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DYNAMICAL ANALYSIS OF AN ECO-EPIDEMIOLOGICAL MODEL EXPERIENCING THE CROWDING EFFECT OF INFECTED PREY

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Abstract: Most eco-epidemiological models use a bi-linear functional response, also known as the simple law of mass action, to describe the transmission of an infection. The non-linear incidence rate considers the infected individuals' crowding effect and prevents the contact rate's unboundedness by choosing suitable parameters. This paper aims to construct an Eco-Epidemiological model following the nonlinear incidence rate suggested by Gumel and Moghadas 2003. The model also offers a reasonable, realistic approach to the ecological systems in the world as we follow the Holling type II for the predator-susceptible prey interaction and the simple mass action low for the predator for the predator-infected prey interaction as the infected prey would be weak. The time for finding it would be significantly more than the time needed to catch the healthy prey. We proved the solutions' positivity and existence and our model's boundedness. The equilibrium points are determined with the feasibility conditions for each. Local stability has been analysed using Routh Hurwitz, and a Lyapunov function has been constructed to study global stability according to La Salle theorem. Different types of bifurcation are observed using Sotomayor's and Hopf theorems. The numerical analysis of the solution was carried out using fourth-order Runge-Kutta. The simulations that we performed using MATLAB 2022a supported our theoretical findings.

Keywords: predator-prey; eco-epidemiological model; nonlinear incidence rate; local stability; global stability; hopf bifurcation; transcritical bifurcation.

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1. INTRODUCTION

The dynamic relationship between predator and prey is considered one of the most fundamental relationships in ecology, as this relationship is one of the top-studied subjects by ecologists. The Lotka-Volterra system is the first fundamental prey-predator system; it goes back to the First World War [1].

The Lotka-Volterra simple mathematical model can be described as the following:

$$\frac{dS}{dT} = \alpha S - mSP$$
$$\frac{dP}{dT} = \theta mSP - dP \quad (1)$$

S: The size of prey population

P: The size of predator population

T: Time

α: Prey's growth rate

m: The predation rate

θ: Conversion factor which denotes the number of newly born predators for each killed prey

d: Predator's natural death rate

The previous system (1) and its variants were extensively studied by many researchers aiming to understand the various ways in which predator-prey interactions take place [2]–[5]. However, the representation in (1) does not explicitly incorporate the impact of transmittable illnesses on predator-prey interdependence. While valuable insights into interaction dynamics have been gleaned, the lack of consideration for infectious factors represents a limitation. Infectious diseases represent significant determinants shaping populace progression, as Research has shown that low-magnitude perturbations originating from environmental sources can induce oscillations in population levels around an average threshold. In contrast, high-intensity disturbances emanating from the surrounding habitat may result in total elimination of the population [6]. The work of [7] and [8] studied a predator-prey model where the prey population is subjected to disease, and the transmission rate of the infection follows the law of mass action. The study shows that the disease in the prey population can induce chaotic interactions.

To expand upon the classic Lotka-Volterra predator-prey model, various formulations of functional responses have been proposed which refine how consumption rates relate to prey abundance levels. One such example is the Holling type II functional response, which depicts a saturating applicable form as prey becomes more plentiful. Generalized variants of this specific

functional response have also been devised to capture additional nuances [9]–[11]. Accounting for non-linearities in the predation relationship through customized functional responses allows for more sophisticated representations of harvesting effects than the simple linear assumption of the basic Lotka-Volterra equations. Continued exploration of diverse functional responses remains an intriguing avenue for developing an increasingly refined understanding of predator-prey population dynamics.

Other studies have also explored the dynamics of a predator-prey system incorporating a disease in either the prey or the predator [12]–[14]. Nevertheless, these studies use the bilinear incidence rate to describe the transmission of the disease. Nonetheless, the bilinear incidence rate doesn't take into account the crowding effect of the infected individuals. Gumel and Moghadas [15] proposed the nonlinear incidence rate, which accounts for how the crowding effect, thereby more realistically portraying pathogen transmission dynamics as population densities fluctuate over space and time. The added nuance of factoring density effects represents an advantageous enhancement over the more straightforward law of mass action formulation for capturing important epidemiological subtleties.

Accordingly, we propose a predator-prey model that includes two key elements:

1) Holling type II functional response to describe the susceptible prey predation rate.

2) Nonlinear incidence rate for the infection transmission.

Accounting for such delays provides a more complete characterization of fundamental ecological interactions that inevitably involve consumption history effects. Incorporating time delays into our proposed predator-prey model with a nonlinear incidence rate could yield additional insights into how delays interact with and amplify the impact of other complexity features in the system. This represents a promising direction for future research to generate a highly sophisticated and representative theoretical framework,

The nonlinear incidence rate was studied in [16], where the predator is considered to consume the infected prey only, and the predation followed Crowley-Martin-type functional response.

In this study, we formulate an eco-epidemiological model characterizing the interaction between a predator population and a prey population impacted by disease. The proposed model adopts a Holling type II functional response to represent the predation dynamics between predators and susceptible prey, aiming to realistically capture prey defense behaviors that can impede consumption rates. Meanwhile, a simple mass-action scheme governs the predator-infected prey interaction, as the infection is presumed to render exposed prey less able to escape or elude capture

by predators. We assume disease transmission occurs via direct contact between susceptible and infected prey. Specifically, disease spread follows a nonlinear incidence rate first established by Gumel and Moghadas in [15] that accounts for inhibitory crowding effects among infected individuals. By integrating different functional response assumptions tied to disease state, along with density-dependent transmission dynamics, the model aims to offer a more nuanced depiction of predator-prey-pathogen interaction compared to mass-action formulations.

This paper offers a thorough analysis of the dynamics of the eco-epidemiological while still considering the crowding effect; such a model can be applied in a wide range of ecosystems where fatal diseases appear in the prey population.

2. THE MATHEMATICAL MODEL

We seek to develop an eco-epidemiological model mainly consisting of a prey and predator. The prey specie is divided into two sub-species 1) susceptible prey (S) and 2) infected prey (I). The predators are considered to be healthy at all times.

1. When there is no disease, the prey population follows the logistical growth function with a carrying capacity of $K > 0$ and a substantial growth rate constant of $\alpha > 0$.

2. the prey population consists of two groups; susceptible prey $S(t)$ and infected prey $I(t)$. The total number of preys at any given time t, denoted as $X(t)$, can be expressed as the sum of these subgroups: $X(t) = S(t) + I(t)$. Additionally, it is posited that only susceptible prey retains the capacity for reproduction, allowing their numbers to rise up to environmental carrying capacity. We also presume infected prey to be incapable of recovery, reproduction or resource competition during the afflicted stage. These assumptions aim to realistically capture the anticipated functional limitations on prey impacted by the disease factor

3. The disease transmission between susceptible and infected prey is modeled using a nonlinear incidence rate formula of the form $\frac{\beta IS}{1+I}$. This formulation was originally proposed by Gumel and Moghadas in 2003 and has since been widely adopted. In this expression, βI represents the infection force exerted by infected individuals, while the $\frac{1}{1+I}$ term accounts for inhibition caused by crowding of infected prey. This approach is deemed more realistic than solely relying on a basic bilinear rate βSI, as it considers how crowding of infected prey can hamper transmission in addition to preventing unlimited contact rates through judicious parameter selection fitting the modeled

system. By integrating density dependence effects, the incidence rate provides a reasonable depiction of disease spread dynamics versus simplistic mass action assumptions.

4. We assumed the predator consumes both the infected and susceptible prey as it is unable of identifying the infected prey from healthy.

5. We presumed that infected prey would be physically weakened by illness, making them easier targets. Accordingly, we considered that predators could immediately overtake infected prey, for simplicity, we treated the time spent by predators handling or subduing infected individuals as essentially negligible. This zero-handling time assumption resulted in the predation equation following a Volterra functional response dynamic, consistent with a Holling Type I functional response.

6. The model stipulates that the predators' consumption of the healthy(susceptible) prey is happening according to a Holling type II functional response.

The eco-epidemiological model:

$$\frac{dS}{dT} = \alpha S\left(1 - \frac{S}{k}\right) - \frac{\beta SI}{1+I} - \frac{m_1 SP}{h+S} \tag{2}$$

$$\frac{dI}{dT} = \frac{\beta SI}{1+I} - m_2 IP - d_1 I$$

$$\frac{dP}{dT} = \frac{e_1 m_1 SP}{h+S} + e_2 m_2 IP - d_2 P$$

$$S, I, P \geq 0 \tag{3}$$

$$\alpha, \beta, m_1, m_2, d_1, d_2, e_1, e_2, h > 0 \tag{4}$$

$\boldsymbol{S}$: The size of prey population

$\boldsymbol{P}$: The size of predator population

$\boldsymbol{T}$: Time

$\boldsymbol{\alpha}$: Prey's growth rate

$\boldsymbol{m_1}$: The predation rate of the susceptible prey

$\boldsymbol{m_2}$ The predation rate of the infected prey

$\boldsymbol{e_1}$: Conversion factor of the susceptible prey

$\boldsymbol{e_2}$: Conversion factor of the infected prey

$\boldsymbol{h}$: Half saturation constant

$\boldsymbol{d_1}$: Infected natural death rate

$\boldsymbol{d_2}$: Predator's natural death rate

3. THE DIMENSIONLESS FORM OF THE MODEL

In this section we seek to reduce the number of the parameters by obtaining the dimensionless form of our model making the analysis simpler

Let's assume that:

(5) $$sk = S, ik = I, pk = P, T = \frac{t}{\alpha}$$

Substituting into (2):

(6) $$\frac{ds}{dt} = s(1-s) - \frac{a_2 si}{a_3 + i} - \frac{b_1 sp}{c_1 + s}$$
$$\frac{di}{dt} = \frac{a_2 si}{a_3 + i} - b_2 ip - c_2 i$$
$$\frac{dp}{dt} = \frac{e_1 b_1 sp}{c_1 + s} + e_2 b_2 ip - c_3 p$$

Where,

(7) $$a_2 = \frac{\beta}{\alpha} > 0, a_3 = \frac{1}{k} > 0\ ,\ b_1 = \frac{m_1}{\alpha} > 0, b_2 = \frac{m_2 k}{\alpha} > 0, c_1 = \frac{h}{k} > 0\ ,\ c_2 = \frac{d_1}{\alpha} > 0, c_3 = \frac{d_2}{\alpha} > 0$$

4. POSITIVITY AND BOUNDEDNESS

In this section we aim to ensure that our model is well-posed by proving its positivity and boundness

Existence and uniqueness of the solution

Theorem 3.1. *All solutions of system* (*6*) *corresponding to the initial conditions* $s(0), i(0), p(0) \geq 0$ *exist and are unique in the period* $[0, \xi]$ *where* $0 < \xi < \infty$ *and* $s(t), i(t), p(t) \geq 0$.

$$\begin{aligned} s(0) &= s_0 \geq 0 \\ i(0) &= i_0 \geq 0 \\ p(0) &= p_0 \geq 0 \\ t &\geq 0 \end{aligned} \tag{8}$$

From (6) we write:

$$\begin{aligned} \frac{ds}{dt} &= s(1-s) - \frac{a_2 si}{a_3+i} - \frac{b_1 sp}{c_1+s} = g_1(x) \\ \frac{di}{dt} &= \frac{a_2 si}{a_3+i} - b_2 ip - c_2 i = g_2(x) \\ \frac{dp}{dt} &= \frac{e_1 b_1 sp}{c_1+s} + e_2 b_2 ip - c_3 p = g_3(x) \end{aligned} \tag{9}$$

The phase space for the system (9) is

$R_+^3 = \{(s,i,p) \in R^3 : s \geq 0, i \geq 0, p \geq 0\}$ Obviously, the functions $f_1(x), f_2(x), f_3(x)$ are continuos functions and their partial derivatives exist and are also continuous on the phase space R^3 ;therefor these functions are Lipschitzian on R_+^3 and so, the system (9) with the non-negative initial condition (8) has a unique solution on $[0, \epsilon]$, where $0 < \epsilon < \infty$ [17]. Integrating (9) with respect to initial conditions, we get

$$\begin{aligned} s(t) &= s(0)e^{\int_0^t f_1(s(x),i(x),p(x).dx} \geq 0 \\ i(t) &= i(0)e^{\int_0^t f_2(s(x),i(x),p(x).dx} \geq 0 \\ p(t) &= p(0)e^{\int_0^t f_3(s(x),i(x),p(x).dx} \geq 0 \end{aligned} \tag{10}$$

Where from (8) we have

$$\begin{aligned} s(0) &= s_0 \geq 0 \\ i(0) &= i_0 \geq 0 \\ p(0) &= p_0 \geq 0 \\ t &\geq 0 \end{aligned}$$

Which proves the theorem.

Uniformly boundedness

The boundedness suggests that our eco-epidemiological model is biologically well-behaved. The boundedness of the system (6) is guaranteed by theorem 3.2.

Theorem 3.2. *All the solutions of the system (6) which starts in R_+^3 are uniformly bounded.*

Proof: for the proof of this theorem, we study two different cases for the initial value of s

Case1:

Let $s(0) \leq 1$ and we claim $s(t) \leq 1$

We prove by contradiction; let's assume $s(t) \geq 1, then \exists\ t_1, t_2\ such\ that\ s(t_1) = 1\ and\ s(t_2) > 1\ then \forall t \in (t_1, t_2]\ we\ say\ s(t) > 1\ is\ true$

from (10) we can write,

$$s(t) = s(0)e^{\int_0^t f_1(s(x),i(x),p(x)).dx} = s(0)\left[e^{\int_0^{t_1} f_1(s(x),i(x),p(x)).dx + \int_{t_1}^t f_1(s(x),i(x),p(x)).dx}\right]$$

(11) $$s(t) = s(t_1)\left[e^{\int_{t_1}^t f_1(s(x),i(x),p(x)).dx}\right]$$

We have $s(t_1) = 1$ then (11) becomes:

(12) $$s(t) = e^{\int_{t_1}^t f_1(s(x),i(x),p(x).dx}$$

but $s(t) > 1$ as in our assumption and

(13) $$f_1\big(s(t), i(t), p(t)\big) = s(t)\big(1 - s(t)\big) - \frac{a_2 s(t) i(t)}{a_3 + i(t)} - \frac{b_1 s(t) p(t)}{c_1 + s} < 0$$

Going back to (12) we find $s(t) < 1$, contradiction.

Case 2:

Let $s(0) > 1$ and we claim $\lim_{t\to\infty} sup\, s(t) \leq 1$

Suppose it is not true then $s(t) > 1$ and $\forall\ t > 0$ and so $f_1(t) < 0$

From l_1 we have:

(14) $$\frac{ds}{dt} = s(1 - s) - \frac{a_2 si}{a_3 + i} - \frac{b_1 sp}{c_1 + s}$$

$since\ s(t) > 1$ then,

(15) $$\frac{ds}{dt} < s(1 - s) < 0$$

Integrating (15) and making $t \to \infty$ we get $s(t) < 1$ when $t \to \infty$ contradiction. Hence,

$\lim_{t\to\infty} sup\, s(t) \leq 1$

Now to show that the population sizes of s, i, p are bounded, it is enough to prove the uniformly boundness of the total population size $\Gamma = s + i + p$ for all $t \geq 0$.

(16) $$\Gamma' = s' + i' + p'\ where\ ' = \frac{d}{dt}$$

From (16):

$$\Gamma' = s(1-s) - \frac{b_1 sp}{c_1+s}(1-e_1) - b_2 ip(1-e_2) - c_2 i - c_3 p \leq s(1-s) - c_2 i - c_3 p$$

we take $\varrho = \min\{c_2, c_3\}$

$\Gamma' + \varrho\Gamma \leq s(1-s) - c_2 i - c_3 p + \varrho(s+i+p) \leq s(1-s)(a_1 s - 1) - c_2 i - c_3 p +$

$\varrho(s+i+p) \leq s[(1-s)+\varrho] - (c_2-\varrho)i - (c_3-\varrho)p \leq s[(1-s)+\varrho] \leq -s^2 + s(1+\varrho) \leq$

$1 + \varrho \; where \; 0 \leq s \leq 1$

$\Gamma' + \varrho\Gamma \leq 1 + \varrho = \theta \; constant$

$\Gamma' + \varrho\Gamma \leq \theta \implies \Gamma' \leq \theta - \varrho\Gamma$

following the help of the theory of differential inequality [18], we obtain

(17) $$0 < \Gamma < \Gamma(0)e^{-\varrho t} - \frac{\theta}{\varrho}e^{-\varrho t} + \frac{\theta}{\varrho}$$

Where $\Gamma(0)$ denotes the initial value of total population.

now when $t \to \infty$ in (17) we get $\Gamma < \frac{\theta}{\varrho}$

(18) $$\Gamma < \frac{1+\varrho}{\varrho}$$

Which indicates that the total population size $\Gamma(t)$ takes of the function f that starts with the initial value $\Gamma(0)$ at the initial time $t = 0$ is bounded by the value $\frac{1+\varrho}{\varrho}$ as the time t grows to infinity. Thus, from (17) and (18) it can be concluded that $\Gamma(t)$ is bounded as

(19) $$0 \leq \Gamma(t) \leq \frac{1+\varrho}{\varrho} .$$

As in (19), we can confirm that $\frac{1+\varrho}{\varrho}$ is an upper bound of $\Gamma(t)$. Therefore, the feasible solution for our system (6) stays in the positively invariant region Ω, where $\Omega=\{s,i,p) \in R_+^3 : \Gamma \leq \frac{1+\varrho}{\varrho} + \xi \quad \forall \xi > 0$. Thus, our system in (6) is biologically meaningful and mathematically well-posed in the domain Ω. Examining the population fluctuations within the designated theoretical parameters demonstrates the model's properties in that conceptual space proving the theorem. This verification substantiates the theoretical proposition. In essence, one may consolidate the key consequence of this proposition as the model maintains finite values for all $(t \geq 0)$.

5. EQUILIBRIUM ANALYSIS

The system (6) has the following equilibrium states:

1) The trivial equilibrium point $E_0(0,0,0)$

2) The axial equilibrium point $E_1(1,0,0)$,

3) The susceptible prey-free equilibrium point $(E_2(0,\frac{c_3}{e_2b_2},-\frac{c_2}{b_2})$

4) The disease-free equilibrium point $E_3(\bar{s},0,\bar{p}\,)$

$\bar{s}=\frac{c_3c_1}{(b_1e_1-c_3)}$

$\bar{p}=\frac{c_1e_1(e_1b_1-c_3(1+c_1))}{(b_1e_1-c_3)^2}$

5) The predator-free equilibrium points (p-free equilibrium points) $E_4(s^*,i^*,0)$

Where $s^*=\frac{c_2}{a_2}\left(a_3+the\ roots\ of\ the\ \ quadratic\ equation(v_2z^2+v_1z+v_0)\right)$

$$i^*\ is\ the\ positive\ roots\ of\ the\ \ cubic\ equation(v_2z^2+v_1z+v_0)$$

$$v_2=c_2$$
$$v_1=a_2^2-a_2+2c_2a_3$$
$$v_0=a_3^2c_2-a_3*a_2$$

6) Interior equilibrium points $\tilde{E}(\tilde{s},\tilde{\imath},\tilde{p})$

$\tilde{s}=-c_1\frac{(e_2b_2X-c_3)}{e_2b_2X+(e_1b_1-c_3)}$

$\tilde{\imath}=the\ positive\ roots\ of\ the\ equation(u_3Z^3+u_2Z^2+u_1Z+u_0)$

$\tilde{p}=-\frac{\left(e_2b_2(a_3c_2X+c_1a_2X+c_2X^2)+c_2b_1e_1(a_3+X)-c_3(a_3c_2+a_2c_1+c_2X)\right)}{b_2(e_2b_2X+(e_1b_1-c_3))(a_3+X)}$

$X=the\ roots\ of\ the\ equation(u_3Z^3+u_2Z^2+u_1Z+u_0)$

$u_3=b_2^2e_2^2c_2$

$u_2=-\left(\left(-e_1c_1^2+\left((a_2-1)e_1-a_2e_2\right)c_1-a_3c_2e_2\right)b_2-2c_2*(b_1e_1-c_3)\right)b_2e_2$

$u_1=a_3c_1e_1e_2b_2^2(c_1+1)+\Big(-b_1c_1e_1^2`(a_2-1)+\left(-c_3c_1^2+\left((a_2-1)c_3+a_2b_1e_2\right)c_1+$

$2a_3b_1c_2e_2\right)e_1-2c_3e_2*(a_2c_1+a_3c_2)\Big)b_2+c_2(b_1e_1-c_3)^2$

$u_0=\left(c_2c_3^2+e_1\left((b_1e_1-c_3)(b_1c_2+b_2c_1)-c_3b_2c_1^2\right)\right)a_3-a_2c_1c_3(b_1e_1-c_3)$

Feasibility

Obviously, the equilibria E_0,E_1 are feasible while E_2 is not feasible so there will be no need to further analyze it, E_3 is feasible under the conditions $c_3(1+c_1)<b_1e_1$

To determine the feasibility of E_4 we can say that the predator-free equilibrium points (p-free equilibrium points) $E_4(s^*, i^*, 0)$exists if and only if there is a solution where $s^*, i^* > 0$ and $p^* = 0$ to the following algebraic nonlinear system:

$$\begin{aligned} & s(1-s) - \frac{a_2 si}{a_3+i} - \frac{b_1 sp}{c_1+s} = 0 \\ & \frac{a_2 si}{a_3+i} - b_2 ip - c_2 i = 0 \\ & \frac{e_1 b_1 sp}{c_1+s} + e_2 b_2 ip - c_3 p = 0 \end{aligned} \quad (20)$$

Substituting $s = s^*, i = i^* \; and \; p = 0$ to the system (20) we get

$$\begin{aligned} & s^*(1-s^*) - \frac{a_2 s^* i^*}{a_3 + i^*} = 0 \\ & \frac{a_2 s^* i^*}{a_3 + i^*} - c_2 i^* = 0 \end{aligned} \quad (21)$$

By summing the equations in (21) we get,

$$s^*(1-s^*) - c_2 i^* = 0 \quad (22)$$

$$i^* = \frac{s^*(1-s^*)}{c_2} \quad (23)$$

But $i^* > 0$ then from (23) we can write:

$$\frac{s^*(1-s^*)}{c_2} > 0 \quad (24)$$

$$s^*(1-s^*) > 0 \quad (25)$$

Which leads to

$$0 < s^* < 1 \quad (26)$$

Therefor E_4 is feasible under the condition

$0 < s^* < 1$ &$0 < i^* < \frac{1}{4c_2}$

For the interior equilibrium point $\tilde{E}(\tilde{s}, \tilde{\imath}, \tilde{p})$ is feasible if and only if there is a solution where $\tilde{s}, \tilde{\imath}, \tilde{p} > 0$ to the following algebraic nonlinear system:

$\tilde{s}(1-\tilde{s}) - \frac{a_2 \tilde{s}\tilde{\imath}}{a_3+\tilde{\imath}} - \frac{b_1 \tilde{s}\tilde{p}}{c_1+\tilde{s}} = 0 \; \ldots (r_1)$

$\frac{a_2 \tilde{s}\tilde{\imath}}{a_3+\tilde{\imath}} - b_2 \tilde{\imath}\tilde{p} - c_2 \tilde{\imath} = 0 \quad \ldots (r_2)$

$\frac{e_1 b_1 \tilde{s}\tilde{p}}{c_1+\tilde{s}} + e_2 b_2 \tilde{\imath}\tilde{p} - c_3 \tilde{p} = 0 \quad \ldots (r_3)$

From $r_3$we get

(27) $$\tilde{\imath} = \frac{1}{e_2 b_2}\left(c_3 - \frac{e_1 b_1 \tilde{s}}{c_1 + \tilde{s}}\right)$$

When $\tilde{\imath} > 0$, if $c_3 < b_1 e_1$ then

(28) $$\tilde{s} < \frac{c_1 c_3}{(e_1 b_1 - c_3)}$$

If $c_3 > b_1 e_1$ then

(29) $$\tilde{s} > \frac{c_1 c_3}{(e_1 b_1 - c_3)}$$

From r_2 we get

(30) $$\tilde{p} = \frac{a_2 \tilde{s}}{b_2(a_3 + \tilde{\imath})} - \frac{c_2}{b_2}$$

$when\ \tilde{p} > 0\ then$

(31) $$\tilde{\imath} < \frac{a_2 \tilde{s} - a_3 c_2}{c_2}$$

Since $\frac{a_2 \tilde{s} - a_3 c_2}{c_2} > \tilde{\imath} > 0$ then,

(32) $$\tilde{s} > \frac{a_3 c_2}{a_2}$$

Therefor $\tilde{E}$ is feasible under the either the conditions $\frac{a_3 c_2}{a_2} < \tilde{s} < 1\ when\ c_3 > b_1 e_1\ \&\ 0 < \tilde{\imath} < \frac{a_2 \tilde{s} - a_3 c_2}{c_2}$ or $\frac{a_3 c_2}{a_2} < \tilde{s} < \frac{c_1 c_3}{(e_1 b_1 - c_3)}\ when\ c_3 < b_1 e_1\ \&\ 0 < \tilde{\imath} < \frac{a_2 \tilde{s} - a_3 c_2}{c_2}$.

6. LOCAL STABILITY ANALYSIS

In this section we shall discuss the local stability of the equilibrium points we determined in the previous section. We will use the notation LAS instead of the term locally asymptotically stable. The Jacobean matrix for the system (6),

$$J = \begin{pmatrix} a_{1,1} & a_{1,2} & a_{1,3} \\ a_{2,1} & a_{2,2} & a_{2,3} \\ a_{3,1} & a_{3,2} & a_{3,3} \end{pmatrix}$$

$$a_{1,1} = 1 - 2s\ - \frac{a_2 i}{a_3 + i} - \frac{b_1 p}{c_1 + s} + \frac{b_1 s p}{(c_1 + s)^2}$$

$a_{1,2} = -\frac{a_2 s}{a_3 + i} + \frac{a_2 s i}{(a_3 + i)^2}$

$a_{1,3} = -\frac{b_1 s}{c_1 + s}\ ,\ a_{2,1} = \frac{a_2 i}{a_3 + i}$

$a_{2,2} = \frac{a_2 s}{a_3 + i} - \frac{a_2 s i}{(a_3 + i)^2} - b_2 p - c_2,\ a_{2,3} = -b_2 i$

$a_{3,1} = \frac{e_1 b_1 p}{c_1+s}\left(1 - \frac{s}{c_1+s}\right),\ a_{3,2} = e_2 b_2 p$

$a_{3,3} = \frac{e_1 b_1 s}{c_1+s} + e_2 b_2 i - c_3$

Now we analyze around each equilibrium point,

Around $E_0(0,0,0)$

$J_0 = \begin{pmatrix} 1 & 0 & 0 \\ 0 & -c_2 & 0 \\ 0 & 0 & -c_3 \end{pmatrix}$, and the eigenvalues are $\lambda_1 = 1,\ \lambda_2 = -c_2,\ \lambda_3 = -c_3\ \ \lambda_1 > 0 \implies E_0$ is unstable.

Around $E_1(1,0,0)$

$J_1 = \begin{pmatrix} -1 & -\frac{a_2}{a_3} & -\frac{b_1}{c_1+1} \\ 0 & \frac{a_2}{a_3} - c_2 & 0 \\ 0 & 0 & \frac{e_1 b_1}{c_1+1} - c3 \end{pmatrix}$, and the eigenvalues are $\lambda_1 = -1,\ \lambda_2 = \frac{e_1 b_1}{c_1+1} - c_3,\ \lambda_3 = -c_2 + \frac{a_2}{a_3}$

$$\lambda_2 < 0\ if c_3 > \frac{e_1 b_1}{c_1 + 1}\ , \qquad \lambda_3 < 0\ if c_2 > \frac{a_2}{a_3}$$

$if\ c_2 > \frac{a_2}{a_3}\ \&\ c_3 > \frac{e_1 b_1}{c_1+1}\ then\ \lambda_i < 0\ \Rightarrow E_1$ is LAS.

Around $E_3(\bar{s}, 0, \bar{p}\,)$

$$J_3 = \begin{pmatrix} H_{1,1} & H_{1,2} & H_{1,3} \\ H_{2,1} & H_{2,2} & H_{2,3} \\ H_{3,1} & H_{3,2} & H_{3,3} \end{pmatrix}$$

$H_{1,1} = -(c_1(e_1 b_1 + c_3) - (e_1 b_1 - c_3))\frac{c_3}{(b_1 e_1 - c_3) b_1 e_1}$ if $0 < c_1 < \frac{e_1 b_1 - c_3}{e_1 b_1 + c_3} \ldots \rho_1$ *then* $H_{1,1} > 0$

if $c_1 > \frac{e_1 b_1 - c_3}{e_1 b_1 + c_3} \ldots \rho_2$ *then* $H_{1,1} < 0$

if $c_1 = \frac{e_1 b_1 - c_3}{e_1 b_1 + c_3} \ldots \rho_3$ *then* $H_{1,1} = 0$

$H_{1,2} = -a_2 c_3 \frac{c_1}{(b_1 e_1 - c_3) * a_3} < 0$

$H_{1,3} = -\frac{c_3}{e_1} < 0$

$H_{2,2} = \frac{a_2 c_3 (b_1 e_1 - c_3) - a_3 b_2 e_1 (b_1 e_1 - c_3(c_1+1))}{a_3 (b_1 e_1 - c_3)^2} - c_2$ if $\frac{a_2 c_3 (b_1 e_1 - c_3) - a_3 b_2 e_1 (b_1 e_1 - c_3(c_1+1))}{a_3 (b_1 e_1 - c_3)^2} > c_2 \ldots \rho_4$

then, $H_{2,2} > 0$ if $\frac{a_2 c_3 (b_1 e_1 - c_3) - a_3 b_2 e_1 (b_1 e_1 - c_3(c_1+1))}{a_3 (b_1 e_1 - c_3)^2} < c_2 \ldots \rho_5$ then $H_{2,2} < 0$

$H_{3,1} = \frac{e_1 b_1 - c_3(c_1+1)}{b_1} > 0$

$H_{3,2} = e_2 b_2 c_1 e_1 \frac{b_1 e_1 - c_3(c_1+1)}{(b_1 e_1 - c_3)^2} > 0$

$H_{2,1} = H_{2,3} = H_{3,3} = 0$

And the characteristic equation is

$\lambda^3 + L_2\lambda^2 + L_1\lambda + L_0 = 0$

$L_2 = -(H_{1,1} + H_{2,2} + H_{3,3}) = -(H_{1,1} + H_{2,2})$

$L_1 = H_{1,1}H_{2,2} + H_{1,1}H_{3,3} + H_{2,2}H_{3,3} - H_{1,2}H_{2,1} - H_{2,3}H_{3,2} - H_{1,3}H_{3,1} = H_{1,1}H_{2,2} - H_{1,3}H_{3,1}$

$L_0 = H_{1,3}H_{3,1}H_{2,2} + H_{1,2}H_{2,1}H_{3,3} + H_{1,1}H_{2,3}H_{3,2} - H_{1,3}H_{2,1}H_{3,2} - H_{1,1}H_{2,2}H_{3,3} - H_{1,2}H_{3,1}H_{2,3}$
$= H_{1,3}H_{3,1}H_{2,2}$

According to Routh Hurwitz Stability Criteria E_3 is LAS if $L_2, L_0 > 0$ and $L_1L_2 > L_0$

If $L_0 > 0$ then $H_{1,3}H_{3,1}H_{2,2} > 0$

$H_{1,3} < 0 \; and \; H_{3,1} > 0 \; then \; H_{2,2} < 0 \; ... (\eta_1)$ which means that ρ_5 should be satisfied.

If $L_2 > 0$ and taking (η_1) into account then $H_{1,1} + H_{2,2} < 0 \; ... (\eta_2)$ which means either ρ_5 and ρ_2 are satisfied or ρ_5, ρ_1 and $H_{1,1} < H_{2,2}$

If $L_1L_2 > L_0$ then $-(H_{1,1}H_{2,2} - H_{1,3}H_{3,1})(H_{1,1} + H_{2,2}) > H_{1,3}H_{3,1}H_{2,2} \Rightarrow$

$-H_{1,1}H_{2,2}(H_{1,1} + H_{2,2}) + H_{1,3}H_{3,1}H_{1,1} > 0 \Rightarrow H_{1,1}\left(H_{1,3}H_{3,1} - H_{2,2}(H_{1,1} + H_{2,2})\right) > 0 \; ... (\eta_3)$

Using the conditions $(\eta_1), (\eta_2)$ in (η_3) we get that (η_3) is satisfied only when $H_{1,1} < 0$ which means if ρ_5 and ρ_2 are satisfied then E_3 is LAS

Around $E_4(s^*, i^*, 0)$

$$J_4 = \begin{pmatrix} W_{1,1} & W_{1,2} & W_{1,3} \\ W_{2,1} & W_{2,2} & W_{2,3} \\ W_{3,1} & W_{3,2} & W_{3,3} \end{pmatrix}$$

$W_{1,1} = 1 - 2s^* - \frac{a_2 i^*}{a_3 + i^*}$

$W_{1,2} = -\frac{a_2 s^*}{a_3 + i^*} + \frac{a_2 s^* i^*}{(a_3 + i^*)^2} < 0 \;,\; W_{1,3} = -\frac{b_1 s^*}{c_1 + s^*} < 0 \;\; W_{2,1} = \frac{a_2 i^*}{a_3 + i^*} > 0$

$W_{2,2} = \frac{a_2 s^*}{a_3 + i^*} - \frac{a_2 s^* i^*}{(a_3 + i^*)^2} - c_2, \;\; W_{2,3} = -b_2 i^* < 0$

$W_{3,1} = 0, \;\; W_{3,2} = 0, \;\; W_{3,3} = \frac{e_1 b_1 s^*}{c_1 + s^*} + e_2 b_2 i^* - c_3$

$L_2 = -(W_{1,1} + W_{2,2} + W_{3,3})$

$L_1 = W_{1,1}W_{2,2} + W_{1,1}W_{3,3} + W_{2,2}W_{3,3} - W_{1,2}W_{2,1} - W_{2,3}W_{3,2} - W_{1,3}W_{3,1} = W_{1,1}W_{2,2} + W_{1,1}W_{3,3} + W_{2,2}W_{3,3} - W_{1,2}W_{2,1}$

$L_0 = W_{1,3}W_{3,1}W_{2,2} + W_{1,2}W_{2,1}W_{3,3} + W_{1,1}W_{2,3}W_{3,2} - W_{1,3}W_{2,1}W_{3,2} - W_{1,1}W_{2,2}W_{3,3} - W_{1,2}W_{3,1}W_{2,3} = W_{1,2}W_{2,1}W_{3,3} - W_{1,1}W_{2,2}W_{3,3}$

According to Routh Hurwitz Stability Criteria E_4 is LAS if $L_2, L_0 > 0$ and $L_1L_2 > L_0$

If $L_2 > 0$ then $W_{1,1} + W_{2,2} + W_{3,3} < 0 \ldots (\rho_1)$

If $L_0 > 0$ then $W_{1,2}W_{2,1}W_{3,3} - W_{1,1}W_{2,2}W_{3,3} > 0 \ldots (\rho_2)$

If $L_1L_2 > L_0$ then $-(W_{1,1} + W_{2,2} + W_{3,3})(W_{1,1}W_{2,2} + W_{1,1}W_{3,3} + W_{2,2}W_{3,3} - W_{1,2}W_{2,1}) > W_{1,2}W_{2,1}W_{3,3} - W_{1,1}W_{2,2}W_{3,3} \ldots (\rho_3)$

E_4 is LAS with the conditions $(\rho_1), (\rho_2), (\rho_3)$.

Around $\tilde{E}(\tilde{s}, \tilde{\imath}, \tilde{p})$

$$\tilde{J} = \begin{pmatrix} T_{1,1} & T_{1,2} & T_{1,3} \\ T_{2,1} & T_{2,2} & T_{2,3} \\ T_{3,1} & T_{3,2} & T_{3,3} \end{pmatrix}$$

$T_{1,1} = 1 - 2\tilde{s} - \frac{a_2\tilde{\imath}}{a_3+\tilde{\imath}} - \frac{b_1\tilde{p}}{c_1+\tilde{s}} + \frac{b_1\tilde{s}\tilde{p}}{(c_1+\tilde{s})^2}$

$T_{1,2} = -\frac{a_2\tilde{s}}{a_3+i^*} + \frac{a_2\tilde{s}\tilde{\imath}}{(a_3+\tilde{\imath})^2} < 0, T_{1,3} = -\frac{b_1\tilde{s}}{c_1+\tilde{s}} < 0$

$T_{2,1} = \frac{a_2\tilde{\imath}}{a_3+\tilde{\imath}} > 0$

$T_{2,2} = \frac{a_2\tilde{s}}{a_3+\tilde{\imath}} - \frac{a_2\tilde{s}\tilde{\imath}}{(a_3+\tilde{\imath})^2} - b_2\tilde{p} - c_2$ *substituting the value of* $\tilde{p}$ *from* (r_2) *we get* $T_{2,2} = -\frac{a_2\tilde{s}\tilde{\imath}}{(a_3+\tilde{\imath})^2} < 0$, $T_{2,3} = -b_2\tilde{\imath} < 0$

$T_{3,1} = \frac{e_1b_1\tilde{p}}{c_1+\tilde{s}}\left(1 - \frac{\tilde{s}}{c_1+\tilde{s}}\right) > 0, T_{3,2} = e_2b_2\tilde{p} > 0$

$T_{3,3} = \frac{e_1b_1\tilde{s}}{c_1+\tilde{s}} + e_2b_2\tilde{\imath} - c_3$

$L_2 = -(T_{1,1} + T_{2,2} + T_{3,3})$

$L_1 = T_{1,1}T_{2,2} + T_{1,1}T_{3,3} + T_{2,2}T_{3,3} - T_{1,2}T_{2,1} - T_{2,3}T_{3,2} - T_{1,3}T_{3,1}$

$L_0 = T_{1,3}T_{3,1}T_{2,2} + T_{1,2}T_{2,1}T_{3,3} + T_{1,1}T_{2,3}T_{3,2} - T_{1,3}T_{2,1}T_{3,2} - T_{1,1}T_{2,2}T_{3,3} - T_{1,2}T_{3,1}T_{2,3}$

According to Routh Hurwitz Stability Criteria E_4 is LAS if $L_2, L_0 > 0$ *and* $L_1L_2 > L_0$.

7. BIFURCATION ANALYSIS

In this section we aim to explore some of the bifurcations that might occur in our system (6) using two theories 1) Sotomayor's theorem [19] and 2) the Hopf Bifurcation Theorem [1]. To apply

Sotomayor's theorem, we must first verify that one of the eigenvalues associated with the Jacobian matrix evaluated at a bifurcation equilibrium point is equal to zero.

Let $V = (v_1, v_2, v_3)^T \; and \; U = (u_1, u_2, u_3)^T$ represent the eigenvectors of the Jacobian J and its transpose J^T, respectively, calculated at the equilibrium point under examination.

Let $f = (f_1, f_2, f_3)^T$ where,

$f_1 = s(1-s) - \frac{a_2 si}{a_3+i} - \frac{b_1 sp}{c_1+s}$

$f_2 = \frac{a_2 si}{a_3+i} - b_2 ip - c_2 i$

$f_3 = \frac{e_1 b_1 sp}{c_1+s} + e_2 b_2 ip - c_3 p$

Theorem 7.1. System (6) experience a transcritical bifurcation with respect to the bifurcation parameter b_1 around $E_1(1,0,0)$ if, $b_1 = \frac{c_3(c_1+1)}{e_1} = b_{1_{[TC1]}}$ keeping the following condition,

$c_2 > \frac{a_2}{a_3}$

Proof.

For $E_1(1,0,0)$ we have the following eigenvalues, are $\lambda_1 = -1,\ \lambda_2 = \frac{a_2}{a_3} - c_2,\ \lambda_3 = \frac{e_1 b_1}{c_1+1} - c_3$

$$J_1 = \begin{pmatrix} -1 & -\frac{a_2}{a_3} & -\frac{b_1}{c_1+1} \\ 0 & \frac{a_2}{a_3} - c_2 & 0 \\ 0 & 0 & \frac{e_1 b_1}{c_1+1} - c3 \end{pmatrix}$$

If we take $b_1 = \frac{c_3(c_1+1)}{e_1}$ the eigenvalues can be written as, are $\lambda_1 = -1,\ \lambda_2 = \frac{a_2}{a_3} - c_2,\ \lambda_3 = 0$ and the Jacobean matrix can be written as,

$$J_1 = \begin{pmatrix} -1 & -\frac{a_2}{a_3} & -\frac{b_1}{c_1+1} \\ 0 & \frac{a_2}{a_3} - c_2 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

Now the eigenvector corresponding to $\lambda = 0$ for $J_1;\ \lambda * V = J * V$

$$V = \left(1, 0, -\frac{(c_1+1)}{b_1}\right)^T$$

the eigenvector corresponding to $\lambda = 0$ for $J_1^T;\ \lambda * U = J^T * U$

$$U = (0,0,1)^T$$

Let's calculate f_{b_1};

$$f_{b_1}\left(E_1, b_{1_{[TC_1]}}\right) = \begin{Bmatrix} 0 \\ 0 \\ 0 \end{Bmatrix}$$

And then we can write,

(33) $$\Omega_1 = W^T * f_{b_1}\left(E_1, b_{1_{[TC_1]}}\right) = 0$$

(34) $$\Omega_2 = W^T * \left[Df_{b_1}\left(E_1, b_{1_{[TC_1]}}\right)V\right] = \frac{-e_1}{b_1} \neq 0$$

(35) $$\Omega_3 = W^T * \left[Df_{b_1}\left(E_1, b_{1_{[TC_1]}}\right)(V,V)\right] = -2\frac{c_1 e_1}{(c_1+1)} \neq 0$$

From (33), (34) and (35) and according to Sotomayor's theory a transactional bifurcation occurs at $E_1(1,0,0)$ for $b_1 = b_{1_{[TC_1]}}$.

Hopf Bifurcation at E_3

To study the Hopf bifurcation at E_3 we will first assume that D is a bifurcation parameter for some system with the following characteristic equation corresponding to some equilibrium point say $E(s,i,p)$ of the system (6) is

(36) $$\lambda^3 + G_1(D)\,\lambda^2 + G_2(D)\lambda + G_3 = 0 \ldots$$

We can now state the Hopf Bifurcation Theorem as it applies to our analysis:

Theorem 7.2 (Hopf Bifurcation Theorem) [1]. Suppose functions $C_1(B), C_2(B), C_3(B)$ are continuous with respect to parameter B within a neighborhood $N_q(D_0)\ of\ D_0 \in R$, where $D > 0$. If the characteristic equation (36) exhibits:

i) A complex-conjugate pair of eigenvalues $\lambda = k(D) + il(D)(with\ k(D), l(D) \in R)$ such that they turn into purely imaginary eigenvalues at $D = D_0$ and $\frac{dk}{dD}|_{D=D_0} = 0$

ii) the remaining eigenvalue is negative at $D = D_0$, will occur around equilibrium point E at $D = D_0$.

Theorem 7.3. Provided the disease-free equilibrium point $E_3(\bar{s}, 0, \bar{p})$ satisfies the biological constraints of positivity, a simple Hopf bifurcation will emerge around around the equilibrium point E_3 at $c_1 = c_{H1} = \frac{e_1 * b_1 - c_3}{(e_1 * b_1 + c_3)} = 1 - \frac{2c_3}{(e_1 * b_1 + c_3)}$ on the condition that c_{H1} is positive.

Proof.

One of J_3 eigenvalue is represented as

$\frac{a_2c_3(b_1e_1-c_3)-a_3b_2e_1(b_1e_1-c_3(c_1+1))}{a_3(b_1e_1-c_3)^2} - c_2 < 0$ according to the local stability condition (η_1)

While the remaining two eigenvalues are the solutions of a quadratic equation of the form:

(37) $$\left(\lambda^2 - H_{1,1}\lambda - H_{1,3}H_{3,1}\right) = 0$$

Consider:

$$\tilde{J}_3 = \begin{pmatrix} H_{1,1} & H_{1,3} \\ H_{3,1} & 0 \end{pmatrix}$$

1) $tr\left(\tilde{J}_3|_{c_1=c_{H1}}\right) = 0$

2) $det\left(\tilde{J}_3|_{c_1=c_{H1}}\right) = \frac{c_3}{e_1}\frac{e_1*b_1-c_3*(c_1+1)}{b_1} > 0$

3) When $(c_1 = c_{H1})$ the characteristic equation (37) becomes $\lambda^2 + det\left(\tilde{J}_3|_{c_1=c_{H1}}\right) = 0$ with purely imaginary roots.

4) $\frac{d}{dc_1}tr(\tilde{J}_3)|_{c_1=c_{H1}} = (e_1 * b_1 + c_3) * \frac{c_3}{(b_1*e_1-c_3)*b_1*e_1} \neq 0$

Therefore, we can say that the Hopf bifurcation theorem conditions are all satisfied. Hence the Theorem 7.3 is proved.

8. GLOBAL STABILITY

Through this section we will discuss the global asymptotically stability (GAS) of the equilibrium points which we proved their locally asymptotically stability in section (5).

Theorem 8.1. If $E_1(1,0,0)$ is LAS then it is Globally asymptotically stable (GAS) in Π_1:

$$\Pi_1 = \{(s,i,p) \in R^3_+, where\ c_3 > \frac{b_1}{c_1+1}\}$$

Proof.

We took a Lyapunov function used by many other researchers such as [20], [21] to analyse the global stability of various forms predator-prey models, and modified it to fit our model as the following:

$$L_1(s,i,p) = (s - 1 - \ln s) + i + p$$

Here $L_1(s,i,p)$, is a positive definite function for all (s,i,p) other than (1,0,0)

Computing the time derivative of L along the solutions of the system (6) will give us;

$$\frac{dL_1}{dt} = \frac{s-1}{s}\left(s(1-s) - \frac{a_2si}{a_3+i} - \frac{b_1sp}{c_1+s}\right) + \frac{a_2si}{a_3+i} - b_2ip - c_2i + \frac{e_1b_1sp}{c_1+s} + e_2b_2ip - c_3p =$$

$$s(1-s) - \frac{a_2si}{a_3+i} - \frac{b_1sp}{c_1+s} - (1-s) + \frac{a_2i}{a_3+i} + \frac{b_1p}{c_1+s} + \frac{a_2si}{a_3+i} - b_2ip - c_2i + \frac{e_1b_1sp}{c_1+s} + e_2b_2ip - c_3p =$$

$-(1-s)^2+\left(\frac{a_2}{a_3+i}-c_2\right)i+\frac{b_1sp}{c_1+s}(e_1-1)+b_2ip(e_2-1)+\left(\frac{b_1}{c_1+s}-c_3\right)p$

in Π_1 we have $c_3>\frac{b_1}{c_1+1}$

And from the local stability conditions we have $c_2>\frac{a_2}{a_3}>\frac{a_2}{a_3+i}$

We get $\frac{dL_1}{dt}<0$

Also $L_1(1,0,0)=0$. As this equilibrium point $E_1=(1,0,0)$ is the lone solution to model (6) satisfying the condition $s=1$, LaSalle's invariance principle [22] entails GAS.

Theorem 8.2. If $E_3(\bar{s},0,\bar{p})$ exists and is LAS then it is Globally asymptotically stable (GAS) in Π_3:

$\Pi_3=\{(s,i,p)\in R_+^3: 0<s<\frac{c_1c_3}{(b_1e_1-c_3)}, p<\frac{c_1e_1(e_1b_1-c_3(1+c_1))}{(b_1e_1-c_3)^2}, A\frac{a_2\bar{s}}{a_3}<c_2\}$

Proof.

Let us consider a suitable Lyapunov function

$L_3(s,i,p)=A\left(s-\bar{s}-\bar{s}\ln\frac{s}{\bar{s}}\right)+i+B\left(p-\bar{p}-\bar{p}\ln\frac{p}{\bar{p}}\right)$, Where $A>1, B=\frac{1}{e_2\bar{p}}$

L_3 is obviously positive definite and continuous on Π_3

Furthermore, $\frac{dL_3}{dt}=A\left(1-\frac{\bar{s}}{s}\right)\left(s(1-s)-\frac{a_2si}{a_3+i}-\frac{b_1sp}{c_1+s}\right)+\frac{a_2si}{a_3+i}-b_2ip-c_2i+$

$B\left(1-\frac{\bar{p}}{p}\right)\left(\frac{e_1b_1sp}{c_1+s}+e_2b_2ip-c_3p\right)=$

$A(s-\bar{s})(1-s)-A\frac{a_2si}{a_3+i}-A\frac{b_1ps}{c_1+s}+A\frac{a_2\bar{s}i}{a_3+i}+A\bar{s}\frac{b_1p}{c_1+s}+\frac{a_2si}{a_3+i}-b_2ip-c_2i+B(p-\bar{p})e_2b_2i+$

$B(p-\bar{p})\left(\frac{(e_1b_1-c_3)s-c_3c_1}{c_1+s}\right)=$

$A(s-\bar{s})(1-s)+\frac{a_2si}{a_3+i}(1-A)+\frac{b_1p}{c_1+s}(\bar{s}-s)+i\left(A\frac{a_2\bar{s}}{a_3+i}-c_2\right)+b_2i(B(p-\bar{p})e_2-p)+$

$B(p-\bar{p})\left(\frac{(e_1b_1-c_3)s-c_3c_1}{c_1+s}\right)<A(s-\bar{s})(1-s)+\frac{a_2si}{a_3+i}(1-A)+\frac{b_1p}{c_1+s}(\bar{s}-s)+$

$i\left(A\frac{a_2\bar{s}}{a_3+i}-c_2\right)+b_2i\left(\frac{p(1-\bar{p})}{\bar{p}}-1\right)+Bs(p-\bar{p})\left(\frac{e_1b_1-c_3(1+c_1)}{c_1+s}\right)$

In Π_3 we have $p<\bar{p},\ s<\bar{s}, A\frac{a_2\bar{s}}{a_3}<c_2$

Since $p<\bar{p}\Rightarrow\frac{p}{\bar{p}}<1$ and $1-\bar{p}<1$ then $\frac{p(1-\bar{p})}{\bar{p}}<1$

And from the feasibility conditions we have $c_3(1+c_1)<b_1e_1$

We get $\frac{dL_1}{dt} < 0$

Also $L_3(\bar{s}, 0, \bar{p}) = 0$. As this equilibrium point $E_3(\bar{s}, 0, \bar{p})$ is the lone solution to model (6) satisfying the condition $s = \bar{s}$ and $p = \bar{p}$, LaSalle's invariance principle [22] entails GAS.

9. NUMERICAL SIMULATION

We carried the numerical simulation using fourth-order Runge-Kutta subjected to the positive initial conditions $s(0) = s_0,\ i(0) = i_0,\ p(0) = p_0$ using MATLAB R2022a. we executed numerical simulations to verify our analytic theoretical findings with a hypothetical, biologically set of parameters:

$(a_2 = 0.02, a_3 = 0.002, b_1 = 1.1, b_2 = 500, c_1 = 0.1, c_2 = 11, c_3 = 0.4, e_1 = 0.35, e_2 = 0.5)$, with the initial conditions $(s_0 = 0.65, i_0 = 0.4, p_0 = 0.2)$, and then varying some of the parameters' value according to the feasibility, LAS and GAS conditions of each feasible equilibrium point we have.

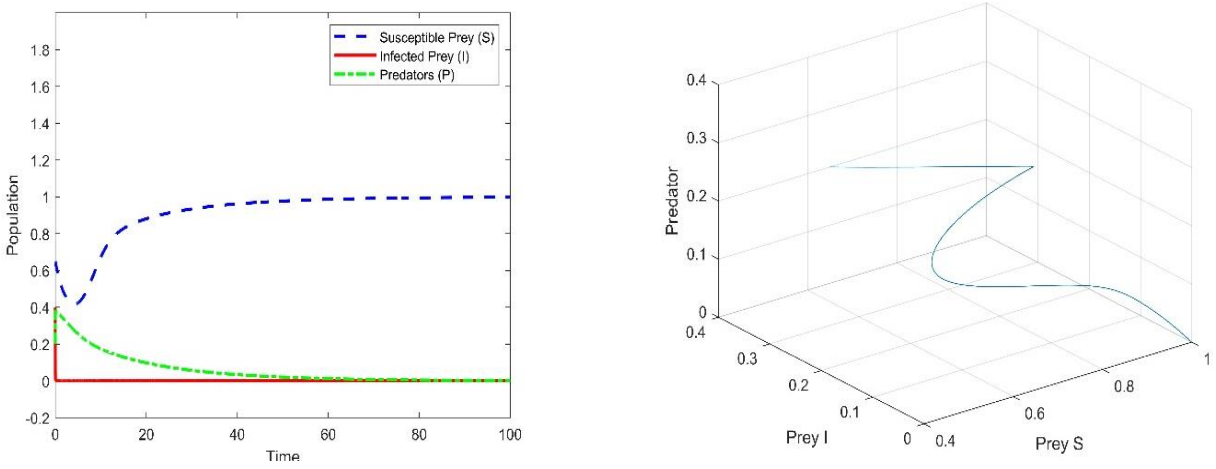

Fig. 1. Globally stable behaviour of E_1
$(a_2 = 0.02, a_3 = 0.002, b_1 = 1.1, b_2 = 500, c_1 = 0.1, c_2 = 11, c_3 = 0.4, e_1 = 0.35, e_2 = 0.5)$

For the mentioned set of parameters, we notice that the trajectories start from (0.65,0.4,0.2) and converges to the equilibrium point in which only the susceptible prey survives while the infected prey and the predator wash out of the system, demonstrating a stable equilibrium.

We can notice that while the infected prey is still in the system the predator population increases but, as soon as the infected prey population is about to wash out of the system the predator

population number starts dropping until it washes out of the system as well a while after the extinction of the infected prey. Furthermore, the susceptible prey grows and reaches its stable state a while before the extinction of the predator (see Fig 1). When the predation rate of the susceptible prey b_1 crosses the value $(b_1 = 1.257142857142857)$ where E_1 loses its stability and undergoes a transcritical bifurcation (see Fig 2)

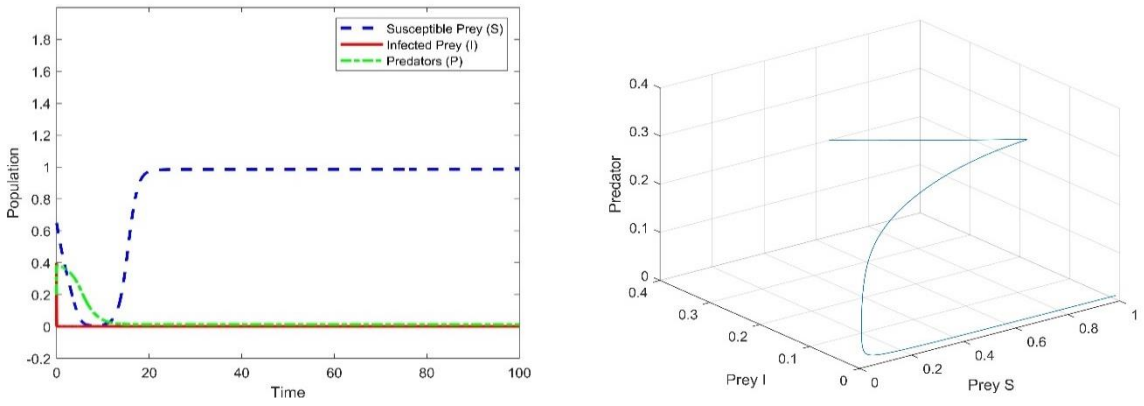

Fig. 2. E_1, E_3 changing stability for $b_1 = 1.257142857142857$

As a result of the transcritical bifurcation that the system undergoes E_1, E_3 change their stability statues for $b_1 = 1.257142857142857$ where the system converges toward a stable state around E_3 for $b_1 > b_{1_{TC}}$ where both the predator and the susceptible prey survives and the infected prey is washed out of the system (see Fig. 3). When we raise the value of b_1 for a certain value say $b_1 > 1.4$ the system goes into unstable state and a limit cycle is born as we can see in fig.4.

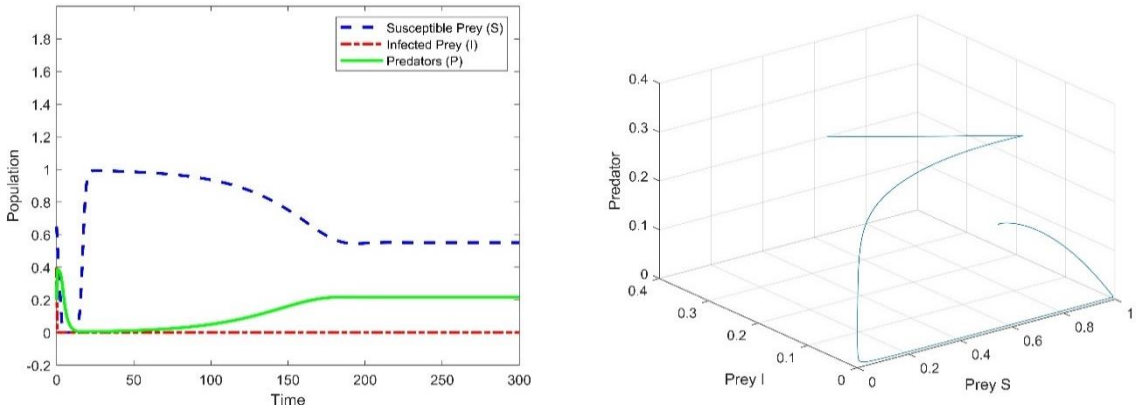

Fig. 3. E_3 Gaining stability for $b_1 = 1.35 > b_{1_{TC}}$

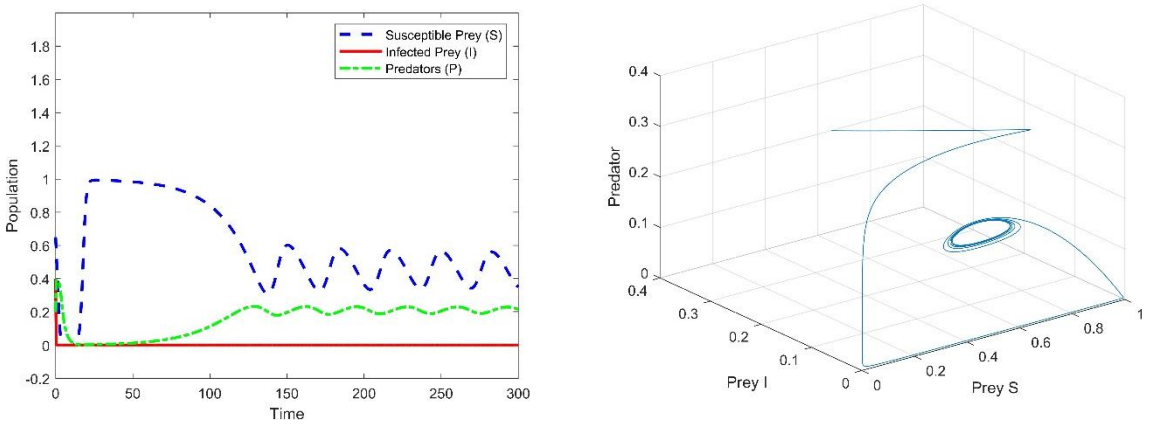

Fig. 4. Unstable behaviour for Gaining stability for $b_1 = 1.4$

Figure 5, show that the equilibrium point E_3 is stable with parameters' values of $(a_2 = 0.02, a_3 = 0.002, b_1 = 0.35, b_2 = 500, c_1 = 0.102, c_2 = 0.3, c_3 = 0.1, e_1 = 0.35, e_2 = 0.5)$, with the initial conditions $(s_0 = 0.65, i_0 = 0.4, p_0 = 0.2)$. The previous parameters' values satisfy the stability conditions of E_3. In the absences of the infected prey, the system experiences a Hopf bifurcation as the parameter c_1 crosses the critical value $c_1 = c_{H1} = 0.101123595$ (see Figure 6) and a limit cycle is born around E_3, (see Figure 7).

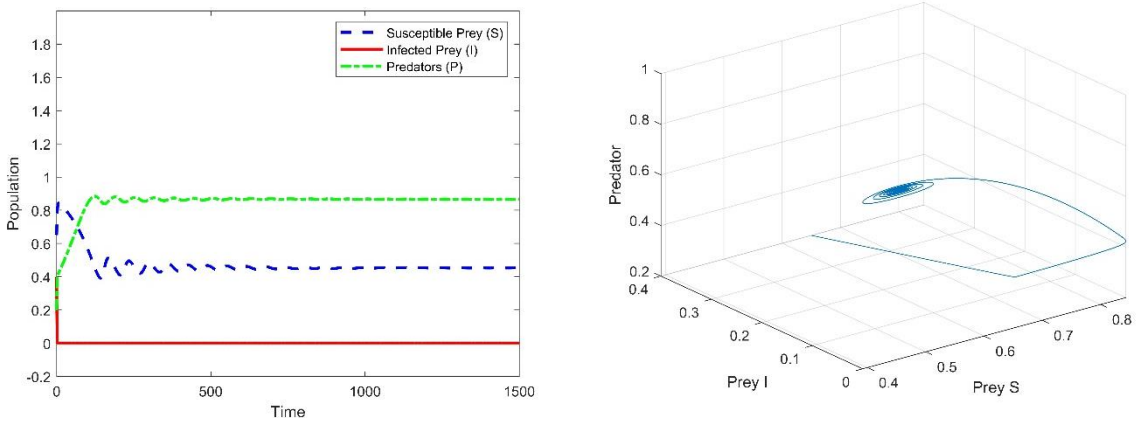

Fig. 5. Globally stable behaviour of E_3
$(a_2 = 0.02, a_3 = 0.002, b_1 = 0.35, b_2 = 500, c_1 = 0.102, c_2 = 0.3, c_3 = 0.1, e_1 = 0.35, e_2 = 0.5)$

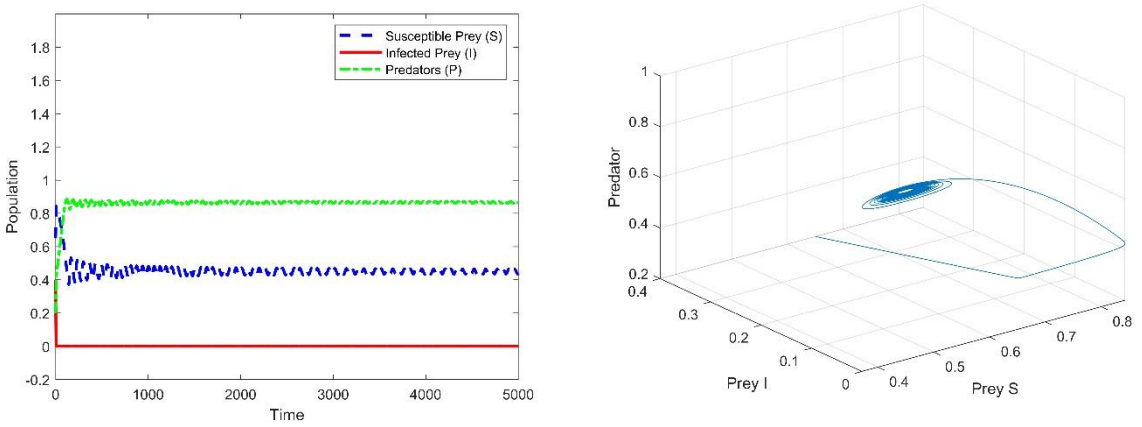

Fig. 6. Hopf bifurcation in E_3 for $c_1 = c_{H1} = 0.101123595$

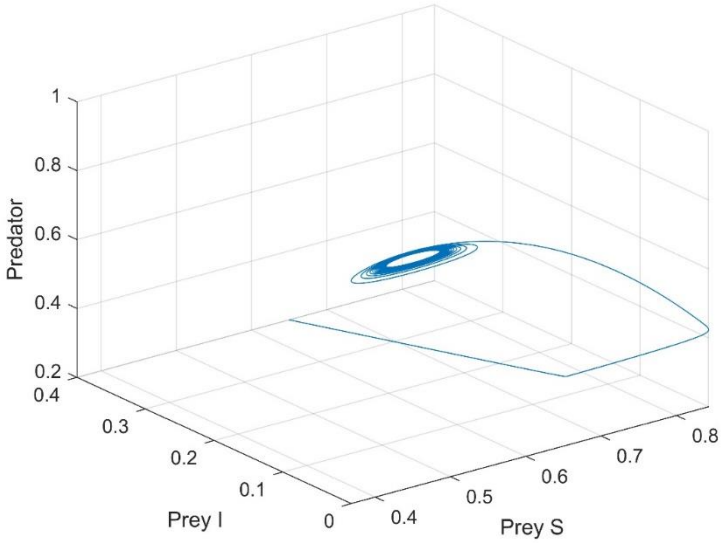

Fig. 7. limit cycle around E_3 for $c_1 = 0.101$

Figure 8, show that the equilibrium point E_4 is stable with parameters' values of $(a_2 = 1, a_3 = 0.002, b_1 = 0.35, b_2 = 3.5, c_1 = 0.1, c_2 = 0.2, c_3 = 0.4, e_1 = 0.35, e_2 = 0.5)$, with the initial conditions $(s_0 = 0.65, i_0 = 0.4, p_0 = 0.2)$.

The previous parameters' values satisfy the stability conditions of E_4. The susceptible prey survives the system for the previous parameters' values and achieve a stable state at $s \cong 0.2$ for $t = 125$, shortly before the predator washes out (See Figure. 9)

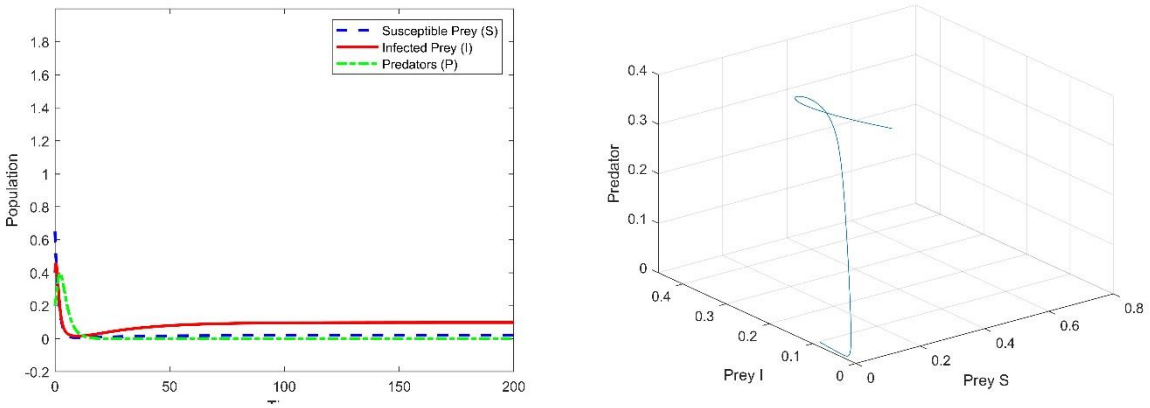

Fig. 8. Stable behaviour of E_4
$(a_2 = 1, a_3 = 0.002, b_1 = 0.35, b_2 = 3.5, c_1 = 0.1, c_2 = 0.2, c_3 = 0.4, e_1 = 0.35, e_2 = 0.5)$

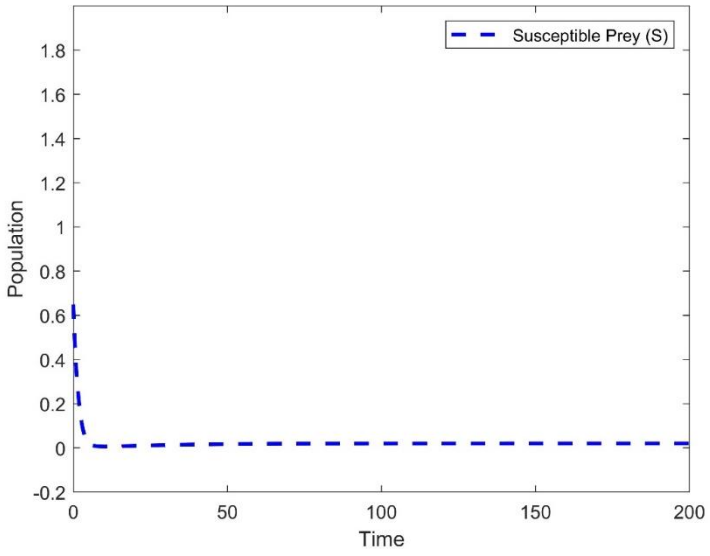

Fig. 9. Stable state of the susceptible prey (s=0.2) around E_4 for $(a_2 = 1, a_3 = 0.002, b_1 = 0.35, b_2 = 3.5, c_1 = 0.1, c_2 = 0.2, c_3 = 0.4, e_1 = 0.35, e_2 = 0.5)$

Figure 10, show that the equilibrium point E_5 is stable with parameters' values of $(a_2 = 0.5, a_3 = 0.002, b_1 = 0.35, b_2 = 25, c_1 = 0.1, c_2 = 0.025, c_3 = 0.4, e_1 = 0.35, e_2 = 0.35)$, with the initial conditions. The previous parameters' values satisfy the stability conditions of E_5. For the previous parameters' values, we can see how the system experience a stable co-

existence equilibrium with the infected prey population number dropping down to a stable state of $i \approx 0.0345$, it can be noticed that the system reaches equilibria very fast around $(t \approx 13)$.

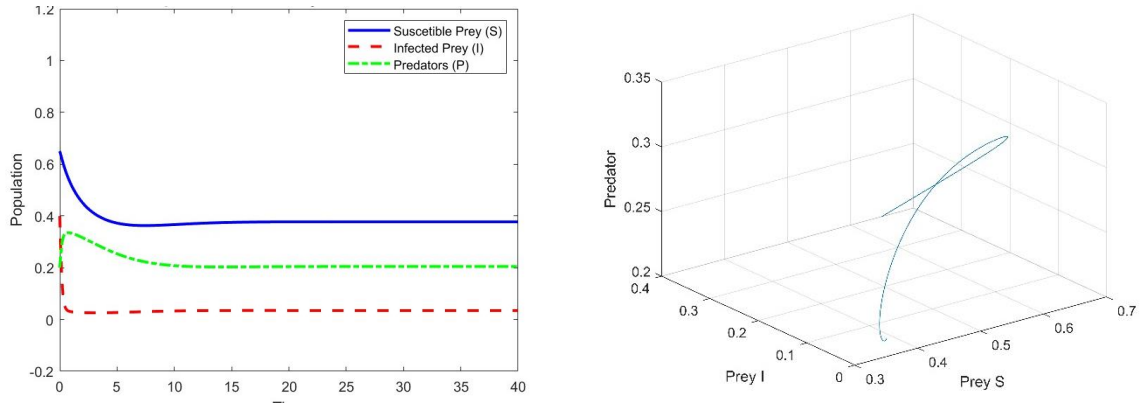

Fig. 10. Stable behaviour of E_5
$(a_2 = 0.5, a_3 = 0.002, b_1 = 0.35, b_2 = 25, c_1 = 0.1, c_2 = 0.025, c_3 = 0.4, e_1 = 0.35, e_2 = 0.35)$

10. RESULTS

In this study, we analysed an eco-epidemiological model characterizing the interaction between a predator population and prey afflicted by disease. Rather than assume infection transmission followed a simple mass action formulation proportional to susceptible and infected prey populations, we adopted a nonlinear incidence rate of the form $\frac{\beta SI}{1+I}$ rooted in density dependence effects. We also accounted for infection potentially rendering prey more vulnerable by stipulating predators consumed infected prey according to mass action dynamics, while predation upon uninfected prey obeyed a Holling Type II functional response. Overall, this work aimed to generate a more nuanced understanding of predator-prey interactions complicated by disease spread according to the nonlinear incidence rate.

We proved that our model is ecologically well-posed as we showed the positiveness and boundness of the proposed model, determined the equilibrium points where we found:

1- one equilibrium at the origin E_0 which is feasible but unstable

2- one axial equilibrium E_1 which is feasible and globally asymptotically stable we also established the conditions for the trancritical bifurcation which occur at E_1

3- three planar equilibria E_2, E_3, E_4 where E_2 is not feasible while E_3 and E_4 are feasible and GAS for some conditions which we established. We also established the conditions at which the E_3 go through a Hopf bifurcation resulting in a limit cycle around E_3

4- Finally, one interior equilibrium point at least which is feasible and stable for some parameters' values as it was shown through the numerical analysis

Our findings align with the findings of [16], [23], as we have shown the impact of infection and predation rates on our model. We also conducted thoroughly analysis to our proposed model exploring interesting bifurcations occurring around E_1 and E_3.

11. DISCUSSION

We found that the predation rate highly affects our proposed model as it could to a different type of bifurcations as we showed that for high enough predation rate a transcritical bifurcation occurs where E_1 and E_3 change stability and the solution converges into a state where both the susceptible prey and the predator survives instead of only the susceptible prey surviving the system. While raising the predation rate for a value higher than a certain limit destabilize the system leading to the born of a limit cycle. We can also notice the direct relation between the Hopf bifurcation constant and the predation rate, showing the important role of the predation rate in creating a limit cycle and presenting a Hopf bifurcation to the proposed model highlighting the important role of the predation rate.

The infection rate on the other hand plays an important role in the dynamic of the system where for different values of the infection rate β and for some set of the parameters' values as we seen in the numeric simulations the system can converge into one of three states, i.e. If we decreased the value of the infection rate less than a certain value say β_1 it can lead to the extinction of the infected prey and two stability cases one where only the susceptible prey survives the system and the second where both the susceptible prey and the predator survives; When the infection rate reaches the value β_1 and in the period $\beta_1 < \beta < \beta_2$ the infected prey survives the extinction leading to state of co-existence stability between the three species; Finally, when the infection rate hits a certain value β_2 and in period say $\beta_2 < \beta < \beta_3$ it leads to a high decrease in the susceptible prey numbers causing the predator to extinct and stabilizing the system. Furthermore, when the predation parameter goes higher than a β_3 it destabilizes the system creating a limit cycle.

Moreover, this model can be better improved by studying different factors and the way they affect this model (such as harvesting, immigration, Allee effect, refugee effect, etc.).

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CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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