

A MODEL FOR VECTOR-BORNE DISEASES: OPTIMAL TREATMENT AND PREVENTION

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ABSTRACT

In this paper we study the dynamics of a vector-transmitted disease under two assumptions. We first look at time dependent prevention and treatment efforts where optimal control theory is applied. Using analytical and numerical techniques, it is shown that there are control efforts for treatment of hosts and prevention of host-vector contacts with minimal cost and side effects. Then we considered the autonomous counter part of the first mode and here we calculated an epidemiological parameter. Based on this parameter, we established conditions for the global stability of the disease-free equilibrium point of the model. The two general models are applied to get results on a malaria disease. Using this data several numerical results about the optimal control functions, the state variables and solutions of the autonomous version are given.

INTRODUCTION

Vector-borne diseases have been and are among the leading causes of death that they remain challenges for many countries in the world, claiming the lives of millions of people every year. The health as well as the socioeconomic impacts of emerging and re-emerging vector-borne diseases is significant. It affects workforce and resources, and hence, it hinders developments. Therefore, it is among the major concerns for several health organizations including, the World Health Organization, Center for Disease Control and National Health Institute.

The geographic location and adaptation of vectors is vast and diverse. Vectors are found in areas ranging from tropical to temperate zones and at different landscapes. Although mosquitoes are commonly known vectors as a pathway for widely known diseases, others, like tsetse flies, blackflies, fleas, all blood-sucking vectors, constitute potential health hazard to mankind and sometimes to pets and livestock. While mosquitoes are common in swampy areas with vegetation where moisture and shade are available and the temperature is warm, tsetse flies which enjoy the same environment are abundant in tropical Africa [2,3].

Accordingly the diseases that are transmitted by vectors are worldwide problems. For example, dengue fever is common in tropical climates including the southern part of the United States in the Texas and Florida regions. It is transmitted by mosquito *Aedes aegypti* while malaria which is carried by female *Anopheles* mosquito is prevalent mainly in Africa and some parts of Asia [7,9]. Similarly, sleeping sickness which is carried by tsetse flies among human beings, horses and cattle [3] is common in the sub Saharan countries of Africa. West Nile virus is also

abundant worldwide including a recent outbreak in North America and Canada (Bowman et al. [5]).

Most commonly used practices of combating vector-borne diseases focus on the reduction of vectors and raising the public's awareness about prevention of host-vector contacts. A number of field and laboratory research have been conducted about vector control to find the most effective approaches to reduce vector population. This includes practicing and monitoring the efficacy of larvaciding, adultciding, spraying pesticide [10]. There are some mathematical models (discrete and continuous) of some vector-borne diseases. Some of these models are general like the discrete model studied by Blayneh and Jang [4] and others focus on specific diseases such as Malaria [6]. Other continuous models have addressed dengue fever [7]. Using a system of differential equations, Bowman et al have given a detailed analysis of the West Nile virus [5].

Given that most vectors, such as mosquitoes use favorable climatic conditions to flourish [3], combating efforts of vector-borne diseases are more effective and economical if practiced in such a way that they are in phase with climatic changes, which enhance vector outbreak. Because of these reasons, we choose the treatment and prevention control functions in one of our models to be piecewise continuous during the duration when they are administered. This duration of time is considered finite. Seasonally varying control practice could limit the side effects that any medicine may have on the individuals under treatment, and it could also cut the cost incurred due to prevention, including limiting ecological damage. Recently some epidemiological models have used control theory, most of which focus on HIV disease [1] and tuberculosis [8], but to our knowledge, optimal control theory has not been implemented to study vector-borne diseases. On the other hand, since the cycle of vector-borne diseases is going on for years thus, predicting the asymptotic dynamics of the disease in times calls for models that are capable of predicting the possibility of eradicating the disease in the long run. To address this we considered autonomous system of differential equations.

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The system of ordinary differential equations is given by

$$\begin{aligned}
\frac{dx_1}{dt} &= \delta_1 g(N) - \frac{\beta \phi y_3 x_1 (1 - u_1(t))}{N} - \mu x_1 + \psi x_4 \\
\frac{dx_2}{dt} &= \frac{\beta \phi y_3 x_1 (1 - u_1(t))}{N} - dx_2 - \mu x_2 \\
\frac{dx_3}{dt} &= dx_2 - (r + r_0 u_2(t)) x_3 - (\alpha + \mu) x_3 \\
\frac{dx_4}{dt} &= (r + r_0 u_2(t)) x_3 - (\mu + \psi) x_4 \\
\frac{dy_1}{dt} &= \delta_2 h(P) - \frac{\phi \theta x_3 y_1 (1 - u_1(t))}{N} - \gamma y_1 \\
\frac{dy_2}{dt} &= \frac{\phi \theta x_3 y_1 (1 - u_1(t))}{N} - \gamma y_2 - k y_2 \\
\frac{dy_3}{dt} &= k y_2 - \gamma y_3,
\end{aligned} \tag{1}$$

with initial conditions $x_i(0) \geq 0$, $i = 1, \dots, 4$ and $y_i(0) \geq 0$, $i = 1, 2, 3$. The control functions u_1 and u_2 represent time dependent efforts of prevention and treatment, respectively practiced

on a time interval $[0, T]$. Note that $1 - u_1(t)$ describes the failure rate of prevention efforts practiced and $u_2(t)$ is treatment effort where, $0 \leq r_0 \leq 1$ is the proportion of effective treatment. It is assumed that the recovery rate of infectious individuals is proportional to the treatment effort with proportionality constant r_0 .

The rest of the parameters in the model are given as follows. δ_1, δ_2 : per capita recruitment rate of the host and the vector population, respectively, where $g(N)$ and $h(P)$ are density dependent factors for the host and the vector, respectively; β : probability that susceptible hosts become infected after contact with an infected vector, ϕ : the average number of contact made to a host by a single vector per unit time. Thus, $\frac{\phi P}{N}$ is contact rate of vectors per host in a unit time. As $\frac{y_3}{P}$ is the proportion of infectious vectors, $x_1 \frac{\phi P}{N} \frac{y_3}{P}$ describes the total number of contacts between infectious vectors and hosts per unit time. Finally, $\beta x_1 \frac{\phi P}{N} \frac{y_3}{P}$ is the infection rate of hosts. However, the prevention effort $u_1(t)$ has impact on the reduction of the infection rate with a failure probability of $(1 - u_1(t))$. Which means while prevention efforts are practiced, the infection rate is $\beta x_1 \frac{\phi P}{N} \frac{y_3}{P} (1 - u_1(t)) = \frac{\beta \phi y_3 x_1 (1 - u_1(t))}{N}$.

The immunity of treated hosts is lost at a rate of ψ per unit time, making treated individuals susceptible to the disease. Our model assumes that infectious individuals at acute stage could die at a rate of α . The natural recovery rate r is assumed to satisfy $0 \leq r < r_0$. Note that when there is no treatment, $\frac{1}{r}$ is the mean length that a host remains infectious before it naturally recovers to join the recovered group, x_4 .

The probability that susceptible vectors get infection through contact with infectious hosts is given by θ . Thus, assuming that vectors get infected by contacting only infectious hosts, since $\frac{x_3}{N}$ is the proportion of infectious hosts, we can see that $\frac{\theta \phi x_3 y_1}{N} (1 - u_1(t))$ is the infection rate of vectors through contact with infectious hosts. We further assume that the recovery rate is greater than the disease induced mortality rate.

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