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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 epidemiology of Rift Valley Fever (RVF). RVF is an Old World, mosquito-borne disease affecting both livestock and humans. The model is an ordinary differential equation model for two populations of mosquito species, those that can transmit vertically and those that cannot, and for one livestock population. 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 illness in domestic animals (e.g., sheep, cattle, goats) and humans. Outbreaks of Rift Valley fever (RVF) are associated with widespread morbidity and mortality in livestock and morbidity in humans. Identified in Kenya in 1930 [1], RVF is often considered a disease primarily of sub-Saharan Africa, though outbreaks occurred in Egypt in 1977 and 1997 [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 significant 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 enable us to examine the potential dynamics of RVF should it appear in the Western Hemisphere [10].

In Africa, the disease is spread by a number of mosquito species to livestock such as cattle, sheep and goats. Some of these mosquito species are infected only directly through feeding on infectious livestock, while others species also can be infected at birth by vertical transmission, i.e., mother-to-offspring [11]. RVF in livestock will cause abortions in pregnant animals and mortality rates as high as 90% in young animals and 30% in adults [12]. While humans can be infected with RVF, we restrict our focus in this study to livestock 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 livestock 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 population #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 livestock 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 [13]. 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 livestock 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 (Q_1) 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 livestock 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

$$\begin{aligned}
\frac{dP_1}{dt} &= b_1 (N_1 - q_1 I_1) - \theta_1 P_1 \\
\frac{dQ_1}{dt} &= b_1 q_1 I_1 - \theta_1 Q_1 \\
\frac{dS_1}{dt} &= \theta_1 P_1 - d_1 S_1 - \frac{\beta_{21} S_1 I_2}{N_2} \\
\frac{dE_1}{dt} &= -d_1 E_1 + \frac{\beta_{21} S_1 I_2}{N_2} - \varepsilon_1 E_1 \\
\frac{dI_1}{dt} &= \theta_1 Q_1 - d_1 I_1 + \varepsilon_1 E_1 \\
\frac{dN_1}{dt} &= (b_1 - d_1) N_1
\end{aligned}$$

Livestock hosts

$$\begin{aligned}
\frac{dS_2}{dt} &= b_2 N_2 - \frac{d_2 S_2 N_2}{K_2} - \frac{\beta_{12} S_2 I_1}{N_1} - \frac{\beta_{32} S_2 I_3}{N_3} \\
\frac{dE_2}{dt} &= -\frac{d_2 E_2 N_2}{K_2} + \frac{\beta_{12} S_2 I_1}{N_1} + \frac{\beta_{32} S_2 I_3}{N_3} - \varepsilon_2 E_2
\end{aligned}$$

$$\frac{dI_2}{dt} = -\frac{d_2 I_2 N_2}{K_2} + \varepsilon_2 E_2 - \gamma_2 I_2 - \mu_2 I_2$$
$$\frac{dR_2}{dt} = -\frac{d_2 R_2 N_2}{K_2} + \gamma_2 I_2$$
$$\frac{dN_2}{dt} = N_2(b_2 - \frac{d_2 N_2}{K_2}) - \mu_2 I_2$$

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:

β_{12} = adequate contact rate: *Aedes* to livestock
β_{21} = adequate contact rate: livestock to *Aedes*
β_{23} = adequate contact rate: livestock to *Culex*
β_{32} = adequate contact rate: *Culex* to livestock
$1/d_1$ = lifespan of *Aedes* mosquitoes
$1/d_2$ = lifespan of livestock animals
$1/d_3$ = lifespan of *Culex* mosquitoes
b_1 = number of *Aedes* eggs laid per day
b_2 = daily birthrate in livestock
b_3 = number of *Culex* eggs laid per day
K_2 = carrying capacity of livestock
$1/\varepsilon_1$ = incubation period in *Aedes*
$1/\varepsilon_2$ = incubation period in livestock
$1/\varepsilon_3$ = incubation period in *Culex*
$1/\gamma_2$ = infectiousness period in livestock
μ_2 = RVF mortality rate in livestock
q_1 = 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 $\beta_{ij} = c_x f_x r_{ij}/g_x$, 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 livestock, r_{ij} is the

rate of successful RVF transmission per bite from population i to j, and g_x 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 parameters. Using numerical simulation based on parameter estimates obtained from the literature, we have investigated the expected vector and host species prevalence 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, $\mathscr{R}_0$, is derived to assess the stability of the disease free equilibrium. $\mathscr{R}_0$ represents the number of secondary cases that are caused by a single infectious case introduced into a completely susceptible population [14, 15]. When $\mathscr{R}_0 < 1$, if a disease is introduced, there are insufficient new cases per case, and the disease cannot invade the population. When $\mathscr{R}_0 > 1$, the disease may become endemic; the greater $\mathscr{R}_0$ is above 1, the less likely stochastic fade out of the disease is to occur. Unlike values of $\mathscr{R}_0$ for strictly directly-transmitted diseases, the magnitude of the reproduction ratio does not necessarily scale in proportion to the intensity of epidemic/epizootic transmission.

It is possible to compute an analytical expression for the basic reproduction number, $\mathscr{R}_0$, for this model by combining two previously published techniques [16, 17]. Since the model incorporates both vertical and horizontal transmission, $\mathscr{R}_0$ for the system is the sum of the $\mathscr{R}_0$ values for each mode of transmission determined separately [16],

$$\mathscr{R}_0 = \mathscr{R}_{0,V} + \mathscr{R}_{0,H}.$$

To compute each component of $\mathscr{R}_0$, we express the model equations in vector form as the difference between the rate of new infection in compartment i, $\mathscr{F}_i$, and the rate of transfer between compartment i and all other compartment due to other processes, $\mathscr{V}_i$ [17]. First, we calculate the basic reproduction number for the vertical transmission route, $\mathscr{R}_{0,V}$. 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 [17],

$$\frac{d}{dt}\begin{bmatrix} Q_1 \\ E_1 \\ I_1 \end{bmatrix} = \mathscr{F}_V - \mathscr{V}_V = \begin{bmatrix} 0 \\ 0 \\ \theta_1 Q_1 \end{bmatrix} - \begin{bmatrix} -b_1 q_1 I_1 + \theta_1 Q_1 \\ \varepsilon_1 E_1 + d_1 E_1 \\ -\varepsilon_1 E_1 + d_1 I_1 \end{bmatrix}.$$

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

$$\mathbf{F}_V = \begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ \theta_1 & 0 & 0 \end{bmatrix}, \quad \mathbf{V}_V = \begin{bmatrix} \theta_1 & 0 & -b_1 q_1 \\ 0 & d_1 + \varepsilon_1 & 0 \\ 0 & -\varepsilon_1 & d_1 \end{bmatrix}.$$

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

$$\mathscr{R}_{0,V} = \frac{b_1 q_1}{d_1}.$$

Next, we calculate the horizontal transmission basic reproduction number, $\mathscr{R}_{0,H}$. For this mode of transmission we must evaluate the exposed and infectious compartments of the *Aedes*, *Culex* and livestock populations. Disease related mortality within the livestock population results in a non-constant livestock population size. To simplify the calculation of $\mathscr{R}_0$, we transform our system to consider the percent of the population made up by each compartment, $x_i = \frac{X_i}{N_i}$, where X_i is a compartment of population i,

$$\frac{d}{dt}\begin{bmatrix} e_1 \\ i_1 \\ e_2 \\ i_2 \\ e_3 \\ i_3 \end{bmatrix} = \mathscr{F}_H - \mathscr{V}_H = \begin{bmatrix} \beta_{21} s_1 i_2 \\ 0 \\ \beta_{12} s_2 i_1 + \beta_{32} s_2 i_3 \\ 0 \\ \beta_{23} s_3 i_2 \\ 0 \end{bmatrix} - \begin{bmatrix} d_1 e_1 + \varepsilon_1 e_1 \\ d_1 i_1 - \varepsilon_1 e_1 \\ d_2 k_2 e_2 + \varepsilon_2 e_2 \\ -\varepsilon_2 e_2 + d_2 k_2 i_2 + \gamma_2 i_2 + \mu_2 i_2 \\ d_3 e_3 + \varepsilon_3 e_3 \\ d_3 i_3 - \varepsilon_3 e_3 \end{bmatrix},$$

where $k_2 \equiv \frac{N_2}{K_2}$. As before, we calculate the matrices $\mathscr{F}_H$ and $\mathscr{V}_H$,

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

$$\mathbf{V}_H = \begin{bmatrix} d_1 + \varepsilon_1 & 0 & 0 & 0 & 0 & 0 \\ -\varepsilon_1 & d_1 & 0 & 0 & 0 & 0 \\ 0 & 0 & d_2 k_2 + \varepsilon_2 & 0 & 0 & 0 \\ 0 & 0 & -\varepsilon_2 & d_2 k_2 + \gamma_2 + \mu_2 & 0 & 0 \\ 0 & 0 & 0 & 0 & d_3 + \varepsilon_3 & 0 \\ 0 & 0 & 0 & 0 & -\varepsilon_3 & d_3 \end{bmatrix}.$$

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

$$\mathscr{R}_{0,H} = \sqrt{\frac{\varepsilon_2}{(d_2 k_2 + \varepsilon_2)(d_2 k_2 + \gamma_2 + \mu_2)} \Big(\frac{\varepsilon_1 \beta_{12} \beta_{21}}{d_1 (d_1 + \varepsilon_1)} + \frac{\varepsilon_3 \beta_{32} \beta_{23}}{d_3 (d_3 + \varepsilon_3)} \Big)}.$$

Thus, we get

$$\mathscr{R}_0 = \frac{b_1 q_1}{d_1} + \sqrt{\frac{\varepsilon_2}{(d_2 k_2 + \varepsilon_2)(d_2 k_2 + \gamma_2 + \mu_2)} \Big(\frac{\varepsilon_1 \beta_{12} \beta_{21}}{d_1 (d_1 + \varepsilon_1)} + \frac{\varepsilon_3 \beta_{32} \beta_{23}}{d_3 (d_3 + \varepsilon_3)} \Big)}.$$

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 [18, 15].

Biologically, we understand the expression for $\mathscr{R}_0$ as follows: the $\mathscr{R}_{0,V}$ corresponds 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 (q_1). $\mathscr{R}_{0,H}$ is comprised of two parts, corresponding to the *Aedes*-livestock interaction and the *Culex*-livestock interaction. The terms $\frac{\epsilon_j}{d_j + \epsilon_j}$ represent the probability of adult *Aedes* ($j = 1$) or *Culex* ($j = 3$) mosquitoes surviving through the extrinsic incubation period to the point where they can become

infectious. Similarly, the term $\frac{\epsilon_2}{d_2k_2+\epsilon_2}$ corresponds to the probability that livestock survive to the point where they are infectious. The $\frac{\beta_{12}}{d_1}$ 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 livestock animal is bitten by *Aedes* or *Culex* species during the time these vectors are infectious is $\frac{\beta_{2j}}{d_2k_2+\gamma_2+\mu_2}$ for $j = 1$ and 3, respectively.

4. Model sensitivity analysis

Many of the parameters for this model cannot be estimated directly from existing 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 [19, 20, 21]. 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 parameters. From these results, we calculated a variety of metrics of model sensitivity including $\mathscr{R}_0$, 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), γ (period of infectiousness in livestock) and d_3, d_1 (vector lifespan) (Table 2). Averaging $\mathscr{R}_0$ over all parameter sets gives a mean of 1.19 (95% confidence interval: 1.18, 1.21) and a median of 1.11 (Figure 2). $\mathscr{R}_0$ ranged from 0.037 to 3.743.

5. Numerical Simulations

To explore the behavior of RVF when introduced into a naïve 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 1000 susceptible livestock animals, 1000 *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 endemic 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 livestock population. The contact rate for the other simulations were lower, corresponding to settings where there are other suitable 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 livestock 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 $\beta_{12} = 0.15$ $\beta_{21} = 0.15$ $\beta_{23} = 0.15$ $\beta_{32} = 0.05$. We also use a case fatality rate of 0.25 or 0.15 which gives us $\mu_2 = 0.0312$ 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 0.05%, 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 involved 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

Parameter	(Range)	Units	Reference
β_{12}	(0.0021, 0.2762)	1/day	[23, 24, 25, 26, 27, 28, 29]
β_{21}	(0.0021, 0.2429)	1/day	[23, 24, 25, 26, 30, 27, 31]
β_{23}	(0.0000, 0.3200)	1/day	[24, 25, 26, 30, 27, 31, 32]
β_{32}	(0.0000, 0.0960)	1/day	[24, 25, 26, 27, 32]
$1/d_1$	(3, 60)	days	[33, 34, 27]
$1/d_2$	(360, 3600)	days	[35]
$1/d_3$	(3, 60)	days	[33, 34, 27]
b_1	d_1	1/day	
b_2	d_2	1/day	
b_3	d_3	1/day	
$1/\varepsilon_1$	(4, 8)	days	[36]
$1/\varepsilon_2$	(1, 6)	days	[37]
$1/\varepsilon_3$	(4, 8)	days	[36]
$1/\gamma_2$	(1, 5)	days	[12]
μ_2	(0.025, 0.1)	1/day	[12, 37]
q_1	(0.0, 0.1)	—	[38]
$1/\theta_1$	(5, 15)	days	[27]
$1/\theta_3$	(5, 15)	days	[27]

TABLE 2. Results of sensitivity testing using partial rank correlation coefficients. Results were comparable for all metrics; only those for $\mathscr{R}_0$ are shown.

parameter	$\mathscr{R}_0$ PRCC	Significance
β_{12}	25.66	$p < 0.001$
β_{21}	26.28	$p < 0.001$
β_{32}	13.21	$p < 0.001$
β_{23}	14.52	$p < 0.001$
$1/\gamma_2$	-10.55	$p < 0.001$
$1/d_1$	-11.82	$p < 0.001$
$1/d_3$	-8.54	$p < 0.001$
μ_2	-2.42	$p < 0.02$

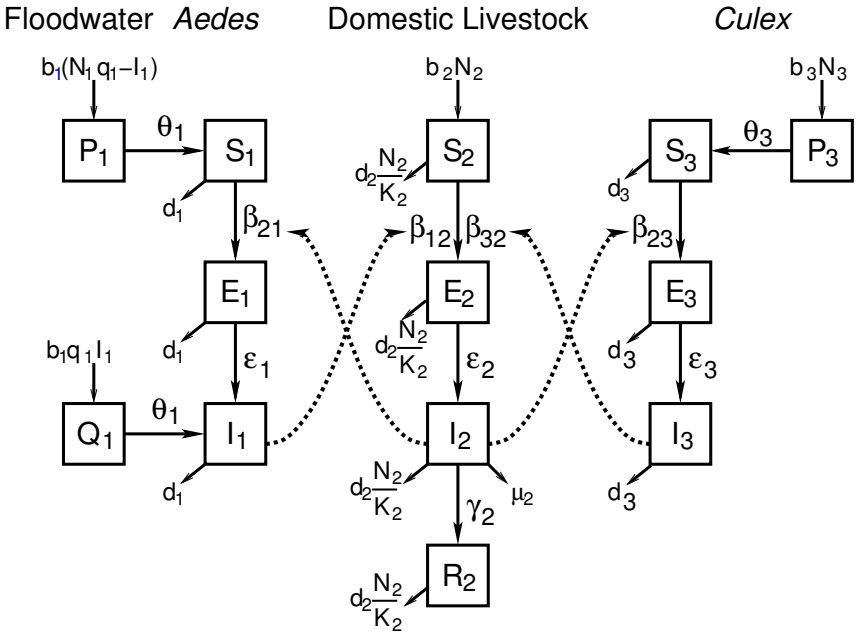

FIGURE 1. Flow diagram of the Rift Valley Fever model

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REFERENCES

[1] R. Daubney, J. R. Hudson, and P. C. Garnham. Enzootic hepatitis or Rift Valley fever: an undescribed virus disease of sheep, cattle and man from East Africa. *J. Pathol. Bacteriol.*, 34:545–579, 1931.

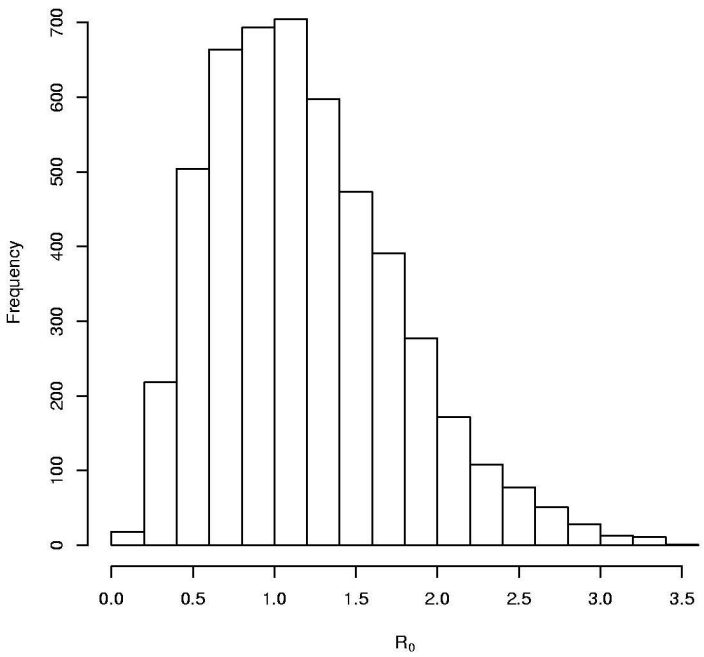


FIGURE 2. Distribution of $\mathscr{R}_0$ values pooling a total of 5000 sets of parameters. The mean is 1.193 (95% confidence interval: 1.177, 1.209) and a median of 1.113. The maximum value is 3.743 and then minimum 0.037.

[2] J. M. Meegan, R. H. Watten, and L. H. Laughlin. Clinical experience with Rift Valley fever in humans during the 1977 Egyptian epizootic. In T. A. Swartz, M. A. Klingberg, N. Goldblum, and C. M. Papier, editors, *Contr. Epidem. Biostatist.*, volume 3, pages 114–123, 1981.

[3] V. Chevalier, S. de la Rocque, T. Baldet, L. Vial, and F. Roger. Epidemiological processes involved in the emergence of vector-borne diseases: West nile fever, rift valley fever, japanese encephalitis and crimean-congo haemorrhagic fever. *Rev Sci Tech.*, 23(2):535–55, 2004.

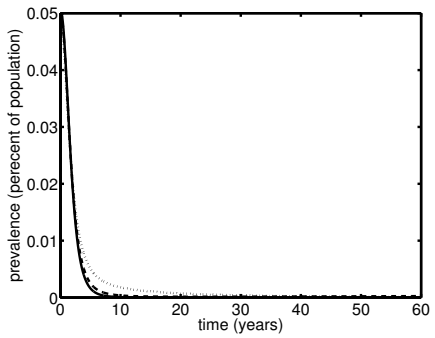
[4] P. G. Jupp, A. Kemp, A. Grobbelaar, P. Leman, F. J. Burt, A. M. Alahmed, D. Al Mujalli, M. Al Khamees, and R. Swanepoel. The 2000 epidemic of Rift Valley fever in Saudi Arabia: mosquito vector studies. *Med. Vet. Entomol.*, 16:245–252, 2002.

[5] A. I. Al-Afaleq, E. M. E. A. Elzein, S. M Mousa, and A. M. Abbas. A retrospective study of Rift Valley fever in Saudi Arabia. *Rev. Sci. Tech.*, 22(3):867–871, 2003.

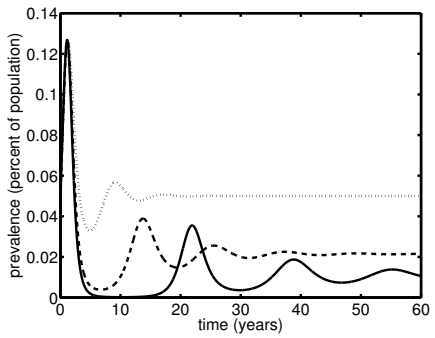
[6] M. Al-Hazmi, E. A. Ayoola an M. Abdurahman, S. Banzal, J. Ashraf, A. El-Bushra, A. Hazmi, M. Abdullah, H. Abbo, A. Elamin, E.-T. Al-Sammani, M. Gadour, C. Menon, M. Hamza, I. Rahim, M. Hafez, M. Jambavalikar, H. Arishi, and A. Aqeel. Epidemic Rift Valley fever in Saudi Arabia: A clinical study of severe illness in humans. *Clin. Infect. Dis.*, 36:245–52, 2003.

[7] T. A. Madani, Y. Y. Al-Mazrou, M. H. Al-Jeffri, A. A. Mishkhas, A. M. Al-Rabeah, A. M. Turkistani, M. O. Al-Sayed, A. A. Abodahish, A. S. Khan, T. G. Ksiazek, and O. Shobokshi. Rift Valley fever epidemic in Saudi Arabia: Epidemiological, clinical, and laboratory characteristics. *Clin. Infect. Dis.*, 37:1094–1092, 2003.

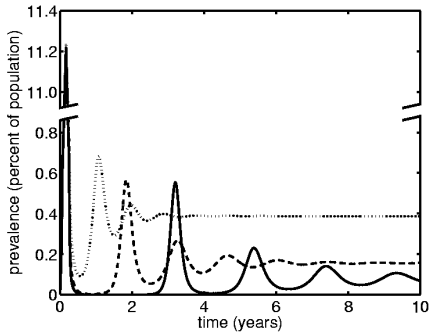
[8] D. J. Gubler. The global emergence/resurgence of aboviral diseases as public health problems. *Arch. Med. Res.*, 33:330–342, 2002.



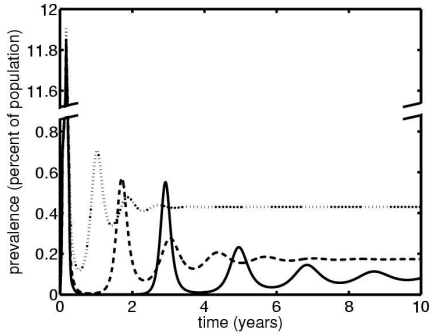
(a) Lower β_{ij} and sheep fatality estimates



(b) Lower β_{ij} and cattle fatality estimates



(c) Higher β_{ij} and sheep fatality estimates



(d) Higher β_{ij} and cattle fatality estimates

FIGURE 3. Results of numerical simulations for cattle and sheep. Livestock lifespan is indicated for 10 years (solid line), 5 years (dashed line) and 2 years (dotted line).

[9] J. H. Rappole, S. R. Derrickson, and Z. Hubálek. Migratory birds and spread of West Nile virus in the Western Hemisphere. *Emerg. Infect. Diseases*, 6(4):319–328, 2000.

[10] J. A. House, M. J. Turell, and C. A. Mebus. Rift Valley fever: Present status and risk to the Western Hemisphere. *Ann. N. Y. Acad. Sci.*, 653:233–242, 1992.

[11] C. J. Peters. Emergence of Rift Valley fever. In J. F. Salazzo and B. Bodet, editors, *Factors in the Emergence of Arbovirus Diseases*, pages 253–263. Elsevier, 1997.

[12] B. J. Erasmus and J. A. W. Coetzer. The symptomatology and pathology of Rift Valley fever in domestic animals. In T. A. Swartz, M. A. Klingberg, N. Goldblum, and C. M. Papier, editors, *Contr. Epidem. Biostatist.*, volume 3, pages 77–82, 1981.

[13] M. L. Wilson. Rift Valley fever virus ecology and the epidemiology of disease emergence. *Ann. N. Y. Acad. Sci.*, 740:169–180, 1994.

[14] R. M. Anderson and R. M. May. *Infectious Diseases of Humans: Dynamics and Control*. Oxford University Press, Oxford, 1991.

[15] J. M. Heffernan, R. J. Smith, and L. M. Wahl. Perspectives on the basic reproductive ratio. *J. R. Soc. Interface*, 2:281–293, 2005.

[16] M. Lipsitch, M. A. Nowak, D. Ebert, and R. M. May. The population dynamics of vertically and horizontally transmitted parasites. *Proc. R. Soc. B*, 260:321–327, 1995.
[17] P. van den Driessche and J. Watmough. Reproduction numbers and sub-threshold endimic equilibria for compartmental models of disease transmission. *Math. Biosci.*, 180:29–48, 2002.
[18] O. Diekmann, H. Heesterbeek, and H. Metz. The legacy of Kermack and McKendrick. In D. Mollison, editor, *Epidemic Models: Their Structure and Relation to Data*, pages 95–115. Cambridge University Press, Cambridge, UK, 1995.
[19] S. M. Blower and H. Dowlatabadi. Sensistivity and uncertainty analysis of complex models of disease transmission: an HIV model, as an example. *Int. Stat. Rev.*, 62(2):229–243, 1994.
[20] M. A. Sanchez and S. M. Blower. Uncertainty and sensitivity analysis of the basic reproductive rate: Tuberculosis as an example. *Am. J. Epidemiol.*, 145(12):1127–1137, 1997.
[21] David Gammack, Jose L. Segovia-Juarez, Suman Ganguli, Simeone Marino, and Denise Kirschner. Understanding the immune response in tuberculosis using different mathematical models and biological scales. *SIAM Journal of Multiscale Modeling and Simulation*, 3(2):312–345, 2005.
[22] F. E. McKenzie. Why model malaria? *Parasitol. Today*, 16(12):511–516, 2000.
[23] D. V. Canyon, J. L. K. Hii, and R. Muller. The frequency of host biting and its effect on oviposition and survival in *Aedes aegypti* (Diptera: Culicidae). *Bull. Entomol. Res.*, 89(1):35–39, 1999.
[24] R. O. Hayes, C. H. Tempelis, A. D. Hess, and W. C. Reeves. Mosquito host preference studies in Hale County, Texas. *Am. J. Trop. Med. Hyg.*, 22(2):270–277, 1973.
[25] C. J. Jones and J. E. Lloyd. Mosquitoes feeding on sheep in southeastern Wyoming. *J. Am. Mosq. Control Assoc.*, 1(4):530–532, 1985.
[26] L. A. Magnarelli. Host feeding patterns of Connecticut mosquitoes (Diptera: Culicidae). *Am. J. Trop. Med. Hyg.*, 26(3):547–552, 1997.
[27] H. D. Pratt and C. G. Moore. *Vector-Borne Disease Control: Mosquitoes, Of Public Health Importance And Their Control*. U.S. Department of Health and Human Services, Atlanta, GA, 1993.
[28] M. J. Turell, C. L. Bailey, and J. R. Beaman. Vector competence of a Houston, Texas strain of *Aedes albopictus* for Rift Valley fever virus. *J. Am. Mosq. Control Assoc.*, 4(1):94–96, 1988.
[29] M. J. Turell, M. E. Faran, M. Cornet, and C. L. Bailey. Vector competence of Senegalese *Aedes fowleri* (Diptera:Culicidae) for Rift Valley fever virus. *J. Med. Entomol.*, 25(4):262–266, 1988.
[30] B. M. McIntosh and P. G. Jupp. Epidemiological aspects of Rift Valley fever in south aftrica with reference to vectors. In T. A. Swartz, M. A. Klingberg, N. Goldblum, and C. M. Papier, editors, *Contr. Epidem. Biostatist.*, volume 3, pages 92–99, 1981.
[31] M. J. Turell and C. L. Bailey. Transmission studies in mosquitoes (Diptera:Culicidae) with disseminated Rift Valley fever virus infections. *J. Med. Entomol.*, 24(1):11–18, January 1987.
[32] J. W. Wekesa, B. Yuval, and R. K. Washino. Multiple blood feeding by *Anopheles freeborni* and *Culex tarsalis* (Diptera:Culicidea): Spatial and temporal variation. *J. Med. Entomol.*, 34(2):219–225, 1997.
[33] M. Bates. *The Natural History of Mosquitoes*. Peter Smith, Gloucester, MA, 1970.
[34] C. G. Moore, R. G. McLean, C. J. Mitchell, R. S. Nasci, T. F. Tsai, C. H. Caslisher, A. A. Marfin, P. S. Moorse, and D. J. Gubler. *Guidelines for Arbovirus Surveillance Programs in the United Sates*. Center for Disease Control and Prevention, April 1993.
[35] O. M. Radostits. *Herd Healthy: Food Animal Production Medicine*. W. B. Saunders Company, Philidelphia, PA, third edition, 2001.
[36] M. J. Turell and B. H. Kay. Susceptibility of slected strains of Australian mosquitoes (Diptera: Culicidae) to Rift Valley fever virus. *J. Med. Entomol.*, 35(2):132–135, 1998.
[37] C. J. Peters and K. J. Linthicum. Rift Valley fever. In G. W. Beran, editor, *Handbook of Zoonoses*, B: Viral, pages 125–138. CRC Press, second edition, 1994.
[38] J. E. Freier and L. Rosen. Verticle transmission of dengue virus by the mosquitoes of the *Aedes scutellaris* group. *Am. J. Trop. Med. Hyg.*, 37(3):640–647, 1987.

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