Electronic supplementary material for
Localized Rift Valley fever virus persistence explains epidemic and interepidemic dynamics and guides control strategies

This PDF file includes:

Supporting text
Tables S1 to S6
Figures S1 to S13
Legends for Datasets S1
ESM References

Supporting Information Text

Detailed methods and materials

We developed a deterministic, ordinary differential equation (ODE)-based model with two mosquito vectors and a single host driven by realistic climate forcing. Specifically, to drive the vector population dynamics and simulate the RVFV dynamics during a 34-year period, we used rainfall and temperature data (described below) from a representative farm in central South Africa. The vector-borne component is conceptually based on the MacDonald malaria model [1] with the addition of transovarial transmission in one vector species, more development stages and climate forcing to drive the population dynamics of both vector species. A single, large sheep flock situated within a single pan (a depression in the ground that fills with water during rain) in central South Africa was selected as the appropriate closed epidemiological unit. The transovarial *Aedes* vector was modelled with a univoltine life cycle and with the *Culex* vector hatching shortly after the *Aedes*. We used a susceptible (S), infected (I) and recovered (R) model in one host species with two life stages: adult sheep (S) and lambs (L) and a susceptible (S), exposed (E), infected (I) model for the two vector species: *Aedes* mosquitoes (A) and *Culex* mosquitoes (C) with multiple life stages (see Table S1 for all state variable definitions; Figure S1). *Aedes* can transmit RVFV both transovarially and horizontally, while *Culex* can only transmit RVFV horizontally. Summaries of the number of hosts or vectors infected were calculated over a yearly timeframe that represents the hatching season in South Africa (September 1 – August 31).

Model Equations

The parameters for the model are given in Table S4. The equations describing the sheep (which were compartmentalized as susceptible, SS, infected, IS, recovered, RS, or vaccinated, VS) and lamb populations (which were compartmentalized as susceptible, SL, infected, IL, recovered, RL, those with maternal immunity, AL, or vaccinated, VL) are as follows.

Adult sheep (SS, IS, RS and VS) are assumed to arise through maturation of lambs at per capita rate (g); are sold at per capita rate ($SoldS$) and subject to natural mortality at per capita rate (μS). Adult susceptible sheep (SS) are infected via bites from *Culex* mosquitoes at per capita rate βSLC per infected *Culex* mosquito (IC), and via bites from *Aedes* mosquitoes at per capita rate βSLA per infected *Aedes* mosquito (IA). The transmission rates βSLC and βSLA are given by Equations S27 and S29. Adult susceptible sheep (SS) are also lost to vaccination at the per capita rate (vax). Adult infected sheep (IS) recover from infection at per capita rate (σ) and are subject to a disease specific mortality rate of (ρS). Adult vaccinated sheep (VS) arise through maturation of vaccinated lambs (VL) at per capita rate (g).

Lambs (SL, IL, RL, VL, AL) are assumed to be purchased at a per capita rate (buyL); mature at a per capita rate (g), are subject to a per capita mortality rate (μL) and all (except IL) arise through birth at the per capita rate (bL), which is moderated by the carrying capacity ($NLmax$); Susceptible lambs (SL) also arise

from lambs born with maternal immunity that has waned at per capita rate (ωMA). Susceptible lambs (SL) are infected via bites from *Aedes* and *Culex* mosquitoes at per capita rates βSLA and βSLC per infected *Aedes* (IA) and *Culex* mosquito (IC), respectively. Susceptible lambs (SL) are also lost to vaccination at the per capita rate (vax). Infected lambs (IL) recover from infection at per capita rate (σ) and are subject to a disease specific mortality rate of (ρL). Lambs that have maternal immunity (AL) are born at the per capita rate of bL from the population of recovered sheep (RS), vaccinated sheep (VS) and the proportion of infected sheep (IS) that did not abort (1- $abort$). Lambs with maternal immunity (AL) are subject to loss by immunity waning at a per capita rate (ωMA) and the per capita lamb mortality rate (μL). Vaccinated lambs (VL) arise through the vaccination of susceptible lambs (SL) at per capita rate (vax).

$$\frac{dSS}{dt} = g \cdot SL - \text{soldS} \cdot SS - \beta SLC \cdot \text{SS} \cdot IC - \beta SLA \cdot SS \cdot IA - \mu S \cdot SS \quad \text{(S1)}$$

$$\frac{dIS}{dt} = g \cdot IL - \text{soldS} \cdot IS + \beta SLC \cdot SS \cdot IC + \beta SLA \cdot SS \cdot IA - \mu S \cdot IS - \sigma \cdot IS - \rho S \cdot IS \quad \text{(S2)}$$

$$\frac{dRS}{dt} = g \cdot RL - \text{soldS} \cdot RS - \mu S \cdot RS + \sigma \cdot IS \quad \text{(S3)}$$

$$\frac{dVS}{dt} = g \cdot VL - \text{soldS} \cdot VS - \mu S \cdot VS \quad \text{(S4)}$$

$$\frac{dSL}{dt} = bL \cdot \left(1 - \frac{NS}{NLmax}\right) \cdot SS + \omega MA \cdot AL + \text{buyL} - g \cdot SL - \beta SLC \cdot SL \cdot IC - \beta SLA \cdot SL \cdot IA - \mu L \cdot SL - \text{vax} \cdot SL \quad \text{(S5)}$$

$$\frac{dIL}{dt} = \beta SLC \cdot SL \cdot IC + \beta SLA \cdot SL \cdot IA - g \cdot IL - \mu L \cdot IL - \sigma \cdot IL - \rho L \cdot IL \quad \text{(S6)}$$

$$\frac{dRL}{dt} = \sigma \cdot IL - g \cdot RL - \mu L \cdot RL \quad \text{(S7)}$$

$$\frac{dAL}{dt} = b \cdot \left(1 - \frac{NS}{NLmax}\right) \cdot (RS + ((1 - Abort) \cdot IS + VS)) - \omega MA \cdot AL - \mu L \cdot AL \quad \text{(S8)}$$

$$\frac{dVL}{dt} = vax \cdot SL - g \cdot VL - \mu L \cdot VL \quad \text{(S9)}$$

The equations for the *Aedes* free-living stages are given, followed by the equations for the egg stages. These were compartmentalized as susceptible and infected larvae and pupae (SALP and IALP), young adults (prior to first feeding), (SAY and IAY), adults (SA and IA), eggs that are mature enough to hatch (SAE and IAE), and eggs that were laid this season and are not mature enough to hatch yet (newSAE and newIAE), respectively. There is also a compartment for exposed adults (EA).

Susceptible and infected *Aedes* larvae and pupae (SALP and IALP, respectively) hatch from mature susceptible and infected *Aedes* eggs (SAE and IAE, respectively) at per capita rate bhA during the period eggs are permitted to hatch indicated by $EndA$ = 1) as defined by the weather data. The population growth

is restricted by the carrying capacity ($NALPmax$), which acts on the total population of *Aedes* larvae and pupae. *Aedes* larvae and pupae develop and emerge as young adults (AY) at the per capita rate $devALP$ and are lost at the per capita mortality rate (μALP). The larval parameters $devALP$ and μALP with the daily temperature and therefore are included in the model as time-dependent variables. Likewise, the hatching parameter $EndA$ is also determined by the climate factors that trigger hatching.

Susceptible and infected young adult *Aedes* (SAY and IAY, respectively) develop and emerge from susceptible and infected *Aedes* larvae and pupae (SALP and IALP, respectively) at per capita rate $devALP$. They become susceptible and infected adults (SA and IA, respectively) once they are ready to take their first blood meal at the per capita rate of $waitA$ and are subject to the adult *Aedes* mortality rate (μA).

Adult *Aedes* (SA, IA, and EA) are assumed to arise at a per capita rate $waitA$, which captures the rate at which newly emerged *Aedes* become ready to take their first blood meal and are subject to natural mortality at per capita rate (μA). Susceptible adult *Aedes* (SA) become exposed (EA) at the per capita rate βASL. βASL is defined by Equation S28. Exposed *Aedes* become infected (IA) at the per capita extrinsic incubation rate (ε).

New susceptible and infected *Aedes* eggs (newSAE and newIAE) are laid by susceptible and infected adult *Aedes* (SA and IA), respectively, at per capita rate $EgAE$ with $EgNAE$ eggs laid during each oviposition. The new susceptible and infected *Aedes* eggs (newSAE and newIAE desiccate and mature into susceptible and infected eggs (SAE and IAE), respectively, that are capable of hatching at the per capita rate αAE and are subject to the per capita *Aedes* egg mortality rate (μAE). The population of mosquitoes that can lay new susceptible and infected eggs is defined by the transovarial transmission rate (q), whereby new susceptible *Aedes* eggs (newSAE) are laid by the entire population of susceptible and exposed adult *Aedes* (SA) and (1-q) of the infected adult *Aedes* (IA) and new infected *Aedes* eggs (newIAE) are laid by the proportion of infected adult *Aedes* (IA) that transmit the virus transovarially (q). The susceptible and infected *Aedes* eggs (SAE and IAE) are subject to the per capita *Aedes* egg mortality rate (μAE).

The climate dependent parameters are included as time-varying and are indicated as bold in the vector equations.

$$\frac{dSALP}{dt} = bhA \cdot EndA \cdot SAE \cdot \left(1 - \frac{NALP}{NALPmax}\right) - devALP \cdot SALP - \mu ALP \cdot SALP \quad \text{(S10)}$$

$$\frac{dSAY}{dt} = devALP \cdot SALP - \text{waitA} \cdot SAY - \mu A \cdot SAY \quad \text{(S11)}$$

$$\frac{dSA}{dt} = \text{waitA} \cdot SAY - \beta ASL \cdot SA \cdot (IL + IS) - \mu A \cdot SA \quad \text{(S12)}$$

$$\frac{dEA}{dt} = \beta ASL \cdot SA \cdot (IL + IS) - \varepsilon \cdot EA - \mu A \cdot EA \quad \text{(S13)}$$

$$\frac{dIALP}{dt} = bhA \cdot EndA \cdot IAE \cdot \left(1 - \frac{NALP}{NAEmax}\right) - devALP \cdot IALP - \mu ALP \cdot IALP \quad \text{(S14)}$$

$$\frac{dIAY}{dt} = devALP \cdot IALP - \text{waitA} \cdot IAY - \mu A \cdot IAY \quad \text{(S15)}$$

$$\frac{dIA}{dt} = waitA \cdot IAY + \varepsilon \cdot EA - \mu A \cdot IA \quad \text{(S16)}$$

$$\frac{dSAE}{dt} = \alpha AE \cdot newSAE - bhA \cdot EndA \cdot SA - \mu AE \cdot SAE \quad \text{(S17)}$$

$$\frac{dIAE}{dt} = \alpha AE \cdot newIAE - bhA \cdot EndA \cdot IAE - \mu AE \cdot IAE \quad \text{(S18)}$$

$$\frac{dnewSAE}{dt} = EgAE \cdot EgNAE \cdot (SA + EA + ((1 - q) \cdot IA)) - \alpha AE \cdot newSAE - \mu AE \cdot newSAE \quad \text{(S19)}$$

$$\frac{dnewIAE}{dt} = EgAE \cdot EgNAE \cdot q \cdot IA - \alpha AE \cdot newIAE - \mu AE \cdot newIAE \quad \text{(S20)}$$

The equations for the *Culex* free-living stages are given, followed by the equations for the egg stages. These were compartmentalized as susceptible eggs (SCE), larvae and pupae (SCLP) and young adults prior to first feeding (SCY). Adults we compartmentalized as (SC), exposed (EC) and infected (IC).

Susceptible *Culex* larvae and pupae (SCLP) hatch from susceptible *Culex* eggs (SCE) at per capita rate bhC during weather permissive periods ($hatchC = 1$). The population growth is limited by the carrying capacity ($NCLPmax$), which acts on the larval and pupae stage (SCLP). *Culex* larvae and pupae (SCLP) develop and emerge as young adults (SCY) at the per capita rate $devCLP$ and are lost at the per capita mortality rate (μCLP).

Susceptible young adult *Culex* (SCY) develop and emerge from susceptible *Culex* larvae and pupae (SCLP) at per capita rate $devCLP$. They become susceptible *Culex* adults (SC) once they are ready to take their first blood meal at the per capita rate of $waitC$ and are subject to the adult *Culex* mortality rate (μC).

Susceptible adult *Culex* (SC) are assumed to arise at a per capita rate $waitC$, which captures the rate of when young *Culex* are ready to take their first blood meal. All adult *Culex* (SC, IC, and EC) are subject to natural mortality at per capita rate (μC). Susceptible adult *Culex* (SC) become exposed (EC) at the per capita rate βCSL. βCSL is defined by Equation S30. Exposed *Culex* become infected (IC) at the per capita extrinsic incubation rate (ε).

Susceptible *Culex* eggs (SCE) are laid by susceptible and exposed *Culex* (SC) at per capita rate $EgCE$ with $EgNCE$ eggs laid during each oviposition. Infection with RVFV impacts the fertility of infected *Culex* adults[2] and only a proportion (1-δ) of infected *Culex* (IC) lay eggs (SCE). The susceptible *Culex* eggs are subject to the per capita *Culex* egg mortality rate (μCE).

$$\frac{dSCLP}{dt} = bhC \cdot hatchC \cdot SCE \cdot \left(1 - \frac{SCLP}{NCLPmax}\right) - devCLP \cdot SCLP - \mu CLP \cdot SCLP \quad \text{(S21)}$$

$$\frac{dSCY}{dt} = devCLP \cdot SCLP - \text{waitC} \cdot SCY - \mu C \cdot SCY \quad \text{(S22)}$$

$$\frac{dSC}{dt} = \text{waitC} \cdot SCY - \beta CSL \cdot SC \cdot (IL + IS) - \mu C \cdot SC \quad \text{(S23)}$$

$$\frac{dEC}{dt} = \beta CSL \cdot SC \cdot (IL + IS) - \epsilon \cdot EC - \mu C \cdot EC \quad \text{(S24)}$$

$$\frac{dIC}{dt} = \epsilon \cdot EC - \mu C \cdot IC \quad \text{(S25)}$$

$$\frac{dSCE}{dt} = EgCE \cdot EgNCE \cdot (SC + EC) + \delta \cdot EgCE \cdot EgNCE \cdot IC - bhC \cdot \text{hatchC} \cdot SCE - \mu CE \cdot SCE \quad \text{(S26)}$$

The frequency dependent transmission rates were calculated as follows:

$$\beta SLA = \frac{\text{biteA}}{NS+NL} \cdot \theta SLA \quad \text{(S27)}$$

$$\beta ASL = \frac{\text{biteA}}{NS+NL} \cdot \theta ASL \quad \text{(S28)}$$

$$\beta SLC = \frac{\text{biteC}}{NS+NL} \cdot \theta SLC \quad \text{(S29)}$$

$$\beta CSL = \frac{\text{biteC}}{NS+NL} \cdot \theta CSL \quad \text{(S30)}$$

The initial conditions for each population were: SS = 360, RS = 240, SL = 140, AL = 60, SAE = 17,946,000, IAE =54,000, SCE = 1000 and all other initial populations were zero. The host population was set at 800 as this was the average size of the flocks we surveyed in 2015 [3] and 2017 (Rostal et al., unpublished). It was assumed that the seroprevalence was 0.30 in adult sheep and that 25% of these recovered sheep had lambs with maternal antibodies at the start of the simulation. In total, 25% of the flock were assumed to be lambs.

Regarding the vectors, the simulation starts at the end of the winter season in South Africa, so only mosquito eggs were assumed to be present. Mosquito population sizes and the proportion of infected mosquitoes, which are believed to be low compared to the susceptible mosquitoes, have never been

quantified (especially for the number or proportion of infected eggs, larvae and pupae). We found that for simulations where infection persisted, the initial conditions had little impact on the long-term dynamics. However, during the parameter estimation process, we iteratively adjusted the initial *Aedes* egg population size so that the initial conditions matched the median generated by the best fitting models.

Climate-Driven Hatching and Development Rates

The hatching of both *Aedes* and *Culex* were triggered by rainfall once the temperature exceeded a threshold of 20ºC. To simulate the univoltine life cycle [4], seasonal hatching of *Aedes* was triggered early in the rainy season [5-7], once 38 mm cumulative rainfall had fallen during seven days, while the mean daily temperature was greater than 20ºC. *Aedes* eggs hatched for nine days (Table S2). The climate parameters that trigger hatching, development and mortality are have important impacts on vector populations. Further, for floodwater mosquitoes the parameters will vary from pan to pan depending on the local hydrological system (e.g., soil texture and composition, current water saturation levels, the saline levels within the pan etc.). Currently, there are no data available at the local pan level to provide good parameter values. Thus, we estimated these parameters based on ***preliminary simulations*** that resulted in expected patterns of the vector population dynamics (Figure S12). The variables estimated based on these preliminary simulations are indicated in Tables S3 and S4 and were used to represent sufficient rainfall to allow the larvae to hatch following flooding in the *Aedes*, and to ensure that some amount of standing water remained in containers or natural water sources for *Culex* to continue hatching.

The delay (H) in the hatching of *Culex* eggs after the *Aedes* start hatching was set at 7 days [8, 9], provided the temperature is greater than 20ºC and there has been greater than 2 mm of cumulative rainfall over three days (to ensure there is standing water present). We simulated the process of *Culex* reproduction throughout the entire year provided the temperature and rainfall thresholds continued to be met.

To simulate the temporal changes in standing water area on which *Culex* can lay their eggs, we used the proportion, *hatchC*, where

$$hatchC = \frac{\text{10 day cumulative rainfall}}{\text{the maximum 10 day cumulative rainfall for that particular year}}. \quad \text{(S31)}$$

$hatchC$ was multiplied by bhC (the reciprocal of the *Culex* hatching time) to support hatching during the periods.

The daily development rates of both *Aedes* and *Culex* larvae and pupae ($devLP$, which represents both $devALP$/ $devCLP$ in days^{-1}) were modelled as temperature-dependent and were calculated based on the equation (Eq. S2), where K is the daily mean temperature in Kelvin, R is the universal gas constant (1.987

cal·K^{-1}·mol^{-1}), K_{C25} is the 298.15 Kelvins (25°C), and the species dependent parameters ρ_{025}, and HA, HH and TH are given in Rueda *et al*. [10] (Table S3):

$$devLP = \frac{\rho_{025} \cdot \frac{K}{K_{C25}} \cdot \exp\left(\frac{HA}{R} \cdot \left(\frac{1}{K_{C25}} - \frac{1}{K}\right)\right)}{1 + \exp\left(\frac{HH}{R} \cdot \left(\frac{1}{TH} - \frac{1}{K}\right)\right)} \quad \text{(S32)}$$

The rate formula developed by Rueda *et al*. [10] was based on data from *Aedes aegypti* and *Culex quinquefasciatus*. To adjust for a difference in species development time, the development rate (*devALP*) for *Aedes mcintoshi* was increased by a factor, f_{Am} (Table S3), as that species develops at a faster rate [8]. Similarly, the development rate (*devCLP*) for *Culex pipiens* is slower than that for *C. quinquefasciatus* [11] and therefore it was multiplied by f_{Cp} (Table S3) .

The larval/pupal mortality rates for *Aedes* and *Culex* (μLP, which represents both μALP/μCLP) were derived from the following equation for each genus (parameters are defined in Table S4):

$$\phi = \frac{\text{devLP}}{\text{devLP} + \mu\text{LP}} \quad \text{(S33)}$$

using the genus-specific development rate and survival fractions (ϕ) obtained from the literature (Table S4). The demographic dynamics of the simulated vector populations (Figure S12) were consistent with patterns of population growth expected.

Assumptions Made about Host and Vector Populations

The following assumptions were made about the sheep population:

- Only susceptible lambs were imported into the flock ($buyL$), resulting in a closed system that RVFV cannot be imported into.
- Rams were sold upon reaching adulthood such that all adult sheep in the flock were female ($SoldS$).
- There was no specific lambing season as farmers in South Africa may manage their sheep to lamb three times every two years (Cordel, pers. comm.) Therefore, lamb births occur at a constant rate.
- Lambs (AL) born to recovered ewes had maternal immunity that waned (ωMA) before they mature into sheep.
- No exposed compartment was included among the sheep populations as the incubation period of RVFV in lambs is only 12-20 hours[12].
- If the vaccination scenario is run, only lambs are vaccinated, and they mature into vaccinated adult sheep.

- Further, the vaccine is assumed to be 100% efficacious with no waning of immunity.
- The vaccine is introduced at a constant daily rate (vax; as are all other rate parameters). This allows us to keep the proportion of vaccinated sheep ($vax.prop$) constant throughout the simulation.

The following assumptions were made about the *Aedes* and *Culex* mosquito populations:

- The carrying capacity ($NALPmax/NCLPmax$) acts via competition and predation on the number of larval and pupae stages.
- Hatching triggers ($endA/hatchC$) and development and mortality rates ($devALP/devCLP$ and $\mu ALP/\mu CLP$) were driven by rainfall and temperature as described above [10].
- Larvae and pupae stages were modelled together as one stage ($ALP/\ CLP$).
- Newly metamorphized adult mosquitoes (AY/CY) did not take their first blood meal until after the first three days (waitA)/waitC).
- *Aedes* eggs do not hatch in the same hatching period as they were laid because the eggs require a period for embryogenesis and enter a period of dormancy or diapause (αAE) until the next flood [13]. They first go into the *newSAE*/*newIAE* compartment until the dormancy period is completed.

Parameters Used in the Model

The values used for the model parameters were primarily obtained from the literature (Table 4). For those that were not available in the literature we used two processes for estimations: preliminary simulation and Latin hypercube estimation. ***Preliminary simulation:*** is described in the climate section and was used primarily to select climate-related parameters that result in the expected population dynamics in the vector populations (a peak of floodwater *Aedes* mosquitoes followed by an increase in the abundance of *Culex* mosquitoes that remain for rest of the season and dropping to very low numbers or zero over the winter (approximately June-August; see Figure S12). For parameters that could have a more substantial impact of the RVFV dynamics in the system, we used ***Latin hypercube estimation*** as described here. For six parameters, transovarial transmission, the mortality rate of *Aedes* eggs, *Aedes* and *Culex* bite rates, and *Aedes* and *Culex* carrying capacity, no reliable estimates were available. To identify realistic estimates, we used Latin hypercube sampling to evaluate 80,000 combinations of these six parameters. The results were evaluated by six criteria: mean annual seroprevalence was between 1-40%, the virus persisted for the entire simulation, at least one outbreak "spike" was detected, the ratio of infected *Aedes* eggs remain relatively constant during the simulation and the mean seroprevalence remained relatively constant during the simulation. An outbreak was considered a "spike" if the ratio of infected animals in that year compared to the average number of infected animals in the three years before and after was 2:1 or greater. The population of *Aedes* eggs and mean seroprevalence was determined to be "relatively constant" if the ratio of the mean value from the first half of the simulation to that of the second half of the simulation was within 0.9-1. For the infected *Aedes* eggs we averaged the annual maximum number of eggs. For the

seroprevalence we averaged the annual mean seroprevalence. There were three general set of patterns that arose among the six simulations that satisfied the six criteria (Figure S13). We evaluated each of them visually. Though the model was not developed as a predictive model, we selected the model that had an outbreak pattern that best matched the historical record of outbreaks in this part of South Africa as our exemplar simulation. The parameter estimates from the exemplar simulation were used to model all simulations presented, except where specific parameters were changed to evaluate the different scenarios.

Unpublished data: We have established a cohort of unvaccinated sheep in the Free State of South Africa (2016-2024). Every three months we sampled the sheep and administered a questionnaire to the farm owner/manager. The questionnaire asked about the number of births and deaths, the number of sheep purchased and the total number of sheep on the farm. This information was averaged over time (through 2019) and used for this study. The total number of sheep on the farm was also compared to the average from the data collected in Ngoshe et al [3], which presented a cross-sectional study of farms from which these cohort sheep farms were selected. The cohort sheep questionnaire data was similar to that administered by Ngoshe et al [3], though modified for follow up every three months. While this data remains unpublished, ethical approval to administer the questionnaires to the sheep owners/managers was provided by the following: US Hummingbird Institutional Review Board (no. 2014–25 24/11/2014), US DTRA Research Oversight Board (CT 2014–33 27/01/2015), SA Witwatersrand and Pretoria Universities Human Ethics Committee (M140306 30/04/2014; 140/2018 11/06/2018), and SA Provincial Departments of Health Free State and Northern Cape (NC2015/001 09/02/2015; 04/04/2015). Voluntary written consent was obtained from all participants included in the study.

Expert opinion/consultation

The values used in the exemplar simulation were reviewed by a group by RVF experts working in South Africa on the Understanding Rift Valley Fever in South Africa project in 2017 and again by all coauthors prior to publication.

Sensitivity Analysis

Three types of sensitivity analyses were conducted. First, we varied one or two parameters at a time and evaluated persistence and mean annual seroprevalence across the 34-year simulation. The parameters included in this first analysis were the transovarial transmission fraction, *Aedes* bite rate, host-to-*Aedes* and *Aedes*-to-host transmission parameters, external incubation rate (EIR) and the proportion of the flock vaccinated.

Second, we used Latin hypercube sampling to explore the model parameter space [14, 15]. Fourteen parameters were included in the sensitivity analysis: σ, ρS, ρL, μA, ϵ, θASL, θSLA, θCSL, θSLC, $biteA$,

$biteC$, μAE, q, and μC. The parameters were varied across a range that was 50% above and below the value used in the simulation (or 1.0 if 50% above a proportion was greater than 1.0; see Table 5). During these simulations the proportion of the flock that was vaccinated was set to 0. The sensitivity analysis consisted of 4000 iterations. Each parameter was evaluated based on its effect on six outcomes: RVFV persistence (years), mean annual seroprevalence, proportion of infected eggs at end of simulation, mean outbreak length, mean outbreak size, and the maximum single outbreak size. These outcomes were calculated for the full 34-year simulation interval. The results were then analyzed using a partial coefficient correlation analysis [14]. The third analysis was to explore the effect various parameters had on R_0, as described below.

Estimation of the Basic Reproduction Number, R_0

R_0 was calculated using the next generation matrix (NGM) approach [16]. The first step was to use a version of the model equations with constant rather than varying birth rates and evaluate R_0 at a fixed population size. This process was necessary as the carrying capacity parameter in the *Aedes* and *Culex* equations allowed for rapid population changes. This allowed us to characterise R_0 at the simulated, constant susceptible populations into which RVFV was introduced. The birth rate necessary to maintain a constant population size are provided in equations S34-S36:

$$\text{birthNSL} = \frac{(NS + NL) \cdot (\mu L + vax + g) \cdot (\mu S + g) \cdot (\mu S + soldS)}{NS \cdot (\mu S + soldS + g) \cdot (\mu S + vax + g)} \quad \text{(S34)}$$

$$\text{birthA} = \frac{\mu A \cdot (\mu A + waitA) \cdot (\mu AE + bhA) \cdot (\mu AE + \alpha AE) \cdot (\mu ALP + devALP)}{\text{bhA} \cdot \text{devALP} \cdot \text{waitA} \cdot \alpha AE} \quad \text{(S35)}$$

$$\text{birthC} = \frac{\mu C \cdot (\mu C + waitC) \cdot (\mu CE + bhC) \cdot (\mu CLP + devCLP)}{\text{bhC} \cdot devCLP \cdot waitC} \quad \text{(S36)}$$

Briefly, to calculate R_0, we first calculated the disease-free equilibrium (DFE) proportions of the host and vector population stages. The vaccinated lamb and sheep populations were set to 0 for the R_0 calculation. We then linearized the infectious subsystem using the birth rates calculated as described above. We calculated the Jacobian matrix and decomposed it into the T matrix (the transmission parameters that describes the production of new infections) and Σ matrix (the development part that describes changes in state) [16]. The NGM with large domain $K_L = -T\Sigma^{-1}$ was calculated and we used the NGM with small domain K_S (using only the rows and columns of K_L belonging to new states-at-infection) to calculate R_0. We then solved R_0 numerically using set population sizes, the mean population size of sheep and lambs and for each species of vector the 34-year mean population and the mean annual peak population size, calculated across timesteps during which there was at least one adult mosquito present. Though the mean peak populations of *Aedes* and *Culex* occurred at different times in the simulation, the estimate of R_0 did not allow the incorporation of temporal variation in the populations and therefore we included the mean peak populations from each mosquito species.

We investigated the sensitivity of R_0 at fixed host and vector population sizes by varying a single parameter in increments of 25% from 50% below and above the parameter value used in the model (Table S4) while holding the remaining parameters constant. All parameters were included in the sensitivity analysis. We used the 14 parameters that were varied for the Latin hypercube sensitivity analysis (Table S5) to examine the range of R_0 values that could be generated as each parameter was varied. For seven of these variables, *Aedes* and *Culex* mortality, bite rates and *Aedes*- and *Culex*-to-host and transovarial transmission fraction, we conducted a sensitivity analysis (parameters were varied from 0.25 and 0.5 times below to above the value used in the simulation) to examine their effects on R_0 in populations of *Aedes* and *Culex*, only *Aedes*, or only *Culex* at the mean and mean peak mosquito population levels. To explore the parameter space around transovarial transmission we produced a contour plot of transovarial transmission, *Aedes* bite rate (0.01-0.45) and seroprevalence. Specifically, we investigated the effect of varying TOT with *Aedes* bite rate on mean seroprevalence, long-term (34-year) persistence, short-term ($R_0 \geq 1$) persistence, the seasonal R_0, and when the seasonal R_0 was at unity. We defined the seasonal R_0 as the value of R_0 when transovarial transmission was set at zero and all other parameters were the same as used for the in the R_0 calculation. Seasonal R_0 was used to examine whether transovarial transmission was necessary for long-term persistence.

The vaccination threshold that drove RVFV to extinction was identified using simulation (18%). To explore the extinction timeframe indicated by the vaccination simulations we ran an additional simulation to evaluate the system at 13.5% (25% lower than the rate identified by simulation) and 54% (three times higher than the rate identified by simulation) vaccination coverage to highlight how infection can persist in the face of very low vaccination coverage and how very high coverage can drive RVFV extinct quickly. In our simulations, we maintained a constant rate of vaccination in order to achieve a constant coverage. The persistence of RVFV in the host and *Aedes* egg populations was assessed for varying vaccination coverage percentages. For the host population, a conservative estimate of greater or equal to one infected host per year indicated persistence. In the *Aedes* egg population, we ran several different simulations varying the initial population of the infected *Aedes* eggs to determine the population threshold required to support viral expansion in the host population. We found that a population of 14,400 infected eggs was sufficient support RVFV expansion. This threshold was used to determine whether there was a sufficient infected *Aedes* egg population to infect hosts in the presence of a sufficiently susceptible host population.

Data and Analysis

We used daily rainfall data from January 1, 1983 to April 30, 2017 from a representative farm in the western Free State (latitude: -28.40, longitude: 26.26; rounded for privacy) extracted from National Oceanic and Atmospheric Administration - Climate Prediction Center (NOAA-CPC) African Rainfall

Climatology (ARC) data records [17, 18]. The ARC data were produced using a combination of METEOSAT cold cloud duration satellite data and rainfall gauge measurements to produce gridded rainfall estimates at a 0.1° x 0.1° spatial resolution since 1983 (see Figure S1A). The temperature data were collected by the nearest local weather stations maintained under the South African Weather Service (SAWS) at the Glenn College site in Bloemfontein, Free State (latitude: -28.95, longitude: 26.33). The stations (Glen College AWS and Glen College AGR) provided the maximum and minimum temperatures in degrees Celsius. As it was the more complete data set, the Glen College AGR station data were used, and the Glen College AWS data were only used if data were missing from the AGR station (SAWS unpublished data, 2017). Temperature data were available from April 1, 1915, to August 2017; however, the dates used were restricted to match the timeframe of the rainfall data (see Figure S1B).

The target seroprevalence was estimated based on the collated seroprevalence data associated with the Clark et al.[19] review of all studies done across Africa. We limited the data to studies that were classified as interepidemic studies. One study, Ksiazek et al.[20], was noted to be misclassified as it is presenting outbreak data and the data from this study was excluded. For a second study, we noted that the incorrect seroprevalence was listed in the Clark et al. [19]review, (the reference to Mroz et al.[21], listed the seroprevalence as 50%, but upon further review of the study, it was noted that the seroprevalence reported was 12.6% in cattle) and we corrected this. With the updated dataset, we calculated the 95th percentile interval (2.5-97.5%) of the data per ruminant species and for the combined ruminant distribution. We used the percent interval calculated for sheep (1-40%) as our target seroprevalence range for the system (excluding 0% as this indicates there was no RVFV persistence.

Data analysis was completed in R version 4.1.2 [22] using Rstudio [23]. The package zoo 1.8-8 [24] was used to calculate the cumulative rainfall data, the ode solver was used from the deSolve 1.28 package [25], the climate data and other daily data (e.g. the hatching triggers) were introduced into the simulation through the use of the approxfun() function (deSolve) and the figures were made using ggplot2 3.3.2 [26]. The lhs 1.1.1 package [27] was used to produce the Latin hypercube for the sensitivity analysis. The resultant data were analyzed by calculating the partial correlation coefficients using the pcc() function from the sensitivity 1.22.1 package [28]. wxMaxima 5.43.0 [29] was used to conduct the R_0 analysis.

Supplemental Tables

Table S1 Definition of the state variables used in the model.

Variable	Population represented
Sheep and Lambs	
AL	Lambs with temporary maternal immunity
SL	Susceptible lambs
IL	Infected lambs
RL	Recovered lambs
VL	Vaccinated lambs
SS	Susceptible sheep
IS	Infected sheep
RS	Recovered sheep
VS	Vaccinated sheep
Aedes Mosquitoes	
SA	Susceptible adult *Aedes*
EA	Exposed adult *Aedes*
IA	Infected adult *Aedes*
SAY	Susceptible young *Aedes* that have not had their first blood meal
IAY	Infected young *Aedes* that have not had their first blood meal
SALP	Susceptible *Aedes* larvae/pupae
IALP	Infected *Aedes* larvae/pupae
newSAE	Newly laid susceptible *Aedes* eggs that cannot hatch yet
SAE	Susceptible *Aedes* eggs that can hatch
newIAE	Newly laid infected *Aedes* eggs that cannot hatch yet
IAE	Infected *Aedes* eggs that can hatch
Culex Mosquitoes	
SC	Susceptible adult *Culex*
EC	Exposed adult *Culex*
IC	Infected adult *Culex*
SCY	Susceptible young *Culex* that have not had their first blood meal
SCLP	Susceptible *Culex* larvae/pupae
SCE	Susceptible *Culex* eggs

Table S2. Parameter values for the climate forcing to drive the hatching and development of the mosquitoes as described in the text.

Description	Value	Unit	Reference
Number of days that *Aedes* eggs can hatch	9	days	[8]
Number of cumulative days of rainfall needed to initiate hatching of *Aedes* eggs	7	days	Preliminary simulation*
Minimum amount of rainfall over 7 days, above which *Aedes* can hatch	38	mm	Preliminary simulation*
Minimum temperature above which both *Aedes* and *Culex* eggs can hatch	20	Celsius	[10]
Number of cumulative days of rainfall needed to initiate and maintain hatching of *Culex* eggs	3	days	Preliminary simulation*
Minimum amount of rainfall over the 3 days, above which *Culex* can hatch	2	mm	Preliminary simulation*
Number of days after the *Aedes* eggs start hatching that the *Culex* can start hatching (H)	7	days	[8]

*Preliminary simulation was used to identify parameter estimates that supported stable vector population dynamics and is described in the Climate-Driven Hatching and Development Rates Section of the Supplemental Information.

Table S3. Parameter values for calculating the larval development rates (Eq. S2). Rueda *et al.* [10] confirmed the Sharpe & DeMichele model [30] with high temperature inhibition with laboratory raised *Aedes* and *Culex* larvae. Their model was built to estimate the development rate per day as the temperature changed (Eq. S2).

Variable	Value	Description	Reference
ρ_{025_A}	0.1546	Effect of temperature on *Aedes* larval development estimated by a linear regression	[10]
HA_A	33255.57	Effect of temperature on *Aedes* larval development estimated by a linear regression	[10]
TH_A	301.67	Effect of temperature on *Aedes* larval development estimated by a linear regression	[10]
HH_A	50543.49	Effect of temperature on *Aedes* larval development estimated by a linear regression	[10]
f_{Am}	1.392704	Factor by which *Aedes mcintoshi* develops faster than *Culex quinquefasciatus*	Preliminary simulation*
ρ_{025_C}	0.21945	Effect of temperature on *Culex* larval development estimated by a linear regression	[10]
HA_C	28049.98	Effect of temperature on *Culex* larval development estimated by a linear regression	[10]
TH_C	298.6	Effect of temperature on *Culex* larval development estimated by a linear regression	[10]
HH_C	35362.18	Effect of temperature on *Culex* larval development estimated by a linear regression	[10]
f_{Cp}	0.7650069	Factor by which *Culex pipiens* develops faster than *Culex quinquefasciatus*	Preliminary simulation*

*Preliminary simulation was used to identify parameter estimates that supported stable vector population dynamics and is described in the Climate-Driven Hatching and Development Rates Section of the Supplemental information.

Table S4. Parameter values for the SEIR model of mosquitoes, sheep, and lambs (Eqs. S4-S33).

Parameter	Value	Description	Unit	Reference
σ	0.25	Recovery rate	days^{-1}	[31]
g	0.005	Maturation rate of lambs to sheep	days^{-1}	[32]
μS	0.00014	Sheep mortality rate	days^{-1}	Rostal *et al* unpublished†
$soldS$	0.002	Percent of sheep sold	days^{-1}	Set to maintain sheep population constant
ρS	0.063	RVFV specific mortality rate in sheep	days^{-1}	[33]
bL	0.004	Birth rate in sheep	days^{-1}	[34]
$buyL$	0.508	Number of lambs purchased	days^{-1}	Rostal *et al* unpublished†
μL	0.00004	Lamb mortality rate	days^{-1}	Rostal *et al* unpublished†
ρL	0.751	RVFV specific mortality rate in lambs	days^{-1}	[35]
Abort	0.9	Abortion fraction among pregnant ewes infected with RVFV	No units	[33]
ωMA	0.008	Maternal antibodies Waning rate	days^{-1}	[36]
vax.prop	0	Proportion of the sheep and lamb populations desired to be vaccinated	No units	Set by user as needed
vax	0	The daily rate of vaccination to maintain *vax.prop*: $\frac{vax.prop \cdot (mu_L + g)}{(1 - vax.prop)}$	days^{-1}	Calculated from *vax.prop*
$NLmax$	800	Herd size maintained by the carrying capacity acting on lambs	No units	Rostal *et al* unpublished†
$EgAE$	0.333	Egg laying Rate for *Aedes circumluteolus*	days^{-1}	[37]
$EgNAE$	34.583	Number of female eggs per adult Asian *Aedes lineatopennis*	No units	[38]
μA	0.07	*Aedes* mortality rate	days^{-1}	[9, 39]
μAE	0.0008378592	*Aedes* egg mortality rate	days^{-1}	Latin hypercube estimation‡
$NALPmax$	223,880.6	Carrying capacity acting on the number of *Aedes* larvae and pupae that can survive in the available habitat	No units	Latin hypercube estimation‡
ϕA	0.36	Fraction of *Aedes mcintoshi* larvae/pupae that survive	No units	[7]
bhA	0.23	Rate that *Aedes* eggs hatch based the approximately 5-day interval between flooding and initial larval detection in the pan	days^{-1}	[8]
αAE	0.033	The rate at which newly laid eggs desiccate and become able to hatch	days^{-1}	Set to prevent newly laid eggs from hatching during the same annual hatching period in which they were laid

Parameter	Value	Description	Unit	Reference
$devALP$	0.068	Mean development rate of *Aedes* - calculated daily based on ambient temperature (Eq. S2)	days^{-1}	[10]
$waitA$	0.333	Reciprocal of the number of days (3) it takes before a newly emerged *Aedes* takes its first bite (delay due to mating, host-seeking etc.)	days^{-1}	[40-42]
μALP	0.12	Mean mortality rate in *Aedes* larvae/pupae on days when there was at least 1 susceptible or infected *Aedes* larvae/pupae - calculated daily based on ambient temperature (Eq. S3)	days^{-1}	Estimated from $devALP$
biteA	0.07108191	*Aedes* bite rate on the flock of sheep	days^{-1}	Latin hypercube estimation‡
ϵ	0.083	Extrinsic incubation rate in mosquitoes	days^{-1}	[43]
θASL	0.41	Probability of transmission to *Aedes lineatopennis/mcintoshi* from the host	No units	[44, 45]
θSLA	0.16	Probability of transmission to the host from *Aedes lineatopennis/mcintoshi*	No units	[44]
q	0.7444736	Transovarial transmission fraction	No units	Latin hypercube estimation‡
$EndA$	[1,0]	When the value is one, *Aedes* eggs hatch; represents the univoltine hatching of *Aedes*	No units	Estimated by climate thresholds
$EgCE$	0.333	Egg laying rate of *Culex*	days-1	[43]
$EgNCE$	118	Mean number of female eggs laid by *Culex*	No units	[11]
δ	0.8	Fraction of RVFV infected *Culex* that lay eggs	No units	[2]
ϕC	0.291	Fraction of *Culex* that survive larval/pupal stages	No units	[11]
bhC	0.667	Reciprocal of the *Culex* hatching time	days^{-1}	[11]
$devCLP$	0.06	Mean development time for *Culex* pupae/larvae - calculated daily based on ambient temperature (Eq. S2)	days^{-1}	[10]
$waitC$	0.333	Reciprocal of the number of days (3) it takes before a newly emerged *Culex* takes its first bite (delay due to mating, host-seeking etc.)	days^{-1}	[40-42]
μC	0.063	Adult *Culex* mortality rate	days^{-1}	[9, 43, 46]
μCLP	0.145	Mean mortality rate in *Culex* larvae/pupae - calculated daily based on ambient temperature (Eq. S3)	days^{-1}	Estimated from $devCLP$
μCE	0.146	*Culex* egg mortality rate - calculated using $\mu_{CE} = bh_C \cdot \frac{1-\psi_C}{\psi_C}$	days^{-1}	Estimated as per the equation
$NCLPmax$	110,013.6	Carrying capacity acting on the number of *Culex* larvae and pupae that can survive in the available habitat	No units	Latin hypercube estimation‡
θCSL	0.71	Probability of transmission to *Culex* from the host	No units	[44, 45, 47-50]
θSLC	0.31	Probability of transmission to the host from *Culex*	No units	[44, 45, 50]

Parameter	Value	Description	Unit	Reference
biteC	0.04952358	*Culex* bite rate on the sheep	$days^{-1}$	Latin hypercube estimation‡
hatchC	0-1	When the value is greater than zero, *Culex* eggs hatch at the specified proportion	No units	Estimated by climate thresholds

†Latin hypercube was used to identify parameter estimates that produced realistic patterns of RVF epidemics, seroprevalences and RVFV persistence as described in the Parameters Used in the Model Section of the Supplemental information.
‡The unpublished survey data referenced as Rostal et al. is described in the Parameters Used in the Model Section of the Supplemental information.
Bold font indicates a time-varying parameter.

Table S5 Ranges of parameters used in the Latin hypercube and R_0 sensitivity analyses. The parameters were varied across a range that was 50% above and below the value used in the exemplar simulation (with a maximum of 1.0 if 50% above a proportion was greater than 1.0).

Parameter	Range used in Latin hypercube
σ	0.125-0.375
ρS	0.0315-0.0945
ρL	0.3755-1.0
ϵ	0.0415-.01245
$biteA$	0.06-0.18
θASL	0.205-0.615
θSLA	0.08-0.24
μA	0.035-0.105
μAE	0.00005-.0015
q	0.35-1.0
$biteC$	0.0175-0.0525
θCSL	0.35-1.0
θSLC	0.0175-0.155
μC	0.0315-.0945

Table S6 Simulated estimates of the mean annual seroprevalence, mean annual maximum host-vector ratio, and the mean proportion of infected mosquitoes and eggs across the entire exemplary simulation.

Factor	Mean (Range)
Mean annual seroprevalence	22.8% (10.4-58.6%)
Mean annual maximum host-vector ratio	1:180 (1:51-1:286)
Mean annual proportion of infected *Aedes* eggs	0.003 (0.002-0.003)
Mean annual proportion of infected adult *Aedes*	0.007 (0.002-0.04)
Mean annual proportion of infected adult *Culex*	0.002 (0-0.035)

Supplemental Figures:

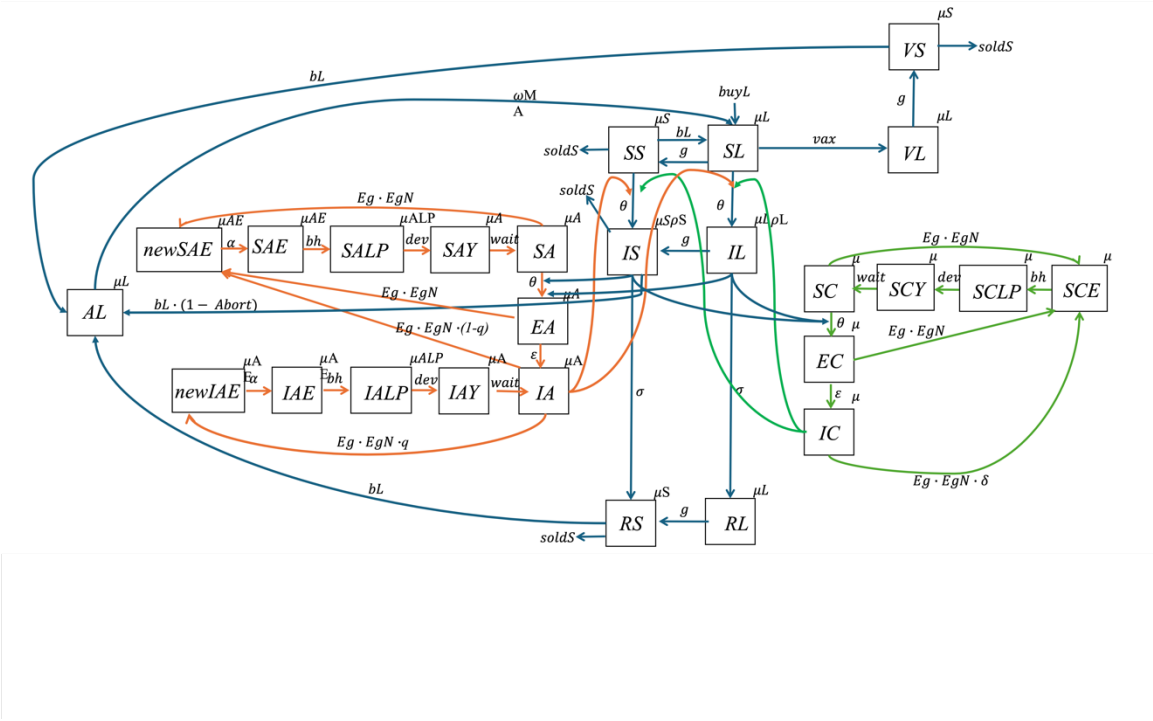

Fig. S1 A diagram of the RVFV transmission model. The infectious states of Susceptible (S), Exposed (E), Infected (I), Maternal Antibodies (A) and Vaccinated (V) are represented for sheep (S), lambs (L), *Aedes* vectors (A) and *Culex* vectors (C). All of the life stages of are represented. For the *Aedes* populations we have susceptible and infected new ($newSAE$, $newIAE$) and mature eggs (SAE, IAE); larvae/pupae ($SALP$, $IALP$); young (non-feeding) adults (SAY, IAY) and adult *Aedes* (SA, IA), respectively, as well exposed adults (EA). For the *Culex,* we have susceptible eggs (SCE), larvae/pupae ($SCLP$), young (non-feeding) adults (SCY) and adult *Culex* (SC). *Aedes* eggs are initially laid in a compartment of new eggs that require desiccation ($newSAE$, $newIAE$) before moving to the infected egg compartments that can hatch (SAE, IAE). For the hosts, we have susceptible (SS), infected (IS), recovered (RS) and vaccinated (VS) sheep. We have susceptible (SL), infected (IL), recovered (RL) and vaccinated (VL) lambs as well as lambs with maternal immunity (AL). The state variables are defined in Table S1, and the parameters are defined in Table S4.

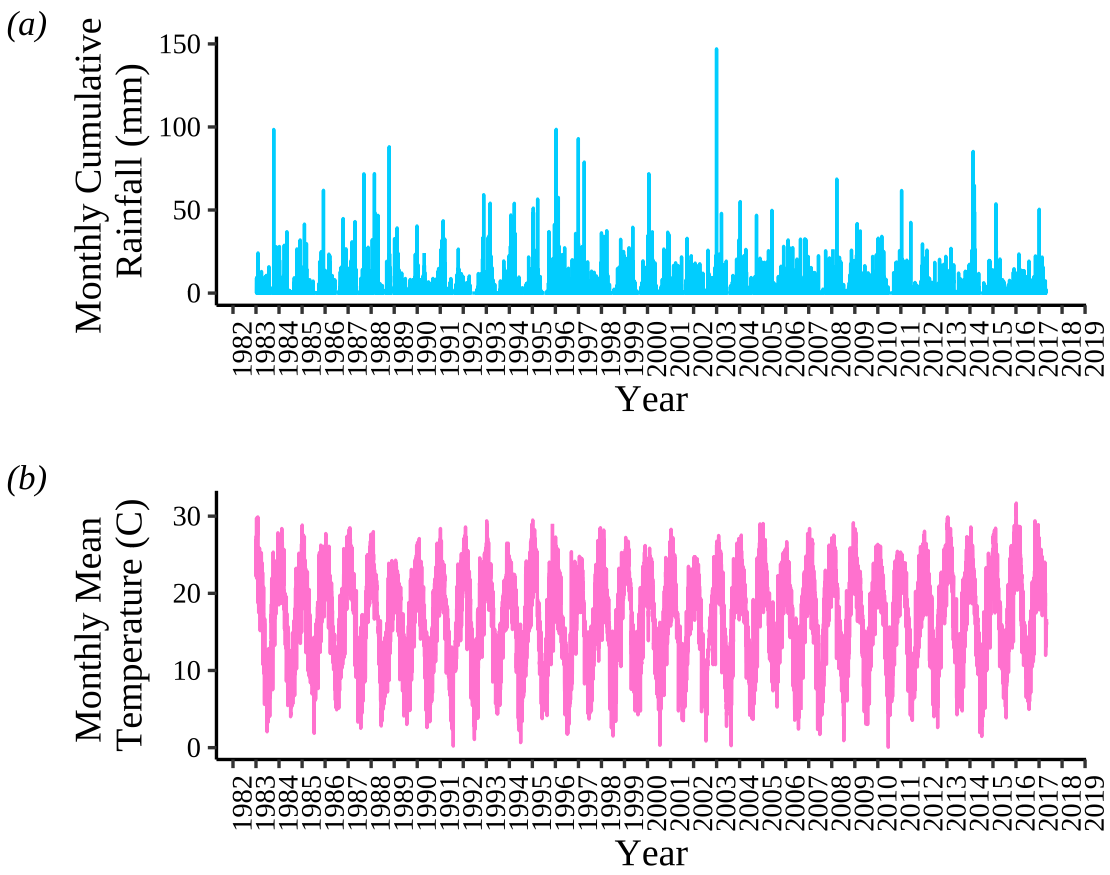

Fig. S2 Monthly A) total rainfall and B) mean temperature by year from 1983-2017.

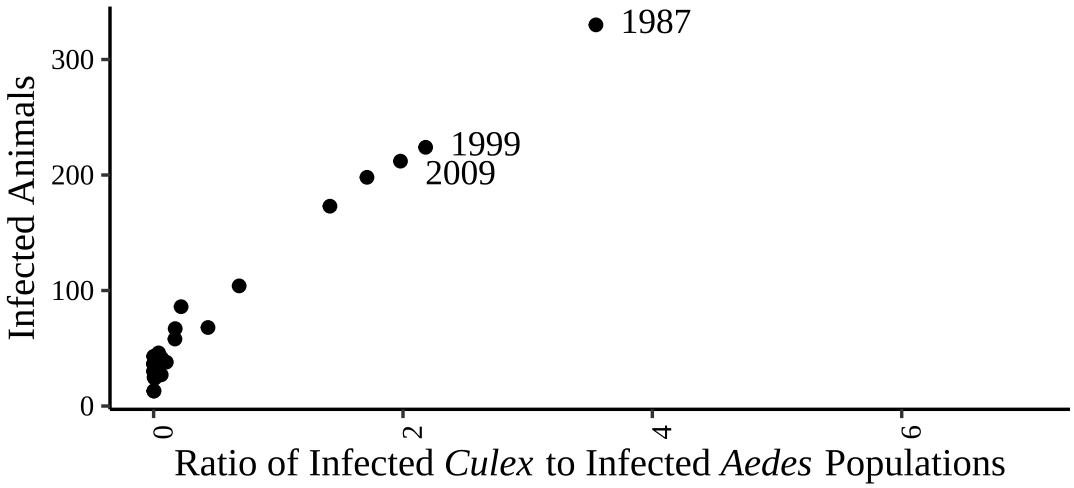

Fig. S3 The total number of infected hosts by the peak population sizes of infected Culex and Aedes for each year of the simulation. The year of the outbreak is indicated for all outbreaks with a ratio of infected Culex and Aedes >2.

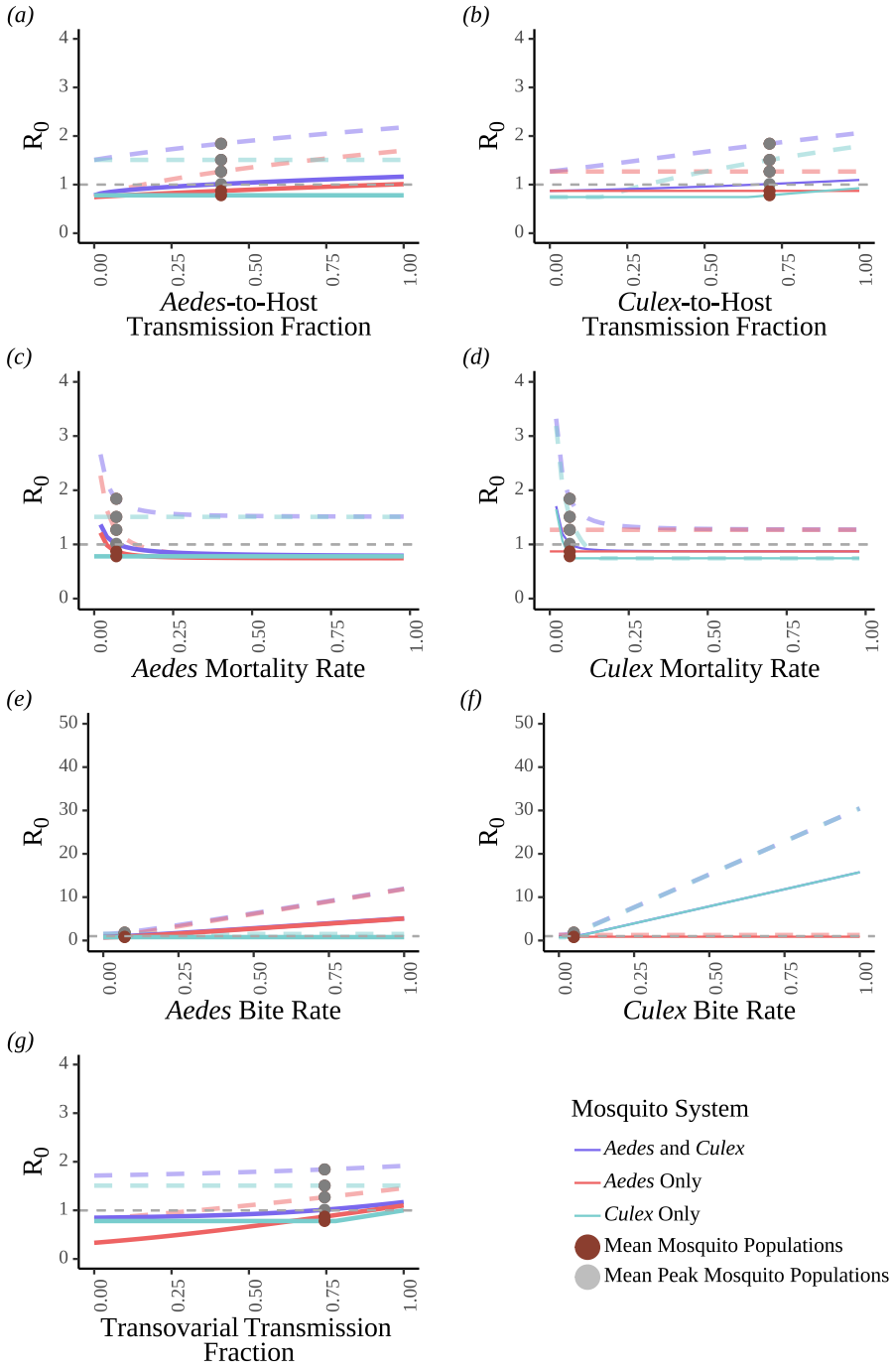

Fig. S4 Sensitivity analysis of R_0 illustrate how R_0 changes in relation to A) Aedes-host transmission fraction; B) Culex-host transmission fraction; C) the Aedes mortality rate; D) Culex mortality rate; E) Aedes bite rate; F) Culex bite rate and G) transovarial transmission fraction. The red line represents the system with Aedes mosquitoes only, the teal line represents the system with Culex mosquitoes only and the purple line represents the system with both Aedes and Culex mosquitoes. The solid lines indicate R_0 at the mean mosquito population, whereas the dashed lines represent the value of R_0 using the mean peak mosquito population. The brown dots indicate the R_0 at the parameter value used in our simulation. The dashed gray line indicates R_0 equal to one.

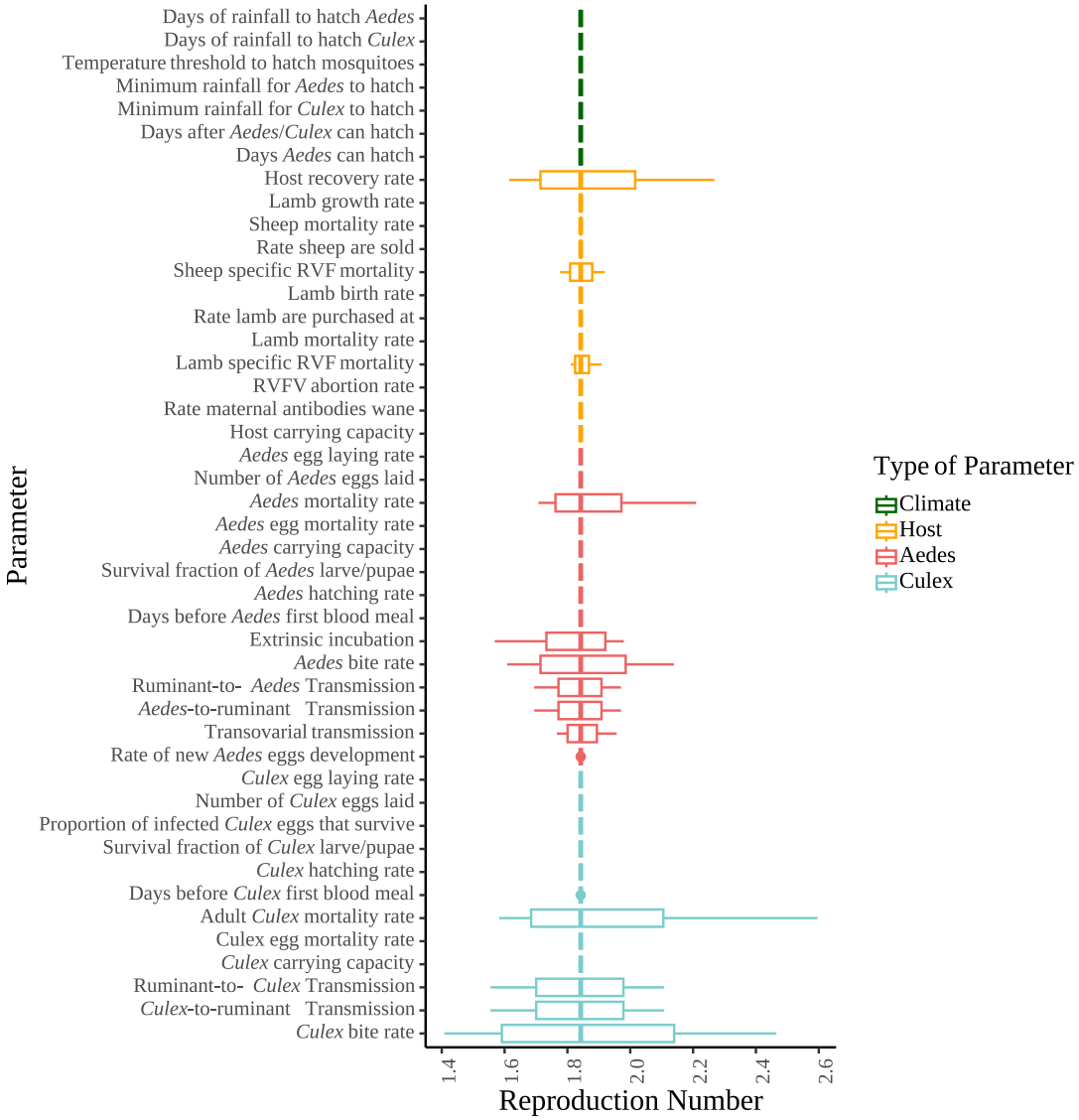

Fig. S5 The value of R_0 across a range of values for each parameter used in the model and calculated at a fixed vector and host population size. The parameters are characterized as those impacting the hatching of the mosquitoes (climate [green]), and those impacting the populations of the hosts (orange), *Aedes* (red) and *Culex* (light blue).

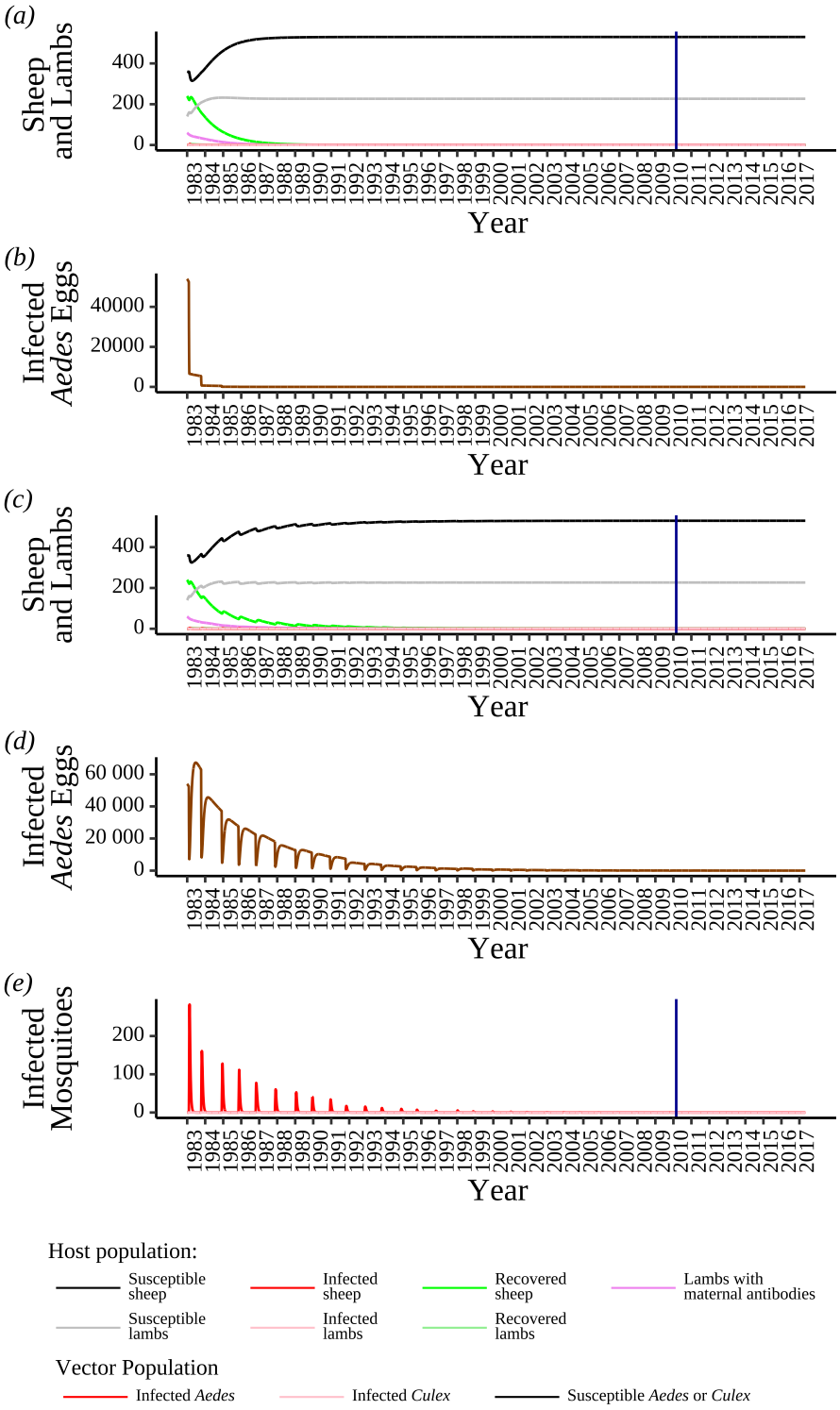

Fig. S6 The dynamics of RVFV in sheep (A) and infected *Aedes* egg (B) populations in a system with no transovarial transmission. The dynamics of RVFV in sheep (C), infected *Aedes* egg (D) and infected mosquito (E) populations in a system with no horizontal transmission. The gray line indicates the estimated threshold for a population of infected *Aedes* eggs to establish RVFV infection in the host population.

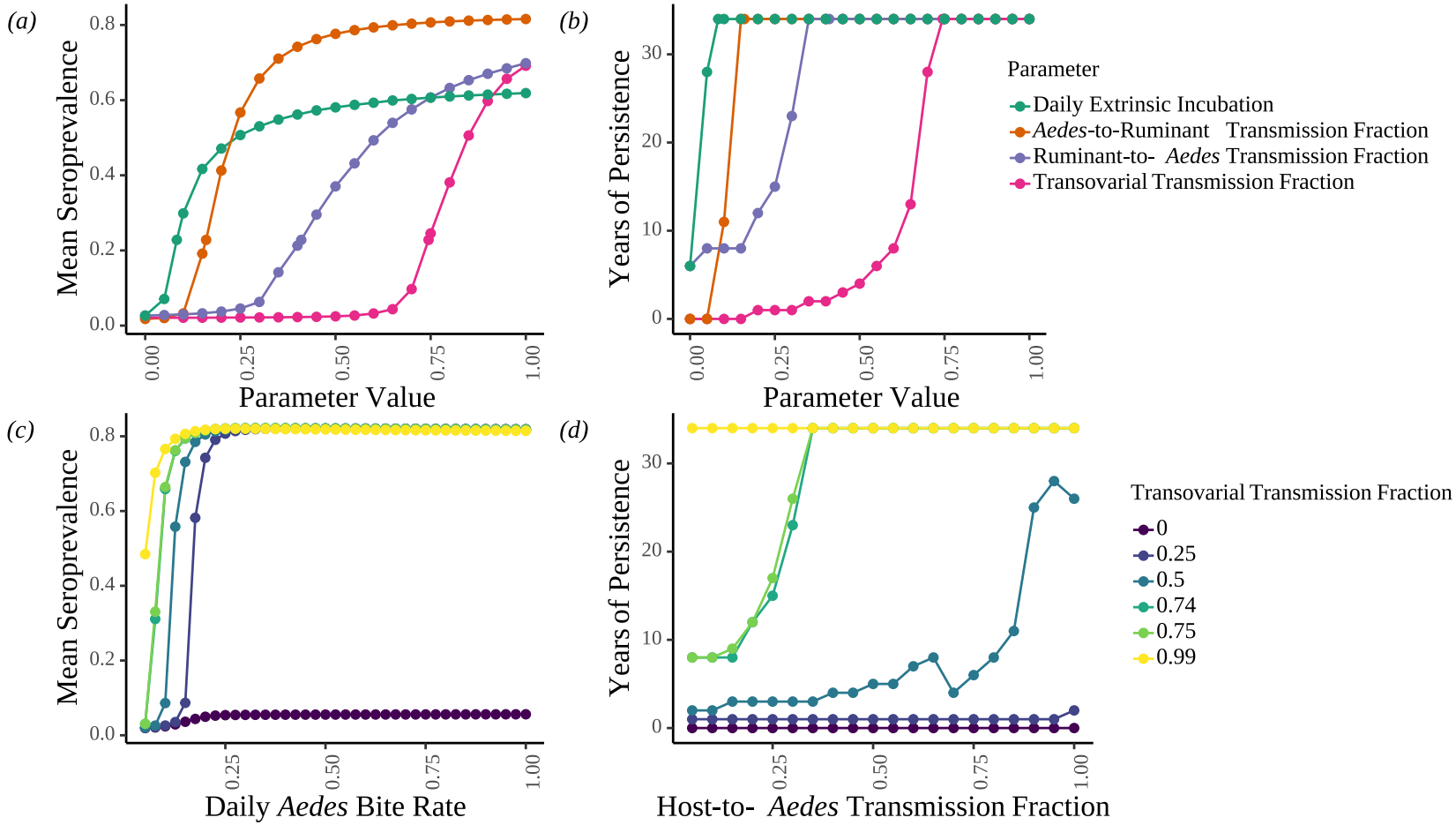

Fig. S7 The sensitivity of the mean seroprevalence (A) and persistence (B) of the system to the transovarial transmission fraction, horizontal transmission parameters, *Aedes* bite rate and the extrinsic incubation rate. The seroprevalence and persistence of RVFV in the system are more sensitive when considering the parameters together, e.g., sensitivity of seroprevalence to the *Aedes* bite rate and transovarial transmission fraction (C) and persistence to the host-to-vector and transovarial transmission fractions (D).

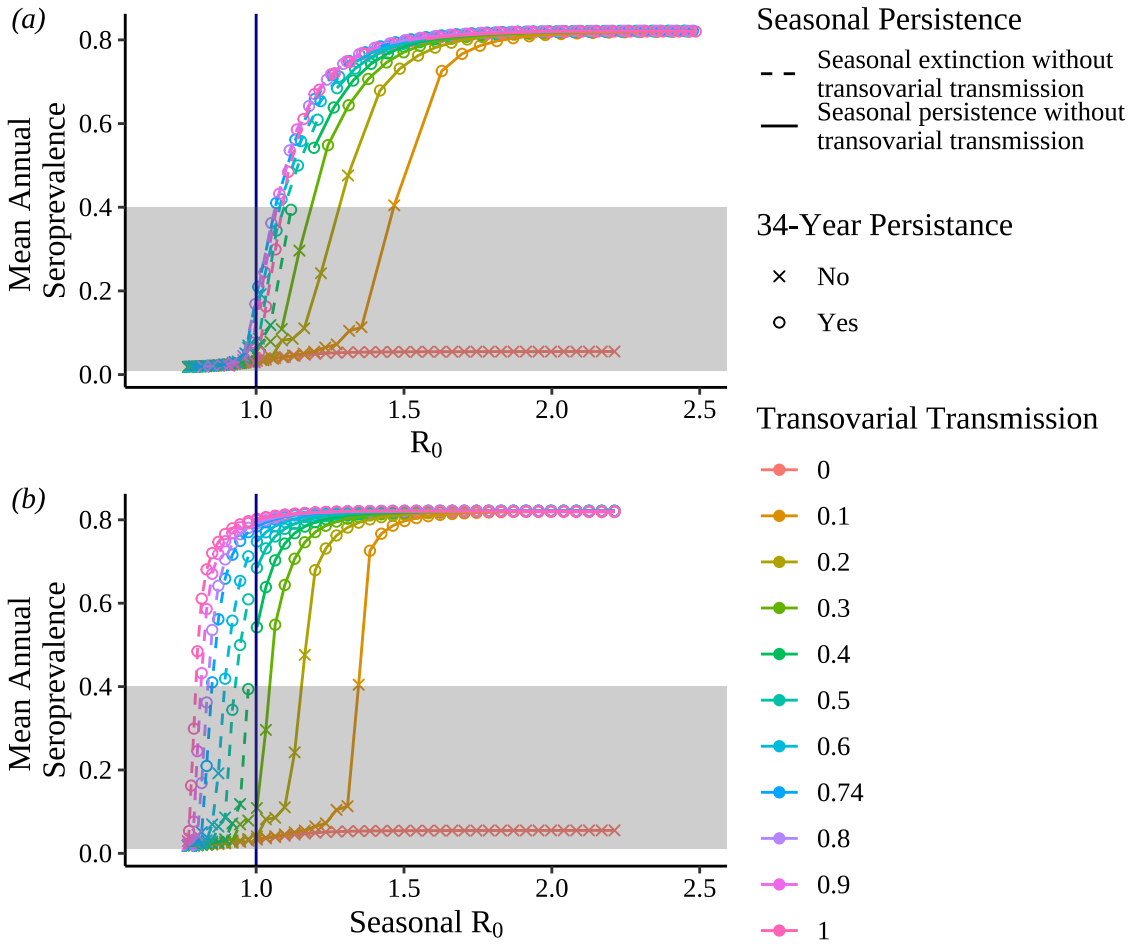

Fig. S8 Changes in mean annual seroprevalence and R_0 as transovarial transmission and *Aedes* bite rate (0.01-0.45 bites per day) changes. Persistence occurs once R_0 crosses unity (top) with mean annual seroprevalences in the desired range (5-40%, shaded area) obtained for transovarial transmission fraction values > 0.5. However, for these parameter ranges, the seasonal R_0 (i.e., the estimated R_0 excluding the contribution of transovarial transmission) is < 1 (bottom). When the seasonal R_0 exceeds one, this leads to large outbreaks and a lack of long-term persistence. Thus, achieving long term persistence and observed seroprevalences appears to require limited seasonal spread and an overwintering mechanism via transovarial transmission which boosts R_0 above unity. Simulations with long-term RVFV persistence in the host population (the final year of the simulation) are indicated by points, whereas "x" indicates RVFV did not persist. Simulations with seasonal persistence (seasonal R_0 is greater than unity) are indicated by the dashed lines and those that did not have seasonal persistence are indicated with a solid line. The blue line indicates R_0 at unity.

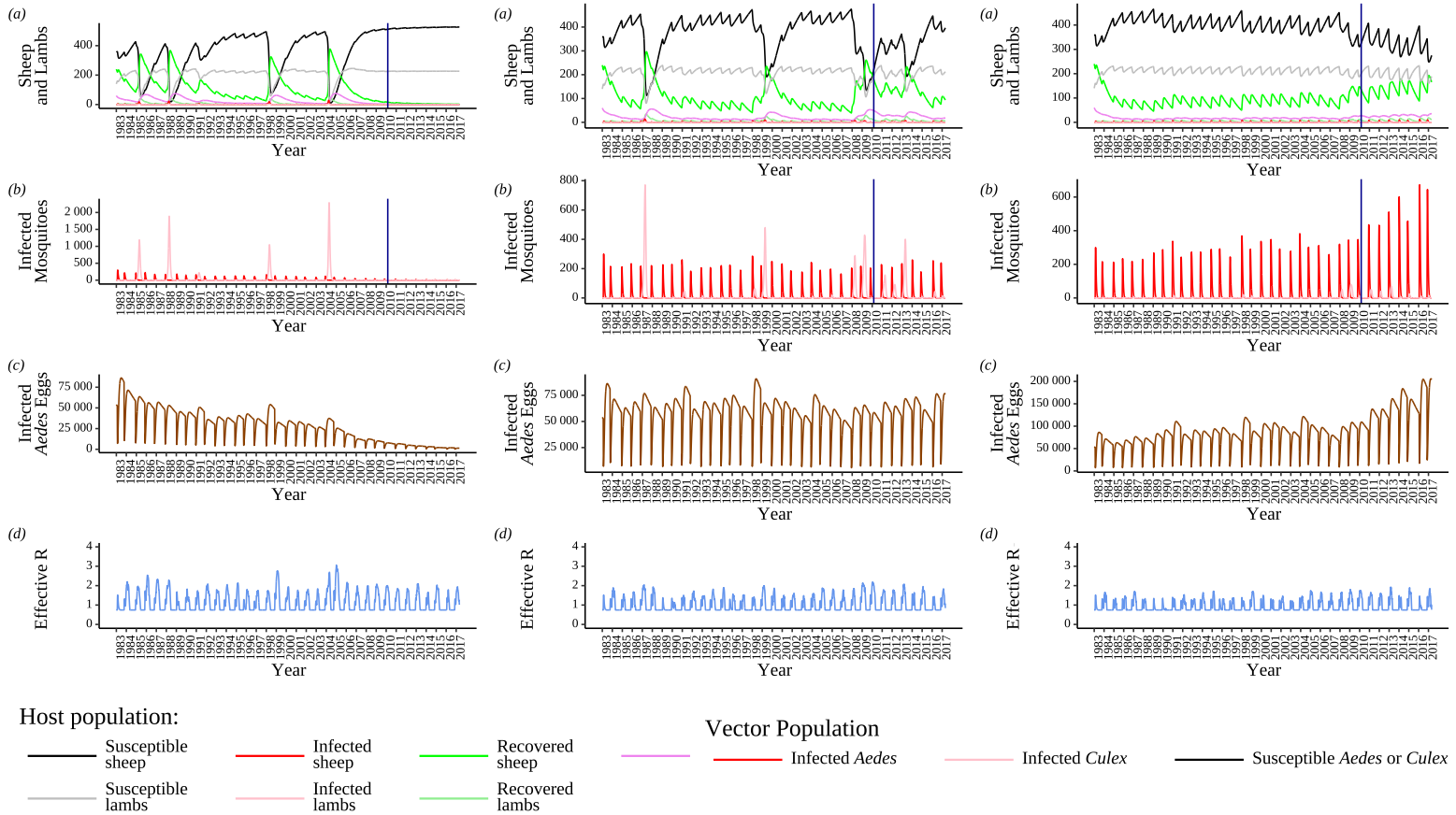

Fig. S9 The full 34-year simulation of A) hosts, B) infected mosquitoes, C) infected *Aedes* eggs and D) the effective reproduction number using 75% of the *Culex* mortality rate (left), the *Culex* mortality rate at the value used for the model simulation (middle) and 125% of the *Culex* mortality rate (right).

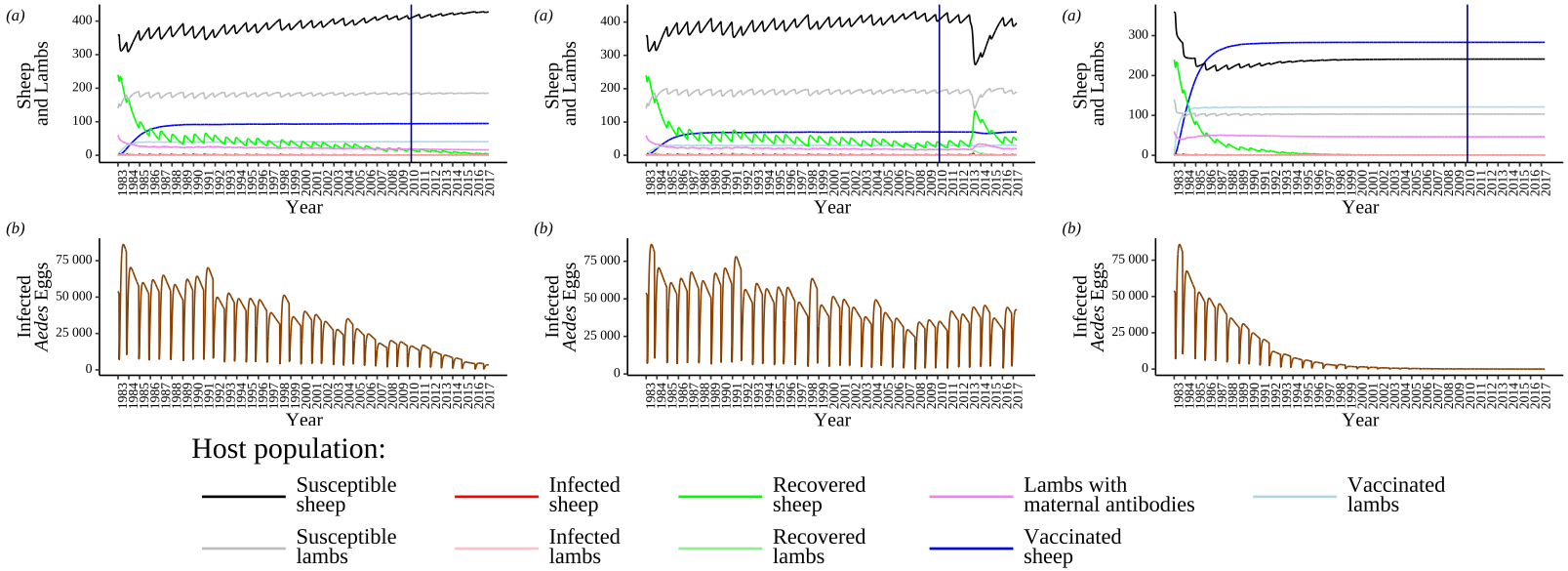

Fig. S10 A full 34-year simulation of A) hosts and B) infected *Aedes* eggs when Left: the proportion of animals vaccinated is maintained at 13.5%, Center: the proportion of animals vaccinated is maintained at 18% and Right: the proportion of animals vaccinated is maintained at 54%.

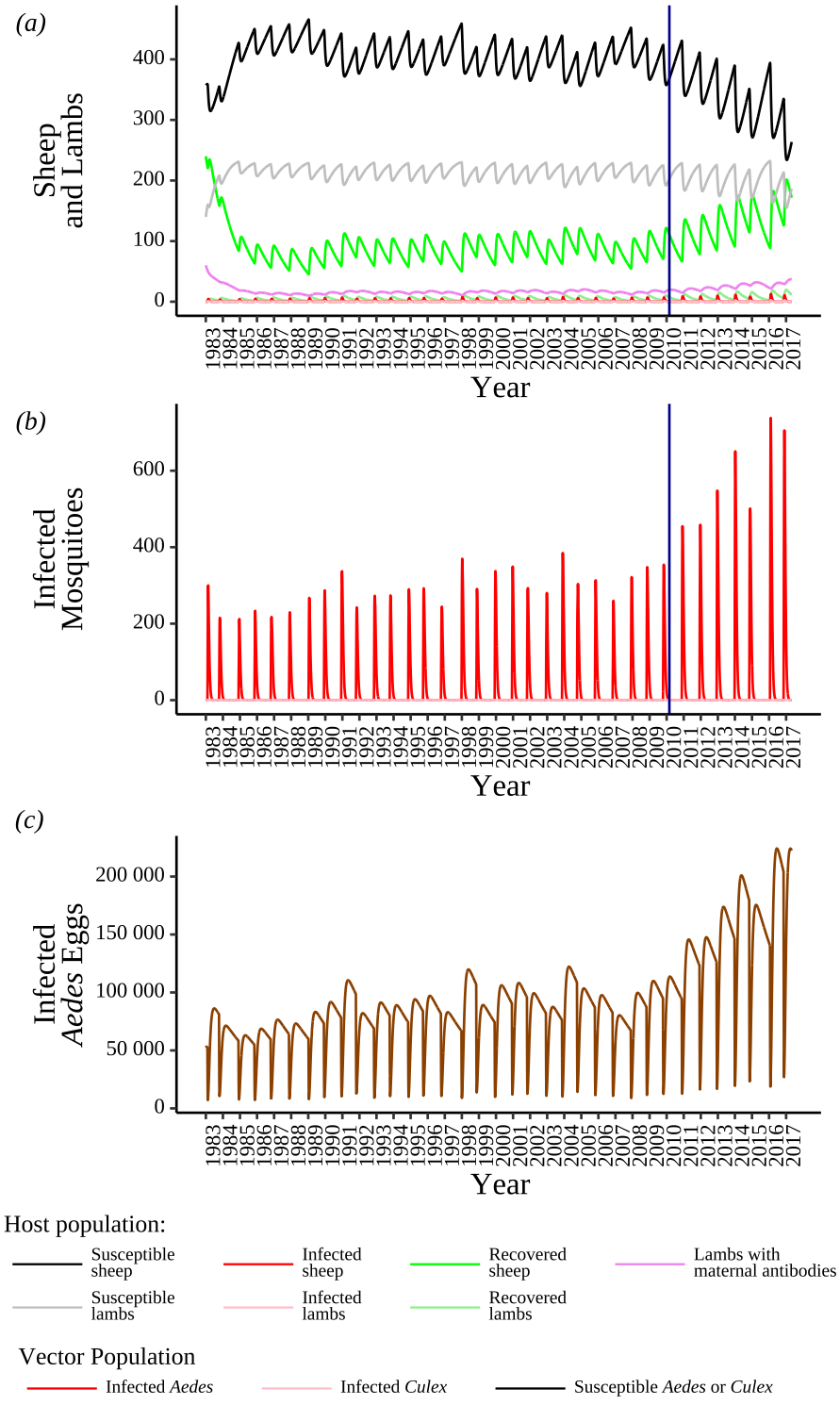

Fig. S11 A simulation of dynamics when only *Aedes* are able to transmit the virus (both vertically and horizontally). The full 34-year simulation of A) hosts and B) infected *Aedes* and C) infected *Aedes* eggs. In this simulation, infection rates remain low (<10%) during the first 20 years. Eventually the infection pressure increases the infection rate in hosts, vectors and eggs. No large outbreaks are seen during this simulation.

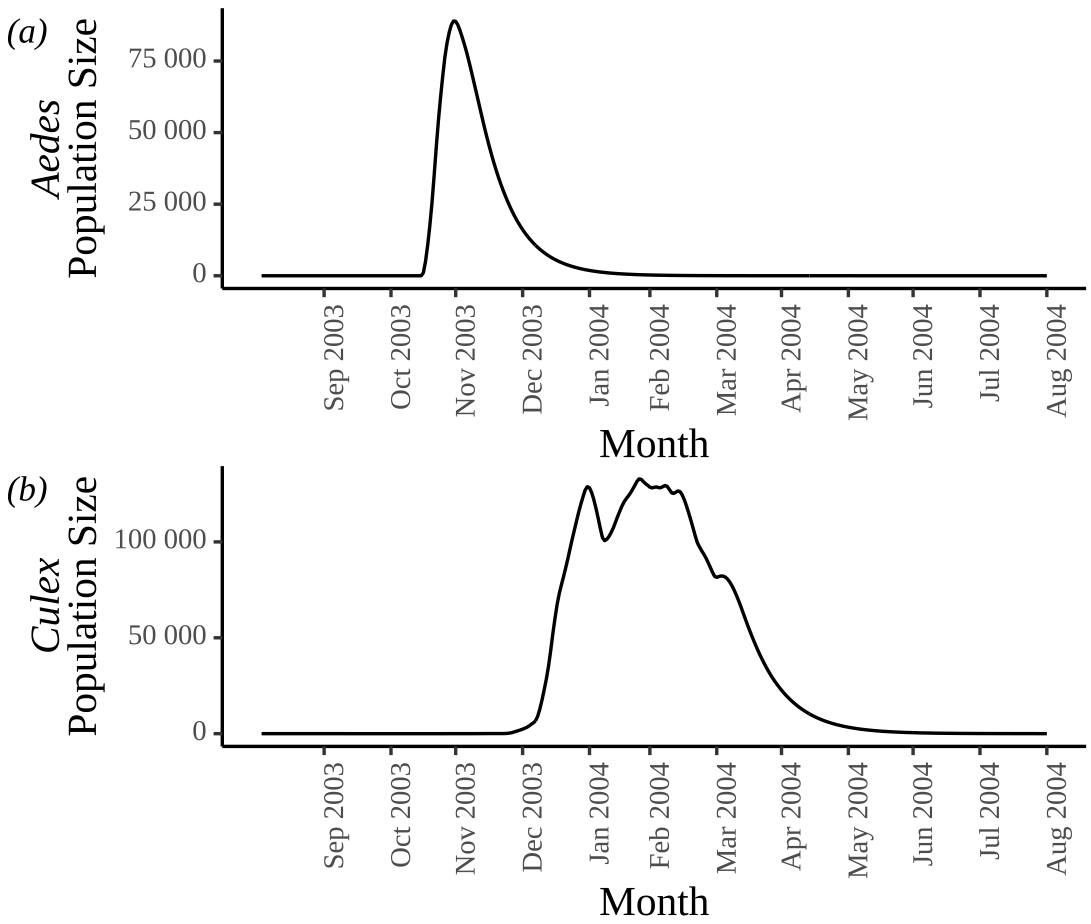

Fig. S12 The total population of adult A) *Aedes* and B) *Culex* mosquitoes over a randomly selected year (September 2003- August 2004).

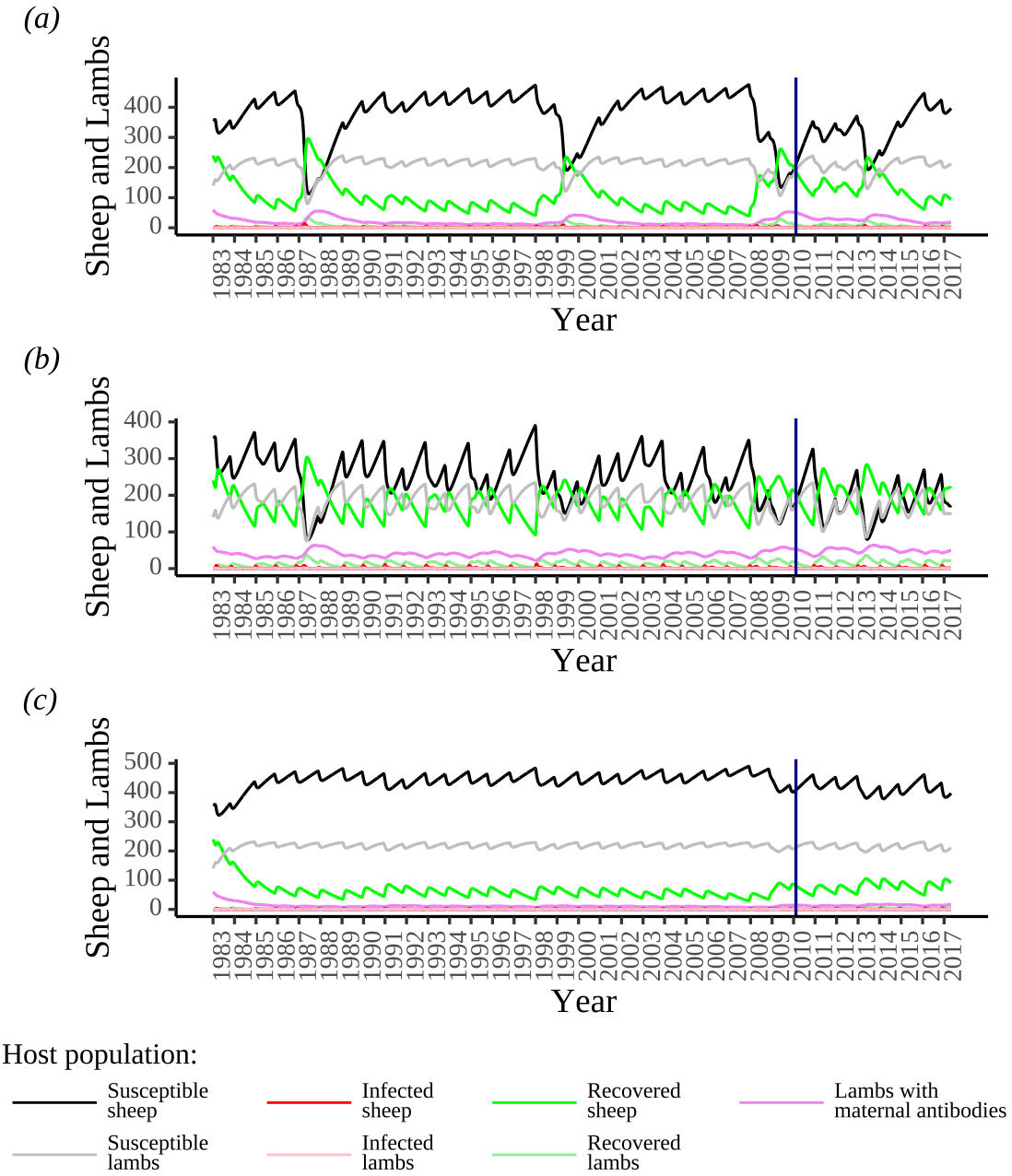

Fig. S13 The simulations to provide parameter values for the six unknown parameters and satisfied the assessment criteria had three broad patterns A) large outbreaks with intervening periods of low-level transmission (our exemplar scenario), B) high rates of annual transmission resulting in frequent large outbreaks, or C) low-level transmission with occasional and small outbreaks.

Dataset S1 (separate file). The data and code for this project are archived with Zenodo, Version v2.0.1. DOI: 10.5281/zenodo.15632526.

Software S1 (separate file). The data and code for this project are archived with Zenodo, Version v2.0.1. DOI: 10.5281/zenodo.15632526.

SI References

[1] Macdonald, G. 1952 The analysis of equilibrium in malaria. *Tropical Diseases Bulletin* **49**, 813-829.
[2] Turell, M.J., Gargan, T.P. & Bailey, C.L. 1985 *Culex pipiens* (Diptera: Culicidae) morbidity and mortality associated with Rift Valley fever virus infection. *Journal of Medical Entomology* **22**, 332-337. (doi:10.1093/jmedent/22.3.332).
[3] Ngoshe, Y.B., Avenant, A., Rostal, M.K., Karesh, W.B., Paweska, J.T., Bagge, W., van Vuren, P.J., Kemp, A., Cordel, C. & Msimang, V. 2020 patterns of Rift Valley fever virus seropositivity in domestic ruminants in central South Africa four years after a large outbreak. *Scientific reports* **10**, 1-13.
[4] Crans, W.J. 2004 A classification system for mosquito life cycles: Life cycle types for mosquitoes of the northeastern United States. *Journal of Vector Ecology* **29**, 1-10.
[5] Linthicum, K.J., Davies, F.G., Kairo, A. & Bailey, C.L. 1985 Rift Valley fever virus (family *Bunyaviridae*, genus *Phlebovirus*). Isolations from diptera collected during an inter-epizootic period in Kenya. *The Journal of Hygiene* **95**, 197-209.
[6] Logan, T., Linthicum, K., Thande, P., Wagateh, J., Nelson, G. & Roberts, C. 1991 Egg hatching of *Aedes* mosquitoes during successive floodings in a Rift Valley fever endemic area in Kenya. *Journal of the American Mosquito Control Association* **7**, 109-112.
[7] Linthicum, K.J., Davies, F.G. & Kairo, A. 1985 Observations on the dispersal and survival of a population of *Aedes lineatopennis* (Ludlow) (Diptera: Culicidae) in Kenya. *Bulletin of Entomological Research* **75**, 661-670.
[8] Linthicum, K.J., Davies, F.G., Bailey, C.L. & Kairo, A. 1984 Mosquito species encountered in a flooded grassland dambo in Kenya. *Mosquito News* **44**, 228-232.
[9] Ba, Y., Diallo, D., Kebe, C.M.F., Dia, I. & Diallo, M. 2005 Aspects of bioecology of two Rift Valley fever virus vectors in Senegal (West Africa): *Aedes vexans* and *Culex poicilipes* (Diptera: Culicidae). *Journal of Medical Entomology* **42**, 739-750.
[10] Rueda, L., Patel, K., Axtell, R. & Stinner, R. 1990 Temperature-dependent development and survival rates of *Culex quinquefasciatus* and *Aedes aegypti* (Diptera: Culicidae). *Journal of Medical Entomology* **27**, 892-898.
[11] Gomez, C., Rabinovich, J.E. & Machado-Allison, C.E. 1977 Population analysis of *Culex pipiens fatigans* Wied. (Diptera: Culicidae) under laboratory conditions. *Journal of Medical Entomology* **13**, 453-463. (doi:10.1093/jmedent/13.4-5.453).
[12] Easterday, B., McGavran, M., Rooney, J. & Murphy, L. 1962 The pathogenesis of Rift Valley fever in lambs. *American Journal of Veterinary Research* **23**, 470-479.
[13] Clements, A.N. 1992 *The biology of mosquitoes: Development, nutrition and reproduction*. London, Chapman & Hall; 509 p.
[14] Marino, S., Hogue, I.B., Ray, C.J. & Kirschner, D.E. 2008 A methodology for performing global uncertainty and sensitivity analysis in systems biology. *Journal of Theoretical Biology* **254**, 178-196. (doi:https://doi.org/10.1016/j.jtbi.2008.04.011).

[15] Drake, J.M. & Rohani, P. 2019 Sensitivity analysis of deterministic models through Latin hypercube sampling: A model for the spread of Ebola virus disease. (pp. 1-11. Mountain View, California, Creative Commons.
[16] Diekmann, O., Heesterbeek, J. & Roberts, M.G. 2010 The construction of next-generation matrices for compartmental epidemic models. *Journal of the Royal Society Interface* **7**, 873–885.
[17] Love, T.B., Kumar, V., Xie, P. & Thiaw, W. 2004 A 20-year daily Africa precipitation climatology using satellite and gauge data. In *14th Conference on Applied Climatology* (p. 5.4, Citeseer.
[18] Novella, N.S. & Thiaw, W.M. 2016 A seasonal rainfall performance probability tool for famine early warning systems. *Journal of Applied Meteorology and Climatology* **55**, 2575-2586.
[19] Clark, M.H., Warimwe, G.M., Di Nardo, A., Lyons, N.A. & Gubbins, S. 2018 Systematic literature review of Rift Valley fever virus seroprevalence in livestock, wildlife and humans in Africa from 1968 to 2016. *PLoS Neglected Tropical Diseases* **12**, e0006627.
[20] Ksiazek, T., Jouan, A., Meegan, J., Le Guenno, B., Wilson, M., Peters, C., Digoutte, J., Guillaud, M., Merzoug, N.O. & Touray, E. 1989 Rift Valley fever among domestic animals in the recent West African outbreak. *Research in Virology* **140**, 67-77.
[21] Mroz, C., Gwida, M., El-Ashker, M., Ziegler, U., Homeier-Bachmann, T., Eiden, M. & Groschup, M. 2017 Rift Valley fever virus infections in Egyptian cattle and their prevention. *Transboundary and Emerging Diseases* **64**, 2049-2058.
[22] R Core Team. 2019 R: A language and environment for statistical computing. (3.5.0 ed. Vienna, Austria., R Foundation for Statistical Computing.
[23] RStudio Team. 2015 RStudio: Integrated Development for R. (Boston, MA, RStudio, Inc.
[24] Zeileis, A. & Grothendieck, G. 2005 zoo: S3 Infrastructure for Regular and Irregular Time Series. *Journal of Statistical Software* **14**, 1-27. (doi:10.18637/jss.v014.i06).
[25] Soetaert, K., Petzoldt, T. & Setzer, R.W. 2010 Solving Differential Equations in R: Package deSolve. *Journal of Statistical Software* **33**, 1-25. (doi:10.18637/jss.v033.i09).
[26] Wickham, H. 2009 ggplot2: Elegant Graphics for Data Analysis. (Springer-Verlag New York.
[27] Carnell, R. 2018 lhs: Latin Hypercube Samples. (version 0.16 ed, R package.
[28] Iooss, B., Janon, A., Pujol, G., Boumhaout, K., Da Veiga, S., Delage, T., Fruth, J., Gilquin, L., Guillaume, J., Le Gratiet, L., et al. 2018 sensitivity: Global Sensitivity Analysis of Model Outputs. (version 1.15.2 ed, R package.
[29] Maxima.sourceforge.net. 2014 Maxima, a Computer Algebra System. (5.34.1 ed.
[30] Sharpe, P.J. & DeMichele, D.W. 1977 Reaction kinetics of poikilotherm development. *Journal of Theoretical Biology* **64**, 649-670.
[31] Wichgers Schreur, P.J., van Keulen, L., Kant, J., Oreshkova, N., Moormann, R.J.M. & Kortekaas, J. 2016 Co-housing of Rift Valley fever virus infected lambs with immunocompetent or immunosuppressed lambs does not result in virus transmission. *Frontiers in Microbiology* **7**. (doi:10.3389/fmicb.2016.00287).
[32] Kilborn, S.H., Trudel, G. & Uhthoff, H. 2002 Review of growth plate closure compared with age at sexual maturity and lifespan in laboratory animals. *Journal of the American Association for Laboratory Animal Science* **41**, 21-26.

[33] Swanepoel, R. & Coetzer, J. 2004 Rift Valley fever. In *Infectious Diseases of Livestock* (eds. J. Coetzer & R. Tustin), pp. 1037-1070, 2nd ed. Cape Town, Oxford University Press
[34] Cloete, S.W.P. & Olivier, J.J. 2010 South African sheep and wool industries. In *The International Sheep and Wool Handbook* (ed. D.J. Cottle), pp. 95-112. Nottingham, United Kingdom, Nottingham University Press.
[35] Easterday, B.C. 1965 Rift Valley fever. *Advanced Veterinary Science* **10**, 65-127.
[36] Weiss, K. 1962 Studies on Rift Valley fever. Passive and active immunity in lambs. *Onderstepoort Journal of Veterinary Research* **29**, 3-9.
[37] Zavortink, T.J. 1990 Biosystematics of *Aedes (Neomelaniconion)*. (pp. 1-37, University of San Francisco.
[38] Jitpakdi, A., Junkum, A., Pitasawat, B., Komalamisra, N., Rattanachanpichai, E., Chaithong, U., Tippawangkosol, P., Sukontason, K., Puangmalee, N. & Choochote, W. 2005 Laboratory colonization of *Aedes lineatopennis*. *Southeast Asian Journal of Tropical Medicine and Public Health* **36**, 158-166.
[39] Arum, S.O., Weldon, C.W., Orindi, B., Tigoi, C., Musili, F., Landmann, T., Tchouassi, D.P., Affognon, H.D. & Sang, R. 2016 Plant resting site preferences and parity rates among the vectors of Rift Valley fever in northeastern Kenya. *Parasites & Vectors* **9**, 310. (doi:10.1186/s13071-016-1601-7).
[40] Chapman, H.C. & Woodard, D.B. 1965 Blood feeding and oviposition of some floodwater mosquitoes in Louisiana: Laboratory studies. *Mosquito News* **25**, 259-262.
[41] Subra, R. 1981 Biology and control of C*ulex pipiens quinquefasciatus** Say, 1823 (*Diptera, Culicidae*) with special reference to Africa. *International Journal of Tropical Insect Science* **1**, 319-338.
[42] Dziem, G.M. & Cupp, E.W. 1983 Laboratory bionomics of *Culex* (Melanoconion) *taeniopus*. *Mosquito News* **43**, 170-175.
[43] Gad, A.M., Hassan, M.M., El Said, S., Moussa, M.I. & Wood, O.L. 1987 Rift Valley fever virus transmission by different Egyptian mosquito species. *Transactions of the Royal Society of Tropical Medicine and Hygiene* **81**, 694-698. (doi:https://doi.org/10.1016/0035-9203(87)90460-3).
[44] McIntosh, B.M., Jupp, P.G., Dossantos, I. & Barnard, B.J.H. 1980 Vector studies on Rift Valley fever virus in South Africa. *South African Medical Journal* **58**, 127-132.
[45] McIntosh, B.M., Jupp, P.G., Anderson, D. & Dickinson, D.B. 1973 Rift Valley fever. 2. Attempts to transmit virus with seven species of mosquito. *Journal of the South African Veterinary Association* **44**, 57-60.
[46] Dohm, D.J., Romoser, W.S., Turell, M.J. & Linthicum, K.J. 1991 Impact of stressful conditions on the survival of *Culex pipiens* exposed to Rift Valley fever virus. *Journal of the American Mosquito Control Association* **7**, 621-623.
[47] Jupp, P., Kemp, A., Grobbelaar, A., Leman, P., Burt, F., Alahmed, A., Mujalli, D., Khamees, M. & Swanepoel, R. 2002 The 2000 epidemic of Rift Valley fever in Saudi Arabia: Mosquito vector studies. *Medical and Veterinary Entomology* **16**, 245-252.
[48] Gargan, T.P., Bailey, C.L., Higbee, G.A., Gad, A. & El Said, S. 1983 The effect of laboratory colonization on the vector-pathogen interactions of Egyptian *Culex pipiens* and Rift Valley fever virus. *Am J Trop Med Hyg* **32**, 1154-1163. (doi:10.4269/ajtmh.1983.32.1154).

[49] Jupp, P.G. & Cornel, A.J. 1988 Vector competence tests with Rift Valley fever virus and five South African species of mosquito. *Journal of the American Mosquito Control Association* **4**, 4-8.
[50] McIntosh, A.C., Jupp, P.G. & Dos Santos, I. 1983 Field and laboratory evidence implicating Culex zombaensis and Aedes circumluteolus as vectors of Rift Valley fever virus in coastal South Africa. *South African Journal of Science* **79**, 61-64.