MATHEMATICAL ANALYSIS OF INTEGRATED CONTROL MEASURES MITIGATING RIFT VALLEY FEVER SPREAD IN LIVESTOCK

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Abstract. This study explores the complex transmission dynamics of Rift Valley fever (RVF), focusing on the interactions between mosquitoes and livestock through the application of a compartmental model. The research incorporates the implementation of isolation for infected individuals as a key control measure. By calculating the basic reproduction number R_0 using the next-generation matrix method, the study illuminates the conditions for achieving a disease-free equilibrium state. It is found that the disease-free equilibrium is locally asymptotically stable when ($R_0<1$) indicating that RVF can potentially be controlled within a livestock population if R_0 is kept below this critical threshold. Conversely, when ($R_0>1$) the disease may become endemic, emphasizing the crucial need to monitor and maintain R_0 at levels below 1. Additionally, the study performs a sensitivity analysis to identify key parameters that are essential for livestock policymakers and veterinary professionals to consider. Through numerical simulations, the research evaluates the effectiveness of early detection and isolation of infected livestock, in conjunction with other integrated control strategies. These simulations provide valuable insights into the dynamic behavior of RVF, aiding in the formulation of effective strategies for the management and prevention of the disease.
Keywords: *detection, reproduction number, equilibrium points, isolation, Rift-Valley fever, sensitivity*

Introduction

Rift Valley fever (RVF) is a viral zoonotic disease with considerable impacts on public health, livestock production, and economic stability, primarily in Africa and the Arabian Peninsula. The causative agent, the Rift Valley fever virus (RVFV), belongs to the *Phlebovirus* genus within the *Bunyaviridae* family (Adeyeye et al., 2011; Mpeshe et al., 2011; Bird et al., 2009). Transmission of RVFV primarily occurs through the bites of infected mosquitoes, affecting both animals and humans. Domesticated livestock, such as cattle, sheep, goats, and camels, are particularly vulnerable, although humans

can also contract the virus. Infected mosquitoes, notably those from the *Aedes* and *Culex* genera, serve as the primary vectors. Furthermore, the virus can spread through direct contact with tissues or bodily fluids of infected animals, increasing the risk for individuals working closely with livestock (Bird and McElroy, 2016). The first case of RVF was recorded in Kenya in 1931, and since then, the disease has been reported in various parts of Africa and other regions. Notable outbreaks occurred in Madagascar in 1979, the Arabian Peninsula in 2000, and the Comoros archipelago in 2007 (Sissoko et al., 2009; Nanyingi et al., 1931). The introduction of RVFV to Egypt in 1977, through the trade of infected animals, and a significant outbreak in Kenya, Somalia, and Tanzania during 1997-1998; exacerbated by El Niño-related flooding-highlighted the global importance of RVF (Kanouté et al., 2017). In 2000, RVF spread beyond Africa for the first time, reaching Saudi Arabia and Yemen, again via the trade of infected livestock from the Horn of Africa. This raised serious concerns about the potential spread of the virus to other regions, including Asia and Europe.

Given the broad impact of RVF on both human and animal health, it is critical to develop comprehensive strategies for surveillance, control, and prevention to mitigate its negative consequences on public health and the economy (WHO, 2018). In animals, RVF can lead to severe clinical manifestations, including fever, high mortality rates in newborns, abortion storms in pregnant livestock, and hepatitis (Oguntolu et al., 2022). In humans, the disease typically presents as a febrile illness, with symptoms such as fever, headache, muscle pain, joint pain, photophobia, and gastrointestinal distress. Severe cases may result in complications like hemorrhagic fever, encephalitis, retinitis, or even death. Populations at heightened risk, including farmers, herders, veterinarians, and slaughterhouse workers, face increased exposure due to their close contact with infected animals. Numerous researchers have leveraged mathematical models as essential tools for studying the epidemiology of diseases across various populations (Adesola et al., 2024a, 2024b; Ajao et al., 2023; Fawzy and Helmy, 2022; Musibau et al., 2022; Akinwumi et al., 2021; Métras et al., 2017; Adesanya et al., 2016a, 2016b; Adewale et al., 2016; 2015a; 2015b; 2015c; Gachohi et al., 2016; Pedro et al., 2016; 2014; Fischer et al., 2013; Bird et al., 2009). Previous research has extensively examined the transmission dynamics and contagious characteristics of Rift Valley fever (RVF) in both livestock and human populations.

In a study, an eco-epidemiological compartmental mathematical model was developed, incorporating ambient temperature and water availability, with empirical environmental data from Kenya to mirror real-world conditions accurately. This model effectively captures the intermittent nature of RVF occurrences, shedding light on the low-level virus circulation that often goes undetected, with occasional reemergence after prolonged periods. The study provides valuable insights into the complex dynamics of RVF in relation to environmental factors, underscoring the sporadic nature of its outbreaks. Similarly, EFSA AHAW Panel et al. (2021) conducted a study to evaluate the effectiveness of surveillance and control strategies for RVF in both Mayotte and the continental European Union (EU), utilizing mathematical models as a part of the assessment. This research aimed to assess the efficiency of various approaches in preventing and controlling RVF outbreaks within these regions. The present study aims to develop a comprehensive model that incorporates the effectiveness of isolating infected and infectious livestock, with the goal of enhancing our understanding of how this isolation strategy influences disease dynamics within

animal populations. Our objective is to explore and quantify the impact of isolating infected and infectious animals on reducing the spread of the disease among livestock.

Model formulation

In developing our model, we assume that livestock primarily contract the infection through contact with infectious mosquitoes. Additionally, a uniform natural death rate, denoted by δ, is applied across all compartments (*Table 1* and *Table 2*). Our model includes two main populations: livestock and mosquitoes. The livestock population is divided into six compartments: S_L=susceptible, V_L=vaccinated, E_L=exposed, SY_L=symptomatic, AY_L=asymptomatic, I_L=isolated, R_L=recovered. Each compartment represents a different stage of infection or immunity within the livestock population. The mosquito (vector) population is subdivided into two compartments: S_V=susceptible, I_V=infected, infected, capturing the different states mosquitoes can occupy regarding their susceptibility to and infection with the virus. This detailed compartmentalization allows us to accurately model the complex dynamics of disease transmission between livestock and mosquitoes, providing a more comprehensive understanding of Rift Valley fever's spread and informing effective control strategies.

Livestock

$$\frac{dS_L}{dt} = \pi_L - \lambda_L S_L - \mu_L S_L + \omega_L R_L$$

$$\frac{dE_L}{dt} = (1-\theta_L)\lambda_L S_L - (\kappa_L + \mu_L)E_L$$

$$\frac{dAy_L}{dt} = (1-\alpha_L)\kappa_L E_L - (\mu_L + \delta_L + \overline{\sigma_L})Ay_L$$

$$\frac{dSy_L}{dt} = \theta_L \lambda_L S_L + \alpha_L \kappa_L E_L - (\mu_L + \delta_L + \sigma_L + \gamma_L)Sy_L \qquad \text{Eq. (1)}$$

$$\frac{dI_L}{dt} = \gamma_L Sy_L - (\mu_L + \delta_L + \Lambda_L)I_L$$

$$\frac{dR_L}{dt} = \sigma_L Sy_L + \overline{\sigma_L} Ay_L + \Lambda_L I_L - (\mu_L + \omega_L)R_L$$

Vector

$$\frac{dS_V}{dt} = \pi_V - \lambda_V S_V - \mu_V S_V \qquad \text{Eq. (2)}$$

$$\frac{dI_V}{dt} = \lambda_V S_V - \mu_V I_V$$

Where;

$$\lambda_L = \frac{\beta_{VL}\phi I_V}{N_L}$$

$$\lambda_V = \frac{\beta_{LV}\phi(E_L + Ay_L + Sy_L)}{N_V}$$

Table 1. *Description of variables.*

Variables	Decription
S_L	Susceptible Livestock Individual
E_L	Exposed Livestock Individuals
Sy_L	Symptomatic Livestock Individual
Ay_L	Asymptomatic Livestock Individual
I_L	Isolated Livestock Individual
R_L	Recovered Livestock Individual
S_V	Susceptible Vector Individual
I_V	Infected Vector Individuals

Table 2. *Description of parameters.*

Parameters	Description
β_{VL}	Probability of transmission from an infectious vector to susceptible livestock
β_{LV}	Probability of transmission from an infectious livestock to susceptible vector
ϕ	Biting rate of vector
π_L	Recruitment rate of Livestock
π_V	Recruitment of vector
λ_L	Force of infections of livestock
λ_V	Force of infections of vector
μ	Natural death
ω_L	Loss of Immunity
θ_L	Active infection
κ_L	Progression rate
α_L	Detection rate of asymptomatic
δ_L	Induced death from disease
γ_L	Isolation of Symptomatic individual
σ_L	Recovery of Symptomatic individual
$\overline{\sigma_L}$	Recovery of Asymptomatic individual
Λ	Recovery of Isolated individual

For a more thorough analysis, the following representation is used (*Figure 1*):

$$\frac{dS_L}{dt} = \pi_L - \lambda_L S_L - \mu_L S_L + \omega_L R_L$$

$$\frac{dE_L}{dt} E_L' = (1-\theta_L)\lambda_L S_L - K_1 E_L$$

$$\frac{dAy_L}{dt} = (1-\alpha_L)\kappa_L E_L - K_2 Ay_L$$

$$\frac{dSy_L}{dt} = \theta_L \lambda_L S_L + \alpha_L \kappa_L E_L - K_3 Sy_L$$

$$\frac{dI_L}{dt} = \gamma_L Sy_L - K_4 I_L$$

$$\frac{dR_L}{dt} = \sigma_L Sy_L + \overline{\sigma_L} Ay_L + \Lambda_L I_L - K_5 R_L \qquad \text{Eq. (3)}$$

$$\frac{dS_V}{dt} = \pi_V - \lambda_V S_V - \mu_V S_V$$

$$\frac{dI_V}{dt} = \lambda_V S_V - \mu_V I_V$$

Where;

$K_1 = (\kappa_L + \mu_L)$

$K_2 = (\mu_L + \delta_L + \overline{\sigma_L})$

$K_3 = (\mu_L + \delta_L + \sigma_L + \gamma_L)$

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

$K_5 = (\mu_L + \omega_L)$

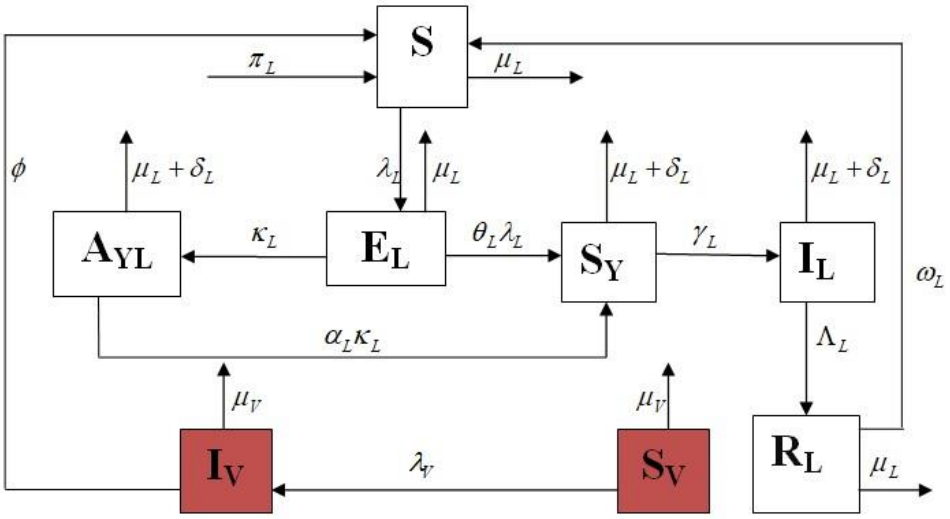

Figure 1. *Schematic Diagram of the RVF Model.*

Analysis of the model

The close set $D = D_L \times D_V \subset R_+^8$ is positive invariant for the model Eq. (3) with non-negative initial condition in R_+^8. Consider the biologically-feasible region $D = D_L \times D_V \subset R_+^8$ with:

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

$$D_V = \left\{ \left(S_V, I_V \right) \in R_+^2 : N_V \le \frac{\pi_V}{\mu} \right\} \quad \text{Eq. (5)}$$

We will demonstrate that *D* is positive invariant (i.e all solutions within *D* remain within *D* for all time *t>0*). Therefore:

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

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

Where

$$N_L = S_L + E_L + Ay_L + Sy_L + I_L + R_L$$

$$N_V = S_V + I_V$$

It follows that:

$$\frac{dN_L}{dt} \le \pi_L - \mu N_L \quad \text{Eq. (6)}$$

$$\frac{dN_V}{dt} \le \pi_V - \mu_V N_V \quad \text{Eq. (7)}$$

A standard comparison theorem can be utilized to demonstrate that:

$$N_L(t) \le N_L(0) e^{-N_L t} + \frac{\pi_L}{\mu_L} (1 - e^{-N_L t})$$

$$N_V(t) \le N_V(0) e^{-N_V t} + \frac{\pi_V}{\mu_V} (1 - e^{-N_V t})$$

In particular,

$$N_L(t) \leq \frac{\pi_L}{\mu_L}$$

$$N_V(t) \leq \frac{\pi_V}{\mu_V}$$

If $N_L(0) \leq \frac{\pi_L}{\mu_L}$ and $N_V(0) \leq \frac{\pi_V}{\mu_V}$

Hence, all solutions of the model with the initial condition persist within this region for $t>0$. This suggests that the region D is positive-invariant, and thus, the model can be deemed epidemiologically and mathematically well-posed.

Disease free equilibrium

The model Eq. (3) has a disease free equilibrium which is derived by setting all the right hand sides of the Eq. (3) to zero which is given by:

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

Existence of endemic equilibrium point of R-V fever

The endemic equilibrium is given point is given below:

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

$$E_L^{**} = \frac{(1-\theta_L)\lambda_L^{**} S_L^{**}}{K_1}$$

$$A_L^{**} y_L = \frac{(1-\alpha_L)\kappa_L E_L^{**}}{K_2}$$

$$S^{**} y_L = \frac{\theta_L \lambda_L^{**} S_L^{**} + \alpha_L \kappa_L E_L^{**}}{K_3}$$

$$I_L^{**} = \frac{\gamma_L Sy_L^{**}}{K_4}$$

$$R_L^{**} = \frac{\sigma_L S^{**} y_L + \overline{\sigma_L} A^{**} y_L + \Lambda_L I_L^{**}}{K_5}$$

$$S_v^{**} = \frac{\pi_V}{\lambda_v^{**} + \mu_v^{**}}$$

$$I_V^{**} = \frac{\lambda_v^{**} S_v^{**}}{\mu_V}$$

Thus, the endemic equilibrium arises whenever the basic reproduction number R_0>1.

Basic reproduction number

Using next generation matrix method (Olopade et al., 2022; 2021a; 2021b; 2017; 2016). The non-negative matrix F (representing new infection terms) and the non-singular matrix V (depicting other remaining transfer terms) of the model Eq. (3) are given respectively:

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & (1-\theta_L)\beta_{VL}\phi\mu_V \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \theta_L\beta_{VL}\phi\mu_V \\ 0 & 0 & 0 & 0 & 0 \\ \beta_{LV}\phi\mu_L & \beta_{LV}\phi\mu_L & \beta_{LV}\phi\mu_L & 0 & 0 \end{pmatrix} \quad \text{Eq. (8)}$$

$$V = \begin{pmatrix} K_1 & 0 & 0 & 0 & 0 \\ -(1-\alpha_L)\kappa_L & K_2 & 0 & 0 & 0 \\ -\alpha_L\kappa_L & 0 & K_3 & 0 & 0 \\ 0 & 0 & -\gamma_L & K_4 & 0 \\ 0 & 0 & 0 & 0 & \mu_V \end{pmatrix} \quad \text{Eq. (9)}$$

$$R_{1,2} = \frac{\pm\sqrt{\begin{matrix}K_1K_2K_3\mu_L\beta_{LV}\beta_{VL}(-K_2\alpha_L\kappa_L\theta_L + K_3\alpha_L\kappa_L\theta_L + K_1K_2\theta_L - K_2K_3\theta_L \\ + K_2\alpha_L\kappa_L - K_3\alpha_L\kappa_L - K_3\theta_L\kappa_L + K_2K_3 + K_3\kappa_L)\phi\end{matrix}}}{K_1K_2K_3}$$

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

$$R_0 = \frac{\sqrt{\begin{matrix}K_1K_2K_3\mu_L\beta_{LV}\beta_{VL}(-K_2\alpha_L\kappa_L\theta_L + K_3\alpha_L\kappa_L\theta_L + K_1K_2\theta_L - K_2K_3\theta_L \\ + K_2\alpha_L\kappa_L - K_3\alpha_L\kappa_L - K_3\theta_L\kappa_L + K_2K_3 + K_3\kappa_L)\phi\end{matrix}}}{K_1K_2K_3} \quad \text{Eq. (10)}$$

Is the average number of infections caused by one infected vector introduced into the completely susceptible populations of both livestock and vectors.

Local stability of disease free equilibrim

Theorem 1: The disease free equilibrium of the model Eq. (3) is locally asymptotically stable if $R_0<1$ and unstable if $R_0>1$. To ascertain the local stability of ε_0, we compute the Jacobian matrix corresponding to the Disease-Free Equilibrium. Evaluating the stability of the disease-free equilibrium at:

$$\varepsilon_0 = \left(\frac{(1-\rho)\pi_L}{\mu_L}, 0, 0, 0, 0, 0, \frac{\pi_V}{\mu_V}, 0 \right)$$

$$J(\varepsilon_0) = \begin{pmatrix} -\mu_L & 0 & 0 & 0 & 0 & \omega_L & 0 & 0 \\ 0 & -K_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & (1-\alpha_L)\kappa_L & -K_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha_L\kappa_L & 0 & -K_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma_L & -K_4 & 0 & 0 & 0 \\ 0 & 0 & \overline{\sigma_L} & \sigma_L & \Lambda_L & -K_5 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\mu_V & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_V \end{pmatrix} \quad \text{Eq. (11)}$$

The characteristic Eq. (11) above are obtained as $|J\varepsilon_0 - \lambda I| = 0$, where I is the (8*8) identity matrix. Then, $|J\varepsilon_0 - \lambda I| =$

$$J(\varepsilon_0 - \lambda I) = \begin{pmatrix} -\mu_L - \lambda & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -K_1 - \lambda & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & (1-\alpha_L)\kappa_L & -K_2 - \lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha_L\kappa_L & 0 & -K_3 - \lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma_L & -K_4 - \lambda & 0 & 0 & 0 \\ 0 & 0 & \overline{\sigma_L} & \sigma_L & \Lambda_L & -K_5 - \lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\mu_V - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_V - \lambda \end{pmatrix} \quad \text{Eq. (12)}$$

The Eigen-values are: $-K_1$, $-K_2$, $-K_3$, $-K_4$, $-K_5$, $-\mu_L$ and $-\mu_V$ twice. The disease equilibrium point is locally asymptotically stable, as evidenced by all eigenvalues being real and negative. This stability theorem suggests that if the initial sizes of the model's sub-populations fall within the basin of attraction of the disease-free equilibrium, the disease can be controlled effectively provided $R_0<1$.

Global stability of disease free equilibrium

We examine the global stability of the disease-free equilibrium using the methodology outlined by Philemon et al. (2023). Consequently, the model equations can be reformulated as follows:

$$\frac{dM}{dt} = F(M, I)$$
$$\frac{dI}{dt} = G(M, I)$$

With G(P,0)=0, where $P \in \Re^3$ represents the uninfected classes (S_L, R_L, S_V) and $I \in \Re^5$ represents the infected classes (E_L, AY_L, SY_L, I_L, I_V). Also $\varepsilon_o = (M^*, 0)$ denotes the disease-free equilibrium of the model. The two conditions (H1) and (H2) stated below must be satisfied for the model to be globally stable:

(H1): For $\frac{dM}{dt} = F(M, 0)$, M^* is globally asymptotically stable

(H2): $G(M, I) = AI - \hat{G}(M, I)$, $\quad \hat{G}(M, I) \geq 0$ for $(M, I) \in D$

Where $A = D_I G(M^*, 0)$ is an M-matrix (the off-diagonal elements of A are non-negative) and D is the region is the feasible region where the model is biologically meaningful. If (H1) and (H2) are satisfied, then the following theorem holds.

Theorem 2: The disease-free equilibrium $\varepsilon_o = (M^*, 0)$ is a globally asymptotically stable equilibrium of the model if and that the conditions (H1) and (H2) are satisfied. Now $M=(S_L, R_L, S_V)$ and $I=(E_L, AY_L, SY_L, I_L, I_V)$.

$$F(M, 0) = \begin{pmatrix} (1-\rho)\pi_L - \mu_L S_L \\ 0 \\ 0 \end{pmatrix} \qquad \text{Eq. (13)}$$

$$AI = \begin{pmatrix} -K_1 & 0 & 0 & 0 & (1-\theta_L)\beta_{VL}\phi \\ (1-\alpha_L)\kappa_L & -K_2 & 0 & 0 & 0 \\ \alpha_L\kappa_L & 0 & -K_3 & 0 & \theta_L\beta_{VL}\phi \\ 0 & 0 & \gamma_L & -K_4 & 0 \\ \beta_{LV}\phi & \beta_{LV}\phi & \beta_{LV}\phi & 0 & -\mu_V \end{pmatrix} \begin{pmatrix} E_L \\ Ay_L \\ Sy_L \\ I_L \\ I_V \end{pmatrix} \qquad \text{Eq. (14)}$$

$$\hat{G}(M, I) = \begin{pmatrix} (1-\theta_L)\beta_{VL}\phi\left(1 - \frac{S_L}{N_L}\right) \\ 0 \\ \theta_L\beta_{VL}\phi\left(1 - \frac{S_L}{N_L}\right) \\ 0 \\ \beta_{LV}\phi\left(1 - \frac{S_V}{N_V}\right) \end{pmatrix} \qquad \text{Eq. (15)}$$

Since $0 \le \varepsilon \le 1$, clearly $\hat{G}(M, I) \ge 0$ $\varepsilon_o = \left(\frac{(1-\rho)\pi_L}{\mu_L}, 0, 0 \right)$ then, the model equations exhibit a globally asymptotically stable disease-free equilibrium. Meeting both specified conditions confirms this stability, implying that the disease-free state is globally asymptotically stable. This biological interpretation indicates that Rift Valley fever can be eradicated regardless of the initial sizes of the sub-populations, provided the basic reproduction number is below one.

Sensitivity analysis

Sensitivity analysis is crucial for understanding the impact of each parameter on disease transmission dynamics. By calculating the sensitivity index of parameters relative to the basic reproduction number, we can assess the extent to which each parameter affects disease transmission. This analysis helps identify the most influential parameters on the basic reproduction number, highlighting their role in driving the spread of the disease. By prioritizing interventions and targeting control measures at these critical parameters, we can enhance our strategies for disease control and prevention. This thorough evaluation deepens our understanding of the mechanisms driving transmission dynamics and aids in developing more effective disease management strategies. The normalized forward sensitivity index of a variable $\boldsymbol{\omega}$ that depends differentiably on a parameter P is defined as:

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

Results and Discussion

Numerical analysis

The theoretical calculations of the model are numerically approximated and validated using a fourth-order Runge-Kutta numerical method, implemented through MAPLE 18 software. Validation is carried out by applying a set of predefined estimated parameter values, as outlined in *Table 3* and *Table 4*.

Table 3*. Parameters and values.*

Parmeter	Value	Source
β_{VL}	0.7	Pepin et al. (2010); Jupp et al. (2002)
β_{LV}	0.21	Pepin et al. (2010); Turell et al. (1985)
ϕ	0.25	Chitnis et al. (2013); Ba et al. (2005)
μ_L	0.05	Mehmood et al. (2021); Gaff et al. (2011)
μ_V	0.07	Mehmood et al. (2021); Kasari (2008)
ω_L	0.2	Assumed
Λ	0.2	Assumed
γ_L	0.3	Assumed
θ_L	0.3	Adeyeye et al. (2011); Jupp et al. (2002)
κ_L	0.4	Adeyeye et al. (2011); Jupp et al. (2002)

κ_V	0.07	Chamchod et al. (2016); Turell et al. (1985)
α_L	0.3	Assumed
δ_L	0.10	Adeyeye et al. (2011); Kasari (2008)
$\overline{\sigma_L}$	0.5	Adeyeye et al. (2011); Kasari (2008)
σ_L	0.3	Adeyeye et al. (2011); Kasari (2008)

Table 4. *Numerical Sensitivity Index for RVF.*

Category	Sensitivity value	Sensitivity sign
β_{VL}	1.00000	+
β_{LV}	1.00000	+
ϕ	1.00000	+
θ_L	0.05371	+
κ_L	0.12227	+
α_L	0.00755	+
ρ	-0.12115	-
μ_L	-0.51223	-
δ_L	-0.30122	-
ω_L	-0.00027	-
γ_L	-0.25999	-
σ_L	-0.22598	-
$\overline{\sigma_L}$	-0.04354	-

A non-linear deterministic model for Rift Valley fever, incorporating the detection and isolation of infected livestock from susceptible individuals, is presented and thoroughly examined. This study seeks to highlight the critical role of early detection and isolation in reducing the spread of Rift Valley fever among livestock populations. The model's mathematical and epidemiological validity is demonstrated through the positivity of its solutions, indicating that it is well-posed in both fields. The analysis explores the existence of both disease-free and endemic equilibrium states, with the basic reproduction number serving as a key indicator of disease dynamics. Depending on its value, the model predicts whether the disease will eventually die out or persist within the population (i.e. when $R_0<1$) or spreads (i.e. when $R_0>1$), based on the value of the basic reproduction number. The stability of the disease equilibrium is examined through both local and global stability analyses, confirming its stability. Additionally, numerical simulations of sensitivity analysis, conducted using MAPLE 18 software are performed to identify the key parameters influencing the spread of Rift Valley fever among livestock. The results indicate that parameters with negative indices help reduce the spread of the disease, while those with positive indices increase the basic reproduction number, thereby amplifying transmission.

An analysis of *Table 3* shows that parameters with positive index values elevate the basic reproduction number, potentially leading to an endemic situation when it exceeds one. Three parameters emerge as the most influential in driving the basic reproduction number: the transmission of infection from infectious vectors to susceptible livestock, the transmission from infectious livestock to susceptible vectors, and the biting rate of

vectors. These findings emphasize the crucial role these factors play in the transmission dynamics of Rift Valley fever within livestock populations. *Figure 2* highlights the importance of isolating symptomatic infected individuals, a key epidemiological measure for limiting the spread of infectious diseases. This strategy involves separating symptomatic individuals from the general population to prevent further transmission. Its success depends on timely identification, availability of isolation facilities, compliance with protocols, and the disease's contagiousness. Isolation plays a critical role in controlling outbreaks and safeguarding public health. *Figure 3* shows the reintegration rate of individuals who were isolated due to infection and have since recovered, returning to the general population. This factor is critical in epidemiology as it helps to understand disease transmission dynamics and assess the effectiveness of control measures. Monitoring this rate provides valuable insights into the efficacy of isolation protocols, the duration of post-recovery immunity, and the overall impact on managing infectious disease outbreaks. *Figure 4* highlights the detection of the asymptomatic livestock population, emphasizing the identification and diagnosis of livestock infected with the disease but not showing visible symptoms. This metric is crucial for controlling Rift Valley Fever, as asymptomatic animals can still spread the virus. Enhancing detection rates through widespread testing is essential for managing outbreaks and implementing effective public health strategies.

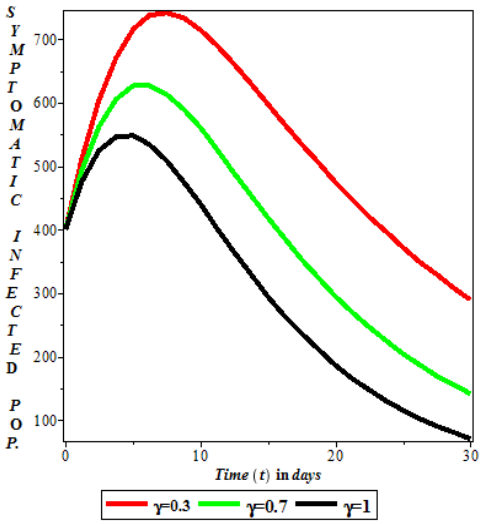

Figure 2. *Symptomatic population with isolation rate* $\gamma_L = 0.3,\ 0.7 \& 1.0$

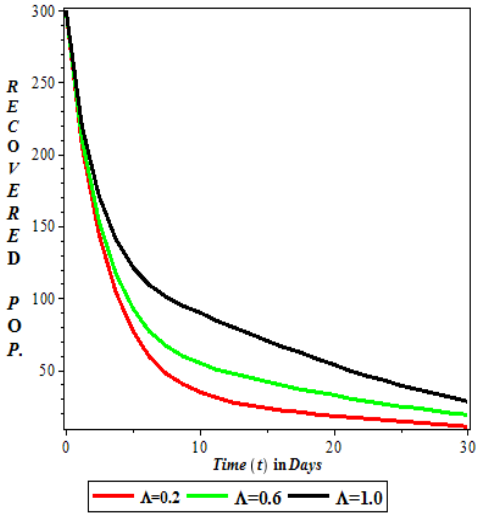

Figure 3. *Recovered population with recovery of isolated individual rate* $\Lambda_L = 0.2,\ 0.6 \& 1.0$

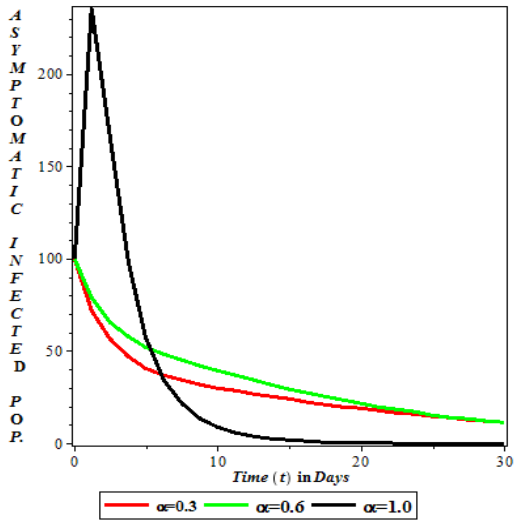

Figure 4. *Asymptomatic population with detection rate* $\alpha_L = 0.3,\ 0.6 \& 1.0$

Figure 5 depicts the symptomatic livestock population alongside their recovery rate, showing the number of animals displaying disease symptoms and eventually recovering over time. This visualization is essential for understanding the progression of Rift Valley fever and evaluating the effectiveness of interventions aimed at livestock recovery. Tracking the recovery rate in symptomatic animals provides critical insights into the disease's dynamics and helps inform strategies for managing and treating affected livestock populations. *Figure 6* represents the asymptomatic livestock population alongside their recovery rate, showing the number of livestock that, while not exhibiting symptoms, recover over time. This recovery may be attributed to the ingestion of certain grasses that unknowingly contain medicinal properties beneficial to the animals' health. Tracking the recovery rate in asymptomatic livestock offers valuable insights into the role of natural remedies and their potential contribution to disease management in livestock populations. *Figure 7* underscores the critical risk posed by infected vectors transmitting diseases to vulnerable livestock, which

exacerbates endemic conditions within the system when effective interventions are lacking. It highlights the livestock population's vulnerability to vector-borne infections and the urgent need for proactive, comprehensive measures to curb their spread and minimize their adverse effects. The figure stresses the importance of implementing timely and targeted actions, such as vector control strategies, vaccination campaigns, early detection of infected livestock, and improved livestock management practices, to safeguard the health and welfare of susceptible animals and reduce the economic impact of disease outbreaks.

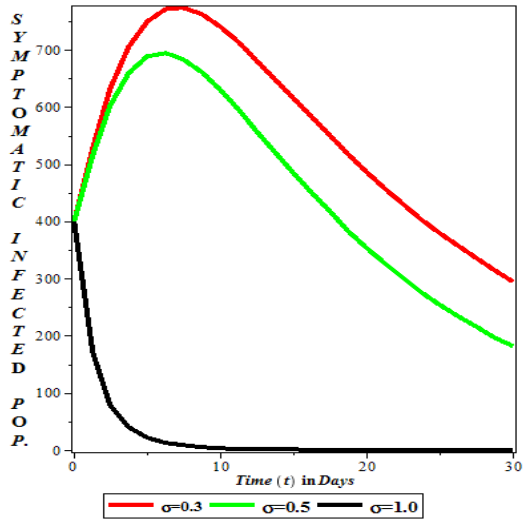

Figure 5. *Symptomatic population with recovery rate* $\sigma_L = 0.3,\ 0.5 \& 1.0$

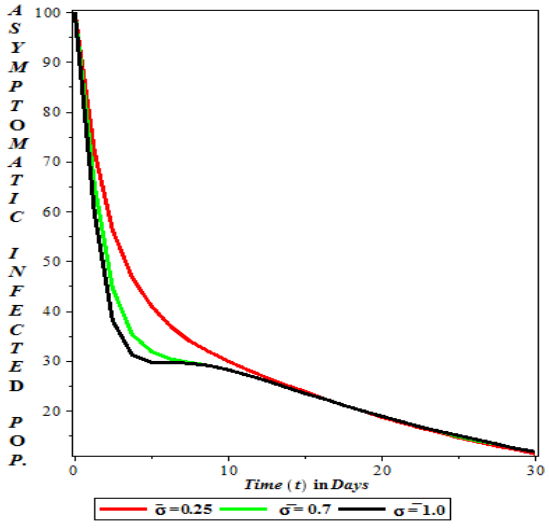

Figure 6. *Asymptomatic population with recovery rate* $\bar{\sigma}_L = 0.25\ 0.7 \& 1.0$

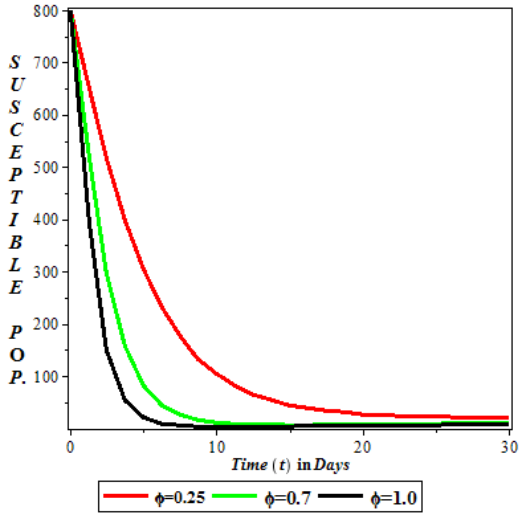

Figure 7. *Susceptible livestock with biting rate* $\phi = 0.25\ 0.7\ \&\ 1.0$

Conclusion

In conclusion, mitigating the impact of Rift Valley fever on livestock populations requires a comprehensive and proactive approach. Key strategies include the early detection and isolation of both asymptomatic and symptomatic infected animals, the implementation of strategic management practices, and the establishment of robust surveillance mechanisms. Veterinary practitioners play a critical role in leading and executing these strategies, ensuring the health and sustainability of livestock farming operations. By prioritizing these proactive measures, stakeholders can effectively reduce the adverse effects of Rift Valley fever on livestock, thereby enhancing agricultural resilience and sustainability.

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Conflict of interest

The authors confirm that there is no conflict of interest involve with any parties in this research study.

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