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Backward bifurcation analysis of epidemiological model with partial immunity

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ABSTRACT

This paper presents a two stage SIS epidemiological model in animal population with bovine tuberculosis (BTB) in African buffalo as a guiding example. The proposed model is rigorously analyzed. The analysis reveals that the model exhibits the phenomenon of backward bifurcation, where a stable disease-free equilibrium (DFE) coexists with a stable endemic equilibrium (EE) when the associated reproduction number ($\mathfrak{R}_v$) is less than unity. It is shown under two special cases of the presented model, that this phenomenon of backward bifurcation does not arise depending on vaccination coverage and efficacy of vaccine. Numerical simulations of the model show that, the use of an imperfect vaccine can lead to effective control of the disease if the vaccination coverage and the efficacy of vaccine are high enough.

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1. Introduction

Bovine tuberculosis (BTB) is a contagious disease caused by a bacterium called *Mycobacterium bovis* (M. bovis), with a wide range of hosts such as domestic livestock, wildlife and humans. Some of such animals include cattle, goats, sheep, Badgers (Meles meles), brushtail possums (Trichosurus vulpecula), deer (Odocoileus virginianus), bison (Bison bison) and African buffalo (Syncerus caffer) which can either be reservoir or spill-over [1]. A reservoir host maintains and spreads infection whereas a spill-over host has a little or no consequence in the maintenance and spread of the infection. However, a spill-over host is referred to a dead-end host when it does not pass on the infection. BTB is a chronic and progressive disease in buffalo that leads to direct or indirect death. In buffalo herds, BTB has a high prevalence of 60% to 92% [2]. It was reported in [3] that the higher the prevalence rate the higher the disease-related mortality and hence a mortality of up to 10% was detected in buffalo herds having a BTB prevalence of at least 50%. The time from infection to death is not known but it varies and depends on the animal's immune response, which can wane by factors such as stress, drought or old age.

As in cattle the main source of BTB transmission in buffalo is by direct contact or by aerosol [3]. Vertical (intrauterine) and pseudo-vertical (through infected milk) transmissions are considered to be rare events in buffalo [2]. The mode of transmission and the route of infection within and between species are generally indicated by the locations of the tuberculous lesions in that species [1].

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In Africa most animals infected with BTB show clinical signs only when the disease has reached an advanced stage. The clinical signs of BTB in buffalo at such stage include: coughing, debilitation, poor body condition or emaciation and lagging when chased by helicopter [1,3].

Park management (Kruger National Park (KNP) and Hluhuwe-iMfolozi Park (HiP)) in South Africa have maintained some control measures such as culling, vaccination and some combination of them to control or eradicate BTB owing to its potential effects on buffalo and other host species [2,4]. However, some modeling work on BTB on buffalo suggests that vaccination may be the best control measure option since BTB may persist in buffalo population even when the population is reduced to low densities [2]. In order to assess the effectiveness of a buffalo vaccination program in South Africa some age structured mathematical models have recently been developed [2,5].

Since there is no clinical evidence which suggests that animals recover from BTB infection [2], we design a simple susceptible–infectious–susceptible (SIS) compartmental model based on the most assumptions of the two stage bovine respiratory syncytial virus epidemiological model presented in [6]. This is for the fact that BTB infection confers partial immunity and spreads among seropositive animals. The basic idea of their model is that for animal diseases which confer partial immunity from initial infection recovery, an animal may becomes lightly infected again without necessarily showing clinical symptoms of the disease. This appear for some diseases at which such seropositive animals may transmit the infection at a lower rate than animals experiencing the infection for the first time. Analysis of the two-stage model in [6] shows the possibility of backward bifurcation, and that the higher of the two subcritical equilibria (one with larger number of infective individuals) is stable whereas the lower one (one with smaller number of infective individuals) is unstable. It is argued in [7] that using two-stage model to incorporate the effect of successive exposure to infectious agents is an oversimplification. This is based on the suggestion in [8] that in some cases greater level of exposure to infectious organism may overcome the immune system and lead to a more subsequent transmission of the disease than a lower exposure. However, a three-stage model for the spread of Bovine respiratory syncytial virus in cattle may be more realistic than the two-stage one. The three-stage extended model considered in [7] is shown to exhibit more complex dynamics such as two subcritical endemic equilibria in the presence of forward bifurcation and multiple supercritical equilibria.

It is to be noted that both the two-stage and its three-stage extension in [6,7] as well as the three compartmental model in [9] are restricted to the situations where the affected population is of constant size. This assumption is reasonable for diseases that either spread quickly (i.e. in less than one year) through the population or those that spread slowly (i.e. over many years) with births approximately balanced by the natural deaths [10–12]. However, for diseases with either high disease-related mortalities or in which the births are not balanced by the deaths, this assumption is not very realistic. Several examples of animal diseases in which disease-related deaths have drastically decreased the population sizes are given in [12]. The two-stage model presented here is an extension of the model presented in [6] by incorporating vital dynamics in a population with varying size which makes the model more realistic and practically relevant. It can also be considered as an extension of the three compartmental model in [9] by considering both differential susceptibility and differential infectivity, respectively. We study this model with an aim to identify causes of backward bifurcation and to assess vaccine impact in the transmission dynamics of an epidemiological model with partial immunity and variable population.

A rigorous analysis of our model reveals some threshold values of the most important parameters in applications (i.e. reproduction number, vaccination coverage and vaccine efficacy). In fact, we obtained a critical value of the vaccinated reproduction number $\mathfrak{R}_v$, denoted by $\mathfrak{R}_v^c$ for the saddle–node bifurcation which is related with appearance and disappearance of the two endemic equilibria. In this setting, reducing $\mathfrak{R}_v$ to a value less than one is no longer sufficient for disease eradication but below $\mathfrak{R}_v^c$. Threshold analysis of the minimum effort for disease eradication taking into account the existence of backward bifurcation is presented in [9,13]. In this paper, we investigate thresholds of vaccination coverage (θ) and vaccine efficacy ($\phi = 1 - \alpha$) in relation to the coexistence region of the stable disease-free equilibrium and endemic equilibrium. These were not reported in [6,7], even though their models exhibit backward bifurcations.

The paper is organized as follows. In Section 2, we formulate the mathematical model and present its basic properties. Detailed analysis of the phenomenon of backward bifurcation and two significant special cases of the proposed model is presented in Section 3. In Section 4, the impact of vaccine is assessed via numerical simulations.

2. Mathematical model

2.1. Formulation of the model

It is well known that some animal infections may confer partial immunity and can spread among seropositive animals. Such type of disease can be modeled by the *SISI* (or $S_1I_1S_2I_2S_2$) compartmental type [6]. Following this approach, we divide the total population N of African buffalo, at time t into four distinct epidemiological classes, namely: those who have never been infected before ($S_1(t)$), those who have experienced at least one previous infection ($S_2(t)$), first time infectious ($I_1(t)$), and at least second time infectious ($I_2(t)$). Hence, the total population at any time t is given by

$$N(t) = S_1(t) + I_1(t) + S_2(t) + I_2(t).$$

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