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Research article

Integral equation modeling of an SEIQHR epidemic system with non-exponentially distributed disease stages

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Abstract: Many infectious diseases, such as COVID-19 and tuberculosis, are characterized by latent and infectious stages, substantial transmission potential, and a non-negligible risk of severe outcomes, which necessitates integrated control strategies including early detection, quarantine, and hospitalization. Compared with exponential distributions, non-exponential distributions provide a more realistic description of disease transmission processes. In this paper, we incorporate general (non-exponential) distributions to describe stage durations associated with infection, recovery, quarantine, and hospital isolation, including Gamma distributions as a special case. Under suitable assumptions, we formulate an SEIQHR epidemic model as a system of integral equations, incorporating natural mortality, disease-induced mortality, and hospital-related mortality. We establish the existence and uniqueness of solutions. An explicit expression of the control reproduction number is derived and used as a threshold quantity for disease control. If the control reproduction number is less than one, the disease-free equilibrium is globally asymptotically stable, otherwise, the endemic equilibrium is globally asymptotically stable. Under the assumption of Gamma-distributed stage durations, we derive an equivalent system of ODEs. Numerical simulations indicate that the shape parameters of the Gamma distributions can significantly affect the control reproduction number.

Keywords: epidemiological model; SEIQHR model; integral equation; non-exponentially distributed disease stages; control reproduction number
Mathematics Subject Classification: 45G15, 92D25, 92D30

1. Introduction

Infectious diseases have repeatedly caused major public health disasters throughout human history, exerting profound impacts on population structure, social order, and economic development. From large-scale outbreaks to long-term disruptions of social systems and the persistent morbidity and mortality burdens observed today, infectious diseases have remained one of the most critical challenges to global health [1]. For example, the Black Death in fourteenth-century Europe resulted in more than 24 million deaths [2]. Influenza has long persisted worldwide in the form of seasonal epidemics; according to the World Health Organization (WHO), approximately one billion influenza infections occur annually, among which about 3–5 million cases are severe, leading to an estimated 290,000–650,000 deaths from respiratory complications each year [3]. Since its emergence in 2019, COVID-19 has rapidly evolved into a global pandemic, causing hundreds of millions of infections and millions of deaths worldwide [4]. Tuberculosis remains one of the leading causes of death from a single infectious agent, with approximately 10.7 million new cases (including adults and children) and about 1.23 million deaths reported globally in 2024 [5]. Mathematical models have been extensively used to investigate how various disease control measures can be implemented more effectively, playing a fundamental role in the prevention and control of infectious diseases [6–8].

Ordinary differential equation (ODE) models are commonly employed to study the dynamics of infectious diseases and evaluate intervention strategies [9, 10]. These models are typically constructed under the assumption that disease-stage durations follow exponential distributions. The classical SEIR model divides the population into susceptible (S), exposed but not yet infectious (E), infectious (I), and recovered or removed (R) compartments. To incorporate control measures such as quarantine and isolation, additional compartments, including quarantine (Q) and hospitalization or isolation (H), are often introduced. Quarantine of individuals suspected of exposure and isolation of symptomatic individuals are among the most fundamental and historically effective strategies for controlling infectious diseases [11]. These measures have been widely applied in the control of numerous diseases [12, 13]. Therefore, explicitly incorporating Q and H into the model enables the assessment of the impact of intervention measures such as contact tracing, isolation, and hospitalization. A substantial body of literature has investigated the effects of quarantine and isolation on disease transmission using extended SEIR-type models [14–19].

However, many models used to assess the impacts of quarantine and isolation are based on the assumption that disease-stage durations are exponentially distributed. Recent studies have shown that assuming Gamma-distributed waiting times, rather than exponential distributions, provides a more realistic description of disease progression [20]. Moreover, Feng et al. [21] demonstrated that quarantine and isolation models based on exponential assumptions may be inappropriate for diseases with relatively long latent and/or infectious periods, particularly when isolation is imperfect. Consequently, the incorporation of non-exponential stage-duration distributions into epidemic models has attracted increasing attention [22–25]. For example, Feng et al. [26] considered arbitrary distributions for transitions associated with the infected and treated stages, while Feng et al. [27] systematically investigated epidemic models with exponential, Gamma, and general stage-duration distributions. Capistran et al. [28] proposed a COVID-19 model incorporating non-exponentially distributed disease stages. Zang et al. [29] developed two multi-group SEIHR models under different latency assumptions and revealed substantial discrepancies between exponential and non-exponential

models in transmission analysis and control evaluation. These studies indicate that modeling disease stages with non-exponential distributions is both theoretically significant and practically relevant.

In addition, layered models based on different discretization schemes have also been widely studied. Various structure-preserving numerical methods, including finite difference approaches, have been developed to ensure desirable properties such as positivity and stability of solutions [30]. Among them, nonstandard finite difference (NSFD) methods are particularly effective in preserving the qualitative dynamics of the underlying continuous system [31–33]. However, such discrete models typically rely on specific time discretization structures and may not be well suited to capture general (non-exponential) distributions of disease-stage durations. In contrast, integral equation formulations provide a more flexible framework for incorporating arbitrary distributions and better reflect the intrinsic heterogeneity in disease progression.

In this study, we develop an SEIR-type epidemic model with quarantine and isolation, incorporating natural mortality, disease-induced mortality, and hospital-related mortality, and assume that the latent, infectious, quarantine, and hospital isolation stages follow arbitrary distributions. We analyze the model dynamics and demonstrate how the shapes of stage-duration distributions affect transmission thresholds and the effectiveness of control strategies, with particular emphasis on the differences between exponential and non-exponential assumptions.

The remainder of this paper is organized as follows. Section 2 presents the model formulation and derives a system of integral equations with general stage-duration distributions. Section 3 investigates the dynamical properties of the model and derives the control reproduction number. Section 4 provides the corresponding ODE system under the assumption of Gamma-distributed stages, derives explicit expressions for the control reproduction number, and illustrates the effects of key parameters on $\mathcal{R}_c$ through numerical simulations. Finally, Section 5 summarizes the main results and discusses possible directions for future research.

2. Model formulation with general stage distributions

2.1. Assumptions on stage distributions

In this section, we establish an SEIR-type epidemic model to describe the disease transmission dynamics under quarantine and hospitalization interventions, while explicitly incorporating disease-induced mortality. The total population is divided into six compartments: susceptible (S), exposed but not yet infectious (E), infectious (I), quarantined (Q), isolated or hospitalized (H), and recovered (R) individuals. Specifically, the quarantine compartment Q represents individuals who are identified through contact tracing or testing and are quarantined before entering the infectious class. The hospitalization compartment H describes individuals who have developed evident symptoms and receive medical intervention in hospitals. Such individuals are typically strictly isolated, and their contact with the general population is significantly reduced, thereby lowering their contribution to disease transmission. Therefore, the inclusion of Q and H allows the model to explicitly capture the effects of intervention measures such as contact tracing, isolation, and hospitalization, and avoids overestimating disease transmission by distinguishing controlled individuals from actively infectious ones.

Susceptible individuals are recruited at rate Λ and become infected through a time-dependent incidence term $\lambda(t)S(t)$. After infection, individuals may progress through infectious, quarantine,

isolation (hospitalization), and recovery stages. All stage durations are governed by general (non-exponential) waiting-time distributions, except for recruitment and infection incidence. We assume that the model involves three per-capita death rates, and that death is governed by constant rates (i.e., exponentially distributed): the natural mortality rate μ, the disease-induced mortality rate for infectious individuals μ_I, and the disease-induced mortality rate for hospitalized individuals μ_H. Natural death is assumed to act throughout all disease stages, whereas disease-induced death occurs only during the infectious and hospitalized stages, with per-capita rates μ_I and μ_H, respectively. Furthermore, the transition $I \to R$ represents recovery without hospitalization, which may include outpatient treatment or community-based care.

The functions are defined as follows: $P_{XY}(t-u)$ denotes the survival function of the residence time in compartment X, that is, the probability that an individual who is in compartment X at time u remains in compartment X without transitioning to compartment Y up to time t, where $0 < u < t$ and $XY \in \{EI, EQ, QH, IH, IR, HR\}$. $-\dot{P}_{XY}(t-u)$ represents the probability density function of the residence time in compartment X, that is, the probability density that an individual who entered compartment X at time u leaves compartment X for compartment Y at time t.

The force of infection at time t is defined as $\lambda(t) = \beta(I(t) + \theta H(t))$, where β denotes the per-capita effective transmission rate from infectious to susceptible individuals, and $\theta \in [0, 1]$ is the isolation inefficiency factor. In particular, $\theta = 1$ corresponds to completely ineffective isolation, while $\theta = 0$ represents perfectly effective isolation. The parameter θ reflects the effectiveness of isolation measures, with smaller values indicating more effective isolation and a reduced contribution of hospitalized individuals to transmission. The transmission dynamics are illustrated in Figure 1.

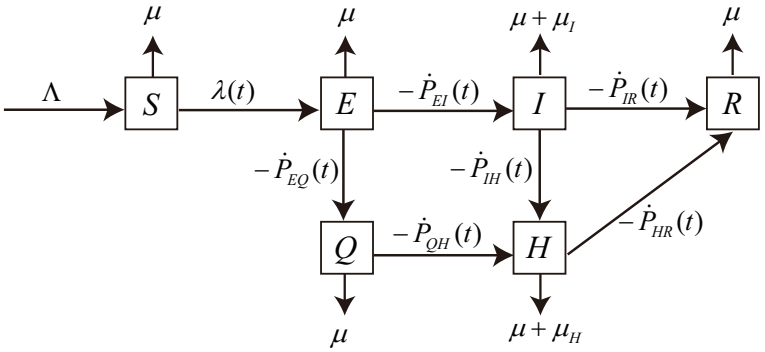

Figure 1. Transfer diagram with general stage distributions.

The probability functions described above admit the following properties:

$$P_i(0) = 1,\ \dot{P}_i(t) \le 0,\ \int_0^\infty P_i(t)dt < \infty,\ i = EI, EQ, QH, IH, IR, HR,$$

where $P_i : [0, \infty) \to [0, 1]$ is nonincreasing and absolutely continuous.

2.2. Derivation of integral equations with general distributions

$X(t)$ denotes the population size of compartment X at time t, where $X \in \{S, E, I, Q, H, R\}$. The total population size is given by

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

The initial conditions at time $t = 0$ are denoted by $X_0 = X(0)$. Assume that

$$I_0 > 0, S_0, E_0, Q_0, H_0, R_0 \geq 0,$$

and $N_0 = N(0) \leq \frac{\Lambda}{\mu}$.

Taking into account the initial number S_0, the total number of susceptible individuals at time t is given by

$$S(t) = \int_0^t [\Lambda - \lambda(t_0)S(t_0)]e^{-\mu(t-t_0)}\, dt_0 + S_0 e^{-\mu t}, \tag{2.1}$$

where the term $[\Lambda - \lambda(t_0)S(t_0)]e^{-\mu(t-t_0)}$ represents the net inflow into the susceptible class at time t_0 who survive until time t. The term $S_0 e^{-\mu t}$ represents the number of individuals remaining in the susceptible stage at time t from the initial susceptible population S_0. Furthermore, a schematic diagram of the infection process is shown in Figure 2.

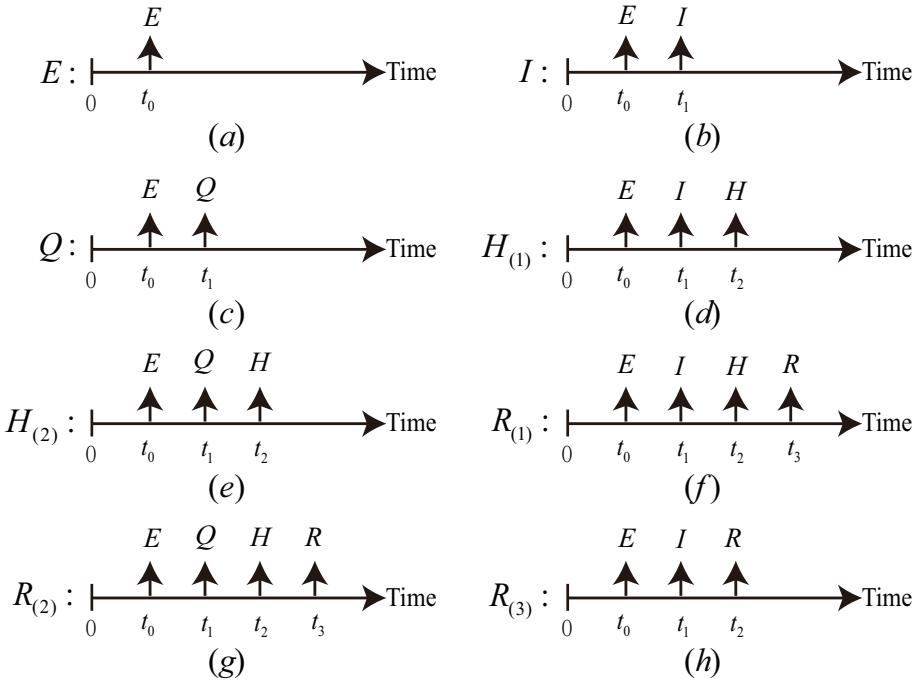

Figure 2. Schematic diagram of the infection process. Panels (a), (b), and (c) depict the E, I, and Q processes, respectively; panels (d) and (e) show the two pathways through which individuals enter compartment H; and panels (f), (g), and (h) represent the three pathways by which individuals enter compartment R. All processes start with the first infection at time t_0.

As shown in Figure 2(a), we assume that an individual becomes exposed at time t_0, where $0 < t_0 < t$. The probabilities that the individual has not yet become infectious, has not yet been quarantined, and is still alive at time t are given by $P_{EI}(t - t_0)$, $P_{EQ}(t - t_0)$, and $e^{-\mu(t-t_0)}$, respectively. Then, the number of individuals in the exposed class (E) at time t who have not died is given by

$$E(t) = \int_0^t \lambda(t_0)S(t_0)P_{EI}(t - t_0)P_{EQ}(t - t_0)e^{-\mu(t-t_0)}dt_0 + E_0 P_{EI}(t)P_{EQ}(t)e^{-\mu t}, \tag{2.2}$$

where the term $E_0P_{EI}(t)P_{EQ}(t)e^{-\mu t}$ represents the number of individuals from the initial exposed population E_0 who remain in the exposed class at time t.

As shown in Figure 2(b), we consider an individual who enters the exposed class at time t_0, and subsequently becomes infectious at time t_1, where $0 < t_0 < t_1 < t$. The probability that an individual who is in the exposed class at time t_0 has not yet been quarantined by time t_1 is given by $P_{EQ}(t_1 - t_0)$. The term $-\dot{P}_{EI}(t_1 - t_0)$ represents the probability density that an individual leaves the exposed class and enters the infectious class at time t_1, given that the individual entered the exposed class at time t_0. The terms $P_{IR}(t - t_1)$ and $P_{IH}(t - t_1)$ denote the probabilities that an individual who entered the infectious class at time t_1 has not yet recovered and has not yet been isolated by time t, respectively. The probability that an infectious individual does not experience disease-induced death from time t_1 to time t is given by $e^{-\mu_I(t-t_1)}$. The probability that an individual survives under natural mortality from time t_0 to time t is given by $e^{-\mu(t-t_0)}$. Then, the number of individuals in the infectious class (I) at time t who have not been isolated, recovered, or removed by either natural or disease-induced death is given by

$$I(t) = \int_0^t \int_0^{t_1} \lambda(t_0)S(t_0)P_{EQ}(t_1 - t_0)[-\dot{P}_{EI}(t_1 - t_0)]P_{IR}(t - t_1)P_{IH}(t - t_1)e^{-\mu_I(t-t_1)}e^{-\mu(t-t_0)}dt_0dt_1 + X_I(t), \tag{2.3}$$

where

$$X_I(t) = \int_0^t E_0P_{EQ}(t_1)[-\dot{P}_{EI}(t_1)]P_{IR}(t - t_1)P_{IH}(t - t_1)e^{-\mu_I(t-t_1)}e^{-\mu t}dt_1 + I_0P_{IR}(t)P_{IH}(t)e^{-\mu t}e^{-\mu_I t}.$$

Here, the term $X_I(t)$ represents the number of individuals originating from the initial exposed population E_0 and initial infectious population I_0 who remain in the infectious class at time t.

As shown in Figure 2(c), we consider an individual who becomes infected and enters the exposed class at time t_0, and is subsequently quarantined at time t_1, where $0 < t_0 < t_1 < t$. The probability that an individual who is in the exposed class at time t_0 has not yet become infectious by time t_1 is given by $P_{EI}(t_1 - t_0)$. The term $-\dot{P}_{EQ}(t_1 - t_0)$ represents the probability density that an individual leaves the exposed class and enters the quarantine class at time t_1, given that the individual entered the exposed class at time t_0. The term $P_{QH}(t - t_1)$ denotes the probability that an individual who entered the quarantined class at time t_1 has not yet been isolated by time t. The probability that an individual survives natural mortality from time t_0 to time t is given by $e^{-\mu(t-t_0)}$. Then, the number of individuals in the quarantine class (Q) at time t who have not been isolated or died is given by

$$Q(t) = \int_0^t \int_0^{t_1} \lambda(t_0)S(t_0)P_{EI}(t_1 - t_0)[-\dot{P}_{EQ}(t_1 - t_0)]P_{QH}(t - t_1)e^{-\mu(t-t_0)}dt_0dt_1 + X_Q(t), \tag{2.4}$$

where

$$X_Q(t) = \int_0^t E_0P_{EI}(t_1)[-\dot{P}_{EQ}(t_1)]P_{QH}(t - t_1)e^{-\mu t}dt_1 + Q_0P_{QH}(t)e^{-\mu t}.$$

Here, the term $X_Q(t)$ represents the number of individuals originating from the initial exposed population E_0 and the initial quarantined population Q_0 who remain in the quarantine class at time t.

As shown in Figure 2(d), we consider an individual who enters the exposed class at time t_0, subsequently becomes infectious at time t_1, and is isolated at time t_2, where $0 < t_0 < t_1 < t_2 < t$.

The probability that an individual who is in the exposed class at time t_0 has not yet been quarantined by time t_1 is given by $P_{EQ}(t_1 - t_0)$. The term $-\dot{P}_{EI}(t_1 - t_0)$ represents the probability density that an individual leaves the exposed class and enters the infectious class at time t_1, given that the individual entered the exposed class at time t_0. The probability that an infectious individual does not experience disease-induced death during the infectious stage from time t_1 to time t_2 is given by $e^{-\mu_I(t_2-t_1)}$. The term $P_{IR}(t_2 - t_1)$ denotes the probability that an individual who entered the infectious class at time t_1 has not yet recovered by time t_2. The term $-\dot{P}_{IH}(t_2 - t_1)$ represents the probability density that an individual leaves the infectious class and enters the isolated class at time t_2, given that the individual entered the infectious class at time t_1. The term $P_{HR}(t - t_2)$ denotes the probability that an individual who entered the isolated class at time t_2 has not yet recovered by time t. The probability that a hospitalized individual does not experience disease-induced death from time t_2 to time t is given by $e^{-\mu_H(t-t_2)}$. The probability that an individual survives natural death from time t_0 to time t is given by $e^{-\mu(t-t_0)}$. Then, under this pathway and ignoring initial conditions, the number of individuals in the isolated class (H) at time t who have not yet recovered or been removed by either natural or disease-induced death is given by

$$H_{(1)}(t) = \int_0^t \int_0^{t_2} \int_0^{t_1} \lambda(t_0)S(t_0)P_{EQ}(t_1 - t_0)[-\dot{P}_{EI}(t_1 - t_0)]e^{-\mu_I(t_2-t_1)}P_{IR}(t_2 - t_1)[-\dot{P}_{IH}(t_2 - t_1)] \times P_{HR}(t - t_2)e^{-\mu_H(t-t_2)}e^{-\mu(t-t_0)}dt_0dt_1dt_2.$$

As shown in Figure 2(e), we consider an individual who enters the exposed class at time t_0, subsequently becomes quarantined at time t_1, and is isolated at time t_2, where $0 < t_0 < t_1 < t_2 < t$. The probability that an individual who is in the exposed class at time t_0 has not yet entered the infectious class by time t_1 is given by $P_{EI}(t_1 - t_0)$. The term $-\dot{P}_{EQ}(t_1 - t_0)$ represents the probability density that an individual leaves the exposed class and enters the quarantined class at time t_1, given that they entered the exposed class at time t_0. The term $-\dot{P}_{QH}(t_2 - t_1)$ represents the probability density that an individual leaves the quarantined class and enters the isolated class at time t_2, given that the individual entered the quarantined class at time t_1. The term $P_{HR}(t - t_2)$ denotes the probability that an individual who entered the isolated class at time t_2 has not yet recovered by time t. The probability that a hospitalized individual does not experience disease-induced death from time t_2 to time t is given by $e^{-\mu_H(t-t_2)}$. The probability that an individual survives natural death from time t_0 to time t is given by $e^{-\mu(t-t_0)}$. Then, under this pathway and ignoring initial conditions, the number of individuals in the isolated class (H) at time t who have not yet recovered or been removed by either natural or disease-induced death is given by

$$H_{(2)}(t) = \int_0^t \int_0^{t_2} \int_0^{t_1} \lambda(t_0)S(t_0)P_{EI}(t_1 - t_0)[-\dot{P}_{EQ}(t_1 - t_0)][-\dot{P}_{QH}(t_2 - t_1)] \times P_{HR}(t - t_2)e^{-\mu_H(t-t_2)}e^{-\mu(t-t_0)}dt_0dt_1dt_2.$$

From the above pathway analysis, the number of individuals in the isolated class (H) at time t who have neither recovered nor been removed by natural or disease-induced death is given by

$$H(t) = H_{(1)}(t) + H_{(2)}(t) + X_H(t), \tag{2.5}$$

where

$$X_H(t) = \int_0^t \int_0^{t_2} E_0P_{EQ}(t_1)[-\dot{P}_{EI}(t_1)]e^{-\mu_I(t_2-t_1)}P_{IR}(t_2 - t_1)[-\dot{P}_{IH}(t_2 - t_1)]P_{HR}(t - t_2)e^{-\mu_H(t-t_2)}e^{-\mu t}dt_1dt_2$$

$$+ \int_0^t \int_0^{t_2} E_0 P_{EI}(t_1)[-\dot{P}_{EQ}(t_1)][-\dot{P}_{QH}(t_2 - t_1)]e^{-\mu_H(t-t_2)} P_{HR}(t - t_2)e^{-\mu t} dt_1 dt_2$$
$$+ \int_0^t I_0 e^{-\mu_I t_2} P_{IR}(t_2)[-\dot{P}_{IH}(t_2)]e^{-\mu_H(t-t_2)} P_{HR}(t - t_2)e^{-\mu t} dt_2$$
$$+ \int_0^t Q_0[-\dot{P}_{QH}(t_2)]e^{-\mu_H(t-t_2)} P_{HR}(t - t_2)e^{-\mu t} dt_2$$
$$+ H_0 e^{-\mu_H t} P_{HR}(t)e^{-\mu t}.$$

Here, the term $X_H(t)$ represents the number of individuals originating from the initial exposed population E_0, the initial infectious population I_0, the initial quarantined population Q_0, and the initial isolated population H_0 who remain in the isolated class at time t.

As shown in Figure 2(f), we consider an individual who enters the exposed class at time t_0, subsequently becomes infectious at time t_1, is isolated at time t_2, and recovers at time t_3, where $0 < t_0 < t_1 < t_2 < t_3 < t$. The probability that an individual who is in the exposed class at time t_0 has not yet been quarantined by time t_1 is given by $P_{EQ}(t_1 - t_0)$. The term $-\dot{P}_{EI}(t_1 - t_0)$ represents the probability density that individuals leave the exposed class and enter the infectious class at time t_1, given that they entered the exposed class at time t_0. The probability that an infectious individual does not experience disease-induced death during the infectious stage from time t_1 to time t_2 is given by $e^{-\mu_I(t_2-t_1)}$. The term $P_{IR}(t_2 - t_1)$ denotes the probability that an individual who entered the infectious class at time t_1 has not yet recovered by time t_2. The term $-\dot{P}_{IH}(t_2 - t_1)$ represents the probability density that individuals leave the infectious class and enter the isolated class at time t_2. The probability that a hospitalized individual does not experience disease-induced death during the isolated stage from time t_2 to time t_3 is given by $e^{-\mu_H(t_3-t_2)}$. The term $-\dot{P}_{HR}(t_3 - t_2)$ represents the probability density that individuals leave the isolated class and enter the recovered class at time t_3, given that they entered the isolated class at time t_2. The probability that an individual survives natural death from time t_0 to time t is given by $e^{-\mu(t-t_0)}$. Then, under this pathway and ignoring initial conditions, the number of individuals in the recovered class (R) at time t who have not yet died is given by

$$R_{(1)}(t) = \int_0^t \int_0^{t_3} \int_0^{t_2} \int_0^{t_1} \lambda(t_0)S(t_0)P_{EQ}(t_1 - t_0)[-\dot{P}_{EI}(t_1 - t_0)]e^{-\mu_I(t_2-t_1)} P_{IR}(t_2 - t_1)$$
$$\times [-\dot{P}_{IH}(t_2 - t_1)]e^{-\mu_H(t_3-t_2)}[-\dot{P}_{HR}(t_3 - t_2)]e^{-\mu(t-t_0)} dt_0 dt_1 dt_2 dt_3.$$

As shown in Figure 2(g), we consider an individual who enters the exposed class at time t_0, subsequently becomes quarantined at time t_1, is isolated at time t_2, and recovers at time t_3, where $0 < t_0 < t_1 < t_2 < t_3 < t$. Under this pathway and ignoring initial conditions, the number of individuals in the recovered class (R) at time t who have not yet died is given by

$$R_{(2)}(t) = \int_0^t \int_0^{t_3} \int_0^{t_2} \int_0^{t_1} \lambda(t_0)S(t_0)P_{EI}(t_1 - t_0)[-\dot{P}_{EQ}(t_1 - t_0)][-\dot{P}_{QH}(t_2 - t_1)]$$
$$\times e^{-\mu_H(t_3-t_2)}[-\dot{P}_{HR}(t_3 - t_2)]e^{-\mu(t-t_0)}\, dt_0 dt_1 dt_2 dt_3.$$

As shown in Figure 2(h), we consider an individual who enters the exposed class at time t_0, becomes infectious at time t_1, and recovers at time t_2, where $0 < t_0 < t_1 < t_2 < t$. By an analogous argument, under this pathway and ignoring initial conditions, the number of individuals in the recovered class (R)

at time t who have not yet died is given by

$$R_{(3)}(t) = \int_0^t \int_0^{t_2} \int_0^{t_1} \lambda(t_0) S(t_0) P_{EQ}(t_1 - t_0)[-\dot{P}_{EI}(t_1 - t_0)] e^{-\mu_I(t_2 - t_1)} P_{IH}(t_2 - t_1)[-\dot{P}_{IR}(t_2 - t_1)] \times e^{-\mu(t-t_0)}\, dt_0 dt_1 dt_2.$$

From the above pathway analysis, the number of individuals in the recovered class (R) at time t who have not yet died is given by

$$R(t) = R_{(1)}(t) + R_{(2)}(t) + R_{(3)}(t) + X_R(t), \tag{2.6}$$

where

$$\begin{aligned} X_R(t) = & \int_0^t \int_0^{t_3} \int_0^{t_2} E_0 P_{EQ}(t_1)[-\dot{P}_{EI}(t_1)] e^{-\mu_I(t_2 - t_1)} P_{IR}(t_2 - t_1)[-\dot{P}_{IH}(t_2 - t_1)] e^{-\mu_H(t_3 - t_2)}[-\dot{P}_{HR}(t_3 - t_2)] \\ & \times e^{-\mu t} dt_1 dt_2 dt_3 \\ & + \int_0^t \int_0^{t_3} \int_0^{t_2} E_0 P_{EI}(t_1)[-\dot{P}_{EQ}(t_1)][-\dot{P}_{QH}(t_2 - t_1)] e^{-\mu_H(t_3 - t_2)}[-\dot{P}_{HR}(t_3 - t_2)] e^{-\mu t} dt_1 dt_2 dt_3 \\ & + \int_0^t \int_0^{t_3} E_0 P_{EQ}(t_2)[-\dot{P}_{EI}(t_2)] e^{-\mu_I(t_3 - t_2)} P_{IH}(t_3 - t_2)[-\dot{P}_{IR}(t_3 - t_2)] e^{-\mu t} dt_2 dt_3 \\ & + \int_0^t I_0 e^{-\mu_I t_3} P_{IH}(t_3)[-\dot{P}_{IR}(t_3)] e^{-\mu t} dt_3 \\ & + \int_0^t \int_0^{t_3} I_0 e^{-\mu_I t_2} P_{IR}(t_2)[-\dot{P}_{IH}(t_2)] e^{-\mu_H(t_3 - t_2)}[-\dot{P}_{HR}(t_3 - t_2)] e^{-\mu t} dt_2 dt_3 \\ & + \int_0^t \int_0^{t_3} Q_0[-\dot{P}_{QH}(t_2)] e^{-\mu_H(t_3 - t_2)}[-\dot{P}_{HR}(t_3 - t_2)] e^{-\mu t} dt_2 dt_3 \\ & + \int_0^t H_0 e^{-\mu_H t_3}[-\dot{P}_{HR}(t_3)] e^{-\mu t} dt_3 + R_0 e^{-\mu t}. \end{aligned}$$

The term $X_R(t)$ represents the number of individuals originating from the initial exposed population E_0, initial infectious population I_0, initial quarantined population Q_0, initial isolated population H_0, and initial recovered population R_0 who remain in the recovered class at time t.

Since survival probabilities are bounded and the corresponding transition densities have finite integrals, all terms generated by the initial population are bounded and multiplied by an exponentially decaying factor. Consequently, these initial contributions, including $S_0 e^{-\mu t}$, $E_0 P_{EI}(t) P_{EQ}(t) e^{-\mu t}$, $X_I(t)$, $X_Q(t)$, $X_H(t)$, and $X_R(t)$, vanish as $t \to \infty$. Therefore, these initial-value terms do not affect the asymptotic dynamics, and Eqs (2.1)–(2.6) reduce to the following integral system.

$$
\begin{aligned}
S(t) &= \int_0^t [\Lambda - \lambda(t_0)S(t_0)]e^{-\mu(t-t_0)}\,dt_0,\\
E(t) &= \int_0^t \lambda(t_0)S(t_0)P_{EI}(t-t_0)P_{EQ}(t-t_0)e^{-\mu(t-t_0)}dt_0,\\
I(t) &= \int_0^t\int_0^{t_1} \lambda(t_0)S(t_0)P_{EQ}(t_1-t_0)[-\dot{P}_{EI}(t_1-t_0)]P_{IR}(t-t_1)P_{IH}(t-t_1)e^{-\mu_I(t-t_1)}e^{-\mu(t-t_0)}dt_0dt_1,\\
Q(t) &= \int_0^t\int_0^{t_1} \lambda(t_0)S(t_0)P_{EI}(t_1-t_0)[-\dot{P}_{EQ}(t_1-t_0)]P_{QH}(t-t_1)e^{-\mu(t-t_0)}dt_0dt_1,\\
H(t) &= \int_0^t\int_0^{t_2}\int_0^{t_1} \lambda(t_0)S(t_0)P_{EQ}(t_1-t_0)[-\dot{P}_{EI}(t_1-t_0)]e^{-\mu_I(t_2-t_1)}P_{IR}(t_2-t_1)[-\dot{P}_{IH}(t_2-t_1)]\\
&\quad\times P_{HR}(t-t_2)e^{-\mu_H(t-t_2)}e^{-\mu(t-t_0)}dt_0dt_1dt_2\\
&\quad+\int_0^t\int_0^{t_2}\int_0^{t_1} \lambda(t_0)S(t_0)P_{EI}(t_1-t_0)[-\dot{P}_{EQ}(t_1-t_0)][-\dot{P}_{QH}(t_2-t_1)]\\
&\quad\times P_{HR}(t-t_2)e^{-\mu_H(t-t_2)}e^{-\mu(t-t_0)}dt_0dt_1dt_2,\\
R(t) &= \int_0^t\int_0^{t_3}\int_0^{t_2}\int_0^{t_1} \lambda(t_0)S(t_0)P_{EQ}(t_1-t_0)[-\dot{P}_{EI}(t_1-t_0)]e^{-\mu_I(t_2-t_1)}P_{IR}(t_2-t_1)\\
&\quad\times[-\dot{P}_{IH}(t_2-t_1)]e^{-\mu_H(t_3-t_2)}[-\dot{P}_{HR}(t_3-t_2)]e^{-\mu(t-t_0)}dt_0dt_1dt_2dt_3\\
&\quad+\int_0^t\int_0^{t_3}\int_0^{t_2}\int_0^{t_1} \lambda(t_0)S(t_0)P_{EI}(t_1-t_0)[-\dot{P}_{EQ}(t_1-t_0)][-\dot{P}_{QH}(t_2-t_1)]\\
&\quad\times e^{-\mu_H(t_3-t_2)}[-\dot{P}_{HR}(t_3-t_2)]e^{-\mu(t-t_0)}\,dt_0dt_1dt_2dt_3\\
&\quad+\int_0^t\int_0^{t_2}\int_0^{t_1} \lambda(t_0)S(t_0)P_{EQ}(t_1-t_0)[-\dot{P}_{EI}(t_1-t_0)]e^{-\mu_I(t_2-t_1)}P_{IH}(t_2-t_1)[-\dot{P}_{IR}(t_2-t_1)]\\
&\quad\times e^{-\mu(t-t_0)}\,dt_0dt_1dt_2.
\end{aligned}
\tag{2.7}
$$

3. Model analysis

3.1. The basic reproduction number

We assume that the per-capita incidence rate is given by

$$\lambda(t) = \beta(I(t) + \theta H(t)), \tag{3.1}$$

where β denotes the effective transmission rate from infectious to susceptible individuals, and $\theta \in [0, 1]$ measures the residual transmissibility of isolated individuals.

To simplify the notation in the later sections, we introduce the following quantities:

$$
\begin{aligned}
T_{EI} &= \int_0^\infty P_{EQ}(s)[-\dot{P}_{EI}(s)]e^{-\mu s}ds, \qquad T_{EQ} = \int_0^\infty P_{EI}(s)[-\dot{P}_{EQ}(s)]e^{-\mu s}ds,\\
T_{IH} &= \int_0^\infty P_{IR}(s)[-\dot{P}_{IH}(s)]e^{-\mu_I s}e^{-\mu s}ds, \quad T_{QH} = \int_0^\infty [-\dot{P}_{QH}(s)]e^{-\mu s}ds,\\
D_I &= \int_0^\infty P_{IR}(s)P_{IH}(s)e^{-\mu_I s}e^{-\mu s}ds, \qquad D_H = \int_0^\infty P_{HR}(s)e^{-\mu_H s}e^{-\mu s}ds.
\end{aligned}
\tag{3.2}
$$

The quantities T_{XY} denote the effective probabilities that an individual survives and transitions from compartment X to compartment Y, accounting for both natural death and stage-specific disease-induced mortality, where $XY \in \{EI, EQ, IH, QH\}$. The quantities D_Z represent the effective mean residence times in compartment Z in the presence of natural death and disease-induced mortality, where $Z \in \{I, H\}$.

Let $A(\tau)$ denote the probability that an individual remains infectious at infection age τ, weighted by the likelihood of survival up to that age. From system (2.7), we can obtain the following equation:

$$A(\tau) = a_I(\tau) + \theta(a_{IH}(\tau) + a_{QH}(\tau)), \tag{3.3}$$

with

$$\begin{aligned}
a_I(\tau) &= \int_0^\tau P_{EQ}(\tau - u)[-\dot{P}_{EI}(\tau - u)]P_{IR}(u)P_{IH}(u)e^{-\mu_I u}e^{-\mu\tau}du,\\
a_{IH}(\tau) &= \int_0^\tau \int_0^{x_1} P_{EQ}(\tau - x_1)[-\dot{P}_{EI}(\tau - x_1)]e^{-\mu_I(x_1-u)}P_{IR}(x_1 - u)[-\dot{P}_{IH}(x_1 - u)]P_{HR}(u)e^{-\mu_H u}e^{-\mu\tau}dudx_1,\\
a_{QH}(\tau) &= \int_0^\tau \int_0^{x_1} P_{EI}(\tau - x_1)[-\dot{P}_{EQ}(\tau - x_1)][-\dot{P}_{QH}(x_1 - u)]P_{HR}(u)e^{-\mu_H u}e^{-\mu\tau}dudx_1.
\end{aligned} \tag{3.4}$$

A simple calculation shows that

$$\begin{aligned}
\int_0^\infty a_I(\tau)d\tau &= \int_0^\infty P_{EQ}(s)[-\dot{P}_{EI}(s)]e^{-\mu s}ds \int_0^\infty P_{IR}(u)P_{IH}(u)e^{-\mu_I u}e^{-\mu u}du = T_{EI}D_I,\\
\int_0^\infty a_{IH}(\tau)d\tau &= \int_0^\infty P_{EQ}(s_1)[-\dot{P}_{EI}(s_1)]e^{-\mu s_1}ds_1 \int_0^\infty P_{IR}(s_2)[-\dot{P}_{IH}(s_2)]e^{-\mu_I s_2}e^{-\mu s_2}ds_2\\
&\quad \times \int_0^\infty P_{HR}(u)e^{-\mu_H u}e^{-\mu u}du\\
&= T_{EI}T_{IH}D_H,\\
\int_0^\infty a_{QH}(\tau)d\tau &= \int_0^\infty P_{EI}(s_1)[-\dot{P}_{EQ}(s_1)]e^{-\mu s_1}ds_1 \int_0^\infty [-\dot{P}_{QH}(s_2)]e^{-\mu s_2}ds_2 \int_0^\infty P_{HR}(u)e^{-\mu_H u}e^{-\mu u}du\\
&= T_{EQ}T_{QH}D_H.
\end{aligned} \tag{3.5}$$

Therefore, the integral $\int_0^\infty A(\tau)\,d\tau$ quantifies the cumulative infectious potential of a typical individual over the entire course of infection. Accordingly, the control reproduction number $\mathcal{R}_c$ can be expressed as

$$\mathcal{R}_c = \beta\frac{\Lambda}{\mu}\int_0^\infty A(\tau)d\tau = \beta\frac{\Lambda}{\mu}[T_{EI}D_I + \theta(T_{EI}T_{IH}D_H + T_{EQ}T_{QH}D_H)]. \tag{3.6}$$

In the absence of control, that is $P_{EQ} = P_{IH} = 1$, we can obtain the basic reproduction number

$$\mathcal{R}_0 = \beta\frac{\Lambda}{\mu}\bar{T}_{EI}\bar{D}_I, \tag{3.7}$$

where $\bar{T}_{EI} = \int_0^\infty [-\dot{P}_{EI}(s)]e^{-\mu s}ds$, $\bar{D}_I = \int_0^\infty P_{IR}(s)e^{-\mu_I s}e^{-\mu s}ds$.

3.2. Well-posedness

Rearranging (2.1), we obtain

$$S(t) = \frac{\Lambda}{\mu}(1 - e^{-\mu t}) + S_0 e^{-\mu t} - \int_0^t \lambda(t_0) S(t_0) e^{-\mu(t-t_0)} dt_0.$$

Moreover, from (2.3) and (2.5), we have

$$\lambda(t) = \beta(X_I(t) + \theta X_H(t)) + \beta \int_0^t \lambda(t_0) S(t_0) A(t - t_0) dt_0.$$

Define

$$f(t) := \frac{\Lambda}{\mu}(1 - e^{-\mu t}) + S_0 e^{-\mu t}, \qquad g(t) := \beta(X_I(t) + \theta X_H(t)).$$

From the definitions of $f(t)$ and $g(t)$, it follows that $f, g \in C([0, \infty))$. Noting that, in (2.1)–(2.6), all compartmental variables can be represented as functionals of $S(t)$ and $\lambda(t)$, the high-dimensional integral system is effectively closed in terms of these two quantities. Therefore, we obtain the following reduced system:

$$\begin{cases} S(t) = f(t) - \displaystyle\int_0^t \lambda(t_0) S(t_0) e^{-\mu(t-t_0)} dt_0, \\ \lambda(t) = g(t) + \beta \displaystyle\int_0^t \lambda(t_0) S(t_0) A(t - t_0) dt_0. \end{cases} \tag{3.8}$$

Theorem 3.1 (Local existence and uniqueness)**.** *Assume that*

$$A(t) \geq 0 \, \textit{for} \, t \geq 0, \qquad A \in L^1(0, \infty).$$

Then, system (3.8) *admits a unique continuous solution*

$$(S, \lambda) \in C([0, T]) \times C([0, T])$$

on some interval $[0, T]$ *with* $T > 0$.

Proof. Let $T_0 > 0$ be fixed. Define

$$X_T := C([0, T]) \times C([0, T]), \qquad 0 < T \leq T_0,$$

equipped with the norm

$$\|(S, \lambda)\|_T := \|S\|_{\infty,T} + \|\lambda\|_{\infty,T},$$

where

$$\|S\|_{\infty,T} := \max_{t\in[0,T]} |S(t)|, \qquad \|\lambda\|_{\infty,T} := \max_{t\in[0,T]} |\lambda(t)|.$$

Then, X_T is a Banach space.

Define the operator $F = (F_1, F_2) : X_T \to X_T$ by

$$\begin{aligned} F_1(S, \lambda)(t) &:= f(t) - \int_0^t \lambda(t_0) S(t_0) e^{-\mu(t-t_0)} \, dt_0, \\ F_2(S, \lambda)(t) &:= g(t) + \beta \int_0^t \lambda(t_0) S(t_0) A(t - t_0) \, dt_0, \end{aligned} \qquad t \in [0, T].$$

Since $f, g \in C([0,\infty))$, they are bounded on $[0, T_0]$. Let

$$M_f := \max_{t\in[0,T_0]} |f(t)|, \qquad M_g := \max_{t\in[0,T_0]} |g(t)|.$$

Choose $R > 0$ such that $R > 2(M_f + M_g)$. For $0 < T \le T_0$, define

$$B_R := \{(S,\lambda) \in X_T : \|(S,\lambda)\|_T \le R\}.$$

Then, B_R is a closed subset of X_T.

Now let $(S,\lambda) \in B_R$. Since $\|S\|_{\infty,T} \le R$, $\|\lambda\|_{\infty,T} \le R$, we have, for every $t \in [0,T]$,

$$|F_1(S,\lambda)(t)| \le |f(t)| + \int_0^t |\lambda(t_0)|\,|S(t_0)|\,e^{-\mu(t-t_0)}\,dt_0 \le M_f + \int_0^t R^2\,dt_0 \le M_f + TR^2,$$

and

$$\begin{aligned}|F_2(S,\lambda)(t)| &\le |g(t)| + \beta\int_0^t |\lambda(t_0)|\,|S(t_0)|\,A(t-t_0)\,dt_0\\ &\le M_g + \beta R^2\int_0^t A(t-t_0)\,dt_0 \le M_g + \beta R^2\alpha(T),\end{aligned}$$

where

$$\alpha(T) := \int_0^T A(\tau)\,d\tau.$$

Since $A \in L^1(0,\infty)$, we have $\alpha(T) \to 0$ as $T \to 0^+$.

Hence, choosing $T \in (0, T_0]$ sufficiently small such that

$$TR^2 + \beta R^2\alpha(T) \le R - (M_f + M_g),$$

we obtain

$$\|F(S,\lambda)\|_T \le M_f + M_g + TR^2 + \beta R^2\alpha(T) \le R.$$

Therefore, $F(B_R) \subset B_R$.

Next, let $(S_1,\lambda_1), (S_2,\lambda_2) \in B_R$. For every $t \in [0,T]$, we have

$$\begin{aligned}&|F_1(S_1,\lambda_1)(t) - F_1(S_2,\lambda_2)(t)|\\ &\le \int_0^t |\lambda_1(t_0)S_1(t_0) - \lambda_2(t_0)S_2(t_0)|\,dt_0\\ &\le \int_0^t \Big(|\lambda_1(t_0)|\,|S_1(t_0) - S_2(t_0)| + |S_2(t_0)|\,|\lambda_1(t_0) - \lambda_2(t_0)|\Big)\,dt_0\\ &\le RT\|(S_1 - S_2, \lambda_1 - \lambda_2)\|_T.\end{aligned}$$

Similarly,

$$\begin{aligned}&|F_2(S_1,\lambda_1)(t) - F_2(S_2,\lambda_2)(t)|\\ &\le \beta\int_0^t |\lambda_1(t_0)S_1(t_0) - \lambda_2(t_0)S_2(t_0)|A(t-t_0)\,dt_0\\ &\le \beta R\alpha(T)\|(S_1 - S_2, \lambda_1 - \lambda_2)\|_T.\end{aligned}$$

Therefore,

$$\|F(S_1, \lambda_1) - F(S_2, \lambda_2)\|_T \leq (TR + \beta R\alpha(T)) \|(S_1 - S_2, \lambda_1 - \lambda_2)\|_T.$$

Again, by choosing $T \in (0, T_0]$ smaller if necessary, we may ensure that

$$TR + \beta R\alpha(T) < 1.$$

Thus, F is a contraction on B_R.

By the Banach fixed-point theorem, F admits a unique fixed point in B_R. Hence, system (3.8) has a unique continuous solution

$$(S, \lambda) \in C([0, T]) \times C([0, T])$$

on some interval $[0, T]$. □

Proposition 3.1 (Local positivity)**.** *Let* (S, λ) *be the local solution of* (3.8) *on* $[0, T]$*. Then,*

$$S(t) \geq 0, \qquad \lambda(t) \geq 0, \qquad t \in [0, T].$$

Moreover,

$$E(t),\ I(t),\ Q(t),\ H(t),\ R(t) \geq 0, \qquad t \in [0, T].$$

Proof. Since $S(t)$ satisfies

$$\dot{S}(t) = \Lambda - \lambda(t)S(t) - \mu S(t), \qquad S(0) = S_0 \geq 0,$$

the variation-of-constants formula gives

$$S(t) = S_0 e^{-\int_0^t (\lambda(\xi)+\mu)\, d\xi} + \int_0^t \Lambda e^{-\int_s^t (\lambda(\xi)+\mu)\, d\xi}\, ds \geq 0.$$

Next, we prove that $\lambda(t) \geq 0$ on $[0, T]$. Suppose, by contradiction, that there exists $t_1 \in [0, T]$ such that

$$\lambda(t_1) < 0.$$

Define

$$\bar{t} := \inf\{t \in [0, t_1] : \lambda(t) < 0\}.$$

Then, $\bar{t}$ is the first time at which λ enters the negative region. By continuity of λ and the strict positivity of λ_0,

$$\lambda(\bar{t}) = 0, \qquad \lambda(s) \geq 0, \quad s \in [0, \bar{t}].$$

Since λ takes negative values after $\bar{t}$, for any sufficiently small $\varepsilon > 0$ such that

$$\lambda(s) < 0, s \in (\bar{t}, \bar{t} + \varepsilon]$$

there exists

$$t_\varepsilon \in [\bar{t}, \bar{t} + \varepsilon]$$

such that

$$\lambda(t_\varepsilon) = \min_{s \in [\bar{t}, \bar{t}+\varepsilon]} \lambda(s) < 0.$$

Evaluating the second equation of (3.8) at $t = t_\varepsilon$, we obtain

$$\lambda(t_\varepsilon) = g(t_\varepsilon) + \beta \int_0^{\bar{t}} \lambda(s)S(s)A(t_\varepsilon - s)\,ds + \beta \int_{\bar{t}}^{t_\varepsilon} \lambda(s)S(s)A(t_\varepsilon - s)\,ds.$$

Since $g(t_\varepsilon) \geq 0$, $S(s) \geq 0$, $A(t_\varepsilon - s) \geq 0$, and $\lambda(s) \geq 0$ for all $s \in [0, \bar{t}]$, the first two terms on the right-hand side are nonnegative. Hence,

$$\lambda(t_\varepsilon) \geq \beta \int_{\bar{t}}^{t_\varepsilon} \lambda(s)S(s)A(t_\varepsilon - s)\,ds.$$

By the choice of t_ε, we have

$$\lambda(s) \geq \lambda(t_\varepsilon), \qquad s \in [\bar{t}, t_\varepsilon].$$

Therefore,

$$\lambda(t_\varepsilon) \geq \beta\,\lambda(t_\varepsilon) \int_{\bar{t}}^{t_\varepsilon} S(s)A(t_\varepsilon - s)\,ds.$$

Since $\lambda(t_\varepsilon) < 0$, dividing by $\lambda(t_\varepsilon)$ reverses the inequality and yields

$$1 \leq \beta \int_{\bar{t}}^{t_\varepsilon} S(s)A(t_\varepsilon - s)\,ds.$$

Since there exists $K > 0$ such that $S(s) \leq K$ for all $s \in [\bar{t}, t_\varepsilon]$, it follows that

$$1 \leq \beta \int_{\bar{t}}^{t_\varepsilon} S(s)A(t_\varepsilon - s)\,ds \leq \beta K \int_{\bar{t}}^{t_\varepsilon} A(t_\varepsilon - s)\,ds.$$

By the change of variable $\tau = t_\varepsilon - s$, we obtain

$$1 \leq \beta K \int_0^{t_\varepsilon - \bar{t}} A(\tau)\,d\tau \leq \beta K \int_0^{\varepsilon} A(\tau)\,d\tau.$$

Since $A \in L^1(0, \infty)$, we have

$$\int_0^{\varepsilon} A(\tau)\,d\tau \to 0 \qquad \text{as } \varepsilon \to 0^+.$$

Choosing $\varepsilon > 0$ sufficiently small, we obtain

$$\beta K \int_0^{\varepsilon} A(\tau)\,d\tau < 1,$$

which contradicts the previous inequality. Therefore,

$$\lambda(t) \geq 0, \qquad t \in [0, T].$$

Since $S(t) \geq 0$ and $\lambda(t) \geq 0$, the integral representations (2.2)–(2.6) consist only of integrals of nonnegative functions. Hence,

$$E(t),\ I(t),\ Q(t),\ H(t),\ R(t) \geq 0, \qquad t \in [0, T].$$

□

Proposition 3.2 (Local boundedness)**.** *Let* (S, λ) *be a nonnegative local solution of* (3.8) *on* $[0, T]$. *Then,* $S(t)$ *and* $\lambda(t)$ *are bounded on* $[0, T]$. *Moreover,* $E(t)$, $I(t)$, $Q(t)$, $H(t)$, *and* $R(t)$ *are also bounded on* $[0, T]$.

Proof. The total population satisfies

$$\dot{N}(t) \leq \Lambda - \mu N(t).$$

By the comparison principle, we obtain

$$N(t) \leq N_0 e^{-\mu t} + \frac{\Lambda}{\mu}(1 - e^{-\mu t}), \qquad t \geq 0.$$

By $N_0 \leq \frac{\Lambda}{\mu}$, we have

$$N(t) \leq \frac{\Lambda}{\mu}, \qquad t \geq 0.$$

Since all compartmental variables are nonnegative, it follows that

$$0 \leq S(t),\ E(t),\ I(t),\ Q(t),\ H(t),\ R(t) \leq N(t) \leq \frac{\Lambda}{\mu}, \qquad t \in [0, T].$$

Hence, all compartmental variables are bounded on $[0, T]$.

Next, by (3.1), we have

$$\lambda(t) = \beta(I(t) + \theta H(t)).$$

Since $I(t)$ and $H(t)$ are bounded on $[0, T]$, it follows that

$$\lambda(t) \leq \beta \frac{\Lambda}{\mu}(1 + \theta) =: M_\lambda, \qquad t \in [0, T].$$

Therefore, $\lambda(t)$ is also bounded on $[0, T]$. □

Theorem 3.2 (Global existence and uniqueness)**.** *System* (3.8) *admits a unique global nonnegative continuous solution on* $[0, \infty)$.

Proof. By Theorem 3.1, there exists a unique local continuous solution. Let $T_{\max} \in (0, \infty]$ be its maximal existence time. Then, the solution is defined on $[0, T]$ for every $T < T_{\max}$, and hence on the maximal interval $[0, T_{\max})$.

By Proposition 3.2, $S(t)$ and $\lambda(t)$ are nonnegative and bounded on every finite interval. In particular, if $T_{\max} < \infty$, then there exists a constant $M > 0$ such that

$$0 \leq S(t) \leq M, \qquad 0 \leq \lambda(t) \leq M, \qquad t \in [0, T_{\max}).$$

Hence, no finite-time blow-up can occur as $t \to T_{\max}$ with $t < T_{\max}$.

Since the solution remains bounded on $[0, T]$ for all $T < T_{\max}$, the same local existence and uniqueness argument as in Theorem 3.1 can be applied on intervals starting sufficiently close to $T_{\max}$. This yields an extension of the solution beyond $T_{\max}$, that is, there exists $\delta > 0$ such that the solution can be continued to $[0, T_{\max} + \delta)$.

This contradicts the maximality of $T_{\max}$. Therefore, $T_{\max} = \infty$, and the solution exists globally on $[0, \infty)$.

The uniqueness follows from the uniqueness of the local solution. □

Remark 3.1. *For any nonnegative initial data, system* (3.8) *admits a unique global nonnegative solution, and this solution remains bounded for all* $t \geq 0$.

3.3. Existence of equilibria

Note that

$$f(t) = \frac{\Lambda}{\mu} + \left(S_0 - \frac{\Lambda}{\mu}\right)e^{-\mu t} \to \frac{\Lambda}{\mu}, \qquad t \to \infty,$$

and

$$g(t) = \beta(X_I(t) + \theta X_H(t)) \to 0, \qquad t \to \infty,$$

since $X_I(t)$ and $X_H(t)$ represent the infectivity contributions generated by the initial infected individuals and decay exponentially. Therefore, system (3.8) is asymptotically equivalent to the following system:

$$\begin{cases} S(t) = \dfrac{\Lambda}{\mu} - \displaystyle\int_0^t \lambda(t_0)S(t_0)e^{-\mu(t-t_0)}\,dt_0, \\ \lambda(t) = \beta \displaystyle\int_0^t \lambda(t_0)S(t_0)A(t-t_0)\,dt_0. \end{cases} \tag{3.9}$$

This limiting system governs the asymptotic behavior of the original system. In particular, it can be used to characterize the equilibria and threshold dynamics. Let S^* and λ^* respectively denote the equilibrium satisfying the system (3.9). Thus, S^* and λ^* satisfy the equations

$$\begin{aligned} S^* &= \frac{\Lambda}{\mu} - \lambda^* S^* \int_0^\infty e^{-\mu s} ds, \\ \lambda^* &= \beta\lambda^* S^* \int_0^\infty A(s) ds. \end{aligned} \tag{3.10}$$

System (3.9) always has the disease-free equilibrium $U_1^* = (S_1^*, \lambda_1^*) = (\frac{\Lambda}{\mu}, 0)$. If $\mathcal{R}_c > 1$, there is a unique endemic equilibrium $U_2^* = (S_2^*, \lambda_2^*)$, where $S_2^* = \frac{\Lambda}{\mu\mathcal{R}_c}$, $\lambda_2^* = \mu(\mathcal{R}_c - 1)$.

3.4. Local stability analysis

For a bounded real-valued function f on $[0, \infty)$, we define $f_\infty = \liminf_{t\to\infty} f(t)$ and $f^\infty = \limsup_{t\to\infty} f(t)$. Then, we have the following lemma.

Lemma 3.1. *[34] Let $f : [0, \infty) \to \mathbb{R}$ be bounded and twice differentiable with a bounded second derivative. Let $t_n \to \infty$ and $f(t_n)$ converge to f^∞ or f_∞ for $n \to \infty$. Then, $f'(t_n) \to 0$, as $n \to \infty$.*

Based on this lemma, we can show the following global attractivity of the disease-free equilibrium when $\mathcal{R}_c < 1$.

Theorem 3.3. *The disease-free equilibrium U_1^* is a global attractor if $\mathcal{R}_c < 1$.*

Proof. Let

$$\lambda^\infty := \lim_{t\to\infty} \tilde{\lambda}(t), \qquad \tilde{\lambda}(t) = \sup_{s\geq t} \lambda(s).$$

We argue by contradiction. Assume that $\lambda^\infty > 0$. Then, there exists a sequence $\{t_n\}$ with $t_n \to \infty$ such that $\lambda(t_n) \to \lambda^\infty$ as $n \to \infty$. By passing to a subsequence if necessary, we may further assume that $t_{n+1} - t_n \to \infty$ as $n \to \infty$.

Obviously, $S(t) \leq \frac{\Lambda}{\mu}$. By Eq (3.9),

$$\begin{aligned}\lambda(t_{n+1}) &= \beta \int_0^{t_{n+1}} \lambda(s)S(s)A(t_{n+1}-s)ds \\ &= \beta \int_0^{t_n} \lambda(s)S(s)A(t_{n+1}-s)ds + \beta \int_{t_n}^{t_{n+1}} \lambda(s)S(s)A(t_{n+1}-s)ds \\ &\leq \beta\frac{\Lambda}{\mu} \int_0^{t_n} \lambda(s)A(t_{n+1}-s)ds + \beta\frac{\Lambda}{\mu} \int_{t_n}^{t_{n+1}} \lambda(s)A(t_{n+1}-s)ds.\end{aligned} \tag{3.11}$$

Since $\lambda(t) = \beta(I(t) + \theta H(t))$, $\lambda(t)$ is bounded on $[0, \infty)$. Then, there is an $M > 0$ such that $\lambda(s) \leq M$ for all $s \in [0, \infty)$.

Then,

$$\begin{aligned}\int_0^{t_n} \lambda(s)A(t_{n+1}-s)ds &\leq M \int_{t_{n+1}-t_n}^{t_{n+1}} A(\tau)d\tau \\ &\leq M \int_{t_{n+1}-t_n}^{\infty} A(\tau)d\tau \to 0, \ \text{ as } \ n \to \infty.\end{aligned} \tag{3.12}$$

Furthermore, we have

$$\beta\frac{\Lambda}{\mu} \int_{t_n}^{t_{n+1}} \lambda(s)A(t_{n+1}-s)ds \leq \beta\frac{\Lambda}{\mu}\tilde{\lambda}(t_n) \int_0^{\infty} A(\tau)d\tau = \tilde{\lambda}(t_n)\mathcal{R}_c. \tag{3.13}$$

Combining (3.11), (3.12), and (3.13), we obtain

$$\lambda(t_{n+1}) \leq \mathcal{R}_c\, \tilde{\lambda}(t_n) + o(1), \quad n \to \infty.$$

Taking the limit superior as $n \to \infty$, and noting that $\tilde{\lambda}(t_n) \to \lambda^{\infty}$, we obtain

$$\lambda^{\infty} \leq \mathcal{R}_c\, \lambda^{\infty}.$$

Since $\mathcal{R}_c < 1$, this implies

$$\lambda^{\infty} = 0,$$

which contradicts the assumption $\lambda^{\infty} > 0$. Therefore,

$$\lim_{t\to\infty} \lambda(t) = 0.$$

Hence,

$$S(t) = \frac{\Lambda}{\mu} - \int_0^{\infty} \lambda(t-s)S(t-s)e^{-\mu s}ds \to \frac{\Lambda}{\mu}, \ \text{ as } \ t \to \infty.$$

This shows that U_1^* is a global attractor. □

Lemma 3.2. *[21] If solutions of a Volterra integral equation $X(t) = F(t) + \int_0^t K(t-s)G(X(s))ds$ exist on $[0, \infty)$ and are bounded, $F(t) \in C[0, \infty)$, $F(t) \to 0$ as $t \to \infty$, $K(t) \in L^1[0, \infty)$, $G(0) = 0$, the Jacobian $J = DG(0)$ of G is nonsingular, and all roots ω of the characteristic equation $\det(I - \int_0^{\infty} e^{-\omega\tau}K(\tau)Jd\tau) = 0$ have negative real parts, then the origin is locally asymptotically stable for this integral equation.*

Theorem 3.4. *If $\mathcal{R}_c > 1$, then the disease-free equilibrium U_1^* is unstable and the endemic equilibrium U_2^* is locally asymptotically stable.*

Proof. Following the approach of Feng, Xu, and Zhao [21], we shift the equilibrium (S^*, λ^*) to the origin by introducing the change of variables

$$\hat{S} = S - S^*, \qquad \hat{\lambda} = \lambda - \lambda^*.$$

Then, the system in terms of $\hat{S}$ and $\hat{\lambda}$ can be rewritten into the following matrix form of a Volterra integral equation:

$$X(t) = F(t) + \int_0^t K(t-s)G(X(s))ds,$$

with

$$X = \begin{pmatrix} \hat{S} \\ \hat{\lambda} \end{pmatrix}, \ F(t) = \begin{pmatrix} \int_{-\infty}^0 \lambda^* S^* e^{-\mu(t-s)} ds \\ -\beta \int_{-\infty}^0 \lambda^* S^* A(t-s) ds \end{pmatrix},$$
$$K(t-s) = \begin{pmatrix} 0 & -e^{-\mu(t-s)} \\ 0 & \beta A(t-s) \end{pmatrix}, \ G(X) = \begin{pmatrix} \hat{S} \\ \hat{\lambda}(\hat{S} + S^*) + \lambda^* \hat{S} \end{pmatrix}.$$

It is easy to verify that $F(t) \to 0$ as $t \to \infty$ and $G(0) = 0$. Then, we have

$$J = DG(0) = \begin{pmatrix} 1 & 0 \\ \lambda^* & S^* \end{pmatrix}.$$

Since $S^* \neq 0$, J is nonsingular. Denote

$$\int_0^\infty e^{-\omega\tau} K(\tau) J d\tau = \begin{pmatrix} -\frac{\lambda^*}{\mu+\omega} & -\frac{S^*}{\mu+\omega} \\ \lambda^* L(\omega) & S^* L(\omega) \end{pmatrix},$$

where

$$L(\omega) = \beta \int_0^\infty e^{-\omega\tau} A(\tau) d\tau.$$

We can obtain the characteristic equation

$$H(\omega) = \det(I - \int_0^\infty e^{-\omega\tau} K(\tau) J d\tau) = 1 + \frac{\lambda^*}{\mu + \omega} - S^* L(\omega) = 0. \tag{3.14}$$

We first perform the stability analysis for the disease-free equilibrium point (S_1^*, λ_1^*). Noticing $(S_1^*, \lambda_1^*) = (\frac{\Lambda}{\mu}, 0)$, we have $L(\omega) = \frac{\mu}{\Lambda}$ by Eq (3.14). Since $L(0) = \frac{\mu}{\Lambda}\mathcal{R}_c$ and $L'(\omega) < 0$ for any real number ω, $H(\omega) = 0$ has a positive real root if $\mathcal{R}_c > 1$. Hence, the disease-free equilibrium $U_1^* = (S_1^*, \lambda_1^*)$ is unstable if $\mathcal{R}_c > 1$.

Now we investigate the stability of the endemic equilibrium (S_2^*, λ_2^*). Since $(S_2^*, \lambda_2^*) = (\frac{\Lambda}{\mu\mathcal{R}_c}, \mu(\mathcal{R}_c - 1))$, the characteristic equation (3.14) simplifies to

$$\frac{\mu\mathcal{R}_c + \omega}{\mu + \omega} = \frac{\Lambda}{\mu\mathcal{R}_c} L(\omega). \tag{3.15}$$

Assume $H(\omega) = 0$ has a root $\omega = a + bi$ with non-negative real part $a \geq 0$. Since $\mathcal{R}_c > 1$, the real part of the left-hand side of (3.15) is

$$\mathrm{Re}\left(\frac{\mu\mathcal{R}_c + \omega}{\mu + \omega}\right) = \frac{(\mu\mathcal{R}_c + a)(\mu + a) + b^2}{(\mu + a)^2 + b^2} > 1,$$

and the real part of the right-hand side of (3.15) is

$$\mathrm{Re}\left(\frac{\Lambda}{\mu\mathcal{R}_c}L(\omega)\right) = \frac{\Lambda}{\mu\mathcal{R}_c}\beta\int_0^\infty e^{-a\tau}\cos b\tau A(\tau)d\tau < \frac{\Lambda}{\mu\mathcal{R}_c}\beta\int_0^\infty A(\tau)d\tau = 1.$$

This contradiction shows that all eigenvalues have negative real parts. Therefore, the endemic equilibrium $U_2^* = (S_2^*, \lambda_2^*)$ is locally asymptotically stable. □

3.5. Global stability analysis

We note that the term $g(t) = \beta(X_I(t) + \theta X_H(t))$ represents the contribution of individuals who were already in infected-related compartments at the initial time. Hence, it can be interpreted as a past-history term. By incorporating this contribution into a prescribed history on $(-\infty, 0]$, the equation for $\lambda(t)$ in system (3.8) can be reformulated as

$$\lambda(t) = \beta\int_{-\infty}^{t}\lambda(s)S(s)A(t-s)\,ds.$$

Equivalently, by the change of variables $\tau = t - s$,

$$\lambda(t) = \beta\int_0^\infty \lambda(t-\tau)S(t-\tau)A(\tau)\,d\tau.$$

We now consider the following equivalent infinite-delay reformulation of system (3.8):

$$\begin{cases}\dot{S}(t) = \Lambda - \mu S(t) - \lambda(t)S(t),\\ \lambda(t) = \beta\displaystyle\int_0^\infty \lambda(t-\tau)S(t-\tau)A(\tau)\,d\tau,\end{cases} \tag{3.16}$$

where the values of $\lambda(t-\tau)S(t-\tau)$ for $t - \tau < 0$ are prescribed by the past history.

We assume that there exists $\bar{\tau} > 0$ such that $A(\tau) = 0$, $\forall\tau > \bar{\tau}$. In other words, $\bar{\tau}$ is the maximal infection age at which an infected individual can still make a nonzero contribution to the force of infection.

Then, system (3.16) can be rewritten as

$$\begin{cases}\dot{S}(t) = \Lambda - \mu S(t) - \lambda(t)S(t),\\ \lambda(t) = \beta\displaystyle\int_0^{\bar{\tau}} \lambda(t-\tau)S(t-\tau)A(\tau)\,d\tau.\end{cases} \tag{3.17}$$

Let $\Omega = C_+([-\bar{\tau}, 0]) \times L_+^1(-\bar{\tau}, 0)$, and define the semiflow $\Phi_t : \Omega \to \Omega$ by

$$(S_t(s), \lambda_t(s)) = (S(t+s), \lambda(t+s)), \qquad s \in [-\bar{\tau}, 0].$$

The norm on Ω is defined by

$$\|(\phi,\psi)\|_\Omega = \sup_{s\in[-\bar\tau,0]}|\phi(s)| + \int_{-\bar\tau}^{0}|\psi(s)|\,ds.$$

Let $\mathcal{R}_c = \beta\frac{\Lambda}{\mu}\int_0^{\bar\tau} A(\tau)\,d\tau$. Since $A(\tau) = 0$ for all $\tau > \bar\tau$, we also have $\mathcal{R}_c = \beta\frac{\Lambda}{\mu}\int_0^{\infty} A(\tau)\,d\tau$, which is consistent with the definition given previously. Then, (3.17) always admits the disease-free equilibrium $U_1^* = (S_1^*, \lambda_1^*) = \left(\frac{\Lambda}{\mu}, 0\right)$. If $\mathcal{R}_c > 1$, then (3.17) admits the endemic equilibrium $U_2^* = (S_2^*, \lambda_2^*) = \left(\frac{\Lambda}{\mu\mathcal{R}_c}, \mu(\mathcal{R}_c - 1)\right)$.

Theorem 3.5. *If $\mathcal{R}_c \le 1$, then the disease-free equilibrium U_1^* is globally asymptotically stable.*

Proof. Define $D = \Phi_{\bar\tau}(\Omega)$ and consider the Lyapunov functional

$$V[S,\lambda] = g\left(\frac{S(0)}{S_1^*}\right) + \int_0^{\bar\tau}\alpha(\tau)\lambda(-\tau)S(-\tau)\,d\tau,$$

where $g(x) = x - 1 - \ln x$, $\alpha(\tau) = \beta\int_\tau^{\bar\tau} A(s)\,ds$. Then, $\alpha(0) = \beta\int_0^{\bar\tau} A(s)\,ds = \frac{\mathcal{R}_c}{S_1^*}$, $\alpha(\bar\tau) = 0$, $\alpha'(\tau) = -\beta A(\tau)$.

Along the trajectories of (3.17),

$$V[S_t,\lambda_t] = g\left(\frac{S_t(0)}{S_1^*}\right) + \int_0^{\bar\tau}\alpha(\tau)\lambda_t(-\tau)S_t(-\tau)\,d\tau = g\left(\frac{S(t)}{S_1^*}\right) + \int_{t-\bar\tau}^{t}\alpha(t-s)\lambda(s)S(s)\,ds.$$

We use the identity

$$g(x) + g\left(\frac{1}{x}\right) = x + \frac{1}{x} - 2 = \frac{(x-1)^2}{x}.$$

Since $\Lambda = \mu S_1^*$, we have

$$\begin{aligned}\frac{d}{dt}g\left(\frac{S(t)}{S_1^*}\right) &= \left(\frac{1}{S_1^*} - \frac{1}{S(t)}\right)\dot S(t)\\ &= -\mu\left[g\left(\frac{S(t)}{S_1^*}\right) + g\left(\frac{S_1^*}{S(t)}\right)\right] - \lambda(t)\left(\frac{S(t)}{S_1^*} - 1\right).\end{aligned}$$

Moreover,

$$\begin{aligned}\frac{d}{dt}\int_{t-\bar\tau}^{t}\alpha(t-s)\lambda(s)S(s)\,ds &= \alpha(0)\lambda(t)S(t) + \int_{t-\bar\tau}^{t}\alpha'(t-s)\lambda(s)S(s)\,ds\\ &= \frac{\mathcal{R}_c}{S_1^*}\lambda(t)S(t) - \beta\int_{t-\bar\tau}^{t}A(t-s)\lambda(s)S(s)\,ds\\ &= \lambda(t)\left(\frac{\mathcal{R}_c S(t)}{S_1^*} - 1\right).\end{aligned}$$

Therefore,

$$\frac{d}{dt}V[S_t,\lambda_t] = -\mu\left[g\left(\frac{S(t)}{S_1^*}\right) + g\left(\frac{S_1^*}{S(t)}\right)\right] + (\mathcal{R}_c - 1)\lambda(t)\frac{S(t)}{S_1^*} \le 0.$$

Hence, $\dot V \le 0$ whenever $\mathcal{R}_c \le 1$. Furthermore, $\dot V = 0$ implies $S(t) = S_1^*$, and, if $\mathcal{R}_c < 1$, $\lambda(t) = 0$. Thus, the largest invariant set in $\{\dot V = 0\}$ is the singleton $\{U_1^*\}$. By LaSalle's invariance principle, U_1^* is globally asymptotically stable in Ω. □

Define

$$\widehat{\Omega} = \left\{(S,\lambda) \in \Omega : \exists a \in [0,\bar{\tau}] \text{ such that } \int_0^{\bar{\tau}} A(\tau+a)\lambda(-\tau)S(-\tau)\,d\tau > 0\right\}.$$

Theorem 3.6. *If $\mathcal{R}_c > 1$, then the endemic equilibrium U_2^* is globally asymptotically stable in $\widehat{\Omega}$.*

Proof. Define $\widehat{D} = \Phi_{\bar{\tau}}(\widehat{\Omega})$ and consider the Lyapunov functional

$$V[S,\lambda] = g\left(\frac{S(0)}{S_2^*}\right) + \int_0^{\bar{\tau}} \delta(\tau)\, g\left(\frac{\lambda(-\tau)S(-\tau)}{\lambda_2^* S_2^*}\right) d\tau,$$

where $\delta(\tau) = \beta\lambda_2^* S_2^* \int_\tau^{\bar{\tau}} A(s)\,ds$. Since $1 = \beta S_2^* \int_0^{\bar{\tau}} A(s)\,ds$, we obtain $\delta(\bar{\tau}) = 0$, $\delta(0) = \lambda_2^*$, $\delta'(\tau) = -\beta\lambda_2^* S_2^* A(\tau)$.

Along the trajectories of (3.17),

$$V[S_t,\lambda_t] = g\left(\frac{S_t(0)}{S_2^*}\right) + \int_0^{\bar{\tau}} \delta(\tau)\, g\left(\frac{\lambda_t(-\tau)S_t(-\tau)}{\lambda_2^* S_2^*}\right) d\tau = g\left(\frac{S(t)}{S_2^*}\right) + \int_{t-\bar{\tau}}^{t} \delta(t-s)\, g\left(\frac{\lambda(s)S(s)}{\lambda_2^* S_2^*}\right) ds.$$

Using $\Lambda = \mu S_2^* + \lambda_2^* S_2^*$, we get

$$\frac{d}{dt} g\left(\frac{S(t)}{S_2^*}\right) = -\mu\left[g\left(\frac{S(t)}{S_2^*}\right) + g\left(\frac{S_2^*}{S(t)}\right)\right] + \lambda_2^*\left[-g\left(\frac{S_2^*}{S(t)}\right) - g\left(\frac{S(t)\lambda(t)}{S_2^*\lambda_2^*}\right) + g\left(\frac{\lambda(t)}{\lambda_2^*}\right)\right].$$

On the other hand,

$$\begin{aligned}
&\frac{d}{dt}\int_{t-\bar{\tau}}^{t} \delta(t-s)\, g\left(\frac{\lambda(s)S(s)}{\lambda_2^* S_2^*}\right) ds \\
=&\delta(0)\, g\left(\frac{\lambda(t)S(t)}{\lambda_2^* S_2^*}\right) + \int_{t-\bar{\tau}}^{t} \delta'(t-s)\, g\left(\frac{\lambda(s)S(s)}{\lambda_2^* S_2^*}\right) ds \\
=&\lambda_2^* g\left(\frac{\lambda(t)S(t)}{\lambda_2^* S_2^*}\right) - \beta\lambda_2^* S_2^* \int_{t-\bar{\tau}}^{t} A(t-s) g\left(\frac{\lambda(s)S(s)}{\lambda_2^* S_2^*}\right) ds.
\end{aligned}$$

Since

$$\beta S_2^* \int_0^{\bar{\tau}} A(\tau)\,d\tau = 1,$$

the measure $d\nu(\tau) = \beta S_2^* A(\tau)\,d\tau$ is a probability measure on $[0,\bar{\tau}]$. By Jensen's inequality,

$$\beta S_2^* \int_{t-\bar{\tau}}^{t} A(t-s) g\left(\frac{\lambda(s)S(s)}{\lambda_2^* S_2^*}\right) ds \geq g\left(\beta S_2^* \int_{t-\bar{\tau}}^{t} A(t-s)\frac{\lambda(s)S(s)}{\lambda_2^* S_2^*}\, ds\right) = g\left(\frac{\lambda(t)}{\lambda_2^*}\right).$$

Therefore,

$$\frac{d}{dt}\int_{t-\bar{\tau}}^{t} \delta(t-s)\, g\left(\frac{\lambda(s)S(s)}{\lambda_2^* S_2^*}\right) ds \leq \lambda_2^*\left[g\left(\frac{\lambda(t)S(t)}{\lambda_2^* S_2^*}\right) - g\left(\frac{\lambda(t)}{\lambda_2^*}\right)\right].$$

Combining the above estimates yields

$$\frac{d}{dt} V[S_t,\lambda_t] \leq -\mu\left[g\left(\frac{S(t)}{S_2^*}\right) + g\left(\frac{S_2^*}{S(t)}\right)\right] - \lambda_2^* g\left(\frac{S_2^*}{S(t)}\right) \leq 0.$$

Hence, the largest invariant set in $\{\dot{V} = 0\}$ consists only of the endemic equilibrium U_2^*. By LaSalle's invariance principle, U_2^* is globally asymptotically stable in $\widehat{\Omega}$. □

4. Model with Gamma distributions

4.1. The system of ODEs

Now we study the Gamma stage distribution case. Let

$$\begin{aligned} P_{EI}(t) &= \sum_{k=1}^{m_1} \frac{(m_1\alpha_1 t)^{k-1} e^{-m_1\alpha_1 t}}{(k-1)!}, P_{IR}(t) = \sum_{k=1}^{n_1} \frac{(n_1\delta_1 t)^{k-1} e^{-n_1\delta_1 t}}{(k-1)!}, \\ P_{QH}(t) &= \sum_{k=1}^{m_2} \frac{(m_2\alpha_2 t)^{k-1} e^{-m_2\alpha_2 t}}{(k-1)!}, P_{HR}(t) = \sum_{k=1}^{n_2} \frac{(n_2\delta_2 t)^{k-1} e^{-n_2\delta_2 t}}{(k-1)!}, \\ P_{EQ}(t) &= \sum_{k=1}^{m_3} \frac{(m_3\xi t)^{k-1} e^{-m_3\xi t}}{(k-1)!}, \quad P_{IH}(t) = \sum_{k=1}^{n_3} \frac{(n_3\phi t)^{k-1} e^{-n_3\phi t}}{(k-1)!}. \end{aligned} \tag{4.1}$$

The following expression will be used frequently:

$$\begin{aligned} -\dot{P}_{EI}(t) &= \frac{(m_1\alpha_1)^{m_1} t^{m_1-1} e^{-m_1\alpha_1 t}}{(m_1-1)!}, -\dot{P}_{IR}(t) = \frac{(n_1\delta_1)^{n_1} t^{n_1-1} e^{-n_1\delta_1 t}}{(n_1-1)!}, \\ -\dot{P}_{QH}(t) &= \frac{(m_2\alpha_2)^{m_2} t^{m_2-1} e^{-m_2\alpha_2 t}}{(m_2-1)!}, -\dot{P}_{HR}(t) = \frac{(n_2\delta_2)^{n_2} t^{n_2-1} e^{-n_2\delta_2 t}}{(n_2-1)!}, \\ -\dot{P}_{EQ}(t) &= \frac{(m_3\xi)^{m_3} t^{m_3-1} e^{-m_3\xi t}}{(m_3-1)!}, \quad -\dot{P}_{IH}(t) = \frac{(n_3\phi)^{n_3} t^{n_3-1} e^{-n_3\phi t}}{(n_3-1)!}. \end{aligned}$$

In the survival functions $P_{EI}(t)$, $P_{QH}(t)$, $P_{EQ}(t)$,$P_{IR}(t)$, $P_{HR}(t)$, and $P_{IH}(t)$, the integers m_1, m_2, m_3, n_1, n_2, and n_3 denote the shape parameters, whereas $1/\alpha_1$, $1/\alpha_2$, $1/\xi$, $1/\delta_1$, $1/\delta_2$, and $1/\phi$ represent the means of the corresponding gamma distributions, respectively. Substituting the above parameters into system (2.7) and differentiating with respect to time, we obtain the following ordinary differential equation model in a compact form which obeys the flowchart of Figure 3.

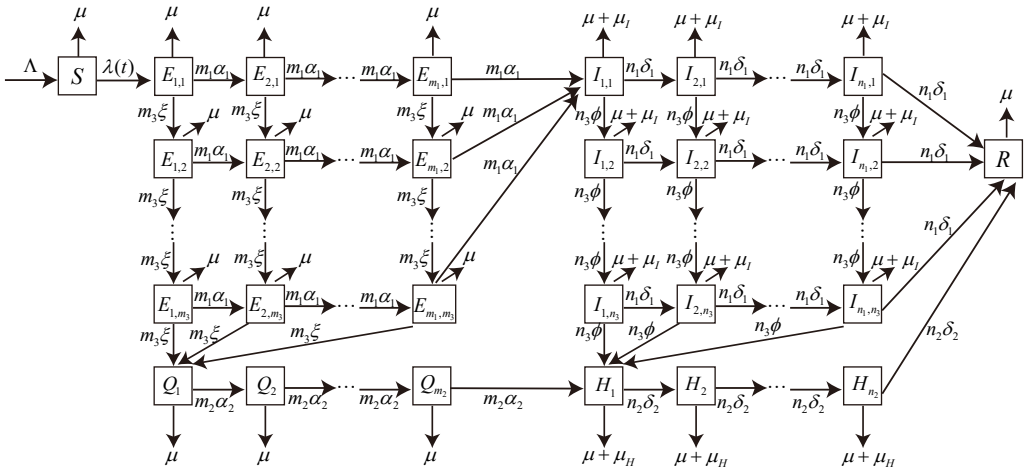

Figure 3. Transfer diagram of the model with Gamma stage distributions.

$$
\begin{aligned}
\dot{S}(t) &= \Lambda - \lambda(t)S(t) - \mu S(t),\\
\dot{E}_{1,1}(t) &= \lambda(t)S(t) - (m_1\alpha_1 + m_3\xi + \mu)\,E_{1,1}(t),\\
\dot{E}_{k,1}(t) &= m_1\alpha_1\,E_{k-1,1}(t) - (m_1\alpha_1 + m_3\xi + \mu)\,E_{k,1}(t),\ k = 2,\dots,m_1,\\
\dot{E}_{1,l}(t) &= m_3\xi\,E_{1,l-1}(t) - (m_1\alpha_1 + m_3\xi + \mu)\,E_{1,l}(t),\ l = 2,\dots,m_3,\\
\dot{E}_{k,l}(t) &= m_1\alpha_1\,E_{k-1,l}(t) + m_3\xi\,E_{k,l-1}(t) - (m_1\alpha_1 + m_3\xi + \mu)\,E_{k,l}(t),\ k = 2,\dots,m_1,\ l = 2,\dots,m_3,\\
\dot{I}_{1,1}(t) &= m_1\alpha_1\sum_{l=1}^{m_3} E_{m_1,l}(t) - (n_1\delta_1 + n_3\phi + \mu_I + \mu)I_{1,1}(t),\\
\dot{I}_{i,1}(t) &= n_1\delta_1\,I_{i-1,1}(t) - (n_1\delta_1 + n_3\phi + \mu_I + \mu)I_{i,1}(t),\ i = 2,\dots,n_1,\\
\dot{I}_{1,j}(t) &= n_3\phi\,I_{1,j-1}(t) - (n_1\delta_1 + n_3\phi + \mu_I + \mu)I_{1,j}(t),\ j = 2,\dots,n_3,\\
\dot{I}_{i,j}(t) &= n_1\delta_1\,I_{i-1,j}(t) + n_3\phi\,I_{i,j-1}(t) - (n_1\delta_1 + n_3\phi + \mu_I + \mu)I_{i,j}(t),\ i = 2,\dots,n_1,\ j = 2,\dots,n_3,\\
\dot{Q}_1(t) &= m_3\xi\sum_{i=1}^{m_1} E_{i,m_3}(t) - (m_2\alpha_2 + \mu)Q_1(t),\\
\dot{Q}_p(t) &= m_2\alpha_2\,Q_{p-1}(t) - (m_2\alpha_2 + \mu)Q_p(t),\ p = 2,\dots,m_2,\\
\dot{H}_1(t) &= m_2\alpha_2 Q_{m_2}(t) + n_3\phi\sum_{i=1}^{n_1} I_{i,n_3}(t) - (n_2\delta_2 + \mu_H + \mu)H_1(t),\\
\dot{H}_q(t) &= n_2\delta_2\,H_{q-1}(t) - (n_2\delta_2 + \mu_H + \mu)H_q(t),\ q = 2,\dots,n_2,\\
\dot{R}(t) &= n_1\delta_1\sum_{j=1}^{n_3} I_{n_1,j}(t) + n_2\delta_2\,H_{n_2}(t) - \mu R(t),
\end{aligned}
\tag{4.2}
$$

where

$$
E(t) = \sum_{k=1}^{m_1}\sum_{l=1}^{m_3} E_{k,l}(t),\quad I(t) = \sum_{i=1}^{n_1}\sum_{j=1}^{n_3} I_{i,j}(t),\quad Q(t) = \sum_{p=1}^{m_2} Q_p(t),\quad H(t) = \sum_{q=1}^{n_2} H_q(t).
$$

Substituting (4.1) into (3.2) and simplifying, we obtain

$$
\begin{aligned}
T_{EI} &= \left(\frac{m_1\alpha_1}{m_1\alpha_1 + m_3\xi + \mu}\right)^{m_1}\sum_{i=0}^{m_3-1}\binom{i+m_1-1}{i}\left(\frac{m_3\xi}{m_1\alpha_1 + m_3\xi + \mu}\right)^{i},\\
T_{EQ} &= \left(\frac{m_3\xi}{m_1\alpha_1 + m_3\xi + \mu}\right)^{m_3}\sum_{j=0}^{m_1-1}\binom{j+m_3-1}{j}\left(\frac{m_1\alpha_1}{m_1\alpha_1 + m_3\xi + \mu}\right)^{j},\\
T_{QH} &= \left(\frac{m_2\alpha_2}{m_2\alpha_2 + \mu}\right)^{m_2},\quad T_{IH} = \left(\frac{n_3\phi}{n_1\delta_1 + n_3\phi + \mu_I + \mu}\right)^{n_3}\sum_{i=0}^{n_1-1}\binom{i+n_3-1}{i}\left(\frac{n_1\delta_1}{n_1\delta_1 + n_3\phi + \mu_I + \mu}\right)^{i},\\
D_I &= \sum_{i=0}^{n_1-1}\sum_{j=0}^{n_3-1}\binom{i+j}{i}\frac{(n_1\delta_1)^i(n_3\phi)^j}{(n_1\delta_1 + n_3\phi + \mu_I + \mu)^{i+j+1}},\quad D_H = \sum_{k=0}^{n_2-1}\frac{(n_2\delta_2)^k}{(n_2\delta_2 + \mu_H + \mu)^{k+1}}.
\end{aligned}
\tag{4.3}
$$

Furthermore, we get the reproduction number $\mathcal{R}_c$ as below:

$$
\mathcal{R}_c = \beta\frac{\Lambda}{\mu}[T_{EI}D_I + \theta(T_{EI}T_{IH}D_H + T_{EQ}T_{QH}D_H)]. \tag{4.4}
$$

In particular, when all shape parameters are set to one, the proposed model reduces to its Markovian counterpart with exponentially distributed stage durations. Letting $m_1 = m_2 = m_3 = n_1 = n_2 = n_3 = 1$, we can obtain

$$P_{EI}(t) = e^{-\alpha_1 t},\ P_{IR}(t) = e^{-\delta_1 t},\ P_{EQ}(t) = e^{-\xi t},\ P_{QH}(t) \ = e^{-\alpha_2 t},\ P_{HR}(t) = e^{-\delta_2 t},\ P_{IH}(t) = e^{-\phi t}. \quad (4.5)$$

Furthermore, we obtain the following ordinary differential equation model in a compact form which obeys the flowchart of Figure 4.

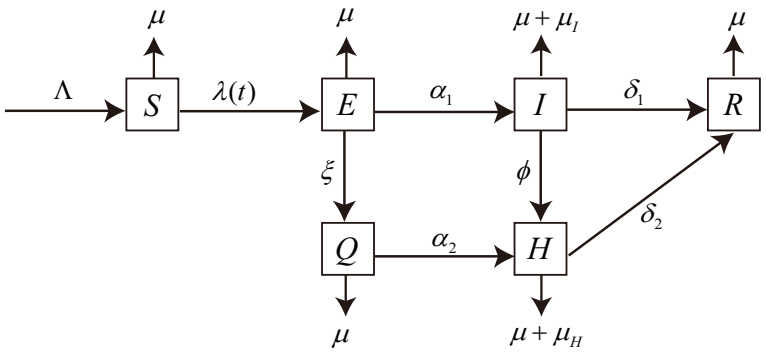

Figure 4. Transfer diagram of the model with exponential stage distributions.

$$\begin{aligned}
\dot{S}(t) &= \Lambda - \lambda(t)S(t) - \mu S(t),\\
\dot{E}(t) &= \lambda(t)S(t) - (\alpha_1 + \xi + \mu)E(t),\\
\dot{I}(t) &= \alpha_1 E(t) - (\delta_1 + \phi + \mu_I + \mu)I(t),\\
\dot{Q}(t) &= \xi E(t) - (\alpha_2 + \mu)Q(t),\\
\dot{H}(t) &= \phi I(t) + \alpha_2 Q(t) - (\delta_2 + \mu_H + \mu)H(t),\\
\dot{R}(t) &= \delta_1 I(t) + \delta_2 H(t) - \mu R(t).
\end{aligned} \quad (4.6)$$

By a straightforward calculation, we obtain the control reproduction number $\mathcal{R}_c^{EDM}$ as follows:

$$\mathcal{R}_c^{EDM} = \beta\frac{\Lambda}{\mu}[\bar{T}_{EI}\bar{D}_I + \theta(\bar{T}_{EI}\bar{T}_{IH}\bar{D}_H + \bar{T}_{EQ}\bar{T}_{QH}\bar{D}_H)], \quad (4.7)$$

where

$$\begin{aligned}
&\bar{T}_{EI} = \frac{\alpha_1}{\xi + \alpha_1 + \mu}, \quad \bar{T}_{EQ} = \frac{\xi}{\xi + \alpha_1 + \mu}, \quad \bar{T}_{IH} = \frac{\phi}{\phi + \delta_1 + \mu_I + \mu},\\
&\bar{T}_{QH} = \frac{\alpha_2}{\alpha_2 + \mu}, \quad \bar{D}_I = \frac{1}{\phi + \delta_1 + \mu_I + \mu}, \quad \bar{D}_H = \frac{1}{\delta_2 + \mu_H + \mu}.
\end{aligned} \quad (4.8)$$

It is worth noting that while the Gamma-distributed case can be reformulated as an equivalent ODE system via the linear chain technique, the dimension of the resulting system increases with the shape parameters, leading to higher computational complexity.

4.2. *Sensitivity analysis of* $\mathcal{R}_c$

In this subsection, we investigate how key control parameters affect the control reproduction number $\mathcal{R}_c$.

By $\frac{\partial \mathcal{R}_c}{\partial \theta} = \beta \frac{\Lambda}{\mu}\left(T_{EI}T_{IH}D_H + T_{EQ}T_{QH}D_H\right) > 0$, the control reproduction number $\mathcal{R}_c$ is an increasing function of θ. A larger value of θ, corresponding to less effective hospitalization and isolation, results in an increased control reproduction number, whereas improved isolation measures (decreasing θ) effectively reduce $\mathcal{R}_c$.

The parameter values used in the numerical simulations are specified as follows. In [35], the tuberculosis case fatality rate during treatment is reported to be 0.24%. Assuming a constant disease-induced mortality rate during hospitalization, the cumulative probability of death over a treatment period T satisfies $0.24\% = 1 - e^{-\mu_H T}$. Taking the treatment duration to be $T = 6$ months (i.e., $T = 0.5$ years), we obtain $\mu_H = 0.0048$ year^{-1}. We further assume that the disease-induced mortality rate during the infectious stage is lower than that during hospitalization, and set $\mu_I = 0.5\,\mu_H = 0.0024$ year^{-1}. The remaining model parameters are specified as follows: $\Lambda = 0.5$ (person $\cdot$ year)$^{-1}$, $\mu = 0.014$ year^{-1}, $\beta = 0.11$ year^{-1}, $\alpha_1 = 0.87$ year^{-1}, $\alpha_2 = 0.09$ year^{-1}, $\delta_1 = 0.09$ year^{-1}, and $\delta_2 = 0.72$ year^{-1}. All these parameter values are taken from [36]. Unless otherwise stated, these baseline parameters are kept fixed throughout, and we focus on the effects of the remaining parameters on the control reproduction number $\mathcal{R}_c$.

To investigate the effects of ξ and ϕ on $\mathcal{R}_c$, we fix the Gamma (Erlang) shape parameters as $n_1 = n_2 = n_3 = m_1 = m_2 = m_3 = 3$ and set $\theta = 0.1$ in model (4.2). Figure 5 illustrates heat maps of the control reproduction number $\mathcal{R}_c$ over the (ξ, ϕ)-plane. As either the detection and quarantine rate ξ or the hospitalization and isolation rate ϕ increases, the threshold contour shifts toward smaller parameter values, indicating a decrease in $\mathcal{R}_c$. Larger ξ leads to earlier detection and quarantine, reflecting enhanced screening and contact tracing capacity. Similarly, larger ϕ accelerates hospitalization and isolation after infection, indicating more effective medical intervention. Overall, strengthening either intervention reduces transmission, with the most pronounced effect when both are enhanced.

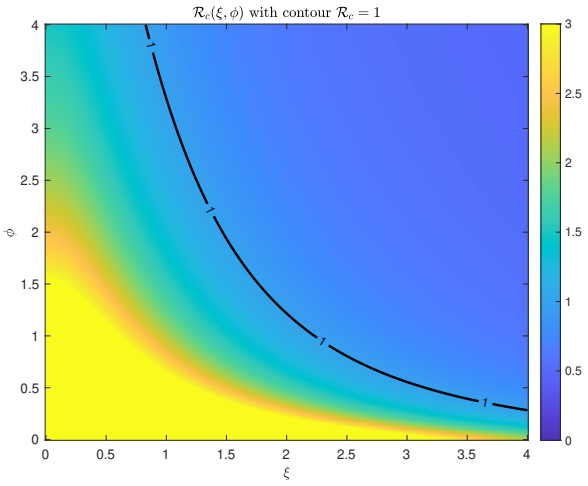

Figure 5. Heat maps of the control reproduction number $\mathcal{R}_c(\xi, \phi)$ under $\theta = 0.1$ and $n_1 = n_2 = n_3 = m_1 = m_2 = m_3 = 3$ for model 4.2. The black dashed curves indicate the threshold $\mathcal{R}_c = 1$.

To investigate the sensitivity of the control reproduction number to the shape parameters, we fix the intervention intensities at $\xi = 3$ and $\phi = 3$ in the following numerical simulations.

We first examine the effects of the shape parameters (n_1, n_2) on $\mathcal{R}_c$. Specifically, we set $\theta = 0.149$ and fix $n_3 = m_1 = m_2 = m_3 = 1$. Figure 6 illustrates heat maps of the control reproduction number $\mathcal{R}_c$ over the (n_1, n_2)-plane. When $n_1 = n_2 = 1$, corresponding to exponentially distributed stage durations, we obtain $\mathcal{R}_c(1, 1) = 0.9843 < 1$, indicating that the disease is controllable under this assumption. However, $\mathcal{R}_c$ increases with both shape parameters n_1 and n_2, suggesting that the exponential assumption may substantially underestimate the transmission potential compared with more realistic Gamma-distributed stage durations.

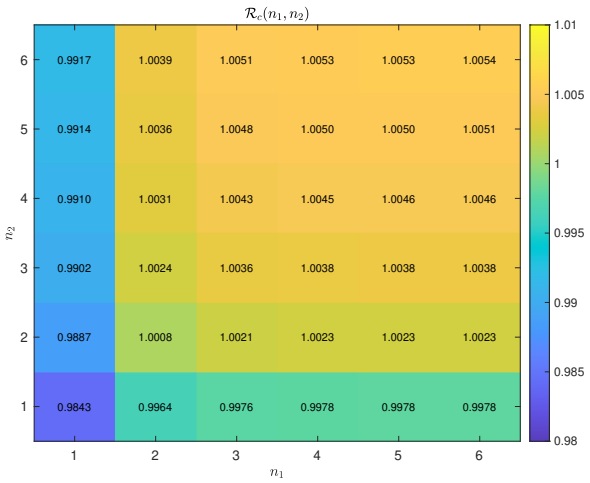

Figure 6. Heat map of the control reproduction number $\mathcal{R}_c$ as a function of the shape parameters (n_1, n_2). The numerical value of $\mathcal{R}_c$ is displayed in each grid cell. The parameters are fixed at $\xi = 3$, $\phi = 3$, $\theta = 0.149$, and $n_3 = m_1 = m_2 = m_3 = 1$.

We then examine the effects of the shape parameters (m_1, m_2) on the control reproduction number $\mathcal{R}_c$. Specifically, we set $\theta = 0.166$ and fix $n_1 = n_2 = n_3 = m_3 = 1$. Figure 7 illustrates heat maps of $\mathcal{R}_c$ over the (m_1, m_2)-plane. When $m_1 = m_2 = 1$, corresponding to exponentially distributed stage durations, we obtain $\mathcal{R}_c(1, 1) = 1.0643 > 1$, indicating that the disease is uncontrollable under this assumption. However, $\mathcal{R}_c$ decreases with increasing m_1 and m_2, suggesting that epidemic models based on exponential assumptions may substantially overestimate the transmission potential compared with models incorporating more realistic Gamma-distributed stage durations.

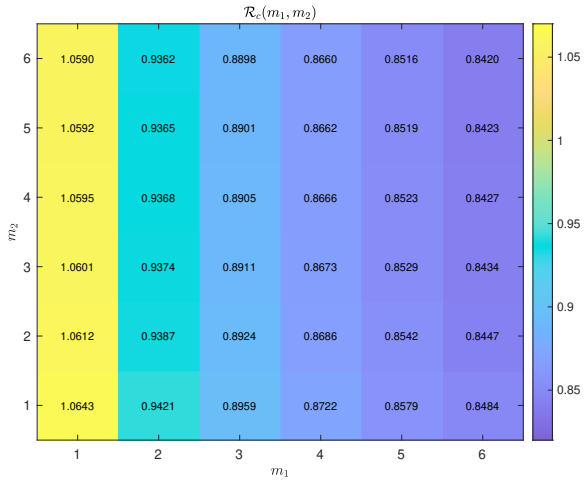

Figure 7. Heat map of the control reproduction number $\mathcal{R}_c$ as a function of the shape parameters (m_1, m_2). The numerical value of $\mathcal{R}_c$ is displayed in each grid cell. The parameters are fixed at $\xi = 3$, $\phi = 3$, $\theta = 0.166$, and $n_1 = n_2 = n_3 = m_3 = 1$.

Finally, we examine the effects of the shape parameters (m_3, n_3) on the control reproduction number $\mathcal{R}_c$. Specifically, we set $\theta = 0.149$ and fix $n_1 = n_2 = m_1 = m_2 = 1$. Figure 8 illustrates heat maps of $\mathcal{R}_c$ over the (m_3, n_3)-plane. When $m_3 = n_3 = 1$, corresponding to exponentially distributed stage durations, we obtain $\mathcal{R}_c(1, 1) = 0.9843 < 1$, indicating that the disease is controllable under this assumption. However, $\mathcal{R}_c$ increases with increasing m_3 and n_3, suggesting that epidemic models based on exponential assumptions may substantially underestimate the transmission potential compared with models incorporating more realistic Gamma-distributed stage durations.

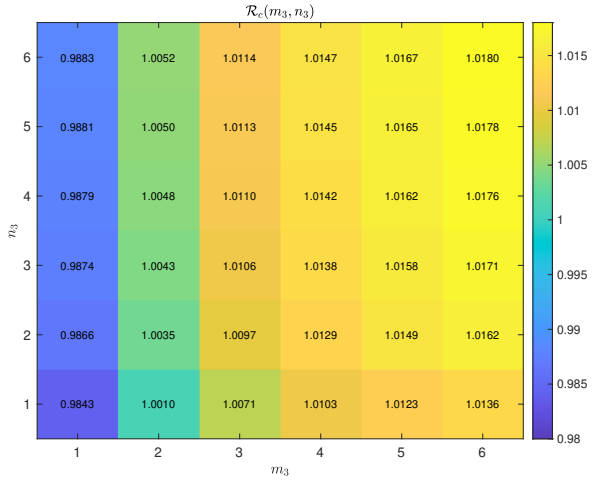

Figure 8. Heat map of the control reproduction number $\mathcal{R}_c$ as a function of the shape parameters (m_3, n_3). The numerical value of $\mathcal{R}_c$ is displayed in each grid cell. The parameters are fixed at $\xi = 3$, $\phi = 3$, $\theta = 0.149$, and $n_1 = n_2 = m_1 = m_2 = 1$.

Taking $n_1 = n_2 = n_3 = m_1 = m_2 = m_3 = 1$ as the baseline, under the parameter setting $\xi = 3$, $\phi = 3$,

and $\theta = 0.149$, we have $\mathcal{R}_c = 0.9843$. Define the relative change rate by

$$\frac{\Delta\mathcal{R}_c}{\mathcal{R}_c} = \frac{\mathcal{R}_c^{\text{new}} - \mathcal{R}_c^{\text{base}}}{\mathcal{R}_c^{\text{base}}}.$$

The calculations show that, when n_1 increases from 1 to 5, $\mathcal{R}_c$ rises from 0.9843 to 0.9978, corresponding to a relative increase of about 1.37%. When the shape parameter n_2 increases from 1 to 5, $\mathcal{R}_c$ rises from 0.9843 to 0.9914, corresponding to a relative increase of about 0.72%. When n_3 increases from 1 to 5, $\mathcal{R}_c$ rises from 0.9843 to 0.9881, corresponding to a relative increase of about 0.39%. When m_3 increases from 1 to 5, $\mathcal{R}_c$ rises from 0.9843 to 1.0123, corresponding to a relative increase of about 2.84%. Therefore, under this parameter set, the effects of these four shape parameters on $\mathcal{R}_c$ can be ranked as

$$m_3 > n_1 > n_2 > n_3.$$

This indicates that the variation in m_3 has the strongest promoting effect on the control reproduction number, whereas the effect of n_3 is relatively weak.

Furthermore, taking $n_1 = n_2 = n_3 = m_1 = m_2 = m_3 = 1$ as the baseline, under the parameter setting $\xi = 3$, $\phi = 3$, and $\theta = 0.166$, we have $\mathcal{R}_c = 1.0643$. When m_1 increases from 1 to 5, $\mathcal{R}_c$ decreases from 1.0643 to 0.8579, corresponding to a relative reduction of about 19.39%. When m_2 increases from 1 to 5, $\mathcal{R}_c$ decreases from 1.0643 to 1.0592, corresponding to a relative reduction of about 0.48%. Hence, under this parameter set, the inhibitory effect of m_1 on $\mathcal{R}_c$ is much stronger than that of m_2, suggesting that m_1 is far more influential on the epidemic threshold, while the effect of m_2 is comparatively limited.

Numerical simulations demonstrate that both control-related parameters and the shape parameters of stage-duration distributions play crucial roles in determining the control reproduction number. Specifically, increasing the ineffective isolation coefficient θ weakens hospital isolation and enhances the transmission ability of hospitalized individuals, resulting in a higher control reproduction number. In contrast, larger values of ξ and ϕ correspond to strengthened quarantine and isolation measures, reflecting earlier quarantine, earlier detection, and earlier isolation, which effectively shorten the infectious period and reduce the control reproduction number. Moreover, under the assumption of Gamma-distributed stage durations, increasing the shape parameters n_1 and n_2 decreases the probability of early recovery, thereby prolonging the effective infectious period and leading to an increase in the control reproduction number. Similarly, larger values of n_3 and m_3 reduce the probability of early quarantine or isolation, allowing infected individuals to remain longer in the community and increasing transmission risk, which further elevates the control reproduction number. Conversely, increasing m_1 and m_2 reduces the probability of prolonged residence in the latent stage, accelerates disease progression, and consequently decreases the control reproduction number.

5. Conclusions

In this study, we assume that the latent, infectious, quarantine, and hospital isolation stages follow non-exponentially distributed durations, and develop an SEIQHR epidemic model formulated as a system of integral equations that incorporates natural mortality, disease-induced mortality, and hospital-related mortality. We derive the basic reproduction number and the control reproduction number for the integral equation model, and investigate the stability of the equilibria. Under the assumption of Gamma-distributed stage durations, we further derive an equivalent system of ordinary

differential equations (ODEs). Our results demonstrate that strengthened control measures can effectively reduce transmission potential. Specifically, improved hospital isolation (corresponding to a decrease in θ), enhanced screening and contact tracing capacity (corresponding to an increase in ξ), and more efficient medical intervention (corresponding to an increase in ϕ) all lead to a reduction in transmission risk. In addition, the shape parameters of stage-duration distributions have a pronounced impact on the control reproduction number. In particular, increasing n_1, n_2, n_3, and m_3 results in an increase in $\mathcal{R}_c$, with m_3 having the most pronounced positive effect. Conversely, increasing m_1 and m_2 leads to a decrease in $\mathcal{R}_c$, with m_1 exerting a stronger inhibitory effect. This suggests that epidemic models based on exponential assumptions may oversimplify disease progression and bias the assessment of epidemic risk and control effectiveness.

The integral equation approach adopted here provides a unified methodology for analyzing epidemic models with general stage-duration distributions. Compared with classical SEIR-type models with exponentially distributed stage durations, the model in this paper incorporates general (non-exponential) distributions and explicitly includes quarantine (Q) and hospitalization (H) compartments, leading to a more realistic description of disease transmission. Moreover, numerical results further demonstrate that incorporating non-exponential stage-duration distributions can significantly affect transmission thresholds and the effectiveness of control measures. Non-exponential distributions capture the variability in latent and infectious periods, particularly for diseases such as COVID-19 and tuberculosis, while the inclusion of Q and H compartments enables explicit modeling of intervention effects. The combination of these features enhances the flexibility and accuracy of the model in describing transmission dynamics under intervention strategies, which cannot be adequately captured by classical SEIR models. Future work may incorporate population heterogeneity, time-dependent control measures, and additional transmission mechanisms, such as environmental transmission or vector-borne transmission, to further enhance the applicability of the proposed framework. In addition, parameter estimation, data fitting, and prediction based on real-world data will be investigated in subsequent work.

Author contributions

Fang Liu: Conceptualization, formal analysis, writing—original draft; Zhen Jin: Methodology, project administration, review and editing. Both authors have read and approved the final version of the manuscript for publication.

Use of Generative-AI tools declaration

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

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Conflict of interest

The authors declare no conflicts of interest.

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