JOURNAL OF ALGEBRAIC STATISTICS
Volume 13, No. 3, 2022, p. 5470-5479
https://publishoa.com
ISSN: 1309-3452

A mathematical study of diseased predators in prey predator models, taking into account the influence of toxicants on the models

Dr. Uday Raj Singh[1]

Associate Professor, Department of Mathematics, C.L. Jain (PG) College, Firozabad

(affiliated to Dr. B.R.A. University), Agra (U.P.), India

Email id-udayrajsinghfzd@gmail.com

Satendra Kumar Suman[2]

Research Scholar, C.L. Jain (PG) College, Firozabad

(affiliated to Dr. B.R.A. University), Agra (U.P.), India

Email id-satendrakumartdl@gmail.com

Received 2022 March 15; **Revised** 2022 April 20; **Accepted** 2022 May 10.

Abstract

In order to investigate the effects of the toxicant on the Lotka-Volterra prey-predator system, a mathematical model has been created. Our working hypothesis was that the populations are composed of three distinct parts: prey, healthy predators, and sick predators. We have obtained all of the equilibriums that are even remotely possible with this model, and we have also established the prerequisites for the presence of an inner equilibria point. We investigated the presence of all equilibrium points as well as their local stability. The findings are weighed against the scenario in which the environmental toxicant was not present. In the final step, analytical results are put through their numerical paces for verification.

Keywords- Lotka-Volterra prey-predator model, pollution environment, Stability, toxicant

1. Introduction-

Mathematical modelling is a powerful tool for describing and analysing the dynamic behaviour of a phenomena. The single-species concept was originally developed by British economist Malthus (1978) and then refined by Verhulst, both around the year 1800. Several attempts at statistically predicting the existence and evolution of species were made at the turn of the twentieth century. Well-known classical figures Lotka and Volterra actually made the first serious effort in this regard. In 1927, they put out the concept of prey and predator. What's more, delay differential equations are frequently employed for modelling the dynamics of living organisms.

The study of predator-prey interactions has been an important part of ecology research for the past three decades. Research into the dynamics of populations and the relationship between prey and predator is an active and important subject of study in the field of applied mathematics.

Mathematical ecology has come a long way since its infancy, when it was pioneered by Lotka (1925) and Volterra (1928). Mathematical analysis was used by Kermack and McKendrick (2027) to study the spread of disease. A new subject, eco-epidemiology, emerged in the late 1990s to examine the intersection of epidemiology and ecological issues. It was Anderson and May (1986) who were the first to merge the two disciplines and create a predator-prey model with population sickness. Separate but related subfields of biology and applied mathematics include mathematical ecology and mathematical epidemiology. To study the intersection of ecology and epidemiology is known as eco-epidemiology. Liu et al.(2003) investigated a single-species model in a contaminated, confined habitat where toxicants are introduced at a constant rate (2003).

JOURNAL OF ALGEBRAIC STATISTICS
Volume 13, No. 3, 2022, p. 5470-5479
https://publishoa.com
ISSN: 1309-3452

In their research, Zhang and Sun (2005) discovered a predator-prey system with a sick predator and a functional response. The predator-prey system was examined by Kant and Kumar (2007), who looked at it from the perspective of migrating prey and predators who were susceptible to virus. In a predator-prey system, Bairagi et al. (2009) looked at how the act of gathering affects the spread of illnesses among a subset of prey. The existence and uniqueness of limit cycles, as well as the global stability of equilibria, were studied by Chen et al. (2010) in a type-II prey-predator model.

In 2012, Naji and Mustafa discussed the dynamics of a nonlinear incidence rate eco-epidemiological model. While assuming that the sensitive prey has the same exchange rate as the sick prey, Johri et al. (2012) studied a Lotka-Volterra type predator-prey model without harvesting or disease in the prey. Hethcote et al. (2014) showed that the presence of infections could modify the biological dynamics of the prey-predator state. Using an eco-epidemiology model, Sharma and Samanta (2015) studied a situation in which a single prey species contracts a disease.

A host-vector epidemic model with a stage structure for the vector was presented and investigated by Zhou and Yao (2015). The dynamical behaviour of a tritrophic food chain model with a predator-prey system was studied by Bera et al. (2016). Eco-epidemiological models have been examined by a plethora of writers, all of whom have focused on infection in the prey population. Subsequent writers, including Kant and Kumar (2017), developed and investigated a predator-prey model that included moving prey and disease infection in both species. There was an infectious disease in the prey population, and Thota (2020) presented a mathematical model for this system. Baishya et al. (2021) constructed a fractional-order model to describe the possible transmission of an infection from prey to predator, and they reviewed the dynamics of this model with respect to its boundedness, uniqueness, and existence of solutions.

2. Mathematical Model-

Two populations are involved in our model: prey, whose population density is represented by $x(t)$ and predators, whose population densities are represented by $y_1(t)$ and $y_2(t)$ where y_1 designates the healthy predator and y_2 represents the infected predator, and t is the time variable. Let the population concentration of the toxin, $u(t)$ and the ambient concentration, c(t), be two variables.

While developing our model, we assume the following assumptions:

$$\frac{dx}{dt} = r\left(1 - \frac{x}{k_1}\right)x - bxy_1 - \mu_1 x \quad (1)$$

$$\frac{dy_1}{dt} = s\left(1 - \frac{y_1+y_2}{k_2}\right)y_1 - \beta y_1 y_2 + \alpha y_2 - \mu_2 y_1 \quad (2)$$

$$\frac{dy_2}{dt} = \beta y_1 y_2 - \alpha y_2 - \mu_3 y_2 - dy_2 - iuy_2 \quad (3)$$

$$\frac{dc}{dt} = Q - \gamma c - \delta c y_2 \quad (4)$$

$$\frac{du}{dt} = \delta c y_2 - \mu_4 u \quad (5)$$

(A_1): Populations of prey expand at a rate consistent with the logistic rule when illness and predators are absent: $r > 0$ and $k_1 > 0$.

(A_2): The assumption here is that the disease is only contagious among predators, and that the spread of it from healthy to afflicted predators follows the standard rule of mass action and environment: $\beta y_1 y_2$, where β is the infectious force.

(A_3): Infected predators are also less active than their healthy counterparts, making it harder for them to catch their prey. Therefore, we think a healthy predator has a higher searching coefficient than a sick one when looking for food.

JOURNAL OF ALGEBRAIC STATISTICS
Volume 13, No. 3, 2022, p. 5470-5479
https://publishoa.com
ISSN: 1309-3452

(A_4): It is believed that the predator's functional response to the prey is of the Lotka-Volterra type.

(A_5): It has been presumed that an infected predator will recover at a pace equal to α.

3. Balance points do exist -

It is not difficult to demonstrate that the system (1-5) contains equilibrium points that are viable from a biological perspective. The following is a discussion of the existence conditions of each of them as well as their respective studies of their local stability:

(3.1) There is never a time when the vanishing equilibrium $\epsilon_0 = (0,0,0,0,0)$ does not exist.

(3.2) The first axial equilibrium $\epsilon_1 = (x, 0,0,0,0)$, $x = \frac{k_1(r-\mu_1)}{r}$ exists provided

$\frac{k_1(r-\mu_1)}{r} > 0 \Rightarrow r - \mu_1 > 0 \Rightarrow r > \mu_1$

(3.3) The second axial equilibrium point with no infection occurred $\epsilon_2 = (0, y_1, 0,0,0)$

$y_1 = \frac{k_2(s-\mu_2)}{s}$ exists provided $\frac{k_2(s-\mu_2)}{s} > 0 \Rightarrow s - \mu_2 > 0 \Rightarrow s > \mu_2$

(3.4) The initial point of planar equilibrium has the equation $\epsilon_3 = (x, y_1, 0,0,0)$, where $y_1 = \frac{k_2(s-\mu_2)}{s}$ and x represents a positive root of the following quadratic equation: $x^2\left(\frac{r}{k_1}\right) - x\left[r - \mu_1 - \frac{bk_2(s-\mu_2)}{s}\right] = 0$

ϵ_3 exists uniquely in interior of XY −plane if $r > \mu_1 + \frac{bk_2(s-\mu_2)}{s}$ and $s > \mu_2$

(3.5) The point of prey-free equilibrium with the toxicant concentration in the population and environment and the occurrence of infection in the predators, denoted by the equation $\epsilon_4 = (0, y_1, y_2, c, u))$, was reached.

$y_1 = \frac{\alpha+\mu_3+d}{\beta} + \frac{i\delta Qy_2}{\mu_4\beta(\gamma+\delta y_2)}$, $c = \frac{Q}{\gamma+\delta y_2}$, $u = \frac{\delta Q y_2}{\mu_4(\gamma+\delta y_2)}$

$A_1 {y_2}^3 + A_2 {y_2}^2 + A_3 y_2 + A_4 = 0$

Where

$A_1 = \alpha k_2 l^2\delta^2 - kl^2 s\delta^2 - ls\delta m - kk_2 l^2\delta^2\beta - k_2 l\delta\beta m$

$A_2 = kk_2 l^2 s\delta^2 - k^2 l^2 s\delta^2 + k_2 ls\delta m - 2kls\delta m - sm^2 - ls\gamma m - kk_2 l^2\delta\beta\gamma - \beta m k_2 l\gamma - kk_2 l^2\delta\beta\gamma + 2\alpha k_2 l^2\delta\gamma - kk_2 l^2\delta^2\mu_2 - k_2 l\delta m$

$A_3 = 2kk_2 sl^2\delta\gamma - 2k^2 sl^2\delta\gamma - 2kls\gamma m - ksl^2\gamma^2 + k_2 sl\gamma m + \alpha k_2 l^2\gamma^2 - kk_2 l^2\mu_2 - k_2 lm - kk_2 l^2\mu_2\delta\gamma$

$A_4 = kk_2 sl^2\gamma^2 - k^2 sl^2\gamma^2 - kk_2 l^2\gamma^2\mu_2$

And $k = \frac{\alpha+\mu_3+d}{\beta}, l = \mu_4\beta, m = i\delta Q$

When $A_4 > 0$ and either $A_3 > 0$ or $A_2 < 0$, a singular instance of ϵ_4 can only be found within the interior of the four-dimensional space denoted by the coordinates $y_1 y_2 cu$

4. Local stability analysis:

The linearization method is going to be used in this part so that we may establish the local stability of the equilibrium points of the system (1-5) here. It is simple to demonstrate that the variational matrix of system (1-5) can be represented by the following formula at the general point (x, y_1, y_2, c, u),

JOURNAL OF ALGEBRAIC STATISTICS
Volume 13, No. 3, 2022, p. 5470-5479
https://publishoa.com
ISSN: 1309-3452

$$J=\begin{bmatrix} r\left(1-\frac{2x}{k_1}\right)-\mu_1 & bx & 0 & 0 & 0 \\ 0 & s\left(1-\frac{2y_1+y_2}{k_2}\right)-\beta y_2-\mu_2 & -\frac{sy_1}{k_2}-\beta y_1+\alpha & 0 & 0 \\ 0 & \beta y_2 & -\alpha-\mu_3-d-iu & 0 & 0 \\ 0 & 0 & -\delta c & -\gamma-\delta y_2 & 0 \\ 0 & 0 & \delta c & \delta y_2 & -\mu_4 \end{bmatrix} \quad (6)$$

(4.1) Under the initial conditions of $\epsilon_0=(0,0,0,0,0)$, the variational matrix of the system (1-5) is

$$J(\epsilon_0)=\begin{bmatrix} r-\mu_1 & 0 & 0 & 0 & 0 \\ 0 & s-\mu_2 & \alpha & 0 & 0 \\ 0 & & -\alpha-\mu_3-d & 0 & 0 \\ 0 & 0 & 0 & -\gamma & 0 \\ 0 & 0 & 0 & 0 & -\mu_4 \end{bmatrix} \quad (7)$$

The Eigen values of $J(\epsilon_0)$ are

$\lambda_1=r-\mu_1,\lambda_2=s-\mu_2,\lambda_3=-\alpha-\mu_3-d,\lambda_4=-\gamma,\lambda_5=-\mu_4$

Therefore, if $r<\mu_1, s<\mu_2$, then the equilibrium $\epsilon_0=(0,0,0,0,0)$ is locally asymptotically stable.

(4.2) Assuming $\epsilon_1=(x,0,0,0,0)$, we have as its variational matrix the solution to the system (1-5):

$$J(\epsilon_1)=\begin{bmatrix} r\left(1-\frac{2x}{k_1}\right)-\mu_1 & bx & 0 & 0 & 0 \\ 0 & s-\mu_2 & \alpha & 0 & 0 \\ 0 & 0 & -\alpha-\mu_3-d & 0 & 0 \\ 0 & 0 & 0 & -\gamma & 0 \\ 0 & 0 & 0 & 0 & -\mu_4 \end{bmatrix}$$

$\lambda_1=r\left(1-\frac{2x}{k_1}\right)-\mu_1,\lambda_2=s-\mu_2,\lambda_3=-\alpha-\mu_3-d,\lambda_4=-\gamma,\lambda_5=-\mu_4$

Consequently, $r<\mu_1+\frac{2xr}{k_1}, s<\mu_2$is a locally asymptotically stable equilibrium if it is satisfied by $\epsilon_1=(x,0,0,0,0)$

(4.3) At $\epsilon_2=(0,y_1,0,0,0)$, the variational matrix for the system (1-5) is

$$J(\epsilon_2)=\begin{bmatrix} r-\mu_1 & 0 & 0 & 0 & 0 \\ 0 & s\left(1-\frac{2y_1}{k_2}\right)-\mu_2 & -\frac{sy_1}{k_2}-\beta y_1+\alpha & 0 & 0 \\ 0 & 0 & -\alpha-\mu_3-d & 0 & 0 \\ 0 & 0 & 0 & -\gamma & 0 \\ 0 & 0 & 0 & 0 & -\mu_4 \end{bmatrix}$$

$\lambda_1=r-\mu_1,\lambda_2=s\left(1-\frac{2y_1}{k_2}\right)-\mu_2,\lambda_3=-\alpha-\mu_3-d,\lambda_4=-\gamma,\lambda_5=-\mu_4$

So, $\epsilon_2=(0,y_1,0,0,0)$is a locally asymptotically stable equilibrium if $r<\mu_1, s<\mu_2+\frac{2y_1s}{k_2}$

(4.4) At the point when $\epsilon_3=(x,y_1,0,0,0)$, the variational matrix for the system (1-5) may be written as

$$J(\epsilon_3)=\begin{bmatrix} r\left(1-\frac{2x}{k_1}\right)-\mu_1 & bx & 0 & 0 & 0 \\ 0 & s\left(1-\frac{2y_1}{k_2}\right)-\mu_2 & -\frac{sy_1}{k_2}-\beta y_1+\alpha & 0 & 0 \\ 0 & 0 & -\alpha-\mu_3-d & 0 & 0 \\ 0 & 0 & 0 & -\gamma & 0 \\ 0 & 0 & 0 & 0 & -\mu_4 \end{bmatrix}$$

JOURNAL OF ALGEBRAIC STATISTICS
Volume 13, No. 3, 2022, p. 5470-5479
https://publishoa.com
ISSN: 1309-3452

$\lambda_1 = r\left(1-\frac{2x}{k_1}\right)-\mu_1, \lambda_2 = s\left(1-\frac{2y_1}{k_2}\right)-\mu_2, \lambda_3 = -\alpha-\mu_3-d, \lambda_4 = -\gamma, \lambda_5 = -\mu_4$

Therefore, $\epsilon_2 = (0, y_1, 0,0,0)$ represents a locally asymptotically stable equilibrium if the following conditions are met: $r < \mu_1 + \frac{2xr}{k_1}, s < \mu_2 + \frac{2y_1 s}{k_2}$

(4.5) At the point when $\epsilon_4 = (0, y_1, y_2 c, u)$, the variational matrix for the system (1-5) may be written as

$$J(\epsilon_5) = \begin{bmatrix} r\left(1-\frac{2x}{k_1}\right)-\mu_1 & bx & 0 & 0 & 0 \\ 0 & s\left(1-\frac{2y_1+y_2}{k_2}\right)-\beta y_2-\mu_2 & -\frac{sy_1}{k_2}-\beta y_1+\alpha & 0 & 0 \\ 0 & \beta y_2 & -\alpha-\mu_3-d & 0 & 0 \\ 0 & 0 & -\delta c & -\gamma-\delta y_2 & 0 \\ 0 & 0 & \delta c & \delta y_2 & -\mu_4 \end{bmatrix}$$

It's not hard to see how this could work. This corresponds to the equilibrium point (ϵ_1), for which the characteristic equation of J_1 is:

$\lambda^5 + k_1\lambda^4 + k_2\lambda^3 + k_3\lambda^2 + k_4\lambda + k_5 = 0$

Where

$k_1 = \mu_4 + \gamma + \delta y_2 + \frac{s(2y_1+y_2)}{k_2} + \beta y_2 + \mu_2 + \frac{2xr}{k_1} + \mu_1 + \alpha + \mu_3 + d - (r+s)$

$k_2 = \mu\rho + \mu\tau + \mu\psi + \mu\omega + \rho\tau + \rho\psi + \rho\omega + \tau\psi + \tau\omega + \psi\omega - \theta\sigma$

$k_3 = \mu\sigma\theta + \theta\rho\sigma + \theta\sigma\psi - \mu\rho\tau - \mu\rho\psi - \mu\rho\omega - \mu\tau\psi - \mu\tau\omega - \mu\psi\omega - \rho\tau\psi - \rho\tau\omega - \rho\psi\omega - \tau\psi\omega$

$k_4 = \mu\rho\tau\psi + \mu\rho\tau\omega + \mu\rho\psi\omega + \mu\tau\psi\omega + \rho\tau\psi\omega - \theta\mu\rho\sigma - \theta\mu\sigma\psi - \theta\rho\sigma\psi$

$k_5 = \theta\mu\rho\sigma\psi - \mu\rho\tau\psi\omega$Where

$\psi = r\left(1-\frac{2x}{k_1}\right)-\mu_1 ; \varepsilon = bx ; \tau = s\left(1-\frac{2y_1+y_2}{k_2}\right)-\beta y_2-\mu_2 ; \sigma = \frac{sy_1}{k_2}-\beta y_1+\alpha ; \theta = \beta y_2 ; \omega = -\alpha-\mu_3-d ; \rho = -\gamma-\delta y_2 ; \phi = \delta y_2 ; \mu = -\mu_4$

$\lambda_1 = \psi, \lambda_2 = \mu, \lambda_3 = \rho, \lambda_4 = \frac{1}{2}\left(\tau+\omega-\sqrt{4\theta\sigma+\tau^2-2\tau\omega+\omega^2}\right)$

$\lambda_5 = \frac{1}{2}\left(\tau+\omega-\sqrt{4\theta\sigma+\tau^2-2\tau\omega+\omega^2}\right)$

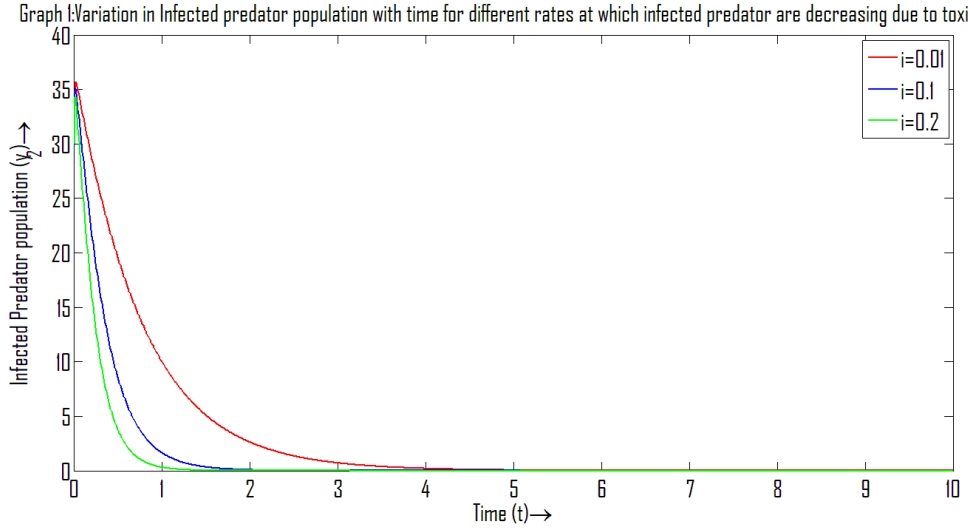
All the coefficients in the characteristic equation are unambiguously positive. If the following hold true, then ϵ_1 is locally asymptotically stable according to the Routh Hurwitz criteria:

$k_1 > 0, k_1k_2 - k_3 > 0, k_1k_2k_3 + k_1(k_5 - k_1k_4) - {k_3}^2 > 0$

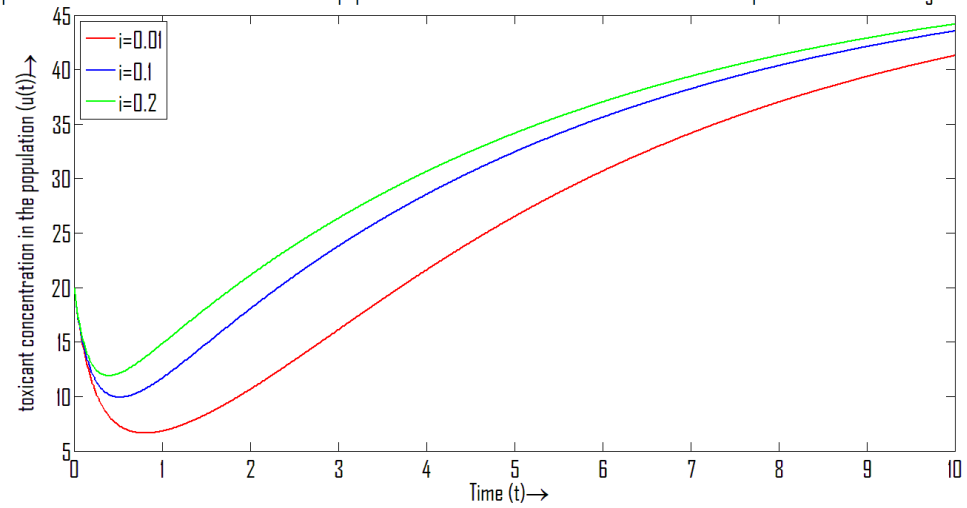
5. Results and Discussion-

JOURNAL OF ALGEBRAIC STATISTICS
Volume 13, No. 3, 2022, p. 5470-5479
https://publishoa.com
ISSN: 1309-3452

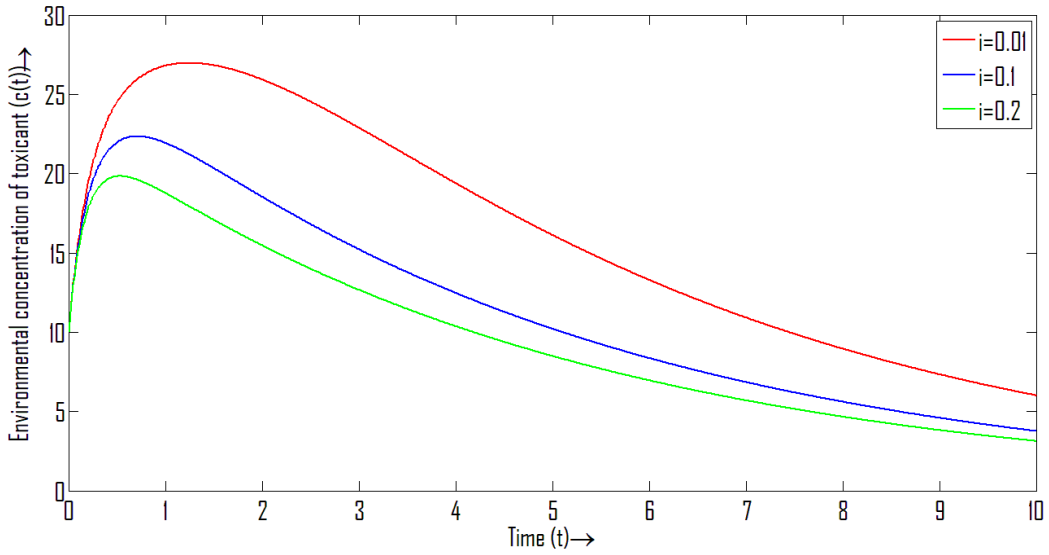
Graph 1:Variation in Infected predator population with time for different rates at which infected predator are decreasing due to toxicant



Graph 2:Variation in toxicant concentration in the population with time for different rates at which infected predator are decreasing due to toxicant

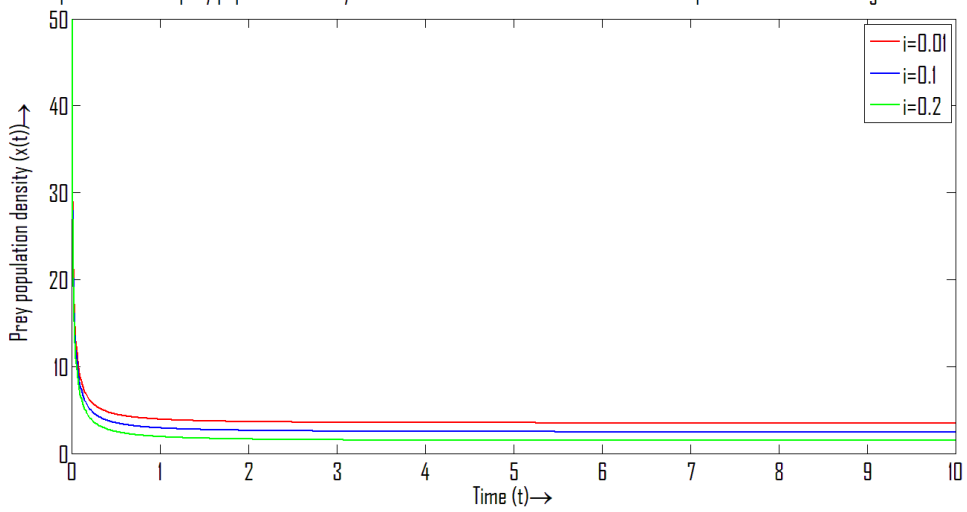


Graph 3:Variation in environmental concentration of toxicant with time for different rates at which infected predator are decreasing due to toxicant

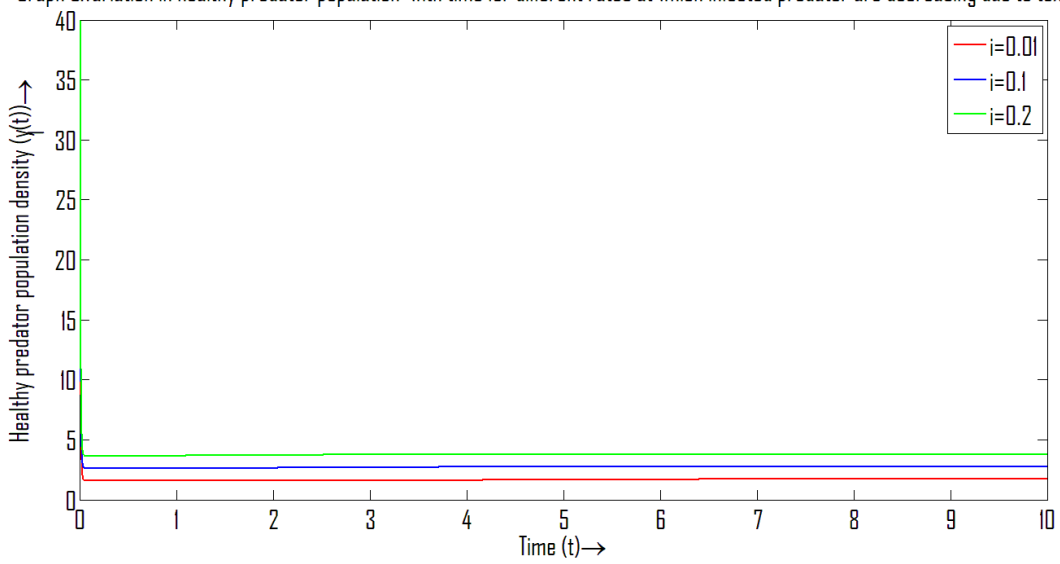


JOURNAL OF ALGEBRAIC STATISTICS
Volume 13, No. 3, 2022, p. 5470-5479
https://publishoa.com
ISSN: 1309-3452

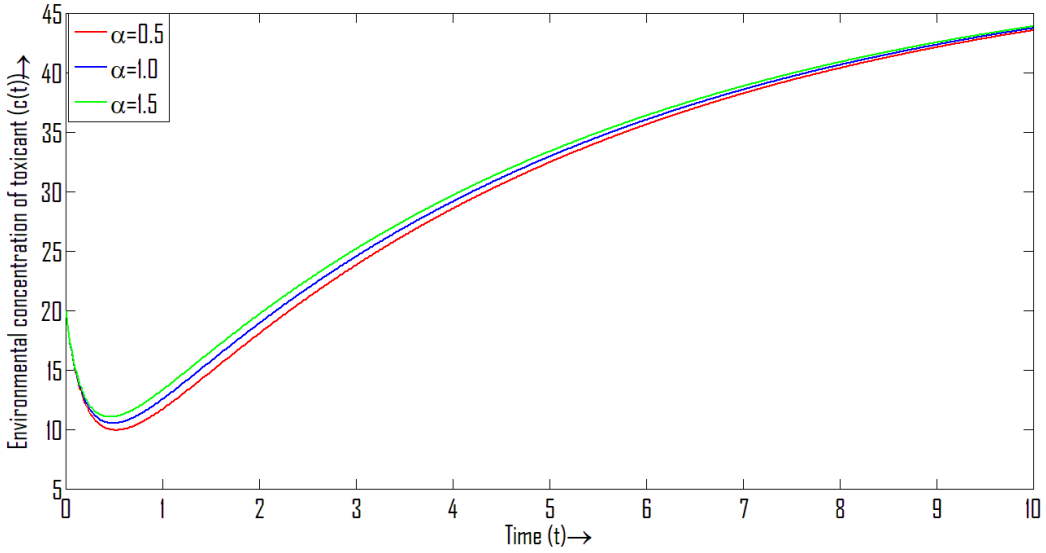
Graph 4:Variation in prey population density with time for different rates at which infected predator are decreasing due to toxicant



Graph 5:Variation in healthy predator population with time for different rates at which infected predator are decreasing due to toxicant

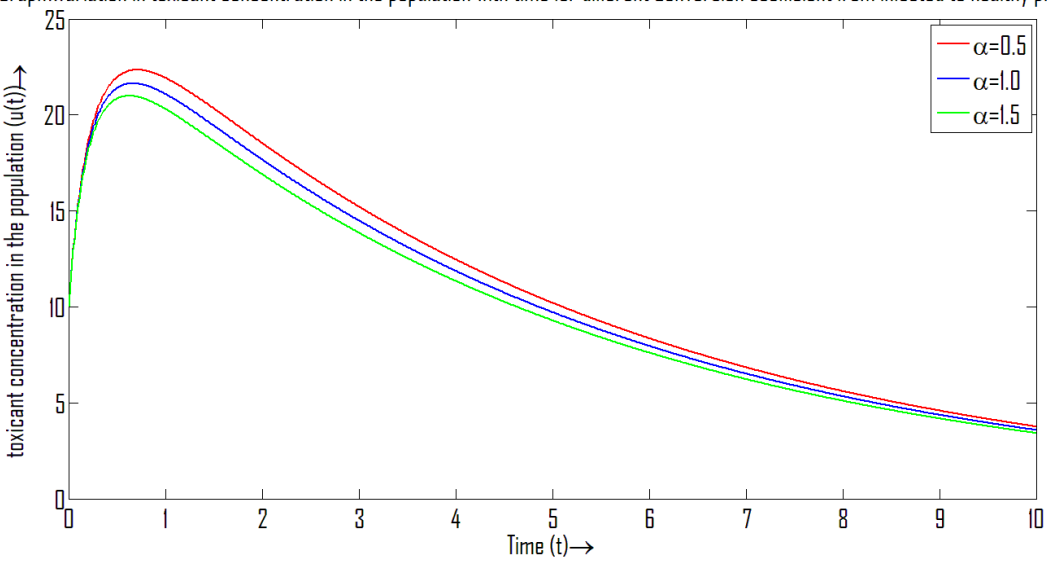


Graph 6:Variation in environmental concentration of toxicant with time for different conversion coefficient from infected to healthy predator



JOURNAL OF ALGEBRAIC STATISTICS
Volume 13, No. 3, 2022, p. 5470-5479
https://publishoa.com
ISSN: 1309-3452

Graph7:Variation in toxicant concentration in the population with time for different Conversion coefficient from infected to healthy predator



We provide a mathematical model in order to examine the impact of a toxicant on a prey-predator system when the population of predators is already afflicted with an illness. The analysis of the mathematical model shows that when a predator is sick with a disease, the level of equilibrium between the prey and the predator decreases due to the toxicant's negative effect on the system. This is shown by a lowering of the level of equilibrium between the prey and the predator. It has been mathematically demonstrated that the vulnerable prey population cannot expand without simultaneously growing the infected predator population. As a result, if the susceptible prey population grows, the infected predator population must shrink, and vice versa.

To better understand the effects of varying the parameters on the overall dynamics of the proposed model and to depict the aforementioned analytical findings, we turn to numerical simulation in this part. Specifically, the goals of this study are to verify the analytical results we have acquired and to determine the range of control parameters that influence the system's dynamics. Therefore, the model is solved numerically for different values of the parameters and initial conditions. A positive asymptotically stable equilibrium point is observed to be reached by the model for the set of hypothetical parameters given below.

r	Rate of growth that is inherent to the prey	1.1
k_1	The carrying capacity of the prey habitat	0.7
k_2	Capacity of the ecosystem to support predators	0.4
b	Coefficient of predation on healthy populations of prey	0.6
i	The rate at which infectious diseases are killing out predators because of toxicants	0.01
β	The infectious agents and their forces	0.9
α	The ratio of infected to healthy predators' conversion coefficient	0.5
μ_1	The proportion of prey that is lost due to natural causes	0.2
μ_2	The rate of natural mortality experienced by healthy predator populations	0.3
μ_3	The percentage of afflicted predators that die out naturally	0.5

JOURNAL OF ALGEBRAIC STATISTICS
Volume 13, No. 3, 2022, p. 5470-5479
https://publishoa.com
ISSN: 1309-3452

μ_4	The amount of time it takes for the toxin to be naturally removed from the organism.	0.2
d	Rate of mortality among infected predators as a result of infection	0.2
γ	The amount of the environmental toxin that is depleted by its natural processes	0.2
δ	The pace at which an organism takes up a toxicant	0.1
Q	The amount of the harmful substance that is introduced into the environment from outside sources	10
s	Rate of growth that is inherent to a healthy predator	1.2

6. Concluding Remarks-

The Lotka-Volterra Predator-Prey Model is an oversimplified representation of the real world's intricate ecological web. This theory argues that each predator has just one potential prey and vice versa, and that there are no environmental factors such as disease, climate change, pollution, etc. However, the model can be refined by include additional variables; doing so will allow us to obtain a more accurate representation of the ecosystem. However, as the number of variables increases, the model becomes more complicated and additional time and effort are needed to solve it. It also reveals the unique connection between biology and mathematics. Incorporating a toxicant into the Lotka-Volterra predator-prey model improves the realism of our simulations.

REFERENCES-

1. Anderson R.M., May R.M.(1986): "The invasion, persistence, and spread of infectious diseases within animal and plant communities", Philosophical Transaction of Royal society B biological sciences, 314:533–570
2. Liu B., Chen L., Zhang Y. (2003): "The effect of impulsive toxicant input on a population in a polluted environment", Journal of biological systems,11(3):265-274
3. Bairagi N., Chaudhuri S., Chattopadhyay J. (2009): "Harvesting as a disease control measure in an eco epidemiological system-A theoretical study", Mathematical Biosciences, 217:134-144
4. Baishya C., Achar S.J., Veeresha P., Prakasha D.G.(2021): "Dynamics of a fractional epidemiological model with disease infection in both the populations", Chaos: An Interdisciplinary Journal of Nonlinear Science, 31,043130:1-1-24
5. Bera S.P., Maiti A., Samanta G.P.(2016): "Dynamics of a food chain model with herd behaviour of the prey Model, Earth Syst. Environ., 2:1–9
6. Chen L., Chen F., Chen L. (2010): "Qualitative analysis of a predator– prey model with Holling type II functional response incorporating a constant prey refuge", Nonlinear Analysis: Real World Applications,11:246–252
7. Hethcote H.W., Wang W., Han L., Ma Z. (2014): "A predator-prey model with infected prey. Theoretical population biology, 34:849–858
8. Johri A., Trivedi N., Sisodiya A., Sing B., Jain S.(2012): "Study of a prey-predator model with disease prey", International Journal contemporary Mathematical sciences ,7:489–498
9. Kant S. and Kumar V. (2017): Stability analysis of predator-prey system with migrating prey and disease infection in both species, Applied mathematical modeling, 42:509–539.
10. Kermack W.O., McKendrick A.G.(1927): "Contributions to the mathematical theory of epidemics-part", Proceeding of Royal Society A , 115:700–721
11. Lotka, A.J. (1925): "Elements of Physical Biology", Nature, 116, 461
12. Malthus T.R. (1798): An Essay on the Principle of Population, J. Johnson in St. Paul's Churchyard, London, 1798.
13. Naji R. K. and Mustafa A. N. (2012): The dynamics of an eco-epidemiological model with nonlinear incidence rate, Journal of Applied Mathematics, Article ID 852631, 1–24.

14. S. Wang, Z. Ma, and W. Wang (2018): "Dynamical behavior of a generalized eco-epidemiological system with prey refuge," Advances in Difference Equations, Article ID 244
15. Sharma S., Samanta G.P.(2015): "Analysis of a two prey one predator system with disease in the first prey population", International Journal of Dynamics and control , 3:210–224
16. Thota S. (2020): " A Mathematical Study on a Diseased Prey-Predator Model with Predator Harvesting", Asian Journal of Fuzzy and Applied Mathematics, 8(2): 16-21
17. Volterra, V. (1928): "Variations and fluctuations of the number of individuals in animal species living together", ICES Journal of Marine Science, 3(1):3–51
18. Zhang J.S., Sun L.(2005): "Analysis of eco-epidemiological model with epidemic in the predator. Journal of Biomathematics, 20:157–164
19. Zhou F.,Yao H. (2015): "Dynamical behavior of a stage-structure epidemic model," International Journal of Nonlinear Science, 19(2):91–99