

## A discrete-time rodent-hantavirus model structured by infection and developmental stages

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### Abstract.

Hantaviruses are a group of viruses that infect wild rodents without causing any apparent illness or disease. New discrete-time models for the spread of hantavirus in a rodent population are formulated and analyzed. The models are structured by the stages of the infection, the stages of development, and the sex of the rodent. The basic reproduction number  $\mathcal{R}_0$  is computed for the deterministic model and a condition is given for a simplified model with males only to be permanent. A stochastic model is also formulated. Numerical simulations illustrate the differences between the deterministic and stochastic models and the dynamics in the male and female rodents. It is shown, in the numerical examples, that a transcritical bifurcation occurs at  $\mathcal{R}_0 = 1$  and a unique enzootic equilibrium exists when  $\mathcal{R}_0 > 1$ . The sensitivity of the equilibrium values to changes in the parameters is also investigated.

### §1. Introduction

Hantaviruses are viruses that persistently infect wild rodents without causing any apparent illness or disease. However, they do produce disease in humans, either hantavirus pulmonary syndrome (HPS in the Americas) or hemorrhagic fever with renal syndrome (HFRS in Europe and Asia). Approximately 30 different hantaviruses are recognized throughout the world [22]. The widespread occurrence of HPS and its potential for severe human illness has identified it as an emerging disease with a significant public health threat.

Each hantavirus is generally associated with a single rodent host known as the reservoir host. Our goal is to formulate a realistic model for hantavirus in the reservoir host that will help us better understand how to prevent the spread of this disease to the human population. The new

model is a system of difference equations, structured by the stages of the infection (susceptible and infected), the stages of development (juvenile, subadult, and adult) and the sex of the rodent (male and female). Other models for the rodent-hantavirus interaction have applied differential equations with stages for infection [1, 2, 3, 24] and males and females [4]. Our model is the first to consider the developmental stages of the rodent in a discrete-time formulation. Hantavirus spread is primarily through infections acquired during post-weaning intraspecific encounters [8]. Studies suggest that there are distinct differences in males and females in their duration of shedding and viremia [10, 14, 15, 21, 25], as well as differences in prevalence of hantavirus antibodies. In addition, males exhibit more aggressive behavior than females [5, 9, 10, 21, 25]. Inclusion of developmental stages and gender differences in our model will account for the differences in hantavirus seroprevalence seen in the field data.

In the next section, a susceptible-infected (SI) epidemic model with developmental stages and gender is formulated. The basic reproduction number  $\mathcal{R}_0$  is computed for this model. It is shown for a simplified model with males only that the system is permanent. In section 3, a stochastic model is formulated. Then, in section 4, numerical simulations of the deterministic and stochastic models illustrate the differences in the models and of the persistent infection dynamics between males and females. It is shown that a transcritical bifurcation occurs at  $\mathcal{R}_0 = 1$  and a unique enzootic equilibrium when  $\mathcal{R}_0 > 1$ . The sensitivity of the enzootic equilibrium values to changes in parameter values is investigated numerically.

## §2. SI deterministic model

### 2.1. Description of model

Rodents are classified as susceptible or infected (and infectious) and further classified by gender, and stage of development. For male rodents, the variables  $Y$  = juveniles,  $C$  = subadults, and  $M$  = adults. For female rodents,  $J$  = juveniles,  $D$  = subadults,  $F$  = adults. We assume the juvenile and subadult stages are nonreproductive. The juvenile stage is when newborn rodents are in the nest. The subadult stage is after juveniles leave the nest but prior to reproduction. We assume that juvenile rodents have a small chance of contracting the virus in the nest and that subadults mature quickly to adulthood, so that the virus is only transmitted in the adult stage [5]. Data indicates that rodents infected with hantavirus may remain infected for life [6, 18], therefore there is no recovery stage in the model. A subscript  $S$  (susceptible) or  $I$  (infected

and infectious) on the variables  $M, F$  denote the infection status of the adult classes. For example,  $M_S =$  susceptible adult males. The total male population is denoted as  $N_M = Y + C + M_S + M_I$  and the total female population as  $N_F = J + D + F_S + F_I$ . The total population size is equal to  $N = N_M + N_F$ . Each developmental and infectious stage is followed over discrete time intervals  $t = 1, 2, \dots$ , where  $[t, t + 1]$  equals one month. We assume rodents remain juveniles for one month and subadults for one month.

To model births, let

$$B(M, F) = \frac{2bMF}{M + F}$$

be the total number of births for  $M = M_S + M_I$  reproductive males and  $F = F_S + F_I$  reproductive females. The function  $B$  is known as a harmonic mean birth function, where  $b$  is the average litter size [7]. Juvenile rodents become reproductive adults in approximately three months. Therefore, the birth function is divided by three.

To model the infection process, we assume that the number of encounters between susceptible and infected rodents are distributed randomly among the rodent population. Then the number of encounters follows a Poisson distribution,

$$p(i) = \frac{\exp(-\lambda)\lambda^i}{i!}, \quad i = 0, 1, 2, \dots,$$

where  $i$  is the number of infective encounters (that result in spread of infection from an infectious individual to a susceptible individual) and  $\lambda$  is the average number of infective encounters per susceptible individual in the population during the interval  $[t, t + 1]$ . Since it only takes one successful encounter to become infected, the probability a susceptible rodent becomes infected is  $1 - p(0)$ . Female and male behavioral and immune system differences suggest that the interaction of the virus with its host may be sexually dimorphic [13, 16]. Aggressive encounters between males contribute to higher seroprevalence in males; therefore, it is reasonable to assume contacts among males are greater than among females [5, 9, 10, 21, 25]. The seroprevalence data in Paraguay also show a distinct difference between males and females (seroprevalence in *Akodon montensis* males  $\approx 20\%$  versus females  $\approx 6\%$  for years 2005 and 2006, unpublished data). Generally, male seroprevalence is approximately 3 to 5 times greater than female. If the average number of infective encounters by all susceptible individuals with infected males or females satisfies the law of mass action, then  $\lambda S = (\beta_M M_I + \beta_F F_I)S$ , where

contact with infected males or females is differentiated by  $\beta_M$  or  $\beta_F$ , respectively. Hence,

$$1 - p(0) = 1 - \exp(-\beta_M M_I - \beta_F F_I),$$

where  $\beta_M \gg \beta_F$ . Encounters between infected and susceptible rodents may also depend on the particular developmental stage. Therefore, the parameters  $\beta_M$  and  $\beta_F$  may depend on  $C, M, D$  or  $F$ . We differentiate these infective encounters by assuming different values for the parameters,  $\beta_{\nu M}$  or  $\beta_{\nu F}$  for contacts between susceptible rodents in stages  $\nu = C, M, D, F$ , respectively.

Density-dependent mortality takes the form of the well-known Beverton-Holt growth equation. For example, in the simple case where the population is not structured by stage or sex, then

$$N(t + 1) = \frac{(b/6 + 1)N(t)K}{K + (b/6)N(t)} = (b/6 + 1)N(t)L(N(t)),$$

where  $K$  is the carrying capacity. The constant  $b/6$  appears because  $(b/2)/3$  is the average litter size per rodent per month. In addition to the density-dependent mortality, every stage has probability  $\mu_i$ ,  $0 < \mu_i \leq 1$ ,  $i = Y, C, M, J, D, F$ , of surviving to the next time interval. There are no deaths from hantavirus in the rodent population; hantavirus appears to have no effect on rodent survival [8]. Births, infections, survival, and transitions in time  $[t, t + 1]$  are followed by density-dependent deaths.

Based on the preceding assumptions, the SI difference equation model for the rodent population has the following form:

$$\begin{aligned} Y(t + 1) &= \frac{1}{3} \left[ \frac{B(M(t), F(t))}{2} \right] L(N(t)) \\ C(t + 1) &= [\mu_Y Y(t)] L(N(t)) \\ M_S(t + 1) &= [\mu_C \exp(-\beta_{CF} F_I(t) - \beta_{CM} M_I(t)) C(t) \\ &\quad + \mu_M \exp(-\beta_{MF} F_I(t) - \beta_{MM} M_I(t)) M_S(t)] L(N(t)) \\ M_I(t + 1) &= [\mu_C (1 - \exp(-\beta_{CF} F_I(t) - \beta_{CM} M_I(t))) C(t) \\ &\quad + \mu_M \{(1 - \exp(-\beta_{MF} F_I(t) - \beta_{MM} M_I(t))) M_S(t) + M_I(t)\}] L(N(t)) \\ J(t + 1) &= \frac{1}{3} \left[ \frac{B(M(t), F(t))}{2} \right] L(N(t)) \\ D(t + 1) &= [\mu_J J(t)] L(N(t)) \\ F_S(t + 1) &= [\mu_D \exp(-\beta_{DF} F_I(t) - \beta_{DM} M_I(t)) D(t) \\ &\quad + \mu_F \exp(-\beta_{FF} F_I(t) - \beta_{FM} M_I(t)) F_S(t)] L(N(t)) \\ F_I(t + 1) &= [\mu_D (1 - \exp(-\beta_{DF} F_I(t) - \beta_{DM} M_I(t))) D(t) \\ &\quad + \mu_F \{(1 - \exp(-\beta_{FF} F_I(t) - \beta_{FM} M_I(t))) F_S(t) + F_I(t)\}] L(N(t)). \end{aligned}$$

The initial conditions are positive and parameters are positive (except  $\beta_{ij} \geq 0$ ). Therefore, it easily follows that solutions to (1)-(2) are positive for  $t \geq 0$ .

**2.2. Basic reproduction number**

The next generation approach [11, 19, 20] is used to compute the basic reproduction number,  $\mathcal{R}_0$ , the number of secondary infections caused by one infectious individual in an entirely susceptible population [12]. If  $\mathcal{R}_0 < 1$ , then the disease-free equilibrium (DFE) is locally asymptotically stable and if  $\mathcal{R}_0 > 1$ , the DFE is unstable.

To compute  $\mathcal{R}_0$ , first the DFE needs to be calculated. At the DFE,  $\bar{M}_I = 0 = \bar{F}_I$ . The remaining equilibrium values are

$$\bar{Y} = \frac{\bar{B}\bar{L}}{6}, \bar{C} = \frac{\mu_Y \bar{B}\bar{L}^2}{6}, \bar{M}_S = \frac{\mu_C \mu_Y \bar{B}\bar{L}^3}{6(1 - \mu_M \bar{L})}, \bar{J} = \frac{\bar{B}\bar{L}}{6},$$

$$\bar{D} = \frac{\mu_J \bar{B}\bar{L}^2}{6}, \bar{F}_S = \frac{\mu_D \mu_J \bar{B}\bar{L}^3}{6(1 - \mu_F \bar{L})}.$$

The value  $\bar{B}$  is the birth function evaluated at the DFE and  $\bar{L} = K/[K + (b/6)E]$ , where  $E$  is the total population size at the DFE. For the case,  $\mu_i \equiv \mu$  for all stages  $i = Y, C, M, J, D, F$ , we show that there exists a unique DFE under certain restrictions on  $\mu$ . In this case, the male and female equilibrium values are equal. Thus,  $\bar{B} = b\bar{M}$  and  $E = 2(\bar{M} + \bar{C} + \bar{Y})$ . A unique DFE exists if  $E$  is positive and unique. Applying the equation for  $\bar{M}$ , it can be shown that  $E$  is the unique positive real root of the following cubic equation:

$$b^3 E^3 + 6Kb^2(3 - \mu)E^2 + 36K^2b(3 - 2\mu)E - 36K^3(b\mu^2 + 6\mu - 6) = 0.$$

It follows from Descartes' rule of signs that there is a unique positive real root of this equation if the constant term in the preceding cubic equation satisfies

$$(3) \quad \mu > \frac{\sqrt{9 + 6b} - 3}{b}.$$

This unique equilibrium value  $E$  can be computed explicitly. In general,  $0 < E \leq K$ .

To form the next generation matrix, the eight state variables are divided into infectious states,  $\vec{X} = (M_I, F_I)^T$ , and noninfectious states,  $(Y, C, M_S, J, D, F_S)^T$ . Focusing on the infectious states, new infections  $\vec{F}$  are separated from other transitions  $\vec{T}$ ,  $\vec{X}(t + 1) = \vec{F}(t) + \vec{T}(t)$ , where

$\vec{\mathbf{F}}(t)$  is

$$(4) \quad \begin{pmatrix} \mu_C(1 - \exp(-\beta_{CF}F_I(t) - \beta_{CM}M_I(t)))C(t) \\ \quad \quad \quad + \mu_M(1 - \exp(-\beta_{MF}F_I(t) - \beta_{MM}M_I(t)))M_S(t) \\ \mu_D(1 - \exp(-\beta_{DF}F_I(t) - \beta_{DM}M_I(t)))D(t) \\ \quad \quad \quad + \mu_F(1 - \exp(-\beta_{FF}F_I(t) - \beta_{FM}M_I(t)))F_S(t) \end{pmatrix} L(N(t))$$

and  $\vec{\mathbf{T}}(t) = (\mu_M M_I(t), \mu_F F_I(t))^T$ . Computing the Jacobian matrices of  $\vec{\mathbf{F}}$  and  $\vec{\mathbf{T}}$  with respect to the infectious states and evaluating at the DFE we obtain matrices  $\mathcal{F}$  and  $\mathcal{T}$ ,

$$(5) \quad \begin{pmatrix} \mu_C\beta_{CM}\bar{C} + \mu_M\beta_{MM}\bar{M}_S & \mu_C\beta_{CF}\bar{C} + \mu_M\beta_{MF}\bar{M}_S \\ \mu_D\beta_{DM}\bar{D} + \mu_F\beta_{FM}\bar{F}_S & \mu_D\beta_{DF}\bar{D} + \mu_F\beta_{FF}\bar{F}_S \end{pmatrix} \bar{L}$$

and  $\begin{pmatrix} \mu_M & 0 \\ 0 & \mu_F \end{pmatrix} \bar{L}$ ,

respectively. Matrices  $\mathcal{F}$  and  $\mathcal{T}$  are nonnegative and  $\rho(\mathcal{T}) < 1$ . Thus  $\mathcal{I} - \mathcal{T}$  is invertible. Matrix  $\mathcal{F}(\mathcal{I} - \mathcal{T})^{-1}$  is the next generation matrix and the basic reproduction number,  $\mathcal{R}_0 = \rho(\mathcal{F}(\mathcal{I} - \mathcal{T})^{-1})$ , is the spectral radius of the next generation matrix [20], i.e.

$$\mathcal{R}_0 = \rho \left( \begin{array}{cc} \frac{(\mu_C\beta_{CM}\bar{C} + \mu_M\beta_{MM}\bar{M}_S)\bar{L}}{1 - \mu_M\bar{L}} & \frac{(\mu_C\beta_{CF}\bar{C} + \mu_M\beta_{MF}\bar{M}_S)\bar{L}}{1 - \mu_F\bar{L}} \\ \frac{(\mu_D\beta_{DM}\bar{D} + \mu_F\beta_{FM}\bar{F}_S)\bar{L}}{1 - \mu_M\bar{L}} & \frac{(\mu_D\beta_{DF}\bar{D} + \mu_F\beta_{FF}\bar{F}_S)\bar{L}}{1 - \mu_F\bar{L}} \end{array} \right).$$

We have given the general form for  $\mathcal{R}_0$ , assuming a unique DFE exists. For the case  $\mu_i \equiv \mu$ ,  $i = Y, C, M, J, D, F$ , we have shown that a unique DFE exists. In this case,  $\bar{C} = \bar{D}$  and  $\bar{M}_S = \bar{F}_S$ . Due to male aggressive behavior, a reasonable assumption is that transmission is greater between two males than between males and females or two females. Hence, if we let  $\mu_i \equiv \mu$ ,  $\beta_M$  be the male transmission coefficients and  $\beta$  be all other transmission coefficients, then the basic reproduction number has a relatively simple form,

$$(6) \quad \mathcal{R}_0 = \left[ \frac{\beta_M + \beta}{2} + \frac{1}{2} \sqrt{4\beta^2 + (\beta_M - \beta)^2} \right] \frac{\mu\bar{L}(\bar{C} + \bar{M}_S)}{1 - \mu\bar{L}}.$$

The expression (6) reduces to a more recognizable form if  $\beta_M = \beta$ ,

$$\mathcal{R}_0 = \frac{\beta\mu\bar{L}}{1 - \mu\bar{L}} 2(\bar{C} + \bar{M}_S).$$

That is,  $\mathcal{R}_0$  is the number of infective contacts by an infectious individual  $\beta\bar{N} = 2\beta(\bar{C} + \bar{M}_S)$  during the individuals infectious period  $\mu\bar{L}/(1 - \mu\bar{L})$  (i.e., number of secondary infections caused by one infectious individual in an entirely susceptible population).

**2.3. Permanence**

Next, we focus on long-term survival of the population known as permanence or uniform persistence. Model (1) and (2) is permanent if there exist  $\delta_1 > 0$  and  $\delta_2 > 0$  such that  $\delta_1 < \liminf_{t \rightarrow \infty} N(t) \leq \limsup_{t \rightarrow \infty} N(t) < \delta_2$  for all positive initial conditions. We simplify model (1) and (2), and consider only the males, assuming there are sufficient number of females, where the birth function is replaced by  $B = bM$ . The simplified model (1) can be expressed in the form  $\vec{x}(t + 1) = A_x \vec{x}(t)$ , where  $\vec{x} = (Y, C, M)^T$  and  $M = M_S + M_I$ . Matrix  $A_x$  depends continuously on  $\vec{x}$  and the simplified model is dissipative. Let  $A_0$  denote  $A_x$  evaluated at the extinction equilibrium,  $\vec{x} = \vec{0}$ ,

$$A_0 = \begin{pmatrix} 0 & 0 & b/6 \\ \mu_Y & 0 & 0 \\ 0 & \mu_C & \mu_M \end{pmatrix}.$$

The form of  $A_0$  and the fact that all parameters are positive implies that  $A_0$  is irreducible. Hence, the simplified model is permanent if  $A_0$  has a dominant eigenvalue greater than one (Theorem 3, pg. 519, [17]). The dominant eigenvalue of  $A_0$  is greater than one if and only if the inherent net reproduction number

$$(7) \quad \mathcal{R} = \frac{b}{6} \mu_C \mu_Y + \mu_M > 1.$$

Inequality (7) is equivalent to (3) if the probability of survival between stages  $\mu_i \equiv \mu$  for  $i = Y, C, M$ . In this case, the DFE exists and is feasible if and only if the simplified model is permanent.

**§3. SI stochastic model**

A discrete-time stochastic model is formulated based on (1) and (2). Random variables  $Y, C, \dots, F_I$  are assumed to be discrete with values in  $\{0, 1, \dots\}$ . Similar assumptions apply to the stochastic model as in the deterministic model, except the births, deaths, and infections are expressed as probabilities. For example, a rodent in stage  $Y$  survives to stage  $C$  during the interval  $[t, t + 1]$  with probability  $\mu_Y$  and does not survive with probability  $1 - \mu_Y$ . An adult male susceptible rodent becomes infected with probability  $P_M = 1 - \exp(-\beta_{MF}F_I(t) - \beta_{MM}M_I(t))$  and does not become infected with probability  $1 - P_M$ .

For the birth process, we assume reproduction depends on the number of mating units at time  $t$ ,  $\lceil B(M(t), F(t))/3 \rceil$ , where  $\lceil u \rceil$  denotes the smallest integer greater than or equal to  $u$ . We assume each mating unit gives birth to  $r$  offspring with probability  $b_r$ ,  $r = 0, 1, \dots$ , where  $\sum_{r=0}^{\infty} b_r = 1$ . In addition, the mean of the offspring distribution,  $\{b_r\}$ , is  $b = \sum_{r=0}^{\infty} r b_r$ , where  $b$  is the average litter size. An offspring may be a male or female with probability  $1/2$ .

§4. Numerical examples

Several numerical examples illustrate the dynamics of the SI deterministic and stochastic models. Baseline parameter values are given in Table 1 for the first simulation (Fig. 1). Because seroprevalence of hantavirus is approximately 3 to 5 times greater in males than females, the contact rates are adjusted. Surprisingly, the male contact rates need to be much greater than 3 to 5 times the females contact rates to obtain a seroprevalence of 3 to 5 times higher in males. Contact rates vary depending on hypothesized interactions among the rodent population. Some contact rates are set to zero indicating that some of the developmental stages have little or no interaction. The survival probability  $\mu = 0.95$  is the same for all stages.

Param.	Value	Param.	Value
$K$	200 rodents	$\mu$	$\frac{0.95}{\text{month}}$
$\beta_{CM}$	$\frac{0.02}{(\text{month})(\text{rodent})}$	$\beta_{DM}$	0
$\beta_{CF}$	0	$\beta_{DF}$	0
Param.	Value	Param.	Value
$b$	3 rodents	$b_r$	$\begin{cases} 1/7 & r = 0, 1, \dots, 6 \\ 0 & \text{otherwise} \end{cases}$
$\beta_{MM}$	$\frac{0.01}{(\text{month})(\text{rodent})}$	$\beta_{FM}$	$\frac{0.002}{(\text{month})(\text{rodent})}$
$\beta_{MF}$	$\frac{0.004}{(\text{month})(\text{rodent})}$	$\beta_{FF}$	$\frac{0.004}{(\text{month})(\text{rodent})}$

Table 1. Baseline parameter values.

In Fig. 1(a), the number of infected males at  $t = 5$  years is approximately 4 times greater than females. The deterministic solution approaches an enzootic equilibrium. The total proportion of the population infected is  $(\bar{M}_I + \bar{F}_I)/\bar{N} \approx 17\%$ . In Fig. 1(b), one sample path of the stochastic model is graphed with the mean of 1000 stochastic sample paths. Notice that the mean is close to the deterministic solution. One sample path illustrates the variability in the number infected.

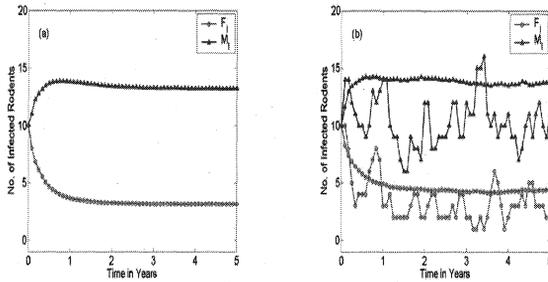


Fig. 1. (a) Deterministic solution,  $\mathcal{R}_0 = 1.55$ . (b) One stochastic sample path and the mean of 1000 sample paths.

In Fig. 2(a), the proportion of infected male and female rodents is graphed as a function of  $\mathcal{R}_0$ , where  $\mathcal{R}_0$  is a function of the contact rates. At  $\mathcal{R}_0 = 1$ , there is a transcritical bifurcation. It is clear from the graph that the proportion of infected males is about four times that of females when  $\mathcal{R}_0 \approx 2$ . As  $\mathcal{R}_0$  increases as a function of the contact rates, the gap between infected male and female rodents decreases.

In Figs. 2(b) and 2(c), the enzootic equilibrium  $\bar{N}$  is graphed as a function of the male and female contact rates, carrying capacity, and litter size. In Fig. 2(b), the infected proportion quickly plateaus for relatively small values of the contact rates. Because juveniles and subadults are not infected, the entire population does not become infected. In Fig. 2(c), if carrying capacity  $K$  is fixed and litter size  $b$  increases, we see that the epidemic dies out. We can attribute this phenomenon to the fact that the population size is fixed but a large proportion of the offspring are not infected. A large number of births “flushes” out the population, increasing the number of susceptible juvenile and subadult rodents while decreasing the number of infected adult rodents. The size of the infected population is greatest when the carrying capacity is large, but the litter size is small.

### §5. Discussion

In this investigation, we formulated new discrete-time, stage-structured SI epidemic models (deterministic and stochastic) for the spread of hantavirus in a rodent population. The stochastic model illustrates the variability in number infected over time (Fig. 1(b)) when births, deaths, and transitions are modeled via a discrete-time Markov process. The inclusion of juvenile and subadult stages in the model reduces the

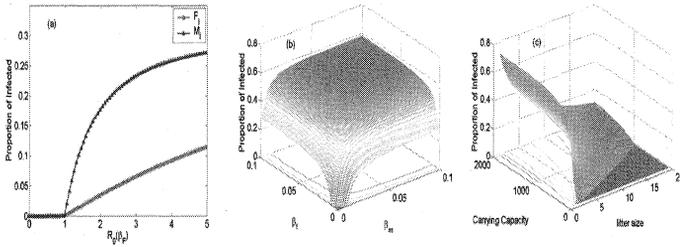


Fig. 2. (a) Graph of the proportion of infected rodents as a function of  $R_0$ ;  $\beta_M = 10\beta_F$ , where  $\beta_M = \beta_{MF} = \beta_{MM} = \beta_{CM} = \beta_{CF}$  and  $\beta_F = \beta_{FF} = \beta_{FM} = \beta_{DM} = \beta_{DF}$ . Parameters are the same as in Table 1, except for the contact rates. (b) Proportion of infected rodents,  $(M_I + F_I)/N$ , at the enzootic equilibrium as a function of  $\beta_M$  and  $\beta_F$ . (c) Proportion of infected rodents at the enzootic equilibrium as a function of carrying capacity  $K$  and litter size  $b$ .

DFE of the population to a level below  $K$ ,  $E \leq K$ . In addition, because the juvenile and subadult stages can not be infected, it is impossible for all rodents in the population to be infected. To control the spread of the infection, the most reasonable strategy is to reduce the population size. A population reduction invariably decreases the contact rates. The model simulations show that the proportion infected increases with  $K$  but decreases with litter size. However, this result depends on the independence of litter size  $b$  and carrying capacity  $K$ , which may not be true in general. Large population explosions (large number of births) often occur during periods of highly favorable environmental conditions [23]. Favorable environmental conditions are equivalent to a high  $K$  value in our models.

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