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Backward Bifurcation of an Age-Structured Epidemic Model with Partial Immunity

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For many infectious diseases, including malaria and COVID-19, the host may experience more than one episode of infection, where reinfection occurs due to waning immunity. In this paper, we propose a new age-structured epidemic model to investigate the dynamics of such diseases with multiple infections. The model is based on a system of partial differential equations that describes the interplay between completely susceptible individuals, temporarily immune individuals, and infected individuals at different stages. The model incorporates both time and age-dependent variables and parameters. We derive the basic reproduction number and conduct rigorous analyses on the equilibrium solutions and their stability properties. Specifically, we study the global asymptotic stability of the disease-free equilibrium and obtain the explicit conditions for the occurrence of a backward bifurcation. Our findings could provide useful insights into the effects of disease prevention and intervention strategies such as vaccination campaigns.

Keywords: Age structure; epidemic model; reinfection; backward bifurcation.

1. Introduction

In contrast to those communicable diseases that could lead to long-lasting immunity upon recovery, most bacterial infections and some viral infections confer only temporary immunity. Consequently, hosts who contract such pathogens may experience more than one episode of infection, where the second or subsequent period of infection may or may not exhibit clinical symptoms [Mumtaz *et al.*, 2020; Bhatia *et al.*, 2020; Ren *et al.*, 2022]. Similarly, vaccines for many infectious diseases may only provide protection for the host for a certain period of time, after which the immunity gained from vaccination would fade or wane.

There have been several modeling studies devoted to diseases with multiple episodes of infection. For example, Greenhalgh *et al.* [2000] extended the classical SIS model to study animal infections with incomplete (or, temporary) immunity. Their analysis showed that a backward bifurcation may exist at the point where the basic reproduction number equals unity. Safan *et al.* [2013] modeled vaccination-based control of diseases that involve primary and secondary infections. They found that the reinfection level played

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a critical role in shaping the rich pattern of the disease dynamics. In addition, De Jong *et al.* [1996] developed a stochastic model to investigate the spread of bovine respiratory syncytial virus among cattle and the persistence of the virus in the host population.

Generally speaking, the susceptibility to an infectious disease and the severity of infection vary from host to host, depending on individual immune traits. Among the various factors that impact the infection, variation by age is particularly important. Because of the differences in immune functions, individuals from different age groups may react differently to an infection. For example, older adults and children are especially vulnerable to infectious diseases due to their weakened and immature immune systems, respectively [Rahman *et al.*, 2021]. Other differences related to age, such as social behavior, contact pattern, survival capacity, and recovery ability, may also lead to different levels of disease risk.

Although many mathematical epidemiological models have incorporated the impact of age on disease dynamics (e.g. [Busenberg *et al.*, 1991; Castillo Chavez *et al.*, 1989; Castillo-Chavez & Feng, 1998; Cai *et al.*, 2020; Cruz-Pacheco *et al.*, 2014; Li & Gupur, 2004; Li *et al.*, 2023; Wang, 2022; Yang *et al.*, 2019, 2023]), there are relatively few studies concerned with the application of age-structured models to infections with incomplete immunity. An age-structured modeling framework was proposed in [Wang *et al.*, 2019] for pertussis (i.e. whooping cough, a highly contagious bacterial infection of the respiratory tract) that allowed two or three infections during a lifetime. Their analysis of the two-infection model shows that the disease-free equilibrium is globally asymptotically stable when the basic reproduction number is lower than unity. In another age-structured pertussis modeling study [Tian & Wang, 2020], the authors considered the effects of vaccination and covert infection and proved the existence of a backward bifurcation when the covert infection rate was high. Despite these findings, our current understanding of the complex interactions between age, incomplete immunity, and multiple infections, and their implication to disease prevention and intervention, remains limited. Consequently, questions such as what is the long-term impact of a vaccination campaign on disease spread and whether a public health strategy based solely on vaccinating a proportion of newborns can lead to effective control of a disease have not been completely answered.

We design a simple Susceptible–Infectious–Susceptible (SIS) compartmental model based on the most assumptions of the two-stage bovine respiratory syncytial virus epidemiological model presented. To address this knowledge gap, we propose a simple SIS compartmental model with age structure in this paper to study diseases that involve multiple episodes of infections. We introduce two separate compartments that represent the individuals associated with the first (or, primary) infection and those with the second and subsequent infections. All the variables and parameters are assumed to be age-dependent. The model is applied to a heterogeneous host population where a portion of the population has developed immunity for the primary infection but is still susceptible for the subsequent infections. We conduct a rigorous mathematical analysis of this model and resolve the key properties associated with the disease dynamics.

We organize the remainder of this paper as follows. We first present the mathematical formulation of our model in Sec. 2. We then analyze the disease-free equilibrium and endemic equilibrium in Secs. 3 and 4, respectively. We conclude the paper with some discussion in Sec. 5.

2. Mathematical Model

We design a simple SIS compartmental model with age structure, and divide the human population in the model into four compartments related to different stages of infection. Let $S_u(t,a)$ and $S_v(t,a)$ denote, respectively, the densities of individuals who are completely susceptible and those who have temporary immunity at time t and age a. Let $I(t,a)$ and $J(t,a)$ denote, respectively, the densities of individuals with the first infection and those with the secondary/subsequent infection at time t and age a. The density of the total population is then given by

$$N(t,a) = S_u(t,a) + I(t,a) + S_v(t,a) + J(t,a). \tag{1}$$

It is assumed that individuals may have multiple infections during their lifetime. A fraction $1-p$, with $0 < p < 1$, of newborns is temporarily immune to the disease, either due to maternal immunity or vaccination immediate at birth. These individuals enter the susceptible class $S_v(t,a)$ and are protected from the first infection, but may contract

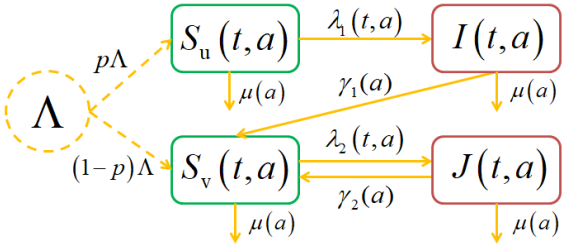

Fig. 1. A schematic for model (2).

subsequent infections. The infectivity from the second and subsequent infections will decrease by a factor of $\sigma \in (0,1)$ due to immune effects. The host contact rate and the probability of exposure to infection per contact are assumed to be age-dependent, denoted by continuous functions $c(a)$ and $\beta(a)$, respectively. A schematic diagram of the model is shown in Fig. 1.

The disease dynamics are described by the following partial differential equations:

$$\begin{aligned}
&\left(\frac{\partial}{\partial a}+\frac{\partial}{\partial t}\right) S_u(t,a)\\
&\quad = -\tilde{\lambda}_1(t,a)S_u(t,a) - \mu(a)S_u(t,a),\\
&\left(\frac{\partial}{\partial a}+\frac{\partial}{\partial t}\right) I(t,a)\\
&\quad = \tilde{\lambda}_1(t,a)S_u(t,a) - \gamma_1(a)I(t,a) - \mu(a)I(t,a),\\
&\left(\frac{\partial}{\partial a}+\frac{\partial}{\partial t}\right) S_v(t,a)\\
&\quad = \gamma_1(a)I(t,a) - \tilde{\lambda}_2(t,a)S_v(t,a) + \gamma_2(a)J(t,a)\\
&\qquad - \mu(a)S_v(t,a),\\
&\left(\frac{\partial}{\partial a}+\frac{\partial}{\partial t}\right) J(t,a)\\
&\quad = \tilde{\lambda}_2(t,a)S_v(t,a) - \gamma_2(a)J(t,a) - \mu(a)J(t,a),
\end{aligned} \tag{2}$$

where $\gamma_1(a)$ and $\gamma_2(a)$ denote the recovery rates of the first infection and the second (and subsequent) infection, respectively, and $\mu(a)$ denotes the natural mortality rate. The infectivity $\lambda_1(t,a)$ for the first infection takes an integral form as

$$\tilde{\lambda}_1(t,a) = c(a)\int_0^\infty \frac{\beta(u)[I(t,u)+J(t,u)]}{N(t,u)du}, \tag{3}$$

and the infectivity for the second infection is assumed to be $\tilde{\lambda}_2(t,a) = \sigma\tilde{\lambda}_1(t,a)$ for some constant $0 < \sigma < 1$. The boundary conditions for system (2) are

$$\begin{aligned}
&S_u(t,0) = p\Lambda, \quad I(t,0) = 0,\\
&S_v(t,0) = (1-p)\Lambda, \quad J(t,0) = 0,
\end{aligned} \tag{4}$$

where $\Lambda > 0$ is the constant influx rate of newborns. Meanwhile, the initial conditions are

$$\begin{aligned}
&S_u(0,a) = S_{u0}(a), \quad I(0,a) = I_0(a),\\
&S_v(0,a) = S_{v0}(a), \quad J(0,a) = J_0(a).
\end{aligned}$$

Assumption 2.1. The functions and parameters in model (2) are all non-negative and satisfy the following conditions:

(i) The natural space for the state variable $S_u(t,\cdot)$, $I(t,\cdot)$, $S_v(t,\cdot)$, $J(t,\cdot)$ is $L^1_+(0,\infty)$, where $L^1_+(0,\infty)$ is the positive cone of functions on $(0,\infty)$.

(ii) The age-dependent parameters $c(a)$, $\beta(a)$, $\gamma_1(a)$, $\gamma_2(a)$ are bounded and belong to $L^1_+(0,\infty)$, and $\mu(a) \in L^1_{\text{Loc},+}(0,\infty)$ with $\int_0^\infty \mu(\sigma)d\sigma = \infty$.

(iii) The initial conditions $S_{u0}(a)$, $I_0(a)$, $S_{v0}(a)$, $J_0(a)$ belong to $L^1_+(0,\infty)$.

Based on Eqs. (1) and (2), we have

$$\begin{aligned}
&\left(\frac{\partial}{\partial a}+\frac{\partial}{\partial t}\right) N(a,t) = -\mu(a)N(a,t),\\
&N(0,t) = \Lambda,\\
&N(a,0) = S_{u0}(a) + I_0(a)\\
&\qquad + S_{v0}(a) + J_0(a)\\
&\qquad =: N_0(a).
\end{aligned} \tag{5}$$

Using the method of characteristics, we get

$$N(a,t) = \begin{cases} N(0,t-a)e^{-\int_0^a \mu(\tau)d\tau}, & t \geq a,\\ N(a-t,0)e^{-\int_0^t \mu(a-t+\eta)d\eta}, & t < a. \end{cases}$$

Note that $N(a,t)$ is continuous if and only if the compatibility condition $N_0(a) = \Lambda$ is satisfied. By Assumption 2.1, we obtain the steady-state solution of model (2)

$$\lim_{t\to\infty} N(a,t) = \Lambda\pi(a),$$

where

$$\pi(a) = e^{-\int_0^a \mu(\tau)d\tau}$$

is the possibility of survival till age a. Without loss of generality, we assume that

$$N(a,t) = N_\infty(a) = \Lambda\pi(a).$$

The critical dynamical properties of our model will be reflected by its equilibrium states. In what follows, we will conduct a detailed mathematical analysis on the equilibrium solutions and their stabilities.

To facilitate the analysis, we first normalize model (2). Let

$$x_u(t,a) = \frac{S_u(t,a)}{N_\infty(a)}, \quad y(t,a) = \frac{I(t,a)}{N_\infty(a)},$$
$$x_v(t,a) = \frac{S_v(t,a)}{N_\infty(a)}, \quad z(t,a) = \frac{J(t,a)}{N_\infty(a)}.$$

Then, the original model (2) is transformed into the following form:

$$\begin{aligned}
&\left(\frac{\partial}{\partial a} + \frac{\partial}{\partial t}\right) x_u(t,a) = -\lambda(t,a)x_u(t,a),\\
&\left(\frac{\partial}{\partial a} + \frac{\partial}{\partial t}\right) y(t,a) = \lambda(t,a)x_u(t,a) - \gamma_1(a)y(t,a),\\
&\left(\frac{\partial}{\partial a} + \frac{\partial}{\partial t}\right) x_v(t,a)\\
&\quad = \gamma_1(a)y(t,a) - \sigma\lambda(t,a)x_v(t,a) + \gamma_2(a)z(t,a),\\
&\left(\frac{\partial}{\partial a} + \frac{\partial}{\partial t}\right) z(t,a)\\
&\quad = \sigma\lambda(t,a)x_v(t,a) - \gamma_2(a)z(t,a),
\end{aligned} \tag{6}$$

where

$$\begin{aligned}
\lambda(t,a) &= c(a)\int_0^\infty \beta(u)[y(t,u) + z(t,u)]du\\
&= c(a)\mathcal{F}(t)
\end{aligned} \tag{7}$$

and

$$\mathcal{F}(t) = \int_0^\infty \beta(u)[y(t,u) + z(t,u)]du.$$

The boundary conditions in (4) become

$$\begin{aligned}
&x_u(t,0) = p, \quad y(t,0) = 0,\\
&x_v(t,0) = 1-p, \quad z(t,0) = 0,\\
&x_u(0,a) = s_{u0}(a), \quad y(0,a) = i_0(a),\\
&x_v(0,a) = s_{v0}(a), \quad z(0,a) = j_0(a),
\end{aligned}$$

where the state space is given by

$$\begin{aligned}
X = \{&(x_u, y, x_v, z) \in L^1_+(0,\infty)\times L^1_+(0,\infty)\\
&\times L^1_+(0,\infty)\times L^1_+(0,\infty), x_u + y + x_v + z = 1\}.
\end{aligned}$$

Now, we first investigate the existence and uniqueness of non-negative solutions for model (6). Let us define the Banach spaces X with the norm $\|\Phi\| = \sum_{i=1}^4 \|\Phi_i\|_1$ for $\Phi(a) = (\Phi_1(a), \Phi_2(a), \Phi_3(a), \Phi_4(a))^T \in X$, where $\|\Phi\|_1 = \int_0^\infty \Phi(a)da$, $\phi(a) \in L^1_+(0,\infty)$, and the superscript T denotes the transpose of a vector. Now, we show the well-posedness of model (6) through recasting the problem in semigroup formulations (see Proposition 6.1 in [Inaba, 2017]). We define a linear operator A: $\mathcal{D}(A) \subset X \to X$ by

$$\begin{aligned}
&(A\Phi)(a)\\
&:= \begin{pmatrix}
-\dfrac{d\Phi_1(a)}{da}\\
-\dfrac{d\Phi_2(a)}{da} - \gamma_1(a)\Phi_2(a)\\
-\dfrac{d\Phi_3(a)}{da} + \gamma_1(a)\Phi_2(a) + \gamma_2(a)\Phi_4(a)\\
-\dfrac{d\Phi_4(a)}{da} - \gamma_2(a)\Phi_4(a)
\end{pmatrix},
\end{aligned}$$

$$\Phi(a) = (\Phi_1(a), \Phi_2(a), \Phi_3(a), \Phi_4(a))^T \in \mathcal{D}(A),$$

where the domain $\mathcal{D}(A)$ is given by

$$\begin{aligned}
\mathcal{D}(A) = \{&\Phi \in X : \Phi_j \text{ is absolutely continuous on}\\
&(0,\infty), \Phi_1(0) = p, \Phi_3(0) = 1-p,\\
&\Phi_2(0) = \Phi_4(0) = 0\}.
\end{aligned}$$

Let $F : X \to X$ be a nonlinear operator defined by

$$(F\Phi)(a) := \begin{pmatrix}
-\lambda(a,t)\Phi_1(a)\\
\lambda(a,t)\Phi_1(a)\\
-\sigma\lambda(a,t)\Phi_3(a)\\
\sigma\lambda(a,t)\Phi_3(a)
\end{pmatrix}, \quad \Phi \in L^1_+(0,\infty),$$

$$\lambda(a) = c(a)\int_0^\infty \beta(\mu)(\Phi_2(\mu) + \Phi_4(\mu))d\mu.$$

Let $\omega(t) = (x_u(\cdot,t), y(\cdot,t), x_v(\cdot,t), z(\cdot,t))^T$ be the state vector of model (6). Model (6) can be

formulated as the following semilinear Cauchy problem on the Banach space X:

$$\frac{d\omega(t)}{dt} = A\omega(t) + F(\omega(t)), \quad \omega(0) = \omega_0 \in X, \tag{8}$$

where $\omega_0(a) = (s_{u0}(a), i_0(a), s_{v0}(a), j_0(a))$. It is easily seen that the linear operator A is the infinitesimal generator of C_0-semigroup $T(t)$ for $t \geq 0$ and nonlinear operator F is continuously Fréchet differentiable on X. To show the existence and uniqueness of positive solutions in model (6), we let

$$M = \{(x_u, y, x_v, z)^T \in X : x_u \geq 0, y \geq 0, \\ x_v \geq 0 \text{ and } z \geq 0\}$$

and

$$M_0 = \{(x_u, y, x_v, z)^T \in X : 0 \leq x_u \leq 1, 0 \leq y \leq 1, \\ 0 \leq x_v \leq 1, \text{ and } 0 \leq z \leq 1\}.$$

Following similar analysis in [Inaba, 2017, Sec. 6.1.2, pp. 291–294] or [Li *et al.*, 2020, Sec. 2.1.1, pp. 23–26], we obtain the following result.

Proposition 1. *The set M_0 is positively invariant with respect to the semiflow defined by the mild solution. The Cauchy problem (8) has a unique solution on X with respect to initial data $\omega_0 \in M \cap \mathcal{D}(A)$.*

3. Disease-Free Equilibrium and Its Stability

Let the disease-free equilibrium be $E_0 = (x_u(a), y(a), x_v(a), z(a))$, where $y(a) = z(a) = 0$ and $\lambda_1(a) = 0$. Then we obtain $\frac{\mathrm{d}x_u(a)}{\mathrm{d}a} = 0$ from the first equation of model (6). Solving this differential equation yields $x_u(a) = x_u(0) = p$. Similarly, from the second equation of system (6), we obtain $x_v(a) = x_v(0) = 1 - p$. Therefore, the disease-free equilibrium E_0 is given by

$$E_0 = (p, 0, 1 - p, 0).$$

Now, we show the asymptotic stability of the disease-free equilibrium. Linearize model (6), and consider the exponential solution

$$\begin{aligned} &\bar{x}_u(t, a) = e^{\alpha t} x_u(a), \quad \bar{y}(t, a) = e^{\alpha t} y(a), \\ &\bar{x}_v(t, a) = e^{\alpha t} x_v(a), \quad \bar{z}(t, a) = e^{\alpha t} z(a), \\ &F_0 = \int_0^\infty \beta(a)[y(a) + z(a)]da. \end{aligned} \tag{9}$$

Under the condition of the disease-free equilibrium, the exponential solution is brought into the linearized system to obtain

$$\begin{aligned} &x_{ua} + \alpha x_u = -\lambda(a)p, \\ &y_a + \alpha y = \lambda(a)p - \gamma_1(a)y(a), \\ &x_{va} + \alpha x_v = \gamma_1(a)y(a) - \sigma\lambda(a)(1 - p) + \gamma_2(a)z(a), \\ &z_a + \alpha z = \sigma\lambda(a)(1 - p) - \gamma_2(a)z(a). \end{aligned} \tag{10}$$

Solving the second and fourth equations in system (10), we have

$$y(a) = pe^{-\int_0^a (\gamma_1(\xi)+\alpha)d\tau} \int_0^a c(\tau)F_0 e^{\int_0^\tau (\gamma_1(\xi)+\alpha)d\xi} d\tau,$$

$$z(a) = \sigma(1 - p)e^{-\int_0^a (\gamma_2(\tau)+\alpha)d\tau} \int_0^a c(\tau) \\ \times F_0 e^{\int_0^\tau (\gamma_2(\xi)+\alpha)d\xi} d\tau.$$

Substitution of $y(a)$ and $z(a)$ into F_0 yields

$$F_0 = \int_0^\infty \beta(a) \left[pe^{-\int_0^a (\gamma_1(\tau)+\alpha)d\tau} \int_0^a c(\tau) \right. \\ \times F_0 e^{\int_0^\tau (\gamma_1(\xi)+\alpha)d\xi} d\tau + \sigma(1 - p) \\ \times e^{-\int_0^a (\gamma_2(\tau)+\alpha)d\tau} \int_0^a c(\tau) \\ \left. \times F_0 e^{\int_0^\tau (\gamma_2(\xi)+\alpha)d\xi} d\tau \right] da.$$

Dividing both sides by F_0, we obtain

$$1 = p \int_0^\infty \beta(a) e^{-\int_0^a (\gamma_1(\xi)+\alpha)d\xi} \\ \times \int_0^a c(\tau) e^{\int_0^\tau (\gamma_1(\xi)+\alpha)d\xi} d\tau da \\ + \sigma(1 - p) \int_0^\infty \beta(a) e^{-\int_0^a (\gamma_2(\xi)+\alpha)d\xi} \\ \times \int_0^a c(\tau) e^{\int_0^\tau (\gamma_2(\xi)+\alpha)d\xi} d\tau da.$$

We define the right-hand side of the above equation as $H(\alpha)$. Consequently, the basic reproduction number of model (6) is given by

$$R_0 = H(0) \\ = p \int_0^\infty \beta(a)\pi_1(a) \int_0^a c(\tau)\pi_1^{-1}(\tau) d\tau da$$

$$+\sigma(1-p)\int_0^\infty \beta(a)\pi_2(a)\int_0^a c(\tau) \times \pi_2^{-1}(\tau)d\tau da, \tag{11}$$

where

$$\pi_1(a) = e^{-\int_0^a \gamma_1(\tau)d\tau} \quad \text{and}$$
$$\pi_2(a) = e^{-\int_0^a \gamma_2(\tau)d\tau}.$$

Biologically, the basic reproduction number R_0 is defined as the average number of secondary infections caused by one infectious individual entering a susceptible population. Mathematically, we have derived R_0 from the characteristic equation $H(\alpha) = 1$. Equation (11) shows that R_0 consists of two parts, quantifying the contributions from two different transmission routes — the first part represents the number of infected individuals produced by the initial susceptible portion p from the completely susceptible population class, while the second part represents the number of infected individuals produced by the initial susceptible portion $(1-p)$ from the population class with temporary immunity.

Using similar methods as those in [Wang *et al.*, 2019], we can establish the following result.

Theorem 3.1. *If $R_0 < 1$, the equilibrium E_0 is locally asymptotically stable. If $R_0 > 1$, the equilibrium E_0 is unstable.*

Proof. By direct calculation, we obtain

$$H'(\alpha) < 0.$$

It is easy to see that $H(\alpha)$ is a decreasing function, and $\lim_{\alpha\to\infty} H(\alpha) = 0$, $\lim_{\alpha\to-\infty} H(\alpha) = \infty$. Hence, there exists a unique real number α_0 such that $H(\alpha_0) = 1$. When $R_0 = H(0) < 1$, we have $\alpha_0 < 0$. Now, we consider a complex root $l = l_1 + il_2$ with $l_2 \neq 0$ such that $H(l) = 1$. We show that the real part l_1 must be negative. Assume, instead, $l_1 \geq 0$. Then

$$\begin{aligned}
1 &= |H(\alpha_0)| = |H(l)| \\
&= \left| p\int_0^\infty\int_0^\tau \beta(\tau)c(\xi)e^{-\int_\xi^\tau \gamma_1(x)dx} \times e^{-(l_1+il_2)(\tau-\xi)}d\xi d\tau + \sigma(1-p)\int_0^\infty\int_0^\tau \beta(\tau) \times c(\xi)e^{-\int_\xi^\tau \gamma_2(x)dx}e^{-(l_1+il_2)(\tau-\xi)}d\xi d\tau \right| \\
&\leq p\int_0^\infty\int_0^\tau \beta(\tau)c(\xi)e^{-\int_\xi^\tau \gamma_1(x)dx}e^{-l_1(\tau-\xi)}d\xi d\tau \\
&\quad + \sigma(1-p)\int_0^\infty\int_0^\tau \beta(\tau)c(\xi) \times e^{-\int_\xi^\tau \gamma_2(x)dx}e^{-l_1(\tau-\xi)}d\xi d\tau \\
&= H(l_1) \leq H(0) = R_0 < 1,
\end{aligned}$$

which is a contradiction. Therefore, $l = l_1 + il_2$ has a negative real part. This shows that when $R_0 < 1$, the disease-free equilibrium E_0 is locally asymptotically stable. In contrast, when $R_0 = H(0) > 1$, the characteristic equation has a positive root, which indicates that the equilibrium E_0 is unstable. ■

We study the global stability of the disease-free equilibrium below for a special case where the two recovery rates $\gamma_1(a)$ and $\gamma_2(a)$ are equal.

Theorem 3.2. *The equilibrium E_0 is globally asymptotically stable when $\frac{1}{\sigma(1-p)+p}R_0 < 1$ and $\gamma_1(a) = \gamma_2(a) = \gamma(a)$.*

Proof. Let $\Psi(t,a)$ denote the probability of an uninfected individual of age a being infected at time t. That is,

$$\Psi(t,a) = c(a)\mathcal{F}(t)(x_u(t,a) + \sigma x_v(t,a)).$$

Since $\sigma \in (0,1)$, we have

$$x_u(t,a) + \sigma x_v(t,a) \leq 1.$$

Then

$$\Psi(t,a) \leq c(a)\mathcal{F}(t). \tag{12}$$

Integrating model (6) along the characteristic lines, we have

$$\begin{aligned}
y(t,a) &= \int_0^a c(s)\mathcal{F}(t-a+s)x_u \times (t-a+s,s)\frac{\pi(a)}{\pi(s)}ds, \quad a < t, \\
z(t,a) &= \int_0^a \sigma c(s)\mathcal{F}(t-a+s)x_v \times (t-a+s,s)\frac{\pi(a)}{\pi(s)}ds, \quad a < t,
\end{aligned} \tag{13}$$

where $\pi(a) = e^{-\int_0^a \gamma(\tau)d\tau}$. Thus,

$$y(t,a) + z(t,a) = \int_0^a \Psi(t-a+s,s)\frac{\pi(a)}{\pi(s)}ds, \quad a < t. \tag{14}$$

Substituting (14) into $\mathcal{F}(t)$, we obtain

$$\begin{aligned}\Psi(t,a) \le c(a)\int_0^t \beta(a)\int_0^a \frac{\pi(a)}{\pi(s)} \\ \times \Psi(t-a+s,s)dsda \\ + c(a)\int_t^\infty \beta(a)(y(t,a)+z(t,a))da.\end{aligned} \tag{15}$$

From the above inequality, we have

$$c(a)\int_t^\infty \beta(a)(y(t,a)+z(t,a))da \to 0 \quad (t\to\infty).$$

Defining

$$\Psi_\infty(a) = \limsup_{t\to\infty}\Psi(t,a)$$

and taking the limit superior when $t\to\infty$ on both sides of (15), we can obtain

$$\Psi_\infty(a) \le c(a)\int_0^\infty \beta(a)\int_0^a \frac{\pi(a)}{\pi(s)}\Psi_\infty(s)dsda. \tag{16}$$

Let

$$\Gamma = \int_0^\infty \beta(a)\int_0^a \frac{\pi(a)}{\pi(s)}\Psi_\infty(s)dsda \tag{17}$$

be a non-negative constant. Then $\Psi_\infty(a)\le c(a)\Gamma$. From (17), we have

$$\begin{aligned}\Gamma &= \int_0^\infty \beta(a)\int_0^a \frac{\pi(a)}{\pi(s)}\Psi_\infty(s)dsda \\ &\le \Gamma\int_0^\infty \beta(a)\int_0^a c(s)\frac{\pi(a)}{\pi(s)}dsda.\end{aligned} \tag{18}$$

Hence,

$$\begin{aligned}\Gamma &\le \Gamma\int_0^\infty \beta(a)\int_0^a \frac{\pi(a)}{\pi(s)}c(s)dsda \\ &= \Gamma\frac{1}{\sigma(1-p)+p}R_0.\end{aligned} \tag{19}$$

When $\frac{1}{\sigma(1-p)+p}R_0<1$, (19) yields $\Gamma=0$. Therefore, we have $\Psi_\infty(a)\equiv 0$. This means that

$$\limsup_{t\to\infty}\Psi(t,a)=0, \quad a\in[0,\infty).$$

From the non-negativity of the solution and (14), we have

$$\lim_{t\to\infty} y(t,a)=0, \quad \lim_{t\to\infty} z(t,a)=0.$$

Similarly,

$$\begin{aligned}x_u(t,a) &= e^{\int_0^a -\lambda_1(t-a+\tau,\tau)d\tau}x_u(t-a,0) \\ &= pe^{\int_0^a -\lambda_1(t-a+\tau,\tau)d\tau},\end{aligned}$$

$$\lim_{t\to\infty} x_u(t,a)=p,$$

and

$$\begin{aligned}x_v(t,a) = e^{-\int_0^a \lambda_2(t-a+\tau,\tau)d\tau} \\ \times\Bigg[\int_0^a \gamma(\eta)(y(t-a+\eta,\eta) \\ + z(t-a+\eta,\eta))e^{\int_0^\eta \lambda_2(t-a+\tau,\tau)d\tau}d\eta \\ + x_v(t-a,0)\Bigg],\end{aligned}$$

$$\lim_{t\to\infty} x_v(t,a)=1-p.$$

This completes the proof. ■

4. Endemic Equilibrium and Backward Bifurcation

We separate the discussion for the two cases $R_0>1$ and $R_0<1$ in the following.

Theorem 4.1. *When the reproduction number $R_0>1$, model (6) has at least one endemic equilibrium E^*.*

Proof. At the equilibrium E^*, model (6) becomes

$$\begin{aligned}\frac{\mathrm{d}x_u(a)}{\mathrm{d}a} &= -\lambda^*(a)x_u(a), \\ \frac{\mathrm{d}y(a)}{\mathrm{d}a} &= \lambda^*(a)x_u(a)-\gamma_1(a)y(a), \\ \frac{\mathrm{d}x_v(a)}{\mathrm{d}a} &= \gamma_1(a)y(a)-\sigma\lambda^*(a)x_v(a)+\gamma_2(a)z(a), \\ \frac{\mathrm{d}z(a)}{\mathrm{d}a} &= \sigma\lambda^*(a)x_v(a)-\gamma_2(a)z(a), \\ \lambda^*(a) &= c(a)\int_0^\infty \beta(a)[y(a)+z(a)]da = c(a)F_0,\end{aligned} \tag{20}$$

where $F_0=\int_0^\infty \beta(a)[y(a)+z(a)]da$. The solutions of the second and fourth equations of model (20) are

$$y(a) = p\int_0^a \lambda^*(\tau)e^{-\int_0^\tau \lambda^*(u)du}\frac{\pi_1(a)}{\pi_1(\tau)}d\tau,$$

$$z(a) = \sigma p \int_0^a \int_0^\eta \int_0^\theta \lambda^*(\eta)\lambda^*(\tau)\gamma_1(\theta) e^{-\int_0^\tau \lambda^*(u)du} \times \frac{\pi_1(\theta)}{\pi_1(\tau)} e^{-\int_\eta^a \sigma\lambda^*(\xi)d\xi} \frac{\pi_2(a)}{\pi_2(\eta)} d\tau d\theta d\eta + \sigma(1-p) \times \int_0^a \lambda^*(\eta) e^{-\int_\eta^a \sigma\lambda^*(\xi)d\xi} \frac{\pi_2(a)}{\pi_2(\eta)} d\eta. \tag{21}$$

Substituting $y(a)$ and $z(a)$ into F_0, we have

$$\begin{aligned} F_0 &= \int_0^\infty \beta(a)[y(a)+z(a)]da \\ &= \int_0^\infty \beta(a) \left[p \int_0^a \lambda^*(\tau) e^{-\int_0^\tau \lambda^*(u)du} \frac{\pi_1(a)}{\pi_1(\tau)} d\tau \right. \\ &\quad + \sigma p \int_0^a \int_0^\eta \int_0^\theta \lambda^*(\eta)\lambda^*(\tau)\gamma_1(\theta) e^{-\int_0^\tau \lambda^*(u)du} \\ &\quad \times \frac{\pi_1(\theta)}{\pi_1(\tau)} e^{-\int_\eta^a \sigma\lambda^*(\xi)d\xi} \frac{\pi_2(a)}{\pi_2(\eta)} d\tau d\theta d\eta + \sigma(1-p) \\ &\quad \left. \times \int_0^a \lambda^*(\eta) e^{-\int_\eta^a \sigma\lambda^*(\xi)d\xi} \frac{\pi_2(a)}{\pi_2(\eta)} d\eta \right] da. \end{aligned} \tag{22}$$

Meanwhile, substitute $\lambda_1^*(a) = c(a)F_0$ into F_0 to obtain

$$\begin{aligned} 1 &= \int_0^\infty \beta(a) \left[p \int_0^a c(\tau) e^{-\int_0^\tau c(u)F_0 du} \frac{\pi_1(a)}{\pi_1(\tau)} d\tau \right. \\ &\quad + \sigma p \int_0^a \int_0^\eta \int_0^\theta c(\eta)c(\tau)F_0\gamma_1(\theta) e^{-\int_0^\tau c(u)F_0 du} \\ &\quad \times \frac{\pi_1(\theta)}{\pi_1(\tau)} e^{-\int_\eta^a \sigma c(\xi)F_0 d\xi} \frac{\pi_2(a)}{\pi_2(\eta)} d\tau d\theta d\eta + \sigma(1-p) \\ &\quad \left. \times \int_0^a c(\eta) e^{-\int_\eta^a \sigma c(\xi)F_0 d\xi} \frac{\pi_2(a)}{\pi_2(\eta)} d\eta \right] da. \end{aligned} \tag{23}$$

We define the right-hand side of Eq. (23) as $Q(F_0)$. Then we obtain

$$\begin{aligned} Q(0) &= p \int_0^\infty \beta(a) \int_0^a c(\tau) \frac{\pi_1(a)}{\pi_1(\tau)} d\tau da + \sigma(1-p) \\ &\quad \times \int_0^\infty \beta(a) \int_0^a c(\tau) \frac{\pi_2(a)}{\pi_2(\tau)} d\tau da \\ &= R_0, \end{aligned}$$

and $\lim_{F_0\to\infty} Q(F_0) = 0$. Therefore, when $R_0 > 1$, $Q(F_0) = 1$ has at least one positive solution. Therefore, there is at least one endemic equilibrium in model (6).

Now, we consider the case $R_0 < 1$. To proceed, we calculate

$$\begin{aligned} Q'(0) &= -p \int_0^\infty \beta(a) \int_0^a c(\tau) \int_0^\tau c(u)du \frac{\pi_1(a)}{\pi_1(\tau)} d\tau da \\ &\quad + \sigma p \int_0^\infty \beta(a) \int_0^a \int_0^\eta \int_0^\theta c(\eta)c(\tau)\gamma_1(\theta) \\ &\quad \times \frac{\pi_1(\theta)}{\pi_1(\tau)} \frac{\pi_2(a)}{\pi_2(\eta)} d\tau d\theta d\eta da - \sigma(1-p) \\ &\quad \times \int_0^\infty \beta(a) \int_0^a c(\eta) \frac{\pi_2(a)}{\pi_2(\eta)} \int_\eta^a \sigma c(\xi) d\xi d\eta da. \end{aligned}$$

Let $Q'(0) = U$. ■

Theorem 4.2. *When the reproduction number $R_0 < 1$, model (6) has at least two endemic steady states if $U > 0$ and $|R_0 - 1|$ is sufficiently small.*

Proof. Define

$$W(F_0, q) = q\frac{Q(F_0)}{R_0} - 1.$$

Then

$$W(0,1) = \frac{Q(0)}{R_0} - 1 = 0, \quad \frac{\partial W}{\partial F_0}(0,1) = \frac{U}{R_0} > 0.$$

Based on the implicit function theorem, there is a unique continuously differentiable function $F_0 = F_0(q)$ defined in a small neighborhood of the number 1 and

$$\left.\frac{\mathrm{d}F_0}{\mathrm{d}q}\right|_{q=1} = -\frac{R_0}{U} < 0.$$

Since $F_0(1) = 0$, there is $\varepsilon > 0$ such that $F_0(q) > 0$ and $W(F_0, q) \equiv 0$ in the small neighborhood of the point $(1-\varepsilon, 1)$. Let $R_0 \in (1-\frac{\varepsilon}{2}, 1)$ and $q \in (1-\varepsilon, 1-\frac{\varepsilon}{2})$. We have

$$Q(\infty) = 0, \quad Q(0) = R_0 < 1, \quad Q(F_0(q)) > 1.$$

So there exist $K_1 \in (0, F_0(q))$ and $K_2 \in (F_0(q), \infty)$ such that $Q(K_1) = Q(K_2) = 1$. A diagram illustrating this scenario is shown in Fig. 2. Therefore, we conclude that model (6) at least has two endemic steady states.

Theorem 4.2 indicates that a backward bifurcation may occur when $R_0 < 1$. Now, we apply the Lyapunov–Schmidt methods and techniques in previous studies [Li *et al.*, 2020; Okuwa *et al.*, 2021; Martcheva & Inaba, 2020; Yang *et al.*, 2022] to conduct a detailed investigation for the existence of a backward bifurcation. ■

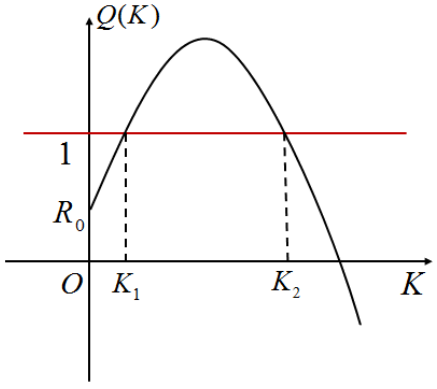

Fig. 2. Model (6) has two endemic equilibria for $R_0 < 1$.

We introduce a branching parameter $\overline{\beta}$ by

$$\beta(\tau) = \overline{\beta}\beta_0(\tau),$$

where $\beta_0(\tau)$ satisfies

$$1 = p\int_0^\infty \beta_0(a)$$

$$\times \int_0^a c(\tau)\frac{\pi_1(a)}{\pi_1(\tau)}d\tau da + \sigma(1-p)$$

$$\times \int_0^\infty \beta_0(a)\int_0^a c(\tau)\frac{\pi_2(a)}{\pi_2(\tau)}d\tau da. \quad (24)$$

Then we have $\overline{\beta} = 1 \Leftrightarrow R_0 = 1$. Here, we define

$$X = L^1(0,\infty)\times\mathbb{R}\times L^1(0,\infty)\times\mathbb{R}\times L^1(0,\infty)$$
$$\times\mathbb{R}\times L^1(0,\infty)\times\mathbb{R},$$
$$X_0 = L^1(0,\infty)\times\{0\}\times L^1(0,\infty)\times\{0\}$$
$$\times L^1(0,\infty)\times\{0\}\times L^1(0,\infty)\times\{0\},$$
$$X_+ = L^1_+(0,\infty)\times\mathbb{R}_+\times L^1_+(0,\infty)\times\mathbb{R}_+$$
$$\times L^1_+(0,\infty)\times\mathbb{R}_+\times L^1_+(0,\infty)\times\mathbb{R}_+,$$
$$X_{0+} = X_0\cap X_+, \quad \phi = (x_u, 0, y, 0, x_v, 0, z, 0)^T.$$

Consider model (6). We define a nonlinear operator $D : X_{0+}\to X_{0+}$ as follows:

$$D(\phi,\overline{\beta}) = \begin{pmatrix} -x_{ua} - \overline{\beta}c(a)\int_0^\infty \beta_0(u)[y(u)+z(u)]du\cdot x_u(a) \\ p - x_u(0) \\ -y_a + \overline{\beta}c(a)\int_0^\infty \beta_0(u)[y(u)+z(u)]du\cdot x_u(a) - \gamma_1(a)y(a) \\ -y(0) \\ -x_{va} + \gamma_1(a)y(a) - \sigma\overline{\beta}c(a)\int_0^\infty \beta_0(u)[y(u)+z(u)]du\cdot x_v(a) + \gamma_2(a)z(a) \\ 1-p-x_v(0) \\ -z_a + \sigma\overline{\beta}c(a)\int_0^\infty \beta_0(u)[y(u)+z(u)]du\cdot x_v(a) - \gamma_2(a)z(a) \\ -z(0) \end{pmatrix}.$$

The derivative of the operator D is given below and applied to any element $h = (e,0,g,0,k,0,l,0)^T \in X_{0+}$, with

$$D_\phi(\phi,\overline{\beta})h = \begin{pmatrix} -e_a - Q_1 \\ -e(0) \\ -g_a + Q_1 - \gamma_1(a)g_1(a) \\ -g(0) \\ -k_a + \gamma_1(a)g(a) - \sigma Q_2 + \gamma_2(a)l(a) \\ -k(0) \\ -l_a + \sigma Q_2 - \gamma_2(a)l(a) \\ -l(0) \end{pmatrix}$$

and

$$Q_1 = \overline{\beta}c(a)e(a)\int_0^\infty \beta_0(u)[y(u)+z(u)]du + \overline{\beta}c(a)x_u(a)\int_0^\infty \beta_0(u)[g(u)+l(u)]du,$$

$$Q_2 = \overline{\beta}c(a)k(a)\int_0^\infty \beta_0(u)[y(u)+z(u)]du + \overline{\beta}c(a)x_v(a)\int_0^\infty \beta_0(u)[g(u)+l(u)]du.$$

Computing the derivative of the operator D at $\phi^0 = (p,0,0,0,1-p,0,0,0)^T$, we obtain

$$D_\phi(\phi^0,\overline{\beta})h = \begin{pmatrix} -e_a - p\overline{\beta}c(a)\int_0^\infty \beta_0(u)[g(u)+l(u)]du \\ -e(0) \\ -g_a + p\overline{\beta}c(a)\int_0^\infty \beta_0(u)[g(u)+l(u)]du - \gamma_1(a)g(a) \\ -g(0) \\ -k_a + \gamma_1(a)g(a) - \sigma(1-p)\overline{\beta}c(a)\int_0^\infty \beta_0(u)[g(u)+l(u)]du + \gamma_2(a)l(a) \\ -k(0) \\ -l_a + \sigma(1-p)\overline{\beta}c(a)\int_0^\infty \beta_0(u)[g(u)+l(u)]du - \gamma_2(a)l(a) \\ -l(0) \end{pmatrix}.$$

Define a linear operator $\mathcal{A} = D_\phi(\phi^0,1) : X_0 \to X$. In order to calculate the eigenvector of $\mathcal{A}$ associated with eigenvalue 0, we solve the characteristic equation

$$\mathcal{A}h = 0.$$

For the sake of convenience, let

$$\Sigma = \int_0^\infty \beta_0(u)(g(u)+l(u))du.$$

Solving g and l, we obtain

$$g(a) = p\Sigma\int_0^a c(\xi)e^{-\int_\xi^a \gamma_1(\eta)d\eta}d\xi,$$

$$l(a) = \sigma(1-p)\Sigma\int_0^a c(\xi)e^{-\int_\xi^a \gamma_2(\eta)d\eta}d\xi.$$

We define

$$\hat{g}_0(a) = p\int_0^a c(\xi)e^{-\int_\xi^a \gamma_1(\eta)d\eta}d\xi,$$

$$\hat{l}_0(a) = \sigma(1-p)\int_0^a c(\xi)e^{-\int_\xi^a \gamma_2(\eta)d\eta}d\xi.$$

Substituting $g(a)$ and $l(a)$ into Σ, we have

$$\begin{aligned}\Sigma &= \int_0^\infty \beta_0(a)[y(a)+z(a)]da \\ &= \Sigma\left(p\int_0^\infty \beta_0(a)\int_0^a c(\xi)e^{-\int_\xi^a \gamma_1(\eta)d\eta}d\xi da \right. \\ &\quad \left. + \sigma(1-p)\int_0^\infty \beta_0(a)\int_0^a c(\xi)e^{-\int_\xi^a \gamma_2(\eta)d\eta}d\xi da\right) \\ &= \Sigma\int_0^\infty \beta_0(a)[\hat{g}_0(a)+\hat{l}_0(a)]da.\end{aligned}$$

If $\Sigma \neq 0$, then

$$1 = \int_0^\infty \beta_0(a)[\hat{g}_0(a)+\hat{l}_0(a)]da,$$

which trivially holds according to (24). Similarly, we also can give other variables

$$\hat{e}_0(a) = -p\int_0^a c(\xi)d\xi,$$

$$\begin{aligned}\hat{k}_0(a) = \int_0^a \Bigg[&-\sigma(1-p)c(\xi) + p\gamma_1(\xi)\int_0^\xi c(\theta) \\ &\times e^{-\int_\theta^\xi \gamma_1(\eta)d\eta}d\theta + \sigma(1-p)\gamma_2(\xi) \\ &\times \int_0^\xi c(\theta)e^{-\int_\theta^\xi \gamma_2(\eta)d\eta}d\theta\Bigg]d\xi.\end{aligned}$$

Hence, $\mathcal{A}h = 0$ has an eigenvector

$$\hat{v}_0 = (\hat{e}_0, 0, \hat{g}_0, 0, \hat{k}_0, 0, \hat{l}_0, 0)^T.$$

(*Note*: *Just as in the ODE case, the components of the eigenvalue* $\hat{v}_0$ *corresponding to positive entries in the disease-free equilibrium may be negative. This is, in general, the case with the PDEs as well.*)

Next, we need to find the adjoint operator $\mathcal{A}^*$. To achieve this goal, we let

$$\psi = (\psi_1, \psi_2, \psi_3, \psi_4, \psi_5, \psi_6, \psi_7, \psi_8)^T \in X^*$$

such that $\langle \mathcal{A}h, \psi, \rangle = \langle h, \mathcal{A}^*\psi \rangle$.

Then we obtain the adjoint operator

$$\mathcal{A}^*\psi = \begin{pmatrix} \psi_{1a} \\ \psi_1(0) \\ A_1 \\ \psi_3(0) \\ \psi_{5a} \\ \psi_5(0) \\ A_2 \\ \psi_7(0) \end{pmatrix}, \tag{25}$$

where

$$\begin{aligned} A_1 &= \psi_{3a} - p\beta_0(a)\int_0^\infty c(\xi)\psi_1(\xi)d\xi + p\beta_0(a)\int_0^\infty c(\xi)\psi_3(\xi)d\xi - \sigma(1-p)\beta_0(a)\int_0^\infty c(\xi)\psi_5(\xi)d\xi \\ &\quad + \sigma(1-p)\beta_0(a)\int_0^\infty c(\xi)\psi_7(\xi)d\xi - \gamma_1(a)\psi_3(a) + \gamma_1(a)\psi_5(a), \\ A_2 &= \psi_{7a} - p\beta_0(a)\int_0^\infty c(\xi)\psi_1(\xi)d\xi + p\beta_0(a)\int_0^\infty c(\xi)\psi_3(\xi)d\xi - \sigma(1-p)\beta_0(a)\int_0^\infty c(\xi)\psi_5(\xi)d\xi \\ &\quad + \sigma(1-p)\beta_0(a)\int_0^\infty c(\xi)\psi_7(\xi)d\xi - \gamma_2(a)\psi_7(a) + \gamma_a\psi_5(a). \end{aligned}$$

Now, we calculate the eigenvector of $\mathcal{A}^*$ associated with eigenvalue 0. Setting $\mathcal{A}^*\psi = 0$. Solving ψ_3 and ψ_7, we obtain

$$\psi_3(a) = \Sigma_1\int_a^\infty \beta_0(\tau)e^{\int_\tau^a \gamma_1(\eta)d\eta}d\tau, \quad \psi_7(a) = \Sigma_1\int_a^\infty \beta_0(\tau)e^{\int_\tau^a \gamma_2(\eta)d\eta}d\tau,$$

where

$$\Sigma_1 = \int_0^\infty c(\xi)[p\psi_3(\xi) + \sigma(1-p)\psi_7(\xi)]d\xi.$$

Substituting $\psi_3(a)$ and $\psi_7(a)$ into Σ_1, we have

$$\begin{aligned} \Sigma_1 &= \int_0^\infty c(\xi)[p\psi_3(\xi) + \sigma(1-p)\psi_7(\xi)]d\xi \\ &= \Sigma_1\left(p\int_0^\infty\int_0^\tau \beta_0(\tau)c(\xi)e^{-\int_\xi^\tau \gamma_1(\eta)d\eta}d\xi d\tau + \sigma(1-p)\int_0^\infty\int_0^\tau \beta_0(\tau)c(\xi)e^{-\int_\xi^\tau \gamma_2(\eta)d\eta}d\xi d\tau\right) \\ &= \Sigma_1\int_0^\infty c(\xi)[p\hat{\phi}_3^*(\xi) + \sigma(1-p)\hat{\phi}_7^*(\xi)]d\xi, \end{aligned}$$

where

$$\psi_3^*(a) = \int_a^\infty \beta_0(\tau)e^{\int_\tau^a \gamma_1(\eta)d\eta}d\tau, \quad \psi_7^*(a) = \int_a^\infty \beta_0(\tau)e^{\int_\tau^a \gamma_2(\eta)d\eta}d\tau.$$

If $\Sigma_1 \neq 0$, then

$$1 = \int_0^\infty c(\xi)[p\hat{\phi}_3^*(\xi) + \sigma(1-p)\hat{\phi}_7^*(\xi)]d\xi,$$

which trivially holds according to (24). Thus, we can obtain the eigenvector $\hat{v}_0^*$ of $\mathcal{A}^*$ associated with eigenvalue 0 as follows:

$$\hat{v}_0^* = (0, 0, \hat{\psi}_3^*(a), \hat{\psi}_3^*(0), 0, 0, \hat{\psi}_7^*(a), \hat{\psi}_7^*(0)).$$

Computing the second derivative for D, we have

$$D_{\phi\overline{\beta}}(\phi, \overline{\beta})h = \begin{pmatrix} -c(a)\left[e(a)\int_0^\infty \beta_0(u)(y(u)+z(u))du + x_u(a)\int_0^\infty \beta_0(u)(g(u)+l(u))du\right] \\ 0 \\ c(a)\left[e(a)\int_0^\infty \beta_0(u)(y(u)+z(u))du + x_u(a)\int_0^\infty \beta_0(u)(g(u)+l(u))du\right] \\ 0 \\ -\sigma c(a)\left[k(a)\int_0^\infty \beta_0(u)(y(u)+z(u))du + x_v(a)\int_0^\infty \beta_0(u)(g(u)+l(u))du\right] \\ 0 \\ \sigma c(a)\left[k(a)\int_0^\infty \beta_0(u)(y(u)+z(u))du + x_v(a)\int_0^\infty \beta_0(u)(g(u)+l(u))du\right] \\ 0 \end{pmatrix}.$$

Therefore, we obtain

$$\begin{aligned} \mathfrak{b} &\overset{\text{def}}{=} \langle F_{\phi\overline{\beta}}(\phi^0, 1)\widehat{v}_0, \widehat{v}_0^*\rangle \\ &= p\int_0^\infty c(a)\int_0^\infty \beta_0(u)(\hat{g}_0(u)+\hat{l}_0(u))du\cdot\psi_3^*(a)da + \sigma(1-p)\int_0^\infty c(a)\int_0^\infty \beta_0(u) \\ &\quad \times(\hat{g}_0(u)+\hat{l}_0(u))du\cdot\psi_7^*(a)da \\ &= \int_0^\infty c(a)[p\psi_3^*(a)+\sigma(1-p)\psi_7^*(a)]da = 1 > 0. \end{aligned}$$

The derivative of the operator F applied to the element $h_2 = (e_2, 0, g_2, 0, k_2, 0, l_2, 0)$ can be computed as follows:

$$D_{\phi\phi}(\phi^0, \overline{\beta})[h, h_2] = \begin{pmatrix} -\overline{\beta}c(a)\int_0^\infty \beta_0(u)(e(a)(g_2(u)+l_2(u)) + e_2(a)(g(u)+l(u)))du \\ 0 \\ \overline{\beta}c(a)\int_0^\infty \beta_0(u)(e(a)(g_2(u)+l_2(u)) + e_2(a)(g(u)+l(u)))du \\ 0 \\ -\sigma\overline{\beta}c(a)\int_0^\infty \beta_0(u)(k(a)(g_2(u)+l_2(u)) + k_2(a)(g(u)+l(u)))du \\ 0 \\ \sigma\overline{\beta}c(a)\int_0^\infty \beta_0(u)(k(a)(g_2(u)+l_2(u)) + k_2(a)(g(u)+l(u)))du \\ 0 \end{pmatrix}.$$

Hence,

$$\begin{aligned}\mathfrak{a} &\stackrel{\text{def}}{=} \langle D_{\phi\phi}(\phi^0,1)[\hat{v}_0,\hat{v}_0],\hat{v}_0^*\rangle \\ &= 2\int_0^\infty c(a)\hat{e}_0(a)\int_0^\infty \beta_0(u)(\hat{g}_0(u)+\hat{l}_0(u))du \\ &\quad\cdot\psi_3^*(a)da + 2\sigma\int_0^\infty c(a)\hat{k}_0(a) \\ &\quad\times\int_0^\infty \beta_0(u)(\hat{g}_0(u)+\hat{l}_0(u))du\cdot\psi_7^*(a)da \\ &= 2\int_0^\infty c(a)[\hat{e}_0(a)\hat{\psi}_3^*(a)+\sigma\hat{k}_0(a)\hat{\psi}_7^*(a)]da.\end{aligned}$$

Lemma 4.1 (see [Martcheva & Inaba, 2020, Theorem 2.1]). *Assume*

(A1) $\mathcal{A}$ *is a closed operator with a simple isolated eigenvalue zero and remaining eigenvalues having negative real parts.* $\hat{v}_0$ *is the unique solution of* $\mathcal{A}v=0$.
(A2) $D(\phi,\overline{\beta})\in C^2(U_0\times I_0,X)$ *for some neighborhood* U_0 *of* ϕ^0 *and internal* I_0 *contains 1.*
(A3) $\hat{v}_0^*\in X^*$ *is the unique positive solution of* $\langle\mathcal{A}\phi,\hat{v}_0^*\rangle=0$, *which indicates that* $\dim(\ker\mathcal{A}^*)=1$, *where* $\ker\mathcal{A}^*=\text{span}\{\hat{v}_0^*\}$.
(A4) $\langle D_{\phi,\overline{\beta}}(\phi^0,1)\hat{v}_0,\hat{v}_0^*\rangle\neq 0$, *where* $D_{\phi,\overline{\beta}}(\phi^0,1)$ *is the second derivative of* D *with respect to* ϕ *and* $\overline{\beta}$.

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

In summary, we obtain the following results.

Theorem 4.3. *Model (6) undergoes a backward bifurcation when* $R_0<1$ *and the following condition holds:*

$$\begin{aligned}&\sigma p\int_0^a c(\xi)d\xi\int_a^\infty \beta_0(\tau)e^{\int_\tau^a\gamma_2(\eta)d\eta}d\tau \\ &\quad>\int_0^a\sigma c(\xi)[\sigma(1-p)e^{-\int_\xi^a\gamma_2(\eta)d\eta} \\ &\quad\quad+pe^{-\int_\xi^a\gamma_1(\eta)d\eta}]d\xi \\ &\quad\quad\times\int_a^\infty\beta_0(\tau)e^{\int_\tau^a\gamma_2(\eta)d\eta}d\tau \\ &\quad\quad+p\int_0^a c(\xi)d\xi\int_a^\infty\beta_0(\tau)e^{\int_\tau^a\gamma_1(\eta)d\eta}d\tau.\end{aligned}$$

Proof. After some manipulation, we obtain

$$\begin{aligned}\hat{k}_0(a) &= \int_0^a c(\xi)[p-\sigma(1-p)e^{-\int_\xi^a\gamma_2(\eta)d\eta} \\ &\quad -pe^{-\int_\xi^a\gamma_1(\eta)d\eta}]d\xi.\end{aligned}$$

Thus,

$$\begin{aligned}&\sigma\hat{k}_0(a)\hat{\psi}_7^*(a)+\hat{e}_0(a)\hat{\psi}_3^*(a) \\ &\quad=\int_0^a\sigma c(\xi)[p-\sigma(1-p)e^{-\int_\xi^a\gamma_2(\eta)d\eta} \\ &\quad\quad-pe^{-\int_\xi^a\gamma_1(\eta)d\eta}]d\xi\int_a^\infty\beta_0(\tau)e^{\int_\tau^a\gamma_2(\eta)d\eta}d\tau \\ &\quad\quad-p\int_0^a c(\xi)d\xi\int_a^\infty\beta_0(\tau)e^{\int_\tau^a\gamma_1(\eta)d\eta}d\tau.\end{aligned}$$

Hence, if the conditions of Theorem 4.3 hold, then $\mathfrak{a}>0$. From Theorem 2.1 in [Martcheva & Inaba, 2020], it follows that model (6) exhibits a backward bifurcation. ■

5. Discussion

In this paper, a simple SIS compartmental model with age-structure is proposed to study the occurrence of multiple infections associated with a single disease. The model introduces different compartments to account for the primary and secondary infections, while incorporating the effects of temporary immunity and different susceptibility levels in the host population. The model allows us to conduct a deep investigation into the complex interplay between multiple infections, incomplete immunity, host heterogeneity, and age-dependent disease dynamics.

Through a detailed mathematical study, we have derived the expression of the basic reproduction number R_0 of the model and analyzed the properties of the trivial and nontrivial equilibrium solutions of the proposed model. Our novel findings include the following: (1) The disease-free equilibrium is globally asymptotically stable when $\frac{1}{\sigma(1-p)+p}R_0<1$, where $(1-p)$ is the portion of newborns who are immune to the first infection; (2) A backward bifurcation may occur under certain conditions. These results are different from typical conclusions of prior age-structured epidemic models with vaccination [Castillo-Chavez & Feng, 1998; Castillo Chavez *et al.*, 1989; Li & Gupur, 2004]. Our results explain why susceptibility to and relapse of many infectious diseases, such as mosquito-borne diseases, are frequently age-dependent. Moreover,

the occurrence of multiple infections may result in backward bifurcation phenomena [Inaba, 2017; Li *et al.*, 2020; Chala & Hamde, 2021].

These results could provide useful guidelines for policy development in public health. For example, suppose that vaccination is used as a major intervention strategy to control an infectious disease and that a portion $(1-p)$ of newborns are vaccinated. Our analytical result (1) stated above shows that the higher the value of p, the larger the domain of the global asymptotic stability for the disease-free equilibrium. Particularly, if $p = 100\%$ (i.e. vaccination coverage is 0), then reducing R_0 below unity would be sufficient to eradicate the disease since the disease-free equilibrium is globally asymptotically stable for $R_0 < 1$. In contrast, if $p = 0$ (i.e. vaccination coverage is 100%), then the global stability domain for the disease-free equilibrium is reduced to $R_0 < \sigma$, where $\sigma < 1$, indicating that control efforts stronger than reducing R_0 below unity by means of other intervention methods would be required to eliminate the disease. This result is somehow counter-intuitive and implies that, although high coverage of a vaccination campaign is generally beneficial in containing the primary infection, it may introduce additional complications (such as possible backward bifurcations) in fighting the disease in the long run, partly due to the waning immunity of the host population. In this regard, the vaccination campaign should be combined with other prevention and intervention methods in order to effectively control the infection and even possibly achieve the goal of eradicating such a disease. Additionally, practical vaccination coverage is always somewhere between 0% and 100%, and the value of the parameter σ, representing the reduction of infectivity in the secondary infection due to immune effects, also plays a role in shaping the degree of disease control measures.

This paper focused on mathematical analysis of the rich dynamics associated with our proposed model. This modeling study can be extended to practical applications in public health and combined with epidemic and surveillance data, if available, for specific diseases, using the mathematical understanding gained from our theoretical analysis to provide meaningful guidelines for effective disease prevention and intervention. On the other hand, although we have analyzed the influence of age-structure, multiple infections and vaccination strategies in the model on the disease spread, there are still other factors worth considering, such as the incubation delay [Brauer *et al.*, 2008, Gupta *et al.*, 2023], complex networks [Chang *et al.*, 2020; Jin *et al.*, 2014], and multistrains [Li *et al.*, 2020; Goel *et al.*, 2023]. We leave these for future research.

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