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RESEARCH PAPER

Rift Valley Fever Optimal Control: Insecticide Spray, Bed Net and Vaccination

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ABSTRACT

A mathematical model for the transmission of rift valley fever was developed and analyzed. The threshold epidemiological pattern of the disease and the equilibriums of the model were determined and the stabilities investigated. A sensitivity analysis of the model was carried out to establish the virtual impact of the model parameters of the spread disease. Optimal control theory was applied to investigate optimal strategies for controlling the spread of rift valley fever using Insecticide Spray, Bed net and vaccination as the control variables. The results obtained from numerical simulation of the model indicated major decline of the transmission of the disease using the three combine control strategies.

Keywords: Rift valley fever, Insecticide Spray, Bed net, Vaccination.

INTRODUCTION

Rift Valley fever (RVF) is an acute, fevercausing viral disease that significantly affects a wide range of animal species including domestic ruminants (such as cattle, buffalo, sheep, goats, and camels) and humans. It causes massive abortions in pregnant animals and death in young animals (CDC, 2007; Chamchod et al., 2014). In humans, it typically causes an influenza-like disease, (associated with headache, fever, muscle and joint pains) or severe illness (associated with hemorrhagic fever, encephalitis or ocular disease) but rarely results to more severe complications resulting to high morbidity and mortality (Shawky, 2000). Not fewer than 30 mosquito species in the genera Aedes, Anopheles, Culex, Eretmapoites, and Mansonia, and other arthropods including sand flies are responsible for spread between domestic ruminants (Fontenille et al., 1998). Though, Aedes and Culex mosquitoes are well thought-out as the main vectors (Abdo-Salem et al., 2011). The disease is caused by the RVF virus, a member of the genus *Phlebovirus* in the family Bunyaviridae. It is acknowledged to occur in epidemics in successions of 5-15 years in the Eastern Africa region and the Horn of Africa. This is mainly following unusual high precipitations that result to continuous flooding (Sumaye et al., 2013) .The disease was first reported among livestock by veterinary officers in Kenya in the early 1900s (CDC, 2007).

As a result of livestock trade and numerous species of mosquitoes that can transmit the disease, there is an emergent apprehension that the disease will re-emerge in non-endemic areas, including the United States (Kasari et al., 2008). Presently, only two types of vaccines are available for livestock: inactivated whole-virus and live-attenuated Smithburn vaccines (Davies, 2006). Inactivated vaccines can be useful to livestock of all ages devoid of causing abortions but they are expensive and repetitive doses are mandatory. Alternatively, live-attenuated vaccines are cheap and effective. They give a permanent immunity with a single dose. Nevertheless, they may lead to fetal abnormality and abortions in pregnant livestock and there is the safety concern of reversion to virulence (Ikegami and Makino, 2009).

Many models have been developed to investigate the risk of Rift valley fever. These

Received 19 October, 2016 Accepted 22 November, 2016 Address Correspondence to: <u>umarchado@gmail.com</u> include the studies of Fischer et al., (2013) developed a discrete mathematical model for the spread of Rift valley fever which is the extension of the model of Gaff et al., (2007). The authors well thought-out only one class of mosquito population and host population. Chitnis et al., (2013) presented two ordinary differential equation models for Rift Valley fever (RVF) spread in cattle and mosquitoes. They extended the model of Chitnis et al., (2006) which includes an asymptomatic host class and vertical transmission in mosquitoes to be appropriate to RVF. The new model accounts for the population dynamics of both livestock and Aedes mosquitoes and seasonal changes in weather that influences the mosquito population size. Gaff et al., (2011) extended the mathematical model for Rift Valley fever initially developed by (Gaff et al., 2007). The extension includes the vector control, livestock vaccination and culling. And other modifications to include logistic growth for the mosquitoes as a substitute to birth and death, since accounting the authors it do not make good judgment when modeling vector control measures.

Barker et al., (2013) developed a Process-based, dynamical mathematical model of virus spread. This is built from the understanding of the relations amongst virus, hosts, and vectors. The model is an extension of the model of Gaff et al., (2007). Gao et al., (2013) developed a simple epidemic three - patch model (Sudan-Nile feast) through animal's movement starting from patch 1 to patch 2 and then from patch 2 to patch 3. The authors aimed at capturing a situation where animals are introduced from Sudan into Egypt from the south and taken north along the Nile for human use, with the risk of a RVF plague if some of them are infected. Mpeshe et al., (2011) extended the deterministic model of Rift valley model of Gaff et al., 2007 where they consider only one class of mosquito population and added human population. In this modeling approach to investigate the optimal control of RVF. We address some aspects that have not been considered in previous mathematical modeling studies. Here, we propose a mathematical model that takes into account the human, mosquito and livestock populations and possibility of abortions in diseased livestock. And suggest possible optimal control for the disease. The sensitivity analysis of the model will be carried out to recognize the most sensitive parameters

for the disease transmission. The paper is organized as follows: Section 2 is dedicated to model depiction and equilibrium analysis. Sensitivity analysis of the model is presented in section 3. In section 4, the existence of control problem stated and then applies the Pontryagin's Maximum Principle to find the necessary conditions for optimal control. Numerical simulations of the models are carried out in section 5 and finally conclusion in section 6.

Model Formulation

The total human population at a time t, denoted by $N_h(t)$, is split into susceptible human $(S_h(t))$, infected humans $(I_h(t))$ and the recovered or immune human $(R_h(t))$.

Hence,
$$N_h(t) = S_h + I_h + R_h$$
. (1)

Similarly, the total Livestock population at a time t, denoted by $N_m(t)$, is split into susceptible Livestock $(S_m(t))$ and infected Livestock $(I_m(t))$ and the recovered or immune Livestock $(R_m(t))$. Thus, $N_m(t) = S_m + I_m + R_m$. (2)

Moreover, the adult *Anopheles* mosquito population at a time t, denoted by $N_v(t)$, is split into susceptible mosquitoes $(S_v(t))$ and infected mosquitoes $(I_v(t))$.

So that $N_v(t) = S_v + I_v$. (3)

Susceptible Humans

The susceptible humans are increased via recruitment of humans (by birth or immigration) into the population at a constant rate Λ_h . They diminished once the susceptible humans acquired the disease after interacting with infected *Anopheles* mosquitoes (at a rate $\beta_h \varepsilon_h c_1$), where β_h is the transmission probability per bite and ε_h is the biting rate of mosquitoes, c_1 is the contact rate of mosquito per human per unit time. The recovery or immune human losses immunity (at a rate δ_h), and turn out to be susceptible again. The susceptible human experiences natural death (at a rate μ_h). This gives:

$$\frac{dS_h}{dt} = \Lambda_h + \delta_h R_h - (\beta_h \varepsilon_h c_1 I_v + \mu_h) S_h, \qquad (4)$$

Infected Humans

The infected humans are generated through infection of susceptible. Infected humans get treated with drugs (at a rate τ) where ($0 \le \tau \le 1$). They suffer a disease-induced death (at a rate μ_0)

and natural death (at a rate μ_h). They progress to recovered class after acquiring immunity at a rate given by \mathcal{G} , where, $0 \le \mathcal{G} < \tau$. Thus, $\frac{dI_h}{dt} = \beta_h \varepsilon_h c_1 I_\nu S_h - (\mathcal{G} + \tau) I_h - (\mu_0 + \mu_h) I_h$, (5)

Recovered Humans

Recovered humans are generated from infected humans that recovered from the disease (at a rate $(\mathcal{B}+\tau)$). They loss immunity (at a rate δ_h), and turn out to be susceptible again. They suffer a natural death (at a rate μ_h).

Hence,
$$\frac{dR_h}{dt} = (\vartheta + \tau)I_h - (\delta_h + \mu_h)R_h, \quad (6)$$

Susceptible Mosquitoes

Susceptible mosquitoes are generated via recruitment of mosquitoes (birth or immigration) at a constant rate Λ_{ν} . They acquired the infections after the interaction with either (infected humans or Livestock), at a rate $(\beta_{\nu}\varepsilon_{\nu}c_{1}(I_{h}+I_{m}))$, where β_{ν} is the probability of *Anopheles* mosquitoes getting infected through

either (infected human or Livestock) and \mathcal{E}_{v} is the biting rate of mosquitoes. It experiences a

natural death (at a rate μ_{ν}).

Therefore,
$$\frac{dS_{\nu}}{dt} = \Lambda_{\nu} - (\beta_{\nu} \varepsilon_{\nu} c_1 (I_h + I_m)) + \mu_{\nu}) S_{\nu}, \quad (7)$$

Infected Mosquitoes

The infected mosquitoes are generated via the infection of the susceptible mosquitoes and diminished by natural death at a rate μ_{ν} .

This gives,
$$\frac{dI_v}{dt} = (\beta_v \varepsilon_v c_1 (I_h + I_m)) S_v - \mu_v I_v$$
, (8)

Susceptible Livestock: The susceptible Livestock are generated via recruitment of Livestock (by birth or immigration) at a constant rate Λ_m . They acquired infection after the interaction with infected mosquitoes (at a rate $\beta_m \varepsilon_m c_2$), where β_m is the transmission probability

per bite and \mathcal{E}_m is the biting rate of mosquitoes, c_2 is the contact rate of mosquitoes per Livestock per unit time. The recovered Livestock losses immunity (at a rate δ_m) and return to susceptible Livestock population. They suffer a natural death (at a rate μ_h).

Thus
$$\frac{dS_m}{dt} = \Lambda_m + \delta_m R_m - (\beta_m \varepsilon_m c_2 I_v + \mu_m) S_m, \quad (9)$$

Infected Livestock: Susceptible Livestock infected with Rift valley fever move to the infected class $(I_m(t))$ (at a rate $\beta_m \varepsilon_m c_2$). They suffer a disease-induced death and abortion of the young ones (at a rate μ_1) and natural death (at a rate μ_m).

Hence,
$$\frac{dI_m}{dt} = \beta_m \varepsilon_m c_2 I_v S_m - (\mu_1 + \mu_m + \gamma_m) I_m, \qquad (10)$$

Recovered Livestock: Infected Livestock progress to recovered class after acquiring immunity as a result of vaccination /treatment (at a rate γ_m). The recovered Livestock population losses immunity (at a rate δ_m) and suffers a natural death (at a rate μ_m).

Thus,
$$\frac{dR_m}{dt} = \gamma_m I_m - (\mu_m + \delta_m) R_m.$$
 (11)

In summary, the basic model for Rift valley fever is given by the subsequent deterministic system of non-linear differential equations.

$$\begin{cases} \frac{dS_h}{dt} = \Lambda_h + \delta_h R_h - (\lambda_h + \mu_h) S_h, \\ \frac{dI_h}{dt} = \lambda_h S_h - (\vartheta + \tau) I_h - (\mu_0 + \mu_h) I_h, \\ \frac{dR_h}{dt} = (\vartheta + \tau) I_h - (\delta_h + \mu_h) R_h, \\ \frac{dS_v}{dt} = \Lambda_v - (\lambda_v + \mu_v) S_v, \end{cases}$$

$$\begin{cases} \frac{dI_v}{dt} = \lambda_v S_v - \mu_v I_v, \\ \frac{dS_m}{dt} = \Lambda_m + \delta_m R_m - (\lambda_m + \mu_m) S_m, \\ \frac{dI_m}{dt} = \lambda_m S_m - (\mu_1 + \mu_m + \gamma_m) I_m, \\ \frac{dR_m}{dt} = \gamma_m I_m - (\mu_m + \delta_m) R_m. \end{cases}$$
(12)

with

$$\begin{cases} \lambda_{h} = \beta_{h} \varepsilon_{h} c_{1} I_{\nu}, \\ \lambda_{\nu} = \beta_{\nu} \varepsilon_{\nu} c_{1} (I_{h} + I_{m}), \\ \lambda_{m} = \beta_{m} \varepsilon_{m} c_{2} I_{\nu}. \end{cases}$$
(13)

as mass action force of infection for human, mosquito and livestock respectively.

The associated model variables and parameters are described in Table 1 and 2 respectively. While figure 1 shows the flow diagram for Rift valley fever transmission.

Table 1 Description of variables of Rift valley fever model

Parameter	Description
S_h	Susceptible human
I_h	Infected human
R_{h}	Recovered/immune
п	human
S_{v}	Susceptible mosquito
I_{v}	Infected mosquito
S_m	Susceptible Livestock
I_m	Infected Livestock
R_m	Recovered/immune
	Livestock

Table 2 Description of parameters for Rift valley fever Model

Parameter	Description	Est. Value	Ref
Λ_h	Recruitment rate of humans	[0.5 1.5]day ⁻¹	Mpeshe et al (2011)
c_1	Effective Contact rate of mosquito to human	$[0.25\ 0.375]^{day^{-1}}$	Bowman et al (2005)
μ_h	Natural death of humans	0.000054 day-1	Xue et al (2012)
9	Recovery rate of humans	0.14 day ⁻¹	Xue et al (2012)
μ_{0}	Disease – induced death of humans	[0.01 0.10] day-1	Bowman et al (2005)
Λ_{v}	Recruitment rate of mosquitoes	[2.0 6.0]	Mpeshe et al (2011)
c_2	Effective Contact rate of human to mosquitoes	$[0.25\ 0.375]\ day^{-1}$	Bowman et al (2005)
Λ_m	Recruitment rate of Livestock	$[0.5 \ 1.5] \ day^{-1}$	Mpeshe et al (2011)
$\mu_{_{v}}$	Natural death of mosquitoes	$a a y^{-1}$	Gaff <i>et al</i> (2007)
$\mu_{_m}$	Natural death of Livestock	$0.0025 day^{-1}$	Gaff <i>et al</i> (2007)
$\mu_{\scriptscriptstyle 1}$	Disease – induced death of Livestock/ abortions	$[0.025 \ 0.10] \ day^{-1}$	Peters and Linthicum (1994)
${\gamma}_m$	Recovery rate of Livestock	$0.14 day^{-1}$	Gaff <i>et al</i> (2007)
τ	Effectiveness of the Anti-rift valley drugs in human	0.8	assumed
$oldsymbol{eta}_h$	Probability of human getting infected	0.03	assumed
${\mathcal E}_h$	Biting rate of humans	0.03	
$\beta_{_{v}}$	Probability of mosquitoes getting infected	0.09	Assumed
\mathcal{E}_{v}	Biting rate of mosquitoes	0.3	Assumed
eta_m	Probability of macaques getting infected	0.03	Assumed
${\mathcal E}_m$	Biting rate of macaques	0.025	Assumed

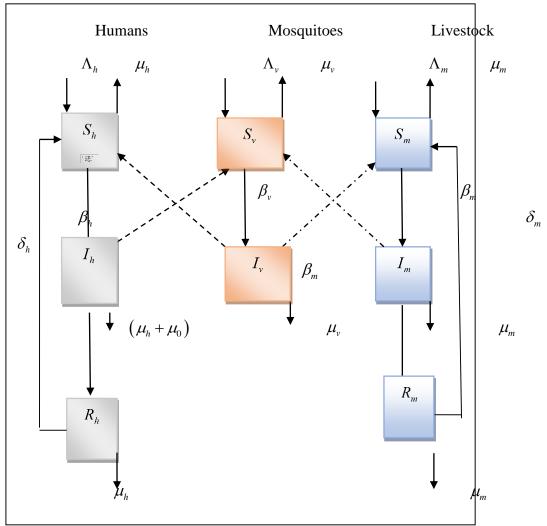


Figure 1: Flow diagram for Rift valley fever transmission

Basic Properties of the Rift Valley Fever Model Invariant Region

The total population sizes N_h, N_v and N_m can be determined by $N_h = S_h + I_h + R_h$, $N_v = S_v + I_v$ and $N_m = S_m + I_m + R_m$ or from the differential equations model $\begin{cases} \frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dI_h}{dt} + \frac{dR_h}{dt} \\ = \Lambda_h - \mu_h N_h - \mu_0 I_h, \end{cases}$ (14)

$$\begin{cases} \frac{dN_v}{dt} = \frac{dS_v}{dt} + \frac{dI_v}{dt} \\ = \Lambda_v - \mu_v N_v, \end{cases}$$
(15)

and

$$\begin{cases} \frac{dN_m}{dt} = \frac{dS_m}{dt} + \frac{dI_m}{dt} + \frac{dR_m}{dt} \\ = \Lambda_m - \mu_m N_m - \mu_1 I_m. \end{cases}$$
(16)

Assuming the disease does not kill, then $(\delta_h = 0)$ thus $\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \mu_0 I_h \le \Lambda_h - \mu_h N_h$ (17)

Lemma The model equation (12)has solutions which are contained in the feasible $\Omega = \Omega_h \times \Omega_v \times \Omega_m$.

Proof: Let $(S_h, I_h, R_h, S_v, I_v, S_m, I_m, R_m) \in \square_+^8$ be any solution of the system with nonnegative initial conditions.

Since
$$\frac{dN_h}{dt} \le \Lambda_h - \mu_h N_h$$
 (18)

Hence, by the standard comparison theorem it can be shown that

 $0 \le N_h \le \frac{\Lambda_h}{\mu_h}$, so that $\Lambda_h - \mu_h N_h \ge K e^{-\mu_h t}$ where *K* is a constant (19) Thus, all possible solutions of the human population of the model equation (12) are in the

region

$$\Omega_{h} = \left\{ \left(S_{h}, I_{h}, R_{h} \right) \in \Box_{+}^{3} : N_{h} \leq \frac{\Lambda_{h}}{\mu_{h}} \right\}.$$
(20)

Similarly, all possible solutions of the mosquito population of the model equation (12) are in the region

$$\Omega_{\nu} = \left\{ \left(S_{\nu}, I_{\nu} \right) \in \Box_{+}^{2} : N_{\nu} \leq \frac{\Lambda_{\nu}}{\mu_{\nu}} \right\}.$$
(21)

As well as all possible solutions of the Livestock population of the model equation (12) are in the region

$$\Omega_m = \left\{ \left(S_m, I_m, R_m \right) \in \Box_+^3 : N_m \le \frac{\Lambda_m}{\mu_m} \right\}.$$
(22)

Thus, the feasible set for model equation (12) is given by

$$\Omega = \begin{cases} \left(S_h, I_h, R_h, S_\nu, I_\nu, S_m, I_m, R_m\right) \in \square_+^8 : S_h, I_h, R_h, S_\nu, I_\nu, S_m, I_m, R_m \ge 0; \\ N_h \le \frac{\Lambda_h}{\mu_h}, N_\nu \le \frac{\Lambda_\nu}{\mu_\nu}, N_m \le \frac{\Lambda_m}{\mu_m} \end{cases} \end{cases}$$
(23)

which is a positively invariant set under the flow induced by the model (12)Therefore the model equation(12) is epidemiologically significant and mathematically well posed in the domain. Thus, in this domain, it is sufficient to consider the dynamics of the flow generated by the model (12). In addition, the usual existence, uniqueness and continuation of results hold for the system.

Positivity of Solutions

Lemma Let the initial data be

 $\{S_h(0), S_v(0), S_m(0) > 0, (I_h(0), I_v(0), I_m(0), R_h(0), R_m(0)) \ge 0\} \in \Omega.$

Then the solution set $\{S_h, I_h, R_h, S_v, I_v, S_m, I_m, R_m\}(t)$ of the model system (12) is positive for all t > 0.

Proof: The first equation of the model (12) gives

$$\begin{cases} \frac{dS_{h}}{dt} = \Lambda_{h} + \delta R_{h} - (\lambda_{h} + \mu_{h}) S_{h} \ge -(\lambda_{h} + \mu_{h}) S_{h} \\ \frac{dS_{h}}{dt} \ge -(\lambda_{h} + \mu_{h}) S_{h} \\ \int \frac{1}{S_{h}} dS_{h} \ge -\int (\lambda_{h} + \mu_{h}) dt \\ S_{h}(t) \ge S_{h}(0) e^{-(\lambda_{h} + \mu_{h})t} \\ S_{h}(t) \ge 0. \end{cases}$$
(24)

The second equation of (12), gives

$$\begin{cases} \frac{dI_h}{dt} = \lambda_h S_h - (\mu_h + \mu_0 + \vartheta + \tau) I_h \ge -(\mu_h + \mu_0 + \vartheta + \tau) I_h \\ \frac{dI_h}{dt} \ge -(\mu_h + \mu_0 + \vartheta + \tau) I_h \\ \int \frac{1}{I_h} dI_h \ge -\int (\mu_h + \mu_0 + \vartheta + \tau) dt \\ I_h(t) \ge I_h(0) e^{-(\mu_h + \mu_0 + \vartheta + \tau)t} \\ I_h(t) \ge 0. \end{cases}$$

(25)

From the third equation of (12)

$$\begin{cases} \frac{dR_{h}}{dt} = (\vartheta + \tau)I_{h} - (\delta_{h} + \mu_{h})R_{h} \geq -(\delta_{h} + \mu_{h})R_{h} \\ \frac{dR_{h}}{dt} \geq -(\delta_{h} + \mu_{h})R_{h} \\ \int \frac{1}{R_{h}}dR_{h} \geq -\int (\delta_{h} + \mu_{h}) dt \\ R_{h}(t) \geq R_{h}(0)e^{-(\delta_{h} + \mu_{h})t} \\ R_{h}(t) > 0. \end{cases}$$

$$(26)$$

It follows that from the fourth equation of (12)

$$\begin{cases} \frac{dS_{\nu}}{dt} = \Lambda_{\nu} - (\lambda_{\nu} + \mu_{\nu})S_{\nu} \ge -(\lambda_{\nu} + \mu_{\nu})S_{\nu} \\ \frac{dS_{\nu}}{dt} \ge -(\lambda_{\nu} + \mu_{\nu})S_{\nu} \\ \int \frac{1}{S_{\nu}}dS_{\nu} \ge -\int (\lambda_{\nu} + \mu_{\nu})dt \\ S_{\nu}(t) \ge S_{\nu}(0)e^{-(\lambda_{\nu} + \mu_{\nu})t} \\ S_{\nu}(t) \ge 0. \end{cases}$$

$$(27)$$

The fifth equation of (12), gives

$$\begin{cases} \frac{dI_{v}}{dt} = \lambda_{v}S_{v} - \mu_{v}I_{v} \ge -\mu_{v}I_{v} \\ \frac{dI_{v}}{dt} \ge -\mu_{v}I_{v} \\ \int \frac{1}{I_{v}}dI_{v} \ge -\int \mu_{v}dt \\ I_{v}(t) \ge I_{v}(0)e^{-\mu_{v}t} \\ I_{v}(t) \ge 0. \end{cases}$$

$$(28)$$

From the sixth equation of (12)

$$\begin{cases} \frac{dS_m}{dt} = \Lambda_m + \delta_m R_m - (\lambda_m + \mu_m) S_m \ge -(\lambda_m + \mu_m) S_m \\ \frac{dS_m}{dt} \ge -(\lambda_m + \mu_m) S_m \\ \begin{cases} \int \frac{1}{S_m} dS_m \ge -\int (\lambda_m + \mu_m) dt \\ S_m(t) \ge S_m(0) e^{-(\lambda_m + \mu_m)t} \\ S_m(t) \ge 0. \end{cases}$$

From the seventh equation of (12)

$$\begin{cases} \frac{dI_m}{dt} = \lambda_m S_m - (\mu_m + \mu_1 + \gamma_m) I_h \ge -(\mu_m + \mu_1 + \gamma_m) I_h \\ \frac{dI_m}{dt} \ge -(\mu_m + \mu_1 + \gamma_m) I_m \\ \begin{cases} \int \frac{1}{I_m} dI_m \ge -\int (\mu_m + \mu_1 + \gamma_m) dt \\ I_m(t) \ge I_m(0) e^{-(\mu_m + \mu_1 + \gamma_m)t} \\ I_m(t) \ge 0. \end{cases}$$

From the eighth equation of (12)

$$\begin{cases} \frac{dR_m}{dt} = \gamma_m I_h - (\delta_m + \mu_m) R_m \ge -(\delta_m + \mu_m) R_m \\ \frac{dR_m}{dt} \ge -(\delta_m + \mu_m) R_m \\ \int \frac{1}{R_m} dR_m \ge -\int (\delta_m + \mu_m) dt \\ R_m(t) \ge R_m(0) e^{-(\delta_m + \mu_m)t} \\ R_m(t) \ge 0. \end{cases}$$
(31)

Furthermore, it is necessary to show that the region Ω is positively invariant so that it

suffices the dynamics of the above system. The right hand sides of equations (14–16) are bounded by $\Lambda_h - \mu_h N_h, \Lambda_v - \mu_v N_v$ and $\Lambda_m - \mu_m N_m$ respectively, it follows that,

$$\frac{dN_{h}}{dt} < 0 \text{ if } N_{h}(t) > \frac{\Lambda_{h}}{\mu_{h}},$$
$$\frac{dN_{v}}{dt} < 0 \text{ if } N_{v}(t) > \frac{\Lambda_{v}}{\mu_{v}} \text{ and}$$
$$\frac{dN_{m}}{dt} < 0 \text{ if } N_{m}(t) > \frac{\Lambda_{m}}{\mu_{m}}.$$

Using the standard comparison theorem (Lakshmikantham *et al.*, 1988) it has been shown that,

$$N_{h} \leq \frac{\Lambda_{h}}{\mu_{h}} \left(1 - e^{(-\mu_{h}t)}\right) + N_{h}(0) e^{(-\mu_{h}t)},$$

$$N_{v} \leq \frac{\Lambda_{v}}{\mu_{v}} \left(1 - e^{(-\mu_{v}t)}\right) + N_{v}(0) e^{(-\mu_{v}t)} \text{ and }$$

$$N_{m} \leq \frac{\Lambda_{m}}{\mu_{m}} \left(1 - e^{(-\mu_{m}t)}\right) + N_{m}(0) e^{(-\mu_{m}t)}.$$

In particular, if

$$N_{h}(0) < \frac{\Lambda_{h}}{\mu_{h}} \text{ then } N_{h}(0) \le \frac{\Lambda_{h}}{\mu_{h}}, \text{ if } N_{v}(0) < \frac{\Lambda_{v}}{\mu_{v}} \text{ then } N_{v}(0) \le \frac{\Lambda_{w}}{\mu_{v}}$$

and $N_{m}(0) < \frac{\Lambda_{m}}{\mu_{m}} \text{ then } N_{m}(0) \le \frac{\Lambda_{m}}{\mu_{m}} \cdot$

Therefore, Ω is positively invariant if $N_h(0) > \frac{\Lambda_h}{\mu_h}, N_v(0) > \frac{\Lambda_v}{\mu_v}$ and $N_m(0) > \frac{\Lambda_m}{\mu_m}$, then either the solution enters Ω in finite time, or $N_h(t)$ approaches $\frac{\Lambda_h}{\mu_h}, N_v(t)$ approaches $\frac{\Lambda_v}{\mu_v}$ and $N_m(t)$ approaches $\frac{\Lambda_m}{\mu_m}$ asymptotically, and the infected state variables I_h, I_v and I_m approaches zero.

Disease – Free Equilibrium (DFE) and Stability Analysis

The steady states of the model are obtained by equating the right hand side of (12) to zero. Thus give the following:

$$\begin{cases} \Lambda_h + \delta_h R_h - (\lambda_h + \mu_h) S_h = 0 \\ \lambda_h S_h - (\vartheta + \tau) I_h - (\mu_0 + \mu_h) I_h = 0 \\ (\vartheta + \tau) I_h - (\delta_h + \mu_h) R_h = 0 \\ \Lambda_v - (\lambda_v + \mu_v) S_v = 0 \\ \lambda_v S_v - \mu_v I_v = 0 \\ \Lambda_m + \delta_m R_m - (\lambda_m + \mu_m) S_m = 0 \\ \lambda_m S_m - (\mu_1 + \mu_m + \gamma_m) I_m = 0 \\ \gamma_m I_m - (\mu_m + \delta_m) R_m = 0 \end{cases}$$
(32)

The Existence of the Trivial Equilibrium State

Provided that the recruitments rates of the populations are non-zero, the populations will not die out. This implies that there is no trivial equilibrium point, thus

$(S_h, I_h, R_h, S_v, I_v, S_m, I_m, R_m) \neq (0, 0, 0, 0, 0, 0, 0)$ Disease-Free Equilibrium State E_0

The disease-free equilibrium points (*DFE*) are equilibrium-point solutions wherever there is no disease (Rift valley fever). The infected compartment in the human, mosquito and Livestock populations that are the infected, that is; I_h, I_v and I_m in the model equation. Thus, the (*DFE*) of the basic model is given by, in the model equation(12). Thus, the (*DFE*) of the basic model (12) is given by,

$$E_{0} = (S_{h}^{*}, I_{h}^{*}, R_{h}^{*}, S_{v}^{*}, I_{v}^{*}, S_{m}^{*}, I_{m}^{*}, R_{m}^{*}) = \left(\frac{\Lambda_{h}}{\mu_{h}}, 0, 0, \frac{\Lambda_{v}}{\mu_{v}}, 0, \frac{\Lambda_{m}}{\mu_{m}}, 0, 0\right),$$
(33)

This represents the state where there exist no infectivity in a community and it is acknowledged as the disease-free equilibrium point (*DFE*).

Basic Reproduction Number R₀

The linear stability of the disease can be established using the next generation operator method (Van den Driesch & Warmouth 2002) on the model equation (1), the matrix F and V, for the new infection terms and the remaining transfer terms, are respectively, given by

$$F = \begin{pmatrix} 0 & \beta_h \varepsilon_h c_1 S_h^* & 0 \\ \beta_\nu \varepsilon_\nu c_1 S_\nu^* & 0 & \beta_\nu \varepsilon_\nu c_1 S_\nu^* \\ 0 & \beta_m \varepsilon_m c_2 S_m^* & 0 \end{pmatrix},$$
$$V = \begin{pmatrix} (\vartheta + \tau + \mu_0 + \mu_h) & 0 & 0 \\ 0 & \mu_\nu & 0 \\ 0 & 0 & (\mu_1 + \mu_m + \gamma_m) \end{pmatrix},$$

The basic reproduction number, denoted by $R_0 = \rho(FV^{-1})$, where ρ denotes the spectral radius, is given by

$$\mathbf{R}_{0} = \sqrt{\frac{(\beta_{h}\varepsilon_{h}\Lambda_{h}\mu_{m}((\mu_{1}+\mu_{m}+\gamma_{m})+\beta_{m}\varepsilon_{m}c_{2}\Lambda_{m}\mu_{h}(\vartheta+\tau+\mu_{0}+\mu_{h}))\beta_{\nu}\varepsilon_{\nu}c_{1}\Lambda_{\nu})}{(\mu_{1}+\mu_{m}+\gamma_{m})(\vartheta+\tau+\mu_{0}+\mu_{h})\mu_{m}\mu_{h}\mu_{\nu}^{2}}}$$
(34)

However, for the purpose of backward bifurcation of multi-host, the force of infection of the mosquito can be split into two. The first for human and the other for Livestock (see, for instance, the work of Blayneh *et al.*, 2010). Thus yields

$$\begin{cases} \lambda_{vh} = \beta_v \varepsilon_v c_1 I_h \\ \lambda_{vm} = \beta_v \varepsilon_v c_1 I_m \end{cases}$$

Human- mosquito interactions basic reproduction number

$$F_{1} = \begin{pmatrix} 0 & \frac{\beta_{h}\varepsilon_{h}c_{1}\Lambda_{h}}{\mu_{h}} \\ \frac{\beta_{\nu}\varepsilon_{\nu}c_{1}\Lambda_{\nu}}{\mu_{\nu}} & 0 \end{pmatrix}$$
$$V_{1} = \begin{pmatrix} \vartheta + \tau + \mu_{0} + \mu_{h} & 0 \\ 0 & \mu_{\nu} \end{pmatrix}$$

The basic reproduction number, denoted by $R_0 = \rho(FV^{-1})$, where ρ denotes the spectral radius, is given by

$$R_{0h} = \sqrt{\frac{\beta_h \varepsilon_h \Lambda_h \beta_v \varepsilon_v \Lambda_v c_1^2}{\mu_h (\vartheta + \tau + \mu_0 + \mu_h) \mu_v^2}}$$
(35)

Livestock -mosquito interactions basic reproduction number

$$F_{2} = \begin{pmatrix} 0 & \frac{\beta_{m}\varepsilon_{m}c_{2}\Lambda_{h}}{\mu_{h}} \\ \frac{\beta_{v}\varepsilon_{v}c_{1}\Lambda_{v}}{\mu_{v}} & 0 \end{pmatrix}$$
$$V_{2} = \begin{pmatrix} \gamma_{m} + \mu_{1} + \mu_{m} & 0 \\ 0 & \mu_{v} \end{pmatrix}$$

The basic reproduction number, denoted by $R_0 = \rho(FV^{-1})$, where ρ denotes the spectral radius, is given by

$$\mathbf{R}_{0m} = \sqrt{\frac{\beta_m \varepsilon_m \Lambda_m c_2 \beta_\nu \varepsilon_\nu \Lambda_\nu c_1}{\mu_m (\gamma_m + \mu_1 + \mu_m) \mu_\nu^2}}$$
(36)

using Theorem 2, of (Van den Driessche & Watmough 2002) the following result is established.

Theorem 1 The *DFE* of the model (12), given (33), is locally asymptotically stable (LAS) if $R_0 < 1$, and unstable if $R_0 > 1$.

The threshold quantity R_0 is the basic reproduction number of the disease. It is the expected average number of new infections produced by infective mosquito when introduced into a completely susceptible population. In the next section we consider the sensitivity analysis of the model parameters.

Sensitivity Analysis of Model Parameters

Sensitivity of each parameter is examined with respect to the basic reproduction number R_0 . In this way, the parameters that are more sensitive to the disease transmission are identified. And by either reducing or increasing such parameters will as well reduce or increase the transmission of the disease. Sensitivity index of the basic reproduction number R_0 , with respect to each parameter is computed as given in Table 1 for the model equation $^{(12)}$

Definition: The normalized forward sensitivity index of a variable with respect to a parameter is the ratio of the relative change in the variable to the relative change in the parameter. When the variable is a differentiable function of the parameter, the sensitivity index perhaps is on the other hand defined using partial derivatives as:

$$\Upsilon^{\mathbf{R}_r}_{\beta_{mr}} = \frac{\partial \mathbf{R}_r}{\partial \beta_{mr}} \times \frac{\beta_{mr}}{\mathbf{R}_r}$$

Sensitivity indices of R_0

Thus, the Sensitivity of R_0 to each of the (18) different parameters described in table 1 are determined using drug resistance basic reproduction number of the basic model (12) stated;

$$\mathbf{R}_{0} = \sqrt{\frac{(\beta_{h}\varepsilon_{h}\Lambda_{h}\mu_{m}((\mu_{1} + \mu_{m} + \gamma_{m}) + \beta_{m}\varepsilon_{m}c_{2}\Lambda_{m}\mu_{h}(\theta + \tau + \mu_{0} + \mu_{h}))\beta_{v}\varepsilon_{v}c_{1}\Lambda_{v})}{(\mu_{1} + \mu_{m} + \gamma_{m})(\theta + \tau + \mu_{0} + \mu_{h})\mu_{m}\mu_{h}\mu_{v}^{2}}}$$

The Sensitivity indices of R_0 with respect to $\Lambda_{\nu}, \beta_h, \beta_{\nu}, c_1, \varepsilon_{\nu}, \mu_{\nu}$ for example are,

$$\begin{cases} \Gamma_{\Lambda_{\nu}}^{\mathfrak{R}_{0}} \coloneqq \frac{\partial \mathfrak{R}_{0}}{\partial \Lambda_{\nu}} \times \frac{\Lambda_{\nu}}{\mathfrak{R}_{0}} = \frac{1}{2} \\ \Gamma_{\beta_{h}}^{\mathfrak{R}_{0}} \coloneqq \frac{\partial \mathfrak{R}_{0}}{\partial \beta_{h}} \times \frac{\beta_{h}}{\mathfrak{R}_{0}} = \frac{1}{2} \\ \Gamma_{\beta_{\nu}}^{\mathfrak{R}_{0}} \coloneqq \frac{\partial \mathfrak{R}_{0}}{\partial \beta_{\nu}} \times \frac{\beta_{\nu}}{\mathfrak{R}_{0}} = \frac{1}{2} \\ \Gamma_{c_{1}}^{\mathfrak{R}_{0}} \coloneqq \frac{\partial \mathfrak{R}_{0}}{\partial c_{1}} \times \frac{c_{1}}{\mathfrak{R}_{0}} = \frac{1}{2} \\ \Gamma_{\varepsilon_{\nu}}^{\mathfrak{R}_{0}} \coloneqq \frac{\partial \mathfrak{R}_{0}}{\partial \varepsilon_{\varepsilon_{\nu}}} \times \frac{\varepsilon_{\nu}}{\mathfrak{R}_{0}} = \frac{1}{2} \\ \Gamma_{\varepsilon_{\nu}}^{\mathfrak{R}_{0}} \coloneqq \frac{\partial \mathfrak{R}_{0}}{\partial \varepsilon_{\varepsilon_{\nu}}} \times \frac{\varepsilon_{\nu}}{\mathfrak{R}_{0}} = \frac{1}{2} \\ \Gamma_{\mu_{\nu}}^{\mathfrak{R}_{0}} \coloneqq \frac{\partial \mathfrak{R}_{0}}{\partial \mu_{\nu}} \times \frac{\mu_{\nu}}{\mathfrak{R}_{0}} = 1 \end{cases}$$
(37)

These parameters do not depend on any parameter values, while the remaining parameter depends of other parameters for their values. The parameters values are given on Table 3

Table 3: Sensitivity index of R_0

		•	
	Parameter	value	Sensitivity index
1	μ_{v}	0.025	-1
2	μ_h	0.000054	0.500028
3	${\cal E}_h$	0.03	0.50000
4	Λ_v	2.0	0.50000
5	eta_h	0.03	0.5000
6	$oldsymbol{eta}_{_{v}}$	0.09	0.5000
7	c_1	0.25	0.5000
8	\mathcal{E}_{v}	0.3	0.5000
9	τ	0.8	-0.4210
10	9	0.14	-0.07368
11	μ_0	0.01	-0.005262
12	Λ_m	0.5000	1.1864×10 ⁻⁸
13	$eta_{\scriptscriptstyle m}$	0.3	1.1864×10^{-8}
14	c_2	0.25	1.1864×10^{-8}
15	${\mathcal E}_m$	0.025	1.1864×10^{-8}
16	γ_m	0.14	-8.194×10^{-9}
17	μ_1	0.06	-3.5117×10^{-9}
18	μ_{m}	0.0027	-1.5803×10^{-10}

The parameters are arranged in ascending order, which is from the most sensitive to the least. The most sensitive parameter is the natural death rate of mosquitoes, μ_v followed by natural death of human μ_h and closely followed by contact rate

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of mosquito per human per unit , C_1 other important parameters include, , recruitment rate of mosquitoes ${}^{\Lambda_v}$ and probability of human getting infected, ${}^{\beta_h}$. The least sensitive parameter is the natural death of Livestock ${}^{\mu_m}$.

The sensitivity index of \Re_0 with respect to probability of mosquitoes getting infected (β_v) is +0.5, this implies that decreasing (or increasing) the (β_v) by 10%, decreases (or increases) \Re_0 by 5% this is the same with recruitment rate of mosquitoes Λ_v and biting rate of mosquitoes \mathcal{E}_v . Since $\Upsilon_{\mu_v}^{\Re_0} = -1$, increasing (or decreasing) μ_v by 10%, decreases (or increases) the \Re_0 by 10%.

Therefore, reducing the number of contacts between human and livestock with mosquitoes, through reducing the number of mosquitoes, this will reduce the contact and biting rate with both human and livestock. Thus, the number of bits that human and livestock will receive will have a large effect on disease transmission.

Model for Optimal Control of rift valley fever

In this section, the basic model (12) to include the possible interventions in order to reduce or limit the proliferation of the disease and the sudden increase of the infected humans and livestock in a population was extended. The associated control model to derive optimal control with minimal cost was formulated. The control efforts u_1, u_2 and u_3 represent bed nets for the susceptible humans and recovered humans, insecticide spray for the mosquito population and vaccination for the susceptible livestock respectively. The model equation (12) becomes

$$\frac{dS_{h}}{dt} = (1-u_{1})\Lambda_{h} + \delta_{h}R_{h} - (\beta_{h}\varepsilon_{h}c_{1}I_{v} + \mu_{h})S_{h}$$

$$\frac{dI_{h}}{dt} = \beta_{h}\varepsilon_{h}c_{1}I_{v}S_{h} - (\vartheta + \tau)I_{h} - (\mu_{0} + \mu_{h})I_{h}$$

$$\frac{dR_{h}}{dt} = (1-u_{1})(\vartheta + \tau)I_{h} - (\delta_{h} + \mu_{h})R_{h}$$

$$\frac{dS_{v}}{dt} = (1-u_{2})\Lambda_{h} - (1-u_{2})(\beta_{v}\varepsilon_{v}c_{1}(I_{h} + I_{m})S_{v}) - \mu_{v}S_{v}$$

$$\frac{dI_{v}}{dt} = (1-u_{2})\beta_{v}\varepsilon_{v}c_{1}(I_{h} + I_{m})S_{v} - \mu_{v}I_{v}$$

$$\frac{dS_{m}}{dt} = (1-u_{1})\Lambda_{m} + \delta_{m}R_{m} - (\beta_{m}\varepsilon_{m}c_{2}I_{v} + \mu_{m})S_{m}$$

$$\frac{dI_{m}}{dt} = \beta_{m}\varepsilon_{m}c_{2}I_{v}S_{m} - (\mu_{1} + \mu_{m} + \gamma_{m})I_{m}$$

$$\frac{dR_{h}}{dt} = (1-u_{1})\gamma_{m}I_{m} - (\delta_{m} + \mu_{m})R_{m}$$
(38)

The factor of $1-u_1(t)$ is a control function representing use of bed net at reducing infectivity in humans. The factor of $1-u_2(t)$ is a control function representing insecticide spray for reducing the mosquito population. The factor of $1-u_3(t)$ is a control function representing vaccination for livestock. This will reduce the contact rate between human and mosquitoes and the livestock and sequentially will reduce infection. Investigating the optimal control efforts that would be needed to control of rift valley fever, an optimal control problem with the objective (cost) functional is given by

$$J(u) = \int_{0}^{t} \left[A_1 I_h + A_2 I_m + B_1 u_1^2 + B_2 u_2^2 + B_3 u_2^3 \right] dt.$$

(39)

Where *T* is the final time and the coefficients A_1, A_2, B_1, B_2, B_3 are positive weights to balance the factors. The aim is to minimize the number of infected humans and livestock I_h, I_m while minimizing the cost of controls $u_1(t), u_2(t), u_3(t)$. Thus, we seek an optimal controls u_1^*, u_2^*, u_3^* such that

$$J(u_1^*, u_2^*, u_2^*) = \min_{u_1, u_2, u_3} \{ J(u_1, u_2, u_3) | u_1, u_2, u_3 \in u \}$$

Where the control set is defined as

 $U = \left\{ (u_1^*, u_2^*, u_3^*) \middle| u_i(t) \text{ is Lebesgues measurable}, i = 1, 2, 3, 0 \le u_i(t) \le 1, t \in [0, T] \right\}.$ (40)

Subject to the system (38) and appropriate initial

conditions. The basic framework of this problem is to characterize the optimal control.

Existence of Control Problem

The term ${}^{A_1I_h, A_2I_m}$ is the cost of infection of rift valley fever while ${}^{B_1u_1^2, B_2u_2^2}$ and ${}^{B_3u_1^2}$ are the costs of bed net, insecticide spray and vaccination respectively. The necessary conditions that an optimal control must satisfy come from the Pontryagin's Maximum Principle. The principle converts (38-39) into a problem of minimizing point wise a Hamiltonian H, with respect to u.

$$\begin{split} H &:= A_{1}I_{h} + A_{2}I_{m} + B_{1}u_{1}^{2} + B_{2}u_{2}^{2} + B_{2}u_{3}^{2} + \lambda_{1}((1-u_{1})\Lambda_{h} + \delta_{h}R_{h} - (\beta_{h}\varepsilon_{h}c_{1}I_{v} + \mu_{h})S_{h}) \\ &\quad + \lambda_{2}(\beta_{h}\varepsilon_{h}c_{1}I_{v}S_{h} - (9+\tau)I_{h} - (\mu_{0} + \mu_{h})I_{h}) \\ &\quad + \lambda_{3}((1-u_{1})(9+\tau)I_{h} - (\delta_{h} + \mu_{h})R_{h}) \\ &\quad + \lambda_{4}(((1-u_{2})\Lambda_{v} - (1-u_{2})(\beta_{v}\varepsilon_{v}c_{1}(I_{h} + I_{m})S_{v}) - \mu_{v}S_{v}) \\ &\quad + \lambda_{5}((1-u_{2})\beta_{v}\varepsilon_{v}c_{1}(I_{h} + I_{m})S_{v} - \mu_{v}I_{v}) \\ &\quad + \lambda_{6}((1-u_{1})\Lambda_{m} + \delta_{m}R_{m} - (\beta_{m}\varepsilon_{m}c_{2}I_{v} + \mu_{m})S_{m}) \\ &\quad + \lambda_{7}(\beta_{m}\varepsilon_{m}c_{2}I_{v}S_{m} - (\mu_{1} + \mu_{m} + \gamma_{m})I_{m}) \\ &\quad + \lambda_{8}((1-u_{1})\gamma_{m}I_{m} - (\delta_{m} + \mu_{m})R_{m}) \end{split}$$

(41)

Where $\lambda_i = 0$, for i = 1, 2, 3, 4 are adjoint variables or co-state variables.

Theorem 2 Given an optimal control u^* and solutions $(S_h^*, I_h^*, R_h^*, S_v^*, I_v^*, S_m^*, I_m^*, R_m^*)$ of the corresponding state system ⁽³⁸⁾ that minimizes $J(u_1, u_2, u_3)$ over U. Then there exists adjoint variables $\lambda_i = 0$, for i = 1, 2, ..., 8 satisfying

$$\begin{aligned} -\dot{\lambda_{1}} &= -\lambda_{1} \left(-\beta_{h} \varepsilon_{h} c_{1} I_{v} - \mu_{h} \right) - \lambda_{2} \beta_{h} \varepsilon_{h} c_{1} I_{v} I_{h} \\ -\dot{\lambda_{2}} &= \lambda_{3} \mu_{1} (\vartheta + \tau) - (\beta_{h} \varepsilon_{h} c_{1} I_{v} S_{h} + (-2\vartheta - 2\tau) I_{h} - \mu_{0} - \mu_{h}) \lambda_{2} - S_{v} \beta_{v} \varepsilon_{v} (\lambda_{4} - \lambda_{5}) (\mu_{2} - 1) c_{1} - A_{1} - \lambda_{3} \\ -\dot{\lambda_{3}} &= -\lambda_{1} \delta_{h} - \lambda_{3} (\mu_{h} - \delta_{h}) \\ -\dot{\lambda_{4}} &= -(c_{1} \beta_{v} (\lambda_{4} - \lambda_{5}) (I_{h} + I_{m}) \varepsilon_{v} + \lambda_{4} \mu_{0}) (\mu_{2} - 1) \\ -\dot{\lambda_{5}} &= -S_{h} c_{1} \beta_{h} (\lambda_{2} I_{h} - \lambda_{1}) \varepsilon_{h} + S_{m} c_{2} \beta_{m} (\lambda_{6} - \lambda_{7}) \varepsilon_{m} + \lambda_{5} \mu_{v} \\ -\dot{\lambda_{6}} &= \beta_{m} c_{2} I_{v} (\lambda_{6} - \lambda_{7}) \varepsilon_{m} + \lambda_{6} \mu_{m} \\ -\dot{\lambda_{7}} &= -S_{v} \beta_{v} \varepsilon_{v} (\lambda_{4} - \lambda_{5}) (\mu_{2} - 1) c_{1} + (\mu_{1} + \mu_{m} + \gamma_{m}) \lambda_{7} + \lambda_{8} (\mu_{3} - 1) \gamma_{m} - A_{2} \\ -\dot{\lambda_{8}} &= (\mu_{m} - \delta_{m}) \lambda_{8} - \lambda_{6} \delta_{m} \end{aligned}$$

and with transversality conditions

$$\lambda_i = 0, \, for \, i = 1, \dots, 8 \tag{42}$$

and the controls u^{*} satisfy the optimality condition

$$\begin{cases} u_1^* = \max\left\{0, \min\left(1, \frac{\lambda_1 \Lambda_h}{2B_1}\right)\right\} \\ u_2^* = \max\left\{0, \min\left(1, \frac{-c_1 \beta_\nu (\lambda_4 - \lambda_5) (I_h + I_m) \varepsilon_\nu - \lambda_4 \mu_\nu) S_\nu + \lambda_4 \Lambda_\nu}{2B_2}\right)\right\} \\ u_3^* = \max\left\{0, \min\left(1, \frac{\lambda_6 \Lambda_m + \lambda_m I_m}{2B_3}\right)\right\} \end{cases}$$

$$(43)$$

Proof The governing equations of the adjoints variables are obtained by differentiation of the Hamiltonian function, evaluated at the optimal control. And with transversality conditions

$$\lambda_i = 0, for i = 1, \dots, 8$$
(44)

On the interior of the control set, where 0 < u < 1, we have

Hence, it is obtained (see Lenhart & Workman 2007)

$$\begin{cases} u_1^* = \max\left\{0, \min\left(1, \frac{\lambda_1 \Lambda_h}{2B_1}\right)\right\} \\ u_2^* = \max\left\{0, \min\left(1, \frac{-c_1 \beta_v (\lambda_4 - \lambda_5) (I_h + I_m) \varepsilon_v - \lambda_4 \mu_v) S_v + \lambda_4 \Lambda_v}{2B_2}\right)\right\} \\ u_3^* = \max\left\{0, \min\left(1, \frac{\lambda_6 \Lambda_m + \lambda_m I_m}{2B_3}\right)\right\} \end{cases}$$

In the next section, we consider the numerical simulations.

Numerical Simulations

In this section, the numerical simulations are presented to illustrate the analytical results. Next, we examine numerically the effect of the optimal control strategies on the spread of rift valley fever in a population.

Strategy A: Optimal use of bed nets Strategy B: Optimal use of insecticide spray Strategy C: Optimal use of vaccination.

Optimal use of bed nets (u_1)

With this control strategy, use of bed nets ${}^{(u_1)}$ is used to optimize the objective functional J, while the control insecticide spray ${}^{(u_2)}$ and vaccination ${}^{(u_3)}$ are set to zero. In figure 2, the result shows a significant difference in the S_h and R_h with optimal control strategy compared to S_h and R_h without control. It was observed in figure 2(a) that the susceptible humans ${}^{(I_h)}$ decrease as a result of control strategy against the increase in the uncontrolled case. In figure 2(b) similar situation was also observed in the case of recovered humans.

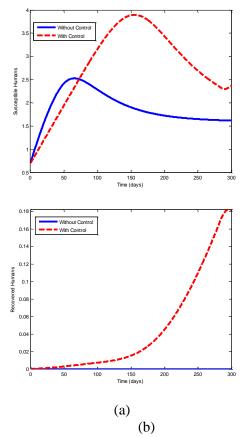


Figure 2: Simulations of the Rift valley fever model showing effect of optimal bed nets to control on the spread of the disease.

Optimal use of the insecticide spray (u_2)

In this control strategy, use of insecticide spray (u_2) is used to optimize the objective functional J, while the control bed nets (u_1) and vaccination (u_3) are set to zero. In figure 3, the result shows a significant difference in the S_v and I_v with optimal control strategy compared to S_v and I_v without control. It was observed in figure 3(a) that the susceptible mosquitoes S_v decrease as a result of control strategy against the increase in the uncontrolled case. In figure 3(b) similar situation was also observed in the case of infected mosquitoes.

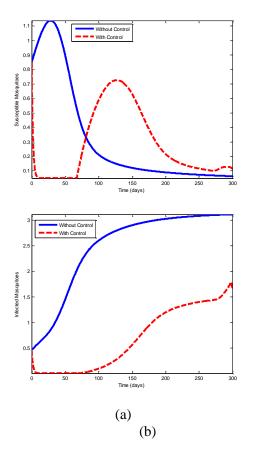


Figure 3: Simulations of the Rift valley fever model showing effect of optimal use of the insecticide spray on the spread of disease.

Optimal Use vaccination (u_3)

In this control strategy, use of vaccination (u_3) is used to optimize the objective functional J, while the control bed nets (u_1) and insecticide spray (u_2) are set to zero. In figure 4, the result shows a significant difference in the S_m and I_m with optimal control strategy compared to S_m and I_m without control. It was observed in figure 4(a) that the susceptible livestock decrease as a result of control strategy against the increase in the uncontrolled case. In figure 4(b) similar situation was also observed in the case of recovered livestock

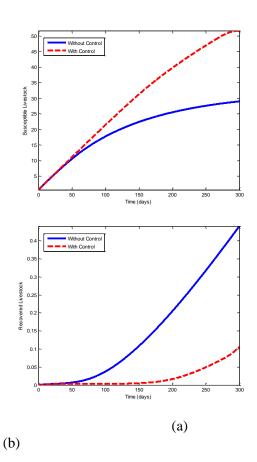


Figure 4: Simulations of the Rift valley fever model showing effect of optimal use of vaccination spray on the spread of disease.

Conclusion

In this paper, a deterministic mathematical model for Rift valley fever controls is developed. The model investigated the interaction of the human, mosquitoes and Livestock. And, it is established that the model is locally asymptotically stable when $\Re_0 < 1$ and unstable for $\Re_0 > 1$. The numerical simulation results have shown that the best control strategies for control of the disease combination of the three control strategies. However, the implication of using all the controls is that additional cost will be incurred. The optimal control for the disease is now the combination of two strategies. That is strategy A and Strategy B. It can be concluded therefore, to control the Rift valley fever; the most costeffective all of the strategies is the use of bed nets (strategy A) and insecticide spray (Strategy B)

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