Mathematical Model to Assess the Relative Effectiveness of Rift Valley Fever Countermeasures

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**ABSTRACT**

Mathematical modeling of infectious diseases is increasingly used to explicate the mechanics of disease propagation, impact of controls, and sensitivity of countermeasures. The authors demonstrate use of a Rift Valley Fever (RVF) model to study efficacy of countermeasures to disease transmission parameters. RVF is a viral infectious disease that propagates through infected mosquitoes and primarily affects animals but also humans. Vaccines exist to protect against the disease but there is lack of data comparing efficacy of vaccination with alternative countermeasures such as managing mosquito population or destroying infected livestock. This paper presents a compartmentalized multispecies deterministic ordinary differential equation model of RVF propagation among livestock through infected *Aedes* and *Culex* mosquitoes and exercises the model to study the efficacy of vector adulticide, vector larvicide, livestock vaccination, and livestock culling on livestock population. Results suggest that livestock vaccination and culling offer the greatest benefit in terms of reducing livestock morbidity and mortality.

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1. INTRODUCTION

Rift Valley fever virus is a mosquito-borne pathogen that causes widespread febrile illness and mortality in domestic animals such as sheep, cattle and goats as well as humans (Gaff, 2007). Rift Valley fever virus was first described in peer-reviewed research in 1930 (Daubney, 1931) and was generally considered a disease primarily of sub-Saharan and southern Africa (Gaff, 2007). Then 1977, the disease moved outside of sub-Saharan and southern Africa with an outbreak occurring in Egypt; since then, outbreaks have occurred in Saudi Arabia and Yemen proving it to be a virus able to invade ecologically diverse regions (Gaff, 2007).

Over the past few decades, significant changes in the distribution and intensity of Rift Valley fever (RVF) have been recorded (WHO, 2007). Since the isolation of the virus in 1931 in the Rift Valley in Kenya, it has been held responsible for several epizootics in small ruminants, causing abortions and stillborns in the ovine species in Eastern and Southern Africa (Gerdes, 2004). Epizootics first occurred in regions of high altitude such as South Africa in 1951 (which resulted in the death of an estimated 100,000 sheep), Zimbabwe in 1958, Nigeria in 1958, and Chad and Cameroon in 1967 (WHO, 2007). Until the 1970s human infection remained low, and the agent mostly affected breeders in contact with affected or dead animals. In 1973, after the first source of infection appeared in the White Nile in Sudan, a human epidemic soon began in South Africa with the first recognized human deaths (Peters, 1994). Human outbreaks then occurred in Egypt in 1977 causing 598 human deaths (Gerdes, 2004), in 1987 causing 200 human deaths, and in Kenya and Somalia in 1997 causing 478 human deaths (CDC, 1998). In 2000, cases of RVF were discovered in Saudi Arabia and Yemen marking the spread of the disease outside of Africa and the Rift Valley (Jupp, 2002). By November 2000, over 500 cases of serious RVF were discovered in Saudi Arabia, with 87 deaths. In Yemen, between August and November 2000, there were over 1,000 suspected occurrences of the disease among humans. The result of the outbreak in Yemen was 121 deaths. Since 2000, outbreaks have occurred in Kenya and Somalia (2006), Tanzania and Sudan (2007) and Madagascar and South Africa (2008).

The potential for an exotic arbovirus to be introduced and widely established across North America can be inferred by the introduction and rapid spread of West Nile viral activity across North America in 1999 (Turell, 2008). Currently, Rift Valley fever is listed as a Category A agent on the Center for Disease Control bioterrorism list and is therefore considered a major threat to the United States. Despite the existence of several vaccines, which can protect against RVF, livestock vaccination is not currently a standardized activity in the United States. This leaves livestock culling of both exposed and infected animals as the only viable after-the-fact countermeasures. The disease is spread by infected mosquitoes whose population can vary widely, and such variance is hard to control as it primarily depends on environment factors. Should RVF be intentionally or unintentionally introduced in the Continental United States, exposed livestock is likely to encompass a significant percentage of all livestock near the vicinity of first appearance. Under such a scenario, the potential economic disruption due to loss of livestock and any subsequent trade restrictions is significant.

Disease control and public health intervention measures for RVF entail three primary countermeasures: the application of insecticide to vector populations targeting either adult mosquitoes or mosquito larvae, livestock vaccination, or finally culling of exposed and/or infected animals. Each of these countermeasures has an associated a-priori cost and corresponding posterior effectiveness; however, there is little research that allows a quantitative cost-benefit analysis among these countermeasures. In this paper, we extend a mathematical model of RVF to include transmission of
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