

Nonlinear Modeling and Stability Analysis of Simultaneous Effects of Reprotoxins in Dynamic Systems of Biological Species at Fixed Points and Their Control

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Received March 4, 2022; Revised August 17, 2022; Accepted September 19, 2022

Cite This Paper in the Following Citation Styles

(a): [1] Chetna Singh, Alok Agrawal, Amresh Kumar, Anuj Kumar Agarwal, Piyush Kumar Tripathi, "Nonlinear Modeling and Stability Analysis of Simultaneous Effects of Reprotoxins in Dynamic Systems of Biological Species at Fixed Points and Their Control," *Environment and Ecology Research*, Vol. 10, No. 5, pp. 519 - 541, 2022. DOI: 10.13189/eer.2022.100501.

(b): Chetna Singh, Alok Agrawal, Amresh Kumar, Anuj Kumar Agarwal, Piyush Kumar Tripathi (2022). *Nonlinear Modeling and Stability Analysis of Simultaneous Effects of Reprotoxins in Dynamic Systems of Biological Species at Fixed Points and Their Control*. *Environment and Ecology Research*, 10(5), 519 - 541. DOI: 10.13189/eer.2022.100501.

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Abstract Using a non-linear differential model, investigations were conducted to determine the harmful effects of two reprotoxins simultaneously in the reproduction process of the biological species. Reprotoxins are chemicals that have been shown to cause cancer in various parts of the reproductive system due to their effect on biological species and getting affected as their multistep protection network fails. The simultaneous effect shows various harmful effects depending upon the increasing concentration level or the quantity of uptake doses. Reprotoxin enters the body in various ways and produces deformities in various parts of the reproductive system. It can be seen in various forms including additive form, multiplicative form, or synergistic form. Both direct and indirect reprotoxin have a harmful impact on the reproduction process. Here, in this paper, we provide stability analysis at the equilibrium points for numerical solutions that characterise solutions of dynamical behaviour based on the parameters that are being used in the formulation of the systems. Toxication and detoxification, as well as some repair, occur inside the cell, organ, or other sites of the reproduction system. This includes enhanced protein synthesis to replace damaged or

non-functioning proteins. The goal of this work is to determine the circumstances or the level of growing reprotoxin at which the system is said to become entirely destabilized. To comprehend the movement of the trajectory path in the attractor basin towards or away from the fixed point, different mathematical structures have been developed. It concludes that the level of a species' population may be preserved by regulating the amount of various reprotoxins in the environment.

Keywords Mathematical Model, Reproductive Toxicant, Reproduction, Biological Species, Mathematical Structure

1. Introduction

The biological population of both aquatic and terrestrial systems that is impacted by toxicants released into the environment by external sources or produced by the species itself can be predicted very precisely using mathematical models. Due to the development of factories

and other human activities, the environment has become contaminated. The rising demand from the species, businesses and manufacturers releases numerous toxicants, including reprotoxins. Environmental conditions are adversely impacted by industrialization and the associated activities. The environment is constantly contaminated with reproductive toxicants such bisphenol-A, phthalates, heavy metals, herbicides, pesticides, fertilizers, and others. The release of reproductive toxicants is detrimental to the species' capacity to reproduce. The female reproductive system is rapidly changing, and many of its essential parts are still poorly understood. The most complex biological process is reproduction, which allows a species to grow and create more of its own kind. In order to maintain ecological balance, it is important to increase the population of a particular species because it depends on another for a variety of reasons. Exposure to reprotoxins can result in ovarian failure, infertility, growth retardation, intrauterine foetal mortality, birth defects, and pregnancy loss. Infertility, spontaneous miscarriages, developmental problems, irregular menstrual cycles, and contaminated breast milk can all result from the exposure to carbon disulfide. The number of inhabitants of a specific species decreases, resulting in an environmental imbalance. The researchers used nonlinear models to determine the impact of one or more toxicants on the species [5, 6, 14, 19, 23, 26, and 37]. The absorption concentration of toxicants by biological species affects the density growth rate linearly [18 & 19]. Shukla and Dubey [37] discovered that when two toxicants impact the same species, they have a simultaneous effect, with one toxicant being more harmful than the other. Shukla and Agrawal suggested the mathematical model in which they discovered that the species releases toxicants, and these toxicants [7] interact with one another. Agarwal [8] discovered that the effect of toxicants on biological species produces more detrimental effects than the other. Widdows [40] collected samples of mussels in several parts of the Venice Lagoon and determined that it was contaminated by various toxicants (For instance, Chromium, Mercury, Cadmium, Iron, Manganese, or chlorinated hydrocarbons) that caused a decrease in the population of species compared to other parts of the Lagoon. Dickman and Rygiel [16] evaluated the influence of toxic metals and oily debris in the Niagara River on the larvae of an invertebrate species of midge (chironomids) and discovered that roughly 26% of the chironomids were deformed from 10 to 800 meters downstream. The majority of studies have focused on a single toxicant, although in reality, the species lives under the influence of multiple toxicants, including the study of *Eurytemora affinis* development. Okamura and Aoyama [2] studied the influence of two heavy metals, cadmium and chromium, on algal growth in a synergistic effect. Munkittrick [31] discovered a link between the three acute lethality bioassays and the Microtox test in terms of correlation and relative sensitivity. Cairns [10] also looked

on the combined and individual effects of chlorine and ammonia on the aquatic environment. It has also been discovered that when the concentration of toxicants rises, the number of protozoa decreases by roughly 20%. Atlas [3] also focused on the impact of environmental toxins on microbial populations and estimated that the percentage of varieties lower than in other undisturbed reference communities. Woin and Bronmark [41] investigated how DDT and MCPA affected snail reproduction. Snails were discovered in a eutrophic wetland in southern Sweden. The effect of DDT and MCPA seems to have been producing its effect on the distribution of the snail, but has a lesser effect on the mortality rate. Abdul Rahman and Habib [1] investigated the Allelopathic effect of alfalfa *Medicago Sativa* blade grass (*Imperata cylindrical*) on soil microflora, finding that the toxic substance produced is not in large quantities but has a strong influence on the soil microflora, causing damage to the source and the progression of neighbouring plant development. Barlow and Sullivan [9] reproductive issues caused by industrial chemical exposures are not confined to pregnant women; males and females can be affected at any time. Lead, methyl mercury, beryllium, polychlorinated biphenyls, carbon monoxide, benzene, chlordecone, dibromochloropropane, and hexa-chlorobenzene are all recognized as being harmful to human reproduction. Huaping and Ma Zhien [21] utilized the mathematical modeling to evaluate the impact of pollution in a two-biological competitive ecosystem. Dubey [13] investigated the chemical defenses mechanism in which two competing species release toxicants on one another, affecting one another's survival in the same habitat. Okamura and Aoyama [32], the amount of one metal taken up in the cells and the rate of growth inhibition increased as the metal concentrations in the medium increased. The amount of one metal in the cells increased as a result of the presence of the other metal. The rate of growth inhibition increased as a result. The amount of Cr accumulated in each fraction was almost comparable in the absence of Cr. In both fractions, the addition of Cr increased the Cd concentration by 40%. According to Hyne and Wilson [22], the Australian bass is at risk of dying early due to the pollution of estuarine water with aluminium from acid sulphate soil leachate. When the simultaneous impacts of two toxicants are studied in a nonlinear dynamical system, Chattopadhyay [12] found that one produces a more detrimental effect than the other. Veeramachaneni [39] discovered that several chemical substances have a contaminating influence on the male reproduction process. Patil and David [33] investigated the various movements of fish in toxic media and discovered that the fish exhibited a variety of movements, including random, irregular, circular swimming motion, hyperexcitability, and the majority of the density was found moving in the bottom, causing the loss of equilibrium. Hartwell [20] investigated *Eurytemora affinis*, which grew in a variety of locations

and drained through chambers in a variety of locations, polluting rivers of the Chesapeake Bay. Sun [38] researched on the morphological abnormalities in a Taiwan river. These malformations were thought to be indicators for internal organ malfunction in various species. Lindley [29] investigated two organochlorine compounds, one of which is a respiratory uncoupler and the other a non-polar narcotic. Pentachlorophenol (PCP) and 1, 2-dichlorobenzene (DCB) are two organochlorine chemicals chosen to investigate the calanoid copepods, a neritic planktonic and the toxicity of estuarine eggs. The toxicity of estuarine eggs increases with the concentration of the two compounds (pesticides). Agrawal and Shukla [6] observed that toxicants are increasing from external sources, and as their toxicity nature increases in the atmosphere, so does their effectiveness on species, resulting in a wide range of harmful effects on the various functions of biological species. Ronit and Eldad [35] studied the impacts of inhibiting acetyl-cholinesterase and its activities on morphological and behavioural changes that occur during the reproduction process. Kumar [26] discovered the effect of various toxicants released into the environment through external sources (e.g., toxic metals, radioactive wastes, volcanic activity, biomass burning, and so on), and also the consequence of the species itself (e.g., vehicular exhaust, industries and domestic waste, fertilizer, etc.). It has been observed that their effect on the life of the subclass causes various internal and external deformities that are responsible for the dysfunctioning of the various activities of the organ in species. It was discovered that their internal disorders, such as decreased reproduction capability, asthma, cancer, and so on, were more severe than the morphological deformation, which caused the species' mortality rate to decrease below the carrying capacity. Kumar [27] investigated whether the simultaneous effect reduces the density of the species below the carrying capacity. Agrawal [5] discovered that reproduction toxicants from external sources affect the fertility rate in subclass of the species, and toxicants levels rise due to this species is more likely to become extinct from the ecosystem. Singh [36] has also researched that if the reproductive toxicant from the species' activities increases into the environment, the deformed population becomes equal to the total population density, raising the most critical situation in the environment. Situation approaches in the system where complete density undergoes deformities in the reproduction part due to hormonal changes responsible for reproduction process resulting in various physical and structural deformations. This rapidly decreases population density below the carrying capacity. It observed that the concentration of reprotoxin rises with the increasing demand of the species due to the growing population in a specific area. The

simultaneous effects of a wide range of reprotoxins on the biological species, and human body, on the reproductive system, have never been examined in a population environment. Due to their chemical reactivity, the majority of these compounds are cytotoxic, carcinogenic, or mutagenic, and reproductive toxicity is typically underestimated. So here we investigate the simultaneous hazardous effects of two reprotoxins which have an antagonistic effect on biological species and most of the species lose their reproduction capability, causing a decline in total population density and a decrease in the number of inhabitants in many species. The purpose of this paper is to discuss three important situations for the simultaneous effect of two reprotoxins, and it includes mathematical structures to enable us to understand the movement of the trajectory path in the region of attraction or attractor basin.

2. Mathematical Model

Consider a habitat in an ecosystem with a specific population density. Their growth is affected by the increasing rate of simultaneous effects of two reproductive toxicants emitting into the environment. Due to these effects in various subspecies groups, one capable of reproduction and other losing its reproductive capability as reprotoxin thresholds increased over time (t). $N_f(t)$ is denoting the subspecies capable of reproduction and $N_d(t)$ subspecies losing its capability of reproduction. It also assumed that one reprotoxin is increased due to the growing population of the species, and the other by the increasing demand of these populations. Some of this reprotoxin is emitted constantly, while some are in split or instantaneous form. Here, one reproductive toxicant $T_1(t)$ is discharging into the atmosphere with the emission rate λ by species itself and the other reproductive toxicant $T_2(t)$ from some external sources due to the growing demands of population i.e. Q at time t . $U_1(t)$ & $U_2(t)$ are the uptake concentration of reproductive toxicant of $T_1(t)$ and $T_2(t)$ respectively by species $N(t)$ at time t . The carrying capacity density $K(T_1, T_2)$ is associated with environmental concentrations of reprotoxins $T_1(t)$ and $T_2(t)$. Here, reprotoxin causes harm either directly or indirectly by structural similarity and chemical reaction. Reprotoxin misleads biological systems, including the reproduction process, by acting as an agonist and antagonist for the hormones involved for the reproduction process during the direct activation process. The reproductive processes are more sensitive and have a higher risk of cancer in the indirect activation phase. The proposed model is based on the increasing of reprotoxins effect from the various sources in the environment. This decreases the capability of reproduction in various species.

- In the logistic growth model, $\frac{dN}{dt} = \left(r - \frac{rN}{K}\right)N$, the term $\frac{rN^2}{K}$ is known as the crowding term due to competition within self.
- Overall per capita reproduction failure rate is $\frac{r_1 N_f U}{N_d} - d - \alpha$, where N_f is the subclass of the total population and capable in reproduction and N_d density of those population that becomes incapable of reproduction after the effect of reprotoxin.
- Where $r = b - d$ (*birth rate - death rate*) is the growth rate of N .

The assumptions are made taking into account the previous findings, facts and natural phenomenon discussed earlier in [4,5]. Therefore the proposed model is given below:

$$\begin{aligned} \frac{dN_f}{dt} &= \left[r - (r_1 U_1 + r_2 U_2) - \frac{1}{K(T_1, T_2)} [rN - (b + \alpha)N_d] \right] N_f \\ \frac{dN_d}{dt} &= \left(\frac{(r_1 U_1 + r_2 U_2)N_f}{N_d} - d - \alpha - \frac{1}{K(T_1, T_2)} [rN - (b + \alpha)N_d] \right) N_d \\ \frac{dT_1}{dt} &= \lambda N - \delta_1 T_1 - \gamma_1 N T_1 + \pi_1 v_1 N U_1 \\ \frac{dT_2}{dt} &= Q - \delta_2 T_2 - \gamma_2 N T_2 + \pi_2 v_2 N U_2 \\ \frac{dU_1}{dt} &= \gamma_1 N T_1 - \beta_1 U_1 - v_1 N U_1 \\ \frac{dU_2}{dt} &= \gamma_2 N T_2 - \beta_2 U_2 - v_2 N U_2 \end{aligned} \tag{2.1}$$

$$N(t) = N_f(t) + N_d(t), N_f(0), N_d(0), T_1, T_2 \geq 0, U_1 \geq c_1 T_1, U_2 \geq c_2 T_2, 0 < \pi_1 < 1, 0 < \pi_2 < 1$$

Here, α is the mortality rate of species in the presence of two reproductive toxicants at the same. δ_1 & δ_2 are the natural depletion rates coefficient of $T_1(t)$ & $T_2(t)$ respectively, β_1 & β_2 denote the natural depletion rates coefficient of $U_1(t)$ & $U_2(t)$ respectively, γ_1 & γ_2 are the depletion rates coefficient of $U_1(t)$ and $U_2(t)$ respectively due to the intake of reproductive toxicant by the total population N , v_1 & v_2 are the depletion rates coefficient of $U_1(t)$ & $U_2(t)$ respectively due to the decomposition of some members of N and π_1 & π_2 are fractions of the depletion of $U_1(t)$ & $U_2(t)$ due to decay of some members of N which reintegrated with environmental reprotoxins $T_1(t)$ & $T_2(t)$. Since, $N(t) = N_f(t) + N_d(t)$, then the model (2.1) is reduced as:

$$\begin{aligned} \frac{dN}{dt} &= [rN - (\alpha + b)N_d] \left[1 - \frac{N}{K(T_1, T_2)} \right] \\ \frac{dN_d}{dt} &= (r_1 U_1 + r_2 U_2)(N - N_d) - (\alpha + d)N_d - [rN - (\alpha + b)N_d] \frac{N_d}{K(T_1, T_2)} \\ \frac{dT_1}{dt} &= \lambda N - \delta_1 T_1 - \gamma_1 N T_1 + \pi_1 v_1 N U_1 \\ \frac{dT_2}{dt} &= Q - \delta_2 T_2 - \gamma_2 N T_2 + \pi_2 v_2 N U_2 \\ \frac{dU_1}{dt} &= \gamma_1 N T_1 - \beta_1 U_1 - v_1 N U_1 \\ \frac{dU_2}{dt} &= \gamma_2 N T_2 - \beta_2 U_2 - v_2 N U_2 \end{aligned} \tag{2.2}$$

In the assumed model (1), $c_1, c_2 > 0$ are constants for the initial concentration $U_1(0)$ & $U_2(0)$ with the initial density of biological population $N(0)$. The carrying capability function $K(T_1, T_2)$ has the following properties:

$$K(0,0) = K_0 > 0, K(T_1, T_2) > 0, \frac{\partial K(T_1, T_2)}{\partial T_1} < 0, \frac{\partial K(T_1, T_2)}{\partial T_2} < 0 \tag{2.3}$$

Here, K_0 is the maximum population of biological species for proposed system (2.2) when the reprotoxic effect on the species is negligible. To discuss the solution of reduced model (2.2) at the fixed point, we find out the region of attraction Ω (i.e. the attractor region or basin where the proposed system is completely defined). The determination and result of the region of attraction for the assumed system (2.2) is clearly shown in lemma (2.1)

Lemma.2.1.

The region of attraction of proposed system is denoted by Ω and

$$\Omega = \left\{ (N, N_d, T, U) : 0 \leq N \leq K_0, 0 \leq N_d \leq \frac{K_0(r_1 + r_2)(\lambda K_0 + Q)}{[(r_1 + r_2)(\lambda K_0 + Q) + (\alpha + d)\delta_m]}, 0 \leq (T_1 + T_2 + U_1 + U_2) \leq \frac{(\lambda K_0 + Q)}{\delta_m} \right\},$$

where $\delta_m = \min(\delta_1, \delta_2, \beta_1, \beta_2)$ attracts solutions in the interior of the positive orthant. This is the region where the movement of the trajectory path with the increasing reprotoxin can be easily seen at the fixed point.

Proof

The first equation of model (2.1):

$$\frac{dN}{dt} = [rN - (\alpha + b)N_d] \left[1 - \frac{N}{K(T_1, T_2)} \right]$$

Gives:

$$\frac{dN}{dt} \leq rN \left\{ 1 - \frac{N}{K(T_1, T_2)} \right\}$$

Thus, $\limsup_{t \rightarrow \infty} N(t) \leq K_0$.

The second equation for model (2.1) is given below:

$$\frac{dN_d}{dt} = (r_1U_1 + r_2U_2)(N - N_d) - (\alpha + d)N_d - [rN - (\alpha + b)N_d] \frac{N_d}{K(T_1, T_2)}$$

Gives:

$$\begin{aligned} \frac{dN_d}{dt} &\leq (r_1U_1 + r_2U_2)N - (r_1U_1 + r_2U_2 + \alpha + d)N_d \\ &\leq \frac{K_0}{\delta_m} (r_1 + r_2)(\lambda K_0 + Q) - \left[\frac{1}{\delta_m} (r_1 + r_2)(\lambda K_0 + Q) + (\alpha + d) \right] N_d \end{aligned}$$

Thus, $\limsup_{t \rightarrow \infty} N_d(t) \leq \frac{K_0(r_1+r_2)(\lambda K_0+Q)}{[(r_1+r_2)(\lambda K_0+Q)+(\alpha+d)\delta_m]}$

Now we add the last four equations of model (2.1), which are given below:-

$$\begin{aligned} \frac{dT_1}{dt} + \frac{dT_2}{dt} + \frac{dU_1}{dt} + \frac{dU_2}{dt} &= \lambda N + Q - \delta_1 T_1 - \delta_2 T_2 - \beta_1 U_1 - \beta_2 U_2 - (1 - \pi_1)v_1 N U_1 - (1 - \pi_2)v_2 N U_2 \\ &\leq (\lambda K_0 + Q) - \delta_m (T_1 + T_2 + U_1 + U_2). \end{aligned}$$

Where, $\delta_m = \min\{\delta_1, \delta_2, \beta_1, \beta_2\}$ Thus, $\limsup_{t \rightarrow \infty} (T_1 + T_2 + U_1 + U_2) \leq \frac{(\lambda K_0 + Q)}{\delta_m}$.

3. Stability Analysis

Understanding the proposed system's dynamic behaviour requires finding the solution to the system. It is possible to qualitatively determine how the dynamical system will behave close to each equilibrium point. To find the equilibrium point, all of the variables' derivatives are set to zero. If the output is regulated, that is, if both the input and the output are constrained, the system solution is stable. The trajectory route will move in a direction that eventually converges to the equilibrium while also keeping sufficiently close if the starting point is close to the equilibrium point.

3.1. Equilibrium Points of Model

Now let us determine to characterise at each fixed point in terms of the dynamical behaviours that are responsible for the system's stability in the region of the attraction. The dynamic system is stable if solutions near a fixed point of proposed system move close to the fixed point as time approaches infinity. The equations (2.2) are solved analytically to obtain the best approximate solution for the suggested biological species model. As a result, multiple analytical approaches for the best estimated outcome of the suggested model are included in this study. If the output is controlled, that is, both input and output are bounded; the system's solution is stable. Three non-negative equilibrium points are obtained in the proposed model, as shown below.

$$E_1 = \left\{ 0, 0, 0, \frac{Q}{\delta_2}, 0, 0 \right\}, E_2 = \{ \tilde{N}, \tilde{N}_d, \tilde{T}_1, \tilde{T}_2, \tilde{U}_1, \tilde{U}_2 \}, E_3 = \{ N^*, N_d^*, T_1^*, T_2^*, U_1^*, U_2^* \}$$

Here, the existence of E_1 is obvious. Now we check the existence of uniqueness and the existence at the equilibrium points E_2 & E_3 which would be providing the positive value of $\{ \tilde{N}, \tilde{N}_d, \tilde{T}_1, \tilde{T}_2, \tilde{U}_1, \tilde{U}_2 \}$ & $\{ N^*, N_d^*, T_1^*, T_2^*, U_1^*, U_2^* \}$ respectively.

Existence and uniqueness of E_2 : Here $\tilde{N}, \tilde{N}_d, \tilde{T}_1, \tilde{T}_2, \tilde{U}_1$ & \tilde{U}_2 are the solutions of the dynamical system of equations.

$$N = K(T_1, T_2) \quad (2.4a)$$

$$N_d = K(T_1, T_2) \quad (2.4b)$$

$$T_1 = \frac{\lambda N(\beta_1 + v_1 N)}{f_1(N)} = g_1(N) \quad (2.4c)$$

$$T_2 = \frac{Q(\beta_2 + v_2 N)}{f_2(N)} = g_2(N) \quad (2.4d)$$

$$U_1 = \frac{\lambda \gamma_1 N^2}{f_1(N)} = h_1(N) \quad (2.4e)$$

$$U_2 = \frac{Q \gamma_2 N}{f_2(N)} = h_2(N) \quad (2.4f)$$

where, $f_1(N) = \{\delta_1 \beta_1 + (\gamma_1 \beta_1 + \delta_1 v_1)N + \gamma_1 v_1(1 - \pi_1)N^2\}$ (2.4g)

$$f_2(N) = \{\delta_2 \beta_2 + (\gamma_2 \beta_2 + \delta_2 v_2)N + \gamma_2 v_2(1 - \pi_2)N^2\} \quad (2.4h)$$

Let, $F_1(N) = N - K(T_1, T_2) = N - K(g_1(N), g_2(N))$ (2.5)

From the above, it is clear that

$$F_1(0) < 0 \text{ and } F_1(K_0) > 0 \quad (2.6)$$

$$F_1(N) = N - K(T_1, T_2) = N - K(g_1(N), g_2(N)) \quad (2.7)$$

$$\frac{d}{dN} F_1(N) = \left[1 - \left\{ \frac{\partial K}{\partial T_1} \frac{dg_1}{dN} + \frac{\partial K}{\partial T_2} \frac{dg_2}{dN} \right\} \right] \quad (2.8)$$

From, equations (2.4c & 2.4d), we get

$$\frac{dg_1}{dN} = \frac{\lambda}{f_1^2(N)} \{\delta_1(\beta_1 + v_1 N)^2 + \gamma_1 v_1 \pi_1 \beta_1 N^2\} > 0 \quad (2.9a)$$

$$\frac{dg_2}{dN} = \frac{-\gamma_2 Q}{f_2^2(N)} \{(1 - \pi_2)(\beta_2 + v_2 N)^2 + \pi_2 \beta_2^2\} < 0 \quad (2.9b)$$

Equations (2.8, 2.9a & 2.9b) imply that $\frac{d}{dN} F_1(N) > 0$, as well as root N in system of equations (2.4a – 2.4f) will be unique, if the following condition holds, i.e.

$$\frac{\partial K}{\partial T_2} \frac{dg_2}{dN} < 1 + \left| \frac{\partial K}{\partial T_1} \frac{dg_1}{dN} \right| \quad (2.10)$$

From the above equations (2.6, 2.8) it is clear that $F_1(N) = 0$ has a unique root N in the interval $[0, K_0]$ under a certain condition (2.10). Since all the above conditions are satisfied so this suggests that knowing the value of \tilde{N} , all the other values i.e. $\tilde{N}_d, \tilde{T}_1, \tilde{T}_2, \tilde{U}_1$ & \tilde{U}_2 can be computed from the equations (2.4b – 2.4h). From the above, it is clear that there exists the value for $\tilde{N}, \tilde{N}_d, \tilde{T}_1, \tilde{T}_2, \tilde{U}_1$ & \tilde{U}_2 .

Now, we further check for the positive solution at the fixed point E_3 .

Existence and uniqueness of E_3 : Here $N^*, N_d^*, T_1^*, T_2^*, U_1^*$ & U_2^* are the solutions of the dynamical system of equations.

$$N = K(T_1, T_2) \quad (2.11a)$$

$$N_d = \frac{(r_1 U_1 + r_2 U_2)}{(\alpha + b)} K(T_1, T_2) \quad (2.11b)$$

and T_1, T_2, U_1, U_2 are the same as (2.4c – 2.4f).

Since the value of N in equation (2.11a) is the same as in equation (2.4a). So, the existence and uniqueness equilibrium point $E_3(N^*, N_d^*, T_1^*, T_2^*, U_1^*$ & $U_2^*)$ is the same as existence and uniqueness of the fixed point $E_2(\tilde{N}, \tilde{N}_d, \tilde{T}_1, \tilde{T}_2, \tilde{U}_1, \tilde{U}_2)$.

3.2. Local Stability Equilibrium Analysis

The jacobian matrix M_1 corresponding to the equilibrium point $E_1 = (0, 0, 0, \frac{Q}{\delta_2}, 0, 0)$ is given below:

$$M_1 = \begin{bmatrix} r & -(\alpha + b) & 0 & 0 & 0 & 0 \\ 0 & -(\alpha + d) & 0 & 0 & 0 & 0 \\ \lambda & 0 & -\delta_1 & 0 & 0 & 0 \\ \frac{-\gamma_2 Q}{\delta_2} & 0 & 0 & -\delta_2 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\beta_1 & 0 \\ \frac{\gamma_2 Q}{\delta_2} & 0 & 0 & 0 & 0 & -\beta_2 \end{bmatrix}$$

Here, the eigenvalues of jacobian matrix M_1 are $r, -(\alpha + d), -\delta_1, -\delta_2, -\beta_1, -\beta_2$. As it is clearly seen that all the eigenvalues are not negative at this equilibrium point therefore the system has a saddle point and is unstable. The system has a locally unstable manifold in the direction of N and a locally stable manifold in the direction of $N_d - T_1 - T_2 - U_1 - U_2$ space. Therefore the proposed system is unstable at equilibrium point E_2 . The jacobian matrix M_2 corresponding to the equilibrium point $E_2 = \tilde{N}, \tilde{N}_d, \tilde{T}_1, \tilde{T}_2, \tilde{U}_1$ & \tilde{U}_2 is given below:

$$M_2 = \begin{bmatrix} l_{11} & 0 & l_{13} & l_{14} & 0 & 0 \\ l_{21} & l_{22} & l_{23} & l_{24} & 0 & 0 \\ l_{31} & 0 & l_{33} & 0 & l_{35} & 0 \\ l_{41} & 0 & 0 & l_{44} & 0 & l_{46} \\ l_{51} & 0 & l_{53} & 0 & l_{55} & 0 \\ l_{61} & 0 & 0 & l_{64} & 0 & l_{66} \end{bmatrix}$$

$$l_{11} = d + \alpha, l_{13} = -(\alpha + d) \left(\frac{\partial K}{\partial T_1} \right)_{E_2}, l_{14} = -(\alpha + d) \left(\frac{\partial K}{\partial T_2} \right)_{E_2}, l_{21} = (r_1 \tilde{U}_1 + r_2 \tilde{U}_2) - r,$$

$$l_{22} = -(r_1 \tilde{U}_1 + r_2 \tilde{U}_2) + \alpha + b, l_{23} = -(\alpha + d) \left(\frac{\partial K}{\partial T_1} \right)_{E_2}, l_{24} = -(\alpha + d) \left(\frac{\partial K}{\partial T_2} \right)_{E_2}$$

$$l_{31} = \lambda - \gamma_1 \tilde{T}_1 + \pi_1 v_1 \tilde{U}_1, l_{33} = -(\delta_1 + \gamma_1 K(\tilde{T}_1, \tilde{T}_2)), l_{35} = \pi_1 v_1 K(\tilde{T}_1, \tilde{T}_2)$$

$$l_{41} = -\gamma_2 \tilde{T}_2 + \pi_2 v_2 \tilde{U}_2, l_{44} = -(\delta_2 + \gamma_2 K(\tilde{T}_1, \tilde{T}_2)), l_{46} = \pi_2 v_2 K(\tilde{T}_1, \tilde{T}_2)$$

$$l_{51} = \gamma_1 \tilde{T}_1 - v_1 \tilde{U}_1, l_{53} = \gamma_1 K(\tilde{T}_1, \tilde{T}_2), l_{55} = -(\beta_1 + v_1 K(\tilde{T}_1, \tilde{T}_2)),$$

$$l_{61} = \gamma_2 \tilde{T}_2 - v_2 \tilde{U}_2, l_{64} = \gamma_2 K(\tilde{T}_1, \tilde{T}_2), l_{66} = -(\beta_2 + v_2 K(\tilde{T}_1, \tilde{T}_2))$$

On solving the above jacobian matrix M_2 , we conclude that one of the eigenvalues is non-negative and equal to $2(\alpha + b) > 0$. This shows that the proposed system of biological species is unstable at the equilibrium point E_2 . Now we determine the eigenvalue of the M_3 corresponding to the fixed point $E_3 = (N^*, N_d^*, T_1^*, T_2^*, U_1^*, U_2^*)$ is:

$$M_3 = \begin{bmatrix} m_{11} & m_{12} & m_{13} & m_{14} & 0 & 0 \\ m_{21} & m_{22} & m_{23} & m_{24} & m_{25} & m_{26} \\ m_{31} & 0 & m_{33} & 0 & m_{35} & 0 \\ m_{41} & 0 & 0 & m_{44} & 0 & m_{46} \\ m_{51} & 0 & m_{53} & 0 & m_{55} & 0 \\ m_{61} & 0 & 0 & m_{64} & 0 & m_{66} \end{bmatrix}$$

Moreover,

$$m_{11} = -r \left\{ \frac{2N^*}{K(T_1^*, T_2^*)} - 1 \right\} + \frac{(\alpha + b)N_d^*}{K(T_1^*, T_2^*)}, m_{12} = -(\alpha + b) \left\{ 1 - \frac{N^*}{K(T_1^*, T_2^*)} \right\},$$

$$m_{13} = -[rN^* - (\alpha + b)N_d^*] \frac{N^*}{K^2(T_1^*, T_2^*)} \left(\frac{\partial K}{\partial T_1} \right)_{E_3},$$

$$m_{14} = -[rN^* - (\alpha + b)N_d^*] \frac{N^*}{K^2(T_1^*, T_2^*)} \left(\frac{\partial K}{\partial T_2} \right)_{E_3}, m_{15} = 0, m_{16} = 0$$

$$m_{21} = (r_1 U_1^* + r_2 U_2^*) - \frac{rN_d^*}{K(T_1^*, T_2^*)}, m_{22} = -(r_1 U_1^* + r_2 U_2^* + \alpha + d) + (\alpha + b) \frac{2N_d^*}{K(T_1^*, T_2^*)},$$

$$\begin{aligned}
 m_{23} &= [rN^* - (\alpha + b)N_d^*] \left[\frac{N_d^*}{K^2(T_1^*, T_2^*)} \right] \left(\frac{\partial K}{\partial T_1} \right)_{E_3}, \\
 m_{24} &= [rN^* - (\alpha + b)N_d^*] \left[\frac{N_d^*}{K^2(T_1^*, T_2^*)} \right] \left(\frac{\partial K}{\partial T_2} \right)_{E_3}, \\
 m_{25} &= r_1(N^* - N_d^*), m_{26} = r_2(N^* - N_d^*), m_{31} = \lambda - \gamma_1 T_1^* + \pi_1 v_1 U_1^*, m_{32} = 0, \\
 m_{33} &= -(\delta_1 + \gamma_1 N^*), m_{34} = 0, m_{35} = \pi_1 v_1 N^*, m_{36} = 0 \\
 m_{41} &= -\gamma_2 T_2^* + \pi_2 v_2 U_2^*, m_{42} = 0, m_{43} = 0, m_{44} = -(\delta_2 + \gamma_2 N^*), m_{45} = 0 \\
 m_{46} &= \pi_2 v_2 N^*, m_{51} = \gamma_1 T_1^* - v_1 U_1^*, m_{52} = 0, m_{53} = \gamma_1 N^*, m_{54} = 0 \\
 m_{55} &= -(\beta_1 + v_1 N^*), m_{56} = 0, m_{61} = \gamma_2 T_2^* - v_2 U_2^*, m_{62} = 0, m_{63} = 0, m_{64} = \gamma_2 N^*, \\
 m_{55} &= 0, m_{66} = -(\beta_2 + v_2 N^*)
 \end{aligned}$$

Here, M_3 is the jacobian matrix corresponding to the fixed point E_3 . Therefore, the polynomial equation of M_3 can be given as,

$$P(x) = x^6 + B_1 x^5 + B_2 x^4 + B_3 x^3 + B_4 x^2 + B_5 x + B_6. \quad (2.12)$$

Where,

$$\begin{aligned}
 B_1 &= -(m_{11} + m_{22} + m_{33} + m_{44} + m_{55} + m_{66}) \\
 B_2 &= (m_{11} + m_{22})(m_{55} + m_{66}) + m_{22}(m_{33} + m_{44}) + (m_{33} + m_{55})(m_{44} + m_{66}) \\
 &\quad + \left| \frac{m_{11}m_{12}}{m_{21}m_{22}} \right| + \left| \frac{m_{11}m_{13}}{m_{31}m_{33}} \right| + \left| \frac{m_{11}m_{14}}{m_{41}m_{44}} \right| + \left| \frac{m_{33}m_{35}}{m_{53}m_{55}} \right| + \left| \frac{m_{44}m_{46}}{m_{64}m_{66}} \right| \\
 B_3 &= m_{14}m_{41}m_{33} - m_{11}m_{55}m_{66} - m_{12}(m_{25}m_{51} + m_{26}m_{61}) - m_{22}(m_{33} + m_{55})(m_{44} + m_{66}) + m_{13} \left| \frac{m_{31}m_{35}}{m_{51}m_{55}} \right| \\
 &\quad + m_{14} \left| \frac{m_{41}m_{46}}{m_{61}m_{66}} \right| - m_{31} \left| \frac{m_{12}m_{13}}{m_{22}m_{23}} \right| - m_{41} \left| \frac{m_{12}m_{14}}{m_{22}m_{24}} \right| - m_{55} \left| \frac{m_{11}m_{14}}{m_{41}m_{44}} \right| - (m_{44} + m_{66}) \left| \frac{m_{11}m_{13}}{m_{31}m_{33}} \right| \\
 &\quad - (m_{11} + m_{22} + m_{44} + m_{66}) \left| \frac{m_{33}m_{35}}{m_{53}m_{55}} \right| - (m_{11} + m_{22} + m_{33} + m_{55}) \left| \frac{m_{44}m_{46}}{m_{64}m_{66}} \right| \\
 &\quad - (m_{33} + m_{44} + m_{55} + m_{66}) \left| \frac{m_{11}m_{12}}{m_{21}m_{22}} \right| \\
 B_4 &= m_{12} \left\{ m_{26}m_{61}(m_{33} + m_{55}) + m_{25}m_{51}(m_{44} + m_{66}) + m_{31} \left| \frac{m_{23}m_{25}}{m_{53}m_{55}} \right| + m_{41} \left| \frac{m_{24}m_{26}}{m_{64}m_{66}} \right| - m_{51} \left| \frac{m_{23}m_{25}}{m_{33}m_{66}} \right| \right. \\
 &\quad \left. - m_{61} \left| \frac{m_{24}m_{26}}{m_{44}m_{46}} \right| \right\} + m_{31}(m_{44} + m_{66}) \left| \frac{m_{12}m_{13}}{m_{22}m_{23}} \right| + m_{41}(m_{33} + m_{55}) \left| \frac{m_{12}m_{14}}{m_{22}m_{24}} \right| \\
 &\quad - m_{13}(m_{22} + m_{44} + m_{66}) \left| \frac{m_{31}m_{35}}{m_{51}m_{55}} \right| - m_{14}(m_{22} + m_{33} + m_{55}) \left| \frac{m_{41}m_{46}}{m_{61}m_{66}} \right| \\
 &\quad + \left\{ (m_{33} + m_{55})(m_{44} + m_{66}) + \left| \frac{m_{33}m_{35}}{m_{53}m_{55}} \right| + \left| \frac{m_{44}m_{46}}{m_{64}m_{66}} \right| \right\} \\
 &\quad \left| \frac{m_{11}m_{12}}{m_{21}m_{22}} \right| + \left\{ m_{11}m_{66} + m_{22}(m_{44} + m_{66}) + \left| \frac{m_{11}m_{14}}{m_{41}m_{44}} \right| + \left| \frac{m_{44}m_{46}}{m_{64}m_{66}} \right| \right\} \left| \frac{m_{33}m_{35}}{m_{53}m_{55}} \right| \\
 &\quad + \left\{ m_{11}m_{55} + m_{22}(m_{33} + m_{55}) + \left| \frac{m_{11}m_{13}}{m_{31}m_{33}} \right| \right\} \left| \frac{m_{44}m_{46}}{m_{64}m_{66}} \right| \\
 B_5 &= (m_{33} + m_{55}) \left\{ m_{14}m_{22} \left| \frac{m_{41}m_{46}}{m_{61}m_{66}} \right| + m_{61}m_{12} \left| \frac{m_{24}m_{26}}{m_{44}m_{46}} \right| - m_{12}m_{41} \left| \frac{m_{24}m_{26}}{m_{64}m_{66}} \right| \right\} \\
 &\quad + (m_{44} + m_{66}) \left\{ m_{12}m_{51} \left| \frac{m_{23}m_{25}}{m_{33}m_{35}} \right| - m_{12}m_{31} \left| \frac{m_{23}m_{25}}{m_{53}m_{55}} \right| \right\} \\
 &\quad + m_{13} \left\{ m_{22}(m_{44} + m_{66}) + \left| \frac{m_{44}m_{46}}{m_{64}m_{66}} \right| \right\} \left| \frac{m_{31}m_{35}}{m_{51}m_{55}} \right| - \left\{ m_{12}m_{25}m_{51} + m_{31} \left| \frac{m_{12}m_{13}}{m_{22}m_{23}} \right| \right\} \left| \frac{m_{44}m_{46}}{m_{64}m_{66}} \right| \\
 &\quad - \left\{ (m_{44} + m_{66}) \left| \frac{m_{33}m_{35}}{m_{53}m_{55}} \right| + (m_{33} + m_{55}) \left| \frac{m_{44}m_{46}}{m_{64}m_{66}} \right| \right\} \left| \frac{m_{11}m_{12}}{m_{21}m_{22}} \right| \\
 &\quad - \left\{ (m_{11} + m_{22}) \left| \frac{m_{44}m_{46}}{m_{64}m_{66}} \right| + m_{12}m_{26}m_{61} + m_{41} \left| \frac{m_{12}m_{14}}{m_{22}m_{24}} \right| - m_{14} \left| \frac{m_{41}m_{46}}{m_{61}m_{66}} \right| \right\} \left| \frac{m_{33}m_{35}}{m_{53}m_{55}} \right| \\
 B_6 &= \left| \frac{m_{11}m_{12}}{m_{21}m_{22}} \right| \left| \frac{m_{33}m_{35}}{m_{53}m_{55}} \right| \left| \frac{m_{44}m_{46}}{m_{64}m_{66}} \right| + \left| \frac{m_{12}m_{14}}{m_{22}m_{24}} \right| \left| \frac{m_{33}m_{35}}{m_{53}m_{55}} \right| \left| \frac{m_{41}m_{46}}{m_{61}m_{66}} \right| \\
 &\quad + \left| \frac{m_{31}m_{35}}{m_{51}m_{55}} \right| \left| \frac{m_{12}m_{13}}{m_{22}m_{23}} \right| \left| \frac{m_{44}m_{46}}{m_{64}m_{66}} \right| - m_{12}m_{26} \left| \frac{m_{41}m_{44}}{m_{61}m_{64}} \right| - m_{12}m_{25} \left| \frac{m_{31}m_{33}}{m_{51}m_{53}} \right| \left| \frac{m_{44}m_{46}}{m_{64}m_{66}} \right|.
 \end{aligned}$$

Where, $\left| \frac{m_1 m_2}{m_3 m_4} \right| = m_1 m_4 - m_2 m_3$

If all the roots of the characteristic equation $p(x)$ are negative or negative real parts if and only if it satisfies all the conditions given below:

- i). $B_j > 0, j = 1, \dots, 6$ (2.13a)
- ii). $H_2 = B_1B_2 - B_3 > 0$ (2.13b)
- iii). $H_3 = B_1B_2B_3 + B_1B_5 - B_3^2 - B_1^2B_4 > 0$ (2.13c)
- iv). $H_4 = (B_1B_2 - B_3)(B_3B_4 - B_2B_5) + (B_1B_2 - B_3)B_1B_6 - (B_1B_4 - B_5)^2 > 0$ (2.13d)
- v). $H_5 = B_3(B_1B_2 - B_3)(B_4B_5 - B_3B_6) + B_3B_5(B_2B_5 - B_1B_6) + B_1B_3B_6(B_1B_4 - 2B_5) - B_1(B_2B_5 - RB_1B_6)^2 - B_5(B_1B_4 - B_5)^2 > 0$ (2.13e)

If all the above conditions of the Routh-Hurwitz Criterion are satisfied for the jacobian matrix M_3 at fixed point E_3 then the system has existence of local stability.

3.3. Global Stability Analysis and Conditions

The solution, which emerges from varying distance from the fixed point, is being used to determine the system's long-term dynamical behaviour (La-Salle and Lefschetz 1961). It enables in the determination of the system's stability without integrating the differential equation explicitly. On the basis of the energy variation in the system, this method expresses the rate of the assumed system's stability. This reveals that, under certain conditions, the fixed E_3 is globally asymptotically stable, i.e., it moves the entire trajectory path towards the attractor basin's equilibrium point. The direct technique establishes a specific condition in which the dynamical system is supposed to be global stable and provides the region of attraction or basin of attractor.

Theorem 3.1:

We consider the following a continued and locally positive definite function,

$$V(N, N_d, T_1, T_2, U_1, U_2) = \left\{ N - N^* - N^* \log \frac{N}{N^*} \right\} + \frac{1}{2}(N_d - N_d^*)^2 + \frac{1}{2}(T_1 - T_1^*)^2 + \frac{1}{2}(U_1 - U_1^*)^2 + \frac{1}{2}(T_2 - T_2^*)^2 + \frac{1}{2}(U_2 - U_2^*)^2 \quad (2.14)$$

Proof:

Differentiating $V(N, N_d, T_1, T_2, U_1, U_2)$ with time t in the dynamical system, we get

$$\begin{aligned} \frac{dV}{dt} &= \frac{1}{N}(N - N^*) \frac{dN}{dt} + (N_d - N_d^*) \frac{dN_d}{dt} + (T_1 - T_1^*) \frac{dT_1}{dt} + (T_2 - T_2^*) \frac{dT_2}{dt} + (U_1 - U_1^*) \frac{dU_1}{dt} + (U_2 - U_2^*) \frac{dU_2}{dt} \\ \frac{dV}{dt} &= \frac{1}{N} \{ rN - (\alpha + b)N_d \} \left\{ 1 - \frac{N}{K(T_1, T_2)} \right\} (N - N^*) \\ &+ \left[(r_1U_1 + r_2U_2)(N - N_d) - (\alpha + d)N_d - \{ rN - (\alpha + b)N_d \} \frac{N_d}{K(T_1, T_2)} \right] (N_d - N_d^*) \\ &+ (\lambda N - \delta_1T_1 - \gamma_1NT_1 + \pi_1v_1NU_1)(T_1 - T_1^*) \\ &+ (Q - \delta_2T_2 - \gamma_2NT_2 + \pi_1v_1NU_1)(T_2 - T_2^*) \\ &+ (\gamma_1NT_1 - \beta_1U_1 - v_1NU_1)(U_1 - U_1^*) \\ &+ (\gamma_2NT_2 - \beta_2U_2 - v_2NU_2)(U_2 - U_2^*) \\ \frac{dV}{dt} &= -\frac{r}{K(T_1^*, T_2^*)}(N - N^*)^2 - \left\{ (r_1U_1^* + r_2U_2^*) + \frac{rN^*}{K(T_1^*, T_2^*)} + (\alpha + d) - \frac{(\alpha + b)(N_d + N_d^*)}{K(T_1^*, T_2^*)} \right\} \\ &(N_d - N_d^*)^2 - (\delta_1 + \gamma_1N^*)(T_1 - T_1^*)^2 - (\delta_2 + \gamma_2N^*)(T_2 - T_2^*)^2 - (\beta_1 + \gamma_1N^*)(U_1 - U_1^*)^2 \\ &- (\beta_2 + \gamma_2N^*)(U_2 - U_2^*)^2 + \left\{ -(\alpha + b) + \frac{(\alpha + b)}{K(T_1^*, T_2^*)} + (r_1U_1^* + r_2U_2^*) - \frac{rN_d^*}{K(T_1^*, T_2^*)} \right\} (N - N^*)(N_d - N_d^*) \\ &+ [\lambda + \{ (\alpha + b)N_d - rN \} \eta_1(T_1, T_2) - \gamma_1T_1 + \pi_1v_1U_1] (N - N^*)(T_1 - T_1^*) \\ &+ [\{ (\alpha + b)N_d - rN \} \eta_2(T_1^*, T_2) - \gamma_2T_2 + \pi_2v_2U_2] (N - N^*)(T_2 - T_2^*) \\ &+ (\gamma_1T_1 - v_1U_1)(N - N^*)(U_1 - U_1^*) + (\gamma_2T_2 - v_2U_2)(N - N^*)(U_2 - U_2^*) \\ &+ [(\alpha + b)N_d^2 - rN^*N_d] \eta_1(T_1, T_2)(N_d - N_d^*)(T_1 - T_1^*) \\ &+ [(\alpha + b)N_d^2 - rN^*N_d] \eta_2(T_1^*, T_2)(N_d - N_d^*)(T_2 - T_2^*) \\ &+ r_1(N - N_d)(N_d - N_d^*)(U_1 - U_1^*) + r_2(N - N_d)(N_d - N_d^*)(U_2 - U_2^*) \\ &+ (\gamma_1 + \pi_1v_1)N^*(T_1 - T_1^*) \\ &+ (\gamma_2 + \pi_2v_2)N^*(T_2 - T_2^*)(U_2 - U_2^*) \end{aligned}$$

$$\eta_1(T_1, T_2) = \begin{cases} \frac{1}{\frac{K(T_1, T_2)}{T_1 - T_1^*} - \frac{1}{K(T_1^*, T_2)}}, & T_1 \neq T_1^* \\ \frac{-1}{K^2(T_1^*, T_2)} \frac{\partial k(T_1^*, T_2)}{\partial T_1}, & T_1 = T_1^* \end{cases} \quad \&$$

$$\eta_2(T_1^*, T_2) = \begin{cases} \frac{1}{\frac{K(T_1^*, T_2)}{T_2 - T_2^*} - \frac{1}{K(T_1^*, T_2^*)}}, & T_2 \neq T_2^* \\ \frac{-1}{K^2(T_1^*, T_2^*)} \frac{\partial k(T_1^*, T_2^*)}{\partial T_2}, & T_2 = T_2^* \end{cases}$$

By applying mean value theorem, the inequalities satisfy the following equations:

$$[\eta_1(T_1, T_2)] \leq \frac{k_1}{K_m^2} \quad \& \quad [\eta_2(T_1^*, T_2)] \leq \frac{k_2}{K_m^2}$$

Now the derivative of $V((N, N_d, T_1, T_2, U_1, U_2), t)$ can be written in term of sum of quadratic, which are as given below:-

$$\begin{aligned} \frac{dV}{dt} = & -\frac{1}{2}a_{11}(N - N^*)^2 + a_{12}(N - N^*)(N_d - N_d^*) - \frac{1}{2}a_{22}(N_d - N_d^*)^2 \\ & -\frac{1}{2}a_{11}(N - N^*)^2 + a_{13}(N - N^*)(T_1 - T_1^*) - \frac{1}{2}a_{33}(T_1 - T_1^*)^2 \\ & -\frac{1}{2}a_{11}(N - N^*)^2 + a_{14}(N - N^*)(T_2 - T_2^*) - \frac{1}{2}a_{44}(T_2 - T_2^*)^2 \\ & -\frac{1}{2}a_{11}(N - N^*)^2 + a_{15}(N - N^*)(U_1 - U_1^*) - \frac{1}{2}a_{55}(U_1 - U_1^*)^2 \\ & -\frac{1}{2}a_{11}(N - N^*)^2 + a_{16}(N - N^*)(U_2 - U_2^*) - \frac{1}{2}a_{66}(U_2 - U_2^*)^2 \\ & -\frac{1}{2}a_{22}(N_d - N_d^*)^2 + a_{23}(N_d - N_d^*)(T_1 - T_1^*) - \frac{1}{2}a_{33}(T_1 - T_1^*)^2 \\ & -\frac{1}{2}a_{22}(N_d - N_d^*)^2 + a_{24}(N_d - N_d^*)(T_2 - T_2^*) - \frac{1}{2}a_{44}(T_2 - T_2^*)^2 \\ & -\frac{1}{2}a_{22}(N_d - N_d^*)^2 + a_{25}(N_d - N_d^*)(U_1 - U_1^*) - \frac{1}{2}a_{55}(U_1 - U_1^*)^2 \\ & -\frac{1}{2}a_{22}(N_d - N_d^*)^2 + a_{26}(N_d - N_d^*)(U_2 - U_2^*) - \frac{1}{2}a_{66}(U_2 - U_2^*)^2 \\ & -\frac{1}{2}a_{33}(T_1 - T_1^*)^2 + a_{35}(T_1 - T_1^*)(U_1 - U_1^*) - \frac{1}{2}a_{55}(U_1 - U_1^*)^2 \\ & -\frac{1}{2}a_{44}(T_2 - T_2^*)^2 + a_{46}(T_2 - T_2^*)(U_2 - U_2^*) - \frac{1}{2}a_{66}(U_2 - U_2^*)^2 \end{aligned}$$

Where,

$$a_{11} = \frac{2}{5} \frac{r}{K(T_1^*, T_2^*)}, a_{22} = \frac{2}{5} \left\{ (r_1 U_1^* + r_2 U_2^*) + \frac{r N^*}{K(T_1^*, T_2^*)} + (\alpha + d) - \frac{(\alpha + b)(N_d + N_d^*)}{K(T_1^*, T_2^*)} \right\}$$

$$a_{33} = \frac{2}{3} (\delta_1 + \gamma_1 N^*), a_{44} = \frac{2}{3} (\delta_2 + \gamma_2 N^*), a_{55} = \frac{2}{3} (\beta_1 + v_1 N^*), a_{66} = \frac{2}{3} (\beta_2 + v_2 N^*)$$

$$a_{12} = \left\{ -(\alpha + b) + \frac{(\alpha + b)}{K(T_1^*, T_2^*)} + (r_1 U_1^* + r_2 U_2^*) - \frac{r N_d^*}{K(T_1^*, T_2^*)} \right\},$$

$$a_{13} = [\lambda + \{(\alpha + b)N_d - rN\}\eta_1(T_1, T_2) - \gamma_1 T_1 + \pi_1 v_1 U_1],$$

$$a_{14} = [(\alpha + b)N_d - rN\}\eta_2(T_1^*, T_2) - \gamma_2 T_2 + \pi_2 v_2 U_2], a_{15} = (\gamma_1 T_1 - v_1 U_1), a_{16} = (\gamma_2 T_2 - v_2 U_2)$$

$$a_{23} = [(\alpha + b)N_d^2 - rN^*N_d]\eta_1(T_1, T_2), a_{24} = [(\alpha + b)N_d^2 - rN^*N_d]\eta_2(T_1^*, T_2)$$

$$a_{25} = r_1(N - N_d), a_{26} = r_2(N - N_d), a_{35} = (\gamma_1 + \pi_1 v_1)N^*, a_{46} = (\gamma_2 + \pi_2 v_2)N^*$$

Thus, $\frac{dV}{dt}$ will be negative definite provided

$$a_{12}^2 < a_{11}a_{22}, a_{13}^2 < a_{11}a_{33}, a_{14}^2 < a_{11}a_{44}, a_{15}^2 < a_{11}a_{55}, a_{16}^2 < a_{11}a_{66}, a_{23}^2 < a_{22}a_{33}, a_{24}^2 < a_{22}a_{44},$$

$$a_{25}^2 < a_{22}a_{55}, a_{26}^2 < a_{22}a_{66}, a_{35}^2 < a_{33}a_{55}, a_{46}^2 < a_{44}a_{66}$$

The $V\{(N, N_d, T_1, T_2, U_1, U_2), t\}$ is a locally positive definite function as well as decrescent. Since this function is strictly increasing and continuous in the region of attraction i.e. all the trajectory path of the variables is found to be moving continuously in the basin of attraction. Also the above is true as time tends to infinity.

Since $\dot{V}((N, N_d, T_1, T_2, U_1, U_2), t)$ is negative. Therefore $V\{(N, N_d, T_1, T_2, U_1, U_2), t\}$ is a positive definite function for above conditions.

Theorem 3.2:

In addition to the assumptions, let $K(T)$ satisfy the following inequalities in the region of attraction Ω :

$$K_m \leq K(T_1, T_2) \leq K_0, 0 \leq -\frac{\partial K(T_1, T_2)}{\partial T_1} \leq \kappa_1, 0 \leq -\frac{\partial K(T_1, T_2)}{\partial T_2} \leq \kappa_2$$

Here, K_m, K_0, κ_1 & κ_2 are positive constants.

If all the inequalities are held by the proposed system (2), then it is globally stable at fixed point E_3 under the condition given below

$$\begin{aligned} & \left[r_1 U_1^* + r_2 U_2^* - (\alpha + b) \left\{ 1 - \frac{1}{K(T_1^*, T_2^*)} \right\} - \frac{r N_d^*}{K(T_1^*, T_2^*)} \right]^2 \\ & < \frac{4}{25} \frac{r}{K(T_1^*, T_2^*)} \left[r_1 U_1^* + r_2 U_2^* + (\alpha + d) + \frac{r N^*}{K(T_1^*, T_2^*)} - \frac{\alpha + b}{K(T_1^*, T_2^*)} \left\{ \frac{K_0(r_1 + r_2)(\lambda K_0 + Q)}{(r_1 + r_2)(\lambda K_0 + Q) + (\alpha + d)\delta_m} + N_d^* \right\} \right] \end{aligned} \tag{2.15a}$$

$$\left[\lambda + (\pi_1 v_1 - \gamma_1) \frac{(\lambda K_0 + Q)}{\delta_m} + \left\{ (\alpha + b) \frac{K_0(r_1 + r_2)(\lambda K_0 + Q)}{[(r_1 + r_2)(\lambda K_0 + Q) + (\alpha + d)\delta_m]} - r K_0 \right\} \frac{\kappa_1}{K_m^2} \right]^2 < \frac{4}{15} \frac{r}{K(T_1^*, T_2^*)} (\delta_1 + \gamma_1 N^*) \tag{2.15b}$$

$$\left[(\pi_1 v_1 - \gamma_1) \frac{(\lambda K_0 + Q)}{\delta_m} + \left\{ (\alpha + b) \frac{K_0(r_1 + r_2)(\lambda K_0 + Q)}{[(r_1 + r_2)(\lambda K_0 + Q) + (\alpha + d)\delta_m]} - r K_0 \right\} \frac{\kappa_2}{K_m^2} \right]^2 < \frac{4}{15} \frac{r}{K(T_1^*, T_2^*)} (\delta_2 + \gamma_2 N^*) \tag{2.15c}$$

$$\left[(\gamma_1 - v_1) \frac{(\lambda K_0 + Q)}{\delta_m} \right]^2 < \frac{4}{15} \frac{r}{K(T_1^*, T_2^*)} (\beta_1 + v_1 N^*) \tag{2.15d}$$

$$\left[(\gamma_2 - v_2) \frac{(\lambda K_0 + Q)}{\delta_m} \right]^2 < \frac{4}{15} \frac{r}{K(T_1^*, T_2^*)} (\beta_2 + v_2 N^*) \tag{2.15e}$$

$$\begin{aligned} & \left[\left((\alpha + b) \left\{ \frac{K_0(r_1 + r_2)(\lambda K_0 + Q)}{[(r_1 + r_2)(\lambda K_0 + Q) + (\alpha + d)\delta_m]} \right\} - \frac{r N^* K_0(r_1 + r_2)(\lambda K_0 + Q)}{[(r_1 + r_2)(\lambda K_0 + Q) + (\alpha + d)\delta_m]} \right) \frac{\kappa_1}{K_m^2} \right]^2 \\ & < \frac{4}{15} (\delta_1 + \gamma_1 N^*) \left[r_1 U_1^* + r_2 U_2^* + (\alpha + d) + \frac{r N^*}{K(T_1^*, T_2^*)} - \frac{\alpha + b}{K(T_1^*, T_2^*)} \left\{ \frac{K_0(r_1 + r_2)(\lambda K_0 + Q)}{(r_1 + r_2)(\lambda K_0 + Q) + (\alpha + d)\delta_m} + N_d^* \right\} \right] \end{aligned} \tag{2.15f}$$

$$\begin{aligned} & \left[\left((\alpha + b) \left\{ \frac{K_0(r_1 + r_2)(\lambda K_0 + Q)}{[(r_1 + r_2)(\lambda K_0 + Q) + (\alpha + d)\delta_m]} \right\} - \frac{r N^* K_0(r_1 + r_2)(\lambda K_0 + Q)}{[(r_1 + r_2)(\lambda K_0 + Q) + (\alpha + d)\delta_m]} \right) \frac{\kappa_2}{K_m^2} \right]^2 \\ & < \frac{4}{15} (\delta_2 + \gamma_2 N^*) \left[r_1 U_1^* + r_2 U_2^* + (\alpha + d) + \frac{r N^*}{K(T_1^*, T_2^*)} - \frac{\alpha + b}{K(T_1^*, T_2^*)} \left\{ \frac{K_0(r_1 + r_2)(\lambda K_0 + Q)}{(r_1 + r_2)(\lambda K_0 + Q) + (\alpha + d)\delta_m} + N_d^* \right\} \right] \end{aligned} \tag{2.15g}$$

$$\begin{aligned} & \left[r_1 \left(K_0 - \frac{r N^* K_0(r_1 + r_2)(\lambda K_0 + Q)}{[(r_1 + r_2)(\lambda K_0 + Q) + (\alpha + d)\delta_m]} \right) \right]^2 \\ & < \frac{4}{15} (\beta_1 + v_1 N^*) \left[r_1 U_1^* + r_2 U_2^* + (\alpha + d) + \frac{r N^*}{K(T_1^*, T_2^*)} - \frac{\alpha + b}{K(T_1^*, T_2^*)} \left\{ \frac{K_0(r_1 + r_2)(\lambda K_0 + Q)}{(r_1 + r_2)(\lambda K_0 + Q) + (\alpha + d)\delta_m} + N_d^* \right\} \right] \end{aligned} \tag{2.15h}$$

$$\begin{aligned} & \left[r_2 \left(K_0 - \frac{r N^* K_0(r_1 + r_2)(\lambda K_0 + Q)}{[(r_1 + r_2)(\lambda K_0 + Q) + (\alpha + d)\delta_m]} \right) \right]^2 \\ & < \frac{4}{15} (\beta_2 + v_2 N^*) \left[r_1 U_1^* + r_2 U_2^* + (\alpha + d) + \frac{r N^*}{K(T_1^*, T_2^*)} - \frac{\alpha + b}{K(T_1^*, T_2^*)} \left\{ \frac{K_0(r_1 + r_2)(\lambda K_0 + Q)}{(r_1 + r_2)(\lambda K_0 + Q) + (\alpha + d)\delta_m} + N_d^* \right\} \right] \end{aligned} \tag{2.15i}$$

$$[(\gamma_1 + \pi_1 v_1) N^*]^2 < \frac{4}{9} (\delta_1 + \gamma_1 N^*) (\beta_1 + v_1 N^*) \tag{2.15j}$$

$$[(\gamma_2 + \pi_2 v_2) N^*]^2 < \frac{4}{9} (\delta_2 + \gamma_2 N^*) (\beta_2 + v_2 N^*) \tag{2.15k}$$

If all the above inequalities are held then the assumed model (2.2) is global asymptotically stable or the system would be remaining stable if the system disturbed for large range at E_3 . Here, eq. (14) \Rightarrow (2.15a – 2.15k). Hence V is a continuous and increasing function, whose domain is Ω under the conditions (2.15a – 2.15k). So, E_3 is uniformly asymptotically globally stable in Ω under the conditions (2.15a – 2.15k) hold.

4. Numerical Simulation

Understanding and predicting the complicated behaviour of dynamical systems necessitate the use of mathematical models. This model is useful for tasks including forecasting, characterizing system reactions, and optimizing system solutions. Now, in order to maximize the solution of the suggested model's dynamical system, the proprietary programming language MATLAB is utilized to provide various types of output. Simulations were carried out utilizing a computer and the MATCONT and MATLAB software. We now assume the function's carrying capacity, which is as follows:

$$K(T_1, T_2) = K_0 - \frac{b_{11}T_1}{1+b_{12}T_1} - \frac{b_{21}T_2}{1+b_{22}T_2} \quad (2.16)$$

Choosing the value of

$$K_0 = 10.0, \quad b_{11} = 0.2, \quad b_{12} = 1.0, \\ b_{21} = 0.1, \quad b_{22} = 2.0,$$

The value of K_m is chosen in such a way that $K(T) \leq K_m \leq K_0$ and a set of the parameters are taken such that all are positive constant.

$$b = .044, d = .01, r_1 = .035, r_2 = 0.032, \lambda = 0.001, \alpha \\ = .0008, \delta_1 = .0029, \delta_2 = 0.09, \pi_2 \\ = .05, \beta_1 = .03,$$

$$\pi_2 = .05, Q = 0.005, \beta_2 = 0.006, K_0 = 10.0, \\ \gamma_1 = .000045, \gamma_2 = 0.001, \\ v_1 = .0001, v_2 = .0001, \pi_1 = .005 \quad (2.17)$$

Table 1 shows the variance of total population and deformed reproductive population (t) for the proposed system, which increases the probability of extinction of various species from the ecosystem. The total population decreases while the deformed reproductive population increases as the reproductive toxicant increases into the environment. Fig.1 shows the simultaneous reaction of two reproductive toxicants on the total population vs. time for given values of Q and λ , keeping all other parameters constant. The trajectory direction changes continually with the changes in the effect of reproductive toxicant. It moves away from the fixed point as the simultaneous reaction increases at the targeted site of the reproductive organ in biological species. Initially, the amount of reproductive toxicants emitted into the environment is less, so their simultaneous effect on reproduction process is relatively low tending less decreasing in the total population. As the simultaneous effect increases, so do the abnormalities in the reproductive systems and the infertility increases. Fig.2 shows the variation of the deformed reproductive population with time lag for the changing values of Q & λ and keeping other parameters constant.

Fig. 3 is a phase diagram between N and N_d , that illustrates how the total population density and the deformed reproductive population change over time. In the

phase diagram, the rate of the reproductive toxicant into the ecosystem increases which increases the deformed reproduction population. As a result, both populations reach their peak and then begin to decline to their minimum value whereas if the toxicant rate is extremely high, both population decrease. It clearly shows that as the simultaneous effect of reproductive toxicants increases at the targeted sites of the reproduction system of a species, a critical state develops in which both chemical reactivity and structural alterations occur severely in the reproductive area of the species. As a result, the chemical reactivity or structural alterations at the targeted organ are substantially increases, usually in the form of synergistic, antagonistic form, resulting in increased infertility and death rate in biological species. If the population density decreases, the environmental pollution also decreases, and it helps the biological species to regain their original population. Fig.4 (a) shows that if the value of β_1 & β_2 changes (increase or decrease), then the equilibrium point in the region of attraction remains the same whatever be the value of λ & Q . Therefore there is no effect and the total population remains the same for the increasing value of β_1 & β_2 . Fig. 4(b) shows that if the value of β_1 & β_2 at $\lambda = .3$ & $Q = .3$ increases by keeping the other parameters constant, infertility rate decreases. It shows that as natural depletion rate coefficient of U_1 & U_2 increases, the deformities in reproduction decrease. Fig. 4(c) shows that if the value of the natural depletion rate coefficient of T_1 & T_2 increases at $\lambda = .1$ & $Q = .1$, its uptake by the biological species decreases. Fig. 4(d) shows that if the value of the natural depletion rate coefficient of T_1 & T_2 decreases ultimately, its uptake by the biological species increases, it increases the rate of the deformed reproduction density at $\lambda = .045$ & $Q = .047$. If the washout rate of the reproductive toxicants by some natural processes decreases, the concentration of the toxicants in the environment increases, and it increases the rate of deformed reproduction population. Fig. 5(a) shows the variation of deformed reproduction population for the changing of value of α at $\lambda = .003$ & $Q = .01$. If the concentration of the reproductive toxicants increases, its uptake doses by the species also increase and it increases the mortality rate of the deformed reproductive population. Fig.5 (b) shows that as the mortality rate increases, the deformed reproductive population decreases due to the increased number of death of the deformed density. The multiple plots on MATLAB appear to suggest that as the rate of reproductive toxicants increases, then their simultaneous reaction at the targeted reproductive part increases. The loss of reproduction capacity increases and the total population decreases below the carrying capacity, increasing the chance of extinction from the community. Surface plots illustrate the combined effect of three variables, which is considerable and has an adverse impact on the assumed system, which consists of one dependent variable and two independent variables. Out of these three, two are independent, while the third is dependent and is represented in matrix or vector form, with the new surface colour function represented by a new matrix notation. The colour data is displayed on the Z-axis,

which is also known as the surface height; thus, colour and surface height are proportionate. To visualize mathematical structure over a rectangular region, we use a surface with a contour in this graphic. The surface with contour develops coloured parametric surfaces based on the total population, deformed density, and two reproductive toxicants, with the colour determined by the rectangular grid's height. Here, three cases have been discussed (i) the reproductive toxicant is constant i.e. $\lambda = .01$ and the value of Q is increased continuously, (ii) the

reproductive toxicant by the external sources is constant i.e. $Q = .1$ and λ increases continuously in the environment and (iii) both the reproductive toxicants increases together parallel and continuously. Here, all three cases have been plotted using MATLAB. Fig.6 & Fig.7 are related to the first case in which reprotoxin by the external sources is increasing continuously and reprotoxin by the species is constant. Fig.6 & Fig.7 are surfaces plot of model (2.2) in which the total population, deformed density, and Q are the three variables.

Table.1. $N_1, N_2, N_d, T_1, T_2, U_1$ & U_2 for different value of Q & λ

N	N_d	T_1	T_2	U_1	U_2	Q	λ
9.8198	1.6910	1.3418	0.1002	0.1914	0.0318	.01	.001
9.7411	4.9799	4.0126	0.3009	0.5679	0.0946	.03	.003
5.8023	4.4035	10.5290	0.4176	0.8990	0.0792	.04	.01
3.9137	2.9695	16.7659	0.0213	0.9708	0.0027	.002	.02
2.3353	1.4814	27.2146	0.2169	0.8384	0.0149	.02	.05
2.2482	1.7482	24.5608	1.0849	0.8548	0.0832	.1	.04
1.4258	1.0890	23.9923	10.9388	0.5114	0.5187	1	.06
0.8001	0.7287	43.4226	0.2201	0.7028	0.0076	.02	.1
0.0831	0.0520	118.0158	0.7771	0.1780	0.0024	.04	1

Figure 1. Variation of Total Population w.r.t Time(t)

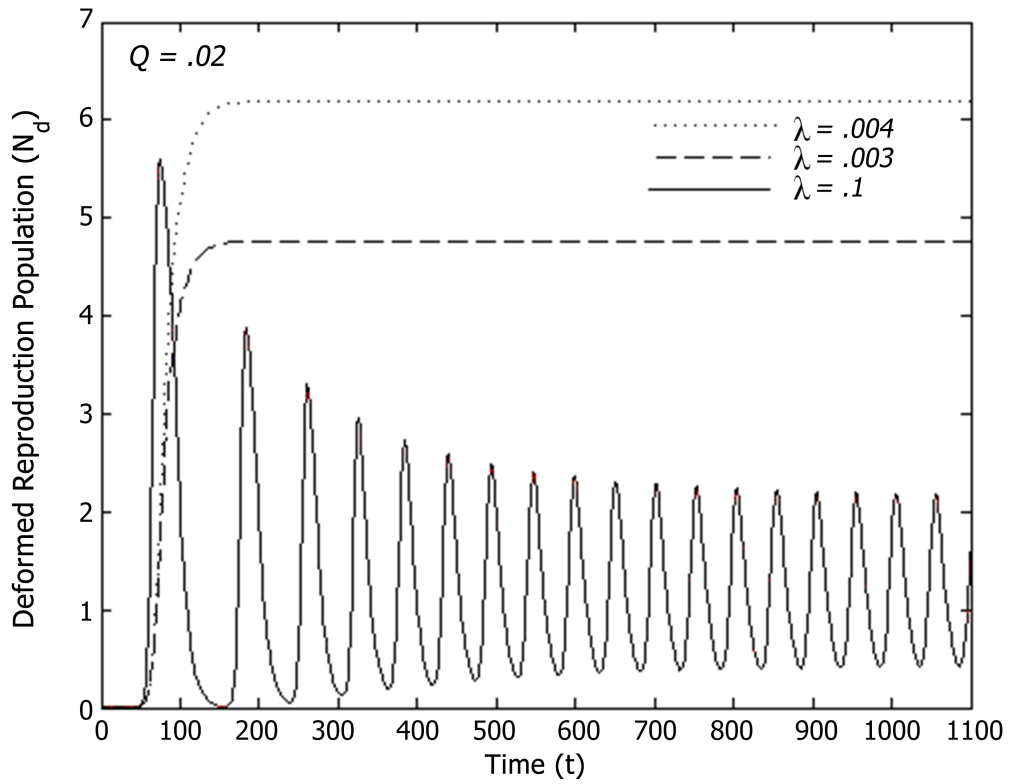


Figure 2. Variation of Deformed Reproduction population w.r.t Time (t)

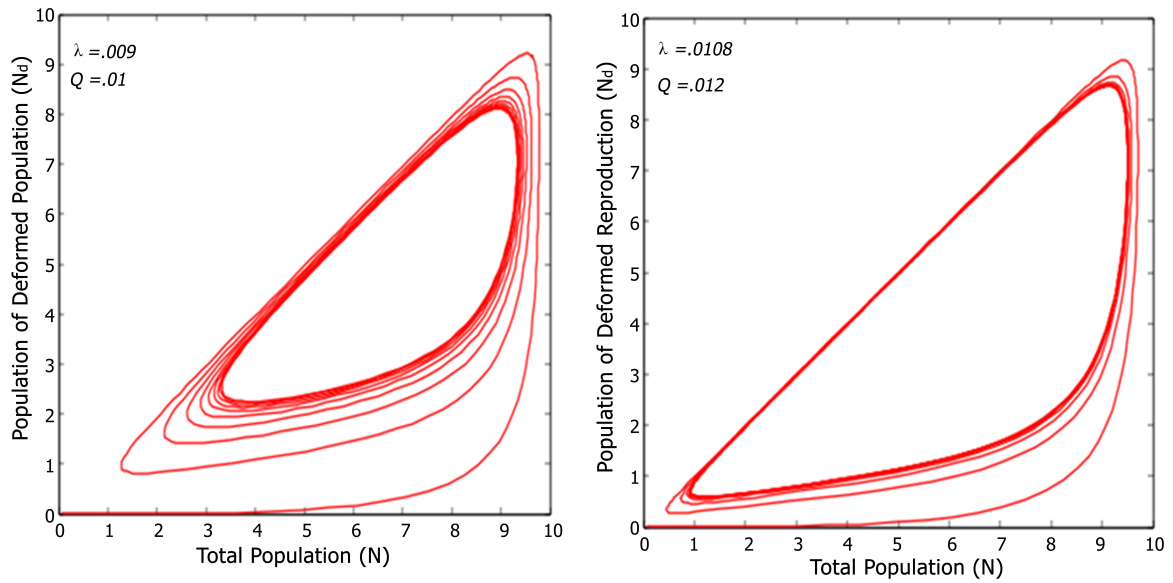


Figure 3. Phase Diagram “Variation of Total Population versus deformed Population with respect to Time (t)

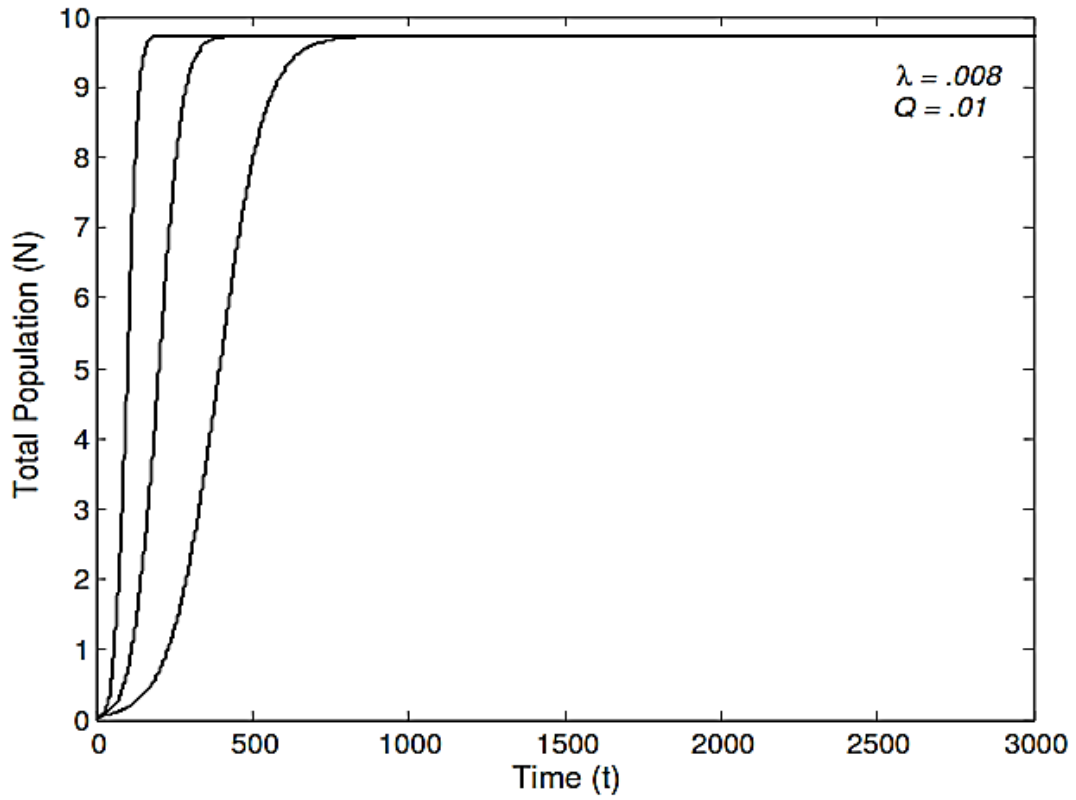


Figure 4(a). Total Population versus time(t) for changed value of β_1 & β_2

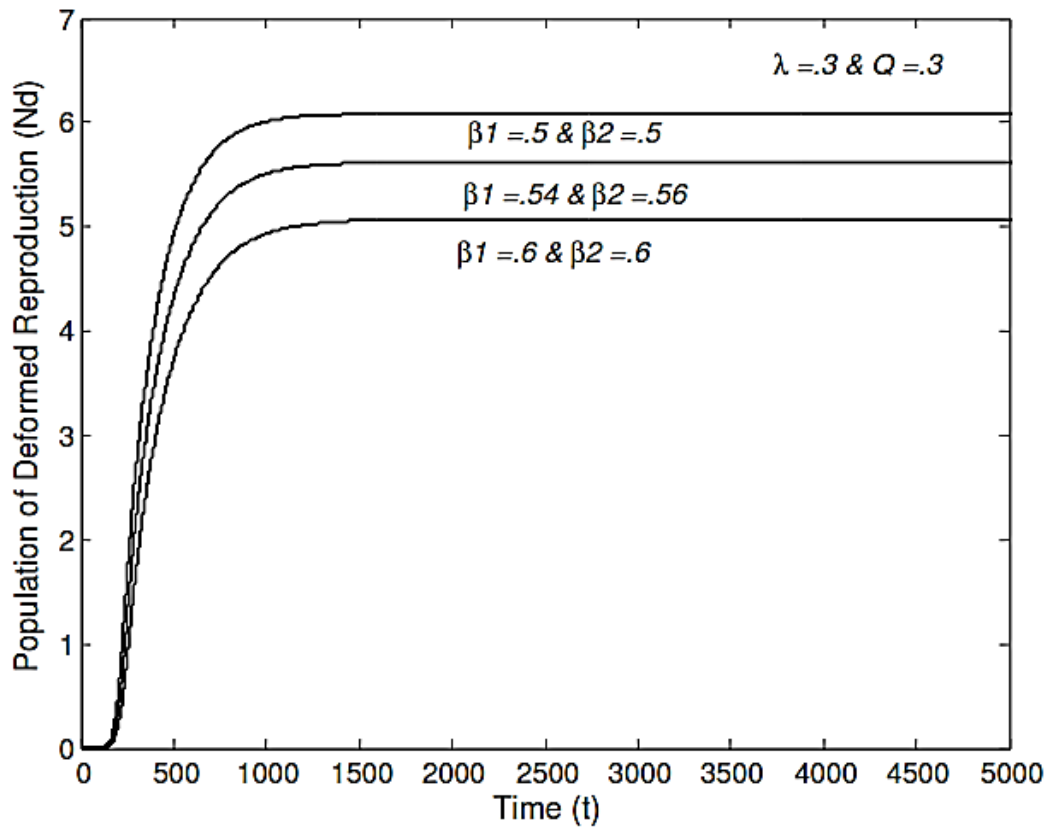


Figure 4(b). Deformed Reproduction Population versus time(t) for the changed value of β_1 & β_2

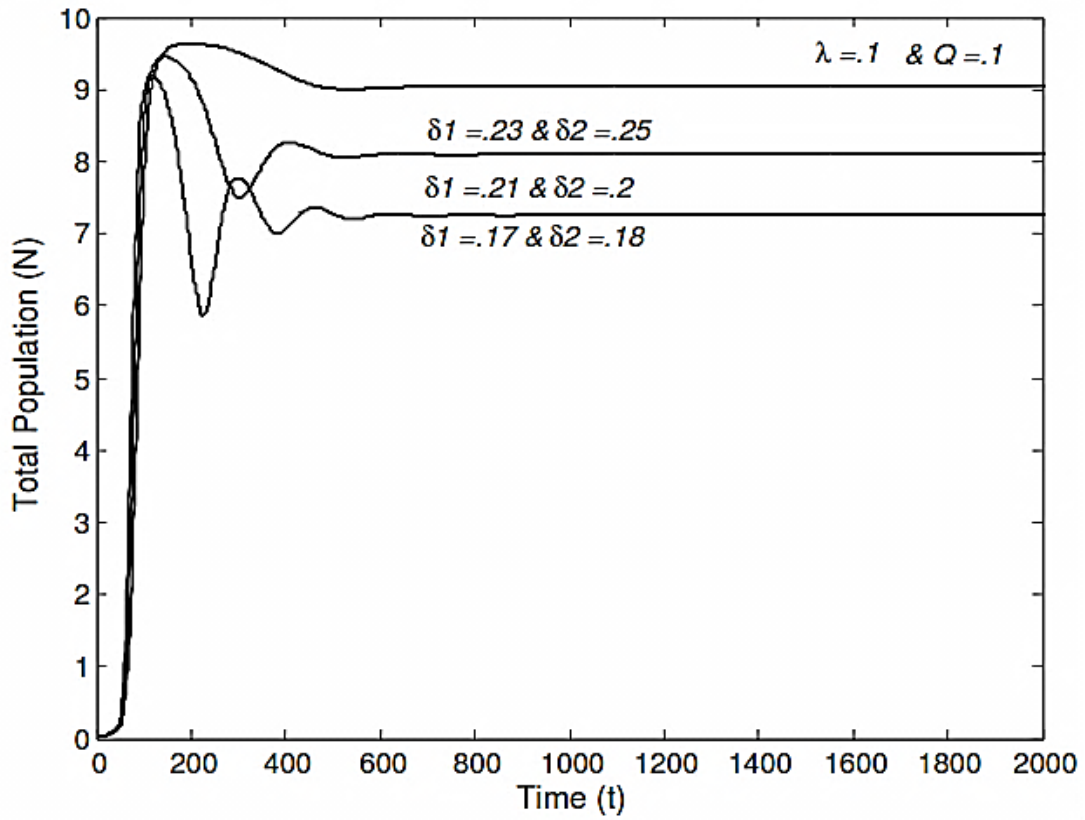


Figure 4(c). Total population versus time (t) versus for changed value of δ_1 & δ_2

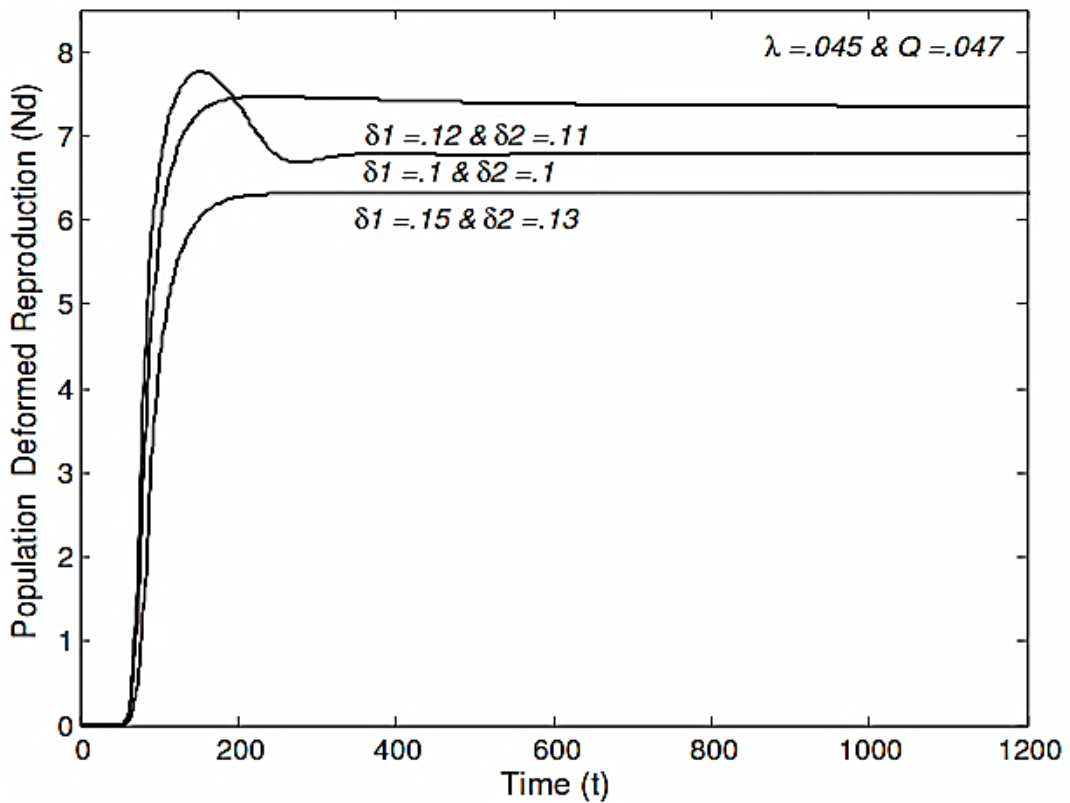


Figure 4(d). Deformed reproduction population versus time (t) for the changed value of δ_1 & δ_2

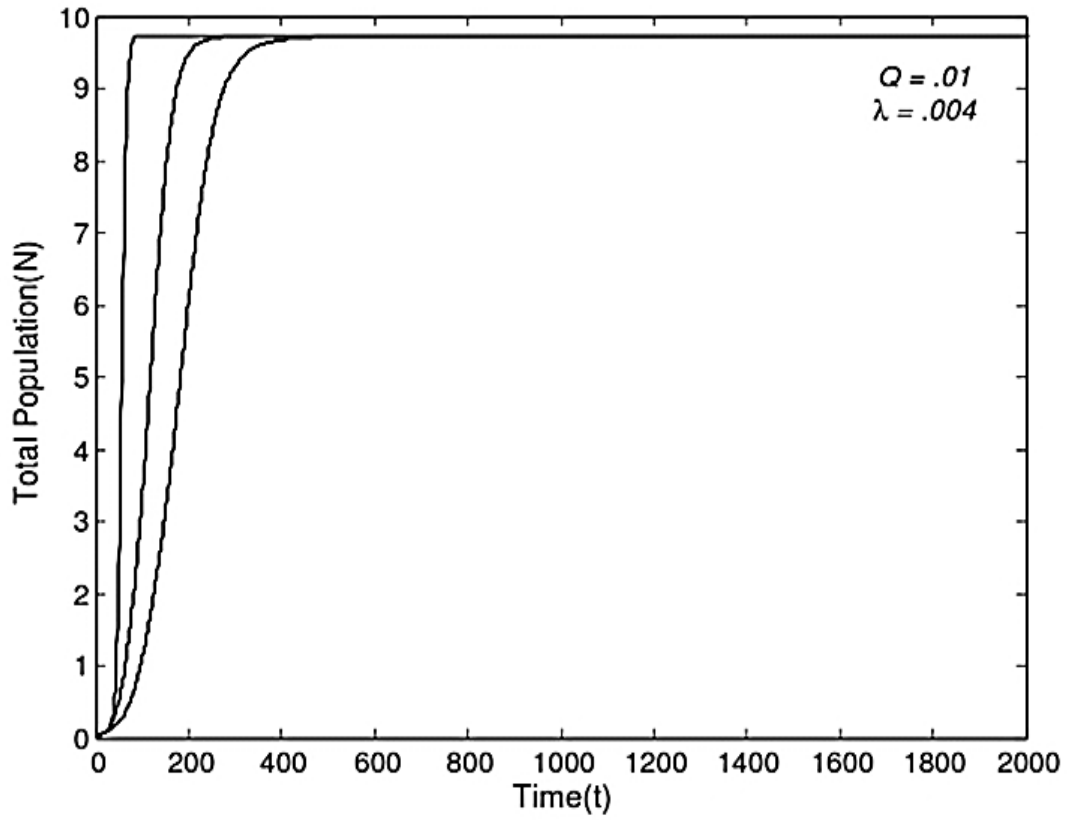


Figure 5(a). Total Population versus Time(t) for changed value of α

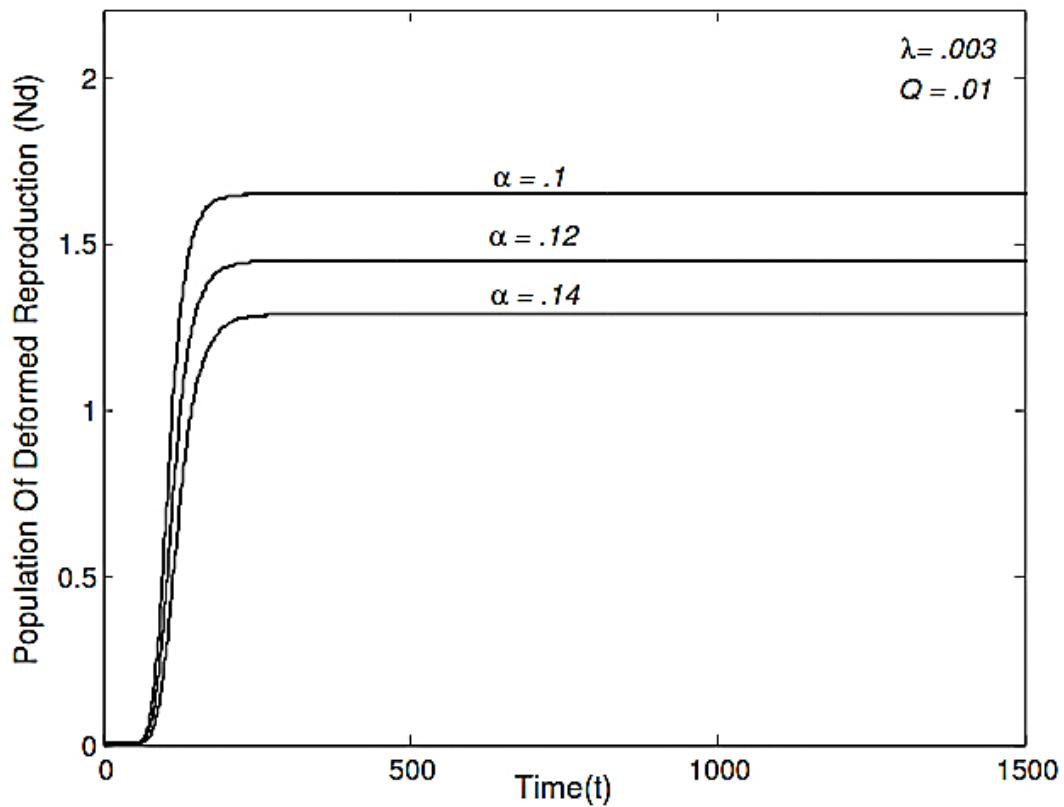


Figure 5(b). Deformed Reproduction versus Time (t) for changed value of α

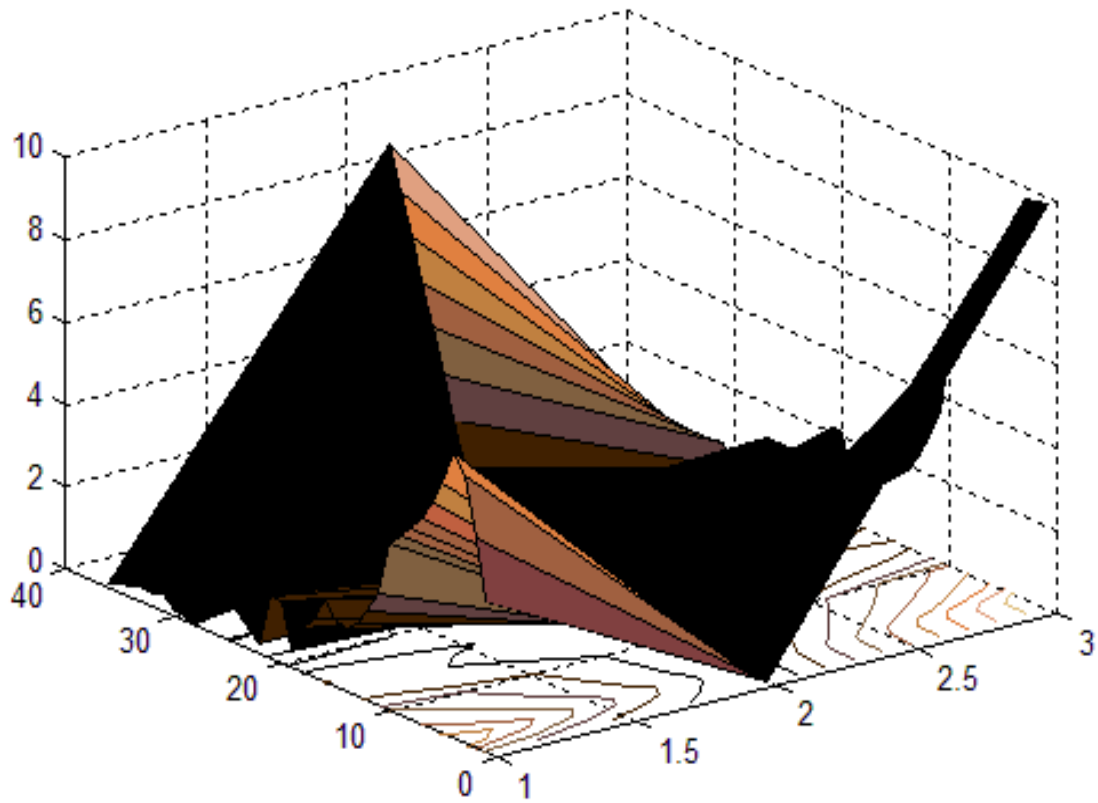


Figure 6. 3D Surface Plot with Contour of Dynamical Population, Reprotoxin(external)

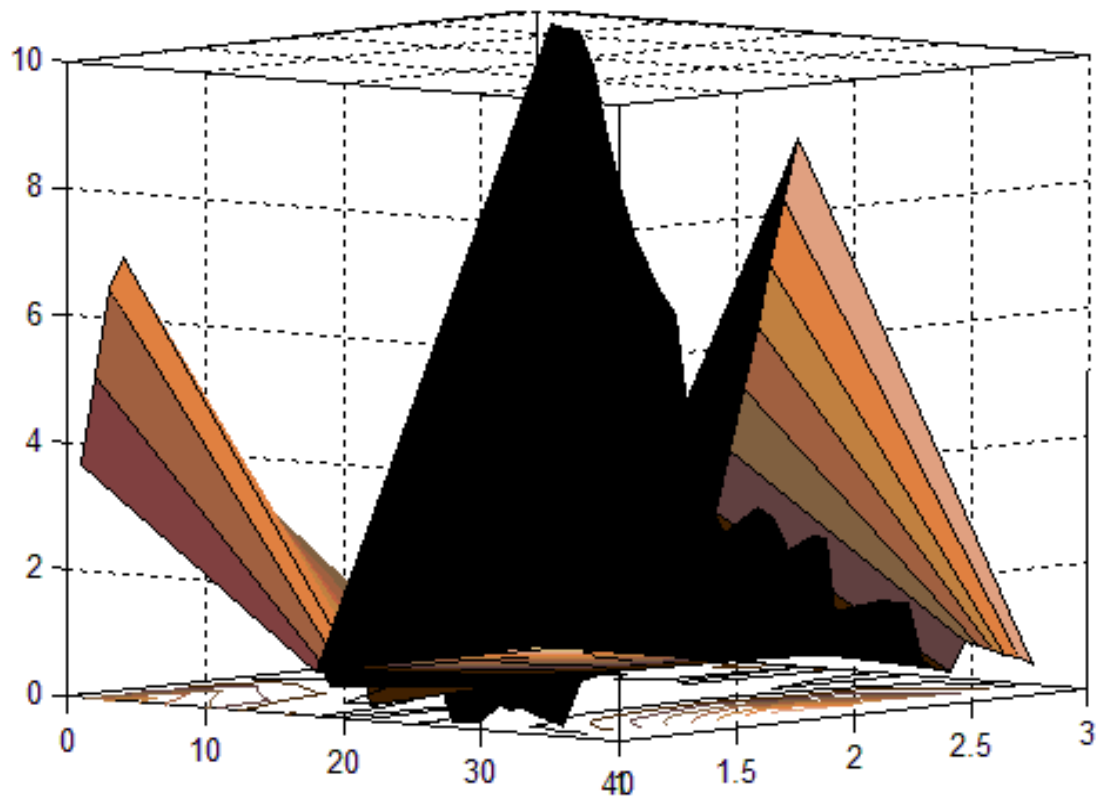


Figure 7. Rotation of 3D Surface Plot with contour of the dynamical system

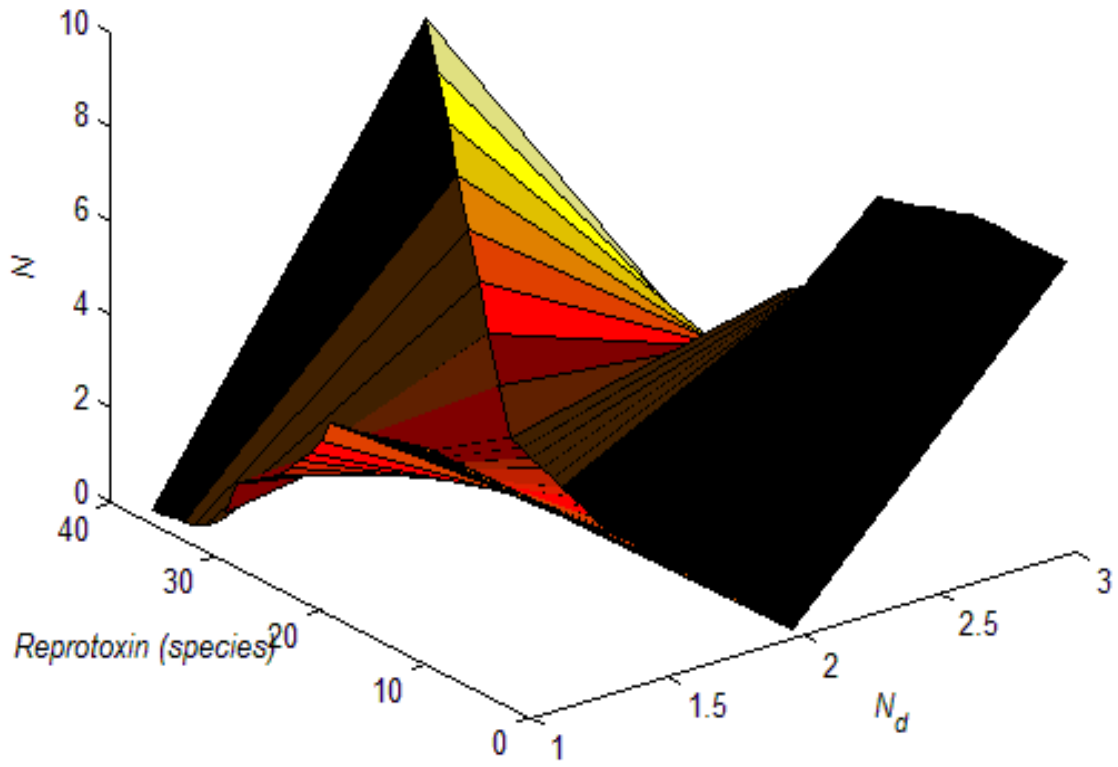


Figure 8. 3D Surface Plot with Contour of Dynamical System, Reprotoxin (species)

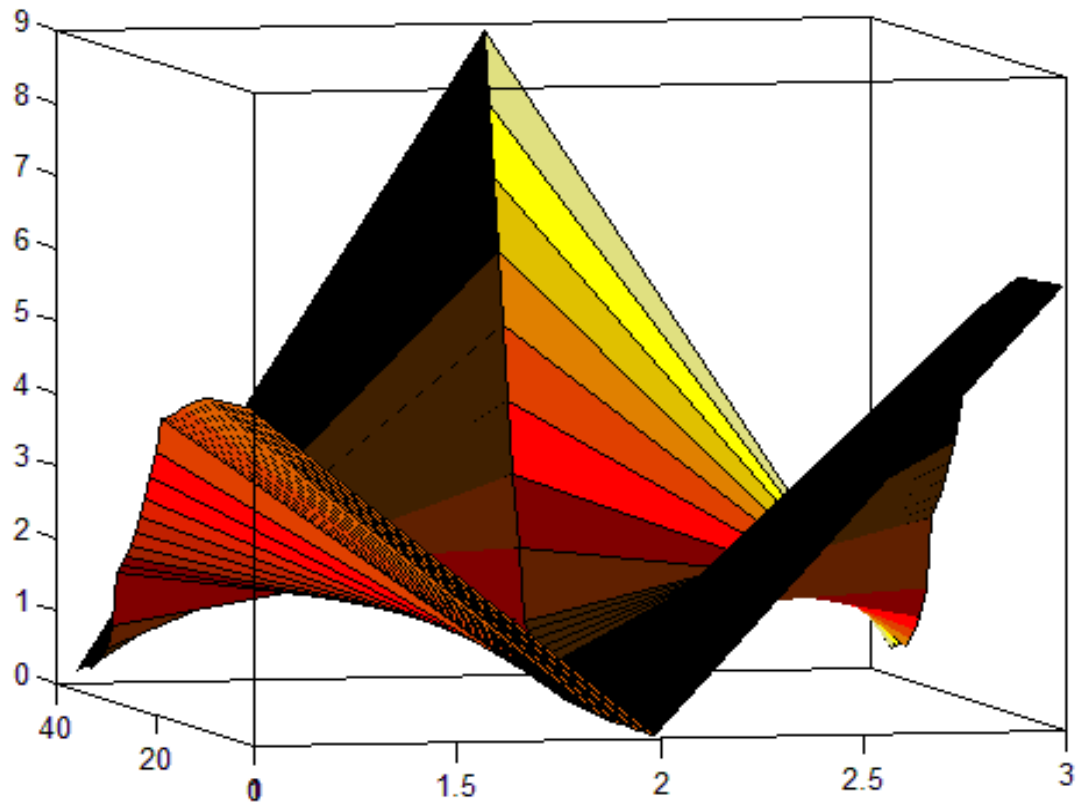


Figure 9. Rotation of Surface Plot of Dynamical System, Reprotoxin (species)

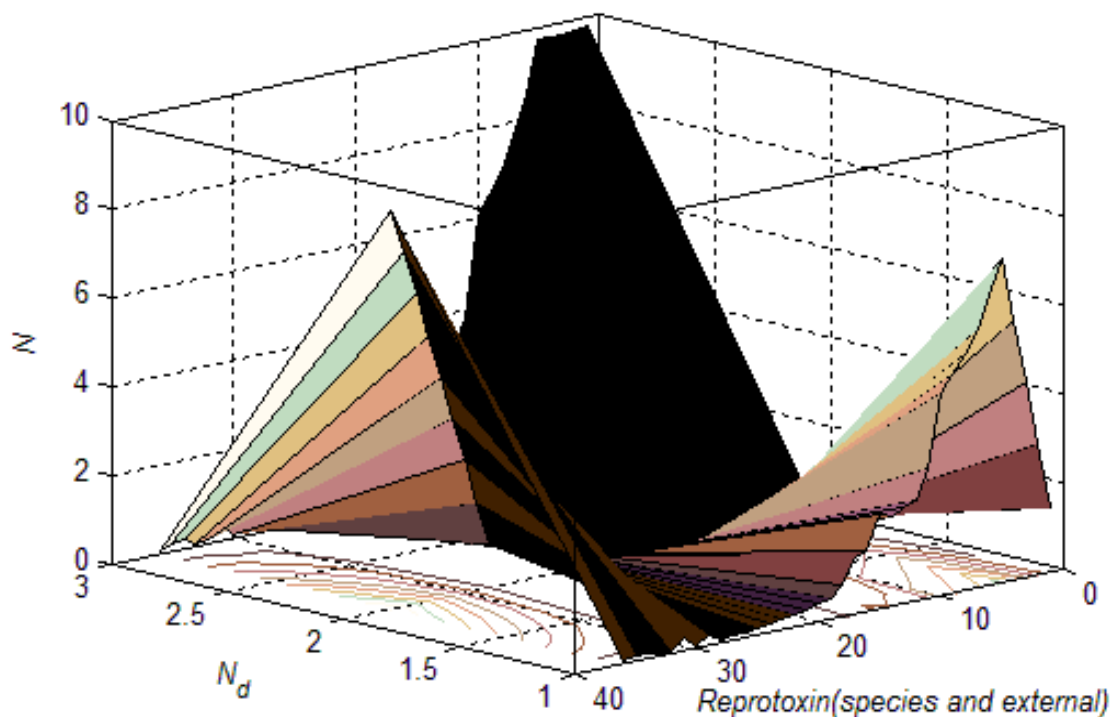


Figure 10. 3D Surface with contour of Dynamical System, Reprotoxin (species and external)

It is concluded that the system's trajectory path moves toward the equilibrium point in the region of attraction, allowing the system to remain stable. The second case is illustrated in Fig.8 and Fig.9 where the three variables are total population, deformed reproductive population, and λ . This plot concludes that if the reproductive toxicants from external sources are constant and the reproductive toxicant by the species is constantly emitted into the environment, the dynamic system is stable as time t tends to infinity and trajectory path moves toward the equilibrium point to maintain the stability of the system. The third case is depicted in Fig.10 in which the total population, deformed reproductive population, and reproductive toxicants (Q and λ both increasing together) are the three variables or matrix representations. This plot concludes that if both the reproductive toxicants produced by the species and the external sources continuously released into the environment, the dynamical system is asymptotically stable as time t tends to infinity, but becomes unstable if the reproductive toxicants rate exceeds the allowable level and the system's trajectory path moves away from the fixed point in the attractor basin, allowing the system to become unstable. From the above, it is clear that the third condition is more harmful and hazardous for biological species than the others, and it is more likely to result the extinction of the species.

5. Result and Discussion

Reprotoxin affects the reproduction, either directly or

indirectly via metabolism and uptake, transmission, conversion (toxification and detoxification), disposal, and recovery are the physiological processes that generate reproductive toxicity. The flow of energy, matter, and information that biological cells and organs require to function properly is interrupted by reprotoxins. The response of a biological species to a reprotoxin is complicated. As a result of these factors, there are multiple incidental exposures of toxicants into the environment in huge quantities, causing the species to have severe reproductive issues. Some of the reprotoxin that affect biological species, mostly in their reproductive organs, are discussed here and are essential to our daily life. This reprotoxin is released into the environment when organic resources such as coal, fuel, and wood are burned incompletely. Polycyclic aromatic hydrocarbons impair the reproduction process through indirect mechanisms such as hepatic and ovarian enzymes. Polycyclic aromatic hydrocarbons have an indirect negative impact. Polycyclic aromatic hydrocarbons stimulate hepatic and reproductive enzymes, as well as microsomal monooxygenases. Nicotine-stimulated receptors appear to be responsible for nicotine's action on the reproductive process. This has two effects: it causes the posterior pituitary to release epinephrine, which resembles the effect of nicotine on human reproductive process. Hazardous metals like cadmium, lead, arsenic, and mercury can harm the reproductive system. Metals like cadmium, lead, and mercury are direct-acting reproductive pollutants that are harmful to both developing and adult reproductive systems. Endometrial alteration, diminished fertility, obstructed

implantation, follicle necrosis, and other reproductive organ damage are examples of their direct effect at reproductive organs such as the ovary, uterus, and hypothalamus. When organophosphate and chlorinated pesticides were stored at the Apopka Lake site, it was found that they had a harmful effect on reproduction. The estrus cycle of young female rats was disturbed after postnatal treatment with mercuric chloride, resulting in diestrus lengthening. Researchers later found that mercury levels in reproductive macrophages, and granulosa indicated that there was direct reproductive impairment. DDT (dichlorodiphenyltrichloroethane) and other chemicals have the ability to alter the endocrine system and cause reproduction abnormalities in alligators. Reprotoxins have detrimental effects on the reproduction process, therefore DBCP (1, 2-Dibromo-3-chloropropane) was banned from use in U.S. crops. Diethylstilbestrol, a non-steroidal hormone used to treat cancer, has a negative impact on reproductive process, causing infertility, miscarriage, infant mortality, and premature birth. Diethylstilbestrol is an estrogenic chemical that acts on the reproduction system as a direct-acting reprotoxin. Oral contraceptive is a type of "reprotoxin" that prevents early follicular reproductive hormone surges from occurring. Oral Contraceptives work by blocking the release of reproductive hormones through feedback inhibition. Hormone-releasing substances (HRPs) can increase or decrease steroid hormone secretion or clearance by stimulating or inhibiting hepatic or reproductive hormone enzyme systems. This effect is especially important in situations where reprotoxin is teratogenic or fetotoxic. Reprotoxins can have a variety of adverse outcomes, and the majority of people are exposed to reprotoxin at work or during drug treatment. Alkylating chemicals, which are utilized in the chemical industry and to treat a variety of neoplastic and non-neoplastic disorders, are chemically reactive reprotoxins. The chance of sterility after certain types of cancer treatment increases the risk of malignant tumours in reproduction parts of the species. Oxidation by the antioxidant enzyme monooxygenases, which enhances the polarity of the reprotoxin, is one of the strategies used to reduce the harmful effect of reprotoxin in species. Chemically bonded metabolites are formed when polar molecules are conjugated or removed without additional processing. For assessing human reproductive risks, the process of hazard identification and characterization is critical. By providing a fast biological reaction to hazardous exposure, detoxification processes limit the amount of chemically reactive substances in the reproductive system. Detoxification mechanisms are an instantaneous biological response to toxic exposure. The body possesses systems for detoxification; however these mechanisms can be ineffective in some situations, resulting in cell, organ, or species harm. Repair may be achieved after harmful damage has occurred. To replace non-functioning proteins damaged by the toxicant, repair could be as easy as increasing or renewing protein

production. For DNA damage, more physiologically sophisticated repair mechanisms have evolved. Detoxification includes DNA damage detection as well as enzymes for removing and replacing damaged DNA. From the above research, it is concluded that both the external sources and species itself are increasing the emission rate of reprotoxin into the environment because of which the density of the population decreases below the carrying capacity.

6. Conclusions

The various harmful effects of reprotoxin on the reproduction species of biological species are discussed in this research using a nonlinear model. As the rate of reprotoxin increases in the environment, the rate of its uptake also increases, affecting the reproduction process in species. Reproductive toxicity is a global issue which is exacerbated by a reprotoxin discharging into the atmosphere by the external sources and the species itself. It is indicated in the various plots that the increase in the magnitude of parameters Q , λ , α , and decrease in some parameters may destabilize the system. It has been proven that a non-trivial fixed point exists, and its steadiness has been investigated. In the emission of two toxicants, the population settles down to fixed point but as the toxicant levels increase, the stability of the proposed dynamical system decreases. These reprotoxins have an adverse effect on the reproduction process (at first sub-lethal, then deadly), as well as progressive reproductive organ damage in subsequent generations. This study concludes if reprotoxin from both the external sources and species continues to increase in the environment; their harmful effect on the population increases, increasing the system's instability. It also concludes that the density of species declines below the carrying capacity, increasing the risk of extinction of the various species over time. Several reprotoxins have been shown to cause cancer in the various part of reproductive system due to the effect of reprotoxin in biological species and gets affected as its multistep protection network fails to prohibit the harmful disruption of the flow of matter, energy or crucial information required for successful reproduction. It concludes that the level of species population may be preserved by regulating the amount of various reprotoxins in the environment. This can only be achieved by cleansing the environment at the source and removing reprotoxin from the ecosystem. This research concludes that the harmful effects of numerous types of reprotoxins must be regulated in order for biological species to survive on the planet.

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