CHAPTER: 08

STABILITY ANALYSIS OF DISEASED PREY-PREDATOR MODEL IN A POLLUTED ENVIRONMENT

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ABSTRACT

When one or both predator and prey populations are afflicted by disease, it is especially important to understand the dynamics of predator-prey interactions in ecosystems increasingly affected by anthropogenic pollution. To better understand the intricate relationship between ecological and environmental aspects, this abstract proposes a theoretical framework for a sick prey-predator model in a polluted environment. Our model takes into account the interplay between prey, predator, and disease dynamics. The assumption is made that a contaminant has an influence on prey populations, either directly or indirectly. Diseases can spread between populations when predators and their prey come into contact with one another. Rates of predation, disease transmission, pollution exposure, and survival are all represented by factors we include. Our model's results show that pollution can alter the dynamics between prey and predator, leaving the system more vulnerable to disease outbreaks. Oscillations, extinction events, and changes in disease incidence are just a few examples of the complex and non-intuitive ecological patterns that can emerge when these elements interact. Because of their domino effect on ecosystems, environmental degradation and disease dynamics in the context of predator-prey interactions are highlighted in this study. To evaluate and lessen the effects of pollution on natural ecosystems, it is essential to conduct extensive ecological and environmental monitoring. Finally, in ecosystems where pollution constraints are increasing, understanding the dynamics of a diseased prey-predator relationship is critical for creating effective conservation and management solutions. This study offers important insights for sustainable ecosystem management and conservation by adding to our knowledge of how ecological systems react to anthropogenic perturbations.

Keywords: *Local Stability, Equilibrium points, Lotka-Volterra predator-prey model, Routh and Hurwitz criteria*

1. INTRODUCTION

Human activities are becoming a major source of environmental contamination, which poses a growing danger to the delicate balance of the world's ecosystems. Predator-prey interactions, which are frequently exacerbated by the presence of illnesses, have thus become a prominent area of ecological study. The importance of understanding and tackling the complex

problems currently facing ecosystems is emphasized by this introductory section, which lays the groundwork for future investigation of a sick prey-predator model in a polluted setting. Predator-prey relationships are an essential part of the ecological balance that has developed over eons. External influences such as climate change, habitat destruction, and broad pollutant release not only affect these interdependent connections but also threaten ecological stability. Pollution, in the form of chemical pollutants, heavy metals, and other industrial wastes, is a growing hazard to both terrestrial and aquatic ecosystems and is one of the most significant of these stressors. Released into the ecosystem without adequate control, pollutants can accumulate in species across all trophic levels, with far-reaching ecological consequences. Predator populations can be impacted when contaminants reduce the viability and abundance of their prey. And there's evidence that pollution can have an indirect effect on predator species, leading to decreases in population and even extinction. As a result of this dynamic interaction, the complexity of predator-prey relationships and ecosystem stability is amplified in the setting of a contaminated environment. Moreover, diseases present yet another level of complexity. Environmental stresses such as pollution can affect the dynamics of disease among predator and prey populations by influencing characteristics such as population density, host resistance, and transmission rates. To investigate the interplay between pollution, predators, and disease, a diseased prey-predator model in a polluted environment provides a useful theoretical framework. Facing global environmental concerns requires a thorough comprehension of this intricate relationship. There is a double whammy of danger to biodiversity and human health when ecosystems are stressed by pollution: not only do they run the risk of experiencing severe population swings and extinctions, but they may also serve as reservoirs for new infectious illnesses. Michael et al. in (2010) studied a general predator–prey system exhibiting fast evolution in either the predator or the prey.

Sinha et al. (2010) evaluated the combined influence of environmental toxicant and illness on prey-predator system. In this study, we assume that both prey and predator populations are impacted by environmental toxins, with diseased prey being more susceptible to the toxins and predators than their healthy counterparts. It was proposed and investigated by Naji and Mustafa in 2013. They hypothesized that the disease spread from prey to prey via direct contact at an exponential rate, although predators feed on their victims in accordance with a Holling type II functional response.

A sick prey-predator model with generic Holling type interactions was investigated by Sahoo and Poria (2014). They determined the system's permanence and impermanence by deriving the local stability requirements of equilibrium points. To examine the effects of rapid evolution on the body size of consumers and to investigate the impact of density-mediate indirect effect on the population dynamics and trait dynamics, Wang et al. (2017) formulated and explored an eco-evolutionary resource-consumer-predator trophic cascade model incorporating the rapid evolution. Mortoja et al. (2019) created a model of predator-prey interactions with a lag time. For a non-delayed system, they analyzed the crowding coefficient and found a Hopf bifurcation. Predator and prey (bacteria and ciliates) in the microbial system interact, and Kaitala et al. (2020) used the quantitative trait evolution model to simulate observational data, coming to the conclusion that this interaction is best explained as a coevolutionary process, in which both species evolved. A predator-prey model was presented and examined by Abdulkadhim and Al-Husseiny (2021). They talked about how pollution and immigration have both contributed to the spread of disease among predators. Using a predator-prey evolutionary model with a continuous phenotypic trait and a pulsed pollution discharge environment, Wang et al. (2021) studied the impact of pollution on the body size of prey. The effects of toxic compounds on species growth with time delays were the focus of Santra et al.'s (2023) consideration of a stage-structured predator-prey model in a toxic environment.

The complex ecological dynamics of such systems are the focus of this study. We want to understand the mechanisms and emergent features of these interrelated ecological elements by creating a mathematical model that mimics the interactions between polluted environments, ill prey, and their predators. In the end, this research helps us better understand how ecosystems react to human-caused disruptions and opens the door to more efficient conservation and management measures that will protect the diverse web of life on Earth.

2. MATHEMATICAL FORMULATION

This model is an expansion of the Lotka-Volterra predator-prey model that takes into account the impacts of pollution and disease on the populations of prey and predators. It is referred to as the Diseased Prey-Predator Model in a polluted environment.

The following is an example of how the equations for the Diseased Prey-Predator Model can be spelled out in a polluted environment:

Prey Equation:

$$\frac{dP}{dt} = rP - \frac{a_1 P}{1+h_1 P} - \frac{a_2 PQ}{1+h_2 P} - d_1 P \quad (1)$$

Predator Equation:

$$\frac{dQ}{dt} = -sQ + \frac{b_1 PQ}{1+h_3 Q} - \frac{b_2 Q}{1+h_4 Q} - d_2 Q \quad (2)$$

3. EQUILIBRIUM POINTS

Within the framework of a sick prey-predator model situated in an environment contaminated by pollutants, equilibrium points, alternatively referred to as steady states or fixed points, denote population and pollution levels wherein the dynamics of the system persist unchangingly across time. At points of equilibrium, the rates of change for all variables, including prey, predator, disease, and pollution, reach a state of equilibrium where they cease to change. The comprehension of the model's long-term behavior necessitates a thorough understanding of equilibrium points. Multiple equilibrium points can exist within a system, and analyzing their properties can yield valuable insights about the system's stability and behavior.

$$rP - \frac{a_1 P}{1+h_1 P} - \frac{a_2 PQ}{1+h_2 P} - d_1 P = 0$$

$$-sQ + \frac{b_1 PQ}{1+h_3 Q} - \frac{b_2 Q}{1+h_4 Q} - d_2 Q = 0$$

$$Q = 0$$

$$rP - \frac{a_1 P}{1+h_1 P} - d_1 P = 0$$

$$P\left[r - \frac{a_1}{1+h_1 P} - d_1\right] = 0$$

$$P = 0$$

$$r - \frac{a_1}{1+h_1 P} - d_1 = 0$$

$$\frac{a_1}{1+h_1 P} = r - d_1$$

$$1 + h_1 P = \frac{a_1}{r-d_1}$$

$$h_1 P = \frac{a_1}{r-d_1} - 1$$

$h_1P = \frac{a_1 - r + d_1}{r - d_1}$

$P = \frac{a_1 - r + d_1}{(r - d_1)h_1}$

$-sQ + \frac{b_1PQ}{1+h_3Q} - \frac{b_2Q}{1+h_4Q} - d_2Q = 0$

$P = 0$

$-sQ - \frac{b_2Q}{1+h_4Q} - d_2Q = 0$

$Q\left[-s - \frac{b_2}{1+h_4Q} - d_2\right] = 0$

$Q = 0, -s - \frac{b_2}{1+h_4Q} - d_2 = 0$

$\frac{b_2}{1+h_4Q} = s + d_2$

$1 + h_4Q = \frac{b_2}{s+d_2}$

$h_4Q = \frac{b_2}{s+d_2} - 1$

$h_4Q = \frac{b_2 - s - d_2}{s+d_2}$

$Q = \frac{b_2 - s - d_2}{h_4(s+d_2)}$

The equilibrium points are $(0,0)$ and $\left[\frac{a_1 - r + d_1}{(r - d_1)h_1}, \frac{b_2 - s - d_2}{h_4(s+d_2)}\right]$

4. STABILITY ANALYSIS

Stability analysis of a diseased prey-predator model in a polluted environment extends the concepts of traditional predator-prey models to consider the influence of pollution and disease on the population dynamics of the prey and predator species. To analyze the stability of such a model, you can follow a similar process to the one described in the previous answer, but with some modifications to account for the disease and pollution effects.

$\frac{dP}{dt} = rP - \frac{a_1P}{1+h_1P} - \frac{a_2PQ}{1+h_2P} - d_1P$

Predator Equation:

$\frac{dQ}{dt} = -sQ + \frac{b_1PQ}{1+h_3Q} - \frac{b_2Q}{1+h_4Q} - d_2Q$

We have the Jacobian matrix (3) for the system (1)-(2) as

$$J = \begin{bmatrix} r - \frac{a_1}{1+h_1P} - \frac{a_2Q}{1+h_2P} - d_1 & -\frac{a_2P}{1+h_2P} \\ \frac{b_1Q}{1+h_3Q} & -s + \frac{b_1P}{1+h_3Q} - \frac{b_2}{1+h_4Q} - d_2 \end{bmatrix} \quad (3)$$

4.1 The variational matrix (3) at $E_1 = (0,0)$ is given by

$$J(E_1) = \begin{bmatrix} r - a_1 - d_1 & 0 \\ 0 & -s - b_2 - d_2 \end{bmatrix} \quad (4)$$

$\lambda_1 = r - a_1 - d_1, \lambda_2 = -(s + b_2 + d_2)$

The all the eigen values of matrix (4) are not negative. Therefore $E_1 = (0,0)$ is locally unstable.

4.2 The variational matrix (3) at $E_2 = \left[\frac{a_1-r+d_1}{(r-d_1)h_1}, \frac{b_2-s-d_2}{h_4(s+d_2)}\right]$ is given by

$$J(E_2) = \begin{bmatrix} r - \frac{a_1}{1+h_1P} - \frac{a_2Q}{1+h_2P} - d_1 & -\frac{a_2P}{1+h_2P} \\ \frac{b_1Q}{1+h_3Q} & -s + \frac{b_1P}{1+h_3Q} - \frac{b_2}{1+h_4Q} - d_2 \end{bmatrix}$$

$$\frac{a_1}{1+h_1P} = \frac{a_1}{1+h_1\left[\frac{a_1-r+d_1}{(r-d_1)h_1}\right]} = \frac{a_1}{1+\frac{a_1-r+d_1}{r-d_1}} = \frac{a_1(r-d_1)}{r-d_1+a_1-r+d_1} = r - d_1$$

$$\frac{a_2Q}{1+h_2P} = \frac{a_2}{1+h_2\left[\frac{a_1-r+d_1}{(r-d_1)h_1}\right]}\left[\frac{b_2-s-d_2}{h_4(s+d_2)}\right] = \frac{a_2(r-d_1)h_1}{(r-d_1)h_1+h_2(a_1-r+d_1)}\frac{b_2-s-d_2}{h_4(s+d_2)}$$

$$r - \frac{a_1}{1+h_1P} - \frac{a_2Q}{1+h_2P} - d_1 = -\frac{a_2(r-d_1)h_1}{(r-d_1)h_1+h_2(a_1-r+d_1)}\frac{b_2-s-d_2}{h_4(s+d_2)} = -\frac{a_2(r-d_1)h_1(b_2-s-d_2)}{(r-d_1)h_1+h_2(a_1-r+d_1)}\frac{1}{h_4(s+d_2)}$$

$$r - \frac{a_1}{1+h_1P} - \frac{a_2Q}{1+h_2P} - d_1 = \frac{a_2h_1(r-d_1)(b_2-s-d_2)}{h_4(s+d_2)[(r-d_1)(h_1-h_2)+a_1h_2\,]}$$

$$\frac{a_2P}{1+h_2P} = \frac{a_2}{1+h_2\left[\frac{a_1-r+d_1}{(r-d_1)h_1}\right]}\left[\frac{a_1-r+d_1}{(r-d_1)h_1}\right] = \frac{a_2h_1(r-d_1)(a_1-r+d_1)}{h_1(r-d_1)[(r-d_1)(h_1-h_2)+a_1h_2\,]}$$

$$\frac{b_1Q}{1+h_3Q} = \frac{b_1}{1+h_2\left[\frac{b_2-s-d_2}{h_4(s+d_2)}\right]}\left[\frac{b_2-s-d_2}{h_4(s+d_2)}\right] = \frac{b_1h_4(s+d_2)(b_2-s-d_2)}{(r-d_1)h_1+h_2(a_1-r+d_1)}\frac{1}{h_4(s+d_2)} = \frac{b_1h_4(s+d_2)(b_2-s-d_2)}{h_4(s+d_2)[(h_4-h_2)(s+d_2)+h_2b_2]}$$

$$\frac{b_2}{1+h_4Q} = \frac{b_2}{1+\left[\frac{b_2-s-d_2}{(s+d_2)}\right]} = \frac{b_2(s+d_2)}{s+d_2+b_2-s-d_2} = s + d_2$$

$$-s + \frac{b_1P}{1+h_3Q} - \frac{b_2}{1+h_4Q} - d_2 = \frac{b_1P}{1+h_3Q} = \frac{b_1}{1+h_3\left[\frac{b_2-s-d_2}{h_4(s+d_2)}\right]}\left[\frac{a_1-r+d_1}{(r-d_1)h_1}\right] = \frac{b_1h_4(s+d_2)(a_1-r+d_1)}{h_1(r-d_1)[(s+d_2)(h_4-h_3)+h_3b_2\,]}$$

$$J(E_2) = \begin{bmatrix} \frac{a_2h_1(r-d_1)(b_2-s-d_2)}{h_4(s+d_2)[(r-d_1)(h_1-h_2)+a_1h_2\,]} & -\frac{a_2h_1(r-d_1)(a_1-r+d_1)}{h_1(r-d_1)[(r-d_1)(h_1-h_2)+a_1h_2\,]} \\ \frac{b_1h_4(s+d_2)(b_2-s-d_2)}{h_4(s+d_2)[(h_4-h_2)(s+d_2)+h_2b_2]} & \frac{b_1h_4(s+d_2)(a_1-r+d_1)}{h_1(r-d_1)[(s+d_2)(h_4-h_3)+h_3b_2\,]} \end{bmatrix}$$

$$J(E_3) = a_2h_1(r-d_1)b_1h_4(s+d_2)\begin{bmatrix} \frac{(b_2-s-d_2)}{h_4(s+d_2)[(r-d_1)(h_1-h_2)+a_1h_2]} & -\frac{(a_1-r+d_1)}{h_1(r-d_1)[(r-d_1)(h_1-h_2)+a_1h_2]} \\ \frac{(b_2-s-d_2)}{h_4(s+d_2)[(h_4-h_2)(s+d_2)+h_2b_2]} & \frac{(a_1-r+d_1)}{h_1(r-d_1)[(s+d_2)(h_4-h_3)+h_3b_2]} \end{bmatrix}$$

$$J(E_3) = \frac{a_2h_1(r-d_1)b_1h_4(s+d_2)}{h_4(s+d_2)h_1(r-d_1)}\begin{bmatrix} \frac{(b_2-s-d_2)}{[(r-d_1)(h_1-h_2)+a_1h_2]} & -\frac{(a_1-r+d_1)}{[(r-d_1)(h_1-h_2)+a_1h_2]} \\ \frac{(b_2-s-d_2)}{[(h_4-h_2)(s+d_2)+h_2b_2]} & \frac{(a_1-r+d_1)}{[(s+d_2)(h_4-h_3)+h_3b_2]} \end{bmatrix}$$

$$J(E_3) = \begin{bmatrix} \frac{a_2b_1(b_2-s-d_2)}{[(r-d_1)(h_1-h_2)+a_1h_2]} & -\frac{a_2b_1(a_1-r+d_1)}{[(r-d_1)(h_1-h_2)+a_1h_2]} \\ \frac{a_2b_1(b_2-s-d_2)}{[(h_4-h_2)(s+d_2)+h_2b_2]} & \frac{a_2b_1(a_1-r+d_1)}{[(s+d_2)(h_4-h_3)+h_3b_2]} \end{bmatrix} \quad (5)$$

$$J(E_3) = \begin{bmatrix} \frac{(b_2-s-d_2)}{[(r-d_1)(h_1-h_2)+a_1h_2]} - \lambda & -\frac{(a_1-r+d_1)}{[(r-d_1)(h_1-h_2)+a_1h_2]} \\ \frac{(b_2-s-d_2)}{[(h_4-h_2)(s+d_2)+h_2b_2]} & \frac{(a_1-r+d_1)}{[(s+d_2)(h_4-h_3)+h_3b_2]} - \lambda \end{bmatrix}$$

$$\left[\frac{(b_2-s-d_2)}{[(r-d_1)(h_1-h_2)+a_1h_2]} - \lambda\right]\left[\frac{(a_1-r+d_1)}{[(s+d_2)(h_4-h_3)+h_3b_2]} - \lambda\right] + \frac{(a_1-r+d_1)}{[(r-d_1)(h_1-h_2)+a_1h_2]}\frac{(b_2-s-d_2)}{[(h_4-h_2)(s+d_2)+h_2b_2]} = 0$$

$$\left[\frac{-(s+d_2-b_2)}{[(r-d_1)(h_1-h_2)+a_1h_2]} - \lambda\right]\left[\frac{-(r-d_1-a_1)}{[(s+d_2)(h_4-h_3)+h_3b_2]} - \lambda\right] + \frac{-(r-d_1-a_1)}{[(r-d_1)(h_1-h_2)+a_1h_2]}\frac{-(s+d_2-b_2)}{[(h_4-h_2)(s+d_2)+h_2b_2]} = 0$$

$$\left[\frac{(s+d_2-b_2)}{[(r-d_1)(h_1-h_2)+a_1h_2]} + \lambda\right]\left[\frac{(r-d_1-a_1)}{[(s+d_2)(h_4-h_3)+h_3b_2]} + \lambda\right] + \frac{(r-d_1-a_1)(s+d_2-b_2)}{[(r-d_1)(h_1-h_2)+a_1h_2][(h_4-h_2)(s+d_2)+h_2b_2]} = 0$$

$$\lambda^2 + \lambda\left[\frac{(s+d_2-b_2)}{\{(r-d_1)(h_1-h_2)+a_1h_2\}} + \frac{(r-d_1-a_1)}{\{(s+d_2)(h_4-h_3)+h_3b_2\}}\right] + \frac{2(r-d_1-a_1)(s+d_2-b_2)}{\{(r-d_1)(h_1-h_2)+a_1h_2\}\{(h_4-h_3)(s+d_2)+h_3b_2\}} = 0$$

The characteristic equation of jacobian matrix (5) is

$$\lambda^2 + k_1\lambda + k_2 = 0 \quad (6)$$

Where

$$k_1 = \frac{a_2b_1(s+d_2-b_2)}{\{(r-d_1)(h_1-h_2)+a_1h_2\}} + \frac{a_2b_1(r-d_1-a_1)}{\{(s+d_2)(h_4-h_3)+h_3b_2\}}$$

$$k_2 = \frac{2a_2{}^2b_1{}^2(r-d_1-a_1)(s+d_2-b_2)}{\{(r-d_1)(h_1-h_2)+a_1h_2\}\{(h_4-h_3)(s+d_2)+h_3b_2\}}$$

The characteristic equation (6) is comprised solely of positive coefficients for its variables. Hence, in accordance with the criteria established by Routh and Hurwitz, it can be concluded that the endemic equilibrium points exhibit local asymptotic stability.

5. RESULTS AND DISCUSSION

Now, let us proceed to develop a rudimentary MATLAB code for the purpose of simulating this model through the utilization of numerical integration techniques. Appropriate parameter values and initial conditions will be selected.

$$r = 0.1, K = 1000, a_1 = 0.02, a_2 = 0.01, h_1 = 500, h_2 = 500, d_1 = 0.05, s = 0.1, b_1 = 0.01$$

$h_3 = 100, b_2 = 0.01, h_4 = 100, d_2 = 0.02$

Initial conditions $P_0 = 800, Q_0 = 200$

Table 1: Input Parameters and their meanings

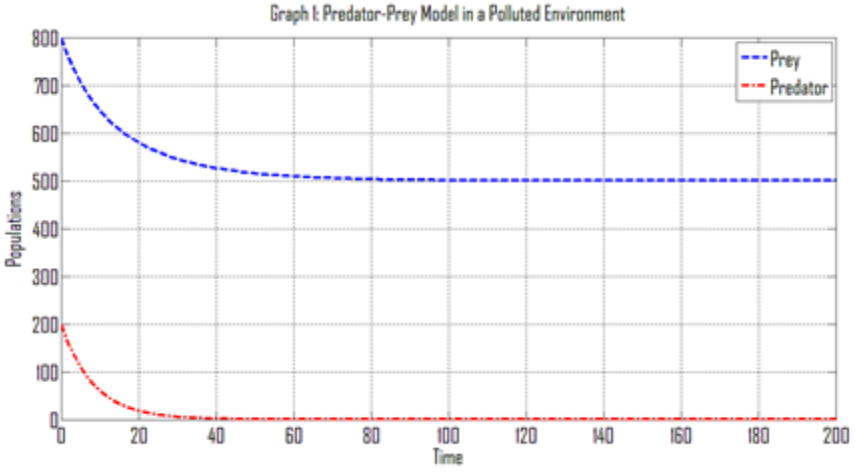
S.No.	Inputs	Meaning
1.	P	Population of prey
2.	Q	Population of predators
3.	r	Intrinsic growth rate of prey
4.	K	Carrying capacity of prey
5	a_1	Disease-induced mortality of prey due to pollution
6.	a_2	Disease-induced mortality of prey due to predation
7.	h_1	Half-saturation constant for pollution effect on prey
8.	h_2	Half-saturation constant for predation effect on prey
9.	d_1	Natural death rate of prey
10.	d_2	Natural death rate of predators
11.	s	Death rate of predators in the absence of prey
12.	b_1	Rate of increase in predator population due to predation
13.	b_2	Disease-induced mortality of predators due to pollution

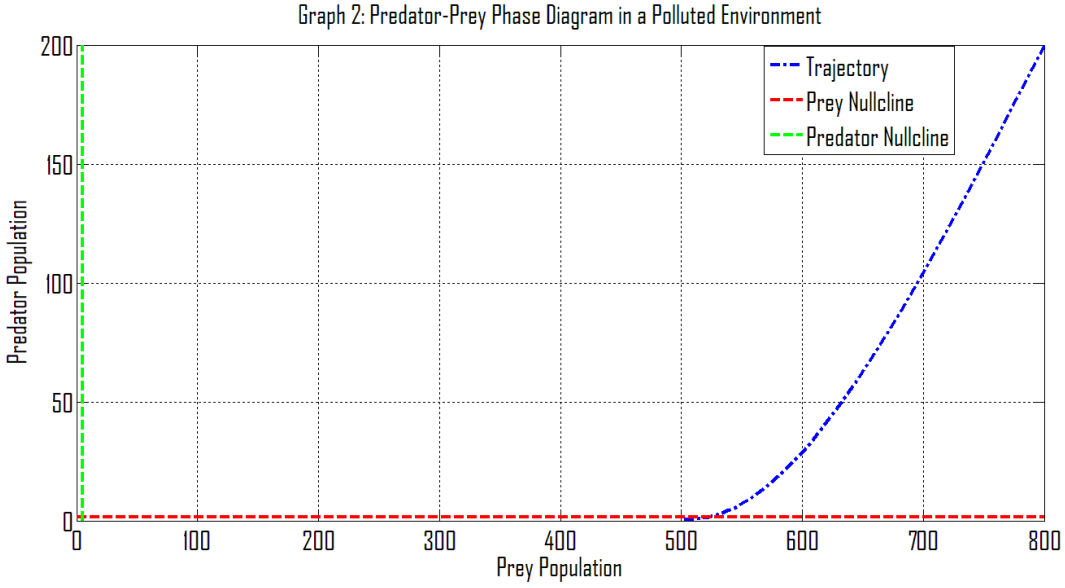
The graph (1) illustrates the dynamics of predator and prey populations in a sick Prey-Predator Model inside a polluted environment, offering a visual depiction of the interplay and fluctuations of these populations in response to many ecological and environmental conditions. The prevailing approach in this model is employing a system of differential equations to elucidate the temporal dynamics of prey and predator populations under the influence of illnesses and pollution.

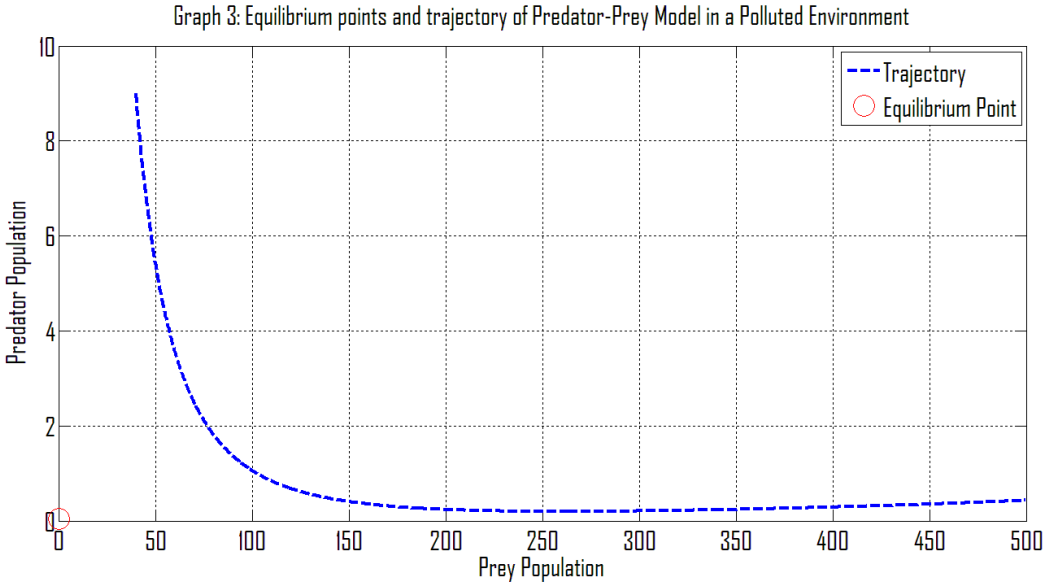
One notable characteristic exhibited by the graph (2) is the occurrence of predator-prey cycles. These cycles illustrate the oscillating patterns of population dynamics between predators and prey across a certain period. The augmentation of prey populations results in an increased availability of food resources for predators, hence leading to a subsequent rise in the predator

population. As the populations of predators increase, they exert heightened predation pressure on their prey, resulting in a subsequent decline in the numbers of prey individuals. The fall in prey populations subsequently results in a reduction in predator populations, hence establishing a cyclical trend.

The equilibrium points in a prey-predator model inside a polluted environment exhibit distinct characteristics that are contingent upon the model's parameters and underlying assumptions. In order to comprehend the intricate dynamics of such systems, more examination and numerical simulations are frequently necessary.







6. CONCLUDING REMARKS

The investigation of a diseased prey-predator model inside a contaminated environment has unveiled the complex and frequently unexpected manners in which natural systems react to human-induced disruptions. This study highlights the necessity of adopting a comprehensive methodology towards ecosystem management and preservation, which considers the intricate interactions among pollution, predator-prey relationships, and disease propagation. In conclusion, our examination of this subject reveals a number of significant facts and consequences. The widespread occurrence of contaminants in various ecosystems is a significant and urgent worldwide challenge. The presence of these contaminants has a disruptive impact on the intricate equilibrium of predator-prey dynamics, hence influencing the well-being and population numbers of species across various trophic levels. The findings of our study serve to underscore the pressing necessity of addressing both the origins and ramifications of environmental degradation. The study emphasizes the capacity of certain species to withstand the adverse effects of pollution and disease, whilst others exhibit increased susceptibility. It is imperative to comprehend the underlying factors that contribute to these discrepancies in order to facilitate effective conservation endeavors and ensure the preservation of biodiversity. The maintenance of stable predator-prey relationships is crucial for the sustenance of healthy ecosystems. The presence of stressors caused by pollution can result in oscillations in population sizes, the extinction of species, and changes in ecological dynamics. It is imperative to acknowledge the ramifications of these disruptions on the well-being of ecosystems in order to sustain biodiversity and ecological services. This study elucidates the role of pollution as a catalyst in the genesis and dissemination of diseases among wildlife populations. These diseases may potentially impact human health, highlighting the interdependence of ecosystems and the "One Health" principle. The results emphasize the significance of conducting thorough environmental monitoring and formulating solutions to alleviate the adverse effects of pollution on ecosystems. In the context of adaptive management approaches, it is imperative to take into account not only the mitigation of pollutants but also the safeguarding of crucial species within the impacted food web. The present study establishes a fundamental basis for further exploration of the precise processes that drive stress and disease transmission in ecosystems as a result of pollution exposure. Subsequent investigations may delve into the ramifications of diverse pollutant categories, fluctuating patterns of disease propagation, and the plausible occurrence of synergistic or antagonistic interactions. In summary, the examination of diseased prey-predator models within contaminated habitats highlights the complex and

interconnected dynamics that drive the ecological systems. In light of the pressing issues of pollution and environmental degradation, the insights derived from this research can serve as a valuable resource to inform and direct our endeavors in safeguarding and rehabilitating ecosystems. By acknowledging the intricate equilibrium inherent in these intricate systems, we move closer to achieving a more sustainable and mutually beneficial cohabitation with the natural environment, thereby ensuring the welfare of both wildlife and human beings within a dynamic and evolving ecosystem.

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