ELSEVIER



Chaos, Solitons & Fractals

Chaos, Solitons and Fractals Nonlinear Science, and Nonequilibrium and Complex Phenomena

journal homepage: www.elsevier.com/locate/chaos

Global stability of an epidemic model with age-dependent vaccination, latent and relapse



Yingke Li^{a,b}, Zhidong Teng^{a,*}, Cheng Hu^a, Qing Ge^b

^a College of Mathematics and Systems Science, Xinjiang University, Urumqi 830046, People's Republic of China ^b College of Mathematics and Physics, Xinjiang Agriculture University, Urumqi 830052, People's Republic of China

ARTICLE INFO

Article history: Received 4 June 2017 Revised 10 October 2017 Accepted 26 October 2017

Keywords: Age-dependent SVEIR epidemic model Vaccination Latent Relapse Permanence Global stability

ABSTRACT

The vaccination, latent and relapse period are three important factors affecting the whole disease development. In this paper, we propose an SVEIR epidemic model with continuous age-dependent vaccination, latency and relapse, at the same time, the nonlinear incidence rate is also considered. Uniform persistence of the model is proved by reformulating it as the so called Volterra integral equations. The basic reproduction number \mathcal{R}_0 , which completely determines the global dynamics of the model, is derived. By using Lyapunov functionals, the global stability of the equilibria is obtained. Namely, the disease-free equilibrium is globally asymptotically stable if $\mathcal{R}_0 < 1$, while if $\mathcal{R}_0 > 1$ the endemic equilibrium is globally asymptotically stable. Finally, two numerical examples support our main analytical results.

© 2017 Elsevier Ltd. All rights reserved.

1. Introduction

Vaccination is considered as one of the most practical and efficient strategies to prevent and control the spread of many diseases, such as measles, pertussis, influenza, Hepatitis B virus(HBV) and human tuberculosis (TB) (See [1–3]). The spectacular successful cases was seen to be eradication of small-pox in 1977 (See [4]). Many models of ordinary differential equations (ODEs) have been proposed to better understand how vaccination impact disease transmission dynamics and their prevention (See [5–9]). For example, Gao et al. in [7] formed an pulse vaccination SEIRS model and discussed the condition for eradicating the disease, Liu and his coauthors in [8] built two SVIR epidemic models to investigate the immunity impacts by impulsive and continuous strategies. Most of these compartmental models are formulated as ODEs under the base assumption that individuals in each compartment are homogeneously mixed.

It has been recognised that the waning of vaccine-induced immunity has been one of the principal reasons for reemergence of some children epidemic such as pertussis, rubella, measles and chickenpox (See [10]). Naturally, researchers incorporate vaccination and the waning immunity in modeling disease dynamics (See [10–15]). As a matter of fact, a suitable assumption on the wan-

https://doi.org/10.1016/j.chaos.2017.10.027 0960-0779/© 2017 Elsevier Ltd. All rights reserved. ing of vaccine-induced immunity is that immunity depends on age of an individual for vaccination. Taking the age of vaccination into consideration, researchers obtain many models of partial differential equations (PDEs). Here, we just list a few of the recent works (See [10–18,26,29–33,36]). It is known that the period for individuals in latent compartment is different from one to one, which relys on distinct infectious disease and individuals situation. For tuberculosis, the latent period may takes months, years or even decades before one being infectious (See [24]). Furthermore, for the infectious, such as tuberculosis and herpes virus infection, the removed individual often have higher relapse rate (See [23,24]). Accordingly, the reactivation is regarded as an important character for both some animal and human diseases in mathematical modeling (See [12,25]). Therefore, it is necessary to consider the sojourn time and relapse structure in modeling.

Another significant aspect in disease modeling is comprehending how the infected and the susceptible population mutual effect and impact the disease dynamics (See [19]). Mathematically, these can be captured by the incidence rate of the disease, defined as the average number of new cases of a disease per unit period off cf time. Models with standard incidence rates and bilinear (namely, mass action) incidence rates have extensively studied recently. Liu et al. in [12] proposed an age-dependent epidemic model with bilinear incidence and investigate how the basic production number determines the global stability of epidemic dynamics. For more

^{*} Corresponding author. E-mail address: zhidong1960@163.com (Z. Teng).



Fig. 1. Flow diagram of our model, where $\Sigma \omega_1 V$, $\Sigma \omega_2 E$ and $\Sigma \omega_3 R$ represent $\int_0^\infty \omega_1(a)\nu(t,a)da, \int_0^\infty \omega_2(b)e(t,b)db$ and $\int_0^\infty \omega_3(c)r(t,c)dc$, respectively.

models with different nonlinear incidence rate, we here only refer to literatures [11,13,14,20–22].

Motivated by the previous works of Liu and Wang [12,13], there have been few literatures investigating the effects of agedependent vaccination, latency and relapse on global stability of models with nonlinear incidence so far. Moreover, It is stressed that the vaccination of susceptible population plays a central role in the evolution of vaccine-induced immunity [26]. So, to understand the effect of these aspects on the dynamics of the epidemics, we propose and investigate an age-dependent SVEIR model of PDEs coupled with ODEs in this paper which takes these elements under consideration.

This work is organized as follows. In Section 2, we propose a novel age-dependent SVEIR model with ages of vaccination, latency and relapse. In Section 3, some preliminaries for our main results are presented, such as the existence of equilibria and the basic reproduction number for the model. In Section 4, we investigate uniform persistence of the model. We analyse the global stability of the disease-free equilibrium and endemic equilibrium in Section 5 and Section 6, respectively. Finally, simulations and discussions are made in Section 7.

2. Model formulation

The total population under consideration is divided into five disjoint subclasses: the susceptible (S), vaccinated (V), latent (E), infectious (I) and recovered (R), respectively. Let S(t) be the number of the susceptible at time t, v(t, a) be the density of the vaccinated at time t with vaccination age a, e(t, b) be the density of the latent at time t, with latent age b, I(t) be the number of the infectious at time t, and r(t, c) be the density of the recovered at time t with relapse age c.

All recruitment into the population is into the susceptible class and occurs with a constant flux Λ . Then they enter into a compartment where individuals are exposed upon the disease but not yet infected, this compartment is often called latent part. Assume that the incidence rate is nonlinear form and all new infections enter the latent class at latent age zero. It is also assumed that the newly vaccinated individuals enter the vaccinated class *V* at vaccination age zero, then the total number of vaccinated individuals within the vaccinated subclass at time *t* is $\int_0^{+\infty} v(a, t) da$. Suppose the vaccine-induced immunity wanes rate is dependent on age of vaccination and given by $\omega_1(a)$, thus the total number of waning of immunity which progress into the susceptible class alive reads $\int_0^{+\infty} \omega_1(a)v(t, a) da$.

Similarly, for the density of the latent e(t, b) at time t with latent age b and the density of the recovered r(t, c) at time t with relapse age c, the total number of latent individuals within the latent subclass and the total number of recovered individuals within the recovered subclass at time t are $\int_0^{+\infty} e(b, t)db$ and $\int_0^{+\infty} r(c, t)dc$, respectively. The age-dependent removal rate from latent subclass and relapse rate from removed subclass are given by $\omega_2(b)$ and $\omega_3(c)$, respectively. Thus the quantity of individuals who progress and the relapse into the infectious class alive read $\int_0^{+\infty} \omega_2(b)e(t, b)db$ and $\int_0^{+\infty} \omega_3(c)e(t, c)dc$, respectively. Our model is described by the following diagram in Fig. 1.

From Fig. 1 we can establish the following system of ODEs coupled with PDEs:

$$\begin{cases} \frac{dS(t)}{dt} = \Lambda - (\mu + \xi)S(t) - \beta S(t)f(I(t)) + \int_0^\infty \omega_1(a)v(t, a)da, \\ \frac{\partial v(t, a)}{\partial t} + \frac{\partial v(t, a)}{\partial a} = -(\omega_1(a) + \mu)v(t, a), \\ \frac{\partial e(t, b)}{\partial t} + \frac{\partial e(t, b)}{\partial b} = -(\omega_2(b) + \mu)e(t, b), \\ \frac{dI(t)}{dt} = \int_0^\infty \omega_2(b)e(t, b)db - (\mu + \delta + k)I + \int_0^\infty \omega_3(c)r(t, c)dc, \\ \frac{\partial r(t, c)}{\partial t} + \frac{\partial r(t, c)}{\partial c} = -(\omega_3(c) + \mu)r(t, c) \\ v(t, 0) = \xi S(t), \ e(t, 0) = \beta S(t)f(I(t)), \ r(t, 0) = kI(t), t \ge 0 \\ S(0) = S_0, \ v(0, a) = v_0(a), \ e(0, b) = e_0(b), \ I(0) = I_0, \\ r(0, c) = e_0(c), \end{cases}$$
(1)

where $S_0, I_0 \in \mathbb{R}_+ = [0, \infty)$, and $v_0(a), e_0(b), e_0(c) \in L^1_+$, where $L^1_+ = L^1_+(0, \infty)$ denotes the space of all Lebesgue integrable functions $\phi : (0, \infty) \to \mathbb{R}_+$. Positive constants ξ, μ, δ and k are the vaccination rate of the susceptible individuals, the natural death rate of population, the disease induced death rate and the recovery rate from the infectious class, respectively. The incidence rate of a disease, defined as the average number of new cases per unit of time, we here take the nonlinear rate $\beta Sf(I)$, where β is the probability of infection by every time contact. It is biologically motivated that we make the following assumptions.

 $(\mathbb{A}_1) \ a \in [0, \hat{a}], b \in [0, \hat{b}]$ and $c \in [0, \hat{c}]$, where \hat{a}, \hat{b} and \hat{c} are the maximum ages of vaccination, latency and relapse, respectively. If $\hat{a} = \infty, \hat{b} = \infty$ and $\hat{c} = \infty$, then v(t, a) = 0, e(t, b) = 0 and r(t, c) = 0 for all enough large a, b and c, respectively.

(A₂) Functions $\omega_i(l) \in L_1^+$ are bounded with essential bounds $\bar{\omega}_i$ and Lipschitz continuous with Lipschitz constants M_{ω_i} for all $l \ge 0$ and i = 1, 2, 3.

 (\mathbb{A}_3) There exists a constant $\mu_0 > 0$ such that $\omega_i(l) \ge \mu_0$ for all $l \ge 0$ and i = 1, 2, 3.

 (\mathbb{A}_4) Function f(I) is nonnegative and twice differentiable for all $I \in [0, \infty)$ with f(I) = 0 if and only if I = 0, $f'(I) \ge 0$ and $f''(I) \le 0$ for all $I \ge 0$.

Remark 1. It is clear that for the bilinear incidence rate f(I) = I and the saturated incidence rate $f(I) = \frac{I}{1+\alpha I}$, where $\alpha > 0$ is a constant, assumption (A₄) is satisfied.

3. Preliminaries

The phase space X for model (1) is defined by $X = \mathbb{R}_+ \times L^1_+ \times L^1_+ \times \mathbb{R}_+ \times L^1_+$ equipped with the norm by

$$\|(x_1, x_2, x_3, x_4, x_5)\|_{\mathbb{X}} = |x_1| + \int_0^\infty |x_2(a)| da + \int_0^\infty |x_3(b)| db$$
$$+ |x_4| + \int_0^\infty |x_5(c)| dc$$

for any $(x_1, x_2, x_3, x_4, x_5) \in \mathbb{X}$. The initial conditions in model (1) can be rewritten as $x_0 = (S_0, \nu_0(\cdot), e_0(\cdot), I_0, r_0(\cdot)) \in \mathbb{X}$. It is easily see that for model (1)

$$\nu(0,0) = \xi S_0 = \nu_0(0), \ e(0,0) = \beta S_0 f(I_0) = e_0(0), \ c(0,0)$$
$$= kI_0 = c_0(0).$$
(2)

The standard existence, uniqueness, nonnegativity and continuability of solutions for model (1) are satisfied [27]. Thus, model (1) has a unique nonnegative solution $\mathcal{F}(t, x_0) = (S(t), v(t, \cdot), e(t, \cdot), I(t), r(t, \cdot))$ for all $t \ge 0$ with the initial condition $\mathcal{F}(0, x_0) = x_0 \in \mathbb{X}$. We have

Download English Version:

https://daneshyari.com/en/article/8254327

Download Persian Version:

https://daneshyari.com/article/8254327

Daneshyari.com