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ANALYZING THE MATHEMATICAL IMPACT OF VACCINATION IN MITIGATING RIFT VALLEY FEVER SPREAD AMONG LIVESTOCK

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The persistence mechanisms of Rift Valley fever (RVF), a zoonotic arboviral hemorrhagic fever, are not fully understood and need thorough quantification at both local and broader geographical scales. Rift Valley Fever (RVF) is a viral zoonosis primarily transmitted by mosquitoes, predominantly affecting livestock with the potential to impact humans. The virus has the capacity for rapid spread, posing a potential epidemic threat to both human and the livestock. The transmission dynamics of Rift Valley Fever (RVF) involving mosquitoes and livestock are investigated and analyzed through a compartmental model, with vaccination considered as a control measure. The basic reproduction number (R_0) is calculated using the next-generation matrix, indicating that the disease-free equilibrium state is locally asymptotically stable when ($R_0 < 1$). This suggests that Rift Valley Fever could be controlled in a livestock population where the reproduction number is below 1, but it becomes endemic when ($R_0 > 1$). Sensitivity analysis identifies key parameters for consideration by livestock policy makers and veterinary workers. Numerical

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simulations offer insightful results to delve deeper into the disease dynamics, considering the efficacy of vaccination and other control measures introduced in the model.

Keywords: Rift-Valley Fever, Vaccination, Equilibrium Points, Reproduction Number, Stability, Sensitivity

INTRODUCTION

Rift Valley fever (RVF) is a viral disease with implications for both human and livestock health, causing a range of symptoms from mild to severe. Often referred to as enzootic hepatitis of cattle and sheep (Adeyeye et al., 2011; Mpeshe et al., 2011; Pedro et al., 2014, Oguntolu et al., 2022). RVF primarily affects cattle, sheep, goats, camels, African buffalo, and humans. Beyond the direct impact on health, the disease leads to significant economic losses due to mortality and the premature termination of infected animals (Xiao et al., 2015; Tennant et al.,2021). RVF is caused by an arbovirus belonging to the Phlebovirus genus, characterized by periodic outbreaks, primarily in the African continent. The disease manifests as a febrile illness, contributing to abortion in livestock and, in severe cases, a fatal hemorrhagic syndrome in humans (Evans et al., 2008). The first reported cases date back to 1912 when an examination of a sheep scourge in Kenya's Rift Valley revealed the presence of the disease. However, it wasn't until 1931 that the RVF virus was isolated (Kanouté et al., 2017). Subsequent incidents, such as the introduction of the virus to Egypt in 1977 through the trade of diseased animals and a notable outbreak in Kenya, Somalia, and Tanzania in 1997-1998 following El Niño and extensive flooding, underscore the global impact of RVF (Kanouté et al., 2017). In a concerning development, RVF spread outside the African continent in 2000, reaching Saudi Arabia and Yemen after infected animal trade from the Horn of Africa. This marked the first reported instance of the virus extending beyond Africa, prompting fears of potential dissemination to other parts of Asia and Europe. The multifaceted nature of RVF, involving both animal and human health, necessitates comprehensive strategies for surveillance, control, and prevention to mitigate its impact on public health and the economy (WHO 2023). Numerous scholars have employed mathematical models as effective and valuable tools to investigate the epidemiology of diseases across diverse populations (Adesola et al., 2024a, 2024b; Philemon et al., 2023; Ajao et al., 2023; Musbau et al., 2022; Akinwumi et al., 2021; Fawzy

et al., 2019; Métras et al., 2017; Adesanya et al., 2016a; Bird et al., 2016; Gachohi et al., 2016; Pedro et al., 2016; Fischer et al., 2013). Extensive previous studies have comprehensively investigated the transmission dynamics and contagious characteristics of Rift Valley fever in both livestock and human populations. Research conducted by (Nielsen 2021) evaluated the efficacy of surveillance and control measures against Rift Valley fever (RVF) in Mayotte and the continental European Union (EU) through the application of mathematical models. The study aimed to assess the impact and efficiency of various strategies in preventing and managing RVF outbreaks in these regions. In the study conducted by (Métras et al., 2020), a detailed examination of the transmission patterns of Rift Valley fever (RVF) in humans during the outbreaks in Mayotte between 2018 and 2019 was undertaken. The researchers employed a Bayesian approach to provide valuable insights into the dynamics and spread of RVF within the human population during this specific time frame. In their research, (Iacono et al., 2018) constructed an eco-epidemiological compartmental mathematical model that incorporates the influence of ambient temperature and water availability. The model was specifically tailored to a realistic setting, utilizing empirical environmental data obtained from Kenya. By integrating these environmental factors, the model successfully captures the intermittent nature of Rift Valley fever (RVF) occurrence. This phenomenon is elucidated as low-level circulation that remains under the threshold of detection, experiencing intermittent emergence, and at times reappearing after extended intervals. The study provides a comprehensive understanding of the complex dynamics of RVF in relation to environmental factors, shedding light on the sporadic nature of its occurrence.

The objective of this study is to formulate a model that incorporates the effectiveness of vaccinating livestock, aiming to provide a comprehensive understanding of the impact of vaccination on the dynamics of the disease in the animal population. Through this model, we seek to explore and quantify the role of vaccination efficacy in mitigating the spread of the infectious agent among livestock.

In Section 2, the model formulation is detailed, while Section 3 provides mathematical analysis of the model. The results obtained from this study are presented in Section 4, followed by discussions and conclusions in Sections 5.

Model Formulation

In constructing the model, it is assumed that livestock contract the infection through contact with infectious mosquitoes, and a consistent natural death rate denoted by μ, which applies uniformly across all compartments. Further details on parameters can be found in Table 2. The model is divided into two (2) populations; the livestock and mosquito population. The livestock population is subdivided into six (6) compartments, i.e. S_L Susceptible, V_L Vaccinated, E_L Exposed, Sy_L Symptomatic, Ay_L Asymptomatic and R_L Recovered.

The vector population is sub divided into three (3) compartments; S_V Susceptible, E_V Exposed and I_V Infected.

Livestock

$$
\begin{aligned}
S_L' &= (1-\rho)\pi_L - \lambda_L S_L - \mu_L S_L + \omega_L V_L \\
V_L' &= \rho\pi_L - (\mu_L + \omega_L)V_L \\
E_L' &= (1-\theta_L)\lambda_L S_L - (\kappa_L + \mu_L)E_L \\
A'y_L &= (1-\alpha_L)\kappa_L E_L - (\mu_L + \delta_L + \overline{\sigma_L})Ay_L \\
S'y_L &= \theta_L\lambda_L S_L + \alpha_L\kappa_L E_L - (\mu_L + \delta_L + \sigma_L)Sy_L
\end{aligned}
\qquad (1)
$$

$$
R_L' = \sigma_L Sy_L + \overline{\sigma_L} Ay_L - \mu_L R_L
$$

Vector

$$
\begin{aligned}
S_V' &= \pi_V - \lambda_V S_V - \mu_V S_V \\
E_V' &= \lambda_V S_V - (\mu_V + \kappa_V)E_V
\end{aligned}
\qquad (2)
$$

$$
I_V' = \kappa_V E_V - \mu_V I_V
$$

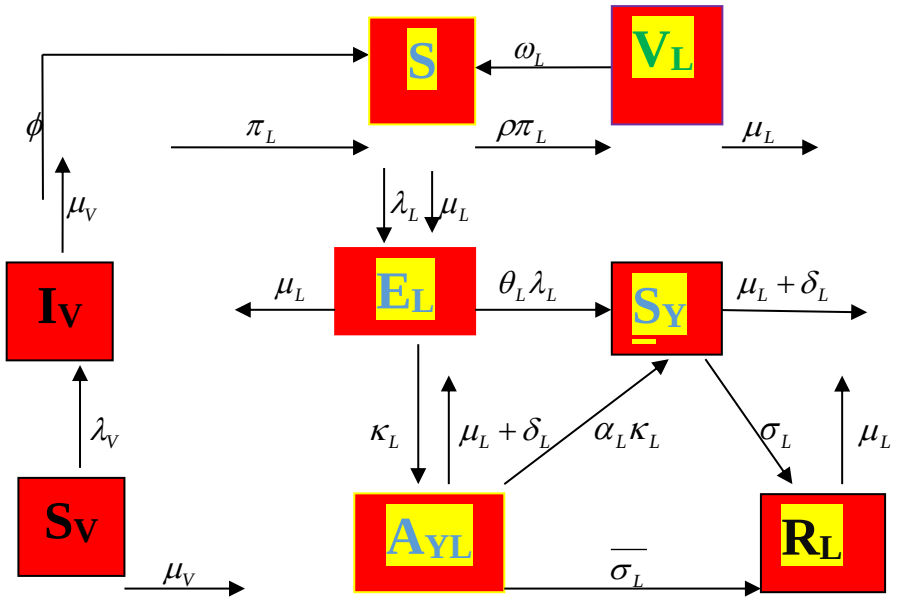
$$
\lambda_L = \frac{\beta_{VL}\phi I_V}{N_L} \quad \text{and} \quad \lambda_V^{**} = \frac{\beta_V\phi(E_L^{**} + Ay_L^{**} + Sy_L^{**})}{N_V}
$$

Table 1. Description of Variables

Variables	Description
S_L	Susceptible Livestock Individual
V_L	Vaccinated Livestock Individual
E_L	Exposed Livestock Individuals
Sy_L	Symptomatic Livestock Individual
Ay_L	Asymptomatic Livestock Individual
R_L	Recovered Livestock Individual
S_V	Susceptible Vector Individual
E_V	Exposed Vector Individuals
I_V	Infected Vector Individuals

Table 2. Description of Parameters

Parameters	Description
β_{VL}	Transmission of infection from an infectious vector to susceptible livestock.
β_{LV}	Transmission of infection from an infectious livestock to susceptible vector.
ϕ	Biting rate of vector
ρ	Vaccine rate
π	Recruitment rate
π_V	Recruitment of vector
λ_L	Force of infections of livestock
λ_V	Force of infections of vector
μ	Natural death
ω_L	Vaccine wanes
θ_L	Active infection
κ_L	Progression rate
α_L	Detection of asymptomatic
δ_L	Induced death from disease
σ_L	Recovery of Symptomatic individual
$\overline{\sigma_L}$	Recovery of Asymptomatic individual



Schematic Diagram of the Rift Valley Fever Model.

For better analysis, the following representation holds

$$S_L' = (1-\rho)\pi_L - \lambda_L S_L - \mu_L S_L + \omega_L V_L$$

$$V_L' = \rho\pi_L - K_1 V_L$$

$$E_L' = (1-\theta_L)\lambda_L S_L - K_2 E_L$$

$$A'y_L = (1-\alpha_L)\kappa_L E_L - K_3 Ay_L$$

$$S'y_L = \theta_L\lambda_L S_L + \alpha_L\kappa_L E_L - K_4 Sy_L \qquad (3)$$

$$R_L' = \sigma_L Sy_L + \overline{\sigma_L} Ay_L - \mu_L R_L$$

$$S_V' = \pi_V - \lambda_V S_V - \mu_V S_V$$

$$E_V' = \lambda_V S_V - K_5 E_V$$

$$I_V' = K_V E_V - \mu_V I_V$$

Where

$K_1 = (\mu_L + \omega_L)$

$K_2 = (\kappa_L + \mu_L)$

$K_3 = (\mu_L + \delta_L + \overline{\sigma_L})$

$K_4 = (\mu_L + \delta_L + \sigma_L)$

$K_5 = (\mu_V + \kappa_V)$

Analysis of the model

Lemma 1: The close set $D = D_L \times D_V \subset R_+^9$ is positive invariant for the model equation (3) with non-negative initial condition in R_+^9.

Proof: Consider the biologically-feasible region $D = D_L \times D_V \subset R_+^9$ with

$$D_L = \left\{ (S_L, V_L, E_L, Ay_L, Sy_L, R_L) \in R_+^6 : N_L \leq \frac{\pi_L}{\mu} \right\} \quad (4)$$

And

$$D_V = \left\{ (S_V, E_V, I_V) \in R_+^3 : N_V \leq \frac{\pi_V}{\mu} \right\} \quad (5)$$

We shall show that D is positive invariance (i.e all solutions in D remain in D for all time $t > 0$). Therefore:

$$\frac{dN_L}{dt} = \pi_L - \mu N_L - \delta_L (Ay_L + Sy_L)$$

and $\frac{dN_V}{dt} = \pi_V - \mu_V N_V$

Where $N_L = S_L + V_L + E_L + Ay_L + Sy_L + R_L$ and $N_V = S_V + E_V + I_V$

It follows that

$$\frac{dN_L}{dt} \leq \pi_L - \mu N_L \quad (6)$$

And

$$\frac{dN_V}{dt} \leq \pi_V - \mu_V N_V \quad (7)$$

A standard comparison theorem can be used to show that

$$N_L(t) \leq N_L(0)e^{-N_L t} + \frac{\pi_L}{\mu_L}(1-e^{-N_L t}) \quad \text{and} \quad N_V(t) \leq N_V(0)e^{-N_V t} + \frac{\pi_V}{\mu_V}(1-e^{-N_V t})$$

In particular $N_L(t) \leq \frac{\pi_L}{\mu_L}$ and $N_V(t) \leq \frac{\pi_V}{\mu_V}$

If $N_L(0) \leq \frac{\pi_L}{\mu_L}$ and $N_V(0) \leq \frac{\pi_V}{\mu_V}$. Therefore all the solutions of the model with initial condition remain there for $t > 0$. This implies that D is positive-invariant in this region and the model can be considered as been epidemiologically and mathematically well posed.

Disease Free Equilibrium

The model equation (3) has a disease free equilibrium which is derived by setting all the right hand sides of the equations in (3) to zero which is given by;

$$\varepsilon_0 = (S_L, V_L, E_L, Ay_L, Sy_L, R_L, S_V, E_V, I_V) = \left(\frac{(1-\rho)\pi_L + \omega_L \rho \pi_L}{K_1 \mu_L}, \frac{\rho \pi_L}{K_1}, 0,0,0,0, \frac{\pi_V}{\mu_V}, 0,0 \right)$$

Basic Reproduction Number

Using next generation matrix, the non negative matrix F (New infection terms) and non singular matrix V (other remaining transfer terms) (Adesanya et al., 2016b; Olopade et al., 2021a, 2021b, 2017, 2016 and 2022) of the model (1) are given respectively;

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & (1-\theta_L)\beta_{VL}\phi\mu_V \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \theta_L\beta_{VL}\phi\mu_V \\ 0 & 0 & 0 & 0 & 0 & 0 \\ \beta_{LV}\phi\mu_L & \beta_{LV}\phi\mu_L & \beta_{LV}\phi\mu_L & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

(8)

And

$$V = \begin{pmatrix} K_2 & 0 & 0 & 0 & 0 & 0 \\ -\kappa_L & K_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & K_4 & 0 & 0 & 0 \\ 0 & -\overline{\sigma_L} & -\sigma_L & \mu_L & 0 & 0 \\ 0 & 0 & 0 & 0 & K_5 & 0 \\ 0 & 0 & 0 & 0 & -\kappa_V & \mu_V \end{pmatrix}$$

(9)

$$R_{1,2} = \frac{\pm\sqrt{K_2K_3K_4K_5\kappa_V\mu_V\beta_{LV}\beta_{VL}(K_2K_3\theta_L - K_3K_4\theta_L - K_4\kappa_L\theta_L + K_3K_4 + K_4\kappa_L)\phi}}{K_2K_3K_4K_5}$$

R_0 is the maximum value of the two Eigen values $R_{1,2}$ hence, the associated reproduction number R_0 for $R-V$ fever model is given by $R_0 = \rho(FV^{-1})$, where ρ is the spectral radius of the dominant Eigen-value of the next generation matrix FV^{-1}. Thus

$$R_0 = \frac{\sqrt{K_2K_3K_4K_5\kappa_V\mu_V\beta_{LV}\beta_{VL}(K_2K_3\theta_L - K_3K_4\theta_L - K_4\kappa_L\theta_L + K_3K_4 + K_4\kappa_L)\phi}}{K_2K_3K_4K_5}$$

(10)

is the average number of infectious due to one infected vector introduced in the completely susceptible populations of livestock and vectors.

Local Stability of Disease Free Equilibrium

Theorem 1: The disease free equilibrium of the model equation (3) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: To determine the local stability of E_0, the Jacobian matrix below is computed corresponding to Disease Free Equilibrium E_0. Considering the stability of the disease free equilibrium at $\left(\frac{(1-\rho)\pi_L+\omega_L\rho\pi_L}{K_1\mu_L},\frac{\rho\pi_L}{K_1},0,0,0,0,\frac{\pi_V}{\mu_V},0,0\right)$.

$$J(E_0)=\begin{pmatrix} -\mu_L & \omega_L & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -K_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -K_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & (1-\alpha_L)\kappa_L & -K_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \alpha_L\kappa_L & -K_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \overline{\sigma_L} & \sigma_L & -\mu_L & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\mu_V & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -K_5 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \kappa_V & -\mu_V \end{pmatrix} \quad (11)$$

The characteristic equation of (11) above are obtained as $|JE_0-\lambda I|=0$, where I is the (9*9) identity matrix. Then, $|JE_0-\lambda I|=$

$$J(E_0)=\begin{pmatrix} -\mu_L-\lambda & \omega_L & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -K_1-\lambda & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -K_2-\lambda & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & (1-\alpha_L)\kappa_L & -K_3-\lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \alpha_L\kappa_L-K_4-\lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \overline{\sigma_L} & \sigma_L-\mu_L-\lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\mu_V-\lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -K_5-\lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \kappa_V-\mu_V-\lambda & 0 \end{pmatrix} \quad (12)$$

The eigen-values are $-\mu_L,-K_1,-K_2-K_3-K_4,-\mu_L,-\mu_V,-K_5-\mu_V$

Hence, the disease equilibrium point is locally asymptotically stable since all the eigen values are real and negative. This theorem implies that if the initial sizes of the sub-

populations of the model are in the basin of attraction of the disease-free equilibrium, the disease is controllable provided $R_o < 1$

Global Stability of Disease Free Equilibrium

Theorem 1: The disease free equilibrium of $R-V$ fever model given by (3) is global asymptotically stable if $R_0 < 1$.

Proof: The comparison theorem (Adewale et al., 2015a; 2015b; 2015c; 2016) shall be used to prove the global stability. The rate of change of variables representing the infected components of equation (3) can be written as;

$$\begin{pmatrix} E_L' \\ A'y_L \\ S'y_L \\ E_V' \\ I_V' \end{pmatrix} = (F-V)\begin{pmatrix} E_L \\ Ay_L \\ Sy_L \\ E_V \\ I_V \end{pmatrix} - \begin{pmatrix} (1-\theta_L)\lambda_L S_L \\ 0 \\ \theta_L \lambda_L S_L \\ \lambda_V S_V \\ 0 \end{pmatrix} \tag{13}$$

Then,

$$\begin{pmatrix} E_L' \\ A'y_L \\ S'y_L \\ E_V' \\ I_V' \end{pmatrix} = (F-V)\begin{pmatrix} -\kappa_L - \mu_L \\ (1-\alpha_L)\kappa_L - \mu_L - \delta_L - \overline{\sigma_L} \\ \alpha_L \kappa_L - \mu_L - \delta_L - \sigma_L \\ -\mu_V - \kappa_V \\ \kappa_V - \mu_V \end{pmatrix} \tag{14}$$

All the Eigen values of matrix $F-V$ have negative real parts. It follows that the linearilized differential inequality system above sis stable whenever $R_0 < 1$. Consequently, by comparison theorem, we have that $E_L = Sy_L = Ay_L = E_V = I_V = 0 \to (0,0,0,0,0)$ as $t \to \infty$. Hence, we have a positive invariant region. It follows that disease free equilibrium is globally asymptotically stable whenever $R_0 < 1$.

Sensitivity Analysis

Sensitivity analysis is a crucial analysis that shows the importance of each parameter to disease transmission. The sensitivity index of parameters with respect to the basic

reproduction number is calculated to known how crucial each parameter is to disease transmission.

Definition: The normalized forward sensitivity index of a variable ω that depends differentiably on a parameter P is defined as

$$X_P^{\omega} = \frac{\partial \omega}{\partial P} \times \frac{P}{\omega} \tag{15}$$

$$X_P^{R_0} = \frac{d\omega}{dP} \times \frac{P}{R_0} \tag{16}$$

The sensitivity index of R_{RVM} with respect to each parameter is calculated using the data in Table 4. The results are presented in Table 3.

Table 3. Numerical Sensitivity Index for RV-Malaria

Parameter	Sensitivity Value	Sign
β_{VL}	1.00000	+
β_{LV}	1.00000	+
ϕ	1.00000	+
ρ	-0.12115	-
μ_L	-0.51223	-
μ_V	-1.25000	-
ω_L	-0.00027	-
θ_L	0.05371	+
κ_L	0.12227	+
κ_V	0.24999	+
α_L	0.00755	+
σ_L	-0.22598	-
$\overline{\sigma_L}$	-0.04354	-

Existence of Endemic Equilibrium Point of $R-V$ Fever

Let the associated reproduction number of the model equation (1) – (9) above given by

$$R_0 = \frac{\sqrt{K_2K_3K_4K_5\kappa_V\mu_V\beta_{LV}\beta_{VL}(K_2K_3\theta_L - K_3K_4\theta_L - K_4\kappa_L\theta_L + K_3K_4 + K_4\kappa_L)\phi}}{K_2K_3K_4K_5}$$

$$S_L' = (1-\rho)\pi_L - \lambda_L S_L - \mu_L S_L + \omega_L V_L$$

(17)

$$V_L' = \rho\pi_L - K_1 V_L$$

(18)

$$E_L' = (1-\theta_L)\lambda_L S_L - K_2 E_L$$

(19)

$$A'y_L = (1-\alpha_L)\kappa_L E_L - K_3 Ay_L$$

(20)

$$S'y_L = \theta_L \lambda_L S_L + \alpha_L \kappa_L E_L - K_4 Sy_L$$

(21)

$$R_L' = \sigma_L Sy_L + \overline{\sigma_L} Ay_L - \mu_L R_L$$

(22)

$$S_V' = \pi_V - \lambda_V S_V - \mu_V S_V$$

(23)

$$E_V' = \lambda_V S_V - K_5 E_V$$

(24)

$$I_V' = K_V E_V - \mu_V I_V$$

(25)

Let $E_{RVF}^{**} = (S_L^{**}, V_L^{**}, E_L^{**}, Sy_L^{**}, Ay_L^{**}, R_L^{**}, S_V^{**}, E_V^{**}, I_V^{**})$ represent any arbitrary endemic equilibrium of model equation (17) to (25).

Let λ_V and λ_L at endemic steady state be denoted by λ_V^{**} and λ_L^{**} given by

$$\lambda_L^{**} = \frac{\beta_L \phi I_V^{**}}{N_V^{**}}$$

(26)

$$\lambda_V^{**} = \frac{\beta_V \phi (E_L^{**} + Ay_L^{**} + Sy_L^{**})}{N_V}$$

(27)

Where $N_L^{**} = S_L^{**} + V_L^{**} + E_L^{**} + Ay_L^{**} + Sy_L^{**} + R_L^{**}$ and $N_V^{**} = S_V^{**} + E_V^{**} + I_V^{**}$

Solving the equations of the model at steady state and re-writing the values of V_L, E_L, Ay_L, Sy_L in terms of $\lambda_L^{**} S_L^{**}$ and re-writing E_V, I_V in terms of $\lambda_v^{**} S_V^{**}$, gives

$$S_L^{**} = \frac{(1-\rho)\pi_L + \omega_L V_L^{**}}{\lambda_L^{**} + \mu_L}$$

(28)

$$V_L^{**} = \frac{\rho\pi_L}{K_1}$$

(29)

$$E_L^{**} = \frac{(1-\theta_L)\lambda_L^{**} S_L^{**}}{K_2}, \quad E_L^{**} = P_1 \lambda_L^{**} S_L^{**}$$

(30)

$$Sy_L^{**} = \frac{\theta_L \lambda_L^{**} S_L^{**} + K_L E_L^{**}}{K_3} = \frac{\theta_L \lambda_L^{**} S_L^{**} + \kappa_L \theta_L \lambda_L^{**} S_L^{**}}{K_2 K_3} = P_2 \lambda_L^{**} S_L^{**}$$

(31)

$$Ay_L^{**} = \frac{(1-\alpha_L) Sy_L^{**}}{K_4} = \frac{(1-\alpha_L)\theta_L \lambda_L^{**} S_L^{**} + \kappa_L \theta_L \lambda_L^{**} S_L^{**}}{K_2 K_3 K_4} = P_3 \lambda_L^{**} S_L^{**}$$

(32)

$$R_L^{**} = \frac{\sigma_L Sy_L^{**} + \overline{\sigma_L} Ay_L^{**}}{\mu_L} = \frac{(K_4\sigma_L + \alpha_L \overline{\sigma_L})\theta_L \lambda_L^{**} S_L^{**} + \kappa_L (1-\alpha_L)\lambda_L^{**} S_L^{**}}{\mu_L K_2 K_3 K_4} = P_4 \lambda_L^{**} S_L^{**}$$

(33)

$$S_V^{**} = \frac{\pi_V}{\lambda_V^{**} + \mu_V}$$

(34)

$$E_V^{**} = \frac{\lambda_V^{**} S_V^{**}}{K_5} = P_5 \lambda_V^{**} S_V^{**}$$

(35)

$$I_V^{**} = \frac{K_V E_V^{**}}{\mu_V} = \frac{K_V \lambda_V^{**} S_V^{**}}{\mu_V K_5} = P_6 \lambda_V^{**} S_V^{**}$$

(36)

Where $P_1 = \frac{\theta_L}{K_2}$, $P_2 = \frac{(1-\alpha_L)+\kappa_L\theta_L}{K_2 K_3}$

$$P_3 = \frac{(1-\alpha_L)\theta_L + K_L\theta_L}{K_2 K_3 K_4}$$

$$P_4 = \frac{(K_4\sigma_L + \alpha_L\overline{\sigma_L})((1-\alpha_L)+\kappa_L\theta_L)}{\mu_L K_2 K_3 K_4}$$

$$P_5 = \frac{1}{K_5}, P_6 = \frac{K_V}{\mu_V K_5}$$

Substituting (30)—(32) into (27)

$$\lambda_V^{**}(1+(P_1+P_2+P_3+P_4)\lambda_L^{**}) = \beta_V\phi(P_1+P_2+P_3)\lambda_L^{**}$$

Let $P_1 + P_2 + P_3 + P_4 = p_8$

$$\lambda_V^{**} = \frac{\beta_V\phi(P_1+P_2+P_3)\lambda_L^{**}}{1+P_8\lambda_L^{**}}$$

(37)

Substituting (36) into (26)

$$\lambda_L^{**}(1+(P_5+P_6)\lambda_V^{**}) = \beta_L\phi P_6\lambda_V^{**}$$

Let $P_5 + P_6 = P_7$

$$\lambda_L(1+P_7\lambda_V^{**}) = \beta_L\phi P_6\lambda_V^{**}$$

(37)

Putting (36) into the right hand of (37), gives;

$$(1+P_7\lambda_V^{**})\ (1+P_8\lambda_L^{**}) = \beta_L\beta_V\phi\gamma P_6(P_1+P_2+P_3)$$

$$(1+P_7\lambda_V^{**})\ (1+P_8\lambda_L^{**}) = R_0^2$$

Hence $1+P_7\lambda_V^{**} = R_0^2$ or $1+P_8\lambda_L^{**} = R_0^2$

So, $\lambda_V^{**} = \frac{R_0^2-1}{P_7} > 0,$ whenever $R_0 > 1$

Or

$$\lambda_L^{**} = \frac{R_0^2-1}{P_8} > 0,$$

(38)

Hence, endemic equilibrium exists whenever $R_0 > 1$.

Numerical Analysis

The numerical approximation and validation of the model's theoretical calculations are conducted through the implementation of a fourth-order Runge-Kutta numerical scheme. This numerical scheme is implemented using the MAPLE 18 program software. The model's calculations are validated using a predefined set of estimated parameter values, as detailed in Table 4.

Table 4. Parameters and Values

Parameter	Value	Source
β_{VL}	0.7	(Pépin et al., 2010 and Jupp et al., 2002)
β_{LV}	0.21	(Pépin et al., 2010 and Jupp et al., 2002)
ϕ	0.25	(41, Chitnis et al., 2013)
ρ	0.6	(Uwishema et al., 2022)
π_L	2000	Assumed
π_V	1000	Assumed
μ_L	0.05	(Gaff et al., 2011 and Marion et al., 2022)
μ_V	0.07	(Mehmood et al., 2021)
ω_L	0.2	Assumed
θ_L	0.3	(Adeyeye et al., 2011and Jupp et al., 2002)
κ_L	0.4	(Adeyeye et al., 2011and Jupp et al., 2002)
κ_V	0.07	(Pépin et al., 2010 and Chamchod et al., 2016)
α_L	0.3	Assumed
δ_L	0.10	(Adeyeye et al., 2011and Kasari et al., 2008)
$\overline{\sigma_L}$	0.25	(Adeyeye et al., 2011and Kasari et al., 2008)
σ_L	0.3	(Adeyeye et al., 2011 and Kasari et al., 2008)

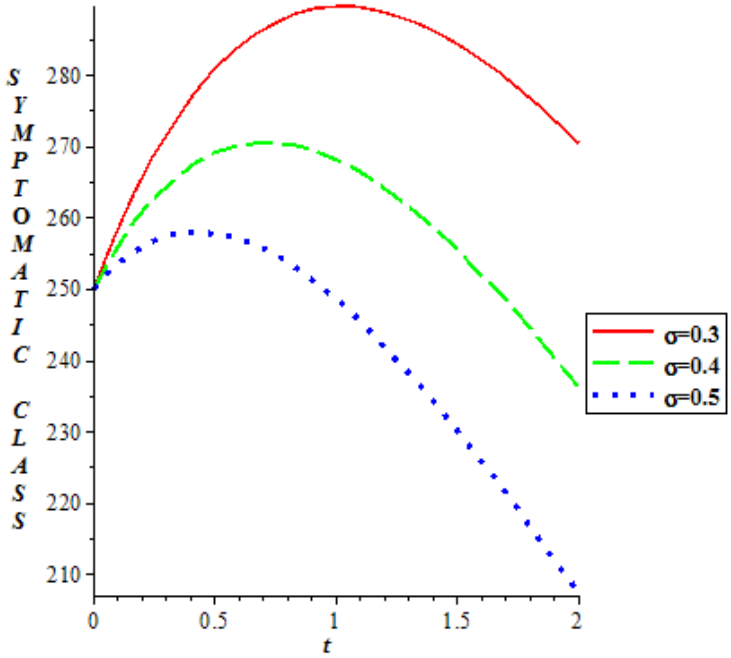


Fig. 1 : Graph of Symptomatic Class against time (t) *at different values of recovery rate* (σ)

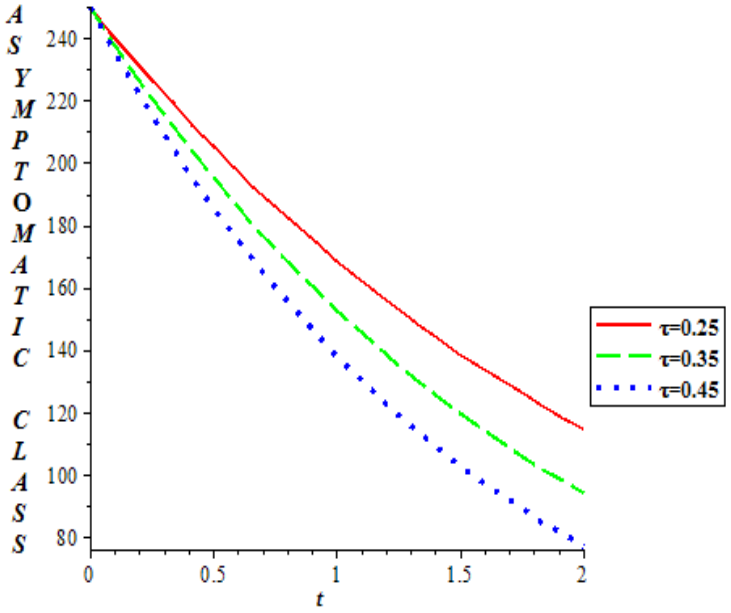


Fig. 2 : Graph of Asymptomatic Class against time (t) *at different values of recovery rate* (τ)

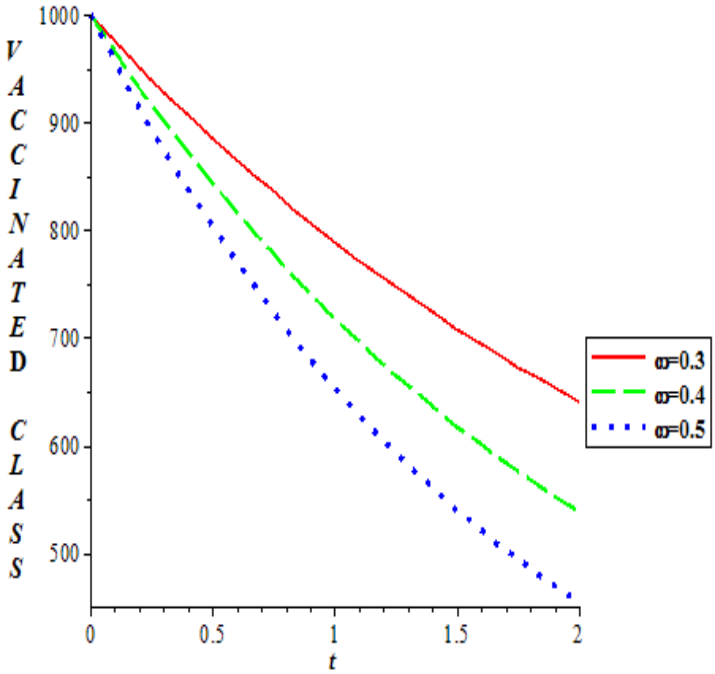


Fig. 3 : Graph of Vaccinated Class against time (t) **at different values of vaccine wanes** (ϖ)

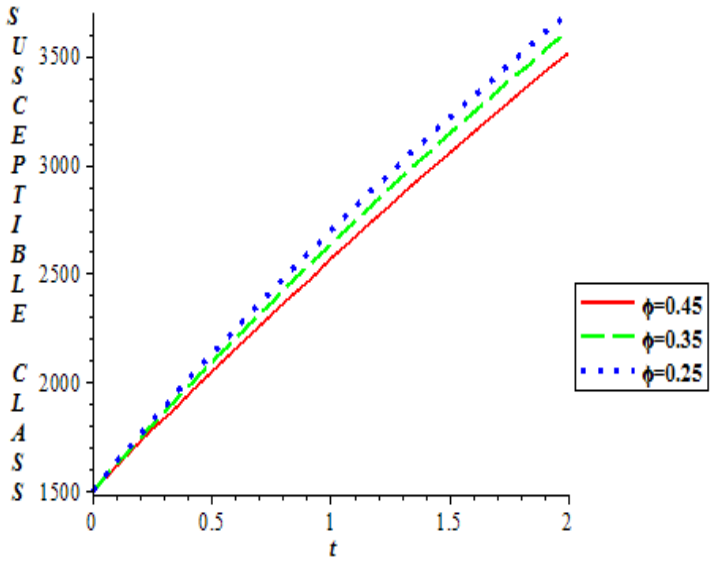


Fig. 4 : Graph of Susceptible Class against time (t) *at different values of mosquito bite* (ϕ)

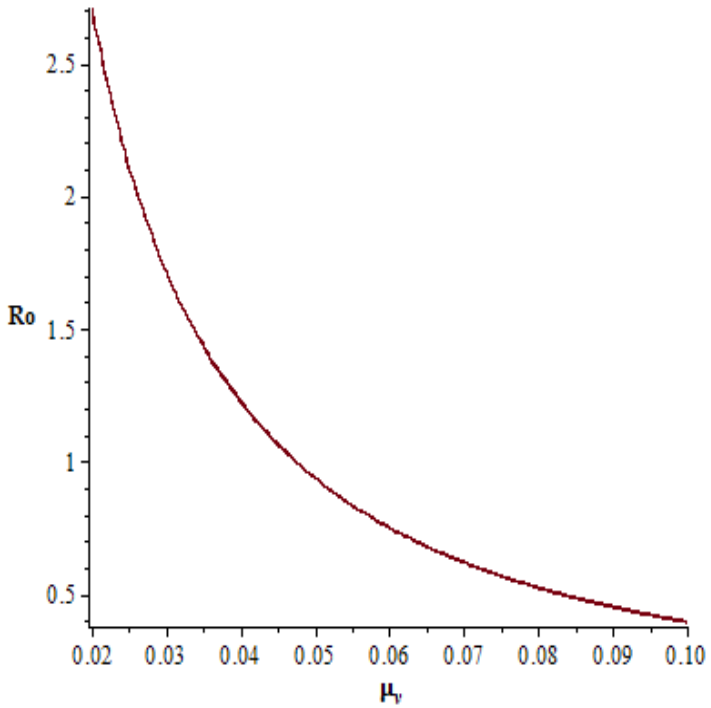


fig.5 : Graph of Basic Reproduction Number Ro against Mosquitoe death at time (t)

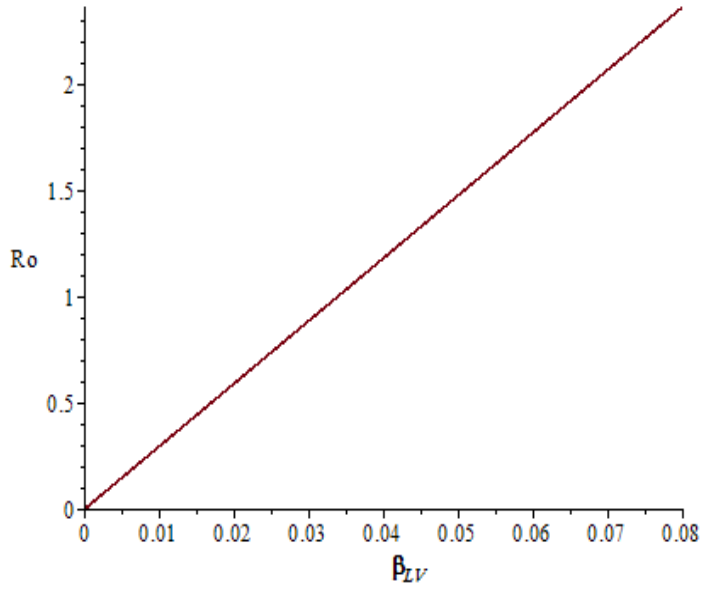


Fig.6 : Graph of Basic Reproduction Number Ro against Contact rate of Livestock to vector at time (t)

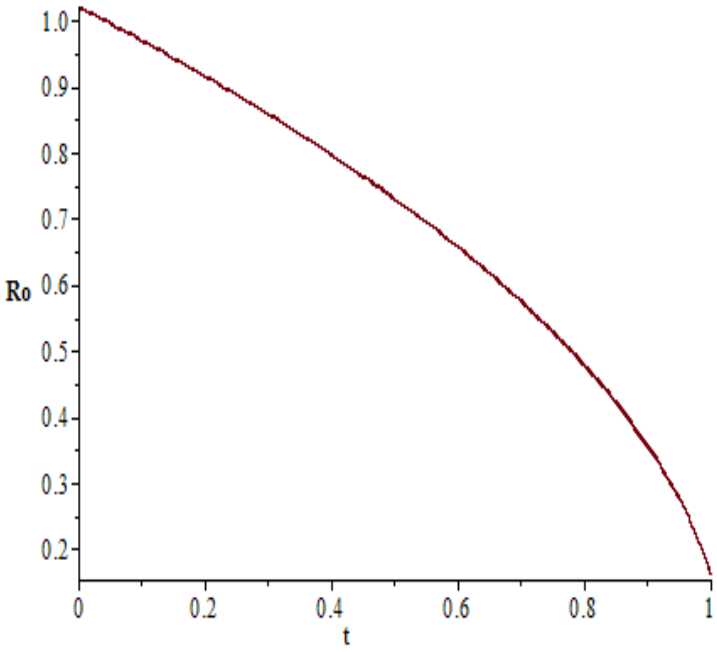


Fig.7 : Graph of Basic Reproduction Number Ro against Vaccine rate at time (t)

RESULTS AND DISCUSSION

Non linear differential deterministic model for rift valley fever in the presence of vaccine is presented and rigorously analyzed for gaining insight on the sensitive parameters that fuel the dynamical spread of the disease in the livestock. The positivity of solution shows that the model is mathematically and epidemiologically well posed. Analyzed the existence of disease free and endemic equilibrium points. Basic reproduction number R_0 which is the average number of new secondary infection generated by a single infected animal during its infectious period determines whether the fever dies out (i.e. when $R_0 < 1$) or spreads (i.e. when $R_0 > 1$). The global stability of endemic equilibrium was analyzed using comparison method.

Numerical simulation of the sensitivity analysis was carried out using MAPLE 18 software, in order to determine the parameters that influence the spread of RV-Fever among the livestock likewise to check how vaccine could be used as a control measure to forestall endemic situation.

It is observed from table 4 that parameters with positive index value will increase the threshold quantity basic reproduction number, which can lead to endemic situation whenever it grows above unity. The three (3) leading parameters to which basic

reproduction number (R_0) is most sensitive are; transmission of infection from infective vectors to susceptible livestock, transmission of infection from infective livestock to susceptible vectors and biting rate of vectors.

The numerical analysis presented in **Figure 1** highlights a significant decrease in the symptomatic class of livestock, and this reduction is attributed to the implementation of early treatment for infected animals. The graph underscores the positive impact of early treatment on enhancing the recovery rate, especially when the treatment is thorough (indicated by $\sigma = 1$). This observation suggests that comprehensive and timely treatment measures can effectively mitigate the symptomatic impact of the disease on the livestock population, contributing to improved recovery outcomes. **Figure 2** illustrates the favorable influence of an increased recovery rate on asymptomatic infected livestock. This positive effect may be attributed to the nutritional content of grass or substances present in their field diet, which could have medicinal properties beneficial to their overall health. The graph suggests that an enhanced recovery rate contributes to a higher proportion of asymptomatic infections among the livestock population, potentially indicating the role of natural elements in supporting their immune system and well-being. **Figure 3** in the results section illustrates the impact of vaccine waning on vaccinated livestock. The graph reveals a positive correlation between higher vaccine waning rates and an increase in both asymptomatic and symptomatic livestock. This highlights the importance of administering vaccines regularly to prevent the development of an endemic situation. The data suggests that maintaining a consistent vaccination schedule is crucial for effectively managing the health of the livestock population and minimizing the occurrence of symptomatic infections. **Figure 4** demonstrates that the biting of infected vectors on susceptible livestock leads to an escalation of the endemic situation within the system when effective intervention measures are lacking. This underscores the critical need for proactive and adequate interventions to mitigate the impact of vector-borne infections on susceptible livestock. The graph emphasizes the importance of implementing measures to control vector exposure and prevent the unchecked spread of infections, highlighting the potential consequences of insufficient intervention in maintaining livestock health. **Figure 5** presents a compelling visual representation of the beneficial impact that targeted mosquito mortality control measures can have on the basic reproduction number. The graph elucidates a clear inverse relationship between the death rate of mosquitoes and the

transmission of the fever in livestock. Notably, as the death rate of mosquitoes' increases, the transmission of the fever in livestock significantly decreases. This underscores the importance of implementing effective control measures targeting mosquito populations to curtail the spread of the disease among livestock. The graph vividly illustrates the potential of mosquito mortality control as a key strategy in mitigating the transmission dynamics of Rift Valley fever within the livestock population. **Figure 6** provides a comprehensive insight into the sensitivity of the transmission rate concerning the basic reproduction number. The graph illustrates a direct correlation between the interaction of infected livestock with susceptible individuals and the level of endemic within the population. Specifically, as the transmission rate increases, indicating heightened interaction between infected and susceptible livestock, there is a corresponding increase in the endemic of the disease. This visual representation underscores the critical role that the transmission rate plays in influencing the basic reproduction number, emphasizing the need for effective measures to reduce interactions between infected and susceptible livestock. **Figure 7** underscores the significance of vaccination as a crucial preventive strategy for veterinary practitioners to curtail the dynamic spread of Rift Valley fever in livestock. The graph vividly illustrates that the introduction of a vaccination strategy contributes to a substantial reduction in the basic reproduction number, bringing it down to a bearable minimum. This visual representation emphasizes the pivotal role of vaccination in mitigating the transmission dynamics of Rift Valley fever, thereby supporting the overall efforts to control and prevent the disease in livestock populations

CONCLUSION

Rigorous vaccination protocols for susceptible livestock are imperative to establish a robust defense against the spread of infectious diseases, particularly considering the potential impact of diseases like Rift Valley fever on livestock populations.

The implementation of regular and scheduled vaccinations is a cornerstone in the toolkit of veterinary practitioners. Timely and periodic vaccination campaigns are crucial to maintain the overall health and well-being of livestock, preventing the onset and transmission of diseases.

The strategic separation of infected livestock from susceptible individuals is a fundamental measure to curtail the propagation of diseases. This involves the identification, isolation,

and appropriate management of infected animals to prevent further transmission within the livestock population.

Veterinary practitioners should adopt a multifaceted approach to intervention strategies, considering a range of measures to effectively combat Rift Valley fever. This may include environmental management, vector control, and prompt response mechanisms to address emerging threats.

Early and accurate detection of asymptomatic livestock is pivotal in disease surveillance. Implementing efficient monitoring systems and diagnostic tools enables the identification of carriers and potential sources of infection, allowing for timely intervention and containment efforts.

In summary, a comprehensive and proactive approach that encompasses vaccination, strategic management practices, and early detection mechanisms is essential for mitigating the impact of Rift Valley fever on livestock populations. Veterinary practitioners must play a critical role in implementing and advocating for these strategies to safeguard the health and sustainability of livestock farming.

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