Electronic Journal of Qualitative Theory of Differential Equations
2026, No. 43, 1–25; https://doi.org/10.14232/ejqtde.2026.1.43 www.math.u-szeged.hu/ejqtde/

An age-structured hand, foot and mouth disease model with partial immunity

Pengmiao Hao[1], **Siwen Ren**[1], **Jianzhi Cao**[✉1] and **Buyu Wen**[2]

[1]Hebei Key Laboratory of Machine Learning and Computational Intelligence, College of Mathematics and Information Science, Hebei University, Baoding, 071002, PR China
[2]School of Information Engineering, Eastern Liaoning University, Dandong, 118001, PR China

Received 25 March 2026, appeared 17 August 2026
Communicated by Michal Fečkan

Abstract. Hand-foot-mouth disease (HFMD) is an infectious disease caused by enteroviruses common in everyone and reinfection can occur. In order to investigate the impact of covert infection and temporary immunity on the transmission of HFMD, we establish an age-structured model, which is based on a system of partial differential equations, describing the interactions among susceptible individuals, latently infected individuals, and temporarily immune individuals. At first, a careful analysis of the existence and local stability of equilibrium states shows the disease-free equilibrium is globally asymptotically stable for $R_0 < 1$, the endemic equilibrium is unstable for $R_0 > 1$, by means of reformulating the model as an abstract Cauchy problem. Then, an application of the Lyapunov–Schmidt approach reveals the conditions for the occurrence of backward bifurcation, and the numerical simulations of obtained conclusions are examined.

Keywords: hand-foot-mouth disease model, Lyapunov–Schmidt approach, backward bifurcation, age structure.

2020 Mathematics Subject Classification: 35B32, 35Q92, 35B40, 92D30.

1 Introduction

Hand-foot-mouth disease disease is a common infectious disease, which is common in children, but adults can also be infected [1]. It can be caused by more than 20 types of viruses in the enterovirus genus, including coxsackievirus, enterovirus 71, and echovirus, all of which are single-stranded positive-sense RNA viruses with strong transmissibility [22]. Enteroviruses spread through multiple routes, including the fecal-oral route, respiratory droplets, and contact with oral or nasal secretions, skin blister fluid, or contaminated objects [29]. After infection, individuals enter an incubation period generally lasting 2 to 5 days. Most cases are mild [8], some patients infected with EV71 may progress to severe complications and even death [18,20]. Recovery confers specific immunity against the infecting virus type, but due to the lack of cross-protection between different enterovirus types, individuals may experience

✉Corresponding author. Email: jzcao@hbu.edu.cn.

multiple episodes of HFMD caused by distinct viral strains [27]. The first case of HFMD was reported in New Zealand in 1957 along with the isolation of the coxsackievirus pathogen, and subsequent outbreaks have been documented globally [16]. Notably, mainland China has experienced high infection rates, over the past decade, HFMD has consistently ranked among the top three Class C diseases in China in both incidence and mortality, representing a major public health threat and consequently driving extensive research into its transmission dynamics [30].

Mathematical modeling serves as an effective tool for analyzing infectious disease dynamics. Most current HFMD models are based on the classical SIR compartmental model, which was originally developed by Kermack and McKendrick while studying the spread of the plague in London. This model pioneered the categorization of the population into three groups (Susceptible-Infected-Removed), laying a crucial foundation for research in infectious disease dynamics [6]. Given the severity of the HFMD outbreak in Sarawak, Malaysia, a simple deterministic model based on the SIR framework was constructed to predict the number of infections and the duration of the epidemic, representing one of the earlier applications of the SIR model to HFMD [21],

$$\begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} = \mu_0 - \mu_1 S - \beta(t) SI + \gamma R, \\ \dfrac{\mathrm{d}I}{\mathrm{d}t} = \beta(t) SI - \omega I - \mu_1 I, \\ \dfrac{\mathrm{d}R}{\mathrm{d}t} = \omega I - \mu_1 R - \gamma R. \end{cases}$$

However, the classical SIR model assumes that individuals become infectious immediately after exposure. In reality, HFMD patients undergo a latent period after infection during which they are no infectious or minimally infectious. As early as 1998, epidemiological surveillance and virological testing in children confirmed that the incubation period for HFMD caused by CVA16 ranges from 2 to 8 days [9]. The study in [10] first introduced an exposed (or latent) compartment (E) into the HFMD transmission model, moving away from the assumption of immediate infectivity and better reflecting the actual transmission dynamics of HFMD. The authors also incorporated the impact of quarantine measures (Q), constructing an SEIQR model. They systematically analyzed the conditions for the existence of disease free and endemic equilibrium points and proved the local stability of these equilibria using the characteristic equation method, thereby enhancing the accuracy and predictive capability of early epidemic dynamics modeling. The study in [3] addressed the limitations of traditional models in capturing the clinical heterogeneity of HFMD. This heterogeneity is characterized by the fact that some infected individuals exhibit typical symptoms such as fever and herpetiform lesions, while a considerable proportion remain asymptomatic yet still capable of transmitting the virus. Asymptomatic individuals (A), due to their concealed status, often evade routine prevention and control measures and can act as hidden sources of infection. To systematically reveal the impact of this pathological heterogeneity on disease transmission dynamics, the authors introduced a separate asymptomatic infected compartment, extending the model to an SIAR framework, this modification resolved prediction biases associated with traditional models. In recent years, many researchers have integrated these two compartments, leading to extensive discussion and application of the SEIAR model [19].

The above HFMD models are primarily based on ordinary differential equations (ODEs). Under this ODE framework, it is assumed that individuals within each compartment are

entirely homogeneous in their epidemiological characteristics and are not influenced by the duration of their stay in that compartment [13,24]. However, literature [25] indicates that the risk of infection among susceptible individuals within the same compartment is heterogeneous. This highlights the high age dependency of incidence. Furthermore, during the transmission of HFMD, individuals in the latent period are asymptomatic. Yet, several days after exposure, once these latent carriers become active, they begin spreading the virus within the population. This latency period may lead to a serious underestimation of HFMD transmission.

Therefore, we introduce an age variable for each compartment to account for the state of individuals within them. Here, age does not refer to the individual's chronological age, but rather to the duration since the individual entered the current compartment. It represents the number of individuals who have been in a given compartment for a specific length of time. By incorporating the duration a within each state, it becomes possible to express factors such as the infectiousness of infected individuals and the risk of activation for exposed individuals as functions of a. This approach offers greater flexibility in model distribution, allowing for dynamic tracking of the evolution of individuals within compartments and their impact on HFMD infection dynamics. This formulation adopts a partial differential equation (PDE) methodology. Recently, continuous models based on age structure have been extensively explored in numerous studies [12,14,17].

Based on the epidemiological characteristics of HFMD, such as age-related differences in incidence and heterogeneity in population exposure risks, reference [23] constructed an age-structured SEIAR infectious disease dynamics model that incorporates the effect of media publicity, as represented by the following differential equation,

$$\begin{cases} \dfrac{\mathrm{d}S(t)}{\mathrm{d}t} = b_r - d_r S(t) - \beta S(t)\big(I(t) + kA(t)\big), \\ \dfrac{\partial E(t,a)}{\partial t} + \dfrac{\partial E(t,a)}{\partial a} = -\chi(a)E(t,a), \quad t \geq 0,\ a \geq 0, \\ \dfrac{\mathrm{d}I(t)}{\mathrm{d}t} = p\displaystyle\int_0^\infty c(a)E(t,a)\,\mathrm{d}a - \big(d_r + \mu + \gamma_1\big)I(t), \quad t \geq 0, \\ \dfrac{\mathrm{d}A(t)}{\mathrm{d}t} = (1-p)\displaystyle\int_0^\infty c(a)E(t,a)\,\mathrm{d}a - \big(d_r + \gamma_2\big)A(t), \quad t \geq 0, \\ \dfrac{\mathrm{d}R(t)}{\mathrm{d}t} = \gamma_1 I(t) + \gamma_2 A(t) - d_r R(t), \quad t \geq 0, \\ E(t,0) = \beta S(t)\big(I(t) + kA(t)\big), \quad t \geq 0, \end{cases}$$

where relevant parameters can be referred to in [23]. The authors discussed the possible existence of both disease free and endemic steady states, analyzed the stability of these states, and verified the theoretical findings through numerical simulations.

Following the aforementioned discussion and expanding the model in [23], this paper develops an age-structured SEIAR model for HFMD incorporating partial immunity. An age variable is introduced into the five compartments to characterize the state of individuals within each compartment, representing the number of individuals in each compartment over a given period. Key parameters such as the infectivity of infectious individuals, the risk of progression from the exposed state, and immune status are expressed as functions of age, denoted by a. The study investigates the existence and stability of steady solutions for the model and derives the basic reproduction number R_0 as the threshold determining disease extinc-

tion or persistence. Furthermore, considering the absence of cross-immunity between virus serotypes and the possibility of repeated HFMD infections, the Lyapunov-Schmidt method [2] is employed to examine whether the temporary immunity of recovered individuals can induce backward bifurcation, as well as the conditions under which such backward bifurcation may arise.

The paper is organized as follows. We transform the model into a Cauchy problem and analyze the well-posedness of its solutions in Section 2. In Section 3, we linearize the model around the equilibria to obtain the normalized system, derive the basic reproduction number R_0, and analyze the existence and stability of the disease-free equilibrium. In Section 4, we investigate the existence conditions and number of endemic equilibria, as well as the conditions for the occurrence of backward bifurcation. Numerical simulations are presented in Section 5 to corroborate our theoretical results. Finally, a summary of our work is given in Section 6.

2 Mathematical model

In this section, we show examples how theorems, definitions, lists and formulae should be formatted. We establish an age-structured SEIAR model comprising five compartments: susceptible, exposed, symptomatically infected, asymptomatically infected, and temporarily recovered. Here, $S(t,a)$, $E(t,a)$, $I(t,a)$, $A(t,a)$ and $R(t,a)$ denote the population density of each compartment at age a and time t, respectively. The total population density satisfies

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

Assume that after infection with the virus, an individual first enters the latent period with a progression rate of σ. Subsequently, they develop symptomatic infection at an age-dependent proportion of $1 - p(a)$ and asymptomatic infection at a proportion of $p(a)$. The contact rate between individuals of different ages is described by the function $\beta(a,a')$, $\gamma_I(a)$ and $\gamma_A(a)$ are the recovery rates of symptomatic and asymptomatic infected individuals respectively. $\mu(a)$ and $b(a)$ refer to the natural mortality rate of the population and the fertility rate at age a, respectively, and recovered individuals acquire temporary immunity, then revert to the susceptible state at a rate of $\xi(a)$. The transmission dynamics of the disease are illustrated in Figure 2.1. The disease transmission dynamics are described by the following system of partial differential equations,

$$\begin{cases} \left(\dfrac{\partial}{\partial a} + \dfrac{\partial}{\partial t}\right) S(t,a) = -\lambda(t,a)S(t,a) - \mu(a)S(t,a) + \xi(a)R(t,a), \\ \left(\dfrac{\partial}{\partial a} + \dfrac{\partial}{\partial t}\right) E(t,a) = \lambda(t,a)S(t,a) - \mu(a)E(t,a) - \sigma E(t,a), \\ \left(\dfrac{\partial}{\partial a} + \dfrac{\partial}{\partial t}\right) I(t,a) = \big(1 - p(a)\big)\sigma E(t,a) - \gamma_I(a)I(t,a) - \mu(a)I(t,a), \\ \left(\dfrac{\partial}{\partial a} + \dfrac{\partial}{\partial t}\right) A(t,a) = p(a)\sigma E(t,a) - \gamma_A(a)A(t,a) - \mu(a)A(t,a), \\ \left(\dfrac{\partial}{\partial a} + \dfrac{\partial}{\partial t}\right) R(t,a) = \gamma_I(a)I(t,a) + \gamma_A(a)A(t,a) - \xi(a)R(t,a) - \mu(a)R(t,a). \end{cases} \tag{2.2}$$

The force of infection for susceptible individuals $\lambda(t,a)$ takes an integral form as

$$\lambda(t,a) = \int_0^\infty \beta(a,a_1)\frac{[\varepsilon_1 E(a_1,t) + I(a_1,t) + \eta_1 A(a_1,t)]}{N(a_1,t)}\,\mathrm{d}a_1$$

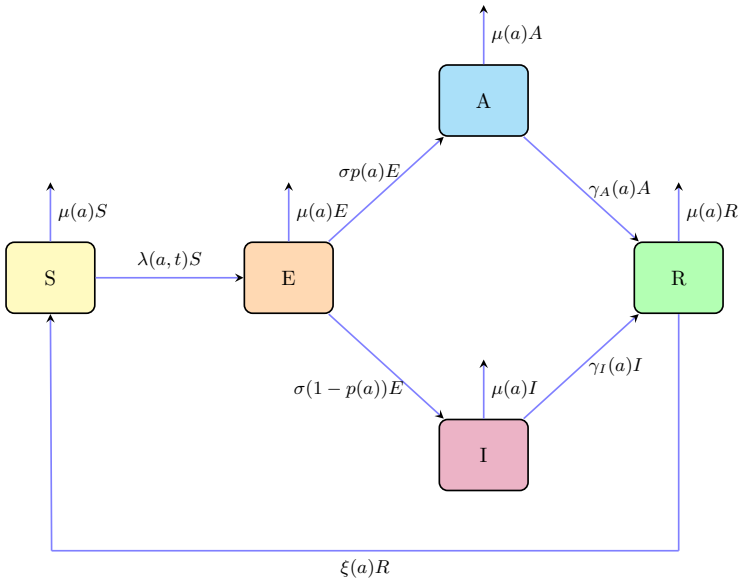

Figure 2.1: SEIAR compartment model.

with $\varepsilon_1 \in (0,1)$ and $\eta_1 \in (0,1)$ representing the infectiousness factors for latent individuals and asymptomatic infectors, respectively. The boundary conditions for system (2.2) are

$$S(0,t) = \int_0^\infty b(a)N(t,a)\,\mathrm{d}a, \quad E(0,t) = I(0,t) = A(0,t) = R(0,t) = 0.$$

Meanwhile, the initial conditions are

$$S(a,0) = S_0(a), \quad E(a,0) = E_0(a), \quad I(a,0) = I_0(a), \quad A(a,0) = A_0(a), \quad R(a,0) = R_0(a).$$

The functions and parameters in model (2.2) satisfy the following conditions:

(i) The age-dependent parameters $\beta(a)$, $\gamma_I(a)$, $\gamma_A(a)$, $\xi(a)$ are bounded and belong to $L^1_+[0,\infty)$, and $\mu(a) \in L^1_{\mathrm{loc}+}(0,\infty)$ with $\int_0^\infty \mu(a)\,\mathrm{d}a = \infty$.

(ii) The natural space for the state variables $S(\cdot,t)$, $E(\cdot,t)$, $I(\cdot,t)$, $A(\cdot,t)$, $R(\cdot,t)$ is $L^1_+[0,\infty)$.

(iii) The initial conditions satisfy $S_0(a)$, $E_0(a)$, $I_0(a)$, $A_0(a)$, $R_0(a)$ belong to $L^1_+[0,\infty)$.

Based on equations (2.1) and (2.2), $N(a,t)$ satisfies

$$\begin{aligned}\left(\frac{\partial}{\partial a} + \frac{\partial}{\partial t}\right) N(a,t) &= -\mu(a)N(a,t),\\ N(0,t) &= \int_0^\infty b(a)N(a,t)da,\\ N(a,0) &= N_0(a) = S_0(a) + E_0(a) + I_0(a) + A_0(a) + R_0(a).\end{aligned}$$

Solving the equation for $N(a,t)$ by the method of characteristics yields

$$N(a,t) = \begin{cases} N(a-t,0)\exp\left(-\int_t^a \mu(a-t+\tau)\,\mathrm{d}\tau\right) & a > t,\\ N(0,t-a)\exp\left(-\int_0^a \mu(\tau)\,\mathrm{d}\tau\right) & a \le t.\end{cases}$$

Note that $N(a,t)$ is continuous when $N_0(a) = \int_0^\infty b(a)N(a,t)$. The steady-state solution of model (2.2) can be derived as $\lim_{t\to\infty} N(a,t) = e^{-\int_0^a \mu(\tau)d\tau}\int_0^\infty b(a)N(a,t)da$. Without loss of generality, it is assumed that $N(a,t) = N_\infty(a) = e^{-\int_0^a \mu(\tau)d\tau}\int_0^\infty b(a)N(a,t)da$. For analytical simplicity, the system (2.2) is subsequently subjected to normalization. Let

$$s(a,t) = \frac{S(a,t)}{N_\infty(a)}, \quad e(a,t) = \frac{E(a,t)}{N_\infty(a)}, \quad i(a,t) = \frac{I(a,t)}{N_\infty(a)}, \quad a(a,t) = \frac{A(a,t)}{N_\infty(a)}, \quad r(a,t) = \frac{R(a,t)}{N_\infty(a)}.$$

This gives

$$\begin{cases} \left(\frac{\partial}{\partial a} + \frac{\partial}{\partial t}\right) s(t,a) = -\lambda(a,t)s(a,t) + \xi(a)r(a,t), \\ \left(\frac{\partial}{\partial a} + \frac{\partial}{\partial t}\right) e(t,a) = \lambda(a,t)s(a,t) - \sigma e(a,t), \\ \left(\frac{\partial}{\partial a} + \frac{\partial}{\partial t}\right) i(t,a) = \big(1-p(a)\big)\sigma e(a,t) - \gamma_I(a)i(a,t), \\ \left(\frac{\partial}{\partial a} + \frac{\partial}{\partial t}\right) a(t,a) = p(a)\sigma e(a,t) - \gamma_A(a)a(a,t), \\ \left(\frac{\partial}{\partial a} + \frac{\partial}{\partial t}\right) r(t,a) = \gamma_I(a)i(a,t) + \gamma_A(a)a(a,t) - \xi(a)r(a,t), \end{cases} \tag{2.3}$$

where

$$\lambda(a,t) = c(a)\int_0^\infty \beta(a')\left[\varepsilon_1 e(a',t) + i(a',t) + \eta_1 a(a',t)\right]da' = c(a)F(t),$$

and the state space is

$$X = \left\{(s,e,i,a,r) \in \left(L^1_+(0,\infty)\right)^5,\ s+e+i+a+r = 1\right\}.$$

The boundary and initial conditions satisfied by system (2.3) are given by

$$\begin{aligned} &s(0,t) = 1, \quad e(0,t) = i(0,t) = a(0,t) = r(0,t) = 0, \\ &s(a,0) = s_0(a), \quad e(a,0) = e_0(a), \quad i(a,0) = i_0(a), \quad a(a,0) = a_0(a), \quad r(a,0) = r_0(a). \end{aligned}$$

Next, system (2.3) is transformed into an abstract Cauchy problem with a non-densely defined operator. The well-posedness of the system is deduced by virtue of the integrated semigroup theory, and the existence and uniqueness of non-negative solutions are investigated. The norm of the Banach space X is defined as follows: $\|\Phi\| = \sum_{i=1}^5 \|\Phi_i\|$, $\Phi(a) = (\Phi_1(a),\Phi_2(a),\Phi_3(a),\Phi_4(a),\Phi_5(a))^T \in X$, with $\|\Phi_i\| = \int_0^\infty \Phi_i(a)da$, $\Phi_i(a) \in L^1_+(0,+\infty)$. A linear operator $A : D(A) \subset X \to X$ is given by

$$A\Phi(a) = \left(-\frac{d}{da}\Phi_1(a),\ -\frac{d}{da}\Phi_2(a),\ -\frac{d}{da}\Phi_3(a),\ -\frac{d}{da}\Phi_4(a),\ -\frac{d}{da}\Phi_5(a)\right)^T,$$

with the domain

$$D(A) = \left\{\Phi \in X \,\middle|\, \Phi_i' \in L^1(0,\infty), \quad \Phi_1(0) = 1, \quad \Phi_j(0) = 0,\ j = 2,3,4,5\right\}.$$

Define a nonlinear operator $F : X \to X$ as

$$F\Phi(a) = \begin{pmatrix} -\lambda(a,t)\Phi_1(a) + \xi(a)\Phi_5(a) \\ \lambda(a,t)\Phi_1(a) - \sigma\Phi_2(a) \\ (1-p(a))\sigma\Phi_2(a) - \gamma_I(a)\Phi_3(a) \\ p(a)\sigma\Phi_2(a) - \gamma_A(a)\Phi_4(a) \\ \gamma_I(a)\Phi_3(a) + \gamma_A(a)\Phi_4(a) - \xi(a)\Phi_5(a) \end{pmatrix}, \quad \Phi \in L^1_+(0,\infty).$$

Let $W = (s, e, i, a, r)^T \in X$, then system (2.3) can be formulated as the following Cauchy problem in X:

$$\frac{dW(t)}{dt} = AW(t) + F(W(t)), \quad W(0) = W_0 \in X, \tag{2.4}$$

where $W_0(a) = (s_0(a), e_0(a), i_0(a), a_0(a), r_0(a))$. It is known that the linear operator A is the infinitesimal generator of the C_0-semigroup $T(t)$ for $t \geq 0$, and the nonlinear operator F is continuously Fréchet differentiable on the space X. Let

$$\Omega = \left\{ (s, e, i, a, r)^T \in X \mid s, e, i, a, r \geq 0 \right\}, \quad \Omega_0 = \left\{ (s, e, i, a, r)^T \in X \mid 0 \leq s, e, i, a, r \leq 1 \right\}.$$

By similar analysis as in [5] or [7], the following conclusion can be drawn.

Proposition 2.1. *For any non-negative initial condition $W_0 \in \Omega \cap D(A)$, system ((2.4)) has a unique continuous solution, and Ω_0 is a positive invariant set of system ((2.4)).*

3 Disease-free equilibrium

This section, the existence and stability of the disease-free equilibrium of system (2.3) are mainly discussed. Let the disease-free equilibrium be $E_0 = \left(s^0(a), e^0(a), i^0(a), a^0(a), r^0(a)\right)$. By the definition of a disease-free equilibrium, $e^0(a) = i^0(a) = a^0(a) = 0$, so $\lambda(a) = 0$. From $\frac{ds^0(a)}{da} = \xi(a) r^0(a)$, $\frac{dr^0(a)}{da} = -\xi(a) r^0(a)$ and $r(0) = 0$, we obtain $r(a) = 0$. Thus, $\frac{ds^0(a)}{da} = 0$, which implies $s^0(a) = s(0) = 1$. Therefore, the disease-free equilibrium is $E_0 = (1, 0, 0, 0, 0)$. Next, the local asymptotic stability of the disease-free equilibrium is analyzed. System (2.3) is linearized about the equilibrium point E_0. Consider the exponential solutions of system (2.3)

$$s(a,t) = s(a)e^{\alpha t}, \quad e(a,t) = e(a)e^{\alpha t}, \quad i(a,t) = i(a)e^{\alpha t}, \quad a(a,t) = a(a)e^{\alpha t}, \quad r(a,t) = r(a)e^{\alpha t}. \tag{3.1}$$

Substituting the exponential solutions (3.1) into the system leads to

$$\begin{cases} s'(a) + \alpha s(a) = -\lambda(a) + \xi(a) r(a), \\ e'(a) + \alpha e(a) = \lambda(a) - \sigma e(a), \\ i'(a) + \alpha i(a) = \sigma\big(1 - p(a)\big) e(a) - \gamma_I(a) i(a), \\ a'(a) + \alpha a(a) = \sigma p(a) e(a) - \gamma_A(a) a(a), \\ r'(a) + \alpha r(a) = \gamma_I i(a) + \gamma_A a(a) - \xi(a) r(a), \end{cases} \tag{3.2}$$

$$\lambda(a) = c(a) F_0, \quad F_0 = \int_0^\infty \beta(u)\big(\varepsilon_1 e(u) + i(u) + \eta_1 a(u)\big) du.$$

Solution of the second, third and fourth equations of system ((3.2)) results in

$$\begin{aligned} e(a) &= e^{-\int_0^a (\sigma+\alpha) d\xi} \int_0^a \lambda(\tau) e^{\int_0^\tau (\sigma+\alpha) d\xi} d\tau, \\ i(a) &= e^{-\int_0^a (\gamma_I(\xi)+\alpha) d\xi} \int_0^a \sigma\big(1 - p(\eta)\big) e(\eta) e^{\int_0^\eta (\gamma_I(\xi)+\alpha) d\xi} d\eta, \\ a(a) &= e^{-\int_0^a (\gamma_A(\xi)+\alpha) d\xi} \int_0^a \sigma p(\eta) e(\eta) e^{\int_0^\eta (\gamma_A(\xi)+\alpha) d\xi} d\eta. \end{aligned}$$

Substituting $e(a)$, $i(a)$ and $a(a)$ into F_0 yields

$$\begin{aligned}F_0 = \int_0^\infty \beta(a) \Bigg[& \varepsilon_1 e^{-\int_0^a (\sigma+\alpha)d\xi} \int_0^a c(\tau) F_0 e^{\int_0^\tau (\sigma+\alpha)d\xi} d\tau \\ & + e^{-\int_0^a (\gamma_I(\xi)+\alpha)d\xi} \int_0^a \sigma(1-p(\eta)) e^{-\int_0^\eta (\sigma+\alpha)d\xi} \times \int_0^\eta c(\tau) F_0 e^{\int_0^\tau (\sigma+\alpha)d\xi} d\tau e^{\int_0^\eta (\gamma_I(\xi)+\alpha)d\xi} d\eta \\ & + \eta_1 e^{-\int_0^a (\gamma_A(\xi)+\alpha)d\xi} \int_0^a \sigma p(\eta) e^{-\int_0^\eta (\sigma+\alpha)d\xi} \times \int_0^\eta c(\tau) F_0 e^{\int_0^\tau (\sigma+\alpha)d\xi} d\tau e^{\int_0^\eta (\gamma_A(\xi)+\alpha)d\xi} d\eta \Bigg] da.\end{aligned}$$

Canceling F_0 from both sides gives

$$\begin{aligned}1 = \int_0^\infty \beta(a) \Bigg[& \varepsilon_1 e^{-\int_0^a (\sigma+\alpha)d\xi} \int_0^a c(\tau) e^{\int_0^\tau (\sigma+\alpha)d\xi} d\tau \\ & + e^{-\int_0^a (\gamma_I(\xi)+\alpha)d\xi} \int_0^a \sigma(1-p(\eta)) e^{-\int_0^\eta (\sigma+\alpha)d\xi} \times \int_0^\eta c(\tau) e^{\int_0^\tau (\sigma+\alpha)d\xi} d\tau e^{\int_0^\eta (\gamma_I(\xi)+\alpha)d\xi} d\eta \\ & + \eta_1 e^{-\int_0^a (\gamma_I(\xi)+\alpha)d\xi} \int_0^a \sigma p(\eta) e^{-\int_0^\eta (\sigma+\alpha)d\xi} \times \int_0^\eta c(\tau) e^{\int_0^\tau (\sigma+\alpha)d\xi} d\tau e^{\int_0^\eta (\gamma_I(\xi)+\alpha)d\xi} d\eta \Bigg] da.\end{aligned}$$

Setting the right-hand side equal to $H(\alpha)$, the basic reproduction number reads

$$\begin{aligned}R_0 = \int_0^\infty \beta(a) \Bigg[& \varepsilon_1 e^{-\int_0^a \sigma d\xi} \int_0^a c(\tau) e^{\int_0^\tau \sigma d\xi} d\tau \\ & + e^{-\int_0^a \gamma_I(\xi) d\xi} \int_0^a \sigma(1-p(\eta)) e^{-\int_0^\eta \sigma d\xi} \int_0^\eta c(\tau) e^{\int_0^\tau \sigma d\xi} d\tau e^{\int_0^\eta \gamma_I(\xi) d\xi} d\eta \\ & + \eta_1 e^{-\int_0^a \gamma_A(\xi) d\xi} \int_0^a \sigma p(\eta) e^{-\int_0^\eta \sigma d\xi} \int_0^\eta c(\tau) e^{\int_0^\tau \sigma d\xi} d\tau e^{\int_0^\eta \gamma_A(\xi) d\xi} d\eta \Bigg] da = H(0).\end{aligned}$$

Theorem 3.1. *If $R_0 < 1$, the disease-free equilibrium E_0 of the system is locally asymptotically stable. If $R_0 > 1$, the disease-free equilibrium E_0 is unstable.*

Proof. Differentiating $H(\alpha)$ with respect to α yields $H'(\alpha) < 0$, which implies that $H(\alpha)$ is a decreasing function with $\lim_{\alpha\to-\infty} H(\alpha) = +\infty$, $\lim_{\alpha\to+\infty} H(\alpha) = 0$. Therefore, there exists a unique real root α_0 such that $H(\alpha_0) = 1$. When $R_0 < 1$, it follows that $\alpha_0 < 0$. A complex root $l = l_1 + il_2$ with $l_2 = 0$ satisfying $H(l) = 1$ is now considered. It is shown that the real part l_1

must be negative. Assume, instead, $l_1 \geq 0$. Then

$$
\begin{aligned}
1 &= |H(l)| \\
&= \left| \int_0^\infty \beta(a) \left[\varepsilon_1 e^{-\int_0^a (\sigma+l_1+il_2)d\xi} \int_0^a c(\tau) e^{\int_0^\tau (\sigma+l_1+il_2)d\xi} d\tau \right.\right. \\
&\quad + e^{-\int_0^a (\gamma_I(\xi)+l_1+il_2)d\xi} \int_0^a \sigma(1-p(\eta)) e^{-\int_0^\eta (\sigma+l_1+il_2)d\xi} \\
&\quad\quad \times \int_0^\eta c(\tau) e^{\int_0^\tau (\sigma+l_1+il_2)d\xi} d\tau e^{\int_0^\eta (\gamma_I(\xi)+l_1+il_2)d\xi} d\eta \\
&\quad + \eta_1 e^{-\int_0^a (\gamma_A(\xi)+l_1+il_2)d\xi} \int_0^a \sigma p(\eta) e^{-\int_0^\eta (\sigma+l_1+il_2)d\xi} \\
&\quad\quad \left.\left. \times \int_0^\eta c(\tau) e^{\int_0^\tau (\sigma+l_1+il_2)d\xi} d\tau e^{\int_0^\eta (\gamma_A(\xi)+l_1+il_2)d\xi} d\eta \right] da \right| \\
&\leq \left| \int_0^\infty \int_0^a \varepsilon_1 \beta(a) e^{-\int_0^a (\sigma+l_1)d\xi} c(\tau) d\tau da \right. \\
&\quad + \int_0^\infty \int_0^a \int_0^\eta \beta(a) e^{-\int_0^a (\gamma_I(\xi)+l_1)d\xi} \sigma(1-p(\eta)) e^{-\int_0^\eta (\sigma+l_1)d\xi} c(\tau) d\tau d\eta da \\
&\quad \left. + \int_0^\infty \int_0^a \int_0^\eta \beta(a) e^{-\int_0^a (\gamma_A(\xi)+l_1)d\xi} \sigma p(\eta) e^{-\int_0^\eta (\sigma+l_1)d\xi} c(\tau) d\tau d\eta da \right| \\
&= |H(l_1)| \leq H(0) = R_0 < 1,
\end{aligned}
$$

this is a contradiction. Therefore, the complex root $l = l_1 + il_2$ has a negative real part, which implies that the disease-free equilibrium E_0 is locally asymptotically stable when $R_0 < 1$. Similarly, when $H(0) = R_0 > 1$, the characteristic equation has a positive root, and thus the disease-free equilibrium E_0 is unstable. □

Remark 3.2. This is a classic epidemiological threshold theorem, which establishes $R_0 = 1$ as the critical point determining whether the disease will break out in the population. Although our system extends from an ordinary differential equation (ODE) model to a partial differential equation model, and the R_0 here depends not only on common parameters but also on age [4], the conclusion remains consistent with that in the ODE model.

Theorem 3.3. *The disease-free equilibrium E_0 is globally asymptotically stable if* $1 + \frac{\mu(a)}{\sigma} - \frac{(1-p(a))\sigma}{\gamma_I(a)} - \frac{p(a)\sigma}{\gamma_A(a)} > 0$.

Proof. Consider the following Lyapunov functional $L(t) = \int_0^\infty \left[\frac{E(a,t)}{\sigma} + \frac{I(a,t)}{\gamma_I(a)} + \frac{A(a,t)}{\gamma_A(a)} \right] da$, $L(t) = 0$ if and only if $E(a,t) = I(a,t) = A(a,t) = 0$. Then

$$
\begin{aligned}
\frac{\mathrm{d}L}{\mathrm{d}t} &= \int_0^\infty \left[\frac{\lambda(a,t)S(a,t)}{\sigma} - E(a,t) - \frac{\mu(a)E(a,t)}{\sigma} \right. \\
&\quad \left. + \frac{(1-p(a))\sigma E(a,t)}{\gamma_I(a)} - \frac{\mu(a)I(a,t)}{\gamma_I(a)} + \frac{p(a)\sigma E(a,t)}{\gamma_A(a)} - A(a,t) - \frac{\mu(a)A(a,t)}{\gamma_A(a)} \right] \mathrm{d}a \\
&\quad - \int_0^\infty \left(\frac{1}{\sigma} \frac{\partial E(a,t)}{\partial a} + \frac{1}{\gamma_I(a)} \frac{\partial I(a,t)}{\partial a} + \frac{1}{\gamma_A(a)} \frac{\partial A(a,t)}{\partial a} \right) \mathrm{d}a.
\end{aligned}
$$

By the Fundamental Theorem of Calculus, with

$$\lim_{a\to+\infty} E(a,t)=0,\quad \lim_{a\to+\infty} I(a,t)=0,\quad \lim_{a\to+\infty} A(a,t)=0,$$
$$E(0,t)=0,\qquad I(0,t)=0,\qquad A(0,t)=0,$$

it follows that $\int_0^\infty \left[\frac{1}{\sigma}\frac{\partial E(a,t)}{\partial a}+\frac{1}{\gamma_I(a)}\frac{\partial I(a,t)}{\partial a}+\frac{1}{\gamma_A(a)}\frac{\partial A(a,t)}{\partial a}\right]da=0$. This gives

$$\frac{\mathrm{d}L(t)}{\mathrm{d}t}=\int_0^\infty\Bigg[-E(a,t)\left(1+\frac{\mu(a)}{\sigma}-\frac{(1-p(a))\sigma}{\gamma_I(a)}-\frac{p(a)\sigma}{\gamma_A(a)}\right)$$
$$-I(a,t)\left(1+\frac{\mu(a)}{\gamma_I(a)}\right)-A(a,t)\left(1+\frac{\mu(a)}{\gamma_A(a)}\right)\Bigg]\mathrm{d}a.$$

Analysis is conducted on the behavior near the disease-free equilibrium $E_0=(s^0(a),0,0,0,0)$, for which $\lambda(a,t)=0$, therefore, if $1+\frac{\mu(a)}{\sigma}-\frac{(1-p(a))\sigma}{\gamma_I(a)}-\frac{p(a)\sigma}{\gamma_A(a)}>0$, then $\frac{dL}{dt}<0$, and the disease-free equilibrium E_0 of the system is globally asymptotically stable. □

Remark 3.4. This theorem provides a stronger sufficient condition, under which the disease will eventually die out regardless of how many people are infected initially. This suggests that we can achieve the complete eradication of the disease by substantially increasing the recovery rate or reducing the proportion of latent individuals progressing to the infectious stage, without being affected by the initial scale of infection.

4 Endemic equilibrium and backward bifurcation

This section mainly discusses the conditions for the existence of the endemic equilibrium, as well as the conditions for the occurrence of backward bifurcation.

Theorem 4.1. *The system* (2.3) *possesses at least one endemic equilibrium* E^* *if the reproduction number* $R_0>1$.

Proof. At the equilibrium $E^*=(s^*(a),e^*(a),i^*(a),a^*(a),r^*(a))$, system (2.3) reduces to

$$\begin{cases}\dfrac{\mathrm{d}s^*(a)}{\mathrm{d}a}=-\lambda^*(a)s^*(a)+\xi(a)r^*(a),\\[2mm]\dfrac{\mathrm{d}e^*(a)}{\mathrm{d}a}=\lambda^*(a)s^*(a)-\sigma e^*(a),\\[2mm]\dfrac{\mathrm{d}i^*(a)}{\mathrm{d}a}=\sigma\big(1-p(a)\big)e^*(a)-\gamma_I(a)i^*(a),\\[2mm]\dfrac{\mathrm{d}a^*(a)}{\mathrm{d}a}=\sigma p(a)e^*(a)-\gamma_A(a)a^*(a),\\[2mm]\dfrac{\mathrm{d}r^*(a)}{\mathrm{d}a}=\gamma_I(a)i^*(a)+\gamma_A(a)a^*(a)-\xi(a)r^*(a),\end{cases}\tag{4.1}$$

$$\lambda^*(a)=c(a)\int_0^\infty\beta(a)\big[\varepsilon_1e^*(u)+i^*(u)+\eta_1a^*(u)\big]\mathrm{d}u=c(a)F_0,$$

and the initial conditions are $s^*(0) = 1$, $e^*(0) = i^*(0) = a^*(0) = r^*(0) = 0$. Solution of system (4.1) yields

$$\begin{aligned}
s^*(a) &= e^{-\int_0^a \lambda^*(\epsilon)\mathrm{d}\epsilon}\left[\int_0^a e^{\int_0^\eta \lambda^*(\epsilon)\mathrm{d}\epsilon}\xi(\eta)r^*(\eta)\mathrm{d}\eta + 1\right],\\
e^*(a) &= e^{-\sigma a}\int_0^a \lambda^*(\eta)s^*(\eta)e^{\sigma\eta}\mathrm{d}\eta,\\
i^*(a) &= e^{-\int_0^a \gamma_I(\epsilon)\mathrm{d}\epsilon}\int_0^a \sigma\big(1-p(\eta)\big)e^*(\eta)e^{\int_0^\eta \gamma_I(\epsilon)\mathrm{d}\epsilon}\mathrm{d}\eta,\\
a^*(a) &= e^{-\int_0^a \gamma_A(\epsilon)\mathrm{d}\epsilon}\int_0^a \sigma p(\eta)e^*(\eta)e^{\int_0^\eta \gamma_A(\epsilon)\mathrm{d}\epsilon}\mathrm{d}\eta,\\
r^*(a) &= e^{-\int_0^a \xi(\epsilon)\mathrm{d}\epsilon}\int_0^a \left(\gamma_I(\eta)i^*(\eta)+\gamma_A(\eta)a^*(\eta)\right)e^{\int_0^\eta \xi(\epsilon)\mathrm{d}\epsilon}\mathrm{d}\eta,
\end{aligned}$$

by substituting $e^*(a)$, $i^*(a)$, $a^*(a)$ and $r^*(a)$ into $s^*(a)$ gives

$$\begin{aligned}
s^*(a) = e^{-\int_0^a \lambda^*(\epsilon)d\epsilon}\Bigg[&\int_0^a e^{\int_0^\eta \lambda^*(\epsilon)d\epsilon}\xi(\eta)e^{-\int_0^\eta \xi(\epsilon)d\epsilon}\\
&\times\int_0^\eta\Bigg(\gamma_I(\tau)e^{-\int_0^\tau \gamma_I(\epsilon)d\epsilon}\int_0^\tau \sigma(1-p(s))e^{-\sigma s}\times\int_0^s \lambda^*(\theta)s^*(\theta)e^{\sigma\theta}d\theta e^{\int_0^s \gamma_I(\epsilon)d\epsilon}ds\\
&+\gamma_A(\tau)e^{-\int_0^\tau \gamma_A(\epsilon)d\epsilon}\int_0^\tau \sigma p(s)e^{-\sigma s}\times\int_0^s \lambda^*(\theta)s^*(\theta)e^{\sigma\theta}d\theta e^{\int_0^s \gamma_A(\epsilon)d\epsilon}ds\Bigg)e^{\int_0^\tau \xi(\epsilon)d\epsilon}d\tau d\eta + 1\Bigg].
\end{aligned}$$

In addition, inserting $e^*(a)$, $i^*(a)$, $a^*(a)$ and $\lambda^*(a) = c(a)F_0$ into F_0 results in

$$\begin{aligned}
F_0 &= \int_0^\infty \beta(a)\big(\varepsilon_1 e(a) + i(a) + \eta_1 a(a)\big)da\\
&= \int_0^\infty \beta(a)\big(\varepsilon_1 e^{-\sigma a}\int_0^a F_0 c(\eta)s^*(\eta)e^{\sigma\eta}d\eta\\
&\quad + e^{-\int_0^a \gamma_I(\epsilon)d\epsilon}\int_0^a \sigma(1-p(\eta))e^{-\sigma\eta}\int_0^\eta F_0 c(\tau)s^*(\tau)e^{\sigma\tau}d\tau e^{\int_0^\eta \gamma_I(\epsilon)d\epsilon}d\eta\\
&\quad + \eta_1 e^{-\int_0^a \gamma_A(\epsilon)d\epsilon}\int_0^a \sigma p(\eta)e^{-\sigma\eta}\int_0^\eta F_0 c(\tau)s^*(\tau)e^{\sigma\tau}d\tau e^{\int_0^\eta \gamma_A(\epsilon)d\epsilon}d\eta\big)da.
\end{aligned}$$

Canceling F_0 from both sides of the equation leads to

$$\begin{aligned}
1 = \int_0^\infty \beta(a)\Bigg(&\varepsilon_1 e^{-\sigma a}\int_0^a c(\eta)s^*(\eta)e^{\sigma\eta}\mathrm{d}\eta\\
&+ e^{-\int_0^a \gamma_I(\epsilon)\mathrm{d}\epsilon}\int_0^a \sigma\big(1-p(\eta)\big)e^{-\sigma\eta}\int_0^\eta c(\tau)s^*(\tau)e^{\sigma\tau}\mathrm{d}\tau e^{\int_0^\eta \gamma_I(\epsilon)\mathrm{d}\epsilon}\mathrm{d}\eta\\
&+ \eta_1 e^{-\int_0^a \gamma_A(\epsilon)\mathrm{d}\epsilon}\int_0^a \sigma p(\eta)e^{-\sigma\eta}\int_0^\eta c(\tau)s^*(\tau)e^{\sigma\tau}\mathrm{d}\tau e^{\int_0^\eta \gamma_A(\epsilon)\mathrm{d}\epsilon}\mathrm{d}\eta\Bigg)\mathrm{d}a.
\end{aligned}$$

Define the right-hand side of the equality as $Q(F_0)$. Setting $F_0 = 0$, then

$$\begin{aligned}
Q(0) = \int_0^\infty \beta(a)\Bigg(&\varepsilon_1 e^{-\sigma a}\int_0^a c(\eta)e^{\sigma\eta}\mathrm{d}\eta\\
&+ e^{-\int_0^a \gamma_I(\epsilon)\mathrm{d}\epsilon}\int_0^a \sigma\big(1-p(\eta)\big)e^{-\sigma\eta}\int_0^\eta c(\tau)e^{\sigma\tau}\mathrm{d}\tau e^{\int_0^\eta \gamma_I(\epsilon)\mathrm{d}\epsilon}\mathrm{d}\eta\\
&+ \eta_1 e^{-\int_0^a \gamma_A(\epsilon)\mathrm{d}\epsilon}\int_0^a \sigma p(\eta)e^{-\sigma\eta}\int_0^\eta c(\tau)e^{\sigma\tau}\mathrm{d}\tau e^{\int_0^\eta \gamma_A(\epsilon)\mathrm{d}\epsilon}\mathrm{d}\eta\Bigg)\mathrm{d}a = R_0.
\end{aligned}$$

Moreover, $\lim_{F_0\to+\infty} Q(F_0) = 0$. Therefore, when $R_0 > 1$, the equation $Q(F_0) = 1$ has at least one positive solution, which implies that the model admits at least one endemic equilibrium. □

We now consider the case where $R_0 < 1$. Differentiating $Q(F_0)$ with respect to F_0 and setting $F_0 = 0$ gives $Q'(0) = -\int_0^a c(\epsilon)d\epsilon$. Let $Q'(0) = U$.

Theorem 4.2. *When the basic reproduction number $R_0 < 1$, if $U > 0$ and $|R_0 - 1|$ is sufficiently small, then the system (2.3) admits at least two endemic equilibria.*

Proof. Let $W(F_0, q) = q\frac{Q(F_0)}{R_0} - 1$, then $W(0,1) = \frac{Q(0)}{R_0} - 1 = 0$, $\frac{\partial W}{\partial F_0}\Big|_{(0,1)} = \frac{U}{R_0} > 0$. By the implicit function theorem, there exists a unique implicit function $F_0 = F_0(q)$ in a neighborhood of the point $(0,1)$ for $W(F_0, q)$, which satisfies

$$\frac{\partial F_0}{\partial q}\Big|_{q=1} = -\frac{R_0}{U} < 0,$$

together with $W(F_0(q), q) = 0$ and $F_0(1) = 0$. Thus, there exists $\varepsilon > 0$ such that $F_0(q) > 0$ for all $q \in (1-\delta, 1)$ and $W(F_0, q) = 0$. Let $R_0 \in \left(1-\frac{\delta}{2}, 1\right)$ and $q \in \left(1-\varepsilon, 1-\frac{\varepsilon}{2}\right)$. Then we have $F_0(q) > 0$, $\lim_{F_0\to+\infty} Q(F_0) = 0$, $Q(0) = R_0 < 1$, which implies $Q(F_0(q)) = \frac{R_0}{q} > 1$. Therefore, there exist $K_1 \in (0, F_0(q))$ and $K_2 \in (F_0(q), +\infty)$ such that $Q(K_1) = Q(K_2) = 1$. Consequently, the system (2.3) admits at least two endemic equilibria. □

Remark 4.3. It is generally believed that $R_0 < 1$ is sufficient to eliminate the disease, but Theorem 4.2 implies that merely reducing R_0 below 1 may not be enough to eradicate it. If the current infection base is large, the system may evolve to a stable endemic equilibrium. Therefore, controlling the disease may require reducing R_0 to a critical value smaller than 1, or simultaneously drastically reducing the number of current infected individuals.

Theorem 4.2 indicates that a backward bifurcation may occur when $R_0 < 1$. Now employ the Lyapunov–Schmidt method and its techniques to conduct a detailed investigation into the existence of such a backward bifurcation.

A bifurcation parameter $\beta(\tau) = \bar{\beta}\beta_0(\tau)$ is now introduced, where $\beta_0(\tau)$ satisfies

$$\begin{aligned}1 = \int_0^\infty \beta_0(a)\Bigg[&\varepsilon_1 e^{-\int_0^a \sigma \mathrm{d}\xi}\int_0^a c(\tau)e^{\int_0^\tau \sigma \mathrm{d}\xi}\mathrm{d}\tau\\ &+ e^{-\int_0^a(\gamma_I(\xi)+\alpha)\mathrm{d}\xi}\int_0^a \sigma\big(1-p(\eta)\big)e^{-\int_0^\eta(\sigma+\alpha)\mathrm{d}\xi}\int_0^\eta c(\tau)e^{\int_0^\tau \sigma \mathrm{d}\xi}\mathrm{d}\tau e^{\int_0^\eta \gamma_I(\xi)\mathrm{d}\xi}\mathrm{d}\eta\\ &+ \eta_1 e^{-\int_0^a \gamma_A(\xi)\mathrm{d}\xi}\int_0^a \sigma p(\eta)e^{-\int_0^\eta \sigma \mathrm{d}\xi}\int_0^\eta c(\tau)e^{\int_0^\tau \sigma \mathrm{d}\xi}\mathrm{d}\tau e^{\int_0^\eta \gamma_A(\xi)\mathrm{d}\xi}\mathrm{d}\eta\Bigg]\mathrm{d}a.\end{aligned}$$

It follows that $R_0 = 1$ when $\bar{\beta} = 1$. Next, the following spaces are defined:

$$\begin{aligned}X &= L^1_{(0,\infty)} \times \mathbb{R} \times L^1_{(0,\infty)} \times \mathbb{R} \times L^1_{(0,\infty)} \times \mathbb{R} \times L^1_{(0,\infty)} \times \mathbb{R} \times L^1_{(0,\infty)} \times \mathbb{R},\\ X_0 &= L^1_{(0,\infty)} \times \{0\} \times L^1_{(0,\infty)} \times \{0\} \times L^1_{(0,\infty)} \times \{0\} \times L^1_{(0,\infty)} \times \{0\} \times L^1_{(0,\infty)} \times \{0\},\\ X_+ &= L^1_{+(0,\infty)} \times \mathbb{R}_+ \times L^1_{+(0,\infty)} \times \mathbb{R}_+ \times L^1_{+(0,\infty)} \times \mathbb{R}_+ \times L^1_{+(0,\infty)} \times \mathbb{R}_+ \times L^1_{+(0,\infty)} \times \mathbb{R}_+,\\ X_{0+} &= X_0 \cap X_+, \quad \Phi = (s, 0, e, 0, i, 0, a, 0, r, 0)^T \in X_0.\end{aligned}$$

A nonlinear operator $D : X_{0+} \to X_{0+}$ takes the form of

$$D(\Phi, \bar{\beta}) = \begin{pmatrix} -s_a - \bar{\beta}c(a)\int_0^\infty \beta_0(u)\big[\varepsilon_1 e(u) + i(u) + \eta_1 a(u)\big]\mathrm{d}u\, s(a) + \xi(a)r(a) \\ 1 - s(0) \\ -e_a + \bar{\beta}c(a)\int_0^\infty \beta_0(u)\big[\varepsilon_1 e(u) + i(u) + \eta_1 a(u)\big]\mathrm{d}u\, s(a) - \sigma e(a) \\ -e(0) \\ -i_a + \big(1 - p(a)\big)\sigma e(a) - \gamma_I(a)i(a) \\ -i(0) \\ -a_a + p(a)\sigma e(a) - \gamma_A(a)a(a) \\ -a(0) \\ -r_a + \gamma_I(a)i(a) + \gamma_A(a)a(a) - \xi(a)r(a) \\ -r(0) \end{pmatrix}.$$

The derivative of the operator D is evaluated on an arbitrary element

$$h = (x, 0, y, 0, z, 0, k, 0, l, 0)^T \in X_{0+}.$$

This gives

$$D_\Phi(\Phi, \bar{\beta})h = \begin{pmatrix} -x_a - F_1 + \xi(a)l(a) \\ -x(0) \\ -y_a + F_1 - \sigma y(a) \\ -y(0) \\ -z_a + \big(1 - p(a)\big)\sigma y(a) - \gamma_I(a)z(a) \\ -z(0) \\ -k_a + p(a)\sigma y(a) - \gamma_A(a)k(a) \\ -k(0) \\ -l_a + \gamma_I(a)z(a) + \gamma_A(a)k(a) - \xi(a)l(a) \\ -l(0) \end{pmatrix},$$

where

$$\begin{aligned} F_1 = {} & \bar{\beta}c(a)\int_0^\infty \beta_0(u)\big[\varepsilon_1 e(u) + i(u) + \eta_1 a(u)\big]\mathrm{d}u\, x(a) \\ & + \bar{\beta}c(a)\int_0^\infty \beta_0(u)\big[\varepsilon_1 y(u) + z(u) + \eta_1 k(u)\big]\mathrm{d}u\, s(a). \end{aligned}$$

Compute the derivative of the operator D at the disease-free equilibrium $E_0 = (1, 0, 0, 0, 0)^T$, let $\Phi^0 = (1, 0, 0, 0, 0, 0, 0, 0, 0, 0)^T$, it follows that

$$D_\Phi(\Phi^0, \bar{\beta})h = \begin{pmatrix} -x_a - \bar{\beta}c(a)\int_0^\infty \beta_0(u)\big[\varepsilon_1 y(u) + z(u) + \eta_1 k(u)\big]\mathrm{d}u + \xi(a)l(a) \\ -x(0) \\ -y_a + \bar{\beta}c(a)\int_0^\infty \beta_0(u)\big[\varepsilon_1 y(u) + z(u) + \eta_1 k(u)\big]\mathrm{d}u - \sigma y(a) \\ -y(0) \\ -z_a + \big(1 - p(a)\big)\sigma y(a) - \gamma_I(a)z(a) \\ -z(0) \\ -k_a + p(a)\sigma y(a) - \gamma_A(a)k(a) \\ -k(0) \\ -l_a + \gamma_I(a)z(a) + \gamma_A(a)k(a) - \xi(a)l(a) \\ -l(0) \end{pmatrix}.$$

Let $\mathcal{A}$ denote the linear operator $D_\Phi(\Phi^0, 1)$. For the sake of simplicity in calculation, let

$M = \int_0^\infty \beta_0(u)\big[\varepsilon_1 y(u) + z(u) + \eta_1 k(u)\big]du$. Solving the system of equations $\mathcal{A}h = 0$ yields

$$
\begin{aligned}
x(a) = \int_0^a \Bigg(& -Mc(\omega) + \xi(\omega) e^{-\int_0^\omega \xi(\epsilon)\mathrm{d}\epsilon} \\
& \times \int_0^\omega e^{-\int_0^\mu \xi(\epsilon)\mathrm{d}\epsilon} \Bigg[\gamma_I(\mu) M \int_0^\mu e^{-\int_\tau^\mu r_I(\epsilon)\mathrm{d}\epsilon} \sigma\big(1-p(\tau)\big) \times \int_0^\tau e^{-\int_\theta^\tau \sigma \mathrm{d}\eta} c(\theta)\mathrm{d}\theta\mathrm{d}\tau \\
& + \gamma_A(\mu) M \int_0^\mu e^{-\int_\tau^\mu r_A(\epsilon)\mathrm{d}\epsilon} \sigma p(\tau) \times \int_0^\tau e^{-\int_\theta^\tau \sigma \mathrm{d}\eta} c(\theta)\mathrm{d}\theta\mathrm{d}\tau \Bigg] \mathrm{d}\mu \Bigg) \mathrm{d}\omega, \\
y(a) = {} & M \int_0^a e^{-\int_\theta^a \sigma \mathrm{d}\eta} c(\theta)\mathrm{d}\theta, \\
z(a) = {} & M \int_0^a e^{-\int_\tau^a r_I(\epsilon)\mathrm{d}\epsilon} \sigma\big(1-p(\tau)\big) \int_0^\tau e^{-\int_\theta^\tau \sigma \mathrm{d}\eta} c(\theta)\mathrm{d}\theta\mathrm{d}\tau, \\
k(a) = {} & M \int_0^a e^{-\int_\tau^a r_A(\epsilon)\mathrm{d}\epsilon} \sigma p(\tau) \int_0^\tau e^{-\int_\theta^\tau \sigma \mathrm{d}\eta} c(\theta)\mathrm{d}\theta\mathrm{d}\tau, \\
l(a) = {} & e^{-\int_0^a \xi(\epsilon)\mathrm{d}\epsilon} \int_0^a e^{-\int_0^\mu \xi(\epsilon)\mathrm{d}\epsilon} \Bigg[\gamma_I(\mu) M \int_0^\mu e^{-\int_\tau^\mu r_I(\epsilon)\mathrm{d}\epsilon} \sigma\big(1-p(\tau)\big) \times \int_0^\tau e^{-\int_\theta^\tau \sigma \mathrm{d}\eta} c(\theta)\mathrm{d}\theta\mathrm{d}\tau \\
& + \gamma_A(\mu) M \int_0^\mu e^{-\int_\tau^\mu r_A(\epsilon)\mathrm{d}\epsilon} \sigma p(\tau) \times \int_0^\tau e^{-\int_\theta^\tau \sigma \mathrm{d}\eta} c(\theta)\mathrm{d}\theta\mathrm{d}\tau \Bigg] \mathrm{d}\mu.
\end{aligned}
$$

Further, the following definitions are introduced:

$$
\begin{aligned}
\hat{x}_0(a) = \int_0^a \Bigg[& -c(\omega) + \xi(\omega) e^{-\int_0^\omega \xi(\varepsilon)\mathrm{d}\varepsilon} \int_0^\omega e^{\int_0^\mu \xi(\varepsilon)\mathrm{d}\varepsilon} \\
& \times \Bigg(\gamma_I(\mu) \int_0^\mu e^{-\int_\tau^\mu r_I(\varepsilon)\mathrm{d}\varepsilon} \sigma\big(1-p(\tau)\big) \int_0^\tau e^{-\int_\theta^\tau \sigma \mathrm{d}\eta} c(\theta)\mathrm{d}\theta\mathrm{d}\tau \\
& + \gamma_A(\mu) \int_0^\mu e^{-\int_\tau^\mu r_A(\varepsilon)\mathrm{d}\varepsilon} \sigma p(\tau) \int_0^\tau e^{-\int_\theta^\tau \sigma \mathrm{d}\eta} c(\theta)\mathrm{d}\theta\mathrm{d}\tau \Bigg) \mathrm{d}\mu \Bigg] \mathrm{d}\omega, \\
\hat{y}_0(a) = {} & \int_0^a e^{-\int_\theta^a \sigma \mathrm{d}\eta} c(\theta)\mathrm{d}\theta, \\
\hat{z}_0(a) = {} & \int_0^a e^{-\int_\tau^a r_I(\epsilon)\mathrm{d}\epsilon} \sigma\big(1-p(\tau)\big) \int_0^\tau e^{-\int_\theta^\tau \sigma \mathrm{d}\eta} c(\theta)\mathrm{d}\theta\mathrm{d}\tau, \\
\hat{k}_0(a) = {} & \int_0^a e^{-\int_\tau^a r_A(\epsilon)\mathrm{d}\epsilon} \sigma p(\tau) \int_0^\tau e^{-\int_\theta^\tau \sigma \mathrm{d}\eta} c(\theta)\mathrm{d}\theta\mathrm{d}\tau, \\
\hat{l}_0(a) = {} & e^{-\int_0^a \xi(\varepsilon)\mathrm{d}\varepsilon} \int_0^a e^{\int_0^\mu \xi(\varepsilon)\mathrm{d}\varepsilon} \Bigg(\gamma_I(\mu) \int_0^\mu e^{-\int_\tau^\mu r_I(\varepsilon)\mathrm{d}\varepsilon} \sigma\big(1-p(\tau)\big) \int_0^\tau e^{-\int_\theta^\tau \sigma \mathrm{d}\eta} c(\theta)\mathrm{d}\theta\mathrm{d}\tau \\
& + \gamma_A(\mu) \int_0^\mu e^{-\int_\tau^\mu r_A(\varepsilon)\mathrm{d}\varepsilon} \sigma p(\tau) \int_0^\tau e^{-\int_\theta^\tau \sigma \mathrm{d}\eta} c(\theta)\mathrm{d}\theta\mathrm{d}\tau \Bigg) \mathrm{d}\mu.
\end{aligned}
$$

Substitution of $y(a)$, $z(a)$ and $k(a)$ into M yields

$$
\begin{aligned}
M &= \int_0^\infty \beta_0(a) \Big[\varepsilon_1 y(a) + z(a) + \eta_1 k(a) \Big] \mathrm{d}a \\
&= M \int_0^\infty \beta_0(a) \Bigg[\varepsilon_1 \int_0^a e^{-\int_\theta^a \sigma \mathrm{d}\eta} c(\theta)\mathrm{d}\theta + \int_0^a e^{-\int_\tau^a r_I(\varepsilon)\mathrm{d}\varepsilon} \sigma\big(1-p(\tau)\big) \int_0^\tau e^{-\int_\theta^\tau \sigma \mathrm{d}\eta} c(\theta)\mathrm{d}\theta\mathrm{d}\tau \\
&\quad + \eta_1 \int_0^a e^{-\int_\tau^a r_A(\varepsilon)\mathrm{d}\varepsilon} \sigma p(\tau) \int_0^\tau e^{-\int_\theta^\tau \sigma \mathrm{d}\eta} c(\theta)\mathrm{d}\theta\mathrm{d}\tau \Bigg] \mathrm{d}a.
\end{aligned}
$$

If $M \neq 0$, then

$$1 = \int_0^\infty \beta_0(a)\left[\varepsilon_1 \hat{y}_0(a) + \hat{z}_0(a) + \eta_1 \hat{k}_0(a)\right] da. \tag{4.2}$$

Therefore, the linear operator $\mathcal{A}$ has an eigenvector given by

$$\hat{v}_0 = \left(\hat{x}_0(a), 0, \hat{y}_0(a), 0, \hat{z}_0(a), 0, \hat{k}_0(a), 0, \hat{l}_0(a), 0\right)^{\mathrm{T}}.$$

To compute the adjoint operator $\mathcal{A}^*$, let $\psi = (\psi_1, \psi_2, \psi_3, \psi_4, \psi_5, \psi_6, \psi_7, \psi_8, \psi_9, \psi_{10})^{\mathrm{T}}$. From $\langle \mathcal{A}h, \psi \rangle = \langle h, \mathcal{A}^*\psi \rangle$, expanding the inner product gives

$$\begin{aligned}
\langle \mathcal{A}h, \psi \rangle = & \int_0^\infty \left[-x_a - c(a)M + \xi(a)l(a)\right]\psi_1(a)\mathrm{d}a - \psi_2 x(0) \\
& + \int_0^\infty \left[-y_a + c(a)M - \sigma y(a)\right]\psi_3(a)\mathrm{d}a - \psi_4 y(0) \\
& + \int_0^\infty \left[-z_a + \sigma\big(1 - p(a)\big)y(a) - \gamma_I(a)z(a)\right]\psi_5(a)\mathrm{d}a - \psi_6 z(0) \\
& + \int_0^\infty \left[-k_a + \sigma p(a)y(a) - \gamma_A(a)k(a)\right]\psi_7(a)\mathrm{d}a - \psi_8 k(0) \\
& + \int_0^\infty \left[-l_a + \gamma_I(a)z(a) + \gamma_A(a)k(a) - \xi(a)l(a)\right]\psi_9(a)\mathrm{d}a - \psi_{10} l(0) \\
= & \langle h, \mathcal{A}^*\psi \rangle.
\end{aligned}$$

Integrating by parts for the differential equations and assuming $\psi(\infty) = 0$, the adjoint operator is as follows:

$$\mathcal{A}^*\psi = \begin{pmatrix}
\psi_{1a} \\
\psi_1(0) \\
\psi_{3a} - \varepsilon_1\beta_0(a)\int_0^\infty c(u)\psi_1(u)\mathrm{d}u \\
+\varepsilon_1\beta_0(a)\int_0^\infty c(u)\psi_3(u)\mathrm{d}u - \sigma\psi_3(a) \\
+\sigma\big(1 - p(a)\big)\psi_5(a) + \sigma p(a)\psi_7(a) \\
\psi_3(0) \\
\psi_{5a} - \beta_0(a)\int_0^\infty c(u)\psi_1(u)\mathrm{d}u \\
+\beta_0(a)\int_0^\infty c(u)\psi_3(u)\mathrm{d}u - \gamma_I(a)\psi_5(a) + \gamma_I(a)\psi_9(a) \\
\psi_5(0) \\
\psi_{7a} - \eta_1\beta_0(a)\int_0^\infty c(u)\psi_1(u)\mathrm{d}u \\
+\eta_1\beta_0(a)\int_0^\infty c(u)\psi_3(u)\mathrm{d}u - \gamma_A(a)\psi_7(a) + \gamma_A(a)\psi_9(a) \\
\psi_7(0) \\
\psi_{9a} + \xi(a)\psi_1(a) - \xi(a)\psi_9(a) \\
\psi_9(0)
\end{pmatrix}.$$

We then calculate the eigenvector of the adjoint operator $\mathcal{A}^*$ associated with the eigenvalue 0. Setting $\mathcal{A}^*\psi = 0$ leads to

$$\begin{aligned}
\psi_1(a) &= \psi_1(0) = 0, \\
\psi_9(a) &= \psi_9(0)e^{\int_0^a \xi(\epsilon)\mathrm{d}\epsilon}, \\
\psi_5(a) &= -e^{\int_0^a r_I(\varepsilon)\mathrm{d}\varepsilon}\int_a^\infty e^{-\int_0^\tau r_I(\varepsilon)\mathrm{d}\varepsilon}\left(r_I(\tau)\psi_9(\tau) + \beta_0(\tau)\int_0^\infty c(u)\psi_3(u)\mathrm{d}u\right)\mathrm{d}\tau, \\
\psi_7(a) &= -e^{\int_0^a r_A(\varepsilon)\mathrm{d}\varepsilon}\int_a^\infty e^{-\int_0^\tau r_A(\varepsilon)\mathrm{d}\varepsilon}\left(r_A(\tau)\psi_9(\tau) + \eta_1\beta_0(\tau)\int_0^\infty c(u)\psi_3(u)\mathrm{d}u\right)\mathrm{d}\tau,
\end{aligned}$$

$$\psi_3(a) = \Sigma e^{a\sigma} \int_a^\infty e^{-\theta\sigma} \Bigg[\sigma\big(1 - p(\theta)\big) e^{\int_0^\theta r_I(\varepsilon)\mathrm{d}\varepsilon} \times \int_\theta^\infty e^{-\int_0^\tau r_I(\varepsilon)\mathrm{d}\varepsilon} \Big(r_I(\tau)\psi_9(0) e^{\int_0^\tau \xi(\varepsilon)\mathrm{d}\varepsilon} + \beta_0(\tau) \Big) \mathrm{d}\tau + \sigma p(\theta) e^{\int_0^\theta r_A(\varepsilon)\mathrm{d}\varepsilon} \times \int_\theta^\infty e^{-\int_0^\tau r_A(\varepsilon)\mathrm{d}\varepsilon} \Big(r_A(\tau)\psi_9(0) e^{\int_0^\tau \xi(\varepsilon)\mathrm{d}\varepsilon} + \eta_1\beta_0(\tau) \Big) \mathrm{d}\tau - \varepsilon_1\beta_0(\theta) \Bigg] \mathrm{d}\theta,$$

where

$$\Sigma = \int_0^\infty c(u)\psi_3(u)\,\mathrm{d}u.$$

Without loss of generality, it can be assumed that $\psi_9(0) = 0$, which implies that

$$\psi_3(a) = \Sigma e^{a\sigma} \int_a^\infty e^{-\theta\sigma} \Bigg[\sigma\big(1 - p(\theta)\big) e^{\int_0^\theta r_I(\varepsilon)\mathrm{d}\varepsilon} \times \int_\theta^\infty e^{-\int_0^\tau r_I(\varepsilon)\mathrm{d}\varepsilon} \Big(r_I(\tau)\psi_9(0) e^{\int_0^\tau \xi(\varepsilon)\mathrm{d}\varepsilon} + \beta_0(\tau) \Big) \mathrm{d}\tau + \sigma p(\theta) e^{\int_0^\theta r_A(\varepsilon)\mathrm{d}\varepsilon} \times \int_\theta^\infty e^{-\int_0^\tau r_A(\varepsilon)\mathrm{d}\varepsilon} \Big(r_A(\tau)\psi_9(0) e^{\int_0^\tau \xi(\varepsilon)\mathrm{d}\varepsilon} + \eta_1\beta_0(\tau) \Big) \mathrm{d}\tau - \varepsilon_1\beta_0(\theta) \Bigg] \mathrm{d}\theta.$$

Upon substituting $\psi_3(a)$ into the expression for Σ, we arrive at

$$\Sigma = \Sigma \int_0^\infty c(u) e^{u\sigma} \int_u^\infty e^{-\theta\sigma} \Bigg[\sigma\big(1 - p(\theta)\big) e^{\int_0^\theta r_I(\varepsilon)\mathrm{d}\varepsilon} \times \int_\theta^\infty e^{-\int_0^\tau r_I(\varepsilon)\mathrm{d}\varepsilon} \Big(r_I(\tau)\psi_9(0) e^{\int_0^\tau \xi(\varepsilon)\mathrm{d}\varepsilon} + \beta_0(\tau) \Big) \mathrm{d}\tau + \sigma p(\theta) e^{\int_0^\theta r_A(\varepsilon)\mathrm{d}\varepsilon} \times \int_\theta^\infty e^{-\int_0^\tau r_A(\varepsilon)\mathrm{d}\varepsilon} \Big(r_A(\tau)\psi_9(0) e^{\int_0^\tau \xi(\varepsilon)\mathrm{d}\varepsilon} + \eta_1\beta_0(\tau) \Big) \mathrm{d}\tau - \varepsilon_1\beta_0(\theta) \Bigg] \mathrm{d}\theta\,\mathrm{d}u.$$

Let

$$\psi_3^*(a) = e^{a\sigma} \int_a^\infty e^{-\theta\sigma} \Bigg[\sigma\big(1 - p(\theta)\big) e^{\int_0^\theta r_I(\varepsilon)\mathrm{d}\varepsilon} \times \int_\theta^\infty e^{-\int_0^\tau r_I(\varepsilon)\mathrm{d}\varepsilon} \Big(r_I(\tau)\psi_9(0) e^{\int_0^\tau \xi(\varepsilon)\mathrm{d}\varepsilon} + \beta_0(\tau) \Big) \mathrm{d}\tau + \sigma p(\theta) e^{\int_0^\theta r_A(\varepsilon)\mathrm{d}\varepsilon} \times \int_\theta^\infty e^{-\int_0^\tau r_A(\varepsilon)\mathrm{d}\varepsilon} \Big(r_A(\tau)\psi_9(0) e^{\int_0^\tau \xi(\varepsilon)\mathrm{d}\varepsilon} + \eta_1\beta_0(\tau) \Big) \mathrm{d}\tau - \varepsilon_1\beta_0(\theta) \Bigg] \mathrm{d}\theta.$$

It follows that

$$1 = \int_0^\infty c(u)\psi_3^*(u)du. \tag{4.3}$$

Substituting $\psi_3^*(a)$ into $\psi_5(a)$ and $\psi_7(a)$ gives

$$\psi_5^*(a) = -e^{\int_0^a r_I(\varepsilon)\mathrm{d}\varepsilon} \int_a^\infty e^{-\int_0^\tau r_I(\varepsilon)\mathrm{d}\varepsilon} \beta_0(\tau)\,\mathrm{d}\tau,$$

$$\psi_7^*(a) = -e^{\int_0^a r_A(\varepsilon)\mathrm{d}\varepsilon} \int_a^\infty e^{-\int_0^\tau r_A(\varepsilon)\mathrm{d}\varepsilon} \eta_1\beta_0(\tau)\,\mathrm{d}\tau.$$

Therefore, an eigenvector of $\mathcal{A}^*$ corresponding to the eigenvalue 0 is given by

$$v_0^* = \big(0, 0, \psi_3^*(a), \psi_3^*(0), \psi_5^*(a), \psi_5^*(0), \psi_7^*(a), \psi_7^*(0), 0, 0\big).$$

We next compute the mixed partial derivative

$$D_{\Phi\bar{\beta}}(\Phi, \bar{\beta})h = \begin{pmatrix} -c(a)\int_0^\infty \beta_0(u)\big[\varepsilon_1 e(u) + i(u) + \eta_1 a(u)\big]\mathrm{d}u\, x(a) \\ -c(a)\int_0^\infty \beta_0(u)\big[\varepsilon_1 y(u) + z(u) + \eta_1 k(u)\big]\mathrm{d}u\, s(a) \\ 0 \\ c(a)\int_0^\infty \beta_0(u)\big[\varepsilon_1 e(u) + i(u) + \eta_1 a(u)\big]\mathrm{d}u\, x(a) \\ c(a)\int_0^\infty \beta_0(u)\big[\varepsilon_1 y(u) + z(u) + \eta_1 k(u)\big]\mathrm{d}u\, s(a) \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix},$$

and define

$$\begin{aligned} b &= \langle D_{\Phi\bar{\beta}}(\Phi^0, 1)\hat{v}_0, \hat{v}_0^*\rangle \\ &= \int_0^\infty c(a)\bigg(\int_0^\infty \beta_0(u)\big[\varepsilon_1 \hat{y}_0(u) + \hat{z}_0(u) + \eta_1 \hat{k}_0(u)\big]\mathrm{d}u\bigg)\psi_3^*(a)\,\mathrm{d}a. \end{aligned}$$

Moreover, based on the identities (4.2) and (4.3), it can conclude that

$$b = \int_0^\infty c(a)\,\mathrm{d}a > 0.$$

Let $h_2 = (x_2, 0, y_2, 0, z_2, 0, k_2, 0, l_2, 0)$, it can be shown that

$$D_{\Phi\Phi}(\Phi^0, \bar{\beta})[h, h_2] = \begin{pmatrix} -\bar{\beta}c(a)\int_0^\infty \big[\varepsilon_1 y(u) + z(u) + \eta_1 k(u)\big]\mathrm{d}u\, x_2(a) \\ -\bar{\beta}c(a)\int_0^\infty \big[\varepsilon_1 y_2(u) + z_2(u) + \eta_1 k_2(u)\big]\mathrm{d}u\, x(a) \\ 0 \\ \bar{\beta}c(a)\int_0^\infty \big[\varepsilon_1 y(u) + z(u) + \eta_1 k(u)\big]\mathrm{d}u\, x_2(a) \\ \bar{\beta}c(a)\int_0^\infty \big[\varepsilon_1 y_2(u) + z_2(u) + \eta_1 k_2(u)\big]\mathrm{d}u\, x(a) \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}.$$

Set a to be

$$\begin{aligned} a &= \langle D_{\Phi\Phi}(\Phi^0, 1)[\hat{v}_0, \hat{v}_0^*], \hat{v}_0^*\rangle \\ &= 2\int_0^\infty \hat{x}_0(a)c(a)\int_0^\infty \Big[\varepsilon_1 \hat{y}_0(u) + \hat{z}_0(u) + \eta_1 \hat{k}_0(u)\Big]\mathrm{d}u\ \psi_3^*(a)\,\mathrm{d}a \\ &= 2\int_0^\infty \hat{x}_0(a)c(a)\psi_3^*(a)\,\mathrm{d}a. \end{aligned}$$

Lemma 4.4 ([26, Theorem 2.1]). *Assume*

(i) $\mathcal{A}$ *is a closed operator with a simple isolated eigenvalue zero and the remaining eigenvalues having negative real parts.* $\hat{v}_0$ *is the unique solution of* $\mathcal{A}v = 0$.

(ii) $D(\Phi, \bar{\beta}) \in C^2(U_0 \times I_0, X)$ *for some neighborhood* U_0 *of* Φ^0 *and an interval* I_0 *containing* 1.

(iii) $\hat{v}_0^* \in X^*$ *is the unique positive solution of* $\langle \mathcal{A}\phi, \hat{v}_0^* \rangle = 0$, *which implies* $\dim(\ker \mathcal{A}^*) = 1$, *where* $\ker \mathcal{A}^* = \operatorname{span}\{\hat{v}_0^*\}$.

(iv) $\langle D_{\Phi\bar{\beta}}(\Phi^0, 1)\hat{v}_0, \hat{v}_0^* \rangle \neq 0$, *where* $D_{\Phi\bar{\beta}}(\Phi^0, 1)$ *is the mixed partial derivative of* D *with respect to* Φ *and* $\bar{\beta}$.

Then, if $b > 0$, *the bifurcation is backward if and only if* $a > 0$ *and forward if and only if* $a < 0$.

Theorem 4.5. *System (2.3) exhibits a backward bifurcation when* $R_0 < 1$ *and the following conditions are satisfied:*

$$\begin{aligned}
&\int_0^\infty \int_0^a \Bigg[-c(\omega) + \xi(\omega) e^{-\int_0^\omega \xi(\varepsilon)\mathrm{d}\varepsilon} \int_0^\omega e^{\int_0^\mu \xi(\varepsilon)\mathrm{d}\varepsilon} \\
&\quad \times \Bigg(\gamma_I(\mu) \int_0^\mu e^{-\int_t^\mu r_I(\varepsilon)\mathrm{d}\varepsilon} \sigma\big(1 - p(t)\big) \int_0^t e^{-\int_v^t \sigma \mathrm{d}\eta} c(v)\,\mathrm{d}v\,\mathrm{d}t + \gamma_A(\mu) \int_0^\mu e^{-\int_t^\mu r_A(\varepsilon)\mathrm{d}\varepsilon} \sigma p(t) \\
&\quad \times \int_0^t e^{-\int_v^t \sigma \mathrm{d}\eta} c(v)\,\mathrm{d}v\,\mathrm{d}t \Bigg) \mathrm{d}\mu \Bigg] \mathrm{d}\omega \times c(a) e^{a\sigma} \int_a^\infty e^{-\theta\sigma} \Bigg[\sigma\big(1 - p(\theta)\big) e^{\int_0^\theta r_I(\varepsilon)\mathrm{d}\varepsilon} \\
&\quad \times \int_\theta^\infty e^{-\int_0^\tau r_I(\varepsilon)\mathrm{d}\varepsilon} \Big(r_I(\tau)\psi_9(0) e^{\int_0^\tau \xi(\varepsilon)\mathrm{d}\varepsilon} + \beta_0(\tau) \Big) \mathrm{d}\tau + \sigma p(\theta) e^{\int_0^\theta r_A(\varepsilon)\mathrm{d}\varepsilon} \\
&\quad \times \int_\theta^\infty e^{-\int_0^\tau r_A(\varepsilon)\mathrm{d}\varepsilon} \Big(r_A(\tau)\psi_9(0) e^{\int_0^\tau \xi(\varepsilon)\mathrm{d}\varepsilon} + \eta_1 \beta_0(\tau) \Big) \mathrm{d}\tau - \varepsilon_1 \beta_0(\theta) \Bigg] \mathrm{d}\theta\,\mathrm{d}a > 0.
\end{aligned}$$

Proof. Substituting $\psi_3^*(a)$ into the expression for a leads to

$$\begin{aligned}
a &= 2 \int_0^\infty \hat{x}_0(a) c(a) e^{a\sigma} \int_a^\infty e^{-\theta\sigma} \\
&\quad \times \Bigg[\sigma\big(1 - p(\theta)\big) e^{\int_0^\theta r_I(\varepsilon)\mathrm{d}\varepsilon} \int_\theta^\infty e^{-\int_0^\tau r_I(\varepsilon)\mathrm{d}\varepsilon} \Big(r_I(\tau)\psi_9(0) e^{\int_0^\tau \xi(\varepsilon)\mathrm{d}\varepsilon} + \beta_0(\tau) \Big) \mathrm{d}\tau \\
&\quad + \sigma p(\theta) e^{\int_0^\theta r_A(\varepsilon)\mathrm{d}\varepsilon} \int_\theta^\infty e^{-\int_0^\tau r_A(\varepsilon)\mathrm{d}\varepsilon} \Big(r_A(\tau)\psi_9(0) e^{\int_0^\tau \xi(\varepsilon)\mathrm{d}\varepsilon} + \eta_1 \beta_0(\tau) \Big) \mathrm{d}\tau - \varepsilon_1 \beta_0(\theta) \Bigg] \mathrm{d}\theta\,\mathrm{d}a \\
&= 2 \int_0^\infty \int_0^a \Bigg[-c(\omega) + \xi(\omega) e^{-\int_0^\omega \xi(\varepsilon)\mathrm{d}\varepsilon} \int_0^\omega e^{\int_0^\mu \xi(\varepsilon)\mathrm{d}\varepsilon} \\
&\quad \times \Bigg(\gamma_I(\mu) \int_0^\mu e^{-\int_t^\mu r_I(\varepsilon)\mathrm{d}\varepsilon} \sigma\big(1 - p(t)\big) \int_0^t e^{-\int_v^t \sigma \mathrm{d}\eta} c(v)\,\mathrm{d}v\,\mathrm{d}t \\
&\quad + \gamma_A(\mu) \int_0^\mu e^{-\int_t^\mu r_A(\varepsilon)\mathrm{d}\varepsilon} \sigma p(t) \int_0^t e^{-\int_v^t \sigma \mathrm{d}\eta} c(v)\,\mathrm{d}v\,\mathrm{d}t \Bigg) \mathrm{d}\mu \Bigg] \mathrm{d}\omega\, c(a) e^{a\sigma} \int_a^\infty e^{-\theta\sigma} \\
&\quad \times \Bigg[\sigma\big(1 - p(\theta)\big) e^{\int_0^\theta r_I(\varepsilon)\mathrm{d}\varepsilon} \int_\theta^\infty e^{-\int_0^\tau r_I(\varepsilon)\mathrm{d}\varepsilon} \Big(r_I(\tau)\psi_9(0) e^{\int_0^\tau \xi(\varepsilon)\mathrm{d}\varepsilon} + \beta_0(\tau) \Big) \mathrm{d}\tau \\
&\quad + \sigma p(\theta) e^{\int_0^\theta r_A(\varepsilon)\mathrm{d}\varepsilon} \int_\theta^\infty e^{-\int_0^\tau r_A(\varepsilon)\mathrm{d}\varepsilon} \Big(r_A(\tau)\psi_9(0) e^{\int_0^\tau \xi(\varepsilon)\mathrm{d}\varepsilon} + \eta_1 \beta_0(\tau) \Big) \mathrm{d}\tau - \varepsilon_1 \beta_0(\theta) \Bigg] \mathrm{d}\theta\,\mathrm{d}a.
\end{aligned}$$

Hence, if the conditions hold, then $a > 0$. From Lemma 4.4, system exhibits a backward bifurcation. □

Remark 4.6. The conditions for backward bifurcation in non-age-structured infectious disease models usually only depend on global parameters (such as recovery rate and transmissibility); in contrast, the backward bifurcation conditions in this model include age-dependent integral terms and the age heterogeneity of the asymptomatic proportion $p(a)$. The introduction of age structure makes the occurrence of backward bifurcation more dependent on the dynamic coupling of different age groups rather than a single global parameter [10].

5 Numerical simulations

In this section, numerical simulations are conducted to verify the main conclusions drawn above. First, the stability of the equilibrium point of the system is verified, and the effects of age structure and partial immunity on the prevention and control of hand, foot, and mouth disease are analyzed. Second, numerical simulation and analysis are performed on the backward bifurcation of the system. According to the parameter settings in [11], we take $\sigma = 0.25$, $p(a) = 0.6$. The recovery rates $\gamma_I(a)$ and $\gamma_A(a)$ of symptomatic and asymptomatic infected individuals, which were given different values in [15,28,31], in general, are fixed as $\gamma_I(a) = 0.01$ and $\gamma_A(a) = 0.17$ in this paper. For the convenience of simulation, the values of each parameter are adopted as follows: $\mu(a) = b(a) = 0.5$, $\varepsilon_1 = 0.01$, $\eta = 0.05$. Set the initial conditions as $s_0 = 150 + 40\sin(\pi x)$, $e_0 = 200 + \sin(\pi x)$, $i_0 = 10000 \cdot \exp\left(-0.2(x-5)^2\right)$, $a_0 = 80 + 40\sin(\pi x)$, $r_0 = 100 + \sin(\pi x)$. Since the infection rate and immune waning rate are age-dependent, we will simulate by assigning different values to the infection rate and immune waning rate respectively. We choose $\beta(a) = 0.40$, $\xi(a) = 0.32$. A simple calculation yields $R_0 = 2.3306$. While we have $R_0 = 0.0974$ by choosing $\beta(a) = 0.008$, $\xi(a) = 0.05$. According to Theorem 3.1, when the $R_0 < 1$, the disease-free equilibrium E_0 of the system is locally asymptotically stable, implying that the disease will be eliminated eventually; when $R_0 > 1$, the disease-free equilibrium is unstable and the disease breaks out. This conclusion can be verified by the simulation results in Figure 5.1 and 5.2. This indicates that when the initial conditions and other parameters are consistent, changes in the infection rate and immune waning rate caused by age variation will have an impact on the transmission of the disease, and may even lead to the outbreak of the disease.

To further investigate the impact of partial immunity on hand, foot, and mouth disease (HFMD), we only consider the scenario where $R_0 < 1$. With β fixed at 0.7132, the basic reproduction number R_0 is calculated as 0.9462. We analyzed the transmission effects by varying the value of immunity waning rate $\xi(a)$. Numerical simulations of the model were conducted for $\xi(a) = 0.0000$ and $\xi(a) = 0.8766$, respectively, and the transmission trends of HFMD were obtained as shown in Figure 5.3. When partial immune components are incorporated into the model, the model exhibits the characteristic of backward bifurcation. Even if the basic reproduction number $R_0 < 1$, the disease can still persist and become endemic. From an epidemiological perspective, an elevated immunity waning rate means that individuals who have acquired immunity lose their specific immunity more rapidly, leading to a continuous replenishment of the susceptible population. Even under the condition of $R_0 < 1$, a low level endemic prevalence can be maintained. In the development of HFMD prevention and control strategies, in addition to focusing on transmission intensity, attention should also be paid to the impact of immune persistence on the long-term evolution of the epidemic. By strength-

ening vaccination and booster immunization strategies, the rate of immunity waning can be slowed down, thereby effectively reducing the endemic prevalence level of HFMD.

Next, we fix $\xi(a) = 0.8867$ and set the other parameters to satisfy the conditions of Theorem 4.5. Let R_0 be a variable parameter and assign values to it within the range of 0.4 to 2. Two distinct sets of initial conditions are selected for the simulation: the first set is $s_0 = 150 + 40\sin(\pi x)$, $e_0 = 20 + \sin(\pi x)$, $i_0 = 100 \cdot \exp\left(-0.2(x-5)^2\right)$, $a_0 = 80 + 40\sin(\pi x)$, $r_0 = 100 + \sin(\pi x)$; the second set is $s_0 = 150 + 40\sin(\pi x)$, $e_0 = 200 + \sin(\pi x)$, $i_0 = 15000 \cdot \exp\left(-0.2(x-5)^2\right)$, $a_0 = 80 + 40\sin(\pi x)$, $r_0 = 100 + \sin(\pi x)$. The Figure 5.4 shows that within a certain range where $R_0 < 1$, the disease exhibits a significant characteristic of backward bifurcation under different initial conditions: when the initial infection level is high, a stable endemic steady state can still be formed even if $R_0 < 1$; when the initial infection level is low, the disease cannot maintain the transmission chain and gradually dies out. When $R_0 > 1$, regardless of the initial infection level, the two different sets of initial conditions will eventually lead to disease outbreak and convergence to the endemic steady state, and no disease extinction solution will appear. This indicates that when $\xi(a) \neq 0$ and the conditions of Theorem 4.5 are satisfied, backward bifurcation occurs, and the progression of the disease is related to the initial infection scale. Under high infection levels, an endemic state can still form even if $R_0 < 1$, which is consistent with the conclusion of Theorem 4.5. From an epidemiological perspective, the early monitoring and rapid response of the disease are particularly important. In the early stage of the disease, if the infected population can be detected and controlled in a timely manner and the initial infection level is reduced, the formation of a stable endemic state can be effectively avoided. When formulating prevention and control measures, we should not only focus on reducing the basic reproduction number R_0 but also strengthen the monitoring of recovered individuals, take appropriate measures to prolong the duration of immunity, and further block the transmission chain of the disease.

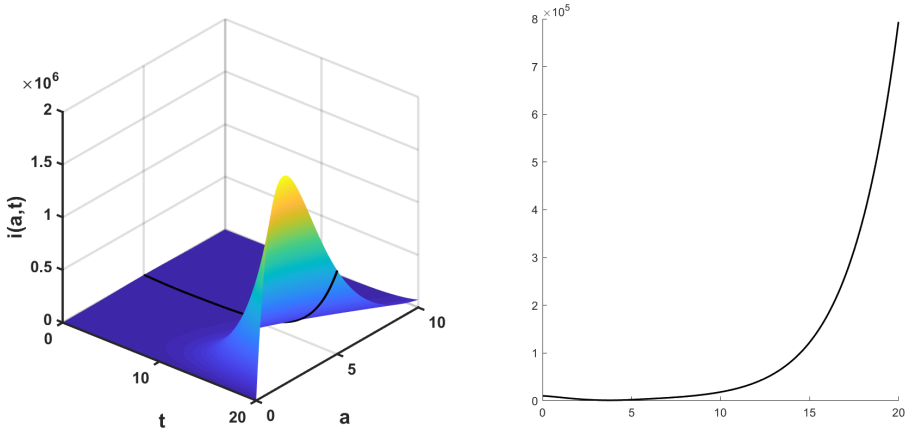

Figure 5.1: $R_0 > 1$: Evolution of infected individuals $i(a,t)$ with time t and age a; the right panel shows the vertical cross-section at age $a = 5$.

6 Conclusions

Age is a key factor influencing the transmission dynamics of HFMD. To reveal the effect of age on its epidemic pattern, this paper constructs an age-structured SEIAR model and

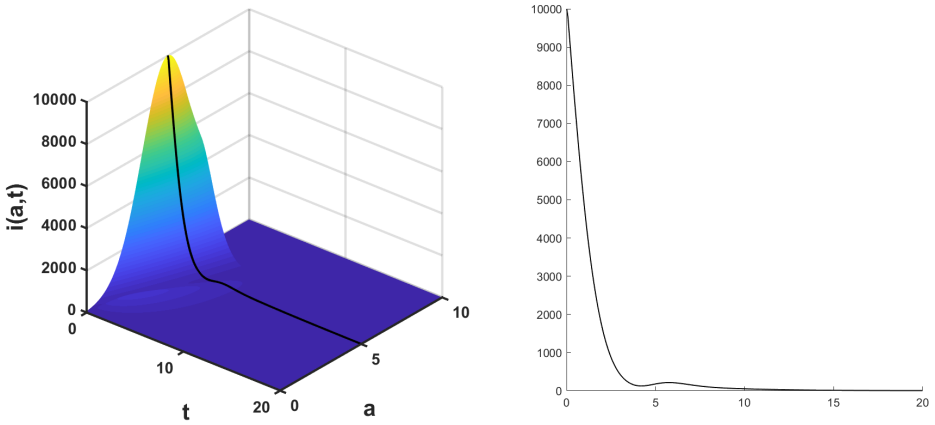

Figure 5.2: $R_0 < 1$: Evolution of infected individuals $i(a,t)$ with time t and age a; the right panel shows the vertical cross-section at age $a = 5$.

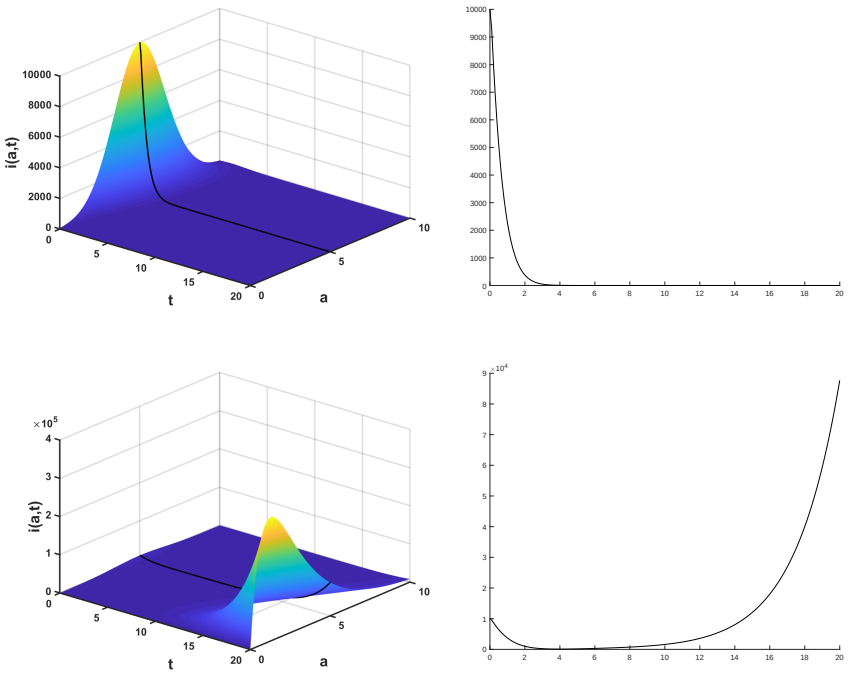

Figure 5.3: Solutions of $i(a,t)$ under different value conditions when $R_0 < 1$, along with the vertical cross-section at $a = 5$.

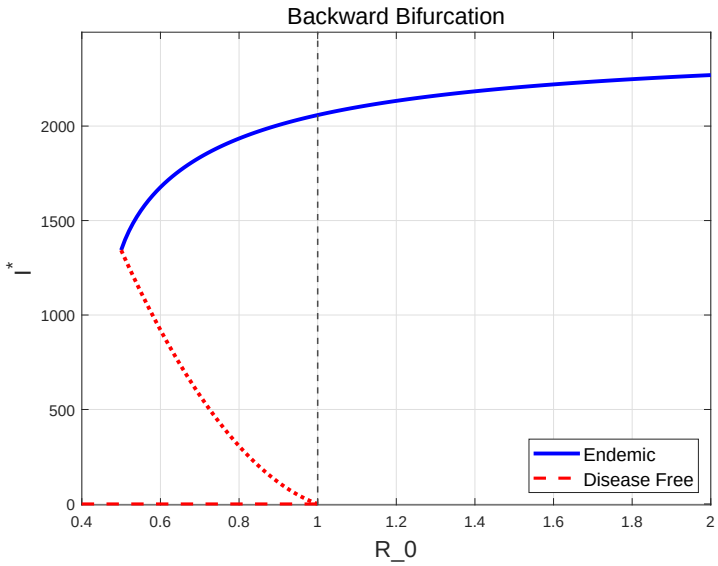

Figure 5.4: Backward bifurcation of the model.

systematically analyzes the stability and backward bifurcation behavior of the model. First, we reformulate the model as an abstract Cauchy problem to analyze the well-posedness of the system's solutions, and then derive the basic reproduction number R_0, which serves as the threshold for determining the extinction or persistence of HFMD. In the stability analysis of equilibria, Theorem 3.1 shows that the disease-free equilibrium E_0 is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$, and this classic threshold conclusion still holds in the partial differential age-structured model. Theorem 3.3 further provides a sufficient condition for the global asymptotic stability of the disease-free equilibrium, which can exclude the possibility of backward bifurcation under specific scenarios.

Subsequent analysis focuses on the backward bifurcation phenomenon. Theorem 4.1 proves that there exists at least one endemic equilibrium E^* when $R_0 > 1$, while Theorem 4.2 reveals that even when $R_0 < 1$, the system may still have two endemic equilibria under certain conditions, which directly provides a theoretical basis for the occurrence of backward bifurcation. This indicates that merely reducing R_0 to below 1 is not sufficient to eradicate the disease, the reason lies in the combined effect of two key factors: on the one hand, the age structure introduces transmission heterogeneity across different age cohorts; the younger age groups, as high-risk populations, have a much higher transmission potential than other age groups and serve as important carriers for the persistent spread of the disease. On the other hand, partial immunity prolongs the duration of susceptibility in the population and reduces the effective recovery rate, leading to a decline in the population's resistance to the disease and further increasing the likelihood of its sustained endemicity. Instead, it needs to be controlled to a level lower than 1, or the number of current infected individuals must be significantly reduced simultaneously. Theorem 4.5 clarifies the specific conditions for the occurrence of backward bifurcation in the system, and further confirms that the introduction of age structure makes the occurrence of backward bifurcation no longer dependent on a single global parameter, but rather on the dynamic interactions between different age groups. Furthermore, the inclusion of partial immunity creates an age-specific immune profile, alters the transmission and susceptibility characteristics of different age groups, and thereby influences the occurrence and development of backward bifurcation.

Acknowledgements

This work was supported by the National Natural Science Foundation of China (12201274).

References

[1] M. O. Adewole, F. A. Abdullah, M. K. M. Ali, Dynamics of hand, foot and mouth disease in children under 15 years old: A case study of Malaysia using age-structured modelling approach, *Appl. Math. Model.* **125**(2024), 728–749. https://doi.org/10.1016/j.apm.2023.10.002; MR4658364; Zbl 1572.92025

[2] L. M. Cai, M. R. Lei, J. Wang, Backward bifurcation of an age-structured epidemic model with partial immunity, *Internat. J. Bifur. Chaos Appl. Sci. Engrg.* **35**(2025), No. 8, Paper No. 2550095, 15 pp. https://doi.org/10.1142/S0218127425500956; MR4917403

[3] N. Halder, N. Roy, Compartmental modeling of hand, foot and mouth infectious disease (HFMD), *Res. J. Appl. Sci.* **5**(2012), No. 3, 177–182. https://doi.org/10.3923/rjasci.2010.177.182

[4] Z. Huang, M. Wang, L. Qiu, N. Wang, Z. Zhao et al., Seasonality of the transmissibility of hand, foot and mouth disease: a modelling study in Xiamen City, China, *Epidemiol. Infect.* **147**(2019), e327. https://doi.org/10.1017/S0950268819002139

[5] H. Inaba, *Age-structured population dynamics in demography and epidemiology*, Springer, Singapore, 2017. https://doi.org/10.1007/978-981-10-0188-8; MR3616174

[6] W. O. Kermack, A. G. McKendrick, A contribution to the mathematical theory of epidemics, *Proc. Roy. Soc. London Ser. A* **115**(1927), No. 772, 700–721. https://doi.org/10.1098/rspa.1927.0118

[7] X.-Z. Li, J. Yang, M. Martcheva, *Age structured epidemic modeling*, Springer, Cham, 2020. https://doi.org/10.1007/978-3-030-42496-1; MR4223785

[8] L. Li, H. Yin, Z. An, Z. Feng, Considerations for developing an immunization strategy with enterovirus 71 vaccine, *Vaccine* **33**(2015), No. 9, 1107–1112. https://doi.org/10.1016/j.vaccine.2014.10.081

[9] C.-C. Liu, H.-W. Tseng, S.-M. Wang, J.-R. Wang, I.-J- Su, An outbreak of enterovirus 71 infection in Taiwan, 1998: epidemiologic and clinical manifestations, *J. Clin. Virol.* **17**(2000), No. 1, 23–30. https://doi.org/10.1016/S1386-6532(00)00068-8

[10] J. Liu, Threshold dynamics for a HFMD epidemic model with periodic transmission rate, *Nonlinear Dynam.* **64**(2011), No. 1–2, 89–95. https://doi.org/10.1007/s11071-010-9848-6; MR2782960; Zbl 1280.92062

[11] T. Saki, Q. Liao, T. P. Van Boeckel, W. Xing, J. Sun, et al., Hand, foot, and mouth disease in China: Modeling epidemic dynamics of enterovirus serotypes and implications for vaccination, *PLOS Med.* **13**(2016), No. 2, e1001958. https://doi.org/10.1371/journal.pmed.1001958

[12] S. Sharma, G. P. Samanta, Analysis of a hand–foot–mouth disease model, *Int. J. Biomath.* **10**(2017), No. 2, 47 pp. https://doi.org/10.1142/s1793524517500164

[13] L. Shi, H. Zhao, D. Wu, Dynamical analysis for a reaction-diffusion HFMD model with nonsmooth saturation treatment function, *Commun. Nonlinear Sci. Numer. Simul.* **95**(2021), Paper No. 105593, 37 pp. https://doi.org/10.1016/j.cnsns.2020.105593; MR4200552

[14] R. Shi, T. Lu, Dynamic analysis and optimal control of a fractional order model for hand-foot-mouth disease, *J. Appl. Math. Comput.* **64**(2020), No. 1–2, 565–590. https://doi.org/10.1007/s12190-020-01369-w; MR4149416

[15] C. Sudarat, W. Surapa, The modelling of hand, foot, and mouth disease in contaminated environments in Bangkok, Thailand, *Comput. Math. Methods Med.* 2018 (2018), 1–8. https://doi.org/10.1155/2018/5168931

[16] G.-Q. Sun, H.-T. Zhang, L.-L. Chang, Z. Jin, H. Wang, et al., On the dynamics of a diffusive foot-and-mouth disease model with nonlocal infections, *SIAM J. Appl. Math.* **82**(2022), No. 4, 24. https://doi.org/10.1137/21M1412992; Zbl 1497.35482

[17] Y. Tang, H. Lian, S. Jing, Z. Ma, Hopf bifurcation of an age-structured hand-foot-mouth disease model in a contaminated environment, *Internat. J. Bifur. Chaos Appl. Sci. Engrg.* **34**(2024), No. 16, No. 2450196, 24. https://doi.org/10.1142/S0218127424501967; MR4845530

[18] A. Wang, D. Bai, J. He, S. R. Smith?, Optimal control of bi-seasonal hand, foot, and mouth disease in mainland China suggests transmission from children and isolating older infected individuals are critical, *J. Math. Biol.* **89**(2024), No. 1, 43. https://doi.org/10.1007/s00285-024-02141-5

[19] J. Wang, Y. Xiao, Z. Peng, Modelling seasonal HFMD infections with the effects of contaminated environments in mainland China, *Appl. Math. Comput.* **274**(2016), 615–627. https://doi.org/10.1016/j.amc.2015.11.035; MR3433162

[20] X. Wang, J. Qian, Y. Mi, Y. Li, Y. Cao, K. Qiao, Correlations of PSGL-1 VNTR polymorphism with the susceptibility to severe HFMD associated with EV-71 and the immune status after infection, *Virol. J.* **21**(2024), No. 1, 1–8. https://doi.org/10.1186/s12985-024-02461-4

[21] Y. C. Wang, F. C. Sung, Modeling the infections for enterovirus in Taiwan, *Am. J. Epidemiol.* **163**(2006), S192–c. https://doi.org/10.1093/aje/163.suppl_11.S192-c

[22] F. Xia, F. Deng, H. Tian, W. He, Y. Xiao, et al., Estimation of the reproduction number and identification of periodicity for HFMD infections in northwest China, *J. Theoret. Biol.* **484**(2020), 110027. https://doi.org/10.1016/j.jtbi.2019.110027

[23] D. Yan, Y. Jin, H. Cao, Y. Cao, Stability and Hopf bifurcation for a delayed hand-foot-mouth disease model with continuous age-structure in the exposed class, *Nonlinear Anal. Real World Appl.* **84**(2025), No. 104310, 17 pp. https://doi.org/10.1016/j.nonrwa.2024.104310, MR4847210

[24] J. Yang, Y. Chen, F.-Q. Zhang, Stability analysis and optimal control of a hand–foot–mouth disease (HFMD) model, *J. Appl. Math. Comput.* **41**(2013), No. 1–2, 99–117. https://doi.org/10.1007/s12190-012-0597-1, MR3017111, Zbl 1300.34118

[25] J. Yang, M. Gong, G.-Q. Sun, Asymptotical profiles of an age-structured foot-and-mouth disease with nonlocal diffusion on a spatially heterogeneous environment, *J. Differential Equations* **377**(2023), 71–112. https://doi.org/10.1016/j.jde.2023.09.001

[26] J. Yang, M. Zhou, X. Li, Backward bifurcation of an age-structured epidemic model with partial immunity: The Lyapunov–Schmidt approach, *Appl. Math. Lett.* **133**(2022), No. 108292, 7 pp. https://doi.org/10.1016/j.aml.2022.108292; MR4447678

[27] T. Yang, T. Xie, H. Li, X. Song, L. Yue, et al., Immune responses of a CV-A16 live attenuated candidate strain and its protective effects in rhesus monkeys, *Emerg. Microbes Infect.* **9**(2020), No. 1, 2136–2146. https://doi.org/10.1080/22221751.2020.1823889

[28] R. Yin, H. Wu, L. Zhang, H.-L. Li, Y. Luo, Z. Teng, A cholera model coupling within-host and between-host with age-dependent and asymptomatic infections, *J. Franklin Inst.* **361**(2024), No. 107283, 23 pp. https://doi.org/10.1016/j.jfranklin.2024.107283, MR4804754

[29] T. Zhang, Z. Zhang, Z. Yu, Q. Huang, D. Gao, Effects of behaviour change on HFMD transmission, *J. Biol. Dyn.* **17**(2023), No. 1, Article ID 2244968, 28 pp. https://doi.org/10.1080/17513758.2023.2244968, Zbl 1521.92098

[30] X. Zhang, R. Sun, et al., Clinical characteristics of fatal cases of hand, foot and mouth disease in children, *Front. Pediatr.* **13**(2025), 522164. https://doi.org/10.3389/fped.2025.1522164

[31] H. Zhao, L. Shi, J. Wang, K. Wang, A stage structure HFMD model with temperature-dependent latent period, *Appl. Math. Model.* **93**(2021), 745–761. https://doi.org/10.1016/j.apm.2021.01.010, MR4200500