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Multiseason transmission for Rift Valley fever in North America

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ABSTRACT

Rift Valley fever is a vector-borne disease, primarily found in West Africa, that is transmitted to humans and domestic livestock. Its similarities to the West Nile virus suggest that establishment in the developed world may be possible. Rift Valley fever has the potential to invade North America, where seasons play a role in disease persistence. The values for the basic reproductive number show that, in order to eradicate the disease, the survival time of mosquitoes must decrease below 8.67 days. Mechanisms such as aggressive spraying that decreases the mosquito population can contain an outbreak. Otherwise, Rift Valley fever is likely to establish itself as a recurring seasonal outbreak. Rift Valley fever poses a potential threat to North America that would require aggressive interventions in order to prevent a recurring seasonal outbreak.

KEYWORDS

Impulsive differential equations; mathematical model; Rift Valley fever; seasons; spraying

1. Introduction

The emergence of West Nile virus in North America has drawn attention to the possibility for other mosquito-borne pathogens to expand their natural ranges (Morse, 1995; Patz et al., 2005; Favier et al., 2006; Moutailler et al., 2008; Turell, Dohm et al., 2008). The West Nile virus first appeared in the United States in 1999, causing acute illness in 62 individuals, 7 of whom died (Roche, 2002). Outbreaks of encephalitis caused by the West Nile virus have occurred in the late summer and early autumn months yearly in New York City since 1999 (Karpati et al., 2004). By the end of 2002, West Nile virus activity had been reported in all but four continental U.S. states, with more than 3,500 human cases reported (Mostashari et al., 2003).

Of the many potential mosquito-borne pathogens able to invade North America like the West Nile virus, one particular concern is Rift Valley fever, an arthropod-borne viral zoonosis, which has seen an expansion in its geographic range and virulence since its formal identification in Kenya's Rift

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Valley in 1931 (House et al., 1992; Favier et al., 2006; Moutailler et al., 2008; Turell, Dohm et al., 2008). Incidence of Rift Valley fever is believed to have a particularly strong connection to climate variation and has been linked with El Niño-southern oscillation events or incidents of localized heavy rainfall (House et al., 1992; Traoré-Lamizana et al., 2001; Porphyre et al., 2005; Despommiers et al., 2007; Anyamba et al., 2009). Prior to 1977, incidents of Rift Valley fever had primarily been concentrated in sub-Saharan Africa and were mainly a concern of the agricultural industry. Infection was fatal in young domestic ruminants and initiated abortion in pregnant animals, while cases were mild in humans (Meegan, 1980; House et al., 1992; Moutailler et al., 2008; Turell, Dohm et al., 2008). In 1977, Rift Valley fever's potential to spread beyond its endemic range in sub-Saharan Africa was confirmed when it appeared without apparent precedent in Egypt. Unlike previously documented outbreaks, human infection exceeded 200,000 cases, 600 of which were fatal (Meegan, 1980; House et al., 1992; Traoré-Lamizana et al., 2001; Favier et al., 2006; Gaff et al., 2007). Since then, Rift Valley fever has become endemic in previously unexposed areas of Western and Eastern Africa. It reached the Arabian Peninsula in the early 21st century (Gerdes, 2004). Rift Valley fever's success at establishing its endemicity in novel environments, with a wide variety of arthropods capable of acting as its vectors, constitutes a serious threat (House et al., 1992; Turell, Dohm et al., 2008; Turell, Linthicum et al., 2008).

Rift Valley fever is an arthropod-borne viral zoonosis: it is transmitted between humans and animals by an arthropod vector (House et al., 1992; Meegan, 1980; Gerdes, 2004). Rift Valley fever can infect a fairly wide range of vertebrate hosts and has an even wider range of insect vectors: more than 40 species of mosquitoes from 8 different genera have been isolated in the field (Meegan, 1980; House et al., 1992; Gerdes, 2004; Turell, Dohm et al., 2008; Turell, Linthicum et al., 2008). Rift Valley fever's success at establishing its endemicity in novel environments is due in part to its flexibility in both hosts and vectors (Balkhy and Memish, 2003). Mosquitoes belonging to the Aedes species are an important vector since they may vertically transmit infection to their young through transovarial transmission, enabling the maintenance of Rift Valley fever during inter-epizootic periods (House et al., 1992; Traoré-Lamizana et al., 2001; Bowman et al., 2005). Culex species and those belonging to Eretmapodites are believed to be strongly involved with epizootic outbreaks (House et al., 1992; Traoré-Lamizana et al., 2001; Bowman et al., 2005). Both the Aedes and Culex species are known to transmit the West Nile virus. Rift Valley fever can also be transmitted through aerosol exposure from handling infected carcasses. Laboratory workers, veterinarians, and individuals involved with meat processing are particularly vulnerable to this form of infection (House et al., 1992; Traoré-Lamizana et al., 2001; Moutailler et al., 2008; Turell, Dohm et al., 2008; Turell, Linthicum et al., 2008).

Rift Valley fever has a very wide range of viable vertebrate hosts, as might be expected, considering the diversity of its vectors (Balkhy and Memish, 2003). Rift Valley fever's most significant host species are domestic ruminants such as sheep and cattle (House et al., 1992; Balkhy and Memish, 2003; Turell, Dohm et al., 2008; Turell, Linthicum et al., 2008). Newborn lambs and goats are most susceptible to disease, followed by calves and sheep. Moderate disease occurs in adult cattle, sheep, goats, humans, water buffalo, and rats. Humans and ruminants are capable of developing enough viremia to infect mosquitoes. Camels, horses, pigs, cats, dogs, guinea pigs, rabbits, hedgehogs, and monkeys are susceptible but do not necessarily display clinical disease. Birds, with the exception of pigeons and chickens, and reptiles are believed to be resistant to infection (House et al., 1992; Gerdes, 2004). Balkhy and Memish (2003) showed that fatality in adult cattle and sheep can be as high as 30%, while young animals may experience 100% fatality. Gerdes (2004) showed that the disease is most significant in young animals, particularly lambs and goats, which may experience 70–100% mortality. Newborn animals experience 100% fatality, and most epidemics are defined by high incidences of abortion (Gerdes, 2004). Prior to the Egyptian outbreak in 1977, the disease in humans had mostly been asymptomatic or mild, often manifesting as influenza-like symptoms. Complications such as encephalitis may occur, and fewer than 1% of cases may present hemorrhagic fever (House et al., 1992; Balkhy and Memish, 2003; Gerdes, 2004; Turell, Dohm et al., 2008).

In order for a vector-borne emerging infectious disease to become endemic in a new location, there must be enough susceptible hosts and vectors (House et al., 1992). Due to Rift Valley fever's large range of viable hosts and vectors, its potential to establish itself is high compared to other vector-borne diseases. Gaff et al. (2007) modeled Rift Valley fever transmission using ordinary differential equations for two populations of mosquito species — those that can transmit vertically and those that cannot — and one population of domestic livestock animals with disease-dependent mortality. They found the stability of the disease-free equilibrium and identified parameters affecting the stability. They showed that, for any given contact rate, there is a low level of endemic prevalence, which implies that the disease could persist if introduced into an isolated system.

Bicout and Sabatier (2004) and Zell (2004) demonstrated that the incidence of many vector-borne infectious diseases shows seasonality, and extreme weather events are often accompanied by additional outbreaks. Favier et al. (2006) assessed the possibility of endemicity without wild animals providing a permanent virus reservoir. Using a deterministic model, endemicity without a permanent virus reservoir is impossible in a single site except when there is a strictly periodic rainfall pattern, but it is possible when there are herd movements and sufficient inter-site variability in rainfall, which drives mosquito emergence.

2. The model

We use a compartmental model derived from Gaff et al. (2007) with three populations: the human, domestic livestock, and mosquito populations (Figure 1). The model has four different classes: the S classes are susceptible individuals, the E classes are infected but non-infectious individuals, the I classes are infectious individuals, and the R classes are recovered and immune individuals. When initially infected, the individual is non-infectious for a while; only individuals in class I can infect the susceptible populations. Once recovered from the disease, the individual has lifelong immunity.

The human population is described by

$$\begin{cases} S'_H(t) = \Lambda_H - d_H S_H(t) - \beta_{LH} \frac{S_H(t) I_L(t)}{N_L(t)} - \beta_{MH} S_H(t) I_M(t) \\ E'_H(t) = \beta_{LH} \frac{S_H(t) I_L(t)}{N_L(t)} + \beta_{MH} S_H(t) I_M(t) - d_H E_H(t) - \varepsilon_H E_H(t) \\ I'_H(t) = \varepsilon_H E_H(t) - d_H I_H(t) - \mu_H I_H(t) - \gamma_H I_H(t) \\ R'_H(t) = \gamma_H I_H(t) - d_H R_H(t), \end{cases} \tag{1}$$

where

$$N_L(t) = S_L(t) + E_L(t) + I_L(t) + R_L(t).$$

The domestic livestock population is described by

$$\begin{cases} S'_L(t) = \Lambda_L - d_L S_L(t) - \beta_{ML} S_L(t) I_M(t) \\ E'_L(t) = \beta_{ML} S_L(t) I_M(t) - d_L E_L(t) - \varepsilon_L E_L(t) \\ I'_L(t) = \varepsilon_L E_L(t) - d_L I_L(t) - \mu_L I_L(t) - \gamma_L I_L(t) \\ R'_L(t) = \gamma_L I_L(t) - d_L R_L(t). \end{cases} \tag{2}$$

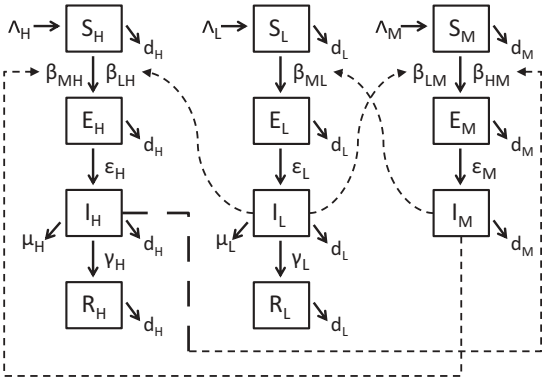

Figure 1. The model. The index "H" refers to human, "L" to livestock, and "M" to mosquitoes. S_H, S_L, and S_M are the susceptible populations, E_H, E_L, and E_M are the exposed populations, I_H, I_L, and I_M are the infected populations, and R_H and R_L are the recovered populations (mosquitoes do not recover from the disease). Other parameters are listed in Table 2.

The Aedes mosquito population is described by

$$\begin{cases} S'_M(t) = \Lambda_M - d_M S_M(t) - \beta_{LM} S_M(t) I_L(t) - \beta_{HM} S_M(t) I_H(t) \\ E'_M(t) = \beta_{LM} S_M(t) I_L(t) + \beta_{HM} S_M(t) I_H(t) - d_M E_M(t) - \varepsilon_M E_M(t) \\ I'_M(t) = \varepsilon_M E_M(t) - d_M I_M(t). \end{cases} \tag{3}$$

Each population has infection rate β_j $(j = H, L, M)$. The natural death rates are d_j, the disease-induced death rates μ_j, the rates at which a noninfectious individual becomes infectious ε_j, and the recovery rates γ_j. The mosquito population contains no R class, because mosquitoes never clear the infection once they are infected. A summary of the parameters and units are given in Table 1.

The birth rates in the system are constant sources of susceptible individuals Λ_i for $i = H, L, M$; in the absence of disease, the susceptible populations converge to the disease-free equilibrium at an exponential rate. We also include fetal death caused by Rift Valley fever infection in the domestic livestock population, which is not included in Gaff et al. (2007). Human and livestock populations are unlikely to be well-mixed (meaning that each infected individual has equal chance of infecting every susceptible individual), so we use standard incidence for transmission. All other terms, including the interactions between mosquitoes and hosts, are mass-action because the mosquito and host populations are assumed to be well-mixed. The spread of Rift Valley fever is much more likely between mosquitoes and hosts than between hosts because the interaction between host and mosquito populations is higher in a given area than host to host interaction.

Table 1. Definition of parameters.

Parameter	Units	Definition
Λ_H	population × days^{-1}	Human birth rate
Λ_L	population × days^{-1}	Immigration of livestock
Λ_M	population × days^{-1}	Mosquito birth rate
$1/d_H$	days	Human survival time
$1/d_L$	days	Livestock survival time
$1/d_M$	days	Mosquito survival time
μ_H	days^{-1}	Disease death rate for humans
μ_L	days^{-1}	Disease death rate for livestock
β_{MH}	(days × population)$^{-1}$	Infection rate from mosquitoes to humans
β_{LH}	(days × population)$^{-1}$	Infection rate from livestock to humans
β_{ML}	(days × population)$^{-1}$	Infection rate from mosquitoes to livestock
β_{LM}	(days × population)$^{-1}$	Infection rate from livestock to mosquitoes
β_{HM}	(days × population)$^{-1}$	Infection rate from humans to mosquitoes
$1/\varepsilon_H$	days	Human incubation time
$1/\varepsilon_L$	days	Livestock incubation time
$1/\varepsilon_M$	days	Mosquito incubation time
$1/\gamma_H$	days	Human recovery time
$1/\gamma_L$	days	Livestock recovery time

3. Asymptotic behavior: One-season model

The model has two equilibria: the disease-free equilibrium and the endemic equilibrium (developed in the Appendix). Several solutions could exist for the endemic equilibrium, resulting in a backward bifurcation. If this is the case, then lowering the basic reproductive number below 1 may no longer be sufficient for control (Li et al., 2011). This presents a serious complication when a disease is already endemic because the outcome depends on initial conditions. In the case of Rift Valley fever, we only consider sufficiently small perturbations away from the disease-free equilibrium because the disease is present in North America. This contrasts with the situation in Africa, where the disease is already established, so more rigorous control efforts would be required.

The Jacobian matrix is computed at the disease-free equilibrium (expressed in the Appendix). For fixed parameters and state variables, the stability changes as the mosquito death rate d_M varies. For the values used in Table 2, the Routh–Hurwitz conditions are satisfied for the characteristic equation

$$f(\alpha) = \alpha^6 + c_1\alpha^5 + c_2\alpha^4 + c_3\alpha^3 + c_4\alpha^2 + c_5\alpha + c_6, \tag{4}$$

when $0.15 < d_M < 0.68$. The disease-free equilibrium is locally stable in this region.

Table 2. Range of parameters.

Parameter	Sample Value	Range	Units	References
Λ_H	$1000 \times d_H$	$1000 \times d_H$	people days^{-1}	(Bowman et al., 2005)
Λ_L	0.1	0.01–0.5	livestock days^{-1}	Assumed
Λ_M	$10000 \times (1/14)$	200–1000	mosquitoes days^{-1}	(Gaff et al., 2007)
$1/d_H$	80×365	70–90	days	(Bowman et al., 2005)
$1/d_L$	20×365	10–25	days	(Gaff et al., 2007)
$1/d_M$	varies	3–60	days	(Gaff et al., 2007)
μ_H	0.01	0.01–0.1	days^{-1}	(Directors of Health Promotion and Education, 2013)
μ_L	1/25	1/10–1/40	days^{-1}	(Gaff et al., 2007)
β_{MH}	0.2762/1000	0.2762/10000–0.2762/100	days^{-1}	(Bowman et al., 2005)
β_{LH}	10^{-5}	10^{-5}–10^{-3}	days^{-1}	(Xue et al., 2012)
β_{ML}	β_{MH}	β_{MH}	days^{-1}	(Gaff et al., 2007)
β_{LM}	$\beta_{ML}/8$	$\beta_{ML}/8$	days^{-1}	(Gaff et al., 2007)
β_{HM}	$\beta_{MH}/10$	$\beta_{MH}/10$	days^{-1}	(Xue et al., 2012)
$1/\varepsilon_H$	5	4–6	days	(Bowman et al., 2005), (CDC, 2003)
$1/\varepsilon_L$	2	1–3	days	(Gaff et al., 2007), (CDC, 2003)
$1/\varepsilon_M$	6	4–8	days	(Gaff et al., 2007)
$1/\gamma_H$	5	2–7	days	(Bowman et al., 2005), (CDC, 2003)
$1/\gamma_L$	3	1–5	days	(Gaff et al., 2007)

We calculated R_0, the average number of secondary infections that any single infected individual will cause by using the next-generation method (van den Driessche and Watmough, 2002). The solution (developed in the Appendix) to the characteristic polynomial gives a cubic equation with one real solution and two complex conjugates. The value for R_0 is the largest modulus of our eigenvalues (Greenhalgh, 1996).

4. Multiple-season model

4.1. *Impulse conditions*

In most of northern America, the mosquito population drops during the winter months. This fact is taken into account though impulsive differential equations (Lakshmikantham et al., 1989; Bainov and Simeonov, 1989, 1993, 1995). At impulse times t_k:

$$\Delta x = x\left(t_k^+\right) - x\left(t_k^-\right) = f\left(t_k, x\left(t_k^-\right)\right), \tag{5}$$

where $f(t, x)$ maps the solution before the impulse, $x\left(t_k^-\right)$, to $x\left(t_k^+\right)$. We reset the mosquito, human, and domestic livestock populations at the beginning of each summer.

We consider two seasons: the summer season, when the mosquitoes will infect hosts, and the winter season, when the mosquitoes die from the cold weather. During the winter, there is no vector to spread the disease; the domestic livestock and human populations increase, as abortions no longer occur.

The mosquitoes decrease to zero with the cold but reappear at the beginning of summer. The Aedes mosquito has very few offspring surviving through the winter (Dohm et al., 2002). We assume that all adult mosquitoes die during winter.

At time t_k,

$$\begin{cases} \Delta S_H = r_1 S_H^- + p_1\left(E_H^- + I_H^- + R_H^-\right) \\ \Delta E_H = -E_H^- \\ \Delta I_H = -I_H^- \\ \Delta R_H = E_H^- + I_H^- - p_1\left(E_H^- + I_H^- + R_H^-\right), \end{cases} \tag{6}$$

where S_H, E_H, I_H and R_H are the susceptible, exposed, infected, and recovered human populations. The susceptible population increases by a fraction r_1. Because the infection period is short, by the end of winter, the total infected population has recovered so that $I_H = 0$. A fraction p_1 of the recovered offspring is susceptible.

At time t_k,

$$\begin{cases} \Delta S_L = r_2 S_L^- + c + p_2\left(E_L^- + I_L^- + R_L^-\right) \\ \Delta E_L = -E_L^- \\ \Delta I_L = -I_L^- \\ \Delta R_L = E_L^- + I_L^- - p_2\left(E_L^- + I_L^- + R_L^-\right), \end{cases} \tag{7}$$

where S_L, E_L, I_L, and R_L are the susceptible, exposed, infected, and recovered domestic livestock populations, respectively. Because infection causes death in livestock, the susceptible livestock increases by a fixed amount c, reflecting the purchase of livestock for breeding.

The opposite happens with the mosquito population. By the end of winter season 1, few mosquitoes remain. At time t_k,

$$\begin{cases} \Delta S_M = -r_3 S_M^- + p_3 I_M^- \\ \Delta E_M = -E_M^- \\ \Delta I_M = -(r_3 + p_3) I_M^-, \end{cases} \tag{8}$$

where S_M and I_M are the susceptible and infected mosquito populations; r_3 is the total decrease in the mosquito population; and p_3 is the proportion of mosquitoes who are born with no infection from an original infected mosquito. The total number of exposed mosquitoes who have offspring is zero, because a mosquito only stays in the exposed class for a short period of time.

The basic reproductive number for the one-season model has similar properties to that of the extended model. In the one-season model, R_0 larger than 1 causes an outbreak of Rift Valley fever in the human and livestock populations. The dynamics in the first season of the extended model are the same for subsequent seasons. The reset initial conditions for the second season do not change the equilibrium points for this season and, because R_0 is greater than 1, we have an outbreak in the second season.

5. Simulations

5.1. *One-season model*

We calculated the effects on the human and domestic livestock populations by having infected mosquitoes enter North America. The initial site of infection is a small, fictitious coastal city. All the values used in the simulations are shown in Table 1. Each farmer buys one cow every 10 days, so $\Lambda_L = 0.1$. We take β_{HM} 10 times smaller than β_{MH} because the infection rate from human to mosquito is smaller than from mosquito to human. The infection rate from livestock to mosquito is likewise smaller than mosquito to livestock. Mosquitoes infect humans and livestock at the same rate.

Using the values in Table 2, Figure 2 shows the effect of decreasing the survival time of mosquitoes. As the survival time of mosquitoes is decreased (due to mosquito spraying), R_0 is reduced below 1.

For Figure 3, we choose the mosquito survival time to be such that $R_0 < 1$, which caused no outbreaks within the year and led to eradication of the disease. For Figure 4, we choose the mosquito survival time to be such that $R_0 > 1$, which caused a disease outbreak at the beginning of the year, but the disease was still eradicated after the outbreak.

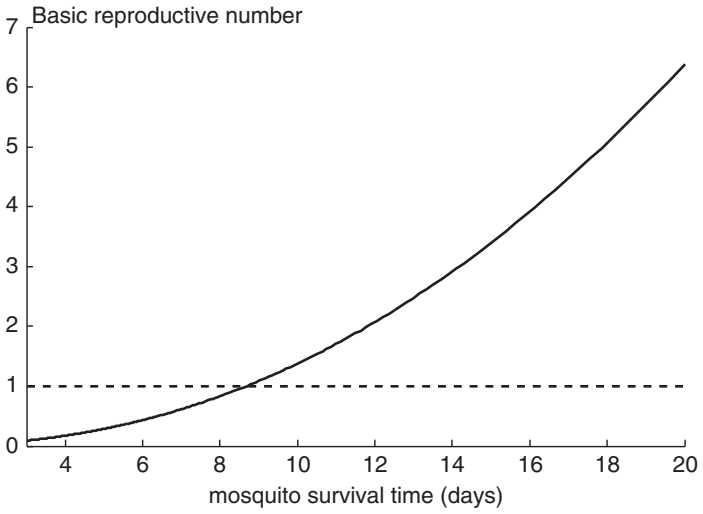

Figure 2. The effects of mosquito survival time on the basic reproductive number, R_0. R_0 drops below 1 if the mosquito survival time is sufficiently small. All other parameters are set to their median values listed in Table 2.

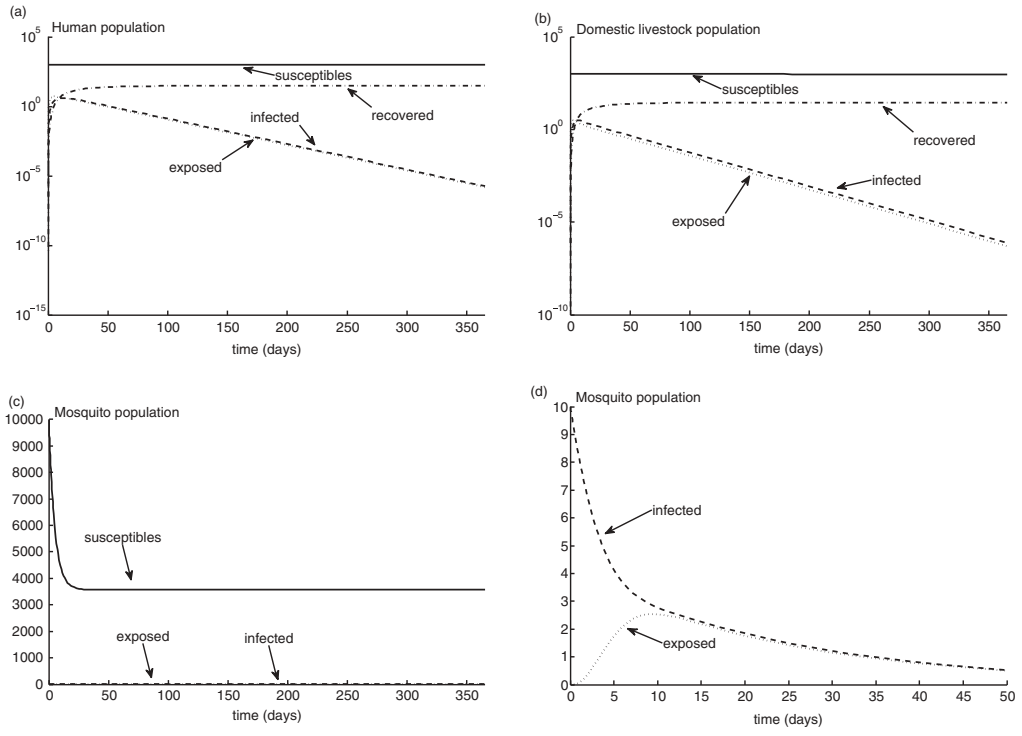

Figure 3. The behavior of (a) the human population, (b) the domestic livestock population, and (c) the mosquito population when the mosquito survival time is such that $R_0 < 1$ for one season (assuming winter does not change the dynamics of populations). The solid lines represent the susceptible populations, the dotted lines the exposed populations, the dashed lines the infected populations, and the dash-dot lines are the recovered populations. (d) The initial behavior of the mosquito population when $R_0 < 1$.

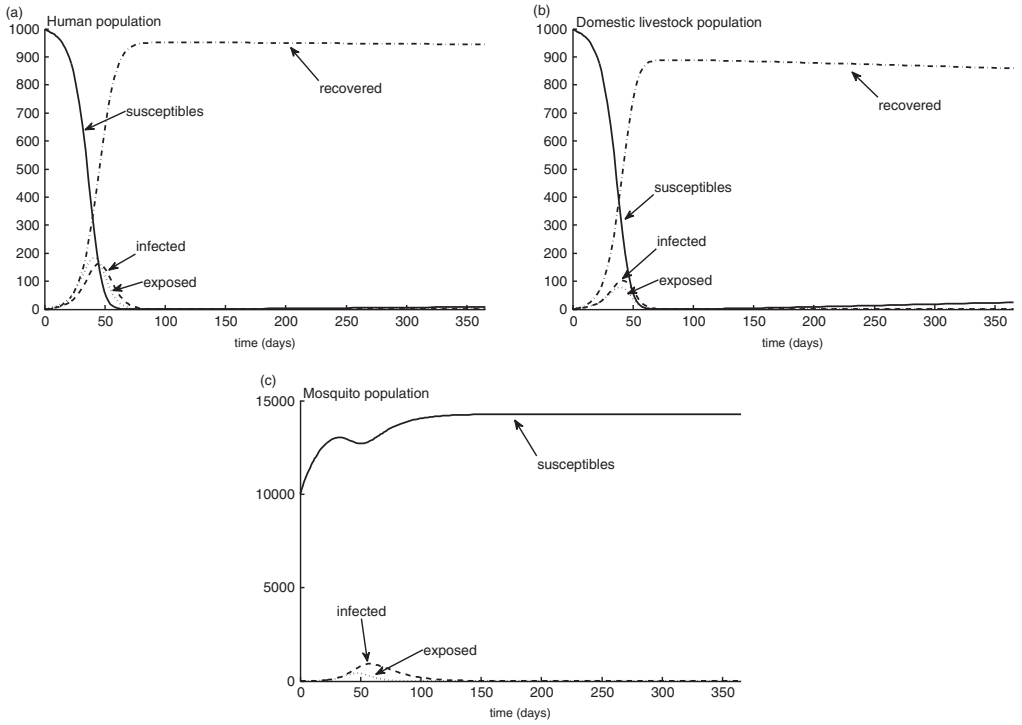

Figure 4. The behavior of (a) the human population, (b) the domestic livestock population and (c) the mosquito population when the mosquito survival time is such that $R_0 > 1$ for one season (assuming winter does not change the dynamics of populations). The solid lines represent the susceptible populations, the dotted lines the exposed populations, the dashed lines the infected populations, and the dash-dot lines are the recovered populations.

For $R_0 < 1$, the mosquito population in Figure 3c decreases. Because mosquitoes do not recover from the disease, and the mosquito survival time is such that $R_0 < 1$, we take the death rate to be large enough so that the mosquitoes do not have enough time to infect the human and domestic livestock populations in order to have an outbreak. Because infection between humans and livestock is nonexistent, it suggests that the disease-free equilibrium is stable and Rift Valley fever does not become endemic.

For a small value of d_M resulting in $R_0 > 1$, the disease causes a decrease in the susceptible human and domestic livestock populations, which follows an increase in the recovered human and domestic livestock populations, because both populations have lifelong immunity (Figure 4a and 4b). The infection does not completely eradicate the virus, which suggests that the endemic equilibrium is stable.

5.2. *Multiple-season model*

We allow 2% of the second generation of susceptible mosquitoes to survive the winter. Meanwhile, the susceptible human population increases by 10%,

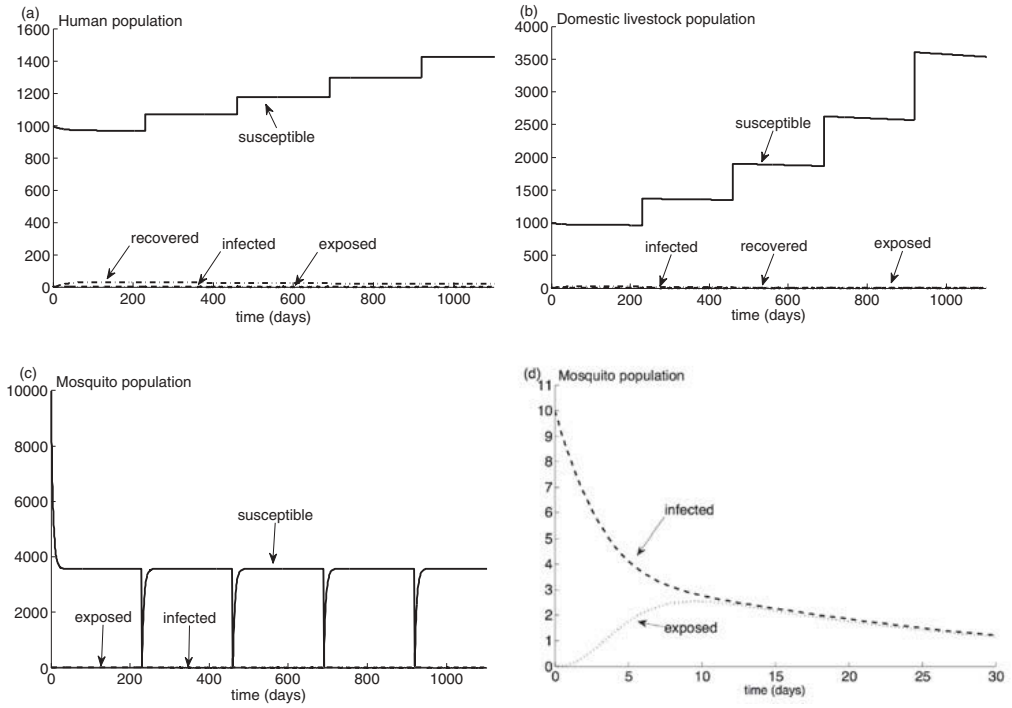

Figure 5. The behavior of (a) the human population, (b) the domestic livestock population, and (c) the mosquito population when the mosquito survival time is such that $R_0 < 1$ for multiple seasons. The solid lines represent the susceptible populations, the dotted lines the exposed populations, the dashed lines the infected populations, and the dash-dot lines the recovered populations. (d) The initial behavior of the mosquito population when $R_0 < 1$.

the infected population decreases to zero, and 10% of the recovered population becomes susceptible. The domestic livestock population grows at the same rate as the human population. The susceptible population increases by 40% during the winter months, so 40% of the recovered population becomes susceptible. The domestic livestock increases by 10% to account for the buying of livestock due to deaths in the previous season.

Figure 5 shows the effects of choosing the mosquito survival time to be such that $R_0 < 1$, which causes no outbreaks within 5 years and leads to eradication of the disease. Figure 6 shows the effects of choosing the mosquito survival time to be such that $R_0 > 1$, which causes a disease outbreak every year in the human and domestic livestock populations.

Figure 5c shows the effects of impulses on the mosquito population by decreasing 98% of all the susceptible mosquitoes during the winter seasons when $R_0 < 1$. For both the human and domestic livestock populations, the disease-free equilibrium is stable (Figures 5a and 5b). The only difference is a change in the susceptible populations due to the discontinuous increase in populations during the winter months.

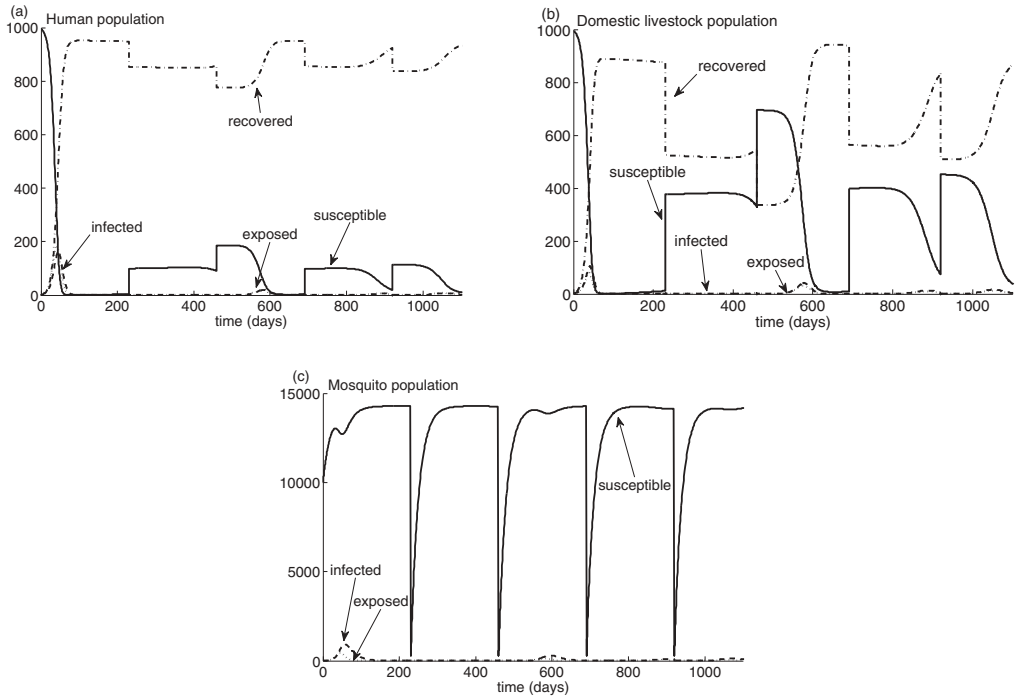

Figure 6. The behavior of (a) the human population, (b) the domestic livestock population, and (c) the mosquito population when the mosquito survival time is such that $R_0 > 1$ for multiple seasons. The solid lines represent the susceptible populations, the dotted lines the exposed populations, the dashed lines the infected populations, and the dash-dot lines the recovered populations.

For $R_0 > 1$, outbreaks occur every year. The first outbreak is larger than subsequent years, but the infected populations remain endemic. Infection persists in the mosquito population even though 98% of the infected are removed from the model due to winter (Figure 6c).

5.3. *Sensitivity*

Latin Hypercube Sampling is a statistical sampling that allows for an efficient analysis of parameter variations across simultaneous uncertainty ranges in each parameter; partial rank correlation coefficients rank the coefficients by the degree of influence each has on the outcome, regardless of whether that influence increases or decreases the effect. Latin Hypercube Sampling is most efficient if the outcome variable is a monotonic function of each of the input parameters (Blower and Dowlatabadi, 1994). Stein (1985) showed that, for many simulations, Latin Hypercube Sampling is the most efficient design, even if the outcome variable is not monotonic. Figure 7a shows the partial rank correlation coefficient sensitivity for all parameters for 1000 runs. R_0 is most sensitive to the mosquito death rate d_M, as Figure 7b shows.

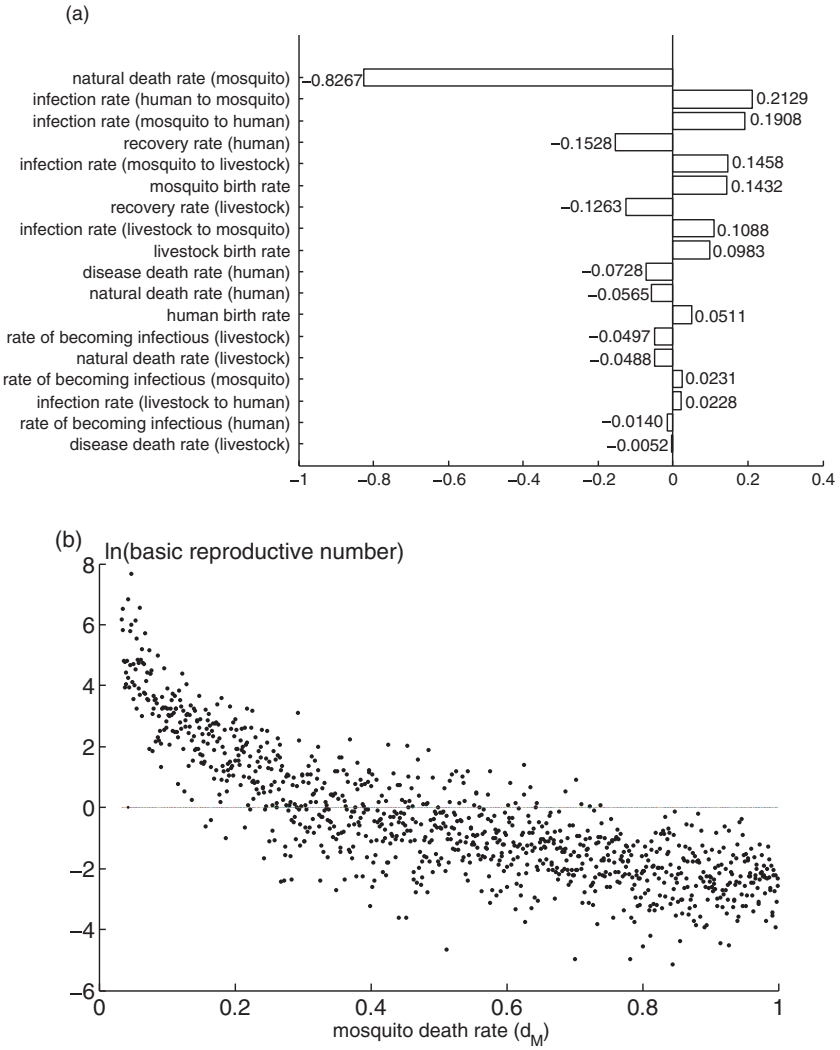

Figure 7. (a) Partial rank correlation coefficient sensitivity analysis on R_0 for all parameters. (b) The effect of the disease death rate d_M on R_0 using Monte Carlo simulations with parameters drawn using Latin Hypercube Sampling.

6. Conclusion

An outbreak of Rift Valley fever in North America is theoretically possible: only a very small number of infected mosquitoes is needed to establish the virus in North America. As seen with West Nile virus, a disease can establish itself with a mosquito as a vector, and humans and livestock as hosts. The virulence of West Nile virus in North America demonstrates the potential of other mosquito-borne pathogens.

We showed that reducing the survival time of mosquitoes below approximately 8.67 days would be sufficient to control an imported outbreak of Rift Valley fever. This could be achieved through spraying insecticides. Insecticide control of mosquitoes has been effective in reducing malaria

worldwide between the 1940s and 1960s (Trigg and Kondrachine, 1998; Mabaso et al., 2004; Macintyre et al., 2006; Al-Arydah and Smith?, 2011). Female mosquitoes live between two weeks and a month, depending on warmth and moisture (CDC, 2012), so reducing this duration to less than 8.67 days results in disease eradication.

The multiple season model shows the same results. If Rift Valley fever can enter North America, an outbreak is likely to occur unless it can be controlled quickly. Furthermore, if Rift Valley fever invades North America once, it is likely to break out again each year. The sufficient condition given in Section A.2 of the Appendix to have eigenvalues with negative real part (ensuring that the disease-free equilibrium is stable) is $d_M > 0.15$, where d_M is the mosquito death rate. This threshold is different from the one found using R_0 ($d_M > 0.115$). The difference is due to the condition used in Section A.2 of the Appendix, which is sufficient but not necessary in order to have all eigenvalues negative. A sufficient and necessary threshold could be calculated from the Routh–Hurwitz conditions if fewer parameters were involved.

While the behavior of the system is consistent with expectations, most parameters have little effect on R_0. Density-independence and mass-action terms limit the model to small populations. We used initial values corresponding to a small coastal population in North America instead of the entire North American population. We ignored the presence of cities.

Our results suggest that countries in the developed world face ongoing threats of disease introduction. As global travel increases the movement of humans, livestock, and vectors around the world, the potential for new outbreaks is heightened. We must be ready to face these challenges.

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A. Appendix

A.1. Equilibria

The model has two equilibria. The disease-free equilibrium is $\left(S_H^*, E_H^*, I_H^*, R_H^*, S_L^*, E_L^*, I_L^*, R_L^*, S_M^*, E_M^*, I_M^*\right) = \left(\frac{\Lambda_H}{d_H}, 0, 0, 0, \frac{\Lambda_L}{d_L}, 0, 0, 0, \frac{\Lambda_M}{d_M}, 0, 0\right)$. The endemic equilibrium is

$$\left(S_H^*, E_H^*, I_H^*, R_H^*, S_L^*, E_L^*, I_L^*, R_L^*, S_M^*, E_M^*, I_M^*\right), \tag{9}$$

where I_M^* is the solution to

$$\rho I_M^3 + \eta I_M^2 + \delta I_M + \omega = 0 \tag{10}$$

with

$$\xi_1 = \frac{\beta_{LM}\varepsilon_L\beta_{ML}\Lambda_L}{(d_L+\mu_L+\gamma_L)(d_L+\varepsilon_L)} \tag{11}$$

$$\xi_2 = \frac{\beta_{HM}\varepsilon_L\Lambda_L}{(d_H+\mu_H+\gamma_H)(d_H+\varepsilon_H)} \tag{12}$$

and

$$\rho = d_M(d_M+\varepsilon_M)\left(\xi_1\beta_{MH}\beta_{ML}+\beta_{ML}^2 d_M\beta_{MH}+\xi_2\beta_{MH}\beta_{ML}^2\right) \tag{13}$$

$$\begin{aligned}\eta = {} & d_M^2(d_M+\varepsilon_M)\beta_{ML}\left(\xi_1+\beta_{ML}+2d_L\beta_{MH}\right)\\ & + d_M(d_M+\varepsilon_M)\beta_{ML}\left(\xi_1\xi_2+\xi_2\beta_{MH}d_L+\xi_1 d_H\right)+\xi_1^2 d_M(d_M+\varepsilon_M)\\ & - \left(\beta_{ML}\varepsilon_L\Lambda_L - d_L d_M(d_M+\varepsilon_M)\right)\left(\xi_1\beta_{MH}+\xi_2\beta_{MH}\beta_{ML}\right)\end{aligned} \tag{14}$$

$$\begin{aligned}\delta = {} & d_M^2 d_L(d_M+\varepsilon_M)\left(\xi_1+2\beta_{ML}+d_L\beta_{MH}\right)\\ & - \left(\beta_{ML}\varepsilon_L\Lambda_L - d_L d_M(d_M+\varepsilon_M)\right)\left(\xi_1\xi_2+\xi_2\beta_{MH}d_L+\xi_1 d_H\right)\\ & - \beta_{MH}d_L\varepsilon_M\Lambda_M\left(\xi_1+\xi_2\beta_{ML}\right)-\xi_1^2\varepsilon_M\Lambda_M\end{aligned} \tag{15}$$

$$\omega = d_M^2 d_L^2(d_M+\varepsilon_M)-\xi_1 d_H d_L\varepsilon_M\Lambda_M-\xi_2\beta_{MH}d_L^2\varepsilon_M\Lambda_M-\xi_1\xi_2 d_L\varepsilon_M\Lambda_M, \tag{16}$$

where

$$S_H^* = \frac{\Lambda_H}{d_H+\beta_{MH}I_M^*+\beta_{LH}I_L^*} \tag{17}$$

$$E_H^* = \frac{\beta_{MH}S_H^*I_M^*+\beta_{LH}S_H^*I_L^*/N_L^*}{d_H+\varepsilon_H} \tag{18}$$

$$I_H^* = \frac{\varepsilon_H E_H^*}{d_H+\mu_H+\gamma_H} \tag{19}$$

$$R_H^* = \frac{\gamma_H I_H^*}{d_H} \tag{20}$$

$$S_L^* = \frac{\Lambda_L}{d_L+\beta_{ML}I_M^*} \tag{21}$$

$$E_L^* = \frac{\beta_{ML}S_L^*I_M^*}{d_L+\varepsilon_L} \tag{22}$$

$$I_L^* = \frac{\varepsilon_L E_L^*}{d_L+\mu_L+\gamma_L} \tag{23}$$

$$R_L^* = \frac{\gamma_L I_L^*}{d_L} \tag{24}$$

$$S_M^* = \frac{\Lambda_M}{d_M+\beta_{HM}I_M^*+\beta_{LM}I_L^*} \tag{25}$$

$$E_M^* = \frac{\beta_{HM}S_M^*I_H^*+\beta_{LM}S_M^*I_L^*}{d_M+\varepsilon_M} \tag{26}$$

and where I_M^* is the solution to Eq. (10).

A.2. Stability of the disease-free equilibrium

The Jacobian matrix evaluated at the disease-free equilibrium is $J_{DFE} = \left(J^{(1)}_{DFE} | J^{(2)}_{DFE}\right)$, where

$$J^{(1)}_{DFE} = \begin{pmatrix} -d_H & 0 & 0 & 0 & 0 & 0 \\ 0 & -d_H - \varepsilon_H & 0 & 0 & 0 & 0 \\ 0 & \varepsilon_H & -d_H - \mu_H - \gamma_H & 0 & 0 & 0 \\ 0 & 0 & \gamma_H & -d_H & 0 & 0 \\ 0 & 0 & 0 & 0 & -d_L & 0 \\ 0 & 0 & 0 & 0 & 0 & -d_L - \varepsilon_L \\ 0 & 0 & 0 & 0 & 0 & \varepsilon_L \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\beta_{HM} S^*_M & 0 & 0 & 0 \\ 0 & 0 & \beta_{HM} S^*_M & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{27}$$

$$J^{(2)}_{DFE} = \begin{pmatrix} -\beta_{LH} S^*_H / S^*_L & 0 & 0 & 0 & -\beta_{MH} S^*_H \\ \beta_{LH} S^*_H / S^*_L & 0 & 0 & 0 & \beta_{MH} S^*_H \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\beta_{ML} S^*_L \\ 0 & 0 & 0 & 0 & \beta_{ML} S^*_L \\ -d_L - \mu_L - \gamma_L & 0 & 0 & 0 & 0 \\ \gamma_L & -d_L & 0 & 0 & 0 \\ -\beta_{LM} S^*_M & 0 & -d_M & 0 & 0 \\ \beta_{LM} S^*_M & 0 & 0 & -d_M - \varepsilon_M & 0 \\ 0 & 0 & 0 & \varepsilon_M & -d_M \end{pmatrix}. \tag{28}$$

The matrix has the characteristic equation

$$0 = \det\left(J_{DFE}\left(S^*_H, E^*_H, I^*_H, R^*_H, S^*_L, E^*_L, I^*_L, R^*_L, S^*_M, E^*_M, I^*_M\right) - \alpha I_{11}\right) \tag{29}$$

$$= (-d_H - \alpha)^2 (-d_L - \alpha)^2 (-d_M - \alpha) f(\alpha), \tag{30}$$

where $f(\alpha)$ is the determinant of $(M_1|M_2)$ where

$$M_1 = \begin{pmatrix} -d_H - \varepsilon_H - \alpha & 0 & 0 \\ \varepsilon_H & -d_H - \mu_H - \gamma_H - \alpha & 0 \\ 0 & 0 & -d_L - \varepsilon_L - \alpha \\ 0 & 0 & \varepsilon_L \\ 0 & \beta_{HM} \frac{\Lambda_M}{d_M} & 0 \\ 0 & 0 & 0 \end{pmatrix} \tag{31}$$

$$M_2 = \begin{pmatrix} \beta_{LH} \frac{\Lambda_H d_L}{d_H \Lambda_L} & 0 & \beta_{MH} \frac{\Lambda_H}{d_H} \\ 0 & 0 & 0 \\ 0 & 0 & \beta_{ML} \frac{\Lambda_L}{d_L} \\ -d_L - \mu_L - \gamma_L - \alpha & 0 & 0 \\ \beta_{LM} \frac{\Lambda_M}{d_M} & -d_M - \varepsilon_M - \alpha & 0 \\ 0 & \varepsilon_M & -d_M - \alpha \end{pmatrix} \tag{32}$$

and S^*_H, S^*_L, and S^*_M are the disease-free equilibrium values. Solving for $f(\alpha)$, we have

$$f(\alpha) = \alpha^6 + c_1 \alpha^5 + c_2 \alpha^4 + c_3 \alpha^3 + c_4 \alpha^2 + c_5 \alpha + c_6, \tag{33}$$

where

$$c_1 = 2d_H + \mu_H + \gamma_H + \varepsilon_H + 2d_L + \varepsilon_L + \mu_L + \gamma_L \tag{34}$$

$$\begin{aligned} c_2 = {} & \big(d_H + \varepsilon_H + \gamma_H\big)(d_H + \varepsilon_H) + \big(2d_H + \varepsilon_H + \mu_H + \gamma_H\big)\big(2d_L + \varepsilon_L + \mu_L + \gamma_L\big) \\ & + \big(2d_H + \varepsilon_H + \mu_H + \gamma_H\big)(2d_M + \varepsilon_M) + (d_L + \varepsilon_L)\big(d_L + \mu_L + \gamma_L\big) \\ & + \big(2d_L + \varepsilon_L + \mu_L + \gamma_L\big)(2d_M + \varepsilon_M) + d_M(d_M + \varepsilon_M) \end{aligned} \tag{35}$$

$$\begin{aligned} c_3 = {} & \big(d_H + \mu_H + \gamma_H\big)(d_H + \varepsilon_H)\big(2d_L + \varepsilon_L + \mu_L + \gamma_L\big) \\ & + \big(d_H + \mu_H + \gamma_H\big)(d_H + \varepsilon_H)(2d_M + \varepsilon_M) \\ & + (d_L + \varepsilon_L)\big(d_L + \mu_L + \gamma_L\big)\big(2d_H + \mu_H + \varepsilon_H + \gamma_H\big) \\ & + \big(2d_L + \mu_L + \varepsilon_L + \gamma_L\big)(2d_M + \varepsilon_M)\big(2d_H + \mu_H + \varepsilon_H + \gamma_H\big) \\ & + d_M(d_M + \varepsilon_M)\big(2d_H + \mu_H + \varepsilon_H + \gamma_H\big) \\ & + (d_L + \varepsilon_L)(2d_M + \varepsilon_M)\big(d_L + \mu_L + \gamma_L\big) \\ & + d_M(d_M + \varepsilon_M)\big(2d_L + \varepsilon_L + \mu_L + \gamma_L\big) \end{aligned} \tag{36}$$

$$\begin{aligned} c_4 = {} & \big(d_H + \mu_H + \gamma_H\big)(d_H + \varepsilon_H)(d_L + \varepsilon_L)\big(d_L + \mu_L + \gamma_L\big) \\ & + (d_H + \varepsilon_H)\big(d_H + \mu_H + \gamma_H\big)(2d_M + \varepsilon_M)\big(2d_L + \varepsilon_L + \mu_L + \gamma_L\big) \\ & + d_M(d_M + \varepsilon_M)(d_H + \varepsilon_H)\big(d_H + \mu_H + \gamma_H\big) \\ & + (d_L + \varepsilon_L)(2d_M + \varepsilon_M)\big(d_L + \mu_L + \gamma_L\big)\big(2d_H + \mu_H + \varepsilon_H + \gamma_H\big) \\ & + d_M(d_M + \varepsilon_M)\big(2d_H + \mu_H + \varepsilon_H + \gamma_H\big)\big(2d_L + \mu_L + \varepsilon_L + \gamma_L\big) \\ & + d_M(d_M + \varepsilon_M)(d_L + \varepsilon_L)\big(d_L + \mu_L + \gamma_L\big) - \varepsilon_H\varepsilon_M\beta_{HM}\beta_{MH}\frac{\Lambda_M}{d_M}\frac{\Lambda_H}{d_H} \\ & - \varepsilon_L\varepsilon_M\beta_{LM}\beta_{ML}\frac{\Lambda_L}{d_L}\frac{\Lambda_M}{d_M} \end{aligned} \tag{37}$$

$$\begin{aligned} c_5 = {} & \big(d_H + \mu_H + \gamma_H\big)(d_H + \varepsilon_H)(d_L + \varepsilon_L)(2d_M + \varepsilon_M)\big(d_L + \mu_L + \gamma_L\big) \\ & + d_M(d_M + \varepsilon_M)\big(d_H + \mu_H + \gamma_H\big)(d_H + \varepsilon_H)\big(2d_L + \varepsilon_L + \mu_L + \gamma_L\big) \\ & + d_M(d_M + \varepsilon_M)\big(d_L + \mu_L + \gamma_L\big)(d_L + \varepsilon_L)\big(2d_H + \varepsilon_H + \mu_H + \gamma_H\big) \\ & - \varepsilon_H\varepsilon_M\beta_{HM}\beta_{MH}\frac{\Lambda_M}{d_M}\frac{\Lambda_H}{d_H}\big(2d_L + \varepsilon_L + \mu_L + \gamma_L\big) \\ & - \varepsilon_L\varepsilon_M\beta_{LM}\beta_{ML}\frac{\Lambda_L}{d_L}\frac{\Lambda_M}{d_M}\big(2d_H + \varepsilon_H + \mu_H + \gamma_H\big) \end{aligned} \tag{38}$$

and

$$\begin{aligned} c_6 = {} & d_m(d_M + \varepsilon_M)\big(d_H + \mu_H + \gamma_H\big)(d_H + \varepsilon_H)\big(d_L + \mu_L + \gamma_L\big)(d_L + \varepsilon_L) \\ & - \varepsilon_L\varepsilon_M\beta_{LM}\beta_{ML}\frac{\Lambda_L}{d_L}\frac{\Lambda_M}{d_M}\big(d_H + \mu_H + \gamma_H\big)(d_H + \varepsilon_H) \\ & - \varepsilon_H\varepsilon_M\beta_{MH}\beta_{HM}\frac{\Lambda_H}{d_H}\frac{\Lambda_M}{d_M}\big(d_L + \mu_L + \gamma_L\big)(d_L + \varepsilon_L) \\ & - \varepsilon_H\varepsilon_L\varepsilon_M\beta_{LH}\beta_{HL}\beta_{HM}\frac{\Lambda_M}{d_M}\frac{\Lambda_H}{d_H}. \end{aligned} \tag{39}$$

Necessary and sufficient conditions for all the zeros of $f(\alpha)$ to have negative real parts are that c_4, c_5, and c_6 be bigger than zero (because c_1, c_2, and c_3 are strictly positive), and

$$a_1a_2a_3 + a_1a_5 > a_3^3 + a_1^2a_4 \tag{40}$$

$$\begin{aligned} & a_1a_6a_2\big(2a_1a_5 - a_3^2\big) + a_1^2a_6(a_4a_3 - a_1a_6) + a_1a_5(a_4a_5 - 3a_3a_6) + a_6a_3^3 \\ & + a_5^2a_1a_4 > a_1^2a_5a_4^2 + a_5(a_3a_4 - a_5a_2)(a_3 - a_1a_2). \end{aligned} \tag{41}$$

If these Routh–Hurwitz conditions are satisfied, the disease-free equilibrium is stable (Truccolo et al., 2003; Allen, 2006). For the values used in Table 2, c_4, c_5, and c_6 vary depending on parameter values and the state variables S_H^*, S_L^*, and S_M^*. By fixing the state variables, the values for d_M (mosquito death rate) can be varied in order to change c_4, c_5, and c_6 from positive to negative. The stability changes depending on the mosquito survival time. Numerically, c_4, c_5, and c_6 are positive when $d_M > 0.15$ (Figure A1), and the two inequalities are satisfied when $0 < d_M < 0.68$. The disease-free equilibrium is stable if $0.15 < d_M < 0.68$.

A.3. Basic reproductive number

The model has six infected populations, E_H, I_H, E_L, I_L, E_M, and I_M. The vector $\mathcal{F}$ representing new infections and the vector $\mathcal{V}$ representing transfers between compartments are given by

$$\mathcal{F} = \begin{pmatrix} \beta_{LH}\frac{S_H^* I_L^*}{N_L^*} + \beta_{MH} S_H^* I_M^* \\ 0 \\ \beta_{ML} S_L^* I_M^* \\ 0 \\ \beta_{LM} S_M^* I_L^* + \beta_{HM} S_M^* I_H^* \\ 0 \end{pmatrix} \tag{42}$$

$$\mathcal{V} = \begin{pmatrix} (d_H + \varepsilon_H) E_H^* \\ (d_H + \mu_H + \gamma_H) I_H^* - \varepsilon_H E_H^* \\ (d_L + \varepsilon_L) E_L^* \\ (d_L + \mu_L + \gamma_L) I_L^* - \varepsilon_L E_L^* \\ (d_M + \varepsilon_M) E_M^* \\ d_M I_M^* - \varepsilon_M E_M^* \end{pmatrix}. \tag{43}$$

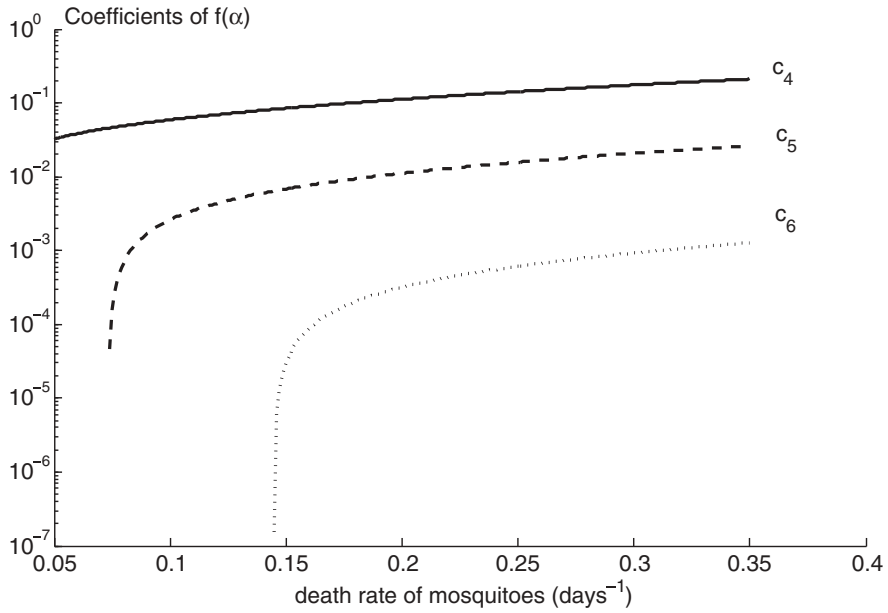

Figure A1. Sign change for certain parameters in $f(\alpha)$. When c_6 becomes negative (at around 0.15), both c_4 and c_5 are still positive. The values of c_4 and c_5 only become negative after c_6 has already fallen below zero.

The matrices F and V are

$$F = \begin{pmatrix} 0 & 0 & 0 & \beta_{LH}S_H^*/S_L^* & 0 & \beta_{MH}S_H^* \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \beta_{ML}S_L^* \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_{HM}S_M^* & 0 & \beta_{LM}S_M^* & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{44}$$

$$V = \begin{pmatrix} d_H+\varepsilon_H & 0 & 0 & 0 & 0 & 0 \\ -\varepsilon_H & d_H+\mu_H+\gamma_H & 0 & 0 & 0 & 0 \\ 0 & 0 & d_L+\varepsilon_L & 0 & 0 & 0 \\ 0 & 0 & -\varepsilon_L & d_L+\mu_L+\gamma_L & 0 & 0 \\ 0 & 0 & 0 & 0 & d_M+\varepsilon_M & 0 \\ 0 & 0 & 0 & 0 & -\varepsilon_M & d_M \end{pmatrix}, \tag{45}$$

where S_H^*, S_L^*, and S_M^* are the disease-free equilibrium values. Next, $FV^{-1} = \left(FV_{(1)}^{-1}|FV_{(2)}^{-1}\right)$ was calculated, and the eigenvalue with the largest modulus is the value of R_0. We have

$$FV_{(1)}^{-1} = \begin{pmatrix} 0 & 0 & \frac{\beta_{LH}S_H^*\varepsilon_L}{S_L^*(d_L+\mu_L+\gamma_L)(d_L+\varepsilon_L)} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \\ \frac{\beta_{HM}S_M^*\varepsilon_H}{(d_H+\varepsilon_H)(d_H+\mu_H+\gamma_H)} & \frac{\beta_{HM}S_M^*}{d_H+\mu_H+\gamma_H} & \frac{\beta_{LM}S_M^*\varepsilon_L}{(d_L+\varepsilon_L)(d_L+\mu_L+\gamma_L)} \\ 0 & 0 & 0 \end{pmatrix} \tag{46}$$

$$FV_{(2)}^{-1} = \begin{pmatrix} \frac{\beta_{LH}S_H^*}{S_L^*(d_L+\mu_L+\gamma_L)} & \frac{\beta_{MH}S_H^*\varepsilon_M}{d_M(d_M+\varepsilon_M)} & \frac{\beta_{MH}S_H^*}{d_M} \\ 0 & 0 & 0 \\ 0 & \frac{\beta_{ML}S_L^*\varepsilon_M}{d_M(d_M+\varepsilon_M)} & \frac{\beta_{ML}S_L^*}{d_M} \\ 0 & 0 & 0 \\ \frac{\beta_{LM}S_M^*}{d_L+\mu_L+\gamma_L} & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}. \tag{47}$$

The characteristic polynomial is

$$\det\left(FV^{-1} - \alpha I_6\right) = \alpha^3 g(\alpha), \tag{48}$$

where $g(\alpha)$ is the determinant of

$$\begin{pmatrix} -\alpha & \frac{\beta_{LH}S_H^*\varepsilon_L}{S_L^*(d_L+\mu_L+\gamma_L)(d_L+\varepsilon_L)} & \frac{\beta_{MH}S_H^*\varepsilon_M}{d_M(d_M+\varepsilon_M)} \\ 0 & -\alpha & \frac{\beta_{ML}S_L^*\varepsilon_M}{d_M(d_M+\varepsilon_M)} \\ \frac{\beta_{HM}S_M^*\varepsilon_H}{(d_H+\varepsilon_H)(d_H+\mu_H+\gamma_H)} & \frac{\beta_{LM}S_M^*\varepsilon_L}{(d_L+\varepsilon_L)(d_L+\mu_L+\gamma_L)} & -\alpha \end{pmatrix}. \tag{49}$$

Solving for α, we have:

$$-\alpha^3 + A\alpha + B = 0, \tag{50}$$

where

$$\begin{aligned} A = &\frac{\beta_{LM}S_M^*\varepsilon_L}{(d_L+\varepsilon_L)(d_L+\mu_L+\gamma_L)}\frac{\beta_{ML}S_L^*\varepsilon_M}{d_M(d_M+\varepsilon_M)} \\ &+ \frac{\beta_{HM}S_M^*\varepsilon_H}{(d_H+\varepsilon_H)(d_H+\mu_H+\gamma_H)}\frac{\beta_{MH}S_H^*\varepsilon_M}{d_M(d_M+\varepsilon_M)} \end{aligned} \tag{51}$$

$$B = \frac{\beta_{HM} S_M^* \varepsilon_H}{(d_H + \varepsilon_H)(d_H + \mu_H + \gamma_H)} \frac{\beta_{LH} S_H^* \varepsilon_L}{(d_L + \mu_L + \gamma_L)(d_L + \varepsilon_L)} \frac{\beta_{ML} \varepsilon_M}{d_M (d_M + \varepsilon_M)}. \tag{52}$$

Because A and B are positive, the equation $\alpha^3 = A\alpha + B$ has a real solution (Figure A2).

Eq. (50) has solutions

$$\alpha_1 = \frac{1}{6}\xi^{\frac{1}{3}} + 2A\xi^{-\frac{1}{3}} \tag{53}$$

$$\alpha_2 = \frac{1}{6}\left(-\frac{1}{2} + i\frac{3^{\frac{1}{2}}}{2}\right)\xi^{\frac{1}{3}} + 2A\left(-\frac{1}{2} - i\frac{3^{\frac{1}{2}}}{2}\right)\xi^{-\frac{1}{3}} \tag{54}$$

$$\alpha_3 = \frac{1}{6}\left(-\frac{1}{2} - i\frac{3^{\frac{1}{2}}}{2}\right)\xi^{\frac{1}{3}} + 2A\left(-\frac{1}{2} + i\frac{3^{\frac{1}{2}}}{2}\right)\xi^{-\frac{1}{3}}, \tag{55}$$

where

$$\xi = 108B + 12\left(81B^2 - 12A^3\right)^{\frac{1}{2}}. \tag{56}$$

The value for R_0 is the largest modulus of the eigenvalues α_i (Greenhalgh, 1996).

Case 1. If $81B^2 - 12A^3 > 0$, with $M = \frac{1}{6}\xi^{\frac{1}{3}}$ and $N = 2A\xi^{-\frac{1}{3}}$, then

$$\alpha_1 = M + N \tag{57}$$

$$\alpha_{2,3} = -\frac{1}{2}(M + N) \pm \frac{3^{\frac{1}{2}}}{2} i(M - N) \tag{58}$$

and the modulus of each eigenvalue is

$$|\alpha_1| = \left((M + N)^2\right)^{\frac{1}{2}} \tag{59}$$

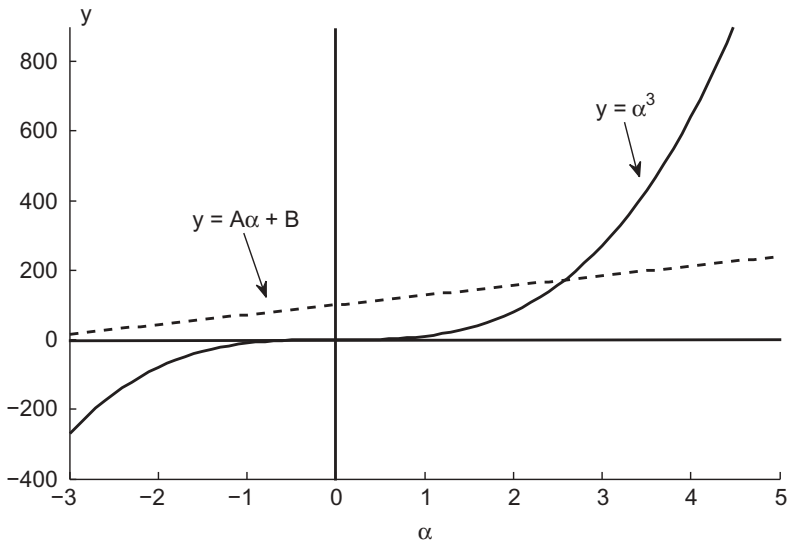

Figure A2. Intersection of the line $y = A\alpha + B$ with the cubic polynomial $y = \alpha^3$ when A and B are positive.

$$= \left(M^2 + 2MN + N^2\right)^{\frac{1}{2}} \tag{60}$$

and

$$|\alpha_{2,3}| = \left(\frac{1}{4}(M+N)^2 + \frac{3}{4}(M-N)^2\right)^{\frac{1}{2}} \tag{61}$$

$$= \frac{1}{2}\left(4N^2 - 4MN + 4M^2\right)^{\frac{1}{2}} \tag{62}$$

$$= \left(M^2 - 2MN + N^2\right)^{\frac{1}{2}}, \tag{63}$$

where $|\alpha_1| > |\alpha_{2,3}|$. $R_0^{(1)} = |\alpha_1|$, where $R_0^{(1)}$ is the basic reproductive number if $81B^2 - 12A^3$ is positive.

Case 2. If $81B^2 - 12A^3 < 0$ then $|\alpha_1| = |\alpha_{2,3}|$. $R_0^{(2)} = |\alpha_1|$, where $R_0^{(2)}$ is the basic reproductive number if $81B^2 - 12A^3$ is negative. We use the fact that $|\alpha_1| = (\alpha_1\bar{\alpha}_1)^{\frac{1}{2}}$ and polar coordinates to find the modulus. Because $r = \left((108B)^2 + 12^2|81B^2 - 12A^3|\right)^{\frac{1}{2}}$ and $\cos\theta = \frac{108B}{r}$,

$$\alpha_1 = \frac{1}{6}r^{\frac{1}{3}}\exp\left(\frac{i\theta}{3}\right) + 2Ar^{-\frac{1}{3}}\exp\left(-\frac{i\theta}{3}\right) \tag{64}$$

$$\bar{\alpha}_1 = \frac{1}{6}r^{\frac{1}{3}}\exp\left(-\frac{i\theta}{3}\right) + 2Ar^{-\frac{1}{3}}\exp\left(i\frac{\theta}{3}\right) \tag{65}$$

and so

$$R_0^{(2)} = |\alpha_1| \tag{66}$$

$$= \frac{1}{36}r^{\frac{1}{3}} + \frac{A}{3}\cos\frac{2\theta}{3} + 4A^2r^{-\frac{2}{3}}. \tag{67}$$

We have

$$R_0 = |\alpha_1|. \tag{68}$$

A.4. Effects of reducing winter to an impulse

The seasonal changes occurring after an introduction of Rift Valley fever to North America reduce the dynamics of the human, livestock, and mosquito populations in the winter months to a single impulse. We compare this seasonal-jump model to a continuous ODE model for both the summer and winter months with impulses only at the end of each season.

In a continuous model for the winter with $S_M = E_M = I_M = \Lambda_M = 0$ at the end of winter, the susceptible, exposed, and infected mosquitoes tend to zero exponentially fast. Figure A3 shows the dynamics of the continuous winter season for the susceptible mosquito population.

At the end of summer, the susceptible mosquito population is at S_M^-. Throughout the winter, the mosquito population decreases exponentially. At the end of the winter season, $S_M^{\text{ends}} = S_M^- \exp(-rT)$, where T is the duration of winter. A proportion p_3 of offspring of the infected mosquitoes hatch at the end of winter, which increases the total population of mosquitoes before the next summer season to $S_M^+ = S_M^- \exp(-rT) + p_3 I_M$. If $r_3 = 1 - \exp(-rT)$, the two models give the same results for the mosquito population. The same results apply to the human and livestock populations.

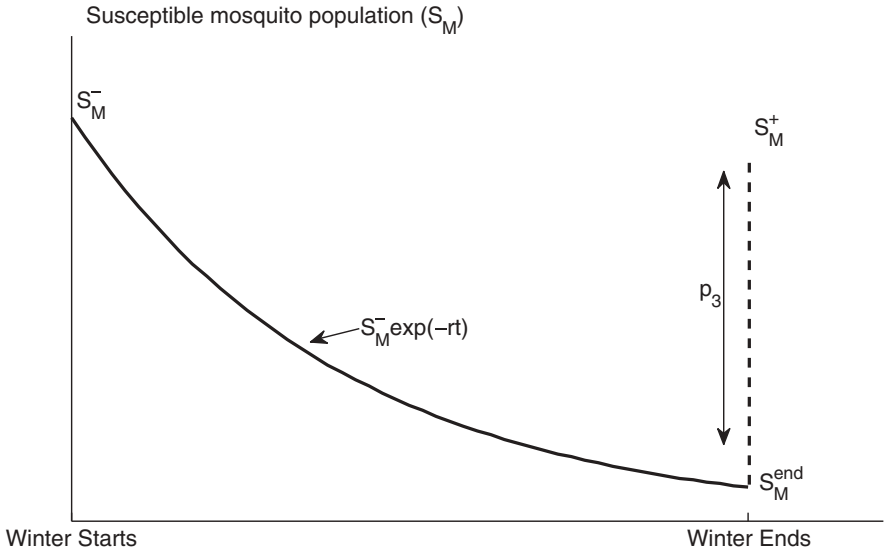

Figure A3. Comparison between full-time model and seasonal-jump model. The curve $S_M = S_M^- \exp(-rt)$ reflects the dynamics of the susceptible mosquito population during the winter months. At the end of the winter $\left(S_M^{\text{end}}\right)$, the mosquito population increases by p_3. S_M^- is the value before the impulse for the seasonal-jump model and S_M^+ is the value after the impulse.