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The dynamics of vector-borne relapsing diseases

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ABSTRACT

In this paper, we describe the dynamics of a vector-borne relapsing disease, such as tick-borne relapsing fever, using the methods of compartmental models. After some motivation and model description we provide a proof of a conjectured general form of the reproductive ratio R_0, which is the average number of new infections produced by a single infected individual. A disease free equilibrium undergoes a bifurcation at $R_0 = 1$ and we show that for an arbitrary number of relapses it is a transcritical bifurcation with a single branch of endemic equilibria that is locally asymptotically stable for R_0 sufficiently close to 1. Furthermore, we show there is no backwards bifurcation. We then show that these results can be extended to variants of the model with an example that allows for variation in the number of relapses before recovery. Finally, we discuss implications of our results and directions for future research.

1. Introduction

Many mathematical models dealing with the spread of infectious diseases show a rich variety of dynamics that arise from various nonlinear interactions or temporal forcing [1]. Vector-borne diseases are additionally complex with interactions between host and vector species [2]. Tick-borne relapsing fever (TBRF) is an example of a system that incorporates such complex interactions in a multiple host-vector community.

In North America, TBRF is caused by several species of spiral-shaped bacteria (*Borrelia* spp.) that are transmitted to their hosts through the bite of an infected vector, the soft ticks of the genus *Ornithodoros*. Once infected with the bacteria, ticks remain infectious for extended periods and possibly for life [3]. Most human cases occur in the summer months and are often associated with sleeping in rustic cabins in mountainous areas of the Western United States [4]. The model presented in this paper is motivated by a system located on Wild Horse Island, Flathead Lake, Lake County, Montana (WHI), where the presence of this pathogen has been confirmed [5]. The island harbors two host species, the red squirrel (*Tamiasciurus hudsonicus*) and the deer mouse (*Peromyscus maniculatus*) and a single vector species (*O. hermsi*), which is thought to control the disease patterns on the island. See [3] for more details.

Compartmental models, such as the SIR models with susceptible, infectious, and removed compartments, have been applied to many disease and disease-like systems in an effort to examine system dynamics [6–13]. In these epidemic models, susceptible individuals pass into the infected class and then transition to the removed class. For some diseases, recovered individuals may relapse through a reactivation of infection and revert back to an infected class. TBRF is a system in which relapse always occurs, but between different infected classes caused by the bacteria's antigenic variation [14–16]. The advantage of antigenic variation is to extend the length of infection so that the host will still be infected at the next interaction with a susceptible vector [17,18]. The questions that we raise are: (1) How do the number of relapses affect disease dynamics? and (2) How do these dynamics differ from a vector-borne disease with no relapses?

Given a mathematical model for disease spread, the disease reproduction number, R_0, is an essential summary parameter. It is defined as the average number of secondary infections produced when one infected individual is introduced into a host population in which all individuals are susceptible [19]. When $R_0 < 1$, the disease free equilibrium (DFE), at which the population remains in the absence of disease, is locally asymptotically stable. However, if $R_0 > 1$, then the DFE is unstable and invasion is always possible [20] and a new endemic equilibrium (EE) exists.

A key assumption for the host-vector disease modeling is the definition of the transmission term, which represents the contact between hosts and vectors. The formulation of the transmission term directly affects the reproduction number R_0. For host-vector disease models, the transmission term includes vector biting rate f, which controls the disease transmission both from the vector-to-host and from the host-to-vector. The TBRF model follows frequency-dependent transmission

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assumptions through the biting rate, since a blood meal is required approximately once every three months regardless of the host population density. Following this framework, it is reasonable to assume that a host would experience an increasing number of bites as the vector population increased [3]. While our work here shares techniques with previous work done on staged progression models [7,13,21–24], the key difference is the addition of vectors.

In this paper, we confirm a general form for R_0, that was conjectured in [3] when there is a single host species in the system, following the methodology of Van den Dreissche and Watmough for general compartmental disease models [25] which is then extended with an arbitrary number of relapsing states. From this we show how R_0 depends on the number of relapses and the various parameters in the model. These results are similar in form to [26] (Our thanks to the reviewer for bringing this to our attention), though there is no age-dependence in the host population in our model. Our most novel results classify the bifurcation at $R_0 = 1$, showing that it is transcritical with an exchange of stability between a disease free equilibrium and an endemic equilibrium. We also show that there is a unique endemic equilibrium for each value of $R_0 > 1$. We finally consider a variation of the model which accounts for differing number of relapses before recovery, and close with discussion and future work.

2. Single host vector model

To begin constructing the model we first make assumptions motivated by the spread of TBRF on WHI. We assume that new infections only occur when an infected vector bites a susceptible host or when a susceptible vector bites an infected host. We also assume that when a vector becomes infected, it is infected for life. Furthermore, we assume that the transmission terms are frequency dependent through the biting rate f. The infected hosts relapse into infected compartments sequentially at a rate α_i and recover from the disease at rate γ. The total populations of hosts and vectors are assumed to remain constant are denoted by N and $\widetilde{N}$ respectively (throughout the paper we will indicate quantities corresponding to the vectors with a $\sim$). The infection dynamics in a single host-vector system with $j-1$ relapsing rates for $j \geq 1$ infected compartments involve the number of susceptible hosts $S(t)$, infectious hosts $I_k(t)$, removed hosts $R(t)$, susceptible vectors $\widetilde{S}(t)$, and infected vectors $\widetilde{I}(t)$, where the total host population is $N = S + \sum_{k=1}^{j} I_k + R$ and the total vector population $\widetilde{N} = \widetilde{S} + \widetilde{I}$. A conceptual model for this scheme is given in Fig. 2.1. The equations for the model are as follows: first the host equations

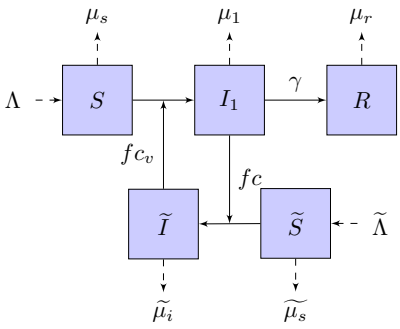
$$\begin{aligned}
S' &= \Lambda - fc_v\widetilde{I}\frac{S}{N} - \mu_s S,\\
I_1' &= fc_v\widetilde{I}\frac{S}{N} - \alpha_1 I_1 - \mu_1 I_1,\\
I_2' &= \alpha_1 I_1 - \alpha_2 I_2 - \mu_2 I_2,\\
&\vdots\\
I_{j-1}' &= \alpha_{j-2} I_{j-2} - \alpha_{j-1} I_{j-1} - \mu_{j-1} I_{j-1},\\
I_j' &= \alpha_{j-1} I_{j-1} - \gamma I_j - \mu_j I_j,\\
R' &= \gamma I_j - \mu_r R,
\end{aligned} \tag{2.1}$$

and the vector equations:

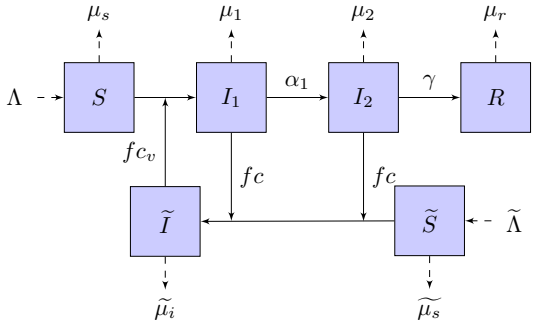
$$\begin{aligned}
\widetilde{S}' &= \widetilde{\Lambda} - \frac{fc\widetilde{S}}{N}\sum_{k=1}^{j} I_k - \widetilde{\mu}_s\widetilde{S},\\
\widetilde{I}' &= \frac{fc\widetilde{S}}{N}\sum_{k=1}^{j} I_k - \widetilde{\mu}\widetilde{I}.
\end{aligned} \tag{2.2}$$

The growth rates Λ and $\widetilde{\Lambda}$ are follows

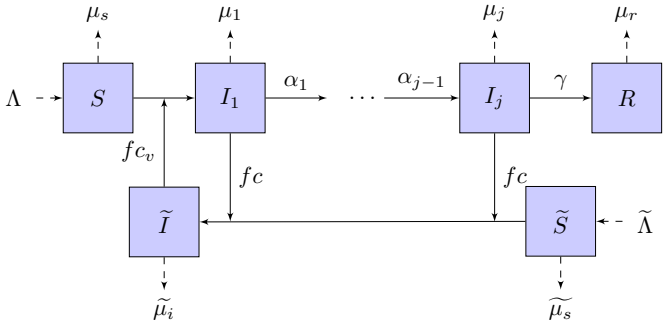
$$\Lambda = \mu_s S + \sum_{i=1}^{j} \mu_j I_j + \mu_r R, \tag{2.3}$$



(a) 0 relapses.



(b) 1 relapse



(c) $j-1$ relapses

Fig. 2.1. Conceptual models for the cross-infection dynamics between a single host-vector system, which includes (a) no relapses between $j = 1$ infected compartments, (b) 1 relapse between $j = 2$ infected compartments, and (c) $j-1$ relapses between j infected compartments. Dashed lines are the vital rates for each population, where solid lines refer to interaction rates between compartments.

$$\widetilde{\Lambda} = \widetilde{\mu}_s\widetilde{S} + \widetilde{\mu}_I\widetilde{I}. \tag{2.4}$$

Under these assumptions it is clear that the total host and vector populations are constant. These growths rates allow us to have a constant population with differential mortality, as in [27]. We let $\overline{S}$ and $\overline{S}_v$ be the constant populations for the hosts and vectors respectively. It is then easy to see that (Table 1)

$$(S, I_1, \ldots, I_j, R, \widetilde{S}, \widetilde{I}) = (\overline{S}, 0, \ldots, \overline{S}_v, 0)$$

is a fixed point of the system. This is known as the Disease Free Equilibrium (DFE). To investigate the stability of the DFE we calculate R_0 for arbitrary j.

3. R_0 for the single host-vector system with $j-1$ relapses.

3.1. Dimensionless form

To ease some calculation we will put Eqs. (2.1) and (2.2) in