Deterministic and stochastic analysis of an eco-epidemiological model

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Abstract Chronic wasting disease (CWD) is a contagious prion disease among the deer family that has the potential to disrupt the ecosystems where deer occur in abundance. To understand the dynamics of this emerging infectious disease, we consider a simple eco-epidemic model where the host population is infected by CWD. Boundedness of the system is established. The structure of equilibria and their linearized stability are investigated. The persistence condition is discussed. By constructing a suitable Lyapunov function, we discuss the global stability of the endemic equilibrium. Local bifurcation (transcritical) around the boundary equilibria is developed. Sufficient conditions for the existence of Hopf-bifurcation are derived. Further, we have also introduced white type of noise into the system to investigate stochastic stability. This suggests that the deterministic model is robust with respect to stochastic perturbation. Some numerical simulations are performed to validate our results.

Keywords Prey–predator model · Chronic wasting disease (CWD) · Global stability · Hopf-bifurcation · Stochastic stability

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1 Introduction

Chronic wasting disease (CWD) is a fatal infectious prion disease among the deer family [1]. It was first identified as a clinical disease in captive mule deer in 1967 in a wildlife research facility in northern Colorado, USA and southeastern Wyoming [2]. In 1980, the disease was determined to be in the family of transmissible spongiform encephalopathies (TSEs) along with other well-known TSEs like transmissible mink encephalopathy (TME) in Mink, bovine spongiform encephalopathy (BSE) or “mad-cow disease” and both Creutzfeldt–Jakob disease (CJD) and variant Creutzfeldt–Jakob disease (vCJD) in humans. CWD is observed in many species of cervids, like white-tailed deer (*Odocoileus virginianus*), black-tailed deer (*Odocoileus columbianus*), mule deer (*Odocoileus hemionus*), Rocky Mountain elk (*Cervus elaphus nelsoni*) and Shira’s moose (*Alces alces shirasi*) [2, 3]. In 1996, besides Colorado and Wyoming, chronic wasting disease was found for the first time in a captive elk farm in Saskatchewan [1]. It was also found in free-ranging deer in the southwestern corner of Nebraska in May 2001. Infectious agents of CWD are neither bacteria nor viruses but are hypothesized by prions. It is transmitted through direct, animal-to-animal contact, vertically, from mother to offspring and indirectly, via environmental contamination [4, 5]. The disease has a long incubation period (12–34 months [6]) and it causes a spongy degeneration in the brain of infected animals, resulting in emaciation, abnormal behavior, loss of bodily functions, and ultimately death [3]. Clinical signs (e.g., staggering, lowered head/ears, lack of fear, drooling) only appear in the late stages of this disease [7, 8]. As the disease persists for a long time and cannot be prevented by vaccines or any other ways, the disease bears the term chronic. Furthermore, in this disease there is a gradual loss of weight, increased thirst and urination, excessive salivation, trouble walking, and hence the disease is wasting. These two facts justify the particular name CWD of this disease.

In the mid-1990s, CWD had been detected among free-ranging deer and elk in the north-eastern Colorado and southeastern Wyoming [9]. According to hunter-harvested animal surveillance, the overall prevalence of the disease in this area from 1996 through 1999 was estimated at approximately 5% in mule deer, 2% in white-tailed deer, and < 1% in elk [9]. This disease can be highly transmissible within captive deer and elk populations. A prevalence of > 90% was reported among mule deer in facilities where the disease has been endemic for > 2 years [1, 10]. The origin and transmission mechanism of the prions causing CWD is incompletely understood. Research on CWD epidemics in captive deer and elk has shown that CWD prions can remain infectious in the environment for at least 2 years [11] and thus it has the potential to disrupt the ecosystems where deer occur in abundance. Recent works on CWD can be found in [12–14]. Through numerical simulations, Hobbs [15] suggested that CWD can be removed from an infected elk population allowing selective predation by wolves. It was remarked in [16, 17] that increasing mortality rates in diseased population can delay disease transmission and decrease disease prevalence. This motivated Hobbs to modify the model of Miller et al. [4] by introducing density-dependent effects on recruitment into the population, where the model is based on three approximations, the density of infected population, susceptible population, and the mass of infectious material in the environment. The explicit form of predation term is not considered in [15]. It is a well-known fact that predation on a species subjected to an infection can affect both the infection prevalence and population dynamics. As the disease is fatal and causes harm to the deer population, elk, and many other cervids, thus at present, there is no known

cure for the disease, and many aspects of the disease are still unclear. So if we remove the infected animals from the population by predation, then we can protect the animals from the disease. It is reasonable to develop an appropriate eco-epidemiological model that can give some insights of the disease dynamics. Research on eco-epidemic models [18–24] has been addressed on direct transmission but indirect transmission on eco-epidemic model has not been focused so much. Thus, our main aim in this article is to control CWD transmission through predation by developing eco-epidemic model with indirect transmission.

It is a well-known fact that population dynamics are often affected by environmental noise, which is an important component in an ecosystem. There has been a growing interest to study how noise affects the population dynamics. Hence, stochastic differential equation models play a major role in the field of biology and ecology. In deterministic models, parameters are all deterministic irrespective of environmental fluctuation and hence it is very difficult to predict the future dynamics of the system correctly [25]. Therefore, many authors introduced randomness in deterministic models to reveal the effect of environmental variability [25–29].

The paper is structured as follows. In Section 2 we formulate our proposed model. Boundedness of the system is given in Section 3. In Section 4 we discuss dynamical behavior of the boundary as well as interior equilibrium point. Local bifurcation analysis around the boundary equilibrium point is given in Section 5. In Sections 6 and 7 we discuss the Hopf-bifurcation and persistence, respectively. Stochastic analysis of the model is derived in Section 8. We perform some numerical simulation in Section 9. A short discussion is presented in Section 10.

2 The eco-epidemic model

Chronic wasting disease (CWD) is a fatal disease of deer, elk, and moose. Experimentally, it can be shown that CWD is transmitted to susceptible animals from residues of excreta left in the environment by infected animals and their carcasses, which is more appropriate than traditional models of direct, animal-to-animal contact [11, 30]. Thorough observation of these diseases can be difficult because prolonged epizootic can result in low, usually undetected level of infection morbidity or mortality. To understand the incident and spatial dynamics of chronic wildlife disease, long-term studies will be helpful. However, it is very difficult to carry out the investigation in natural populations due to financial and logical constraints. For this reason, such complexity modeling disease dynamics may be the only practical way to measure the spatial and temporal patterns of chronic disease in wildlife. It suggests an alternative transmission mechanism and explores the spread of the disease. Much work has been done to model direct transmission, but despite the fact that CWD prions can remain infectious in the environment for years, relatively little information exists about the potential effects of indirect transmission of CWD dynamics. So in the present study, we consider a prey–predator system where the prey population is divided into two groups, infected and non-infected, and a third dynamic variable, which represents the mass of infectious material in the environment. Our model is based on traditional susceptible-infected-recovered (SIR) models of disease transmission in humans [31–33]. The model is described by a system of four ordinary differential equations. The predator

functional response is assumed to be Holling type II. In view of above, we propose the following model:

$$\begin{aligned}\frac{dS}{dt} &= S \left[r \left(1 - \frac{S+I}{k} \right) - \beta E \right] \\ \frac{dI}{dt} &= \beta S E - \mu I - \frac{aIP}{m+I} \\ \frac{dE}{dt} &= \varepsilon I - \tau E \\ \frac{dP}{dt} &= P \left(-d + \frac{bI}{m+I} \right)\end{aligned}\quad (1)$$

with initial condition $S(0) > 0$, $I(0) > 0$, $E(0) > 0$, $P(0) \geq 0$. Here, S denotes the density of the susceptible animals and I represents the infected ones. E is the mass of infectious material in the environment, and P is the density of the predator.

The first equation describes a logistic growth of the prey, with per-capita birth rate r and environmental carrying capacity k . Susceptible prey population become infected when it comes into contact with the infectious material in the environment. β denotes the indirect transmission coefficient for the disease.

The second equation represents the evolution of the infected populations. The only positive contribution comes from the susceptible class, which are infected. Infected populations are affected by natural death at the rate μ . The infected prey is more vulnerable than susceptible prey, as they are very weak and can be caught more easily than healthy prey. We assume that the predator population consumes the infected prey population with Holling type II functional response, which is $\frac{aI}{m+I}$, where m is half saturation constant and a denotes the maximal predator per-capita consumption rate, i.e., the maximum number of prey population can be eaten by a predator in each unit time.

The first term of third equation shows that deposition of infectious material through excretion by infected animals. The second term denotes the loss of infectious material from the environment.

In the last equation when there is no prey, predator population suffers a natural death at the rate d . In the second term, b represents the conversion efficiency of the predator. Our model is also applicable to propagation of other disease like tuberculosis in livestock [34], avian influenza [35], vibrio cholerae [36, 37], viral hepatitis A [38], etc. Biological significance and units of the model parameters are given in Table 1.

3 Boundedness of the system

In theoretical eco-epidemiology, boundedness of a system implies that the system is biologically valid and well behaved. The following lemma ensures the boundedness of system (1).

Lemma 1 *All the solutions of system (1), which start in R_+^4 are uniformly bounded within a region Ω defined by $\Omega = \{(S, I, E, P) \in R_+^4 : 0 \leq W(t) \leq \frac{k(r+1)}{v}\}$ where $v = \min\{1, \frac{\mu}{2}, \tau, d\}$.*

Proof Let $(S(t), I(t), E(t), P(t))$ be one of the solutions of system (1). As $\frac{dS}{dt} \leq rS(1 - \frac{S+I}{k})$, we have $\lim_{t \rightarrow \infty} S(t) \leq k$. $\square$

Table 1 Parameters of model (1) and their units

Parameter	Definition	Units
r	maximum per-capita growth rate	time^{-1}
k	environmental carrying capacity of the prey	number
β	the indirect transmission coefficient for the disease	$\text{mass}^{-1}\text{time}^{-1}$
μ	the death rate of the infected animals through CWD	time^{-1}
a	predation coefficient	$\text{mass}^{-1}\text{time}^{-1}$
b	the conversion coefficient of the predator	time^{-1}
m	half saturation constant	number
ε	the per-capita rate of excretion of infectious material by infected animals	time^{-1}
τ	the mass specific rate of loss of infectious material from the environment	time^{-1}
d	death rate of predator	time^{-1}

Let a time-dependent function $W(t) = S(t) + I(t) + \frac{\mu}{2\epsilon} E(t) + \frac{a}{b} P(t)$, we get,

$$\frac{dW}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{\mu}{2\epsilon} \frac{dE}{dt} + \frac{a}{b} \frac{dP}{dt}$$

By using system (1),

$$\begin{aligned} \frac{dW}{dt} &= rS\left(1 - \frac{S+I}{k}\right) - \frac{\mu}{2} I - \frac{\mu\tau}{2\epsilon} E - \frac{a}{b} dP \\ &\leq rS - \frac{\mu}{2} I - \frac{\mu\tau}{2\epsilon} E - \frac{a}{b} dP \\ &= (r+1)S - \left[S + \frac{\mu}{2} I + \frac{\mu\tau}{2\epsilon} E + \frac{a}{b} dP\right] \\ &\leq (r+1)k - vW, \text{ where } v = \min\left\{1, \frac{\mu}{2}, \tau, d\right\}. \end{aligned}$$

Hence, we have

$$\frac{dW}{dt} + vW \leq (r+1)k.$$

Now, applying the theory of differential inequality in [39], we get,

$$0 \leq W(t) \leq \frac{(r+1)k}{v} + \frac{W(S(0), I(0), E(0), P(0))}{e^{vt}}$$

$$0 \leq W(t) \leq \frac{(r+1)k}{v} \quad (t \rightarrow \infty).$$

Hence, all the solution of the system will enter in the region $\Omega = \{(S, I, E, P) : 0 \leq W(t) \leq \frac{k(r+1)}{v}\}$.

4 Equilibria and Stability Analysis

Here we discuss the stability of boundary equilibria and the interior equilibrium point of system (1). The system has four equilibrium points, one is a population-free equilibrium

point $E_0(0, 0, 0, 0)$, which always exists. One is susceptible prey-only equilibrium point $E_1(k, 0, 0, 0)$ and the other one is predator-free equilibrium point $E_2(\bar{S}, \bar{I}, \bar{E}, 0)$.

$$\text{where } \bar{S} = \frac{\mu\tau}{\beta\epsilon}, \bar{I} = \frac{\tau\bar{E}}{\epsilon}, \text{ and } \bar{E} = \frac{r(k\beta\epsilon - \mu\tau)}{\beta(r\tau + k\epsilon\beta)}.$$

E_2 is feasible provided $k > \frac{\mu\tau}{\epsilon\beta}$. Thus, the carrying capacity of the environment is high enough to support the predator-free equilibrium.

The interior equilibrium point is given by $E^*(S^*, I^*, E^*, P^*)$ where,

$$S^* = \frac{kr\tau(b-d) - \beta\epsilon dm k - r\tau dm}{r\tau(b-d)}, I^* = \frac{dm}{b-d}, E^* = \frac{\epsilon dm}{\tau(b-d)} \text{ and } P^* = \frac{bm(\beta\epsilon S^* - \mu\tau)}{a\tau(b-d)}.$$

Clearly, the interior equilibrium point E^* is feasible if $b > \frac{d(kr\tau + \beta\epsilon mk + r\tau m)}{kr\tau}$ and $S^* > \frac{\mu\tau}{\beta\epsilon}$.

4.1 Stability analysis

We will now study the dynamical behavior of the system around the boundary and interior equilibrium points. The variational matrix for system (1) at an arbitrary point (S, I, E, P) is given by

$$V(S, I, E, P) = \begin{pmatrix} r - \frac{2rS}{k} - \frac{rI}{k} - \beta E & -\frac{rS}{k} & -\beta S & 0 \\ \beta E & -\mu - \frac{maP}{(m+I)^2} & \beta S & -\frac{aI}{m+I} \\ 0 & \epsilon & -\tau & 0 \\ 0 & \frac{mbP}{(m+I)^2} & 0 & -d + \frac{bI}{m+I} \end{pmatrix}.$$

Lemma 2 (i) E_0 is always unstable, (ii) E_1 is locally asymptotically stable if $\tau\mu > \beta k\epsilon$ and unstable if the inequality is reversed.

Proof (i) System (1) has the following variational matrix at E_0

$$V(E_0) = \begin{pmatrix} r & 0 & 0 & 0 \\ 0 & -\mu & 0 & 0 \\ 0 & \epsilon & -\tau & 0 \\ 0 & 0 & 0 & -d \end{pmatrix}.$$

Then the characteristic equation of $V(E_0)$ is

$$(\lambda - r)(\lambda + \mu)(\lambda + \tau)(\lambda + d) = 0.$$

Clearly, it has only one positive root and the other three negative roots. Therefore the population-free equilibrium point E_0 is always unstable.

(ii) The variational matrix of system (1) at E_1 is given by

$$V(E_1) = \begin{pmatrix} -r & -r & -\beta k & 0 \\ 0 & -\mu & \beta k & 0 \\ 0 & \epsilon & -\tau & 0 \\ 0 & 0 & 0 & -d \end{pmatrix}.$$

Thus, the characteristic equation of $V(E_1)$ is given by

$$(\lambda + r)(\lambda + d)\{\lambda^2 + \lambda(\tau + \mu) + \tau\mu - \beta k\epsilon\} = 0.$$

Here, all the eigenvalues of the above equation has negative real part if $\tau\mu > \beta k\epsilon$. So the equilibrium point E_1 is locally asymptotically stable if the above condition holds otherwise unstable. $\square$

Lemma 3 E_2 is locally asymptotically stable if (i) $\tau < \frac{k\beta\epsilon}{\mu} < r$ and (ii) $d < \frac{b\bar{I}}{m+\bar{I}}$.

Proof The variational matrix of system (1) at E_2 is given by

$$V(E_2) = \begin{pmatrix} -\frac{r\bar{S}}{k} - \frac{r\bar{S}}{k} - \beta\bar{S} & 0 \\ \beta\bar{E} & -\mu & \beta\bar{S} & -\frac{a\bar{I}}{m+\bar{I}} \\ 0 & \epsilon & -\tau & 0 \\ 0 & 0 & 0 & -d + \frac{b\bar{I}}{m+\bar{I}} \end{pmatrix}.$$

Therefore, the characteristic equation of $V(E_2)$ is

$$\left(\lambda + d - \frac{b\bar{I}}{m+\bar{I}}\right)(\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3) = 0. \quad (2)$$

$$\text{where } \begin{cases} a_1 = r\frac{\bar{S}}{k} + \mu + \tau > 0 \\ a_2 = r\frac{\bar{S}}{k}(\mu + \tau + \beta\bar{E}) > 0 \\ a_3 = \frac{r\beta\tau\bar{S}\bar{E}}{k} + \beta^2\epsilon\bar{S}\bar{E} > 0. \end{cases} \quad (3)$$

Clearly, one of the eigenvalues, say $-d + \frac{b\bar{I}}{m+\bar{I}}$, which is negative, follows from the assumption. Further, we note that $a_1, a_2, a_3 > 0$ and also $a_1a_2 - a_3 > 0$ by the inequality (i) of Lemma 3 and hence all the eigenvalues of the characteristic (2) have negative real parts. Therefore, the equilibrium point E_2 is locally asymptotically stable. $\square$

Finally, the variational matrix at the interior equilibrium point E^* can be written as

$$V(E^*) = \begin{pmatrix} -\frac{rS^*}{k} & -\frac{rS^*}{k} & -\beta S^* & 0 \\ \beta E^* & -\mu - \frac{maP^*}{(m+I)^2} & \beta S^* & -\frac{aI^*}{m+I^*} \\ 0 & \epsilon & -\tau & 0 \\ 0 & \frac{mbP^*}{(m+I^*)^2} & 0 & 0 \end{pmatrix}.$$

Then the characteristic equation of $V(E^*)$ becomes

$$\lambda^4 + p_1\lambda^3 + p_2\lambda^2 + p_3\lambda + p_4 = 0 \quad (4)$$

$$\text{where } \begin{cases} p_1 = -b_{11} - b_{22} - b_{33} > 0 \\ p_2 = b_{11}b_{22} + b_{11}b_{33} - b_{23}b_{32} - b_{24}b_{42} + b_{22}b_{33} - b_{12}b_{21} \\ p_3 = b_{11}b_{23}b_{32} + b_{11}b_{24}b_{42} - b_{11}b_{22}b_{33} + b_{24}b_{42}b_{33} + b_{12}b_{21}b_{33} - b_{13}b_{21}b_{32} \\ p_4 = -b_{11}b_{24}b_{42}b_{33} > 0 \end{cases} \quad (5)$$

$$b_{11} = -\frac{rS^*}{k}, b_{12} = -\frac{rS^*}{k}, b_{13} = -\beta S^*, b_{21} = \beta E^*, b_{22} = -\mu - \frac{maP^*}{(m+I^*)^2}, b_{23} = \beta S^*, b_{24} = -\frac{aI^*}{m+I^*}, b_{32} = \epsilon, b_{33} = -\tau, b_{42} = \frac{mbP^*}{(m+I^*)^2}, b_{14} = b_{31} = b_{34} = b_{41} = b_{43} = b_{44} = 0.$$

Clearly, $p_1 > 0$, $p_3 > 0$, $p_4 > 0$. The expressions for $p_1p_2 - p_3$ and $p_1p_2p_3 - p_1^2p_4$ in system parameters are quite complicated and hence we cannot say about the signs of the above expressions. So we will show local stability of the interior equilibrium point by giving a numerical example.

We now state and prove the global stability of the interior equilibrium point with the help of Lyapunov function. From the global stability criteria, one can ensure that disease is endemic.

Theorem 1 Suppose the interior equilibrium point E^* is locally asymptotically stable in R_+^4 , then it is globally asymptotically stable if the following conditions hold :

- (i) $\mu > \frac{P^*}{m+I^*}$
- (ii) $4(\mu - \frac{P^*}{m+I^*})(\frac{r}{k} + \frac{\beta}{2}) > \tau(\frac{r}{k} + \beta E^*)^2 + \frac{3\beta}{4}(\beta k + \epsilon)(\frac{r}{k} + \beta E^*) + \frac{r}{k}(\beta k + \epsilon)^2$
- (iii) $\det(M) > 0$.

Proof Define a positive definite real valued function

$$V(S, I, E, P) = S - S^* - S^* \ln \frac{S}{S^*} + \frac{1}{2}(I - I^*)^2 + \frac{1}{2}(E - E^*)^2 + \frac{1}{2}(P - P^*)^2 \quad (6)$$

V will be a Lyapunov function if $\dot{V} \leq 0$ when $(S, I, E, P) \neq (0, 0, 0, 0)$. Now the time derivative of V along the solution of (1) is given by

$$\begin{aligned} \frac{dV}{dt} &= (S - S^*) \frac{S}{S^*} + (I - I^*)\dot{I} + (E - E^*)\dot{E} + (P - P^*)\dot{P} \\ &= (S - S^*)\{r(1 - \frac{S+I}{k}) - \beta E\} + (I - I^*)\{\beta S E - \mu I - \frac{aIP}{m+I}\} \\ &\quad + (E - E^*)\{\epsilon I - \tau E\} + (P - P^*)\{P(-d + \frac{bI}{m+I})\} \\ &= -\frac{r}{k}(S - S^*)^2 - \frac{r}{k}(S - S^*)(I - I^*) - \beta(S - S^*)(E - E^*) + \beta S(I - I^*)(E - E^*) \\ &\quad + \beta E^*(S - S^*)(I - I^*) - \mu(I - I^*)^2 - \frac{I(P - P^*)(I - I^*)}{m+I} \\ &\quad - \frac{mP^*(I - I^*)^2}{(m+I)(m+I^*)} + \epsilon(I - I^*)(E - E^*) \\ &\quad - \tau(E - E^*)^2 - d(P - P^*)^2 + \frac{bI(P - P^*)^2}{m+I} + \frac{P^*(I - I^*)(P - P^*)}{(m+I)(m+I^*)} \\ &\leq -\frac{r}{k}(S - S^*)^2 - \{\mu - \frac{P^*}{m+I^*}\}(I - I^*)^2 - \tau(E - E^*)^2 - \{d - \frac{bk}{m}\}(P - P^*)^2 \\ &\quad + (\frac{r}{k} + \beta E^*)|S - S^*||I - I^*| + \beta|S - S^*||E - E^*| + (\beta k + \epsilon)|I - I^*||E - E^*| \\ &\quad + (\frac{k}{m} + \frac{P^*}{m(m+I^*)})|E - E^*||P - P^*| \\ &= -X^T M X, \end{aligned}$$

where $X^T = \{|S - S^*|, |I - I^*|, |E - E^*|, |P - P^*|\}$ and $M = [a_{ij}]_{4 \times 4}$. Elements of the matrix M are given by

$$\begin{aligned} a_{11} &= \frac{r}{k}, \quad a_{22} = \mu - \frac{P^*}{m+I^*}, \quad a_{33} = \tau, \quad a_{44} = d - \frac{bk}{m}, \quad a_{12} = a_{21} = -\frac{1}{2}\left(\frac{r}{k} + \beta E^*\right), \\ a_{13} &= a_{31} = -\frac{\beta}{2}, \quad a_{14} = a_{41} = 0, \quad a_{23} = a_{32} = -\frac{\beta k + \epsilon}{2}, \\ a_{24} &= a_{42} = -\frac{1}{2}\left(\frac{k}{m} + \frac{P^*}{m(m+I^*)}\right), \quad a_{34} = a_{43} = 0. \end{aligned}$$

Hence, M is positive definite if

- (i) $\mu > \frac{P^*}{m+I^*}$
- (ii) $4(\mu - \frac{P^*}{m+I^*})(\frac{r\tau}{k} + \frac{\beta}{2}) > \tau(\frac{r}{k} + \beta E^*)^2 + \frac{3\beta}{4}(\beta k + \epsilon)(\frac{r}{k} + \beta E^*) + \frac{r}{k}(\beta k + \epsilon)^2$ and
- (iii) $\det(M) > 0$.

Conditions of the theorem imply that M is positive definite and hence $\dot{V} < 0$ and consequently, V is a Lyapunov function with respect to all solutions in the interior of the positive orthant. So E^* is globally asymptotically stable. $\square$

Bifurcation analysis deals with a structurally unstable system. This subject is a branch of mathematics that ensures a certain qualitative change of the dynamical systems with respect to some parameters. A small change in parameter creates a topological transform. In case of local bifurcation theory, it is known that the system depends on control parameters as parameter varies, but it is still unknown in case of non-hyperbolic equilibria. Different types of bifurcations can be obtained depending on model parameters [42]. In this work, the parameters d and β have been chosen for the transcritical bifurcation and β has been taken for Hopf-bifurcation of system (1).

5 Local bifurcation analysis

In this section, we shall use Sotomayor's theorem [40] to investigate the local bifurcation around the equilibrium points of system (1). The existence of non-hyperbolic equilibrium point is a necessary but not sufficient condition for bifurcation to occur. Therefore, we choose a parameter that gives zero eigenvalue to the variational matrix at the equilibria. Now rewrite system (1) in the form:

$$\begin{aligned} \frac{dX}{dt} &= F(X) \text{ where } X = (S, I, E, P)^t \text{ and } F = (F_1, F_2, F_3, F_4) \text{ where} \\ F_1 &= S \left[r \left(1 - \frac{S+I}{k} \right) - \beta E \right], \quad F_2 = \beta S E - \mu I - \frac{a I P}{m+I}, \quad F_3 = \epsilon I - \tau E, \\ F_4 &= P \left(-d + \frac{b I}{m+I} \right) \end{aligned}$$

Then, according to variational matrix of system (1), we obtain the following for non-zero vector $V = (v_1, v_2, v_3, v_4)^t$:

$$\begin{aligned} D^2 F(S, I, E, P)(V, V) &= \frac{\partial^2 F}{\partial S^2} v_1^2 + \frac{\partial^2 F}{\partial S \partial I} v_1 v_2 + \frac{\partial^2 F}{\partial I \partial S} v_2 v_1 + \frac{\partial^2 F}{\partial I^2} v_2^2 + \frac{\partial^2 F}{\partial S \partial E} v_1 v_3 \\ &\quad + \frac{\partial^2 F}{\partial E \partial S} v_3 v_1 + \frac{\partial^2 F}{\partial S \partial P} v_1 v_4 + \frac{\partial^2 F}{\partial P \partial S} v_4 v_1 + \frac{\partial^2 F}{\partial E^2} v_3^2 \\ &\quad + \frac{\partial^2 F}{\partial I \partial E} v_2 v_3 + \frac{\partial^2 F}{\partial E \partial I} v_3 v_2 + \frac{\partial^2 F}{\partial I \partial P} v_2 v_4 + \frac{\partial^2 F}{\partial P \partial I} v_4 v_2 \\ &\quad + \frac{\partial^2 F}{\partial E \partial P} v_3 v_4 + \frac{\partial^2 F}{\partial P \partial E} v_4 v_3 + \frac{\partial^2 F}{\partial P^2} v_4^2 \end{aligned}$$

Now, after simple calculations we obtain that,

$$D^2F(S, I, E, P)(V, V) = \begin{pmatrix} -\frac{2r}{k}v_1^2 - \frac{2r}{k}v_1v_2 - 2\beta v_1v_3 \\ 2\beta v_1v_3 \\ 0 \\ \frac{2bm}{(m+I)^2}v_2v_4 \end{pmatrix}$$

and

$$D^3F(S, I, E, P)(V, V, V) = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

Thus, system (1) has no pitchfork bifurcation. The local bifurcation near the equilibrium points is investigated in the following theorems:

Theorem 2 *System (1) undergoes a transcritical bifurcation near the axial equilibrium point E_1 but no saddle node bifurcation when the parameter β crosses the critical value $\beta^* = \frac{\mu\tau}{k\epsilon}$.*

Proof One of the eigenvalues of the variational matrix $V(E_1)$ will be zero if $\beta = \beta^* = \frac{\mu\tau}{k\epsilon}$. Now the variational matrix of system (1) at E_1 with zero eigenvalue is given by

$$V(E_1) = \begin{pmatrix} -r & -r & -\beta k & 0 \\ 0 & -\mu & \beta k & 0 \\ 0 & \epsilon & -\tau & 0 \\ 0 & 0 & 0 & -d \end{pmatrix}$$

Let $V = (v_1, v_2, v_3, v_4)^t$ be the eigenvector corresponding to eigenvalue $\lambda = 0$. Thus $V = (\frac{\tau(r+\mu)}{\epsilon r}, \frac{\tau}{\epsilon}, 1, 0)^t$. Also, let $W = (w_1, w_2, w_3, w_4)^t$ represents the corresponding eigenvector of $V(E_1)^t$ to the eigenvalues of $\lambda = 0$. Hence $V(E_1)^t W = 0$ gives that $W = (0, \frac{\epsilon}{\mu}, 1, 0)^t$. Now $F_\beta(E_1, \beta^*) = (0, 0, 0, 0)^t$, $F_\beta(E_1, \beta)$ represents the derivative of $F = (F_1, F_2, F_3, F_4)^t$ with respect to β . Then we get $W^t[F_\beta(E_1, \beta^*)] = 0$. $\square$

Thus, according to Sotomayor's theorem, system (1) has no saddle-node bifurcation at $\beta = \beta^*$.

Again

$$DF_\beta(E_1, \beta^*) = \begin{pmatrix} 0 & 0 & -k & 0 \\ 0 & 0 & k & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

Then, $W^t[DF_\beta(E_1, \beta^*)V] = \frac{k\epsilon}{\mu} \neq 0$.

Now

$$D^2F(E_1, \beta^*)(V, V) = \begin{pmatrix} -\frac{2r}{k}v_1^2 - \frac{2r}{k}v_1v_2 - 2\beta v_1v_3 \\ 2\beta v_1v_3 \\ 0 \\ 0 \end{pmatrix}$$

Therefore, $W^t[D^2F(E_1, \beta^*)(V, V)] = 2\beta v_1v_3w_3 \neq 0$.

Thus, according to Sotomayor's theorem, system (1) has a transcritical bifurcation at E_1 when the parameter β crosses the critical value β^* . Furthermore, as the characteristic equation of $V(E_1)$ has no purely imaginary eigenvalues, no Hopf bifurcation can occur.

Theorem 3 System (1) admits a transcritical bifurcation but no saddle-node bifurcation around the equilibrium point E_2 when d crosses the critical value $\bar{d} = \frac{b\bar{I}}{m+I}$.

Proof Proof is similar to the proof of Theorem 2. $\square$

6 Hopf-bifurcation around E^*

In this section, we will study whether the Hopf-bifurcation occurs or not of system (1). In our system, β is a crucial parameter; we will show that Hopf bifurcation occurs for system (1) at a critical value $\beta = \beta_0$ using the result developed in [41, 42].

Theorem 4 If the indirect transmission rate β of the disease crosses a critical value β_0 , then the system undergoes a Hopf-bifurcation around its positive equilibrium point E^* if the following conditions hold:

- (i) $p_i(\beta_0) > 0, \forall i = 1, 2, 3, 4$,
- (ii) $p_1(\beta_0)p_2(\beta_0) > p_3(\beta_0)$,
- (iii) $p_1(\beta_0)p_2(\beta_0)p_3(\beta_0) - p_3^2(\beta_0) + p_1^2(\beta_0)p_4(\beta_0) = 0$, and
- (iv) $\frac{du_i}{d\beta} \neq 0, i=1,2,3,4$ at $\beta = \beta_0$.

Proof Consider, the eigenvalues of the characteristic (4) is of the form $\lambda_i = u_i + i v_i$. Put these in (4) and separating real and imaginary parts we get

$$u^4 - 6u^2v^2 + v^4 + p_1(u^3 - 3uv^2) + p_2(u^2 - v^2) + p_3u + p_4 = 0 \quad (7)$$

$$-4uv^3 + 4u^3v + p_1(3u^2 - v^2)v + 2p_2uv + p_3v = 0 \quad (8)$$

From (8), we get $f(u) = v^2 = \frac{1}{4u+p_1}(4u^3 + 3p_1u^2 + 2p_2u + p_3)$

Substituting the value of v^2 in (7), we get

$$u^4(4u + p_1)^2 + (4u^3 + 3p_1u^2 + 2p_2u + p_3)^2 + (4u^3 + 3p_1u^2 + 2p_2u + p_3)(-6u^2 - 3p_1u - p_2)(4u + p_1) + (p_1u^3 + p_2u^2 + p_3u + p_4)(4u + p_1)^2 = 0$$

Now, differentiating w.r.t β at $\beta = \beta_0$ we get,

$$\left[\frac{du}{d\beta} \right]_{\beta=\beta_0} = \frac{(-\frac{d}{d\beta})(p_1p_2p_3 - p_3^2 - p_1^2p_4)}{4p_3(p_2 - p_3) - 2p_1(p_2^2 - p_1p_3) + 8p_1p_2} \neq 0$$

So the system has a Hopf-bifurcation at a critical value β_0 around the interior equilibrium point E^* . $\square$

The conditions for obtaining Hopf-bifurcation are too unwieldy and complicated to be biologically interpretable. Thus, to analyze the model, we numerically simulated population dynamics with β less than (Fig. 4a) and greater than (Fig. 4b) β_0 .

7 Persistence

Persistence means long-term survival of all populations in the future time that does not depend on the initial populations. Mathematically, it means that strictly positive solutions

do not have omega limit points on the boundary of the non-negative cone. We have shown the persistence by using Butler–McGehee lemma [43].

Theorem 5 Suppose $d < \frac{b\bar{I}}{m+I}$, then system (1) is uniformly persistent.

Proof Consider, α be any point in the positive octant and $o(\alpha)$ be any orbit through the point $\alpha = (S, I, E, P)$. $\Omega(\alpha)$ is the omega limit set of $o(\alpha)$, which is bounded. Suppose E_0 is not in $\Omega(\alpha)$. If it belongs to the region $\Omega(\alpha)$ then by Butler–McGehee lemma [43], there exist a point u in $\Omega(\alpha) \cap W^s(E_0)$ where $W^s(E_0)$ be the strong manifold of E_0 . Since $o(u)$ lies in $\Omega(\alpha)$ and $W^s(E_0)$ is the $I - E - P$ plane, we conclude that $o(u)$ is unbounded, which is a contradiction.

Similarly, we can show that $E_1 \notin \Omega(\alpha)$. If $E_1 \in \Omega(\alpha)$ then by Butler–McGehee lemma [43], there exist a point u in $\Omega(\alpha) \cap W^s(E_1)$ where $W^s(E_1)$ be the strong manifold of E_1 . Since $o(u)$ lies in $\Omega(\alpha)$ and $W^s(E_1)$ is the $S - P$ direction, we conclude that $o(u)$ is unbounded, which is a contradiction. Next we show that $E_2 \notin \Omega(\alpha)$, by the condition $d < \frac{b\bar{I}}{m+I}$, E_2 is a saddle point. $W^s(E_2)$ is the P direction, hence $o(u)$ is unbounded in $\Omega(\alpha)$, again a contradiction.

Thus, $\Omega(\alpha)$ lies in the positive octant and system (1) is persistent. Since system (1) is bounded, by the main theorem of Butler et al. [44], the system is uniformly persistent.

The condition of Theorem 5 indicates that if the death rate of the predator remains a certain threshold value then none of the populations facing extinction. $\square$

Remark 1 If the condition of Theorem 5 is reversed, then system (1) becomes impermanent [45]. This indicates extinction of at least one population from the system.

8 Stochastic analysis of the model

Stochastic perturbations have been introduced in some of the model parameters [21, 46, 47]. Here we allow stochastic perturbations of the variables S, I, E, P around their positive interior equilibrium point E^* , when it is feasible and locally asymptotically stable. Locally stability of E^* is implied by the existence condition of E^* . So in the model we assume that stochastic perturbation of the variables around their values at E^* are of white noise type, which are proportional to the distances of S, I, E, P from values S^*, I^*, E^*, P^* . So system (1) can be written as

$$\begin{aligned} dS &= S \left[r \left(1 - \frac{S+I}{K} \right) - \beta E \right] dt + \sigma_1 (S - S^*) d\xi_t^1 \\ dI &= \left[\beta S E - \mu I - \frac{aIP}{m+I} \right] dt + \sigma_2 (I - I^*) d\xi_t^2 \\ dE &= [\varepsilon I - \tau E] dt + \sigma_3 (E - E^*) d\xi_t^3 \\ dP &= \left[P \left(-d + \frac{bI}{m+I} \right) \right] dt + \sigma_4 (P - P^*) d\xi_t^4 \end{aligned} \quad (9)$$

where $\sigma_i, i=1,2,3,4$ are real constants, $\xi_t^i = \xi_i(t)$, $i=1,2,3,4$ are independent from each other in the standard Wiener processes [48]. We determine whether the dynamical nature of the continuous model (1) is robust with respect to such a kind of stochasticity by investigating the asymptotic stochastic stability behavior of the equilibrium E^* for (9) and by comparing

the results with those obtained for system (1). We will consider (9) as the Ito stochastic differential system.

8.1 Stochastic stability of the positive equilibrium

The SDEs system of (9) can be centered at E^* by the change of the variables

$$u_1 = S - S^*, \quad u_2 = I - I^*, \quad u_3 = E - E^*, \quad u_4 = P - P^*. \quad (10)$$

The linearized SDEs around the positive E^* take the form

$$du(t) = f(u(t))dt + g(u(t))d\xi(t) \quad (11)$$

where $u(t) = \text{col}(u_1(t), u_2(t), u_3(t), u_4(t))$ and

$$f(u(t)) = \begin{pmatrix} -\frac{rS^*}{K} & -\frac{rS^*}{K} & -\beta & 0 \\ \beta E^* & -\mu - \frac{maP^*}{(m+I^*)^2} & \beta S^* - \frac{aI^*}{m+I^*} & \\ 0 & \epsilon & -\tau & 0 \\ 0 & \frac{mbP^*}{(m+I^*)^2} & 0 & 0 \end{pmatrix} u(t)$$

$$g(u(t)) = \begin{pmatrix} \sigma_1 u_1 & 0 & 0 & 0 \\ 0 & \sigma_2 u_2 & 0 & 0 \\ 0 & 0 & \sigma_3 u_3 & 0 \\ 0 & 0 & 0 & \sigma_4 u_4 \end{pmatrix}$$

The positive equilibrium in (11) corresponding to the trivial solution $u(t)=0$.

Let $U=(t \geq t_0) \times R^n$, $t_0 \in R^+$. Consider $V \in C_2^0(U)$ a twice differential function with respect to u and a continuous function with respect to t .

Now we require the following theorem to prove the asymptotically mean square stability of trivial solution of (11).

Theorem 6 [49] Assume that $V(t, u) \in C_2^0(U)$ satisfying the inequalities

$$K_1|u|^p \leq V(t, u) \leq K_2|u|^p, \quad (12)$$

$$LV(t, u) \leq -K_3|u|^p, \quad K_i > 0, \quad p > 0, \quad i = 1, 2, 3. \quad (13)$$

Then, the trivial solution of (11) is exponentially p -stable for $t \geq 0$.

Here, if $p = 2$, then the trivial solution of (11) is globally asymptotically stable in probability.

Again with the reference of [49], we get

$$LV(t, u) = \frac{\partial V(t, u)}{\partial t} + f^T(u) \frac{\partial V(t, u)}{\partial u} + \frac{1}{2} Tr \left[g^T(u) \frac{\partial^2 V(t, u)}{\partial u^2} g(u) \right]$$

where

$$\frac{\partial V}{\partial u} = \text{Col} \left(\frac{\partial V}{\partial u_1}, \frac{\partial V}{\partial u_2}, \frac{\partial V}{\partial u_3}, \frac{\partial V}{\partial u_4} \right),$$

$$\frac{\partial^2 V(t, u)}{\partial u^2} = \frac{\partial^2 V}{\partial u_j \partial u_i}, \quad i, j = 1, 2, 3, 4.$$

and T represents transposition.

Now we state asymptotic mean square stability of the zero solution of (11).

Theorem 7 Assume that $\sigma_1^2 < \frac{2rS^*}{K}$, $\sigma_2^2 < 2\left(\mu + \frac{maP^*}{(m+I^*)^2}\right)$, $\sigma_3^2 < 2\tau$, $\sigma_4^2 < d + 2d_1P^* - \frac{bI^*}{m+I^*}$ and $(\frac{rS^*}{K} - \frac{1}{2}\sigma_1^2)(\tau - \frac{1}{2}\sigma_3^2) > \frac{1}{4}\beta S^*w_1$. Then the zero solution of (11) asymptotically mean square stable.

Proof Consider a Lyapunov function

$$V(u) = \frac{1}{2}(w_1u_1^2 + w_2u_2^2 + w_3u_3^2 + w_4u_4^2)$$

where w_i , $i = 1, 2, 3, 4$ all are real positive constants. It is easy to check that inequalities (12) hold true with $p = 2$.

Therefore

$$\begin{aligned} LV(u) = & w_1[-\frac{rS^*}{K}u_1 - \frac{r}{K}S^*u_2 - \beta S^*u_3]u_1 \\ & + w_2[\beta E^*u_1 - (\mu + \frac{maP^*}{(m+I^*)^2})u_2 + \beta S^*u_3 - \frac{aI^*}{m+I^*}u_4]u_2 \\ & + w_3[\epsilon\mu_2 - \tau\mu_3]u_3 \\ & + w_4[\frac{bI^*}{(m+I^*)^2}u_2 - (d + 2d_1P^* - \frac{bI^*}{m+I^*})u_4]u_4 \\ & + \frac{1}{2}Tr[g^T(u)\frac{\partial^2 V}{\partial u^2}g(u)] \end{aligned}$$

$$\text{Note that } \frac{\partial^2 V}{\partial u^2} = \begin{pmatrix} w_1 & 0 & 0 & 0 \\ 0 & w_2 & 0 & 0 \\ 0 & 0 & w_3 & 0 \\ 0 & 0 & 0 & w_4 \end{pmatrix}$$

and hence

$$g^T(u)\frac{\partial^2 V}{\partial u^2}g(u) = \begin{pmatrix} w_1\sigma_1u_1^2 & 0 & 0 & 0 \\ 0 & w_2\sigma_2u_2^2 & 0 & 0 \\ 0 & 0 & w_3\sigma_3u_3^2 & 0 \\ 0 & 0 & 0 & w_4\sigma_4u_4^2 \end{pmatrix}$$

So

$$\frac{1}{2}Tr[g^T(u)\frac{\partial^2 V}{\partial u^2}g(u)] = \frac{1}{2}[w_1\sigma_1^2u_1^2 + w_2\sigma_2^2u_2^2 + w_3\sigma_3^2u_3^2 + w_4\sigma_4^2u_4^2]. \quad (14)$$

Now if we choose, $w_1\frac{r}{K}S^* = w_2\beta E^*$, $w_2\beta S^* = w_3\epsilon$, and $w_2\frac{a}{m+I^*} = w_4\frac{b}{(m+I^*)^2}$, from (14) it is easy to check that,

$$\begin{aligned} LV(u) = & -\left(\frac{rS^*}{K} - \frac{1}{2}\sigma_1^2\right)w_1u_1^2 - \left[\mu + \frac{maP^*}{(m+I^*)^2} - \frac{1}{2}\sigma_2^2\right]w_2u_2^2 \\ & - \left[\tau - \frac{1}{2}\sigma_3^2\right]w_3u_3^2 - \left[d + 2d_1P^* - \frac{bI^*}{m+I^*} - \frac{1}{2}\sigma_4^2\right]w_4u_4^2 - \beta S^*w_1u_1u_3 \\ = & a_{11}u_1^2 - a_{12}u_1u_3 - a_{22}u_2^2 - a_{31}u_2^2 - a_{32}u_4^2 \end{aligned}$$

where, $a_{11} = -(\frac{rS^*}{K} - \frac{1}{2}\sigma_1^2)w_1$, $a_{31} = [\mu + \frac{maP^*}{(m+I^*)^2} - \frac{1}{2}\sigma_2^2]w_2$, $a_{22} = [\tau - \frac{1}{2}\sigma_3^2]w_3$, $a_{12} = \beta S^*w_1$, $a_{32} = [d + 2d_1P^* - \frac{bI^*}{m+I^*} - \frac{1}{2}\sigma_4^2]w_4$.

It is always negative if $a_{11}a_{22} > \frac{1}{4}a_{12}^2$ that is if $(\frac{rS^*}{K} - \frac{1}{2}\sigma_1^2)(\tau - \frac{1}{2}\sigma_3^2) > \frac{1}{4}\beta S^*w_1$. Hence, the result follows from Theorem 6. $\square$

In this next section, we have performed some numerical simulations to verify our results obtained earlier. Numerical simulations are performed with the help of Matlab R2014a software package for hypothetical set of data.

9 Numerical simulation

In most eco-epidemiological studies, the infection rate plays a crucial role in describing the dynamics of the system. Keeping this in mind, we varied the infection rate β to observe different type of behavior of system (1). Suppose $r = \frac{14}{5}$, $k = 7$, $\mu = 1.5$, $a = 1$, $m = 1$, $\varepsilon = 1$, $\tau = 1$, $d = 0.5$, $b = 1$, our results show that the predator population will become extinct for the parametric values of $\beta = 0.25$ (see Fig. 1a). By increasing the value of β , we observe that the system has a positive equilibrium $(1, 1, 1, 1)$ at $\beta = 2$, which is locally asymptotically stable (see Fig. 1b). A three-dimensional phase diagram represents the stability of system (1) (see Fig. 1c).

Fig. 1 Influence of indirect transmission rate β on predator population: **a** extinction of predator population for $\beta = 0.25$ **b** for $\beta = 2.0$ time series plot indicate coexists of all the species and **c** phase portrait shows local stability of endemic equilibrium point E^* . Other model parameters are $r = \frac{14}{5}$, $k = 7$, $\mu = 1.5$, $a = 1$, $m = 1$, $\varepsilon = 1$, $\tau = 1$, $d = 0.5$, $b = 1$

Fig. 2 The interior equilibrium point E^* does not exist and system (1) is impermanent for parametric values $r = \frac{14}{5}$, $k = 7$, $\beta = 2.5$, $\mu = 1.5$, $a = 1$, $m = 1$, $\varepsilon = 1$, $\tau = 1$, $d = 0.5$, $b = 1$

We observed that by increasing the amount of transmission rate $\beta = 2.5$ stabilizes the system and the interior equilibrium point does not exist, consequently system (1) is impermanent (see Fig. 2).

Again, if we take $\beta = 2.07$ and all other parameter remains the same, then system (1) has a positive equilibrium point $(\frac{1}{3}, 1, \frac{1}{3})$ and we observe that all the species of system (1) enter into an oscillatory state from a stable situation and in this case the system is uniformly persistent (permanent). A three-dimensional phase diagram represents the limit cycle oscillations of system (1) (see Fig. 3).

Furthermore, the interior equilibrium point E^* undergoes a Hopf bifurcation and stability changes occur when the parameter β exceeds its critical value $\beta_0 = 2.049$. To demonstrate the dynamical behaviors of system (1), we have plotted the bifurcation diagram of the system in (β, S, I) plane and its projections on (β, S) and (β, I) plane in Fig. 4.

To show the interplay between the disease transmission rate β and the half saturation constant m in the stability of the system around the interior equilibrium E^* , we plot the stability region of E^* in $\beta - m$ parametric space which shown in Fig. 5. The red region indicates the stable coexistence of all populations.

Fig. 3 Limit cycle appears and the system is permanent for the parametric values $r = \frac{14}{5}$, $k = 7$, $\beta = 2.07$, $\mu = 1.5$, $a = 1$, $m = 1$, $\varepsilon = 1$, $\tau = 1$, $d = 0.5$, $b = 1$

Fig. 4 Phase portrait and bifurcation diagram: **a** system is stable when $\beta = 2.030 < \beta_0 = 2.049$, **b** system loses its stability when $\beta = 2.050 > \beta_0 = 2.049$, **c** bifurcation diagram of system (1) in (β, S, I) plane and its projection on (β, S) and (β, I) planes for $\beta \in (1.5, 2, 5)$ and other model parameters are $r = \frac{14}{5}$, $k = 7$, $\mu = 1.5$, $a = 1$, $m = 1$, $\varepsilon = 1$, $\tau = 1$, $d = 0.5$, $b = 1$

Fig. 5 Stability region of system (1) in β – m plane. The red region represents the stability of the coexistence equilibrium and the green region represents the instability of the coexistence equilibrium point. The model parameters are $r = \frac{14}{5}$, $k = 7$, $a = 1$, $m = 1$, $\varepsilon = 1$, $\tau = 1$, $d = 0.5$, $b = 1$, $\mu \in (0.4, 2)$ and $\beta \in (1, 3)$

10 Discussion

In this paper, we have generalized an existing eco-epidemic model of the dynamics of chronic wasting disease affecting an animal population. Including the dynamics of the predator population, we arrive at a four-dimensional system of coupled nonlinear differential equations. To eradicate diseases, vaccination and prevention policies are mainly used [50], but for fatal diseases like CWD, there is still no vaccination programme. However, it may be possible for a disease affecting a population by allowing in the environment another population which is the predator of the former. It is a well-known fact that infected prey individuals become easy to catch by the predator. This property has been considered in a various number of eco-epidemiological models [18, 20, 23, 24, 29, 51]. In our model, we have considered that the predator consumes infected prey only to control the disease [52, 53]. The predation process follows Holling type II response function. The proposed model is shown to be biologically well-behaved as the system is bounded. The local stability of the system in different steady states has been discussed. Further, the system cannot collapse for any values of parameters, as the population-free equilibrium E_0 is never stable. It is observed that if the carrying capacity of the environment remains below a certain threshold value ($k < \frac{\tau\mu}{\beta\epsilon}$), then the susceptible prey-only equilibrium E_1 is an attractor. If the predator has a large death rate ($d > \frac{b\bar{I}}{m+\bar{I}}$), then predator-free equilibrium E_2 cannot be stable. We have also derived the conditions under which the coexistence equilibrium is globally asymptotically stable, i.e., the disease persists in the populations. For disease eradication, such type of conditions should be avoided. Sotomayor's theorem [40] is applied to ensure the existence of transcritical bifurcation. This bifurcation transforms a susceptible prey-only equilibrium point into an unstable equilibrium point and the predator-free equilibrium to an unstable one. The local existence of limit cycle is obtained through Hopf-bifurcation when the transmission rate β crosses a critical value. Miller et al. [4] address a model for CWD dynamics without considering the growth equations of predator populations for obtaining coexistence results. Furthermore, they have not investigated the effect of environmental noise on the system. These two aspects are analyzed in our model. We have chosen selective predation to eradicate disease in our system. Still non-selective predation can be effective in controlling the disease.

To investigate the effect of environmental noise on the system, we apply stochastic perturbation around the positive equilibrium. Constructing a suitable Lyapunov function and using Ito's formula, we note that the deterministic model is robust with respect to stochastic perturbation. The criterion of stochastic stability depends on the intensities of noise σ_i , $i = 1, 2, 3, 4$. If the intensities of noise are small, the stochastic stability can be achieved under certain conditions. Further, the stochastic stability of the system shows that the intensities σ_i , $i = 1, 2, 3, 4$ have the tolerance limits.

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Compliance with ethical standards

Conflict of interests The authors declare that they have no conflicts of interest.

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