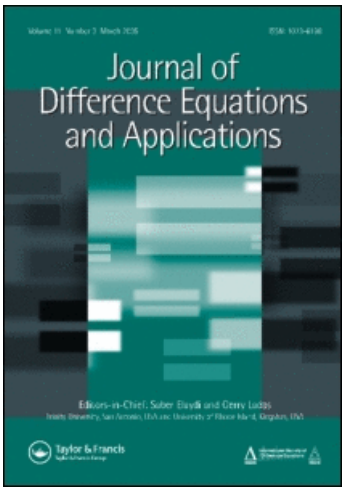

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The basic reproduction number in some discrete-time epidemic models

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Dedicated to Gerry Ladas on the occasion of his 70th birthday

The next generation matrix approach for calculating the basic reproduction number $\mathcal{R}_0$ is summarized for discrete-time epidemic models. This approach is applied to six disease models developed for the study of two emerging wildlife diseases: hantavirus in rodents and chytridiomycosis in amphibians. Two of the models include discrete spatial patches. For each model, $\mathcal{R}_0$ is calculated in terms of the model parameters. For $\mathcal{R}_0 < 1$, if a small number of infectives is introduced, then the wildlife disease dies out. Global stability of the disease-free equilibrium is verified for a special case of the SI hantavirus model when $\mathcal{R}_0 < 1$. In addition, a numerical example indicates that there is a transcritical bifurcation at $\mathcal{R}_0 = 1$, with the disease dying out if $\mathcal{R}_0 < 1$ but tending to an endemic level if $\mathcal{R}_0 > 1$.

Keywords: basic reproduction number; chytridiomycosis; discrete-time epidemic model; epidemic model on patches; hantavirus; next generation matrix

AMS Subject Classification: 39A11; 92D30

1. Introduction

The basic reproduction number, generally denoted as $\mathcal{R}_0$, is one of the most important parameters in the study of mathematical epidemiology. The basic reproduction number can be used to assess whether a newly infectious disease can invade a population and to estimate the final size of an SIR-type epidemic [21,33,39]. For example, when $\mathcal{R}_0 < 1$, the disease-free equilibrium (DFE) is locally asymptotically stable and when $\mathcal{R}_0 > 1$, it is unstable [21,39].

The next generation matrix approach has been very useful in determining a biologically meaningful formula for the basic reproduction number in the case of continuous-time epidemic models, i.e., systems of differential equations [21,39]. However, the next generation matrix approach is not well known in the study of discrete-time epidemic models. Although calculation of $\mathcal{R}_0$ via the next generation matrix approach has been applied infrequently in the study of discrete-time epidemic models [20,29,40], this approach has been applied frequently in the study of discrete-time population models. In population models, the basic reproduction number determines local stability or instability of the extinction equilibrium [17,18,31].

In this investigation, we describe briefly the next generation matrix approach for calculating $\mathcal{R}_0$ in discrete-time epidemic models. Then, we apply this approach to several discrete-time epidemic models, developed for the study of two emerging diseases of wildlife, hantavirus and chytridiomycosis. Hantaviruses, carried by wild rodents, do not affect their rodent hosts but result in serious, life-threatening diseases in humans, either hantavirus pulmonary syndrome

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or hemorrhagic fever with renal syndrome [37]. Chydriomycosis is a fungal pathogen that infects the skin of amphibians, causing it to slough off and very often resulting in death of the infected animal [38]. The chytrid fungus does not infect humans. For these discrete-time epidemic models, the calculation of $\mathcal{R}_0$ is new.

2. Next generation matrix approach

Let $X = (x_1, x_2, \ldots, x_n)^{\mathrm{T}}$ denote the n states of a population with regard to their disease status, i.e., healthy and susceptible, infectious, recovered, etc. Let

$$X(t+1) = G(X(t)), \quad t = 0, 1, \ldots, \tag{1}$$

describe the dynamics of the states of the population over discrete time intervals, where $G : \mathbf{R}_+^n \to \mathbf{R}_+^n$ and $G \in C^1(\mathbf{R}_+^n)$ for $\mathbf{R}_+^n = \{Y = (y_1, y_2, \ldots, y_n) | y_j \geq 0, \ j = 1, 2, \ldots, n\}$. Suppose the states are ordered so that the first m states, $m < n$, denoted as $X_0 = (x_1, \ldots, x_m)^{\mathrm{T}}$, are the infected (e.g., exposed, infectious) states and the remaining $n - m$ states are the uninfected states denoted as $X_1 = (x_{m+1}, \ldots, x_n)^{\mathrm{T}}$. Hence, (1) can be expressed as

$$\begin{pmatrix} X_0(t+1) \\ X_1(t+1) \end{pmatrix} = \begin{pmatrix} G_0(X(t)) \\ G_1(X(t)) \end{pmatrix}.$$

For example, consider an SEIR epidemic model, where S, E, I and R represent the number of susceptible, exposed (latent), infectious and recovered individuals, respectively. Then, the vector $X = (E, I, S, R)^{\mathrm{T}}$, where $X_0 = (E, I)^{\mathrm{T}}$ and $X_1 = (S, R)^{\mathrm{T}}$.

We assume there exists a unique disease-free equilibrium (DFE) of system (1), where $X_0 = 0$ and $X_1 > 0$. In addition, we assume that linearizing the discrete system (1) about the DFE yields the linearized system

$$Y(t+1) = JY(t), \tag{2}$$

where J is the $n \times n$ Jacobian matrix evaluated at the DFE. Matrix J has the following form:

$$J = \begin{pmatrix} F + T & O \\ A & C \end{pmatrix}, \tag{3}$$

where the $m \times m$ submatrices F and T are non-negative, O is the zero matrix, and $F + T$ is irreducible. Matrices F and T are obtained from differentiation with respect to states X_0 and evaluation at the DFE. The important step here is to identify the terms in G_0 that correspond to those in F and those in T. Letting $G_0(X(t)) = \mathcal{F}(t) + \mathcal{T}(t)$, where $\mathcal{F}$ is the vector of new infections that survive the time interval and $\mathcal{T}$ is the vector of all other transitions (e.g., recovery, disease-related deaths), these vectors lead to F and T, respectively. The notation for F and T comes from the literature on matrix population models [15,17,18], where F is known as the fertility matrix and T as the transition matrix.

We assume, in the absence of disease, that the DFE is locally asymptotically stable. That is, the spectral radius of C is less than one, i.e., $\rho(C) < 1$. In addition, we require that $\rho(T) < 1$, but this assumption generally follows from the form of T. Therefore, we assume

$$\rho(C), \rho(T) < 1. \tag{4}$$

Since J is block triangular and $\rho(C) < 1$, stability of the linearized system, $Y(t+1) = JY(t)$, depends on the eigenvalues of the matrix $F + T$, and is independent of matrix A. We can apply theory developed from matrix population dynamics to provide a definition of the basic reproduction number [17,18,31]. Matrix $Q = F(\mathbb{I} - T)^{-1}$ is known as the next generation matrix, where $\mathbb{I}$ is the $m \times m$ identity matrix. Since $\rho(T) < 1$,

$$Q = F(\mathbb{I} + T + T^2 + \cdots).$$

Let I_0 be the vector of initial number or density of infectious individuals, then

$$QI_0 = F(I_0 + TI_0 + T^2I_0 + \cdots),$$

represents the distribution of all infections accumulated during the lifespan of the population [31]. The basic reproduction number $\mathcal{R}_0$ is defined as the spectral radius of matrix Q, that is,

$$\mathcal{R}_0 = \rho(F[\mathbb{I} - T]^{-1}) = \rho(Q). \tag{5}$$

In the discrete-population dynamics literature, $\mathcal{R}_0$ is also referred to as the net reproductive rate or inherent net reproductive number [17,18,31] and in the mathematical epidemiology literature, $\mathcal{R}_0$ is also referred to as the basic reproduction ratio or basic reproductive rate [9,27].

It follows from Theorem 3.3 in Li and Schneider ([31], p. 455) and from Theorem 3 in Cushing and Yicang ([18], p. 115) that $\rho(F + T) = r < 1$ if and only if $\mathcal{R}_0 < 1$. In fact, the relationship between r and $\mathcal{R}_0$ satisfies one of the three inequalities [31]:

$$r = \mathcal{R}_0 = 1, \quad 1 < r \leq \mathcal{R}_0, \quad \text{or} \quad 0 \leq \mathcal{R}_0 \leq r < 1.$$

These results are summarized in the following theorem.

THEOREM 2.1. *Suppose the system of difference equation* (1) *has a unique DFE and that linearization of the system about the DFE yields system* (2)*, where matrix J is given by* (3) *with matrices F and T non-negative, $F + T$ is irreducible, and matrices C and T satisfying* (4)*. Then, the basic reproduction number for system* (1) *as defined in* (5)*. In addition, the DFE of system* (1) *is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

The local stability of the DFE can also be characterized by applying the Schur–Cohn (Jury) criteria to the Jacobian matrix $F + T$ in (3), see, for example, [23]. However, the criteria are usually algebraically difficult to apply and require that several inequalities be satisfied. In addition, the criteria do not provide biological insight into the disease dynamics. On the other hand, the basic reproduction number, as given by (5), provides a single biological meaningful threshold value for the disease to die out when an infectious individual is introduced into an entirely susceptible population ($\mathcal{R}_0 < 1$).

We present six discrete-time models developed for the study of disease in wildlife populations, four models for hantavirus in rodents and two models for chytridiomycosis in amphibians. For each of these models, the conditions of Theorem 2.1 are shown to be satisfied and the basic reproduction number is calculated.

3. Hantavirus models

Hantaviruses are zoonotic pathogens, carried by wild rodents. There are approximately 30 different hantaviruses recognized throughout the world [34,37]. Each hantavirus is generally carried by a single rodent species known as the reservoir population [16]. Human infection

occurs primarily through the inhalation of aerosolized saliva or excreta of infectious rodents. Infection in humans results in either hemorrhagic fever with renal syndrome or hantavirus pulmonary syndrome [37].

A variety of different models has been developed to study the spread of hantavirus in rodent populations. They have taken the form of differential equations [1–3,6,7,35,36], some of which are stochastic [3,7], difference equations, and discrete-time Markov chains [40]. The model of Wesley *et al.* [40] is the first discrete-time model to be applied to hantavirus that includes stages for juveniles, subadults, and adults. In the present investigation, we do not consider the developmental stages; only adults are modelled. As in many of the previous models, we make the following assumptions regarding the biology of the rodents and the epizoology of the infection. The infection and persistence of hantavirus in its rodent host has little or no effect on rodent survival, i.e., there are no disease-related deaths [1–3,7,26,35,36,40]. There is no vertical transmission from mother to offspring [1–3,7,35,36,40]. Male aggressiveness results in greater contact among males than between males and females or among females [3,7,40]. There are equal numbers of males and females, mating is random and rodents become reproductive soon after birth [3,7]. Therefore, non-reproductive stages are not included. We formulate SI, SIR and SEIR models and an SI patch model and show that Theorem 2.1 can be applied. In addition, for the SI, SIR and SEIR models, we prove that the total population size approaches a positive constant.

3.1 SI model

Let S_m, S_f, I_m, I_f, $N_m = S_m + I_m$, and $N_f = S_f + I_f$ be the number of susceptible males, susceptible females, infectious males, infectious females, total number of males and total number of females, respectively. Then $N = S_m + I_m + S_f + I_f$ is the total population size. Let B denote a harmonic mean birth function

$$B \equiv B(N_m, N_f) = \frac{2bN_mN_f}{N}, \tag{6}$$

where $b > 0$ is the average litter size ([15], p. 574) and $N > 0$. The harmonic mean birth function is one of the most commonly used birth functions in demography because of its biologically reasonable properties, e.g., $B(0, N_f) = 0 = B(N_m, 0)$ and $B(N/2, N/2) = bN/2$. We assume the sex ratio at birth is approximately 1:1. The time interval $[t, t+1]$ is approximately the gestation period plus the time until sexual maturity, which is on the order of two to three months.

The probability of infection is based on the Poisson probability distribution. Let

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

where k is the number of encounters that result in infection and λ is the average number of encounters per susceptible individual during the time interval $[t,t+1]$ [40]. Since, it takes at least one effective encounter to become infectious, the probability that a susceptible rodent becomes infectious is $1 - p(0)$. If the average number of encounters by susceptible males with infectious males or females satisfies the law of mass action, then $\lambda S_m = (\beta_m I_m + \beta I_f)S_m$, where the spread of infection depends on whether contact is with an infectious male or infectious female, β_m *versus* β, respectively. This assumption leads to density-dependent transmission for

susceptible males

$$p(0) = \exp(-\beta_{\mathrm{m}} I_{\mathrm{m}} - \beta I_{\mathrm{f}}).$$

Due to male aggressiveness, it is assumed that contact between males is much greater than between males and females or between females; hence,

$$\beta_m \gg \beta > 0, \tag{7}$$

and for susceptible females

$$p(0) = \exp(-\beta I_{\mathrm{m}} - \beta I_{\mathrm{f}}).$$

Density-dependent transmission is often assumed for wildlife models because of the highly fluctuating population densities. The exponential form for $p(0)$ appears in other discrete-time epidemic models (e.g., [24,25,28,40]).

We assume that the birth and infection process is followed by density-dependent survival. It is reasonable to assume that density-dependent survival is logistic. Logistic growth is modelled by including a factor, which has a Beverton–Holt form ([15], p. 506):

$$D(N) = \frac{K}{K + (b/2)N}, \tag{8}$$

where K is the carrying capacity and $1 + (b/2)$ is the exponential of the intrinsic growth rate, $1 + (b/2) = \mathrm{e}^r$, or equivalently $\ln(1 + b/2) = r$. All individuals are subject to the same density-dependent survival. This is a realistic assumption for our hantavirus models because no disease-related deaths and no developmental stages are included.

Based on the preceding assumptions, the discrete-time SI hantavirus model takes the following form:

$$\begin{aligned}
S_{\mathrm{m}}(t+1) &= \left[\frac{B}{2} + \exp(-\beta_{\mathrm{m}} I_{\mathrm{m}}(t) - \beta I_{\mathrm{f}}(t)) S_{\mathrm{m}}(t)\right] D(N) \\
I_{\mathrm{m}}(t+1) &= \left[(1 - \exp(-\beta_{\mathrm{m}} I_{\mathrm{m}}(t) - \beta I_{\mathrm{f}}(t))) S_{\mathrm{m}}(t) + I_{\mathrm{m}}(t)\right] D(N) \\
S_{\mathrm{f}}(t+1) &= \left[\frac{B}{2} + \exp(-\beta I_{\mathrm{m}}(t) - \beta I_{\mathrm{f}}(t)) S_{\mathrm{f}}(t)\right] D(N) \\
I_{\mathrm{f}}(t+1) &= \left[(1 - \exp(-\beta I_{\mathrm{m}}(t) - \beta I_{\mathrm{f}}(t))) S_{\mathrm{f}}(t) + I_{\mathrm{f}}(t)\right] D(N).
\end{aligned} \tag{9}$$

The initial conditions are non-negative, $S_j(0) \geq 0$, $I_j(0) \geq 0$ for $j = \mathrm{m}, \mathrm{f}$, and the parameters are positive. It is clear that a unique non-negative solution exists to (9) with $N_j(t) \geq 0$ for $j = \mathrm{m}, \mathrm{f}$ and $t \geq 0$. Written in vector form, $X(t+1) = G(X((t))$, where the states are reordered so that the male and female infectious states are the first two components, $\mathrm{X} = (I_{\mathrm{m}}, I_{\mathrm{f}}, S_{\mathrm{m}}, S_{\mathrm{f}})^{\mathrm{T}}$. Model (9) is known as an SI epidemic model; susceptible individuals become infectious but do not recover.

We show that the total population size of model (9) follows closely logistic growth (Beverton–Holt growth). That is, asymptotically,

$$N(t+1) \approx \frac{(1 + b/2)KN(t)}{K + (b/2)N(t)},$$

so that $\lim_{t\to\infty} N(t) = K$. Solutions approach the carrying capacity.

THEOREM 3.1. *In the SI model* (9), $\lim_{t\to\infty} N_m(t) = K/2 = \lim_{t\to\infty} N_f(t)$.

Proof. Let $u(t) = N_m(t) - N_f(t)$ so that $N(t) + u(t) = 2N_m(t)$ and $N(t) - u(t) = 2N_f(t)$. Then

$$u(t+1) = \frac{u(t)K}{K + (b/2)N(t)}. \tag{10}$$

Hence, $u(t+1) \leq u(t)$. Either $N_m(0) = N_f(0)$ or $N_m(0) \neq N_f(0)$. For the case $N_m(0) = N_f(0)$, $u(t) = 0 = u(t)/N(t)$ for $t \geq 0$. For the case $N_m(0) \neq N_f(0)$, we assume without loss of generality that $N_m(0) > N_f(0)$. The sequence $\{u(t)\}_{t=0}^{\infty}$ is decreasing and bounded below by zero. Hence, this sequence has a limit, which we denote as u^*. Also note that

$$N(t+1) > \frac{N(t)K}{K + (b/2)N(t)}. \tag{11}$$

Thus, dividing $u(t+1)$ by $N(t+1)$ and applying (10) and (11) yield

$$\frac{u(t+1)}{N(t+1)} < \frac{u(t)}{N(t)} < 1 \tag{12}$$

for $t \geq 0$. The sequence $\{u(t)/N(t)\}_{t=0}^{\infty}$ is decreasing and bounded below by zero. Denote the limit of this sequence by L. Inequality (12), $u(t) > 0$, and $N(t) > 0$ imply $0 < L \leq 1$.

We have already shown $L = 0$ and $u^* = 0$ for the case $N_m(0) = N_f(0)$. Now, we show $L = 0$ and $u^* = 0$ for the case $N_m(0) > N_f(0)$. Let $\epsilon > 0$ and choose T such that for $t \geq T$, $\left(\frac{u(t)}{N(t)}\right)^2 < L^2 + \epsilon < 1$. Summing the equations in (9),

$$\begin{aligned} N(t+1) &= \big(B(N_m(t), N_f(t)) + N(t)\big)\frac{K}{K + (b/2)N(t)} \\ &= \left(\frac{b[N^2(t) - u^2(t)]}{2N(t)} + N(t)\right)\frac{K}{K + (b/2)N(t)} \end{aligned} \tag{13}$$

$$> \left(\frac{b}{2}[1 - L^2 - \epsilon] + 1\right)\frac{KN(t)}{K + (b/2)N(t)} = H_\epsilon(N(t)). \tag{14}$$

Consider the difference equation $s(t+1) = H_\epsilon(s(t))$. The function $H_\epsilon(s)$ is monotone increasing in s and has a unique positive fixed point $s^* = K(1 - L^2 - \epsilon) > 0$. It follows for $s(0) = N(0)$ that $\lim_{t\to\infty} s(t) = s^*$. By comparison with the inequality in (14), it follows that $\liminf_{t\to\infty} N(t) \geq s^*$. In addition, it follows from (10) that $u(t+1) \leq u(t)c$, where $c = K/(K + (b/2)s^*) < 1$ which implies $\lim_{t\to\infty} u(t) = 0 = u^*$. Consequently, $u^* = 0$ and $N(t) \geq s^* > 0$ imply $\lim_{t\to\infty} \frac{u(t)}{N(t)} = 0 = L$.

Let $\epsilon > 0$ and choose T such that $t \geq T$ implies $\left(\frac{u(t)}{N(t)}\right)^2 < \epsilon$. Then applying (13) and (14), the following inequalities are obtained:

$$\left(\frac{b}{2}[1 - \epsilon] + 1\right)\frac{KN(t)}{K + (b/2)N(t)} \leq N(t+1) \leq \left(\frac{b}{2} + 1\right)\frac{KN(t)}{K + (b/2)N(t)}. \tag{15}$$

The left and right sides of (15) are monotone increasing functions of N, $N \geq 0$. It follows by a comparison result from [8] (Lemma 1, p. 201) that

$$[1 - \epsilon]K \leq \liminf_{t\to\infty} N(t) \leq \limsup_{t\to\infty} N(t) \leq K.$$

Since ϵ is arbitrary, $\lim_{t\to\infty} N(t) = K$ and since $\lim_{t\to\infty} u(t) = 0$, the conclusion of the theorem follows. □

For model (9), we apply the next generation matrix approach to calculate the basic reproduction number $\mathcal{R}_0$ and show that the conditions of Theorem 2.1 are satisfied. The unique DFE is given by $\bar{S}_m = K/2 = \bar{S}_f$, where $\bar{I}_m = 0 = \bar{I}_f$. Calculating the Jacobian matrix J evaluated at the DFE, as in (3), leads to the following submatrices:

$$F = \frac{K/2}{1 + (b/2)} \begin{pmatrix} \beta_m & \beta \\ \beta & \beta \end{pmatrix}, \tag{16}$$

$T = \dfrac{1}{1 + (b/2)}\mathbb{I}$, O is the 2×2 zero matrix, and $C = T$, where $\mathbb{I}$ is the 2×2 identity matrix. It is straightforward to see that the conditions of Theorem 2.1 are satisfied, namely that matrices F and T are non-negative, $F + T$ is irreducible, and $\rho(T), \rho(C) < 1$. It follows that $\mathcal{R}_0 = \rho(F(\mathbb{I} - T)^{-1})$, where

$$\mathcal{R}_0 = \frac{\beta_m + \beta + \sqrt{(\beta_m - \beta)^2 + 4\beta^2}}{2b} K. \tag{17}$$

Note that $\mathcal{R}_0$ depends on the litter size, carrying capacity and transmission coefficients. The basic reproduction number for an SI stage-structured hantavirus model studied in [40] has a form similar to (17). In their model, given that the density-independent survival is the same for all stages and that the transmission coefficients are distinguished only by males and females, β_m and β, then $\mathcal{R}_0$ in the model of Wesley *et al.* (equation (6) in [40]) reduces to (17). In addition, note that $\mathcal{R}_0$ is an increasing function of the transmission parameters, β and β_m, a decreasing function of the litter size b and an increasing function of the carrying capacity K. This latter result is due to the assumption of density-dependent transmission. If the carrying capacity is kept fixed but births increase, the infectious population is decreased (and hence, $\mathcal{R}_0$ is decreased) because newborns are not infectious. Under the alternate assumption of frequency-dependent transmission, namely $\lambda S = (\beta_m I_m + \beta I_f)S/N$ for $S = S_m$ and $S = S_f$, then the basic reproduction number is $\mathcal{R}_0/K$, where $\mathcal{R}_0$ is defined in (17).

The following corollary is a direct consequence of Theorem 2.1.

COROLLARY 3.2. *Let $\mathcal{R}_0$ be defined as in* (17). *If $\mathcal{R}_0 < 1$, then the DFE of* (9) *is locally asymptotically stable and if $\mathcal{R}_0 > 1$, it is unstable.*

For the SI hantavirus model (9) we can show the results in Corollary 3.2 are equivalent to the Schur–Cohn criteria for stability of the DFE. Linearizing system (9) about the DFE yields the Jacobian matrix (3). The eigenvalues of $F + T$ are the solutions to $z^2 + a_1 z + a_2 = 0$, where $a_1 = -[(\beta_m + \beta)K/2 + 2]/(1 + b/2) < 0$ and $a_2 = [(\beta_m - \beta)\beta K^2/4 + (\beta_m + \beta)K/2 + 1]/(1 + b/2)^2 > 0$. All roots of this polynomial equation lie inside the unit circle iff the three inequalities are satisfied: $1 + a_1 + a_2 > 0$, $1 - a_1 + a_2 > 0$ and $1 - a_2 > 0$ ([15], p. 522).

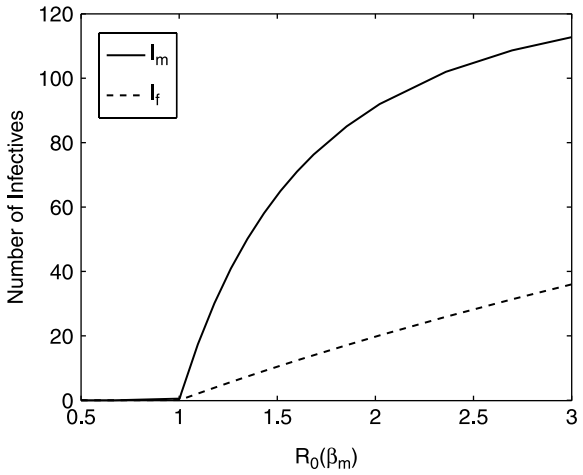

Figure 1. Bifurcation diagram for the SI hantavirus model, where stable solutions I_m and I_f are graphed as a function of $\mathcal{R}_0(\beta_m)$, $\beta = \beta_m/10$, $b = 6$, and $K = 1000$.

The third inequality is required if the roots are complex conjugates. Since, all roots are real ($a_1^2 > 4a_2$), only the first two inequalities need to be satisfied. The second inequality is true. The first is true iff $\mathcal{R}_0 < 1$. At $\mathcal{R}_0 = 1$ a root crosses the unit circle at $z = 1$, thus we expect a transcritical bifurcation ([15], p. 528). The bifurcation diagram in Figure 1 illustrates this bifurcation by showing the stable solutions I_m and I_f as a function of $\mathcal{R}_0(\beta_m)$, where $\beta = \beta_m/10$ and all other parameter values are fixed. The disease persists when $\mathcal{R}_0 > 1$.

The particular form of $\mathcal{R}_0$ in (17) depends on how the disease is transmitted between the sexes. The first two terms in the numerator of $\mathcal{R}_0$ represent transmission between the same sex. The terms in the square root involve same sex transmission and heterosexual transmission. For example, suppose we identify the transmission coefficient by the sex of the infectious individual and the sex of the susceptible individual, e.g., $\beta_{mf} I_m S_f$. Then for general $D(N)$, the matrix F in (16) has the form

$$F = D(K)K/2 \begin{pmatrix} \beta_{mm} & \beta_{fm} \\ \beta_{mf} & \beta_{ff} \end{pmatrix},$$

and $T = D(K)\mathbb{I}$. The basic reproduction number is

$$\mathcal{R}_0 = \frac{\beta_{mm}/2 + \beta_{ff}/2 + (1/2)\sqrt{(\beta_{mm} - \beta_{ff})^2 + 4\beta_{mf}\beta_{fm}}}{1 - D(K)} D(K)K/2.$$

If there is only heterosexual transmission, then the preceding expression for $\mathcal{R}_0$ reduces to the geometric mean of $\beta_{mf}D(K)K/2/[1 - D(K)]$ and $\beta_{fm}D(K)K/2/[1 - D(K)]$. That is,

$$\mathcal{R}_0 = \frac{\sqrt{\beta_{mf}D(K)K/2\beta_{fm}D(K)K/2}}{1 - D(K)}, \tag{18}$$

which simplifies to $\mathcal{R}_0 = \sqrt{\beta_{mf}\beta_{fm}}K/b$ for $D(K)$ as in (8). Alternately, if the model assumptions are relaxed so that infectious males have the same transmission coefficient β_m, regardless

of contact with a susceptible male or female, then for general $D(N)$ the matrix F in (16) has the form

$$F = D(K)K/2\begin{pmatrix} \beta_{\mathrm{m}} & \beta \\ \beta_{\mathrm{m}} & \beta \end{pmatrix},$$

and $T = D(K)\mathbb{I}$. The basic reproduction number is the sum of a reproduction number for males and for females,

$$\mathcal{R}_0 = \frac{\beta_{\mathrm{m}} D(K)K/2}{1 - D(K)} + \frac{\beta D(K)K/2}{1 - D(K)}. \tag{19}$$

Each term in (19) is the product of the virus transmission parameter modified by survival, the population number, and the lifetime survival of infectious individuals (all evaluated at the DFE). Lifetime survival is computed from the infinite sum $1/(1 - D(K)) = \sum_{j=0}^{\infty} [D(K)]^j$. In the extreme case $\beta_{\mathrm{m}} = \beta$, where both males and females have the same transmission coefficient, the basic reproduction number simplifies to $\mathcal{R}_0 = \beta D(K)K/(1 - D(K)) = \beta K/(b/2)$ for $D(K)$ as in (8). In general, for model (9), because the relationship (7) holds,

$$\mathcal{R}_0|_{\beta=0} < \mathcal{R}_0 < \mathcal{R}_0|_{\beta=\beta_{\mathrm{m}}}.$$

Under more restrictive model assumptions, we can show $\mathcal{R}_0 < 1$ implies global stability of the DFE. In the following theorem, we assume the population size is constant, the number of births is constant and the transmission parameter is the same for males and females.

THEOREM 3.3. *Let $N(0) = K$, $B = bK/2$, $D(N)$ be given by* (8)*, and $\beta_m = \beta$ in model* (9)*. If $\mathcal{R}_0 = \beta K/(b/2) < 1$, then the DFE of* (9) *is globally asymptotically stable.*

Proof. Summing all of the difference equations in (9) for $t = 0$ leads to $N(1) = [B + N(0)]$ $D(N(0)) = K$. Hence, for time $t \geq 0$, it follows that $N(t) = K$. Let $I = I_{\mathrm{m}} + I_{\mathrm{f}}$. Then, summing the difference equations for $I_{\mathrm{m}}(t)$ and $I_{\mathrm{f}}(t)$ and substituting $N(t) = K$ yields

$$I(t+1) = \frac{K - \mathrm{e}^{-\beta I(t)}(K - I(t))}{1 + b/2} = f(I(t)).$$

Note that $f(0) = 0$ and $f(I) > 0$ for $0 < I \leq K$. If $\mathcal{R}_0 = \beta K/(b/2) < 1$, then

$$f'(I) = \frac{\mathrm{e}^{-\beta I}(\beta(K - I) + 1)}{1 + b/2} < 1.$$

Thus, $0 < f(I) < I$ for $I > 0$. Consequently, the infectious individuals approach zero, $\lim_{t\to\infty} I_{\mathrm{m}}(t) = 0 = \lim_{t\to\infty} I_{\mathrm{f}}(t)$.

Let $u(t) = N_{\mathrm{m}}(t) - N_{\mathrm{f}}(t)$. The identity (10) and $N(t) = K$ imply $u(t+1) = u(t)/(1 + b/2)$. Hence, $\lim_{t\to\infty} u(t) = 0$. Since, the infectious individuals approach zero, $N_{\mathrm{m}}(t)$ and $N_{\mathrm{f}}(t)$ approach $S_{\mathrm{m}}(t)$ and $S_{\mathrm{f}}(t)$, respectively. It follows that $\lim_{t\to\infty} S_{\mathrm{m}}(t) = K/2 = \lim_{t\to\infty} S_{\mathrm{f}}(t)$; solutions approach the DFE. □

Another form for the probability of infection is assumed by Lewis *et al.* [29] in a discrete-time model for West Nile virus, where mosquito vectors and the bird reservoir population are

modelled. In their model using subscripts v, r for vector, reservoir, respectively, the probability that a susceptible mosquito avoids infection arising from a single bite is $1 - \alpha_v$ and the probability a susceptible mosquito avoids infection from the entire infected bird population in one time step is

$$(1 - \alpha_v)^{\beta_r I_r/N_r}.$$

In our infection process, with density-dependent transmission, the probability that the entire rodent population avoids infection is

$$\exp(-\beta_{\mathrm{m}} I_{\mathrm{m}} - \beta I_{\mathrm{f}}).$$

Comparing these two probabilities under the assumption of density-dependent transmission and one infectious group, the probability of no infection based on the model of Lewis *et al.* [29] is approximately $(1 - \alpha_v)^{\beta_r I}$, whereas this probability, based on our Poisson approximation, is $\exp(-\beta I)$. These approximations are the same if $(1 - \alpha_v)^{\beta_r} = \exp(-\beta)$.

3.2 SIR model

We generalize the SI hantavirus model of the previous subsection to an SIR model, where rodents recover from infection. This assumption may not be realistic for all hantaviruses but may be a better model in some cases. The hantavirus known as Sin Nombre virus has been shown to cause a persistent infection in its rodent host, *Peromyscus maniculatus* [34]. However, for some hantaviruses it has been noted that the highest viral load often occurs in the first few months after infection [36]. After this short period of time, rodents shed virus at much lower levels and transmission of the virus is reduced. Thus, this latter stage may be approximated by a recovered class. Let R_{m} and R_{f} denote the number of male and female rodents that recover from the disease, respectively. Let γ_{m} and γ_{f} be the probability of recovery for males and females, respectively, with

$$0 \leq \gamma_{\mathrm{m}}, \quad \gamma_{\mathrm{f}} \leq 1.$$

For this model, $N_{\mathrm{m}} = S_{\mathrm{m}} + I_{\mathrm{m}} + R_{\mathrm{m}}$, $N_{\mathrm{f}} = S_{\mathrm{f}} + I_{\mathrm{f}} + R_{\mathrm{f}}$ and $N = N_{\mathrm{m}} + N_{\mathrm{f}}$ are the total number of males, total number of females and total population size, respectively. Then, the SIR system of difference equations takes the following form:

$$\begin{aligned}
S_{\mathrm{m}}(t+1) &= \left[\frac{B}{2} + \exp(-\beta_{\mathrm{m}} I_{\mathrm{m}} - \beta I_{\mathrm{f}}) S_{\mathrm{m}}\right] D(N) \\
I_{\mathrm{m}}(t+1) &= \big([1 - \exp(-\beta_{\mathrm{m}} I_{\mathrm{m}} - \beta I_{\mathrm{f}})] S_{\mathrm{m}} + (1 - \gamma_{\mathrm{m}}) I_{\mathrm{m}}\big) D(N) \\
R_{\mathrm{m}}(t+1) &= \big[\gamma_{\mathrm{m}} I_{\mathrm{m}} + R_{\mathrm{m}}\big] D(N) \\
S_{\mathrm{f}}(t+1) &= \left[\frac{B}{2} + \exp(-\beta I_{\mathrm{m}} - \beta I_{\mathrm{f}}) S_{\mathrm{f}}\right] D(N) \\
I_{\mathrm{f}}(t+1) &= \big([1 - \exp(-\beta I_{\mathrm{m}} - \beta I_{\mathrm{f}})] S_{\mathrm{f}} + (1 - \gamma_{\mathrm{f}}) I_{\mathrm{f}}\big) D(N) \\
R_{\mathrm{f}}(t+1) &= \big[\gamma_{\mathrm{f}} I_{\mathrm{f}} + R_{\mathrm{f}}\big] D(N),
\end{aligned} \tag{20}$$

where the birth and survival functions B and D are defined in (6) and (8), respectively. The t dependence of the variables in the right side of (20) is omitted for simplicity. Initial conditions are non-negative, $S_j(0) \geq 0$, $I_j(0) > 0$, $R_j(0) \geq 0$, $j = \mathrm{m}, \mathrm{f}$, and parameters are positive; hence, solutions are non-negative for $t \geq 0$.

It can be shown in a manner similar to the proof of Theorem 3.1 that N_m and N_f approach $K/2$ as $t \to \infty$. Hence, the DFE for the SIR model is $\bar{S}_m = K/2 = \bar{S}_f$ with other states equal to zero. Ordering the states as (I_m, I_f, S_m, S_f, R_m, R_f), the submatrices in the Jacobian matrix (3) are $T = \dfrac{1}{1 + (b/2)}\operatorname{diag}(1 - \gamma_m, 1 - \gamma_f)$, F is given by (16), O is the 2×4 zero matrix, and $C = \dfrac{1}{1 + (b/2)}\mathbb{I}$, where $\mathbb{I}$ is the 4×4 identity matrix. Applying the next generation matrix approach in Theorem 2.1, the basic reproduction number is

$$\mathcal{R}_0 = \frac{\beta_m K/4}{b_{\gamma_f}} + \frac{\beta K/4}{b_{\gamma_f}} + \frac{K/4\sqrt{[\beta_m b_{\gamma_f} + \beta b_{\gamma_m}]^2 - 4\beta(\beta_m - \beta)b_{\gamma_f} b_{\gamma_m}}}{b_{\gamma_f} b_{\gamma_m}}, \tag{21}$$

where $b_{\gamma_m} = (b/2) + \gamma_m$ and $b_{\gamma_f} = (b/2) + \gamma_f$.

The results for model (20) follow from Theorems 2.1 and 3.1.

COROLLARY 3.4. *Let $\mathcal{R}_0$ be defined as in* (21). *In the SIR model* (20), $\lim_{t\to\infty} N_m(t) = K/2 = \lim_{t\to\infty} N_f(t)$. *In addition, if $\mathcal{R}_0 < 1$, then the DFE of model* (20) *is locally asymptotically stable and if $\mathcal{R}_0 > 1$, it is unstable.*

The basic reproduction number (21) is a decreasing function of γ_m and γ_f; recovery reduces the probability of an outbreak. If there is no recovery for males and females, $\gamma_m = 0 = \gamma_f$ in (21), then the basic reproduction number for the SI model is obtained.

3.3 SEIR model

Next, we generalize the SIR hantavirus model of the previous subsection to an SEIR model. The total population size is $N = N_m + N_f$, where now $N_m = S_m + E_m + I_m + R_m$ and $N_f = S_f + E_f + I_f + R_f$ are the total number of males and females in the population, respectively, with individuals that have been exposed to the disease but are not yet infectious denoted as E_m (males) and E_f (females). The length of the latent period is assumed to be the same for males and females, but the infectious period for males is longer than for females [3,7]. The probability of becoming infectious in the time interval $[t, t + 1]$ is δ, $0 \le \delta \le 1$. The same assumptions regarding the other variables and parameters are taken as in the SIR model of the previous subsection. The discrete-time SEIR model applies to hantavirus if the latent period is greater than the gestation period plus the time to sexual maturity. For many hantavirus infections, the latent period may be very short and so, for the SEIR model to be applicable, the time period $[t, t + 1]$ may need to be shortened implying that developmental stages other than adults may need to be included.

The SEIR model for males has the following form:

$$\begin{aligned}
S_m(t+1) &= \left[\frac{B}{2} + \exp(-\beta_m I_m - \beta I_f)S_m\right] D(N) \\
E_m(t+1) &= \left([1 - \exp(-\beta_m I_m - \beta I_f)]S_m + (1 - \delta)E_m\right) D(N) \\
I_m(t+1) &= \left[\delta E_m + (1 - \gamma_m)I_m\right] D(N) \\
R_m(t+1) &= \left[\gamma_m I_m + R_m\right] D(N).
\end{aligned} \tag{22}$$

A similar set of difference equations applies to the females, where β_m and γ_m are replaced by β and γ_f, respectively. Initial conditions are non-negative, $S_j(0) \ge 0$, $E_j(0) + I_j(0) \ge 0$, $R_j(0) \ge 0$

for $j = \mathrm{m}, \mathrm{f}$, and parameters are positive so that solutions to the SEIR model are non-negative for $t \geq 0$. It can be shown in a manner similar to the proof of Theorem 2.1 that the total population size approaches the carrying capacity K.

Next, we compute the basic reproduction number for model (22). At the DFE $\bar{S}_m = K/2 = \bar{S}_f$ and all other states are zero. We order the states as follows: $(E_\mathrm{m}, E_\mathrm{f}, I_\mathrm{m}, I_\mathrm{f}, S_\mathrm{m}, S_\mathrm{f}, R_\mathrm{m}, R_\mathrm{f})$. Then, the submatrices in the Jacobian matrix J in (3) are $C = \dfrac{1}{1 + (b/2)}\mathbb{I}$, O is the 4×4 zero matrix,

$$F = \frac{K/2}{1 + (b/2)} \begin{pmatrix} 0 & 0 & \beta_\mathrm{m} & \beta \\ 0 & 0 & \beta & \beta \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

and

$$T = \frac{1}{1 + (b/2)} \begin{pmatrix} 1 - \delta & 0 & 0 & 0 \\ 0 & 1 - \delta & 0 & 0 \\ \delta & 0 & 1 - \gamma_\mathrm{m} & 0 \\ 0 & \delta & 0 & 1 - \gamma_\mathrm{f} \end{pmatrix},$$

where $\mathbb{I}$ is the 4×4 identity matrix. The matrices satisfy the conditions stated in Theorem 2.1. The basic reproduction number is given by

$$\mathcal{R}_0 = \frac{\beta_\mathrm{m}\delta K/4}{b_{\gamma_\mathrm{m}} b_\delta} + \frac{\beta\delta K/4}{b_{\gamma_\mathrm{f}} b_\delta} + \frac{\delta K/4\sqrt{[\beta_\mathrm{m} b_{\gamma_\mathrm{f}} + \beta b_{\gamma_\mathrm{m}}]^2 - 4\beta(\beta_\mathrm{m} - \beta) b_{\gamma_\mathrm{f}} b_{\gamma_\mathrm{f}}}}{b_{\gamma_\mathrm{f}} b_{\gamma_\mathrm{m}} b_\delta}, \tag{23}$$

where $b_{\gamma_\mathrm{f}} = (b/2) + \gamma_\mathrm{f}$, $b_{\gamma_\mathrm{m}} = (b/2) + \gamma_\mathrm{m}$ and $b_\delta = (b/2) + \delta$.

The results for the discrete SEIR model (22) are then summarized as in Corollary 3.4, but with $\mathcal{R}_0$ given as in (23). Note that letting δ/b_δ tend to 1 gives the previous $\mathcal{R}_0$, defined in (21) for the SIR model. The delay δ introduced through inclusion of an exposed class results in a decrease of $\mathcal{R}_0$.

3.4 SI patch model

In the previous models, we assumed that the spatial environment is homogeneous. Here, we formulate a new model for animals that move among n spatial regions or patches. The patches are determined by habitat or environmental requirements for the species. Within each patch, there are births, deaths, and disease spread as in model (9), but in addition rodents move between the patches.

Let $m_{ji} \geq 0$ and $f_{ji} \geq 0$ denote the probabilities that a male and a female rodent, respectively, move from patch i to patch j, $j \neq i$. Let $m_{ii} \leqslant 1 - \sum_{j \neq i} m_{ji} \geq 0$ and $f_{ii} \leqslant 1 - \sum_{j \neq i} f_{ji} \geq 0$ denote the probabilities that a male and a female rodent, respectively, do not move out of patch i. Because movement may involve a cost (animals leaving patch i may die before reaching patch j),

we assume

$$0 < \sum_{j=1}^{n} m_{ji} \leq 1 \text{ and } 0 < \sum_{j=1}^{n} f_{ji} \leq 1 \tag{24}$$

for $i = 1, \ldots, n$. Denote the $n \times n$ dispersal matrices as $\mathbb{M} = (m_{ij})$ and $\mathbb{F} = (f_{ij})$. We assume that $\mathbb{M}$ and $\mathbb{F}$ are irreducible, i.e., the patches are connected. Hence, it follows that $\rho(\mathbb{M}), \rho(\mathbb{F}) \leq 1$. In the hantavirus model, there are no disease-related deaths, hence, movement of a susceptible animal is not differentiated from an infectious animal.

To formulate an n-patch model, let the time interval $[t, t+1]$ be divided into two steps, $[t, \hat{t}]$, $[\hat{t}, t+1]$, where $t < \hat{t} < t+1$. Births, deaths and infection occur during the first step followed by movement during the second step. Discrete-time models with growth followed by dispersal among two patches have been studied by others [5,13,14,22]. The patch dynamics in the first time step, $[t, \hat{t}]$, as generalized from (9), are given by the system

$$\begin{aligned}
S_m^i(\hat{t}) &= \left[\frac{B^i}{2} + \exp(-\beta_{\mathrm{m}}^i I_{\mathrm{m}}^i(t) - \beta I_{\mathrm{f}}^i(t)) S_{\mathrm{m}}^i(t)\right] D^i(N^i) \\
I_{\mathrm{m}}^i(\hat{t}) &= \left[(1 - \exp(-\beta_{\mathrm{m}}^i I_{\mathrm{m}}^i(t) - \beta^i I_{\mathrm{f}}^i(t))) S_{\mathrm{m}}^i(t) + I_{\mathrm{m}}^i(t)\right] D^i(N^i) \\
S_{\mathrm{f}}^i(\hat{t}) &= \left[\frac{B^i}{2} + \exp(-\beta^i I_{\mathrm{m}}^i(t) - \beta^i I_{\mathrm{f}}^i(t)) S_{\mathrm{f}}^i(t)\right] D^i(N^i) \\
I_{\mathrm{f}}^i(\hat{t}) &= \left[(1 - \exp(-\beta^i I_{\mathrm{m}}^i(t) - \beta I_{\mathrm{f}}^i(t))) S_{\mathrm{f}}^i(t) + I_{\mathrm{f}}^i(t)\right] D^i(N^i)
\end{aligned} \tag{25}$$

for $i = 1, \ldots n$, where

$$B^i = \frac{2b^i N_{\mathrm{m}}^i N_{\mathrm{f}}^i}{N^i} \text{ and } D^i(N^i) = \frac{K^i}{K^i + (b^i/2)N^i}.$$

The superscript i on the parameter or the disease class means it is associated with patch i. Movement among the patches occurs during the second time step, $[\hat{t}, t+1]$. Let $S_r = (S_r^1, \ldots, S_r^n)^{\mathrm{T}}$ and $I_r = (I_r^1, \ldots, I_r^n)^{\mathrm{T}}$, where $r = \mathrm{m}, \mathrm{f}$. Then, the second step of the model is

$$S_{\mathrm{m}}(t+1) = \mathbb{M} S_{\mathrm{m}}(\hat{t}), \quad S_{\mathrm{f}}(t+1) = \mathbb{F} S_{\mathrm{f}}(\hat{t}), \quad I_{\mathrm{m}}(t+1) = \mathbb{M} I_{\mathrm{m}}(\hat{t}), \text{ and } I_{\mathrm{f}}(t+1) = \mathbb{F} I_{\mathrm{f}}(\hat{t}).$$

Because of the large number of parameters, it is difficult to verify the existence of a unique DFE and to determine a simple expression for the basic reproduction number. In the special case that the demographic parameters are independent of patch, $b^i \equiv b$ and $K^i \equiv K$, and movement does not result in any deaths, namely,

$$\sum_{j=1}^{n} m_{ji} = 1 \text{ and } \sum_{j=1}^{n} f_{ji} = 1,$$

then the unique DFE is independent of patch and is given by

$$\bar{S}_m^i = K/2 = \bar{S}_f^i, \quad i = 1, \ldots, n.$$

In this case, the assumptions of Theorem 2.1 are satisfied and the basic reproduction number can be computed. Let $X_0 = (I_{\mathrm{m}}, I_{\mathrm{f}})$ and $X_1 = (S_{\mathrm{m}}, S_{\mathrm{f}})$. We define the following $n \times n$ matrices: $\mathbb{M}_{\beta_{\mathrm{m}}} = (m_{ij}\beta_{\mathrm{m}}^i)$, $\mathbb{M}_{\beta} = (m_{ij}\beta^i)$, $\mathbb{F}_{\beta} = (f_{ij}\beta^i)$ and O_n is the zero matrix. Then, the submatrices

in the Jacobian matrix (3) are

$$F = \frac{K/2}{1+(b/2)} \begin{pmatrix} \mathbb{M}_{\beta_m} & \mathbb{M}_{\beta} \\ \mathbb{F}_{\beta} & \mathbb{F}_{\beta} \end{pmatrix}, \tag{26}$$

$$T = \frac{1}{1+(b/2)} \begin{pmatrix} \mathbb{M} & O_n \\ O_n & \mathbb{F} \end{pmatrix}, \tag{27}$$

O is the $2n \times 2n$ zero matrix, and $C = T$. The basic reproduction number for this special case of the n-patch model is given by (5), where F and T are defined in (26) and (27). A simple expression for $\mathcal{R}_0$ cannot be computed unless $\mathbb{M}$ and $\mathbb{F}$ have a simple form, but $\mathcal{R}_0$ can easily be computed for a given set of parameter values. In the limiting case, where there is no movement, i.e., $\mathbb{M}$ and $\mathbb{F}$ approach the $n \times n$ identity matrix, it follows that each patch has its own basic reproduction number $\mathcal{R}_i$, namely

$$\mathcal{R}_i = \frac{\beta_m^i + \beta^i + \sqrt{(\beta_m^i - \beta^i)^2 + 4(\beta^i)^2}}{2b} K.$$

Dispersal can spread a disease through space in that an outbreak in one patch can be spread to other patches. On the other hand, if $\mathcal{R}_0 < 1 < \mathcal{R}_i$ for some i, then dispersal can also reduce the probability of an outbreak for the entire system. Similar complicated dependence of $\mathcal{R}_0$ on movement between patches is found for other discrete-time models (e.g., [13,14]) and continuous-time models (e.g., [4,10]).

4. Chytridiomycosis model

Chytridiomycosis is a fungal infection of amphibians that has been associated with mass die-offs in the United States, Australia, Central America and Europe [11,12,19,30,38]. *Batrachochytrium dendrobatidis*, the fungal pathogen responsible for chytridiomycosis, was identified in 1998 from dead and dying frogs in Australia and Panama [32]. The fungus attacks the keratin in the skin of amphibians [19]. Because keratin is present only in the mouthparts of the larval stage of amphibians, this stage is not as susceptible to infection as the post-metamorphic stages [11].

Discrete-time, deterministic and stochastic models have been developed for amphibian populations infected by chytridiomycosis [24,25] but the basic reproduction numbers were not computed for these models. We now consider a simplified model for fungal infection in amphibians, originally formulated by Emmert and Allen [25], which we describe briefly and then compute the basic reproduction number.

4.1 SI model

Let A_S and A_I denote the density of adult amphibians that are either susceptible or infected by the fungal pathogen. Let F denote the density of the fungal pathogen present in the environment, either as motile zoospores or on the keratin of dead amphibians. The sex ratio of males and females is constant, only females are modelled. Because eggs and tadpoles present in a chytrid-infected pond will become infected, we assume infected adult females give birth to infected offspring. The egg and tadpole stages experience the highest mortality. Hence, we assume density-dependent birth and survival functions for the susceptible and infected adults: $B_S(N) = b_S\phi(N)$ and $B_I(N) = b_I\phi(N)$, $0 < b_I \leq b_S$, where N is the adult population size,

$N = A_S + A_I$ (as distinct from our hantavirus models). In addition, ϕ: $[0,\infty) \rightarrow (0,1]$ is a strictly decreasing function of N satisfying $\phi(0) = 1$ and $\lim_{N\rightarrow\infty}\phi(N) = 0$. Two reasonable forms for the density-dependent function ϕ are Beverton–Holt and Ricker forms, namely

$$\phi(N) = \frac{1}{1+cN} \text{ and } \phi(N) = \exp(-cN), \quad c > 0, \tag{28}$$

respectively (see [15], p. 506).

The probability of infection is based on the Poisson distribution with density-dependent transmission (as for our hantavirus models). The probability that adults do not become infected is

$$\exp(-\beta[\omega_A A_I + \omega_F F]) = \exp(-\beta\omega \cdot I),$$

where $\omega \cdot I = \omega_A A_I + \omega_F F$, $\omega_{A,}\omega_F \geq 0$ and $\omega_A + \omega_F > 0$. The fungus will not persist in the environment unless there is a source of keratin from infected amphibians.

The parameters p_S, p_I and p_F are probabilities of survival during the time interval $[t, t+1]$ for susceptible and infected adult amphibians and for free-living fungi, respectively

$$0 < p_I < p_S < 1, \quad 0 < p_F < 1.$$

The number of new fungal zoospores coming from infected animals is $b_F A_I$, $b_F > 0$.

The adult model has the following form [25]:

$$\begin{aligned} A_S(t+1) &= [B_S(N(t)) + p_S \exp(-\beta\omega \cdot I)]A_S(t) \\ A_I(t+1) &= [B_I(N(t)) + p_I]A_I(t) + p_S(1 - \exp(-\beta\,\omega \cdot I))A_S(t) \\ F(t+1) &= b_F A_I(t) + p_F F(t). \end{aligned} \tag{29}$$

Initial conditions are non-negative, $A_S(0) > 0$, $A_I(0) > 0$, $F(0) \geq 0$, and all parameters are positive. Hence, solutions are non-negative for $t \geq 0$ The time interval $[t, t+1]$ is the time between reproductive episodes. For amphibians that breed only once per year the time interval is one year. In the model of Emmert and Allen [25], the interval $[t, t+1]$ was subdivided further into a birth interval $[t, t_1]$ and a survival interval $[t_1, t+1]$ during which time no births occur, $B_S \equiv 0 \equiv B_I$. Here, we assume only one time interval $[t, t+1]$.

It is straightforward to verify conditions for existence of a unique DFE for system (29). A positive unique DFE $\bar{A}$ exists if and only if $b_S > 1 - p_S$ (maximal number of births/adult during the interval $[t, t+1]$ is greater than the probability of dying). The DFE is given by

$$\bar{A} = \phi^{-1}\left(\frac{1-p_S}{b_S}\right) > 0. \tag{30}$$

If $b_S \leq 1 - p_S$, then $b_I < 1 - p_I$. Extinction is a consequence of the following inequalities:

$$\begin{aligned} A_S(t+1) &\leq [b_S\phi(A_S(t)) + p_S]A_S(t) \\ A_I(t+1) &< [b_I + p_I]A_I(t). \end{aligned}$$

If $b_I < 1 - p_I$ then $\lim_{t\rightarrow\infty} A_I(t) = 0$. It follows from system (29) that $\lim_{t\rightarrow\infty} F(t) = 0$ In addition, $\{A_S(t)\}_{t=0}^{\infty}$ is a monotone decreasing sequence. The sequence must converge to a fixed point of $A_S = (b_S\phi(A_S) + p_S)A_S$. But the only fixed point of this equation is $A_S = 0$.

We summarize these results in the following theorem.

THEOREM 4.1. *Consider system* (29).

(a) If $b_S \leq 1 - p_S$, then $\lim_{t\to\infty}(A_S(t), A_I(t), F(t)) = (0, 0, 0)$.
(b) If $b_S > 1 - p_S$, then a unique DFE $(\bar{A}, 0, 0)$ exists, where $\bar{A}$ is given by (30).

To compute the basic reproduction number for system (29), the variables are ordered as (A_I, F, A_S), because A_I and F give rise to new infections. Computing the Jacobian matrix J in (3),

$$F = \begin{pmatrix} \beta\omega_A p_S \bar{A} + B_I(\bar{A}) & \beta\omega_F p_S \bar{A} \\ b_F & 0 \end{pmatrix},$$

$T = \text{diag}(p_I, p_F)$, O is the 2×1 zero matrix, and

$$C = p_S + B_S(\bar{A}) + \bar{A}B_S'(\bar{A}).$$

Note that $\rho(T) < 1$ is automatic here. For the conditions of Theorem 2.1 to be satisfied, the scalar C must lie in the interval $(-1,1)$, the condition for stability in the absence of disease. Assuming this restriction holds, the basic reproduction number for system (29) is $\mathcal{R}_0 = \rho(F(\mathbb{I} - T)^{-1})$, which gives

$$\mathcal{R}_0 = \frac{\beta\omega_A p_S \bar{A} + B_I(\bar{A})}{2(1 - p_I)} + \frac{1}{2}\sqrt{\frac{(\beta\omega_A p_S \bar{A} + B_I(\bar{A}))^2}{(1 - p_I)^2} + \frac{4b_F\beta\omega_F p_S \bar{A}}{(1 - p_I)(1 - p_F)}}. \tag{31}$$

The stability results for system (29) are a direct consequence of Theorem 2.1.

COROLLARY 4.2. *Let* $\mathcal{R}_0$ *be defined by* (31). *Assume* $b_S > 1 - p_S$ *and*

$$|p_S + B_S(\bar{A}) + \bar{A}B_S'(\bar{A})| < 1. \tag{32}$$

Then the unique DFE $(\bar{A}, 0, 0)$ *of system* (29)*, where* $\bar{A}$ *is defined in* (30)*, is locally asymptotically stable if* $\mathcal{R}_0 < 1$ *and is unstable if* $\mathcal{R}_0 > 1$.

In the case of a Beverton–Holt functional form $B_S(\bar{A}) + \bar{A}B_S'(\bar{A}) = b_S/(1 + c\bar{A})^2$. Then, condition (32) holds if and only if $b_S > 1 - p_S$ showing that the restriction on the birth function (32) is not required for the Beverton–Holt functional form. However, in the case of a Ricker functional form (28), the additional restriction (32) must be imposed (for stability in the absence of disease).

We now consider two special cases of transmission, where the basic reproduction number has a simple form that can easily be interpreted. In the first case, suppose $\omega_A = 0$, $\omega_F > 0$ and $B_I \equiv 0$. That is, infection occurs only through contact with free-living fungi. In this case, the basic reproduction number simplifies to

$$\mathcal{R}_0 = \sqrt{\frac{b_F\beta\omega_F p_S \bar{A}}{(1 - p_I)(1 - p_F)}},$$

the geometric mean of two reproduction numbers $b_F/(1 - p_F)$ and $\beta\omega_F p_S \bar{A}/(1 - p_I)$. The fungus acts as a vector and for the infection to persist, it must survive in the vector population and in the host population. The fractions $1/(1 - p_F)$ and $1/(1 - p_I)$ can be expressed as infinite sums,

e.g., $1/(1-p_{\mathrm{I}}) = \sum_{k=0}^{\infty} p_{\mathrm{I}}^k$, the lifetime survival of the infected animals. Thus, the two reproduction numbers have a simple recognizable form.

In the second special case, suppose $\omega_{\mathrm{F}} = 0$ and $\omega_{\mathrm{A}} > 0$, where infection occurs only through contact with infected animals A_{I}. Then F has rank one, and the basic reproduction number simplifies to

$$R_0 = \frac{\beta\omega_{\mathrm{A}} p_{\mathrm{S}} \bar{A}}{1-p_{\mathrm{I}}} + \frac{B_{\mathrm{I}}(\bar{A})}{1-p_{\mathrm{I}}}.$$

The basic reproduction number is the sum of horizontal transmission (first term) and vertical transmission (second term) numbers.

4.2 SI patch model

The SI chytridiomycosis model of the previous subsection can be generalized to an n-patch model in a manner similar to the SI hantavirus patch model in Section 3.4. For amphibians, movement occurs between breeding ponds and depends on whether the animal is susceptible or infected. Let $\mathbb{M}^{\mathrm{S}} = (m_{ij}^{\mathrm{S}})$ and $\mathbb{M}^{\mathrm{I}} = (m_{ij}^{\mathrm{I}})$ denote the $n \times n$ dispersal matrices for susceptible and infected amphibians, where $0 \le m_{ij}^{\mathrm{I}} \le m_{ij}^{\mathrm{S}} \le 1$, $j \ne i$, $0 \le m_{ii}^{\mathrm{S}} \le m_{ii}^{\mathrm{I}} \le 1$,

$$\sum_{j=1}^{n} m_{ji}^{\mathrm{S}} \le 1 \text{ and } \sum_{j=1}^{n} m_{ji}^{\mathrm{I}} \le 1.$$

Assume $\mathbb{M}^{\mathrm{S}}$ and $\mathbb{M}^{\mathrm{I}}$ are irreducible. We divide the time interval into two steps, $[t, \hat{t}]$ and $[\hat{t}, t+1]$, as in the SI hantavirus patch model, and we assume that births, deaths and infections occur in the first step. Then, the first step of the n-patch model is a generalization of (29) to patch i:

$$\begin{aligned}
A_{\mathrm{S}}^i(\hat{t}) &= [B_{\mathrm{S}}^i(N^i(t)) + p_{\mathrm{S}}^i \exp(-\beta^i \omega^i \cdot I^i)] A_{\mathrm{S}}^i(t) \\
A_{\mathrm{I}}^i(\hat{t}) &= [B_{\mathrm{I}}^i(N^i(t)) + p_{\mathrm{I}}^i] A_{\mathrm{I}}^i(t) + p_{\mathrm{S}}^i(1 - \exp(-\beta^i \omega^i \cdot I^i)) A_{\mathrm{S}}^i(t) \\
F^i(\hat{t}) &= b_{\mathrm{F}}^i A_{\mathrm{I}}^i(t) + p_{\mathrm{F}}^i F^i(t)
\end{aligned}$$

for $i = 1, \ldots, n$. Let $A_{\mathrm{S}} = (A_{\mathrm{S}}^1, \ldots, A_{\mathrm{S}}^n)^{\mathrm{T}}$, $A_{\mathrm{I}} = (A_{\mathrm{I}}^1, \ldots, A_{\mathrm{I}}^n)^{\mathrm{T}}$ and $\mathbb{F} = (F^1, \ldots, F^n)^{\mathrm{T}}$. Since there is no movement of the fungi, the second step of the n-patch model is

$$A_{\mathrm{S}}(t+1) = \mathbb{M}^{\mathrm{S}} A_{\mathrm{S}}(\hat{t}), \quad A_{\mathrm{I}}(t+1) = \mathbb{M}^{\mathrm{I}} A_{\mathrm{I}}(\hat{t}), \quad \text{and} \quad \mathbb{F}(t+1) = \mathbb{F}(\hat{t}).$$

To verify that a unique DFE exists for this general n-patch model is difficult due to the large number of parameters. For the special case where demographic parameters for the susceptible animals are patch independent, then the results from the previous subsection can be applied. That is, assume $B_{\mathrm{S}}^i \equiv B_{\mathrm{S}}$ and $p_{\mathrm{S}}^i \equiv p_{\mathrm{S}}$, where $B_{\mathrm{S}}(N) = b_{\mathrm{S}}\phi(N)$ with ϕ satisfying the properties described in the previous subsection, and

$$\sum_{j=1}^{n} m_{ji}^{\mathrm{S}} = 1.$$

Then, it can be shown that a unique DFE is patch independent and has the same form as in (30). That is,

$$\bar{A}^i = \phi^{-1}\left(\frac{1-p_S}{b_S}\right) = \bar{A}, \quad i = 1, \ldots, n,$$

provided $b_S > 1 - p_S$. Let matrices F^i and C^i denote the matrices in the previous subsection, where the parameters and states may depend on patch i. That is,

$$F^i = \begin{pmatrix} \beta^i \omega_A^i p_S \bar{A} + B_I^i(\bar{A}) & \beta^i \omega_F^i p_S \bar{A} \\ b_F^i & 0 \end{pmatrix} = \begin{pmatrix} f_{11}^i & f_{12}^i \\ b_F^i & 0 \end{pmatrix}$$

and

$$C^i = p_S + B_S(\bar{A}) + \bar{A}B_S'(\bar{A}).$$

Note that C^i is a patch-independent scalar. Define the $n \times n$ matrices $\mathbb{M}^I_{f_{11}} = (m^I_{ij} f^j_{11})$, $\mathbb{M}^I_{f_{12}} = (m^I_{ij} f^j_{12})$, $\mathbb{M}^I_{p_I} = (m^I_{ij} p^j_I)$, $\mathbb{M}_{b_F} = \mathrm{diag}(b^1_F, \ldots, b^n_F)$ and $\mathbb{M}_{p_F} = \mathrm{diag}(p^1_F, \ldots, p^n_F)$. Then the submatrices F, T, O and C in the Jacobian matrix (3) for the n-patch model are

$$F = \begin{pmatrix} \mathbb{M}^I_{f_{11}} & \mathbb{M}^I_{f_{12}} \\ \mathbb{M}_{b_F} & O_n \end{pmatrix}, \quad T = \begin{pmatrix} \mathbb{M}^I_{p_I} & O_n \\ O_n & \mathbb{M}_{p_F} \end{pmatrix},$$

O is the $2n \times n$ zero matrix, and $C = C^i \mathbb{M}^S$. It is easy to see that $\rho(T) < 1$ and $F + T$ is irreducible. For the case of Beverton–Holt death rate, $\rho(C) < 1$. Hence, Theorem 2.1 applies and the basic reproduction number can be computed using (5) from the matrices F and T derived above. This can easily be computed for a set of given parameter values.

5. Conclusion

The next generation matrix approach has been applied extensively to continuous-time epidemic models to calculate the basic reproduction number, but it is not as well-known in discrete-time epidemic models. However, this approach has been applied widely in the study of discrete-time population models, where the basic reproduction number determines local stability of the extinction equilibrium. This theory allows the next generation matrix approach to be easily extended to discrete-time epidemic models. In this investigation, we describe briefly the next generation matrix approach for calculating the basic reproduction number for general discrete-time epidemic models. Theorem 2.1 summarizes how this approach is used to calculate the basic reproduction number, and provides sufficient conditions so that $\mathcal{R}_0 < 1$ implies local asymptotic stability of the DFE and $\mathcal{R}_0 > 1$ implies instability.

It is interesting to note that the value of $\mathcal{R}_0$ given by equation (23) for the discrete-time SEIR hantavirus model (22) is the same as the basic reproduction number in a continuous-time SEIR hantavirus model (system of differential equations) [7]. The system of differential equations for

the males has the following form:

$$\frac{dS_m}{dt} = \frac{B(N_m, N_f)}{2} - S_m d(N) - S_m(\beta_m I_m + \beta I_f)$$
$$\frac{dE_m}{dt} = -E_m d(N) + S_m(\beta_m I_m + \beta I_f) - \delta E_m$$
$$\frac{dI_m}{dt} = \delta E_m - I_m d(N) - \gamma_m I_m$$
$$\frac{dR_m}{dt} = \gamma_m I_m - R_m d(N),$$

where $d(N) = a + CN$ is a density-dependent natural death rate, $a > 0$ is the per capita, density-independent death rate, and $c > 0$ is the per capita, density-dependent death rate [7]. The unique DFE is $\bar{S}_m = K/2 = \bar{S}_f$, where $K = (b/2 - a)/c$, $0 < a < b/2$, and b is the average litter size. Unlike the difference equations, where the survival probability is multiplied by the population size after infection, in the differential equations, the death rate is subtracted from the rate of population growth. The basic reproduction number for the preceding system of differential equations together with analogous equations for the females is found by the next generation matrix in [39] to be equal to $\mathcal{R}_0$ in (23). Analogous continuous-time SI and SIR hantavirus models, formulated according to the preceding SEIR model, have the same basic reproduction number as in the discrete-time SI and SIR models studied here, namely formulae (17) and (21), respectively.

In this investigation, we applied the next generation matrix approach to six different models that have applications to two emerging wildlife diseases, hantavirus in rodents (SI, SIR, SEIR models and SI patch model) and chytridiomycosis in amphibians (SI model and SI patch model). The conditions of Theorem 2.1 were shown to hold for each of the models. This is the first time that the basic reproduction numbers have been calculated for these discrete-time models. A numerical example for the SI hantavirus model indicates for the bifurcation parameter β_m ($\beta = \beta_m/10$) that a transcritical forward bifurcation occurs at $\mathcal{R}_0 = 1$, showing endemic disease for $\mathcal{R}_0 > 1$. We verified for a special case of the SI hantavirus model that $\mathcal{R}_0 < 1$ implies global stability of the DFE.

The functional form of the basic reproduction number is shown to depend on the various model parameters and the modelling assumptions. The assumptions about births, recovery, latent period, male *versus* female transmission, vector transmission, horizontal *versus* vertical transmission and dispersal have an impact on the magnitude of $\mathcal{R}_0$ and ultimately, on the likelihood of an outbreak.

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