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A Comparative Study on the Impact of Infection on the Stability of Eco-epidemiological Models

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Abstract. This study investigates the stability of eco-epidemiological systems by analyzing a deterministic ordinary differential equation (ODE) model of predator-prey interactions with disease transmission. The model incorporates both Beddington-DeAngelis and Holling Type IV functional responses. Addressing a critical gap in understanding coexistence dynamics under disease pressure, we employ Lyapunov stability theory and Routh-Hurwitz criteria to demonstrate that fatal prey infection universally precludes three-species coexistence, regardless of functional response type. Our results reveal that system dynamics exhibit sensitive dependence on infection and predation rates, showing heightened extinction risks due to prey defense mechanisms. These findings advance theoretical ecology by quantifying disease-predation tradeoffs and provide predictive stability thresholds with direct implications for ecosystem management and conservation strategies in disease-affected environments.

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Key Words and Phrases: Eco-epidemiological Model, Prey-predator, Local Stability, Beddington-DeAngelis, Holling Type IV

1. Introduction

Ecology and epidemiology, though distinct, share several common features and are increasingly integrated into the field of eco-epidemiology, which addresses both ecological and epidemiological dimensions, highlighting the profound influence of disease on ecological systems from both mathematical and ecological standpoints [1], [2]. The introduction of a parasite into a population of hosts and its subsequent behavior are intricately shaped by interactions with other community members, particularly predators. The intensity of predation plays a critical role in determining community structure and ecosystem characteristics. Within host-parasite systems, predation can have a substantial impact, modifying the population dynamics of both hosts and parasites, potentially acting as a barrier to parasite establishment [3], [4].

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In theoretical investigations of host-parasite-predator interactions, predator behavior is frequently modeled in a simplified manner. A key component in these models is the functional response, defined as the rate at which a predator consumes prey per unit of time [4]. Recent advances in discrete-time modeling [5] and conformable derivative approaches [6] have expanded our understanding of these dynamics. This response captures characteristics of both predator and prey behavior, shaped by factors such as prey escape mechanisms, habitat complexity, and handling time. The present study introduces a host-parasite-predator model to explore the influence of infection rate, attack rate, and the nature of the predator functional response on the system's behavior [7].

This research contributes original insights through an eco-epidemiological model that analyzes dual predation (susceptible versus infected prey) and disease transmission to predators, while comparing Beddington-DeAngelis and Holling Type IV functional responses. Our analysis identifies critical stability thresholds that reveal how predator behavior mediates disease-driven ecosystem collapse, advancing both theoretical ecology and conservation applications.

Venturino (1995) examined SIS models in which disease transmission occurs within the prey population, incorporating logistic growth for both prey and predator populations, with predators exclusively feeding on infected prey. Hethcote et al. (2004) explores a predator-prey model modification to include logistic growth in the prey population and an SIS disease framework, under the assumption that infected prey is more vulnerable to predation and serves as a resource for predator population growth [2], [8]. However, these models possess certain limitations, as predators generally consume both infected and susceptible prey. Feeding on infected prey can adversely affect predator populations, often resulting in negative growth. Whenever a parasite causes infection in the prey population, the infection can transmit to the predator population via their interactions, potentially causing the collapse of both populations when the infection is lethal. Our work introduces and analyzes a mathematical model incorporating these eco-epidemiological features. Investigating and comparing the model's dynamics to identify critical system parameters and their ranges is our objective, enabling the prediction of various theoretical outcomes arising from the interplay among susceptible prey, infected prey, and predators.

In this study, two functional responses are employed. The first is the Beddington-DeAngelis functional response, an extension of the Holling Type II response, which accounts for mutual interference among predators. Our model describes how the rate of prey consumption by predators is influenced not only by prey density but also by the density of the predators themselves. The key feature is the inclusion of an additional term that accounts for the reduction in predation efficiency due to interactions among predators, such as competition or interference. This functional response is particularly useful in ecological modeling as it provides a more realistic representation of predator-prey dynamics, especially in environments where predator density significantly influences predation rates [9], [10], [11].

The following form expresses the Beddington-DeAngelis functional response:

$$\varphi(S,P) = \frac{\alpha S}{S + bP + c} \tag{1}$$

The second functional response utilized is the Holling Type IV, a model employed in ecology to characterize how a predator's rate of prey consumption varies in response to changes in prey density. Unlike the simpler Holling Types I, II, and III, which describe linear, hyperbolic, and sigmoidal responses respectively, Holling Type IV incorporates a more complex scenario where the predator's consumption rate initially increases with prey density, then decreases after reaching a peak. This decrease can be due to factors like predator satiation or increased handling time as prey becomes more abundant [12], [13]. This type of functional response is particularly useful in modeling situations where predators experience diminishing returns in prey capture efficiency at high prey densities, reflecting more realistic ecological interactions. The generalized Holling Type IV functional response [14], [15], [16] is given as:

$$\varphi(S) = \frac{\alpha S}{1 + a_1 S + a_2 S^2}, \quad a_2 > 0, \quad a_1 > -2\sqrt{a_2}, \quad \alpha > 0.$$
 (2)

The Holling Type IV response function $\varphi(S)$, also referred to as the Monod-Haldane response function [14], is positive for S > 0 and increases within the interval $S \in [0, 1/\sqrt{a_2}]$. The function reaches its maximum value at $S = 1/\sqrt{a_2}$, decreases for $S > 1/\sqrt{a_2}$, and approaches zero as S tends toward infinity. The phenomenon, when prey populations grow large enough, they become more capable of avoiding, protecting, or defending themselves against predators, is captured in this response function [17], [18].

To formulate our model, which extends classical predator-prey frameworks by integrating disease dynamics in prey, we consider a system where predators preferentially consume infected individuals due to their reduced escape ability. The Beddington-DeAngelis functional response accounts for predator interference, while the Holling Type IV functional response captures prey defense mechanisms at high densities.

This formulation aligns with empirical observations where disease alters predation efficiency and justifies the inclusion of biologically meaningful terms such as: λIS (disease transmission), γIP (predation on infected prey), we depended on the model used in [19], based on the assumptions in [19] and the functional responses we use in this work as discussed in Section 2 and 3.

2. First model analysis

In this section, the boundedness, finding equilibria, and their stability are described with respect to the Beddington-DeAngelis functional response.

The eco-epidemiological model incorporating the Beddington-DeAngelis functional response:

$$\begin{cases}
\frac{dS}{dT} &= rS\left(1 - \frac{S+I}{K}\right) - \lambda IS - \frac{\alpha SP}{S+bP+c}, \\
\frac{dI}{dT} &= \lambda IS - \gamma IP - d_1 I, \\
\frac{dP}{dT} &= \frac{\alpha mSP}{S+bP+c} - m\gamma IP - d_2 P,
\end{cases}$$
(3)

where system (3) is examined under the initial conditions S(0) > 0, I(0) > 0, and P(0) > 0.

Here, S represents the susceptible population, I corresponds to the infected population, and P indicates the predator population at time T. To maintain biological validity, all parameters in the model are positive, and their specific biological interpretations are detailed in Table 1.

Parameter	Biological Meaning	
r	Intrinsic growth rate	
K	Environmental carrying capacity	
λ	Infectious contact rate	
α	Maximal relative increase of predation	
b	Magnitude of interference among predators	
c	Half-saturation constant	
γ	Attack rate on infected prey	
d_1	Natural death rate	
d_2	Natural mortality rate of predator	
\overline{m}	Conversion efficiency	

Table 1: Biological interpretations of all parameters.

The following theorem demonstrates that the linear combination of susceptible prey, infected prey, and predator populations remains below a finite value, indicating the boundedness of solutions of system (3).

Theorem 1. The solution (S(t), I(t), P(t)) is uniformly bounded for initial condition $(S_0, I_0, P_0) \in \mathbb{R}^3_+$.

Proof. Consider the function $W(t) = S(t) + I(t) + \frac{1}{m}P(t)$, which is defined from $\mathbb{R}_0^+ \to \mathbb{R}_0^+$ and is differentiable on the interval (0,t). The derivative $\frac{dW(t)}{dT}$ along the trajectory of the system (2.1) can be written as:

$$\frac{dW(t)}{dT} = rS\left(1 - \frac{S+I}{K}\right) - \lambda IS - \frac{\alpha SP}{S+bP+c} + \lambda IS - \gamma IP - d_1I + \frac{\alpha SP}{S+bP+c} - \gamma IP - \frac{d_2}{m}P.$$

Now, for any a > 0, we have

$$\frac{dW(t)}{dT} + aW(t) = rS - \frac{rS^2}{K} - \frac{rSI}{K} - 2\gamma IP - d_1I - \frac{d_2}{m}P + aS + aI + \frac{a}{m}P$$

$$\leq S\left(r + a - \frac{r}{K}S\right) - (d_1 - a)I - \left(\frac{d_2 - a}{m}\right)P \leq S\left(r + a - \frac{rS}{K}\right),$$

where $0 < a < \min\{d_1, d_2\}$. The maximum value of the expression $S\left(r + a - \frac{rS}{K}\right)$ is $\frac{K(r+a)^2}{4r}$.

Hence,

$$\frac{dW(t)}{dT} + aW(t) \le \frac{K(r+a)^2}{4r}, \quad \forall t \in (0,T).$$

Let

$$H(t,y) = \frac{K(r+a)^2}{4r} - ay,$$

which satisfies the Lipschitz condition. Clearly,

$$\frac{dW(t)}{dT} = \frac{K(r+a)^2}{4r} - aW(t) = H(t, W(t)), \quad \forall t \in (0, T).$$

Let

$$\frac{dX}{dT} = H(t, X) = \frac{K(r+a)^2}{4r} - aX$$
 and $X_0 = W(S_0, I_0, P_0)$.

The linear ordinary differential equation has a solution

$$X(t) = \frac{M}{a} (1 - e^{-at}) + X_0 e^{-at}.$$

Since X(t) is bounded on (0,T), by the comparison theorem [20],

$$W(t) \le X(t) = \frac{M}{a} (1 - e^{-at}) + W(S_0, I_0, P_0)e^{-at}, \quad \forall t \in (0, T_M).$$

Thus, for $t \to \infty$, we have $0 < W(t) < \frac{M}{a}$. Hence, all solutions are bounded uniformly on \mathbb{R}_0^+ .

For non-dimensionless system (3), we apply the transformation:

$$s = \frac{S}{K}, \quad i = \frac{I}{K}, \quad p = \frac{P}{K}, \quad T = \frac{t}{\lambda K},$$

to obtain the following dimensionless system:

$$\begin{cases}
\frac{ds}{dt} = es\left(1 - (s+i)\right) - si - \frac{n_1 sp}{s + bp + d}, \\
\frac{di}{dt} = si - wip - d_3i, \\
\frac{dp}{dt} = \frac{mn_1 sp}{s + bp + d} - mwpi - d_4p,
\end{cases} \tag{4}$$

where

$$e = \frac{r}{\lambda K}$$
, $n_1 = \frac{\alpha}{\lambda K}$, $d = \frac{c}{K}$, $w = \frac{\gamma}{\lambda}$, $d_3 = \frac{d_1}{\lambda K}$, and $d_4 = \frac{d_2}{\lambda K}$.

2.1. Equilibria

The system (4) exhibits the following equilibrium points:

• $E_0 = (0,0,0)$ and $E_1 = (1,0,0)$ always exist.

- $E_2 = \left(d_3, \frac{e(1-d_3)}{e+1}, 0\right)$ exists where $d_3 < 1$.
- $E_3 = (\bar{s}, 0, \bar{p})$ exists in the interior of \mathbb{R}^3_+ if there is a positive solution to the following set of equations:

$$e(1-\bar{s}) = \frac{n_1\bar{p}}{\bar{s} + b\bar{p} + d},\tag{5}$$

$$\frac{mn_1\bar{s}}{\bar{s} + b\bar{p} + d} = d_4. \tag{6}$$

From equation (6), we get:

$$\bar{p} = \frac{(mn_1 - d_4)\bar{s} - dd_4}{bd_4}. (7)$$

By substituting the value of \bar{p} into equation (5), a set of polynomial equations is obtained:

$$bm\bar{s}^2 + (mn_1 - bm - d_4)\bar{s} - dd_4 = 0.$$
(8)

Equation (8) has a positive root:

$$\bar{s} = \frac{(d_4 + bm - mn_1) + \sqrt{(d_4 + bm - mn_1)^2 + 4bmdd_4}}{2bm}.$$

So, $E_3 = (\bar{s}, 0, \bar{p})$ exists in the interior of \mathbb{R}^3_+ if and only if \bar{p} is given by equation (7) and \bar{s} satisfies:

$$0 < \frac{dd_4}{mn_1 - d_4} < \bar{s} < 1. \tag{9}$$

• The interior equilibrium point $E_* = (s^*, i^*, p^*)$ exists if and only if there is a positive solution to the following nonlinear equations:

$$e(1 - (s+i)) - i - \frac{n_1 p}{F} = 0, (10)$$

$$s - wp - d_3 = 0, (11)$$

$$\frac{mn_1s}{F} - mwi - d_4 = 0, (12)$$

where F = s + bp + d.

From equation (11), we get:

$$p^* = \frac{s^* - d_3}{w}.$$

Substituting the value of p^* into equation (12) yields:

$$i^* = \frac{(wmn_1 - d_4(w+b))s^* - d_4(dw - bd_3)}{mw((w+b)s^* + (dw - bd_3))}.$$

By substituting p^* and i^* into equation (10), we get a polynomial equation:

$$P(s^*) = A_1 s^{*2} + A_2 s^* + A_3,$$

where

$$A_1 = mwe(w+b),$$

$$A_2 = mwe(dw - bd_3) + (e+1)(mwn_1 - d_4(w+b) + mwn_1 - mwe(w+b)),$$

$$A_3 = (e+1)(-d_4(dw - bd_3)) - mwn_1d_3 - mwe(dw - bd_3).$$

Clearly, the equation $P(s^*) = 0$ has a positive root s^* if $dw - bd_3 > 0$. Hence, $E_* = (s^*, i^*, p^*)$ exists if:

$$d_3 < 1$$
, $dw - bd_3 > 0$, and $s^* > \max \left\{ d_3, \frac{d_4(dw - bd_3)}{mwn_1 - wd_4 - bd_4} \right\}$. (13)

2.2. Stability analysis

The general variational matrix of the system at (s, i, p) is $J = [u_{ij}]_{3\times 3}$, where:

$$\begin{split} u_{11} &= s \left(-e + \frac{n_1 p}{F^2} \right) + e (1 - s - i) - i - \frac{n_1 p}{F}, \quad u_{12} = -s (e + 1), \\ u_{13} &= \frac{-n_1 s (s + d)}{F^2}, \quad u_{21} = i, \quad u_{22} = s - w p - d_3, \quad u_{23} = -w i, \\ u_{31} &= \frac{m n_1 p (b p + d)}{F^2}, \quad u_{32} = -m w p, \quad u_{33} = \frac{-m n_1 b s p}{F^2} + \frac{m n_1 s}{F} - m w i - d_4, \end{split}$$

and F = s + bp + d.

Theorem 2. The system (4) is unstable around E_0 for all parameter values, as evidenced by the eigenvalues:

$$\lambda_1 = e > 0$$
, $\lambda_2 = -d_3 < 0$, and $\lambda_3 = -d_4 < 0$.

Theorem 3. The system (4) is locally asymptotically stable around $E_1 = (1,0,0)$, if $d_3 > 1$ and $mn_1 < (1+d)d_4$.

Proof. The eigenvalues of $J(E_1)$ are:

$$\lambda_1 = -e, \quad \lambda_2 = 1 - d_3, \quad \text{and} \quad \lambda_3 = \frac{mn_1}{1+d} - d_4.$$

Then E_1 is stable where $d_3 > 1$ and $mn_1 < (1+d)d_4$. Additionally, E_1 is globally asymptotically stable if condition (9) does not hold.

Theorem 4. The system (4) is locally asymptotically stable around $E_2 = \left(d_3, \frac{e(1-d_3)}{e+1}, 0\right)$, if:

$$n_1 < \frac{d_3 + d}{md_3} \left(\frac{mwe(1 - d_3)}{e + 1} + d_4 \right).$$
 (14)

Proof. The variational matrix about the equilibrium point $E_2 = \left(d_3, \frac{e(1-d_3)}{e+1}, 0\right)$ yields:

$$\lambda_{23} = \frac{mn_1d_3}{d_3+d} - \frac{mwe(1-d_3)}{e+1} - d_4,$$

where the eigenvalues λ_{21} and λ_{22} are roots of the polynomial equation:

$$\lambda^2 + ed_3\lambda + ed_3(1 - d_3) = 0,$$

and satisfy the following relations:

$$\lambda_{21} + \lambda_{22} = -ed_3 < 0$$
 and $\lambda_{21} \cdot \lambda_{22} = ed_3(1 - d_3) > 0$ (since $d_3 < 1$).

Thus, all eigenvalues possess negative real parts provided that condition (14) is satisfied.

Theorem 5. The system (4) is locally asymptotically stable around $E_3 = (\bar{s}, 0, \bar{p})$ if one of the following conditions holds:

$$d_3 \ge 1$$
 and $\frac{d_4}{m} < n_1 < \frac{e}{\bar{p}} (\bar{s} + b\bar{p} + d)^2$, (15)

$$d_3 < 1$$
 and $\frac{d_4}{m} \left(\frac{d}{w\bar{p} + d_3} + 1 \right) < n_1 < \frac{e}{\bar{p}} (\bar{s} + b\bar{p} + d)^2.$ (16)

Proof. By substituting E_3 in the variational matrix, we obtain $J(E_3) = [a_{ij}]_{3\times 3}$, where:

$$a_{11} = \bar{s} \left(\frac{n_1 \bar{p}}{(\bar{s} + b \bar{p} + d)^2} - e \right), \quad a_{12} = -(e+1)\bar{s} \quad (<0), \quad a_{13} = \frac{-n_1 \bar{s}(\bar{s} + d)}{(\bar{s} + b \bar{p} + d)^2} \quad (<0),$$

$$a_{22} = \bar{s} - w \bar{p} - d_3, \quad a_{31} = \frac{m n_1 \bar{p}(b \bar{p} + d)}{(\bar{s} + b \bar{p} + d)^2} \quad (>0), \quad a_{32} = -m w \bar{p} \quad (<0),$$

$$a_{33} = \frac{-b m n_1 \bar{s} \bar{p}}{(\bar{s} + b \bar{p} + d)^2} \quad (<0), \quad a_{21} = a_{23} = 0.$$

The characteristic equation associated with this variational matrix can be expressed as:

$$(\lambda - a_{22})[(\lambda - a_{11})(\lambda - a_{33}) - a_{13}a_{31}] = 0,$$

or equivalently:

$$(\lambda - a_{22})[\lambda^2 + (a_{11} + a_{33})\lambda + a_{11}a_{33} - a_{13}a_{31}] = 0.$$

The eigenvalues of $J(E_3)$ satisfy the following relations:

$$\lambda_{32} = a_{22}, \quad \lambda_{31} + \lambda_{33} = a_{11} + a_{33}, \quad \lambda_{31} \cdot \lambda_{33} = a_{11}a_{33} - a_{13}a_{31}.$$

We analyze the following cases:

- If $d_3 \geq 1$, then $\lambda_{32} = a_{22} = \bar{s} w\bar{p} d_3 < 0$ (since $\bar{s} < 1$ from (2.7)). If $n_1 < \frac{e}{\bar{p}}(\bar{s} + b\bar{p} + d)^2$, then $a_{11} < 0$, and we achieve $\lambda_{31} + \lambda_{33} < 0$ and $\lambda_{31} \cdot \lambda_{33} > 0$. Hence, for $d_3 \geq 1$ with $\frac{d_4}{m} < n_1 < \frac{e}{\bar{p}}(\bar{s} + b\bar{p} + d)^2$, E_3 exists and is locally asymptotically stable.
- If $d_3 < 1$, then $\lambda_{32} = a_{22} = \bar{s} w\bar{p} d_3 < 0$ where $\frac{dd_4}{mn_1 d_4} < \bar{s} < w\bar{p} + d_3$. We obtain $\frac{d_4}{m} \left(\frac{d}{w\bar{p} + d_3} + 1 \right) < n_1$. As in the previous case, if $n_1 < \frac{e}{\bar{p}} (\bar{s} + b\bar{p} + d)^2$, then $a_{11} < 0$, and we achieve $\lambda_{31} + \lambda_{33} < 0$ and $\lambda_{31} \cdot \lambda_{33} > 0$. Hence, for $d_3 < 1$ with $\frac{d_4}{m} \left(\frac{d}{w\bar{p} + d_3} + 1 \right) < n_1 < \frac{e}{\bar{p}} (\bar{s} + b\bar{p} + d)^2$, E_3 exists and is locally asymptotically stable.

Theorem 6. For all parameter values, the equilibrium point E_* is unstable.

Proof. By substituting E_* in the variational matrix, we obtain $J(E_*) = [b_{ij}]_{3\times 3}$, where:

$$b_{11} = s^* \left[\frac{n_1 p^*}{F^{*2}} - e \right], \quad b_{12} = -(e+1)s^* < 0, \quad b_{13} = \frac{-n_1 s^* (s^* + d)}{F^{*2}} < 0,$$

$$b_{21} = i^* > 0, \quad b_{22} = 0, \quad b_{23} = -wi^* < 0, \quad b_{31} = \frac{mn_1 p^* (bp^* + d)}{F^{*2}} > 0,$$

$$b_{32} = -mwp^* < 0, \quad b_{33} = \frac{-bmn_1 s^* p^*}{F^{*2}} < 0,$$

and $F^* = s^* + bp^* + d$.

The characteristic equation of $J(E_*)$ can be expressed as:

$$\lambda^3 + D_1\lambda^2 + D_2\lambda + D_3 = 0,$$

where:

$$D_1 = -(b_{11} + b_{33}),$$

$$D_2 = b_{11}b_{33} - b_{13}b_{31} - b_{23}b_{32} - b_{12}b_{21},$$

$$D_3 = b_{11}b_{23}b_{32} - b_{23}b_{31}b_{12} - b_{13}b_{21}b_{32} + b_{12}b_{21}b_{33}.$$

By substituting the values of b_{ij} into D_3 , we obtain:

$$D_{3} = \left(\frac{n_{1}s^{*}p^{*}}{F^{*2}} - s^{*}e\right)mw^{2}F^{*}p^{*} - \frac{mwn_{1}s^{*}(s^{*} + d)i^{*}p^{*}}{F^{*2}} + \frac{mn_{1}(e+1)s^{*}p^{*}i^{*}}{F^{*2}}\left(bs^{*} - w(bp^{*} + d)\right).$$

$$(17)$$

Since $s^* = wp^* + d_3$ and simplifying equation (17), we obtain:

$$D_{3} = -mw^{2}es^{*}i^{*}p^{*} + \frac{mwn_{1}s^{*}i^{*}p^{*}}{(F^{*})^{2}} [wp^{*} - (s^{*} + d)]$$

$$+ \frac{mn_{1}(e+1)s^{*}i^{*}p^{*}}{(F^{*})^{2}} [b(wp^{*} + d_{3}) - wbp^{*} - dw]$$

$$= -mw^{2}es^{*}i^{*}p^{*} - \frac{mwn_{1}es^{*}i^{*}p^{*}}{(F^{*})^{2}} [d_{3} + d] + \frac{mn_{1}(e+1)}{(F^{*})^{2}}s^{*}i^{*}p^{*} [bd_{3} - dw]$$

From the existence condition (13) of E_* , $bd_3 - dw < 0$. Thus, D_3 becomes negative, which violates the Routh-Hurwitz criterion for stability. Therefore, the equilibrium point E_* is unstable for all parameter values.

3. Second model analysis

In this section, the boundedness, finding equilibria, and their stability are described with respect to the Holling type IV functional response.

Eco-Epidemiological Model with Holling Type IV Functional Response

The eco-epidemiological model with Holling Type IV functional response is given by:

$$\begin{cases}
\frac{dS}{dT} = rS\left(1 - \frac{S+I}{K}\right) - \lambda IS - \frac{\alpha SP}{1 + a_1 S + a_2 S^2}, \\
\frac{dI}{dT} = \lambda IS - \gamma IP - d_1 I, \\
\frac{dP}{dT} = \frac{\alpha mSP}{1 + a_1 S + a_2 S^2} - m\gamma IP - d_2 P,
\end{cases}$$
(18)

where the system (18) is analyzed under the initial conditions S(0) > 0, I(0) > 0, and P(0) > 0.

For this functional response, we use parameters a_1 and a_2 instead of parameters b_1 and b_2 used in system (3). The rest of the parameters remain the same. The parameters a_1 and a_2 represent handling times and inhibitory effects, respectively.

Theorem 7. The solution (S(t), I(t), P(t)) is uniformly bounded for initial conditions $(S_0, I_0, P_0) \in \mathbb{R}^3_+$.

Proof. As in Theorem 1.

By applying the same transformation as in the previous model, the system (18) can be expressed in the following dimensionless form:

$$\begin{cases}
\frac{ds}{dt} = es(1 - (s + i)) - si - \frac{n_2 sp}{c_1 + c_2 s + s^2}, \\
\frac{di}{dt} = si - wip - d_3 i, \\
\frac{dp}{dt} = \frac{mn_2 sp}{c_1 + c_2 s + s^2} - mwpi - d_4 p,
\end{cases}$$
(19)

where:

$$n_2=\frac{\alpha}{\lambda a_2k^2},\quad c_1=\frac{1}{a_2k^2},\quad c_2=\frac{a_1}{a_2k},\quad e=\frac{r}{\lambda k},\quad w=\frac{\gamma}{k},\quad d_3=\frac{d_1}{\lambda k},\quad \text{and}\quad d_4=\frac{d_2}{\lambda k}.$$

3.1. Equilibria

The system (19) exhibits the following equilibrium points:

- $E'_0 = (0,0,0), E'_1 = (1,0,0)$ always exist.
- $E_2' = \left(d_3, \frac{e(1-d_3)}{e+1}, 0\right)$ exists where $d_3 < 1$.

• $E_3^{\pm} = (s_3^{\pm}, 0, p_3^{\pm})$ exist where

$$s_3^{\pm} = \frac{mn_2 - c_2d_4 \pm \sqrt{B}}{2d_4}, \quad p_3^{\pm} = \frac{em}{d_4^2} \left[(1 + c_2)d_4 - mn_2 \right] s_3^{\pm} + c_1d_4,$$

where $B = (mn_2 - c_2d_4)^2 - 4d_4^2c_1$, under the following condition:

$$\frac{d_4}{m}\left(c_2 + 2\sqrt{c_1}\right) < n_2 < \frac{d_4}{m}(1 + c_2). \tag{20}$$

• The interior equilibrium point $E'_* = (\bar{s}, \bar{i}, \bar{p})$ exists if and only if there exists a positive solution to the system:

$$e(1 - (s+i)) - i - \frac{n_2 p}{G} = 0, (21)$$

$$s - wp - d_3 = 0, (22)$$

$$s - wp - d_3 = 0,$$

$$\frac{mn_2s}{G} - mwi - d_4 = 0,$$
(22)

where $G = c_1 + c_2 s + s^2$.

From equation (21), we have $s + i \le 1$, and from equation (22), we have $\bar{p} = \frac{\bar{s} - d_3}{w}$. Then, p < 1 if:

$$\bar{s} > d_3. \tag{24}$$

From equations (21) and (23), we obtain:

$$emwG - emwG\bar{s} - (e+1)(mn_2\bar{s} - d_4G) - mwn_2\bar{p} = 0,$$
 (25)

$$mn_2\bar{s} - d_4G = mwG\bar{i}. (26)$$

By substituting equation (26) into (25), we obtain the cubic polynomial:

$$P(\bar{s}) = A_3 \bar{s}^3 - A_2 \bar{s}^2 - A_1 \bar{s} - A_0,$$

where:

$$A_0 = (emw^2 + (e+1)wd_4) c_1 + mwn_2d_3 > 0,$$

$$A_1 = w(e+1)(d_4c_2 - mn_2) + mw(ewc_2 - ewc_1 - n_2),$$

$$A_2 = emw^2(1 - c_2) + (e+1)wd_4,$$

$$A_3 = emw^2.$$

Clearly, $P(0) = -A_0 < 0$ and:

$$P(c_1) = c_1 w(e+1) \left[mn_2 - d_4 c_2 - d_4 c_1 - d_4 \right] + mw \left[ewc_1(c_1^2 + c_1 c_2 - c_2 - 1) - n_2(c_1 - d_3) \right].$$

Hence, $P(c_1) > 0$ if:

$$n_2 > \max\left\{\frac{d_4(c_2 + c_1 + 1)}{m}, \frac{ewc_1(1 + c_1 - c_1c_2 - c_1^2)}{c_1 - d_3}\right\}.$$
 (27)

Since $P(\bar{s})$ is a continuous function on $[0, c_1]$, P(0) < 0, and $P(c_1) > 0$, by the Intermediate Value Theorem, there exists \bar{s} , $0 < \bar{s} < c_1$, such that $P(\bar{s}) = 0$.

Thus, $E_* = (\bar{s}, \bar{i}, \bar{p})$ exists if conditions (24) and (27) hold.

3.2. Stability Analysis

The variational matrix of the system (19) is $J' = [u'_{ij}]_{3\times 3}$, where:

$$\begin{split} u'_{11} &= s \left[\frac{n_2 p(c_2 + 2s)}{G^2} - e \right] + e(1 - (s+i)) - i - \frac{n_2 p}{G}, \quad u'_{12} = -s(e+1) < 0, \quad u'_{13} = -\frac{n_2 s}{G} < 0, \\ u'_{21} &= i > 0, \quad u'_{22} = s - wp - d_3, \quad u'_{23} = -wi < 0, \quad u'_{31} = \frac{mn_2 p(c_1 - s^2)}{G^2}, \quad u'_{32} = -mwp < 0, \\ u'_{33} &= \frac{mn_2 s}{G} - mwi - d_4. \end{split}$$

Theorem 8. The system (19) is unstable around $E'_0 = (0,0,0)$ for all parameter values.

Proof. The eigenvalues of E_0' are $\lambda_1 = e > 0$, $\lambda_2 = -d_3 < 0$, and $\lambda_3 = -d_4 < 0$. Thus, E_0' is unstable.

Theorem 9. The system (19) is locally asymptotically stable around $E_1' = (1,0,0)$, if $d_3 > 1$ and $n_2 < \frac{d_4}{m}(1 + c_1 + c_2)$.

Proof. The eigenvalues of E_1' are $\lambda_1 = -e < 0$, $\lambda_2 = 1 - d_3$, and $\lambda_3 = \frac{mn_2}{1 + c_1 + c_2} - d_4$. Thus, E_1' is stable if $d_3 > 1$ and $n_2 < \frac{d_4}{m}(1 + c_1 + c_2)$.

Theorem 10. The system (19) is locally asymptotically stable around $E'_2 = \left(d_3, \frac{e(1-d_3)}{e+1}, 0\right)$ if and only if:

$$n_2 < \frac{d_4}{md_3}(c_1 + c_2d_3 + d_3^2).$$

Proof. From the variational matrix at E'_2 , we have the eigenvalue:

$$\lambda_3 = \frac{mn_2d_3}{c_1 + c_2d_3 + d_3^2} - \frac{emw(1 - d_3)}{e + 1} - d_4,$$

which is negative if:

$$n_2 < \frac{d_4}{md_3}(c_1 + c_2d_3 + d_3^2).$$

The other eigenvalues λ_1 and λ_2 are the roots of:

$$\lambda^2 + ed_3\lambda + ed_3(1 - d_3) = 0,$$

with $\lambda_1 + \lambda_2 = -ed_3 < 0$ and $\lambda_1\lambda_2 = ed_3(1 - d_3) > 0$. Hence, λ_1 and λ_2 are either negative real numbers or complex conjugates with negative real parts. By the Routh-Hurwitz criterion, E_2' is locally asymptotically stable.

Theorem 11. The system (19) is locally asymptotically stable around $E_3^- = (s_3^-, 0, p_3^-)$, if one of the following holds:

$$d_3 \ge 1, \quad s_3^- < \sqrt{c_1}, \quad and \quad n_2 < \frac{ep_3^-(c_2 + 2s_3^-)}{G_3^2},$$

$$d_3 < 1, \quad s_3^- < \min\{\sqrt{c_1}, wp_3^- + d_3\}, \quad and \quad n_2 < \frac{ep_3^-(c_2 + 2s_3^-)}{G_2^2}.$$

Proof. The variational matrix around $E_3^- = (s_3^-, 0, p_3^-)$ is:

$$\begin{bmatrix} s_3^- \left(\frac{n_2 p_3^- (c_2 + 2 s_3^-)}{G_3^2} - e\right) & -(e+1) s_3^- & -\frac{n_2 s_3^-}{G_3} \\ 0 & s_3^- - w p_3^- - d_3 & 0 \\ \frac{m n_2 p_3^- (c_1 - (s_3^-)^2)}{G_3^2} & -m w p_3^- & 0 \end{bmatrix},$$

where $G_3 = c_1 + c_2 s_3^- + (s_3^-)^2$. The eigenvalue in the *i*-direction is $\lambda_2 = s_3^- - w p_3^- - d_3$. and for:

$$J_{13}' = \begin{bmatrix} s_3^- \left(\frac{n_2 p_3^- (c_2 + 2 s_3^-)}{G_3^2} - e\right) & -\frac{n_2 s_3^-}{G_3} \\ \frac{m n_2 p_3^- (c_1 - (s_3^-)^2)}{G_3^2} & 0 \end{bmatrix},$$

we have:

$$\operatorname{tr}(J'_{13}) = s_3^- \left(\frac{n_2 p_3^- (c_2 + 2s_3^-)}{G_3^2} - e \right),$$
$$\det(J'_{13}) = \frac{m n_2^2 p_3^- s_3^- (c_1 - (s_3^-)^2)}{G_3^3}.$$

For $d_3 \geq 1$: We have $s_3^- < 1$, so $s_3^- < d_3$. Hence, $\lambda_2 = s_3^- - w p_3^- - d_3 < 0$. Also, $\det(J_{13}') > 0$ and $\operatorname{tr}(J_{13}') < 0$ where $s_3^- < \sqrt{c_1}$ and $n_2 < \frac{ep_3^-(c_2 + 2s_3^-)}{G_3^2}$, respectively. For $d_3 < 1$: When $s_3^- < \min\{\sqrt{c_1}, w p_3^- + d_3\}$, then $\lambda_2 = s_3^- - w p_3^- - d_3 < 0$ and $\det(J_{13}') > 0$. Moreover, if $n_2 < \frac{ep_3^-(c_2 + 2s_3^-)}{G_3^2}$, then $\operatorname{tr}(J_{13}') < 0$.

Theorem 12. The system (19) is unstable around $E_3^+ = (s_3^+, 0, p_3^+)$.

Proof. From the existence condition (20) of $E_3^+ = (s_3^+, 0, p_3^+)$,

$$mn_2 > d_4(c_2 + 2\sqrt{c_1}).$$

and:

$$s_3^+ = \frac{mn_2 - c_2d_4 + \sqrt{(mn_2 - c_2d_4)^2 - 4c_1d_4^2}}{2d_4} > \frac{mn_2 - c_2d_4}{2d_4}.$$

Thus:

$$s_3^+ > \frac{c_2 d_4 + 2d_4 \sqrt{c_1} - c_2 d_4}{2d_4} = \sqrt{c_1}.$$

Therefore, $s_3^+ > \sqrt{c_1}$, so $\det(J'_{13})$ is negative, which means that λ_1 or λ_3 is positive.

Theorem 13. The system (19) is unstable around $E'_* = (\bar{s}, \bar{i}, \bar{p})$ for all parameter values.

Proof. From the variational matrix $J'(E'_*) = [b'_{ij}]_{3\times 3}$, the characteristic polynomial corresponding to $J'(E'_*)$ is:

$$\lambda^3 + H_1\lambda^2 + H_2\lambda + H_3 = 0,$$

where:

$$H_1 = -b'_{11}, \quad H_2 = b'_{12}b'_{21} + b'_{23}b'_{32} + b'_{13}b'_{31}, \quad H_3 = b'_{11}b'_{23}b'_{32} - b'_{31}b'_{12}b'_{23} - b'_{13}b'_{21}b'_{32}.$$

And

$$b'_{11} = \bar{s} \left[\frac{n_2 \bar{p}(c_2 + 2\bar{s})}{\bar{G}^2} - e \right], \quad b'_{12} = -\bar{s}(e+1) < 0, \quad b'_{13} = -\frac{n_2 \bar{s}}{\bar{G}} < 0, \quad b'_{21} = \bar{i} > 0,$$

$$b'_{22} = 0, \quad b'_{23} = -w\bar{i} < 0, \quad b'_{31} = \frac{mn_2 \bar{p}(c_1 - \bar{s}^2)}{\bar{G}^2}, \quad b'_{32} = -mw\bar{p} < 0, \quad b'_{33} = 0,$$

where $\bar{G} = c_1 + c_2 \bar{s} + \bar{s}^2$.

Now if $b'_{11}>0$, then $H_1=-b'_{11}$ is negative, which means one of the eigenvalues is positive. This implies that the equilibrium $E'_*=(\bar s,\bar i,\bar p)$ is unstable. If $b'_{11}>0$, then the stability of $E'_*=(\bar s,\bar i,\bar p)$ depends on the sign of b'_{31} . For b'_{31} , from the existence condition of E'_* . Since $0<\bar s< c_1$ and 0< s+i<1, it follows that $\bar s<1$. Hence, $\bar s^2<\bar s$ and $\bar s^2< c_1$, which implies:

$$b_{31}' = (c_1 - \bar{s})^2 > 0.$$

Then, H_3 is negative. Thus, by the Routh-Hurwitz criterion, one of the eigenvalues of the Jacobian matrix around E'_* is positive, and therefore $E'_* = (\bar{s}, \bar{i}, \bar{p})$ is unstable.

4. Numerical simulation

Based on the conditions for existence and the analysis of stability for the equilibrium points, the parameters d_3 , n_1 , and n_2 are recognized to be important in models (4) and (19). However, a direct comparison of the dynamics between models (3) and (18) in the d_3 - n_1 and d_3 - n_2 parameter planes is not feasible. To address this, we first reformulate all the conditions outlined in the respective theorems using the original system parameters, as outlined in Table 2, and then compare the results in the α - λ parameter plane.

For both the Beddington-DeAngelis and Holling type IV functional responses, it is noted that the trivial equilibrium point E_0 invariably remains an unstable saddle for all parameter values. If $\lambda < \frac{d_1}{K}$, E_1 is asymptotically stable if $\alpha < \frac{d_2}{m} \left(1 + \frac{c}{K}\right)$ for the Beddington-DeAngelis functional response and $\alpha < \frac{d_2}{m} \left(a_2K + \frac{1}{K} + a_1\right)$ for the Holling type IV functional response.

It is important to observe that the net reproductive ratio, R_0 , is defined as $R_0 = \frac{\lambda S}{d_1}$ for each case. The number of new infections generated directly from a single infected prey is represented by R_0 . Whenever $R_0 < 1$, the disease tends to die out, whereas if $R_0 > 1$,

Equilibria	Beddington-DeAngelis	Holling Type IV
E_0	Unstable	Unstable
E_1	$\lambda < \frac{d_1}{K} \text{ and }$ $\alpha < \frac{d_2}{m} \left(1 + \frac{c}{K} \right)$	$\lambda < \frac{d_1}{K}$ and $\alpha < \frac{d_2}{m} \left(a_2 K + \frac{1}{K} + a_1 \right)$
E_2	$\lambda > \frac{d_1}{K}$ and $\alpha < \frac{d_1 + c\lambda}{md_1} \left(\frac{mr\gamma(1 - \frac{d_1}{\lambda K})}{\lambda(c + K)} + d_2 \right)$	$\lambda > \frac{d_1}{K}$ and $\alpha < \frac{d_2}{m} \left(\lambda + a_1 d_1 + \frac{a_2 d_1^2}{\lambda} \right)$
E_3	$\bullet \lambda \leq \frac{d_1}{K} \text{ and }$ $\frac{d_2}{m} < \alpha < \frac{r}{\bar{p}} (\bar{s} + b\bar{p} + \frac{c}{K})^2$ $\bullet \lambda > \frac{d_1}{K} \text{ and }$ $\frac{d_2}{m} \left(\frac{c\lambda}{\gamma K \bar{p} + d_1} + 1 \right) < \alpha < \frac{r}{\bar{p}} (\bar{s} + b\bar{p} + \frac{c}{K})^2$	With respect to E_3^- : • $\lambda \leq \frac{d_1}{K}$, $s_3^- < \frac{1}{K\sqrt{a_2}}$, and $\alpha < \frac{ra_2^2K^2p_3^-(a_1 + 2a_2Ks_3^-)}{(1/K + a_1s_3^- + a_2K(s_3^-)^2)^2}$ • $\lambda > \frac{d_1}{K}$, $s_3^- < \min\left\{\frac{1}{K\sqrt{a_2}}\right\}$, $\frac{1}{\lambda K}(\lambda \gamma p_3^- + d_1), \text{ and}$ $\alpha < \frac{ra_2^2K^2p_3^-(a_1 + 2a_2Ks_3^-)}{(1/K + a_1s_3^- + a_2K(s_3^-)^2)^2}$ With respect to E_3^+ : Unstable
E_*	Unstable	Unstable

Table 2: Stability Conditions for Equilibria

the disease persists and becomes endemic within the host population. Additionally, it is evident that the net reproductive ratio increases proportionally with the susceptible population, S. Consequently, if the basic reproductive ratio remains below 1 even at the maximum host population level K (i.e., $\frac{\lambda K}{d_1} < 1$ or $\lambda < \frac{d_1}{K}$), the infection is unable to spread within the host population. From a biological perspective, this means that when both the infection rate and the rate at which susceptible prey are encountered are sufficiently low, the infected and predator populations cannot persist. As a result, the system reaches an equilibrium where only healthy prey remains.

To validate and illustrate the observed results, we provide an example using the following fixed parameter values:

$$r = 4$$
, $K = 100$, $b = 0.8$, $c = 20$, $a_1 = -0.1$,
 $a_2 = 0.01$, $\gamma = 0.06$, $d_1 = 0.2$, $m = 0.6$, $d_2 = 0.1$, (28)

while varying only one ecological parameter, α , and one epidemiological parameter λ . Based on the specified parameter values, it is evident that the equilibrium E_1 remains

stable when α is below 0.2 for the Beddington-DeAngelis functional response and below 0.151667 for the Holling type IV functional response, provided that $\lambda < 0.002$. By setting $\lambda = 0.001$ and selecting $\alpha = 0.15$ for Beddington-DeAngelis and $\alpha = 0.1$ for Holling type IV, we observe that all trajectories originating from the initial condition (50, 50, 50) converge to the equilibrium point where the susceptible prey population S persists as a stable equilibrium. This convergence demonstrates that E_1 is globally asymptotically stable for both functional responses. Naturally, in the absence of both predation and infection, the equilibrium density of the susceptible population asymptotically approaches its carrying capacity K. The stability of E_1 is further illustrated in Figures 1 and 2 for the Beddington-DeAngelis and Holling type IV functional responses, respectively.

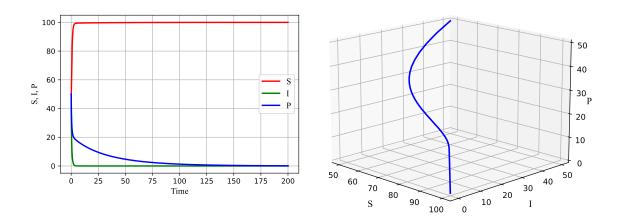


Figure 1: Stability of E_1 with Beddington-DeAngelis functional response.

For the specified set of parameter values (28), it is observed that the stability of the equilibrium E_2 requires the value of λ to exceed 0.002. By selecting $\lambda=0.008$, it is further determined that α must remain below 0.75 for the Beddington-DeAngelis functional response and below 0.00633 for the Holling type IV functional response. Consequently, with $\lambda=0.008$ and α set to 0.7 and 0.006 for the Beddington-DeAngelis and Holling type IV responses, respectively, all trajectories originating from the initial condition (50, 50, 50) converge to the predator-free equilibrium E_2 . At this equilibrium, the susceptible and infected prey populations coexist in a stable state. This convergence demonstrates that E_2 is globally asymptotically stable for both functional responses. The stability of E_2 is visually confirmed in Figures 3 and 4, which depict the dynamics for the Beddington-DeAngelis and Holling type IV functional responses, respectively.

In the case of a lower infection rate, it is observed that the stability of the equilibrium E_3 requires the value of λ to be less than 0.002. By selecting $\lambda = 0.001$, it is further determined that α should lie within the interval (0.1666, 283) for the Beddington-DeAngelis functional response and be less than 866 for the Holling type IV functional response.

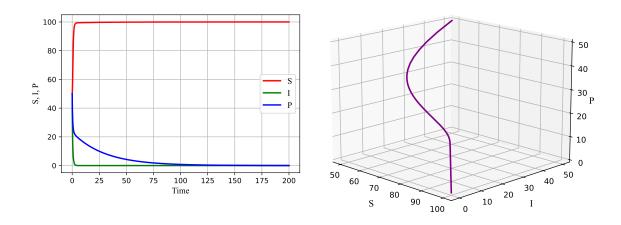


Figure 2: Stability of E_1 with Holling-type IV functional response.

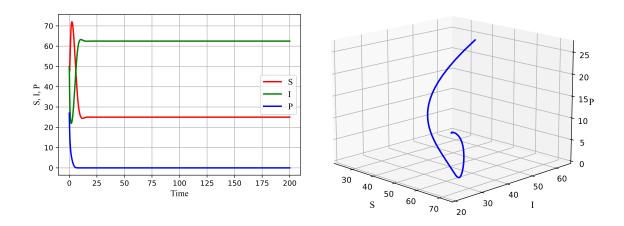


Figure 3: Stability of E_2 with Beddington-DeAngelis functional response.

Consequently, with $\lambda=0.001$ and $\alpha=0.3$, all trajectories converge to the disease-free equilibrium E_3 , where the susceptible prey and predator populations coexist in a stable state. This demonstrates that E_3 is globally asymptotically stable for both functional responses under the same infection and attack rates. The stability of E_3 is illustrated in Figures 5 and 6, which depict the dynamics for the Beddington-DeAngelis and Holling type IV functional responses, respectively.

For both functional responses, it is evident that the interior equilibrium, representing the coexistence of all three species, remains unstable across all parameter values. This

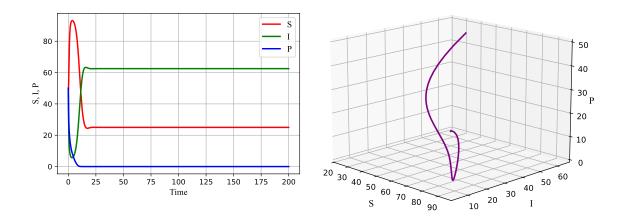


Figure 4: Stability of E_2 with Holling-type IV functional response.

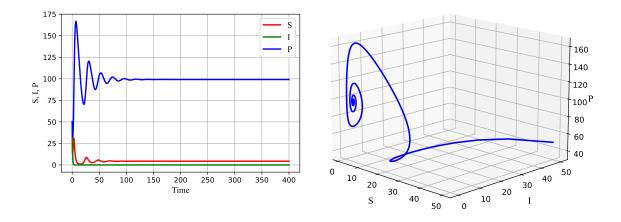


Figure 5: Stability of E_3 with Beddington-DeAngelis functional response.

equilibrium acts as a hyperbolic saddle point.

5. Conclusion

The instability of the interior equilibrium in an ecological system arises from the detrimental effects of infected prey on the predator population, which hinders their coexistence. Our model demonstrates that infection can disrupt previously stable predator-prey dynamics, while predators have the potential to disrupt otherwise stable interactions between hosts and parasites. These dynamics are critically influenced by the infection

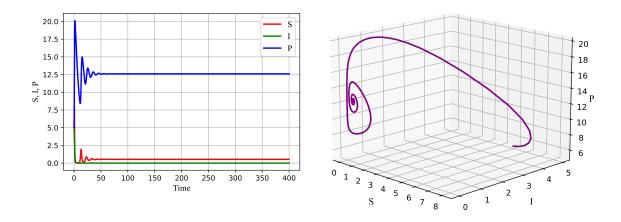


Figure 6: Stability of E_3^- with Holling-type IV functional response.

rate and the predator's attack rate on susceptible prey. The analysis demonstrates that achieving coexistence in host-parasite-predator systems, where prey is affected by a lethal disease, is not feasible. Instead, only specific configurations such as healthy, disease-free, predator-free, or oscillating disease-free systems can be attained by precisely controlling these two critical parameters. Additionally, the biological control paradox is not inherently present in eco-epidemiological models, and our framework can be extended to various host-parasite-predator systems by incorporating different functional responses.

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