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Research article

Stability and bifurcation analysis of a diffusive modified Leslie-Gower prey-predator model with prey infection and Beddington DeAngelis functional response

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A R T I C L E I N F O

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ABSTRACT

In this paper, we present and analyze a spatio-temporal eco-epidemiological model of a prey predator system where prey population is infected with a disease. The prey population is divided into two categories, susceptible and infected. The susceptible prey is assumed to grow logistically in the absence of disease and predation. The predator population follows the modified Leslie-Gower dynamics and predates both the susceptible and infected prey population with Beddington-DeAngelis and Holling type II functional responses, respectively. The boundedness of solutions, existence and stability conditions of the biologically feasible equilibrium points of the system both in the absence and presence of diffusion are discussed. It is found that the disease can be eradicated if the rate of transmission of the disease is less than the death rate of the infected prev. The system undergoes a transcritical and pitchfork bifurcation at the Disease Free Equilibrium Point when the prey infection rate crosses a certain threshold value. Hopf bifurcation analysis is also carried out in the absence of diffusion, which shows the existence of periodic solution of the system around the Disease Free Equilibrium Point and the Endemic Equilibrium Point when the ratio of the rate of intrinsic growth rate of predator to prey crosses a certain threshold value. The system remains locally asymptotically stable in the presence of diffusion around the disease free equilibrium point once it is locally asymptotically stable in the absence of diffusion. The Analytical results show that the effect of diffusion can be managed by appropriately choosing conditions on the parameters of the local interaction of the system. Numerical simulations are carried out to validate our analytical findings.

1. Introduction

It is known that infectious diseases can affect ecological systems and regulate population density. Thus, studying the influence of epidemiological factors on the dynamics of prey-predator interactions plays a crucial role for better understanding of the eco-system. Because of these, mathematical modelling of epidemics has become a very important subject of research after the seminal model of Kermack-McKendric [1] on SIRS systems. Anderson and May [2] were the pioneers for investigating the invasion, persistence, and spread of infectious diseases by formulating an eco-epidemiological prey-predator model. At recent times, many researchers have proposed and studied epidemic and eco-epidemiological models [3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15]. For example, Shaikh et al. [6] have investigated the dynamics of an eco-epidemiological system with disease in competitive prey species. They have considered hyperbolic mortality of predators and Holling type II

functional response and showed the local and global stability of the feasible equilibria and the existence of Hopf bifurcation at both the endemic and Disease Free Equilibrium Points.

On the other hand, in reality, prey and predator species, both infected and healthy ones, are in-homogeneously distributed in different ecological space at a given time and interact with other organisms present within their spatial domain. This consideration involves diffusion process which can be quite intricate as different concentration levels of prey and predator cause different population movements [16]. Thus, this movement or diffusion process must be incorporated in temporal eco-epidemiological models that do not represent space explicitly. Thus, the resulting eco-epidemiological models are represented by reaction diffusion equations.

The spatio-temporal dynamics of a prey-predator system with disease has been investigated by many researchers [17, 18, 19, 20, 21, 22, 23]. Ko et al. [17] have considered a ratio-dependent prey-predator sys-

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tem with infection in the prey population and studied the asymptotic behavior of constant solutions, whereas R.K. Upadhyaya and P. Roy [24] developed a reaction-diffusion eco-epidemiological model of preypredator interaction and found out the occurrence of temporal chaos at a fixed point in space. Raw et al. [18] have studied the dynamical complexities and formation of pattern in a spatial eco-epidemiological prey-predator system with prey infection and harvesting. They investigated conditions for the existence of Turing instability. R.K. Upadhyaya et al. [21] designed a spatial model to study a damaged diffusive ecoepidemiological system of Tilapia and Pelican populations in Salton Sea, California, USA. They observed the existence of Hopf bifurcation and investigated conditions for the Turing instability of the system. Li et al. [20] have considered an eco-epidemiological prey-predator system with infection in predator population and delay, and found the parameter ranges for the occurrence of Turing patterns.

Leslie and Gower [25] introduced a predator prey model where the predator grows logistically with carrying capacity depending on the availability of variable resource, the number of prey. The Leslie-Gower model is a ratio dependent model which has a singularity at the origin. However, when the predator is provided with an alternative food apart from its favored food, it gives rise to modified Leslie-Gower predator dynamics. Now, the model is mathematically free from any singularity and well behaved.

In recent years, more attention has been paid to the study of the dynamics of an eco-epidemiological Leslie-Gower predator-prey interactions [10, 19, 26, 27, 28, 29, 30, 31, 32]. However, most of the works focus on the non-spatial dynamics of an eco-epidemiological Leslie-Gower prey predator dynamics.

The novelty of this paper is the consideration of prey infection, with nonlinear incidence rate, mixed type of functional response: Beddington-DeAngelis type functional response and Holling type II functional response for the susceptible and infected prey population, respectively, and the modified Leslie-Gower predator dynamics. Thus, the main aim of this paper is to study the spatio-temporal dynamics of a diffusive eco-epidemiological predator-prey system with prey infection, Beddington-DeAngelis type functional response and the modified Leslie-Gower type predator dynamics.

The organization of this paper is as follows: in section 2, Mathematical Model Formulation is discussed. Section 3 deals with the analysis of the temporal system: existence and boundedness of solutions, the local and global stability analysis of the biologically feasible equilibrium points and bifurcation analysis of the system (3). Section 4 is devoted to the analysis of the spatio-temporal system: persistence properties of solutions and local stability of equilibrium points of the system (2) are discussed. Numerical simulation results are presented in section 5. Lastly, conclusions are given in section 6.

2. Mathematical model formulation

Let N(X,T) and W(X,T) represent the total prey population densities and the predator population density, respectively at time T and position X in a habitat $\Omega \subset \mathcal{R}_+$ and the prey population is infected with a disease. We took the following assumptions to formulate our eco-epidemiological model.

- 1. In the presence of disease, the prey population is divided into two groups: Susceptible prey U and infected prey V. Therefore the total prey population is N = U + V.
- 2. The susceptible prey is capable of reproducing and hence grows logistically with carrying capacity K and intrinsic growth rate r_1 . The infected population does not recover and the disease is not genetically inherited. The infected prey is removed by death at a natural rate d.
- 3. We assume that the disease transmission follows the non-linear incidence rate $\frac{dUV}{1+bV}$. In this incidence rate the number of effective contacts between infective and susceptible individuals is assumed

Table 1. Biological meaning of parameters.

Parameters	Biological meaning
<i>r</i> ₁	The intrinsic growth rate of Susceptible prey,
Κ	Environmental carrying capacity of prey,
а	Infection rate of prey,
b	Measure of Inhibition of prey,
с	Predation rate of Predator on susceptible prey
В	Saturation constant
ω	Predator interference
Α	Search rate
h	Handling time
d	Natural death rate of infected prey
<i>r</i> ₂	Maximum per capita growth rate of the predator
S	Residual loss in predator population due to severe
	scarcity of its favorite food
<i>s</i> ₂	Conversion factor of susceptible prey into predator
<i>s</i> ₃	Conversion factor of infected prey into predator
D_U	Diffusion coefficient of susceptible prey
D_V	Diffusion coefficient of infected prey
D_{W}	Diffusion coefficient of predator

to saturate at high infective levels due to crowding of infective individuals.

- 4. Predator predates both susceptible and infected prey following the Beddington-DeAngelis functional response and Holling type-II functional response, respectively. The Beddington-DeAngelis functional response is used to capture the mutual interference of predators. Whereas, Holling type II functional response is used because the infected preys are relatively accessible for predation, as they are weak to escape from the predator.
- 5. The predator dynamics follow the modified Leslie-Gower dynamics with intrinsic growth rate r_2 and carrying capacity proportional to the density of the susceptible and infected prey populations.
- 6. The susceptible prey, infected prey and predator population moves in the habitat with constant diffusion coefficients D_U , D_V and D_W , respectively.

Based on the above assumptions and parameters (Table 1), the spatio-temporal eco-epidemiological model is given by the following set of reaction diffusion equations.

$$\begin{cases} U_T - D_U \Delta U = r_1 \left(1 - \frac{U}{K} \right) U - \frac{aUV}{1 + bV} - \frac{cUW}{B + U + \omega W}, \\ V_T - D_V \Delta V = \frac{aUV}{1 + bV} - \frac{AWV}{1 + AhV} - dV, \\ W_T - D_W \Delta W = r_2 \left(1 - \frac{W}{s + s_2 U + s_3 V} \right) W, \\ U_v = V_v = W_v = 0 \\ U(X, 0) = U_0(X) \ge 0, V(X, 0) = V_0(X) \ge 0, \\ W(X, 0) = W_0(X) \ge 0, \end{cases}$$
(1)

where $\Omega \subset \mathcal{R}_+$ is a bounded region with smooth boundary $\partial\Omega$, and all the parameters in the model are assumed to be positive. ν is the outward unit normal vector to the boundary $\partial\Omega$. The admissible initial data $U_0(X)$, $V_0(X)$ and $W_0(X)$ are continuous functions on $\overline{\Omega}$. The homogeneous Neumann boundary condition means that the system (1) is self-contained and has no population flux across the boundary $\partial\Omega$.

Introduce the following non-dimensional variables and parameters so as to reduce the number of parameters of the system (1):

$$\begin{split} u &= \frac{U}{K}, v = \frac{V}{K}, w = \frac{W}{K}, t = r_1 T, x = X \sqrt{\frac{r_1}{D_U}}, D_2 = \frac{D_V}{D_U} \\ D_3 &= \frac{D_W}{D_U}, \alpha = \frac{aK}{r_1}, \kappa = bK, \beta = \frac{B}{K}, \gamma = \frac{c}{r_1}, \theta = \frac{AK}{r_1}, \\ \eta &= \frac{r_2}{r_1}, \sigma = AhK, s_1 = \frac{s}{K}, \delta = \frac{d}{r_1}. \end{split}$$

The system (1) is transformed to the following non-dimensional system.

$$\begin{cases} u_t - \Delta u = (1 - u)u - \frac{\alpha uv}{1 + \kappa v} - \frac{\gamma uw}{\beta + u + \omega w}, \\ v_t - D_2 \Delta v = \frac{\alpha uv}{1 + \kappa v} - \frac{\theta vw}{1 + \sigma v} - \delta v, \\ w_t - D_3 \Delta w = \eta \left(1 - \frac{w}{s_1 + s_2 u + s_3 v} \right) w, \\ u_v = v_v = w_v = 0, \\ u(x, 0) = u_0(x) \ge 0, v(x, 0) = v_0(x) \ge 0, \\ w(x, 0) = w_0(x) \ge 0. \end{cases}$$

$$(2)$$

3. Analysis of the temporal system

It is ecologically and epidemiologically reasonable to study the local dynamics of a predator-prey system before proceeding to the study of its spatio-temporal dynamics. Thus the temporal dynamics of the system (1), which involves only the interaction terms, is

$$\begin{cases} \frac{du}{dt} = (1-u)u - \frac{\alpha uv}{1+\kappa v} - \frac{\gamma uw}{\beta+u+\omega w}, \\ \frac{dv}{dt} = \frac{\alpha uv}{1+\kappa v} - \frac{\theta vw}{1+\sigma v} - \delta v, \\ \frac{dw}{dt} = \eta \left(1 - \frac{w}{s_1 + s_2 u + s_3 v}\right) w, \\ u(0) = u_0 \ge 0, v(0) = v_0 \ge 0, w(0) = w_0 \ge 0. \end{cases}$$
(3)

Let us define

$$G_{1}(u, v, w) = (1 - u)u - \frac{\alpha uv}{1 + \kappa v} - \frac{\gamma uw}{\beta + u + \omega w},$$

$$G_{2}(u, v, w) = \frac{\alpha uv}{1 + \kappa v} - \frac{\theta vw}{1 + \sigma v} - \delta v,$$

$$G_{3}(u, v, w) = \eta \left(1 - \frac{w}{s_{1} + s_{2}u + s_{3}v}\right) w.$$
(4)

3.1. Positive invariance and boundedness

Theorem 3.1. All solutions of the system (3) with positive initial conditions exist and remain positive.

Proof. Let (u(t), v(t), w(t)) be a solution of the system (3). One can easily show that the functions G_1, G_2 and G_3 are continuous functions and locally Lipschitizian on \mathcal{R}_+^3 . Therefore, the solution of the system (3) with positive initial condition exists and is unique. Moreover, it can be shown that these solutions exist for all t > 0 and stay positive. \Box

Theorem 3.2. All solutions of the system (3) which initiate in \mathcal{R}^3_+ are uniformly bounded in the region

$$\begin{split} \Theta &= \left\{ (u,v,w) \in \mathcal{R}_+^3 : u \leq 1 + \varepsilon_1, v(t) \leq \frac{\alpha}{\delta \kappa} + \varepsilon_2, \\ w(t) \leq s_1 + s_2 + \frac{s_3 \alpha}{\delta \kappa} + \varepsilon_3, \forall \varepsilon_1, \varepsilon_2, \varepsilon_3 > 0 \right\}. \end{split}$$

Proof. Let (u(t), v(t), w(t)) be any solution of the system (3) with positive initial conditions. Since $\frac{du}{dt} \le u(1-u)$, by the comparison principle we have $\lim_{t\to\infty} u(t) \le 1$. Thus there exists $t_1 >> 1$ such that $u(t) \le 1 + \varepsilon_1$ for some arbitrarily small $\varepsilon_1 > 0$.

From the second equation of the system (3), we have

$$\frac{dv}{dt} + \delta v \le \frac{\alpha u v}{1 + \kappa v} \le \frac{\alpha (1 + \varepsilon_1)}{\kappa}.$$

Applying Gronwall's inequality [33], we have

$$0 < v(t) < \frac{\alpha(1+\varepsilon_1)}{\delta\kappa} \left(1-e^{-\delta t}\right) + v(0)e^{-\delta t}.$$

Since $\varepsilon_1 > 0$ is arbitrarily small and for letting $t \to \infty$, we have $\lim_{t\to\infty} v(t) \le \frac{\alpha}{\delta\kappa}$. Thus there exists $t_2 >> 1$ such that $v(t) \le \frac{\alpha}{\delta\kappa} + \varepsilon_2$ for some arbitrarily small $\varepsilon_2 > 0$.

From the third equation of the system (3), we have

$$\frac{dw}{dt} \leq \frac{\eta \left(s_1 + s_2(1 + \varepsilon_1) + s_3 \left(\frac{\alpha}{\delta \kappa} + \varepsilon_2 \right) - w \right) w}{s_1 + s_2(1 + \varepsilon_1) + s_3 \left(\frac{\alpha}{\delta \kappa} + \varepsilon_2 \right)}.$$

By the comparison principle, we get

$$\lim_{t\to\infty} w(t) \leq s_1 + s_2(1+\varepsilon_1) + s_3\left(\frac{\alpha}{\delta\kappa} + \varepsilon_2\right).$$

Since $\varepsilon_1 > 0$ and $\varepsilon_2 > 0$ are arbitrarily small, we have $\lim_{t\to\infty} w(t) \le s_1 + s_2 + s_3 \frac{\alpha}{\delta \kappa}$. Thus there exists $t_3 >> 1$ such that $w(t) \le s_1 + s_2 + s_3 \frac{\alpha}{\delta \kappa} + \varepsilon_3$ for some arbitrarily small $\varepsilon_3 > 0$.

Hence all solutions of the system (3), starting in \mathcal{R}_{+}^{3} are uniformly bounded for all $t \ge 0$ and eventually confined in the region

$$\begin{split} \Theta &= \left\{ (u, v, w) \in \mathcal{R}_{+}^{3} : u \leq 1 + \varepsilon_{1}, v(t) \leq \frac{\alpha}{\delta \kappa} + \varepsilon_{2}, \\ w(t) \leq s_{1} + s_{2} + \frac{s_{3}\alpha}{\delta \kappa} + \varepsilon_{3}, \forall \varepsilon_{1}, \varepsilon_{2}, \varepsilon_{3} > 0 \right\}. \quad \Box \end{split}$$

3.2. Extinction criteria

Theorem 3.3. The disease will be removed from the system (3) if $\alpha < \delta$.

Proof. From the second equation of the system (3), we have

$$\frac{dv}{dt} \le \left(\frac{\alpha u}{1+\kappa v} - \delta\right) v \le (\alpha - \delta)v.$$

And hence,

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$$\frac{dv}{dt} + (-\alpha + \delta)v \le 0.$$

Applying Gronwall's inequality [33], we have $0 \le v(t) < v(0)e^{(\alpha-\delta)t}$. Thus, for $t \to \infty$, we have $0 \le v(t) \le 0$, if $\alpha < \delta$. Therefore, $v(t) \to 0$ as $t \to \infty$ if $\alpha < \delta$. Hence, the theorem. \Box

3.3. Equilibrium points and reproduction number

3.3.1. Equilibrium points

The temporal system (3) has the following six biologically feasible equilibrium points.

- 1. The Extinction Equilibrium Point $E_0 = (0, 0, 0)$, which exists always.
- 2. The Infection and Predator Free Equilibrium Point $E_1 = (1, 0, 0)$, which exists always.
- 3. The Prey Free Equilibrium Point $E_2 = (0, 0, s_1)$, which exists always.
- 4. The Predator Free Equilibrium Point $E_3 = (u^*, v^*, 0)$, where $u^* = \frac{\delta(1+\kappa v^*)}{a}$ and v^* is the unique positive root of the quadratic equation

$$\delta^2 \kappa^2 v^2 + (\alpha^2 \delta - \alpha \delta \kappa + 2\delta^2 \kappa) v - \delta(\alpha - \delta) = 0.$$
⁽⁵⁾

Equation (5) will have a unique positive root if $\alpha > \delta$. Thus, E_3 exists if $\alpha > \delta$.

5. The Disease Free Equilibrium Point $E_4 = (\hat{u}, 0, \hat{w})$, where

$$\hat{u} = \frac{-B_1 + \sqrt{B_1^2 + 4(1 + s_2\omega)C_1}}{2(1 + s_2\omega)}, \\ \hat{w} = s_1 + s_2\hat{u}$$
(6)

with

 $B_1 = \beta + s_2 \gamma + \omega(s_1 - s_2) - 1, \ C_1 = \beta - s_1(\gamma - \omega).$

It can be observed that \hat{u} is positive if $C_1 > 0$. Thus, E_4 exists if

$$\beta > s_1(\gamma - \omega). \tag{7}$$

6. The Endemic Equilibrium Point $E_5 = (\tilde{u}, \tilde{v}, \tilde{w})$, where

$$\tilde{w} = s_1 + s_2 \tilde{u} + s_3 \tilde{v},$$

$$\tilde{u} = \frac{(1 + \kappa \tilde{v})(\delta + s_1 \theta + (s_3 \theta + \sigma \delta) \tilde{v})}{\alpha - s_2 \theta + (\alpha \sigma - s_2 \theta \kappa) \tilde{v}}$$
(9)

and \tilde{v} is the unique positive root of the polynomial

$$A_5v^5 + A_4v^4 + A_3v^3 + A_2v^2 + A_1v + A_0 = 0.$$
 (10)

The coefficients A_i (*i* = 1, 2, 3, 4, 5) are given in Supplementary Appendix.

3.3.2. Reproduction number

The basic reproduction number R_0 is obtained by using the next generation matrix method [34]. The temporal system (3) has one infected state, v, and two uninfected states, u and w. Therefore, the flux of newly infected is \mathcal{F} , where $\mathcal{F} = \frac{auv}{\kappa v+1}$; other entering and leaving fluxes is \mathcal{V} , where $\mathcal{V} = \frac{\theta vw}{\sigma v+1} + \delta v$.

The Next generation matrix is then $K = FV^{-1}$, where

$$F = \left(\frac{\partial \mathcal{F}}{\partial v}\right)\Big|_{E_4 = (\hat{u}, 0, \hat{w})} \text{ and } V = \left(\frac{\partial \mathcal{V}}{\partial v}\right)\Big|_{E_4 = (\hat{u}, 0, \hat{w})}$$

The reproduction number R_0 is the spectral radius of the generation matrix *K*, which can be given as [34]

$$\mathbf{R}_0 = \rho(K) = \frac{\alpha \hat{u}}{\delta + \theta(s_1 + s_2 \hat{u})}.$$
(11)

3.4. Local stability analysis

In this subsection, the local dynamics of the system (3) around the biologically feasible equilibrium points is investigated. The Jacobean matrix of the system (3) at any arbitrary point (u, v, w) is given as

$$J(u, v, w) = \begin{pmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & a_{23} \\ a_{31} & a_{32} & a_{33} \end{pmatrix},$$

where

$$\begin{split} a_{11} &= 1 - 2u - \frac{\alpha v}{\kappa v + 1} - \frac{\gamma w (\beta + w\omega)}{(\beta + u + w\omega)^2}, \\ a_{12} &= -\frac{\alpha u}{(\kappa v + 1)^2}, \quad a_{13} = -\frac{\gamma u (\beta + u)}{(\beta + u + w\omega)^2}, \\ a_{21} &= \frac{\alpha v}{\kappa v + 1}, a_{22} = -\delta + \frac{\alpha u}{(\kappa v + 1)^2} - \frac{\theta w}{(\sigma v + 1)^2}, \\ a_{23} &= -\frac{\theta v}{\sigma v + 1}, \quad a_{31} = \frac{\eta s 2 w^2}{(s_1 + s_2 u + s_3 v)^2}, \\ a_{32} &= \frac{\eta s_3 w^2}{(s_1 + s_2 u + s_3 v)^2}, a_{33} = \frac{\eta (s_1 + s_2 u + s_3 v - 2w)}{s_1 + s_2 u + s_3 v}. \end{split}$$

Theorem 3.4. The Extinction Equilibrium Point $E_0 = (0,0,0)$ and the Infected Prey and Predator Free Equilibrium Points $E_1 = (1,0,0)$ are unstable saddle points.

Proof. The eigenvalues of the Jacobean matrices $J(E_0)$ and $J(E_1)$ are $1, -\delta, \eta$ and $-1, \alpha - \delta, \eta$, respectively. Therefore, both the equilibrium points E_0 and E_1 are unstable saddle equilibrium points as J has a positive eigenvalue at the respective equilibrium points.

Theorem 3.5. The Prey Free Equilibrium Point $E_2 = (0, 0, s_1)$ is locally asymptotically stable provided $\beta < s_1(\gamma - \omega)$. Otherwise, it is unstable.

Proof. The eigenvalues of the Jacobean matrix $J(E_2)$ are $-\eta, 1 - \frac{s_1\gamma}{\beta+s_1\omega}, -\delta-s_1\theta$. Thus, all eigenvalues will be negative provided $1 - \frac{s_1\gamma}{\beta+s_1\omega}$ is negative. Therefore, E_2 will be locally asymptotically stable provided $\beta < s_1(\gamma - \omega)$. However, if $\beta > s_1(\gamma - \omega)$, then E_2 becomes unstable. \square

Theorem 3.6. The Predator Free Equilibrium Point $E_3 = (u^*, v^*, 0)$ is unstable.

Proof. One of the eigenvalues of the Jacobean matrix $J(E_3)$ is $\eta > 0$. Therefore, the equilibrium point E_3 is unstable.

Theorem 3.7. The Disease Free Equilibrium Point $E_4 = (\hat{u}, 0, \hat{w})$ is locally asymptotically stable if

$$R_0 < 1, \ \eta > a_{11},$$
 (12)

where

$$a_{11} = \left(\frac{\gamma \hat{w}}{(\beta + \hat{u} + \omega \hat{w})^2} - 1\right) \hat{u}.$$

Proof. The characteristic equation of the Jacobean matrix $J(E_4)$ is

$$(a_{22} - \lambda)(\lambda^2 + (\eta - a_{11})\lambda - (a_{11} + s_2 a_{13})\eta = 0,$$
(13)

where

$$a_{13} = -\frac{\gamma \hat{u}(\beta + \hat{u})}{(\beta + \hat{u} + \omega \hat{w})^2}, \quad a_{22} = \delta(s_1 + s_2 \theta \hat{u})(\mathbf{R}_0 - 1).$$
(14)

The roots of the characteristics equation (13) are

$$\lambda_{1,2} = \frac{a_{11} - \eta \pm \sqrt{(a_{11} - \eta)^2 + 4\eta(a_{11} + s_2 a_{13})}}{2},$$

$$\lambda_3 = \delta(s_1 + s_2 \theta \hat{u})(\mathbf{R}_0 - 1).$$
(15)

It is clear that λ_3 becomes negative for $R_0 < 1$ and $\lambda_{1,2}$ will have negative real part if $a_{11} - \eta < 0$ and $a_{11} + s_2 a_{13} < 0$. Now,

$$\begin{split} a_{11} + s_2 a_{13} &= -\frac{\hat{u}((1+s_2\omega)^2 \hat{u}^2 + B\hat{u} + C)}{(\beta + \hat{u} + \omega \hat{w}^2)}; \\ B &= 2(\beta + s_1\omega)(1+s_2\omega), \ C &= (\beta + s_1\omega)^2 + \gamma(s_2\beta - s_1). \end{split}$$

Since $2(1 + s_2\omega)\hat{u} > 1 + s_2\omega - (\beta + s_2\gamma + s_1\omega)$ and $\beta - s_1(\gamma - \omega) > 0$ (cf. (6) and (7)), we have

 $B\hat{u}+C>(1+s_2\omega)(\beta-s_1(\gamma-\omega))>0$

This implies $a_{11} + s_2 a_{13} < 0$.

Therefore, the Disease Free Equilibrium Point E_4 will be locally asymptotically stable if $R_0 < 1$ and $\eta > a_{11}$ (i.e. condition (12) holds). Hence the theorem.

Theorem 3.8. The Endemic Equilibrium Point $E_5 = (\tilde{u}, \tilde{v}, \tilde{w})$ is locally asymptotically stable if

$$\phi_2 > 0, \ \phi_0 > 0, \ \phi_1 \phi_2 - \phi_0 > 0,$$
 (16)

where

$$\begin{split} \phi_2 &= -a_{11} - a_{22} - \eta, \\ \phi_1 &= a_{11}a_{22} - a_{21}a_{12} - \eta(s_2a_{13} + s_3a_{23} + a_{11} + a_{22}), \\ \phi_0 &= \eta(a_{11}a_{22} - a_{12}a_{21} + s_3(a_{11}a_{23} - a_{13}a_{21}) \\ &+ s_2(a_{13}a_{22} - a_{12}a_{23})) \end{split}$$

and a_{ij} (i = 1,3; j = 1,2,3) are the entries of the Jacobean matrix $J(E_5)$ which are given as

$$\begin{split} a_{11} &= \tilde{u} \left(\frac{\gamma \tilde{w}}{(\beta + \tilde{u} + \omega \tilde{w})^2} - 1 \right), \quad a_{12} = -\frac{\alpha \tilde{u}}{(\kappa \tilde{v} + 1)^2} \\ a_{13} &= -\frac{\gamma \tilde{u}(\beta + \tilde{u})}{(\beta + \tilde{u} + \omega \tilde{w})^2}, a_{21} = \frac{\alpha \tilde{v}}{\kappa \tilde{v} + 1}, \\ a_{22} &= -\frac{\alpha \kappa \tilde{u}}{(\kappa \tilde{v} + 1)^2} + \frac{\theta \sigma \tilde{w}}{(\sigma \tilde{v} + 1)^2}, \quad a_{23} = -\frac{\theta \tilde{v}}{\sigma \tilde{v} + 1}. \end{split}$$

Proof. The characteristic equation of the Jacobean matrix $J(E_5)$ is

$$\lambda^{3} + \phi_{2}\lambda^{2} + \phi_{1}\lambda + \phi_{0} = 0.$$
(17)

According to Routh-Hurwitz criteria, all the roots of the characteristics equation (17) have negative real parts if and only if $\phi_2 > 0$, ϕ_0 and $\phi_1\phi_2 - \phi_0 > 0$.

Therefore, the Endemic Equilibrium Point E_5 is locally asymptotically stable provided the condition (16) is satisfied. Hence the result. \Box

3.5. Global stability

In this subsection the global stability of the Disease Free Equilibrium Point and the Endemic Equilibrium Point is investigated.

Theorem 3.9. The Disease Free Equilibrium Point E_4 is globally asymptotically stable if

$$\alpha < \delta, \quad s_3 \left(1 + \frac{\sigma \alpha}{\delta \kappa} \right) < s_1 \theta,$$

$$\frac{2\gamma s_1 + s_2 \beta + s_2 (1 + 2\gamma) \hat{\mu}}{\chi_1 \chi_2 \chi_3} > \frac{\beta^2 (4s_1 + s_2^2) + s_1^2}{(\beta s_1)^2},$$
(18)

where

$$\chi_1 = \beta + u + \omega w, \quad \chi_2 = \beta + \hat{u} + \omega \hat{w}, \quad \chi_3 = s_1 + s_2 u + s_3 w.$$

Proof. Consider a Lyapunov function

$$S(u, v, w) = \left(u - \hat{u} - \hat{u} \ln \frac{u}{\hat{u}}\right) + v + \left(w - \hat{w} - \hat{w} \ln \frac{w}{\hat{w}}\right).$$
(19)

Differentiating equation (19) with respect to time *t* along the solutions of the temporal system (3) yields

$$\frac{dS}{dt} = A(u-\hat{u})^2 + C(w-\hat{w})^2 + B(u-\hat{u})(w-\hat{w}) + Lv$$
$$= C\left(w-\hat{w} + \frac{B}{2C}(u-\hat{u})\right)^2 + \frac{4AC - B^2}{4C}(u-\hat{u})^2 + Lv$$

where

$$A = \frac{\gamma \hat{w}}{\chi_1 \chi_2} - 1, \quad B = \frac{s_2}{\chi_3} - \frac{\beta + \hat{u}}{\chi_1 \chi_2} \quad C = -\frac{1}{\chi_3},$$
$$L = \frac{s_3 (w - \hat{w})}{\chi_3} + \frac{\alpha \hat{u}}{1 + \kappa v} - \delta - \frac{\theta w}{1 + \sigma v}.$$

Since, C < 0, $\frac{dS}{dt}$ will be negative if $4AC - B^2 > 0$ and L < 0. Now, under condition (18), we have

$$4AC - B^{2} = \frac{2\gamma s_{1} + s_{2}\beta + s_{2}(1+2\gamma)\hat{u}}{\chi_{1}\chi_{2}\chi_{3}} - \left(\frac{4}{\chi_{3}} + \frac{s_{2}^{2}}{\chi_{3}^{2}} + \frac{(\beta+\hat{u})^{2}}{\chi_{1}^{2}\chi_{2}^{2}}\right)$$
$$> \frac{2\gamma s_{1} + s_{2}\beta + s_{2}(1+2\gamma)\hat{u}}{\chi_{1}\chi_{2}\chi_{3}} - \frac{\beta^{2}(4s_{1} + s_{2}^{2}) + s_{1}^{2}}{(\beta s_{1})^{2}} > 0$$

and

$$\begin{split} L &\leq \alpha - \delta + \left(\frac{s_3}{s_1} - \frac{\theta}{1 + \frac{\sigma \alpha}{\delta \kappa}}\right) w \\ &= \alpha - \delta + \left(\frac{s_3(1 + \frac{\sigma \alpha}{\delta \kappa}) - \theta s_1}{s_1(1 + \frac{\sigma \alpha}{\delta \kappa})}\right) w < 0. \end{split}$$

Therefore, by Lyapunov theorem, the Disease Free Equilibrium Point E_4 is globally asymptotically stable if condition (18) holds. Hence the theorem.

Theorem 3.10. *The Endemic Equilibrium Point* E_5 *is globally asymptotically stable if*

$$l_{11} > 0, \ l_{22} > 0, \ l_{12}^2 < l_{11}l_{22}, \ l_{13}^2 < l_{11}l_{33}, \ l_{23}^2 < l_{22}l_{33},$$
 (20)

where

$$\begin{split} l_{11} &= 1 - \frac{\gamma \tilde{w}}{\chi_1}, l_{12} = \frac{\alpha \kappa \tilde{v}}{2(1 + \kappa \tilde{v})}, l_{13} = \frac{s2}{2\chi_4} - \frac{\beta + \tilde{u}}{2\chi_1}, \\ l_{23} &= \frac{s_3}{2\chi_4} - \frac{\theta}{2\chi_3}, l_{22} = -\frac{\theta \sigma \tilde{w}}{\chi_3} + \frac{\alpha \kappa \tilde{u}}{\chi_2}, l_{33} = \frac{1}{\chi_4} \\ \chi_1 &= (\beta + u + \omega w)(\beta + \tilde{u} + \omega \tilde{w}), \chi_2 = (1 + \kappa v)(1 + \kappa \tilde{v}), \\ \chi_3 &= (1 + \sigma v)(1 + \sigma \tilde{v}), \quad \chi_4 = s_1 + s_2 u + s_3 w. \end{split}$$

Proof. Consider a Lyapunov function

$$S(u, v, w) = \left(u - \hat{u} - \hat{u} \ln \frac{u}{\hat{u}}\right) + \left(v - \hat{v} - \hat{v} \ln \frac{v}{\hat{v}}\right) + \left(w - \hat{w} - \hat{w} \ln \frac{w}{\hat{w}}\right).$$
(21)

Differentiating equation (21) with respect to time *t* along the solutions of the temporal system (3) results

$$\begin{aligned} \frac{dS}{dt} &= -l_{11} \left(u - \hat{u} \right)^2 - l_{22} \left(v - \hat{v} \right)^2 + l_{12} (u - \hat{u}) (v - \hat{v}) \\ &- l_{33} \left(w - \hat{w} \right)^2 + l_{13} (u - \hat{u}) (w - \hat{w}) + l_{23} (v - \hat{v}) (w - \hat{w}) \\ &= \mathbf{P}^T M \mathbf{P}, \end{aligned}$$

where

$$\mathbf{P} = (u - \hat{u}, v - \hat{v}, w - \hat{w}), \quad M = \begin{pmatrix} -l_{11} & l_{12} & l_{13} \\ l_{12} & -l_{22} & l_{23} \\ l_{13} & l_{23} & -l_{33} \end{pmatrix}.$$

Now, $\frac{dS}{dt}$ is negative if and only if the matrix *M* is negative definite. The sufficient conditions for the matrix *M* to be negative definite are

$$l_{11} > 0, \ l_{22} > 0, \ l_{33} > 0, \ l_{12}^2 < l_{11}l_{22}, \ l_{13}^2 < l_{11}l_{33}, \ l_{23}^2 < l_{22}l_{33}.$$

Since, $l_{33} > 0$, *M* is negative definite if condition (20) holds. Thus, $\frac{dS}{dt}$ becomes negative if condition (20) holds.

Therefore, by Lyapunov theorem, the Endemic Equilibrium Point E_5 is globally asymptotically stable if condition (20) holds. Hence the theorem.

3.6. Bifurcation analysis

In this subsection the local bifurcation at the Disease Free Equilibrium Point and the Endemic Equilibrium Points is discussed with the help of Sotomayor Theorem [35].

Theorem 3.11. When the bifurcation parameter α passes through the critical value $a^* = \frac{\delta(s_1+s_2\theta\hat{u})}{\hat{u}}$ (i.e. $R_0 = 1$), the temporal system (3) at the Disease Free Equilibrium Point $E_4 = (\hat{u}, 0, \hat{w})$ has

- 1. no saddle-node bifurcation,
- 2. a transcritical bifurcation if $\alpha g \neq \theta h + \kappa \delta(s_1 + s_2 \theta \hat{u}) \theta \sigma \hat{w}$,
- 3. a pitchfork bifurcation if $\alpha g = \theta h + \kappa \delta(s_1 + s_2 \theta \hat{u}) \theta \sigma \hat{w}$, $\sigma \neq \kappa$ and $\sigma \hat{w} \neq h$,

where

$$g = \frac{\delta(s_1 + s_2\theta\hat{u}) - s_3a_{13}}{a_{11} + s_2a_{13}}, \quad h = \frac{s_2\delta(s_1 + s_2\theta\hat{u}) + s_3a_{11}}{a_{11} + s_2a_{13}},$$
$$a_{11} = \frac{\gamma\hat{u}\hat{u}}{(\beta + \hat{u} + \omega\hat{u})^2} - \hat{u}, \quad a_{13} = -\frac{\gamma\hat{u}(\beta + \hat{u})}{(\beta + \hat{u} + \omega\hat{u})^2}.$$

Proof. The Jacobean matrix at E_4 is

$$J(E_4) = \begin{pmatrix} a_{11} & -\alpha \hat{u} & a_{13} \\ 0 & \delta(s_1 + s_2 \theta \hat{u})(\mathbf{R}_0 - 1) & 0 \\ s_2 \eta & s_3 \eta & -\eta \end{pmatrix}.$$

If the bifurcation parameter $\alpha = \alpha^*$, then the Jacobean matrix $J(E_4)$ has a zero eigenvalue and can be given as

$$J(E_4)_{\alpha^*} = \begin{pmatrix} a_{11} & -\alpha \hat{u} & a_{13} \\ 0 & 0 & 0 \\ s_2 \eta & s_3 \eta & -\eta \end{pmatrix}.$$

Let $P = (p_1, p_2, p_3)^T$ be an eigenvector corresponding to the eigenvalue $\lambda = 0$. Hence, $J(E_4)_{a^*} P = 0$ gives $P = p_2(g, 1, h)$ where p_2 is any nonzero real number. Similarly, assume $Q = (q_1, q_2, q_3)^T$ be the corresponding eigenvector of $J(E_4)_{a^*}^T$. Thus, from $J(E_4)_{a^*}^T Q = 0$, we get $Q = (0, q_2, 0)^T$ where q_2 is any nonzero real number.

Let $\mathbf{u}(t) = (u(t), v(t), w(t))^T$ and $\mathbf{G} = (G_1, G_2, G_3)^T$, where G_1, G_2 and G_3 are given in equation (4). Then, the temporal system (3) can be given as $\mathbf{u}' = \mathbf{G}(\mathbf{u})$. Now

$$\frac{d\mathbf{G}}{d\alpha} = \mathbf{G}_{\alpha} = \left(-\frac{uv}{\kappa v+1}, \frac{uv}{\kappa v+1}, 0\right)^{T}.$$

This implies $\mathbf{G}_{\alpha}(E_4, \alpha^*) = (0, 0, 0)^T$. Then, we have $Q^T \mathbf{G}_{\alpha}(E_4, \alpha^*) = (0, 0, 0)^T$.

Thus, by Sotomayor's theorem, the temporal system (3) has no saddle-node bifurcation near the Disease Free Equilibrium Point near $\alpha = \alpha^*$.

The derivative of \mathbf{G}_{α} with respect to \mathbf{u} evaluated at (E_4,α^*) is given as

$$D\mathbf{G}_{\alpha}(E_4, \alpha^*) = \begin{pmatrix} 0 & -\hat{u} & 0\\ 0 & \hat{u} & 0\\ 0 & 0 & 0 \end{pmatrix}.$$

Hence, $Q^T[D\mathbf{G}_{\alpha}(E_4, \alpha^*)P] = p_2 q_2 \hat{u} \neq 0.$

The second derivative of **G** with respect to **u** evaluated at (E_4, α^*) is obtained as

$$D^{2}\mathbf{G}((E_{4}, \alpha^{*}))(P, P) = 2p_{2}(b_{11}, b_{21}, b_{31})^{T}$$

where

$$\begin{split} b_{11} &= \left(\frac{\gamma \hat{w}(\beta + \hat{w}\omega)}{(\beta + \hat{u} + \omega \hat{w})^3} - 1\right) g^2 - \alpha^* g + \kappa \delta(s_1 + s_2 \theta \hat{u}) \\ &- \frac{\gamma (\beta (\beta + \hat{u}) + \omega \hat{w} (\beta + 2\hat{u}))gh}{(\beta + \hat{u} + \omega \hat{w})^3} + \frac{\gamma \hat{u} \omega (\beta + \hat{u})h^2}{(\beta + \hat{u} + \omega \hat{w})^3}, \\ b_{21} &= \alpha^* g - \theta h + \theta \sigma \hat{w} - \kappa \delta(s_1 + s_2 \theta \hat{u}), \\ b_{31} &= \frac{\eta}{s_1 + s_2 \hat{u}} \left(-s_2^2 g^2 - s_2 s_3 g + 2s_2 g h + 2s_3 h - s_3^2 - h^2\right). \end{split}$$

Thus,

$Q^{T}[D^{2}\mathbf{G}((E_{4},\alpha^{*}))(P,P)] = 2p_{2}q_{2}b_{21}.$

Therefore, by Sotomayor's theorem, a transcritical bifurcation occurs at E_4 when the bifurcation parameter α passes the critical value α^* if $\alpha^* \neq \frac{\theta h - \theta \alpha \hat{w} + \kappa \delta(s_1 + s_2 \theta \hat{u})}{\epsilon^*}$.

However, if $\alpha^* = \frac{\theta h - \theta \sigma i \hat{v} + \kappa \delta(s_1 + s_2 \theta \hat{u})}{g}$, then after some algebraic calculations, we get

$$Q^{T}[D^{3}\mathbf{G}((E_{4},\alpha^{*}))(P,P.P)] = 6\theta(\sigma-\kappa)(h-\sigma\hat{w}).$$

Therefore, by Sotomayor's theorem, a pitchfork bifurcation occurs at E_4 when the bifurcation parameter α passes the critical value α^* if $\sigma \neq \kappa$ and $\sigma \hat{w} \neq h$. Hence, the theorem. \Box

Theorem 3.12. The temporal system (3) undergoes Hopf Bifurcation around the Disease Free Equilibrium Point $E_4 = (\hat{u}, 0, \hat{w})$, when the bifurcation parameter η crosses the critical value $\eta_{cr} = a_{11}$, where $a_{11} = \left(\frac{\gamma \hat{w}}{(\theta + \hat{u} + \omega \hat{w})^2} - 1\right) \hat{u}$.

Proof. From the eigenvalues of the Jacobean matrix $J(E_4)$, which are given in (15), we can see that λ_3 is real, λ_1 , λ_2 are purely imaginary if and only if there is a critical value of $\eta = \eta_{cr} = a_{11}$. Thus, at $\eta = \eta_{cr}$, we have $\lambda_1 = i\sqrt{p_2}$, $\lambda_2 = -i\sqrt{p_2}$, $\lambda_3 = \delta(s_1 + s_2\theta\hat{u})(\mathbb{R}_0 - 1)$, where $p_2 = -(a_{11} + s_2a_{13})$ and $a_{13} = \left(\frac{\gamma\hat{w}}{(\hat{\rho}+\hat{u}+a\hat{w})^2} - 1\right)\hat{u}$.

Now, differentiating equation (13) with respect to η gives

$$\begin{split} \left[\frac{d\lambda}{d\eta}\right]_{\eta=\eta_{cr}} &= -\left[\left[\frac{\lambda\dot{p}_1 + \dot{p}_2}{2\lambda + p_1}\right]_{\lambda=i\sqrt{p_2}}\right]_{\eta=\eta_{cr}} \\ &= \left[\frac{2p_2}{p_1^2 + 4p_2}\right]_{\eta=\eta_{cr}} + i\left[\frac{2p_1\sqrt{p_2}}{p_1^2 + 4p_2}\right]_{\eta=\eta_{cr}} \\ &= 0.5 \neq 0, \end{split}$$

where $p_1 = -(a_{11} + s_2 a_{13})$. This implies $Re\left[\frac{d\lambda}{d\eta}\right]_{\eta = \eta_{cr}} = 0.5 \neq 0$.

Therefore, the temporal system (3) undergoes a Hopf Bifurcation around the Disease Free Equilibrium Point at a certain critical value of the parameter $\eta = \eta_{cr}$.

Theorem 3.13. The temporal system (3) will experience a Hopf Bifurcation around the Endemic Equilibrium Point E_5 when the bifurcation parameter η passes the critical value $\eta = \eta_{cr}$ if the following conditions hold.

(i) $\phi_2(\eta) > 0$ and $\phi_0(\eta) > 0$ at $\eta = \eta_{cr}$, (ii) $H(\eta) := \phi_1(\eta)\phi_2(\eta) - \phi_0(\eta) = 0$ at $\eta = \eta_{cr}$,

(iii)
$$\left[\frac{dH(\eta)}{d\eta}\right]_{\eta=\eta_{cr}} \neq 0,$$

where ϕ_2, ϕ_1, ϕ_0 are as defined in theorem (3.8).

Proof. From conditions (i) and (ii), it follows that the temporal system (3) will have one negative root and two purely imaginary roots. Thus, for $\eta = \eta_{cr}$, the characteristic equation of the Jacobean matrix $J(E_5)$ given in (17), must be written as $(\lambda^2 + \phi_1)(\lambda + \phi_2) = 0$, and gives the three roots: $\lambda_1 = i\sqrt{\phi_1}$, $\lambda_2 = -i\sqrt{\phi_1}$ and $\lambda_3 = -\phi_2$.

For all values of the bifurcation parameter η , the eigenvalues $\lambda_{1,2}$ are of the form $\lambda_1 = q_1(\eta) + iq_2(\eta)$ and $\lambda_2 = q_1(\eta) - iq_2(\eta)$, in which $q_1(\eta)$ and $q_2(\eta)$ are real. Substituting $\lambda = q_1(\eta) + iq_2(\eta)$ in to the characteristics equation (17) gives

$$(q_1(\eta) + iq_2(\eta))^3 + \phi_2(\eta)(q_1(\eta) + iq_2(\eta))^2 + \phi_1(\eta)(q_1(\eta) + iq_2(\eta)) + \phi_0(\eta) = 0.$$
(22)

Differentiating equation (22) with respect to the bifurcation parameter η and separating real and imaginary parts gives

$$A(\eta)\dot{q}_{1}(\eta) - B(\eta)\dot{q}_{2}(\eta) + C(\eta) = 0,$$
(23)

$$B(\eta)\dot{q}_{1}(\eta) + A(\eta)\dot{q}_{2}(\eta) + D(\eta) = 0, \qquad (24)$$

where

$$\begin{split} A(\eta) &= \phi_1(\eta) + 2\phi_2(\eta)q_1(\eta) + 3(q_1^2(\eta) - q_1^2(\eta)), \\ B(\eta) &= 2(\phi_2(\eta) + 3q_1(\eta))q_2(\eta), \\ C(\eta) &= (q_1^2(\eta) - q_2^2(\eta))\phi_2(\eta) + q_1(\eta)\phi_1(\eta) + \phi_0(\eta), \\ D(\eta) &= (2q_1(\eta)\phi_2 + \phi_1(\eta))q_2(\eta). \end{split}$$

Solving the simultaneous equations (23) and (24) for \dot{q}_1 gives

$$Re\left[\frac{d\lambda}{d\eta}\right]_{\eta=\eta_{cr}} = -\left[\frac{A(\eta)C(\eta) + B(\eta)D(\eta)}{A^2(\eta) + B^2(\eta)}\right]_{\eta=\eta_{cr}}$$
$$= \left[\frac{\phi_1(\eta)\dot{\phi}_2(\eta) + \phi_2(\eta)\dot{\phi}_1(\eta) - \dot{\phi}_0}{2(\phi_1^2(\eta) + \phi_2^2(\eta)}\right]_{\eta=\eta_{cr}}$$

$$= -\left[\frac{\frac{dH(\eta)}{d\eta}}{2(\phi_1^2(\eta) + \phi_2^2(\eta)}\right]_{\eta=\eta_{rr}} \neq 0.$$

Hence the theorem. $\hfill\square$

4. Analysis of the spatio-temporal system

In this section, the spatio-temporal dynamics of the system (2) is investigated.

4.1. Persistence and boundedness

To study the existence of a positively invariant attracting region, the boundedness and the persistence property of solutions of the spatiotemporal system (2), the following lemma is used [36].

Lemma 4.1. Let f(s) be a positive C^1 function for $s \ge 0$, and let d > 0, $\eta \ge 0$ be constants. Further, let $T \in [0, \infty)$ and $\Phi \in C^{2,1}(\Omega \times (T, \infty)) \cap C^{1,0}(\overline{\Omega} \times [T, \infty))$ be a positive function.

1 If Φ satisfies

$$\begin{cases} \Phi_t - d\Delta \Phi \leq \Phi^{1+\eta} f(\Phi)(\vartheta - \Phi), & (x,t) \in \Omega \times (T,\infty), \\ \Phi_v = 0, & (x,t) \in \partial \Omega \times [T,\infty), \end{cases}$$

and the constant $\vartheta > 0$, then $\limsup_{t \to \infty} \max_{\overline{\Omega}} \Phi(., t) \le \vartheta$.

 $2~\mbox{\it If}~\Phi$ satisfies

$$\begin{cases} \Phi_t - d\Delta \Phi \ge \Phi^{1+\eta} f(\Phi)(\vartheta - \Phi), & (x,t) \in \Omega \times (T,\infty), \\ \Phi_y = 0, & (x,t) \in \partial \Omega \times [T,\infty), \end{cases}$$

and the constant $\vartheta > 0$, then $\liminf_{t \to \infty} \min_{t \to \infty} \Phi(., t) \ge \vartheta$.

3 If Φ satisfies

$$\begin{cases} \Phi_t - d\Delta \Phi \leq \Phi^{1+\eta} f(\Phi)(\vartheta - \Phi), & (x,t) \in \Omega \times (T,\infty), \\ \Phi_v = 0, & (x,t) \in \partial \Omega \times [T,\infty), \end{cases}$$

and the constant $\vartheta \leq 0$, then $\limsup_{t \to \infty} \max_{\overline{\Omega}} \Phi(.,t) \leq 0$.

Theorem 4.2. All solutions of (2) initiating in \mathbb{R}^3_+ are ultimately bounded and eventually enter into the positively invariant attracting region

$$\Sigma = [0, 1] \times \left[0, \frac{\alpha}{\delta \kappa}\right] \times \left[0, s_1 + s_2 + \frac{\alpha s_3}{\delta \kappa}\right]$$

Proof. We have to show that for $(u(x,0), v(x,0), w(x,0)) \in \Sigma$, $(u(x,t), v(x,t), w(x,t)) \in \Sigma \ \forall t \ge 0$. It is straightforward to see that $u(x,t) \ge 0$, $v(x,t) \ge 0$ and $w(x,t) \ge 0$ since the initial values u(x,0), v(x,0) and w(x,0) are nonnegative. Now from the first equation of system (2), we have

 $u_t - \Delta u \le u(1-u).$

Thus, by Lemma 4.1 we have

$$\limsup_{t \to \infty} \max_{\overline{\Omega}} u(.,t) \le 1.$$
⁽²⁵⁾

Thus, for any given $\epsilon > 0$ there exists $t_1 >> 1$, such that $u(x,t) \le 1 + \epsilon$, for $(x,t) \in \overline{\Omega} \times [t_1, \infty)$. As a result, for $(x,t) \in \overline{\Omega} \times [t_1, \infty)$, the equation of v satisfies

$$v_t - D_2 \Delta v \leq \frac{\alpha(1+\varepsilon)v}{1+\kappa v} - \delta v \leq \delta \left(\frac{\alpha(1+\varepsilon)}{\delta \kappa} - v\right).$$

Since $\varepsilon > 0$ is arbitrary, Lemma 4.1 yields

$$\limsup_{t \to \infty} \max_{\overline{\Omega}} v(.,t) \le \frac{\alpha}{\delta \kappa}.$$
(26)

Thus, for any given $\varepsilon_1 > 0$ there exists $t_2 >> 1$, such that $v(x,t) \le \frac{\alpha}{\delta \kappa} + \varepsilon_1$, for $(x,t) \in \overline{\Omega} \times [t_2, \infty)$. As a result, for $(x,t) \in \overline{\Omega} \times [t_2, \infty)$, the equation of *w* satisfies

$$\begin{split} \psi_t - D_3 \Delta w &\leq \eta \left(1 - \frac{w}{s_1 + (1 + \varepsilon)s_2 + (\frac{\alpha}{\delta\kappa} + \varepsilon_1)s_3} \right) w \\ &= \eta \left(\frac{s_1 + (1 + \varepsilon)s_2 + (\frac{\alpha}{\delta\kappa} + \varepsilon_1)s_3 - w}{s_1 + (1 + \varepsilon)s_2 + (\frac{\alpha}{\delta\kappa} + \varepsilon_1)s_3} \right) w. \end{split}$$

Since $\varepsilon > 0$ and $\varepsilon_1 > 0$ are arbitrary, Lemma 4.1 yields

$$\limsup_{t \to \infty} \max_{\overline{\Omega}} w(.,t) \le s_1 + s_2 + \frac{\alpha s_3}{\delta \kappa}.$$
(27)

Hence, the proof is complete. \Box

и

Definition 4.1. The system (2) is said to be persistent if for any nonnegative initial data (u(x, 0), v(x, 0), w(x, 0)) with, $(u(x, 0), v(x, 0), w(x, 0)) \neq (0, 0, 0)$, there exist positive constants σ_0 , σ_1 and σ_2 such that the solutions (u(x, t), v(x, t), w(x, t)) of (2) satisfy

 $\liminf_{t\to\infty}\min_{\overline{\Omega}} u(.,t) \ge \sigma_0, \liminf_{t\to\infty}\min_{\overline{\Omega}} v(.,t) \ge \sigma_1, \liminf_{t\to\infty}\min_{\overline{\Omega}} w(.,t) \ge \sigma_2.$

Theorem 4.3. The system (2) is persistent if

$$l_u = 1 - (\alpha/\kappa) - (\gamma/\omega) > 0, \ \alpha l_u - \left(\theta\left(s_1 + s_2 + \frac{\alpha s_3}{\delta\kappa}\right) + \delta\right) > 0.$$

Proof. Since $u_t - \Delta u \ge u(1 - (\alpha/\kappa) - (\gamma/\omega) - u)$ and $1 > (\alpha/\kappa) + (\gamma/\omega)$, by Lemma 4.1,

$$\liminf_{t \to \infty} \min_{\overline{\Omega}} u(.,t) \ge 1 - (\alpha/\kappa) - (\gamma/\omega) = l_u > 0.$$
(28)

Thus, for any $0 < \varepsilon < l_{\mu}$, there exists $t_0 >> 1$ such that

$$u(x,t) \ge l_u - \varepsilon, \text{ in } \overline{\Omega} \times [t_0,\infty).$$

For $(x, t) \in \overline{\Omega} \times [t_0, \infty)$, the second equation of system (2) gives

$$\begin{split} -D_{2}\Delta v &\geq \left(\frac{\alpha u}{1+\kappa v} - \theta w - \delta\right) v\\ &\geq \left(\frac{\alpha u}{1+\kappa v} - \theta \left(s_{1} + s_{2} + \frac{\alpha s_{3}}{\delta \kappa}\right) - \delta\right) v\\ &\geq \left(\frac{\alpha (l_{u} - \varepsilon)}{1+\kappa v} - \theta \left(s_{1} + s_{2} + \frac{\alpha s_{3}}{\delta \kappa}\right) - \delta\right) v\\ &\geq f\left(\frac{\alpha (l_{u} - \varepsilon) - \theta \left(s_{1} + s_{2} + \frac{\alpha s_{3}}{\delta \kappa}\right) - \delta}{\left(\theta \left(s_{1} + s_{2} + \frac{\alpha s_{3}}{\delta \kappa}\right) + \delta\right) \kappa} - v\right), \end{split}$$

where

 v_t

$$f = \frac{\left(\theta\left(s_1 + s_2 + \frac{\alpha s_3}{\delta \kappa}\right) + \delta\right)\kappa \iota}{1 + \kappa \upsilon}$$

Since ε is arbitrary, we have

$$\liminf_{t \to \infty} \min_{\overline{\Omega}} v(.,t) \ge \frac{\alpha l_u - \left(\theta\left(s_1 + s_2 + \frac{\alpha s_3}{\delta \kappa}\right) + \delta\right)}{\left(\theta\left(s_1 + s_2 + \frac{\alpha s_3}{\delta \kappa}\right) + \delta\right)\kappa} = l_v > 0.$$
(29)

For given $\epsilon^* > 0$, there exists $t_1 \ge t_0$ such that

$$v(.,t) \ge l_v - \varepsilon^*.$$

Similarly, the third equation of the system (2) gives

$$\begin{split} w_t - D_3 \Delta w &\geq \eta \left(1 - \frac{w}{s_1 + s_2(l_u - \varepsilon) + s_3(l_v - \varepsilon *)} \right) w \\ &= \eta \left(\frac{s_1 + s_2(l_u - \varepsilon) + s_3(l_v - \varepsilon *) - w}{s_1 + s_2(l_u - \varepsilon) + s_3(l_v - \varepsilon *)} \right) w. \end{split}$$

Since ε and ε^* are arbitrary, we have

$$\liminf_{t \to \infty} \min_{\overline{\Omega}} w(.,t) \ge s_1 + s_2 l_u + s_3 l_v = l_w > 0.$$
(30)

Hence, from (28), (29) and (30), it follows that the system (2) is persistent. \Box

4.2. Local stability

In this subsection, the stability of the constant steady states of the spatio-temporal system (2) is discussed. It is easy to see that the constant steady states of the spatio-temporal system (2) are the six biologically feasible equilibrium points of the temporal system (3).

Now, denote $\mathbf{u} = (u(x, y, t), v(x, y, t), w(x, y, t))^T$ and $\mathbf{G}(\mathbf{u}) = (G_1(\mathbf{u}), G_2(\mathbf{u}), G_3(\mathbf{u}))^T$. Let $0 = \mu_0 < \mu_1 < \mu_2 < \mu_3 < ...$ be the eigenvalues of the operator $-\Delta$ on Ω under the homogeneous Neumann boundary condition. $O(\mu_i)$ is the eigenspace corresponding to the eigenvalue μ_i , $\mathbf{X}_{ij} := \{\mathbf{c}.\psi_{ij} : \mathbf{c} \in \mathbf{R}^3\}$, where $\{\psi_{ij}\}$ are orthonormal basis of \mathbf{X}_i for $j = 1, 2, 3, ..., dim[O_i], \mathbf{X} := \{\mathbf{u} = (u, v, w) \in [C^1(\overline{\Omega})]^3 | \frac{\partial \mathbf{u}}{\partial v} = 0 \text{ on } \partial\Omega\}$, and $\sum_{\substack{\alpha \in \mathcal{M} \\ \alpha \in \mathcal{M}}} \frac{dim[O(\mu_i)]}{\alpha + 1}$

so
$$\mathbf{X} = \bigoplus_{i=0}^{\infty} \mathbf{X}_i$$
, where $\mathbf{X}_i = \bigoplus_{j=1}^{\min\{O(\mathbf{A}_i)\}} \mathbf{X}_{ij}$.

Linearization of the system (2) at E_n (n = 0(1)5) yields

 $\mathbf{u} = \mathbf{L}\mathbf{u}; \quad \mathbf{L} = \mathbf{D} + J(E_n), \quad \mathbf{D} = diag(1, D_2, D_3),$

where $J(E_n)$ is the Jacobean Matrix which is defined in section 3.4. The eigenspace $\mathbf{X}_i, i \ge 0$, is invariant under the operator \mathbf{L} . λ is an eigenvalue of \mathbf{L} on \mathbf{X}_i if and only if it is an eigenvalue of the matrix $\mathbf{L}_i = -\mu_i \mathbf{D} + J(E_n)$.

Theorem 4.4. The equilibrium points E_0 , E_1 and E_3 are unstable.

Proof. For i = 0, the operator \mathbf{L}_i at $E_0 E_1$ and E_3 has a common positive eigenvalue $\eta > 0$. This shows that the equilibrium points E_0 , E_1 and E_3 are unstable. \Box

Theorem 4.5. The equilibrium point $E_2 = (0, 0, s_1)$ is uniformly asymptotically stable if $\beta + s_1 \omega < s_1 \gamma$.

Proof. The eigenvalues of the operator \mathbf{L}_i at E_2 are $\lambda_{1i} = -\delta - s_1\theta - D_2\mu_i$, $\lambda_{2i} = -\eta - D_3\mu_i$ and $\lambda_{3i} = 1 - \frac{s_1\gamma}{\beta + s_1\omega} - \mu_i$. Hence, all the eigenvalues will be negative if $\beta + s_1\omega < s_1\gamma$. Thus, there exist some positive numbers ρ_i such that $Re\{\lambda_{1i}\}, Re\{\lambda_{2i}\}, Re\{\lambda_{3i}\} \le -\rho_i \forall i$.

Let $\rho = \min\{\rho_i\}$. Then, $\rho > 0$ and $Re\{\lambda_{1i}\}, Re\{\lambda_{2i}\}, Re\{\lambda_{3i}\} \le -\rho \forall i$. Consequently, the spectrum of **L** lies in $\{Re\lambda \le -\rho\}$. Thus, Theorem 5.1.1. of Dan Henry (p. 98) [37] concludes the uniform asymptotically stability of E_2 .

Remark 4.1. If $\beta + s_1 \omega < s_1 \gamma$ then the temporal stability of equilibrium point E_2 ensures the uniform stability of the spatio-temporal system (2) in the vicinity of E_2 . That is, diffusion do not have an effect on the stability of the locally asymptotically stable equilibrium point E_2 .

Theorem 4.6. The Disease Free Equilibrium Point $E_4 = (\hat{u}, 0, \hat{w})$ is uniformly asymptotically stable if

 $\mathbf{R}_0 < 1, a_{11} < 0. \tag{31}$

Proof. The characteristics equation of the operator L_i at E_4 is

where

$$(\lambda - (\delta(s_1 + s_2\theta\hat{u})(\mathbf{R}_0 - 1) - \mu_i D_2))(\lambda^2 + Tr_i\lambda + det_i) = 0,$$
(32)

$$Tr_i = (a_{11} - \eta) - (1 + D_3)\mu_i,$$

 $det_i = -\eta(a_{11} + s_2 a_{13}) + (\eta - a_{11} D_3)\mu_i + D_3 \mu_i^2$

and a_{11} and a_{13} are as in Theorem 3.7.

Hence, under condition (31) and the fact that $a_{11} + s_2a_{13} < 0$ (cf. Theorem 3.7), we can see that $Tr_i < 0$, $det_i > 0$ and $(\delta(s_1 + s_2\theta\hat{u})(\mathbf{R}_0 - 1) - \mu_i D_2) < 0$ for all $i \ge 0$. From the Routh-Hurwitz criterion it follows that, for each $i \ge 0$, all the three roots λ_{1i} , λ_{2i} and λ_{3i} have negative real parts. Thus, there exist some positive numbers ρ_i such that $Re\{\lambda_{1i}\}, Re\{\lambda_{2i}\}, Re\{\lambda_{3i}\} \le -\rho_i \ \forall i$.

Let $\rho = \min\{\rho_i\}$. Then, $\rho > 0$ and $Re\{\lambda_{1i}\}, Re\{\lambda_{2i}\}, Re\{\lambda_{3i}\} \le -\rho \forall i$. Consequently, the spectrum of **L** lies in $\{Re\lambda \le -\rho\}$. Thus, Theorem 5.1.1. of Dan Henry (p. 98) [37] concludes the uniform asymptotically stability of E_4 . \Box

Theorem 4.7. The Endemic Equilibrium Point $E_5 = (\tilde{u}, \tilde{v}, \tilde{w})$ is uniformly asymptotically stable if

$$a_{11} < 0, \quad a_{22} < 0,$$
 (33)

where a_{11} , a_{12} and a_{22} are as defined in Theorem 3.8.

Proof. The characteristics equation of \mathbf{L}_i at the Endemic Equilibrium Point E_5 is given by

$$\lambda^3 + P_{2i}\lambda^2 + P_{1i}\lambda + P_{0i} = 0, \tag{34}$$

where

$$\begin{split} \phi_{2i} &= (1+D_2+D_3)\mu_i + \phi_2, \\ \phi_{1i} &= (D_2+D_3(1+D_2))\mu_i^2 - (a_{22}(1+D_3) + a_{11}(D_2+D_3))\mu_i \\ &+ \eta(1+D_2)\mu_i + \phi_1, \\ \phi_{0i} &= D_2 D_3 \mu_i^3 + (\eta D_2 - (a_{22}+a_{11}D_2)D_3)\mu_i^2 - a_{12}a_{21}D_3\mu_i \\ &+ (a_{11}a_{22}D_3 + (-a_{23}s_3 - a_{22} - (a_{11}+a_{13}s_2)D_2))\eta\mu_i + \phi_0 \end{split}$$

and a_{rs} (r = 1, 3; s = 1, 2, 3), ϕ_2 , ϕ_1 and ϕ_0 are as defined in Theorem 3.8. Algebraic manipulations and simplifications give

$$\phi_{1i}\phi_{2i} - \phi_{0i} = M_1\mu_i^3 + M_2\mu_i^2 + M_3\mu_i^1 + M_4,$$

where

$$\begin{split} M_1 &= (1+D_2)(1+D_3)(D_2+D_3), \\ M_2 &= -a_{11}(D_2+D_3)(2+D_2+D_3) - a_{22}(1+D_3)(1+2D_2+D_3) \\ &+ \eta(1+D_2)(1+D_2+2D_3), \\ M_3 &= -\eta s_2(1+D_3) - s_3\eta a_{23}(D_2+D_3) - a_{12}a_{21}(1+D_2) \\ &+ (a_{11}^2-a_{22}\eta)D_2 + a_{11}a_{22}(a_{11}+a_{22}-2\eta)D_3 \\ &+ (a_{22}-\eta)(a_{22}+(2a_{11}-\eta)(1+D_2)), \end{split}$$

 $M_4 = (a_{11} + a_{22})(a_{12}a_{21} - a_{11}a_{22}) + ((a_{11} + a_{22})^2 + s_3a_{11}a_{13})\eta$

 $+\,s_2a_{12}a_{23}+s_3a_{22}a_{23}+a_{13}(s_2a_{23}+s_3a_{21}))\eta\\$

$$+(-a_{11}-a_{22}-s_2a_{a13}-s_3a_{23})\eta^2.$$

Now, under condition (33) and the fact that $a_{12} > 0$, $a_{13} < 0$, $a_{21} > 0$, $a_{23} < 0$ and

$$a_{21} + s_2 a_{13} = \frac{\delta + s_1 \theta + (s_3 \theta + \sigma \delta) \tilde{v}}{\tilde{u}(1 + \sigma \tilde{v})} > 0,$$

we have $\phi_2 > 0$, $\phi_1 > 0$, $\phi_0 > 0$ and M_n (n = 1, 2, 3, 4) > 0. This implies

 $\phi_{2i} > 0, \ \phi_{1i} > 0, \ \phi_{0i} > 0, \ \phi_{1i}\phi_{2i} - \phi_{0i} > 0, \ \forall i \ge 0.$

Hence, the Routh-Hurwitz criterion implies that, for each $i \ge 0$, all the three roots λ_{1i} , λ_{2i} and λ_{3i} have negative real parts. Thus, there exist



Fig. 1. Transcritical Bifurcation diagram of the temporal system (3) around the Disease Free Equilibrium Point E_4 for the data set as in (35) and $\eta = 0.25$ with respect to α .



Fig. 2. Hopf Bifurcation diagram of the temporal system (3) around the Disease Free Equilibrium Point E_4 for the data set as in (35) with respect to η .

some positive numbers ρ_i such that $Re\{\lambda_{1i}\}, Re\{\lambda_{2i}\}, Re\{\lambda_{3i}\} \le -\rho_i \forall i \ge 0$. Let $\rho = \min\{\rho_i\}$. Then, $\rho > 0$ and $Re\{\lambda_{1i}\}, Re\{\lambda_{2i}\}, Re\{\lambda_{3i}\} \le -\rho \forall i \ge 0$. Consequently, the spectrum of **L** lies in $\{Re\lambda \le -\rho\}$.

Therefore, by Theorem 5.1.1. of Dan Henry (p. 98) [37], the Endemic Equilibrium Point is uniform asymptotically stability. \Box

5. Numerical simulation

In this section, we present some numerical simulation results of the temporal system (3) and the spatio-temporal system (2) to support our analytical findings stated in the previous sections. The numerical simulations are performed with the help of MATLAB-R2014a, Mathematica-11 and MatCont-6pt1 software packages.

5.1. Temporal system

In this subsection, we consider the following two sets of parametric values.

$$\begin{aligned} \alpha &= 0.03, \gamma = 1.5, \theta = 0.6, \delta = 0.1, \sigma = 1, \kappa = 1, \\ s_1 &= 0.1, s_2 = 0.5, s_3 = 0.4, \beta = 0.15, \omega = 0.2, \\ \alpha &= 0.8, \gamma = 0.6, \theta = 0.6, \delta = 0.1, \sigma = 1, \kappa = 0.1, \\ s_1 &= 0.1, s_2 = 0.2, s_3 = 0.4, \beta = 0.1, \omega = 0.1. \end{aligned}$$
(36)

For the data set (35), the Disease Free Equilibrium Point $E_4 = (0.23954, 0, 0.21977)$ exists. For $\eta = 0.25$, the temporal system (3) undergoes a Transcritical bifurcation around E_4 at $\alpha = 0.96794813$ as shown in Fig. 1. Whereas, for the data set (35), the temporal system (3) undergoes a Hopf bifurcation about E_4 when the parameter η crosses the critical value $\eta = \eta_{cr} = 0.18067445$ as shown in Fig. 2.



Fig. 3. Stability behavior of the temporal system (3) around E_4 for the parametric values as in (35) and $\eta = 0.25$. (a) The times series solution (b) The phase portrait showing the local asymptotically stability of E_4 .



Fig. 4. The phase portrait of the temporal system (3) showing the global asymptotic stability of the system (3) around the point E_4 for the parametric values as in (35) and $\eta = 0.25$.

From the Hopf bifurcation diagram (cf. Fig. 2), we can infer that the temporal system (3) exchanges stability when the bifurcation parameter η crosses its threshold value $\eta_{cr} = 0.18067445$. The local stability analysis also shows that, for the parametric values as in (35), the Disease Free Equilibrium Point E_4 will be locally asymptotically stable for $\eta > \eta_{cr} = 0.18067445$ and unstable for $\eta < \eta_{cr} = 0.18067445$. The existence of Hopf bifurcation ensures the existence of periodic solution, leading to the existence of a limit cycle, for $\eta < \eta_{cr} = 0.18067445$. Fig. 3 shows the local stability of the temporal system (3) for the parametric values as in (35) and $\eta = 0.25$. Moreover, the temporal system (3) is globally asymptotically stable around E_4 as shown in Fig. 4.

Fig. 5 shows the existence of periodic solution of the system (3) around the Disease Free Equilibrium Point E_4 for the parametric values as in (35) and $\eta = 0.1$.

For the data set (36), the Endemic Equilibrium Point $E_5 = (0.303278, 0.357855, 0.303798)$ exists. For $\eta = \eta_{cr} = 0.32517$, the temporal system (3) undergoes a Hopf bifurcation around E_5 (cf. Fig. 6).



Fig. 5. Dynamical behavior of the temporal system (3) around E_4 for the parametric values as in (35) and $\eta = 0.1$. (a) The time series solution (b) The phase portrait showing the existence of a limit cycle.





The Hopf bifurcation diagram (cf. Fig. 6) of the temporal system (3) around the Endemic Equilibrium Point E_5 shows the existence of exchange of stability of the temporal system (3) around E_5 when the bifurcation parameter η passes its threshold value $\eta = \eta_{cr} = 0.32517$. From the local stability analysis of the temporal system (2), one can see that, for the parametric values as in (36), the Endemic Equilibrium Point E_5 is locally asymptotically stable for $\eta > \eta_{cr} = 0.32517$ and unstable for $\eta < \eta_{cr} = 0.32517$. The existence of Hopf bifurcation ensures the existence of periodic solution, leading to the existence of a limit cycle, for $\eta < \eta_{cr} = 0.32517$.

The bifurcation diagram (cf. Fig. 7 and Fig. 8) of the temporal system (3) around the Endemic Equilibrium Point E_5 shows the existence of exchange of stability of the temporal system (3) around E_5 when the bifurcation parameter α passes its threshold value $\alpha = \alpha_{cr} = 0.7474$. Moreover, it can be seen that the Endemic Equilibrium Point disappears when the value of α is less 0.312. In this case, a trans-critical bifurcation occurs at the Endemic Equilibrium Point. From the local stability



Fig. 7. Bifurcation diagram of the temporal system (3) around the Endemic Equilibrium Point E_5 for the data set as in (36), except α , and $\eta = 0.2$ with respect to α .



Fig. 8. The Hopf bifurcation diagram of the temporal system (3) around the Endemic Equilibrium Point E_5 for the data set as in (36), except α , and $\eta = 0.2$ with respect to α .

analysis of the temporal system (3), one can see that, for the parametric values as in (36) and $\eta = 0.2$, the Endemic Equilibrium Point E_5 is locally asymptotically stable for $0.312 < \alpha < \alpha_{cr} = 0.7474$ and unstable for $\alpha > \alpha_{cr} = 0.7474$. The existence of Hopf bifurcation ensures the existence of periodic solution, leading to the existence of a limit cycle, for $\alpha > \alpha_{cr} = 0.7474$.

Fig. 9 shows the local stability of the temporal system (3) for the parametric values as in (36) and $\eta = 0.6$. Moreover, the temporal system (3) is globally asymptotically stable around E_5 as shown in Fig. 10. Fig. 11 shows the existence of periodic solution of the system (3) around the Endemic Equilibrium Point E_5 for the parametric values as in (36) and $\eta = 0.2$.

Fig. 12 shows the stability of the temporal system (3) around the Endemic Equilibrium Point for the parametric values as in (36) except $\alpha = 0.7$ and $\eta = 0.2$. Thus, from Figs. 11 and 12, we can observe that a decrease in the amount of prey infection leads to damping of the oscillation and results in the stability of the temporal system (3) around the Endemic Equilibrium Point.

5.2. Diffusive system

In this subsection, numerical simulation results of the stability of the spatio-temporal system (2) around the Disease Free Equilibrium Point and the Endemic Equilibrium Point are presented.

Fig. 13 shows that the spatio-temporal system (2) is locally asymptotically stable around the Disease Free Equilibrium Point $E_4 = (0.368648, 0, 0.284324)$ for the parametric values as in (35) except $\beta = 0.25$, $\eta = 0.25$, $D_2 = 0.01$ and $D_3 = 10$. Fig. 13(a), (b) and (c) represent the time series solution of the spatio-temporal system (2) around



Fig. 9. Stability behavior of the temporal system (3) around E_5 for the parametric values as in (36) and $\eta = 0.6$. (a) The times series solution (b) The phase portrait showing the local asymptotically stability of E_5 .



Fig. 10. The phase portrait of the temporal system (3) showing the global asymptotic stability of the system (3) around the point E_5 for the parametric values as in (36) and $\eta = 0.6$.

the Disease Free Equilibrium Point E_4 at spatial locations x = 500,2000 and x = 4000, respectively. Fig. 13(d) represents the spatial distribution of the species at t = 600.

Fig. 14 shows that the spatio-temporal system (2) is locally asymptotically stable around the Endemic Equilibrium Point $E_5 = 0.409029, 0.374422, 0.331575$ for the parametric values as in (36) except $\kappa = 0.9, \eta = 0.325, D_2 = 0.01$ and $D_3 = 10$. Fig. 14(a), (b) and (c) represent the time series solution of the spatio-temporal system (2) around the Endemic Equilibrium Point E_5 at spatial locations x = 500, 2000 and x = 4000, respectively. Fig. 14(d) represents the spatial distribution of the species at t = 600.

Fig. 15 shows that the spatio-temporal system (2) is spatially unstable around the Endemic Equilibrium Point $E_5 = (0.315978, 0.362293, 0.308113)$, leading to the formation one dimensional chaotic pattern, for the parametric values as in (36) except $\kappa = 0.2$, $\eta = 0.1$, $D_2 = 0.01$ and $D_3 = 10$, and for the heterogeneous initial distribution

$$u(x,0) = 0.315978 + 10^{-8}(x - 1200)(x - 2800)$$



Fig. 11. Dynamical behavior of the temporal system (3) around E_5 for the parametric values as in (36) and $\eta = 0.2$. (a) The time series solution (b) The phase portrait showing the existence of a limit cycle.



Fig. 12. Stability behavior of the temporal system (3) around E_5 for the parametric values as in (36) except $\alpha = 0.7$ and $\eta = 0.2$. (a) The times series solution (b) The phase portrait showing the local asymptotically stability of E_5 .



Fig. 13. Dynamical behavior of the spatio-temporal system (2) around E_4 for the parametric values as in (35) except $\beta = 0.25$, $\eta = 0.25$, $D_2 = 0.01$ and $D_3 = 10$. (a) Time series solution at x = 500 (b) Time series solution at x = 2000 (c) Time series solution at x = 4000 (d) Spatial distribution at time t = 600.



Fig. 14. Dynamical behavior of the spatio-temporal system (2) around E_5 for the parametric values as in (36) except $\kappa = 0.9$, $\eta = 0.325$, $D_2 = 0.01$ and $D_3 = 10$. (a) Time series solution at x = 500 (b) Time series solution at x = 2000 (c) Time series solution at x = 4000 (d) Spatial distribution at time t = 600.



Fig. 15. Emergence of one dimensional chaotic pattern for the parametric values as in (36) except $\kappa = 0.2$, $\eta = 0.1$, $D_2 = 0.01$ and $D_3 = 10$.

v(x, 0) = 0.362293

 $w(x,0) = 0.308113 + 10^{-8}(x - 1200)(x - 2800).$

6. Conclusions

In this paper, a spatio-temporal eco-epidemiological model with Beddington-DeAngelis functional response and the modified Leslie-Gower type predator dynamics under homogeneous Newman boundary condition is considered. The prey population is assumed to be infected with a disease and the disease spread in the system according to the nonlinear incidence rate.

It is observed that the temporal system (3) has six biologically feasible equilibrium points: E_0, E_1, E_2, E_3, E_4 and E_5 . It is also seen that the six biologically feasible equilibrium points of the temporal system (3) are also the constant equilibrium points of the spatio-temporal system (2). The equilibrium points E_0, E_1 and E_3 are unstable both in the presence and absence of diffusion. The prey free equilibrium point is locally asymptotically stable if and only if $\beta < s_1(\gamma - \omega)$, in the presence and absence of diffusion. The local and global stability conditions for the Disease Free Equilibrium Point and Endemic Equilibrium Point of the temporal system (3) are obtained. Moreover, the local stability conditions for the Disease Free Equilibrium Point and Endemic Equilibrium Point of the spatio-temporal system (2) are established.

From the results of Theorem 3.7 and Theorem 4.6, we can conclude that the presence of diffusion will not have an effect on the dynamics of the system (3) around the Disease Free Equilibrium Point as long as the reproduction number is less than unity and the first entry of the corresponding Jacobean matrix is negative.

The infected prey will be extinct if the prey infection rate is less than the death rate of the infected prey (cf. Theorem 3.3). The stability of the Disease Free Equilibrium Point implies that total extinction of the species is not possible and hence the introduction of infected prey into the system may act as a biological control. The Bifurcation analysis of the temporal system (3) shows that the temporal system (3) undergoes a transcritical, pitchfork and Hopf bifurcations under certain conditions.

Numerical simulations are performed to support the analytical results. The numerical simulation results show the existence of transcritical and Hopf bifurcation of the temporal system (3) around the Disease Free Equilibrium Point (cf. Fig. 1 and Fig. 2), and the existence of Hopf bifurcation (cf. Fig. 6 and Fig. 7) and trans-critical bifurcation (cf. Fig. 7) at the Endemic Equilibrium Point. The emergence of chaotic pattern for the system (2) is shown in Fig. 15.

We conclude that the prey infection rate, α , has both stabilizing and destabilizing effect on the Endemic Equilibrium Point. When it is less that its critical value α_{cr} , the susceptible prey, infected prey and predator will coexist and approaches to the Endemic Equilibrium Point. However, when the prey infection rate passes through some critical value, the Endemic Equilibrium Point loses its stability and Hopf bifurcations occurs. The three population exhibits an oscillatory behavior. Wang et al. [38] points out that the increase of the infectious rate can lead to the lost of stability. Thus, our results are inline with the results of Wang et al. [38].

The main novelty between our work and other recent works is the inclusion of prey infection, with nonlinear incidence rate, mixed type of functional response for the susceptible and infected prey population, and the modified Leslie-Gower predator dynamics. These additional ecological components enrich the dynamics of the system and make the system more realistic than the existing models.

Both analytical and numerical simulation results show the complex and rich dynamics of the system under consideration. The future work can be carried out by incorporating the horizontal and vertical disease transmission to the predator population with ecological factors like refuge, additional food and delay and the formation of spatial and spatio-temporal patterns.

Declarations

Author contribution statement

Dawit Melese and Shiferaw Feyissa: Conceived and designed the experiments; Performed the experiments; Analyzed and interpreted the data; Contributed materials, analysis tools or data; Wrote the paper.

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References

- W.O. Kermack, A.G. McKendrick, A contribution to the mathematical theory of epidemics, Proc. R. Soc. A, Math. Phys. Eng. Sci. 115 (772) (1927) 700–721.
- [2] R.M. Anderson, R.M. May, The population dynamics of microparasites and their invertebrate hosts, Philos. Trans. R. Soc. B, Biol. Sci. 291 (1054) (1981) 451–524.
- [3] L. Wang, R. Xu, G. Feng, Modelling and analysis of an eco-epidemiological model with time delay and stage structure, J. Appl. Math. Comput. 50 (1–2) (2016) 175–197.
- [4] D. peng Gao, N. jing Huang, S.M. Kang, C. Zhang, Global stability analysis of an SVEIR epidemic model with general incidence rate, Bound. Value Probl. 2018 (1) (2018).
- [5] A. Meskaf, O. Khyar, J. Danane, K. Allali, Global stability analysis of a two-strain epidemic model with non-monotone incidence rates, Chaos Solitons Fractals 133 (2020) 109647.
- [6] A.A. Shaikh, H. Das, S. Sarwardi, Dynamics of an eco-epidemiological system with disease in competitive prey species, J. Appl. Math. Comput. 62 (1–2) (2020) 525–545.
- [7] C. Maji, D. Mukherjee, D. Kesh, Deterministic and stochastic analysis of an ecoepidemiological model, J. Biol. Phys. 44 (1) (2018) 17–36.
- [8] C. Maji, D. Kesh, D. Mukherjee, Bifurcation and global stability in an eco-epidemic model with refuge, Energy Ecol. Environ. 4 (3) (2019) 103–115.
- [9] A.P. Maiti, C. Jana, D.K. Maiti, A delayed eco-epidemiological model with nonlinear incidence rate and Crowley–Martin functional response for infected prey and predator, Nonlinear Dyn. 98 (2019) 1137–1167.

- [10] A. Mondal, A.K. Pal, G.P. Samanta, On the dynamics of evolutionary Leslie-Gower predator-prey eco-epidemiological model with disease in predator, Ecol. Genet. Genom. 10 (2019) 100034.
- [11] R. Yang, M. Liu, C. Zhang, A diffusive predator-prey system with additional food and intra-specific competition among predators, Int. J. Biomath. 11 (4) (2018) 1–28.
- [12] S. Saha, A. Maiti, G.P. Samanta, A Michaelis-Menten predator-prey model with strong Allee effect and disease in prey incorporating prey refuge, Int. J. Bifurc. Chaos 28 (6) (2018).
- [13] S. Saha, G.P. Samanta, Analysis of a predator-prey model with herd behavior and disease in prey incorporating prey refuge, Int. J. Biomath. 12 (1) (2019).
- [14] S. Mondal, A. Maiti, G.P. Samanta, A predator-prey model with strong Allee effect and disease in prey population, Int. J. Ecol. Econ. Stat. 40 (2) (2019) 92–112.
- [15] S. Mondal, N. Bairagi, G.M. 'Guerekata, Global stability of a Leslie-Gower-type fractional order tritrophic food chain model, Fract. Differ. Calc. 1 (2019) 149–161, http://arxiv.org/abs/1905.11035.
- [16] Y.H. Fan, W.T. Li, Global asymptotic stability of a ratio-dependent predator-prey system with diffusion, J. Comput. Appl. Math. 188 (2) (2006) 205–227.
- [17] W. Ko, W. Choi, I. Ahn, Asymptotic behavior of a diffusive eco-epidemiological model with an infected prey population, Adv. Differ. Equ. 227 (1) (2017) 1–20.
- [18] S.N. Raw, B. Tiwari, P. Mishra, Dynamical complexities and pattern formation in an eco-epidemiological model with prey infection and harvesting, J. Appl. Math. Comput. (2020).
- [19] N. Babakordi, H.R. Zangeneh, M.M. Ghahfarokhi, Multiple bifurcation analysis in a diffusive eco-epidemiological model with time delay, Int. J. Bifurc. Chaos 29 (3) (2019) 1–23.
- [20] J. Li, Z. Jin, G.Q. Sun, L.P. Song, Pattern dynamics of a delayed eco-epidemiological model with disease in the predator, Discrete Contin. Dyn. Syst., Ser. S 10 (5) (2017) 1025–1042.
- [21] R.K. Upadhyay, J. Datta, Y. Dong, Y. Takeuchi, Emergence of spatial patterns in a damaged diffusive eco-epidemiological system, Int. J. Bifurc. Chaos 28 (9) (2018) 1–24.
- [22] B. Mukhopadhyay, R. Bhattacharyya, Dynamics of a delay-diffusion prey-predator model with disease in the prey, J. Appl. Math. Comput. 17 (1–2) (2005) 361–377.
- [23] D. Mukherjee, Effect of diffusion on a two-species eco-epidemiological model, Math. Comput. Model. Dyn. Syst. 11 (4) (2005) 447–457.

- [24] R.K. Upadhyay, P. Roy, Disease spread and its effect on population dynamics in heterogeneous environment, Int. J. Bifurc. Chaos 26 (1) (2016) 1–27.
- [25] P. Leslie, J. Gower, The properties of a stochastic model for the predator-prey type of interaction between two species, Biometrica 47 (1960) 219–235.
- [26] A. Suryanto, I. Darti, Dynamics of Leslie-Gower pest-predator model with disease in pest including pest-harvesting and optimal, Int. J. Math. Math. Sci. 2019 (2019) 1–9.
- [27] S. Sarwardi, M. Haque, E. Venturino, A Leslie-Gower Holling-type II ecoepidemic model, J. Appl. Math. Comput. 35 (1–2) (2011) 263–280.
- [28] A. Kang, Y. Xue, J. Fu, Dynamic behaviors of a Leslie-Gower ecoepidemiological model, Discrete Dyn. Nat. Soc. 2015 (1) (2015).
- [29] S. Sharma, G.P. Samanta, A Leslie-Gower predator-prey model with disease in prey incorporating a prey refuge, Chaos Solitons Fractals 70 (1) (2015) 69–84.
- [30] C. Wei, J. Liu, S. Zhang, Analysis of a stochastic eco-epidemiological model with modified Leslie–Gower functional response, Adv. Differ. Equ. 2018 (1) (2018) 1–17.
- [31] A.A. Shaikh, H. Das, N. Ali, Study of LG-Holling type III predator–prey model with disease in predator, J. Appl. Math. Comput. 58 (1–2) (2018) 235–255, arXiv:1706. 09144 [abs].
- [32] X. Zhou, J. Cui, X. Shi, X. Song, A modified Leslie-Gower predator-prey model with prey infection, J. Appl. Math. Comput. 33 (1–2) (2010) 471–487.
- [33] G. Birkhoff, G.-C. Rota, Ordinary Differential Equations, 4th edition, John Wiley & Sons, Inc., Boston, 1989.
- [34] O. Diekmann, J.A. Heesterbeek, M.G. Roberts, The construction of next-generation matrices for compartmental epidemic models, J. R. Soc. Interface 7 (47) (2010) 873–885.
- [35] J. Sotomayor, Generic Bifurcations of Dynamical Systems, Academic Press, Inc., 1973.
- [36] M. Wang, P.Y. Pang, Global asymptotic stability of positive steady states of a diffusive ratio-dependent prey-predator model, Appl. Math. Lett. 21 (11) (2008) 1215–1220.
- [37] D. Henry, Geometric Theory of Semilinear Parabolic Equations, vol. 14, Lecture Notes in Mathematics, vol. 840, 1982.
- [38] S. Wang, Z. Ma, W. Wang, Dynamical behavior of a generalized eco-epidemiological system with prey refuge, Adv. Differ. Equ. 2018 (1) (2018) 1–20.