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| RESEARCH ARTICLE

Healthy Predator Crowding on a Delayed Non-autonomous Eco-epidemic System

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| ABSTRACT
This article presents a delayed non-autonomous eco-epidemiological prey-predator model with predator infection. The author considered incubation delay of infection in predator over-crowding among healthy species. The article contains the system solution's existence, boundedness, and uniform persistence under certain conditions. The numerical simulation confirms analytical findings. The analysis of autonomous and non-autonomous models for the same incubation delays shows that the autonomous system leads stability to bifurcation while crossing the threshold value to incubation delay. However, the non-autonomous model leads from stability to chaos while crossing threshold values. Finally, simulating the effect of healthy predator crowding shows that increasing healthy predator crowding helps to remove infection from the environment.

| KEYWORDS

Incubation delay, Periodic solutions, Chaos, Crowding

| ARTICLE INFORMATION

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1. Introduction

Eco-epidemiology is a rapidly evolving field that examines the interplay between ecological dynamics and infectious diseases, particularly through the lens of predator-prey interactions [16]. Understanding how infectious agents spread within these communities is essential for predicting and managing disease dynamics. The transmission of pathogens often occurs via prey species, which can significantly impact predator health and alter overall population dynamics [21]. This complexity necessitates sophisticated modeling approaches that accurately reflect these interactions [1]. Delayed differential equations have emerged as a powerful tool, enabling researchers to incorporate critical aspects such as incubation periods and seasonal variations into their models [2]. Yuan and Mohamad [23, 15] analyze population dynamics through delay differential equations, emphasizing their utility in understanding ecological interactions. Moreover, Kumar et al. [14] investigate the transmission dynamics of tooth cavities in humans, illustrating how mathematical models can address health-related issues across various contexts.
In recent years, the role of incubation delays(the time between infection and the onset of

infectiousness) has gained prominence in eco-epidemiological research [3]. Greenhalgh et.al. [8] emphasize the significance of these delays in determining infection persistence within populations. Their findings indicate that the timing of disease transmission relative to reproductive and ecological parameters can critically influence the stability and health of predator-prey systems. Such delays can impact oscillatory behavior and population stability [7], suggesting that the timing of infections may induce cyclical dynamics that either stabilize or destabilize populations. To enhance the realism of these models, researchers have increasingly incorporated time-varying parameters that reflect seasonal environmental shifts, such as periodic prey growth and fluctuating predation rates. For instance, [24] utilize delay models to examine the effects of Wolbachia on dengue spread in mosquitoes, highlighting the role of environmental factors in shaping disease dynamics.

Recent reviews have identified key principles that underlie successful eco-epidemiological models. [6, 19, 4, 22] provide a comprehensive overview of these principles, such as the utilization of logistic prey growth, bilinear incidence rates for disease transmission, and predator functional responses consistent with empirical observations. Furthermore, the understanding of stability within predator-prey dynamics has been enriched by studies such as those by [13] and [5]. These works investigate the implications of disease presence in predator populations, shedding light on how these dynamics influence overall ecosystem health.

The proposed study aims to present a three-compartment non-autonomous eco- epidemiological model that incorporates incubation delay of disease transmission in predators and crowding among healthy species. The research reveals that for the same value of incubation delay; the non-autonomous model exhibits chaos instead of bifurcation.

Also, the healthy predator overcrowding helps to remove infection from the environment.

The flow of the research paper is as follows: The section 2 presents a non-autonomous eco- epidemic model, including initial conditions and parameter descriptions. Section 3 is the preliminaries section with two subsections. The subsection 3.1 tells about the existence and

uniqueness of the system's solution (1) whereas the subsection 3.2 shows the permanence and uniform persistence of the system (1). In section 4, we have investigated the non-autonomous system (1) and the corresponding autonomous model numerically to verify our analysis. Section 5 talks about the analytical and numerical results and their real-world understanding.

2. Formation of mathematical model: The present section contains a delayed non-autonomous prey-predator model incorporating healthy prey, diseased prey, and an infected predator with population densities $\boldsymbol{S(t)}$, $\boldsymbol{P(t)}$, and $\boldsymbol{I(t)}$, respectively. All three populations lie within the considered region for time $\boldsymbol{t > 0}$. The parameter description of the proposed model is: $\boldsymbol{r}$ is the logistic growth rate of the prey species, and $\boldsymbol{\lambda}$ is the disease contact rate among predators. Here, we consider that the spread of disease follows the linear functional response $\boldsymbol{\lambda S(t)P(t)}$. The fatality rate of prey $\boldsymbol{\eta_1}$ and the mortality rates of healthy and infected predators are $\boldsymbol{\eta_2}$ and $\boldsymbol{\eta_3}$ respectively. The healthy and infected predator hunts prey at the rate of $\boldsymbol{\alpha_1}$ and $\boldsymbol{\alpha_2}$, respectively. The conversion rate of prey to the healthy predator and infected predator is $\boldsymbol{\gamma_1}$ and $\boldsymbol{\gamma_2}$, respectively. Finally, $\boldsymbol{d_1}$ and $\boldsymbol{d_2}$ are crowding coefficients of prey and healthy predator. Here, we didn't consider the crowding of an infected predator as it is incapable move another location in search of food. All the earlier assumptions lead to the underlying non-autonomous model.

$$\left.\begin{aligned}
\frac{dS(t)}{dt} &= b(t)S(t) - \alpha_1(t)P(t)S(t) - \alpha_2(t)S(t)I(t) - \eta_1(t)S(t) - d_1S^2(t),\\
\frac{dP(t)}{dt} &= \gamma_1(t)S(t)P(t) - \lambda(t)P(t)I(t) - d_2(t)P^2(t) - \eta_2(t)P(t),\\
\frac{dI(t)}{dt} &= \lambda(t)P(t-\tau)I(t-\tau) + \gamma_2(t)S(t)I(t) - \eta_3(t)I(t).
\end{aligned}\right\} \quad (1)$$

The author considered all system parameters positive, real, bounded, and continuous. All the

model parameters are time-dependent because these parameters are affected by seasonal factors like air pressure, rain, temperature, etc. The author shows seasonality in the model by taking all model parameters as sinusoidal functional response presented in 12.

Further, since an infected individual takes time to become infectious, there is a time lag between infection and the beginning of infectiousness called incubation delay. the author incorporate time τ (1). Where τ is the discrete-time delay. Consider $\jmath: [-\tau, 0] \to \mathbb{R}^3$ as a Banach space of continuous functions, denoted by $\mathbb{C}$, equipped with the norm defined as:

$$\| \jmath \| = \sup_{-\tau \le \varphi \le 0} \{|\jmath_1(\varphi)|, |\jmath_2(\varphi)|, |\jmath_3(\varphi)|\},$$

Where $\jmath = (\jmath_1, \jmath_2, \jmath_3)$ and the initial conditions of system (1) are given as $S(\varphi) = \jmath_1(\varphi)$, $P(\varphi) = \jmath_2(\varphi)$, $I(\varphi) = \jmath_3(\varphi)$, $\varphi \in [-\tau, 0]$. Consider $\jmath = (\jmath_1, \jmath_2, \jmath_3) \in \mathbb{C}([-\tau, 0], \mathbb{R}^3)$ as the initial function of continuous functions mapping the interval $[-\tau, 0]$ into $\mathbb{R}^3$. For biological premises, the initial functions are considered to be:

$$\jmath_i(\varphi) \ge 0, \varphi \in [-\tau, 0], i = 1,2,3. \quad (2)$$

3. Preliminaries: The following subsections demonstrate fundamental results such as the permanence, existence, and uniqueness of the solution to model $(\boldsymbol{1})$.

3.1 Existence and uniqueness: The system $(\boldsymbol{1})$ possesses a unique solution over the interval $[-\boldsymbol{\tau}, \infty)$ for the initial conditions $(\boldsymbol{2})$.

Proof. We know that the right hand side of the system (1) is locally Lipschitzian and completely continuous over $\mathbb{C}$, has a unique solution $(S(t), P(t), I(t))$ of model (1) satisfying initial conditions (2) on $[0, \alpha)$ where $0 < \alpha \leq +\infty$.

Now, referring to the first equation of model (1), we can observe that:

$$S(t) = S(0) \quad \exp \int_0^t [b(v) - \alpha_1(v)P(v) - \alpha_2(v)I(v) - \eta_1(v) - d_1S(v)]dv > 0 \quad \forall \quad t \geq 0.$$

Also, using model (1)'s second equation, we have

$$P(t) = P(0) \exp \int_0^t [\gamma_1(v)S(v) - \lambda(v)I(v) - d_2(v)P(v) - \eta_2(v)]dv > 0 \quad \forall \quad t \geq 0.$$

Next, we need to show that $I(t) > 0 \quad \forall\, t \geq 0$. Suppose that $I(t) \leq 0 \quad \forall \quad t \geq 0$, then $\exists$ a $t_1 > 0$ such that $I(t_1) = 0$ and $I(t) \geq 0 \quad \forall t \in [-\tau, t_1]$. Moreover,

$$\frac{dI(t)}{dt} \geq \gamma_2(t)S(t)I(t) - \eta_3(t)I(t) \qquad \forall t \in [0, t_1]. \tag{3}$$

$$I(t) \geq I(0) \quad \exp \int_0^t [\gamma_2(v)S(v) - \eta_3(v)]dv > 0 \qquad \forall t \in [0, t_1].$$

This contradicts our initial supposition. Thus, $I(t) > 0, \quad \forall \quad t \geq 0$.

3.2 Permanence: This subsection explores the enduring nature of the system $(\mathbf{1})$, using the initial conditions $(\mathbf{2})$. We shall demonstrate how the system will exhibit uniform persistence under specific conditions. Moreover, all integrants of system $(\mathbf{1})$ will survive over a long time under certain conditions. For a bounded and continuous function, $\boldsymbol{g(t)}$ is specified on the interval. $[\mathbf{0}, +\infty)$, let $\boldsymbol{g^l = inf_{t\leq 0} g(t)}$ and $\boldsymbol{g^u = sup_{t\geq 0} g(t)}$.

Definition 2.1 If p_i and q_i, $i = 1,2,3$, are positive constants that ensure:

$$p_1 \leq \lim_{t\to\infty}\inf S(t) \leq \lim_{t\to\infty}\sup S(t) \leq q_1,$$

$$p_2 \leq \lim_{t\to\infty}\inf P(t) \leq \lim_{t\to\infty}\sup P(t) \leq q_2,$$

$$p_3 \leq \lim_{t\to\infty}\inf I(t) \leq \lim_{t\to\infty}\sup I(t) \leq q_3,$$

holds for each solution $(S(t), P(t), I(t))$ of model (1) satisfying initial conditions (2), then model described by (1) is considered uniformly persistent. Let's consider the system [20],

$$\dot{y} = \mu y(t - \tau) - \beta y(t) - \sigma y^2(t),$$

where $\mu, \beta, \sigma, \tau > 0; y(t) > 0$ for $-\tau \leq t \leq 0$. Then, we have

$$\lim_{t\to\infty} y(t) = \begin{cases} \dfrac{\mu - \beta}{\sigma} & \mu > \beta \\ 0 & \mu < \beta. \end{cases}$$

Assume that $X(t) = (S(t), P(t), I(t))$ represent solution of the model (1) satisfying initial conditions (2). Consider that model (1) adheres to the following conditions:

$$\frac{\eta_2^l}{(\gamma_1-\lambda)^u} < K^* = max\left\{\left(\frac{b^u}{d_1^l}\right), \left(\frac{b^u}{d_1^l}\right)\left(\frac{\gamma_2^u}{\eta_3^l}\right)\right\} \quad (4)$$

then ∃ a $\Upsilon_3 > 0$ so that

$$S(t), P(t) < K_3, I(t) \leq K_4 \quad \forall t \geq \Upsilon_3, \quad (5)$$

where $K_3 > K^*$ and $K_4 > \frac{(\gamma_1-\lambda)^u K^* - \eta_2^l}{d_2^l}$.

Proof. Let $K_1 > \left(\frac{b^u}{d_1^l}\right)$. Considering the system's first equation (1), we have $\dot{S}(t) \leq S(t)[b(t) - d_1(t)S(t)] \leq S(t)\left[b^u - d_1^l S(t)\right]$. Thus, if $S(0) \leq K_1$, then $S(t) \leq K_1, \quad \forall t \geq 0$. If $S(0) > K_1$ and let $-\beta_1 = K_1\left(b^u - d_1^l K_1\right), \beta_1 > 0$, then ∃ an $\in > 0$, such that if $t \in [0, \in), S(t) > K_1$ and we have $\dot{S}(t) < -\beta_1 < 0$. Therefore, ∃ a $\Upsilon_1 > 0$ and $\Upsilon_3 > 0$ such that $S(t) \leq K_1$ and $P(t) \leq K_4 \quad \forall t \geq \Upsilon_1, \Upsilon_3$.

Using system (1)'s third equation, we get

$$\dot{I}(t) \leq \gamma_2^u(t) K_1 I(t) - \eta_3^l I(t), \quad t \geq \Upsilon_1 + \tau. \quad (6)$$

We deduce from Lemma (3.2) that ∃ a $\Upsilon_2 \geq \Upsilon_1 + \tau$ so that $I(t) \leq K_2, \quad \forall t \geq \Upsilon_2$, where $K_2 > \frac{\gamma_2^u K_1}{\eta_3}$.

It is feasible to select K_1 sufficiently in proximity to $\left(\frac{b^u}{d_1^l}\right)$. Hence, $S(t), I(t) \leq K_3$, where

$$K_3 > K^* = max\left\{\left(\frac{b^u}{d_1^l}\right), \left(\frac{b^u}{d_1^l}\right)\left(\frac{\gamma_2^u}{\eta_3^l}\right)\right\}, \quad \forall t \geq \Upsilon_2.$$

Using system (1)'s second equation, we obtain

$$\dot{P}(t) \leq P(t)\left[(\gamma_1 - \lambda)^u K_3 - \eta_2^l - d_2^l P(t)\right] \quad \forall t \geq \Upsilon_3.$$

Therefore, ∃ a $\Upsilon_3 > 0$ so that $P(t) \leq K_4 \quad \forall t \geq \Upsilon_3$, where $K_4 > \frac{(\gamma_1-\lambda)^u K^* - \eta_2^l}{d_2^l}$, since K_3 can be chosen sufficiently close to K^*.

The system (1), considering initial condition (2), exhibits uniform persistence when the subsequent condition is satisfied:

$$\frac{\eta_2^l}{(\gamma_1 - \lambda)^2} < K^* = \max\left\{\left(\frac{b^u}{d_1^l}\right), \left(\frac{b^u}{d_1^l}\right)\left(\frac{\gamma_2^u}{\eta_3^l}\right)\right\}$$

$$< \min\left\{\frac{d_2^l b^l + \alpha_1^u \eta_2^l - \frac{(\alpha_2+\eta_1)^u \eta_2^u d_2^u}{(\gamma_1-\lambda)^l}}{(\eta_1-\lambda)^u d_2^l + \alpha_1^u (\gamma_1-\lambda)^u}, \frac{\lambda^l (d_2^l b^l + \alpha_1^u \eta_2^l) + \gamma_2^u \eta_2^l (\alpha_2+\eta_1)^u}{\lambda\{(\alpha_2+\eta_1)^u d_2^l + \alpha_1^u (\gamma_1-\lambda)^u\} + (\gamma_1-\lambda)^u \gamma_2^u (\alpha_2+\eta_1)^u}\right\} \quad (7)$$

Proof. Assume that $X(t) = (S(t), P(t), I(t))$ is a solution of the model (1) satisfying initial conditions (2), with the help of Theorem 3.2, we have

$$\dot{S}(t) \geq S(t)[(b^l - (\alpha_2 - \eta_1)^u K_3 + \alpha_1^u K_4) - d_1^u], \quad \forall t \geq \Upsilon_3.$$

If $(b^l - (\alpha_2 - \eta_1)^u K_3 + \alpha_1^u K_4) > 0$, then we can chose m_1 such that

$$0 < m_1 < \frac{[b^l - (\alpha_2 - \eta_1)^u K_3 + \alpha_1^u K_4]}{d_1^u} \Rightarrow (b^l - (\alpha_2 - \eta_1)^u K_3 + \alpha_1^u K_4) - d_1^u m_1 > 0.$$

If $S(\Upsilon_3) \geq m_1$, then $S(t) \geq m_1 \quad \forall t \geq \Upsilon_3$. If $S(\Upsilon_3) < m_1$, and let $\mu_1 = S(\Upsilon_3)[b^l - \lambda^u K_3 - \alpha_1^u K_4 - \eta_1] - \eta_1^u m_1 > 0$, then ∃ an $\in > 0$, such that $S(t) < m_1$, and $\dot{S} > \mu_1 > 0, \quad \forall t \in [\Upsilon_3, \Upsilon_3 + \in)$. Therefore, ∃ a $\Upsilon_4 > \Upsilon_3 > 0$, such that $S(t) \geq m_1 \quad \forall t \geq \Upsilon_4$.

Using system (1)'s third equation, we obtain

$$\dot{I}(t) \geq \lambda^l K_3 P(t-\tau) I(t-\tau) + \gamma_2^u m_1 I(t) - \eta_3^u I(t). \tag{8}$$

$$\geq \lambda^l K_3 P(t-\tau) I(t-\tau) - \in_2 I^2(t) + \gamma_2^u m_1 I(t) - \eta_3^u I(t) \quad \forall t \geq \Upsilon_4 + \tau, \tag{9}$$

where $\in_2$ represents any non-negative real number. Suppose that $\lambda^l m_1 > \alpha_2^u K_3 + \eta^u$, then by using Theorem (3.2) we may deduce that ∃ a $\Upsilon_5 \geq \Upsilon_4 + \tau$ so that $P(t) \geq m_2, \quad \forall t \geq \Upsilon_5$, where $m_2 < \frac{\lambda^l M_3 + \alpha_2^u m_1 + \eta_3^u}{\in_2}$. Hence,

$$S(t), P(t) \geq m_3, \quad \forall t \geq \Upsilon_5,$$

where $m_3 < m^* = min\{m_1, m_2\}$.

Using system (1)'s second equation, we obtain

$$\dot{P}(t) \geq P(t)[(\gamma_1 - \lambda)^l m_3 - \eta_2^u - d_2^u P(t)].$$

If $(\gamma_1 - \lambda)^l m_3 > \eta_2^u$, then we can select m_4 so that $0 < m_4 < \frac{(\gamma_1 - \lambda)^l m_3 - \eta_2^u}{d_2^u}$. If $P(\Upsilon_5) \geq m_4$, then $P(t) \geq m_4, \quad 0.25em \forall t \geq \Upsilon_5$. If $P(\Upsilon_5) < m_4$ and suppose $\mu_2 = P(\Upsilon_5)[(\gamma_1 - \lambda)^l m_3 - \eta_2^u - m_4 d_2^u] > 0$, then ∃ an $\in_3 > 0$ such that $P(t) < m_4$ and $\dot{P}(t) > \mu_2 > 0, \quad 0.25em \forall t \in [\Upsilon_5, \Upsilon_5 + \in_3)$. Thus, ∃ a $\Upsilon_6 > \Upsilon_5 > 0$ such that $\Upsilon(t) \geq m_4 \quad \forall t > \Upsilon_6$. After considering the above analysis, we establish that ∃ a $\Upsilon_6 > 0$, ensuring that each solution of system (1) satisfying initial conditions (2) eventually enters and remains in the area.

$$\Omega = \{(S(t), P(t), I(t)) | m \leq S(t) \leq K, m \leq P(t) \leq K, m \leq I(t) \leq K\}, \quad \forall t > \Upsilon_6,$$

where $m = min\{m_3, m_4\}$ and $K = max\{K_3, K_4\}$.

4. Numerical Simulation

The present section performs numerical simulation of the non-autonomous (1). Here, we numerically simulate the different properties of the proposed system (1). These properties are stability, bifurcation, periodic oscillation, chaos, and the effect of predator overcrowding in a periodic environment. In the present model, we consider parameters to be time-dependent. The reason behind this consideration is the temperature fluctuation throughout the year due to seasonal changes. Since the behavior of this temperature fluctuation is periodic, we consider all the time-dependent parameters as a sinusoidal function. The parameter description is as follows:

$$r(t) = r\left(1 + \sigma_1 \sin\left(\frac{2\pi t}{365}\right)\right), \lambda(t) = \lambda\left(1 + \sigma_2 \sin\left(\frac{2\pi t}{365}\right)\right),$$

$$\eta_1(t) = \eta_1\left(1 + \sigma_3 \sin\left(\frac{2\pi t}{365}\right)\right), \eta_2(t) = \eta_2\left(1 + \sigma_4 \sin\left(\frac{2\pi t}{365}\right)\right),$$

$$\eta_3(t) = \eta_3\left(1 + \sigma_5 \sin\left(\frac{2\pi t}{365}\right)\right), \alpha_2(t) = \alpha_2\left(1 + \sigma_7 \sin\left(\frac{2\pi t}{365}\right)\right),$$

$$\gamma_1(t) = \gamma_1\left(1 + \sigma_8 \sin\left(\frac{2\pi t}{365}\right)\right), \gamma_1(t) = \gamma_2\left(1 + \sigma_9 \sin\left(\frac{2\pi t}{365}\right)\right),$$

$$d_1(t) = d_1\left(1 + \sigma_{10} \sin\left(\frac{2\pi t}{365}\right)\right), d_2(t) = d_2\left(1 + \sigma_{11} \sin\left(\frac{2\pi t}{365}\right)\right). \tag{10}$$

We first study the autonomous version of the system (1) with the following biological realist parameters set:

$$r = 6.5, \lambda = 0.2, \alpha_1 = 0.55, \eta_1 = 0.1, \alpha_2 = 0.4, \eta_2 = 0.21, \gamma_1 = 0.45, \gamma_2 = 0.08,$$

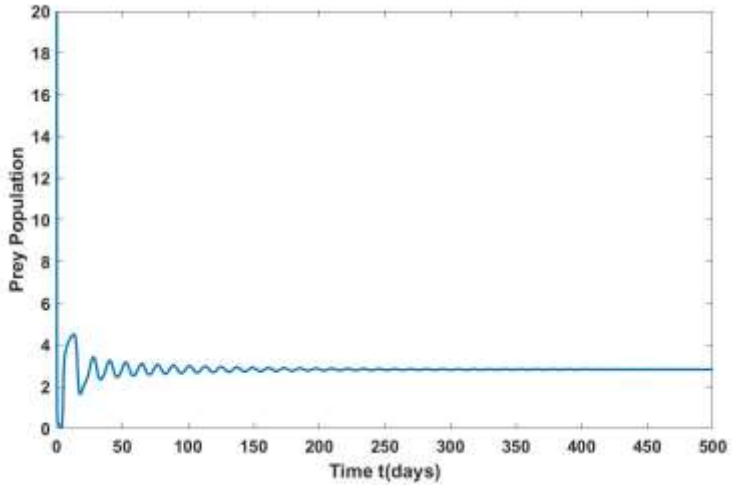
$$\eta_3 = 0.51, d_1 = 1.29, d_2 = 0.05 \quad (11)$$

The simulation results reveal that the interior equilibrium of the autonomous version of the model is stable for the incubation delay τ =1.3411155 for the variable set (11) Fig. 1. For the same variable set, the interior equilibrium becomes unstable while crossing the threshold value of incubation delay τ =3.1411155 and undergoes hopf bifurcation for τ =3.1511155 Fig. 2. Additionally, we explore the behavior of the non-autonomous model. We plot the solution trajectories of the model (1) for the variable set (11) along with (12).

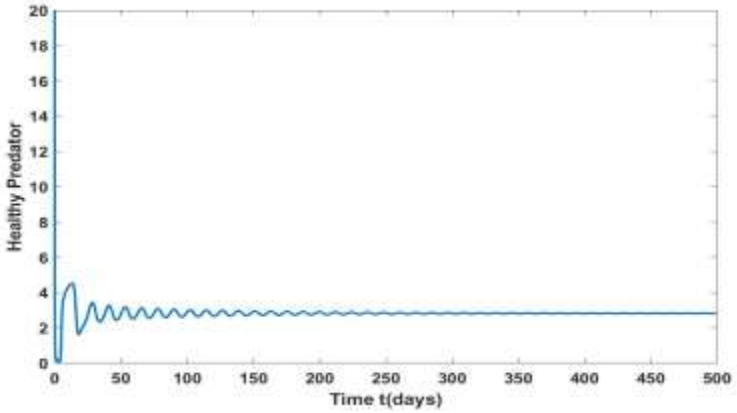
$$\sigma_1 = 0.1, \sigma_2 = 0.4, \sigma_3 = 0.052, \sigma_4 = 0.052, \sigma_5 = 0.66, \sigma_6 = 0.1, \sigma_7 = 0.1, \sigma_8 = 0.1,$$

$$\sigma_9 = 0.1, \sigma_{10} = 0.1, \sigma_{11} = 0.1. \quad (12)$$

The non-autonomous model (1) exhibits periodic oscillation for incubation delay $\tau = 1.3411155$. However, the system shows chaotic behavior for incubation delays τ =3.1511155. Finally, we check the effect of increasing predator crowding. We plot solution trajectories for the variable set (11) and (12) except predator crowding coefficient $d_2 = 2.5$ and see that the infected predator starts disappearing from the system while increasing predator crowding coefficient Fig (5).



(a)



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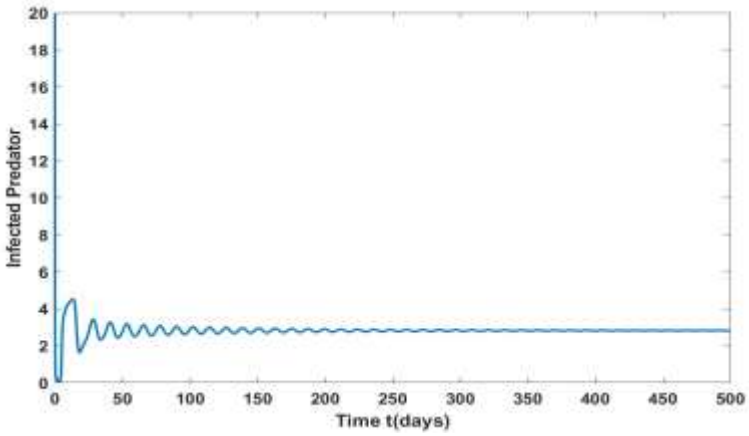
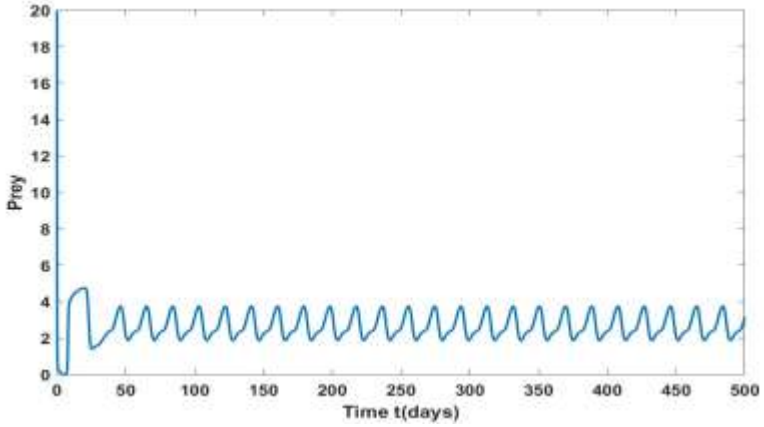
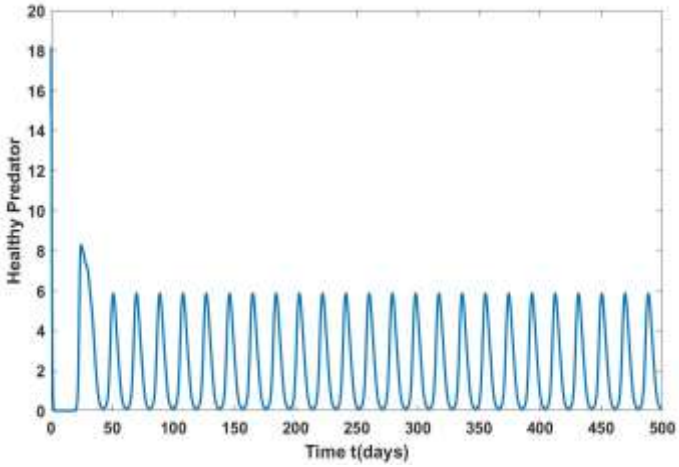


(c)

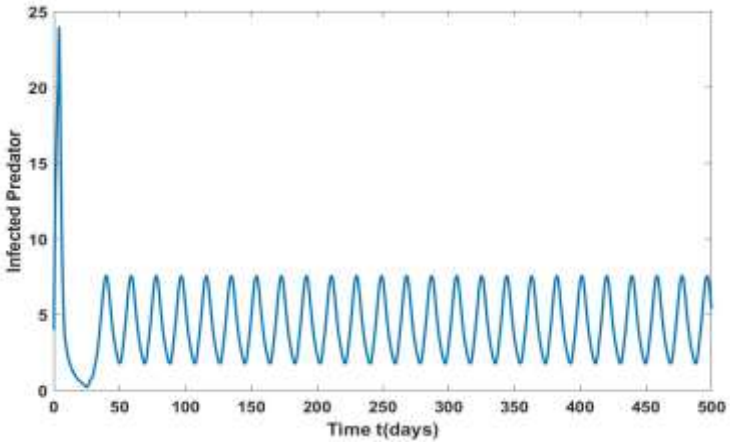
Figure 1: Solution trajectories of the model (1) showing stability for $\tau = 1.3411155$in the absence of seasonality for the parameter set (11) (a) Prey (b) Healthy Predator (c) Infected Predator.



(a)

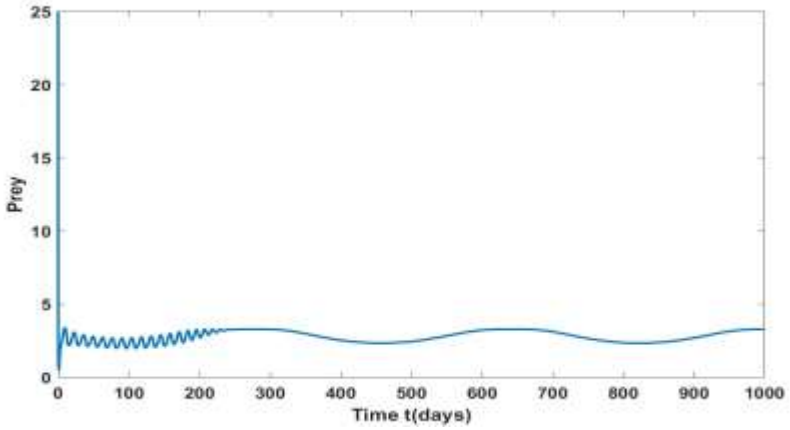


(b)

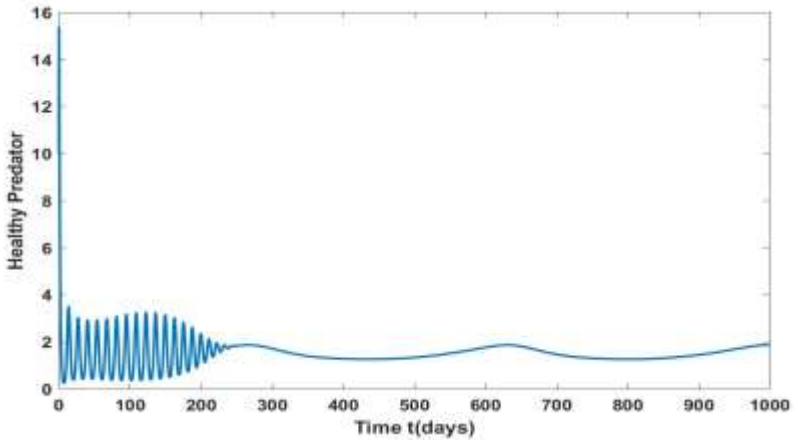


(c)

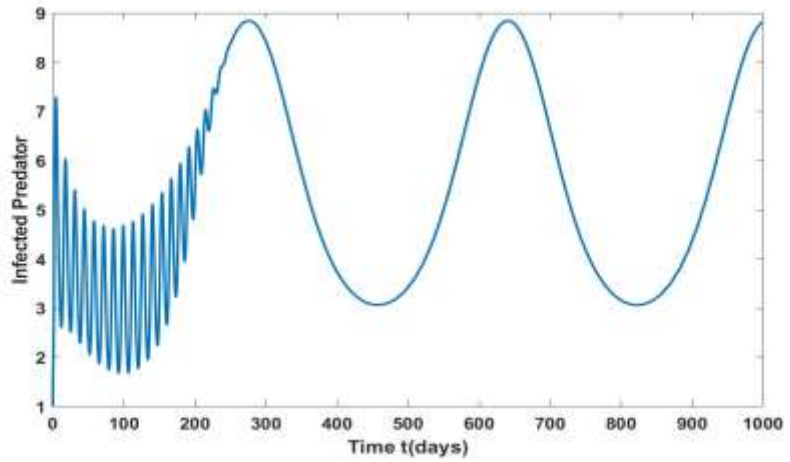
Figure 2: Solution trajectories of the model (1) showing bifurcation for τ =3.1511155in the absence of seasonality for the parameter set (11) (a) Prey (b) Healthy Predator (c) Infected Predator.



(a)

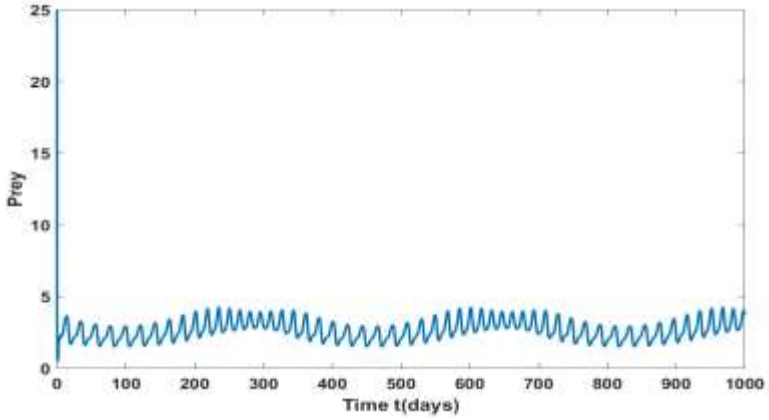


(b)

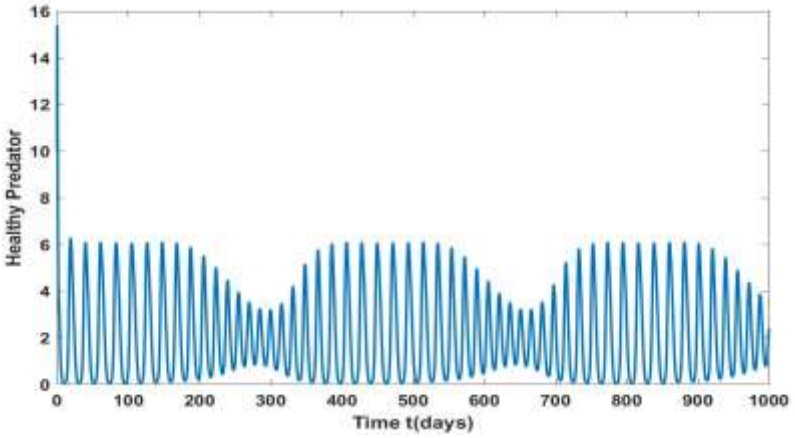


(c)

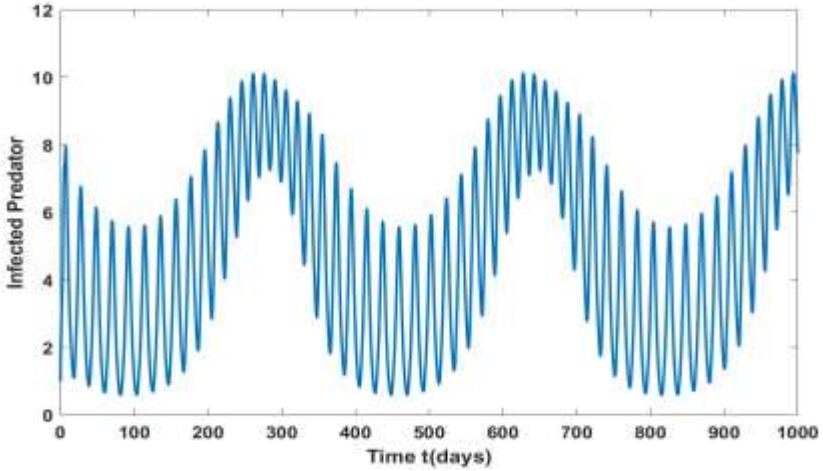
Figure3: Periodic oscillation of population of the non-autonomous model (1) for incubation delay τ =1.3411155 for the parameter set (11) and (12) (a) Prey (b) Healthy Predator (c) Infected Predator.



(a)

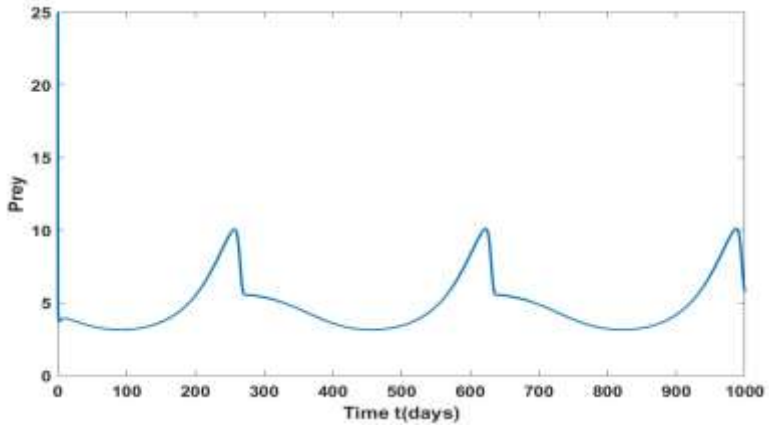


(b)

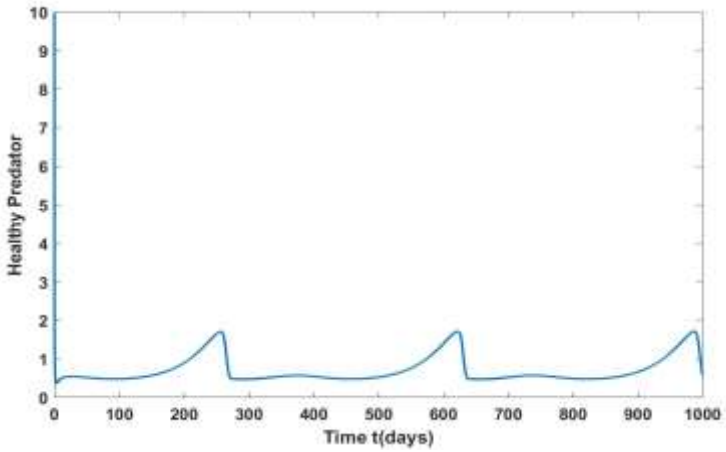


(c)

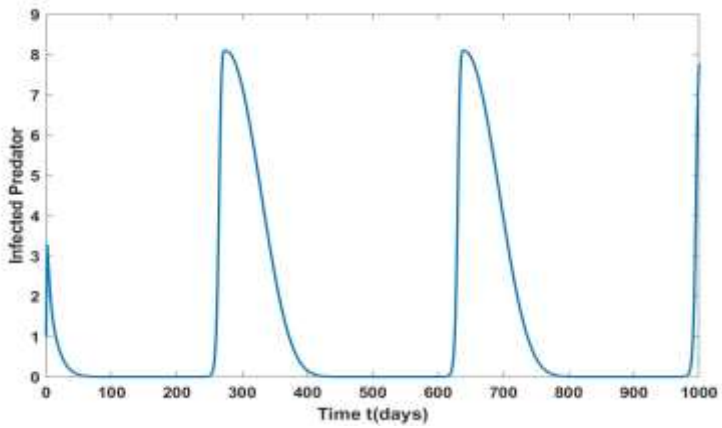
Figure 4: Chaotic behavior of population of the non-autonomous model (1) for incubation delay τ =3.1511155 for the parameter set (11) and (12)(a) Prey (b) Healthy Predator (c) Infected Predator.



(a)



(b)



(c)

Figure 5: Effect of healthy predator crowding on the non-autonomous model (1) for the parameter set (11) and (12) except $d_2 = 2.5$ (a) Prey (b) Healthy Predator (c) Infected Predator.

5. Results: Research shows that many biological processes embodying various delays like latent delay, gestation delay, and incubation delay show more complicated dynamics than non-delayed systems [18, 12, 10]. Various seasonal factors like air pressure, moisture, sunlight, and rainfall also affect system variables. Thus, we also need to study the effect of these parameters on the system. The author includes those mentioned earlier in the proposed system using the sine wave function. Hence, an autonomous system becomes non-autonomous[17, 9, 11].

The present model offers a three-compartment delayed non-autonomous system with infected predator incorporating disease transmission among predators is not an instantaneous process. When a healthy predator comes in contact with an infected predator, it will take an incubation lag to become infectious.

The author mathematically established the system's existence, uniqueness, and permanence and numerically explored all these properties. The author concludes that the delayed autonomous system shows stable behavior for incubation delay τ =1.3411155. Nevertheless, it reveals bifurcation while passing through the critical value of incubation delay τ =3.1511155. Biologically, the system offers stability for any disease having a small incubation period. Additionally, the author simulates the non-autonomous model for the incubation delay mentioned earlier and reveals that the autonomous model (1) becomes periodic. Further, it shows chaotic behavior while increasing the incubation period. Thus, the delayed non-autonomous system offers periodic oscillation instead of stability and chaos instead of bifurcation. Hence, the delayed non-autonomous system gives a more realistic situation than the delayed autonomous one. Finally, we analyze the effect of healthy predator crowding on the system. Increasing predator over-crowding controls the infected predator population and hence controls the spread of disease in the environment. When healthy predators are over-crowded in a particular habitat, they consume the resources as they are more accessible to the resources due to their fitness, while infected predators are not. Thus, the infected predator population starts decreasing and exits from the environment after some time.

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