Commun Nonlinear Sci Numer Simulat 43 (2017) 271–281

Contents lists available at ScienceDirect

Commun Nonlinear Sci Numer Simulat

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

Research paper

Global stability and a comparison of SVEIP and delayed SVIP epidemic models with indirect transmission

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ARTICLE INFO

Article history:
Received 8 January 2015
Revised 29 June 2016
Accepted 13 July 2016
Available online 15 July 2016

Keywords:
Delayed system
Basic reproduction number
Global stability
Numerical analysis

ABSTRACT

The latent period is one of the important risk factors considered in epidemiological research literatures. In general, a latent period can be modelled by incorporating a delay effect (delay system), or by introducing an exposed class defined as *E*. In this paper, a susceptible-vaccinated-exposed-infectious-pathogen (SVEIP) dynamic model and its corresponding delayed SVIP model are proposed. Under biologically motivated assumptions, the stability of equilibria is investigated by the global Lyapunov functions and functionals, and the dynamical properties of two systems are found to depend entirely on the basic reproduction numbers R_0^1 and R_0^2: if $R_0^1(R_0^2) \leq 1$, the disease-free equilibrium is globally asymptotically stable; if $R_0^1(R_0^2) > 1$, the endemic equilibrium exists and is globally asymptotically stable, which implies time delay span has no effect on the stability of equilibria in delay system. Finally, a comparison between SVEIP and delayed SVIP epidemic model is made by numerical analysis, elaborating the epidemiological significance of these results.

1. Introduction

Animal diseases are widely distributed throughout the world, which have caused huge economic losses and serious public health problems. For example, around 60,000 people die annually from rabies, and the new cases of human brucellosis reach 500,000 [1–3]. Currently, the prevention and control of animal disease is one of the issues of health authorities to pay close attention. Although the strategies taken by various countries and regions are not identical, the vaccination is one of the most common control measures and has been actively applied by many countries to the prevention and control of FMD, rabies, brucellosis and other diseases. Therefore, the study on the impact of vaccination for specific animal disease has attracted the attention of many scholars, yielding many theoretical results to guide the prevention and control of specific animal disease (see [4–7]). However, most studies have assumed that the vaccinated animals are not infected in the quantitative assessment of the effectiveness of vaccination. In fact, it is an unreasonable assumption because the effective rate of the vaccination is less than 100%, for example, vaccine B.suis strain 2 can only protect 82% of animal from brucella [8]. Therefore, it is a very important risk factor that the vaccinated animals can be infected.

For the majority of animal disease, there is an incubation period when some animal diseases have no clinical features and infectious force and the infected animals can not be detected by a serological test, such as bovine brucellosis. Therefore, the impact of latency is one of the key research contents. In the study of infectious disease with the dynamic model, if time

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http://dx.doi.org/10.1016/j.cnsns.2016.07.010
1007-5704/

delay is exponentially distributed, the latent period is expressed by an extra class which is defined as E. But in reality, it often appears that an assumption of a constant delay is more reasonable, this will result in a delay differential equation. For the prevention and control of animal diseases, no matter how the incubation period is presented, it is important to investigate the global dynamics of mathematical models which are used to describe infectious animal diseases.

Many authors have formulated various animal epidemic models with direct and indirect transmission (contact transmission and pathogen infection), in which the kinetic analysis have been carried out extensively(see [9–14]). However, there is few researches by now studying the influence of the effectiveness of vaccination and the impact of latency, and no particular delay dynamic model investigating the prevalence of animal diseases. To study the influence of the two factors on the dynamics of disease transmission, two dynamic models with indirect transmission for the spread of some animal diseases are proposed based on the different representation of latencies. In fact, the treatment of infected animals is rarely attempted because of the high cost, so the standard control measures are vaccination, test and culling. Therefore, animal population is classified into four compartments: the susceptible compartment $S(t)$, the vaccinated compartment $V(t)$, the exposed compartment $E(t)$ (that is, he latent period is represented by an extra class E) and the infectious compartment $I(t)$. In addition, infectious animal can shed pathogen into the environment through the abortion or animal secretions, which can survive for several weeks, or even months in the feces or contaminated environment under the suitable conditions. Pathogen can be *harvested* by susceptible individuals that become infected individuals. Therefore, an infectious animal generates infection in two ways: the direct and indirect modes of transmission. Let $P(t)$ denote pathogens in the environment. So we have the following system:

$$\begin{cases} \frac{dS}{dt} = A - S(f(I) + g(P)) - \mu S - \nu S + \delta V, \\ \frac{dV}{dt} = \nu S - \epsilon V(f(I) + g(P)) - \mu V - \delta V, \\ \frac{dE}{dt} = (S + \epsilon V)(f(I) + g(P)) - (\mu + \sigma)E, \\ \frac{dI}{dt} = \sigma E - \varphi(I), \\ \frac{dP}{dt} = h(I) - \omega(P). \end{cases} \tag{1}$$

The elimination rate of infectious animals, including the disease induced death rate, is denoted by $\varphi(I)$. Define $h(I)$ as the pathogen shedding rate of infectious animals. $\omega(P)$ represents the disinfection rate and decaying rate of pathogen in the environment. If a constant delay is used to express the latency of animal diseases, the following delay differential equation can be obtained:

$$\begin{cases} \frac{dS}{dt} = A - S(f(I) + g(P)) - \mu S - \nu S + \delta V, \\ \frac{dV}{dt} = \nu S - \epsilon V(f(I) + g(P)) - \mu V - \delta V, \\ \frac{dI}{dt} = (S(t-\tau) + \epsilon V(t-\tau))(f(I(t-\tau)) + g(P(t-\tau)))e^{-\mu\tau} - \varphi(I), \\ \frac{dP}{dt} = h(I) - \omega(P). \end{cases} \tag{2}$$

The initial condition of system (2) is given as

$$\begin{cases} S(\theta) = \phi_1(\theta), V(\theta) = \phi_2(\theta), I(\theta) = \phi_3(\theta), P(\theta) = \phi_4(\theta), \\ \phi_k(\theta) \geq 0, \theta \in [-\tau, 0], \phi_k(0) > 0, k = 1, 2, 3, 4, \end{cases} \tag{3}$$

where $(\phi_1, \phi_2, \phi_3, \phi_4) \in C([-\tau, 0], \Re^4_{+0})$, the space of continuous functions mapping the interval $[-\tau, 0]$ into $\Re^4_{+0}$ equipped with the sup-norm, where $\Re^4_{+0} = \{(x_1, x_2, x_3, x_4) : x_i \geq 0, i = 1, 2, 3, 4\}$.

In this paper, under very general and biologically plausible assumptions on the incidence, shedding rate of pathogen, and removal rate function, and assuming the functions $\frac{f}{\varphi}$, $\frac{g}{\omega}$ and $\frac{h}{\varphi}$ are nonincreasing, the uniqueness of the endemic equilibrium is shown, and the global stability of equilibria are determined by the basic reproduction numbers R_0^1 and R_0^2: if $R_0^1(R_0^2) \leq 1$, the disease-free equilibrium of two systems is globally asymptotically stable; if $R_0^1(R_0^2) > 1$, there exists a unique endemic equilibrium for each system which is globally asymptotically stable.

This work is structured as follows. In Section 2, model assumptions and the basic reproduction number are given for system (1) and (2). Section 3 and Section 4 show the global stability of equilibria of system (1) and (2). Section 5 is numerical analysis and comparison between SVEIP model and its corresponding delayed SVIP model. A brief summary is given in Section 6.

2. The assumptions and the basic reproduction number

To make biological sense for our models, the functions f, g, φ, h and ω are assumed to be sufficiently smooth and satisfy the following hypotheses:

(H_1) $f(0) = g(0) = 0$, and $f(I)$, $g(P) \geq 0$, $f'(I)$, $g'(P) \geq 0$ for I, $P \geq 0$.
(H_2) $h(0) = 0$, $h(I) > 0$, $h'(I) > 0$ for $I > 0$.
(H_3) $\varphi(0) = 0$, and $\varphi'(I) > 0$ for $I \geq 0$; there exists constant $\mu_1 > 0$ such that $\varphi(I) \geq \mu_1 I$.
(H_4) $\omega(0) = 0$, and $\omega'(P) > 0$ for $P \geq 0$.