Global threshold dynamics of an SIVS model with waning vaccine-induced immunity and nonlinear incidence

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Abstract

Vaccination is the most effective method of preventing the spread of infectious diseases. For many diseases, vaccine-induced immunity is not life long and the duration of immunity is not always fixed. In this paper, we propose an SIVS model taking the waning of vaccine-induced immunity and general nonlinear incidence into consideration. Our analysis shows that the model exhibits global threshold dynamics in the sense that if the basic reproduction number is less than 1, then the disease-free equilibrium is globally asymptotically stable implying the disease dies out; while if the basic reproduction number is larger than 1, then the endemic equilibrium is globally asymptotically stable indicating that the disease persists. This global threshold result indicates that if the vaccination coverage rate is below a critical value, then the disease always persists and only if the vaccination coverage rate is above the critical value, the disease can be eradicated.

Key words: Waning immunity; vaccination age; nonlinear incidence; Lyapunov functional.

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1 Introduction

Mathematical models describing the dynamics of infectious diseases are of great public health importance because they provide insights on implementing practical and efficient disease-control strategies [1]. Vaccination is one of the most effective methods to prevent and control the spread of many infections [2–5]. Many early epidemic models assumed that vaccination imparts permanent immunity [6–8]. The baseline assumption for most compartmental models incorporating vaccination is that individuals in each compartment are homogeneously mixed. For example, Liu et al. [9] proposed two SVIR models with permanent immunity to investigate the impacts of both continuous and impulsive vaccination policies. Xiao and Tang [10] built a simple SIV model including susceptible, infected and imperfectly vaccinated classes and assuming a nonlinear incidence rate. They showed that complex dynamics can be induced by imperfect vaccination. Gao et al. [11] formulated an SEIRS model and examined the possibility of eradicating an infectious disease by implementing the pulse vaccination strategy.

It has been recognized that the waning of vaccine-induced immunity has been one of the major causes for reemergence of some childhood diseases such as measles, rubella and pertussis [12–14]. For chickenpox, the varicella vaccine only provides temporary immunity, permanent immunity can only be gained via recovery from natural chickenpox disease. For those previously vaccinated children, if the vaccine-induced immunity is under protective level or completely lost, which can be identified by an antibody titer test, then they may become vulnerable to the infections again [15]. It is thus of great importance to incorporate the waning of vaccine-induced immunity into disease modeling to understand how the waning of vaccine-induced immunity impacts the disease dynamics. In this regard, some delay differential equation models have been proposed under the assumption that the vaccine-induced immunity lasts for a fixed time period [16]. If the immunity duration is not fixed, similar to the age-of-infection [17], the age-of-vaccination is taken into consideration. This usually results in a partial differential equation model. See, for example, [18–21].

Another crucial aspect in disease modeling is understanding how the population behavior and the infectivity of the disease impact the disease dynamics [22,23]. Mathematically, this can be captured by the incidence rate of a disease, defined as the average number of new cases per unit of time. Two typical incidence rates are the bilinear (or mass action) and the standard incidence rates [24]. It has been commonly accepted that the bilinear incidence rate βSI (where β is the transmission rate, S is the Susceptible, I is the infected) is more appropriate for communicable diseases such as H5N1 [25], SARS [26], Hand, foot, and mouth disease [27], but not for sexually transmitted infections [28];

while the standard incidence rate $\frac{\beta SI}{S+I}$ seems to be a good approximation if the number of available partners is large but each individual cannot make more contacts than is practically feasible. In practice, these two limiting incidence rates are both over simplified and cannot capture some important features of disease transmission. For example, if the number of infectious individuals is too large, then there should be a saturation effect. Thus a saturated incidence rate $Sf(I)$ with $f(I) = \frac{I}{1+\alpha I}$ seems more appropriate[29]. For models with various nonlinear incidence rates, we refer to [30–34].

In this work, we consider an SIRVS model that takes both the waning of vaccine-induced immunity and nonlinear incidence into consideration. More specifically, the nonlinear incidence is assumed to be of the form $Sf(I)$ with f satisfying

(A1) For $x \in \mathbb{R}_+$, $f(x) \geq 0$ with equality if and only if $x = 0$, $f'(x) \geq 0$, and $f''(x) \leq 0$.

Note that the same assumption has also been made in [35,36]. Typical such functions include $f(I) = \beta I$, and $f(I) = \frac{\beta I}{1+\alpha I}$.

It follows from Assumption (A1) and the Mean Value Theorem that

$$f'(x)x \leq f(x) \leq f'(0)x \text{ for } x \in \mathbb{R}_+. \tag{1.1}$$

The above inequality will be used in proving our main result. Indeed it can be used to modify the arguments for the case with bilinear incidence rate considered in [18–20,37].

The population under consideration is classified into four disjoint compartments, namely, susceptible, infected, recovered and vaccinated classes. We use $S(t)$, $I(t)$ and $R(t)$ to denote the population sizes of susceptible compartment, infected compartment, and recovered compartment at time t, respectively. It is assumed that susceptible individuals are vaccinated at the rate of $\phi \geq 0$. We let $v(t, a)$ denote the population size of the vaccinated compartment at time t with the vaccination age a and assume that vaccine-induced immunity wanes at the rate of $\varepsilon(a)$, which satisfies the following property

(A2) $\varepsilon : [0, \infty) \to [0, \infty)$ is bounded, nondecreasing and piecewise continuous with possibly finite many jumps.

Further, we assume that all newly recruited individuals, including the newborns, are susceptible and the recruitment rate is $\Lambda > 0$. The natural death rate is $\mu > 0$ and the disease induced death rate is δ. The infected individuals are assumed to enter the recovered class at a rate of γ. Since for some diseases such as chickenpox, the recovered individuals gain permanent disease-induced immunity and we focus on how the waning of vaccine-induced immunity affects

the disease dynamics, in this work, we assume that the recovered individuals do not enter the susceptible class.

Based on the above assumptions, our SIVS model with vaccination-age and nonlinear incidence is described by

$$\begin{cases} \frac{dS(t)}{dt} = \Lambda - \mu S(t) - S(t)f(I(t)) - \phi S(t) + \int_0^\infty \varepsilon(a)v(t,a)da, \\ \frac{dI(t)}{dt} = S(t)f(I(t)) - (\mu + \gamma + \delta)I(t), \\ \frac{dR(t)}{dt} = \gamma I(t) - \mu R(t), \\ \frac{\partial v(t,a)}{\partial t} + \frac{\partial v(t,a)}{\partial a} = -(\mu + \varepsilon(a))v(t,a), \\ v(t,0) = \phi S(t), \\ S(0) = S_0 \geq 0, I(0) = I_0 > 0, R(0) = R_0 \geq 0, v(0,\cdot) = v_0(\cdot) \in L^1_+, \end{cases} \tag{1.2}$$

where L^1_+ is the set of integrable functions from $(0,\infty)$ into $\mathbb{R}_+ = [0,\infty)$.

Note that the third equation of system (1.2) does not appear in the remaining three equations. This allows us to consider the following sub-system

$$\begin{cases} \frac{dS(t)}{dt} = \Lambda - \mu S(t) - S(t)f(I(t)) - \phi S(t) + \int_0^\infty \varepsilon(a)v(t,a)da, \\ \frac{dI(t)}{dt} = S(t)f(I(t)) - (\mu + \gamma + \delta)I(t), \\ \frac{\partial v(t,a)}{\partial t} + \frac{\partial v(t,a)}{\partial a} = -(\mu + \varepsilon(a))v(t,a), \\ v(t,0) = \phi S(t), \\ S(0) = S_0 \geq 0, I(0) = I_0 > 0, v(0,\cdot) = v_0(\cdot) \in L^1_+, \end{cases} \tag{1.3}$$

PDE models with bilinear incidence rates related to (1.3) have been extensively studied in the literature. For some recent work, we refer to [19–21,38–40] and the references therein. Establishing global dynamics of PDE models with age-structure has been a challenging task due to the lack of well-established tools. The difficulties increase when the PDE disease model has a nonlinear incidence rate. The purpose of this paper is to make an attempt to establish global dynamics for (1.3).

It follows from [41,42] that system (1.3) has a unique continuous solution if the initial conditions satisfy the compatibility condition

$$v_0(0) = \phi S_0$$

In the sequel, we always assume the above compatibility condition is satisfied. Tools from Browne and Pilyugin [43] can be employed to show that the solution of (1.3) exists on $\mathbb{R}_+$ and it is nonnegative. Thus we can define a solution

semiflow $\Phi : \mathbb{R}_+ \times (\mathbb{R}_+ \times \mathbb{R}_+ \times L^1_+) \to \mathbb{R}_+ \times \mathbb{R}_+ \times L^1_+$ of (1.3) as

$$\Phi(t, (S_0, I_0, v_0)) = (S(t), I(t), v(t, \cdot))$$

for $t \in \mathbb{R}_+$ and $(S_0, I_0, v_0) \in \mathbb{R}_+ \times \mathbb{R}_+ \times L^1_+$.

Let $N(t) = S(t) + I(t) + \int_0^\infty v(t, a)da$, then it follows from (1.3) that

$$\frac{dN(t)}{dt} \leq \Lambda - \mu N(t),$$

which implies that $\limsup_{t\to\infty} N(t) \leq \frac{\Lambda}{\mu}$. Let

$$\Gamma = \{(S_0, I_0, v_0(\cdot)) \in \mathbb{R}_+ \times \mathbb{R}_+ \times L^1_+ : \ v_0(0) = \phi S_0, \ N(0) \leq \tfrac{\Lambda}{\mu}\}.$$

Then it is straightforward to show that Γ is positively invariant and attracts all positive solutions of Φ of (1.3). Therefore, in the following, we restrict our attention to solutions of (1.3) with initial conditions in Γ.

The rest of this paper is organized as follows. In Section 2, we discuss the existence and local stability of equilibria of (1.3). In Section 3, we establish the global dynamics of (1.3). We conclude this work in Section 4 with some discussions.

2 Existence and local stability of equilibria

Define $\pi : \mathbb{R}_+ \to \mathbb{R}_+$ as

$$\pi(a) = e^{-\mu a - \int_0^a \varepsilon(s)ds}$$

and

$$K = \int_0^\infty \varepsilon(a)\pi(a)da.$$

Then

$$K \leq \int_0^\infty \varepsilon(a)e^{-\int_0^a \varepsilon(s)ds}da = 1. \tag{2.1}$$

An equilibrium $(\bar{S}, \bar{I}, \bar{v}(\cdot))$ of (1.3) must satisfy the following equations

$$\begin{cases} 0 = \Lambda - \mu\bar{S} - \bar{S}f(\bar{I}) - \phi\bar{S} + \int_0^\infty \varepsilon(a)\bar{v}(a)da, \\ 0 = \bar{S}f(\bar{I}) - (\mu + \gamma + \delta)\bar{I}, \\ \frac{d\bar{v}(a)}{da} = -(\mu + \varepsilon(a))\bar{v}(a), \\ \bar{v}(0) = \phi\bar{S}. \end{cases} \tag{2.2}$$

It follows from the third and the fourth equations of (2.2) that

$$\bar{v}(a) = \phi \bar{S} \pi(a),$$

while by the first and the third equations of (2.2), we obtain

$$\bar{S} = \frac{\Lambda}{f(\bar{I}) + \mu + \phi(1-K)}.$$

Substituting the above into the second equation of (2.2) yields $g(\bar{I}) = 0$, where

$$g(I) \triangleq f(I)[\Lambda - (\mu + \gamma + \delta)I] - (\mu + \delta)[\mu + \phi(1-K)]I.$$

By (A1), $\bar{I} = 0$ clearly is a solution to $g(I) = 0$. Thus

$$E_0 = \left(\frac{\Lambda}{\mu + \phi(1-K)}, 0, \frac{\phi\Lambda}{\mu + \phi(1-K)} \pi(a) \right)$$

is the disease-free equilibrium.

In order to obtain the condition for the existence of an endemic equilibrium, we define the basic reproduction number as

$$\mathcal{R}_0 = \frac{\Lambda f'(0)}{(\mu + \gamma + \delta)(\mu + \phi(1-K))}. \tag{2.3}$$

In epidemiology, the basic reproduction number $\mathcal{R}_0$ gives the average number of cases that one typical infectious individual generates, if introduced into a susceptible population, over the whole infectious period $\frac{1}{\mu+\gamma+\delta}$.

If $(S^*, I^*, v^*(\cdot))$ is an endemic equilibrium, then we must have $I^* \in (0, \frac{\Lambda}{\mu+\delta})$ satisfying $h(I^*) = 0$, where

$$h(I) = \frac{g(I)}{I} = \frac{f(I)}{I}[\Lambda - (\mu + \gamma + \delta)I] - (\mu + \delta)[\mu + \phi(1-K)].$$

Note that

$$\begin{aligned} \lim_{I \to 0^+} h(I) &= f'(0)\Lambda - (\mu + \gamma + \delta)[\mu + \phi(1-K)] \\ &= (\mu + \gamma + \delta)[\mu + \phi(1-K)](R_0 - 1) \end{aligned}$$

and

$$\lim_{I \to \frac{\Lambda}{\mu+\gamma+\delta}-} h(I) = -(\mu + \gamma + \delta)[\mu + \phi(1-K)] < 0 \text{ (since } K < 1).$$

Moreover, for $I \in (0, \frac{\Lambda}{\mu+\gamma+\delta})$, it follows from (1.1) that

$$h'(I) = \frac{[\Lambda - (\mu + \gamma + \delta)I][f'(I)I - f(I)] - f(I)(\mu + \gamma + \delta)I}{I^2} < 0.$$

Therefore, $h(I) = 0$ has an unique positive solution in $(0, \frac{\Lambda}{\mu+\gamma+\delta})$ if and only if $\mathcal{R}_0 > 1$. Let I^* be a unique solution of $h(I) = 0$ in $(0, \frac{\Lambda}{\mu+\gamma+\delta})$. Then system (1.3) admits a unique endemic equilibrium $E^* = (S^*, I^*, \phi S^* \pi(a))$, where $S^* = \frac{\Lambda}{f(I^*)+\mu+\phi(1-K)}$.

Summarizing the above analysis, we have the following result concerning the existence of equilibria.

Theorem 2.1 *Consider system* (1.3) *with* $\mathcal{R}_0$ *defined in (2.3). If* $\mathcal{R}_0 \leq 1$, *then there is a unique equilibrium, which is the disease-free equilibrium* E_0*; while if* $\mathcal{R}_0 > 1$, *then there are two equilibria, the disease-free equilibrium* E_0 *and the endemic equilibrium* E^*.

Next, we study the local stability of the equilibria obtained in Theorem 2.1. Linearizing system (1.3) at an equilibrium $\bar{E} = (\bar{S}, \bar{I}, \bar{v}(\cdot))$ deduces the associated characteristic equation as

$$0 = \begin{vmatrix} \lambda + \mu + f(\bar{I}) + \phi(1 - \hat{K}(\lambda)) & \bar{S} f'(\bar{I}) \\ -f(\bar{I}) & \lambda + \mu + \gamma + \delta - \bar{S} f'(\bar{I}) \end{vmatrix},$$

where

$$\hat{K}(\lambda) = \int_0^\infty \varepsilon(a)\pi(a)e^{-\lambda a} da.$$

Then the equilibrium $\bar{E}$ is locally (asymptotically) stable if all eigenvalues of the characteristic equation have negative real parts and it is unstable if at least one eigenvalue has a positive real part.

Theorem 2.2 *Consider system* (1.3) *with* $\mathcal{R}_0$ *defined in (2.3). If* $\mathcal{R}_0 < 1$, *then the disease-free equilibrium* E_0 *is locally asymptotically stable and if* $\mathcal{R}_0 > 1$, *the unique endemic equilibrium* E^* *is locally asymptotically stable.*

Proof. The characteristic equation at E_0 is

$$0 = \begin{vmatrix} \lambda + \mu + \phi(1 - \hat{K}(\lambda)) & f'(0)S^0 \\ 0 & \lambda + \mu + \gamma + \delta - S^0 f'(0) \end{vmatrix},$$

where $S^0 = \frac{\Lambda}{\mu+\phi(1-K)}$. It is clear that one eigenvalue is

$$\lambda = (\mu + \gamma + \delta)(\frac{S^0 f'(0)}{\mu + \gamma + \delta} - 1) = (\mu + \gamma + \delta)(R_0 - 1)$$

and all other eigenvalues satisfy

$$\lambda + \mu + \phi = \phi \hat{K}(\lambda). \tag{2.4}$$

We claim that all roots of (2.4) have negative real parts. Otherwise, let λ_0 be a root of (2.4) with $\mathrm{Re}(\lambda_0) \geq 0$. Then the module of the left hand side of (2.4) is $|\lambda_0 + \mu + \phi| > \phi$, while the module of the right hand side of (2.4) is $|\phi\hat{K}(\lambda_0)| \leq \phi K \leq \phi$. This leads to a contradiction. This proves our claim and hence E_0 is locally asymptotically stable when $\mathcal{R}_0 < 1$.

The characteristic equation at E^* is

$$
\begin{aligned}
0 = & \begin{vmatrix} \lambda + \mu + f(I^*) + \phi(1 - \hat{K}(\lambda)) & S^* f'(I^*) \\ -f(I^*) & \lambda + \mu + \gamma + \delta - S^* f'(I^*) \end{vmatrix} \\
= & \left[\lambda + \mu + \phi(1 - \hat{K}(\lambda))\right] (\lambda + \mu + \gamma + \delta - S^* f'(I^*)) \\
& + f(I^*)(\lambda + \mu + \gamma + \delta) \\
= & (\lambda + \mu + \phi(1 - \hat{K}(\lambda)) + f(I^*))(\lambda + \mu + \gamma + \delta) \\
& - S^* f'(I^*) \left[\lambda + \mu + \phi(1 - \hat{K}(\lambda))\right] \\
= & \left[\lambda + \mu + \phi(1 - \hat{K}(\lambda)) + f(I^*)\right] (\lambda + \mu + \gamma + \delta) \\
& - \frac{(\mu + \gamma + \delta) f'(I^*) I^*}{f(I^*)} \left[\lambda + \mu + \phi(1 - \hat{K}(\lambda))\right],
\end{aligned}
$$

where $S^* = \frac{(\mu+\gamma+\delta)I^*}{f(I^*)}$ was used. Next, we show that the characteristic equation has no eigenvalues with nonnegative real parts. By way of contradiction, assume that there is one eigenvalue λ_1 with $\mathrm{Re}(\lambda_1) \geq 0$. Then

$$
\left|1 + \frac{f(I^*)}{\lambda_1 + \mu + \phi(1 - \hat{K}(\lambda_1))}\right| |\lambda_1 + \mu + \gamma + \delta| = \left|\frac{(\mu + \gamma + \delta) f'(I^*) I^*}{f(I^*)}\right|. \quad (2.5)
$$

It follows from (1.1) and the right hand of (2.5) that

$$
\left|\frac{(\mu + \gamma + \delta) f'(I^*) I^*}{f(I^*)}\right| \leq \mu + \gamma + \delta.
$$

On the other hand, by the left hand of (2.5), we have

$$
\left|1 + \frac{f(I^*)}{\lambda_1 + \mu + \phi(1 - \hat{K}(\lambda_1))}\right| |\lambda_1 + \mu + \gamma + \delta| > \mu + \gamma + \delta,
$$

since $\left|1 + \frac{f(I^*)}{\lambda_1+\mu+\phi(1-\hat{K}(\lambda_1))}\right| > 1$. This leads to a contradiction. This completes the proof. $\square$

3 Global stability analysis

We use the Fluctuation Lemma to establish the global stability of the disease-free equilibrium E_0. To this end, we first introduce the notation

$$\psi_\infty = \liminf_{t\to\infty} \psi(t) \qquad \text{and} \qquad \psi^\infty = \limsup_{t\to\infty} \psi(t).$$

and then state the Fluctuation Lemma as below.

Lemma 3.1 (Fluctuation Lemma [44]) *Let $\psi : \mathbb{R}_+ \to \mathbb{R}$ be a bounded and continuously differentiable function. Then there exist sequences $\{s_n\}$ and $\{t_n\}$ such that $s_n \to \infty$, $t_n \to \infty$, $\psi(s_n) \to \psi_\infty$, $\psi'(s_n) \to 0$, $\psi(t_n) \to \psi^\infty$, and $\psi'(t_n) \to 0$ as $n \to \infty$.*

We also need the following lemma to prove our Theorem 3.3.

Lemma 3.2 ([41]) *Suppose $f : \mathbb{R}_+ \to \mathbb{R}$ is a bounded function. Then*

$$\limsup_{t\to\infty} \int_0^t k(\theta) f(t-\theta) d\theta \le f^\infty \|k\|_1.$$

where $\|k\|_1 = \int_0^\infty k(s)ds$.

Theorem 3.3 *If $\mathcal{R}_0 < 1$, then the disease-free equilibrium E_0 of (1.3) is globally asymptotically stable.*

Proof. It follows from Theorem 2.2 that it suffices to show that E_0 is attractive in Γ. Let $(S(t), I(t), v(t,a))$ be a solution of (1.3) with $(S_0, I_0, v_0(\cdot)) \in \Gamma$. Integrating the second equation of (1.3) with the boundary condition yields

$$v(t,a) = \begin{cases} \phi S(t-a)\pi(a), & t \ge a, \\ v_0(a-t)\frac{\pi(a)}{\pi(a-t)}, & t < a, \end{cases} \tag{3.1}$$

With the assistance of the Fluctuation Lemma, it is easy to get

$$S^\infty \le \frac{\Lambda}{\mu + \phi(1-K)}.$$

It follows from (1.1) that

$$\begin{aligned} \tfrac{dI(t)}{dt} &= S(t)f(I(t)) - (\mu+\gamma+\delta)I \\ &\le \tfrac{\Lambda}{\mu+\phi(1-K)} f'(0)I - (\mu+\gamma+\delta)I \\ &= (\mu+\gamma+\delta)[\mathcal{R}_0 - 1]I. \end{aligned}$$

This leads to $I^\infty \to 0$.

Lemma 3.1 implies that there exists a sequence $\{t_n\}$ such that $t_n \to \infty$, $S(t_n) \to S_\infty$, and $S'(t_n) \to 0$ as $n \to \infty$. Note that $\lim_{n\to\infty} I(t_n) = 0$. Thus

$$\begin{aligned}\frac{dS(t_n)}{dt} &= \Lambda - (\mu+\phi)S(t_n) + \phi\int_0^{t_n}\varepsilon(a)S(t_n-a)\pi(a)da \\ &\quad + \int_{t_n}^{\infty}\varepsilon(a)v_0(a-t_n)\frac{\pi(a)}{\pi(a-t_n)}da - S(t_n)f(I(t_n))\end{aligned}$$

and $\lim_{n\to\infty}\int_{t_n}^{\infty}\varepsilon(a)v_0(a-t_n)\frac{\pi(a)}{\pi(a-t_n)}da = 0$. Let $n \to \infty$, then

$$\begin{aligned}0 &\geq \Lambda - [\mu+\phi]S_\infty + \phi\int_0^{\infty}\varepsilon(a)S_\infty\pi(a)da - S_\infty f(I^\infty) \\ &= \Lambda - [\mu+\phi(1-K)]S_\infty - S_\infty f(I^\infty).\end{aligned}$$

Note that $I^\infty \to 0$, we then have $\frac{\Lambda}{\mu+\phi(1-K)} \leq S_\infty \leq S^\infty \leq \frac{\Lambda}{\mu+\phi(1-K)}$. That is, $\lim_{t\to\infty} S(t) = \frac{\Lambda}{\mu+\phi(1-K)}$. It follows from (3.1) that

$$\lim_{t\to\infty} v(t,a) = \frac{\phi\Lambda}{\mu+\phi(1-K)}\pi(a).$$

Therefore, $(S(t), I(t), v(t,\cdot)) \to E_0$ in $\mathbb{R}_+ \times \mathbb{R}_+ \times L^1_+$ as $t \to \infty$. This completes the proof. □

Next we study the permanence of (1.3). Define $\rho : \Gamma \to \mathbb{R}_+$ as

$$\rho(S, I, v) = f(I), \qquad \text{for } (S, I, v) \in \Gamma.$$

Let

$$\Gamma_0 = \{(S_0, I_0, v_0) \in \Gamma : \text{there exists a } t_0 \in \mathbb{R}_+ \text{ such that } \rho(\Phi(t_0, (S_0, I_0, v_0))) > 0\}.$$

Obviously, if $(S_0, I_0, v_0) \in \Gamma \setminus \Gamma_0$, then $(S(t), I(t), v(t,\cdot)) \to E_0$ as $t \to \infty$.

Definition 3.1 ([45]) If there exists an $\epsilon > 0$, independent of the initial conditions, such that

$$\limsup_{t\to\infty} \rho(\Phi(t, (S_0, I_0, v_0(\cdot)))) > \epsilon \text{ (respectively, } \liminf_{t\to\infty} \rho(\Phi(t, (S_0, I_0, v_0))) > \epsilon),$$

then (1.3) is called to be **uniformly weakly ρ-persistent** (respectively, **uniformly strongly ρ-persistent**), for $(S_0, I_0, v_0) \in \Gamma_0$.

Proposition 3.4 *If $\mathcal{R}_0 > 1$, then (1.3) is uniformly weakly ρ-persistent.*

Proof. If $\mathcal{R}_0 > 1$, we can choose an $\epsilon_0 > 0$ such that

$$\left(\frac{\Lambda}{\mu+\phi(1-K)+\epsilon_0} - \epsilon_0\right) f'(f^{-1}(\epsilon_0)) - (\mu+\gamma+\delta) > 0. \tag{3.2}$$

By contradiction, assume that there exists $(S_0, I_0, v_0) \in \Gamma_0$ with

$$\limsup_{t\to\infty} \rho(\Phi(t, (S_0, I_0, v_0))) \le \frac{\epsilon_0}{2}.$$

Then there exists $t_0 \in \mathbb{R}_+$ such that

$$\rho(\Phi(t, (S_0, I_0, v_0))) \le \epsilon_0 \qquad \text{for } t \ge t_0.$$

Without loss of generality, we can assume that $t_0 = 0$. In fact, this can be achieved by replacing the initial condition with $\Phi(t_0, (S_0, I_0, v_0))$. Therefore, for $t \ge t_0 = 0$, it follows from (1.1) that

$$f(I(t)) \le \epsilon_0, \quad I(t) \le f^{-1}(\epsilon_0). \tag{3.3}$$

Furthermore, if $\{t_n\}$ is a sequence such that $S(t_n) \to S_\infty$, then the Fluctuation Lemma together with (3.1) shows that

$$\frac{dS(t_n)}{dt} \ge \Lambda - (\mu+\phi)S(t_n) - \epsilon_0 S(t_n) + \phi \int_0^{t_n} \varepsilon(a)S(t_n - a)\pi(a)da.$$

Combining Lemma 3.1 and the above inequality, we obtain $S_\infty \ge \frac{\Lambda}{\mu+\phi(1-K)+\epsilon_0}$. Thus there exists $t_1 \ge t_0$ such that $S(t) \ge \frac{\Lambda}{\mu+\phi(1-K)+\epsilon_0} - \epsilon_0$ for $t \ge t_1$. Again, by replacing the initial condition, we can assume that $S(t) \ge \frac{\Lambda}{\mu+\phi(1-K)+\epsilon_0} - \epsilon_0$ for $t \in \mathbb{R}_+$. This, together with (A1), (3.1) and (3.3), leads to

$$\begin{aligned}
\frac{dI(t)}{dt} &= S(t)f(I(t)) - (\mu+\gamma+\delta)I(t) \\
&\ge \left(\frac{\Lambda}{\mu+\phi(1-K)+\epsilon_0} - \epsilon_0\right) f(I(t)) - (\mu+\gamma+\delta)I(t) \\
&\ge \left(\frac{\Lambda}{\mu+\phi(1-K)+\epsilon_0} - \epsilon_0\right) f'(I(t))I(t) - (\mu+\gamma+\delta)I(t) \\
&= \left[\left(\frac{\Lambda}{\mu+\phi(1-K)+\epsilon_0} - \epsilon_0\right) f'(f^{-1}(\epsilon_0)) - (\mu+\gamma+\delta)\right] I(t)
\end{aligned}$$

for $t \in \mathbb{R}_+$. It follows from the positivity of I_0 that $\liminf_{t\to\infty} I(t) = +\infty$. This contradicts with the boundedness of I. The proof is complete. □

It follows from Proposition 3.4 that $\Phi(t, \Gamma_0) \subseteq \Gamma_0$, and hence it induces a semiflow on Γ_0. Next we show that the solution semiflow Φ has a global compact attractor $\mathcal{A}$ in Γ_0. To this end, we first introduce two lemmas.

Lemma 3.5 ([46, Theorem 3.4.6]) *Let $T(t)$ be a semigroup acting on $X = \mathbb{R}^2_+ \times L^1_+(0,\infty)$. If $T(t) : X \to X$, $t \in \mathbb{R}_+$ is asymptotically smooth, point*

dissipative and orbits of bounded sets are bounded, then there exists a global attractor.

A semiflow is *asymptotically smooth* if each forward invariant bounded closed set is attracted by a nonempty compact set.

Lemma 3.6 ([46, Lemma 3.2.3]) *For each $t \in \mathbb{R}_+$, suppose $T(t) = S(t) + U(t) : X \to X$ has the property that $U(t)$ is completely continuous and there is a continuous function $k : \mathbb{R}_+ \times \mathbb{R}_+ \to \mathbb{R}_+$ such that $k(t, r) \to 0$ as $t \to \infty$ and $|S(t)x| \le k(t, r)$ if $|x| < r$. Then $T(t)$, $t \in \mathbb{R}_+$, is asymptotically smooth.*

Proposition 3.7 *If $\mathcal{R}_0 > 1$, then there exists a global attractor $\mathcal{A}$ for the solution semiflow Φ of (1.3) in Γ_0.*

Proof. By Lemma 3.5, we need to show that the semiflow Φ is asymptotically smooth. This can be achieved by employing Lemma 3.6 as follows.

For $t \in \mathbb{R}_+$ and $(S_0, I_0, v_0) \in \Gamma_0$, define

$$\begin{aligned}\hat{\Phi}(t, (S_0, I_0, v_0)) &= (0, 0, \hat{v}(t, \cdot)),\\ \tilde{\Phi}(t, (S_0, I_0, v_0)) &= (S(t), I(t), \tilde{v}(t, \cdot)),\end{aligned}$$

where

$$\tilde{v}(t,a) = \begin{cases} v(t,a) & \text{for } 0 \le a \le t \\ 0 & \text{for } t < a \end{cases} = \begin{cases} \phi S(t-a)\pi(a) & \text{for } 0 \le a \le t \\ 0 & \text{for } t < a \end{cases} \tag{3.4}$$

and

$$\hat{v}(t,a) = v(t,a) - \tilde{v}(t,a) = \begin{cases} 0 & \text{for } 0 \le a \le t, \\ v_0(a-t)\frac{\pi(a)}{\pi(a-t)} & \text{for } t < a. \end{cases} \tag{3.5}$$

Then $\Phi = \hat{\Phi} + \tilde{\Phi}$. Obviously, both $\hat{v}$ and $\tilde{v}$ are nonnegative. By (3.5), we obtain

$$\begin{aligned}\|\hat{\Phi}(t, (S_0, I_0, v_0))\| &= \|\hat{v}(t, \cdot)\|_1 \\ &= \int_t^\infty v_0(a-t)\frac{\pi(a)}{\pi(a-t)}da \\ &= \int_0^\infty v_0(a)\frac{\pi(a+t)}{\pi(a)}da \\ &\le e^{-\mu t}\int_0^\infty v_0(a)da \\ &= e^{-\mu t}\|v_0\|_1\end{aligned}$$

$$\leq e^{-\mu t}\|(S_0, I_0, v_0)\|$$

and hence $\hat{\Phi}$ satisfies the assumption in Lemma 3.6.

Next we show that $\tilde{\Phi}$ is completely continuous. This means that for any fixed $t \in \mathbb{R}_+$ and any bounded set $\mathcal{B} \subseteq \Gamma_0$, the set $\mathcal{B}_t \triangleq \{\tilde{\Phi}(t, (S_0, I_0, v_0)) : (S_0, I_0, v_0) \in \mathcal{B}\}$ is precompact. It is sufficient to show that $\mathcal{B}_{t,v} = \{\tilde{v}(t, \cdot) : (S(t), I(t), \tilde{v}(t, \cdot)) \in \mathcal{B}_t\}$ is precompact. This can be achieved by applying the Fréchet-Kolmogrov Theorem [47]. Firstly, it follows from the definitions of $\tilde{\Phi}$ and Γ_0 that $\mathcal{B}_{t,v}$ is bounded. This implies that the first condition of the Fréchet-Kolmogrov Theorem holds. Secondly, it is easy to see that $\int_t^\infty \tilde{v}(t, a)da = 0$. Thus the third condition of the Fréchet-Kolmogrov Theorem is satisfied. Finally, to verify the second condition of the Fréchet-Kolmogrov Theorem, we show that $\mathcal{B}_{t,v}$ is uniformly continuous $\tilde{\Phi}$ or

$$\lim_{h\to 0^+} \|\tilde{v}(t, \cdot) - \tilde{v}(t, \cdot + h)\|_1 = 0 \qquad \text{uniformly in } \mathcal{B}_{t,v}. \tag{3.6}$$

Equation (3.6) holds automatically when $t = 0$ since $\tilde{v}(0, \cdot) = 0$ by (3.4). As a result, we only need to consider the case with $t > 0$. Let $h \in (0, t)$. Then

$$\begin{aligned}
&\|\tilde{v}(t, \cdot) - \tilde{v}(t, \cdot + h)\|_1 \\
=&\int_0^\infty |\tilde{v}(t, a) - \tilde{v}(t, a + h)|da \\
=&\phi \int_0^{t-h} |S(t - a - h)\pi(a + h) - S(t - a)\pi(a)|da + \phi \int_{t-h}^t S(t - a)\pi(a)da \\
\leq&\phi \int_0^{t-h} S(t - a - h)|\pi(a + h) - \pi(a)|da + \phi\frac{\Lambda}{\mu + \phi(1 - K)}h \\
&+\phi \int_0^{t-h} |S(t - a - h) - S(t - a)|\pi(a)da \\
\leq&\phi\frac{\Lambda}{\mu + \phi(1 - K)}\mu h + \phi \int_0^{t-h} |S(t - a - h) - S(t - a)|\pi(a)da \\
&+\phi\frac{\Lambda}{\mu + \phi(1 - K)}h
\end{aligned}$$

as $S(t) \leq \frac{\Lambda}{\mu+\phi(1-K)}$ for $t \in \mathbb{R}_+$ and $|\pi(a + h) - \pi(a)| \leq 1 - e^{-\int_a^{a+h} \mu ds} \leq \mu h$. This estimate, together with the uniform continuity of $S(t)$, immediately yields (3.6). The proof is complete. □

By Proposition 3.4, Proposition 3.7, and [48, Theorem 3.2], we immediately have

Theorem 3.8 *System* (1.3) *is uniformly strongly ρ-persistent if $\mathcal{R}_0 > 1$.*

A *total trajectory* of Φ is a function $X : \mathbb{R} \to \mathbb{R}_+ \times L^1_+$ such that $\Phi(s, X(t)) = X(t + s)$ for all $t \in \mathbb{R}$ and all $s \in \mathbb{R}_+$.

Corollary 3.9 *Suppose $\mathcal{R}_0 > 1$. Let $(S(t), I(t), v(t,\cdot))$ be a total trajectory in $\mathcal{A}$. Then there exists an $\varepsilon_0 > 0$ such that $S(t)$, $I(t)$, $v(t,0) > \varepsilon_0$ for all $t \in \mathbb{R}$.*

Proof. It follows from $I(t) \le \frac{\Lambda}{\mu}$ and Assumption (A1) that

$$\frac{dS(t)}{dt} \ge \Lambda - (\mu + \phi + f(\frac{\Lambda}{\mu}))S(t)$$

and hence $S_\infty \ge \frac{\Lambda}{\mu+\phi+f(\frac{\Lambda}{\mu})}$. This provides a lower bound ε_1 for the S-coordinate for any point in $\mathcal{A}$. Theorem 3.8 implies that there exists $\varepsilon_2 > 0$ such that $\rho(S(t), I(t), v(t,\cdot)) > \varepsilon_2$ for all $t \in \mathbb{R}$. Thus $f'(0)I(t) \ge f(I(t)) \ge \varepsilon_2$ and hence $I(t) \ge \frac{\varepsilon_2}{f'(0)}$. Note that $v(t,0) = \phi S \ge \phi\varepsilon_1$. We can take $\varepsilon_0 = \min\{\varepsilon_1, \frac{\varepsilon_2}{f'(0)}, \phi\varepsilon_1\}$ to complete the proof. □

Remark 3.1 Corollary 3.9 establishes the persistence of system (1.3) when the basic reproduction number $\mathcal{R}_0 > 1$. This implies that there always exist infectious individuals when the basic reproduction number is larger than 1. This result is crucial as it implies that the Lyapunov functional to be used in proving the global stability of the endemic equilibrium and its derivative along solutions of system (1.3) are meaningful at infinity.

Define $\varphi : (0, \infty) \to \mathbb{R}$ as

$$\varphi(x) = x - 1 - \ln x.$$

It is well-known that φ attains a global minimum only at 1 with $\varphi(1) = 0$ and $\varphi(x) > 0$ for $x \ne 1$.

Lemma 3.10 ([35, Proposition A.1]) *Define*

$$F(I) = \varphi\left(\frac{f(I)}{f(I^*)}\right) - \varphi\left(\frac{I}{I^*}\right).$$

If Assumption (A1) is satisfied, then $F(I) \le 0$ for all $I > 0$.

Now we are in the position to prove the following result.

Theorem 3.11 *If $\mathcal{R}_0 > 1$, then the endemic equilibrium E^* is globally asymptotically stable in Γ_0.*

Proof. By Theorem 2.2 and Proposition 3.7, it suffices to show $\mathcal{A} = \{E^*\}$. Let $X(t) = (S(t), I(t), v(t,\cdot))$ be a total trajectory in $\mathcal{A}$. By Corollary 3.9, there exists $\epsilon_0 > 0$, for any $t \in \mathbb{R}$, such that $0 \le \varphi(x) < \epsilon_0$ for $x = \frac{S(t)}{S^*}$, $\frac{I(t)}{I^*}$, and $\frac{v(t,0)}{v^*(0)}$. Note that $\frac{v(t,a)}{v^*(a)} = \frac{v(t-a,0)\pi(a)}{v^*(0)\pi(a)} = \frac{v(t-a,0)}{v^*(0)}$, which implies that $0 \le \varphi(x) \le \epsilon_0$ for any $t \in \mathbb{R}$ and $a \in \mathbb{R}_+$.

Let

$$\alpha(a) = \int_a^\infty \varepsilon(s)v^*(s)ds.$$

Then

$$\frac{d\alpha(a)}{da} = -\varepsilon(a)v^*(a).$$

Define

$$V(t) = S^*V_S(t) + I^*V_I(t) + V_v(t),$$

where $V_S(t) = \varphi(\frac{S(t)}{S^*})$, $V_I(t) = \varphi(\frac{I(t)}{I^*})$ and $V_v(t) = \int_0^\infty \alpha(a)\varphi(\frac{v(t,a)}{v^*(a)})da$. Then V is bounded.

Next we show that the upper-right derivative $\frac{dV(t)}{dt}$ along the solution is non-positive. We first have

$$\begin{aligned}
\frac{dV_S(t)}{dt} &= \left(1 - \frac{S^*}{S(t)}\right)\left[\Lambda - (\mu+\phi)S(t) - S(t)f(I) + \int_0^\infty \varepsilon(a)v(t,a)da\right] \\
&= \left(1 - \frac{S^*}{S(t)}\right)\left[(\mu+\phi)S^* + S^*f(I^*(a)) - \int_0^\infty \varepsilon(a)v^*(a)da\right. \\
&\quad \left. -(\mu+\phi)S(t) - S(t)f(I) + \int_0^\infty \varepsilon(a)v(t,a)da\right] \\
&= -(\mu+\phi)S^*(\frac{S^*}{S} + \frac{S}{S^*} - 2) + S^*f(I^*)[1 - \frac{S(t)f(I(t))}{S^*f(I^*)} - \frac{S^*}{S} \\
&\quad + \frac{f(I(t))}{f(I^*)}] + \int_0^\infty \varepsilon(a)v^*(a)\left[\frac{v(t,a)}{v^*(a)} - \frac{S^*v(t,a)}{S(t)v^*(a)} - 1 + \frac{S^*}{S}\right]da.
\end{aligned}$$

Differentiating V_I with respect to t and noting that $\mu + \gamma + \delta = \frac{S^*f(I^*)}{I^*}$, we obtain

$$\begin{aligned}
\frac{dV_I(t)}{dt} &= \left(1 - \frac{I^*}{I(t)}\right)[Sf(I(t)) - (\mu+\gamma+\delta)I] \\
&= \left(1 - \frac{I^*}{I(t)}\right)\left[Sf(I(t)) - \frac{S^*f(I^*)I}{I^*}\right] \\
&= S^*f(I^*)[1 + \frac{S(t)f(I(t))}{S^*f(I^*)} - \frac{S(t)f(I(t))I^*}{S^*f(I^*)I(t)} - \frac{I(t)}{I^*}].
\end{aligned}$$

The derivative of V_v can be calculated as follows

$$\begin{aligned}
\frac{dV_v(t)}{dt} &= \int_0^\infty \alpha(a)\frac{\partial\varphi(\frac{v(t,a)}{v^*(a)})}{\partial t}da \\
&= \int_0^\infty \alpha(a)(1 - \frac{v^*(a)}{v(t,a)})\frac{1}{v^*(a)}\frac{\partial v(t,a)}{\partial t}da
\end{aligned}$$

$$
\begin{aligned}
&= -\int_0^\infty \alpha(a)(1-\frac{v^*(a)}{v(t,a)})\frac{v(t,a)}{v^*(a)}[\frac{v_a(t,a)}{v(t,a)}+\mu+\varepsilon(a)]da \\
&= -\int_0^\infty \alpha(a)(\frac{v(t,a)}{v^*(a)}-1)[\frac{v_a(t,a)}{v(t,a)}+\mu+\varepsilon(a)]da,
\end{aligned}
$$

where $v_a(t,a)=\frac{\partial v(t,a)}{\partial a}$. With the assistance of

$$
\frac{\partial}{\partial a}(\frac{v(t,a)}{v^*(a)}) = (\frac{v(t,a)}{v^*(a)}-1)[\frac{v_a(t,a)}{v(t,a)}+\mu+\varepsilon(a)],
$$

$\frac{d}{da}(\alpha(a)) = -\varepsilon(a)v^*(a)$, and integration by parts, we have

$$
\begin{aligned}
\frac{dV_v(t)}{dt} &= -\int_0^\infty \alpha(a)\frac{\partial}{\partial a}(\frac{v(t,a)}{v^*(a)})da \\
&= -\alpha(a)\varphi(\frac{v(t,a)}{v^*(a)})|_{a=0}^{a=\infty} + \int_0^\infty \varphi(\frac{v(t,a)}{v^*(a)})\frac{d\alpha(a)}{da}da \\
&= -\alpha(a)\varphi(\frac{v(t,a)}{v^*(a)})|_{a=\infty} + \alpha(0)\varphi(\frac{v(t,0)}{v^*(0)}) \\
&\quad -\int_0^\infty \varepsilon(a)v^*(a)\varphi(\frac{v(t,a)}{v^*(a)})da.
\end{aligned}
$$

Using $\alpha(0)=\int_0^\infty \varepsilon(a)v^*(a)da$ yields

$$
\begin{aligned}
\frac{dV_v(t)}{dt} &= -\alpha(a)\varphi(\frac{v(t,a)}{v^*(a)})|_{a=\infty} + \int_0^\infty \varepsilon(a)v^*(a)\varphi(\frac{v(t,0)}{v^*(0)})da \\
&\quad -\int_0^\infty \varepsilon(a)v^*(a)\varphi(\frac{v(t,a)}{v^*(a)})da.
\end{aligned}
$$

Summing up $\frac{dV_S}{dt}$, $\frac{dV_I}{dt}$, and $\frac{dV_v}{dt}$ yields

$$
\begin{aligned}
\tfrac{dV}{dt} &= -(\mu+\phi)S^*(\tfrac{S^*}{S}+\tfrac{S}{S^*}-2) - \alpha(a)\varphi(\tfrac{v(t,a)}{v^*(a)})|_{a=\infty} \\
&\quad +S^*f(I^*)[2-\tfrac{S^*}{S}+\tfrac{f(I(t))}{f(I^*)}-\tfrac{S(t)f(I(t))I^*}{S^*f(I^*)I(t)}-\tfrac{I(t)}{I^*}] \\
&\quad +\textstyle\int_0^\infty \varepsilon(a)v^*(a)[\varphi(\frac{v(t,0)}{v^*(0)})-\varphi(\frac{v(t,a)}{v^*(a)})+\frac{v(t,a)}{v^*(a)}-\frac{S^*v(t,a)}{S(t)v^*(a)}-1+\frac{S^*}{S}]da \\
&= -(\mu+\phi(1-K))S^*(\tfrac{S^*}{S}+\tfrac{S}{S^*}-2) - \alpha(a)\varphi(\tfrac{v(t,a)}{v^*(a)})|_{a=\infty} \\
&\quad +S^*f(I^*)[\varphi(\tfrac{f(I(t))}{f(I^*)})-\varphi(\tfrac{I(t)}{I^*})-\varphi(\tfrac{S^*}{S})-\varphi(\tfrac{S(t)f(I(t))I^*}{S^*f(I^*)I(t)})] \\
&\quad -\textstyle\int_0^\infty \varepsilon(a)v^*(a)\varphi(\frac{S^*v(t,a)}{S(t)v^*(a)})da.
\end{aligned}
$$

It follows from Lemma 3.10, the properties of φ and $\int_0^\infty \varepsilon(a)v^*(a)da = \phi S^*K$ that $\frac{dV(t)}{dt}\le 0$. Therefore V is nonincreasing. Since V is bounded on $X(\cdot)$, the $\omega-$limit set of $X(\cdot)$ must be contained in $\mathcal{M}$, the largest invariant subset of

$\{\frac{dV}{dt} = 0\}$. It follows from $\frac{dV}{dt} = 0$ that $S(t) = S^*$ and $v(t,a) = v^*(a)$. Thus $\frac{dS(t)}{dt} = 0$ in $\mathcal{M}$. This implies that

$$0 = \Lambda - (\mu + \phi)S^* - S^* f(I(t)) + \int_0^\infty \varepsilon(a)v^*(a)da$$

for $t \in \mathbb{R}$, which yields $f(I(t)) = f(I^*)$ for all $t \in \mathbb{R}$. This, together with monotonicity of $f(x)$ stated in (A1) implies that $I(t) = I^*$ for all t. Therefore, $\mathcal{M} = \{E^*\}$.

The above analysis indicates that the $\omega-$limit set of $X(\cdot)$ consists of just the endemic equilibrium E^* and hence $V(X(t)) \geq V(E^*)$ for all $t \in \mathbb{R}$. Thus $\mathcal{A} = \{E^*\}$. The proof is complete. $\square$

Remark 3.2 Theorem 3.11 indicates that the endemic equilibrium E^* attracts all solutions of (1.3), if $\mathcal{R}_0 > 1$. This, together with Theorem 3.3 and Theorem 2.2, implies that the global stability of equilibria for system (1.3) is completely determined by the reproduction number $\mathcal{R}_0$. This result partially extends the previous existing results for disease models with age structure [4,18] in which only local stability results are established.

4 Discussion

In this paper we have incorporated the waning of vaccine-induced immunity and nonlinear incidence rate into an age-structured SIVS model. It has been shown that the disease dynamics is completely determined by the basic reproduction number: if the reproduction number is less than 1, the disease-free equilibrium E_0 is globally asymptotically stable and the disease dies out; while if the basic reproduction number is larger than 1, then the endemic equilibrium E^* is globally asymptotically stable and the disease persists.

Similar to many disease models, our newly studied model exhibits global threshold dynamics. Unlike ordinary differential equation models, our PDE model requires more subtle analysis to achieve the global dynamics. Further, our result does shed some light on how to manage vaccination coverage rate to control the spread of diseases. Based on our analysis, there exists a threshold value

$$\phi_0 := \frac{\Lambda f'(0) - \mu(\mu + \gamma + \delta)}{(\mu + \gamma + \delta)(1 - K)}$$

for the vaccine coverage rate. Clearly, the vaccination coverage required to eradicated the disease is higher than that when the waning of vaccine-induced immunity is neglected (i.e., $K = 0$). Thus setting the vaccination coverage ϕ as ϕ_0 with $K = 0$ is not sufficient for the disease to die out.

To illustrate our theoretical result, we take chickenpox related disease parameters for the purpose of numerical computation. More precisely, we set

$$\mu = \frac{1}{75}\ \text{year}^{-1},\ \gamma = 26\ \text{year}^{-1},\ \delta = 0.014\ \text{year}^{-1}.$$

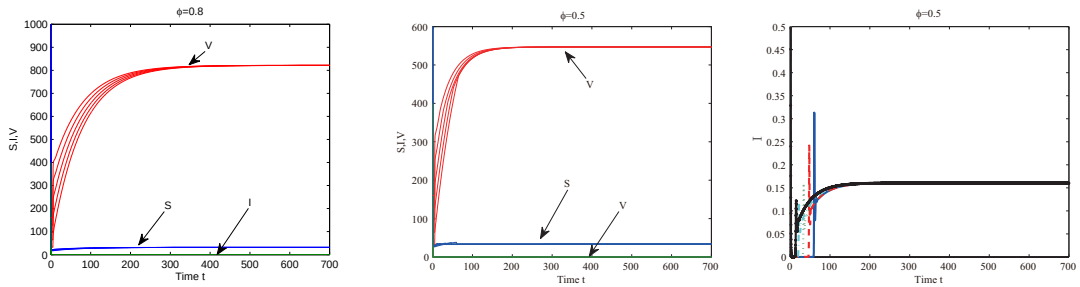
This means the average individual lifespan is 75 years, and the infection lasts for about 2 weeks (in the range of $10 \sim 21$ days) and among every $10,000$ infected individuals, 140 died each year as a result of chickenpox (in the range of $100 \sim 150$ in the USA [49]). For chickenpox virus, clinic data suggests the vaccinated individuals gain protective immunity during the first 5 years, and the immunity gradually wanes since then [50]. We take the following form for $\varepsilon(a)$:

$$\varepsilon(a) = \begin{cases} 0, & 0 < a \leq 5, \\ \frac{1}{65}, & a > 5. \end{cases}$$

This implies, the immunity is completely lost after 70 years. The incidence function $f(I)$ is taken as a saturation function given below

$$f(I) = \frac{0.8I}{1 + 0.2I}.$$

The birth rate is taken as $\Lambda = 13$ per 1000 people per year in the USA according to the World Bank data [51]. With these specified parameters values, the computed critical vaccination coverage is $\phi_0 = \%77.43$ (with $K \approx 0.50$), which is twice as high as that of the case when the waning of vaccine-induced immunity is neglected (the resulting coverage is $\%38.62$). This clearly indicates the waning of vaccine-induced immunity should be taken into consideration in designing practical vaccination strategies. Numerical simulations are presented in Figure 1.



(a) (b) (c)

Fig. 1. Numerical solutions of (1.3) with five different sets of initial conditions. The parameters are listed in the text and ϕ varies from 0.8 to 0.1. (a) $\phi = 0.8 > \phi_0$; (b) $\phi = 0.5 < \phi_0$, (c) $\phi = 0.5 < \phi_0$,.

Our analysis shows that the immunity waning function $\varepsilon(a)$ crucially influences the critical vaccination coverage ϕ_0. Much clinic work should be further conducted to reveal how the vaccine-induced immunity wanes for diseases such as chickenpox so that we may gain better description on the immunity waning function $\varepsilon(a)$ and thus come up with better estimate on the critical vaccination coverage ϕ_0 to eradicate the disease.

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References

[1] R.M. Anderson, R.M. May, Infectious Diseases of Humans: Dynamics and Control, Oxford University Press, 1992.

[2] M. Haber, I.M. Longini, M.E. Halloran, Measures of the effects of vaccination in a randomly mixing population, Int. J. Epidemiol., 20 (1991) 300-319.

[3] B. Shulgin, L. Stone, Z. Agur, Pulse vaccination strategy in the SIR epidemic model. Bull. Math. Biol., 60 (1998) 1123-1148.

[4] M. Iannelli, M. Martcheva, X.Z. Li, Strain replacement in an epidemic model with super-infection and perfect vaccination, Math. Biosci., 195 (1) (2005) 23-46.

[5] C.A. Iliano V, R.O Marcelo, Transmission blocking vaccines to control insect-borne diseases: a review, Mem. Inst. Oswaldo Cruz, 105(1)(2010) 1-12.

[6] M.E. Alexander, C. Bowman, S.M. Moghadas, R. Summers, A.B. Gumel, B.M. Sahai, A vaccination model for transmission dynamics of influenza, SIAM J. Appl. Dyn. Syst., 3 (2004) 503-524.

[7] B. Buonomo, A. dOnofrio, D. Lacitignola, Global stability of an SIR epidemic model with information dependent vaccination, Math. Biosci., 216 (2008) 9-16.

[8] G. Huang, X. Liu, Y. Takeuchi, Lyapunov functions and global stability for age-structured HIV infection model, SIAM J. Appl. Math., 72 (2012) 25-38.

[9] X. Liu, Y. Takeuchi, S. Iwami, SVIR epidemic models with vaccination strategies, J. Theor. Bio., 253 (2008) 1-11.

[10] Y. Xiao, S. Tang, Dynamics of infection with nonlinear incidence in a simple vaccination model, Nonlinear Anal. Real World Appl., 11 (2010) 4154-4163.

[11] S. Gao, L. Chen, J. J. Nieto, A. Torres, Analysis of a delayed epidemic model with pulse vaccination and saturation incidence, Vaccine, 24 (2006) 6037-6045.

[12] J. R. Kremer, F. Schneider, C. P. Muller, Waning antibodies in measles and rubella vaccineesa longitudinal study, Vaccine, 24(2006), 2594–2601.

[13] F.R. Mooi, N.A.T. van der Maas, H.E. de Melker, Pertussis resurgence: waning immunity and pathogen adaptation two sides of the same coin, Epidemiol. Infect., 142(2014), 685–694.

[14] A. M. Wendelboe, A. van Rie, S. Salmaso, J. A. Englund, Duration of immunity against pertussis after natural infection or vaccination, Pediatr Infect Dis J. 24(2005), S58–S61.

[15] D. Miron, I. Lavi, R. Kitov, A. Hendler, Vaccine effectiveness and severity of varicella among previously vaccinated children during outbreaks in day-care centers with low vaccination coverage, Pediatr Infect Dis J. 24(2005): 233-236.

[16] M. L. Taylor, T. W. Carr, An SIR epidemic model with partial temporary immunity modeled with delay, J. Math. Biol., (2009) 59:841-880.

[17] W.O. Kermack, A.G. McKendrick, Contributions to the mathematical theory of epidemics I, Proc. Roy Soc. Ser. A, 115 (1927) 700-721.

[18] X. Li, J. Wang, M. Ghosh, Stability and bifurcation of an SIVS epidemic model with treatment and age of vaccination, Appl. Math. Model., 34 (2010) 437-450.

[19] X. Duan, S. Yuan, X. Li, Global stability of an SVIR model with age of vaccination. Appl. Math. Comput., 226 (2014) 528-540.

[20] X. Duan, S. Yuan, Z. Qiu, J. Ma, Global stability of an SVEIR epidemic model with ages of vaccination and latency, Comput. Math. Appl., 68 (2014) 288-308.

[21] H. Gulbudak, M. Martcheva, A Structured avian influenza model with imperfect vaccination and vaccine induced asymptomatic infection, Bull. Math. Biol., 76 (10) (2014) 2389-2425.

[22] R.M. Anderson, R.M. May, The invasion, persistence, and spread of infectious diseases within animal and plant communities, Philos. Trans. R. Soc. Lond. B, 314 (1986), 533-570.

[23] A. d'Onofrio, Vaccination policies and nonlinear force of infection: generalization of an observation by Alexander and Moghadas (2004), Appl. Math. Comput. 168 (2005) 613-622.

[24] F. Brauer, C. Castillo-Chavez, Mathematical Models in Population Biology and Epidemiology (Texts in Applied Mathematics), Second Edition, Springer-Verlag, New York, 2011.

[25] M. Martcheva, Avian Flu: Medeling and implications for control, J. Biol. Syst., 22 (2014) 151-175.

[26] W. Wang, S. Ruan, Simulating the SARS outbreak in Beijing with limited data, J. Theor. Biol., 227 (2004)369-379.

[27] Q. Zhu, Y.T. Hao, J.Q. Ma, S.C. Yu, Y. Wang, Surveillance of Hand, Foot, and Mouth Disease in Mainland China (2008-2009), Biomed. Environ. Sci., 24(4), (2011) 349-356.

[28] F. Liljeros, C.R. Edling, L.A. Nunes Amaral, Sexual networks: implications for the transmission of sexually transmitted infections, Microb. Infect., 5(2) (2003) 189-196.

[29] V. Capasso, G. Serio, A generalization of the Kermack-Mackendric deterministic model, Math. Biosci., 42 (1978) 43-61.

[30] W.M. Liu, S.A. Levin, Y. Iwasa, Influence of nonlinear incidence rates upon the behavior of SIRS epidemiological models, J. Math. Biol., 23 (1986) 187-204.

[31] D. Xiao, S. Ruan, Global analysis of an epidemic model with nonmonotone incidence rate, Math. Biosci., 208(2) (2007) 419-429.

[32] P. van den Driessche, J. Watmough, A simple SIS epidemic model with a backward bifurcation, J. Math. Biol., 40 (2000) 525-540.

[33] L. Wu, Z. Feng, Homoclinic bifurcation in an SIQR model for childhood diseases, J. Differ. Equations, 168 (2000), 150-167.

[34] S. Ruan, W. Wang, Dynamical behavior of an epidemic model with a nonlinear incidence rate, J. Differ. Equations, 188(1) (2003) 135-163.

[35] R.P. Sigdel, C.C. McCluskey, Global stability for an SEI model of infectious disease with immigration, Appl. Math. Comput., 243 (2014) 684-689.

[36] Y. Enatsu and Y. Nakata, Stability and bifurcation analysis of epidemic models with saturated incidence rates: An application to a nonmonotone incidence rate, Math. Biosci. Eng., 11 (2014) 785-805.

[37] P. Magal, C.C. McCluskey, G.F. Webb, Lyapunov functional and global asymptotic stability for an infection-age model, Appl. Anal., 89 (2010) 1109-1140.

[38] Y. Chen, J. Yang, F. Zhang, The global stability of an SIRS model with infection age, Math. Biosci. Eng., 11 (2014) 449-469.

[39] C.C. McCluskey, Global stability for an SIR epidemic model with delay and nonlinear incidence, Nonlinear Anal. Real World Appl., 11 (2010) 3106-3109.

[40] G. Huang, Y. Takeuchi, W. Ma, D. Wei, Global stability for delay SIR and SEIR epidemic models with nonlinear incidence rate, Bull. Math. Biol., 72 (2010) 1192-1207.

[41] M. Iannelli, Mathematical Theory of Age-structured Population Dynamics, in Applied Mathematics Monographs 7, comitato Nazionale per le Scienze Matematiche, Consiglio Nazionale delle Ricerche (C.N.R.), Giardini, Pisa, 1995.

[42] P. Magal, Compact attrators for time periodic age-structured population models, Electron. J. Differ. Equations, 65 (2001) 1-35.

[43] C.J. Browne, S.S. Plyugin, Global analysis of age-structured within-host virus model, Discrete Contin. Dyn. Syst. Ser. B, 18 (2013) 1999-2017.

[44] W.M. Hirsch, H. Hanisch, J.P. Gabriel, Differential equation models of some parasitic infections: Methods for the study of asymptotic behavior, Comm. Pure Appl. Math., 38 (1985) 733-753.

[45] H.L. Smith, H.R. Thieme, Dynamical Systems and Population Persistence, AMS, Providence, 2011.

[46] J.K. Hale, Asymptotic Behavior of Dissipative Systems, AMS, Providence, RI, 1988.

[47] K. Yasida, Functional Analysis, 2nd ed., Berlin-Heidelberg, New York, Springer-Verlag, 1968.

[48] H.R. Thieme, Uniform persistence and permanence for non-autonomous semiflows in population biology, Math. Biosci., 166(2000) 173-201.

[49] http://www.cdc.gov/chickenpox/about/overview.html.

[50] S.S. Chaves, P. Gargiullo,J.X. Zhang,R. Civen,D. Guris,L. Mascola, J.F. Seward, Loss of vaccine-induced immunity to varicella over time, N Engl J Med., 356(11) (2007) 1121-1129.

[51] http://data.worldbank.org/indicator/SP.DYN.CBRT.IN.