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RESEARCH ARTICLE

Persistence in a Discrete-time, Stage-structured Fungal Disease Model

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A discrete-time SI epidemic model, with less than 100% vertical disease transmission, for the spread of a fungal disease in a structured amphibian host population, is analyzed. Criteria for persistence of the population as well as for persistence of the disease are established. Stability results for host extinction and for the disease-free equilibrium are presented. Bifurcation theory is used to establish existence of an endemic equilibrium.

Keywords: Structured populations, SI epidemic model, persistence, vertical transmission, bifurcation

AMS Subject Classification: **39A11,92D30**

1. Introduction

Amphibian populations have been declining world wide for decades [10, 14, 16] due to habitat loss and disease. According to Skerratt et al. [10], the relatively recent global emergence of the fungal disease chytridiomycosis has resulted in "the most spectacular loss of vertebrate biodiversity due to disease in recorded history". Individual frogs contact the disease either when their skin comes into contact with water that contains spores from infected animals, or by direct contact with any infected animal. After infection, the fungus invades the surface layers of the frog's skin, causing damage to the keratin layer.

A family of discrete-time models that incorporate the life-stage structure of an amphibian population, which may include larva (L), juvenile(J) and adult(A) stages, afflicted with a fungal disease were formulated by Allen and Emmert [5]. The most general models were of LJA-SI and LJA-SIR type, meaning they included all three stages and are of SI or SIR epidemic type. A notable feature of the models was the assumption of 100% efficient vertical transmission of disease from infected adults to their larval offspring. The authors also considered several sub-models characterized by simpler population stage structure (JA or even simply A). Their mathematical results establish persistence and extinction of the population, not the disease, but persistence is established in a somewhat narrow sense: the existence of a disease-free equilibrium. Numerous numerical simulations and bifurcation diagrams show that quite complicated dynamic behavior is possible in the models, especially with the Ricker recruitment nonlinearity and large intrinsic birth rates.

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Emmert and Allen extended their models to seasonal models [6], by considering the birth functions, as well as the survival probabilities within stages, and transition probabilities from one stage to another, to be periodic functions. In the same paper, they also analyze some stochastic versions of the models (discrete Markov chains branching processes). Using the 'next generation matrix approach', Allen and van den Driessche obtained explicit formulae for the basic reproductive ratio for the adult model with infection included [1].

Motivated by [5], the authors [9] applied theoretical ideas from persistence theory to the model of Allen and Emmert to establish that (1) the disease cannot lead to the extinction of the amphibian population (persistence of the host), and (2) the disease can become endemic in the population (persistence of the disease) under suitable conditions. Persistence here is used in the stronger, dynamical sense of persistence theory [13, 18]. Along with the natural condition for persistence of the disease, namely that the disease-free state is unstable to invasion of the disease, we were forced to assume that the disease-free dynamics was convergent. More precisely, we assumed that in the absence of the disease, the population settled into a globally attracting fixed point. While we could give sufficient conditions for global convergence, and although simulations suggested it occurred for a much larger parameter set, nonetheless, it is a quite restrictive hypothesis as simulations in [5] clearly demonstrate.

The paper of Thieme et al [15] examines the question of whether a disease can drive a host population to extinction. They propose a traditional ordinary differential equation with mass action incidence but with general disease-free host dynamics that may include an Allee effect. Among many interesting outcomes, one is host eradication due to the disease.

The present paper builds on our earlier work [9]. As noted above, the Emmert-Allen model assumes 100% efficient vertical transmission of the disease from infected host to their offspring. An unusual consequence of this assumption for the LJA-SI model is that, in addition to the usual disease-free invariant set, there exists a dynamical regime (invariant set) in which the host population consists entirely of infected individuals. It seems unlikely that this state could be an attractor for realistic parameter regions and therefore it probably can be ignored on biological grounds since a viable "disease-free" host population is a prerequisite for the emergence of disease. However, this feature certainly complicated the mathematics. Moreover, from a biological point of view, the hypothesis of completely faithful vertical transmission seems unlikely and from the mathematical point of view it leads to a structurally unstable system. For if the model is modified by assuming that a (very small) proportion of offspring of infected adults is born susceptible, then the "all-infectives dynamical regime" disappears. For this reason, we are motivated to modify the Emmert-Allen model as just described, and to reconsider our analysis in [9]. Furthermore, motivated by issues of mathematical tractability, we restrict attention here to the the JA-SI model, dropping consideration of the larval stage.

Our earlier results [9] continue to hold for the modified model and we are able to sharpen some of these. We establish that the disease cannot drive the host to extinction, as we did in [9], and we provide sufficient conditions for the disease to persist in the population which are completely similar to the ones in [9], requiring the restrictive hypothesis of a globally attracting steady state for the disease-free subsystem. We hope to relax this assumption in future work. However, we are able to obtain somewhat better sufficient conditions for this global convergence to hold due to the lower dimensionality of the disease-free system in the absence of a larval stage. Finally, we employ an extension of the Rabinowitz global bifurcation theorem to establish the existence of an endemic disease steady state for all values of the

disease transmission-rate exceeding the critical value at which the disease-free state looses stability. The illuminating application of the Rabinowitz bifurcation theorem to a broad class of models in population dynamics has been championed by Jim Cushing [3, 4]. We dedicate this paper to him on the occasion of his 65th birthday.

2. The SI Juvenile and Adult Model

The Emmert-Allen model [5] considers Juvenile J, and Adult A stages of the host population, labeled with subscripts S, I indicating their status as susceptible or recovered. F denotes density of fungus in the environment. We consider here the case when we have less than 100% vertical transmission. In addition, all parameters in the model are assumed to be positive, except for c_{JS}, c_{JI} (see below), which we allow be zero. The JA-SI model is:

$$\begin{cases} J_S^{n+1} = p_{JS} J_S^n e^{-\beta_J w\cdot I^n} + q_{LS} b_S \phi(T^n) A_S^n + (1-f) q_{LI} b_I \phi(T^n) A_I^n \\ A_S^{n+1} = q_{JS} J_S^n + p_{AS} A_S^n e^{-\beta_A w\cdot I^n} \\ J_I^{n+1} = p_{JS} J_S^n (1 - e^{-\beta_J w\cdot I^n}) + p_{JI} J_I^n + f q_{LI} b_I \phi(T^n) A_I^n \\ A_I^{n+1} = q_{JI} J_I^n + p_{AS} A_S^n (1 - e^{-\beta_A w\cdot I^n}) + p_{AI} A_I^n \\ F^{n+1} = b_F v_J J_I^n + b_F v_A A_I^n + p_F F^n \end{cases} \tag{1}$$

where $T = c_{JS} J_S + c_{JI} J_I + c_{AS} A_S + c_{AI} A_I$, with nonnegative c_{ij}, and the function $\phi(T) : [0, \infty) \to [0, \infty)$ strictly decreasing, and having the property that $x \mapsto x\phi(x)$ is bounded. A common choice is $\phi(x) = e^{-x}$ (Ricker), or $\phi(x) = 1/(1+x)$ (Beverton-Holt). Following [6], we give below a brief explanation of parameters used in the model, where $N = J, A$, and $K = S, I$ (see [6] for more details on this):

- p_{NK}=probability of survival within a stage.
- p_F=probability that fungi survive in the environment without a live host.
- p_N=probability of recovery from the infection.
- q_{NK}=probability of transition among stages, but remaining in the same class (susceptible, infected or immune). Ex: q_{JS} is the probability that a juvenile becomes an adult, but remains susceptible.
- $b_K \phi(T)$=number of eggs/adult that survive to larval stage.
- b_F=birth of fungal zoospores from growth on dead larvae, juveniles, or adults.

The probability of becoming infected in state N, $1 - e^{-\beta_N w\cdot I}$, increases with increasing values of infection in the population and environment, infection represented by the weighted sum of the infected classes and fungus:

$$w \cdot I = w_J J_I + w_A A_I + w_F F.$$

The following inequalities among parameters are assumed:

$$\begin{cases} p_{AS}, p_{AI}, q_{LS}, q_{LI}, q_{JS}, q_{JI}, f < 1 \\ p_{JS} + q_{JS} < 1 \\ p_{JI} + q_{JI} < 1 \end{cases} \tag{2}$$

Let

$$\Delta_S := \frac{q_{LS} q_{JS} b_S}{(1 - p_{JS})(1 - p_{AS})}, \quad \Delta_I := \frac{f q_{LI} q_{JI} b_I}{(1 - p_{JI})(1 - p_{AI})}. \tag{3}$$

Also, we denote (J_S, A_S, J_I, A_I, F) in short, by $\mathbf{x}$. In our next result we analyze the "boundedness" of (1), as well as the existence of fixed points is the boundary invariant sets.

Proposition 2.1: *The following hold:*

a) If $c_{AK} > 0$, $K = S, I$ then (1) has a global attractor of bounded sets. In particular, there is a bounded set that attracts all orbits.
b) $\mathfrak{B}_{IF} := \{\mathbf{x} \in \mathrm{R}^5_+ | J_I = A_I = F = 0\}$ and the $F-axis$ are positively invariant sets for (1).
c) There exists a unique non-trivial boundary fixed point E_S of (1) if and only if $\Delta_S > 1$. When it exists, $E_S = (\bar{J}_S, \bar{A}_S, 0, 0, 0)$, with

$$(\bar{J}_S, \bar{A}_S) = \frac{\phi^{-1}(\frac{1}{\Delta_S})}{c_{JS}(1-p_{AS}) + c_{AS}q_{JS}} (1 - p_{AS}, q_{JS}) . \tag{4}$$

$\mathfrak{B}_{IF}$ represents no disease states, and the $F - axis$ represents no hosts states. Notice that $\mathbf{0}$ is always a fixed point of (1).

2.1. *Disease-free Dynamics*

When infection is not present, the model takes the form

$$\begin{cases} J_S^{n+1} = p_{JS}J_S^n + q_{LS}b_S\phi(T^n)A_S^n \\ A_S^{n+1} = q_{JS}J_S^n + p_{AS}A_S^n \end{cases} \tag{5}$$

where $T = c_{JS}J_S + c_{AS}A_S$.

By Proposition 2.1 it is clear that $(\bar{J}_S, \bar{A}_S)$ is the unique non-zero fixed point of (5).

Parameter Δ_S distinguishes between extinction and survival of the host:

Theorem 2.2: *The following hold:*

a) If $\Delta_S < 1$ then $\boldsymbol{0}$ is a globally asymptotically stable fixed point.
b) If $\Delta_S > 1$ then there exists $\varepsilon > 0$ such that

$$\liminf_{n\to\infty} \min\{J_S^n, A_S^n\} > \varepsilon, \forall\, \mathbf{x}^0 = (J_S^0, A_S^0) \in \mathrm{R}^2_+ \setminus \{\boldsymbol{0}\}.$$

Part *a*) can be found in [5].

In the following theorem we give various sufficient conditions for global stability of E_S.

Theorem 2.3: *Assume $\Delta_S > 1$ and let $\Gamma = \frac{c_{JS}q_{LS}b_S}{c_{AS}p_{JS}}$. In any of the following cases:*

a) $\phi(x) = 1/(1+x)$ and $\Gamma \le \begin{cases} \frac{\Delta_S^2}{\Delta_S - 1}, \text{ if } \Delta_S \le 2 \\ 4, \text{ if } \Delta_S > 2 \end{cases}$,
b) $\phi(x) = e^{-x}$, $\Delta_S \le e$ and $\Gamma \le \frac{\Delta_S}{\ln(\Delta_S)}$,
c) $\phi(x) = e^{-x}$, $\Gamma \le e$ and $\Delta_S \le e^{1 + c_{JS}(1-p_{JS})/(c_{AS}q_{JS})}$,

$(\bar{J}_S, \bar{A}_S)$ is asymptotically stable and attracts all solutions starting in $\mathrm{R}^2_+ \setminus \{\boldsymbol{0}\}$.

Numerical simulations suggest that the conclusion of Theorem 2.3 holds for a much larger set of parameters than required by our hypotheses. However, sim-

ulations in [6] suggest that the dynamics exhibits a period-doubling cascade as parameter b_S is increased.

2.2. *Main results*

We begin by determining the stability of the boundary fixed points of (1). Then we establish sufficient conditions for persistence of the host and for the disease, and finally bifurcation of a positive, endemic equilibrium from E_S.

The Jacobian of (1) at $\mathbf{0}$ is:

$$J(\mathbf{0}) = \begin{pmatrix} p_{JS} & q_{LS}b_S & 0 & (1-f)q_{LI}b_I & 0 \\ q_{JS} & p_{AS} & 0 & 0 & 0 \\ 0 & 0 & p_{JI} & fq_{LI}b_I & 0 \\ 0 & 0 & q_{JI} & p_{AI} & 0 \\ 0 & 0 & b_F v_J & b_F v_A & p_F \end{pmatrix}. \tag{6}$$

Clearly, its spectral radius, $r(J(\mathbf{0}))$ equals $\max\{r(A^S), r(A^I), p_F\}$, where

$$A^S = \begin{pmatrix} p_{JS} & q_{LS}b_S \\ q_{JS} & p_{AS} \end{pmatrix}, \quad A^I = \begin{pmatrix} p_{JI} & fq_{LI}b_I \\ q_{JI} & p_{AI} \end{pmatrix}. \tag{7}$$

The relationship between Δ_K (see (3)) and $r(A^K)$, $K = S, I$ is given in the following:

Lemma 2.4: $(1-\Delta_K)(1-r(A^K)) \geq 0$, *with equality if and only if* $\Delta_K = 1$.

One can view the Δ_K as basic reproductive numbers following [1].

The following matrix, closely related to $J(\mathbf{0})$,

$$B = \begin{pmatrix} p_{JS} & q_{LS}b_S & 0 & (1-f)q_{LI}b_I & 0 \\ q_{JS} & p_{AS} & 0 & 0 & 0 \\ p_{JS} & 0 & p_{JI} & fq_{LI}b_I & 0 \\ 0 & p_{AS} & q_{JI} & p_{AI} & 0 \\ 0 & 0 & b_F v_J & b_F v_A & p_F \end{pmatrix}.$$

is useful for global stability of $\mathbf{0}$.

The 5×5 Jacobian matrix $J(E_S)$ evaluated at E_S decomposes as

$$J(E_S) = \begin{pmatrix} C & D \\ 0 & J_S^{IF} \end{pmatrix},$$

where C determines the stability of E_S as a fixed point of the disease-free system and

$$J_S^{IF} = \begin{pmatrix} p_{JI} + p_{JS}\beta_J w_J \bar{J}_S & p_{JS}\beta_J w_A \bar{J}_S + fq_{LI}b_I\phi(\bar{T}_S) & p_{JS}\beta_J w_F \bar{J}_S \\ q_{JI} + p_{AS}\beta_A w_J \bar{A}_S & p_{AI} + p_{AS}\beta_A w_A \bar{A}_S & p_{AS}\beta_A w_F \bar{A}_S \\ b_F v_J & b_F v_A & p_F \end{pmatrix} \tag{8}$$

pertains to the ability of the disease to invade the disease-free state. We denoted T evaluated at E_S by $\bar{T}_S$.

Below we give a result regarding the stability of the boundary fixed points.

Theorem 2.5: *The following hold:*

a) $\mathbf{0}$ *is asymptotically stable if* $\Delta_S < 1, \Delta_I < 1$ *and unstable if either* $\Delta_S > 1$ *or* $\Delta_I > 1$. *If* $r(B) < 1$ *then* $\mathbf{0}$ *is globally asymptotically stable.*
b) If E_S *is asymptotically stable in* $\mathfrak{B}_{IF}$ *and* $r(J_S^{IF}) < 1$ *then* E_S *is asymptotically stable in* R^5_+.

Now we give sufficient conditions for persistence of the host and for persistence of both the host and the disease.

Theorem 2.6 : *Persistence and Positive Fixed Points for (1):*

(I) If $\Delta_S > 1$ *then there exists* $\varepsilon > 0$ *such that*

$$\liminf_{n\to\infty} \min\{J_S^n, A_S^n\} > \varepsilon, \ \forall\, \mathbf{x}^0 \in \mathrm{R}^5_+ \setminus F - axis.$$

(II) If one of the following holds:
a) $\Delta_S > 1$, $r(J_S^{IF}) > 1$, *and the conclusion of Theorem 2.3 holds.*
b) $\Delta_S < 1$ *and* $\Delta_I > 1$.
then there exists $\varepsilon > 0$ *such that*

$$\liminf_{n\to\infty} \min_{1\le i\le 5} x_i^n > \varepsilon, \ \forall\, \mathbf{x}^0 \in \mathrm{R}^5_+ \setminus (\mathfrak{B}_{IF} \cup F - axis),$$

and (1) has a fixed point in $(R^5_+)^0 = \{\mathbf{x} \in \mathrm{R}^5_+ | x_1, ..., x_5 > 0\}$.

Under the hypotheses of Theorem 2.6 (II) there exists a positive "endemic fixed point", which may or may not be stable. The first two hypotheses in (II)(a) are natural, E_S should exist and be unstable, but the last hypothesis (the conclusion of Theorem 2.3 holds) is too strong; hypotheses (II)(b) are biologically implausible as $\Delta_I < \Delta_S$ is expected.

An alternative approach to obtain a positive fixed point is to use bifurcation theory but then we must select a "bifurcation parameter". If we fix disease-free parameters $(p_{JS}, p_{AS}, q_{LS}, q_{JS}, b_S)$ then E_S is fixed and we may vary disease-associated parameters $(p_{JI}, p_{AI}, p_F, q_{LI}, q_{JI}, b_I, f, w_K, v_K, \beta_K;\ K = J, A)$. The matrix J_S^{IF} is monotone increasing in all these parameters so by Perron-Frobenius Theory, its principle eigenvalue is monotone increasing in these parameters. Roughly speaking, stability of E_S is reduced by the increase of any of these. We select the disease transmission rates $\beta = \beta_J = \beta_A$ for special attention in our next result but this choice is somewhat arbitrary. Other natural choices include the vertical transmission efficiency f, disease-related reduction in maximal reproduction rate $b_I = b_S - \mu$, disease-related increase in adult death $p_{AI} = p_{AS} - \mu$, disease-related decrease in juvenile maturation $q_{JI} = q_{JS} - \mu$, or infection weighting factors w_K, v_K.

Theorem 2.7 : *Let* $\beta_J = \beta_A = \beta > 0$ *and assume that* E_S *is a non-degenerate fixed point of the disease-free system and that* $\Delta_I < \Delta_S$. *Then* $r(\beta) = r(J_S^{IF}(\beta))$ *is continuous and strictly increasing on* $[0, \infty)$ *with* $r(0) < 1$ *and* $r(\infty) = \infty$. *There exists a unique* $\beta_0 > 0$ *such that* $r(\beta_0) = 1$, E_S *is unstable for* $\beta > \beta_0$ *and for each such* β *there exists at least one positive fixed point in* $(\mathrm{R}^5_+)^0$.

As our calculations indicate, for the values of parameters given in Table 1 (c_{NK}, $N = J, A$; $K = S, I$, not given in the table, are all equal to 0.1), the bifurcation for the positive fixed point occurs at a value of β in the interval $(1.16, 1.17)$ (see also Figure 1). By increasing the value of β (in a certain neighborhood of the bifurcation value), the positive fixed point increases in the I components and decreases in the S components, as it can be observed in Figure 2. Our last result

Table 1. Parameters used in the numerical simulations

Susceptible	Infected	Contact	Fungus
$p_{JS} = 0.04$	$p_{JI} = 0.03$	$w_J = 0.4$	$p_F = 0.5$
$p_{AS} = 0.05$	$p_{AI} = 0.04$	$w_A = 0.4$	$v_L = 1$
$q_{LS} = 0.2$	$q_{LI} = 0.1$	$w_F = 0.4$	$v_J = 1$
$q_{JS} = 0.3$	$q_{LI} = 0.1$		$v_A = 1$
$b_S = 18$	$b_I = 9$		$b_F = 10$
	f=0.9		

Figure 1. Bifurcation of a positive fixed point from the disease-free state.

Figure 2. Four orbits with the same initial condition, converging to the disease-free fixed point ($\beta = 1$ and $\beta = 1.1$) and to a positive fixed point ($\beta = 1.2$ and $\beta = 1.3$).

shows that the population decreases at fixed point from E_S level to the endemic fixed point:

Proposition 2.8: *Assume that*

$$p_{JI} \le p_{JS},\ p_{AI} \le p_{AS},\ q_{JI} \le q_{JS},\ q_{LI} b_I \le q_{LS} b_S$$

If $\mathbf{x} = (J_S, A_S, J_I, A_I, F) \in (\mathrm{R}^5_+)^0$ *is a fixed point, then* $T_{\mathbf{x}} \le \bar{T}_S$, *where* $T_{\mathbf{x}}$ *is* T *evaluated at* $\mathbf{x}$, *and* $\bar{T}_S$ *is* T *evaluated at* E_S. *If, in addition,*

$$c_{JS} = c_{JI} = c_{AS} = c_{AI}$$

then $J_S + J_I + A_S + A_I \le \bar{J}_S + \bar{A}_S$.

3. Discussion

As noted in the introduction, the Emmert-Allen model [5] assumes 100% efficient vertical transmission of the disease from infected host to their offspring. Because this seems unlikely to be satisfied we have introduced a vertical transmission efficiency parameter $f \in [0, 1]$ in the JA-SI model of [5]. When $f = 1$ we recover the original model while for $f < 1$ the fraction $1 - f$ of off-spring of infected host begin life disease-free and susceptible.

Our previous work [9] provides sufficient conditions for persistence of the host and for persistence of the disease for both the LJA-SI and LJA-SIR model formulated by Emmert and Allen [5]. These same results carry over to the JA-SI model, modified to include the vertical transmission efficiency parameter, considered here. Specifically, the disease cannot drive the host population to extinction (Theorem 1 (I)) and when the disease can invade the disease-free fixed point then the disease persists provided the disease-free state attracts all non-trivial initial data for the disease-free system (Theorem 1 (IIa)).

By neglecting the larval stage of the full model, which we do here, we are able to obtain somewhat better sufficient conditions for this global convergence to hold in Theorem 2.3 than in the corresponding result in [9]. Strictly speaking the results are not comparable but here we are not forced to assume that one of the c_{JK} vanishes.

Our result guaranteeing persistence of the disease also implies the existence of a positive fixed point-the endemic equilibrium. An alternative approach to showing the existence of this equilibrium is to use bifurcation theory which avoids any assumptions on the disease-free dynamics. We used an extension of the Rabinowitz global bifurcation theorem to establish the existence of an endemic disease steady state for all values of the disease transmission-rate exceeding the critical value at which the disease-free state loses stability. Furthermore, we show analytically and numerically that the host population at the endemic fixed point is less than the host population at the disease-free fixed point. Our numerical calculations show that this reduction can be substantial. The large reduction in host population due to the disease would leave the host vulnerable to extinction in a model that includes demographic stochasticity.

4. Proofs

We focus here on the proofs of Theorem 2.3 and Theorem 2.7 since the others are nearly identical to ones in [9]. Specifically, the proofs of Proposition 2.1, Lemma 2.4, Theorem 2.2, Theorem 2.5, and Theorem 2.6 are similar to Proposition 2.1, Lemma 3.1, Theorem 3.3, Theorem 4.3, Theorem 4.4, respectively, in [9].
We denote by A^T the transpose of matrix A. O is the zero matrix. We call the matrix $A = (a_{ij})_{1\leq i,j\leq m} \in \mathrm{R}^m \times \mathrm{R}^m$

positive, and write $A > O$, if $a_{ij} \geq 0$, $\forall\, i, j \in \{1, ..., m\}$, and $A \neq O$;
strictly positive, and write $A \gg O$, if $a_{i,j} > 0$, $\forall\, i, j \in \{1, ..., m\}$;
non-negative, and write, $A \geq O$, if $a_{i,j} \geq 0$, $\forall\, i, j \in \{1, ..., m\}$.

We define the partial order relation on the set of non-negative matrices as follows: $A \leq B \Leftrightarrow B - A \geq O$. Assume analogous definitions for " $<$ " and " $\ll$ ". Also, we assume the same notation for vectors in R^m. For a differentiable function $f : \mathrm{R}^m \to \mathrm{R}^m$, we denote the derivative of f at $\mathbf{x}$ by $Df(\mathbf{x})$.

Proof: (**of Theorem (2.3)**) Let $\mathbf{x} = (J_S, A_S)^T$ and denote the right hand side of (5) by $Q(\mathbf{x})$. Then

$$DQ(\mathbf{x}) = \begin{pmatrix} a_{11}(\mathbf{x}) & a_{12}(\mathbf{x}) \\ q_{JS} & p_{AS} \end{pmatrix},$$

where $a_{11}(\mathbf{x}) = p_{JS} + c_{JS}q_{LS}b_S\phi'(T)A_S$ and $a_{12}(\mathbf{x}) = q_{LS}b_S(\phi(T) + c_{AS}\phi'(T)A_S)$. Abusing notation, denote $(\bar{J}_S, \bar{A}_S)$ also by E_S. First we show that $DQ(E_S) > \mathbf{0} \Rightarrow E_S$ is asymptotically stable. So assume $DQ(E_S) > \mathbf{0}$. Let

$$A^S(\mathbf{x}) = \begin{pmatrix} p_{JS} & q_{LS}b_S\phi(T) \\ q_{JS} & p_{AS} \end{pmatrix}. \tag{9}$$

Then we have that

$$\mathbf{0} < DQ(E_S) < A^S(E_S),$$

and since both matrices are positive and irreducible, we obtain that

$$r(DQ(E_S)) < r(A^S(E_S)) \qquad (\mathbf{i}).$$

E_S being a positive fixed point of Q implies $(E_S)^T = A^S(E_S)(E_S)^T$, so 1 is an eigenvalue of $A^S(E_S)$. Because $A^S(E_S)$ is positive and irreducible, $r(A^S(E_S)) = 1$, by the Perron-Frobenius theory. Then, using (**i**), we obtain that E_S is asymptotically stable.

We now prove a) and b) simultaneously. First we show that any solution is attracted to the set $[\mathbf{0}, \mathbf{w}] = \{\mathbf{x} \in \mathrm{R}_+^2 | \mathbf{x} \leq \mathbf{w}\}$, where $\mathbf{w} = (\frac{1-p_{AS}}{c_{AS}q_{JS}}\phi^{-1}(\frac{1}{\Delta_S}), \frac{1}{c_{AS}}\phi^{-1}(\frac{1}{\Delta_S}))^T$. Let $\mathbf{y}^0 > \mathbf{0}$, and $\mathbf{x}^0 = \mathbf{y}^0$. We have that

$$\mathbf{x}^{n+1} \leq M^S\mathbf{x}^n + \mathbf{a}^1, \forall\, n \geq 0 \qquad (\mathbf{ii})$$

where $M^S = \begin{pmatrix} p_{JS} & 0 \\ q_{JS} & p_{AS} \end{pmatrix}$, $\mathbf{a}^1 = (\frac{q_{LS}b_S}{c_{AS}}M_1, 0)^T$, $M_1 = \sup_x x\phi(x)$. Then (see proof of Proposition 2.1 in [9]) $\omega(\mathbf{y}^0) \subseteq [\mathbf{0}, \mathbf{w}^1]$ where $\mathbf{w}^1 = (I - M^S)^{-1}\mathbf{a}^1 = (\frac{1-p_{AS}}{c_{AS}q_{JS}}M_1\Delta_S, \frac{1}{c_{AS}}M_1\Delta_S)^T$. Let $\mathbf{y}^1 \in \omega(\mathbf{y}^0)$. Then $\omega(\mathbf{y}^1) \subseteq \omega(\mathbf{y}^0) \subseteq [\mathbf{0}, \mathbf{w}^1]$. Again, consider the solution to (5) starting at $\mathbf{x}^0 = \mathbf{y}^1$. Then

$$\mathbf{x}^{n+1} \leq M^S\mathbf{x}^n + \mathbf{a}^2,$$

where $\mathbf{a}^2 = (M_2\frac{q_{LS}b_S}{c_{AS}}, 0)^T$, with $M_2 = \max_{x\in[0,M_1\Delta_S]} x\phi(x)$. Similarly, we get that $\omega(\mathbf{y}^1) \subseteq [\mathbf{0}, \mathbf{w}^2]$, where $\mathbf{w}^2 = (I - M^S)^{-1}\mathbf{a}^2 = (\frac{1-p_{AS}}{c_{AS}q_{JS}}M_2\Delta_S, \frac{1}{c_{AS}}M_2\Delta_S)^T$. So $\omega(\mathbf{y}^0) \cap [\mathbf{0}, \mathbf{w}^2] \neq \emptyset$. Continuing inductively, we obtain that $\omega(\mathbf{y}^0) \cap [\mathbf{0}, \mathbf{w}^n] \neq \emptyset$, $\forall\, n \geq 0$, where $\mathbf{w}^n = (\frac{1-p_{AS}}{c_{AS}q_{JS}}M_n\Delta_S, \frac{1}{c_{AS}}M_n\Delta_S)^T$, with $M_n = \max_{x\in[0,M_{n-1}\Delta_S]} x\phi(x)$. It is clear that, for both choices of ϕ, $M_2 \leq M_1$. Suppose $M_i \leq M_{i-1}$. Then $M_{i+1} = \max_{x\in[0,M_i\Delta_S]} x\phi(x) \leq \max_{x\in[0,M_{i-1}\Delta_S]} x\phi(x) = M_i$. Thus, by induction, we have $M_{n+1} \leq M_n$, $\forall\, n \geq 1$, which implies that $(M_n)_n$ is a convergent sequence. Hence $(\mathbf{w}^n)_n$ is convergent, and let $\mathbf{w}$ be its limit. In the Beverton-Holt case, $x \mapsto x\phi(x)$ is increasing on R_+, while in the Ricker case, $x \mapsto x\phi(x)$ is increasing on $[0, 1]$, and in this latter case we have $M_n\Delta_S \leq M_1\Delta_S = \Delta_S/e \leq 1$, $\forall\, n \geq 1$. So, in any case, $M_n = \max_{x\in[0,M_{n-1}\Delta_S]} x\phi(x) = M_{n-1}\Delta_S\phi(M_{n-1}\Delta_S) = c_{AS}\mathbf{w}_2^{n-1}\phi(c_{AS}\mathbf{w}_2^{n-1})$. Thus,

$$\mathbf{w}^n = (\frac{1-p_{AS}}{c_{AS}q_{JS}}\Delta_S c_{AS}\mathbf{w}_2^{n-1}\phi(c_{AS}\mathbf{w}_2^{n-1}), \frac{1}{c_{AS}}\Delta_S c_{AS}\mathbf{w}_2^{n-1}\phi(c_{AS}\mathbf{w}_2^{n-1}))^T.$$

Letting n go to infinity we get

$$\mathbf{w} = (\frac{1-p_{AS}}{c_{AS}q_{JS}}\Delta_S c_{AS}\mathbf{w}_2\phi(c_{AS}\mathbf{w}_2), \frac{1}{c_{AS}}\Delta_S c_{AS}\mathbf{w}_2\phi(c_{AS}\mathbf{w}_2))^T.$$

Solving the above equation, we obtain $\mathbf{w} = (\frac{1-p_{AS}}{c_{AS}q_{JS}}\phi^{-1}(\frac{1}{\Delta_S}), \frac{1}{c_{AS}}\phi^{-1}(\frac{1}{\Delta_S}))^T$.
Hence $\omega(\mathbf{y}^0) \cap [\mathbf{0}, \mathbf{w}] \neq \emptyset$. Since $\mathbf{y}^0 > \mathbf{0}$ was arbitrarily chosen, we have that $E_S \in [\mathbf{0}, \mathbf{w}]$. From Theorem 2.2 part *b*) we have that $\mathbf{0} \notin \omega(\mathbf{y}^0)$. Thus, if we show that any solution starting in $[\mathbf{0}, \mathbf{w}] \setminus \{\mathbf{0}\}$ converges to E_S, we are done, because then, using the fact that $\omega(\mathbf{y}^0)$ is compact and invariant, we would have $E_S \in \omega(\mathbf{y}^0) \Rightarrow \omega(\mathbf{y}^0) = \{E_S\}$ (we used that E_S is asymptotically stable). For this, we first show that the system is monotone in $[\mathbf{0}, \mathbf{w}]$. Let $\mathbf{x} \in [\mathbf{0}, \mathbf{w}]$. We discuss the two cases:
The Beverton-Holt case, i.e. $\phi(x) = 1/(1 + x)$. We have $a_{11}(\mathbf{x}) \geq p_{JS} - \frac{c_{JS}q_{LS}b_S}{c_{AS}}\frac{c_{AS}A_S}{(1+c_{AS}A_S)^2}$. For any $\mathbf{x} \in [\mathbf{0}, \mathbf{w}]$ we have $c_{AS}A_S \leq \phi^{-1}(\frac{1}{\Delta_S}) = (\Delta_S - 1)$. The function $x \mapsto x/(1+x)^2$ is increasing on $[0, 1]$ and decreasing on $[1, \infty)$. Thus,

if $\Delta_S \leq 2$, the maximum of $\frac{c_{AS}A_S}{(1+c_{AS}A_S)^2}$ is $\frac{\Delta_S-1}{\Delta_S^2}$, otherwise it is $1/4$. In any case we have $a_{11}(\mathbf{x}) \geq 0$. It is trivial to check that $a_{12}(\mathbf{x}) \geq 0$.
The Ricker case, i.e. $\phi(x) = e^{-x}$. Notice that $c_{AS}A_S \leq \phi^{-1}(\frac{1}{\Delta_S}) = \ln(\Delta_S) \leq 1$. So the maximum of $e^{-c_{AS}A_S}c_{AS}A_S$ is $\frac{\ln(\Delta_S)}{\Delta_S}$, because $x \mapsto xe^{-x}$ is increasing on $[0,1]$.

$$a_{11}(\mathbf{x}) = p_{JS} - c_{JS}q_{LS}b_S e^{-T}A_S \geq p_{JS} - \frac{c_{JS}}{c_{AS}}q_{LS}b_S e^{-c_{AS}A_S}c_{AS}A_S \geq$$
$$\geq p_{JS} - \frac{c_{JS}q_{LS}b_S}{c_{AS}}\frac{\ln(\Delta_S)}{\Delta_S} \geq 0 \Leftrightarrow \Gamma \leq \frac{\Delta_S}{\ln(\Delta_S)}\,.$$

Also we have $a_{12}(\mathbf{x}) = q_{LS}b_S e^{-T}(1 - c_{AS}A_S) \geq 0 \Leftrightarrow A_S \leq 1/c_{AS}$ $(\star)$. Thus, the system is monotone in $[\mathbf{0}, \mathbf{w}]$ (see [11]).
It can be easily checked that $Q(\mathbf{w}) \leq \mathbf{w} \Leftrightarrow \phi(c_{JS}w_1 + c_{AS}w_2) \leq \phi(c_{JS}\bar{J}_S + c_{AS}\bar{A}_S)$, which holds because $E_S \leq \mathbf{w}$. Let $\mathbf{x}^0 \in [\mathbf{0}, \mathbf{w}]\backslash\{\mathbf{0}\}$. Since $\mathbf{x}^n \gg \mathbf{0}, \forall n \geq 1$, without a loss of generality we can assume that $\mathbf{x}^0 \gg \mathbf{0}$. Now we want to show that we can find $\mathbf{u} \gg 0$ such that $\mathbf{u} \leq \mathbf{x^0}$ and $Q(\mathbf{u}) > \mathbf{u}$. $DQ(\mathbf{0}) = A^S$, hence $\lambda := r(DQ(\mathbf{0})) > 1$ (from lemma (2.4)).
$DQ(\mathbf{0})$ being irreducible, it has an eigenvector $\tilde{\mathbf{u}} \gg 0$, corresponding to λ, and let $\mathbf{u} = s\tilde{\mathbf{u}}$, $s > 0$. Using Taylor expansion and the fact that $\mathbf{0}$ is a fixed point of Q we get

$$Q_j(\mathbf{u}) = \sum_{i=1}^{2} \frac{\partial Q_j}{\partial x_i}(\mathbf{c}_j)u_i \qquad j = 1,2 \qquad (\mathbf{iii}),$$

where $\mathbf{c}_j{}'s$ are points on the line segments joining $\mathbf{0}$ and $\mathbf{u}$. Let $\varepsilon > 0$ such that

$$(\lambda - 1)u_j - \varepsilon|\mathbf{u}| > 0;\ j = 1,2 \qquad (\mathbf{iv}).$$

Such an ε exists, because $\lambda > 1$. Q being a $\mathcal{C}^1$ map, DQ is continuous, so we can choose s sufficiently small (hence $\mathbf{u}$ and $\mathbf{c}_j,\ j = 1,2,3$ are small) such that to have both

$$\frac{\partial Q_j}{\partial x_i}(\mathbf{c}_j) \geq \frac{\partial Q_j}{\partial x_i}(\mathbf{0}) - \varepsilon,\ i,j = 1,2$$

and $\mathbf{u} \leq \mathbf{x^0}$. Thus, using$(\mathbf{iii})$ we have $Q(\mathbf{u}) \geq DQ(\mathbf{0})\mathbf{u} - \Upsilon\mathbf{u} > \mathbf{u} \Leftrightarrow \lambda\mathbf{u} - \Upsilon\mathbf{u} > \mathbf{u} \Leftrightarrow$ $(\mathbf{iv})$ holds, where we denoted by Υ the matrix having each element equal to ε.
So we have $\mathbf{u} \leq \mathbf{x}^0 \leq \mathbf{w}$. But then because (5) is monotone, we have that

$$\mathbf{u}^n \leq \mathbf{x}^n \leq \mathbf{w}^n,\ \forall\, n \geq 0 \qquad (\mathbf{v}),$$

where $\mathbf{u}^0 = \mathbf{u}$ and $\mathbf{w}^0 = \mathbf{w}$. Also, $Q(\mathbf{u}) > \mathbf{u} \Rightarrow (\mathbf{u}^n)_n$ is an increasing sequence in R^2_+, and $Q(\mathbf{w}) \leq \mathbf{w} \Rightarrow (\mathbf{w}^n)_n$ is a decreasing sequence in R^2_+. Hence they converge (again, see [11]), and by the continuity of Q, the limit of each sequence must be fixed point of Q, which cannot be other that E_S. Then $(\mathbf{v})$ implies that $\mathbf{x}^n \to E_S$.

c) As above, we have $a_{11}(\mathbf{x}) \geq p_{JS} - \frac{c_{JS}}{c_{AS}}q_{LS}b_S e^{-c_{AS}A_S}c_{AS}A_S \geq p_{JS} - \frac{c_{JS}q_{LS}b_S}{ec_{AS}} \geq 0 \Leftrightarrow \Gamma \leq e$, and using $(\star)$ in b) we conclude that the system is monotone in $[0,\infty) \times [0, 1/c_{AS}]$. Let $g(x) = e^x x$. The curves

$$l_S:\ A_S = \frac{q_{JS}}{1 - p_{AS}}J_S,\ \text{and}\ l_J:\ J_S = \frac{1}{c_{JS}}g^{-1}\left(\frac{c_{JS}q_{LS}b_S}{c_{AS}(1-p_{JS})}e^{-c_{AS}A_S}c_{AS}A_S\right) \quad (10)$$

Figure 3. Dynamics of the disease-free model with Ricker-type nonlinearity, i.e. $\phi(x) = e^{-x}$.

represent the S and J nullclines, respectively. $\Delta_S > 1$ implies $l_S \cap l_J = \{E_S\}$, and $\Delta_S \leq e^{1+c_{JS}(1-p_{JS})/(c_{AS}q_{JS})}$ is equivalent to $\bar{A}_S \leq 1/c_{AS}$, which guarantees that E_S is situated inside the region of monotonicity. Notice that g is increasing on R_+, hence g^{-1} is increasing. So the point on l_J having the largest J_S coordinate is $(\hat{J}_S, 1/c_{AS})$, where

$$\hat{J}_S = \frac{1}{c_{JS}} g^{-1}\left(\frac{c_{JS}q_{LS}b_S}{ec_{AS}(1-p_{JS})}\right).$$

For the points on l_J we have that when $A_S = 0$ then $J_S = \frac{1}{c_{JS}}g^{-1}(0) = 0$, and when $A_S \to \infty$ then $J_S \to 0$. The map $A_S \mapsto e^{-c_{AS}A_S}c_{AS}A_S$ is increasing for $A_S \in [0, 1/c_{AS}]$, and decreasing when $A_S > 1/c_{AS}$. Hence J_S is increasing, as a function of A_S (see (10)), for $A_S \in [0, 1/c_{AS}]$, and decreasing, for $A_S > 1/c_{AS}$. Next, we want to determine the way the points are mapped, depending on their position relative to the nullclines l_S and l_J. If (J_S^n, A_S^n) is "above" l_S, i.e. $A_S^n > \frac{q_{JS}}{1-p_{AS}}J_S^n$ then $A_S^{n+1} < q_{JS}\frac{1-p_{AS}}{q_{JS}}A_S^n + p_{AS}A_S^n = A_S^n$. Similarly, if (J_S^n, A_S^n) is "below" l_S, then $A_S^{n+1} > A_S^n$.
If (J_S^n, A_S^n) is such that $J_S^n < \frac{1}{c_{JS}}g^{-1}(\frac{c_{JS}q_{LS}b_S}{c_{AS}(1-p_{JS})}e^{-c_{AS}A_S^n}c_{AS}A_S^n)$ then $g(c_{JS}J_S^n) < \frac{c_{JS}q_{LS}b_S}{c_{AS}(1-p_{JS})}e^{-c_{AS}A_S^n}c_{AS}A_S^n \Rightarrow J_S^n < p_{JS}J_S^n + q_{LS}b_S e^{-c_{AS}A_S^n - c_{JS}J_S^n}A_S^n = J_S^{n+1}$. Similarly, if (J_S^n, A_S^n) is such that $J_S^n > \frac{1}{c_{JS}}g^{-1}(\frac{c_{JS}q_{LS}b_S}{c_{AS}(1-p_{JS})}e^{-c_{AS^n}A_S}c_{AS}A_S^n)$ then we have that $J_S^{n+1} < J_S^n$. So, the dynamics of the model look like indicated in the Figure 3, where $\mathcal{R}$ is the region bounded by the A_S-axis and by l_J.

Let $\mathbf{x} \in \mathcal{R}$ and $\mathbf{y} = P(\mathbf{x})$. Then $y_1 = Q_1(\mathbf{x})$. Let $\tilde{x}_1$ be such that $(\tilde{x}_1, x_2) \in l_J$. So, $\frac{\partial Q_1}{\partial x_1} = a_{11}(\mathbf{x}) \geq 0 \Leftrightarrow Q_1(x_1, x_2) \leq Q_1(\tilde{x}_1, x_2) = \tilde{x}_1 \leq \hat{J}_S$. Thus, $[0, \hat{J}_S] \times [0, \infty)$ is positively invariant, from which it follows that $\omega(\mathbf{x}) \cap [0, \hat{J}_S] \times [0, 1/c_{AS}] \neq \emptyset$, for any $\mathbf{x} \in \mathrm{R}_+^2$. So, let $\mathbf{x} > \mathbf{0}$, and $\mathbf{x}^0 \in \omega(\mathbf{x})$. From Theorem 2.2, we have $\mathbf{x}^0 \neq \mathbf{0}$, and without a loss of generality, we can assume $\mathbf{x}^0 \gg \mathbf{0}$. Taking $\mathbf{w} = (\hat{J}_S, \hat{A}_S)$, where $\hat{A}_S = \frac{q_{JS}}{1-p_{AS}}\hat{J}_S$ (notice that $Q(\mathbf{w}) \leq \mathbf{w}$) we can show, using the same monotonicity arguments as in the proof of a) and b), that $E_S = \omega(\mathbf{x})$.

□

Proof: (**of Theorem 2.7**) We may express (1) as $\mathbf{x}^{n+1} = A(\mathbf{x}^n; \beta)\mathbf{x}^n$ where the 5×5 matrix A depends on both $\mathbf{x}$ and the parameter β. We want to explore the solution set of $G(\mathbf{x}, \beta) = \mathbf{x} - A(\mathbf{x}, \beta)\mathbf{x} = 0$, especially near $\mathbf{x} = E_S$ so we set $\mathbf{x} = E_S + \mathbf{y}$. Define $F(\mathbf{y}, \beta) = E_S + \mathbf{y} - A(E_S + \mathbf{y}; \beta)(E_S + \mathbf{y}),\ \mathbf{y} \in R^5$. Then $F : \mathrm{R}^5 \times \mathrm{R} \to \mathrm{R}^5$ is continuous. Let $S = \{(\mathbf{y}, \beta) \in \mathrm{R}^5 \times \mathrm{R} | F(\mathbf{y}, \beta) = 0\}$ and note that S contains the "trivial branch" $\mathcal{T} = \{(\mathbf{0}, \beta) | \beta \in \mathrm{R}\}$. Note that $F(\mathbf{y}, \beta)$ can be written as $F(\mathbf{y}, \beta) = \mathbf{y} - f(\mathbf{y}, \beta)$, where $f(\mathbf{y}, \beta) = A(E_S + \mathbf{y}; \beta)(E_S + \mathbf{y}) - E_S$, which is obviously differentiable.

We begin by establishing that a local bifurcation from the trivial branch can occur. We write the Jacobian matrix as

$$F_{\mathbf{y}}(0, \beta) = I - \begin{pmatrix} U & D(\beta) \\ 0 & J_S^{IF}(\beta) \end{pmatrix}, \tag{11}$$

where U is a 2×2 matrix for which one is not an eigenvalue, and $J_S^{IF}(\beta)$ is given in (8). By the Implicit Function Theorem, a bifurcation can occur from $\mathcal{T}$ at $(\mathbf{x}, \beta) = (\mathbf{0}, \beta)$ only if this matrix is singular. Since one is not an eigenvalue of C,

this can only happen when one is an eigenvalue of $J_S^{IF}(\beta)$.

As $J_S^{IF}(\beta)$ has all positive entries and is strictly increasing in β, its spectral radius $r(\beta) = r(J_S^{IF}(\beta))$ is continuous and strictly increasing by Perron-Frobenius Theory (see [2]). $r(\beta)$ lies between the column sums of $J_S^{IF}(\beta)$ (again, see [2]), and so $r(\beta) \to \infty$ as $\beta \to \infty$. $r(0)$ is the spectral radius of

$$\begin{pmatrix} p_{JI} & fq_{LI}b_I\phi(\bar{T}_S) & 0 \\ q_{LI} & p_{AI} & 0 \\ b_F v_J & b_F v_A & p_F \end{pmatrix},$$

which is the maximum of p_F and the spectral radius of the upper right 2×2 block. The spectral radius of this block is greater (less) than one if and only if $\Delta_I\phi(\bar{T}_S)$ is greater (less) than one. But since $\Delta_I < \Delta_S$ we have $\Delta_I\phi(\bar{T}_S) < \Delta_S\phi(\bar{T}_S) = 1$. Hence $r(0) < 1$. Therefore, there exists a unique value of $\beta = \beta_0 > 0$ such that $r(\beta_0) = 1$.

The nullspace of $F_{\mathbf{y}}(0, \beta_0)$, $N(F_{\mathbf{y}}(0, \beta_0))$, equals span$\{\mathbf{v}\}$, where $\mathbf{v} = (\mathbf{v}_S, \mathbf{v}_I)^T$ and $\mathbf{v}_I$ is an eigenvector of $J_S^{IF}(\beta_0)$ corresponding to the eigenvalue one. Hence $\mathbf{v}_I \gg \mathbf{0}$. $N(F_{\mathbf{y}}(0, \beta_0)^T) =$ span$\{\mathbf{z}\}$ where $\mathbf{z} = (0, \mathbf{z}_I)$ and such that $\mathbf{z}_I > 0$ is an eigenvector of $J_S^{IF}(\beta_0)^T$. In order to show that a bifurcation occurs at β_0 from the trivial branch $\mathcal{T}$, it follows by the Crandall-Rabinowitz Theorem that we must show $\mathbf{z}^T F_{\mathbf{y}\beta}(0, \beta_0)\mathbf{v} \neq 0$ (see e.g. [7, Theorem I.5.1]). Calculation gives

$$F_{\mathbf{y}\beta}(0, \beta_0) = -\begin{pmatrix} 0 & D'(\beta_0) \\ 0 & J_S^{IF'}(\beta_0) \end{pmatrix},$$

where

$$J_S^{IF'}(\beta_0) = \begin{pmatrix} p_{JS}\beta_0 w_J \bar{J}_S & p_{JS}\beta_0 w_A \bar{J}_S & p_{JS}\beta_0 w_J \bar{J}_S \\ p_{AS}\beta_0 w_J \bar{A}_S & p_{AS}\beta_0 w_A \bar{A}_S & p_{AS}\beta_0 w_F \bar{A}_S \\ 0 & 0 & 0 \end{pmatrix}.$$

Consequently, $\mathbf{z}^T F_{\mathbf{y}\beta}(0, \beta_0)\mathbf{v} = -\mathbf{z}_I^T J_S^{IF'}(\beta_0)\mathbf{v}_I < 0$. We conclude from the Crandall-Rabinowitz Theorem that there is a local bifurcation of a nontrivial branch of solutions of $F(\mathbf{y}, \beta) = \mathbf{0}$ at $(\mathbf{0}, \beta_0)$ of the form

$$(\mathbf{y}, \beta) = (\mathbf{y}(s) = s\mathbf{v} + o(s), \beta(s)),\ \beta(0) = \beta_0 \tag{12}$$

for any $|s| < \delta$, for some $\delta > 0$. We assume that δ is so small that $\mathbf{y}(s) = (\mathbf{y}_S(s), \mathbf{y}_I(s))$ satisfies $\mathbf{y}_I(s) > 0$ for $s > 0$ and $\mathbf{y}_I(s) < 0$ for $s < 0$. The Crandall-Rabinowitz Theorem also implies that the intersection of a small neighborhood of $(\mathbf{0}, \beta_0)$ with S yields only points of the trivial branch and points of the branch given by (12).

Therefore, in a neighborhood of (E_S, β_0) there are precisely two branches of fixed points of G, the trivial branch and $\mathbf{x} = E_S + \mathbf{y}(s)$ corresponding to $\beta = \beta(s)$.

Now we apply the extension of the Global Bifurcation Theorem of Rabinowitz in Kielhöfer. See [7, Theorem II.5.9] for more details. Denote by $C_{loc}^+ \subset S$ the branch of solutions (12) corresponding to $s \geq 0$ and let $C_{loc}^- \subset S$ denote the branch of solutions (12) corresponding to $s \leq 0$. Let C denote the component of $NT = \overline{S \setminus \mathcal{T}}$ containing $(\mathbf{0}, \beta_0)$. Obviously, $C_{loc}^{\pm} \subset C$. Following [7], Let C^+ denote the maximal component containing $C_{loc}^+ \setminus \{(\mathbf{0}, \beta_0)\}$ in $C \setminus \{(\mathbf{0}, \beta_0)\}$ and C^- denote the maximal component containing $C_{loc}^- \setminus \{(\mathbf{0}, \beta_0)\}$ in $C \setminus \{(\mathbf{0}, \beta_0)\}$. Then $C = C^+ \cup C^- \cup \{(\mathbf{0}, \beta_0)\}$ but $C^+ = C^-$ is not excluded. According to [7, Theorem II.5.9], C^+ (and C^-)

satisfies one of the alternatives (**i**) C^+ is unbounded, or (**ii**) it contains some $(\mathbf{0}, \beta)$ where $\beta \neq \beta_0$, or (**iii**) it contains a pair of points $(\pm\mathbf{y}, \beta)$ where $\mathbf{y} \neq 0$.

Let $P = \{(\mathbf{y}, \beta) \in S | \mathbf{x} = E_S + \mathbf{y} > \mathbf{0}, \beta > 0\}$ and note that it is open in S. Then $C^+_{loc} \setminus \{(\mathbf{0}, \beta_0)\} \subset P$. We claim that $(\mathbf{0}, \beta_0)$ is the only limit point of P not belonging to P. Let $(\mathbf{y}, \beta)$ be a limit point of P and let $\mathbf{x} = E_S + \mathbf{y}$. First note that $\mathbf{x} = E_S + \mathbf{y} = 0$ cannot hold since it is an isolated fixed point by Theorem 2.6 (I). In fact, the same argument that establishes Theorem 2.6 (I) implies that the persistence is robust, meaning that the same epsilon may be used for all parameters β in a neighborhood of a given one (see [13]). Therefore, no sequence $(\mathbf{y}_n, \beta_n) \in P$ with $\beta_n \to \beta$ and $\mathbf{y}_n \to \mathbf{y}$ such that $\mathbf{x}_n = E_S + \mathbf{y}_n \to 0$ exists. More generally, if $\mathbf{x} = E_S + \mathbf{y} \in \partial \mathrm{R}^5_+$ then either $\mathbf{x} = E_S$ or $\mathbf{x} = \mathbf{0}$, since these are the only boundary equilibria. We have already shown that the latter cannot occur. We now show that if $\mathbf{x} = E_S$ then $\beta = \beta_0$. Indeed, the definition of $(\mathbf{y}, \beta)$ as a limit point of P implies that there exists a sequence $\{(\mathbf{y}^n, \beta_n)\}_n \in P$ satisfying $\mathbf{y}^n \to \mathbf{0}$ and $0 < \beta_n \to \beta$, and where $\mathbf{y}^n = (\mathbf{y}^n_S, \mathbf{y}^n_I)^T$ and $\mathbf{x}^n = E_S + \mathbf{y}^n > 0$. Then

$$0 = \frac{1}{\|\mathbf{y}^n\|}[F(\mathbf{y}^n, \beta_n) - F(\mathbf{0}, \beta_n)] = \int_0^1 F_{\mathbf{y}}(s\mathbf{y}^n, \beta_n) ds \frac{\mathbf{y}^n}{\|\mathbf{y}^n\|} .$$

Some subsequence of $\{\frac{\mathbf{y}^n}{\|\mathbf{y}^n\|}\}_n$ converges to a unit vector $\mathbf{u} = (\mathbf{u}_S, \mathbf{u}_I)^T \geq 0$ satisfying $F_{\mathbf{y}}(\mathbf{0}, \beta)\mathbf{u} = \mathbf{0}$. Since $\mathbf{u}_I \neq 0$ (or else $\mathbf{u} = 0$, see (11)) and $J^{IF}_S(\beta)\mathbf{u}_I = \mathbf{u}_I \geq \mathbf{0}$, the Perron-Frobenius Theorem implies that $r(\beta) = 1$ and therefore $\beta = \beta_0$. Hence $(\mathbf{0}, \beta)$ cannot be a limit point of P unless $\beta = \beta_0$. Finally, suppose that $(\mathbf{y}, 0)$ is a limit point of P. We may assume that $\mathbf{x} = E_S + \mathbf{y} = (\mathbf{x}_S, \mathbf{x}_I, F)^T > 0$ since we have already shown that $\mathbf{x}$ cannot be a boundary point of R^5 regardless of the value of $\beta \geq 0$. If this were the case, then

$$\mathbf{x}_S = \begin{pmatrix} p_{JS} & q_{LS} b_S \phi(T) \\ q_{JS} & p_{AS} \end{pmatrix} \mathbf{x}_S + \begin{pmatrix} (1-f) q_{LI} b_I A_I \phi(T) \\ 0 \end{pmatrix},$$

and

$$\mathbf{x}_I = \begin{pmatrix} p_{JI} & f q_{LI} b_I \phi(T) \\ q_{JI} & p_{AI} \end{pmatrix} \mathbf{x}_I.$$

As $\mathbf{x}_I > 0$ the spectral radius of the matrix multiplying $\mathbf{x}_I$ is one and, by assumption, it follows that the matrix V multiplying $\mathbf{x}_S$ has spectral radius greater than one. The latter implies that $\det(I - V) = (1 - p_{JS})(1 - p_{AS})(1 - \Delta_S(T)) < 0$ and, by Cramer's Rule, that $A_S = (x_S)_1 < 0$, a contradiction to $\mathbf{x} > 0$.

The above arguments establish the claim that $(\mathbf{0}, \beta_0)$ is the only limit point of P not belonging to P, so $\overline{P} = P \cup \{(\mathbf{0}, \beta_0)\}$. Therefore $\overline{P}$ and $\overline{S \setminus P}$ can have at most the point $(\mathbf{0}, \beta_0)$ in common but this point is missing from C^+. So, we may express the connected set C^+ as the disjoint union of two subsets: $C^+ = (C^+ \cap \overline{P}) \cup (C^+ \cap \overline{S \setminus P})$; each subset is closed in C^+. One of these sets is empty since C^+ is connected and clearly it is $C^+ \cap \overline{S \setminus P}$. Thus, $C_+ \subset P$ and consequently, alternatives (**ii**) and (**iii**) of [7, Theorem II.5.9] cannot hold. We conclude that C^+ is unbounded. But the existence of a compact attracting set that is independent of β for the dynamics (Proposition 2.1) implies that $\{\mathbf{x} | \mathbf{x} = E_S + \mathbf{y}, (\mathbf{y}, \beta) \in C^+\}$ is bounded. It follows that $\{\beta \geq 0 | (\mathbf{y}, \beta) \in C^+\}$ is unbounded and because it is connected and contains β_0, our final assertion is proved.

□

Proof: (**of Proposition 2.8**) Let $\mathbf{x} = (J_S, A_S, J_I, A_I, F) \gg \mathbf{0}$ be a fixed point and $J = J_S + J_I$ and $A = A_S + A_I$. Then

$$J = p_{JS}J_S + p_{JI}J_I + [q_{LS}b_S A_S + q_{LI}b_I A_I]\phi(T), \text{ and}$$
$$A = q_{JS}J_S + q_{JI}J_I + p_{AS}A_S + p_{AI}A_I.$$

Using the hypotheses, we find that $\mathbf{x}_S = (J, A)^T \gg \mathbf{0}$ satisfies

$$\mathbf{x}_S \leq \begin{pmatrix} p_{JS} & q_{LS}b_S\phi(T_{\mathbf{x}}) \\ q_{JS} & p_{AS} \end{pmatrix} \mathbf{x}_S.$$

By Perron-Frobenius Theory, the spectral radius of the matrix must be greater than or equal to one, or equivalently,

$$\frac{q_{JS}q_{LS}b_S\phi(T_{\mathbf{x}})}{(1-p_{JS})(1-p_{AS})} \geq 1 = \frac{q_{JS}q_{LS}b_S\phi(\bar{T}_S)}{(1-p_{JS})(1-p_{AS})}.$$

It follows that $\phi(T_{\mathbf{x}}) \geq \phi(\bar{T}_S)$ and therefore $T_{\mathbf{x}} \leq \bar{T}_S$. The final assertion follows from the definition of T.

□

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