Modeling the Spatial Spread of Rift Valley Fever in Egypt*

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Abstract

Rift Valley fever (RVF) is a severe viral zoonosis in Africa and the Middle East that harms both human health and livestock production. It is believed that RVF in Egypt is introduced by the importation of infected animals from Sudan. In this paper, we propose a three-patch model for the process that animals enter Egypt from Sudan are moved up the Nile, and then consumed at those population centres. The basic reproduction number for each patch is introduced and then the threshold dynamics of the model are established. We simulate an interesting scenario showing possible explanation to the observed phenomenon on the geographic spread of RVF in Egypt.

Key words. Rift Valley fever, patch model, Egypt, basic reproduction number, threshold dynamics.

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1 Introduction

Rift Valley fever (RVF) is a viral zoonosis of domestic animals (such as cattle, sheep, camels and goats) and humans caused by the RVF virus (RVFV), a member of the genus *Phlebovirus* in the Bunyaviridae family. Initially identified in the Rift Valley of Kenya in 1931, outbreaks of RVF have been reported in sub-Saharan Africa, Egypt, Saudi Arabia and Yemen. These result in significant economic losses due to high mortality and abortion in livestock. The virus is transmitted primarily by the bites of infected female mosquitoes. Several species of *Culex* or *Aedes* mosquitoes are known vectors and some species of *Aedes* can also transmit the virus vertically (mother-to-offspring). Humans can also become infected by direct/indirect contact with the blood or organs of infected animals, but they cannot transmit it. To date, two types of vaccines are available for veterinary use [12], but there is no licensed vaccine for humans. Outbreaks of RVF in East Africa are typically associated with rainfall events [16, 4]. Heavy rainfall is believed to induce outbreaks by raising water levels in low-lying areas sufficiently to allow the hatching of *Aedes* eggs, which can persist during dry periods. Since *Aedes* mosquitoes can transmit RVF vertically, the newly hatched mosquitoes can induce an outbreak once they mature [7]. However, vertical transmission has not been demonstrated in Egypt or Yemen, where outbreaks have also occurred. An alternative hypothesis is that in such regions outbreaks may occur when the disease is introduced by the importation of infected animals [8, 2, 3] or by the use of live virus vaccines [13] together with suitable conditions for transmission, specifically high mosquito densities and the presence of large numbers of host animals [2, 3].

Mathematical models have become an important tool in identifying disease transmission process, assessing infection risk and prevalence, and optimizing control strategies. However, so far little has been done to model and analyze the RVF transmission dynamics [19]. Gaff et al. [9] proposed a compartment model explored the mechanisms of RVFV circulation including *Aedes* and *Culex* mosquitoes and livestock population, in which each adult mosquito population is divided into classes containing susceptible, exposed and infectious individuals and the livestock population is classified as susceptible, exposed, infectious and recovered. To account for vertical transmission in *Aedes* mosquiotes, compartments for uninfected and infected eggs are also included. Meanwhile, only uninfected eggs are included for *Culex* mosquitoes. They derived the basic reproduction number to assess the stability of the disease-free equilibrium and performed sensitivity analysis to determine the most significant model parameters to disease transmission. In [20], Mpheshe et al. modified the model in Gaff et al. [9] to reduce egg classes of mosquitoes, include human population and exclude vertical transmission in mosquitoes. They gave conditions for the stability of the disease-free equilibrium and persistence of the disease. Sensitivity indices of the basic reproduction number and the endemic equilibrium were evaluated to study the relative importance of different factors responsible for RVF transmission and prevalence. It is believed that RVFV is introduced to a disease-free area by insects carried by wind and animal movements through trade [19]. Xue et al. [25] presented a network-based metapopulation model incorporating *Aedes* and *Culex* mosquitoes, livestock and human populations. They tested the model with data from an outbreak of RVF in South Africa and analyzed the sensitivity of the model to its parameters. Recently, Chamchod et al. [5] proposed a simple but innovative model to investigate the emergence of RVF outbreaks, and epizootic and enzootic cycles of RVFV. Many aspects

of their investigation have not been addressed in previous modeling studies. For example, they considered the effect of vaccination on the transmission dynamics of RVFV. However, these models either do not include spatial effects or are too complicated to perform rigorous mathematical analysis.

The main purpose of this paper is to propose a mathematically tractable model with spatial dynamics that can capture the hypothesis that Rift Valley fever outbreaks in Egypt might arise when the importation of large numbers of animals from Sudan coincides with high mosquito densities and there is an introduction of the infection during that period through importation of infected animals, use of live virus vaccines, or some other mechanism. In the next section, we develop a three-patch epidemic model to describe the spatial spread of RVF in Egypt. In Section 3, the basic reproduction number for each patch is calculated and the threshold dynamics of the model will be established. Moreover, the existence and stability of the endemic equilibrium are discussed. In Section 4, we simulate an interesting scenario showing possible explanation to the observed phenomenon on the geographic spread of RVF in Egypt. A brief discussion is given in Section 5.

2 The model

The first outbreak of RVF in Egypt occurred in the Nile Valley and Delta in 1977 [11]. This was the first RVF outbreak recorded outside traditionally affected areas in sub-Saharan Africa. Due to a combination of a lack of experience in dealing with RVF patients and insufficient public health programs, the outbreak caused at least thousands of human infections and hundreds of human deaths [17]. Since then, Egypt has been experiencing continued RVF outbreaks among domestic animals which indicates that the RVFV has become enzootic in Egypt. The imported animals from Sudan and the Horn of Africa were usually not vaccinated against RVFV. Travel time from north-central Sudan, where RVF was epizootic, to livestock markets in southern Egypt (Aswan Province), was less than 5 days, approximating the incubation period of RVFV in sheep [8, 1]. So it is hypothesized that the recurrence of epizootic is mainly caused by the continuous importation of infected animals from Sudan and failure of the locally applied RVF vaccination program [13].

Egypt is an arid country with most of the population concentrated along the Nile, in the Delta and near the Suez Canal. The imported animals enter southern Egypt from northern Sudan, are moved up the Nile, and then consumed at these population centres. Vertical transmission of RVF has not been shown to occur in Egypt [18]. For simplicity, we restrict our focus on the disease transmission between domestic animals and mosquitoes. To capture the idea that more mosquitoes lead to more transmission, it seems most natural to use mass-action transmission terms. The movement timescale of animals is relatively short, so we assume that there is no host reproduction during the journey. Therefore, the density of hosts is determined by movement, mortality, and the rate at which they are introduced, which could be set to depend on demand. We assume that there is no movement for vector population because of their limited mobility. Assume also that the mosquito population satisfies the logistic growth to maintain an equilibrium vector population. For epidemiology, we use a simple SIRS model for hosts and an SI model for vectors.

Based on the above assumptions, we propose a three-patch model (Sudan-Nile-feast) with

Table 1: The state variables in model (2.1) and their descriptions

Symbol	Description
S_i	Number of susceptible animals in patch i at time t
I_i	Number of infectious animals in patch i at time t
R_i	Number of recovered animals in patch i at time t
U_i	Number of susceptible mosquitoes in patch i at time t
V_i	Number of infectious mosquitoes in patch i at time t

animals movement from patch 1 to patch 2 and then from patch 2 to patch 3:

$$\begin{cases} \dfrac{dS_1}{dt} = r - \alpha_1 S_1 V_1 - \mu S_1 + \zeta R_1 - cS_1, \\ \dfrac{dI_1}{dt} = \alpha_1 S_1 V_1 - (\mu + \gamma + \delta) I_1 - cI_1, \\ \dfrac{dR_1}{dt} = \gamma I_1 - (\mu + \zeta) R_1 - cR_1, \\ \dfrac{dU_1}{dt} = \xi_1 (U_1 + V_1) - \dfrac{\xi_1 - \nu_1}{M_1} (U_1 + V_1)^2 - \nu_1 U_1 - \beta_1 I_1 U_1, \\ \dfrac{dV_1}{dt} = -\nu_1 V_1 + \beta_1 I_1 U_1, \end{cases} \tag{2.1a}$$

$$\begin{cases} \dfrac{dS_2}{dt} = cS_1 - \alpha_2 S_2 V_2 - \mu S_2 + \zeta R_2 - cS_2, \\ \dfrac{dI_2}{dt} = cI_1 + \alpha_2 S_2 V_2 - (\mu + \gamma + \delta) I_2 - cI_2, \\ \dfrac{dR_2}{dt} = cR_1 + \gamma I_2 - (\mu + \zeta) R_2 - cR_2, \\ \dfrac{dU_2}{dt} = \xi_2 (U_2 + V_2) - \dfrac{\xi_2 - \nu_2}{M_2} (U_2 + V_2)^2 - \nu_2 U_2 - \beta_2 I_2 U_2, \\ \dfrac{dV_2}{dt} = -\nu_2 V_2 + \beta_2 I_2 U_2, \end{cases} \tag{2.1b}$$

$$\begin{cases} \dfrac{dS_3}{dt} = cS_2 - \alpha_3 S_3 V_3 - \mu S_3 + \zeta R_3 - cS_3, \\ \dfrac{dI_3}{dt} = cI_2 + \alpha_3 S_3 V_3 - (\mu + \gamma + \delta) I_3 - cI_3, \\ \dfrac{dR_3}{dt} = cR_2 + \gamma I_3 - (\mu + \zeta) R_3 - cR_3, \\ \dfrac{dU_3}{dt} = \xi_3 (U_3 + V_3) - \dfrac{\xi_3 - \nu_3}{M_3} (U_3 + V_3)^2 - \nu_3 U_3 - \beta_3 I_3 U_3, \\ \dfrac{dV_3}{dt} = -\nu_3 V_3 + \beta_3 I_3 U_3. \end{cases} \tag{2.1c}$$

The state variables and parameters used in model (2.1) and their descriptions are presented in Table 1 and Table 2, respectively.

Table 2: The parameters in model (2.1) and their descriptions

Symbol	Description
r	Recruitment rate of animals
c	Movement rate of animals
μ	Natural death rate for animals
δ	Disease-induced death rate for animals
γ	Recovery rate for animals
ζ	Rate of loss of immunity for animals
ξ_i	Growth rate of mosquitoes in patch i
ν_i	Natural death rate for mosquitoes in patch i
M_i	Carrying capacity for mosquitoes in patch i
α_i	Transmission rate from vector to host in patch i
β_i	Transmission rate from host to vector in patch i

The total number of mosquitoes in patch i at time t, denoted by $N_i^v(t)$, satisfies

$$\frac{dN_i^v}{dt} = (\xi_i - \nu_i)N_i^v - \frac{\xi_i - \nu_i}{M_i}(N_i^v)^2, i = 1, 2, 3,$$

and it converges to M_i as $t \to \infty$ for any positive initial value. Therefore, we may consider the following reduced system

$$\begin{cases} \frac{dS_1}{dt} = r - \alpha_1 S_1 V_1 - \mu S_1 + \zeta R_1 - cS_1, \\ \frac{dI_1}{dt} = \alpha_1 S_1 V_1 - (\mu + \gamma + \delta) I_1 - cI_1, \\ \frac{dR_1}{dt} = \gamma I_1 - (\mu + \zeta) R_1 - cR_1, \\ \frac{dV_1}{dt} = -\nu_1 V_1 + \beta_1 I_1 (M_1 - V_1), \end{cases} \tag{2.2a}$$

$$\begin{cases} \frac{dS_2}{dt} = cS_1 - \alpha_2 S_2 V_2 - \mu S_2 + \zeta R_2 - cS_2, \\ \frac{dI_2}{dt} = cI_1 + \alpha_2 S_2 V_2 - (\mu + \gamma + \delta) I_2 - cI_2, \\ \frac{dR_2}{dt} = cR_1 + \gamma I_2 - (\mu + \zeta) R_2 - cR_2, \\ \frac{dV_2}{dt} = -\nu_2 V_2 + \beta_2 I_2 (M_2 - V_2), \end{cases} \tag{2.2b}$$

$$\begin{cases} \dfrac{dS_3}{dt} = cS_2 - \alpha_3 S_3 V_3 - \mu S_3 + \zeta R_3 - cS_3, \\ \dfrac{dI_3}{dt} = cI_2 + \alpha_3 S_3 V_3 - (\mu+\gamma+\delta) I_3 - cI_3, \\ \dfrac{dR_3}{dt} = cR_2 + \gamma I_3 - (\mu+\zeta) R_3 - cR_3, \\ \dfrac{dV_3}{dt} = -\nu_3 V_3 + \beta_3 I_3 (M_3 - V_3). \end{cases} \tag{2.2c}$$

Theorem 2.1. *All forward solutions in* $\mathbb{R}^{12}_+$ *of* (2.2) *eventually enter* $\Omega = \Omega_1 \times \Omega_2 \times \Omega_3$, *where* $\Omega_i = \{(S_i, I_i, R_i, V_i) \in \mathbb{R}^4_+ : S_i + I_i + R_i \leq \dfrac{rc^{i-1}}{(\mu+c)^i}, V_i \leq M_i\}, i = 1, 2, 3,$ *and* Ω *is positively invariant for* (2.2).

Proof. Let $N_i^h(t)$ be the total number of host population in patch i at time t. Then we have

$$\frac{dN_1^h}{dt} = r - (\mu + c)N_1^h - \delta I_1 \leq r - (\mu + c)N_1^h$$

and

$$\frac{dN_i^h}{dt} = cN_{i-1}^h - (\mu + c)N_i^h - \delta I_i \leq cN_{i-1}^h - (\mu + c)N_i^h, \ i = 2, 3.$$

By a simple comparison theorem [22], the proof is complete. □

3 Mathematical analysis

It is easy to see that (2.2) has a unique disease-free equilibrium

$$\begin{aligned} E^0 &= (S_1^0, I_1^0, R_1^0, V_1^0, S_2^0, I_2^0, R_2^0, V_2^0, S_3^0, I_3^0, R_3^0, V_3^0) \\ &= (\frac{r}{\mu + c}, 0, 0, 0, \frac{rc}{(\mu + c)^2}, 0, 0, 0, \frac{rc^2}{(\mu + c)^3}, 0, 0, 0). \end{aligned}$$

Note that system (2.2) is in a block-triangular form, the dynamics of patch 1 are independent of patch 2 and patch 3 while the dynamics of patch 2 are independent of patch 3.

3.1 The first patch

Obviously, $E_1^0 = (S_1^0, 0, 0, 0)$ is the unique disease-free equilibrium of subsystem (2.2a). To calculate the basic reproduction number corresponding to (2.2a), we order the infected state variables by (I_1, R_1, V_1). Following the method and notations of van den Driessche and Watmough [24], the linearization of (2.2a) at E_1^0 gives

$$F = \begin{bmatrix} 0 & 0 & \alpha_1 S_1^0 \\ 0 & 0 & 0 \\ \beta_1 M_1 & 0 & 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} \mu + \gamma + \delta + c & 0 & 0 \\ -\gamma & \mu + \zeta + c & 0 \\ 0 & 0 & \nu_1 \end{bmatrix}.$$

Direct calculation yields

$$V^{-1} = \begin{bmatrix} (\mu+\gamma+\delta+c)^{-1} & 0 & 0 \\ \gamma(\mu+\gamma+\delta+c)^{-1}(\mu+\zeta+c)^{-1} & (\mu+\zeta+c)^{-1} & 0 \\ 0 & 0 & \nu_1^{-1} \end{bmatrix}$$

and the basic reproduction number for the first patch equals

$$\mathcal{R}_{10} = \rho(FV^{-1}) = \sqrt{\frac{\alpha_1 S_1^0}{\nu_1} \cdot \frac{\beta_1 M_1}{\mu+\gamma+\delta+c}} = \sqrt{\frac{\alpha_1 r}{(\mu+c)\nu_1} \cdot \frac{\beta_1 M_1}{\mu+\gamma+\delta+c}},$$

which depends on all parameters except ζ, the rate of loss of immunity for animals. $(\mathcal{R}_{10})^2$ is proportional to S_1^0 and M_1, so more mosquitoes and more animals lead to more disease transmission.

Theorem 3.1. *The disease-free equilibrium E_1^0 of* (2.2a) *is globally asymptotically stable in Ω_1 if $\mathcal{R}_{10} \leq 1$ and unstable if $\mathcal{R}_{10} > 1$.*

Proof. It is easy to show the local stability of E_1^0 by verifying (A1)-(A5) in van den Drissche and Watmough [24].

Consider a Lyapunov function $L_1 = \nu_1(\mu+c)I_1 + \alpha_1 r V_1$ on Ω_1. Then

$$\begin{aligned} L_1' &= \nu_1(\mu+c)I_1' + \alpha_1 r V_1' \\ &= \nu_1(\mu+c)\alpha_1 S_1 V_1 - \nu_1(\mu+c)(\mu+\gamma+\delta+c)I_1 - \alpha_1 r\nu_1 V_1 + \alpha_1 r\beta_1 I_1(M_1 - V_1) \\ &= [\nu_1(\mu+c)\alpha_1 S_1 - \alpha_1 r\nu_1]V_1 + [\alpha_1 r\beta_1(M_1 - V_1) - \nu_1(\mu+c)(\mu+\gamma+\delta+c)]I_1 \\ &= \nu_1(\mu+c)\alpha_1(S_1 - S_1^0)V_1 + [\alpha_1 r\beta_1(M_1 - V_1) - \nu_1(\mu+c)(\mu+\gamma+\delta+c)]I_1 \\ &\leq [\alpha_1 r\beta_1(M_1 - V_1) - \nu_1(\mu+c)(\mu+\gamma+\delta+c)]I_1 \quad \text{in} \quad \Omega_1 \\ &\leq [\alpha_1 r\beta_1 M_1 - \nu_1(\mu+c)(\mu+\gamma+\delta+c)]I_1 \\ &= [(\mathcal{R}_{10}^2 - 1)\nu_1(\mu+c)(\mu+\gamma+\delta+c)]I_1 \\ &\leq 0 \quad \text{if} \quad \mathcal{R}_{10} \leq 1. \end{aligned}$$

The largest compact invariant set, denoted by Γ_1, in $\{(S_1, I_1, R_1, V_1) \in \Omega_1 : L_1' = 0\}$ is the singleton $\{E_1^0\}$.

Case 1: $\mathcal{R}_{10} < 1$. Preceding calculation shows that $I_1 \equiv 0$. So

$$\frac{dV_1}{dt} = -\nu_1 V_1 \text{ and } \frac{dR_1}{dt} = -(\mu+\zeta+c)R_1.$$

Backward continuation of a compact invariant set indicates that $V_1 = 0$ and $R_1 = 0$. Thus

$$\frac{dS_1}{dt} = r - (\mu+c)S_1.$$

This means that $S_1 = S_1^0$ and hence $\Gamma_1 = \{E_1^0\}$.

Case 2: $\mathcal{R}_{10} = 1$. The preceding calculation gives either $V_1 \equiv 0$ or $I_1 \equiv 0$. The latter case proceeds as before. Suppose $V_1 \equiv 0$, then $\frac{dV_1}{dt} = \beta_1 I_1 M_1 \equiv 0$ which implies $I_1 = 0$. Once again this can proceed as before.

By LaSalle's Invariance Principle [14], E_1^0 is globally asymptotically stable in Ω_1. □

Theorem 3.2. *If $\mathcal{R}_{10} > 1$, then system (2.2a) has a unique endemic equilibrium, denoted by $E_1^* = (S_1^*, I_1^*, R_1^*, V_1^*)$, which is locally asymptotically stable. Moreover, the disease is uniformly persistent in Ω_1^0, the interior of Ω_1, i.e., there is a constant $\epsilon > 0$ such that any solution of (2.2a) starting at a point of Ω_1^0 satisfies*

$$\liminf_{t\to\infty}(I_1(t), R_1(t), V_1(t)) > (\epsilon, \epsilon, \epsilon).$$

Proof. If $E_1^* = (S_1^*, I_1^*, R_1^*, V_1^*)$ is a positive equilibrium of (2.2a), then it satisfies the following system of algebraic equations

$$\begin{aligned} r - \alpha_1 S_1 V_1 - \mu S_1 + \zeta R_1 - cS_1 &= 0,\\ \alpha_1 S_1 V_1 - (\mu+\gamma+\delta) I_1 - cI_1 &= 0,\\ \gamma I_1 - (\mu+\zeta) R_1 - cR_1 &= 0,\\ -\nu_1 V_1 + \beta_1 I_1 (M_1 - V_1) &= 0. \end{aligned} \tag{3.1}$$

Solving S_1, R_1 and V_1 in terms of I_1 from the last three equations of (3.1), that is,

$$S_1 = \frac{(\mu+\gamma+\delta+c)(\nu_1+\beta_1 I_1)}{\alpha_1\beta_1 M_1},\ R_1 = \frac{\gamma I_1}{\mu+\zeta+c},\ V_1 = \frac{\beta_1 I_1 M_1}{\nu_1+\beta_1 I_1},$$

and substituting them into the first equation, we obtain

$$r - (\mu+\gamma+\delta+c)I_1 - (\mu+c)\frac{\mu+\gamma+\delta+c}{\alpha_1\beta_1 M_1}(\beta_1 I_1 + \nu_1) + \zeta\frac{\gamma}{\mu+\zeta+c} I_1 = 0,$$

which can be simplified to a linear equation

$$\left[(\mu+\gamma+\delta+c) + (\mu+c)\frac{\mu+\gamma+\delta+c}{\alpha_1 M_1} - \frac{\zeta\gamma}{\mu+\zeta+c}\right] I_1 + \left[(\mu+c)\frac{\mu+\gamma+\delta+c}{\alpha_1\beta_1 M_1}\nu_1 - r\right] = 0.$$

The coefficient of I_1 is always positive and the constant part is negative if and only if $\mathcal{R}_{10} > 1$. Hence, system (2.2a) has a unique endemic equilibrium if and only if $\mathcal{R}_{10} > 1$.

Next we study the local stability of E_1^* by using the Routh-Hurwitz criterion. The Jacobian matrix of system (2.2a) at the endemic equilibrium E_1^* is

$$J(S_1^*, I_1^*, R_1^*, V_1^*) = \begin{pmatrix} -\alpha_1 V_1^* - \rho & 0 & \zeta & -\alpha_1 S_1^* \\ \alpha_1 V_1^* & -(\rho+\gamma+\delta) & 0 & \alpha_1 S_1^* \\ 0 & \gamma & -(\rho+\zeta) & 0 \\ 0 & \beta_1(M_1 - V_1^*) & 0 & -\nu_1 - \beta_1 I_1^* \end{pmatrix},$$

where $\rho = \mu + c$ and the corresponding characteristic equation is

$$P_1(\lambda) = (\lambda+\rho+\zeta)(\lambda^3 + b_2\lambda^2 + b_1\lambda + b_0) - \zeta\alpha_1 V_1^*\gamma(\lambda + \nu_1 + \beta_1 I_1^*) = 0,$$

where

$$\begin{aligned} b_2 &= \alpha_1 V_1^* + 2\rho + \gamma + \delta + \nu_1 + \beta_1 I_1^* > 0,\\ b_1 &= (\alpha_1 V_1^* + \rho)(\rho+\gamma+\delta) + (\alpha_1 V_1^* + 2\rho + \gamma + \delta)(\nu_1 + \beta_1 I_1^*) - \alpha_1\beta_1 S_1^*(M_1 - V_1^*),\\ b_0 &= (\alpha_1 V_1^* + \rho)(\rho+\gamma+\delta)(\nu_1 + \beta_1 I_1^*) - \alpha_1\beta_1 S_1^*\rho(M_1 - V_1^*). \end{aligned}$$

It follows from the second and fourth equations of (3.1) that

$$(\rho+\gamma+\delta)\nu_1 = \alpha_1\beta_1 S_1^*(M_1 - V_1^*)$$

and hence

$$\begin{aligned} b_1 &= (\alpha_1 V_1^* + \rho)(\rho+\gamma+\delta+\nu_1) + (\alpha_1 V_1^* + 2\rho + \gamma + \delta)\beta_1 I_1^* > 0, \\ b_0 &= (\rho+\gamma+\delta)(\alpha_1\nu_1 V_1^* + \alpha_1\beta_1 V_1^* I_1^* + \rho\beta_1 I_1^*) > 0 \quad \text{and} \quad b_1 b_2 > b_0. \end{aligned}$$

Then

$$P_1(\lambda) = \lambda^4 + c_3\lambda^3 + c_2\lambda^2 + c_1\lambda + c_0 = 0,$$

where

$$\begin{aligned} c_3 &= \rho + \zeta + b_2 > 0, c_2 = (\rho+\zeta)b_2 + b_1 > 0, \\ c_1 &= (\rho+\zeta)b_1 + b_0 - \zeta\alpha_1 V_1^*\gamma = \rho b_1 + b_0 + \zeta(b_1 - \alpha_1 V_1^*\gamma) > 0, \\ c_0 &= (\rho+\zeta)b_0 - \zeta\alpha_1 V_1^*\gamma(\nu_1 + \beta_1 I_1^*) = \rho b_0 + \zeta(b_0 - \alpha_1 V_1^*\gamma(\nu_1 + \beta_1 I_1^*)) > 0. \end{aligned}$$

Now it suffices to show that $c_1c_2c_3 > c_1^2 + c_3^2c_0$. In fact

$$\begin{aligned} &c_1c_2c_3 - c_1^2 - c_3^2c_0 = c_1(c_2c_3 - c_1) - c_3^2c_0 \\ =&c_1[c_3(\rho+\zeta)b_2 + (b_1b_2 - b_0) + \zeta\alpha_1 V_1^*\gamma] - c_3^2c_0 \\ >&c_1c_3(\rho+\zeta)b_2 - c_3^2c_0 = c_3[c_1(\rho+\zeta)b_2 - c_3c_0] \\ =&c_3[(\rho+\zeta)^2(b_1b_2 - b_0) - \zeta\alpha_1 V_1^*\gamma((\rho+\zeta)b_2 - (\rho+\zeta+b_2)(\nu_1+\beta_1 I_1^*))] \\ >&c_3[(\rho+\zeta)\zeta(b_1b_2 - b_0) - \zeta\alpha_1 V_1^*\gamma(\rho+\zeta)b_2] \\ =&c_3(\rho+\zeta)\zeta(b_1b_2 - b_0 - \alpha_1 V_1^*\gamma b_2) > 0. \end{aligned}$$

Thus, the Routh-Hurwitz criterion implies that all eigenvalues of the characteristic equation have negative real parts. Hence, the endemic equilibrium is locally asymptotically stable.

Finally, the uniform persistence of system (2.2a) in Ω_1^0 can be proved by applying Theorem 4.6 in Thieme [23]. We omit the proof here, since it is similar to that of Theorem 2.5 in Gao and Ruan [10]. □

Remark 3.3. It is worth mentioning that Yang et al. [26] studied a similar vector-host epidemic model with an SIR structure for the host population and without disease-induced host deaths. They used the method of the second additive compound matrix (see [15] and references therein) to establish the global stability of the endemic equilibrium when it exists. Unfortunately, we cannot use that approach to establish the global result because of the higher complexity in our model.

3.2 The second patch

By a simple comparison theorem, we conclude that the disease is uniformly persistent in Ω^0 if it is uniformly persistent in Ω_1^0. Namely, the disease will persist in all three patches if $\mathcal{R}_{10} > 1$. Indeed, it follows from Theorem 3.2 that for any fixed initial data we have

$$\frac{dI_2}{dt} \geq c\epsilon - (\mu+\gamma+\delta+c)I_2$$

for t large enough. So $\liminf_{t\to\infty} I_2(t) \geq c\epsilon/(\mu+\gamma+\delta+c)$. Similarly, we can find positive lower limits for all other variables. If the disease dies out in patch 1, i.e., $\mathcal{R}_{10} \leq 1$, then each solution of (2.2a) with nonnegative initial data converges to E_1^0 and the limiting system of (2.2b) is

$$\begin{aligned}
\frac{dS_2}{dt} &= cS_1^0 - \alpha_2 S_2 V_2 - \mu S_2 + \zeta R_2 - cS_2, \\
\frac{dI_2}{dt} &= \alpha_2 S_2 V_2 - (\mu+\gamma+\delta) I_2 - cI_2, \\
\frac{dR_2}{dt} &= \gamma I_2 - (\mu+\zeta) R_2 - cR_2, \\
\frac{dV_2}{dt} &= -\nu_2 V_2 + \beta_2 I_2 (M_2 - V_2).
\end{aligned} \tag{3.2}$$

Comparing (3.2) with (2.2a), we immediately find that (3.2) possesses a unique disease-free equilibrium $E_2^0 = (S_2^0, I_2^0, R_2^0, V_2^0) = (cS_1^0/(\mu+c), 0, 0, 0) = (rc/(\mu+c)^2, 0, 0, 0)$ and obtain the basic reproduction number of patch 2 as

$$\mathcal{R}_{20} = \sqrt{\frac{\alpha_2 S_2^0}{\nu_2} \cdot \frac{\beta_2 M_2}{\mu+\gamma+\delta+c}} = \sqrt{\frac{\alpha_2 rc}{(\mu+c)^2 \nu_2} \cdot \frac{\beta_2 M_2}{\mu+\gamma+\delta+c}}.$$

If $\mathcal{R}_{10} \leq 1$ and $\mathcal{R}_{20} \leq 1$, then the disease goes extinct in the first two patches; if $\mathcal{R}_{10} \leq 1$ and $\mathcal{R}_{20} > 1$, then the disease dies out in the first patch but persists in the last two patches.

3.3 The third patch

Similarly, if $\mathcal{R}_{10} \leq 1$ and $\mathcal{R}_{20} \leq 1$, we obtain a limiting system of (2.2c) as follows:

$$\begin{aligned}
\frac{dS_3}{dt} &= cS_2^0 - \alpha_3 S_3 V_3 - \mu S_3 + \zeta R_3 - cS_3, \\
\frac{dI_3}{dt} &= \alpha_3 S_3 V_3 - (\mu+\gamma+\delta) I_3 - cI_3, \\
\frac{dR_3}{dt} &= \gamma I_3 - (\mu+\zeta) R_3 - cR_3, \\
\frac{dV_3}{dt} &= -\nu_3 V_3 + \beta_3 I_3 U_3,
\end{aligned} \tag{3.3}$$

System (3.3) has a unique disease-free equilibrium $E_3^0 = (S_3^0, I_3^0, R_3^0, V_3^0) = (cS_2^0/(\mu+c), 0, 0, 0) = (rc^2/(\mu+c)^3, 0, 0, 0)$ and the basic reproduction number of patch 3 is given by

$$\mathcal{R}_{30} = \sqrt{\frac{\alpha_3 S_3^0}{\nu_3} \cdot \frac{\beta_3 M_3}{\mu+\gamma+\delta+c}} = \sqrt{\frac{\alpha_3 rc^2}{(\mu+c)^3 \nu_3} \cdot \frac{\beta_3 M_3}{\mu+\gamma+\delta+c}}.$$

If $\mathcal{R}_{10} \leq 1$, $\mathcal{R}_{20} \leq 1$ and $\mathcal{R}_{30} \leq 1$, then the disease goes extinct in all three patches; if $\mathcal{R}_{10} \leq 1$, $\mathcal{R}_{20} \leq 1$ and $\mathcal{R}_{30} > 1$, then the disease dies out in the first two patches, but persists in the third patch. So we have the following result:

Theorem 3.4. *For the full model (2.2), if $\mathcal{R}_{10} > 1$, the disease persists in all three patches; if $\mathcal{R}_{10} \leq 1$ and $\mathcal{R}_{20} > 1$, the disease dies out in the first patch but persists in the remaining two patches; if $\mathcal{R}_{10} \leq 1$, $\mathcal{R}_{20} \leq 1$ and $\mathcal{R}_{30} > 1$, the disease dies out in the first two patches, but persists in the last patch; if $\mathcal{R}_{10} \leq 1$, $\mathcal{R}_{20} \leq 1$ and $\mathcal{R}_{30} \leq 1$, the disease dies out in all three patches and E^0 is globally asymptotically stable.*

Theorem 3.5. *System (2.2) has a unique endemic equilibrium, denoted $E^* = (S_1^*, I_1^*, R_1^*, V_1^*, S_2^*, I_2^*, R_2^*, V_2^*, S_3^*, I_3^*, R_3^*, V_3^*)$, if and only if $\mathcal{R}_{10} > 1$ and it is locally asymptotically stable when it exists.*

Proof. The necessity is a straightforward consequence of Theorem 3.1. To prove the existence and uniqueness of an endemic equilibrium as $\mathcal{R}_{10} > 1$, it suffices to show that the system

$$\begin{aligned}
\frac{dS_i}{dt} &= cS_{i-1}^* - \alpha_i S_i V_i - \mu S_i + \zeta R_i - cS_i, \\
\frac{dI_i}{dt} &= cI_{i-1}^* + \alpha_i S_i V_i - (\mu + \gamma + \delta) I_i - cI_i, \\
\frac{dR_i}{dt} &= cR_{i-1}^* + \gamma I_i - (\mu + \zeta) R_i - cR_i, \\
\frac{dV_i}{dt} &= -\nu_i V_i + \beta_i I_i (M_i - V_i),
\end{aligned} \tag{3.4}$$

has a unique positive equilibrium for $i = 2, 3$. To compute the constant solution of (3.4), we set the right hand side of each of the four equations equal to zero and direct calculations yield

$$cS_{i-1}^* + cI_{i-1}^* - (\mu+\gamma+\delta+c)I_i - (\mu+c)\frac{(\mu+\gamma+\delta+c)I_i - cI_{i-1}^*}{\alpha_i} \cdot \frac{\beta_i I_i + \nu_i}{\beta_i M_i I_i} + \zeta \frac{cR_{i-1}^* + \gamma I_i}{\mu + \zeta + c} = 0,$$

which can be reduced to a quadratic equation

$$f(I_i) \equiv a_2 I_i^2 + a_1 I_i + a_0 = 0, \tag{3.5}$$

where $a_2 = -\left(1 + \frac{\mu + c}{\alpha_i M_i}\right)(\mu+\gamma+\delta+c) + \zeta \frac{\gamma}{\mu+\zeta+c} < 0$, $a_1 = cS_{i-1}^* + cI_{i-1}^* - \frac{\mu+c}{\alpha_i \beta_i M_i}((\mu+\gamma+\delta+c)\nu_i - cI_{i-1}^*\beta_i) + \zeta \frac{cR_{i-1}^*}{\mu+\zeta+c}$ and $a_0 = \frac{\mu+c}{\alpha_i \beta_i M_i} cI_{i-1}^* \nu_i > 0$.

Thus, (3.5) has exactly one positive root, I_i^*. To check the positivity of other variables, we need to verify that $I_i^* > cI_{i-1}^*/(\mu+\gamma+\delta+c)$, or equivalently, $f(cI_{i-1}^*/(\mu+\gamma+\delta+c)) > 0$. In fact, $f(cI_{i-1}^*/(\mu+\gamma+\delta+c))$ equals

$$\frac{\zeta\gamma c^2 (I_{i-1}^*)^2}{(\mu+\zeta+c)(\mu+\gamma+\delta+c)^2} + \frac{c^2 S_{i-1}^* I_{i-1}^*}{\mu+\gamma+\delta+c} + \frac{\zeta c^2 R_{i-1}^* I_{i-1}^*}{(\mu+\zeta+c)(\mu+\gamma+\delta+c)} > 0.$$

The local stability of the endemic equilibrium $(S_i^*, I_i^*, R_i^*, V_i^*)$ of system (3.4) can be proved in a way similar to that of E_1^* in Theorem 3.2. □

3.4 Model with restriction

Research in RVF indicates that an infection leads to a durable, probably life-long, immunity in animals [21]. In any event, the immunity period is relatively longer than the duration of movement. We may assume that the rate of loss of immunity ζ equals zero and use an SIR model for the host population. In this case, since R_i does not appear in other equations of (2.2), system (2.2) can be reduced to

$$\begin{cases} \dfrac{dS_1}{dt} = r - \alpha_1 S_1 V_1 - \mu S_1 - cS_1, \\ \dfrac{dI_1}{dt} = \alpha_1 S_1 V_1 - (\mu + \gamma + \delta) I_1 - cI_1, \\ \dfrac{dV_1}{dt} = -\nu_1 V_1 + \beta_1 I_1 (M_1 - V_1), \end{cases} \tag{3.6a}$$

$$\begin{cases} \dfrac{dS_2}{dt} = cS_1 - \alpha_2 S_2 V_2 - \mu S_2 - cS_2, \\ \dfrac{dI_2}{dt} = cI_1 + \alpha_2 S_2 V_2 - (\mu + \gamma + \delta) I_2 - cI_2, \\ \dfrac{dV_2}{dt} = -\nu_2 V_2 + \beta_2 I_2 (M_2 - V_2), \end{cases} \tag{3.6b}$$

$$\begin{cases} \dfrac{dS_3}{dt} = cS_2 - \alpha_3 S_3 V_3 - \mu S_3 - cS_3, \\ \dfrac{dI_3}{dt} = cI_2 + \alpha_3 S_3 V_3 - (\mu + \gamma + \delta) I_3 - cI_3, \\ \dfrac{dV_3}{dt} = -\nu_3 V_3 + \beta_3 I_3 (M_3 - V_3). \end{cases} \tag{3.6c}$$

The following result can be proved in a way similar to that of Theorem 4.3 in Yang et al. [26]. Consequently, the disease dynamics of (3.6) are completely determined by the basic reproduction numbers $\mathcal{R}_{i0}$ for $i = 1, 2, 3$.

Theorem 3.6. *For system (3.6), if $\mathcal{R}_{10} > 1$, then the disease persists at an endemic equilibrium level in all three patches ; if $\mathcal{R}_{10} \leq 1$ and $\mathcal{R}_{20} > 1$, then the disease dies out in the first patch but persists at an endemic equilibrium level in the remaining two patches; if $\mathcal{R}_{10} \leq 1$, $\mathcal{R}_{20} \leq 1$ and $\mathcal{R}_{30} > 1$, then the disease dies out in the first two patches but persists at an endemic equilibrium level in the last patch; if $\mathcal{R}_{10} \leq 1$, $\mathcal{R}_{20} \leq 1$ and $\mathcal{R}_{30} \leq 1$, then the disease dies out in all three patches.*

3.5 The relation between $\mathcal{R}_0$ and model parameters

It follows from Theorem 3.4 that the disease dies out in all patches if and only if $\mathcal{R}_{i0} \leq 1$ for $i = 1, 2, 3$. In other words, to eliminate the disease from the whole system, all three threshold parameters $\mathcal{R}_{10}, \mathcal{R}_{20}$ and $\mathcal{R}_{30}$ must be reduced to be less than 1. To do so, we should study how the basic reproduction numbers vary with the model parameters which can help us design highly efficient control strategies. Recall that

$$\mathcal{R}_{i0}^2 = \frac{\alpha_i r c^{i-1}}{(\mu + c)^i \nu_i} \cdot \frac{\beta_i M_i}{\mu + \gamma + \delta + c}, i = 1, 2, 3.$$

Obviously, $\mathcal{R}_{i0}$ is strictly increasing in α_i, β_i, M_i or r, and strictly decreasing in ν_i, μ, γ or δ. An increase in the movement rate, c, will decrease $\mathcal{R}_{10}$. The dependence of $\mathcal{R}_{i0}$ on c becomes more complicated if $i > 1$, since c appears in both the numerator and denominator of the formula for $\mathcal{R}_{i0}^2$.

Proposition 3.7. *For $i > 1$, there exists some c_i^* such that the basic reproduction number $\mathcal{R}_{i0}$ is strictly increasing in c if $c \in (0, c_i^*)$ and strictly decreasing if $c \in (c_i^*, \infty)$. Furthermore, $(i-1)\mu/2 < c_i^* < (i-1)\mu$.*

Proof. Let $g_i(c)$ be the partial derivative of $\mathcal{R}_{i0}^2$ with respect to c. Then

$$\begin{aligned} g_i(c) &= \frac{\alpha_i r \beta_i M_i}{\nu_i} \cdot \frac{\partial}{\partial c}\Big(\frac{c^{i-1}}{(\mu+c)^i(\mu+\gamma+\delta+c)}\Big) \\ &= \frac{\alpha_i r \beta_i M_i}{\nu_i} \cdot c^{i-2} \cdot \frac{(i-1)(\mu+c)(\mu+\gamma+\delta+c) - ci(\mu+\gamma+\delta+c) - c(\mu+c)}{(\mu+c)^{i+1}(\mu+\gamma+\delta+c)^2} \\ &= \frac{\alpha_i r \beta_i M_i}{\nu_i} \cdot c^{i-2} \cdot \frac{i\mu(\mu+\gamma+\delta+c) - (\mu+c)(\mu+\gamma+\delta+c) - c(\mu+c)}{(\mu+c)^{i+1}(\mu+\gamma+\delta+c)^2} \\ &= \frac{\alpha_i r \beta_i M_i}{\nu_i} \cdot c^{i-2} \cdot \frac{-2c^2 - (\gamma+\delta+(3-i)\mu)c + (i-1)\mu(\mu+\gamma+\delta)}{(\mu+c)^{i+1}(\mu+\gamma+\delta+c)^2} \end{aligned}$$

and the sign of $g_i(c)$ is the same as that of

$$h_i(c) = -2c^2 - (\gamma+\delta+(3-i)\mu)c + (i-1)\mu(\mu+\gamma+\delta).$$

Since $h_i(0) = (i-1)\mu(\mu+\gamma+\delta) > 0$, the equation $h_i(c) = 0$ has exactly one positive root, denoted by c_i^*, satisfying $h_i(c) > 0$ if $c \in (0, c_i^*)$ and $h_i(c) < 0$ if $c \in (c_i^*, \infty)$. Note that

$$\begin{aligned} h_i(k\mu) =& -2k^2\mu^2 - (\gamma+\delta+(3-i)\mu)k\mu + (i-1)\mu(\mu+\gamma+\delta) \\ =& [-2k^2 - (3-i)k + (i-1)]\mu^2 - [(\gamma+\delta)k - (i-1)(\gamma+\delta)]\mu \\ =& (k+1)(-2k+i-1)\mu^2 + (i-k-1)(\gamma+\delta)\mu \quad \text{for } k > 0. \end{aligned}$$

In particular, we have

$$h_i((i-1)\mu) = -i(i-1)\mu^2 < 0 \text{ and } h_i((i-1)\mu/2) = (i-1)(\gamma+\delta)\mu/2 > 0,\ i > 1,$$

which implies $c_i^* \in ((i-1)\mu/2, (i-1)\mu)$. □

Remark 3.8. The duration of movement, $1/c$, is about a few weeks or months, while the life span of an animal, $1/\mu$, could be a couple of years or even longer. Namely, the timescale of the movement is very short relative to the host population dynamic timescale. So generally speaking, $\mathcal{R}_{i0}$ is decreasing in c and shortening the duration of host movement could reduce the possibility of a disease spread.

Now we perform a sensitivity analysis of the basic reproduction number $\mathcal{R}_{i0}$ to model parameters to determine how best to reduce initial disease transmission. The normalized forward sensitivity index [6] of $\mathcal{R}_{i0}$ to a parameter p is defined as

$$\Upsilon_p^i = \frac{\partial \mathcal{R}_{i0}}{\partial p} \times \frac{p}{\mathcal{R}_{i0}}.$$

For $i = 1, 2, 3$, we find that $\Upsilon^i_{\alpha_i} = \Upsilon^i_{\beta_i} = \Upsilon^i_{M_i} = \Upsilon^i_r = \frac{1}{2}$, $\Upsilon^i_{\nu_i} = -\frac{1}{2}$, $\Upsilon^i_\gamma = -\frac{\gamma}{2(\mu+\gamma+\delta+c)} > -\frac{1}{2}$ and $\Upsilon^i_\delta = -\frac{\delta}{2(\mu+\gamma+\delta+c)} > -\frac{1}{2}$. In addition, if $c \gg \mu$ then

$$\Upsilon^i_\mu = -\frac{i(\mu+\gamma+\delta+c)\mu + (\mu+c)\mu}{2(\mu+c)(\mu+\gamma+\delta+c)} > -\frac{1}{2}$$

and

$$\Upsilon^i_c = -\frac{2c^2 + (\gamma+\delta+(3-i)\mu)c - (i-1)\mu(\mu+\gamma+\delta)}{2(\mu+c)(\mu+\gamma+\delta+c)} < -\frac{1}{2}.$$

So $\mathcal{R}_{i0}$ is most sensitive to the movement rate c.

4 Numerical simulations

In this section, we conduct numerical simulations to confirm our analytical results. The model uses a daily time step and some of the parameter values are chosen from the data in Gaff et al. [9] and the references therein.

Firstly, we explore the relation between $\mathcal{R}_{i0}$ and the travel rate c. We use the following set of parameter values: $r = 30, \mu = 1.2\times10^{-3}, \delta = 0.1, \gamma = 0.4, \zeta = 5\times10^{-3}, M_1 = 80, M_2 = 1000, M_3 = 100, \nu_i = 0.06, \alpha_i = 0.002$ and $\beta_i = 0.002$ for $i = 1, 2, 3$. Figure 1 shows how the basic reproduction number varies as a function of the livestock movement rate c, in the range $c \in [0, 0.5]$. As predicted by Proposition 3.7, the curve of $\mathcal{R}_{10}$ is constantly decreasing, and the curves of $\mathcal{R}_{20}$ and $\mathcal{R}_{30}$ are increasing for small c and then decreasing.

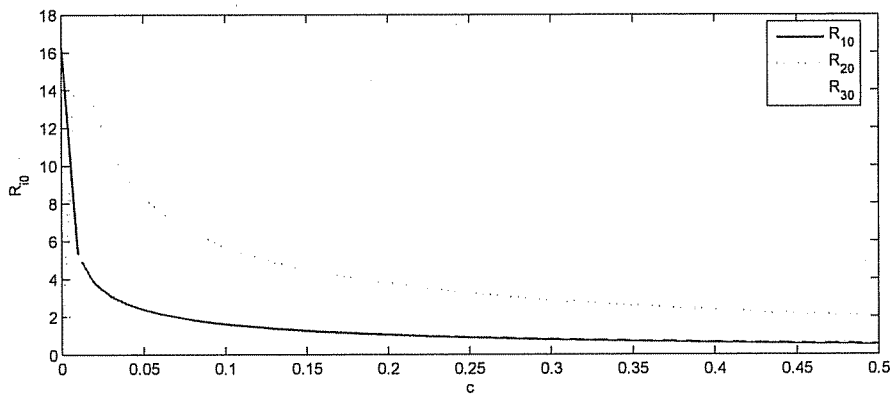

Figure 1: The curves of the basic reproduction number of patch i, $\mathcal{R}_{i0}$, versus c.

Now we fix c at 0.3 and the respective basic reproduction numbers are $\mathcal{R}_{10} = 0.8143 < 1, \mathcal{R}_{20} = 2.8731 > 1$ and $\mathcal{R}_{30} = 0.9067 < 1$. To consider a hypothetical disease invasion scenario, we set the initial data of patches 2 and 3 to zero such that there is no infected animals or mosquitoes in patches 2 and 3 at the beginning of travel. The disease dies out in patch 1, but persists in patches 2 and 3, which is coincident with Theorem 3.4 (see Figures

2 and 3). This may represent an interesting phenomenon regarding the role that animal movement plays in the spatial spread of RVF from Sudan to Egypt. Though the disease is introduced to patch 2 from patch 1, it goes extinct in its origin because of lower mosquito density in patch 1. Patch 2 (the Nile) has high mosquito population density and the disease will reach an endemic level once it appears. Patch 3 cannot sustain a disease alone, but this becomes possible because of continuous immigration of infected animals from patch 2.

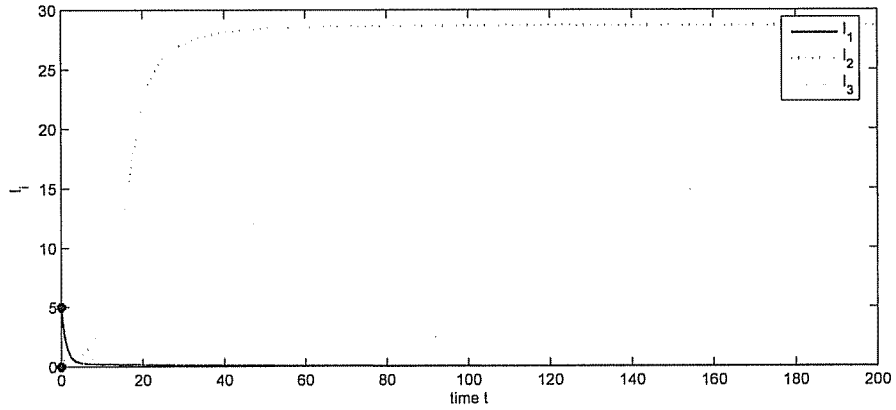

Figure 2: Numerical simulations of system (2.2a) showing I_i vs t. Initial conditions: $S_1(0) = 100, I_1(0) = 5, R_1(0) = 0, V_1(0) = 0$ and $S_2(0) = I_2(0) = R_2(0) = V_2(0) = S_3(0) = I_3(0) = R_3(0) = V_3(0) = 0$. $\mathcal{R}_{10} < 1, \mathcal{R}_{20} > 1$ and $\mathcal{R}_{30} > 1$.

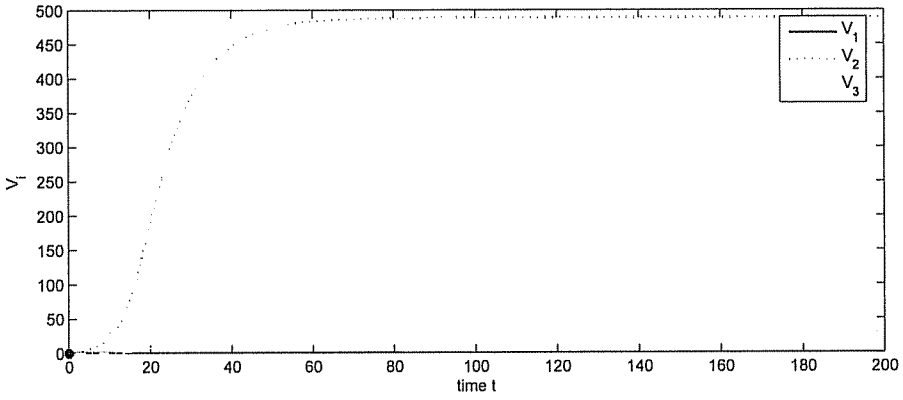

Figure 3: Numerical simulations of system (2.2a) showing V_i vs t. Initial conditions: $S_1(0) = 100, I_1(0) = 5, R_1(0) = 0, V_1(0) = 0$ and $S_2(0) = I_2(0) = R_2(0) = V_2(0) = S_3(0) = I_3(0) = R_3(0) = V_3(0) = 0$. $\mathcal{R}_{10} < 1, \mathcal{R}_{20} > 1$ and $\mathcal{R}_{30} > 1$.

5 Discussion

In this paper, we have formulated a simple epidemic patch model aimed at capturing a scenario where animals are imported into Egypt from the south and taken north along the Nile for human consumption, with the risk of a RVF outbreak if some of them are infected. A similar model might apply to Saudi Arabia and Yemen based on some descriptions [3]. We have evaluated the basic reproduction number for each patch and established the threshold dynamics of the model. It is suggested that a small number of imported infectious animals from Sudan could result in an outbreak of RVF in Egypt. Increasing the recruitment rate of animals, c, or the carrying capacity of mosquitoes, M_i, will increase the basic reproduction number, $\mathcal{R}_{i0}$. So the likelihood of a RVF outbreak is higher when both r and M_i are large. The rate r at which animals are fed in might be determined by demand, which would be large during Muslim festival periods. For example, millions of animals are imported and slaughtered as each devout Muslim must traditionally slaughter one animal during the celebration of Eid al-Adha (also known as the Feast of Sacrifice). The date of Eid al-Adha varies from year to year as it is linked to the Islamic calendar and more attention should be paid to the transmission of RVFV when the rainy season (more mosquitoes) corresponds to the time of the occurrence of festivals [3].

We may assume that some animals starting the journey are recovered. It might be that way even if no sick animals are starting the journey, since recovered ones could be healthy. If this happens, the subsystem (2.2a) will become

$$\begin{cases} \dfrac{dS_1}{dt} = r - \alpha_1 S_1 V_1 - \mu S_1 + \zeta R_1 - cS_1, \\ \dfrac{dI_1}{dt} = \alpha_1 S_1 V_1 - (\mu + \gamma + \delta) I_1 - cI_1, \\ \dfrac{dR_1}{dt} = r_R + \gamma I_1 - (\mu + \zeta) R_1 - cR_1, \\ \dfrac{dV_1}{dt} = -\nu_1 V_1 + \beta_1 I_1 (M_1 - V_1), \end{cases} \tag{5.1}$$

where r_R is a constant recruitment of recovered individuals into patch 1. Let $\tilde{R}_1 = R_1 - r_R/(\mu + \zeta + c)$ and $\tilde{r} = r + \zeta r_R/(\mu + \zeta + c)$. Then (5.1) can be written as

$$\begin{cases} \dfrac{dS_1}{dt} = \tilde{r} - \alpha_1 S_1 V_1 - \mu S_1 - cS_1, \\ \dfrac{dI_1}{dt} = \alpha_1 S_1 V_1 - (\mu + \gamma + \delta) I_1 - cI_1, \\ \dfrac{d\tilde{R}_1}{dt} = \gamma I_1 - (\mu + \zeta) \tilde{R}_1 - c\tilde{R}_1, \\ \dfrac{dV_1}{dt} = -\nu_1 V_1 + \beta_1 I_1 (M_1 - V_1), \end{cases} \tag{5.2}$$

which is qualitatively equivalent to (2.2a). Therefore, all of the aforementioned results still hold for system (5.2) or (5.1) and its associated new full system.

The work presented in this paper enables us to gain useful insights into the spread of RVF among different regions. However, there are other aspects we have not considered in

this study. Can we simplify our SIRS model to an SI/SIR model for hosts? Do we need more detailed epidemiological models, for example SEIR for hosts, SEI for vectors? We may want to think about extending the model to a larger and more realistic patch network, for example if we want to study how changing the network affects disease spread, but we would need to know at least something qualitative about movement patterns of herds to set the movement coefficients. Seasonal effects on mosquito population and time-dependence of animal importation may also be incorporated. Data for disease, vector and animal migration from RVF endemic regions need to be collected so that we can further test the validity of our model.

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