodeficiency Virus) from the 1980s to the present [1,2], SARS (Severe Acute Respiratory Syndrome) in 2002–2003 [3,4], H5N1 influenza (Avian Influenza) in 2005 [5, 6], H1N1 (Swine Flu) in 2009 [7, 8], and Ebola in 2014 [9, 10]. The recent COVID-19 (Coronavirus Disease 2019) pandemic further underscored its critical role, profoundly affecting public health, economies, and societal structures globally.

The foundation of contemporary epidemiological modeling was significantly influenced by the Spanish influenza pandemic of 1918–1919 and the seminal work of Kermack and McKendrick [11,12]. This has led to the introduction of numerous models, with multi-compartment frameworks forming the cornerstone of modern studies and providing essential insights into disease transmission dynamics. Current applications encompass deciphering historical outbreaks, forecasting the trajectories of ongoing and future diseases [13–15], models incorporating nonlinear transmission rates [16, 17], multi-patch models [18, 19], multi-group models addressing population heterogeneity [20], and frameworks integrating vaccination and control measures [21, 22]. Spatiotemporal models further characterize spatial distributions of susceptible and infected individuals by accounting for individual mobility [23, 24]. Comprehensive overviews are available in monographs [25, 26] and review articles [27, 28].

Classical SIR-type (Susceptible-Infected-Recovered) models underpin the development of both single and multi-strain epidemic formulations, typically assuming that recoveries and deaths are proportional to the number infected at time t. To address limitations inherent in these assumptions, delay differential equation (DDE) models have become a powerful mathematical tool. By incorporating explicit delays, DDE models more accurately capture temporal features of disease progression and transmission. They have been widely applied to study infectious diseases like influenza (single and multi-strain) and COVID-19 [29–31] for single-strain models, and [32] for both single-strain and two-strain models with cross-immunity. Models describing interactions between two strains without cross-immunity [33], systems with distributed recovery and death rates (where DDEs provide suitable approximations [29]), delay models with vaccination [34], and dynamics involving periodic transmission rates for single and double strains [35] have also been investigated.

Complementary research has explored economic-demographic dynamical systems [33, 36], illustrating scenarios where lockdown can control epidemic by reducing the number of infectious individuals to its minimum and preserve the economic state of population, while epidemic can lead to economic deterioration.

Epidemic models based on systems of ordinary differential equations (ODEs) that integrate age-specific fertility and mortality rates provide crucial insights into long-term disease dynamics and population impacts. These ODE frameworks capture how birth rates replenish susceptible individuals and how disease-induced mortality alters age structure, influencing transmission potential and endemic equilibria. Incorporating demography into such differential equation models is essential for predicting the evolution of diseases like COVID-19 in specific populations and for evaluating long-term vaccination strategies.

In parallel to these developments, another critical advancement has been the use of DDEs to model fixed time periods inherent to disease biology, such as the duration of infection (τ_1) and acquired immunity (τ_2). Models such as the one proposed by [33, 34] have been instrumental in this area.

While appropriate for studying the pure effects of delays, a significant limitation of such models is their assumption of a closed population without vital dynamics (i.e., no births or natural deaths), which restricts their applicability to short-term outbreaks or hypothetical scenarios. In this paper, we develop a novel epidemic model that synthesizes these two critical strands of research. We extend the established DDE framework [33, 34] by incorporating essential demographic processes, namely, age-specific fertility (γ) and mortality (μ, δ) rates.

Within this context, the present study first introduces an extended epidemic propagation model

with birth rate, natural mortality rate, and disease-induced mortality rate. Then, we consider the specific case without mortality due to infection, and rigorously demonstrate the existence, uniqueness, nonnegativity, and boundedness of its solutions. Subsequent analysis identifies the system's equilibrium states and examines their stability properties under the condition of demographic balance (where fertility rate equals mortality rate). Finally, we present conclusions and outline further research perspectives.

2. Extended epidemic propagation model

In our previous research [33, 34], we have analyzed a closed-population SIR model with fixed delays (infection period τ_1 and immunity period τ_2), given by the system:

$$\begin{aligned}
\frac{dS(t)}{dt} &= -J(t) + J(t-\tau_1-\tau_2),\\
\frac{dI(t)}{dt} &= J(t) - J(t-\tau_1),\\
\frac{dR(t)}{dt} &= J(t-\tau_1) - J(t-\tau_1-\tau_2),\\
J(t) &= \frac{\beta}{N} S(t) I(t),
\end{aligned}$$

where β is infection transmission rate and $J(t)$ represents the number of the new incidences at time t. This model, which assumes a constant population size with no births or natural deaths, is well-suited for modeling short-term outbreaks but is limited in its application to endemic diseases that persist on the timescale of human lifespans. To study long-term dynamics, it is essential to incorporate vital dynamics (births and deaths). Therefore, in this work, we extend the previous framework by introducing demographic parameters, leading to a novel system of DDEs.

We consider a mathematical model of population dynamics that describes the spread of an infectious disease. It is assumed that, at any time t, the population can be partitioned into three mutually exclusive compartments: Susceptible $S(t)$, infected $I(t)$, and recovered $R(t)$ individuals. The total population size at time t is given by

$$N(t) = S(t) + I(t) + R(t), \tag{2.1}$$

where:

- $S(t)$ denotes the number of individuals susceptible to the infection;
- $I(t)$ denotes the number of infected individuals who are capable of transmitting the disease;
- $R(t)$ denotes the number of individuals who have recovered and are temporarily immune.

The transmission of the disease occurs through interactions between susceptible and infected individuals. The number of newly infected individuals at time t is modeled by the function

$$J(t) = \frac{\beta}{N(t)} S(t) I(t),$$

where $\beta > 0$ represents the disease transmission rate, which characterizes the frequency of effective contacts between individuals. The dynamics of the model are further governed by the parameters of Table 1.

Table 1. Parameters of the SIR model (2.4) with delays and demographics.

Parameter	Definition	Reference(s)
β	Disease transmission rate (effective contacts per unit time)	[14, 15, 33, 34]
τ_1	Duration of the infectious period	[33, 34]
τ_2	Duration of the temporary immune period	[33, 34]
γ	Birth rate coefficient (new susceptible individuals per unit time)	[14, 15]
μ	Natural mortality rate (deaths per capita per unit time)	[14, 15]
δ	Disease-induced mortality rate (additional deaths per infected individual per unit time)	[14, 37]

To model mortality, we assume that the lifetime T of an infected individual, measured from the moment of infection, follows an exponential distribution

$$T \sim \text{Exp}(\lambda), \quad \lambda = \mu + \delta,$$

where λ represents the total mortality rate (natural plus disease-induced).

The probability density function of T is given by

$$f_T(t) = \lambda e^{-\lambda t}, \quad t \geq 0,$$

and the probability that an individual survives until time t after infection is:

$$\mathbb{P}(T > t) = \int_t^\infty f_T(u)\, du = e^{-\lambda t}.$$

An important feature of the exponential distribution is its memoryless property:

$$\mathbb{P}(T > s + t \mid T > s) = \mathbb{P}(T > t), \quad \forall s, t \geq 0.$$

This implies that the probability of an event occurring in the future depends only on the elapsed time and not on the history of the process prior to the current moment.

Number of infected individuals. The infectious period lasts for a fixed duration τ_1. At time t, the infected individuals are those who were infected at some time $u \in [t - \tau_1, t]$ but have not died by time t due to either natural (μ) or disease-induced (δ) mortality.

The probability that an individual infected at time u remains alive at time t is given by $e^{-(\mu+\delta)(t-u)}$. Therefore, the number of infected individuals at time t is described by the integral:

$$I(t) = \int_{t-\tau_1}^{t} J(u) e^{-(\mu+\delta)(t-u)} du. \tag{2.2}$$

Number of recovered individuals. The duration of the immune period after recovery is τ_2. At time t, the recovered individuals are those who were infected at some time $u \in [t - \tau_1 - \tau_2, t - \tau_1]$ but have survived both the infectious period of length τ_1 and the subsequent immune period until time t.

The probability of survival until time t is the joint probability of surviving the infectious and immune periods. Using the memoryless property of the exponential distribution, this probability is expressed as the product $e^{-(\mu+\delta)\tau_1} \cdot e^{-\mu(t-\tau_1-u)}$, where $e^{-(\mu+\delta)\tau_1}$ is the probability of surviving the infectious period, and $e^{-\mu(t-\tau_1-u)}$ is the probability of surviving the immune period. Thus, the number of recovered individuals with active immunity at time t is given by

$$R(t) = \int_{t-\tau_1-\tau_2}^{t-\tau_1} J(u)e^{-(\mu+\delta)\tau_1}e^{-\mu(t-\tau_1-u)}du. \tag{2.3}$$

Expressions (2.2) and (2.3) account for time delays due to the durations of the infectious and immune periods, as well as mortality. The number of susceptible individuals at time t can be calculated by substituting (2.2) and (2.3) into (2.1). These integral expressions provide the foundation for deriving a complete system of delay differential equations that describe the dynamics of the epidemic in the population.

Differentiating the expressions for $I(t)$ and $R(t)$. To construct the system of differential equations, we compute the time derivatives of $I(t)$ and $R(t)$. Differentiating (2.2) with respect to t, we apply Leibniz's rule to get

$$\frac{dI(t)}{dt} = \frac{d}{dt}\left(\int_{t-\tau_1}^{t} J(u)e^{-(\mu+\delta)(t-u)}du\right) = J(t) - e^{-(\mu+\delta)\tau_1}J(t-\tau_1) - (\mu+\delta)I(t).$$

Similarly, differentiating (2.3) with respect to t, we obtain

$$\frac{dR(t)}{dt} = e^{-(\mu+\delta)\tau_1}J(t-\tau_1) - e^{-(\mu+\delta)\tau_1}e^{-\mu\tau_2}J(t-\tau_1-\tau_2) - \mu R(t).$$

Number of susceptible individuals. To complete the formulation of the model, we derive an equation for the number of susceptible individuals $S(t)$. Recall that the total population size is $N(t) = S(t) + I(t) + R(t)$, with $I(t)$ and $R(t)$ already defined.

Susceptible individuals are those who are not currently infected but are at risk of infection upon contact with infected individuals. The dynamics of $S(t)$ are influenced by the following factors:

- *Births*: New individuals enter the population at a rate $\gamma N(t)$, and are assumed to be initially susceptible.
- *Infection*: Susceptible individuals become infected at a rate $J(t) = \frac{\beta}{N(t)}S(t)I(t)$.
- *Loss of immunity*: Individuals who complete the immune period return to the susceptible class after a delay of $\tau_1 + \tau_2$, provided they have survived the entire duration. The corresponding flux is $e^{-(\mu+\delta)\tau_1}e^{-\mu\tau_2}J(t-\tau_1-\tau_2)$.
- *Mortality*: Natural mortality reduces the number of susceptible individuals at a rate $\mu S(t)$.

Incorporating all these processes, we derive the differential equation for $S(t)$ as follows

$$\frac{dS(t)}{dt} = \gamma N(t) - J(t) + e^{-(\mu+\delta)\tau_1}e^{-\mu\tau_2}J(t-\tau_1-\tau_2) - \mu S(t),$$

which completes the system of equations and enables the full description of the epidemic dynamics.

Infection propagation is modelled with the system of delay differential equations

$$\frac{dS(t)}{dt} = \gamma N(t) - J(t) + e^{-(\mu+\delta)\tau_1} e^{-\mu\tau_2} J(t-\tau_1-\tau_2) - \mu S(t), \tag{2.4a}$$

$$\frac{dI(t)}{dt} = J(t) - e^{-(\mu+\delta)\tau_1} J(t-\tau_1) - (\mu+\delta) I(t), \tag{2.4b}$$

$$\frac{dR(t)}{dt} = e^{-(\mu+\delta)\tau_1} J(t-\tau_1) - e^{-(\mu+\delta)\tau_1} e^{-\mu\tau_2} J(t-\tau_1-\tau_2) - \mu R(t), \tag{2.4c}$$

$$J(t) = \frac{\beta}{N(t)} S(t) I(t). \tag{2.4d}$$

This system is considered with the following initial conditions:

$$N(\theta) = S(\theta) = N_0, \quad I(\theta) = R(\theta) = 0, \quad \forall \theta \in [-(\tau_1+\tau_2), 0),$$

$$N(0) = N_0 > 0, \quad S(0) = S_0 > 0, \quad I(0) = I_0 \geq 0, \quad R(0) = 0, \quad (S_0 + I_0 = N_0).$$

Remark 1. *By setting $\mu = \gamma = \delta = 0$, the proposed model reduces to the previously studied version in [33].*

3. Demographically stable epidemic propagation model without disease-induced mortality

This section presents the analysis of a special case in which the population is in demographic equilibrium ($\gamma = \mu > 0$), and the disease is non-lethal ($\delta = 0$). We investigate the solution's non-negativity, existence, and uniqueness, and establish theorems concerning the existence and stability of a stationary state.

To analyze the dynamics of the total population size $N(t)$, we differentiate (2.1) with respect to time t and get

$$\frac{dN(t)}{dt} = \frac{d}{dt}\left(S(t) + I(t) + R(t)\right).$$

Substituting the expressions from system (2.4), we obtain:

$$\frac{dN(t)}{dt} = \gamma N(t) - \mu N(t) - \delta N(t).$$

If $\gamma = \mu > 0$ and $\delta = 0$, this expression simplifies to

$$\frac{dN(t)}{dt} = \gamma N(t) - \mu N(t) = 0 \implies N(t) = N_0.$$

This means that demographic processes - birth and natural death - exactly balance each other, and the population size remains constant throughout the disease dynamics, that is,

$$S(t) + I(t) + R(t) = N_0 \ \text{(constant)}. \tag{3.1}$$

We obtain the following system of equations:

$$\frac{dS(t)}{dt} = \gamma N_0 - J(t) + e^{-\mu(\tau_1+\tau_2)} J(t-\tau_1-\tau_2) - \mu S(t), \tag{3.2a}$$

$$\frac{dI(t)}{dt} = J(t) - e^{-\mu\tau_1} J(t-\tau_1) - \mu I(t), \tag{3.2b}$$

$$\frac{dR(t)}{dt} = e^{-\mu\tau_1} J(t-\tau_1) - e^{-\mu(\tau_1+\tau_2)} J(t-\tau_1-\tau_2) - \mu R(t), \tag{3.2c}$$

$$J(t) = \frac{\beta}{N_0} S(t) I(t) \tag{3.2d}$$

with the initial conditions:

$$S(\theta) = N_0 > 0, \quad I(\theta) = 0, \quad R(\theta) = 0, \quad \forall \theta \in [-(\tau_1+\tau_2), 0),$$

$$S(0) = S_0 > 0, \quad I(0) = I_0 \geq 0, \quad R(0) = 0 \quad (S_0 + I_0 = N_0). \tag{3.3}$$

3.1. Nonnegativity, existence, and uniqueness of solution

3.1.1. Nonnegativity of solution

To ensure the biological validity of the model, we must show that all variables remain nonnegative for all $t \geq 0$.

Lemma 2. *The function $J(t) = \frac{\beta}{N_0} S(t) I(t)$ in system (3.2) satisfies the condition $J(t) > 0$ for all $t > 0$, given the initial conditions: $S(0) > 0, I(0) > 0, R(0) = 0$, and $S(\theta) = N_0 > 0, I(\theta) = 0, R(\theta) = 0$ for all $\theta \in [-(\tau_1+\tau_2), 0)$.*

Proof. Assume that the function $J(t)$ becomes nonpositive at some point in time. Then, due to the continuity of $J(t)$ and the initial condition $J(0) = \frac{\beta}{N_0} S(0) I(0) > 0$, there exists a "first" moment $t_0 > 0$ at which the function crosses zero. This moment can be defined as

$$t_0 = \inf\Big\{t > 0 : J(t) = 0\Big\}.$$

It holds that

$$J(t) > 0 \quad \forall t \in [0, t_0),$$

since $J(0) > 0$, and the function does not reach zero before time t_0. Consider two cases corresponding to the condition $J(t_0) = 0$ ($S(t_0) I(t_0) = 0$):

- **Case 1.** If $S(t_0) = 0$, then from equation (3.2a), we get

$$\frac{dS(t_0)}{dt} = \gamma N_0 + e^{-\mu(\tau_1+\tau_2)} J(t_0 - \tau_1 - \tau_2).$$

Note that $J(t_0 - \tau_1 - \tau_2) \geq 0$, since $t_0 - \tau_1 - \tau_2 \in [-(\tau_1+\tau_2), t_0)$. Therefore,

$$\frac{dS(t_0)}{dt} \geq \gamma N_0 > 0.$$

This means there exists $\delta > 0$ such that $S(t) < S(t_0)$ for $t \in (t_0 - \delta, t_0)$ and $S(t) > S(t_0)$ for $t \in (t_0, t_0 + \delta)$. Since $S(t_0) = 0$, it follows that $S(t) < 0$ for $t \in (t_0 - \delta, t_0)$. However, the function $S(t)$ cannot become negative without crossing zero, and since $S(0) > 0$, this contradicts the definition of t_0 as the first time at which $J(t_0) = 0$.

- **Case 2.** If $I(t_0) = 0$, then from the integral form (2.2) for $I(t)$ under $\gamma = \mu > 0$ and $\delta = 0$, we have

$$I(t_0) = \int_{t_0-\tau_1}^{t_0} J(u)e^{-\mu(t_0-u)}du = 0.$$

Since the exponential factor $e^{-\mu(t_0-u)} > 0$ for all $u \in [t_0 - \tau_1, t_0]$, it follows that $J(u) = 0$ almost everywhere on this interval. Due to the continuity of $J(t)$, this implies $J(t) = 0$ for all $t \in [t_0 - \tau_1, t_0]$, which contradicts the definition of t_0 as the first moment where $J(t_0) = 0$.
Thus, the assumption that $J(t)$ becomes nonpositive leads to a contradiction. Therefore, $J(t) > 0$ for all $t > 0$.

□

Theorem 3. *Let the functions $S(t)$, $I(t)$, and $R(t)$ be a solution of system* (3.2) *under the initial conditions $S(0) > 0, I(0) > 0, R(0) = 0$, and $S(\theta) = N_0 > 0$, $I(\theta) = 0$, $R(\theta) = 0$ for all $\theta \in [-(\tau_1 + \tau_2), 0)$. Then, for all $t > 0$, the following inequalities*

$$S(t) > 0, \quad I(t) > 0, \quad R(t) \geq 0$$

hold.

Proof. Assume that the function $S(t)$ becomes nonpositive at some point. By continuity, it must cross the t-axis. If $S(t_1) = 0$ at some time $t_1 > 0$, then

$$\frac{dS(t_1)}{dt} = \gamma N_0 + e^{-\mu(\tau_1+\tau_2)}J(t_1 - \tau_1 - \tau_2) \geq \gamma N_0 > 0.$$

Repeating the same reasoning as in the lemma above, we conclude that $S(t) > 0$ for all $t > 0$.

Now consider the integral expression for the number of infectious individuals given by

$$I(t) = \int_{t-\tau_1}^{t} J(u)e^{-\mu(t-u)}du.$$

Fix an arbitrary $t^* > 0$. Consider two cases depending on the value of $t^* - \tau_1$.

- **Case 1.** $t^* - \tau_1 < 0$. Given the initial conditions where $I(\theta) = 0$ for all $\theta \in [-(\tau_1 + \tau_2), 0)$, the integral becomes as

$$I(t^*) = \int_{0}^{t^*} J(u)e^{-\mu(t^*-u)}du > 0,$$

since $J(u) > 0$ for all $u > 0$ according to Lemma 2.

- **Case 2.** $t^* - \tau_1 \geq 0$. We obtain

$$I(t^*) = \int_{t^*-\tau_1}^{t^*} J(u)e^{-\mu(t^*-u)}du > 0,$$

because $J(u) > 0$ for all $u > 0$ by Lemma 2.

Thus, for any $t > 0$, we have $I(t) > 0$. We also have the integral expression for $R(t)$, derived from (2.3) as follows:

$$R(t) = \int_{t-\tau_1-\tau_2}^{t-\tau_1} J(u)e^{-\mu\tau_1}e^{-\mu(t-\tau_1-u)}du.$$

Applying similar reasoning to that used for $I(t)$, we conclude that $R(t) \geq 0$ for all $t > 0$. □

Remark 4. *From Theorem 3 and equality* (3.1)*, we demonstrate the boundedness of the solution of system* (3.2).

3.1.2. Existence and uniqueness of solution

Now, we proceed to the proof of the existence theorem. We will prove the existence and uniqueness of the solution of system (3.2) for $t \in [0, n(\tau_1 + \tau_2)]$ where $n \in \mathbb{N}$, with the initial conditions (3.3).

Note that if (3.2a) and (3.2b) have unique solutions, then $J(t)$ is uniquely determined. Thus, Eq (3.2c) has a unique solution. Hence, it is sufficient to prove the existence and uniqueness of solution for the Eqs (3.2a) and (3.2b).

Let us set $\theta = \tau_1 + \tau_2$, and let (S_n, I_n, R_n) be the restriction of the solution (S, I, R) on the interval $[(n-1)\theta, n\theta]$, $n \in \mathbb{N}$. We have the following theorem.

Theorem 5. *If there exists a unique solution* $(S_{n-1}, I_{n-1}, R_{n-1})$ *of system* (3.2) *in the domain*

$$\widehat{\Sigma}_{n-1} = \left\{(S, I, R) \in \Sigma^3_{n-1} \ : \ \zeta_{n-1}(t) = \frac{\beta S(t-\theta)I(t-\theta)}{N_0} \geq 0\right\},$$

where Σ_{n-1} *is defined by*

$$\Sigma_{n-1} = \{T_{n-1} \in C([(n-2)\theta, (n-1)\theta], \mathbb{R}) : 0 \leq T_{n-1}(t) \leq N_0, \forall t \in [(n-2)\theta, (n-1)\theta]\},$$

then the system (3.2) *will have a unique solution* (S_n, I_n, R_n) *in the domain*

$$\widehat{\Sigma}_n = \left\{(S, I, R) \in \Sigma^3_n \ : \ \zeta_n(t) = \frac{\beta S(t-\theta)I(t-\theta)}{N_0} \geq 0\right\},$$

where

$$\Sigma_n = \{T_n \in C([(n-1)\theta, n\theta], \mathbb{R}) : 0 \leq T_n(t) \leq N_0, \forall t \in [(n-1)\theta, n\theta]\}, \ n \in \mathbb{N}.$$

Proof. If $t \in [(n-1)\theta, n\theta]$ where $n \in \mathbb{N}$, then $t - \theta \in [(n-2)\theta, (n-1)\theta]$ and $J(t-\theta)$ is known and determined in the previous time interval of the function $J(t)$. Set $\zeta_n(t) = J(t-\theta)$. For $n = 1$, the solution is given by the function on $[-\theta, 0]$, and ζ_1 is given as

$$\zeta_1(x) = \begin{cases} 0 & \text{if } x \in [-\theta, 0), \\ \frac{\beta}{N_0} S(0)I(0) > 0 & \text{if } x = 0. \end{cases}$$

When $t \in [(n-1)\theta, n\theta]$, then system (3.2) becomes

$$\frac{dS(t)}{dt} = \gamma N_0 - J(t) + \zeta_n(t)e^{-\mu(\tau_1+\tau_2)} - \mu S(t), \tag{3.4a}$$

$$\frac{dI(t)}{dt} = J(t) - J(t-\tau_1)e^{-\mu\tau_1} - \mu I(t), \tag{3.4b}$$

$$\frac{dR(t)}{dt} = J(t-\tau_1) - \zeta_n(t)e^{-\mu(\tau_1+\tau_2)} - \mu R(t), \tag{3.4c}$$

$$J(t) = \frac{\beta}{N_0} S(t)I(t), \tag{3.4d}$$

where $\zeta_n(t) = J(t-\theta_1)$ as explained previously. To prove this theorem, we need a mathematical setup of complete metric space, which is defined properly in the following lemma.

Lemma 6. *(Σ_n, d) is a complete metric space with respect to the metric $d(T_n^1, T_n^2)$ defined by*

$$d(T_n^1, T_n^2) = \sup_{t\in[(n-1)\theta, n\theta]} \left\{e^{-\nu t}|T_n^1(t) - T_n^2(t)|\right\}, n \in \mathbb{N}$$

and $\nu \geq 0$ is a constant.

Proof. First, we prove that Σ_n is a complete metric space with respect to the supremum metric given by the equality

$$d_{sup}(T_n^1, T_n^2) = \sup_{t\in[(n-1)\theta, n\theta]} |T_n^1(t) - T_n^2(t)|.$$

Consider a Cauchy sequence $\{T_n^i(t)\}$ in Σ_n. Then, for any $\epsilon > 0$, there exists $M_0 \in \mathbb{N}$ such that

$$d_{sup}(T_n^i, T_n^j) = \sup_{t\in[(n-1)\theta, n\theta]} |T_n^i(t) - T_n^j(t)| < \epsilon \text{ for } i, j \geq M_0.$$

Therefore, for all $t \in [(n-1)\theta, n\theta]$, $\{T_n^i(t)\}$ is a Cauchy sequence in $\mathbb{R}$ and, hence, converges to a real number denoted by $T_n(t)$. Choose any $t \in [(n-1)\theta, n\theta]$. Hence, there exists $C_t \in \mathbb{N}$ such that if $c > C_t$, then $|T_n^c(t) - T_n(t)| < \epsilon/2$. Furthermore, since $\{T_n^i\}$ is a Cauchy sequence in (Σ_n, d_{sup}), there exists M_1 such that

$$d_{sup}(T_n^i, T_n^j) = \sup_{t\in[(n-1)\theta, n\theta]} |T_n^i(t) - T_n^j(t)| < \epsilon/2 \text{ for } i, j \geq M_1.$$

Next, choose $c > max\{M_1, C_t\}$. Then, for all $i \geq M_1$,

$$|T_n^i(t) - T_n(t)| = |T_n^i(t) - T_n^c(t) + T_n^c(t) - T_n(t)| \leq |T_n^i(t) - T_n^c(t)| + |T_n^c(t) - T_n(t)| < \epsilon.$$

Taking supremum over $[(n-1)\theta, n\theta]$ in both sides of the above inequality, we get

$$d_{sup}(T_n^i, T_n) < \epsilon, \text{ for } i \geq M_1.$$

It remains to show that $T_n \in \Sigma_n$. It is clear that for all $i \in \mathbb{N}$, $0 \leq T_n^i(t) \leq N_0$, for all $t \in [(n-1)\theta, n\theta]$. Taking limit as $i \to \infty$, we get $0 \leq T_n(t) \leq N_0$, for all $t \in [(n-1)\theta, n\theta]$. Take any $t_0 \in [(n-1)\theta, n\theta]$. Then,

$$\lim_{t\to t_0} T_n(t) = \lim_{t\to t_0}\lim_{i\to\infty} T_n^i(t) = \lim_{i\to\infty}\lim_{t\to t_0} T_n^i(t) = \lim_{i\to\infty} T_n^i(t_0) = T_n(t_0),$$

which proves that T_n is continuous at t_0. Thus, $T_n \in \Sigma_n$, and, hence, (Σ_n, d_{sup}) is a complete metric space. Next, we have the following relation between the two metrics d and d_{sup} on Σ_n:

$$e^{-n\theta\nu} d_{sup}(T_n^1, T_n^2) \leq d(T_n^1, T_n^2) \leq e^{-(n-1)\theta\nu} d_{sup}(T_n^1, T_n^2),$$

which implies that d and d_{sup} are equivalent metrics. This proves that (Σ_n, d) is a complete metric space. □

We now proceed to prove the existence and uniqueness of solution of system (3.4a) and (3.4b) in the metric space (Σ_n, d). For any given function $T(t) \in \Sigma_n$, the equation

$$\frac{dS(t)}{dt} = \gamma N_0 - \frac{\beta}{N_0} S(t)T(t) + \zeta_n(t)e^{-\mu(\tau_1+\tau_2)} - \mu S(t), \tag{3.5}$$

with the condition $S((n-1)\theta) \geq 0,\ n \in \mathbb{N}$, can be written as

$$\frac{dS(t)}{dt} + (\frac{\beta}{N_0}T(t) + \mu)S(t) = \gamma N_0 + \zeta_n(t)e^{-\mu(\tau_1+\tau_2)},$$

and it has a unique solution given by

$$S_T(t) = (S_T((n-1)\theta) + \int_{(n-1)\theta}^{t} b(x)e^{\Lambda_T(x)}dx)e^{-\Lambda_T(t)}.$$

We set

$$S_T(t) = C_T(t)e^{-\Lambda_T(t)}, \tag{3.6}$$

where

$$C_T(t) = S_T((n-1)\theta) + \int_{(n-1)\theta}^{t} b(x)e^{\Lambda_T(x)}dx, \tag{3.7}$$

$$b(u) = \gamma N_0 + \zeta_n(u)e^{-\mu(\tau_1+\tau_2)}, \tag{3.8}$$

and

$$\Lambda_T(u) = \int_{(n-1)\theta}^{u} (\frac{\beta}{N_0}T(s) + \mu)ds. \tag{3.9}$$

Since $\zeta_n \geq 0$, we can ensure that $S_T(t) \geq 0$, i.e., $S_n \geq 0$, in the current interval. Since the functions T and S_T are positive and bounded, then the function C_T is bounded. There is a positive number $\tilde{c}_T$ such that $C_T(t) \leq \tilde{c}_T : \forall t \geq 0$.

Lemma 7. *Function C_T defined in* (3.7) *is Lipschitz in T.*

Proof. From the boundedness of $T(s)$, $\zeta_n(u)$, and $S_T((n-1)$, we can write $|T(s)| \leq M$ for all s and some $M > 0$ and $|\zeta_n(u)| \leq Z$ for all u and some $Z > 0$. This ensures $b(u)$ is bounded, that is,

$$|b(u)| \leq \gamma N_0 + Ze^{-\mu(\tau_1+\tau_2)} =: B.$$

Since the initial condition depends continuously on T, then $S_T((n-1)\theta)$ is Lipschitz in T, i.e., there exists $K_0 > 0$ such that

$$|S_{T_1}((n-1)\theta) - S_{T_2}((n-1)\theta)| \leq K_0\, d_{sup}(T_1, T_2).$$

Since t is in a bounded interval $[(n-1)\theta, (n-1)\theta + L_0]$, then for some $L_0 > 0$, $|t-(n-1)\theta| \leq L_0$. Next,

$$|C_{T_1}(t) - C_{T_2}(t)| = \left|[S_{T_1}((n-1)\theta) - S_{T_2}((n-1)\theta)] + \int_{(n-1)\theta}^{t} b(x)\left(e^{\Lambda_{T_1}(x)} - e^{\Lambda_{T_2}(x)}\right)dx\right|.$$

Using the triangle inequality, we get

$$|C_{T_1}(t) - C_{T_2}(t)| \leq |S_{T_1}((n-1)\theta) - S_{T_2}((n-1)\theta)| + \left|\int_{(n-1)\theta}^{t} b(x)\left(e^{\Lambda_{T_1}(x)} - e^{\Lambda_{T_2}(x)}\right)dx\right|.$$

We have,

$$|S_{T_1}((n-1)\theta) - S_{T_2}((n-1)\theta)| \leq K_0\, d_{sup}(T_1, T_2),$$

and

$$\int_{(n-1)\theta}^{t} b(x)\left(e^{\Lambda_{T_1}(x)} - e^{\Lambda_{T_2}(x)}\right)dx \leq B\int_{(n-1)\theta}^{t}\left|e^{\Lambda_{T_1}(x)} - e^{\Lambda_{T_2}(x)}\right|dx.$$

The exponential function is Lipschitz on bounded domains. Specifically, $\Lambda_T(x)$ is bounded because $|T(s)| \leq M$ and $|x-(n-1)\theta| \leq L_0$. Thus,

$$|\Lambda_T(x)| \leq \int_{(n-1)\theta}^{x}\left(\frac{\beta}{N_0}M + \mu\right)ds \leq \left(\frac{\beta M}{N_0} + \mu\right)L_0 =: R.$$

The function e^z is Lipschitz on $[-R, R]$ with constant e^R (since $|de^z/dz| = e^z \leq e^R$). So we can write

$$\left|e^{\Lambda_{T_1}(x)} - e^{\Lambda_{T_2}(x)}\right| \leq e^R|\Lambda_{T_1}(x) - \Lambda_{T_2}(x)|.$$

Hence,

$$|\Lambda_{T_1}(x) - \Lambda_{T_2}(x)| = \left|\frac{\beta}{N_0}\int_{(n-1)\theta}^{x}(T_1(s) - T_2(s))ds\right|$$

$$\leq \frac{\beta}{N_0}d_{sup}(T_1, T_2)\,|x-(n-1)\theta| \leq \frac{\beta}{N_0}L_0\,d_{sup}(T_1, T_2).$$

Combining these, we get

$$\left|e^{\Lambda_{T_1}(x)} - e^{\Lambda_{T_2}(x)}\right| \leq e^R\,\frac{\beta}{N_0}L_0\,d_{sup}(T_1, T_2).$$

Therefore, we have

$$\int_{(n-1)\theta}^{t} b(x)\left(e^{\Lambda_{T_1}(x)} - e^{\Lambda_{T_2}(x)}\right)dx \leq B\int_{(n-1)\theta}^{t} e^R\,\frac{\beta}{N_0}L_0\,d_{sup}(T_1, T_2)dx$$

$$\leq Be^R\frac{\beta}{N_0}L_0\,d_{sup}(T_1, T_2)\,L_0 = Be^R\frac{\beta}{N_0}L_0^2\,d_{sup}(T_1, T_2).$$

Finally, we can write

$$|C_{T_1}(t) - C_{T_2}(t)| \leq \left(K_0 + Be^R\frac{\beta}{N_0}L_0^2\right)d_{sup}(T_1, T_2),$$

where $R = \left(\frac{\beta M}{N_0} + \mu\right)L$. Thus, C_T is Lipschitz in T with constant $K = K_0 + Be^R\frac{\beta}{N_0}L_0^2$. □

Note that subscript T is used to denote the unique solution of Eq (3.5) for a given function $T(t) \in \Sigma_n$. Let us denote $J_T(t) = \frac{\beta}{N_0}S_T(t)T(t)$, then the equation

$$\frac{dI(t)}{dt} = \frac{\beta}{N_0}S_T(t)T(t) - \frac{\beta}{N_0}S_T(t-\tau_1)T(t-\tau_1)e^{-\mu\tau_1} - \mu T(t), \tag{3.10}$$

with the condition $I_T((n-1)\theta) \geq 0$ and $t \in [(n-1)\theta, n\theta]$ also has a unique solution, which can be written in the form

$$I_T(t) = I_T((n-1)\theta) + \int_{(n-1)\theta}^{t} H(\xi, T)d\xi,$$

where

$$H(\xi, T) = \frac{\beta}{N_0}C_T(\xi)T(\xi)e^{-\Lambda_T(\xi)} - \frac{\beta}{N_0}C_T(\xi-\tau_1)T(\xi-\tau_1)e^{-\Lambda_T(\xi-\tau_1)} - \mu T(\xi). \tag{3.11}$$

Let us consider the map $L : (\Sigma_n, d) \to (\Sigma_n, d)$ defined by the equality

$$L(T(t)) = I_T((n-1)\theta) + \int_{(n-1)\theta}^{t} H(\xi, T)d\xi, \tag{3.12}$$

where $H(\xi, T)$ satisfies (3.11). Before proceeding further, we verify that L maps (Σ_n, d) into itself.

Lemma 8. *The map $L : (\Sigma_n, d) \to (\Sigma_n, d)$ defined in* (3.12) *is well-defined.*

Proof. We have

$$H(\xi, T) = -\Big(\frac{dS_T(\xi)}{d\xi} + \frac{dR_T(\xi)}{d\xi}\Big).$$

Next,

$$\int_{(n-1)\theta}^{t} H(\xi, T)d\xi = -\Big(\int_{(n-1)\theta}^{t} \frac{dS_T(\xi)}{d\xi}d\xi + \int_{(n-1)\theta}^{t} \frac{dR_T(\xi)}{d\xi}d\xi\Big).$$

Thus,

$$\int_{(n-1)\theta}^{t} H(\xi, T)d\xi = S_T((n-1)\theta) + R_T((n-1)\theta) - (S_T(t) + R_T(t)).$$

Hence,

$$I_T((n-1)\theta) + \int_{(n-1)\theta}^{t} H(\xi, T)d\xi = N_0 - (S_T(t) + R_T(t)).$$

This implies $L(T(t)) = I_T((n-1)\theta) + \int_{(n-1)\theta}^{t} H(\xi, T)d\xi$ lies between 0 and N_0. Let us also note that if $T_1(t), T_2(t) \in \Sigma_n$ and $T_1(t) = T_2(t)$, then $S_{T_1}(t) = S_{T_2}(t)$, and, consequently, $H(\xi, T_1) = H(\xi, T_2)$. Hence, the map L is well-defined. □

Next, we prove that the map $L : (\Sigma_n, d) \to (\Sigma_n, d)$ defined in (3.12) is a contraction.

Lemma 9. *The map $L : (\Sigma_n, d) \to (\Sigma_n, d)$ defined in* (3.12) *is a contraction map.*

Proof. For any two functions $T_1(t), T_2(t) \in \Sigma_n$,

$$|L(T_1(t)) - L(T_2(t))| \leq \int_{(n-1)\theta}^{t} |H(\xi, T_1) - H(\xi, T_2)|d\xi.$$

Then, we have the following estimate:

$$\Big|H(\xi, T_1) - H(\xi, T_2)\Big| =$$

$$\frac{\beta}{N_0}\Big|\Big(C_{T_1}(\xi)T_1(\xi)e^{-\Lambda_{T_1}(\xi)} - C_{T_1}(\xi-\tau_1)T_1(\xi-\tau_1)e^{-\Lambda_{T_1}(\xi-\tau_1)} - \mu N_0 T_1(\xi)\Big)$$

$$-\Big(C_{T_2}(\xi)T_2(\xi)e^{-\Lambda_{T_2}(\xi)} - C_{T_2}(\xi-\tau_1)T_2(\xi-\tau_1)e^{-\Lambda_{T_2}(\xi-\tau_1)} - \mu N_0 T_2(\xi)\Big)\Big|.$$

Thus,

$$\Big|H(\xi, T_1) - H(\xi, T_2)\Big| =$$

$$\frac{\beta}{N_0}\Big|C_{T_1}(\xi)e^{-\Lambda_{T_1}(\xi)}(T_1(\xi)-T_2(\xi))+T_2(\xi)(C_{T_1}(\xi)e^{-\Lambda_{T_1}(\xi)}-C_{T_2}(\xi)e^{-\Lambda_{T_2}(\xi)})$$
$$+C_{T_1}(\xi-\tau_1)e^{-\Lambda_{T_1}(\xi-\tau_1)}(T_2(\xi-\tau_1)-T_1(\xi-\tau_1))+T_2(\xi-\tau_1)(C_{T_2}(\xi-\tau_1)e^{-\Lambda_{T_2}(\xi-\tau_1)}$$
$$-C_{T_1}(\xi-\tau_1)e^{-\Lambda_{T_1}(\xi-\tau_1)})+\mu N_0(T_2(\xi)-T_1(\xi))\Big|.$$

We use the following properties:

- $C_T(\rho)\le\tilde{c}_T$, and $\tilde{c}=\max\{\tilde{c}_{T_1},\tilde{c}_{T_2}\}$ is a uniform bound, so $|C_T(\rho)|\le\tilde{c}$ for all ρ and both T_1 and T_2.
- $|e^{-\rho}|\le 1$ and $|e^{-\rho}-e^{-\mu}|\le|\rho-\mu|$ for $\rho\ge 0,\mu\ge 0$.
- The domain is bounded: $|u-(n-1)\theta|\le\chi$ for some $\chi>0$, so $|\Lambda_{T_1}(u)-\Lambda_{T_2}(u)|\le\frac{\beta}{N_0}\int_{(n-1)\theta}^{u}|T_1(s)-T_2(s)|ds$.
- $|T_i(\xi)|\le M$ for $i=1,2$ and all ξ, with $M>0$.
- C_T is Lipschitz in T by Lemma 3, that is, $|C_{T_1}(u)-C_{T_2}(u)|\le K|T_1(u)-T_2(u)|$ for some $K>0$, uniformly in u.
- $|T_1(\xi)-T_2(\xi)|\le e^{\nu\xi}d(T_1,T_2)$.

Applying the triangle inequality and the above properties, we get

$$\big|H(\xi,T_1)-H(\xi,T_2)\big|\le\frac{\beta}{N_0}\sum_{i=1}^{5}|A_i|,$$

where

- $A_1=C_{T_1}(\xi)e^{-\Lambda_{T_1}(\xi)}(T_1(\xi)-T_2(\xi))$,
- $A_2=T_2(\xi)\Big(C_{T_1}(\xi)e^{-\Lambda_{T_1}(\xi)}-C_{T_2}(\xi)e^{-\Lambda_{T_2}(\xi)}\Big)$,
- $A_3=C_{T_1}(\xi-\tau_1)e^{-\Lambda_{T_1}(\xi-\tau_1)}(T_2(\xi-\tau_1)-T_1(\xi-\tau_1))$,
- $A_4=T_2(\xi-\tau_1)\Big(C_{T_2}(\xi-\tau_1)e^{-\Lambda_{T_2}(\xi-\tau_1)}-C_{T_1}(\xi-\tau_1)e^{-\Lambda_{T_1}(\xi-\tau_1)}\Big)$,
- $A_5=\mu N_0(T_1(\xi)-T_2(\xi))$.

Thus,

- $|A_1|\le\tilde{c}\cdot 1\cdot|T_1(\xi)-T_2(\xi)|=\tilde{c}|T_1(\xi)-T_2(\xi)|\le\tilde{c}e^{\nu\xi}d(T_1,T_2)$,
- $$|A_2|\le|T_2(\xi)|\Big[|C_{T_1}(\xi)-C_{T_2}(\xi)|\cdot|e^{-\Lambda_{T_1}(\xi)}|+|C_{T_2}(\xi)|\cdot|e^{-\Lambda_{T_1}(\xi)}-e^{-\Lambda_{T_2}(\xi)}|\Big]\le$$
$$M\left[Ke^{\nu\xi}d(T_1,T_2)\cdot 1+\tilde{c}\cdot\frac{\beta}{N_0}\chi e^{\nu\xi}d(T_1,T_2)\right]=(MK+\frac{M\tilde{c}\beta\chi}{N_0})e^{\nu\xi}d(T_1,T_2),$$
- $|A_3|\le\tilde{c}\cdot 1\cdot e^{\nu\xi}d(T_1,T_2)=\tilde{c}e^{\nu\xi}d(T_1,T_2)$,
- $$|A_4|\le|T_2(\xi-\tau_1)|[|C_{T_2}(\xi-\tau_1)-C_{T_1}(\xi-\tau_1)|\cdot|e^{-\Lambda_{T_2}(\xi-\tau_1)}|+|C_{T_1}(\xi-\tau_1)|\cdot|e^{-\Lambda_{T_2}(\xi-\tau_1)}$$
$$-e^{-\Lambda_{T_1}(\xi-\tau_1)}|]\le M\left[Ke^{\nu\xi}d(T_1,T_2)\cdot 1+\tilde{c}\cdot\frac{\beta}{N_0}\chi e^{\nu\xi}d(T_1,T_2)\right]$$
$$=(MK+\frac{M\tilde{c}\beta\chi}{N_0})e^{\nu\xi}d(T_1,T_2),$$
- $|A_5|\le\mu N_0e^{\nu\xi}d(T_1,T_2)$.

Hence,

$$\sum_{i=1}^{5}|A_i| \leq \tilde{c}e^{\nu\xi}d(T_1,T_2)+\left(MK+\frac{M\tilde{c}\beta\chi}{N_0}\right)e^{\nu\xi}d(T_1,T_2)+\tilde{c}e^{\nu\xi}d(T_1,T_2)$$

$$+\left(MK+\frac{M\tilde{c}\beta\chi}{N_0}\right)e^{\nu\xi}d(T_1,T_2)+\mu N_0 e^{\nu\xi}d(T_1,T_2)$$

$$=\left(2\tilde{c}+2MK+\frac{2M\tilde{c}\beta\chi}{N_0}+\mu N_0\right)e^{\nu\xi}d(T_1,T_2).$$

Multiplying by $\frac{\beta}{N_0}$, we get

$$\left|H(\xi,T_1)-H(\xi,T_2)\right| \leq \frac{\beta}{N_0}\left(2\tilde{c}+2MK+\frac{2M\tilde{c}\beta\chi}{N_0}+\mu N_0\right)e^{\nu\xi}d(T_1,T_2).$$

This inequality implies the estimate

$$\left|L(T_1(t))-L(T_2(t))\right| \leq \frac{\beta}{N_0}\left(2\tilde{c}+2MK+\frac{2M\tilde{c}\beta\chi}{N_0}+\mu N_0\right)d(T_1,T_2)\int_{(n-1)\theta}^{t}e^{\nu\xi}d\xi$$

$$=\frac{\beta}{N_0}\left(2\tilde{c}+2MK+\frac{2M\tilde{c}\beta\chi}{N_0}+\mu N_0\right)\frac{e^{\nu t}-1}{\nu}d(T_1,T_2)$$

$$\leq \frac{\beta}{N_0}\left(2\tilde{c}+2MK+\frac{2M\tilde{c}\beta\chi}{N_0}+\mu N_0\right)\frac{e^{\nu t}}{\nu}d(T_1,T_2).$$

Therefore,

$$e^{-\nu t}\left|L(T_1(t))-L(T_2(t))\right| \leq \frac{\beta}{N_0}\left(2\tilde{c}+2MK+\frac{2M\tilde{c}\beta\chi}{N_0}+\mu N_0\right)\frac{1}{\nu}d(T_1,T_2).$$

Taking the supremum of both sides, we get

$$d(L(T_1),L(T_2)) \leq \frac{\beta}{N_0}\left(2\tilde{c}+2MK+\frac{2M\tilde{c}\beta\chi}{N_0}+\mu N_0\right)\frac{1}{\nu}d(T_1,T_2).$$

We choose the value of $\nu > 0$ large enough such that $\frac{\beta}{N_0}\left(2\tilde{c}+2MK+\frac{2M\tilde{c}\beta\chi}{N_0}+\mu N_0\right)\frac{1}{\nu} < 1$. Consequently, $L:(\Sigma_n,d)\to(\Sigma_n,d)$ is a contraction map on the complete metric space (Σ_n,d). □

To finish the proof of the existence of solution, we use the following theorem [38].

Theorem 10. *Let (Ω,d) be a complete metric space and let $\psi:\Omega\to\Omega$ be a contraction mapping on Ω. Then, ψ has a unique fixed point $x\in\Omega$ (such that $\psi(x)=x$).*

It follows from this theorem that the map L has a unique fixed point. Thus, there exists a unique function $T_u\in\Sigma$ satisfying the equality $T_u(t)=I_{T_u}((n-1)\theta))+\int_{(n-1)\theta}^{t}H(\xi,T_u)d\xi$, where $H(\xi,T_u)$ is given in (3.11). Besides, we note that $H(\xi,T)$ is a continuous function on $[(n-1)\theta,n\theta]$. Hence, the derivative $\frac{dT_u(t)}{dt}$ exists. This completes the proof of the existence and uniqueness of solution of system (3.2) on $[(n-1)\theta,n\theta]$, where $n\in\mathbb{N}$. □

As a consequence of this theorem, we can guarantee that $I \geq 0$ in the current interval, i.e., $I_n \geq 0$ in the current interval. Also, we have $S_n \geq 0$ in the current interval. Hence, we can say

$$\zeta_{n+1} = \frac{\beta S_{n+1}(t-\theta) I_{n+1}(t-\theta)}{N_0} \geq 0, \text{ in } \Sigma_{n+1}.$$

Consequently, we have the following theorem.

Theorem 11. *There exists a unique solution* (S_u, I_u, R_u) *of system* (3.2) *in the domain* Σ^3*, where* Σ *is defined by*

$$\Sigma = \{T \in C([0, n\theta], \mathbb{R}) : 0 \leq T(t) \leq N_0, \forall t \in [0, n\theta] : \ n \in \mathbb{N}, \ T(t) = 0 : t \in [-\theta, 0)\} = \bigcup_{j=1}^{n} \Sigma_j,$$

and

$$S_u = \sum_{j=1}^{n} S_j \Upsilon_{\left[(j-1)\theta, j\theta\right)}, \quad I_u = \sum_{j=1}^{n} I_j \Upsilon_{\left[(j-1)\theta, j\theta\right)}, \quad R_u = \sum_{j=1}^{n} R_j \Upsilon_{\left[(j-1)\theta, j\theta\right)},$$

where Υ_A *is the indicator function that takes value* 1 *in the set A and* 0 *otherwise.*

3.2. Stationary states of the system

We now consider the possibility of a stationary state for system (3.2), that is, constant values (S^*, I^*, R^*) at which all derivatives vanish. First, note that the system always admits a trivial stationary state corresponding to the complete absence of infection $(S^*, I^*, R^*) = (N_0, 0, 0)$. This solution reflects the situation in which the epidemic does not spread. More interesting is the study of a nontrivial stationary state with $I^* > 0$. We set the righthand sides of Eqs (3.2a)–(3.2c) to zero and assume $J(t) \equiv J^* = \dfrac{\beta}{N_0} S^* I^*$ is constant. We obtain a system of algebraic equations from which the stationary values are expressed as follows:

- Number of susceptible individuals:

$$S^* = \frac{\mu N_0}{\beta(1 - e^{-\mu\tau_1})}. \tag{3.13}$$

- Number of infected individuals:

$$I^* = N_0 \left(1 - \frac{\mu}{\beta(1 - e^{-\mu\tau_1})}\right)\left(\frac{1 - e^{-\mu\tau_1}}{1 - e^{-\mu(\tau_1+\tau_2)}}\right). \tag{3.14}$$

- Number of recovered individuals:

$$R^* = \frac{N_0 \left(1 - \frac{\mu}{\beta(1-e^{-\mu\tau_1})}\right) e^{-\mu\tau_1}(1 - e^{-\mu\tau_2})}{1 - e^{-\mu(\tau_1+\tau_2)}}. \tag{3.15}$$

Thus, the stationary solution (S^*, I^*, R^*) is fully determined by the model parameters and the initial population size N_0. This stationary solution will be positive if the following condition holds:

$$\frac{\mu}{\beta(1 - e^{-\mu\tau_1})} < 1 \quad \Longleftrightarrow \quad \frac{\beta(1 - e^{-\mu\tau_1})}{\mu} > 1.$$

Therefore, we naturally define the basic reproduction number as

$$\Re_0 = \frac{\beta(1 - e^{-\mu\tau_1})}{\mu}. \tag{3.16}$$

Remark 12. *Consider the basic reproduction number $\Re_0$ as a function of the parameter μ. Then,*

$$\lim_{\mu\to 0^+} \Re_0(\mu) = \lim_{\mu\to 0^+} \frac{\beta(1 - e^{-\mu\tau_1})}{\mu} = \beta\tau_1.$$

This result is consistent with the previously obtained value of the basic reproduction number in the model without demographic processes, that is, $\gamma = \mu = \delta = 0$ (see [33, 34]).

Theorem 13. *If $\Re_0 < 1$, then the solution of system* (3.2) *tends to the trivial stationary solution, i.e.,*

$$\lim_{t\to\infty} S(t) = N_0, \quad \lim_{t\to\infty} I(t) = 0, \quad \lim_{t\to\infty} R(t) = 0.$$

Proof. From the nonnegativity of the solutions of system (3.2), it follows that they are bounded. That is,

$$S(t) \le N_0, \quad I(t) < N_0, \quad R(t) < N_0.$$

Then, from the integral form of $I(t)$, we derive the estimate

$$I(t) = \int_{t-\tau_1}^{t} J(u)e^{-\mu(t-u)}du \le \int_{t-\tau_1}^{t} \beta I(t)e^{-\mu(t-u)}du.$$

Define

$$M(t) = \sup_{s\in[t-\tau_1,t]} I(s).$$

Then,

$$I(t) \le \beta M(t) \int_{t-\tau_1}^{t} e^{-\mu(t-u)}du = \beta M(t) \int_0^{\tau_1} e^{-\mu s}ds = \beta M(t)\frac{1 - e^{-\mu\tau_1}}{\mu} = \Re_0 M(t).$$

Using the estimate above, we find an upper bound for $M(t)$ as follows:

$$M(t) = \sup_{s\in[t-\tau_1,t]} I(s) \le \sup_{s\in[t-\tau_1,t]} \Re_0 M(s) = \Re_0 \sup_{s\in[t-2\tau_1,t]} I(s).$$

Thus, we obtain

$$\sup_{s\in[t-\tau_1,t]} I(s) \le \Re_0 \sup_{s\in[t-2\tau_1,t]} I(s), \quad \sup_{s\in[t-2\tau_1,t]} I(s) \le \Re_0 \sup_{s\in[t-3\tau_1,t]} I(s), \quad \dots$$

Therefore, for any $n \in \mathbb{N}$, provided $t - (n + 1)\tau_1 \ge 0$, the following holds:

$$I(t) \le \Re_0 \sup_{s\in[t-\tau_1,t]} I(s) \le \Re_0^n \sup_{s\in[t-(n+1)\tau_1,t]} I(s).$$

Thus, for sufficiently large t and accordingly large $n \in \mathbb{N}$, we have

$$I(t) \le \Re_0^n \sup_{s\in[t-(n+1)\tau_1,t]} I(s).$$

Taking a rough estimate $I(t) \leq N_0$, and noting that $\sup_{s\in[0,t^*]} I(s) \leq N_0$ for any $t^* > 0$, we conclude that as $t \to \infty$ and $\Re_0 < 1$, then

$$\lim_{t\to\infty} I(t) \leq \lim_{n\to\infty} \Re_0^n N_0 = 0.$$

Hence, $\lim_{t\to\infty} I(t) = 0$.

Now, rewriting the differential equation for $R(t)$, we get

$$\frac{dR(t)}{dt} + \mu R(t) = e^{-\mu\tau_1} J(t-\tau_1) - e^{-\mu(\tau_1+\tau_2)} J(t-\tau_1-\tau_2).$$

Denote the righthand side as

$$f(t) = e^{-\mu\tau_1} J(t-\tau_1) - e^{-\mu(\tau_1+\tau_2)} J(t-\tau_1-\tau_2).$$

The general solution of this linear nonhomogeneous ODE is

$$R(t) = e^{-\mu t}\left(R(0) + \int_0^t e^{\mu s} f(s)ds\right) = e^{-\mu t}\int_0^t e^{\mu s} f(s)ds.$$

Since $J(t) = \frac{\beta}{N_0} S(t)I(t)$ and $S(t)$ is bounded above, from $\lim_{t\to\infty} I(t) = 0$, it follows that $\lim_{t\to\infty} J(t) = 0$, and, hence, $\lim_{t\to\infty} f(t) = 0$.

Fix an arbitrary $\varepsilon > 0$. Since $f(t) \to 0$ as $t \to \infty$, there exists $T > 0$ such that for all $s > T$, $|f(s)| < \frac{\varepsilon\mu}{2}$. We split the integral

$$\int_0^t e^{\mu s} f(s)ds = \int_0^T e^{\mu s} f(s)ds + \int_T^t e^{\mu s} f(s)ds.$$

The first term is a constant: $C_1 = \int_0^T e^{\mu s} f(s)ds$. Estimate the second term

$$\left|\int_T^t e^{\mu s} f(s)ds\right| \leq \frac{\varepsilon\mu}{2}\int_T^t e^{\mu s} ds = \varepsilon\frac{e^{\mu t} - e^{\mu T}}{2}.$$

Therefore,

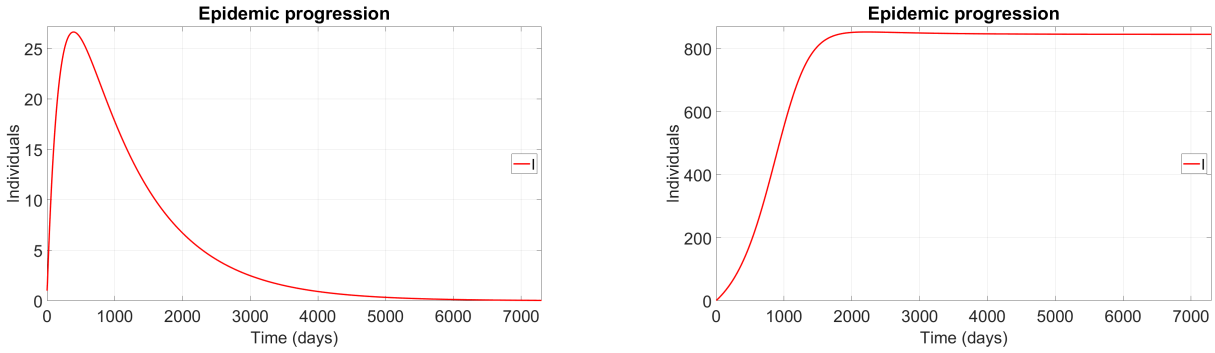
$$|R(t)| \leq e^{-\mu t}\left|C_1 + \varepsilon\frac{e^{\mu t} - e^{\mu T}}{2}\right| \leq e^{-\mu t}|C_1| + \varepsilon\frac{1 - e^{-\mu(t-T)}}{2}.$$

Now choose $T' > 0$ such that $e^{-\mu T'}|C_1| < \frac{\varepsilon}{2}$, i.e., $T' > \frac{1}{\mu}\ln\left(\frac{2|C_1|}{\varepsilon}\right)$. Thus, for $t > \max\{T, T'\}$, we have

$$|R(t)| \leq \frac{\varepsilon}{2} + \frac{\varepsilon}{2} = \varepsilon.$$

Since $\varepsilon > 0$ is arbitrary, we conclude that $\lim_{t\to\infty} R(t) = 0$. From this, it follows that $S(t) \to N_0$ as $t \to \infty$, since $S(t) + I(t) + R(t) = N_0$. □

This theoretical result is further supported by numerical example displayed in Figure 1 which illustrates that basic reproduction number $\Re_0$ has an important property, and it serves as a threshold parameter for the existence of an epidemic. If $\Re_0 < 1$ as in Figure 1(a), then the infection eventually dies out, and if $\Re_0$ equals 1 or slightly larger than 1 as in Figure 1(b), then a transition to a stationary state with the presence of infected individuals is possible.



(a) (b)

Figure 1. Numerical simulation of system (3.2) for the initial conditions $N_0 = 10^6$, $S(t) = N_0$, $I(t) = R(t) = 0\ \forall t < 0$, $S(0) = N_0 - 1$, $I(t) = 1$, $R(t) = 0$, and the parameters $\gamma = \mu = 10^{-3}$, $\tau_1 = 10$, $\tau_2 = 180$; (a) $\beta = 0.098$ ($\Re_0 \approx 0.98$); (b) $\beta = 0.102$ ($\Re_0 \approx 1.015$).

3.3. Stability analysis

Introduce the state vector of the system

$$X(t) = \begin{bmatrix} S(t) \\ I(t) \\ R(t) \end{bmatrix},$$

then system (3.2) can be written in vector form

$$\frac{d}{dt}X(t) = \mathcal{F}(X(t),\ X(t-\tau_1),\ X(t-\tau_1-\tau_2)),$$

where $\mathcal{F} : \mathbb{R}^3 \times \mathbb{R}^3 \times \mathbb{R}^3 \to \mathbb{R}^3$ is a nonlinear mapping. Consider the stationary solution $X^* = \left[S^*,\ I^*,\ R^*\right]^T$, satisfying $\mathcal{F}(X^*, X^*, X^*) = 0$. Adding a small perturbation to it, we get

$$X(t) = X^* + V(t), \quad \|V(t)\| \ll 1.$$

Linearizing the system around X^*, we obtain the approximate system

$$\frac{d}{dt}V(t) = J_1V(t) + J_2V(t-\tau_1) + J_3V(t-\tau_1-\tau_2),$$

where J_1, J_2, J_3 are Jacobian matrices evaluated at X^* with respect to $X(t), X(t-\tau_1), X(t-\tau_1-\tau_2)$, respectively.

Let $V(t) = e^{\lambda t}v$, where $v \in \mathbb{R}^3$. Then,

$$\lambda E v e^{\lambda t} = e^{\lambda t}J_1v + e^{\lambda(t-\tau_1)}J_2v + e^{\lambda(t-\tau_1-\tau_2)}J_3v,$$

where E is the identity matrix. Dividing both sides by $e^{\lambda t}$, we obtain

$$\lambda E v = J_1v + e^{-\lambda\tau_1}J_2v + e^{-\lambda(\tau_1+\tau_2)}J_3v.$$

Therefore, the characteristic equation of the system takes the form

$$\det\left(\lambda E - J_1 - e^{-\lambda\tau_1}J_2 - e^{-\lambda(\tau_1+\tau_2)}J_3\right) = 0. \tag{3.17}$$

The Jacobian matrices J_1, J_2, J_3 evaluated at X^* have the following form:

$$J_1 = \begin{bmatrix} -\frac{\beta}{N_0}I^* - \mu & -\frac{\beta}{N_0}S^* & 0 \\ \frac{\beta}{N_0}I^* & \frac{\beta}{N_0}S^* - \mu & 0 \\ 0 & 0 & -\mu \end{bmatrix}, \quad J_2 = \begin{bmatrix} 0 & 0 & 0 \\ -\frac{\beta}{N_0}e^{-\mu\tau_1}I^* & -\frac{\beta}{N_0}e^{-\mu\tau_1}S^* & 0 \\ \frac{\beta}{N_0}e^{-\mu\tau_1}I^* & \frac{\beta}{N_0}e^{-\mu\tau_1}S^* & 0 \end{bmatrix},$$

$$J_3 = \begin{bmatrix} \frac{\beta}{N_0}e^{-\mu(\tau_1+\tau_2)}I^* & \frac{\beta}{N_0}e^{-\mu(\tau_1+\tau_2)}S^* & 0 \\ 0 & 0 & 0 \\ -\frac{\beta}{N_0}e^{-\mu(\tau_1+\tau_2)}I^* & -\frac{\beta}{N_0}e^{-\mu(\tau_1+\tau_2)}S^* & 0 \end{bmatrix}.$$

Substituting the explicit forms of J_1, J_2, and J_3 into (3.17), we obtain the simplified characteristic equation

$$\begin{aligned}(\lambda+\mu)\Bigg[&\left(\lambda + \frac{\beta}{N_0}I^* + \mu - \frac{\beta}{N_0}I^*e^{-(\lambda+\mu)(\tau_1+\tau_2)}\right)\cdot\left(\lambda + \mu - \frac{\beta}{N_0}S^* + \frac{\beta}{N_0}S^*e^{-(\lambda+\mu)\tau_1}\right) \\ &-\left(\frac{\beta}{N_0}S^* - \frac{\beta}{N_0}S^*e^{-(\lambda+\mu)(\tau_1+\tau_2)}\right)\cdot\left(-\frac{\beta}{N_0}I^* + \frac{\beta}{N_0}I^*e^{-(\lambda+\mu)\tau_1}\right)\Bigg] = 0.\end{aligned} \tag{3.18}$$

Note that the characteristic equation is transcendental, so it can have an infinite number of roots.

Theorem 14. *Let z be an unstable characteristic root of equation* (3.17)*, i.e., $Re(z) \geq 0$. Then, the following estimate holds*

$$|z| \leq \|J_1\| + \|J_2\| + \|J_3\|,$$

where $\|\cdot\|$ denotes the matrix norm induced by the vector norm.

Proof. We define the matrix

$$W(z) = J_1 + e^{-z\tau_1}J_2 + e^{-z(\tau_1+\tau_2)}J_3.$$

Using this matrix, the characteristic Eq (3.17) can be rewritten as:

$$P(z;\tau_1;\tau_2) = \det(zE - W(z)) = 0.$$

This means that z is an eigenvalue of the matrix $W(z)$, and there exists an index $j \in \{1,2,3\}$ such that

$$z = \lambda_j, \quad \text{where } \lambda_j \in \sigma(W(z)),$$

where $\sigma(W(z))$ denotes the spectrum of the matrix $W(z)$.

We can use the property that for any eigenvalue λ of a matrix A ($\lambda \in \sigma(A)$), the inequality $|\lambda| \leq \|A\|$ holds [39]. Then:

$$|z| = |\lambda_j| \leq \|W(z)\| = \|J_1 + e^{-z\tau_1}J_2 + e^{-z(\tau_1+\tau_2)}J_3\|.$$

Applying the triangle inequality for matrix norms, we obtain

$$\|W(z)\| = \|J_1 + e^{-z\tau_1}J_2 + e^{-z(\tau_1+\tau_2)}J_3\| \leq \|J_1\| + \|J_2\| + \|J_3\|.$$

□

Remark 15. *This theorem implies that there exists a bounded region in the right half-plane of the complex plane $\mathbb{C}^+$ that contains all unstable characteristic roots of Eq* (3.17).

From the form of the characteristic Eq (3.18), it follows that

$$\lambda = -\mu$$

or

$$\begin{aligned}\left(\lambda + \frac{\beta}{N_0}I^* + \mu - \frac{\beta}{N_0}I^* e^{-(\lambda+\mu)(\tau_1+\tau_2)}\right)\cdot\left(\lambda + \mu - \frac{\beta}{N_0}S^* + \frac{\beta}{N_0}S^* e^{-(\lambda+\mu)\tau_1}\right)\\ -\left(\frac{\beta}{N_0}S^* - \frac{\beta}{N_0}S^* e^{-(\lambda+\mu)(\tau_1+\tau_2)}\right)\cdot\left(-\frac{\beta}{N_0}I^* + \frac{\beta}{N_0}I^* e^{-(\lambda+\mu)\tau_1}\right) = 0.\end{aligned} \tag{3.19}$$

Introducing the notation,

$$x = \lambda + \mu, \quad a = \frac{\beta}{N_0}I^*, \quad b = \frac{\beta}{N_0}S^*.$$

Then, Eq (3.19) becomes

$$(x + a - ae^{-x(\tau_1+\tau_2)})(x - b + be^{-x\tau_1}) - (b - be^{-x(\tau_1+\tau_2)})(ae^{-x\tau_1} - a) = 0.$$

Expanding the terms, we find

$$x[x + a(1 - e^{-x(\tau_1+\tau_2)}) + b(e^{-x\tau_1} - 1)] = 0.$$

Thus, either

$$\lambda = -\mu$$

or

$$\lambda = -a(1 - e^{-(\mu+\lambda)(\tau_1+\tau_2)}) - b(e^{-(\mu+\lambda)\tau_1} - 1) - \mu. \tag{3.20}$$

Lemma 16. *If $\Re_0 \geq 1$, then Eq (3.20) has no nontrivial positive real roots.*

Proof. Denote the righthand side of Eq (3.20) by $f(\lambda)$. Then,

$$f(\lambda) = -a(1 - e^{-(\mu+\lambda)(\tau_1+\tau_2)}) - b(e^{-(\mu+\lambda)\tau_1} - 1) - \mu,$$

$$f'(\lambda) = -a(\tau_1 + \tau_2)e^{-(\mu+\lambda)(\tau_1+\tau_2)} + b\tau_1 e^{-(\mu+\lambda)\tau_1}.$$

Note that $a \geq 0,\ b > 0$. Therefore,

$$f'(\lambda) \leq b\tau_1 e^{-(\mu+\lambda)\tau_1} = \frac{\mu\tau_1 e^{-(\mu+\lambda)\tau_1}}{1 - e^{-\mu\tau_1}} < \frac{\mu\tau_1 e^{-\mu\tau_1}}{1 - e^{-\mu\tau_1}} < 1, \quad \forall\lambda \in [0, \infty).$$

Equation $f(\lambda) = \lambda$ can be rewritten as

$$g(\lambda) := f(\lambda) - \lambda = 0.$$

We already know that $f'(\lambda) < 1$ for all $\lambda \geq 0$, so

$$g'(\lambda) = f'(\lambda) - 1 < 0, \quad \forall\lambda \in [0, \infty).$$

This means that $g(\lambda)$ is strictly decreasing on $[0, \infty)$. Evaluating $g(\lambda)$ at $\lambda = 0$, we get

$$g(0) = f(0) = -a(1 - e^{-\mu(\tau_1+\tau_2)}) \leq 0.$$

Since $g(\lambda)$ is strictly decreasing, then

$$\forall \lambda > 0 : \quad g(\lambda) < g(0) \leq 0.$$

Therefore, the equation $g(\lambda) = 0$ cannot have real positive solutions. This completes the proof of the lemma. □

The basic reproduction number, $\Re_0$, represents a fundamental epidemic threshold whose value determines whether a disease can establish itself in a population. The condition $\Re_0 > 1$ signifies that the pathogen is capable of sustained transmission, leading to an endemic state. The biological implication of $\Re_0$ is that conditions of the emergence of persistent epidemics are formulated in terms of the pathogen's transmission rate β, the duration of the infectious period τ_1, and the natural mortality rate of the host population μ.

The main observation from the bifurcation plot given by Figure 2(a) is that if $\Re_0 > 1$, system (3.2) could have either periodic solution when $\gamma = \mu$ are less than some critical value μ_c (Figure 2(b),(d)) or damped oscillation which leads to stable endemic solution when $\gamma = \mu$ exceeds that critical value μ_c (Figure 2(c),(e)). It remains to mention that the basic reproduction number corresponding to the parameters values of Figure 2 satisfies $\Re_0 \in [1.9, 2)$.

On the other hand, from the bifurcation graph given in Figure 3(a), we observe that if $\gamma = \mu < \mu_c$ and $\Re_0 > 1$, the system (3.2) could have either damped oscillation (stable endemic solution) when the disease transmission rate β is less than some critical value (Figure 3(b),(d)) or periodic solution when β exceeds that critical value (Figure 3(c),(e)). The basic reproduction number corresponding to the parameters values of Figure 3 satisfies $\Re_0 \in [1, 2)$. The transition to periodic oscillations via a Hopf bifurcation, along with the general theory for delay epidemic models, is studied in works such as [40–42].

Likewise, from the bifurcation graph given in Figure 4(a), we observe that if $\gamma = \mu < \mu_c$ and $\Re_0 > 1$, the system (3.2) could have either damped oscillation (stable endemic solution) when the disease duration τ_1 is less than some critical value (Figure 4(b),(d)) or periodic solution when τ_1 exceeds that critical value (Figure 4(c),(e)). The basic reproduction number corresponding to the parameters values of Figure 4 satisfies $\Re_0 \in (1.04, 3)$.

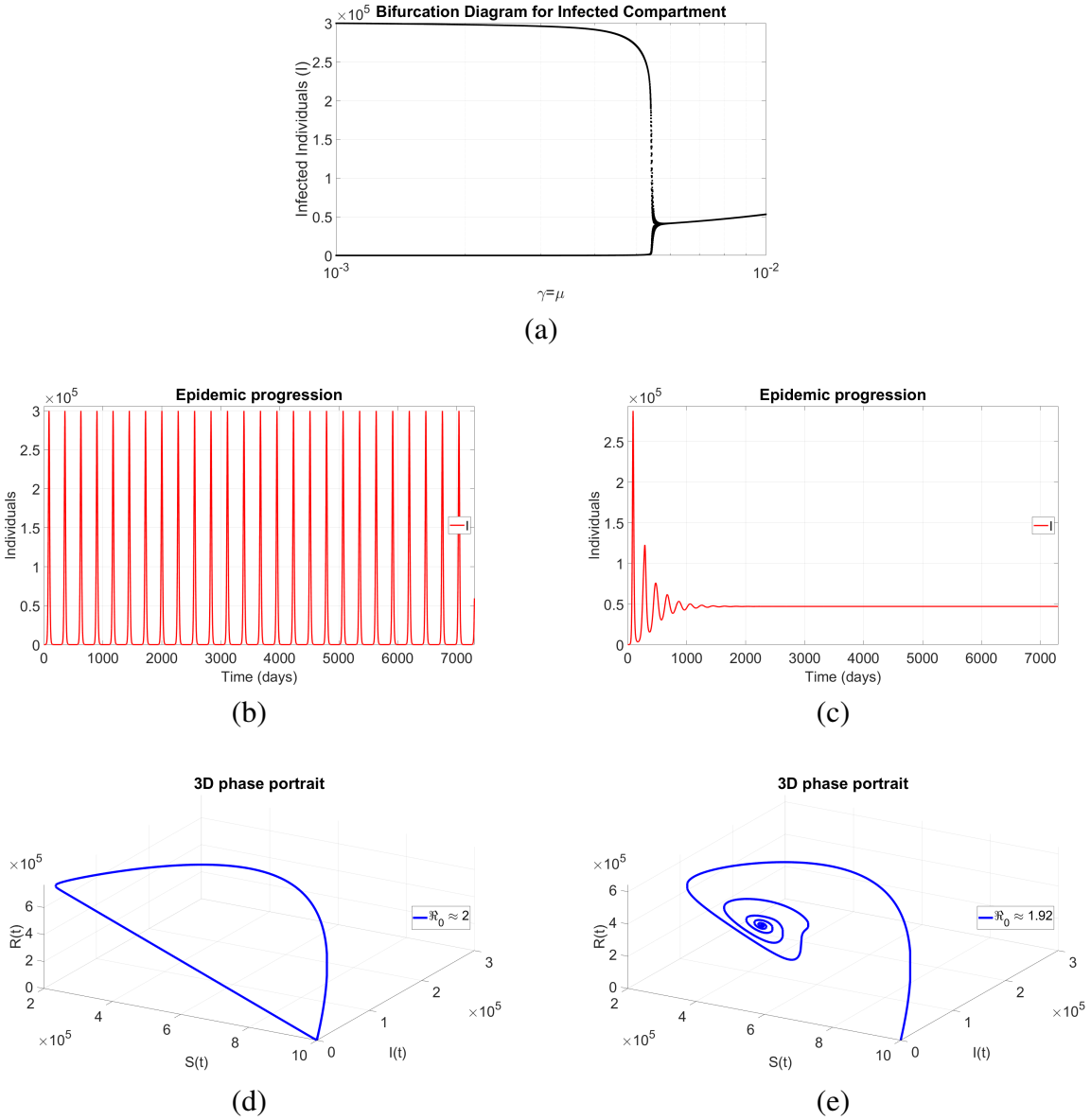


Figure 2. (a) The bifurcation of the infected compartment, that is, we plot the local maximums and local minimums of the oscillations after the graph of infected compartment stabilizes, against $\gamma = \mu$ in system (3.2) for the initial conditions $N_0 = 10^6$, $S(t) = N_0$, $I(t) = R(t) = 0$ $\forall t < 0$, $S(0) = N_0 - 1$, $I(t) = 1$, $R(t) = 0$, and the parameters $\beta = 0.2$, $\tau_1 = 10$, $\tau_2 = 180$; (b),(d) $\gamma = \mu = 10^{-3}$ ($\Re_0 \approx 2$); (c),(e) $\gamma = \mu = 8\times10^{-3}$ ($\Re_0 \approx 1.92$).

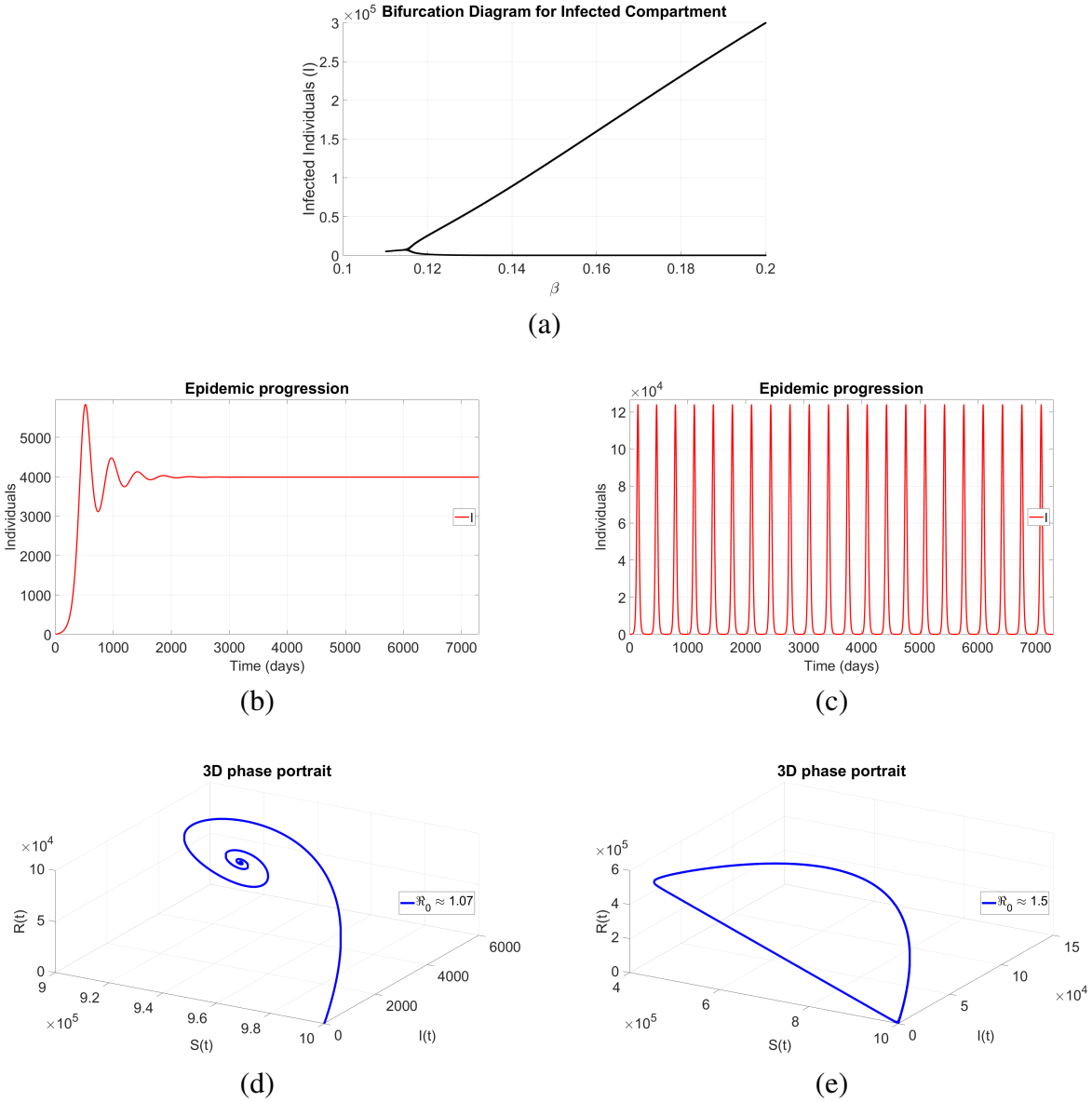


Figure 3. (a) The bifurcation of the infected compartment, that is, we plot the local maximums and local minimums of the oscillations after the graph of infected compartment stabilizes, against the parameter β in system (3.2) for the initial conditions $N_0 = 10^6$, $S(t) = N_0$, $I(t) = R(t) = 0\ \forall t < 0$, $S(0) = N_0 - 1$, $I(t) = 1$, $R(t) = 0$, and the parameters $\gamma = \mu = 10^{-3}$, $\tau_1 = 10$, $\tau_2 = 180$; (b),(d) $\beta = 0.108$ ($\Re_0 \approx 1.07$); (c),(e) $\beta = 0.15$ ($\Re_0 \approx 1.5$).

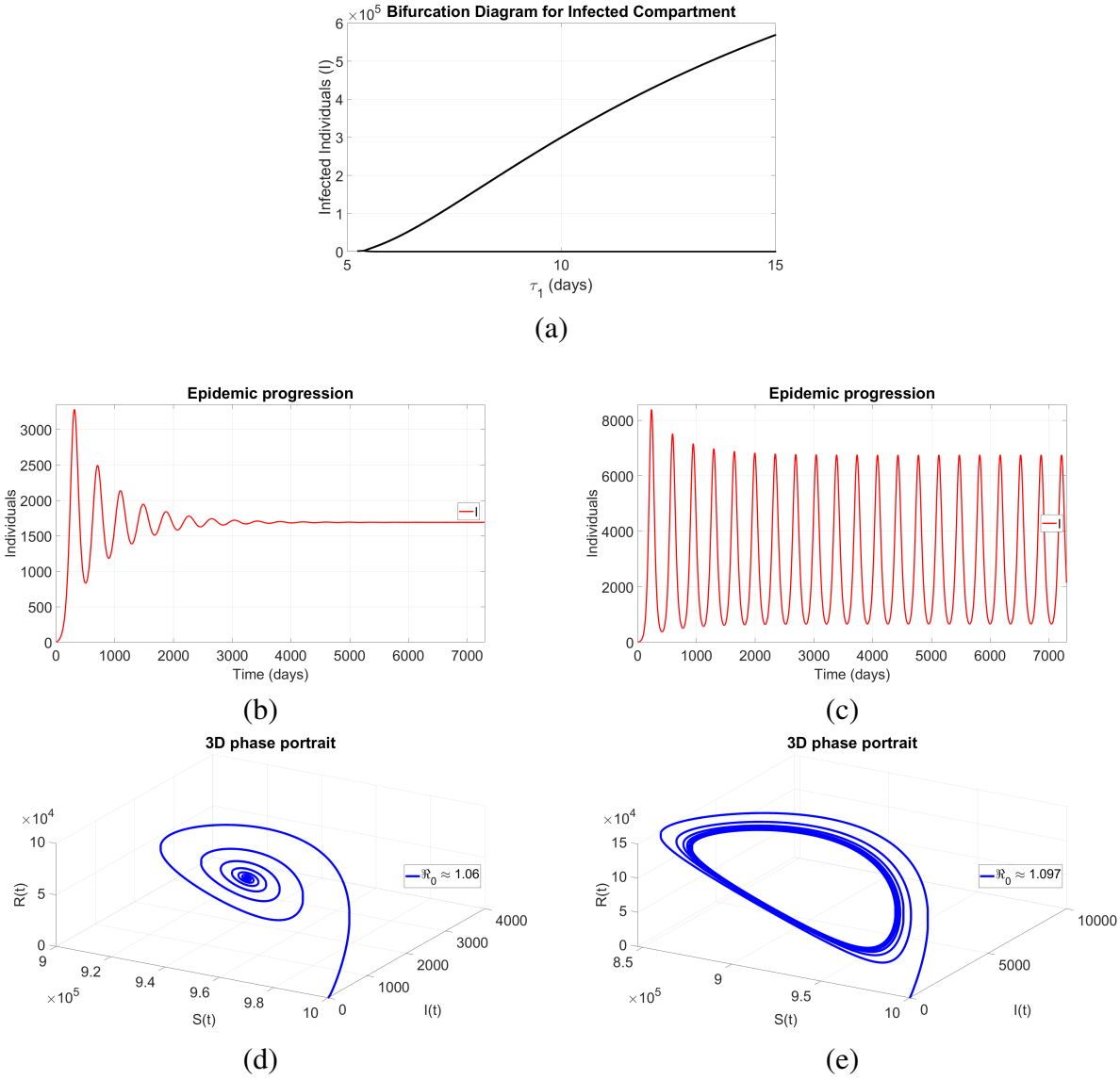


Figure 4. (a) The bifurcation of the infected compartment, that is, we plot the local maximums and local minimums of the oscillations after the graph of the infected compartment stabilizes, against the parameter τ_1 in system (3.2) for the initial conditions $N_0 = 10^6$, $S(t) = N_0$, $I(t) = R(t) = 0$ $\forall t < 0$, $S(0) = N_0 - 1$, $I(t) = 1$, $R(t) = 0$, and the parameters $\beta = 0.2$, $\gamma = \mu = 10^{-3}$, $\tau_2 = 180$; (b),(d) $\tau_1 = 5.3$ ($\Re_0 \approx 1.06$); (c),(e) $\tau_1 = 5.5$ ($\Re_0 \approx 1.097$).

The bifurcation diagrams in Figures 2(a), 3(a), and 4(a) collectively demonstrate that the system undergoes a supercritical Hopf bifurcation for each parameter. This is characterized by a transition from a stable endemic equilibrium to stable periodic oscillations as the respective parameter crosses a critical threshold. Specifically, this transition occurs:

- In Figure 2(a), as the mortality rate $\gamma = \mu$ decreases past the critical value $\mu_c \approx 0.006$.
- In Figure 3(a), as the transmission rate β increases past $\beta_c \approx 0.115$.
- In Figure 4(a), as the infectious period delay τ_1 increases past $\tau_{1c} \approx 5.4$.

Consistently, for parameter $\gamma = \mu$ values above their critical thresholds ($\gamma = \mu = 8 \times 10^{-3} > \mu_c$ in Figure 2(b),(d); and parameters β and τ_1 below their critical thresholds $\beta = 0.108 < \beta_c$ in Figure 3(b),(d); $\tau_1 = 5.3 < \tau_{1c}$ in Figure 4 (b),(d), the solutions converge to a stable equilibrium. Conversely, for values below the critical thresholds ($\gamma = \mu = 10^{-3} < \mu_c$ in Figure 2(c),(e); and above their critical thresholds $\beta = 0.15 > \beta_c$ in Figure 3(c),(e); $\tau_1 = 5.5 > \tau_{1c}$ in Figure 4(c),(e), the solutions converge to a stable limit cycle. The stability analysis of the endemic equilibrium is not solely dependent on $\Re_0$. The same value of $\Re_0 > 1$ can lead to either a stable equilibrium or recurring waves, depending on the specific combination of underlying biological parameters (β, τ_1, μ).

Figures 2–4 show that the oscillation amplitude is highly sensitive to changes in the parameters β, τ_1, and μ near their critical bifurcation thresholds, but this sensitivity diminishes significantly when the parameters are far from these critical values.

One of the fundamental methods of sensitivity analysis is based on computing derivatives of model outputs with respect to input parameters. Considering the stationary values S^*, I^*, and R^* defined in equalities (3.13)–(3.15), we find

$$\frac{\partial S^*}{\partial \beta}/S^* = -\frac{1}{\beta}, \quad \frac{\partial I^*}{\partial \beta}/I^* = \frac{\partial R^*}{\partial \beta}/R^* = \frac{\mu}{\beta(\beta(1-e^{-\mu\tau_1})-\mu)}.$$

Since $\frac{\partial S^*}{\partial \beta}/S^*$ is always negative, meaning S^* decreases as β increases, the magnitude of sensitivity is inversely proportional to β. On the other hand, regarding I^* and R^*, the sign depends on the term $\beta(1-e^{-\mu\tau_1})-\mu$ in the denominator. However, since $\Re_0 > 1$ is the condition for the stationary values I^* and R^* to be positives, that is, $\beta(1-e^{-\mu\tau_1})-\mu > 0$, then both of I^* and R^* increase with β. An analogous procedure, computing the normalized derivatives with respect to τ_1 and μ, can be applied to comprehensively assess the sensitivity of the system to all its key parameters.

4. Discussion

This work presents a comprehensive analysis of an extended SIR model incorporating vital dynamics (birth and death rates) and discrete delays representing the infectious and immune periods. The core contributions are structured as follows. First, a novel system of DDEs is constructed to describe the dynamics of susceptible, infected, and recovered populations, generalizing previous models by including the parameters γ, μ, and δ.

Second, for the foundational special case of a demographically stable population without disease-induced mortality ($\gamma = \mu > 0,\ \delta = 0$), the work rigorously establishes the existence, nonnegativity, and uniqueness of the system's solutions. It further derives the disease-free equilibrium and a formula for the basic reproduction number $\Re_0$, providing the necessary threshold condition for an outbreak.

The disease-free equilibrium loses stability when $\Re_0 > 1$, leading to the emergence of a positive equilibrium. The stability of this endemic state, whether asymptotically stable or exhibiting periodic oscillations, depends not only on the basic reproduction number but also critically on the fertility/mortality rates. Specifically, within the demographically equilibrated system (3.2), the instability of the endemic stationary solution is governed by critical values of the fertility/mortality rate (μ_c), the disease transmission rate (β_c), and the disease duration (τ_{1c}), as visualized in Figures 2–4.

For the critical fertility/mortality rate μ_c, when $\Re_0 > 1$, the endemic equilibrium is locally asymptotically stable if $\mu_c < \mu = \gamma$, but becomes unstable if $\mu = \gamma < \mu_c$, resulting in oscillatory

behavior. The critical value μ_c signifies a demographic tipping point. In populations with a high birth rate (i.e., a young population where μ is effectively high), the model predicts a stable endemic state. In contrast, populations with lower birth rates (e.g., aging populations where μ is lower) are more likely to experience persistent epidemic waves if $\mu < \mu_c$. This underscores the need for public health planning to account for demographic structure, suggesting that aging societies may need to prepare for long-term, oscillatory epidemic patterns even for the same disease.

Regarding the critical transmission rate β_c, stability exhibits a dual dependence, that is, if $\mu = \gamma < \mu_c$, the endemic equilibrium remains locally asymptotically stable when $\beta < \beta_c$, yet loses stability and induces oscillations when $\beta > \beta_c$. That is, reducing transmission (β) through vaccination or social distancing does more than lower case numbers; it can prevent the onset of destabilizing oscillations. The target is not just to push $\Re_0$ below 1, but in vulnerable demographics, to push β below β_c to ensure predictability.

The critical duration τ_{1c} highlights that measures which shorten the infectious period—such as test-to-isolate policies and effective treatments—are not just beneficial; they are essential tools for shifting the system from an unstable, oscillatory regime into a stable one, thereby simplifying long-term health planning.

5. Conclusions

The delay models developed here are generic but slightly more complex than the epidemic components of models in [33, 34] due to additional terms accounting for fertility and mortality. Those works, in which $\gamma = \mu = 0$, demonstrate that the stability analysis of the endemic solution depends completely on the basic reproduction number, namely, if $\Re_0$ exceeds some critical value $\Re_c > 1$, the endemic solution is unstable leading to periodic oscillations. Otherwise, if $1 < \Re_0 < \Re_c$, then the endemic solution is stable with damped oscillations. However as mentioned above, the case is different in the present work, in which the models characterize epidemic progression using six parameters β, τ_1, τ_2 γ, μ, and δ, which can be estimabled from the literature. This approach enables applications to research more complex multi-compartment models (involving distinct susceptible/infected groups) and immuno-epidemic models, particularly concerning infection-induced mortality. Investigating the model's applicability to transmissible diseases also presents a promising avenue for future research.

Author contributions

Anastasia Mozokhina: Methodology, validation, investigation, writing–original draft; Ivan Popravka: Software, validation, formal analysis, investigation, writing–original draft; Masoud Saade: Software, validation, formal analysis, investigation, writing–original draft; Vitaly Volpert: Methodology, investigation, writing—original draft. All authors have read and approved the final version of the manuscript for publication.

Use of Generative-AI tools declaration

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

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Conflict of interest

All authors declare that they have no competing interests.

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