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AN EPIDEMIOLOGICAL MODEL OF RIFT VALLEY FEVER

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Abstract . We present and explore a novel mathematical model of the epi - demiology of Rift Valley Fever (RVF) . RVF is an Old World , mosquito - borne disease affecting both livestock and humans . The model is an ordinary dif - ferential equation model for two populations of mosquito species , those that can transmit vertically and those that cannot , and for one livestock popula - t ion . We analyze the model to find the stability of the disease - free equlibrium and test which model parameters affect this stability most significantly . This model is the basis for future research into the predication of future outbreaks in the Old World and the assessment of the threat of introduction into the New World .

1 . Introduction

Rift Valley fever virus (RVFV ; family : Bunyaviridae , genus *Phlebovirus*) is a mosquito - borne pathogen causing febrile i llness in domestic animals (e . g . , sheep , cattle , goats) and humans . Outbreaks of Rift Valley fever (RVF) are associated with widespread morbidity and mortality in livesto ck and morbidity in humans . Identified in Kenya in 1 930 [1] , RVF is often considered a disease primarily of sub - Saharan Africa , though outbreaks o ccurred in Egypt in 1 977 and 1 997 [2 , 3] . Recent translocation to Saudi Arabia and Yemen [4 , 5 , 6 , 7] demonstrate the ability of RVFV to invade ecologically diverse regions . The virus has never been observed in the Western Hemisphere , and it is feared that introduction could have signifi - cant deleterious impact on human and agricultural health . In light of the recent North American introduction and rapid spread of West Nile virus throughout the continent [8 , 9] , it seems prudent to develop a mathematical model that could en - able us to examine the potential dynamics of RVF should it appear in the Western Hemisphere [1 0] .

In Africa , the disease is spread by a number of mosquito species to livesto ck such as cattle , sheep and goats . Some of these mosquito species are infected only directly through feeding on infectious livesto ck , while others species also can be infected at birth by vertical transmission , i . e . , mother - to - offspring [1 1] . RVF in livestock will cause abortions in pregnant animals and mortality rates as high as 90% in young animals and 30% in adults [1 2] . While humans can be infected with RVF , we restrict our fo cus in this study to livesto ck populations .

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2 . The RVF model

We construct a compartmental , ordinary differential equation (ODE) model of RVFV transmission based on a simplification of the picture described above . The model considers two populations of mosquitoes (one exhibiting vertical transmission and the other not) and a population of livesto ck animals with disease - dependent mortality .

The model is depicted schematically in Figure 1 . One population of vectors represent *Aedes* mosquitoes (model population #1), which can be infected through either vertically or via a blood meal from an infectious host (model population #2). The other vector population is able to transmit RVFV to hosts but not to their offspring ; here we consider it to be a population of *Culex* mosquitoes (model popu - lation #3). Once infectious , mosquito vectors remain infectious for the remainder of their lifespan . Infection is assumed not to affect mosquito behavior or longevity significantly . Hosts , which represent various livesto ck animals , can become infected when fed upon by infectious vectors . Hosts may then die from RVFV infection or recover , whereupon they have lifelong immunity to reinfection [1 3] . Neither age structure nor spatial effects are incorporated into this model .

Populations contain a number of susceptible (S_i), incubating (infected , but not yet infectious)(E_i) and infectious (I_i) individuals , $i = 1, 2, 3$. Infected livesto ck will either die from RVFV or will recover with immunity (R_2). To reflect the vertical transmission in the *Aedes* species , compartments for uninfected (P_1) and infected ($Q1$) eggs are included . As the *Culex* species cannot transmit RVF vertically , only uninfected eggs (P_3) are included . Adult vectors emerge from these compartments at the appropriate maturation rates . The size of each adult mosquito population is $N_i = S_i + E_i + I_i$, for $i = 1$ and 3 . The livestock population is modeled using a logistic population model with a given carrying capacity , K_2. The total livesto ck population size is $N_2 = S_2 + E_2 + I_2 + R_2$.

The system of ODEs representing the populations is given below :

Aedes mosquito vectors

$$\frac{dP_1}{dt} = b_1(N_1 - q1^I1) - \theta_1 P_1$$

$$\frac{dQ1}{dt} = b_1 q1^I1 - \theta_1 Q1$$

$$\frac{dS_1}{dt} = \theta_1 P_1 - d_1 S_1 - \frac{\beta 21^S 1^I 2}{N_2}$$

$$\frac{dE_1}{dt} = -d_1 E_1 + \frac{\beta 21^S 1^I 2}{N_2} - \varepsilon_1 E_1$$

$$\frac{dI_1}{dt} = \theta_1 Q1 - d_1 I_1 + \varepsilon_1 E_1$$

$$\frac{dN_1}{dt} = (b_1 - d_1)N_1$$

Livestock hosts

$$\frac{dS_2}{dt} = b_2 N_2 - \frac{d_2 S_2 N_2}{K_2} - \frac{\beta 12^S 2^I 1}{N_1} - \frac{\beta 32^S 2^I 3}{N_3}$$

$$\frac{dE_2}{dt} = -\frac{d_2 E_2 N_2}{K_2} + \frac{\beta 12^S 2^I 1}{N_1} + \frac{\beta 32^S 2^I 3}{N_3} - \varepsilon_2 E_2$$

$$\frac{dI_2}{dt} = -\frac{d_2 I_2 N_2}{K_2} + \varepsilon_2 E_2 - \gamma 2^I 2 - \mu 2^I 2$$
$$\frac{dR_2}{dt} = -\frac{d_2 R_2 N_2}{K_2} + \gamma 2^I 2$$
$$\frac{dN_2}{dt} = N_2(b_2 - \frac{d_2 N_2}{K_2}) - \mu 2^I 2$$

Culex mosquito vectors

$$\frac{dP_3}{dt} = b_3 N_3 - \theta_3 P_3$$
$$\frac{dS_3}{dt} = \theta_3 P_3 - d_3 S_3 - \frac{\beta 23^S 3^I 2}{N_2}$$
$$\frac{dE_3}{dt} = -d_3 E_3 + \frac{\beta 23^S 3^I 2}{N_2} - \varepsilon_3 E_3$$
$$\frac{dI_3}{dt} = -d_3 I_3 + \varepsilon_3 E_3$$
$$\frac{dN_3}{dt} = (b_3 - d_3) N_3,$$

where :

$\beta 12$ = adequate contact rate : *Aedes* to livesto ck
$\beta 21$ = adequate contact rate : livesto ck to *Aedes*
$\beta 23$ = adequate contact rate : livesto ck to *Culex*
$\beta 32$ = adequate contact rate : *Culex* to livesto ck
$1/d_1$ = lifesp an of *Aedes* mosquitoes
$1/d_2$ = lifesp an of livesto ck animals
$1/d_3$ = lifesp an of *Culex* mosquitoes
b_1 = number of *Aedes* eggs laid per day
b_2 = daily birthrate in livesto ck
b_3 = number of *Culex* eggs laid per day
K_2 = carrying capacity of livesto ck
$1/\varepsilon_1$ = incubation period in *Aedes*
$1/\varepsilon_2$ = incubation period in livestock
$1/\varepsilon_3$ = incubation period in *Culex*
$1/\gamma 2$ = infectiousness period in livesto ck
$\mu 2$ = RVF mortality rate in livesto ck
$q1$ = transovarial transmission rate in *Aedes*
$1/\theta_1$ = development time of *Aedes*
$1/\theta_3$ = development time of *Culex* .

Approximate parameters values for the model are given in Table 1 . Since there are no direct measures for the adequate contact rates , these values are calculated as $\beta_{ij} = c_x f_x r_{ij}/gx$, where $x = i$ or $x = j$ and $i \neq j$ and x is a mosquito population . The value c_x is the feeding rate per gonotrophic cycle of mosquito population x, f_x is the probability that a mosquito of population x will feed on livesto ck , r_{ij} is the

rate of successful RVF transmission per bite from population i to j, and gx is the length of the gonotrophic cycle in days of mosquitoes in population x.

We analyzed the resulting model by computing the fundamental reproduction ratio and sensitivity of model output to variation or uncertainty in biological pa - rameters . Using numerical simulation based on parameter estimates obtained from the literature , we have investigated the expected vector and host species preva - lence in epidemic and endemic situations , as well as the expected risk of epidemic transmission of introduced into virgin areas .

3 . Stability Analysis

For epidemiology models , a quantity , R_0, is derived to assess the stability of the disease free equilibrium .R_0 represents the number of secondary cases that are caused by a single infectious case introduced into a completely susceptible popula - tion [1 4 , 1 5] . When $R_0 < 1$, if a disease is introduced , there are insufficient new cases per case , and the disease cannot invade the population . When $R_0 > 1$, the dis - ease may become endemic ; the greater R_0 is above 1 , the less likely sto chastic fade out of the disease is to o ccur . Unlike values of R_0 for strictly directly - transmitted diseases , the magnitude of the reproduction ratio does not necessarily scale in pro - portion to the intensity of epidemic / epizootic transmission .

It is possible to compute an analytical expression for the basic reproduction number , R_0, for this model by combining two previously published techniques [1 6 , 1 7] . Since the model incorporates both vertical and horizontal transmission , R_0 for the system is the sum of the R_0 values for each mode of transmission determined separately [1 6] ,

$$R_0 = R_{0,V} + R_{0,H}.$$

To compute each component of R_0, we express the model equations in vector form as the difference between the rate of new infection in compartment i, F_i, and the rate of transfer between compartment i and all other compartment due to other processes , V_i[17]. First , we calculate the basic reproduction number for the vertical transmission route , $R_{0,V}$. For this case , the only compartments involved are the infected eggs , exposed adults , and infectious adults of the *Aedes* population . Thus we have , in the notation of reference [1 7] ,

$$\frac{d}{dt}\begin{bmatrix} Q1 \\ E_1 \\ I_1 \end{bmatrix} = F_V - V_V = \begin{bmatrix} 0 \\ 0 \\ \theta_1 Q1 \end{bmatrix} - \begin{bmatrix} -b_1 q1^I 1 + \theta_1 Q1 \\ \varepsilon_1 E_1 + d_1 E_1 \\ -\varepsilon_1 E_1 + d_1 I_1 \end{bmatrix} .$$

The corresponding Jacobian matrices about the disease free equilibrium of the above system are

$$\mathbf{F}_V = \begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ \theta_1 & 0 & 0 \end{bmatrix} , \quad \mathbf{V}_V = \begin{bmatrix} \theta_1 & 0 & -b_1 q1 \\ 0 & d_1 + \varepsilon_1 & 0 \\ 0 & -\varepsilon_1 & d_1 \end{bmatrix} .$$

The basic reproduction number for vertical transmission is calculated as the spectral radius of the next generation matrix , $\mathbf{F_V V_V^{-1}}$,

$$R_{0,V} = \frac{b_1 q1}{d_1} .$$

Next , we calculate the horizontal transmission basic reproduction number , $R_{0,H}$. For this mode of transmission we must evaluate the exposed and infectious com - partments of the *Aedes* , *Culex* and livesto ck populations . Disease related mortality within the livesto ck population results in a non - constant livesto ck population size . To simplify the calculation of R_0, we transform our system to consider the per - cent of the population made up by each compartment , $x_i = \frac{X_i}{N_i}$, where X_i is a compartment of population i,

$$\frac{d}{dt}\begin{bmatrix} e_1 \\ i_1 \\ e_2 \\ i_2 \\ e_3 \\ i_3 \end{bmatrix} = F_H - V_H = \begin{bmatrix} \beta 21^s 1^i 2 \\ 0 \\ \beta 12^s 2^i 1 + \beta 32^s 2^i 3 \\ 0 \\ \beta 23^s 3^i 2 \\ 0 \end{bmatrix} - \begin{bmatrix} d_1 e_1 + \varepsilon_1 e_1 \\ d_1 i_1 - \varepsilon_1 e_1 \\ d_2 k_2 e_2 + \varepsilon_2 e_2 \\ -\varepsilon_2 e_2 + d_2 k_2 i_2 + \gamma 2^i 2 + \mu 2^i 2 \\ d_3 e_3 + \varepsilon_3 e_3 \\ d_3 i_3 - \varepsilon_3 e_3 \end{bmatrix},$$

where $k_2 \equiv \frac{N_2}{K_2}$. As before , we calculate the matrices F_H and V_H,

$$\mathbf{F}_H = \begin{bmatrix} 0 & 0 & 0 & \beta 21 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta 12 & 0 & 0 & 0 & \beta 32 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \beta 23 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix},$$

$$\mathbf{V}_H = \begin{bmatrix} d_1 + \varepsilon_1 & 0 & 0 & 0 & 0 & 0 \\ -\varepsilon_1 & d_1 & 0 & 0 & 0 & 0 \\ 0 & 0 & d_2 k_2 + \varepsilon_2 & 0 & 0 & 0 \\ 0 & 0 & -\varepsilon_2 & d_2 k_2 + \gamma 2 + \mu 2 & 0 & 0 \\ 0 & 0 & 0 & 0 & d_3 + \varepsilon_3 & 0 \\ 0 & 0 & 0 & 0 & -\varepsilon_3 & d_3 \end{bmatrix}.$$

The spectral radius of $\mathbf{F_H V_H^{-1}}$ results in ,

$$R_{0,H} = \sqrt{\frac{\varepsilon_2}{(d_2 k_2 + \varepsilon_2)(d_2 k_2 + \gamma 2 + \mu 2)}\left(\frac{\varepsilon_1 \beta 12 \beta 21}{d_1(d_1 + \varepsilon_1)} + \frac{\varepsilon_3 \beta 32 \beta 23}{d_3(d_3 + \varepsilon_3)}\right)}.$$

Thus , we get

$$R_0 = \frac{b_1 q 1}{d_1} + \sqrt{\frac{\varepsilon_2}{(d_2 k_2 + \varepsilon_2)(d_2 k_2 + \gamma 2 + \mu 2)}\left(\frac{\varepsilon_1 \beta 12 \beta 21}{d_1(d_1 + \varepsilon_1)} + \frac{\varepsilon_3 \beta 32 \beta 23}{d_3(d_3 + \varepsilon_3)}\right)}.$$

The first term in the sum corresponds to direct transmission , i . e . , RVFV travels vertically from *Aedes* to *Aedes* mosquito , whereas the second term corresponds to indirect (vector borne) transmission ; virus transport between vectors is mediated by mammalian hosts . This vector - host - vector viral transmission path is the nature of the square root [1 8 , 1 5] .

Biologically , we understand the expression for R_0 as follows : the $R_{0,V}$ corre - sponds to the product of the mean number of eggs laid over an average floodwater *Aedes* mosquito lifespan ($\frac{b_1}{d_1}$), and the fraction of those eggs that are infected with RVFV transovarially ($q1$). $R_{0,H}$ is comprised of two parts , corresponding to the *Aedes* - livesto ck interaction and the *Culex* - livesto ck interaction . The terms $\frac{\epsilon_j}{d_j + \epsilon_j}$ represent the probability of adult *Aedes* ($j = 1$) or *Culex* ($j = 3$) mosquitoes sur - viving through the extrinsic incubation period to the point where they can become

infectious . Similarly , the term $\frac{\epsilon_2}{d_2 k_2 + \epsilon_2}$ corresponds to the probability that livesto ck survive to the point where they are infectious . The $beta - line_{d_1}^{12}$ represents the mean number
of bites *Aedes* make throughout the course of their lifetimes , and similarly for $\frac{\beta 32}{d_3}$ in the case of *Culex* mosquitoes . Finally , the mean number of times a livesto ck animal is bitten by *Aedes* or *Culex* species during the t ime these vectors are infectious is $\frac{\beta 2j}{d_2 k_2 + \gamma 2^{+} \mu 2}$ for $j = 1$ and 3 , respectively .

4 . Model sensitivity analysis

Many of the parameters for this model cannot be estimated directly from exist - ing research . We employed the technique of Latin hypercube sampling to test the sensitivity of the model to each input parameter in an approach successfully applied in the past to many other disease models [1 9 , 20 , 2 1] . Latin hypercube sampling is a stratified sampling technique that creates sets of parameters by sampling for each parameter according to a predefined probability distribution . For each parameter , we assumed a uniform distribution across the ranges listed in Table 1 . We then
solved the system numerically using a large set ($n = 5000$) of sampled model pa - rameters . From these results , we calculated a variety of metrics of model sensitivity including R_0, maximum number of animals infected , time to reach that maximum and others , to assess the impact of each parameter on the model results . We used the partial rank correlation coefficient to assess the significance of each parameter with respect to each metric . The most significant parameters were found to be $\beta 12, \beta 21, \beta 23, \beta 32, ($ adequate contact rates $), \gamma ($ period of infectiousness in livesto ck $)$
and $d_3, d_1 ($ vector lifespan $)$ (Table 2) . Averaging R_0 over all parameter sets gives a mean of 1.19(95% confidence interval : 1 . 1 8 , 1 . 2 1) and a median of 1 . 1 1 (Figure 2) .
R_0 ranged from 0 . 37 to 3 . 743 .

5 . Numerical S imulations

To explore the behavior of RVF when introduced into a na ïve environment , we conducted numerical simulations of an isolated system (i . e . , no immigration or emigration) . The model uses a daily time step and is solved by a fourth order Runge - Kutta scheme . For each simulation , we start with 1 0 susceptible livesto ck animals , 1 0 *Culex* eggs , 999 *Aedes* susceptible eggs , 1 *Aedes* infected egg and 1 susceptible *Aedes* adult mosquito .

To assess the expected vector and host species prevalence in epidemic and en - demic situations , we ran four simulations . For the first two , we used a relatively high set of values for the adequate contact rates , β_{ij}, which would be appropriate
for settings where mosquitoes feed almost exclusively on the livesto ck population . The contact rate for the other simulations were lower , corresponding to settings where there are other suit able hosts for the mosquito , but these other hosts do not otherwise influence the dynamics of RVF . Each set of contact rates were used for a simulation using the higher RVF - associated mortality of sheep and a simulation using the lower RVF - associated mortality of cattle .

The percent of livesto ck infected through time , for specific simulations , are shown in Figure 3 . For these simulations , we define the " high set for β " as $\beta 12 = 0.48$ $\beta 21 = 0.395 \beta 23 = 0.56 \beta 32 = 0.13$, and " low set for β " as $\beta 12 = 0.15 \beta 21 = 0.15 \beta 23 = 0.15 \beta 32 = 0.5$. We also use a case fatality rate of 0 . 25 or 0 . 1 5 which gives us $\mu 2 = 0.312$ or $\mu 2 = 0.0176$, respectively . For simulations where

β_{ij} is high , the initial outbreaks were sufficiently large that it was necessary to break to y - axis to demonstrate subsequent outbreaks . Figure 3 (a) shows that with lower estimates for contact rates and the death rate associated with sheep , after an initial epidemic reaching a maximum of 0.5%, the disease dies out for all lifespans . Figure 3 (b) shows that with lower estimates for contact rates and the death rate associated with cattle , after an initial epidemic reaching a maximum under 0.13%, the disease remains endemic with multiple epidemics prior to a steady state infection level . The frequency of the subsequent epidemics reflects the turnover rate of the cattle population . Figure 3 (c) shows that with higher β_{ij} values and sheep fatality estimates , after an initial epidemic reaching over 10% infected , there are subsequent epidemics with the final endemic levels of between 0 . 1 and 0.4%. Figure 3 (d) shows that with higher β_{ij} values and cattle fatality estimates , after an initial epidemic reaching over 10% infected , there are subsequent epidemics with the final endemic levels of between 0 . 1 and 0.2%. In all cases , there is transmission following introduction , albeit at low levels in the case of the lower β values . For all but the lower β with sheep mortality cases , the disease attains a low level of endemic prevalence after a sequence of epidemics , suggesting the disease could persist if introduced into an isolated system .

6 . Conclusions

The model presented is a simplified representation of the complex biology in - volved in the epidemiology of RVF . As in all models , much of the value lays in the process of building the model , which forces researchers to carefully state the many assumptions they build their thinking upon [22] . Relaxation of model assumptions such as inclusion of age - structure or spatial variation may demonstrate additional insights . We hope this model and these results will act as a catalyst to further investigation .

Table 1 . Parameters with estimated ranges for numerical simulations

Table ignored!

TABLE 2 . Results of sensitivity testing using partial rank correlation coefficients . Results were comparable for all metrics ; only those for R_0 are shown .

Table ignored!

FIGURE 1 . Flow diagram of the Rift Valley Fever model

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FIGURE 2 . Distribution of R_0 values pooling a total of 5000 sets of parameters . The mean is 1.193(95% confidence interval : 1 . 1 77 , 1 . 209) and a median of 1 . 1 1 3 . The maximum value is 3 . 743 and then minimum 0 . 37 .

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(a) Lower β_{ij} and sheep fatality esti - mates

(b) Lower β_{ij} and cattle fatality esti - mates

(c) Higher β_{ij} and sheep fatality esti - mates

(d) Higher β_{ij} and cattle fatality esti - mates

FIGURE 3 . Results of numerical simulations for cattle and sheep . Livestock lifespan is indicated for 1 0 years (solid line) , 5 years (dashed line) and 2 years (dotted line) .

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