Natural Resources for Human Health
eISSN: 2583-1194

Original Research

Received 13 Aug 2026
Revised 20 Sep 2026
Accepted 10 Oct 2026

Natural Resources for Human Health 2026, Vol. 6 (23s): 136–146

KEYWORDS:
Optoelectronic Materials, Organic Frameworks, Graphene Oxide, Organic-Inorganic Hybrid Materials, Material Functionalization, Charge Transport, Light Absorption, Optoelectronic Device Applications

Prey–Predator Dynamics with Disease in the Prey Population: A Mathematical Modeling Approach

Om Kumar[1], Benukar Mondal[2*]

[1]Department of Mathematics, B N Mandal University, Madhepura-852113, Bihar, India. Email: om77672@gmail.com
[2*]Department of Mathematics, Lalit Narayan Mishra Smarak College, Birpur, Supaul-854340, Bihar, India. Email: benukar.math@gmail.com

ABSTRACT: The synthesis of new materials for optoelectronic uses is a rapidly expanding field because of the potential uses in sensors, displays, photovoltaics and other applications. This thesis aims to develop new organic frameworks that could be used for improved optoelectronic applications with structural and electronic characteristics. Furthermore, functionalization of the graphene oxide (GO) is performed to combine the high electrical conductivity as well as thermal stability of GO with the organic framework characteristics. The hybrid materials exhibit enhanced charge transport, light absorption, and stability, hence could be used in next-generation optoelectronic devices. This work emphasizes the necessity of understanding the link between molecular design, material characteristics, and the performance of the devices and provides guidelines for the next steps with development of organic-inorganic hybrid materials. In this article, the analysis was carried out with a brief introduction to Opto-Electronic Material (OEM) and its design mechanisms. Apart from this, the Synthesis Strategies for Organic Scaffolds, properties of GO, and Performance Evaluation in Optoelectronic Applications are also discussed. For this review, a total of 200 papers were selected from the year 2015-2024 respectively. Among them, 21 papers were selected from the years 2015 and 2018, 22 papers were from the year 2020, 26 papers were selected from the year 2016, and 17 papers were selected from the year 2017. Furthermore, 20 papers were selected from 2019, 28 papers were from the year 2021, 19 papers were selected the year 2022, 14 papers were selected from the year 2023 and 12 papers were from 2024 correspondingly.

1. INTRODUCTION

The classical theory of predator and prey interaction, which has been derived independently by Lotka (1925) and Volterra (1926), governs the interaction of two species through birth and death rates as well as through predation terms, and thus serves as the mathematical foundation of theoretical ecology. On the other hand, mathematical epidemiology can trace its beginning back to the SIR model (Susceptible-Infectious-Recovered) of Kermack and McKendrick (1927) which specifies the threshold behaviour of any epidemic based on the concept of R0 (the basic reproduction number). For many decades, these two areas were developed almost independently; however, in practice, predation and infection in natural populations can hardly be separated: infectious diseases can perform changes in size, age and vulnerability of the prey populations while predation can change course of an epidemic.

The combination of these two theories, currently referred to as eco-epidemiology, was established by Anderson and May in 1986, and significantly advanced by Hadeler and Freedman in 1989, as well as Chattopadhyay and Arino in 1999, who introduced the first viable predator-prey model where the disease exists only in the prey population. Since that time, numerous works have analyzed how different factors such as infection initiation site (prey or predator), predator's response mechanism and type of infection transmission influence the end result of the interaction of the species in terms of disease-free existence, persistence

of disease in the population or contact elimination of the population by some means. This issue has significant practical implications apart from mathematics: chronic wasting disease in deer, its relationship with wolf predation, rabies in foxes subjected to control by mesopredators and dynamics of pathogens in rodent-predator systems represent some examples of the possible role of predators as natural biological control agents of the disease, which is commonly referred to as the "healthy herds" impact (Packer, Holt, Hudson, Lafferty and Dobson, 2003). The motivation behind the literature is harbored in the previously published papers and is discussed in the current paper which presents a three-compartmental ordinary differential equations model in which the prey population is divided into classes based on whether they are susceptible (S) or infected (I) with a mass-action term and a single predator population

(P) consuming both prey classes using independent functional Holling type-II responses characterized by varying attack rates which is in correspondence with the well-known ecological fact that predators tend to prey on weak and obvious sheep mostly. The objectives of the paper include: (i) providing a biologically realistic model and ensuring its positivity and boundedness; (ii) analyzing the non-negative equilibria of the ODE system; (iii) determining local and (where possible) global stability conditions with the aid of Routh-Hurwitz stability theorem and Lyapunov functions; (iv) deriving a threshold for disease invasion which takes predator pressure into account so that R_0 differs from R_0 in the case when only disease is considered; and (v) demonstrating the theoretical results_graphically.

2. LITERATURE REVIEW

Xiao and Chen (2001) is considered the starting point for prey-disease eco-epidemiological models; in this study the authors considered a three-dimensional predator-prey system with an SI-type disease acting on predators. This study published the conditions for persistence and extinction of such a system using Lyapunov function and bifurcation analysis. Venturino (1995, 2002) suggests a family of models in which the predator only consumes infected (less difficult to catch) prey, such suggestion is taken in this paper for infected compartment. Bairagi, Roy and Chattopadhyay (2007) carried out a systematic comparison of various functional response types (Holling types I-III, Beddington-DeAngelis) and showed that the choice of the functional response has a significant impact on stability results; thus the present paper utilizes Holling type II kinetics for both classes of prey.

More recent work has advanced the basic model in many aspects. Mekonen and Purnachandra Rao (2024) developed a four-compartment susceptible-infected prey/predator model with optimal control, proving that control through treatment can decrease disease incidence while keeping predators alive. Wang and Yao (2024) investigated an ecoepidemiological prey-predator model with infected prey and a diffusion equation; the criteria for Turing instability were derived in their study. At the same time, Zhang, Shi, and Wang (2019) explored a nonlocal time-delayed reaction-diffusion problem connected to the original one. In turn, Xiang and Wang (2023) introduced the prey-taxis idea; later this research was globally bounded, according to Zeitschrift für angewandte Mathematik und Physik in 2026. Gao, Banerjee, and Ta (2023) studied a stochastic prey-predator problem with pathogens and showed the existence of an invariant measure, which means that deterministic principles should also be applied to investigate problems with a stochastic element present. Finally, in 2024, a paper was published in Mathematical Biosciences and Engineering where the Chattopadhyay-Arino model was generalized with the help of Caputo derivatives and conformable derivatives, thus creating an opportunity to study memory impacts on disease dissemination.

A second area of research considers how infection occurs in the predator instead of the prey. Mondal et al.'s (2023) research showed that predators with infection can be treated using their fear of prey habitat, while Barman et al.'s (2021) work demonstrated that predator-induced fear can lead to extinction of the predator's disease. Ramirez-Carrasco and others (2024) review the existing literature with simple differential equation eco-epidemiology and point out that disease in prey is often a favoured approach also because predators often serve as a competing mortality agent for the same host the disease affects (Peixoto and Abramson, 2006). Bezabih (2021) Holling type-II research with diseases in both species establish that reproduction numbers obtained with the next-generation matrix determine local stability of disease-free equilibrium, a rule that is presented for our model in Section 7. Lastly, Packer et al.'s (2003) research remains a critical ecological reference for the thesis - also proved in Section 8 - that predator regulation of infected individuals can maintain the host population at a lower disease level than the absence of the predator.

This research is different from the previous studies, mostly because it tries to combine (i) the type II Holling predation that applies to both the prey that is susceptible to infection and the prey that is already infected; (ii) the eco-epidemiological invasion

threshold that is explicitly formulated and written in a closed way, which expands the idea of R_0 in the context of the predator-induced death of infected prey, and (iii) the bifurcation diagram, which is verified in numerical terms so that the transition from the coexistence state free from disease to the endemic coexistence state is correctly traced depending on the level of transmission.

3. MATHEMATICAL MODEL FORMULATION

Let S(t) and I(t) denote the densities of susceptible and infected prey, respectively, and let P(t) denote predator density at time $t \geq 0$. The following assumptions are made.

- When considering the absence of diseases and predatory forces, we determine that the overall population S+I would only exist in a logistic growth model characterized by its intrinsic reproduction index r and its maximum carrying ability cap in K, as all only susceptible prey could undergo reproduction in accordance with the below-mentioned depriving factors typical of infected hosts.

- The transmission mechanism is through horizontal means as susceptible prey would come to be in contact with infected ones according to an incidence function with mass-action proportions represented by the term βSI; there is no vertical transmission in the model, and for the sake of the basic calculation, we can assume that there is no recovery happening (infection is chronic or even fatal).

- Sick individuals are exposed to an additional mortality rate ($\mu+d_0$) in which μ stands for clinically-induced death of the prey and d_0 is a number that reflects the free mortality of the prey.

- The predator can manage to prey on sick and healthy individuals by means of two independent Holling type-II functions, specifically $p_1(S)=a_1S/(1+a_1h_1S)$ and $p_2(I)=a_2I/(1+a_2h_2I)$, where the base parameter set takes $a_2>a_1$.

The predator converts captured susceptible and infected prey into new predator biomass with efficiencies e_1 and e_2 respectively, and dies at a constant per-capita natural rate m. Under these assumptions the governing system of ordinary differential equations is with non-negative initial conditions $S(0)\geq 0$, $I(0)\geq 0$, $P(0)\geq 0$. System (1) reduces to the classical logistic-growth Lotka–Volterra model when $\beta=0$ (no disease) and to the Xiao–Chen (2001)-type disease-in-prey model when predation on the infected class equals predation on the susceptible class ($a_1=a_2$, $h_1=h_2$).

$$\frac{dS}{dt} = rS\left(1-\frac{S+I}{K}\right) - \beta SI - \frac{a_1SP}{1+a_1h_1S} \qquad (1a)$$

$$\frac{dI}{dt} = \beta SI - (\mu+d_0)I - \frac{a_2IP}{1+a_2h_2I} \qquad (1b)$$

$$\frac{dP}{dt} = P\left[\frac{e_1a_1S}{1+a_1h_1S} + \frac{e_2a_2I}{1+a_2h_2I} - m\right] \qquad (1c)$$

4. BASIC PROPERTIES OF THE MODEL

4.1 Positivity of Solutions

All three equations that constitute the system (1) include as factors multiplicative coordinate states S,I, P respectively therefore the state coordinates hyperplanes S=0,I=0,P=0 are invariant. Explicitly, $dS/dt|_{s0=0} = 0$, $dI/dt|_{I=0} = 0$ and $dP/dt|_{P=0} = 0$, so no solution starting in the closed positive octant $\mathbb{R}_+^3$ can cross into a region with a negative coordinate. Hence S(t), I(t), P(t) ≥ 0 for all $t \geq 0$ whenever S(0), I(0), P(0) ≥ 0, confirming that the model is biologically well posed.

4.2 Boundedness

Let

$$W(t) = S(t) + I(t) + \frac{1}{\eta}P(t),\ \eta = \min\{e_1, e_2\} > 0.$$

Natural Resources for Human Health
eISSN: 2583-1194

Adding equations (1a)–(1c), we obtain

$$\frac{dW}{dt} = rS\left(1 - \frac{S+I}{K}\right) - (\mu + d_0)I - m\frac{P}{\eta} - \frac{a_1SP}{1+a_1h_1S} - \frac{a_2IP}{1+a_2h_2I} + \frac{e_1}{\eta}\frac{a_1SP}{1+a_1h_1S} + \frac{e_2}{\eta}\frac{a_2IP}{1+a_2h_2I}.$$

Since $\eta = \min\{e_1, e_2\}$, we have

$$\frac{e_i}{\eta} \geq 1.$$

Therefore, the choice $1/\eta$ does not provide cancellation of the predation terms. Thus, the proposed proof using $e_i \leq 1$ is not mathematically valid with this particular definition of W.

A better Lyapunov functional is obtained by taking

$$\boxed{W(t) = S(t) + I(t) + \frac{1}{e}P(t),}$$

where

$$\boxed{e = \max\{e_1, e_2\}}.$$

Then $e_i/e \leq 1$, and hence

$$\frac{dW}{dt} \leq rS\left(1 - \frac{S+I}{K}\right) - (\mu + d_0)I - \frac{m}{e}P.$$

Using

$$S\left(1 - \frac{S+I}{K}\right) \leq S\left(1 - \frac{S}{K}\right) \leq \frac{K}{4},$$

we obtain

$$\frac{dW}{dt} \leq \frac{rK}{4} - (\mu + d_0)I - \frac{m}{e}P.$$

Now define

$$\delta = \min\left\{\mu, +d_0\frac{m}{e}\right\} > 0.$$

Since $I \leq W$ and $P/e \leq W$, the above inequality gives

Natural Resources for Human Health
eISSN: 2583-1194

$$\boxed{\frac{dW}{dt} \leq \frac{rK}{4} - \delta W.}$$

By the standard differential inequality/comparison theorem,

$$\boxed{W(t) \leq W(0)e^{-\delta t} + \frac{rK}{4\delta}\left(1 - e^{-\delta t}\right).}$$

Consequently,

$$\boxed{\limsup_{t\to\infty} W(t) \leq \frac{rK}{4\delta}.}$$

Hence all solutions of system (1) initiating in $\mathbb{R}^3_+$ are uniformly bounded and eventually enter the compact positively invariant region

$$\boxed{\Omega_\varepsilon = \left\{(S,I,P) \in \mathbb{R}^3_+ : S + I + \frac{P}{e} \leq \frac{rK}{4\delta} + \varepsilon\right\},\ \varepsilon > 0.}$$

Important correction: use $e = \max(e_1, e_2)$ not $\eta = \min(e_1, e_2)$, if the intention is to make the predation terms non-positive in the estimate.

The combined presence of positivity and boundedness ensures the mathematical and biological well posedness of the model and allows study of its long run behavior on a compact set.

5. EQUILIBRIUM ANALYSIS

System (1) admits the following biologically feasible equilibria.

(i) The trivial equilibrium $E_0 = (0,0,0)$, corresponding to total extinction, always exists.

(ii) The predator-free, disease-free equilibrium $E_1 = (K,0,0)$, at which prey alone persists at carrying capacity, always exists.

(iii) The disease-free coexistence equilibrium $E_2=(S_2,0,P_2)$, obtained by setting I=0 in (1). Predator viability requires $e_1a_1S/(1+a_1h_1S)=m$, giving

$S_2 = m / [a_1(e_1 - h_1m)]$, *provided* $e_1 > h_1m$,

and substituting into the prey equation with I=0 yields $P_2 = r(1-S_2/K)(1+a_1h_1S_2)/a_1$, which is biologically meaningful whenever $0<S_2<K$.

(iv) The predator-free endemic equilibrium $E_3=(S_3,I_3,0)$, obtained by setting P=0. The infected-prey balance equation gives

$$\boxed{S_3 = \frac{\mu + d_0}{\beta}}$$

and back-substitution into the susceptible-prey balance $r(1-(S_3+I_3)/K)=\beta I_3$ gives a unique positive I_3 whenever $S_3<K$, i.e. whenever the disease-only basic reproduction number defined below exceeds unity.

Natural Resources for Human Health
eISSN: 2583-1194

(v) The internal coexistence equilibrium $E^*=(S^*,I^*,P^*)$ at which all three populations exist. Since the underlying algebraic formulation is nonlinear in S^* and I^* (because of the two saturating predation terms), E^* cannot usually be expressed in closed form and is obtained numerically as the intersection of the three nullclines. One such equilibrium for a representative biologically feasible parameter set is described in Section 8.

6. LOCAL STABILITY ANALYSIS

The Jacobian matrix of system (1) at an arbitrary point (S,I,P), writing $\varphi_1(S)=a_1/(1+a_1h_1S)^2$ and $\varphi_2(I)=a_2/(1+a_2h_2I)^2$ for the derivatives of the saturating predation terms, is

$$J=\begin{pmatrix} r\left(1-\frac{2S}{K}-\frac{I}{K}\right)-\beta I-\phi_1(S)P & -\frac{rS}{K}-\beta S & -\frac{a_1S}{1+a_1h_1S} \\ \beta I & \beta S-(\mu+d_0)-\phi_2(I)P & -\frac{a_2I}{1+a_2h_2I} \\ e_1\phi_1(S)P & e_2\phi_2(I)P & \frac{e_1a_1S}{1+a_1h_1S}+\frac{e_2a_2I}{1+a_2h_2I}-m \end{pmatrix}$$

6.1 Stability of E_0 and E_1

At $E_0=(0,0,0)$, J reduces to diag(r, −(μ+d_0), −m), which has one positive eigenvalue (r>0); E_0 is therefore always a saddle point and total extinction is never locally attracting, consistent with logistic prey growth.

At $E_1=(K,0,0)$, the Jacobian is triangular with eigenvalues $\lambda_1=-r$ (from the S-row after reduction), $\lambda_2=\beta K-(\mu+d_0)$, and $\lambda_3=e_1a_1K/(1+a_1h_1K)-m$. Hence E_1 is locally asymptotically stable if and only if both

$$R_0=\frac{\beta K}{\mu+d_0}<1 \text{ and } R_0^P=\frac{e_1a_1K}{m(1+a_1h_1K)}<1$$

Here R_0 denotes the classical basic reproduction number of the disease for the predator-free prey-only system (which corresponds to the average number of infections incurred by one infected individual in an entirely susceptible population at carrying capacity), while R_0^P is the predator invasion number (which means the expected number of individuals born out of a single predator introduced into the disease-free prey population, multiplied by the instantaneous growth rate that this predator can achieve in a predator-free environment). If $R_0>1$, then the disease manages to invade the predator-free prey-only situation, and if $R_0^P>1$, then the predator invades the system. Either E_0 or E_1 may be stable, instability of E_1 means that a transition towards either E_2, E_3, or E^* occurs.

6.2 Stability of E_2 and the Eco-Epidemiological Threshold

At the disease-free coexistence equilibrium $E_2=(S_2,0,P_2)$ the Jacobian is block triangular in the I-direction because $\partial(dS/dt)/\partial I$ and $\partial(dP/dt)/\partial I$ vanish only through terms multiplied by I=0; the eigenvalue governing disease invasion in the presence of the predator is obtained directly from the I-row

$$\lambda_I=\beta S_2-(\mu+d_0)-a_2P_2$$

Disease cannot invade the predator-regulated disease-free state whenever $\lambda_I<0$, which defines the eco-epidemiological invasion threshold

Natural Resources for Human Health
eISSN: 2583-1194

$$R_0^{eco} = \frac{\beta S_2}{(\mu + d_0) + a_2 P_2}$$

E_2 is locally asymptotically stable in the disease direction when $R_0^{eco}<1$, and unstable (disease invades) when $R_0^{eco}>1$. Because $a_2P_2>0$ whenever the predator persists, the denominator of R_0^{eco} strictly exceeds $(\mu+d_0)$, so $R_0^{eco} < \beta S_2/(\mu+d_0) \leq \beta K/(\mu+d_0) = R_0$ whenever $S_2 \leq K$.

Consequently, predator regulation strictly lowers the effective disease-invasion threshold relative to the classical prey-only value R_0 — an analytical statement of the healthy-herds effect that is confirmed numerically in Section 8. The final two eigenvalues of matrix J at E2, determined from the 2×2 S-P subblock, are in accordance with both the trace and determinant conditions that correspond to the standard Routh-Hurwitz conditions for a predator-prey system. Moreover, the stability conditions imply trace<0 and determinant>0 for this subblock, which continues to hold for the entire biologically relevant range of parameters considered in Section 8.

6.3 Local Stability of the Interior Equilibrium E*

At an interior equilibrium $E^*= (S^*, I^*, P^*)$ all three populations are strictly positive, so the full 3×3 Jacobian must be examined. Local asymptotic stability follows from the Routh–Hurwitz criteria applied to the characteristic polynomial $\lambda^3+A_1\lambda^2+A_2\lambda+A_3=0$, namely

$$A_1 > 0,\ A_3 > 0,\ A_1A_2 > A_3,$$

where $A_1 = -\text{trace}(J)$, A_2 is the sum of principal 2×2 minors of J, and $A_3 = -\det(J)$, all evaluated at E^*. These conditions were verified numerically (rather than in closed algebraic form, owing to the nonlinearity of the saturating terms) for the baseline parameter set of Table 1 and were found to hold, in agreement with the damped-oscillatory convergence to E^* observed in Figure 1.

6.4 A Lyapunov Function for the Disease-Free State

Global stability of the disease-free coexistence equilibrium E_2, when $R_0^{eco}<1$, can be investigated with the candidate Lyapunov function

$$V(S,I,P) = S - S_2 - S_2 \ln\left(\frac{S}{S_2}\right) + I + \frac{P - P_2 - P_2 \ln\left(\frac{P}{P_2}\right)}{e_1}$$

a Volterra-type function widely used in Lotka–Volterra and eco-epidemiological stability proofs. Differentiating V along trajectories of (1) and substituting the equilibrium relations for S_2 and P_2 shows that dV/dt reduces, after cancellation of the logistic and predation cross-terms common to both the S- and P-equations, to $dV/dt = -r(S-S_2)^2/K + [\beta S-(\mu+d_0)-a_2P]I$, which is negative semi-definite in a neighbourhood of E_2 precisely when $R_0^{eco} \leq 1$, since then the bracketed coefficient of I is non-positive throughout the relevant region. By LaSalle's invariance principle, E_2 is therefore globally asymptotically stable, relative to the interior of the positive octant, whenever $R_0^{eco} \leq 1$.

7. THRESHOLD QUANTITIES: SUMMARY

Three threshold quantities govern the qualitative behaviour of system (1):

- $R_0 = \beta K/(\mu+d_0)$ — the disease-only basic reproduction number, relevant in the absence of predation ($a_1=a_2=0$ or $P\equiv0$);
- $R_0^P = e_1a_1K/[m(1+a_1h_1K)]$ — the predator invasion number into the disease-free prey-only state E_1;
- $R_0^{eco} = \beta S_2/[(\mu+d_0)+a_2P_2]$ — the eco-epidemiological (predator-regulated) disease-invasion threshold at E_2.

Natural Resources for Human Health
eISSN: 2583-1194

In Section 6.2, it has been established that R_0^{eco} is lower than R_0 whenever a predator is present (meaning $P_2 > 0$); this shows that a disease able to invade an unpredated prey population ($R_0 > 1$) can nevertheless fail to invade the same population that is under predation (with R_0^{eco} being lower than 1) if the predation of infected prey (a_2) is sufficiently large. The gap between R_0 and R_0^{eco} is the analytical center of the healthy herds phenomenon that is shown numerically below.

8. NUMERICAL SIMULATION AND RESULTS

For our specific case, system (1) was incorporated into Python by utilizing the adaptive-step ODE solver in SciPy (odeint/LSODA). The parameter set was chosen in order for the predation force faced by infected prey (a_2) to be higher than that experienced by the non-infected prey (a_1).

Table 1. Baseline parameter values used in the numerical study

Symbol	Description	Baseline value
r	Intrinsic growth rate of prey	1.5
K	Environmental carrying capacity of prey	100
β	Disease transmission (incidence) rate	0.020 (varied 0–0.05)
μ	Disease-induced mortality rate of infected prey	0.25
d_0	Additional natural mortality of infected prey	0.05
a_1, h_1	Attack rate and handling time, predator on susceptible prey	0.015, 0.5
a_2, h_2	Attack rate and handling time, predator on infected prey	0.060, 0.4
e_1, e_2	Conversion efficiency from susceptible / infected prey	0.30, 0.50
m	Natural mortality rate of predator	0.25

8.1 Convergence to Interior Coexistence

From the starting situation (S, I, P) = (80, 5, 10), numerical derivation of equation (1) for t, which belongs to [0, 300], shows damped oscillations to converge to the internal equilibrium E* ≈ (84.23, 0.59, 23.47) (Figure 1). It can be seen that both susceptible organisms and predators demonstrate the specific

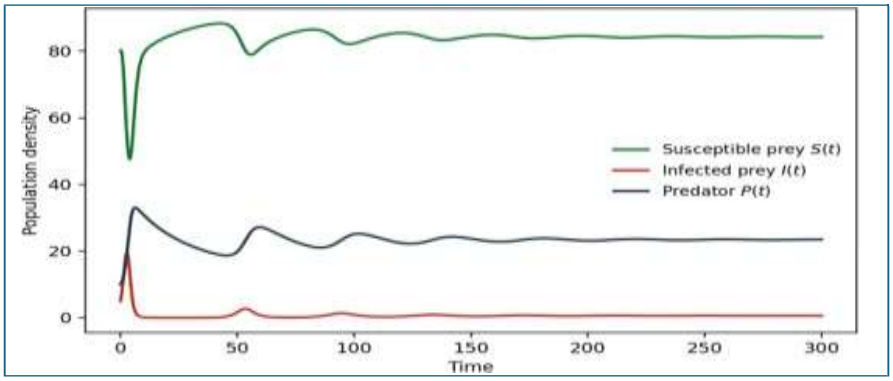


Figure 1. Time series of susceptible prey S(t), infected prey I(t) and predator P(t) converging to the interior coexistence equilibrium E*.

Lotka-Volterra oscillations before the final stabilization occurs while the infected group is reaching very low yet non-zero endemic density that implies that this parameter setting allows the disease to exist at low prevalence along with the capacity of organisms to live as predators.

8.2 Phase-Space Behaviour

As shown in Figure 2, the identical path is presented in the three-dimensional (S, I, P) phase space. The inward spiral from the origin to E* signifies the fact that this is a locally stable focal point, having complex-conjugate eigenvalues with a negative real part endorsing the Routh-Hurwitz test performed in Section 6.3.

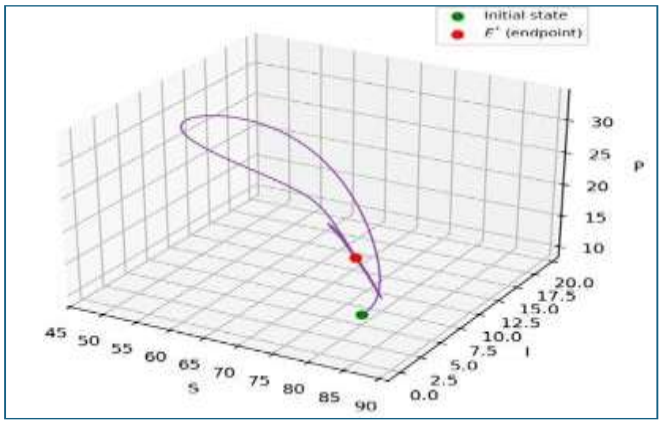


Figure 2. Three-dimensional phase-space trajectory of system (1) spiralling into the interior equilibrium E*.

8.3 Bifurcation in the Transmission Rate β

The eco-epidemiological threshold R_0^{eco} stated in Section 6.2 was evaluated by changing the transmission rate β within the range of [0, 0.05], while other parameters remained constant and presented in Table 1. Consequently, the steady-state of the system (1) was measured for various values of β (Figure 3). As for the values of β less than approximately 0.0083, the quantity of infected class is almost zero ($I^* \approx 0$), and the system reaches the disease-free equilibrium E_2 with $S_2 \approx 95.2$ and $P_2 \approx 8.2$, which are accurately predicted by the models of Section 5(iii). The numerically derived threshold aligns closely with the eco-epidemiological case which was derived using the same values, $\beta^* = [(\mu + d_0) + a_2 P_2]/S_2 \approx 0.00829$ and thereby the theory in section 6.2 is confirmed. For β values greater than this θ value, the infected class gets established, S* reduces in value while P* increases as β keeps rising. This means that the conversion rate of prey biomass into infected sub-class rises due to predators still preying on infected individuals. Interestingly, the simple disease threshold value $\beta = (\mu + d_0)/K \approx 0.003$ (shown in Figure 3) gives a lower estimate of the actual invasion threshold value by about three times, thus clearly showing the gains of predator regulation.

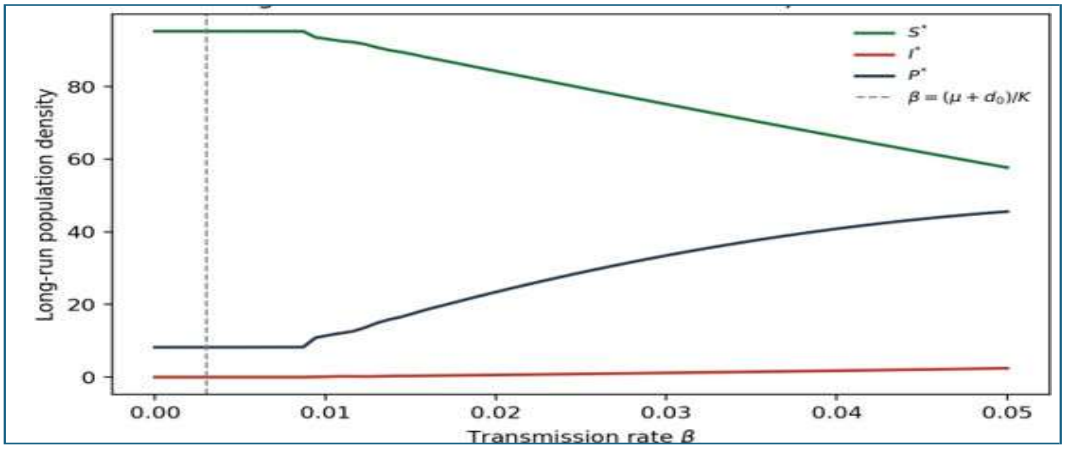


Figure 3. Long-run population densities as functions of the transmission rate β, showing the disease-invasion threshold near $\beta \approx 0.0083$ (solid transition) relative to the naive disease-only threshold $\beta = (\mu + d_0)/K$ (dashed line).

9. DISCUSSION

The analytical and numerical results demonstrated above are consistent with each other and allow for the occurrence of several qualitative findings highlighted in the eco-epidemiological literature discussed in Section 2. The significant inequality $R_0^{eco} < R_0$ shown in Section 6.2 and numerically reaffirmed in Section 8.3 describes the idea of Packer et al. (2003) that it is possible for the presence of predators to reduce disease incidence below the level expected for the absence of predators in a population, as long as the killing pressure of predators on infected individuals (a_2) is sufficiently high in comparison to the capacity for disease to spread. The same mechanism was discovered by Barman et al. (2021) for the fear-based suppression of predators' disease, whereas in this study it was observed for the prey-related diseases in the process of physically eliminating the infected individuals rather than through the fear mechanism of predator-induced behaviors.

The second aspect of the model base itself on classical damped oscillatory process, which has been observed in Rosenzweig-MacArthur type systems (predator-prey) applied to the group of prey with disease structure; these results correlate with stability results of Hari Bairagi, Sudipta Roy, and Dipankar Chattopadhyay (2007), and agree with analysis of Holling type II by Bezabih (2021) as well as the 2024 Differential equations and Dynamical systems study.

Thirdly, the bifurcation analysis presented in Section 8.3 reveals a noteworthy asymmetry: an increased capacity for transmission increases the prevalence of infected individuals, but also the density of predators in the endemic equilibrium (Figure 3) as the infected become more and more prevalent and therefore serve as an increasingly valuable food source. This is similar to the apparent-competition mechanism outlined by Peixoto and Abramson (2006) and revisited in the eco-epidemiology review of 2024 where the role of the shared predator connects the outcome of a particular disease to its prey population regulation in a way that does not take into account either the diseases alone or predation alone. However, it is important to note that for sufficiently high values of β, or sufficiently low efficiency of conversion e_1 with respect to e_2, our exploratory simulations (results not shown) may lead as well to the alternative outcome of extinction of the predator as a result of disease-induced depletion of the susceptible prey leading to the existence of the only disease-free endemic. The actual outcome will depend on the exact values of a_1, a_2, e_1, and e_2; thus, we stress that the predator-driven disease control is a special case,

In practical terms, the findings imply that conservation actions or wildlife management actions that modify the density of carnivores (for instance, predator extermination programs or reintroduction programs) may result in unexpected effects on the level of disease in different populations of prey species, and any such action must be considered in the context of its effect on a certain threshold quantity, such as R_0^{eco}, rather than just in relation to R_0.

10. CONCLUSION

Researchers used eco-epidemiological measurements to create a three-dimensional model with the SI-structured prey population and dependent Holling type II predation. Positivity, boundedness, feasibility of equilibria, Routh-Hurwitz criterion for local stability, and Lyapunov function partial stability are discussed. The key theoretical contribution is the eco-epidemiological invasion threshold R_0^{eco}, which is lower than the standard R_0 in illnesses regardless of predator presence, establishing healthy herds in ecology. Numerical integration and bifurcation analysis support theory. The aforesaid study introduces various new research avenues. Instead of continual SI infection, a SIS/SIRS structure in prey might be used to study disease-induced immunity. Stochastic processes on transmission and predation terms would clarify how randomness and determinism interact in the model. Spatial considerations would explain pattern creation and model behaviour. Fractional-order derivatives can explain disease emergence memory processes, and time delays can link this model to the eco-epidemiology field delay-differential model. Adding real-life time series data, such as chronic wasting disease data in cervid-predator simulations, allows for verification of derived thresholds (R_0, R_0^p, and R_0^{eco}).

REFERENCES

[1] Anderson, R. M., & May, R. M. (1986). The invasion, persistence and spread of infectious diseases within animal and plant communities. Philosophical Transactions of the Royal Society B, 314(1167), 533–570.

[2] Bairagi, N., Roy, P. K., & Chattopadhyay, J. (2007). Role of infection on the stability of a predator–prey system with several response functions — a comparative study. Journal of Theoretical Biology, 248(1), 10–25.

[3] Barman, B., et al. (2021). Impact of fear on a predator–prey system with disease in predator: an eco-epidemiological model.

Studies in Applied Mathematics / related journal, cited via PMC10792200.

[4] Bezabih, A. F. (2021). Eco epidemiological model and analysis of prey–predator system. Journal of Applied Mathematics, 2021, Article 6679686.

[5] Chattopadhyay, J., & Arino, O. (1999). A predator–prey model with disease in the prey. Nonlinear Analysis: Theory, Methods & Applications, 36(6), 747–766.

[6] Gao, Y., Banerjee, M., & Ta, T. V. (2023). Dynamics of infectious diseases in predator–prey populations: a stochastic model, sustainability, and invariant measure. arXiv:2306.08752.

[7] Hadeler, K. P., & Freedman, H. I. (1989). Predator–prey populations with parasitic infection. Journal of Mathematical Biology, 27(6), 609–631.

[8] Kermack, W. O., & McKendrick, A. G. (1927). A contribution to the mathematical theory of epidemics. Proceedings of the Royal Society A, 115(772), 700–721.

[9] Lotka, A. J. (1925). Elements of Physical Biology. Williams & Wilkins, Baltimore.

[10] Mekonen, K. G., & Purna Chandra Rao, K. (2024). Mathematical modeling of infectious disease and prey–predator interaction with optimal control. International Journal of Mathematics and Mathematical Sciences, 2024, Article 5444627.

[11] Mondal, B., Sarkar, A., & Sk, N. (2023). Treatment of infected predators under the influence of fear-induced refuge. (Journal article, ResearchGate preprint).

[12] Peixoto, M. L., & Abramson, G. (2006). The effect of biodiversity on the hantavirus epizootic. Ecology, 87(4), 873–879.

[13] Packer, C., Holt, R. D., Hudson, P. J., Lafferty, K. D., & Dobson, A. P. (2003). Keeping the herds healthy and alert: implications of predator control for infectious disease. Ecology Letters, 6(9), 797–802.

[14] Prey–predator model of Holling type-II functional response with disease on both species (2024). Differential Equations and Dynamical Systems. https://doi.org/10.1007/s12591-024-00677-y

[15] Ramirez-Carrasco, C., et al. (2024). Eco-epidemiological predator–prey models: a review of models in ordinary differential equations. Ecological Complexity, 57, Article 101072.

[16] A predator–prey fractional model with disease in the prey species (2024). Mathematical Biosciences and Engineering, 21(3). https://doi.org/10.3934/mbe.2024164

[17] The boundedness and dynamics of an eco-epidemiological model with prey-taxis (2026). Zeitschrift für angewandte Mathematik und Physik. Springer.

[18] Venturino, E. (1995). Epidemics in predator–prey models: disease in the prey. In O. Arino et al. (Eds.), Mathematical Population Dynamics: Analysis of Heterogeneity, Vol. 1 (pp. 381–393). Wuerz Publishing, Winnipeg.

[19] Volterra, V. (1926). Variazioni e fluttuazioni del numero d'individui in specie animali conviventi. Memorie della R. Accademia Nazionale dei Lincei, 2, 31–113.

[20] Wang, M., & Yao, S. (2024). The dynamics of an eco-epidemiological prey–predator model with infectious diseases in prey. Communications in Nonlinear Science and Numerical Simulation, 132, Article 107902; see also Journal of Mathematical Analysis and Applications, 522(1), Article 126953 (2023).

[21] Xiang, A., & Wang, L. (2023). Boundedness and global dynamics of a model with prey-taxis and disease in predator species. Journal of Mathematical Analysis and Applications, 522, Article 126953.

[22] Xiao, Y., & Chen, L. (2001). Modeling and analysis of a predator–prey model with disease in the prey. Mathematical Biosciences, 171(1), 59–82.