Global properties of a general dynamic model for animal diseases: A case study of brucellosis and tuberculosis transmission

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ABSTRACT

Animal diseases such as brucellosis and tuberculosis can be transmitted through an environmentally mediated mechanism, but the topics of most modeling work are based on infectious contact and direct transmission, which leads to the limited understanding of the transmission dynamics of these diseases. In this paper, we propose a new deterministic model which incorporates general incidences, various stages of infection and a general shedding rate of the pathogen to analyze the dynamics of these diseases. Under the biologically motivated assumptions, we derive the basic reproduction number R_0, show the uniqueness of the endemic equilibrium, and prove the global asymptotically stability of the equilibria. Some specific examples are used to illustrate the utilization of our results. In addition, we elaborate the epidemiological significance of these results, which are very important for the prevention and control of animal diseases.

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1. Introduction

Animal diseases, can be transmitted not only to other animals, but also to humans with exposure to infected animals or ingestion of contaminated water and dust in the environment. For example, brucellosis, caused by intracellular bacteria of the genus Brucella, can infect a wide variety of wild and domestic animal hosts, and is regarded as one of the most important zoonosis [15]. Although some animal diseases are well controlled in some developed countries, such as bovine brucellosis in Australia, most animal diseases are still common clinical diseases in the world [19]. Particularly, many animal diseases represent a significant public health burden on developing countries and continue receiving worldwide attention.

As for infectious animal diseases, the incubation period varies with species and stages of gestation at infection. Progression of the brucellosis infection can take two weeks to five months before the occurrence of the clinical signs, and goes through several distinct stages. The infectivity of an infected animal is determined by two main factors: brucella shed by exposed and infected animals which can survive for several weeks, or even months in the feces or contaminated environment and the frequency at which the host is in contact with others [2,14]. For the brucellosis infection, the transmissibility increases from the exposed stage to the infectious stage, and it is difficult to find the infected animals in the exposed period through the method of serum detection. For the tuberculosis (TB) infection, the usual route of infection is by inhaling infected droplets which are expelled from the lungs by coughing. Because the course of disease is slow, taking months or years to kill an infected animal, an animal can spread the disease to many other herd mates before it begins to manifest clinical

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signs [21]. Therefore, contact with undetected infected domestic animals and ingestion of the pathogen in the environment are the major ways of spreading the disease. On the other hand, it is worth noting note that an asymptomatic disease carrier may have a low pathogen load while maintaining a high infectivity due to risky behaviors.

Transmission dynamics of certain animal diseases are complicated with the multiple interaction between the animal host and the pathogen in the environment, which results in both direct animal-to-animal and indirect environment-to-animal transmission pathways. In addition, the treatment of infected animals is rarely attempted because of the high cost and the larger goal of eliminating the disease. Then the standard control measure applied to animal diseases is vaccination, test and culling. It is noteworthy that these control measures play an important role in the prevalence of some animal diseases and the impact of intervention measures needs to be carefully investigated using mathematical models. The present work aims to understand the global dynamics of these animal diseases in a general mathematical model which has a potential to incorporate different factors into a unified framework. The study tends to be crucial for effective prevention and intervention against animal diseases outbreak.

Many dynamic models have been proposed to study the complex dynamics of certain animal diseases [1,3,5,8,10,16–18]. However, most of these studies focused on analyzing the spread of animal diseases with the direct transmission (contact transmission). Therefore, one difficulty in studying infectious diseases dynamics in animals is the coupling between its multiple transmission pathways which lead to combination of animal-environment epidemiological models. In the present paper, a general dynamic model of infectious animal diseases is proposed involving both direct and indirect transmission, nonlinear incidence, and various infection stages of infectious individuals. On very general and biologically plausible assumptions of the incidence, the birth of individuals, shedding rate of pathogenic bacteria, and removal functions, we analyze the uniqueness of the endemic equilibrium and the global stability of the equilibria, which are determined by the basic reproduction number R_0: if $R_0 \leqslant 1$, the disease-free equilibrium is globally asymptotically stable; if $R_0 > 1$, there exists a unique endemic equilibrium which is the globally asymptotically stable.

This work is structured as follows. Section 1 gives the general introduction of the study background. In Section 2, the dynamic model for infectious diseases is presented. We derive the basic reproduction number and show the uniqueness of the endemic equilibrium in Section 3. In Section 4, the global stability of the disease-free equilibrium and the endemic equilibrium are established. Some examples are shown in Section 5. A brief summary is given in Section 6.

2. The model and notations

Based on common characteristics of the spread of these animal diseases, we divide the animal population into three compartments: the susceptible compartment $S(t)$, the exposed compartment $E(t)$ and the infectious compartment $I(t)$. Normally, exposed and infected animals can shed pathogenic bacteria into the environment through abortion or animal secretions, which can be *harvested* by susceptible individuals who would become infected depending on the ingested dose. Therefore, infected or exposed animals would spread the bacteria in two ways: direct and indirect modes of transmission. Define $B(t)$ as the pathogens in the environment. We propose the following compartmental model for the dynamics of infectious animal diseases based on the combination of an *SEI* model and an environmental component:

$$\begin{cases} \dfrac{dS}{dt} = m(S) - n(S)\big(\rho(E) + f(I) + g(B)\big), \\[2mm] \dfrac{dE}{dt} = n(S)\big(\rho(E) + f(I) + g(B)\big) - (e + \sigma)E, \\[2mm] \dfrac{dI}{dt} = \sigma E - \varphi(I), \\[2mm] \dfrac{dB}{dt} = \omega(E) + h(I) - \theta(B). \end{cases} \tag{1}$$

Here, the intrinsic growth rate of the susceptible class is given by $m(S)$ with all the newly produced animals assumed to be susceptible. $n(S)$ is a contact function. eE is the elimination rate of the exposed class. The transfer rate from the exposed class into the infected class is given by σE. The elimination rate of the infected animals, including the disease induced death rate, is denoted by $\varphi(I)$. We define $\omega(E)$ and $h(I)$ as the pathogen shedding rate of the exposed and infected animals respectively. $\theta(B)$ represents the disinfection rate and decaying rate of pathogen in the environment. Moreover, the functions m, n, ρ, f, g, ϕ, φ, ω, h and θ are assumed to be sufficiently smooth so that solutions to (1) with nonnegative initial conditions exist and are unique. The following basic assumptions are to make the model biologically significant:

(H_1) There exists $S_0 > 0$ such that the equation $m(S_0) = 0$ with

$$\big(m(S) - m(S_0)\big)(S - S_0) < 0 \quad \text{for } S \neq S_0.$$

(H_2) $n(S) \geqslant 0$, for $S \geqslant 0$; $n'(S) > 0$ for $S > 0$.
(H_3) $\rho(0) = f(0) = g(0) = 0$, and $\rho(E)$, $f(I)$, $g(B) > 0$, for $E, I, B > 0$.
(H_4) The functions $\rho(E)$, $f(I)$, $g(B)$ satisfy the following conditions for $E, B, I > 0$: