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Mathematical modelling for the transmission dynamics of Rift Valley fever virus with human host

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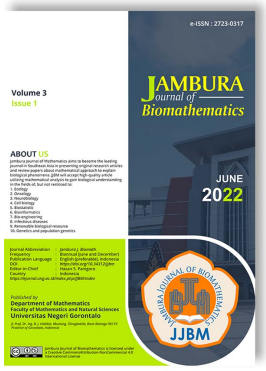

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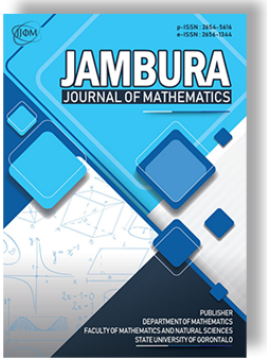

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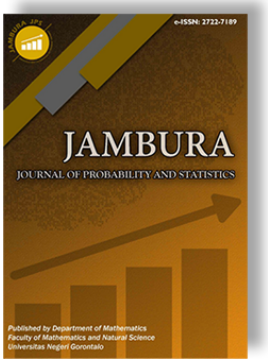

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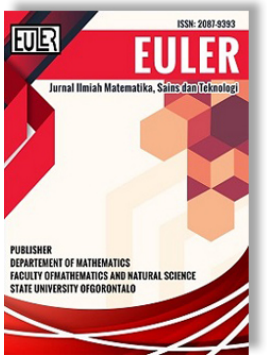

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Research Article

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Mathematical modelling for the transmission dynamics of Rift Valley fever virus with human host

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ABSTRACT. *Rift Valley Fever (RVF) is a viral zoonosis spread primarily by mosquitos that primarily affects livestock but has the potential to affect humans. Because of its potential to spread quickly and become an epidemic, it has become a public concern. In this article, the transmission dynamics of RVF with mosquito, livestock and human host using a compartmental model is studied and analyzed. The basic reproduction number R_0 is computed using next generation matrix and the disease-free equilibrium state is found to be locally asymptotically stable if $R_0 < 1$ which implies that rift valley fever could be put under control in a population where the reproduction number is less than 1. The numerical simulations give insightful results to further explore the dynamics of the disease based on the effect of three interventions; efficacy of vaccination, culling of livestock and trapping of mosquitoes introduced in the model.*

1. Introduction

Rift Valley fever (RVF) is a viral illness that can cause mild to severe symptoms in humans and livestock. RVF is also known as enzootic hepatitis of sheep and cattle [1]. It is an acute, infectious and zoonotic disease of predominantly cattle, sheep, goats, camels, African buffalo (Syncerus caffer) and humans. The disease also results in significant financial losses due to the mortality and early termination of RVF-infected animals. RVF infection is an individual from the Phlebovirus variety. The disease is caused by an arbovirus and is associated with periodic outbreaks that mostly occur on the African continent. It is a febrile disease that is accompanied by abortion in livestock and a severe fatal haemorrhagic syndrome in humans has been observed [2]. The disease was first reported among sheep in Kenya during an examination concerning a scourge among sheep on a ranch in the Rift Valley of Kenya by Montgomery in 1912 and Stordy in 1913 [1], but the disease was not isolated until 1931 [3]. From that moment forward, all incidents in Sub-Saharan Africa have been documented. The RVF infection was introduced to Egypt through diseased animals traded together with the Nile irrigation system framework in 1977, resulting in a dangerous incident. From 1997to 1998, a significant outbreak occurred in Kenya, Somalia and Tanzania following El Niño occasion and broad flooding. Following infected animal exchange from the horn of Africa, RVF spread in September 2000 to Saudi Arabia and Yemen, denoting the primary detailed event of the virus outside the African landmass and raising worries that it could stretch out to different parts of Asia and Europe [4]. Many researchers have utilized mathematical models to study the epidemiology of diseases in different populations, and they have proven to be an effective and useful tool. To gain a better knowledge of the disease's transmission dynamics and control, many models have been built and studied using various methodologies. These studies include the following, [5–12]. There is much previous research that studied critically the transmission dynamics and contagiousness of this disease in different countries alongside its evolution, most especially recently where its dynamics are considered with the COVID-19 outbreak. The analysis of different ways COVID-19 has contributed to the increase in RVF cases and how it has impacted the interventions allocated to the disease comparing it with the status of the disease before the pandemic has been studied [13, 14]. Study in [15] assessed the effectiveness of surveillance and control measures against RVF in Mayotte and in the continental EU using mathematical models. [16] further gave insight into the patterns of the transmission of RVF in humans between the 2018 and 2019 outbreak in Mayotte, using the Bayesian approach, the drivers of RVF were modelled in [17]. [18] gave a review on the endemic and epidemic status of RVF in Egypt, the virus vectors and their ecology, transmission dynamics, risk factors and the ecology of the RVF in terms of animal-human interface, prevention, control measures, use of environmental and climate data in surveillance systems to predict disease outbreaks. [19] developed a Rift Valley fever virus transmission model comprising two hosts, the analysis of the model demonstrates that both periodic and reactive vaccination to be used strategically to effectively control the disease which was also buttressed by [20] while [21] proposed and analysed a deterministic model with mosquito, livestock and human host

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as a system of non-linear ordinary differential equations and the numerical simulations support the analytical results in further exploring theoretically the long-term dynamics of the disease at the population level. [22] developed an eco-epidemiological compartment mathematical model coupled to the dynamics of ambient temperature and water availability and it was applied to a realistic setting using empirical environmental data from Kenya. The model captures the intermittent nature of RVF occurrence, explained as low-level circulation under the threshold of detection, with intermittent emergence and sometimes after long periods. Also, the patterns of seasons and socioeconomic effect on the spread of RVF were investigated by [23], the numerical results present the transmission dynamics of the disease pathogen over both short and long periods of time, particularly during the festival time. Other relevant articles like [24–31], gave more insight into the modelling of this disease with different techniques and approaches in order to better understand the dynamics and evolution of the outbreak in many scenarios. The goal of this work is to develop a model that takes into account three interventions; efficacy of vaccination, culling of livestock and trapping of mosquitoes. The other parts of the article are divided as follows: Section 2 describes the model formulation, Section 3 presents some mathematical analysis of the model, Section 4 displays the results derived from this article, Sections 5 and 6 presents the discussion of the results and conclusion from the article.

2. Methods

In formulating the model, we consider horizontal transmission in mosquitoes; control (culling rate) vector population is also considered. Humans are considered to be a source of infection to mosquitoes (contact rate from humans to vectors is assumed to be almost negligible). We also assume that livestock and humans get infected when they come in contact with infectious vectors and that the natural death rate occurs in all three groups. The model is divided into three populations; the susceptible, S_i and infected, I_i classes, for i = h, l, m for, humans (h), livestock (l) and mosquitoes (m), respectively. The two susceptible populations (humans and livestock) become infected via an infectious mosquito bite at per capita rates β_i. The newborns in each category are recruited at the per capita birth rate of Λ_i and hosts die naturally at per capita rates μ_i. Recovery in livestock is introduced at a constant rate γ_l ; recovery in humans at a constant rate γ_h. The rates for treatment are; livestock τ_l, treated humans τ_h and the vector is trapped at a constant rate δ_m. Since a population dynamics model is considered, all the state variables and parameters are assumed to be non-negative. The pictorial representation of the model formulation is presented in Figure 1. The model equation is given as follows:

$$
\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \frac{\beta_{hm} I_m S_h}{N_h} - \mu_h S_h + \gamma_h R_h \\
\frac{dE_h}{dt} &= \frac{\beta_{hm} I_m S_h}{N_h} - (\varepsilon_h + \mu_h) E_h \\
\frac{dI_h}{dt} &= \varepsilon_h E_h - (\mu_h + \tau_h) I_h \\
\frac{dR_h}{dt} &= \tau_h I_h - (\mu_h + \gamma_h) R_h
\end{aligned}
$$

$$
\begin{aligned}
\frac{dS_l}{dt} &= \Lambda_l - \frac{\beta_{lm} I_m S_l}{N_l} - (\mu_l + v_\varepsilon) S_l + \gamma_l R_l \\
\frac{dE_l}{dt} &= \frac{\beta_{lm} I_m S_l}{N_l} - (\mu_l + \tau_l + \varepsilon_l) E_l \\
\frac{dI_l}{dt} &= \varepsilon_l E_l - (\mu_l + c_l + \tau_l) I_l \\
\frac{dI_R}{dt} &= v_\varepsilon S_l + \tau_l E_l + \tau_l I_l - (\mu_l + \gamma_l) R_l \\
\frac{dS_m}{dt} &= \Lambda_m - \frac{\beta_{ml} I_l S_m}{N_m} - \frac{\beta_{mh} I_h S_m}{N_m} - (\mu_m + \delta_m) S_m \\
\frac{dI_m}{dt} &= \frac{\beta_{ml} I_l S_m}{N_m} + \frac{\beta_{mh} I_h S_m}{N_m} - (\mu_m + \delta_m) I_m
\end{aligned}
\tag{1}
$$

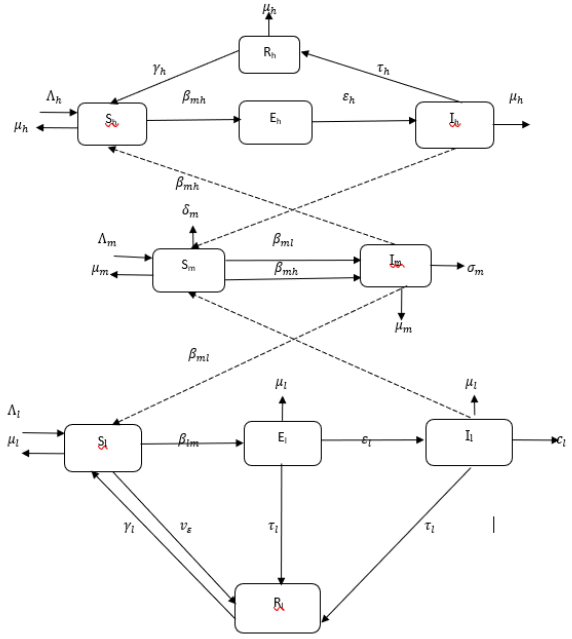

Figure 1. Schematic diagram of the model

3. Mathematical Analysis

3.1. Positivity of the solution

Theorem 1. *Let*

$$
\psi = \left(\begin{array}{l}
(S_h, E_h, I_h, R_h) \in \boldsymbol{R}^4 : S_h(0) > 0, E_h(0) > 0, \\
I_h(0) > 0, R_h(0) > 0, S_h + E_h + I_h + R_h \leq \frac{N_h}{\mu_h} \\
(S_h, E_h, I_h, R_h) \in \boldsymbol{R}^4 : S_h(0) > 0, E_h(0) > 0, \\
I_h(0) > 0, R_h(0) > 0, S_h + E_h + I_h + R_h \leq \frac{N_h}{\mu_h} \\
(S_l, E_l, I_l, R_l) \in \boldsymbol{R}^4 : S_l(0) > 0, E_l(0) > 0, \\
I_l(0) > 0, R_l(0) > 0, S_l + E_l + I_l + R_l \leq \frac{N_l}{\mu_l} \\
(S_m, I_m) \in \boldsymbol{R}^2 : S_m(0) > 0, I_m(0) > 0 \\
S_m + I_m \leq \frac{N_m}{\mu_m}
\end{array} \right)
$$

Then the solutions of $S_h, E_h, I_h, R_h, S_h + E_h + I_h + R_h, S_m, I_m$ *are positive for all* $t \geq 0$.

Table 1. Values for Population-Independent Parameter of the Model

Parameter	Value	Source
μ_l	0.5	Estimated
Λ_l	0.9	Estimated
γ_l	0.25	Estimated
ε_l	0.25	[31]
v_ε	0.25	[23]
β_{ml}	0.25	[31]
Λ_h	0.8	[27]
Λ_m	0.64	[23]
c_l	0.75	[23]
ε_h	0.25	[23]
β_{lm}	0.39	[27]
β_{mh}	0.25	[27]
β_{hm}	0.001	[27]
τ_h	0.25	[23]
τ_l	0.25	[23]
δ_m	0.25	[28]
μ_h	0.01	Assumed
μ_m	0.67	Assumed

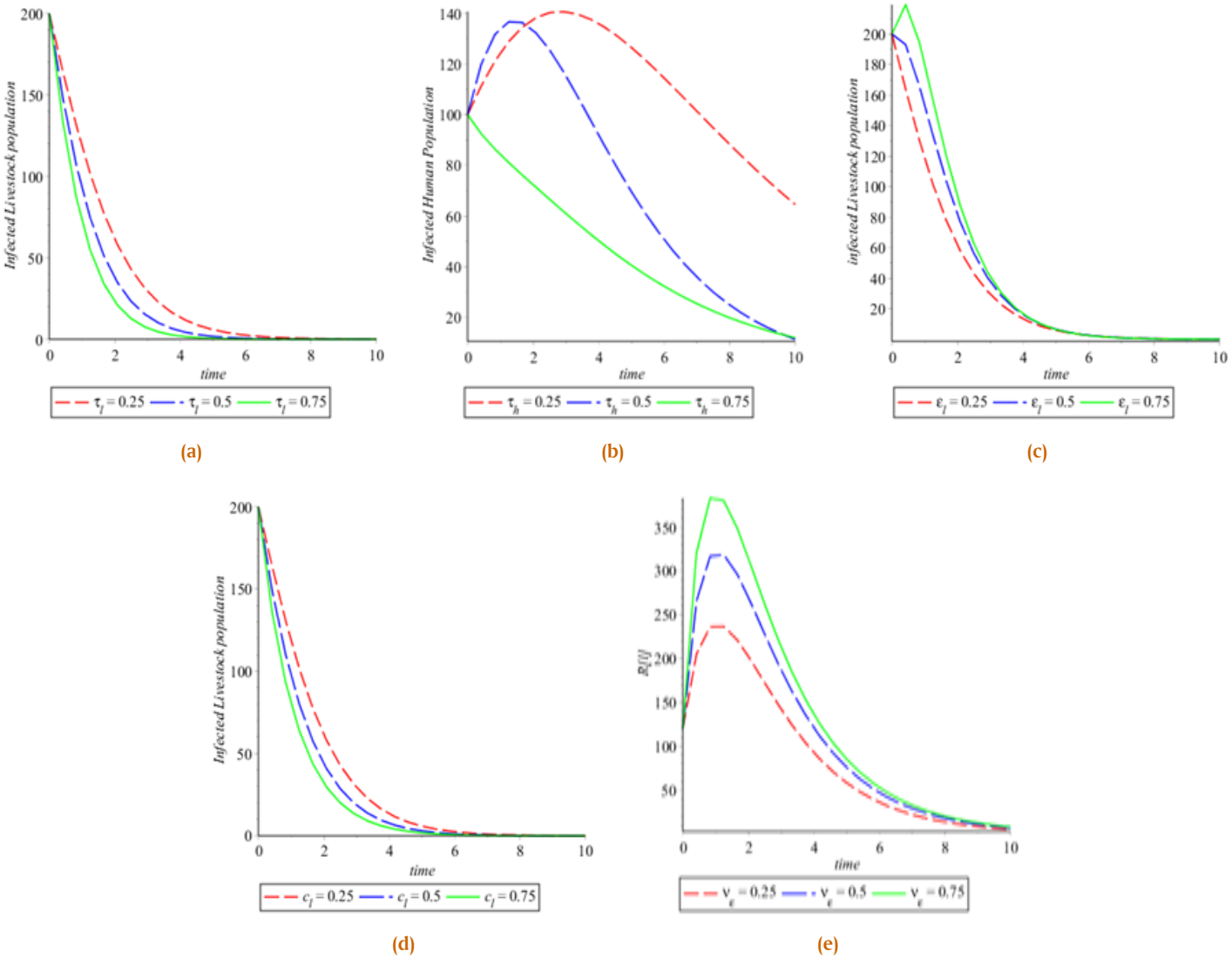

Figure 2. Numerical simulations of model (1) using parameter values in Table 1. **(a)** Effect of treatment on infected livestock population, **(b)** Effect of treatment of humans on infected human population, **(c)** Effect of incubation period on infected livestock population, **(d)** Culling rate of livestock on infected livestock population, and **(e)** Effect of vaccination of livestock on recovered livestock population

Proof.

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \frac{\beta_h m I_m S_h}{N_h} - \mu_h S_h + \gamma_h R_h \\
\frac{dS_h}{dt} &\geq -\mu_h S_h \\
\frac{dS_h}{S_h} &\geq -\mu_h dt \\
\int \frac{dS_h}{S_h} &\geq \int -\mu_h dt \\
S_h(t) &\geq S_h(0)\exp(-\mu_h t) \geq 0
\end{aligned} \tag{2}$$

Similarly, it can be shown that the remaining nine equations in (1) are also positive for all $t \geq 0$. □

3.2. *Effective reproduction number, Rc*

Using Next Generation Matrix [32, 33], the Jacobian of Infection term matrix is given as

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 & \beta_m \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\beta_{lm}k_7}{k_7+v_\varepsilon} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_{mh} & 0 & \beta_{ml} & 0 \end{bmatrix}, \tag{3}$$

and the jacobian of transmission term matrix is

$$V^-1 = \begin{bmatrix} \frac{1}{k_1} & 0 & 0 & 0 & 0 \\ \frac{\varepsilon_h}{k_1k_2} & \frac{1}{k_2} & 0 & 0 & 0 \\ 0 & 0 & \frac{1}{k_5} & 0 & 0 \\ 0 & 0 & \frac{\varepsilon_l}{k_5k_6} & \frac{1}{k_6} & 0 \\ 0 & 0 & 0 & 0 & \frac{1}{k_8} \end{bmatrix}, \tag{4}$$

$$FV^-1 = \begin{bmatrix} 0 & 0 & 0 & 0 & \frac{\beta_{hm}}{k_8} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\beta_{lm}k_7}{k_7+v_\varepsilon} \\ 0 & 0 & 0 & 0 & 0 \\ \frac{\beta_{mh}\varepsilon_h}{k_1k_2} & \frac{\beta_{mh}}{k_2} & \frac{\beta_{ml}\varepsilon_l}{k_5k_6} & \frac{\beta_{ml}}{k_6} & 0 \end{bmatrix}, \tag{5}$$

such that

$$\begin{aligned}
\lambda_1 &= 0,\ \lambda_2 = 0,\ \lambda_3 = 0, \\
\lambda_4 &= \frac{\sqrt{\lambda_{4a}}}{k_1k_2k_5k_6k_8\left(k_7+v_\varepsilon\right)}, \\
\lambda_5 &= -\frac{\sqrt{\lambda_{5a}}}{k_1k_2k_5k_6k_8\left(k_7+v_\varepsilon\right)}, \\
\lambda_{4a} &= k_1k_2k_5k_6k_8\left(k_7+v_\varepsilon\right)\left(\beta_{ml}\beta_{lm}k_1k_2k_7\varepsilon_l\right. \\
&\quad \left.+\beta_{mh}\beta_{hm}k_5k_6k_7\varepsilon_h + \beta_{mh}\beta_{hm}k_5k_6v_\varepsilon\varepsilon_h\right), \\
\lambda_{5a} &= k_1k_2k_5k_6k_8\left(k_7+v_\varepsilon\right)\left(\beta_{ml}\beta_{lm}k_1k_2k_7\varepsilon_l\right. \\
&\quad \left.+\beta_{mh}\beta_{hm}k_5k_6k_7\varepsilon_h + \beta_{mh}\beta_{hm}k_5k_6v_\varepsilon\varepsilon_h\right).
\end{aligned}$$

Clearly, λ_4 is the dominant eigen value

$$R_c = \frac{\sqrt{\lambda_{4a}}}{k_1k_2k_5k_6k_8\left(k_7+v_\varepsilon\right)} \tag{6}$$

3.3. *Disease Free Equilibrium (DFE) State*

Disease free equilibrium states are steady when all the infectious classes in a population are zero, that is; the population comprises of susceptible humans and vectors only. At Disease Free Equilibrium;

$$\begin{aligned}
E^0 &= (S_h, E_h, \mathrm{I}_h, R_h, S_l, E_l, \mathrm{I}_l, R_l, S_m, \mathrm{I}_m) \\
&= \left(\frac{\Lambda_h}{\mu_h}, 0, 0, 0, \frac{\Lambda_l k_7}{k_4k_7 - v_\varepsilon\gamma_l}, 0, 0, \frac{\Lambda_l v_\varepsilon}{k_4k_7 - v_\varepsilon\gamma_l}, \frac{\Lambda_m}{k_8}, 0\right)
\end{aligned} \tag{7}$$

3.4. *Local stability of disease -free equilibrium state*

We investigate the local stability at the equilibrium points.

Proof

Linearizing the model eq. (1) at any arbitrary equilibrium point (E^*) gives the Jacobian

$$J\left(E^o\right) = \begin{bmatrix} a_1 & 0 & 0 & \gamma_h & 0 & 0 & 0 & 0 & 0 & -\beta_{hm} \\ 0 & a_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \beta_{hm} \\ 0 & 0 & a_3 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{\beta_{hm}\varepsilon_h}{k_1} \\ 0 & 0 & 0 & a_4 & 0 & 0 & 0 & 0 & 0 & A_1 \\ 0 & 0 & 0 & 0 & a_5 & 0 & 0 & \gamma_l & 0 & -A_2 \\ 0 & 0 & 0 & 0 & 0 & a_6 & 0 & 0 & 0 & A_2 \\ 0 & 0 & 0 & 0 & 0 & 0 & a_7 & 0 & 0 & A_3 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_8 & 0 & A_5 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_9 & -A_6 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_{10} \end{bmatrix},$$

where $a_1 = -(\mu_h + \lambda)$, $a_2 = -(k_1 + \lambda)$, $a_3 = -(k_2 + \lambda)$, $a_4 = -(k_3 + \lambda)$, $a_5 = -(k_4 + \lambda)$, $a_6 = -(k_5 + \lambda)$, $a_7 = -(k_6 + \lambda)$, $a_8 = -(A_4 + \lambda)$, $a_9 = -(k_8 + \lambda)$, and $a_{10} = A_7 - \lambda$. Therefore, the eigenvalues are

$$\begin{aligned}
\lambda_1 &= -\mu_h < 0, \\
\lambda_2 &= -k_1 = -(\mu_h + \varepsilon_h) < 0, \\
\lambda_3 &= -k_2 = -(\mu_h + \tau_h) < 0 \\
\lambda_4 &= -k_3 = -(\mu_h + \gamma_h) < 0 \\
\lambda_5 &= -k_4 = -(\mu_l + v_\varepsilon) < 0 \\
\lambda_6 &= -k_5 = -(\mu_l + \tau_l + \varepsilon_l) < 0 \\
\lambda_7 &= -k_6 = -(\mu_l + c_l + \tau_l) < 0 \\
\lambda_8 &= -A_4 = -\left(\frac{k_4k_7 - \gamma_l v_\varepsilon}{k_4}\right) \\
&= \frac{-\mu_l(\mu_l + \gamma_l + v_\varepsilon)}{\mu_l + v_\varepsilon} < 0 \\
\lambda_9 &= -k_8 = -(\mu_m + \delta_m) < 0 \\
\lambda_{10} &= A_7 \\
&= \frac{\begin{array}{l} k_5k_6k_7\beta_{hm}\beta_{mh} + k_1k_7\varepsilon_l\beta_{lm}\beta_{ml} + k_5k_6v_\varepsilon\beta_{hm}\beta_{mh} \\ +k_1k_5k_6k_7k_8 + k_1k_5k_6k_8v_\varepsilon \end{array}}{k_1k_5k_6\left(k_7+v_\varepsilon\right)}
\end{aligned}$$

Therefore $R_c < 1$. This implies that $\lambda_{10} < 0$ if $R_c < 1$. Hence, the disease-free equilibrium E^o of the equations is locally asymptotically stable (LAS) if $R_c < 1$.

4. Results

We present in this section the parameters used for the simulation in Table 1 and the graphical representation of various numerical simulations results in Figure 2a. We varied some of the

parameters ($\upsilon_\varepsilon, \varepsilon_l, \tau_h$) of the model using the values between 0 and 1 in order to see the effect of this variation on the model we formulated. Other parameters are presented in Table 1.

5. Discussion of the Results

Figure 2a shows the effect of treating livestock, this causes a decrease in the infected class of livestock. Figure 2b shows the relationship between treatment of humans and infected human population. The higher the rate of treatment, the lower the infected human population. Figure 2c shows the relationship between incubation period and infected livestock. The longer the incubation period, the higher the spread of RVF virus among the vectors and eventually increase in infected livestock population. Figure 2d shows that the increase in the elimination of infected livestock (culling rate), decreases the infected livestock population. Figure 2e shows the efficacy of vaccination on livestock; the more livestock are vaccinated, the more the population of recovered livestock. For the numerical simulations, the time are in weeks.

6. Conclusion

The Rift Valley Model formulated in this work exists in a feasible region where disease free and endemic equilibrium points are obtained and the local stability of disease-free equilibrium was investigated. The positivity of solutions using Wiah's method was also determined. The model has three interventions; efficacy of vaccination, culling of livestock and trapping of mosquitoes. The model showed that disease free equilibrium exists and is locally asymptotically stable whenever it is associated with effective reproduction number, that is Rc < 1 and it has a unique endemic equilibrium when $Rc > 1$. These results have important public health implications, since they determine the severity and outcome of the epidemic that is, clearance or persistence of infection and provide a framework for the design of control strategies. Analysis of the model showed that there exist two possible solutions, namely the disease-free point and the endemic equilibrium point of the forces of infection. Further analysis showed that the disease-free point is locally stable implying that small perturbations and fluctuations on the disease state will always result in the eradication of the disease if $Rc < 1$. In the final analysis, efficacy of vaccination, culling of livestock and trapping intervention program will effectively control the spread of rift valley fever. The result in this article will further guide decision makers to proper manage the outbreak of this disease in different countries.

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