MATHEMATICAL BIOSCIENCES
AND ENGINEERING
Volume **2**, Number **4**, October **2005**

http://www.mbejournal.org/

pp. **703–717**

PREDATOR-PREY DYNAMICS WITH DISEASE IN THE PREY

Peter A. Braza

Department of Mathematics and Statistics, University of North Florida
4567 St. Johns Bluff Road, Jacksonville, FL 32224, USA

(Communicated by Yang Kuang)

Abstract. The Holling-Tanner model for predator-prey systems is adapted to incorporate the spread of disease in the prey. The analysis of the dynamics centers on bifurcation diagrams in which the disease transmission rate is the primary parameter. The ecologically reasonable assumption that the diseased prey are easier to catch enables tractable analytic results to be obtained for the stability of the steady states and the locations of Hopf bifurcation points as a function of the ecological parameters. Two parameters of particular relevance are the ratio of the predator's intrinsic growth rate to the prey's growth rate and the maximum number of infected prey that can be eaten per time. The dynamics are shown to be qualitatively different depending on the comparative size of these parameters. Numerical results obtained with AUTO are used to extend the local analysis and further illustrate the rich dynamics.

1. **Introduction.** The basic Holling-Tanner model for predator-prey interactions is given by the equations:

$$\begin{aligned} \frac{dx}{dt} &= r_1 x\,(1-\frac{x}{K}) - \frac{a\,x\,y}{1+a\,T_h\,x} \\ \frac{dy}{dt} &= r_2 y\,(1-\frac{y}{x/J}) \end{aligned} \tag{1}$$

[1] - [5]. The variables $x(t)$ and $y(t)$ denote the prey and predator densities. The parameters r_1 and r_2 denote the intrinsic linear growth rates and K is the carrying capacity of the prey. The parameter a is the rate of successful search and T_h is the handling time. The Tanner contribution to the model, first proposed by Leslie [6], is the equation describing the predator dynamics. The predator grows exponentially for small predator values and has a prey dependent carrying capacity given by x/J. The parameter J is the number of prey required to support the predator at equilibrium.

To incorporate prey that can be susceptible to and infected with disease, we introduce variables, $S(t)$ and $I(t)$. It is assumed that the disease only affects the prey. The infected prey population will decrease due to the disease and the interactions with the predator but will increase due to the spread of disease among the susceptible prey. The susceptible prey will grow with a logistic component depending on the populations of both prey types but decline due to the transmission of disease and the interactions with the predator.

2000 *Mathematics Subject Classification.* 34C25, 37G15, 92D25 .
Key words and phrases. predator-prey, Holling-Tanner, Hopf bifurcations.

Different rates of successful search a_S and a_I will have to be introduced for the susceptible and infected prey respectively. Two factors that affect the magnitude of the search rates are the speed of movement of the predator relative to the prey and the proportion of attacks that result in a successful capture of the prey [7]. The running premise in this paper is that the predator will find it much easier to search for and catch the diseased prey rather than the merely susceptible prey thus implying that $a_S \ll a_I$. With this premise it will also be the case that $J_I \ll J_S$ since many more susceptible prey would be required to sustain the predator (it is harder to find and catch the susceptible prey). There are three primary factors affecting the handling times: time actually spent pursuing and subduing the prey, time spent eating the prey, and time during which the predator is not hungry [7]. When comparing the handling times for the susceptible prey and infected prey, the first factor comes into play only in such a manner that would imply $T_I < T_S$ but not $T_I \ll T_S$. Finally, using the parameter c to denote the net natural death rate due to disease and β to denote the disease transmission rate, the equations of the Holling-Tanner model with disease in the prey are:

$$\begin{aligned} \frac{dS}{dt} &= r_1 S(1 - \frac{S+I}{K}) - \beta SI - \frac{a_S\, S}{1 + a_I\, T_I\, I + a_S\, T_S\, S}\, Y \\ \frac{dI}{dt} &= -cI + \beta SI - \frac{a_I\, I}{1 + a_I\, T_I\, I + a_S\, T_S\, S}\, Y \\ \frac{dY}{dt} &= r_2 Y\,(1 - \frac{Y}{S/J_S + I/J_I}). \end{aligned} \tag{2}$$

The adaptations of the Holling type II functional response term to include multiple prey types follow from time budget analyses and are shown in [8] - [11].

Other authors use different models to incorporate the disease. Xiao and Chen used simple mass action (terms proportional to $-SY$ and $-IY$) to describe the decline of both prey types in the presence of the predator [12]. They were mostly interested in theoretical issues such as permanence, boundedness of solutions, nature of equilibria, etc. due to time delays attributed to gestational times. In their conclusions it was acknowledged that their results would be more meaningful if a Holling type II functional response was used. Chattopadhyay and Arino did use Holling type II responses but did not include both prey types in each response function as above [13]. They considered the case when the predator eats only the infected prey and their emphasis was on a special case in which the susceptible prey's linear growth rate is very large thus allowing the system to be reduced to two equations.

Hadeler and Freedman used a model in which the parasitically infected prey were more vulnerable to predation than the susceptible prey [14]. They pointed to several studies which provide validity to the assumption that the infected prey are easier to catch. A review article by Holmes and Bethel noted that infected prey are simply less active and therefore more prone to be caught [15]. Some prey would alter their behavior in their habitat in a way that would make them more vulnerable to predation. For example, infected snails would stay on the top of vegetation rather than safely embedded within.

The mathematical and ecological consequences stemming from the assumption that the infected prey are easier to catch have been studied from different viewpoints. This paper provides a further one in which a more precise accounting of the ecological parameter sizes quantifies the assumption and the analysis of the

dynamics is approached from a bifurcation perspective. Although the standard Holling-Tanner model is well established, no one (to this author's knowledge) has considered a predator prey system with disease that incorporates the Tanner model for the predator dynamics.

The central goal of this paper is to study how disease in the prey affects the overall dynamics of the system. This is achieved primarily by means of local bifurcation analysis and secondarily by the use of AUTO and XPPAUT [17] [18]. The paper proceeds by first non-dimensionalizing the variables and identifying a suitable small parameter based on the condition $a_S \ll a_I$. The use of a small parameter enables the ready identification of the influence that the ecological parameters have on the stabilty properties of the steady states and the location of Hopf bifurcation points.

2. **Analysis.** As a first step in the bifurcation analysis, equations (2) need to be non-dimensionalized. New prey and predator variables are defined by:

$$\text{susceptible prey} \quad s = \frac{S}{K}, \qquad \text{infected prey} \quad i = \frac{I}{K}, \qquad \text{predator} \quad y = \frac{Y\,J_I}{K}.$$

The new time is defined by

$$t_{new} = r_1 t_{old},$$

so the ratio of linear growth rates naturally arises as

$$r = \frac{r_2}{r_1}. \tag{3}$$

The scaled death rate and the disease transmission rate are given by,

$$c_{new} = \frac{c_{old}}{r_1} \quad \text{and} \quad \beta_{new} = \frac{K}{r_1}\beta_{old}.$$

Utilizing $a_S \ll a_I$, the small parameter ε will be defined by

$$\varepsilon = \frac{a_S T_S}{a_I T_I} \ll 1.$$

Furthermore, with the definitions,

$$\varepsilon\, j = J_I/J_S, \quad b = \frac{1}{a_S\, T_S\, K}, \quad q_s = \frac{1}{r_1\, J_I\, T_S}, \quad \text{and} \quad q_i = \frac{1}{r_1\, J_I\, T_I},$$

equations (2) become:

$$\begin{aligned} \frac{ds}{dt} &= s\,(1 - s - i) - \beta\, s\, i - \frac{\varepsilon\, q_s\, s}{i + \varepsilon\, s + \varepsilon b}\, y \\ \frac{di}{dt} &= -c\, i + \beta\, s\, i - \frac{q_i\, i}{i + \varepsilon\, s + \varepsilon b}\, y \\ \frac{dy}{dt} &= r\, y\,(1 - \frac{y}{i + \varepsilon\, j\, s}). \end{aligned} \tag{4}$$

As a note, the inverses of the handling times ($1/T_S$ and $1/T_I$) are often described in the literature as the maximum number of prey (of the specified type) that can be eaten per predator per time so the parameters q_s and q_i should be interpreted as scaled versions [1], [7], [16].

The first step in the bifurcation analysis is to find the steady states. The reasonable assumption $\varepsilon \ll 1$ coming from the idea that the infected prey are easier to catch allows for a simpler characterization of the steady states, albeit approximate, and subsequent bifurcation analysis. Since the main emphasis of this paper is to

study the effects of disease on predator-prey dynamics, the disease transmission rate β will be used as the primary bifurcation parameter.

2.1. **The O(1) Steady States.** With the assumption that $\varepsilon \ll 1$, a good way to begin unraveling the bifurcation structure of equations (4) is to first set the parameter ε equal to zero. With this done, equations (4) become:

$$\frac{ds}{dt} = s\,(1 - s - i) - \beta\, s\, i \tag{5}$$

$$\frac{di}{dt} = -c\, i + \beta\, s\, i - q_i\; y \tag{6}$$

$$\frac{dy}{dt} = r\, y\,(1 - \frac{y}{i}) \tag{7}$$

At this order, the infected prey are diminished by the interactions with the predator but the susceptible prey are not. The susceptible prey do decline because of the disease however and thereby increase the ranks of the infected prey. By setting the right hand sides of these equations equal to zero the following two steady states are obtained,

$$s = \frac{c + q_i}{\beta},\; i = y = \frac{\beta - c - q_i}{\beta(1+\beta)} \tag{8}$$

$$s = \frac{c}{\beta},\; i = \frac{\beta - c}{\beta(1+\beta)},\; y = 0, \tag{9}$$

along with two degenerate steady states,

$$s = 1,\; i = y = 0 \tag{10}$$

$$s = i = y = 0. \tag{11}$$

To help visualize the steady states, the susceptible prey s and infected prey i are shown as a function of the disease transmission rate β in Figure 1.

Coexistence of the predator and two prey types is featured in the primary steady state (8) and this steady state is valid only for $\beta \geq c + q_i$. This inequality essentially represents the fact that the disease transmission rate has to be above a threshold value for the infected prey to be sustained. This in turn allows the predator to thrive on its primary source of food, the infected prey. When $\beta = c + q_i$ this steady state matches the degenerate steady state (10) in which the populations of the infected prey and the predator are both zero while the susceptible prey is at its maximum. The second steady state (9) features no predator and the coexistence of the susceptible and infected prey. Although plausible on ecological grounds, it will be shown that this steady state is unstable for all parameter values. The term y/i in equations (7) is indeterminate with the latter two steady states (10 and 11) so higher order analysis will be done in a subsequent section to rectify this.

The stability of the steady states (8) and (9) is determined by linearizing equations (5 - 7) about the steady states. The resulting characteristic equations for the eigenvalues λ are of the form,

$$\lambda^3 - T_1\lambda^2 + T_2\lambda - T_3 = 0, \tag{12}$$

in which T_1 is the trace of the linearized matrix, T_2 is sum of determinants of the cofactor matrices formed from the trace elements, and T_3 is the determinant. The Routh-Hurwitz criteria of $T_1 < 0,\; T_3 < 0$, and $T_1T_2 - T_3 < 0$ are the necessary and sufficient conditions for a steady state to be stable.

For the zero predator steady state (9) in which the susceptible and infected prey coexist at the values $s = c/\beta,\ i = (\beta - c)/(\beta(1+\beta))$, the characteristic equation is

$$\lambda^3 - (r - \frac{c}{\beta})\lambda^2 + \frac{c}{\beta}(\beta - c - r)\lambda - cr\frac{\beta - c}{\beta} = 0.$$

All three conditions for stability cannot be satisfied simultaneously so this steady state is always unstable.

2.2. **The Primary O(1) Steady State.** The primary steady state given in (8), $s = (c + q_i)/\beta,\ i = y = (\beta - c - q_i)/(\beta(1+\beta))$, will yield much more interesting dynamical behavior. The characteristic equation is

$$\lambda^3 + \frac{c + q_i + \beta r - \beta q_i}{\beta}\lambda^2 + \frac{(c+q_i)(\beta - c - 2q_i + r)}{\beta}\lambda + \frac{r(c+q_i)(\beta - c - q_i)}{\beta} = 0.$$

The condition for stability, $T_3 < 0$, implies $\beta > c + q_i$ but this is automatically satisfied since it is merely the condition for the infected prey i and predator y to be positive. The condition $T_1 < 0$ is satisfied if

$$\beta(q_i - r) < (c + q_i). \tag{13}$$

This inequality foreshadows a qualitative change in the bifurcation structure when $r = q_i$.

The requirements on the parameters dictated by the third stability condition $T_1T_2 - T_3 < 0$ are not so evident. However, of particular interest for the present is the case $T_1T_2 - T_3 = 0$ which is a necessary condition for a Hopf bifurcation. Simplified somewhat, the expression is:

$$T_1T_2 - T_3 = \frac{c + q_i}{\beta^2}\left[\beta r(\beta - c - q_i) - (\beta - c - 2q_i + r)(c + q_i + \beta r - \beta q_i)\right]. \tag{14}$$

Because the local frequency of a bifurcating periodic solution is $\sqrt{T_2}$, a Hopf bifurcation can only occur if the additional condition $T_2 > 0$ accompanies $T_1T_2 - T_3 = 0$.

Viewing the disease transmission rate β as the primary bifurcation parameter, a Hopf bifurcation may occur at the following two values:

$$\beta_\pm = \frac{1}{2q_i}\left(D \pm \sqrt{D^2 - 4q_i(c+q_i)(c+2q_i-r)}\right) \tag{15}$$

$$\text{with}\quad D = (c+q_i)(1+q_i) + (q_i - r)^2.$$

On the face of it, it appears that there would be two Hopf bifurcation values of β if $c + 2q_i - r > 0$ but the condition $T_2 > 0$ also imposes the requirement $r < q_i$; if $r > q_i$ there is only one Hopf bifurcation value at β_+.

A useful property is that the steady states do not depend on the ratio r of the linear growth rates but the stability and the location of the Hopf bifurcation point(s) do depend on r. One example in the two Hopf bifurcation point case is that, as r increases to q_i, the value of β_- from (15) decreases to $c + q_i$ which corresponds to the point where the primary steady state (8) intersects the degenerate one (10). Also, the steady state branch is stable when β satisfies $\beta_- < \beta < \beta_+$ and unstable when β satisfies $c + q_i < \beta < \beta_-$ or $\beta > \beta_+$. This is the scenario depicted in Figure 1. As a further note, when $r \to q_i^-$, the stability of the $s = (c + q_i)/\beta$, branch extends all the way from $\beta = \beta_+$ back down to $\beta = c + q_i$ or the $s = 1$ degenerate steady state branch. When $r > q_i$ there is only one Hopf bifurcation at $\beta = \beta_+$ and the steady state branch is stable for $\beta < \beta_+$ but unstable when $\beta > \beta_+$; the stability of the branch is determined by $T_1T_2 - T_3 < 0$ since (13) is automatically satisfied.

In all cases, the bifurcations are supercritical so that the periodic solutions are locally stable. Representative bifurcation diagrams (using the full equations (4)) created with AUTO in XPPAUT [18] are illustrated in Figures 3 - 7.

The remarks are predicated on the assumption that there will be Hopf bifurcation points on the primary $O(1)$ steady state when $r < q_i$ but this need not be the case. It is apparent that the two Hopf bifurcation values of β may coalesce if the discriminant

$$D^2 - 4q_i(c + q_i)(c + 2q_i - r) \tag{16}$$

in expression (15) is equal to zero and indeed, there will be no Hopf bifurcation values of β if the discriminant is less than zero. There are different ways to characterize the ensuing parameter relationships. One way to proceed is to fix q_i and then determine the values of the parameters c and r that make the discriminant less than zero; the results of this can be seen in Figure 2. Bifurcation diagrams (using the full equations (4)) for the case when there is no Hopf bifurcation on the primary branch are shown in Figures 8 and 9. Note that there is a bifurcating periodic solution but it arises from the degenerate steady state $s = 1, i = y = 0$ (10). Understanding how the bifurcations from this steady state are linked with the primary steady state (8) is essential for the proper insight into the dynamics and a complete view of the overall bifurcation diagram.

2.3. **The Degenerate Steady States.** Of some ecological importance is the value of the disease transmission rate β that destabilizes the environment in which the susceptible prey are the only thriving species. The stability properties of the order 1 steady state $s = 1, i = y = 0$ (10), which indicate that only the susceptible prey are present, need to be determined. A more delicate analysis has to be done since the term y/i found in equations (7) is indeterminate. To accomplish this, the variables are expanded in asymptotic series in ε as:

$$\begin{aligned} s(t) &= 1 + s_1(t)\,\varepsilon + s_2(t)\,\varepsilon^2 + \cdots \\ i(t) &= i_1(t)\,\varepsilon + i_2(t)\,\varepsilon^2 + \cdots \\ y(t) &= y_1(t)\,\varepsilon + y_2(t)\,\varepsilon^2 + \cdots \end{aligned} \tag{17}$$

When these expressions are substituted into equations (4), a set of equations is generated at each power of ε. At $O(\varepsilon)$ there are *three* different steady states given by:

$$(s_1, i_1, y_1) = (0, 0, 0) \tag{18}$$

$$(s_1, i_1, y_1) = (-qs/2, 0, 1) \tag{19}$$

and

$$s_1 = \frac{(1+\beta)(2\beta - 2c - q_i)}{\beta - c - q_i} - \frac{(\beta - c)q_s}{q_i}, \quad i_1 = \frac{-2\beta + 2c + q_i}{\beta - c - q_i}, \quad y_1 = \frac{-\beta + c}{\beta - c - q_i}. \tag{20}$$

The parameters b and j were set equal to 1 since their significance is minor and the subsequent analysis will be simplified by fixing the values.

Although not mentioned yet but quite evident is the fact that $s = 1, i = y = 0$ is a steady state of the full equations (4) and the $O(\varepsilon)$ steady state $(s_1, i_1, y_1) = (0, 0, 0)$ in (18) is just the beginning part of its expansion. By considering the characteristic equation, it is easy to see that this steady state is always unstable.

The second steady state $(s_1, i_1, y_1) = (-qs/2, 0, 1)$ given in (19) indicates that a small number of the predator coexists with the bountiful susceptible prey but

no infected prey. Actually, when the equations for this steady state are solved at the higher orders of ε, $i_k = 0$ for all k. In other words, there are no infected prey. The characteristic equation for the eigenvalues of the $O(\varepsilon)$ system linearized about the second steady state is $\lambda^3 - T_1\lambda^2 + T_2\lambda - T_3 = 0$ with the coefficients $T_1 = \beta - c - q_i/2 - 1 - r$, $T_2 = (c + q_i/2 - \beta)(1 + r) + r$, and $T_3 = r(\beta - c - q_i/2)$. The conditions for stability $T_1 < 0$, $T_3 < 0$, and $T_1T_2 - T_3 < 0$ imply that this steady state is unstable when the disease transmission rate satisfies $\beta > c + q_i/2$ and stable when $\beta < c + q_i/2$. Basically this means that if the disease is not transmitted above a sufficiently high rate, the susceptible prey thrives at levels near its capacity, the infected prey are not sustained at all and the predator population is small - just subsisting on the harder to catch susceptible prey.

2.3.1. *The Third Degenerate Steady State.* The third steady state given in (20) has the most interesting bifurcation possibilities. This steady state only makes sense ecologically if the values for i_1 and y_1 are positive ($s = 1 + s_1\,\varepsilon$ will automatically be positive). This imposes the condition on β,

$$c + \frac{q_i}{2} < \beta < c + q_i. \tag{21}$$

The second steady state (19) bifurcates into the third branch at $\beta = c + q_i/2$, and, as will be seen, transfers the stability to the third branch.

The characteristic equation for the $O(\varepsilon)$ linearized system is again of the form, $\lambda^3 - T_1\lambda^2 + T_2\lambda - T_3 = 0$. This time the coefficients are less tractable:

$$\begin{aligned} T_1 &= \left(2(\beta - c)(\beta - c - \frac{q_i}{2}) - q_i(1 + r)\right)/q_i \\ T_2 &= \left[-2(\beta - c)(\beta - c - \frac{q_i}{2}) - r\left(2(\beta - c - \frac{q_i}{2})(\beta - c - q_i) - q_i\right)\right]/q_i \\ T_3 &= 2r(\beta - c - \frac{q_i}{2})(\beta - c - q_i)/q_i. \end{aligned} \tag{22}$$

It is easily seen that the stability condition $T_3 < 0$ is satisfied when $c + q_i/2 < \beta < c + q_i$ which is precisely the domain of β that gives ecologically valid (greater than zero) population values (21). The stability condition $T_1T_2 - T_3 < 0$ is more cluttered with parameters but setting $T_1T_2 - T_3 = 0$ admits four values of β which satisfy $\beta_1 < \beta_2 < \beta_h < \beta_4$. The values β_1 and β_2 are not ecologically valid being less than $c + q_i/2$. The condition $T_1T_2 - T_3 < 0$ is satisfied when $\beta_2 < \beta < \beta_h$ and $\beta > \beta_4$. The stability condition $T_1 < 0$ holds true when $\beta_{T1-} < \beta < \beta_{T1+}$ where $\beta_{T1-} < c + q_i/2$ and $\beta_h < \beta_{T1+} < \beta_4$. The combined effect of all these inequalites is that the third branch is stable when,

$$c + \frac{q_i}{2} < \beta < \beta_h \tag{23}$$

and unstable otherwise.

The value of

$$\beta = \beta_h = c + \frac{q_i}{4} + \frac{1}{4}\sqrt{q_i^2 + 8q_ir} \tag{24}$$

is special since a Hopf bifurcation will occur at $\beta = \beta_h$ if $T_2(\beta_h) > 0$. As noted earlier, this latter condition is necessary because the local frequency of the bifurcating periodic solution is $\sqrt{T_2}$. It can be verified that $T_2(\beta_h) > 0$ only if $r < q_i$. Additionally, as r increases to q_i, β_h increases to $c + q_i$, which is the upper value of β for which the steady state is valid (21) and also the value of β for which the steady state expansion (20) becomes singular.

2.4. **Ecological Meaning for the Condition $r > q_i$.** Before continuing, some ecological meaning needs to be ascribed to the condition $r > q_i$. The parameter r was scaled (3) so that it was proportional to the predator's linear growth rate r_2 and that rate governs the predator's growth especially when the predator population is small as can be seen by considering the original equations (2). The third degenerate steady state branch given in (20) features a small predator population and the stability of that branch extends further as the value of r (r_2) increases. It is reasonable on ecological grounds to suspect that the predator population would be stable if its growth rate r_2 is relatively large. That observation begs the question, "what is relatively large?," - the inequality $r > q_i$ provides a meaningful answer. The third degenerate branch is always stable if $r > q_i$ or it loses its stability at a Hopf bifurcation if $r < q_i$.

The significance of q_i as the dividing point can be found by considering the inequality

$$r_2 > \frac{1}{J_i T_i},$$

which is equivalent to $r > q_i$. Both r_2 and $1/(J_i T_i)$ are rates. Since T_i is the handling time per infected prey and J_i is the number of infected prey needed to sustain the predator at equilibrium, the product $J_i T_i$ is the time to handle all the prey needed to sustain the predator. This means that $1/(J_i T_i)$ can be interpreted as the *sustaining rate* for the predator. It makes sense that a healthy, stable environment for the predator would exist if its natural growth rate r_2 exceeded its sustaining rate. From an ecological viewpoint, an environment is less stable for a predator if there is periodic behavior (which will occur if $r < q_i$) when the population is low since noise could then make the population disappear. This observation is particularly pertinent for the predator population in the third degenerate steady state since its value is small.

On a related note, when considering the primary $O(1)$ steady state (8) in section 2.2, it was pointed out that there was only one Hopf bifurcation point (at $\beta = \beta_+$) when $r > q_i$ and that steady state was stable for values of the disease transmission rate β up to β_+. This stability property agrees with the significance expressed above in which the condition $r > q_i$ is favorable for the predator. Actually, the next section establishes that the primary $O(1)$ steady state and the third degenerate steady state are just parts of the same branch. This implies that, in the $r > q_i$ case, the predator population (in particular) will be stable all the way from nearly depleted value given in (20) to the more robust value in (8) with $\beta = \beta_+$.

2.5. **The Connection Between the Steady States, Patching the Singularity at $\beta = c + q_i$.** The values of the predator and infected prey in the primary $O(1)$ steady state given in (8) are positive (and therefore ecologically relevant) only if the disease transmission rate β satisfies $\beta > c + q_i$. Seemingly contrary to this, the values of the predator and infected prey in the third degenerate steady state given in (20) are positive only if $\beta < c + q_i$. Moreover, the values in the degenerate steady state become singular at $\beta = c + q_i$. It was not brought up in the discussion in section 2.2 but there are $O(\varepsilon)$ corrrections to the primary $O(1)$ steady state (8) that also become singular at $\beta = c + q_i$.

It is readily seen by using numerical techniques such as AUTO that these two branches are really parts of the same branch. The analytic way of showing this is to bridge the singularity by expanding the steady state of the full system (4) in powers of $\varepsilon^{1/2}$ instead of ε. The idea is to accomodate the $O(\varepsilon)$ terms that

become infinite at $\beta = c + q_i$ by putting in larger $O(\varepsilon^{1/2})$ corrections. Noting the form of the highest order values of the primary $O(1)$ steady state (8) and the third degenerate steady state (20), the variables s, i, and y are expanded as:

$$\begin{aligned} s(t) &= 1 + s_1(t)\,\varepsilon^{1/2} + s_2(t)\,\varepsilon^1 + \cdots \\ i(t) &= i_1(t)\,\varepsilon^{1/2} + i_2(t)\,\varepsilon^1 + \cdots \\ y(t) &= y_1(t)\,\varepsilon^{1/2} + y_2(t)\,\varepsilon^1 + \cdots \end{aligned} \tag{25}$$

The disease transmission rate is expanded as

$$\beta = c + q_i + \varepsilon^{1/2} p$$

so that the magnitude of p measures how far β is from the singular value $c + q_i$. When these expressions are substituted into the full system (4) and the coefficients of the powers of $\varepsilon^{1/2}$ are in turn equated to zero, the following steady state values are obtained:

$$s_1 = -\frac{p + \sqrt{p^2 + 4q_i(c+q_i)(1+c+q_i)}}{2(c+q_i)} \tag{26}$$

$$i_1 = y_1 = \frac{p + \sqrt{p^2 + 4q_i(c+q_i)(1+c+q_i)}}{2(c+q_i)(1+c+q_i)}. \tag{27}$$

By the nature of the expansion of β, these expressions are only valid when p is less than $O(1/\varepsilon^{1/2})$.

This analysis provides the patch for the singularities in the expansions at $\beta = c + q_i$, thus establishing analytically that the primary $O(1)$ steady state given in (8) and the third degenerate steady state given in (20) are parts of the same steady state branch.

3. **Numerical Results and Discusssion.** The interplay of the local mathematical results with the ecological parameters has been the focus of this paper. In accord with this analysis, global numerical results are discussed using bifurcation diagrams which are explored using AUTO (in XPPAUT) from the vantage point of either $r > q_i$ or $r < q_i$.

As pointed out, the condition $r > q_i$ is beneficial for the stability of the steady state predator population when it is low in number. A representative bifurcation diagram is illustrated in Figure 3. The steady state is stable until the lone Hopf bifurcation point at β_+ (15). The resulting periodic branch is supercritical and remains stable throughout the domain of interest. Even though the periodic solution is mathematically (deterministically) stable for all β, the system is inherently unstable from an ecological point of view when the population size becomes close to zero since noise from environmental fluctuations can cause one of the species to become extinct [2], [19] This ecological instability could have been independently conjectured on the basis that the disease transmission rate was simply too large to sustain the prey.

There are more exciting bifurcation possibilities when $r < q_i$. As r decreases from a value larger than q_i to one smaller than q_i, two new Hopf bifurcation points emerge at β_h (24) of the third degenerate steady state and β_- (15) of the primary $O(1)$ steady state. The periodic solution branch originating at β_h terminates at β_-. When these β values are relatively close, the resulting periodic solution is stable throughout with a relatively small amplitude as can be seen in Figure 4. As r decreases further from q_i, β_h and β_- separate more causing the periodic solution

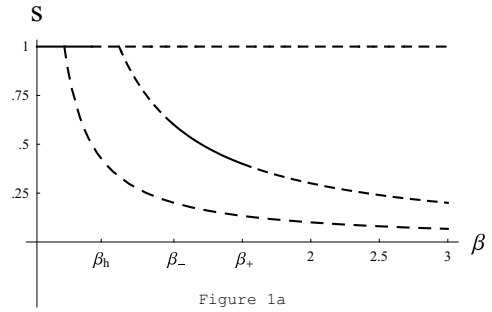

Figure 1a

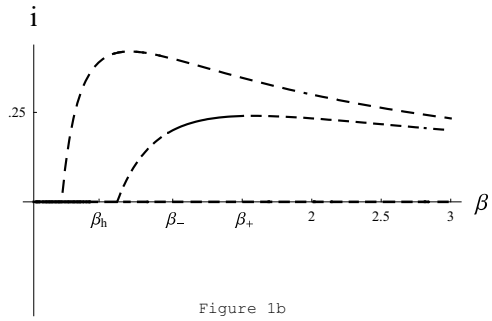

Figure 1b

FIGURE 1. The $O(1)$ steady states are shown for the susceptible (Fig. 1a) and infected prey (Fig. 1b). The figures illustrate the case in which there are three Hopf bifurcation points. The solid curve represents the stable steady state and the dashed curve is the unstable steady state.

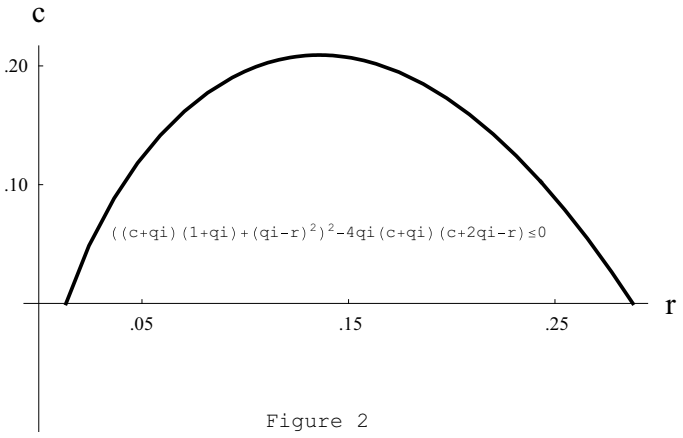

Figure 2

FIGURE 2. There will be two Hopf bifurcation points on the primary $O(1)$ steady state if the point (r, c) lies above the curve and none if (r, c) lies below the curve. The value of q_i was fixed at .40.

amplitude to increase and the branch to lose stability at period doubling points (see Figures 5 and 6). Also to be observed, the periodic solution amplitude of

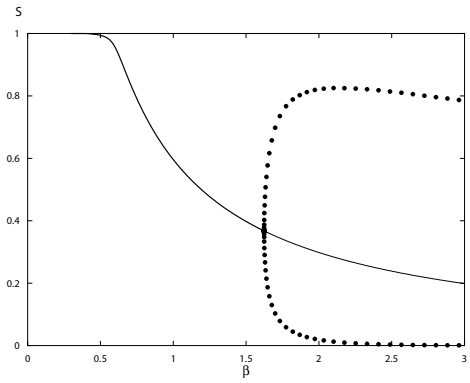

Figure 3a

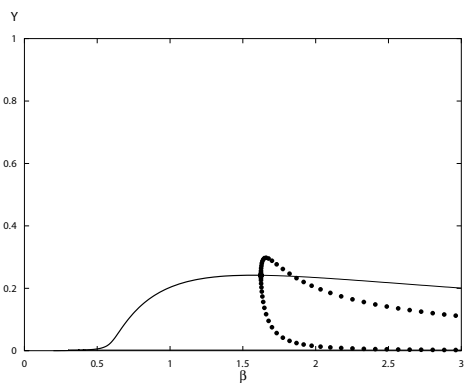

Figure 3b

FIGURE 3. Bifurcation diagrams for the susceptible prey (Fig. 3a) and the predator (Fig. 3b) verses the disease transmission rate are shown for the case $r > q_i$. The steady state is stable all the way to the lone Hopf bifurcation point and the resulting periodic solution is stable throughout. The unstable steady state is represented by the light line. The maximum and minimum of the periodic solutions are shown with solid circles (sometimes appearing as a thick solid curve) denoting that they are stable. The parameters used are $r = .45$, $c = .2$, $q_i = .4$, $\varepsilon = .002$, $q_s = .1$, and $b = j = 1$.

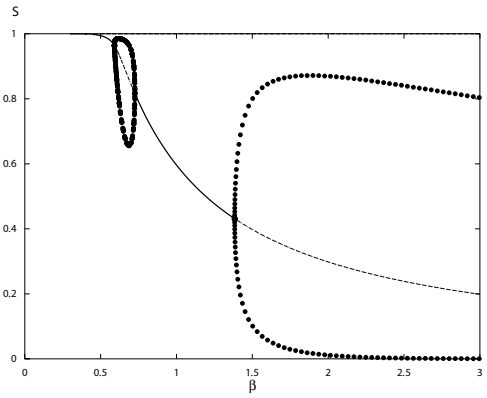

Figure 4

FIGURE 4. The bifurcation diagram of the susceptible prey shows two stable periodic solution branches. The first one has a relatively small amplitude and it stems from the Hopf bifurcation point $\beta = \beta_h$ and terminates at β_-. The second branch starts at β_+ and remains stable. The parameters used are $r = .3$, $c = .2$, $q_i = .4$, $\varepsilon = .002$, $q_s = .1$, and $b = j = 1$.

the susceptible prey approaches 1 and the predator's minimum value approaches zero. Although not part of the figure, the infected prey population also gets close to zero. These latter two factors make the system ecologically unstable but this time the cause can be attributed to the fact that r has become too distant from its sustaining value q_i.

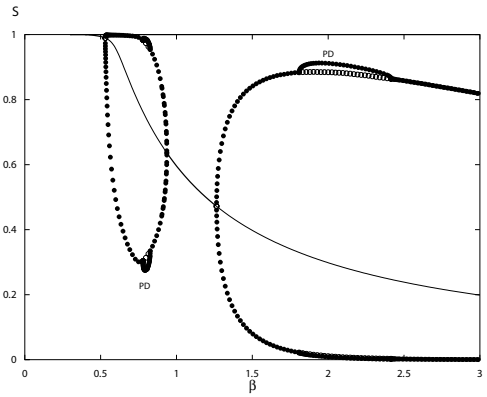

Figure 5

FIGURE 5. The bifurcation diagram of the susceptible prey shows the two periodic solution branches in which the separation of β_h and β_- is greater than in Figure 4 due to a smaller value of r. Both branches lose stability at period-doubling points and the (stable) period-doubling branches are labelled as PD. The parameters used are $r = .2$, $c = .2$, $q_i = .4$, $\varepsilon = .002$, $q_s = .1$, and $b = j = 1$.

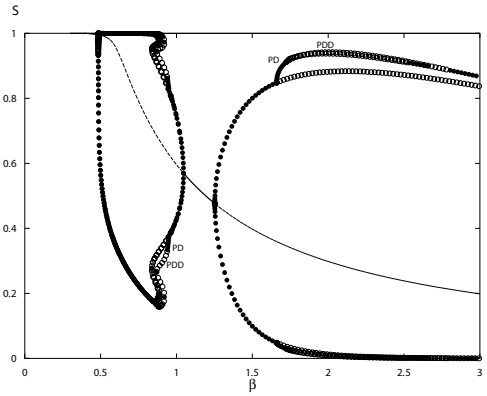

Figure 6

FIGURE 6. The bifurcation diagram of the susceptible prey in which there is a sequence of period doubling points. The value of r is less than in Figure 5 and the consequent separation of β_h and β_- is greater. Unstable periodic solutions are denoted by open circles. The period-doubling branches are labelled as PD and the period-doubling branches bifurcating from the basic period-doubling branches are labelled as PDD. The parameters used are $r = .12$, $c = .2$, $q_i = .4$, $\varepsilon = .002$, $q_s = .1$, and $b = j = 1$.

Mathematically the bifurcation diagrams are more compelling as r decreases from q_i. In Figure 5 it is seen that period-doubling branches emerge as stable attractors in the region in which the periodic solutions were previously stable. In Figure 6 with the same q_i as in Figure 5 but a smaller r, a sequence of period-doubling points give rise to higher period attractors on both periodic branches. Ascribing precise

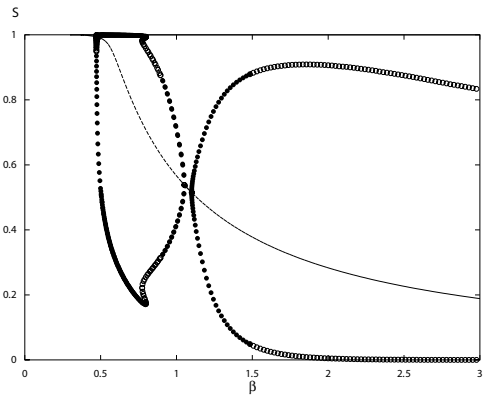

Figure 7

FIGURE 7. The bifurcation diagram of the susceptible prey in which the Hopf bifurcation points β_- and β_+ nearly coalesce. The parameters used are $r = .15$, $c = .17$, $q_i = .4$, $\varepsilon = .002$, $q_s = .1$, and $b = j = 1$.

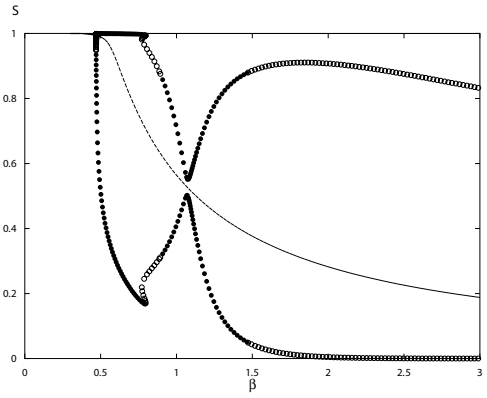

Figure 8

FIGURE 8. The bifurcation diagram of the susceptible prey in which the Hopf bifurcation points β_- and β_+ have passed through each other and disappeared. The lone periodic solution branch arises from the Hopf bifurcation point β_h. The parameters used are $r = .15$, $c = .168$, $q_i = .4$, $\varepsilon = .002$, $q_s = .1$, and $b = j = 1$.

ecological meaning to these mathematical results would be a stretch, but it would be appropriate to infer that the system may simply be in a realm of ecologically interesting dynamics.

Another feature in the $r < q_i$ case is that the Hopf bifurcation points β_- and β_+ on the primary $O(1)$ steady state may coalesce and disappear. The conditions for this occurring were discussed at the end of section 2.2 and the resulting parameter relationships were illustrated in Figure 2. Figure 7 shows the bifurcation diagram in which β_- and β_+ nearly coalesce and Figure 8 is the companion figure in which β_- and β_+ have just disappeared. The interesting feature is that the periodic solution from β_h dips down toward the ghosts of the Hopf bifurcations that were

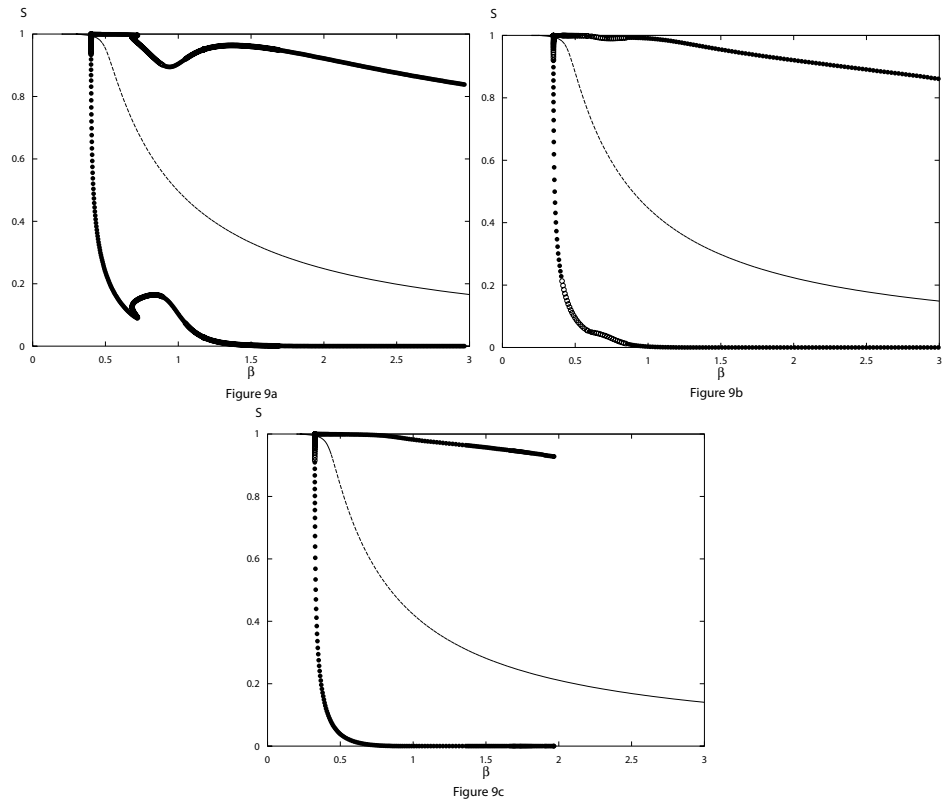

FIGURE 9. As in Figure 8, bifurcation diagrams are shown in which there is one Hopf bifurcation at $\beta = \beta_h$. By decreasing the disease death rate c, the periodic branch loses its secondary bifurcations and eventually becomes entirely stable. Figures 9a, 9b, and 9c all use the values $r = .15$, $q_i = .4$, $\varepsilon = .002$, $q_s = .1$, and $b = j = 1$ but $c = .1$ in Figure 9a, $c = .05$ in Figure 9b, and $c = .025$ in Figure 9c.

at the points β_- and β_+ while the secondary bifurcation features remain the same. Figure 2 was used as a guide to explore this solution more by fixing the values of r and q_i and varying c, the net disease death rate. As c decreases further into the region, the periodic solution branch loses its secondary bifurcation points and eventually becomes entirely stable. This can be seen in the sequence of bifurcation diagrams in Figure 9. Interestingly, with a smaller net death rate due to disease, the system can be ecologically unstable.

From both mathematical and ecological perspectives, the bifurcation possibilities in the Holling-Tanner predator-prey system with disease in the prey are quite rich. A fundamental demarcation in the dynamics occurs at $r = q_i$, which can be interpreted as the predator's (scaled) growth rate, r, equaling its sustaining rate q_i. As discussed, there are many bifurcation possibilities when $r < q_i$, which contrasts with the much simpler solution structure when $r > q_i$. When $r > q_i$, there is a larger range of the disease transmission rate β in which the steady state populations are stable. Those stable steady branches lose stability at a Hopf bifurction for a

relatively large value of β and the emerging periodic solutions are stable. This comparatively uncomplicated stable mathematical behavior makes ecological sense since the condition $r > q_i$ indicates that the predator's rate of growth is greater than its sustaining rate.

REFERENCES

[1] P. A. Braza, *The bifurcation structure of the Holling-Tanner model for predator-prey interactions using two-timing*, SIAM J. Appl. Math. Vol. 63, No. 3 (2003), pp. 889-904.

[2] R. M. May, *Stability and Complexity in Model Ecosystems*, Princeton University Press, Princeton, NJ, 1973.

[3] J. T. Tanner, *The stability and the intrinsic growth rates of of prey and predator populations*, Ecology, 56 (1975), pp. 855-867.

[4] E. Saez and E. Gonzalez-Olivares, *Dynamics of a predator-prey model*, SIAM J. Appl.Math., 59 (1999), pp. 1867-1878.

[5] D. J. Wollkind, J. B. Collings, and J. A. Logan, *Metastability in a temperature-dependent model system for predator-prey mite outbreak interactions on fruit flies*, Bull. Math. Biol., 50 (1988), pp.379-409.

[6] P. H. Leslie, *Some further notes on the use of matrices in population mathematics*, Biometrika, 35, (1948), pp. 213-245.

[7] C. S. Holling, *The functional response of invertebrate predators to prey density*, Mem. Ent. Soc. Can., (1965), pp. 3-60.

[8] M. J. W. Cock, *The assessment of preference*, J. Animal Ecol., 47, (1978), pp. 805-816.

[9] W. W. Murdoch, *The functional response of predators*, J. Appl. Ecol., 10, (1973), pp. 335-342.

[10] J. H. Lawton, J. R. Beddington, and R. Bonser, *Switching in Invertebrate predators*, in *Ecological Stability,* M. B.Usher and M. H. Williamson eds., Chapman and Hall, London, (1974), pp. 141-158.

[11] A. Sikder, *Uniform persistence for sigmoidal diet selection with keystone prey species*, J. Math. Biol. 41, (2000), pp. 25-44.

[12] Y. Xiao and L. Chen, *Modeling and analysis of a predator-prey model with disease in the prey*, Math. Biosciences, 171, (2001) pp. 59-82.

[13] J. Chattopadhyay and O. Arino, *A predator-prey model with disease in the prey*, Nonlinear Anal., 36, (1999), pp. 747-766.

[14] K. P. Hadeler and H. I. Freedman, *Predator-prey populations with parasitic infection*, J. Math. Biol. 27, (1989), pp. 609-631.

[15] J. C. Holmes and W. M. Bethel, *Modification of Intermediate host behaviour by parasites*, in *Behavioural aspects of parasite transmission*, E. V. Canning, C. A. Wright eds., Suppl. I to Zool. f. Linnean Soc., 51, (1972), pp.123-149.

[16] B. W. Kooi, L. D. J. Kuijper, M. P. Boer, S. A. L. M. Kooijman, *Numerical bifurcation analysis of a tri-trophic food web with omnivory*, Math. Biosciences, 177&178, (2002), pp. 201-228.

[17] E. J. Doedel and J. P. Kernevez, *AUTO, Software continuation and bifurcation problems in ordinary differential equations,* Technical report, California Institute of Technology, Pasadena, CA, 1986.

[18] B. Ermentrout, *Simulating, Analyzing, and Animating Dynamical Systems: A Guide to XPPAUT for Researchers and Students,* SIAM Press, Philadelphia, PA, 2002.

[19] B. Spagnolo, D. Valenti, A. Fiasonaro, *Noise in ecosystems: A short review*, Math. Biosciences and Engineering, Vol. 1, No. 1 (2004), pp. 185-211.

Received on January 17, 2005. Revised on September 23, 2005.

E-mail address: pbraza@unf.edu