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THE DYNAMICS OF PREY-PREDATOR MODEL WITH DISEASE IN PREDATOR INVOLVING ALLEE EFFECT AND MIGRATION

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Abstract: The main objective of this paper is to study the dynamical behavior of an eco-epidemiological system involving strong Allee effect and migration in the predator-prey model with the disease in the predator population. The total predator population is divided into two compartments, namely the susceptible predatory population and the infected predator population. The uniform boundedness of the dynamical system is discussed. The stability of the equilibrium points is analyzed. The occurrence of local bifurcation around the equilibrium points is investigated. To verify our analytical findings, numerical simulations are carried out using MATLAB (R2018b) Software.

Keywords: prey-predator model; Allee effect; migration; equilibrium points; local bifurcation.

2020 AMS Subject Classification: 92D40, 92D30, 34D20.

1. INTRODUCTION

Understanding the mechanism underlying the predator-prey system is an essential task in ecology and evolutionary biology. Predator-prey interactions are crucial to ecosystem functioning. It is common knowledge that interactions between predators and prey have been primarily influenced by predation. Prey is consumed by a predator by way of hunting and death in the wild. A mathematical model known as the Lotka-Volterra model [1-2] was first presented by Lotka and Volterra in 1925 to study the evolution of prey and predator populations. Numerous authors have

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examined mathematical models of their relationships based on the existence and significance of prey and predators in nature [3–5]. The models have now been enhanced by scientists through substantial research grounded in real-world contexts, see [6–10]. It is well recognized that the interaction between prey and predator is a remarkable and significant field of research in mathematical modeling and dynamical systems. Therefore, a variety of functional responses (predation rate function) were proposed and used in various biological models, including the prey-dependent type (also known as the Holling type I, type II, and type III) [6], the ratio-dependent type [7], the predator-dependent type (also known as the Beddington-DeAngelis type) [8–9], and many others. The functional response is important in ecological models because it indicates how much each predator attacks prey per unit of time. As a result, numerous researchers have suggested and investigated diverse ecological systems that make use of different kinds of functional reactions, see [10–15] and the references therein.

On the other hand, the biological significance of the study of how diseases affect dynamical systems in ecology cannot be overstated. Numerous prey-predator models that have diseases have recently been examined [16–20]. In fact, Ibrahim and Naji [16] created and investigated a prey-predator model that included infectious illness in the prey and predator harvesting of the Michael Mentence type. While Star et al. [17] proposed and examined an eco-epidemiological system containing an infectious disease that is vertically transmitted. Additionally, the harvesting effort placed on the predator was examined using the Micheal-Mentence kind of harvesting. However, Djilali and Ghanbari [18] talked about how an infectious sickness may have influenced the development of ecological species. They thought about a fractional order computational predator-prey model. Additionally, they made the assumption that the population of prey had contracted a non-fatal contagious condition. A five-compartment prey-predator model including treatment for diseased prey and sick predator was built and analyzed by Bezabih et al. [19]. They used the incidence rates of predation as functional response type II and the incidence rates of disease transmission as simple kinetic mass action functions. A delayed prey-predator system with a SI-type disease in the prey population was later proposed and studied by Hussien and Naji [21]. Using a nonlinear incidence rate, the effect of the incubation period delay on disease transmission was considered. Numerous species are constantly in contact with one another in various ways, which promotes the rapid transmission of disease within the species. While some scientists focused on the study of disease in prey species [22–24], others worked on the study of disease in predator species [25–27]. The presence of disease in both of them has, however, been the subject of

numerous research [28–30].

Allee was the first to articulate the Allee effect in 1931 [31]. He makes reference to a procedure that, for small population densities, lowers the growth rate and raises the death rate. The Allee effect can be attributed to a number of things, including difficulties in partner selection, predator satiation, cooperative defense, etc. Numerous studies conducted in recent years have shown that low population densities are more susceptible to the Allee effect and often vanish as population densities rise. There are two different types of Allee effects. The first is the strong Allee effect, which suggests that the per capita growth rate is negative and that extinction is likely to occur, see for example [4, 32–34]. In fact, Strong Allee effects in the prey species lead to the designation of the region of the system's persistence through the designation of the Allee threshold value, according to Al-Momen and Naji's [4] Sokol-Howell prey-predator model. Later on, a Leslie-Gower predator-prey model with a substantial Allee effect on prey and a fear impact on the predator was proposed by Liu et al. [35]. They came to the conclusion that the system experiences degenerate Bogdanov-Takens bifurcation and that the density of prey species increases with the strength of the fear effect. The weak Allee effect, which is the second phenomenon, shows a decreased per capita growth rate at low population density but still shows a positive trend [36–38]. A recent eco-epidemiological prey-predator system was suggested and examined by Mahmood and Satar [39] when the predator is exposed to the weak Allee effect and harvesting. They noticed that the system exhibits a variety of attractors, including bi-stable, periodic, and stable point behavior.

Additionally, all organisms migrate, which is a very important ecological event. The reasons for migration vary from one species to another, though. Human migration is a result of things like jobs, marriages, education, and other factors. Other species, such as birds, mammals, insects, fish, and reptiles, also migrate as a result of climate change, in search of food, shelter, and other reasons [40]. Keeping the aforementioned in mind, the prey-predator systems must take migration into consideration. Additionally, the process of migration is a significant occurrence that aids the ecosystem in achieving a state of stability. Numerous academics have published work on the prey-predator system with movement in mathematical ecology; see [41–43]. The prey-predator system with movement in both species and sickness in the prey population was further taken into consideration by Arora and Kumar [44]. They observed that as vulnerable prey populations migrated more frequently and the infective prey population tended to decrease over time, the system became unstable. Additionally, as the diseased class of prey displays a fall in its number,

so does the predator population density, and the susceptible prey population grows quickly as a result.

In contrast to the studies mentioned above, the primary goal of this work is to investigate the dynamics of the model when migration and the Allee impact on the prey are present. We developed the mathematical model in section two. In the third section, the reality of equilibrium points is proven. In part four, local stability analysis is examined. Section five establishes the bifurcation analysis of positive equilibrium. In section 6, numerical simulation is seen. Section 7 presents the Conclusion as a final point

2. MATHEMATICAL MODEL

In this section, an eco-epidemiological system consisting of the effect of migration in the predator-prey system with disease in the predator population. We have also incorporated a strong Allee effect in prey population, where $X(T)$ denoted the population size at time T of prey. It is assumed that the predator species is contracted the SI type of infectious disease, therefore the predator is divided into two compartments: the susceptible predator that is denoted to their population size at time T by $Y(T)$, and the second compartment contains the infected predator, which is denoted to their population size at time T by $Z(T)$.

The following hypotheses are adopted to formulate the mathematical model.

$$\begin{aligned} \frac{dX}{dT} &= X \left[r \left(1 - \frac{X}{K} \right) (X - \mu) - \frac{p_1 Y}{b+X} - \frac{p_2 Z}{b+X} - m_1 \right] = X g_1(X, Y, Z) \\ \frac{dY}{dT} &= Y \left[\frac{e_1 p_1 X}{b+X} - \beta Z - d_1 - m_2 \right] = Y g_2(X, Y, Z) \\ \frac{dZ}{dT} &= Z \left[\frac{e_2 p_2 X}{b+X} + \beta Y - d_2 - m_3 \right] = Z g_3(X, Y, Z) \end{aligned} \quad (1)$$

where $X(0) \geq 0, Y(0) \geq 0$, and $Z(0) \geq 0$, with all the parameters, are assumed to be positive constants. Moreover, the functions $g_i, \forall i = 1, 2, 3$ in the right-hand side of the system (1) are continuous and have continuous partial derivatives in the following space $\mathfrak{Q} = \{(X, Y, Z) \in \mathbb{R}^3: X(0) \geq 0, Y(0) \geq 0, Z(0) \geq 0\}$. It is assumed that prey grows logistically with a growth rate $r > 0$ and carrying capacity $K > 0$. The parameter μ represents Allee threshold. Also, migration among prey and predators has been incorporated into the system, where m_1, m_2 , and m_3 are denoted as the migration coefficients in prey, susceptible predator, and infected predator, respectively. The parameters p_1 and p_2 are predation rates of susceptible and infected predators. The parameters e_1 and e_2 are conversion rates of prey into susceptible and infected predators, respectively. The parameters d_1 and d_2 are the natural death rates of susceptible and infected

predators. The parameter β is the infection rate. Finally, the parameter b is the half-saturation constant of both the predators.

Now, the solution of system (1) is proved to be uniformly bounded as shown in the following theorem.

Theorem 1. Every solution of system (1) with the initial condition belonging to $\mathbb{R}_+^3$ is uniformly bounded in the region $\Omega = \{(X, Y, Z) \in \mathbb{R}_+^3 : X \geq 0, Y \geq 0, Z \geq 0\}$

Proof. Assume that $\phi(t) = X(t) + Y(t) + Z(t)$ and $\xi = \min\{r\mu + m_1, d_1 + m_2, d_2 + m_3\}$ then the derivative of W with respect to T can be written as

$$\frac{d\phi(T)}{dT} + \xi\phi(T) \leq X\left[\frac{r(K+\mu)}{K}X - \frac{r}{K}X^2\right] \leq \frac{4r(K+\mu)^3}{27K} = L$$

Integrating the above inequality. We have $\phi(t) \leq \frac{L}{\xi}(1 - e^{-\xi T}) + \phi_0 e^{-\xi T}$, then the direct computation shows that for $T \rightarrow \infty$, we have $\phi(T) \leq \frac{L}{\xi}$, therefore, the solutions of system (1) are uniformly bounded.

3. EQUILIBRIA AND THEIR STABILITY

In this section, the existence of the equilibrium points and stability analysis are discussed. It is observed that system (1) has at most five equilibrium points, which can be stated as follows:

- The origin equilibrium point $S_0 = (0, 0, 0)$ always exists.
- The axial equilibrium points $S_{1i} = (\bar{X}_i, 0, 0); i = 1, 2$, where $\bar{X}_i$ for $i = 1, 2$ are the roots of the equation

$$rX^2 - r(K + \mu)X + K(\mu r + m_1) = 0. \quad (2)$$

Hence, Eq. (2) either have two different positive roots given by

$$\begin{aligned} \bar{X}_1 &= \frac{1}{2} \left((K + \mu) + \sqrt{(K + \mu)^2 - 4 \frac{K}{r} (\mu r + m_1)} \right) \\ \bar{X}_2 &= \frac{1}{2} \left((K + \mu) - \sqrt{(K + \mu)^2 - 4 \frac{K}{r} (\mu r + m_1)} \right) \end{aligned} \quad (3)$$

where

$$(K + \mu)^2 \geq 4 \frac{K}{r} (\mu r + m_1). \quad (4)$$

or no positive roots exist. Clearly, the roots in Eq. (3) are equal $\bar{X}_{1,2} = \frac{1}{2}(K + \mu)$, when the equality in the condition (4) occurs.

- The disease-free equilibrium point, $S_2 = (\hat{X}, \hat{Y}, 0)$, where

$$\hat{X} = \frac{b(d_1+m_2)}{e_1p_1-(d_1+m_2)}, \quad (5)$$

$$\hat{Y} = \frac{1}{Kp_1} \left[(b + \hat{X}) \left(-K(\mu r + m_1) - r(\hat{X})^2 + (K + \mu)r\hat{X} \right) \right] \quad (6)$$

Exists in the XY – plane under the following conditions

$$e_1p_1 > (d_1 + m_2) \quad (7)$$

$$(K + \mu)r\hat{X} > K(\mu r + m_1) + r(\hat{X})^2 \quad (8)$$

- The positive equilibrium point $S_3 = (\tilde{X}, \tilde{Y}, \tilde{Z})$ where

$$\left. \begin{aligned} \tilde{Y} &= \frac{[(d_2+m_3)-e_2p_2]\tilde{X}+b(d_2+m_3)}{\beta(b+\tilde{X})} \\ \tilde{Z} &= \frac{e_1p_1\tilde{X}-(d_1+m_2)(b+\tilde{X})}{\beta(b+\tilde{X})} \end{aligned} \right\}. \quad (9)$$

While $\tilde{X}$ represents a positive root of the equation

$$A_1X^4 + A_2X^3 + A_3X^2 + A_4X + A_5 = 0, \quad (10)$$

where

$$A_1 = -r\beta$$

$$A_2 = -2br\beta + Kr\beta + \mu r\beta,$$

$$A_3 = -b^2r\beta + 2bKr\beta + 2b\mu r\beta - K\mu r\beta - K\beta m_1$$

$$A_4 = b^2Kr\beta + b^2\mu r\beta - 2bK\mu r\beta - 2bK\beta m_1 - Kd_2p_1 - Km_3p_1 \\ + Kd_1p_2 + Km_2p_2 - Ke_1p_1p_2 + Ke_2p_1p_2$$

$$A_5 = -b^2K\mu r\beta - b^2K\beta m_1 - bKd_2p_1 - bKm_3p_1 + bKd_1p_2 + bKm_2p_2$$

Clearly, the positivity of $\tilde{Y}$ and $\tilde{Z}$ are satisfied provided that

$$\left. \begin{aligned} (d_2 + m_3) &> e_2p_2 \\ e_1p_1\tilde{X} &> (d_1 + m_2)(b + \tilde{X}) \end{aligned} \right\} \quad (11)$$

However, Eq. (9) has a unique positive root, given by $\tilde{X}$, provided that one set of the following sets of conditions holds:

$$\left. \begin{aligned} A_3 &> 0; A_4 > 0; A_5 > 0 \\ A_2 &< 0; A_3 < 0; A_5 > 0 \\ A_2 &< 0; A_4 > 0; A_5 > 0 \end{aligned} \right\} \quad (12)$$

4. LOCAL STABILITY ANALYSIS

Now, the local stability analysis of the above feasible equilibrium points of system (1) is studied

using a linearization technique. The Jacobian matrix of system (1) is

$$J = \begin{pmatrix} \frac{r}{K}(2(K+\mu)X - 3X^2) - (\mu r + m_1) - \frac{b(Yp_1 + Zp_2)}{(b+X)^2} & -\frac{Xp_1}{b+X} & -\frac{Xp_2}{b+X} \\ \frac{be_1p_1Y}{(b+X)^2} & -Z\beta - d_1 - m_2 + \frac{Xe_1p_1}{b+X} & -Y\beta \\ \frac{be_2p_2Z}{(b+X)^2} & Z\beta & Y\beta - d_2 - m_3 + \frac{Xe_2p_2}{b+X} \end{pmatrix}, \quad (13)$$

Straightforward computation shows that the Jacobian matrix of system (1) around the origin equilibrium point $S_0 = (0,0,0)$ has the following eigenvalues:

$$\lambda_{01} = -(\mu r + m_1) < 0, \quad \lambda_{02} = -(d_1 + m_2) < 0, \quad \lambda_{03} = -(d_2 + m_3) < 0. \quad (14)$$

Accordingly, all the eigenvalues of the origin equilibrium point are negative and S_0 is always an asymptotically stable. That means the prey and predators (susceptible and infected) will be extinct.

The Jacobian matrix evaluated at the axial equilibrium point $S_{11} = (\bar{X}_1, 0, 0)$ can be written as

$$J(S_{11}) = \begin{pmatrix} \frac{r(K-2\bar{X}_1+\mu)}{K}\bar{X}_1 & -\frac{\bar{X}_1p_1}{b+\bar{X}_1} & -\frac{\bar{X}_1p_2}{b+\bar{X}_1} \\ 0 & -d_1 - m_2 + \frac{\bar{X}_1e_1p_1}{b+\bar{X}_1} & 0 \\ 0 & 0 & -d_2 - m_3 + \frac{\bar{X}_1e_2p_2}{b+\bar{X}_1} \end{pmatrix} \quad (15)$$

The eigenvalues of $J(S_{11})$ are given by $\lambda_{11} = -\frac{r\sqrt{(K+\mu)^2 - 4\frac{K}{r}(\mu r + m_1)}}{K}\bar{X}_1 < 0$, $\lambda_{12} = -(d_1 + m_2) + \frac{\bar{X}_1e_1p_1}{b+\bar{X}_1}$ and $\lambda_{13} = -(d_2 + m_3) + \frac{\bar{X}_1e_2p_2}{b+\bar{X}_1}$, Hence the axial equilibrium point $S_{11} = (\bar{X}_1, 0, 0)$ is locally asymptotically stable under condition (4) and the following conditions hold.

$$\frac{\bar{X}_1e_1p_1}{b+\bar{X}_1} < (d_1 + m_2) \quad (16)$$

$$\frac{\bar{X}_1e_2p_2}{b+\bar{X}_1} < (d_2 + m_3) \quad (17)$$

However,

$$J(S_{12}) = \begin{pmatrix} \frac{r(K-2\bar{X}_2+\mu)}{K}\bar{X}_2 & -\frac{\bar{X}_2p_1}{b+\bar{X}_2} & -\frac{\bar{X}_2p_2}{b+\bar{X}_2} \\ 0 & -d_1 - m_2 + \frac{\bar{X}_2e_1p_1}{b+\bar{X}_2} & 0 \\ 0 & 0 & -d_2 - m_3 + \frac{\bar{X}_2e_2p_2}{b+\bar{X}_2} \end{pmatrix}, \quad (18)$$

Then the eigenvalues of $J(S_{12})$ are given by $\lambda_{11} = \frac{r\sqrt{(K+\mu)^2 - 4\frac{K}{r}(\mu r + m_1)}}{K}\bar{X}_2$, $\lambda_{12} = -(d_1 + m_2) + \frac{\bar{X}_2e_1p_1}{b+\bar{X}_2}$, $\lambda_{13} = -(d_2 + m_3) + \frac{\bar{X}_2e_2p_2}{b+\bar{X}_2}$, since $\lambda_{11} > 0$ under the condition (4). Hence the axial equilibrium point $S_{12} = (\bar{X}_2, 0, 0)$ is a saddle point (unstable).

Now, Jacobian matrix at the disease-free point $S_2 = (\hat{X}, \hat{Y}, 0)$ can be written as

$$J(S_2) = \begin{pmatrix} \hat{X} \left(\frac{r(K+\mu-2\hat{X})}{K} + \frac{p_1\hat{Y}}{(b+\hat{X})^2} \right) & -\frac{p_1\hat{X}}{b+\hat{X}} & -\frac{p_2\hat{X}}{b+\hat{X}} \\ \frac{be_1p_1\hat{Y}}{(b+\hat{Y})^2} & 0 & -\beta\hat{Y} \\ 0 & 0 & \beta\hat{Y} - d_2 - m_3 + \frac{e_2p_2\hat{X}}{b+\hat{X}} \end{pmatrix} \quad (19)$$

Therefore, the characteristic equation of $J(S_2)$ can be determined as follow

$$(\lambda^2 - H_1\lambda + D_1) \left[\beta\hat{Y} - d_2 - m_3 + \frac{e_2p_2\hat{X}}{b+\hat{X}} - \lambda \right] = 0, \quad (20)$$

where $H_1 = \hat{X} \left(\frac{r(K+\mu-2\hat{X})}{K} + \frac{p_1\hat{Y}}{(b+\hat{X})^2} \right)$ and $D_1 = \left(\frac{p_1\hat{X}}{b+\hat{X}} \right) \left(\frac{be_1p_1\hat{Y}}{(b+\hat{Y})^2} \right) > 0$. Hence, the eigenvalues of $J(S_2)$ are given by

$$\left. \begin{aligned} \lambda_{21} &= \frac{H_1}{2} + \frac{1}{2} \sqrt{H_1^2 - 4D_1} \\ \lambda_{22} &= \frac{H_1}{2} - \frac{1}{2} \sqrt{H_1^2 - 4D_1} \\ \lambda_{23} &= \beta\hat{Y} - (d_2 + m_3) + \frac{e_2p_2\hat{X}}{b+\hat{X}} \end{aligned} \right\} \quad (21)$$

According to the forms of these eigenvalues $\lambda_{21}, \lambda_{22}$ and λ_{23} , they have negative real parts and hence S_2 is locally asymptotically stable provided the following conditions hold

$$\beta\hat{Y} + \frac{e_2p_2\hat{X}}{b+\hat{X}} < d_2 + m_3, \quad (22)$$

$$H_1 < 0. \quad (23)$$

Finally, the Jacobian matrix at the positive point $S_3 = (\tilde{X}, \tilde{Y}, \tilde{Z})$ can be written as:

$$J(S_3) = \begin{pmatrix} \tilde{X} \left(\frac{(K+\mu)r}{K} - \frac{2r\tilde{X}}{K} + \frac{p_1\tilde{Y}+p_2\tilde{Z}}{(b+\tilde{X})^2} \right) & -\frac{p_1\tilde{X}}{b+\tilde{X}} & -\frac{p_2\tilde{X}}{b+\tilde{X}} \\ \frac{be_1p_1\tilde{Y}}{(b+\tilde{X})^2} & 0 & -\beta\tilde{Y} \\ \frac{be_2p_2\tilde{Z}}{(b+\tilde{X})^2} & \beta\tilde{Z} & 0 \end{pmatrix} = (r_{ij}) \quad (24)$$

Then the characteristic equation of $J(S_3)$ is given by

$$X^3 + R_1X^2 + R_2X + R_3 = 0, \quad (25)$$

where

$$R_1 = -(r_{11}), R_2 = -(r_{12}r_{21} + r_{13}r_{31} + r_{23}r_{32}), R_3 = -[r_{23}(r_{12}r_{31} - r_{11}r_{32}) + r_{13}r_{21}r_{32}].$$

While $\Delta = R_1R_2 - R_3 = r_{31}(r_{11}r_{13} + r_{12}r_{23}) + r_{21}(r_{11}r_{12} + r_{13}r_{32})$.

Using the Routh–Hurwitz criteria [45], all the eigenvalues of $J(S_3)$ have negative real part if and only if $R_i; (i = 1, 3) > 0$ and $\Delta = R_1R_2 - R_3 > 0$.

Consequently it is easy to verify that S_3 is locally asymptotically stable provided that the following condition:

$$\frac{2r\tilde{X}}{K} > \frac{p_1\tilde{Y}+p_2\tilde{Z}}{(b+\tilde{X})^2} + \frac{(K+\mu)r}{K} \quad (26)$$

$$r_{12}r_{31}-r_{11}r_{32} > 0 \quad (27)$$

$$r_{11}r_{12} + r_{13}r_{32} > 0 \quad (28)$$

Note that, since the system has the origin as unconditional locally asymptotically stable, therefore there is no possibility to study the global stability of the above equilibrium points.

5. BIFURCATION ANALYSIS

In this section, the possibility of occurrence of local bifurcation of system (1) is investigated. Therefore, in the following theorem, the bifurcation parameter is selected so that the equilibrium point becomes a non-hyperbolic point.

Theorem 2. Assume that condition (26) holds. Then system (1) undergoes a saddle-node bifurcation near the positive equilibrium point $S_3 = (\tilde{X}, \tilde{Y}, \tilde{Z})$ when the parameter e_1 passes from the positive value e_1^* provided that the following condition is satisfied

$$\begin{aligned} M_1\sigma_1\zeta_1^2 - \frac{p_1b\zeta_1\zeta_2}{(b+\tilde{X})^2}(\sigma_1 - e_1\sigma_2) - \frac{p_2b\zeta_1}{(b+\tilde{X})^2}(\sigma_1 - e_2) + \beta\zeta_2(1 - \sigma_2) \\ - \frac{b\zeta_1^2}{(b+\tilde{X})^3}(e_1\tilde{Y} + e_2Z^*) \neq 0 \end{aligned} \quad (29)$$

Proof. By using Sotomayor's theorem [46]. If $e_1^* = \frac{(b+\tilde{X})^2}{p_1b\tilde{Y}}B$, where $B = \frac{r_{23}(r_{11}r_{32}-r_{12}r_{31})}{r_{13}r_{32}} > 0$

then $\det(J_{S_3}) = R_3 = 0$, which means one eigenvalue of the Jacobian matrix J_{S_3} is zero.

Let $V = (v_1, v_2, v_3)$ and $\Psi = (\psi_1, \psi_2, \psi_3)$ represent the two eigenvectors corresponding to the zero eigenvalue of J_{S_3} and its transpose $J_{S_3}^T$, respectively. Then it is determined as:

$$V = (\zeta_1, \zeta_2, 1)^t, \text{ where } \zeta_1 = \frac{-r_{23}}{r_{21}} \text{ and } \zeta_2 = \frac{r_{23}r_{11}-r_{13}r_{31}}{r_{12}r_{21}}$$

$$\Psi = (\sigma_1, \sigma_2, 1), \text{ where } \sigma_1 = \frac{-r_{32}}{r_{12}} \text{ and } \sigma_2 = \frac{r_{32}r_{11}-r_{12}r_{31}}{r_{12}r_{21}}$$

Now, direct computation gives that:

$$F_{e_1}(X, e_1) = \left(0, \frac{p_1XY}{b+X}, 0\right)^T, \text{ then } F_{e_1}(S_3, e_1^*) = \left(0, \frac{p_1\tilde{X}\tilde{Y}}{b+\tilde{X}}, 0\right)^T.$$

Therefore, $\Psi^T[F_{e_1}(S_3, e_1^*)] = \frac{p_1\tilde{X}\tilde{Y}}{b+\tilde{X}} \neq 0$.

Now, the second derivative of the system (1), can be written as follows

$$D^2F(X, \theta).(V, V) = \begin{pmatrix} v_1 \left[2 \left(\frac{r(K+\mu-3X)}{K} + \frac{b(Yp_1+Zp_2)}{(b+X)^3} \right) v_1 - \frac{2b(p_1v_2+p_2v_3)}{(b+X)^2} \right] \\ \frac{2be_1p_1v_1(-Yv_1+(b+X)v_2)}{(b+X)^3} - 2\beta v_2v_3 \\ 2\beta v_2v_3 + \frac{2be_2p_2v_1(-Zv_1+(b+X)v_3)}{(b+X)^3} \end{pmatrix}$$

Therefore, according to condition (29), it is simple to verify that

$$\begin{aligned} \Psi^T[D^2F(S_3, e_1^*)(V, V)] &= 2[M_1\sigma_1\zeta_1^2 - \frac{p_1b\zeta_1\zeta_2}{(b+\tilde{X})^2}(\sigma_1 - e_1\sigma_2) - \frac{p_2b\zeta_1}{(b+\tilde{X})^2}(\sigma_1 - e_2) \\ &\quad + \beta\zeta_2(1 - \sigma_2) - \frac{b\zeta_1^2}{(b+\tilde{X})^3}(e_1\tilde{Y} + e_2Z^*)] \neq 0 \end{aligned}$$

where $M_1 = \frac{r(K+\mu-3\tilde{X})}{K} + \frac{b(\tilde{Y}p_1+\tilde{Z}p_2)}{(b+\tilde{X})^3}$. Consequently, system (1) undergoes a saddle-node bifurcation.

6. NUMERICAL SIMULATIONS

In this section, the numerical simulations conduct using MATLAB R2018b software to verify the theoretical results discussed in the previous sections. The main goal is to understand how the system responds to changes in its parameters. For simulation purposes, the following coefficient values were selected below with various sets of initial points.

$$\begin{aligned} r = 1, K = 5, \mu = 0.1, b = 2, p_1 = 0.85, p_2 = 0.4, e_1 = 0.5, \\ e_2 = 0.4, \beta = 0.05, d_1 = 0.1, d_2 = 0.15, m_1 = 0.01, m_2 = 0.05, m_3 = 0.1, \end{aligned} \quad (30)$$

Note that in the following phase portraits, the red and blue dots refer to the various initial points and the asymptotically stable points, respectively. It was noticed that starting from various initial points and using the set (30), the bi-stable case will be appearing and the trajectory approaches asymptotically to various attractors S_0 and S_3 , as shown in figure 1.

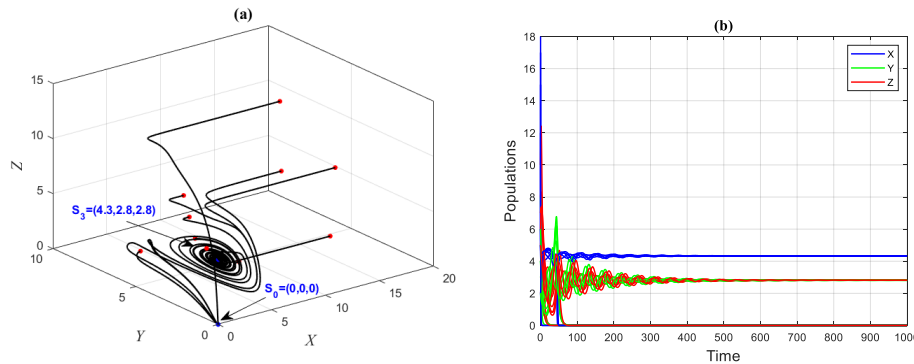


Figure 1. The trajectory of system (1) with the set (30) and various initial points. (a) System (1)

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has a bi-stable case between $S_0 = (0,0,0)$ and $S_3 = (4.3,2.8,2.8)$. (b) Explain the time series for (a).

Now, to understand the impacts of changing the intrinsic growth rate on the dynamics of system (1) with different values of r . It is observed that, the ranges $0 < r \leq 0.63$, $0.63 < r \leq 2.1$ and $2.1 < r$ are investigated respectively. Clearly, the trajectory approaches to S_0 , bi-stable case (S_0 and S_3), and S_3 respectively, as shown in figure 2.

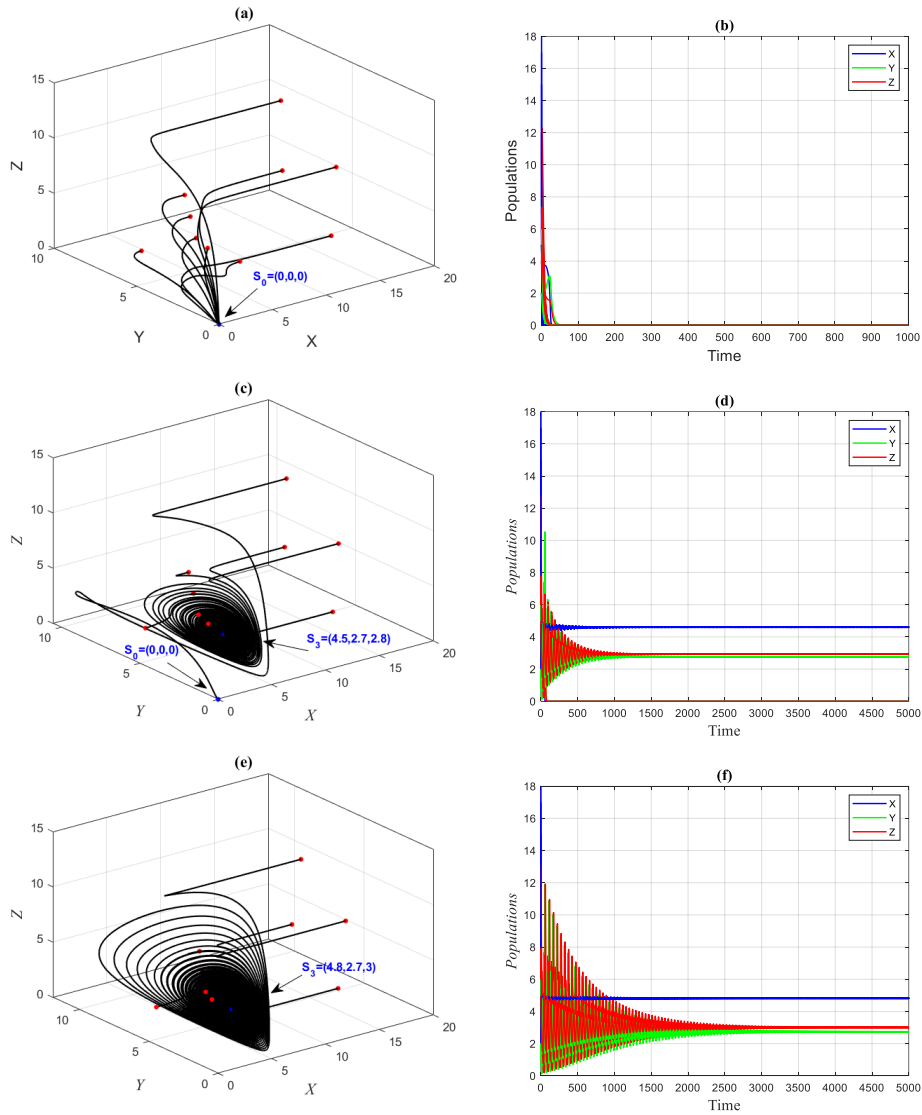


Figure 2. The trajectory of system (1) with the set (30) and numerous values of r . (a) For $r = 0.5$. (b) Explain the time series for (a). (c) For $r = 1.5$. (d) Explain the time series for (c). (e) For $r = 3$. (f) Explain the time series for (e).

The impacts of changing the carrying capacity of system (1) are studied. It is clear that, for $0 < K < 4$, $4 \leq K < 10$ and $10 \leq K$, then the trajectory approaches to S_0 , bi-stable case (S_0 and S_3) and Periodic dynamics in the $Int. \mathbb{R}_+^3$ respectively, as illustrated in figure 3.

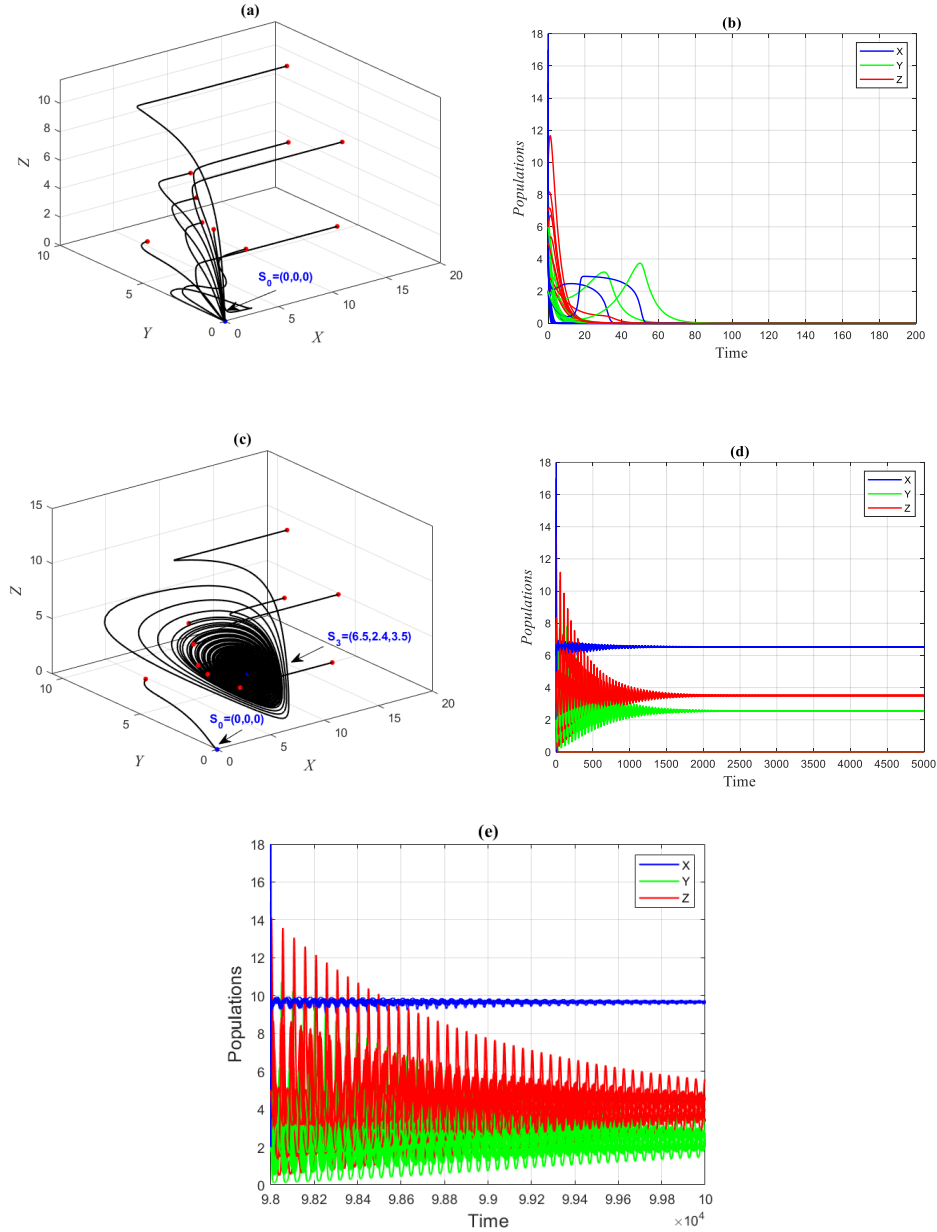


Figure 3. The trajectory of system (1) with the set (30) and numerous values of K . (a) For $K = 3$. (b) Explain the time series for (a). (c) For $K = 7$. (d) Explain the time series for (c). (e) Explain the time series for $K = 10$.

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The impacts of changing the Allee threshold of system (1) are investigated below. Obviously, for $0 < \mu \leq 1$ and $1 < \mu$, then the trajectory approaches to bi-stable case (S_0 and S_3) and S_0 respectively, as shown in figure 4.

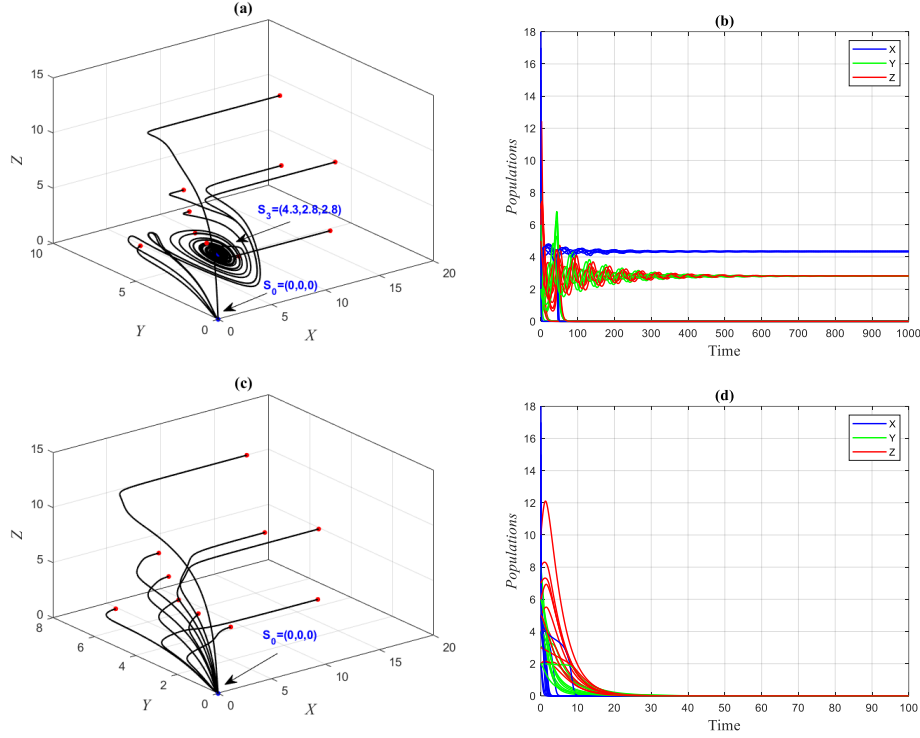
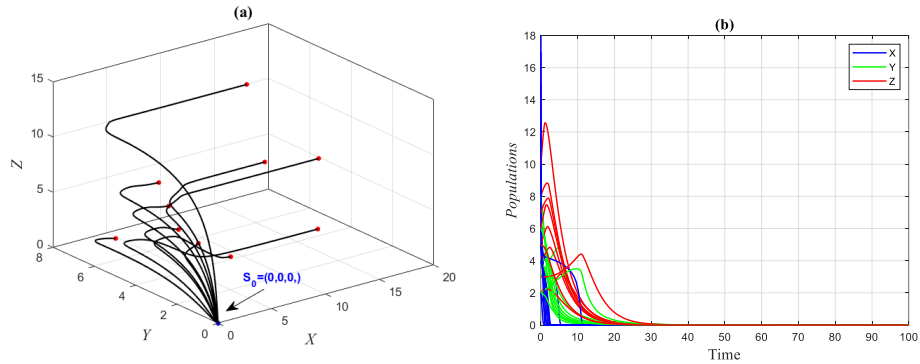


Figure 4. The trajectory of system (1) with the set (30) and numerous values of μ . (a) For $\mu = 0.05$. (b) Explain the time series for (a). (c) For $\mu = 2$. (d) Explain the time series for (c).

However, the effects of changing the half-saturation constant on the dynamics of system (1) is illustrated in figure 5. In the ranges $0 < b \leq 0.8$ and $0.8 < b$. It is observed that the trajectory approaches to S_0 and bi-stable case (S_0 and S_3).



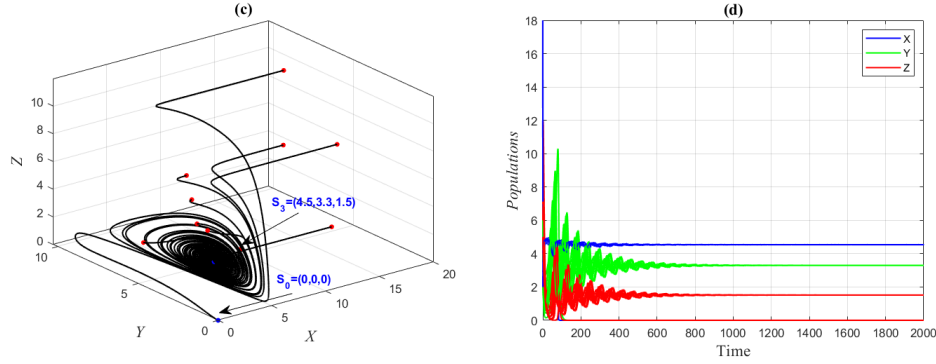


Figure 5. The trajectory of system (1) with the set (30) and numerous values of b . (a) For $b = 0.5$. (b) Explain the time series for (a). (c) For $b = 4$. (d) Explain the time series for (c).

The impacts of changing the predation rate of susceptible predator of system (1) are studied below. Obviously, for $0 < p_1 < 0.5$, $0.5 \leq p_1 < 0.6$ and $0.6 \leq p_1$, then the trajectory approaches to S_1 , S_3 and bi-stable case (see figure 1) respectively, as shown in figure 6.

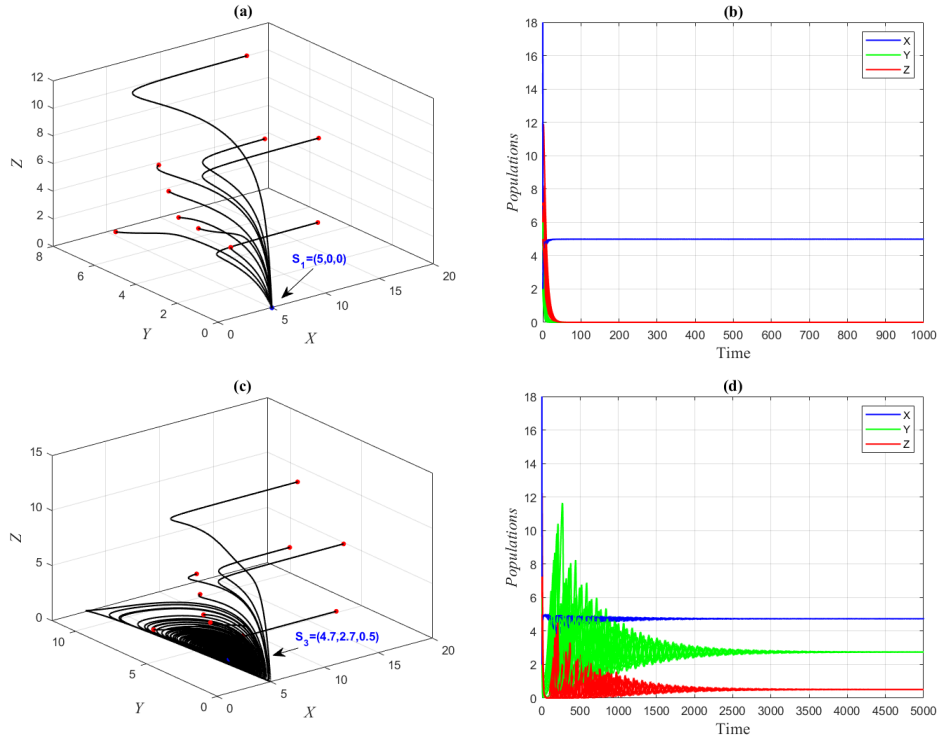


Figure 6. The trajectory of system (1) with the set (30) and numerous values of p_1 . (a) For $p_1 = 0.1$. (b) Explain the time series for (a). (c) For $p_1 = 0.5$. (d) Explain the time series for (c).

Now, the predation rate of infected predator p_2 and the conversion rates of prey into infected predator e_2 have quantitative effects. Therefore, the trajectory of system (1) approaches to bi-

stable case(S_0 and S_3), see figure 1.

Now, the impacts of changing the conversion rates of prey into susceptible predator is investigated. when $0 < e_1 < 0.3$, the trajectory approaches to bi-stable case(S_0 and S_1). Also, the trajectory approaches to bi-stable case (S_0 and S_3) when $0.3 \leq e_1 < 0.9$. For $0.9 \leq e_1$, the trajectory approaches to S_0 see figure 7.

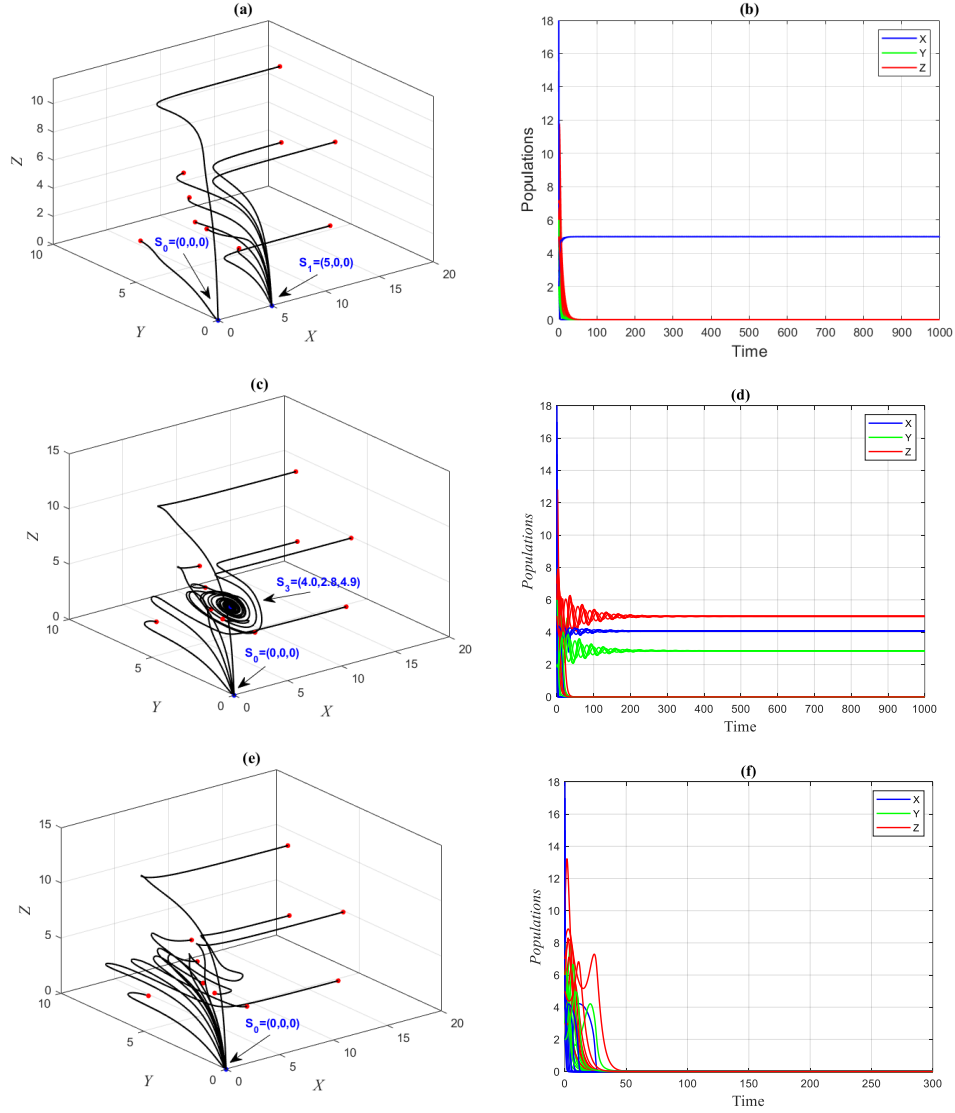


Figure 7. The trajectory of system (1) with the set (30) and numerous values of e_1 . (a) For $e_1 = 0.1$. (b) Explain the time series for (a). (c) For $e_1 = 0.7$. (d) Explain the time series for (c). (e) For $e_1 = 0.9$. (f) Explain the time series for (e).

The effects of changing the infection rate on the dynamics of system (1) is illustrated in figure 8. In the ranges $0 < \beta \leq 0.03$, $0.03 < \beta \leq 0.1$ and $0.1 < \beta$, it is observed that the trajectory

approaches to S_0 , bi-stable case (S_0 and S_3) (see figure 1) and bi-stable case (S_0 and Periodic dynamics in the $Int. \mathbb{R}_+^3$) respectively.

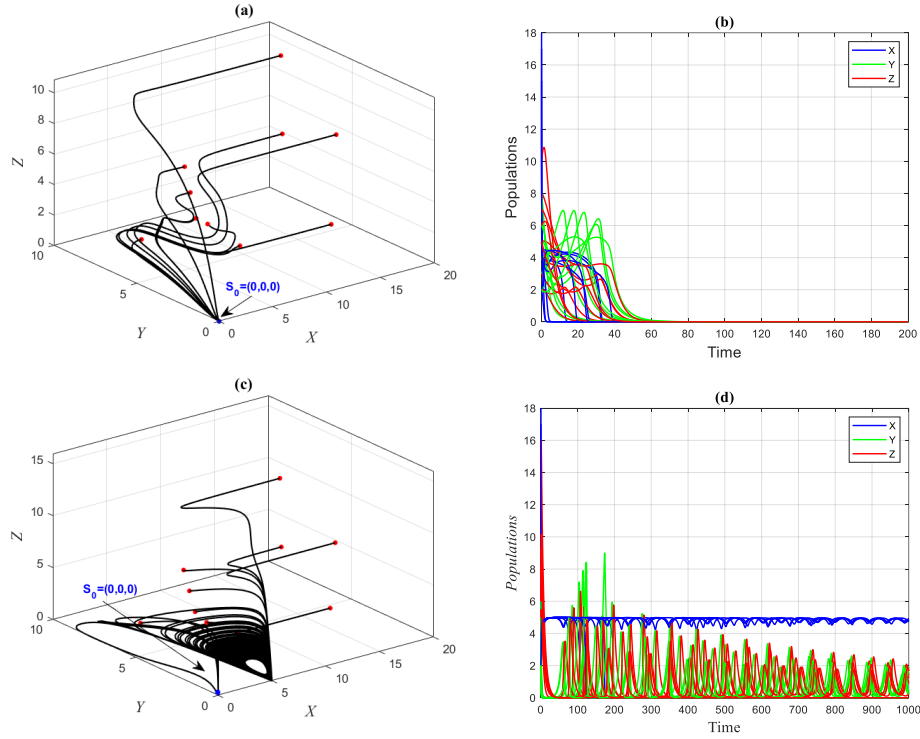
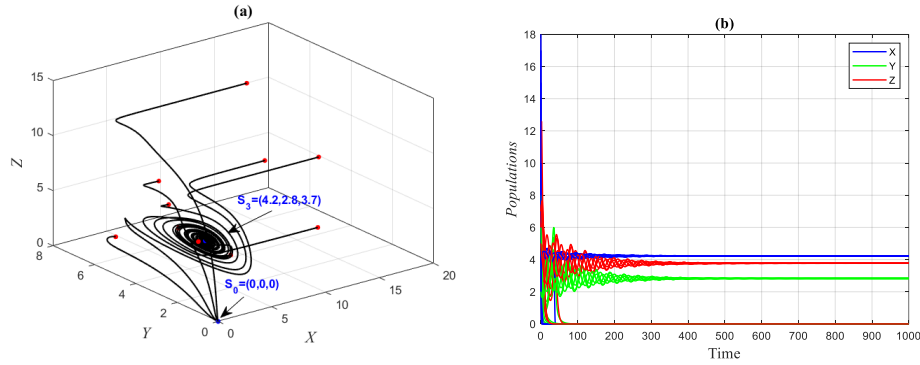


Figure 8. The trajectory of system (1) with the set (30) and numerous values of β . (a) For $\beta = 0.03$. (b) Explain the time series for (a). (c) For $\beta = 0.2$. (d) Explain the time series for (c).

The impacts of changing the natural death rates of susceptible predator of system (1) are studied. It is clear that, for $0 < d_1 < 0.3$ and $0.3 \leq d_1$, then the trajectory approaches to bi-stable case (S_0 and S_3) and S_1 respectively, as illustrated in figure 9.



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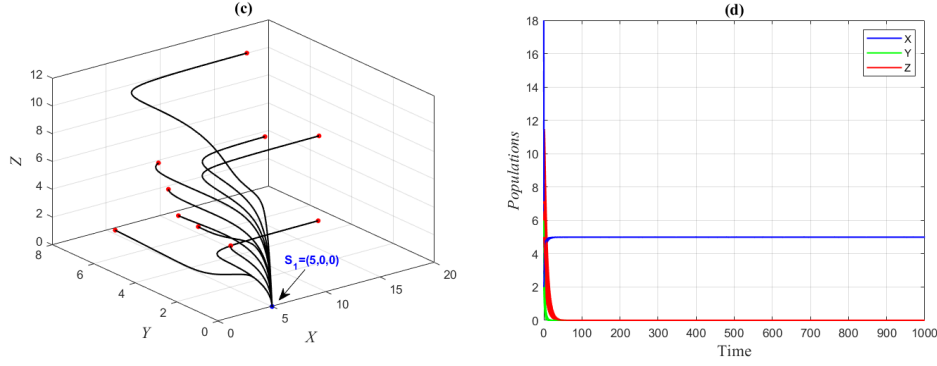


Figure 9. The trajectory of system (1) with the set (30) and numerous values of d_1 . (a) For $d_1 = 0.05$. (b) Explain the time series for (a). (c) For $d_1 = 0.5$. (d) Explain the time series for (c).

The impacts of changing the natural death rates of infected predator of system (1) are investigated below. Obviously, for $0 < d_2 \leq 0.3$ and $0.3 < d_2$, then the trajectory approaches to bi-stable case (S_0 and S_3) and S_0 respectively, as shown in figure 10.

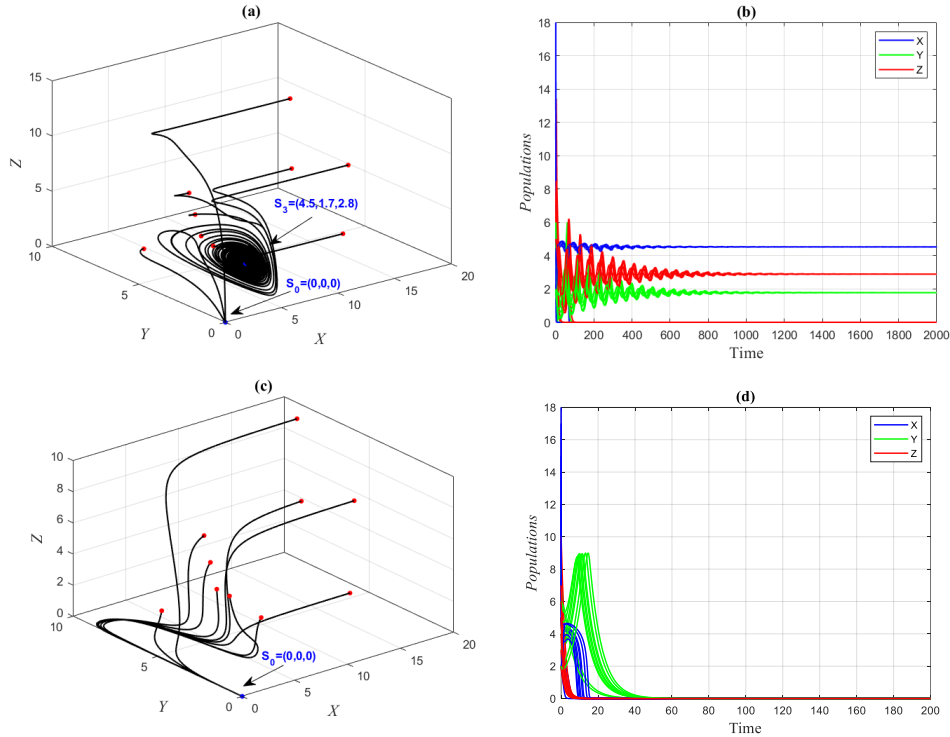


Figure 11. The trajectory of system (1) with the set (30) and numerous values of d_1 . (a) For $d_2 = 0.05$. (b) Explain the time series for (a). (c) For $d_2 = 0.8$. (d) Explain the time series for (c).

Now, the migration coefficient in prey m_1 has similar dynamic case of natural death rates of

infected predator d_2 . Also, the trajectory approaches to bi-stable case (S_0 and S_3) and S_0 , (see figure 11).

The impacts of changing the migration coefficient in susceptible predator m_2 of system (1) are studied below. Obviously, for $0 < m_2 < 0.3$, $0.3 \leq m_2 < 0.4$ and $0.4 \leq m_2$, then the trajectory approaches to bi-stable case (S_0 and S_2), bi-stable case (S_0 and S_1) and S_1 respectively, as shown in figure 12.

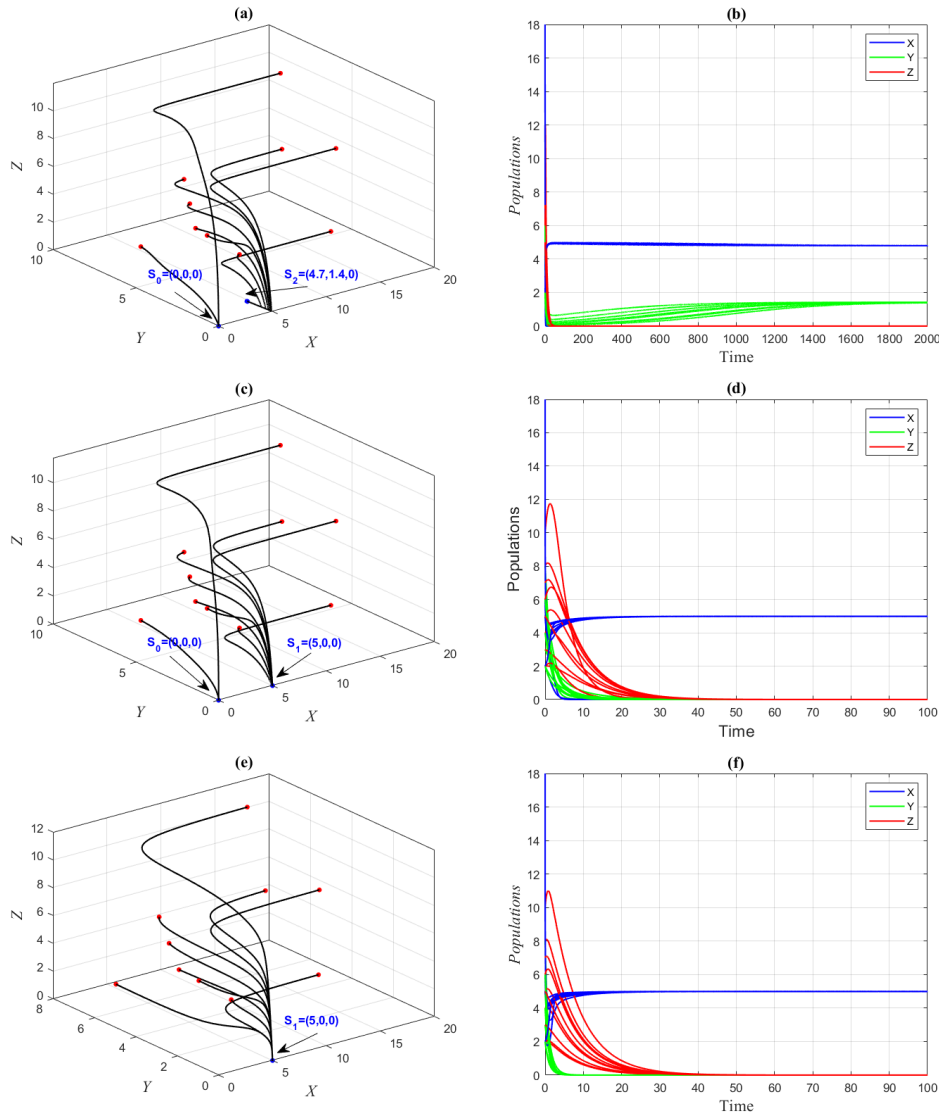


Figure 12. The trajectory of system (1) with the set (30) and numerous values of m_2 . (a) For $m_2 = 0.2$. (b) Explain the time series for (a). (c) For $m_2 = 0.3$. (d) Explain the time series for (c). (e) For $m_2 = 0.9$. (f) Explain the time series for (e).

However, the effects of changing the migration coefficient in infected predator m_3 on the dynamics of system (1) is illustrated in figure 13. In the ranges $0 < m_3 < 0.1$, $0.1 \leq m_3 < 0.2$ and $0.2 \leq m_3$ It is observed that the trajectory approaches to bi-stable case (S_0 and Periodic dynamics in the $Int. \mathbb{R}_+^3$), bi-stable case (S_0 and S_3) (see figure 1) and S_0 , respectively.

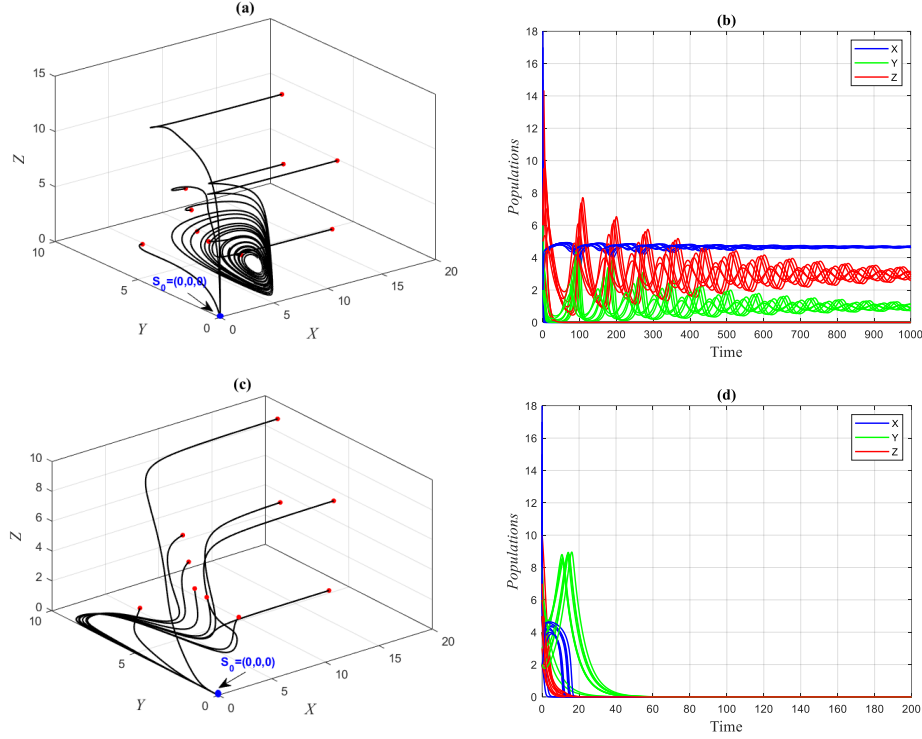


Figure 13. The trajectory of system (1) with the set (30) and numerous values of m_3 . (a) For $m_3 = 0.01$. (b) Explain the time series for (a). (c) For $m_3 = 0.5$. (d) Explain the time series for (c).

7. CONCLUSION

This paper has studied an eco-epidemiological model involving strong Allee effect and migration. The predator is assumed to be contracting the SI type of infectious disease. The properties of solutions are demonstrated, the stability and persistence of equilibrium points are investigated. Also, the local bifurcation is studied. The numerical simulation is investigated by using various initial points with a set of parameters which shown in Eq. (30) and the outcomes are summarized as follows: Depending on the initial points and the set of data (30), system (1) possesses a bi-stable case, represented by two stable equilibrium points S_0 and S_3 .

Decreasing the values of intrinsic growth rate, carrying capacity, the half-saturation constant and

the infection rate indicate to the extinction of both prey and predators (susceptible and infected). Clearly, the trajectory approaches to S_0 . While due to increasing of them, the trajectory approaches to S_3 , periodic dynamic in the $int. R_+^3$, bi-stable case (S_0 and S_3) and bi-stable case (S_0 and periodic dynamic in the $int. R_+^3$), respectively. On the other hand, increasing the values of Allee threshold, the conversion rate of prey into susceptible predator, the natural death rate of infected predator, the migration coefficients in prey and infected predator refer to the extinction of both prey and predators. Also, the trajectory approaches to S_0 . Now, increasing the values of the natural death rate of susceptible predator and the migration coefficient in susceptible predator indicate to the extinction of predators and the trajectory approaches to S_1 .

Decreasing (increasing) the values of the predation rate and the conversion rate of infected predator refer to the same quantitative effects and the trajectory approaches to bi-stable case (S_0 and S_3). Finally, increasing the values of the predation rate of susceptible predator indicates to the extinction of predators and the trajectory approaches to S_1 , while further increasing of the predation rate more than a critical value makes the trajectory approaches to S_3 . As a result of the previous, System (1) is sensible to varies in the values of parameters. we were summarized the conclusion of this paper as follows: The Allee threshold is a major factor that effects on the biological processes. It was noticed that, the presence of higher Allee threshold makes it difficult for persistence.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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