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:

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

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

$$\left[r_1U_1^* + r_2U_2^* - (\alpha+b)\left\{1 - \frac{1}{K(T_1^*,T_2^*)}\right\} - \frac{rN_d^*}{K(T_1^*,T_2^*)}\right]^2$$

$$< \frac{4}{25}\frac{r}{K(T_1^*,T_2^*)}\left[r_1U_1^* + r_2U_2^* + (\alpha+d) + \frac{rN^*}{K(T_1^*,T_2^*)} - \frac{\alpha+b}{K(T_1^*,T_2^*)}\left\{\frac{K_0(r_1+r_2)(\lambda K_0+Q)}{(r_1+r_2)(\lambda K_0+Q)+(\alpha+d)\delta_m} + N_d^*\right\}\right] \quad (2.15a)$$

$$\left[\lambda + (\pi_1\nu_1 - \gamma_1)\frac{(\lambda K_0+Q)}{\delta_m} + \left\{(\alpha+b)\frac{K_0(r_1+r_2)(\lambda K_0+Q)}{[(r_1+r_2)(\lambda K_0+Q)+(\alpha+d)\delta_m]} - rK_0\right\}\frac{\kappa_1}{K_m^2}\right]^2 < \frac{4}{15}\frac{r}{K(T_1^*,T_2^*)}(\delta_1 + \gamma_1 N^*) \quad (2.15b)$$

$$\left[(\pi_1\nu_1 - \gamma_1)\frac{(\lambda K_0+Q)}{\delta_m} + \left\{(\alpha+b)\frac{K_0(r_1+r_2)(\lambda K_0+Q)}{[(r_1+r_2)(\lambda K_0+Q)+(\alpha+d)\delta_m]} - rK_0\right\}\frac{\kappa_2}{K_m^2}\right]^2 < \frac{4}{15}\frac{r}{K(T_1^*,T_2^*)}(\delta_2 + \gamma_2 N^*) \quad (2.15c)$$

$$\left[(\gamma_1 - \nu_1)\frac{(\lambda K_0+Q)}{\delta_m}\right]^2 < \frac{4}{15}\frac{r}{K(T_1^*,T_2^*)}(\beta_1 + \nu_1 N^*) \quad (2.15d)$$

$$\left[(\gamma_2 - \nu_2)\frac{(\lambda K_0+Q)}{\delta_m}\right]^2 < \frac{4}{15}\frac{r}{K(T_1^*,T_2^*)}(\beta_2 + \nu_2 N^*) \quad (2.15e)$$

$$\left[\left((\alpha+b)\left\{\frac{K_0(r_1+r_2)(\lambda K_0+Q)}{[(r_1+r_2)(\lambda K_0+Q)+(\alpha+d)\delta_m]}\right\}^2 - \frac{rN^*K_0(r_1+r_2)(\lambda K_0+Q)}{[(r_1+r_2)(\lambda K_0+Q)+(\alpha+d)\delta_m]}\right)\frac{\kappa_1}{K_m^2}\right]^2$$

$$< \frac{4}{15}(\delta_1 + \gamma_1 N^*)\left[r_1U_1^* + r_2U_2^* + (\alpha+d) + \frac{rN^*}{K(T_1^*,T_2^*)} - \frac{\alpha+b}{K(T_1^*,T_2^*)}\left\{\frac{K_0(r_1+r_2)(\lambda K_0+Q)}{(r_1+r_2)(\lambda K_0+Q)+(\alpha+d)\delta_m} + N_d^*\right\}\right] \quad (2.15f)$$

$$\left[\left((\alpha+b)\left\{\frac{K_0(r_1+r_2)(\lambda K_0+Q)}{[(r_1+r_2)(\lambda K_0+Q)+(\alpha+d)\delta_m]}\right\}^2 - \frac{rN^*K_0(r_1+r_2)(\lambda K_0+Q)}{[(r_1+r_2)(\lambda K_0+Q)+(\alpha+d)\delta_m]}\right)\frac{\kappa_2}{K_m^2}\right]^2$$

$$< \frac{4}{15}(\delta_2 + \gamma_2 N^*)\left[r_1U_1^* + r_2U_2^* + (\alpha+d) + \frac{rN^*}{K(T_1^*,T_2^*)} - \frac{\alpha+b}{K(T_1^*,T_2^*)}\left\{\frac{K_0(r_1+r_2)(\lambda K_0+Q)}{(r_1+r_2)(\lambda K_0+Q)+(\alpha+d)\delta_m} + N_d^*\right\}\right] \quad (2.15g)$$

$$\left[r_1\left(K_0 - \frac{rN^*K_0(r_1+r_2)(\lambda K_0+Q)}{[(r_1+r_2)(\lambda K_0+Q)+(\alpha+d)\delta_m]}\right)\right]^2$$

$$< \frac{4}{15}(\beta_1 + \nu_1 N^*)\left[r_1U_1^* + r_2U_2^* + (\alpha+d) + \frac{rN^*}{K(T_1^*,T_2^*)} - \frac{\alpha+b}{K(T_1^*,T_2^*)}\left\{\frac{K_0(r_1+r_2)(\lambda K_0+Q)}{(r_1+r_2)(\lambda K_0+Q)+(\alpha+d)\delta_m} + N_d^*\right\}\right] \quad (2.15h)$$

$$\left[r_2\left(K_0 - \frac{rN^*K_0(r_1+r_2)(\lambda K_0+Q)}{[(r_1+r_2)(\lambda K_0+Q)+(\alpha+d)\delta_m]}\right)\right]^2$$

$$< \frac{4}{15}(\beta_2 + \nu_2 N^*)\left[r_1U_1^* + r_2U_2^* + (\alpha+d) + \frac{rN^*}{K(T_1^*,T_2^*)} - \frac{\alpha+b}{K(T_1^*,T_2^*)}\left\{\frac{K_0(r_1+r_2)(\lambda K_0+Q)}{(r_1+r_2)(\lambda K_0+Q)+(\alpha+d)\delta_m} + N_d^*\right\}\right] \quad (2.15i)$$

$$[(\gamma_1 + \pi_1\nu_1)N^*]^2 < \frac{4}{9}(\delta_1 + \gamma_1 N^*)(\beta_1 + \nu_1 N^*) \quad (2.15j)$$

$$[(\gamma_2 + \pi_2\nu_2)N^*]^2 < \frac{4}{9}(\delta_2 + \gamma_2 N^*)(\beta_2 + \nu_2 N^*) \quad (2.15k)$$

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) ⇒ (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:

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

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.

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

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

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 λ =.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. $\boldsymbol{\lambda}$ =.01 and the value of Q is increased continuously, (ii) the reproductive toxicant by the external sources is constant i.e. Q = .1 and $\boldsymbol{\lambda}$ 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. $\boldsymbol{N_1, N_2, N_d, T_1, T_2, U_1}$ & $\boldsymbol{U_2}$ for different value of Q & λ

$\boldsymbol{N}$	$\boldsymbol{N_d}$	$\boldsymbol{T_1}$	$\boldsymbol{T_2}$	$\boldsymbol{U_1}$	$\boldsymbol{U_2}$	$\boldsymbol{Q}$	$\boldsymbol{\lambda}$
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

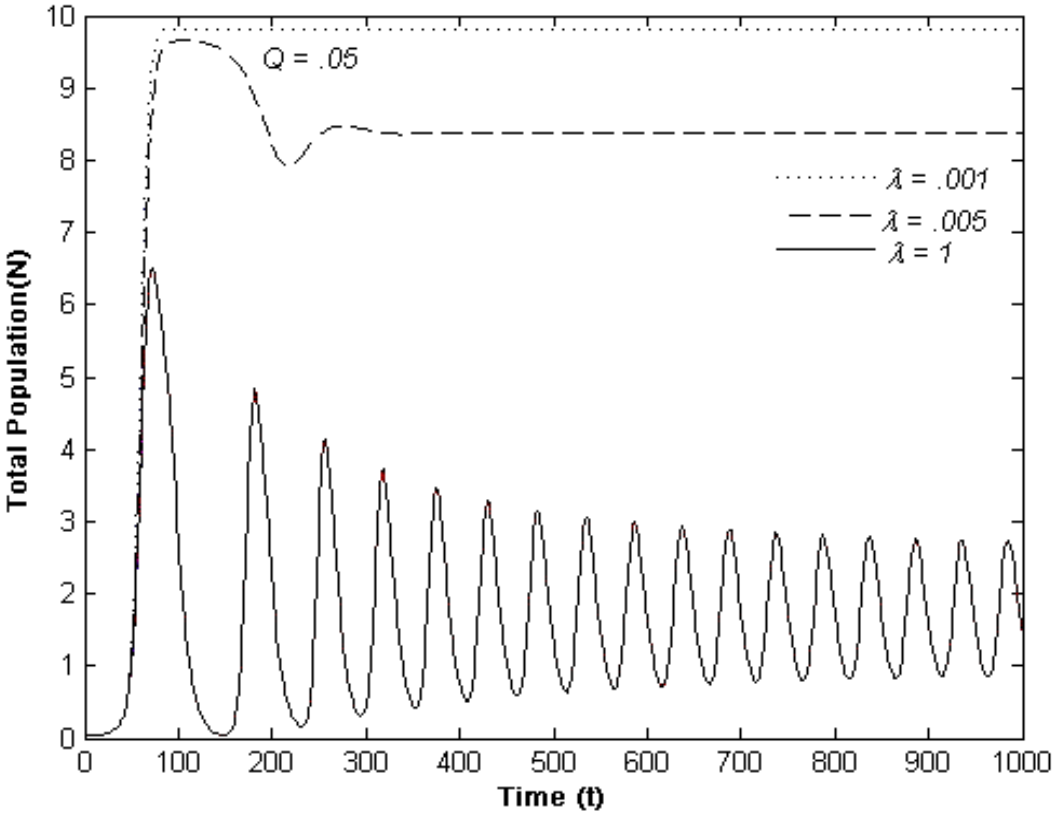

Figure 1. Variation of Total Population w.r.t Time($\boldsymbol{t}$)

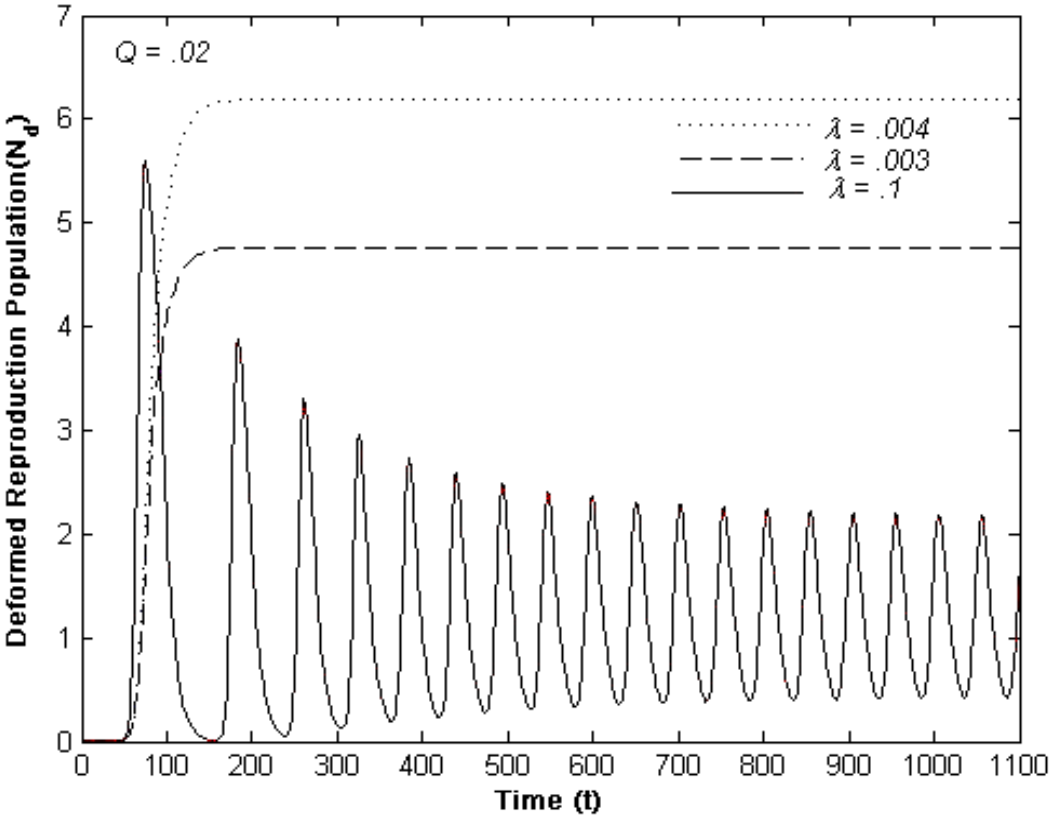

Figure 2. Variation of Deformed Reproduction population w.r.t Time (t)

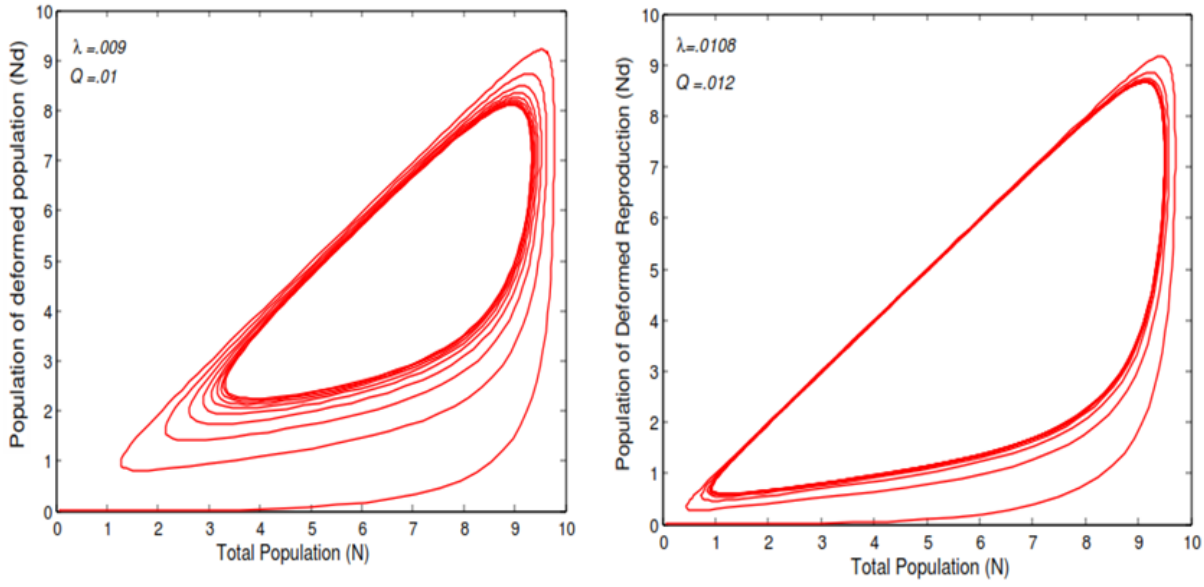

Figure 3. Phase Diagram "Variation of Total Population versus deformed Population with respect to Time (t)

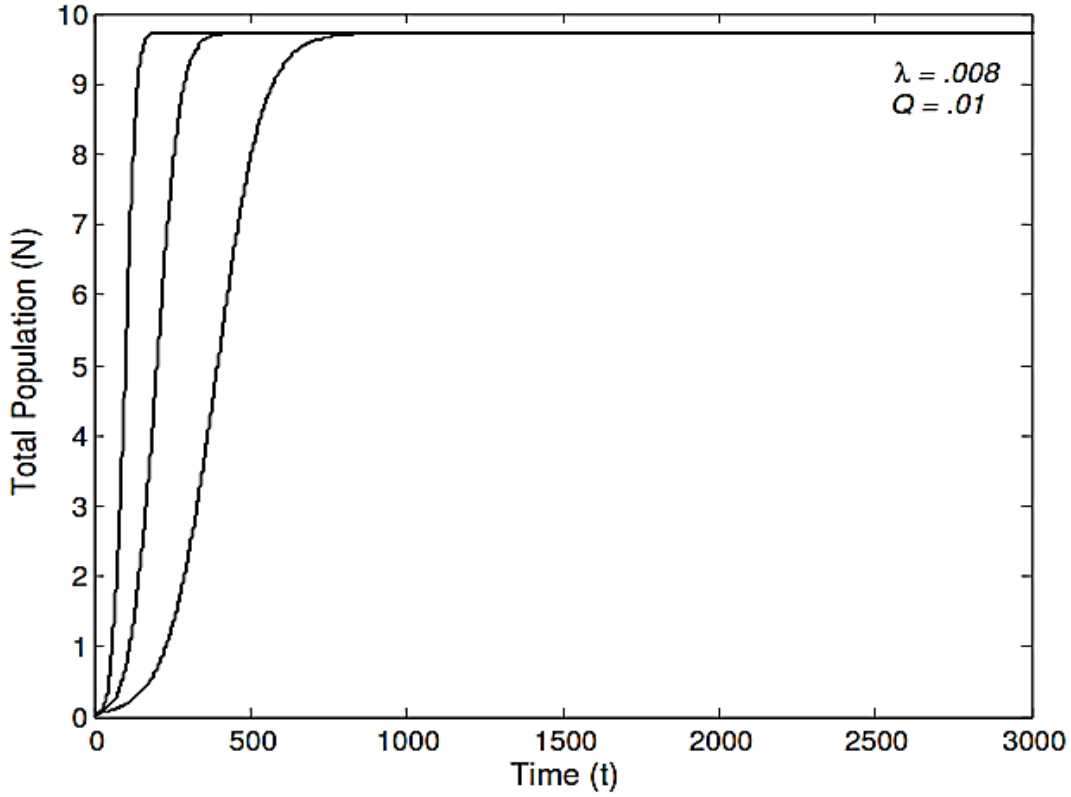

Figure 4(a). Total Population versus time(t) for changed value of β_1 & β_2

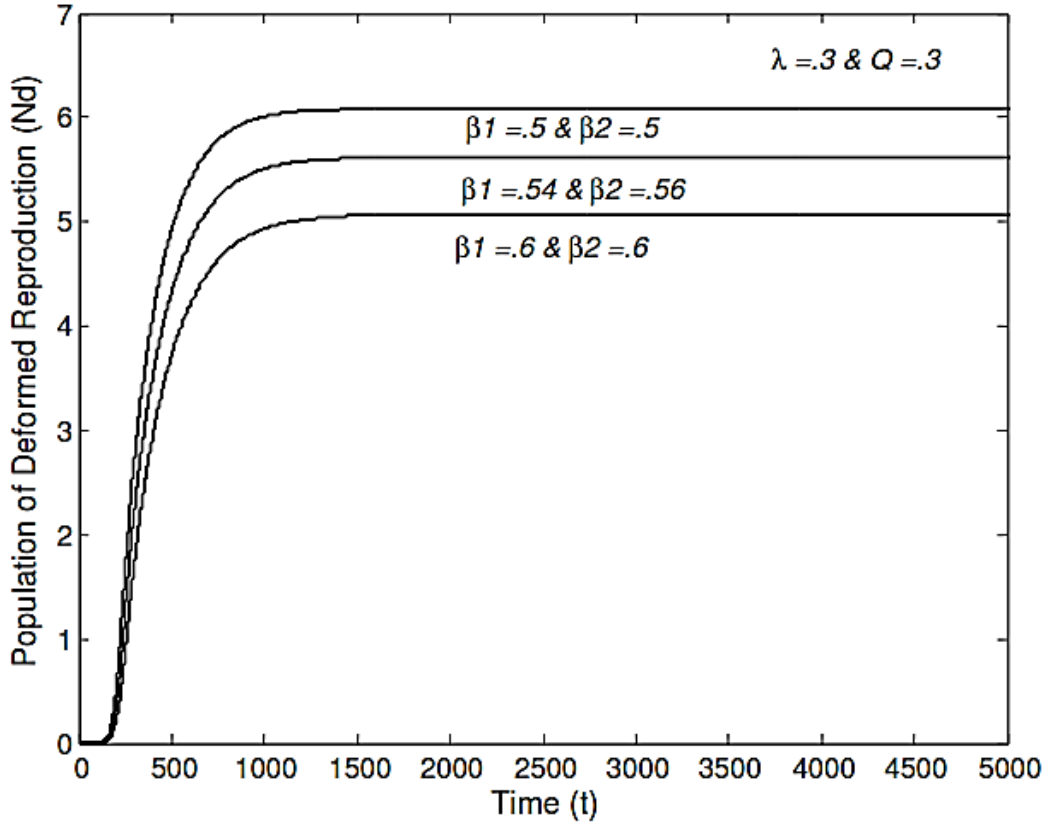

Figure 4(b). Deformed Reproduction Population versus time(t) for the changed value of β_1 & β_2

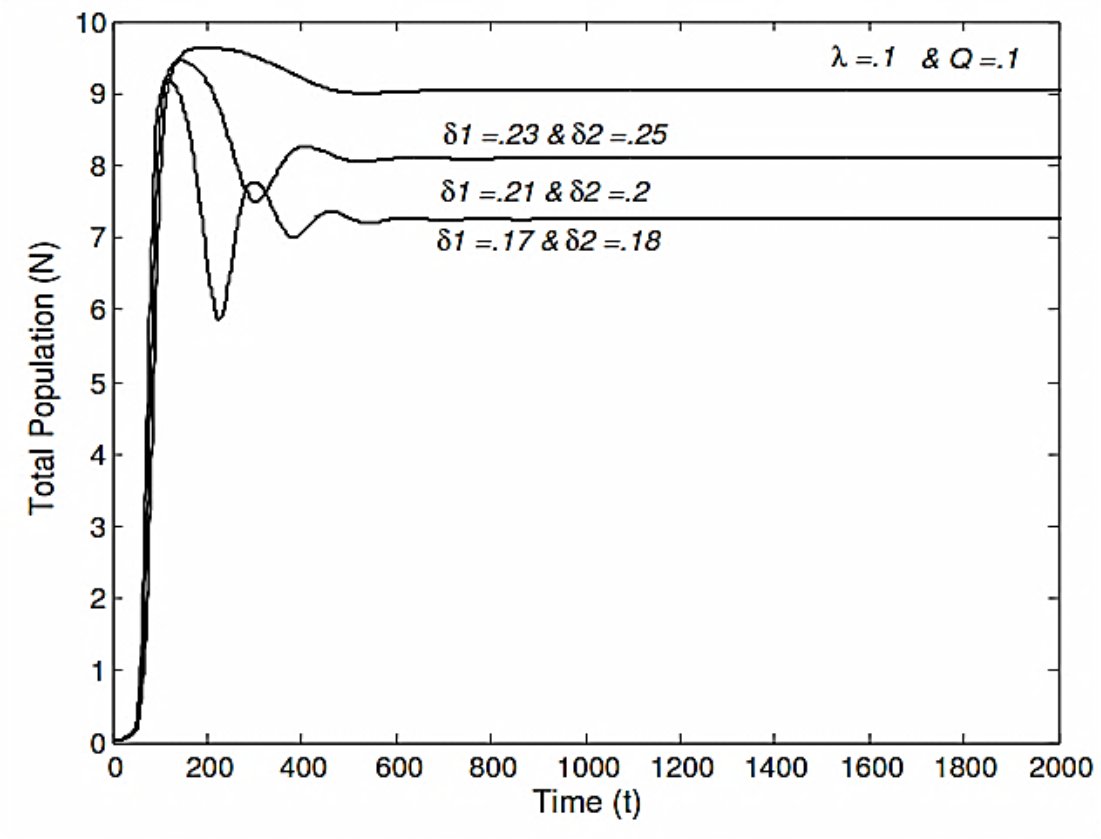

Figure 4(c). Total population versus time (t) versus for changed value of $\boldsymbol{\delta_1}$ & $\boldsymbol{\delta_2}$

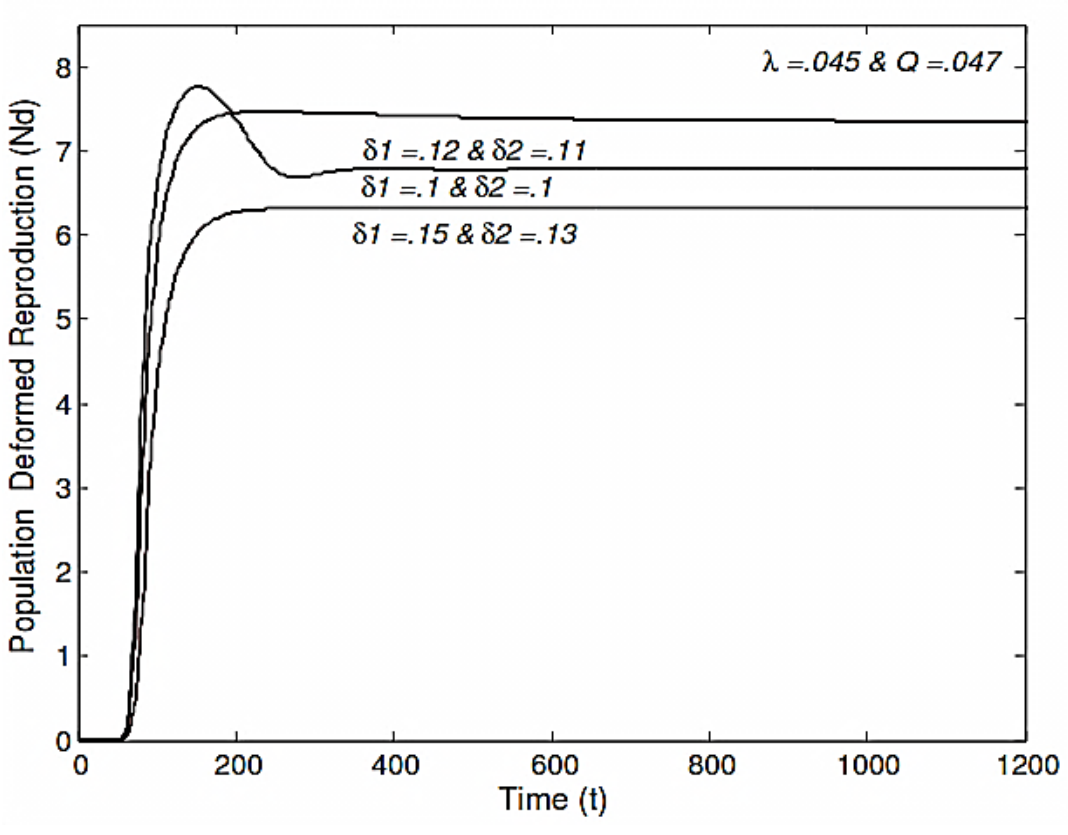

Figure 4(d). Deformed reproduction population versus time (t) $\boldsymbol{for\ the\ changed}$ value of $\boldsymbol{\delta_1}$ & $\boldsymbol{\delta_2}$

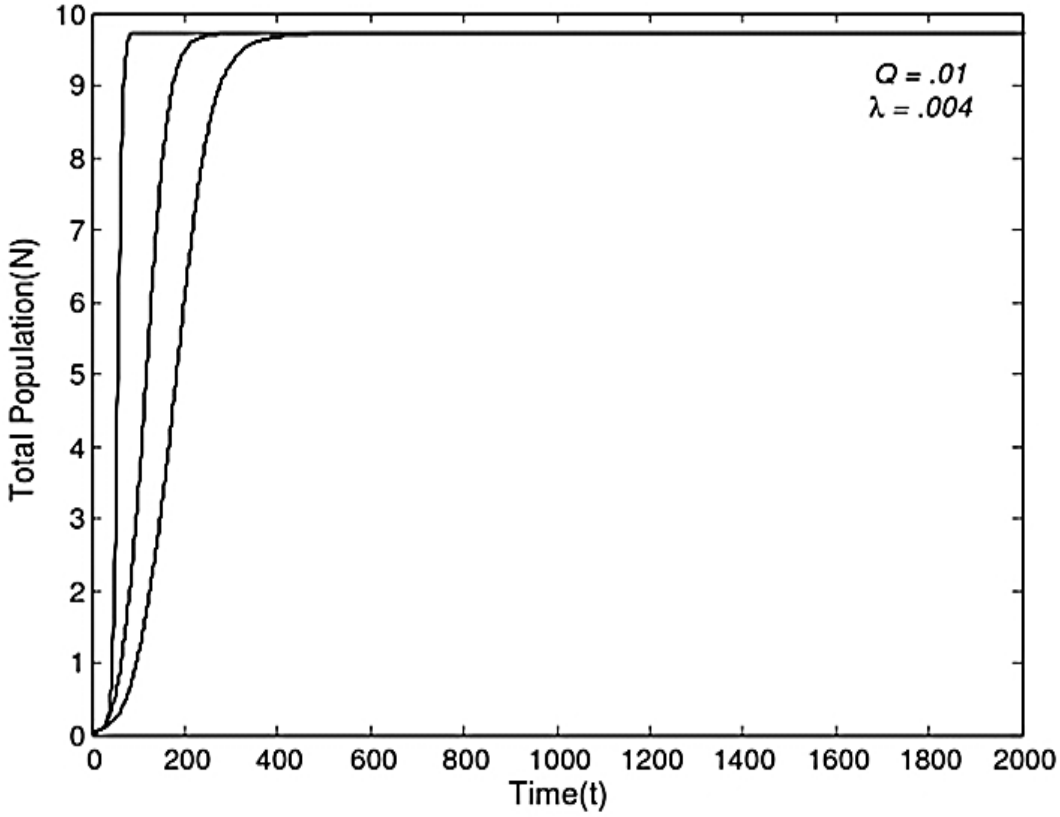

Figure 5(a). Total Population versus Time(t) for changed value of α

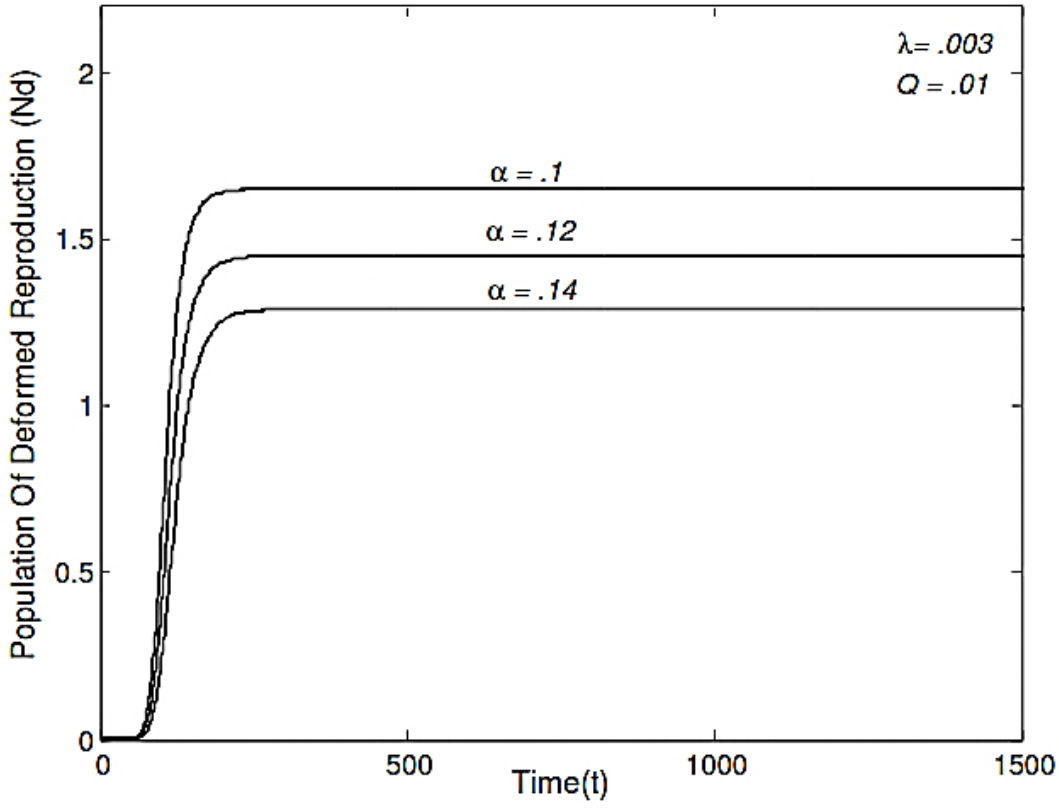

Figure 5(b). Deformed Reproduction versus Time (t) for changed value of α

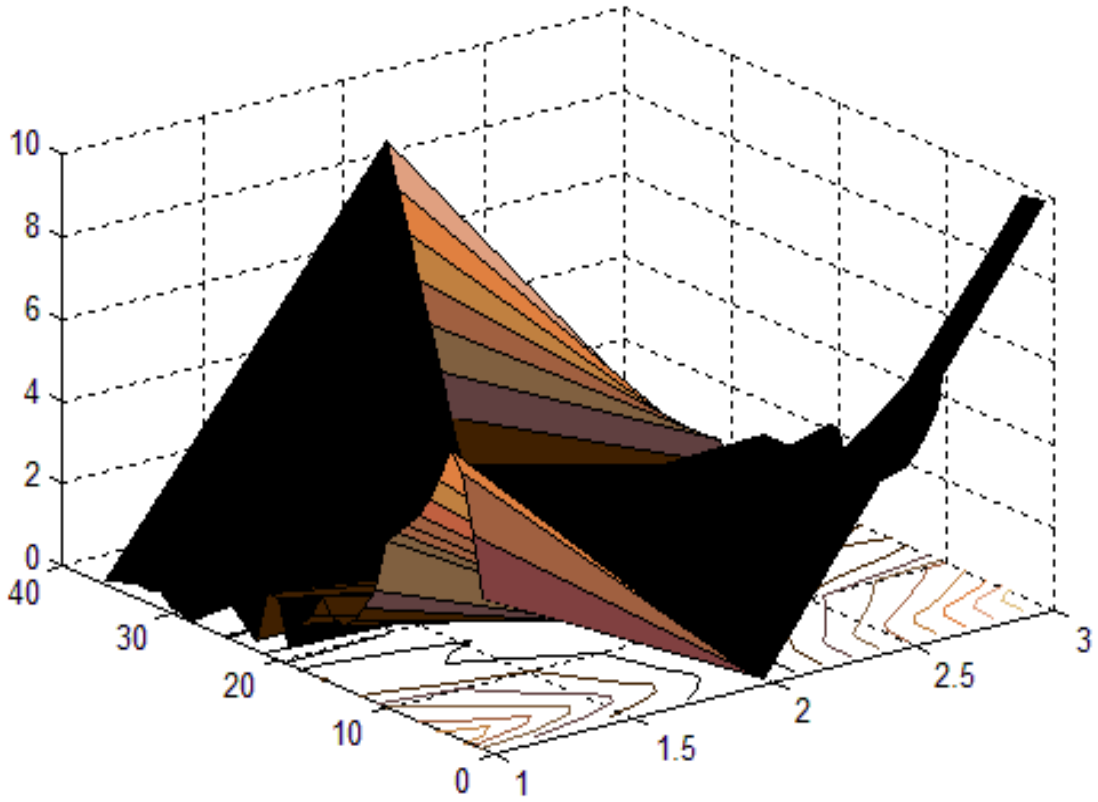

Figure 6. 3D Surface Plot with Contour of Dynamical Population, Reprotoxin(external)

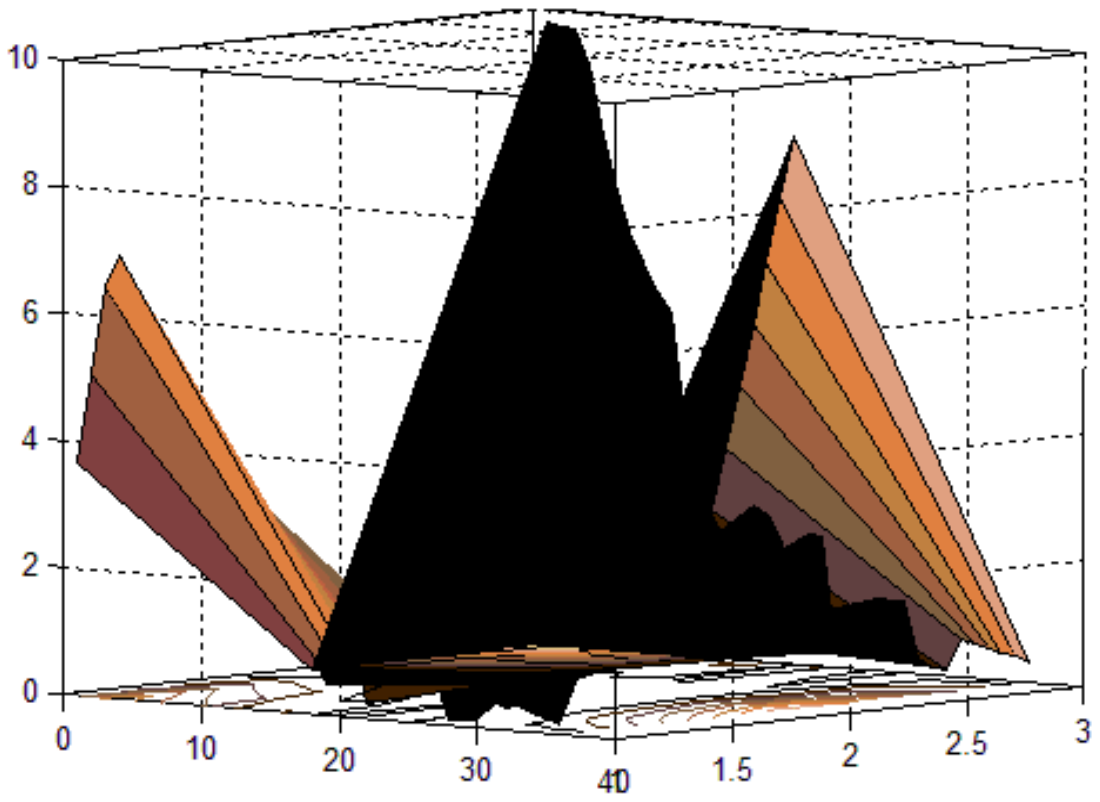

Figure 7. Rotation of 3D Surface Plot with contour of the dynamical system

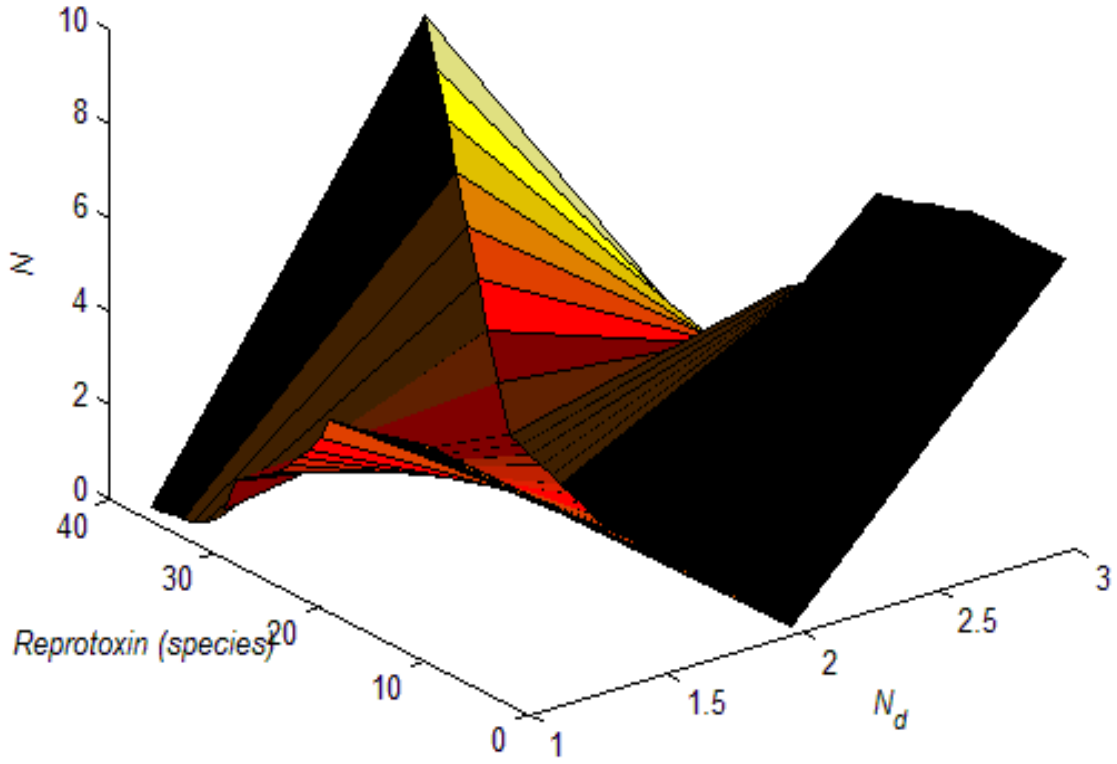

Figure 8. 3D Surface Plot with Contour of Dynamical System, Reprotoxin (species)

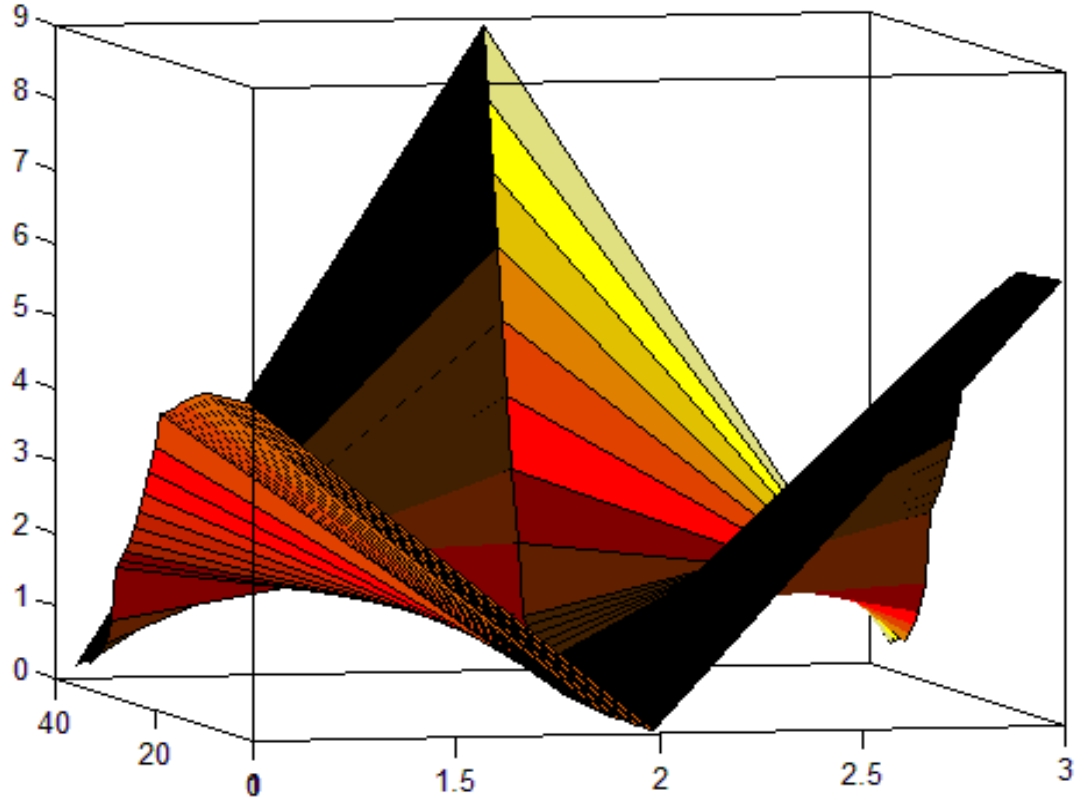

Figure 9. Rotation of Surface Plot of Dynamical System, Reprotoxin (species)

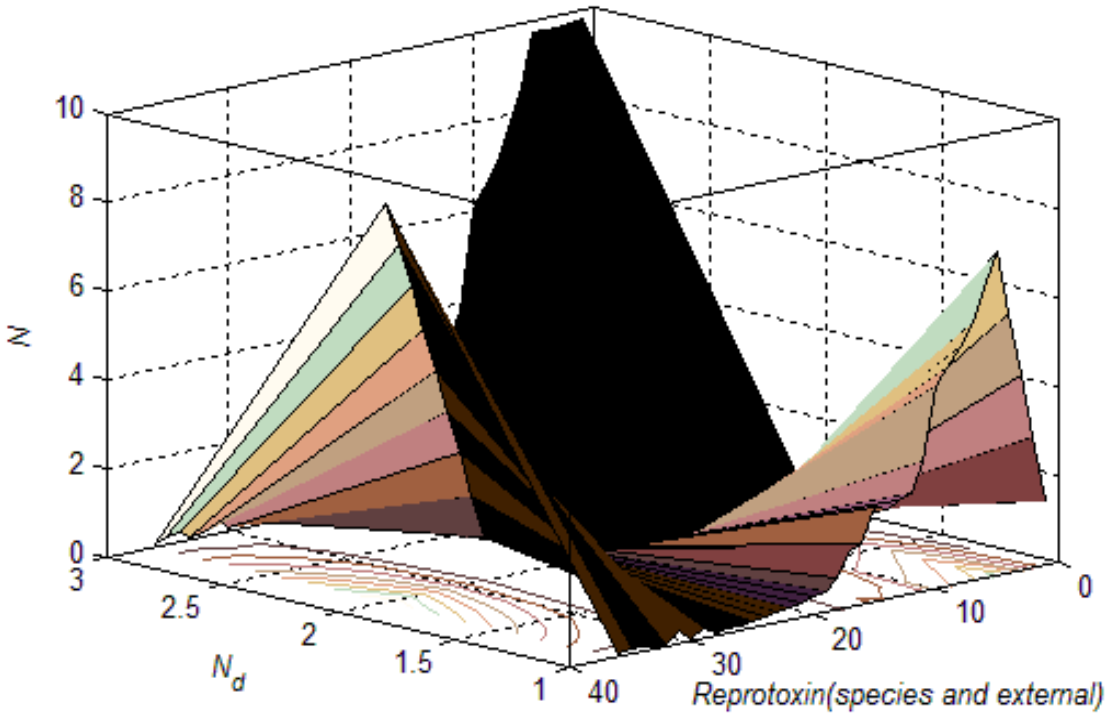

Figure 10. 3D Surface with contour of Dynamical System, Reprotoxin (species and external)

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 $\boldsymbol{\lambda}$. 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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