

# Population Persistence and Extinction in a Discrete-time, Stage-structured Epidemic Model

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Dedicated to Saber Elaydi on the occasion of his 60th birthday

A discrete-time model is formulated for the spread of disease in a structured host population. The host population is subdivided into three developmental stages, larva, juvenile and adult, and each stage can be infected by the pathogen. We investigate conditions on the parameters where either the host population does not survive or the host population survives and is free from the disease. Several different submodels of the full structured epidemic model are studied and conditions are derived for global stability of the extinction equilibrium and local stability of the disease-free equilibrium. Some numerical examples are presented to illustrate the dynamics of the model when the disease-free equilibrium is not stable. The motivation for our model is the spread of a fungal pathogen in amphibian populations.

*Keywords:* Structured population, *SIR* epidemic model, Bifurcation diagram, Populations

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## INTRODUCTION

Continuous-time and age-structured epidemic models have been applied to the study of many human diseases (Refs. [2,6,7,9,20,22–24,32,33] and references therein). It is the purpose of this investigation to develop a discrete-time model for the spread of disease in an animal population structured by developmental stages. Discrete-time and structured models have been successfully applied to insect and vertebrate populations, where for example, the discrete time intervals correspond to reproductive periods or average length of a particular stage [1,10,11].

The study of animal diseases has become increasingly important because of potential disease spread to humans (e.g. zoonotic diseases such as Lyme disease, hantavirus pulmonary syndrome and dengue hemorrhagic fever) and disease impact on wildlife populations [13,21,26,31]. Recently, two emerging diseases have been implicated in the worldwide decline of amphibian populations, chytridiomycosis and ranaviral disease [13,15,30]. Chytridiomycosis is a fungal pathogen that causes a high mortality in

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post-metamorphic amphibians [3,4]. It causes a widespread infection of the skin resulting in hyperkeratosis and sloughing off of the skin [5]. Recent evidence suggests chytridiomycosis is one of the causal factors in amphibian declines in Australia, Central America, North America, South America and Western Europe [3–5,8,12,15,25,27,28]. A comprehensive study of 936 amphibian populations indicated a decreasing trend in populations from 1960 to the present [25]. It is the spread of a fungal pathogen in amphibians that serves as the framework for our model development.

In this paper, a discrete-time model is formulated for the spread of disease in a structured host population. The host population is subdivided into three developmental stages, larva, juvenile and adult, and each stage can be infected by the fungal pathogen. We investigate conditions on the parameters where either the host population does not survive or the host population survives and is not infected.

In the second section, the general disease model is formulated. Then, in the fourth and fifth sections, submodels of the general model are analyzed, including a model restricted to only two stages, juvenile and adult, with and without disease, and a model with all three stages, with and without disease. The stability of the extinction equilibrium and disease-free equilibrium are studied. We present some numerical examples in the sixth section to illustrate possible dynamics of the model when the disease-free equilibrium is unstable. The analyses of the simpler submodels help in the analysis of the more complicated models.

## DESCRIPTION OF THE MODEL

The general disease model is based on an *SIR* epidemic model, where the host population is subdivided according to the disease state; susceptible  $S$ , infected  $I$  and immune or recovered individuals  $R$ . In addition, the host population is subdivided into three developmental stages, larva, juvenile and adult, denoted as  $L$ ,  $J$  and  $A$ , respectively. The subscripts  $S$ ,  $I$  and  $R$  denote the disease state of a particular stage. For example,  $J_S$  denotes susceptible juveniles and  $A_I$  denotes infected adults. It is assumed that the disease is caused by a fungal pathogen denoted by the variable  $F$ . The variable  $F$  denotes fungi density, fungi that persists in the environment on the keratin of dead infected animals; it does not refer to fungi growing on infected living animals. Susceptible individuals can become infected either by direct contact with the fungi or by direct contact with any infected individual. Figure 1 is a compartmental diagram of the full structured model.

We model the changes in the disease state and developmental stages of individuals over discrete time intervals,  $\{0, \Delta t, 2\Delta t, \dots\}$ . During the time interval  $\Delta t$ , an individual may move to the next developmental stage,  $L \rightarrow J$ ,  $J \rightarrow A$ , or change to another disease state,  $S \rightarrow I$ ,  $I \rightarrow R$ . Contacts resulting in infection during the time interval  $\Delta t$  are assumed to follow a Poisson distribution. The probability that a susceptible larva does not become infected during the time interval  $\Delta t$  is  $e^{-\beta_L w \cdot I}$ , where

$$w \cdot I = w_L L_I + w_J J_I + w_A A_I + w_F F, \quad (1)$$

$w_k \geq 0, k = L, J, A, F$ . The expression in Eq. (1) is a weighted sum of the infected stages and the fungi. We have assumed a mass action transmission rate. For many animal populations, it is the case that densities are not constant and hence, it is reasonable to assume a transmission rate that is proportional to the population size [14]. The probability that a susceptible larva does become infected during the time interval  $\Delta t$  is  $1 - e^{-\beta_L w \cdot I}$ .

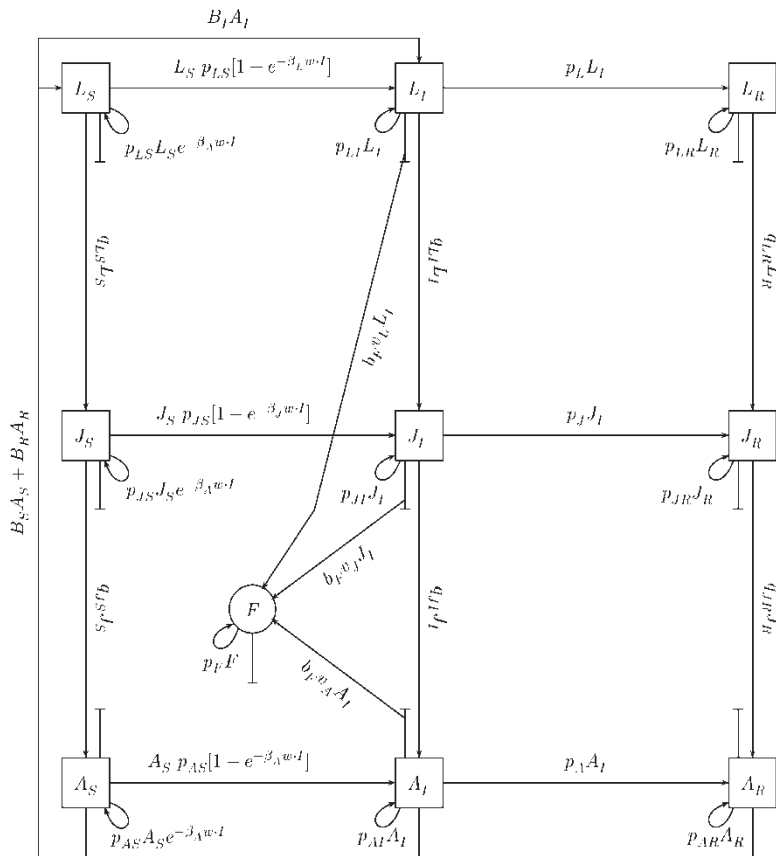


FIGURE 1 A compartmental diagram for the general *SIR* structured epidemic model (5).

The probabilities that a juvenile or an adult do not become infected have similar forms,  $e^{-\beta_j w \cdot I}$  and  $e^{-\beta_A w \cdot I}$ , respectively. The adult stage is the only reproductive stage and infected adults are assumed to reproduce infected eggs which survive to become infected larvae. Immunity is not transferred to newborns (eggs that become larvae); the larvae from immune adults are susceptible.

The birth and survival to the larval stage,  $B_S(T)$ ,  $B_I(T)$  and  $B_R(T)$ , are density-dependent functions of  $T$ , where  $T$  is a weighted sum of the total population size,

$$T = c_{LS}L_S + c_{LI}L_I + c_{LR}L_R + c_{JS}J_S + c_{JI}J_I + c_{JR}J_R + c_{AS}A_S + c_{AI}A_I + c_{AR}A_R, \quad (2)$$

$c_k \geq 0$ . We assume  $B_S(T)$ ,  $B_I(T)$  and  $B_R(T)$  are positive, strictly decreasing functions of  $T \in [0, \infty)$ . In addition,

$$B_S(0) = b_S, \quad B_I(0) = b_I \quad \text{and} \quad B_R(0) = b_R,$$

where  $b_I \leq \min \{b_S, b_R\}$  and  $B_j(T) = b_j \phi(T)$ ,  $j = S, I, R$ . In amphibian populations, predation, competition and cannibalism are especially important during the egg and larval stages. As the population density increases, survival probability to the larval stage decreases. In this paper, two well-known density-dependent functions for  $\phi(T)$  will be considered,

Ricker and Beverton–Holt forms [10]:

$$\text{Ricker : } \phi(T) = e^{-T} \quad (3)$$

$$\text{Beverton–Holt : } \phi(T) = \frac{1}{1+T}. \quad (4)$$

In the model, the fungal growth during the time interval  $\Delta t$  depends on the density of the infected stages,  $L_I$ ,  $J_I$  or  $A_I$ . Fungi in the environment grows on the skin of dead animals, and therefore, fungal growth is a weighted sum of those infected animals that die during the interval  $\Delta t$ ; we denote this as

$$v_L L_I + v_J J_I + v_A A_I,$$

where  $v_k \geq 0$ ,  $k = L, J, A$ . If no animals are infected,  $L_I = 0$ ,  $J_I = 0$  and  $A_I = 0$ , then  $F \rightarrow 0$ , because, in this case,

$$F(t + \Delta t) = p_F F(t), \quad p_F < 1.$$

The *SIR* structured model is given by the following system of difference equations:

$$\begin{aligned} L_S(t + \Delta t) &= L_S p_{LS} e^{-\beta_L w \cdot I} + B_S(T) A_S + B_R(T) A_R \\ L_I(t + \Delta t) &= L_S p_{LS} [1 - e^{-\beta_L w \cdot I}] + p_{LI} L_I + B_I(T) A_I \\ L_R(t + \Delta t) &= p_L L_I + p_{LR} L_R \\ J_S(t + \Delta t) &= q_{LS} L_S + J_S p_{JS} e^{-\beta_J w \cdot I} \\ J_I(t + \Delta t) &= q_{LI} L_I + J_S p_{JS} [1 - e^{-\beta_J w \cdot I}] + p_{JI} J_I \\ J_R(t + \Delta t) &= q_{LR} L_R + p_J J_I + p_{JR} J_R \\ A_S(t + \Delta t) &= q_{JS} J_S + A_S p_{AS} e^{-\beta_A w \cdot I} \\ A_I(t + \Delta t) &= q_{JI} J_I + A_S p_{AS} [1 - e^{-\beta_A w \cdot I}] + p_{AI} A_I \\ A_R(t + \Delta t) &= q_{JR} J_R + p_A A_I + p_{AR} A_R \\ F(t + \Delta t) &= b_F (v_L L_I + v_J J_I + v_A A_I) + p_F F, \end{aligned} \quad (5)$$

where  $w \cdot I$  is given by Eq. (1) and  $T$  is given by Eq. (2). All initial conditions are nonnegative. The state variables on the right-hand side of model (5) are functions of time  $t$  and all of the model parameters are nonnegative. For amphibian populations, it may be the case that the parameters are functions of time, due to environmental variations. However, in this preliminary analysis we assume that all of the parameters are constants with the exception of the birth and survival functions  $B_j(T)$ ,  $j = S, I, R$ . It is straightforward to see that all solutions to model (5) are nonnegative.

The individual parameters are defined below. These parameters represent the probabilities for a change in state or change in density during the time interval  $\Delta t$ .

$p_{Lj}$  = probability a larva remains susceptible, infected or immune,  $j = S, I, R$ .

$p_{Jj}$  = probability a juvenile remains susceptible, infected, or immune,  $j = S, I, R$ .

$p_{Aj}$  = probability an adult remains susceptible, infected, or immune,  $j = S, I, R$ .

$p_L$  = probability a larva recovers from the infection.

- $p_J$  = probability a juvenile recovers from the infection.  
 $p_A$  = probability an adult recovers from the infection.  
 $p_F$  = probability fungi survives when there are no dead infected animals.  
 $q_{Lj}$  = probability a larva becomes a juvenile but remains susceptible, infected, or immune,  
 $j = S, I, R$ .  
 $q_{Jj}$  = probability a juvenile becomes an adult but remains susceptible, infected, or  
immune,  $j = S, I, R$ .  
 $b_F$  = growth of fungi on skin of dead larvae, juveniles, or adults.  
 $B_j(T)$  = number of eggs/adult that survive to larval stage,  $j = S, I, R$ .  
 $\beta_j$  = parameter related to transmission of infection to larvae, juveniles, or adults,  
 $j = L, J, A$ .  
 $v_j, w_i$  = weighting factors, where  $j = L, J, A$  and  $i = L, J, A, F$ .

The probabilities with  $p$  in model (5) involve a transfer between disease states  $S, I$  or  $R$ , whereas the probabilities with  $q$  involve a transfer between stages  $L, J$  or  $A$ . These parameters lie in the interval  $[0, 1)$ . We need some additional constraints on the parameters  $p$  and  $q$  so that they are biologically meaningful.

The restrictions needed on the parameters  $p$  and  $q$  can be seen by considering the dynamics for a susceptible or infected juvenile. It is evident from Fig. 1 that during a time interval  $\Delta t$ , a susceptible juvenile may survive and remain susceptible, with probability  $p_{JS}e^{-\beta_J w^I}$  (they do not become infected). Otherwise, a susceptible juvenile can become infected, with probability  $p_{JS}(1 - e^{-\beta_J w^I})$ , or become a susceptible adult, with probability  $q_{JS}$ . We assume the time interval  $\Delta t$  is small enough so that there cannot be two changes, a change in disease status and a change in developmental stage. Generally, the infection process occurs at a faster rate than the transition to another stage. The probability of juvenile death during the time interval  $\Delta t$  is equal to  $1 - (p_{JS} + q_{JS})$ . Therefore, for susceptible juveniles, we require that  $p_{JS} + q_{JS} < 1$ . Next consider an infected juvenile. An infected juvenile may either survive and remain an infected juvenile, with probability  $p_{JI}$ , become immune, with probability  $p_J$ , or become an infected adult, with probability  $q_{JI}$ , so that  $p_{JI} + p_J + q_{JI} < 1$ . In general, the following eight inequalities must be satisfied:

$$\begin{aligned}
 p_{LS} + q_{LS} < 1, & & p_{LI} + p_L + q_{LI} < 1, \\
 p_{LR} + q_{LR} < 1, & p_{AS} < 1, & \text{and} & p_{JI} + p_J + q_{JI} < 1, \\
 p_{JS} + q_{JS} < 1, & p_{AR} < 1, & & p_{AI} + p_A < 1.
 \end{aligned} \tag{6}$$

In the following sections, various submodels are discussed, a model with only one stage,  $A$ , two stages,  $J$  and  $A$ , and models with all three stages. In all cases, disease-free models are considered first, then  $SI$  epidemic models and finally,  $SIR$  epidemic models.

## THE ADULT MODEL

We begin by simplifying the model to a single adult stage. If no disease is present, the model has the form,

$$A_S(t + \Delta t) = p_{AS} A_S(t) + q_{LS} q_{JS} B_S(T) A_S(t), \tag{7}$$

where  $B_S(T) = b_S\phi(T)$  and  $T \equiv T(t) = c_{AS}A_S(t)$ . Equation (7) does not follow directly from the general model (5). In Eq. (7), it is assumed that only adults are counted and that the time interval  $\Delta t$  is chosen so that the probability eggs survive to the larval stage is  $B_S(T)$ , the probability larvae survive to the juvenile stage is  $q_{LS}$  and the probability juveniles survive to the adult stage is  $q_{JS}$ .

We summarize the dynamics for this simple model (7). Because  $B_S(T) \leq b_S$ , it follows that

$$A_S(t + \Delta t) \leq (p_{AS} + q_{LS}q_{JS}b_S)A_S(t).$$

Therefore, if

$$b_S < \frac{1 - p_{AS}}{q_{LS}q_{JS}} \equiv \Phi_S, \quad (8)$$

then  $\lim_{t \rightarrow \infty} A_S(t) = 0$ . In addition, if the inequality in Eq. (8) is reversed, then there exists a unique positive equilibrium given by

$$\bar{A}_S = \frac{1}{c_{AS}} \phi^{-1} \left( \frac{\Phi_S}{b_S} \right). \quad (9)$$

If the Beverton–Holt functional form is assumed for  $\phi$ , then the inequality  $b_S > \Phi_S$  implies the positive equilibrium is globally asymptotically stable. This follows because the right-hand side of Eq. (7) is a strictly increasing function of  $A_S$ . On the other hand, if the Ricker functional form is assumed, then local stability of the positive equilibrium requires

$$\Phi_S < b_S < \Phi_S e^{2/(1-p_{AS})}.$$

The positive equilibrium becomes unstable when  $b_S = \Phi_S e^{2/(1-p_{AS})}$  and 2-cycles appear. In particular, at  $b_S = \Phi_S e^{2/(1-p_{AS})}$ , there is a period-doubling bifurcation [11,19,29].

When infection is included in model (7) but no immunity, then the model has the form of an *SI* epidemic model,

$$\begin{aligned} A_S(t + \Delta t) &= p_{AS} A_S(t) e^{-\beta_A w^I} + q_{LS} q_{JS} B_S(T) A_S(t) \\ A_I(t + \Delta t) &= p_{AS} A_S(t) (1 - e^{-\beta_A w^I}) + p_{AI} A_I(t) + q_{LI} q_{JI} B_I(T) A_I(t) \\ F(t + \Delta t) &= b_F v_A A_I(t) + p_F F(t), \end{aligned} \quad (10)$$

where  $T \equiv T(t) = c_{AS}A_S(t) + c_{AI}A_I(t)$ . If there is immunity, then the model has the form of an *SIR* epidemic model,

$$\begin{aligned} A_S(t + \Delta t) &= p_{AS} A_S(t) e^{-\beta_A w^I} + q_{LS} q_{JS} (B_S(T) A_S(t) + B_R(T) A_R(t)) \\ A_I(t + \Delta t) &= p_{AS} A_S(t) (1 - e^{-\beta_A w^I}) + p_{AI} A_I(t) + q_{LI} q_{JI} B_I(T) A_I(t) \\ A_R(t + \Delta t) &= p_A A_I(t) + p_{AR} A_R(t) \\ F(t + \Delta t) &= b_F v_A A_I(t) + p_F F(t), \end{aligned} \quad (11)$$

where  $T \equiv T(t) = c_{AS}A_S(t) + c_{AI}A_I(t) + c_{AR}A_R(t)$ . For models (10) and (11), there exists a unique disease-free equilibrium given by  $A_S = \bar{A}_S$ ,  $A_I = 0$ ,  $A_R = 0$  and  $F = 0$ , where  $\bar{A}_S$  is

defined in Eq. (9), provided  $b_S > \Phi_S$ . If, on the other hand,

$$b_S < \Phi_S \quad \text{and} \quad b_I < \frac{1 - p_{AI}}{q_{LS} q_{JS}} \equiv \Phi_I, \quad (12)$$

then the zero or extinction equilibrium is locally asymptotically stable. The extinction equilibrium is also globally asymptotically stable in model (10). Global stability follows in the case of Eq. (10) because the population vector  $X(t) = (A_S(t), A_I(t), F(t))^T$  satisfies

$$X(t + \Delta t) \leq GX(t),$$

where

$$G = \begin{pmatrix} q_{LS}q_{JS}b_S + p_{AS} & 0 & 0 \\ p_{AS} & q_{LI}q_{JI}b_I + p_{AI} & 0 \\ 0 & b_F v_A & p_F \end{pmatrix}$$

has the same eigenvalues as the Jacobian matrix evaluated at the zero equilibrium. The Jacobian matrix is the same as matrix  $G$  with the exception of the (2, 1) element; this element is zero in the Jacobian matrix. If the conditions in Eq. (12) are satisfied, then the eigenvalues of  $G$  satisfy  $|\lambda| < 1$  and  $G^k$  approaches the zero matrix as  $k \rightarrow \infty$ . These results are summarized in the following theorem.

**THEOREM 1** (a) *If  $b_S < \Phi_S$ , then the extinction equilibrium of model (7) is globally asymptotically stable. If  $b_S > \Phi_S$ , then a unique positive equilibrium exists to model (7) and it is globally asymptotically stable in the case of the Beverton–Holt birth function and it is locally asymptotically stable in the case of the Ricker birth function if, in addition,*

$$b_S < \Phi_S e^{2/(1-p_{AS})}.$$

(b) *If  $b_S < \Phi_S$  and  $b_I < \Phi_I$ , then the extinction equilibrium of model (10) is globally asymptotically stable and in model (11), it is locally asymptotically stable. If  $b_S > \Phi_S$ , then a unique positive equilibrium exists to models (10) and (11).*

*The parameters  $\Phi_S$  and  $\Phi_I$  are defined in Eqs. (8) and (12), respectively.*

Note that we have expressed the stability conditions in terms of the parameter  $b_S$ . In the numerical examples, the parameter  $b_S$  is the bifurcation parameter.

## THE JUVENILE AND ADULT MODEL

Next, we analyze the general model (5) with only two developmental stages, juvenile and adult. First the disease-free model is analyzed, then the *SI* epidemic model and finally, the *SIR* epidemic model.

### Disease-free Model

When there is no disease present and only two developmental stages, *J* and *A*, the model takes the form,

$$\begin{aligned} J_S(t + \Delta t) &= p_{JS}J_S(t) + q_{LS}B_S(T)A_S(t) \\ A_S(t + \Delta t) &= q_{JS}J_S(t) + p_{AS}A_S(t), \end{aligned} \quad (13)$$

where  $T \equiv T(t) = c_{JS}J_S(t) + c_{AS}A_S(t)$ . Model (13) does not follow directly from the general model (5). In model (13) it is assumed that only juveniles and adults are counted and that the time interval  $\Delta t$  is chosen so that the probability eggs survive to the larval stage is  $B_S(T)$ , the probability larvae survive to the juvenile stage is  $q_{LS}$ .

It is easy to show that all equilibria lie on the line

$$A = \frac{q_{JS}}{1 - p_{AS}} J.$$

A unique positive equilibrium exists  $(\bar{J}_S, \bar{A}_S)$  given by

$$\bar{J}_S = \frac{1 - p_{AS}}{c_{JS}(1 - p_{AS}) + c_{AS}q_{JS}} \phi^{-1}\left(\frac{\Lambda_S}{b_S}\right)$$

$$\bar{A}_S = \frac{q_{JS}}{c_{JS}(1 - p_{AS}) + c_{AS}q_{JS}} \phi^{-1}\left(\frac{\Lambda_S}{b_S}\right),$$

if

$$\phi^{-1}\left(\frac{\Lambda_S}{b_S}\right) > 0 \quad \text{or} \quad b_S > \Lambda_S, \quad (14)$$

where

$$\Lambda_S \equiv \frac{(1 - p_{JS})(1 - p_{AS})}{q_{LS}q_{JS}}. \quad (15)$$

We note that

$$\bar{T} = (c_{JS}\bar{J}_S + c_{AS}\bar{A}_S) = \phi^{-1}\left(\frac{\Lambda_S}{b_S}\right) \quad (16)$$

and that  $\phi(\bar{T}) = \Lambda_S/b_S$ .

The Jacobian matrix of model (13) has the following form:

$$M = \begin{pmatrix} c_{JS}q_{LS}b_S \frac{\partial \phi(T)}{\partial T} A_S + p_{JS} & q_{LS}b_S (c_{AS} \frac{\partial \phi(T)}{\partial T} A_S + \phi(T)) \\ q_{JS} & p_{AS} \end{pmatrix}. \quad (17)$$

The Jacobian matrix evaluated at  $(0, 0)$  has a particularly simple form

$$M_0 = \begin{pmatrix} p_{JS} & q_{LS}b_S \\ q_{JS} & p_{AS} \end{pmatrix}.$$

Application of the Jury conditions [17,18],  $|\text{Tr}(M_0)| < 1 + \det(M_0) < 2$ , shows that the following inequality must hold for local asymptotic stability of the extinction equilibrium  $(0, 0)$ :

$$b_S < \Lambda_S. \quad (18)$$

Also, notice that whenever inequality (18) is satisfied, there can be no positive equilibria as this violates the condition for existence of a positive equilibrium given by inequality (14).

Denote the vector  $X(t) = (J_S(t), A_S(t))^T$ . Then, it follows that  $X(t + \Delta t) \leq M_0 X(t)$  because  $B_S(T) \leq b_S$ . Hence,

$$X(k + \Delta t) \leq M_0^k X(0).$$

If condition (18) is satisfied, then the eigenvalues of  $M_0$  satisfy  $|\lambda| < 1$ . Hence,  $M_0^k$  approaches the zero matrix as  $k$  approaches infinity. Therefore, if the condition in Eq. (18) is satisfied, the zero equilibrium is globally asymptotically stable.

Next, consider the positive equilibrium. Because  $\partial \phi(T)/\partial T$  appears in the Jacobian matrix,  $M$ , stability criteria for the positive equilibria cannot be evaluated unless a particular functional form for  $B_S(T)$  is chosen. We assume one of two well-known forms, Ricker and Beverton–Holt [10]. In the case of a Ricker function,

$$\phi(T) = e^{-T}.$$

Thus,  $\partial \phi(T)/\partial T = -\phi(T)$  and  $\phi^{-1}(T) = \ln(b_S/\Lambda_S)$ . Because  $\phi(\bar{T})$  satisfies Eq. (16), we have  $[\partial \phi/\partial T]|_{T=\bar{T}} = -\Lambda_S/b_S$ . In addition,

$$\begin{aligned} \bar{J}_S &= \frac{1 - p_{AS}}{c_{JS}(1 - p_{AS}) + c_{AS}q_{JS}} \ln\left(\frac{b_S}{\Lambda_S}\right) \\ \bar{A}_S &= \frac{q_{JS}}{c_{JS}(1 - p_{AS}) + c_{AS}q_{JS}} \ln\left(\frac{b_S}{\Lambda_S}\right). \end{aligned}$$

After evaluating the Jacobian matrix,  $M$ , at the positive equilibrium and applying the Jury conditions, the results are summarized below. Let

$$\bar{C} = q_{JS}c_{AS} - p_{AS}c_{JS}. \quad (19)$$

If  $\bar{C} > 0$ , then the positive equilibrium is locally asymptotically stable if  $b_S < \Lambda_S e^W$ , where

$$W = \frac{(2 - p_{JS} - p_{AS})(c_{JS} + \bar{C})}{\bar{C}(1 - p_{JS})(1 - p_{AS})} > 0. \quad (20)$$

If  $\bar{C} < c_{JS}$ , then the positive equilibrium is locally asymptotically stable if  $b_S < \Lambda_S e^K$ , where

$$K = \frac{2(p_{JS} + p_{AS})(c_{JS} + \bar{C})}{(c_{JS} - \bar{C})(1 - p_{JS})(1 - p_{AS})} > 0. \quad (21)$$

Next, assume that the density-dependent function  $\phi$  has a Beverton–Holt form,

$$\phi(T) = \frac{1}{1 + T}.$$

Thus,  $\partial \phi(T)/\partial T = -(1 + T)^{-2} = -(\phi(T))^2$ ,  $\phi^{-1}(T) = (1/T) - 1$  and

$$\bar{T} = c_{JS}\bar{J}_S + c_{AS}\bar{A}_S = \phi^{-1}\left(\frac{\Lambda_S}{b_S}\right) = \frac{b_S}{\Lambda_S} - 1.$$

Therefore,

$$\bar{J}_S = \frac{1 - p_{AS}}{c_{JS}(1 - p_{AS}) + c_{AS}q_{JS}} \left( \frac{b_S}{\Lambda_S} - 1 \right) \quad (22)$$

$$\bar{A}_S = \frac{q_{JS}}{c_{JS}(1 - p_{AS}) + c_{AS}q_{JS}} \left( \frac{b_S}{\Lambda_S} - 1 \right). \quad (23)$$

Evaluation of the Jacobian matrix,  $M$ , at the positive equilibrium (22) and (23) leads to the following results concerning local asymptotic stability of the positive equilibrium. If  $\bar{C} < c_{JS}$  and  $0 < K < 1$ , then local asymptotic stability requires

$$b_S < \frac{\Lambda_S}{1 - K}, \quad (24)$$

where  $K$  is defined by Eq. (21). We summarize the results in the following theorem.

**THEOREM 2** (a) *If  $b_S < \Lambda_S$ , then the extinction equilibrium of model (13) is globally asymptotically stable.*

(b) *If  $b_S > \Lambda_S$ , then a unique positive equilibrium of model (13) exists and is locally asymptotically stable subject to the following conditions:*

- (i) *For the Ricker birth function, if  $\bar{C} > 0$ , then local asymptotic stability requires  $b_S < \Lambda_S e^W$  and if  $\bar{C} < c_{JS}$ , then local asymptotic stability requires  $b_S < \Lambda_S e^K$ .*
- (ii) *For the Beverton–Holt birth function, if  $\bar{C} < c_{JS}$  and  $0 < K < 1$ , then local asymptotic stability requires  $b_S < \Lambda_S/(1 - K)$ .*

*The parameters  $\Lambda_S$ ,  $\bar{C}$ ,  $W$  and  $K$  are defined in Eqs. (15), (19), (20) and (21), respectively.*

In the special case that the density-dependent function  $B_S(T)$  only depends on the adult population,  $c_{JS} = 0$ , the second criterion in (i) and the stability condition (ii) are not needed. In this case,  $K < 0$ , and local stability in the Beverton–Holt case simplifies to  $b_S > \Lambda_S$  and in the Ricker case to  $\Lambda_S < b_S < \Lambda_S e^W$ . In addition, if  $B_S(T)$  only depends on the juvenile population,  $c_{AS} = 0$ , then the positive equilibrium is locally stable in the Ricker case if  $\Lambda_S < b_S < \Lambda_S e^K$  and in the Beverton–Holt case if  $\Lambda_S < b_S < \Lambda_S/(1 - K)$ .

### The SI Juvenile and Adult Model

Next, infection is included in the juvenile and adult model. The model takes the form,

$$\begin{aligned} J_S(t + \Delta t) &= p_{JS}J_S e^{-\beta_J w I} + q_{LS}B_S(T)A_S \\ J_I(t + \Delta t) &= p_{JS}J_S(1 - e^{-\beta_J w I}) + p_{JI}J_I + q_{LI}B_I(T)A_I \\ A_S(t + \Delta t) &= q_{JS}J_S + p_{AS}A_S e^{-\beta_A w I} \\ A_I(t + \Delta t) &= q_{JI}J_I + p_{AS}A_S(1 - e^{-\beta_A w I}) + p_{AI}A_I \\ F(t + \Delta t) &= b_F(v_J J_I + v_A A_I) + p_F F, \end{aligned} \quad (25)$$

where on the right side the state variables are functions of time  $t$ . The birth functions,  $B_S(T)$  and  $B_I(T)$ , are functions of  $T = c_{JS}J_S + c_{JI}J_I + c_{AS}A_S + c_{AI}A_I + c_FF$ . The disease-free and extinction equilibria of model (25) are the same as those in model (13).

Let  $X(t) = (J_S(t), A_S(t), J_I(t), A_I(t), F(t))^T$ . Then the Jacobian matrix of model (25) evaluated at the zero equilibrium has the form

$$M_0 = \begin{pmatrix} p_{JS} & q_{LS}b_S & 0 & 0 & 0 \\ q_{JS} & p_{AS} & 0 & 0 & 0 \\ 0 & 0 & p_{JI} & q_{LI}b_I & 0 \\ 0 & 0 & q_{JI} & p_{AI} & 0 \\ 0 & 0 & b_F v_J & b_F v_A & p_F \end{pmatrix}.$$

The eigenvalues of  $M_0$  are  $p_F$  and the eigenvalues of the two submatrices:

$$\begin{pmatrix} p_{JS} & q_{LS}b_S \\ q_{JS} & p_{AS} \end{pmatrix} \quad \text{and} \quad \begin{pmatrix} p_{JI} & q_{LI}b_I \\ q_{JI} & p_{AI} \end{pmatrix}. \quad (26)$$

The matrices in Eq. (26) have the same structure and have been completely analyzed for model (13). Hence, for stability of the extinction equilibrium, two inequalities must be satisfied:

$$b_S < \frac{(1 - p_{JS})(1 - p_{AS})}{q_{LS}q_{JS}} \equiv \Lambda_S \quad \text{and} \quad b_I < \frac{(1 - p_{JI})(1 - p_{AI})}{q_{LI}q_{JI}} \equiv \Lambda_I. \quad (27)$$

Also, notice that whenever inequalities (27) are satisfied, there can be no positive disease-free equilibria as this violates the condition for the positive equilibria criteria given in inequality (14).

The *SI* juvenile and adult model is bounded above, that is,  $X(t + \Delta t) \leq GX(t)$ , where

$$G = \begin{pmatrix} p_{JS} & q_{LS}b_S & 0 & 0 & 0 \\ q_{JS} & p_{AS} & 0 & 0 & 0 \\ p_{JS} & 0 & p_{JI} & q_{LI}b_I & 0 \\ 0 & p_{AS} & q_{JI} & p_{AI} & 0 \\ 0 & 0 & b_F v_J & b_F v_A & p_F \end{pmatrix}.$$

Matrix  $G$  has the same eigenvalues as  $M_0$ . Hence, if conditions (27) are satisfied, then the zero equilibrium is globally asymptotically stable.

The special case of the disease-free positive equilibrium will be partially analyzed. This can be accomplished without any assumptions about the functional forms for  $B_S(T)$  and  $B_I(T)$  due to previous work. The eigenvalues of the Jacobian matrix are determined by the eigenvalues of the following submatrices

$$\begin{pmatrix} q_{LS}b_S \frac{\partial \phi(\bar{T})}{\partial T} c_{JS} \bar{A}_S + p_{JS} & q_{LS}b_S \frac{\partial \phi(\bar{T})}{\partial T} c_{AS} \bar{A}_S + q_{LS}b_S \phi(\bar{T}) \\ q_{JS} & p_{AS} \end{pmatrix} \quad (28)$$

and

$$\begin{pmatrix} \beta_{JWJPJS} \bar{J}_S + p_{JI} & q_{LI}b_I \phi(\bar{T}) + \beta_{JWAPJS} \bar{J}_S & \beta_{JWFPJS} \bar{J}_S \\ q_{JI} + \beta_{AWJPAS} \bar{A}_S & \beta_{AWAPAS} \bar{A}_S + p_{AI} & \beta_{AWFPAS} \bar{A}_S \\ b_F v_J & b_F v_A & p_F \end{pmatrix}. \quad (29)$$

Notice that matrix (28) is the same as the Jacobian matrix (17). Hence, all results about matrix (28) with either a Ricker function or a Beverton–Holt function apply. The Jury conditions can be applied to the  $3 \times 3$  matrix (29). However, they cannot be expressed in a simple form that relates to the magnitudes of  $b_S$  and  $b_I$ . Some simple sufficient but not necessary conditions for local asymptotic stability of the positive equilibrium uses matrix norms. If all of the absolute row sums are less than one, then the absolute value all of the eigenvalues of matrix (29) are less than one. These results are summarized below:

$$b_F < \frac{1 - p_F}{v_J + v_A} \quad (30)$$

$$b_S < \frac{(1 - p_{JS})(1 - p_{AS})}{p_L q_{LI} q_{JS}} e^N \quad (31)$$

$$b_I < \frac{1 - p_{JI} - \beta_J p_{JS} \bar{J}_S (w_J + w_A + w_F)}{q_{LI} \phi(\bar{T})}, \quad (32)$$

where

$$N = \frac{(c_{JS} + \bar{C})(1 - p_{AI} - q_{JI})}{q_{JS} \beta_A p_{AS} (w_J + w_A + w_F)}.$$

Note that  $N$  may be negative as could the right-hand side of criteria (32). We summarize these results in the following theorem.

**THEOREM 3** (a) *If  $b_S < \Lambda_S$  and  $b_I < \Lambda_I$ , then the extinction equilibrium of model (25) is globally asymptotically stable.*

(b) *If  $b_S > \Lambda_S$ , then a unique positive disease-free equilibrium exists to model (25) and sufficient conditions for locally asymptotic stability are given by the conditions in Theorem 2(b) and the criteria in Eqs. (30)–(32).*

*The parameters  $\Lambda_S$  and  $\Lambda_I$  are defined in Eq. (27).*

### The SIR Juvenile and Adult Model

The SIR juvenile and adult submodel of model (5) reduces to the following seven difference equations:

$$\begin{aligned} J_S(t + \Delta t) &= p_{JS} J_S e^{-\beta_J w \cdot I} + q_{LS} B_S(T) A_S + q_{LS} B_R(T) A_R \\ J_I(t + \Delta t) &= p_{JS} J_S (1 - e^{-\beta_J w \cdot I}) + p_{JI} J_I + q_{LI} B_I(T) A_I \\ J_R(t + \Delta t) &= p_J J_I + p_{JR} J_R \\ A_S(t + \Delta t) &= q_{JS} J_S + p_{AS} A_S e^{-\beta_A w \cdot I} \\ A_I(t + \Delta t) &= q_{JI} J_I + p_{AS} A_S (1 - e^{-\beta_A w \cdot I}) + p_{AI} A_I \\ A_R(t + \Delta t) &= q_{JIR} J_I + q_{JR} J_R + p_A A_I + p_{AR} A_R \\ F(t + \Delta t) &= b_F (v_J J_I + v_A A_I) + p_F F. \end{aligned} \quad (33)$$

The birth functions,  $B_S(T)$ ,  $B_I(T)$  and  $B_R(T)$ , depend on  $T = c_{JS}J_S + c_{JI}J_I + c_{JR}J_R + c_{AS}A_S + c_{AI}A_I + c_{AR}A_R + c_FF$ . The disease-free and extinction equilibria of model (25) are the same as those in model (13). For this model, we only consider stability of the extinction equilibrium.

The Jacobian matrix evaluated at the zero equilibrium, after suitable rearranging,  $X(t) = (J_S(t), A_S(t), J_R(t), A_R(t), J_I(t), A_I(t), F(t))^T$ , is given by

$$M_0 = \begin{pmatrix} p_{JS} & q_{LS}b_S & 0 & q_{LS}b_R & 0 & 0 & 0 \\ q_{JS} & p_{AS} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & p_{JR} & 0 & p_J & 0 & 0 \\ 0 & 0 & q_{JR} & p_{AR} & q_{JIR} & p_A & 0 \\ 0 & 0 & 0 & 0 & p_{JI} & q_{LI}b_I & 0 \\ 0 & 0 & 0 & 0 & q_{JI} & p_{AI} & 0 \\ 0 & 0 & 0 & 0 & b_F v_J & b_F v_A & p_F \end{pmatrix}.$$

Hence, the eigenvalues of  $M_0$  are  $p_F$ ,  $p_{JR}$ ,  $p_{AR}$ , and the eigenvalues of the two submatrices given in Eq. (26). The local stability results are the same as for the *SI* juvenile and adult model. Thus, the following result is a corollary of Theorem 3.

**COROLLARY 1** *The extinction equilibrium of model (33) is locally asymptotically stable if  $b_S < \Lambda_S$  and  $b_I < \Lambda_I$ , where  $\Lambda_S$  and  $\Lambda_I$  are defined in Eq. (27). If  $b_S > \Lambda_S$ , then a unique positive disease-free equilibrium exists.*

## THE LARVA, JUVENILE AND ADULT MODEL

### Disease-free Model

In this special case of the general model (5), only susceptible larvae, juveniles and adults are included. The model is given by

$$\begin{aligned} L_S(t + \Delta t) &= p_{LS}L_S(t) + B_S(T)A_S(t) \\ J_S(t + \Delta t) &= q_{LS}L_S(t) + p_{JS}J_S(t) \\ A_S(t + \Delta t) &= q_{JS}J_S(t) + p_{AS}A_S(t). \end{aligned} \quad (34)$$

The birth function  $B_S(T)$  depends on  $T \equiv T(t) = c_{LS}L_S(t) + c_{JS}J_S(t) + c_{AS}A_S(t)$ .

Define

$$\Psi_S = \frac{(1 - p_{LS})(1 - p_{JS})(1 - p_{AS})}{q_{LS}q_{JS}}. \quad (35)$$

A unique, positive equilibrium exists to model (34) and is given by

$$\begin{aligned} \bar{L}_S &= \frac{(1 - p_{JS})(1 - p_{AS})}{C} \phi^{-1} \left( \frac{\Psi_S}{b_S} \right) \\ \bar{J}_S &= \frac{q_{LS}(1 - p_{AS})}{C} \phi^{-1} \left( \frac{\Psi_S}{b_S} \right) \\ \bar{A}_S &= \frac{q_{LS}q_{JS}}{C} \phi^{-1} \left( \frac{\Psi_S}{b_S} \right), \end{aligned}$$

where  $C = c_{LS}(1 - p_{JS})(1 - p_{AS}) + c_{JS}q_{JS}(1 - p_{AS}) + c_{AS}q_{LS}q_{JS}$  provided  
 $b_S > \Psi_S$ .

We do not study the stability of the positive equilibrium.

The extinction equilibrium has a Jacobian matrix of the form

$$M_0 = \begin{pmatrix} p_{LS} & 0 & b_S \\ q_{LS} & p_{JS} & 0 \\ 0 & q_{JS} & p_{AS} \end{pmatrix}.$$

Applying the Jury conditions for a  $3 \times 3$  matrix and using extensive manipulations, it can be shown that the eigenvalues of  $M_0$  have magnitude less than one if  $b_S < \Psi_S$ . In addition,  $X(t + \Delta t) \leq M_0 X(t)$ , where  $X(t) = (L_S(t), J_S(t), A_S(t))^T$ . The results of model (34) are summarized in the following theorem.

**THEOREM 4** *If  $b_S < \Psi_S$ , then the extinction equilibrium of model (34) is globally asymptotically stable. If  $b_S > \Psi_S$ , a unique positive disease-free equilibrium exists to model (34). The parameter  $\Psi_S$  is defined in Eq. (35).*

### The *SI* and *SIR* Larva, Juvenile and Adult Models

The *SI* larva, juvenile and adult model is given by the following seven difference equations:

$$\begin{aligned} L_S(t + \Delta t) &= p_{LS}L_S e^{-\beta_L w \cdot I} + B_S(T)A_S \\ L_I(t + \Delta t) &= p_{LS}L_S(1 - e^{-\beta_L w \cdot I}) + p_{LI}L_I + B_I(T)A_I \\ J_S(t + \Delta t) &= q_{LS}L_S + p_{JS}J_S e^{-\beta_J w \cdot I} \\ J_I(t + \Delta t) &= q_{LI}L_I + p_{JS}J_S(1 - e^{-\beta_J w \cdot I}) + p_{JI}J_I \\ A_S(t + \Delta t) &= q_{JS}J_S + p_{AS}A_S e^{-\beta_A w \cdot I} \\ A_I(t + \Delta t) &= q_{JI}J_I + p_{AS}A_S(1 - e^{-\beta_A w \cdot I}) + p_{AI}A_I \\ F(t + \Delta t) &= b_F(v_L L_I + v_J J_I + v_A A_I) + p_F F. \end{aligned} \quad (36)$$

The birth functions  $B_S(T)$  and  $B_I(T)$  depend on  $T = c_{LS}L_S + c_{LI}L_I + c_{JS}J_S + c_{JI}J_I + c_{AS}A_S + c_{AI}A_I$ . The positive disease-free equilibrium is the same as for model (34). Stability is only examined for the case of the extinction equilibrium. The Jacobian matrix evaluated at the extinction equilibrium, after rearrangement,  $X(t) = (L_S(t), J_S(t), A_S(t), L_I(t), J_I(t), A_I(t), F(t))^T$ , is

$$M_0 = \begin{pmatrix} p_{LS} & 0 & b_S & 0 & 0 & 0 & 0 \\ q_{LS} & p_{JS} & 0 & 0 & 0 & 0 & 0 \\ 0 & q_{JS} & p_{AS} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & p_{LI} & 0 & b_I & 0 \\ 0 & 0 & 0 & q_{LI} & p_{JI} & 0 & 0 \\ 0 & 0 & 0 & 0 & q_{JI} & p_{AI} & 0 \\ 0 & 0 & 0 & b_F v_L & b_F v_J & b_F v_A & p_F \end{pmatrix}. \quad (37)$$

The eigenvalues of  $M_0$  are  $p_F$  and those given by the submatrices

$$\begin{pmatrix} p_{LS} & 0 & b_S \\ q_{LS} & p_{JS} & 0 \\ 0 & q_{JS} & p_{AS} \end{pmatrix} \quad \text{and} \quad \begin{pmatrix} p_{LI} & 0 & b_I \\ q_{LI} & p_{JI} & 0 \\ 0 & q_{JI} & p_{AI} \end{pmatrix}. \quad (38)$$

The matrices in Eq. (38) have the same form as those for the disease-free model (34). Hence, for the extinction equilibrium to be locally asymptotically stable requires

$$b_S < \Psi_S \quad \text{and} \quad b_I < \frac{(1 - p_{LI})(1 - p_{JI})(1 - p_{AI})}{q_{LI} q_{JI}} \equiv \Psi_I. \quad (39)$$

It can easily be seen that  $X(t + \Delta t) \leq GX(t)$ , where

$$G = \begin{pmatrix} p_{LS} & 0 & b_S & 0 & 0 & 0 & 0 \\ q_{LS} & p_{JS} & 0 & 0 & 0 & 0 & 0 \\ 0 & q_{JS} & p_{AS} & 0 & 0 & 0 & 0 \\ p_{LS} & 0 & 0 & p_{LI} & 0 & b_I & 0 \\ 0 & p_{JS} & 0 & q_{LI} & p_{JI} & 0 & 0 \\ 0 & 0 & p_{AS} & 0 & q_{JI} & p_{AI} & 0 \\ 0 & 0 & 0 & b_F v_L & b_F v_J & b_F v_A & p_F \end{pmatrix}.$$

Matrix  $G$  has the same eigenvalues as  $M_0$ . Thus, the extinction equilibrium is globally asymptotically stable for Eq. (36) provided the eigenvalues of  $M_0$  (or  $G$ ) have magnitude less than one. Before stating the results we consider the full structured model (5).

For the original model (5), the positive disease-free equilibrium is the same as model (34). Stability is only examined for the case of the zero equilibrium. The associated Jacobian matrix has eigenvalues  $p_{LR}$ ,  $p_{JR}$ ,  $p_{AR}$ ,  $p_F$  and eigenvalues of the submatrices (38). Hence, for the zero equilibrium to be locally asymptotically stable, the inequalities (39) must be satisfied. We have the following corollary to Theorem 4.

**COROLLARY 2** *If the conditions in Eq. (39) are satisfied, then the extinction equilibrium of model (5) is locally asymptotically stable and for Eq. (36) it is globally asymptotically stable. If  $b_S > \Psi_S$ , then a unique positive disease-free equilibrium exists to models (5) and (36).*

## NUMERICAL EXAMPLES

Numerical examples using the Ricker or Beverton–Holt functional forms (Eqs. (3) and (4))  $B_j(T) = b_j \phi(T)$ ,  $j = S, I$  are presented. A random integer-valued initial population size from the set  $\{1, 2, \dots, 20\}$  is assigned to each of the variables. Numerical examples for the disease-free models and the  $SI$  models are discussed. Parameter values used in the numerical examples are given in Table I. The values of the parameters are chosen for illustration

TABLE I Parameter values used in the numerical examples

<i>Susceptible</i>		<i>Infected</i>		<i>Contact</i>		<i>Fungus</i>	
<i>Parameter</i>	<i>Value</i>	<i>Parameter</i>	<i>Value</i>	<i>Parameter</i>	<i>Value</i>	<i>Parameter</i>	<i>Value</i>
$p_{LS}$	0.03	$p_{LI}$	0.02	$w_L$	0.4	$b_F$	10
$p_{JS}$	0.04	$p_{JI}$	0.03	$w_A$	0.4	$p_F$	0.5
$p_{AS}$	0.05	$p_{AI}$	0.04	$w_F$	0.4	$v_L$	1
$q_{LS}$	0.2	$q_{LI}$	0.1	$\beta_L$	1	$v_J$	1
$q_{JS}$	0.3	$q_{JI}$	0.2	$\beta_J$	1	$v_A$	1
$c_{LS}$	0.1	$c_{LI}$	0.1	$\beta_A$	1		
$c_{JS}$	0.1	$c_{JI}$	0.1				
$c_{AS}$	0.1	$c_{AI}$	0.1				

purposes and do not represent actual values. In the bifurcation diagrams, the bifurcation parameter is  $b_S$ . It is assumed that  $b_I = (1/2)b_S$ .

In the first example, the dynamics of the disease-free juvenile and adult model (13) are illustrated for the parameter values listed in Table I and a Ricker birth function. A bifurcation diagram showing the equilibrium total population size for  $b_S \in [12, 26]$  and  $b_S \in [100, 350]$

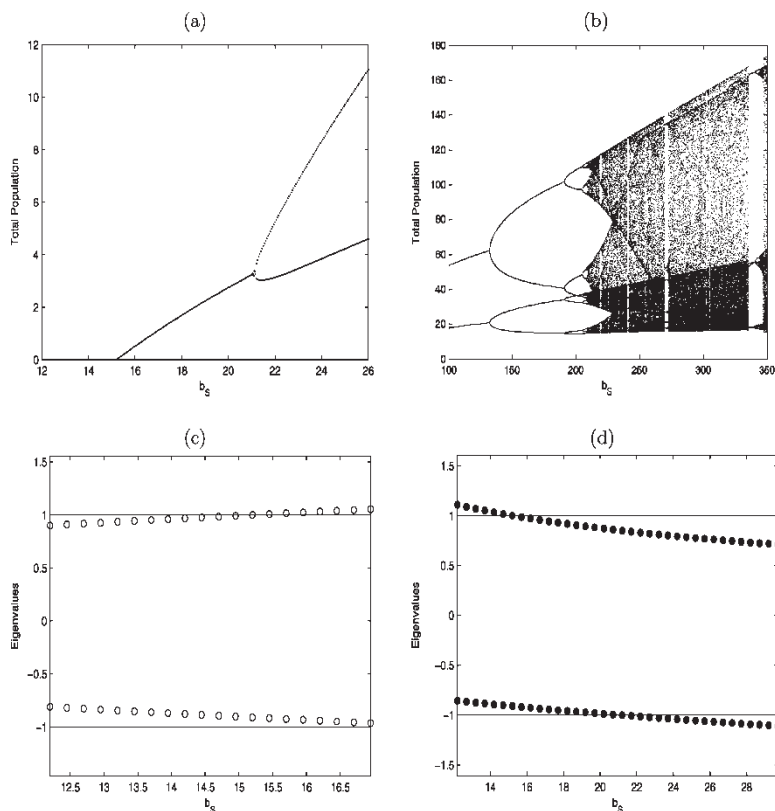


FIGURE 2 A bifurcation diagram for the susceptible juvenile and adult model (13) with the Ricker birth and survival function. (a) and (b) are bifurcation diagrams for the total population as a function of  $b_S$ . (c) is a plot of the real eigenvalues of the Jacobian matrix evaluated at the extinction equilibrium. (d) is a plot of the real eigenvalues of the Jacobian matrix evaluated at the positive equilibrium.

is given in Figures 2(a) and (b). Note that the values of the population size are not significant since the variables can be scaled by a constant. Figure 2(c) plots the two real eigenvalues,  $\lambda_1$  and  $\lambda_2$ , of the Jacobian matrix evaluated at the extinction equilibrium as a function of  $b_S$ . When  $b_S < \Lambda_S = 15.2$ , the extinction equilibrium is stable. At  $b_S = 15.2$ ,  $\lambda_1 = 1$ , a transcritical bifurcation occurs. For  $15.2 < b_S < \Lambda_S \min\{e^W, e^K\} \approx 21.12$ , the positive disease-free equilibrium is stable. Figure 2c plots the two real eigenvalues,  $\lambda_1$  and  $\lambda_2$ , of the Jacobian matrix evaluated at the positive equilibrium. When  $b_S = 21.12$ , there is a period-doubling bifurcation,  $\lambda_2 = -1$ . The period-doubling bifurcation is evident in Figure 2(b).

The dynamics of the *SI* juvenile and adult model (25) are illustrated in the next example with a Ricker birth function. Figures 3(a) and (b) are bifurcation diagrams for the total population as a function of  $b_S$ . Figures 3(c) and (d) are bifurcation diagrams for the susceptible and infected populations, respectively. For  $b_S < \Lambda_S = 15.2$  and  $b_I < \Lambda_I = 46.56$ , the extinction equilibrium is stable, and when  $b_S = 15.2$  a transcritical bifurcation occurs. The disease-free equilibrium is stable for  $15.2 < b_S < 18$ . At  $b_S \approx 18$  another transcritical bifurcation occurs, the disease-free equilibrium becomes unstable and an endemic equilibrium becomes

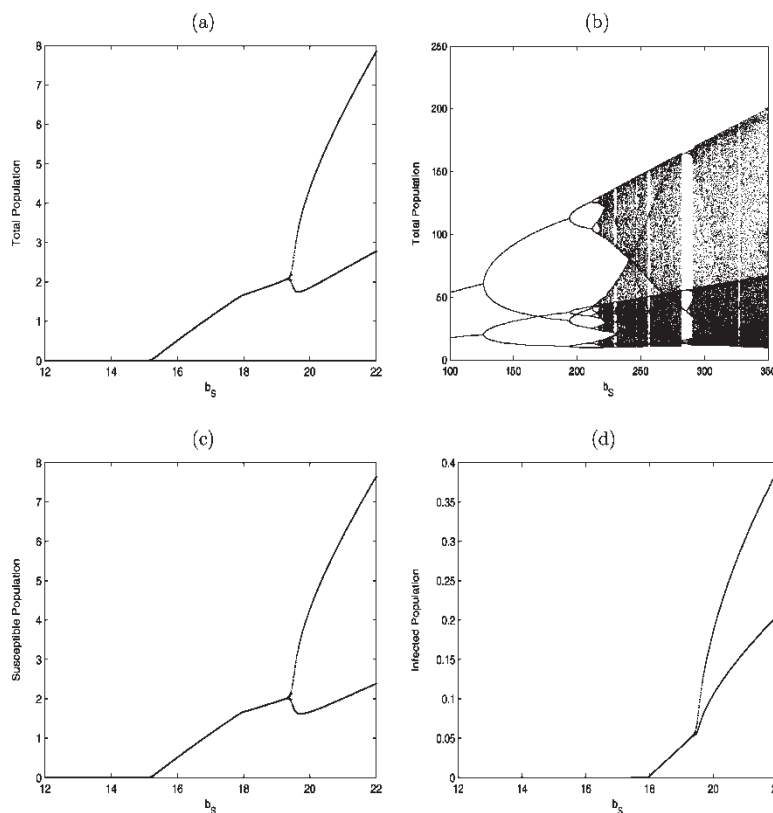


FIGURE 3 A bifurcation diagram for the *SI* juvenile and adult model (25) with the Ricker birth and survival function. (a) and (b) are bifurcation diagrams for the total population as a function of  $b_S$ . (c) and (d) are bifurcation diagrams for the susceptible and infected populations.

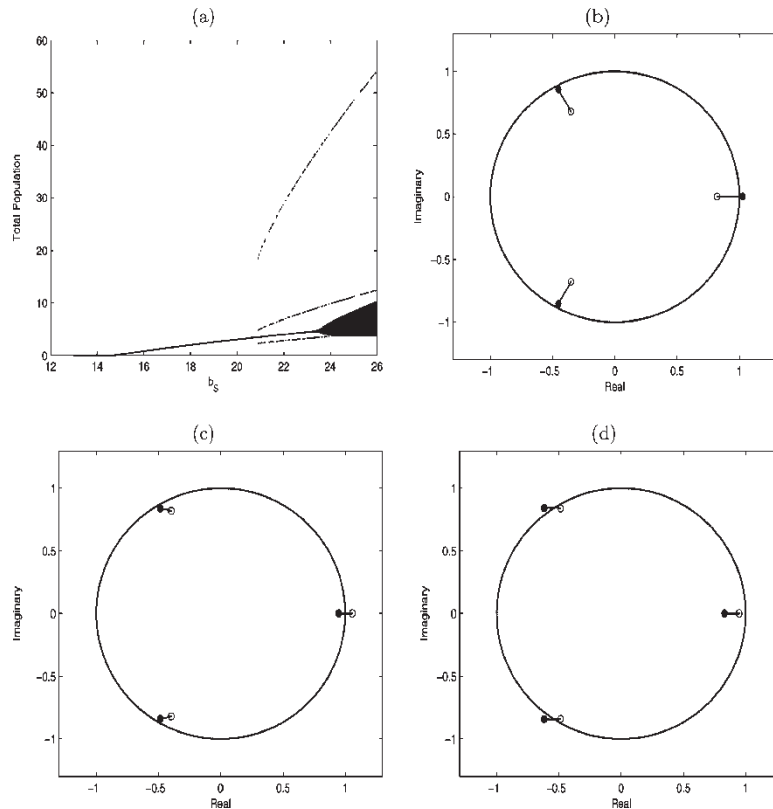


FIGURE 4 A bifurcation diagram for the susceptible larva, juvenile and adult model (34) for the Ricker birth and survival function. (a) is the bifurcation diagram for the total population. (b) is a plot of the complex eigenvalues of the Jacobian matrix evaluated at the extinction equilibrium,  $b_S \in [8, 16]$ . (c) and (d) are plots of the complex eigenvalues at the disease-free positive equilibrium. In (c),  $b_S \in [12, 18]$  and in (d),  $b_S \in [18, 30]$ . In (b)–(d), an open circle indicates the eigenvalue for the minimum value of  $b_S$  and a closed circle indicates the eigenvalue for the maximum value of  $b_S$ .

stable. When  $b_S$  is increased further, there is a bifurcation to a 2-cycle, and it is evident from Figure 3(b) that a period-doubling bifurcation occurs.

Figure 4(a) is a bifurcation diagram for the susceptible larva, juvenile and adult model (34) for  $b_S \in [12, 26]$  with a Ricker birth function. When the bifurcation diagram in Figure 4(a) is graphed for larger values of  $b_S$ , it looks similar to Figures 2(b) and 3(b). Figures 4(b)–(d) are graphs of the real eigenvalue and the two complex eigenvalues of the Jacobian matrices as a function of the bifurcation parameter  $b_S$ . In Figure 4(b), the Jacobian matrix is evaluated at the extinction equilibrium for  $b_S \in [8, 16]$ . In Figure 4(c) and (d) the Jacobian matrix is evaluated at the positive disease-free equilibrium for  $b_S \in [12, 16]$  and for  $b_S \in [18, 30]$ , respectively. The open circles in Figures 4(b)–(d) are the eigenvalues at the minimum value of  $b_S$  and the closed circles are the eigenvalues at the maximum value of  $b_S$ . In Figure 4(b), when the real eigenvalue crosses 1,  $b_S = \Psi_S = 14.744$ , a transcritical bifurcation occurs, the extinction equilibrium becomes unstable and in Figure 4(c) the disease-free positive equilibrium becomes stable. In Figure 4(d), when the pair of complex eigenvalues have magnitude one, the positive equilibrium becomes unstable and the numerical simulations indicate that two different stable solutions appear. Depending on initial conditions, a stable 3-cycle appears or a stable quasiperiodic orbit (see Fig. 5).

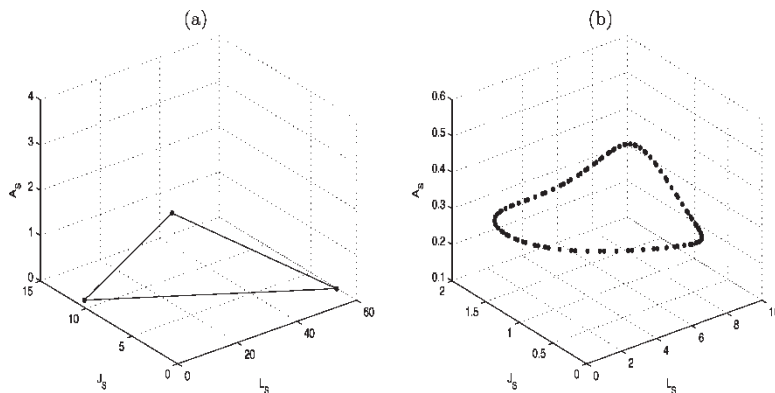


FIGURE 5 Two stable solutions for the susceptible larva, juvenile and adult model (34). The parameter  $b_S = 24$ . (a) is the graph of the stable 3-cycle with initial conditions  $L_S(0) = J_S(0) = A_S(0) = 20$ . (b) is the graph of a stable quasiperiodic orbit with initial conditions  $L_S(0) = J_S(0) = A_S(0) = 10$ .

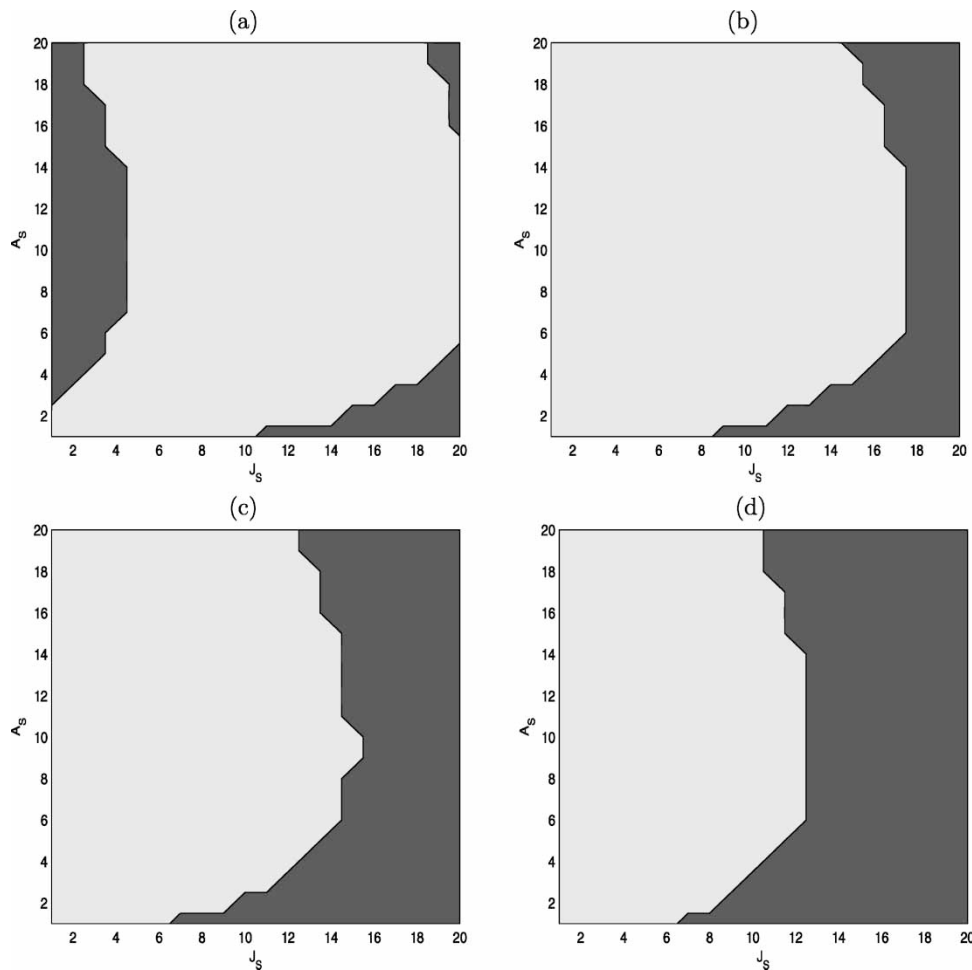


FIGURE 6 The basin of attraction in the susceptible larva, juvenile and adult model (34) for either a stable 3-cycle or a stable quasiperiodic orbit. The dark gray region indicates the initial conditions that give rise to a stable 3-cycle and the light gray region, initial conditions that give rise to a quasiperiodic orbit. The parameter  $b_S = 24$ . In (a)–(d),  $L_S(0) = 5, 10, 15$  and  $20$ , respectively.

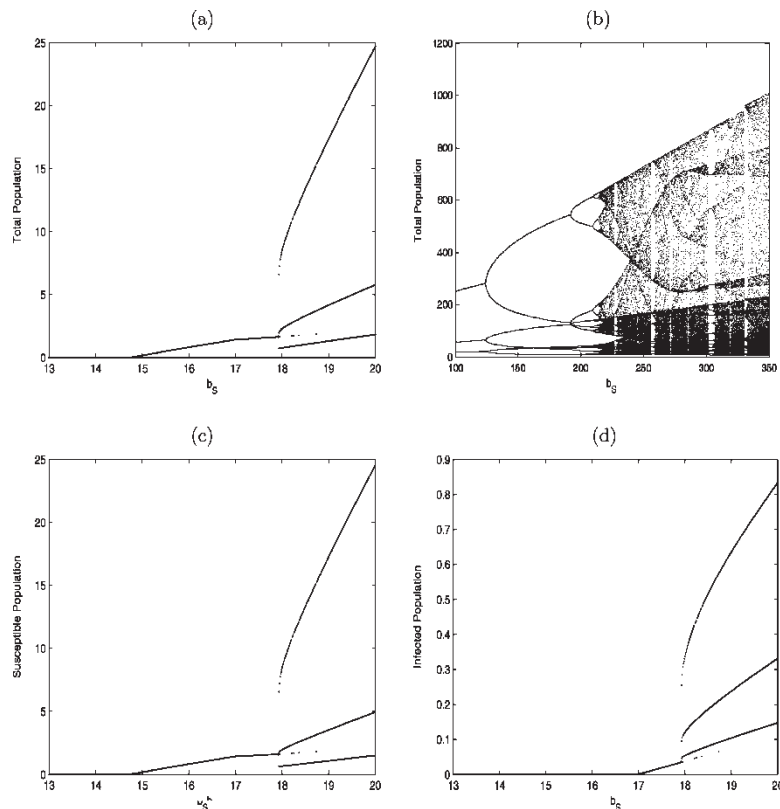


FIGURE 7 A bifurcation diagram for the SI larva, juvenile and adult model (36) for the Ricker birth and survival function. (a) and (b) are bifurcation diagrams for the total population as a function of  $b_S$ . (c) and (d) are bifurcation diagrams for susceptible and infected populations, respectively.

In Figures 6(a)–(d), we plot part of the basin of attraction for the 3-cycle and for the quasiperiodic orbit. The initial number of larvae,  $L_S(0)$ , is set equal to 5, 10, 15, and 20, in Figures 6(a)–(d), respectively. The initial number of juveniles,  $J_S(0)$ , and the initial number of adults,  $A_S(0)$ , are allowed to vary from 1 to 20. Initial conditions in the dark gray region lead to a stable 3-cycle and in the light gray region they lead to a quasiperiodic orbit.

A bifurcation diagram for the SI larvae, juvenile, and adult model (36) is graphed in Figure 7 with a Ricker birth function. Figures 7(a) and (b) are bifurcation diagrams for the total population. Figures 7(c) and (d) are bifurcation diagrams for the susceptible and infected populations, respectively. When the stable positive equilibrium becomes unstable, there is a stable 3-cycle that appears.

The dynamics for the structured epidemic models using a Beverton–Holt birth function yield similar results for values of the bifurcation parameter  $b_S < 100$ . The Beverton–Holt models did not generate the complicated dynamics that are present in the Ricker models. This might be expected because even in the simple single stage adult model the difference between these two types of birth functions is evident. In the numerical examples with a Beverton–Holt birth function, when the positive equilibrium becomes unstable, only 2- or 3-cycles are generated, even for values of  $b_S$  up to 5000.

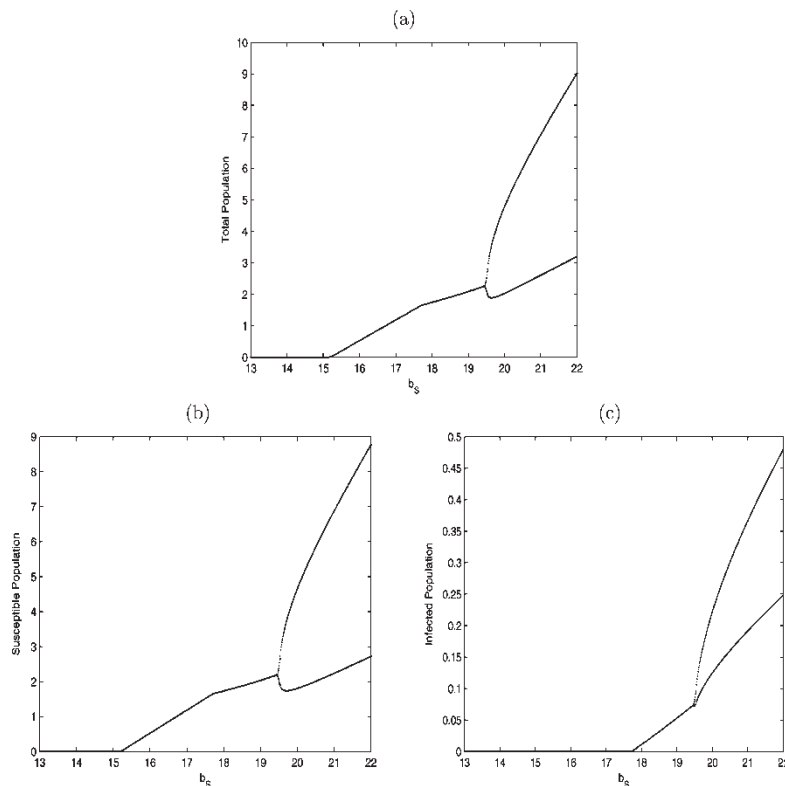


FIGURE 8 A bifurcation diagram for the SI juvenile and adult model (25) for the Beverton–Holt birth and survival function. (a) is a bifurcation diagram for the total population. (b) and (c) are bifurcation diagrams for susceptible and infected populations, respectively.

As a final example, we plot a bifurcation diagram for the *SI* juvenile and adult model (25) using a Beverton–Holt birth function. Figure 8(a)–(c) show the stable equilibria and stable 2-cycles for the total, susceptible and infected populations, respectively. Compare Figure 8 with 3.

## CONCLUSIONS AND FUTURE DIRECTIONS

We have formulated a new discrete-time, structured epidemic model for the spread of a fungal pathogen. In this paper, we assume that the parameters are constant and study the dynamics, analytically and numerically, in some simple cases. When applying the model to amphibian populations, the parameters can be functions of time. For example, the birth and survival functions should be positive only during the reproductive period which can vary considerably between different species (days–weeks–months). One interesting feature of our model is the complex dynamics driven by a high birth rate,  $b_s$ . Amphibian populations can explode after emergence of adults following spring rains and reproduction. This is a time when predation, competition and cannibalism are very high also. Because high birth rates are

not maintained throughout the year, it is not known whether such complex dynamics as predicted by the models can actually occur. We intend to investigate the dynamics in models where the birth and survival functions could be pulse functions or more complex time-dependent functions. In addition, in future work, stochastic models will be formulated and analyzed and the dynamics will be explored for parameter values associated with particular amphibian species.

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