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Modeling and analyzing the effects of seasonality on brucellosis infection

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ABSTRACT

We present a mathematical model for the transmission dynamics of brucellosis that incorporates the effects of seasonality. We analyze the basic reproduction number associated with the time-periodic model and establish results on the threshold dynamics. Meanwhile, we perform an optimal control study on the use of animal vaccination and environmental decontamination as disease control measures against brucellosis infection. Our results show that seasonality plays an important role in shaping the long-term dynamics of brucellosis, which subsequently impacts the design of its optimal control strategies.

1. Introduction

Brucellosis, a fatal disease of humans and animals, is caused by various species of the genus brucella [7]. It is one of the most common bacterial zoonoses worldwide and it poses a major threat to human and animal health, and animal production [13]. Humans are usually infected through consumption of non-pasteurized dairy products and close-contact manipulation of infected animals. In humans, brucellosis is life threatening and exhibits nonspecific symptoms, including intermittent fever, weight loss, depression, hepatomegaly, and splenomegaly [3,8]. Arthritis, spondylitis, osteomyelitis, epididymitis, and orchitis, as well as more severe complications such as neurobrucellosis, liver abscesses, and endocarditis, are also common in some patients [3]. In animals, the transmission occurs when susceptible animals are exposed to infected animals or through ingestion of contaminated water, dust, improperly treated dairy products and so on [8]. Meanwhile, brucellosis is primarily a reproductive disease and is associated with abortion, retained placenta, and impaired fertility in the principal animal hosts [3].

Although tremendous progress has been made in controlling the disease, there are still a number of countries/regions where the infection persists in domestic animals and, consequently, transmission to the human population frequently occurs. Recent reports on animal infections [8] demonstrate that the disease is endemic in the Middle East, Asia, Africa, Latin America, the Mediterranean Basin, and the Caribbean.

Recently, mathematical models have been developed to analyze brucellosis outbreaks in an effort to better understand the intrinsic disease transmission and determine the strength and weakness of current prevention and control strategies. In particular, Hou and co-workers [5] proposed the following system of ordinary differential equations to model the transmission dynamics of brucellosis:

$$\begin{cases} \dot{S}(t) = A - \beta_1[E(t)+I(t)]S(t) - \beta_2 B(t)S(t) \\ \qquad -(\mu+\tau)S(t) + kH(t), \\ \dot{H}(t) = \tau S(t) - \gamma\beta_1[E(t)+I(t)]H(t) - \gamma\beta_2 H(t)B(t) \\ \qquad -(\mu+k)H(t), \\ \dot{E}(t) = \beta_1[S(t)+\gamma H(t)][E(t)+I(t)] \\ \qquad +\beta_2[S(t)+\gamma H(t)]B(t) - (\sigma+\mu)E(t), \\ \dot{I}(t) = \sigma E(t) - (\mu+c)I(t), \\ \dot{B}(t) = \beta_3(E+I) - (d+\delta)B, \end{cases} \tag{1}$$

where $S(t)$, $H(t)$, $E(t)$, and $I(t)$ are the numbers of the susceptible, vaccinated, exposed (latent), and infectious animals at time t, respectively. The total animal population at time t is $N(t) = S(t) + H(t) + E(t) + I(t)$. Further, $B(t)$ is the concentration of brucella in the environment, the parameter A is the recruitment rate, μ is the natural mortality rate, c is the disease-related death rate, τ is the vaccination rate, k is the immunity waning rate, β_1 is the direct disease transmission rate, β_2 is the indirect disease transmission rate, γ is the modification factor, σ is the incubation rate, β_3 is the

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pathogen shedding rate, δ is the environmental decontamination rate, and d represents pathogen decay rate. As highlighted in prior studies [5,17], exposed animals have no clinical manifestations and, without loss of generality, they can be assumed to have the same infectivity as that of the infectious animals.

This work and several other studies (see, for example, [7,8]) have certainly produced many useful results and improved the existing knowledge on brucellosis dynamics. One of the limitations of these models, however, is that they assumed that the model parameters are constant in time, implying that the disease contact rates and pathogen population growth rate, etc., all take fixed values independent of time. In fact, like many other infectious diseases, brucellosis is significantly influence by seasonal variations, and prior studies have demonstrated a strong connection between brucellosis infection and seasonal variations [1,2,20]. Factors such as the seasonal availability of forage which in turn lead to nomadic animal farming may be attributed to seasonality of brucellosis dynamics. Further, the survival of *brucella* in the environment depends critically on humidity, temperature and exposure to UV light. For example, its survival in ideal environments is reported to last up to 135 days, while a field study in the spring in Montana, USA found that Brucella abortus survived in the environment for only 21–81 days [1,2,9]. In addition, an analysis of brucellosis datasets in countries with temperate or cold climates [20] underscores that there is a marked seasonal variation in the incidence of acute brucellosis, with most cases occurring in the spring and summer. Seasonal variations also lead to periodic changes in pastures that induce animal movement and seasonal migration, resulting in disease dynamics not captured by mathematical models with constant model parameters.

From an applied perspective, understanding the mechanisms that link seasonal variations to diseases dynamics may aid in forecasting the long-term human and animal health risks, in developing an effective public health program, and in setting objectives for utilizing limited resources more effectively [10]. So far no published work has discussed the influence of seasonal variation on the transmission dynamics of brucellosis. The purpose of the present paper is to present a general brucellosis model in a periodic environment, by extending the autonomous model proposed in [5] to include seasonal variation in both the pathogen dynamics and the disease transmission pathways. We will then conduct a careful analysis on this periodic model, with a focus on its threshold dynamics characterized by the associated basic reproduction number. In addition, we will explore optimal disease control measures based on animal vaccination and environmental decontamination to contain brucellosis outbreaks, through an optimal control study. Our results are new and, to our knowledge, very little work has appeared so far on the optimal control study of periodic epidemiological models.

The remainder of this paper is organized as follows. In Section 2, we present details of our periodic brucellosis model, followed by an analysis on disease extinction and persistence that are determined by the basic reproduction number. In Section 3, we perform an optimal control study on the use of animal vaccination and environmental decontamination, through both mathematical analysis and numerical simulation. Finally, we conclude the paper with some discussion in Section 4.

2. Model with seasonal variation

2.1. Model framework

Motivated by the model (1), we propose the following non-autonomous dynamical system to describe the transmission dynamics of brucellosis in a time-periodic environment:

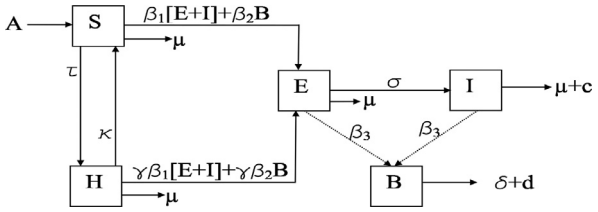

Fig. 1. Flowchart illustrating the dynamics of brucellosis.

$$\begin{cases} \dot{S}(t) = A - \beta_1(t)[E(t)+I(t)]S(t) - \beta_2(t)B(t)S(t) \\ \qquad -(\mu+\tau)S(t) + kH(t), \\ \dot{H}(t) = \tau S(t) - \gamma\beta_1(t)H(t)[E(t)+I(t)] \\ \qquad -\gamma\beta_2(t)H(t)B(t) - (\mu+k)H(t), \\ \dot{E}(t) = \beta_1(t)[S(t)+\gamma H(t)][E(t)+I(t)] + \beta_2(t)[S(t) \\ \qquad +\gamma H(t)]B(t) - (\sigma+\mu)E(t), \\ \dot{I}(t) = \sigma E(t) - (\mu+c)I(t), \\ \dot{B}(t) = \beta_3(t)(E+I) - d(t)B(t) - \delta B(t). \end{cases} \tag{2}$$

All the variables and model parameters are assumed to be positive and they retain the same definitions as in model (1). The model flow diagram is depicted in Fig. 1.

The influence of seasonal variations on the dynamics of the disease is captured by periodic functions $\beta_j(t)$ $(j=1,2,3)$ and $d(t)$. Thus, we assume that $\beta_j(t)$, $(j=1,2,3)$ are periodic continuous functions in t with a period $\omega>0$ (specifically, $\omega=12$ months). Thus,

$$\beta_j(t) = a_j\left[1 + b_j \sin\left(\frac{\pi t}{6}\right)\right], \tag{3}$$

where a_j $(j=1,2,3)$ is the baseline value or the times average of $\beta_j(t)$, and b_j $(0<b_j<1)$ denotes the magnitude of seasonal fluctuations. In addition, we define

$$d(t) = d_0\left[1 + d_1 \sin\left(\frac{\pi t}{6}\right)\right], \tag{4}$$

where d_0 denotes the basic pathogen decay rate without seasonal forcing and d_1 $(0<d_1<1)$ denotes the magnitude of seasonal fluctuations.

2.2. Basic properties of the model

It can be easily verified that the following biologically feasible domain,

$$\Gamma = \left\{(S,H,E,I,B) \in \mathbb{R}^5_+ : S+H+E+I \le \frac{A}{\mu}, \right. \\ \left. B \le \frac{2a_3(1+b_3)A}{\mu[d_0(1+d_1)+\delta]}\right\}, \tag{5}$$

is invariant for system (2). Thus we will study the dynamics of our model in the closed set Γ. In addition, we note that there is a constant influx (at rate A) into the susceptible class. Hence, without loss of generality, we assume that the susceptible population is positive at the initial time; that is,

$$S(0) > 0\,. \tag{6}$$

2.3. Disease-free equilibrium

System (2) has an evident disease-free equilibrium given by $P_0 = (S_0, H_0, 0, 0, 0)$, with

$$S_0 = \frac{A(\mu+k)}{\mu(\mu+\tau+k)}, \quad H_0 = \frac{A\tau}{\mu(\mu+\tau+k)}, \quad \text{and} \\ S_0 + \gamma H_0 = \frac{A(\mu+k+\gamma\tau)}{\mu(\mu+\tau+k)}. \tag{7}$$