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MODELING THE EFFECT OF REPRODUCTIVE TOXICANT IN SUBCLASS AND IS EMITTED INTO THE ENVIRONMENT BY THE BIOLOGICAL SPECIES ITSELF

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Abstract: In this paper, a nonlinear model is analyzed to review of some of the chemical substances and this chemical substance produces harmful effects on the reproductive capability of the biological species. The chemical substance is the reproduction toxicant produced by the biological species itself. As the species is polluting its own environment therefore an increase in reproduction toxicant is directly proportional to the increase in population of the species. This shows various effects on the reproduction behavior of the species depending upon the concentration level or intake quantity by the biological species. This reproduction toxicant enters inside the body through inhalation, skin contact, or ingestion causing reproduction deformities in different parts of the body or the reproduction system of the species after undergoing various different mechanisms (structurally or chemically). Mathematical models have played a vital role in sketching the ecological problem to research and predicting the dynamical behavioral variation of both aquatic and terrestrial ecosystems. Some members of biological species get critically affected and show abnormalities in the reproduction behavior of biological species, like deformity, fecundity, endometriosis, reproductive cancer, etc. Various mathematical tools and stability theory of equation is

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employed to research the decreases within the biological population we conduct stability analysis on the nontrivial equilibrium solution of the model using Lyapunov's direct method and eventually, we conduct numerical simulations so as to check the analytical outcomes with numerical computations, so on substantiate that there is a parameter range that the results are relevant. The reproduction process increases the population of a selected species which in turn increases the reproduction toxicant in the environment, and due to an increased level of reproduction toxicant, the reproduction in species gets lethal and sub-lethal effect because of which the population decreases below the carrying capacity. This paper is to draw attention of the viewer to put a control on the population of species including the human beings and let them to become more conscious in keep the environment free from toxicant effect.

Keywords: mathematical model; reproductive toxicants; carrying capacity; biological species.

2010 AMS Subject Classification: 93A30.

1. INTRODUCTION

Reproduction toxicant is producing various harmful effects on the reproduction of the biological species by yielding several direct and indirect mechanisms on the site of the targeted organ. Consider an ecosystem during which there are two principal biological species, a plant species, and an animal species. The animal species consume and is partially dependent on the plant species. Pollution is also being inputted into the environment .We assume that the environment may be a spatially homogeneous environment and also that there's no migration to or emigration from the ecosystem. Additionally, we supposed that each one of the individuals within either species population is identical. Several factors determine the deleterious effects of environmental toxicant on the functioning of the female reproduction system. There are various types of reproductive toxicant releasing into the atmosphere and are producing a harmful effect on the reproduction of the subspecies. Successful reproduction in species performs a complex series of interdependent physiological, molecular and cellular events. Reproduction in species is vulnerable at many points of the reproductive cycle due to the dreadful effect of reproductive toxins continuously discharging into the atmosphere such as phthalate, bisphenol A, dioxin, heavy metals, pesticide, etc. One of the most common reasons for increasing reproductive toxicant in the atmosphere is external sources or man-made projects by biological species especially human beings have established for their short-time happiness. Some of the reproductive

toxicants causing reproductive deformities in biological species are dibromochloropropane (DBCP), Carbon disulfide, dioxin, phthalate, esters of phthalate, heavy elements like mercury, cadmium, lead, etc., pesticides, radioactive rays, etc., are responsible for the unsuccessful reproduction because of which the population of the particular species declines to create an imbalance in the ecosystem [12, 17]. Many researchers have proposed and mathematical models to investigate the effect of toxicant on biological species [2, 5, 9, 11 and 14]. The research works have been carried out for separate cases such as the effect of single toxicant, more than one toxicants, n-toxicant on a biological species, or two biological species with a competition of thrives in the same atmosphere. Freedman proposed a model [11] to determine the effect of toxicant on species and also on a predator-prey system. Widdows considered sampled mussels in various part of the [22] Venice Lagoon overstretch by chemical toxicant (Cr, Hg, Ni, Cd, Fe, Mn or chlorinated hydrocarbons, etc.) and initiate a significant reduction in their heightening as compared to living in other regions of the Lagoon. Veeramachaneni initiate the serious harmful effects of chemicals on [21] male reproduction. It have been also found that created toxicant media for the [18] fish exhibiting irregular, random, hyperexcitability, circular swimming movements; sink to the bottom and loss of equilibrium. Caudal bending is the prime morphological malformation. Herath have initiate that when the collaborative effect of two [8] toxicant octylphenol (OP) and bisphenol A (BPA), and accessory reproduction organs. This was very crucial phenomenon and was considered using mathematical models for very limited cases proposed a mathematical model to [3, 5] found the effect of a single toxicant discharge from some external sources, on a biological species in which a subspecies of biological species having abnormal symptoms and not capable of reproduction. Lindley have research that when two organochlorine [17] compounds, pentachlorophenol (PCP), a respiratory uncouple, and 1,2-dichlorobenzene (DCB), a non-polar narcotic, were selected for toxicity eggs of estuarine and neritic planktonic calanoid copepods then it is concluded that as the concentration of this two compound rises it reduces the percentage of eggs hatched after exposure to a pesticide. Woin and Bronmark [23] have considered the effect of DDT and MCPA on the reproduction of snail collected from a eutrophic pond in southern Sweden and showed that these pollutants may have no effect on mortality but have a reinitiate effect on the distribution. Additionally, Kumar proposed and investigate a modified model to investigate the effect of toxins on a biological species in which a subspecies [5] of biological species is critically affected. He have also considered the effect of a single toxins on the reproduction of a

biological species in separate cases such as toxins [5] discharge by biological species itself (e.g., industrial, and domestic activities, vehicular exhaust, radioactive wastes, use of pesticides, etc.,) and from some external sources (e.g., volcanic eruptions, forest fires, etc.,), respectively. This research shows that biological species do not have any morphological deformity but are critically affected by these pollutants and develop certain types of diseases such as declined reproductive capability, cancer, asthma, etc. which results in increasing the mortality rate of these biological species. Kumar have initiate that the effect of two toxins on the biological species [15] in which they have revealed that when both toxins level rises, the total density of species decline, the density of the subspecies of species firstly rises and then it decline as the total density decline. In this investigate; it is assumed that the toxins are assumed to be discharge in the atmosphere by biological species itself. Agrawal have considered that the [5] effects of n toxins on a biological population. Agrawal [2] have initiated that the effect of reproductive toxins on biological species discharge from the external sources. Now, we propose and analyses a mathematical model to investigate the effect of a reproductive toxins on the reproduction capacity of the biological species produce by itself. We have assumed that the biological species are adversely affected by these reproductive toxin and some members of the species lose the capability of reproduction due to which the mortality rate rises, decreasing the population of many species. We begin by proposing a new model to study the effects of a pollutant on local animal and plant biomass. We conduct local and global stability analysis on the nontrivial equilibrium solution of the model using the direct Lyapunovs method. And finally, we conduct numerical simulations in order to compare the analytical results with numerical computations, in order to confirm that there is a parameter range for which the results are relevant. The purpose of this review is to put all possibility that environmental reproductive toxicant contribute to adverse reproductive outcomes.

2. MATHEMATICAL MODEL

Let's we take a logistic growth for a selected biological species, here N is total population density of the biological species at time t. The biological species is producing the reproductive toxicant itself and polluting its own environment. When this reproductive toxicant enters the targeted organ in the body it changes the normal function of the organs particularly the reproductive system of the biological species. Reproductive toxicant produces a direct and indirect effect on the reproductive process depending upon the doses of intake in the biological species. This effect either includes

structural similarities or chemical reactivity at or inside the targeted organ. It may produce an adverse effect on the reproductive behavior of biological species by one or more mechanisms (structurally or chemically). The structural similarity mislead the biological process and the chemical reactivity in the targeted organ produces new compound and these compounds are considered more dangerous as compared to the reproductive toxicant and thus produces severe harmful effect in the reproduction process of the biological species. In this system, we have supposed that increasing rate of the intake concentration or doses of the reproductive toxicant by this species is same as the rate of the decrease of the reproductive toxicant in the atmosphere and is taken to be directly proportional to population density as well as the concentration of the toxicant in the environment. It is also assumed that as the uptake of toxicant increases, its carrying capacity decreases with the increased rate of concentration of the reproductive toxicant in the environment. Here, N_d is the population density or a subclass of species facing the problem of infertility after the effect of reproductive toxicant which is a subclass of total population(N). The environmental concentration of the reproductive toxicant assumed to be $T(t)$ at time t. Let $U(t)$ be the intake of reproductive toxicant by the biological species living in assumed system at time t. $N_f(t)$, is the population density which is free from any reproduction deformities. Here, the per capita growth rate of N_f is $r - r_1U = b - d - r_1U$, where d is the natural death rate, b the natural birth rate and r_1 is the rate at which the reproduction capable population of the biological species decreases after the effect of the reproductive toxicant. δ, is denoting the natural depleting rate coefficient of $T(t)$ due to the intake of reproductive toxicant. β, is denoting the natural depleting rate coefficient of $U(t)$ due to the reproductive effect of toxicant. γ is the depleting rate coefficient of $U(t)$ due to uptake of reproductive toxicant by the species, v is the depleting rate coefficient of $U(t)$ due to decay of some members of N, π is a fraction of the depleting of $U(t)$ due to decay of some members of the total population which may reenter into the environment. α is the mortality rate coefficient of the population become infertile due to high reprotoxicity, Q is the rate of discharge of reproductive toxicant into the environment by the species itself . δ, is denoting the natural depleting rate coefficient of $T(t)$ due to the uptake of the reproductive toxicant. The concentration of reproductive toxicant in the environment is continuously decreasing due to its assimilation by the abnormalities in the reproduction process in a subclass of species. These different doses of reproductive toxicant into the body of different species attack various part of the reproductive system and decrease the capability of reproduction which in turn decreases the intrinsic growth in biological species. It is also supposed that the

reproductive toxicant in the environment and their uptake by the species decreases due to some natural factors.

Now by keeping in mind all the above facts of the assumed system affected by the reproductive toxicant and increasing the infertility in species, a model has been proposed and is given below:-

$$\frac{dN_f}{dt} = \left(r - r_1 U - \frac{1}{K(T)}[rN - (b+\alpha)N_d]\right) N_f$$
$$\frac{dN_d}{dt} = \left(\frac{r_1 N_f U}{N_d} - d - \alpha - \frac{1}{K(T)}[rN - (b+\alpha)N_d]\right) N_d$$
$$\frac{dT}{dt} = \lambda N - \delta T - \gamma NT + \pi\nu NU$$
$$\frac{dU}{dt} = \gamma NT - \beta U - \nu NU \qquad (4.1)$$

Where, $N(t) = N_f(t) + N_d(t),\ N_f(0), N_d(0), T(0) \geq 0, U \geq cT(0), c > 0, 0 \leq \pi \leq 1$

Here, $K(T)$ is a function of N and it decreases with increasing reprotoxicity to measure the carrying capacity of the environment. According to the model system (4.1)

$$K(0) = K(0) > 0, \qquad K(T) > 0, \frac{dK}{dT} < 0 \ or \ T > 0 \qquad (4.2)$$

Here, $K_0 = K(0)$ is the greatest value carrying capacity of biological species in reprotoxic free environment. Now we reduces the model (4.1) to make it free from N_f and this can be done by using the fact that $N(\mathrm{t}) = N_f(\mathrm{t}) + N_d(\mathrm{t})$. The reduced model is given below:-

$$\frac{dN}{dt} = [rN - (\alpha + b)N_d]\left[1 - \frac{N}{K(T)}\right],$$
$$\frac{dN_d}{dt} = r_1(N - N_d)U - (\alpha + d)N_d - [rN - (\alpha + b)N_d]\frac{N_d}{K(T)}$$
$$\frac{dT}{dt} = \lambda N - \delta T - \gamma NT + \pi\nu NU$$
$$\frac{dU}{dt} = \gamma NT - \beta U - \nu NU \qquad (4.3)$$

2.1 Dynamical behavior of the model

If the reproductive toxicant enters the body of the biological species, it is distributed to different targeted organ and altered the reproduction process which in turn increases the infertility in species and this effect has been shown by proposed model (4.3). As the proposed model is

nonlinear so determination of the exact solution is almost impossible. For this a solution is obtained at the point motion of the function or the trajectory path move towards the equilibrium points and would provide a long term behavior dynamically for the system and this solution of the problem would not be changed with time and remains the same. If there is any equilibrium point with non-negative value for the system by keeping the right side of proposed model (4.3) equal to zero then there exist the possibility of having solution for the model system (4.3)**.** On solving the proposed model (4.3) it gives three equilibrium point E_1, E_2 and E_3 for this dynamical system which is non-negative. Existence of all the three equilibrium point is as follows.

$$E_1 = (0,0,0,0),\ E_2 = \left\{K(\tilde{T}), K(\tilde{T}), \tilde{T}, \tilde{U}\right\} \text{ \& } E_3 = \left\{K(T^*), \frac{r_1 U^*}{\alpha + b} K(T^*), T^*, U^*\right\}$$

To obtain the local as well as global stability for the system, eigenvalue at the equilibrium point is determine. If all the eigenvalues at the equilibrium points are negative then the proposed system may have the possibilities of existing stability.

2.2 Uniqueness & existence of the model

Let us we now check the existence and uniqueness at the equilibrium point E_3, which is given by $E_3 = \left\{K(T^*), \frac{r_1 U^*}{\alpha+b} K(T^*),\ T^*, U^*\right\}$, would confirm the existence of the positive value of N^*, N_d^*, T^* and U^*.

$$N = K(T), \tag{4.4.a}$$

$$N_d = \frac{r_1 U}{\alpha + b} K(T), \tag{4.4.b}$$

$$T = \frac{\lambda N(\beta + \gamma N)}{\{\beta\delta + (\delta v + \beta\gamma)N + (1-\pi)\gamma v N^2\}} = g(N) \tag{4.4.c}$$

$$U = \frac{\gamma N Q}{\{\beta\delta + (\delta v + \beta\gamma)N + (1-\pi)v\gamma N^2\}} = h(N) \tag{4.4.d}$$

$$f(N) = \{\beta\delta + (\delta v + \beta\gamma)N + (1-\pi)\gamma v N^2\}$$

As the rate of reprotoxic rate into the environment by the increasing population rate of the species rises and its uptake by the biological species increases. The population due to reproduction process gets affected which in turn decreases the carrying capacity of the biological species. Using equations (4.4.a), (4.4.c) and (4.4.d), we get

$$F(N) = N - K\big(g(N)\big) \tag{4.5}$$

$$F'(N) = 1 - \frac{dK}{dT}\frac{dg}{dN} > 0 \tag{4.6}$$

From, equations (4.4.c) we get

$$\frac{dg}{dN} = \frac{-\lambda N\gamma}{f^2(N)}\{\beta^2 + 2\beta v(1-\pi)N + v^2(1-\pi)N^2\} < 0 \tag{4.7}$$

Since

$$\frac{dK}{dT} < 0 \left[\text{from equation (4.2) and } \frac{dg}{dN} < 0 \text{ from equation (4.7)}\right]$$

So,

$$\frac{dF}{dN} = 1 - \frac{dK}{dT}\frac{dg}{dN} > 0 \tag{4.8}$$

Therefore, it is clear from the above equation that the equation $F = 0$ has a unique root say N^*, only if

$$\frac{dK}{dT}\frac{dg}{dN} < 1. \tag{4.9}$$

Hence it is clear from the above that there exit a unique root N^* in the interval $[0, K_0]$ for $F(N) = 0$. Also if condition (4.7) hold then the values N_d^*, T^* and U^* can be obtained using the value of N^* in the equation (4.3). Hence, the proposed model has existence for the given set of condition and the initial value problem has a unique solution.

2.3 Local stability analysis for the system

To understand the dynamic behavior of the proposed system, it is required to find the solution of the system. If the initial point is nearby to the equilibrium point then the motion of the trajectory path is approaching towards not only remain close enough but also eventually converge to the equilibrium. Now to determine the local stability of the model (4.3)**,** variational matrices are to be determining at the corresponding equilibrium in order to obtain the eigenvalues at the equilibrium points.

2.4 Stability at the equilibrium points E_1 & E_2

Let M_1 and M_2 be the variational matrices corresponding to the equilibrium point $E_1 = \left(0, 0, \frac{\varrho}{\delta}, 0\right)$ and $E_2 = \left(K(\tilde{T}), K(\tilde{T}), \tilde{T}, \tilde{U}\right)$ respectively then M_1 and M_2 are given as:-

$$M_1=\begin{bmatrix} r & -(\alpha+b) & 0 & 0 \\ 0 & -(\alpha+d) & 0 & 0 \\ \lambda & 0 & -\delta & 0 \\ 0 & 0 & 0 & -\beta \end{bmatrix} \&$$

$$M_2=\begin{bmatrix} d+\alpha & 0 & (\alpha+d)K'(T) & 0 \\ r_1\tilde{U}-r & -r_1\tilde{U}+\alpha+b & (d+\alpha)K'(T) & 0 \\ \lambda-\gamma\tilde{T}+v\tilde{U}\pi & 0 & -(\delta+\gamma K'(T)) & \pi v K(\tilde{T}) \\ \gamma\tilde{T}-v\tilde{T} & 0 & \gamma K(\tilde{T}) & -(\beta+vK(\tilde{T})) \end{bmatrix}$$

For the variational Matrix M_1, the eigenvalues of the matrix M_1 are r, $-(\alpha+d), -\delta, -\beta$. Since one of the eigenvalue is not negative therefore the system is unstable at E_1. It concludes that the system has saddle point and is locally stable in the direction of *N* with unstable manifold in direction of $N_d - T - U$ space. Since all the eigenvalues are not negative for matrix M_2 at E_2 and one of the eigenvalue is $-r_1\tilde{U}+\alpha+b$ which is positive, therefore the system is unstable at E_1 & E_2.

2.5 Stability at the equilibrium point E_3

We use the linearization technique to linearize the model corresponding to the equilibrium point $E_3 = (N^*, N_d^*, T^*, U^*) = \left\{K(T^*),\ \frac{r_1 U^*}{\alpha+b}K(T^*),\ T^*, U^*\right\}$ by taking the following transformation: $N = N^* + n,\ N_d = N_d^* + n_d,\ T = T^* + \tau, U = U^* + u.$ Here, $n,\ n_d, \tau$ and u are taken as small perturbations around E_3. Therefore, the model system (4.3) can be written in the terms of $n,\ n_d, \tau$ and u are as follows:-

$$\dot{X} = AX + B \tag{4.10}$$

Where, A $=\begin{bmatrix} a_{11} & a_{12} & a_{13} & 0 \\ a_{21} & a_{22} & a_{23} & a_{24} \\ a_{31} & 0 & a_{33} & a_{34} \\ a_{41} & 0 & a_{43} & a_{44} \end{bmatrix},\ X = \begin{bmatrix} n \\ n_d \\ \tau \\ u \end{bmatrix},\ B = \begin{bmatrix} b_1 \\ b_2 \\ b_3 \\ b_4 \end{bmatrix}$

Moreover,

$a_{11} = [r - 2l_1N^* + l_2N_d^*], a_{12} = l_2N^* + \alpha + b, a_{13} = l_3N^* + l_4N_d^*,\ a_{14} = 0,$

$a_{21} = r_1U^* + l_1N_d^*,\ a_{22} = -\{r_1U^* + (\alpha+d)\} + l_1N^* + 2l_2N_d^*,\ a_{23} = l_3N^* + l_4N_d^*,$

$a_{24} = r_1(N^* + N_d^*),\ a_{31} = \lambda - \gamma T^* + \pi v U^*,\ a_{32} = 0, a_{33} = -\{\delta + \gamma N^*\},$

$a_{34} = \pi v N^*,\ a_{41} = \gamma \mathrm{T}^* - v\mathrm{U}^*,\ a_{42} = 0,\ a_{43} = \gamma T^*,\ a_{44} = -\{\beta + vN^*\}$

$l_1 = \frac{-r}{K(T)},\ l_2 = \frac{(\alpha + b)}{K(T)},\ l_3 = \frac{rK'(T)}{K^2(T)},\ l_4 = \frac{-(\alpha + b)K'(T)}{K^2(T)}$

$b_1 = l_1\ n^2 + l_2\ n\, n_d + 2l_3\ N^*(\, n\,\tau + \ n^2\tau) + l_4(N^* n_d\tau + N_d^* n\,\tau + n n_d\tau)$

$b_2 = r_1(n - n_d)u + (l_1\, n + l_2 n_d)n_d + \ l_3(N_d^*\, n\,\tau + N^* n_d\tau + n n_d\tau) + l_4(2N_d^* n_d\tau + n_d^2\tau)$

$b_3 = -\gamma n\,\tau + \pi v n u\,, b_4 = \gamma n\,\tau - v n u$

Equation (4.10) is the combination of linear and non-linear part of the proposed model (4.3) respectively. Where, M_3 is a jacobian matrix corresponding to the equilibrium point E_3. Therefore, the characteristic equation of M_3 corresponding to the equilibrium point E_3 can be written as:-

$$p(x) = x^4 + B_1x^3 + B_2x^2 + B_3x + B_4 \qquad (4.11)$$

Where,

$B_1 = -(m_{11} + m_{12} + m_{13} + m_{14})$

$B_2 = m_{11}(m_{22} + m_{33} + m_{44}) + m_{22}(m_{33} + m_{44}) + m_{33}\, m_{44} - (m_{34}m_{43} + \ m_{12}m_{21} + m_{13}m_{31})$

$B_3 = (m_{33} + m_{44})(m_{12}m_{21} - m_{11}m_{22}) + (m_{11}\ + m_{22})\ + (m_{34}m_{43} - m_{33}m_{44}) - m_{12}(m_{23}m_{31} + \ m_{24}m_{41}) + m_{13}m_{31}(m_{22} + m_{44}) - \ m_{13}m_{34}m_{41.}$

$B_4 = (m_{11}m_{22} - \ m_{12}m_{21})(m_{33}\, m_{44} - m_{34}m_{43}) + m_{12}m_{24}\,(m_{41}m_{33} - \ m_{31}m_{43})(\, m_{31}m_{44} - m_{34}m_{41}\ )(m_{12}\, m_{23} - m_{13}m_{22})\,.$

Now according to Routh-Hurwitz Criterion, we can say that all the roots of the polynomial $p(x)$ are either negative or with negative real parts provided,

$$(i) B_j > 0, \quad j = 1\ to\ 4 \qquad (4.12.\mathrm{a})$$

$$(ii)\ B_1B_2\ > B_3\,, \qquad (4.12.b)$$

$$(iii) B_1B_2B_3 > B_3^2 + B_1^2B_4 \qquad (4.12.c)$$

If all the above conditions are satisfied then the equilibrium point E_3 will be locally asymptotically stable. Hence, when the reprotoxicity level of the system increases it has the ability to regain its population size.

2.6 Global stability analysis

We have found that when the motion or curve of the function starts from point very near to the equilibrium point, the trajectory of the solution not only move close to the equilibrium point but also converges towards the equilibrium . However, in the case of the global stability the initiated point may start from anywhere or from distance state but the solution of the system always converges towards the equilibrium point. For this, the Lyapunov's direct method is taken into consideration. Now, we obtained the future scope for determining the long term dynamical behavior of the solution which starts from a distant state from the equilibrium point (La-Salle and Lefschetz 1961). Lyapunov's direct method sets certain condition showing the existence of global stability of the proposed system. This shows that E_3 is globally asymptotically stable under the certain conditions.

2.7 Global stability conditions

It is not only sufficient to analyze the behavior of the system by using the linearize technique to linearize the system at the equilibrium point, for a nonlinear system to be asymptotically stable. It is also important to determine how much the system can be disturbed far away from the equilibrium point while remaining stable, so for this we determine the region of attraction or basin of attraction. The fundamental concept of the Lyapunov's directs method provide the region of attraction and establish the condition under which the system is supposed to be globally stable. The system is said to be globally stable if it must satisfied all the conditions. Since the proposed system is satisfying all the conditions therefore the system is globally stable.

Lemma 5.1:- The region of attraction or the basin of attraction or the ROA is the set of initial states from which the motion of the system converges not only close to the equilibrium point but also towards the equilibrium point. The region of attraction of the proposed system (4.3) is given as

$$\Omega = \left\{ \left(N, N_{d,} T, U\right): 0 \le N \le K_0, 0 \le N_d \le \frac{k[\lambda K_0 r_1 - (\alpha + b)\delta_m]}{(\alpha + b)\delta_m}, 0 \le \{T(t) + U(t)\} \le \frac{\lambda K_0}{\delta_m} \right\}.$$

Proof: - From the first equation of model system (4.3) we get

$$\frac{dN}{dt} = rN - \frac{rN^2}{K(T)} - \left\{1 - \frac{N}{K(T)}\right\}(\alpha + b)N_d \le rN - \frac{rN^2}{K(T)} \le rN - \frac{rN^2}{K(T)} \qquad (4.13.a)$$

Thus, $limsup_{t\to\infty} N(t) \le K_0$. similarly, from the second system of equation of model, we get

$$\frac{dN_d}{dt} = r_1 NU - r_1 N_d U - (\alpha + d)N_d - [rN - (\alpha + b)N_d]\frac{N_d}{K(T)}$$
$$\le r_1 NU - r_1 N_d U - [rN - (\alpha + b)N_d]\frac{N_d}{K(T)}$$
$$\le r_1 NU - r_1 N_d U + (\alpha + b)\frac{N_d^2}{K(T)} \qquad (4.13.b)$$

Solving the above equation and using binomial theorem we get,

Thus $limsup_{t\to\infty} N_d(t) \le \frac{k[\lambda K_0 r_1 - (\alpha + b)\delta_m]}{(\alpha + b)\delta_m}$.

Now, adding the two equations of model system we get

$$\frac{dT}{dt} + \frac{dU}{dt} = \lambda K_0 - \delta T - \gamma NT + \pi v NU + \gamma NT - \beta U - v = \lambda K_0 - \delta T - \beta U - (1 - \pi)vNU$$
$$\le \lambda K_0 - \delta_m (T + U) \qquad (4.13.c)$$

Where, $\delta_m = \min(\delta, \beta)$. Thus

$$limsup_{t\to\infty}(U + T) = \frac{\lambda K_0}{\delta_m}$$

This proves the lemma.

Theorem 5.1:- In addition to the assumptions of (4.1) and (4.3), let $K(T)$ satisfies the following inequalities in the region of attraction Ω:

$$k_m \le K(T) \le K_0, \& \ |K'(T)| \le k$$

Here k_m, K_0, k are positive constants.

If all the inequalities satisfied by the assumed system (4.3) then it is globally stable at equilibrium point E_3 under the conditions given below-

$$\left[\frac{\alpha+b}{K(T^*)}-r_1U^*+\frac{rk\{\lambda K_0r_1-(\alpha+b)\delta_m\}}{(\alpha+b)\delta_mK(T^*)}-\frac{(\alpha+b)}{N^*}\right]^2$$
$$<\frac{4}{9}\left[\frac{r}{K(T^*)}+\frac{k\{\lambda K_0r_1-(\alpha+b)\delta_m\}p}{\delta_mN_m^2}\right]\left\{r_1U^*+(\alpha+d)+\frac{rN^*}{K(T^*)}\right.$$
$$\left.-\frac{(\alpha+b)N_d^*}{K(T^*)}-\frac{k\{\lambda K_0r_1-(\alpha+b)\delta_m\}}{\delta_mK(T^*)}\right\} \quad (4.14.a)$$

$$\left[\left\{\frac{k\{\lambda K_0r_1-(\alpha+b)\delta_m\}}{\delta_m}-r\lambda K_0\right\}\frac{q}{k_m^2}-\lambda+(-\gamma+\pi v)\frac{\lambda K_0}{\delta_m}\right]^2$$
$$<\frac{4}{9}\left[\frac{r}{K(T^*)}+\frac{k\{\lambda K_0r_1-(\alpha+b)\delta_m\}p}{\delta_mN_m^2}\right][\delta+\gamma N^*] \quad (4.14.b)$$

$$\left[(\gamma-v)\frac{\lambda K_0}{\delta_m}\right]^2<\frac{4}{9}\left[\frac{r}{K(T^*)}+\frac{k\{\lambda K_0r_1-(\alpha+b)\delta_m\}p}{\delta_mN_m^2}\right][\beta+vN^*] \quad (4.14.c)$$

$$\left[\left\{\frac{-rK_0^2\{\lambda K_0r_1-(\alpha+b)\delta_m\}}{(\alpha+b)\delta_m}+(\alpha+b)N_d^{2*}\right\}\frac{q}{k_m^2}\right]$$
$$<\frac{4}{9}\left[\delta+\gamma N^*\right]\left\{r_1U^*+(\alpha+d)+\frac{rN^*}{K(T^*)}-\frac{(\alpha+b)N_d^*}{K(T^*)}\right.$$
$$\left.-\frac{k\{\lambda K_0r_1-(\alpha+b)\delta_m\}}{\delta_mK(T^*)}\right\} \quad (4.14.\mathrm{d})$$

$$\left[\frac{k\lambda K_0r_1^2}{\delta_m}\right]^2<\frac{4}{9}\left\{r_1U^*+(\alpha+d)+\frac{rN^*}{K(T^*)}-\frac{(\alpha+b)N_d^*}{K(T^*)}-\frac{k\{\lambda K_0r_1-(\alpha+b)\delta_m\}}{\delta_mK(T^*)}\right\}\{\beta+vN^*\}$$
$$(4.14.e)$$

$$[\gamma N^* + \pi v N^*]^2 < \frac{4}{9}[\delta + N^*\gamma][\beta + vN^*] \qquad (4.14.f)$$

If all the above inequalities are satisfied by the proposed model (4.3) at E_3 then the system is globally asymptotically stable i.e. for large dissipation of energy the system would remain stable.

Proof of the theorem 5.1:- We consider the following positive definite function,

$$V(N, N_{d,}T, U) = \left\{N - N^* - N^* log\frac{N}{N^*}\right\} + \frac{1}{2}(N_d - N_d^*)^2 + \frac{1}{2}(T - T^*)^2 + \frac{1}{2}(U - U^*)^2 \qquad (4.15.a)$$

Differentiating with respect to t along the system of the solution, we get

$$\frac{dV}{dt} = \left[r - (\alpha + b)\frac{N_d}{N}\right]\left[1 - \frac{N}{K(T)}\right](N - N^*) + \left[\{r_1(N - N_d)U - (\alpha + d)N_d\} - \{rN - (\alpha + b)N_d\}\frac{N_d}{K(T)}\right](N_d - N_d^*) + [\lambda N - \delta T - \gamma NT + \pi v NU](T - T^*) + [\gamma NT - \beta U - vNU](U - U^*)$$

Where,

$$\frac{dV}{dt} = -\left\{\frac{r}{K(T^*)} + (\alpha + b)N_d\xi(\mathrm{N})\right\}(N - N^*)^2 - \left\{r_1U^* + (\alpha + d) + \frac{rN^*}{K(T^*)} - \frac{(\alpha + b)(N_d + N_d^*)}{K(T^*)}\right\}(N_d - N_d^*)^2$$

$$-(\delta + \gamma N^*)(T - T^*)^2 - (\beta + vN^*)(U - U^*)^2 + \left\{\frac{\alpha + b}{K(T^*)} - r_1U^* + \frac{rN_d}{K(T^*)} - \frac{\alpha + b}{N^*}\right\}(N - N^*)(N_d - N_d^*) + \{(\alpha + b)N_d\eta(\mathrm{T}) - rN\eta(\mathrm{T}) - \gamma T + \pi vU\}(N - N^*)(T - T^*) + \{\gamma T - vU\}(N - N^*)(U - U^*) + \{-rNN_d\eta(T) + (\alpha + b)N_d^{2*}\eta(T)\}(N_d - N_d^*)(T - T^*) + \{r_1(N - N_d)\}(N_d - N_d^*)(U - U^*) + \{(\pi v + \gamma)N^*\}(T - T^*)(U - U^*)$$

(4.15. b)

$$\xi(\mathrm{N}) = \begin{cases} \dfrac{\frac{1}{N} - \frac{1}{N^*}}{N - N^*} & , \quad N \neq N^* \\ \dfrac{-1}{(N^*)^2} & , \quad N = N^* \end{cases} \quad \& \quad \eta(\mathrm{T}) = \begin{cases} \dfrac{\frac{1}{K(T)} - \frac{1}{K(T')}}{(T - T^*)}, & T \neq T^* \\ \dfrac{-K'(T)}{K^2(T)} & , T = T^* \end{cases}$$

By using mean value theorem, the inequality satisfies the following equations:

$$|\xi(\mathrm{N})| \leq p/N_m^2, |\,\eta(\mathrm{T})| \leq q/K_m^2 \ .$$

Thus, $\frac{dV}{dt}$ can be written as the sum of the quadratics,

$$\frac{dV}{dt} = -\frac{1}{2}a_{11}(N - N^*)^2 + a_{12}(N - N^*)(N_d - N_d^*) - \frac{1}{2}a_{22}(N_d - N_d^*)^2$$

$$-\frac{1}{2}a_{11}(N - N^*)^2 + a_{13}(N - N^*)(T - T^*) - \frac{1}{2}a_{33}\,(T - T^*)^2$$

$$-\frac{1}{2}a_{11}(N - N^*)^2 + a_{14}(N - N^*)(U - U^*) - \frac{1}{2}\,a_{44}(U - U^*)^2$$

$$-\frac{1}{2}a_{22}(N_d - N_d^*)^2 + a_{23}(N_d - N_d^*)(T - T^*) - \frac{1}{2}a_{33}(T - T^*)^2$$

$$-\frac{1}{2}a_{22}(N_d - N_d^*)^2 + a_{24}(N_d - N_d^*)(U - U^*) - \frac{1}{2}a_{44}(U - U^*)^2$$

$$-\frac{1}{2}a_{33}(T - T^*)^2 + a_{34}(T - T^*)(U - U^*) \ - \frac{1}{2}a_{44}(U - U^*)^2 \qquad (4.15.c)$$

Where,

$$a_{11} = \frac{2}{3}\left\{\frac{r}{K(T^*)} + (\alpha + b)N_d\xi(\mathrm{N})\right\}, a_{22} = \frac{2}{3}\left\{r_1U^* + (\alpha + d) + \frac{rN^*}{K(T^*)} - \frac{(\alpha + b)(N_d + N_d^*)}{K(T^*)}\right\},$$

$$a_{33} = \frac{2}{3}(\delta + \gamma N^*),\ a_{44} = \frac{2}{3}[\beta + vN^*],\ a_{12} = \left\{\frac{\alpha + b}{K(T^*)} - r_1U^* + \frac{rN_d}{K(T^*)} - \frac{\alpha + b}{N^*}\right\},$$

$$a_{13} = \{(\alpha + b)N_d\eta(\mathrm{T}) - rN\eta(\mathrm{T}) - \gamma T + \pi vU\}, a_{14} = [\gamma T - vU],$$

$$a_{23} = \{-rNN_d\eta\,(T) + (\alpha + b)N_d^{2*}\eta\,(T)\}, a_{24} = \{r_1(N^* - N_d^*)\}\,,\ \ a_{34}\ = \{(\pi v + \gamma)N^*\}. \tag{4.15.d}$$

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

$$a_{12}^2 < a_{11}a_{22},\ a_{13}^2 < a_{11}a_{33},\ a_{14}^2 < a_{11}a_{44},\ a_{23}^2 < a_{22}a_{33},\ a_{24}^2 < a_{22}a_{44}\ \ \&\ a_{34}^2 < a_{33}a_{44} \tag{4.15.e}$$

2.8 Numerical Simulation

Now to get long term behavior of the dynamical system of the proposed model a proprietary programming language MATLAB is used to produce various kinds of output. Here simulation of all the analytical results with the help of a computer is done by using MATCONT and MATLAB packages. This will make us provide future predictions and a wider scope of the proposed model (4.3). Now we assume the carrying capability function as

$$K(T) = K_0 - \frac{b_1 T}{1 + b_2 T}$$

The set of parameters is taken in a way such that all are positive constant. Values for the parameters are as given below;

$b = .07\,, d = .003\,,\ \ r_1 = .03\,, \alpha = .001\ \,, \delta =\ .04\,, \gamma = .0001\,, v = .001, \pi = .3, \beta = .09,\ K_0 = \ 10\,, b_1 =\ .04\,, b_2 = 1.2,\ \ \lambda = 1$

All the values are selected in such a way that the value of $\frac{b_1 T}{1 + b_2 T}$ must be less than 1.

Now let's us consider a set of parameter for which the conditions (4.12.a-4.12.b) of local asymptotic stability of interior equilibrium E_3 are satisfied. From the above the system in which the reproductive toxicant is produced by the biological species decreasing the fertility of the species itself has been found locally as well as globally stable at the equilibrium point and this has been shown by establishing Lyapunov's theorem. The following theorem characterizes the non-linear stability behavior of the equilibrium point E_3. Since the solution of the system begins in the positive octant therefore we required the region of attraction or domain which is clearly shown in Lemma.5.1. Few of the equilibrium points for the different value of environmental reproductive toxicant produced by the species are as given below:-

Table.1: Equilibrium points for different value of $\boldsymbol{\lambda}$.

$\boldsymbol{\lambda}$	N	N_d	T	U	1.4	7.9841	7.5349	274.1135	2.2341
.01	9.9752	0.1024	2.4349	0.0243	1.8	7.0325	6.6916	312.1630	2.2688
.04	9.9693	0.4089	9.7340	0.0971	2.1	7.0782	6.5283	357.5535	2.5635
.08	9.9680	0.8173	19.4655	0.1941	3	3.2823	3.2802	260.6702	0.9697
.1	9.9678	1.0217	24.3312	0.2426	6	0.0034	0.0034	0.5372	0.0021
.6	9.9669	6.1267	145.9743	1.4570	9	0.0055	0.0055	1.1810	0.0067
1	9.5384	9.0010	233.0614	2.2336	10	0.0036	0.0036	0.9498	0.0039

Fig.1 and Fig.2 shows the variation of N & N_d with an increasing rate of environmental reprotoxicity corresponding to the time (t). Its uptake increases in biological species due to which the infertility rate increases. It is concluded that initially the infertility rate in population increase due to which the deformed reproductive population increases and the total population increases with increased rate of reprotoxicity level and it attains its greatest height or threshold where it finally attains its equilibrium state. The level of attending steady-state in the deformed reproduction population decreases as the reprotoxicity level increases in species. In Fig.1 when the reproductive toxicant is in between $\boldsymbol{\lambda}$ = .001 to 1 the equilibrium level is same for all values of $\boldsymbol{\lambda}$ i.e. total population remains same but when the production rate of reprotoxin by the biological species rises beyond $\boldsymbol{\lambda}$ = 1 or higher than one then the total population decreases and starts to bifurcate. It includes two level one for which the total population is low and in seconds the total population is at high level. In Fig.2 initially the deformed reproductive population is less for $\boldsymbol{\lambda}$ = .01 but when the production rate of reproductive toxicant by the biological species rises then $\boldsymbol{\lambda}$ increases due to which the deformed reproduction population increases to its maximum threshold, again decreases to it minimum level and asymptotically oscillates as time tends to infinity. Also, Fig.3 is the plot between N and N_d, it shows the stability of dynamical behavior of the species and relative changes between N and N_d. Here, we conclude that with increasing rate of environmental reproductive toxicant of both populations, initially both increases to attain their peak value and then suddenly decreases due to an increase in the mortality rate. This rise and fall in both the population continue till both become stable and attend steady state i.e. its approaches to a solution or a point where all rate tends to zero. It is concluded that as the total

population increases the production rate of reproductive toxicant increases by the biological species due to which the deformed reproduction population increases. It is also noticed that when the total population and deformed population increase to their maximum value, the population density decreases to its minimum value due to the increasing rate of mortality. Again due to less population density, the environment is less reprotoxic and again becomes favorable for the species. This increases the total population density which in turn again increases the deformed reproduction population due to increasing reprotoxicity level. Fig. 4(a) and Fig. 4(b) Shows the plot of N and N_d w.r.t time for very high and low values of reproductive toxicant λ. For very high value of reproductive toxicant produce by the biological species, the deformed reproductive population increases and oscillates asymptotically as time tends to infinity whereas its value is less for low production rate of reproductive toxicants and vice versa in the case total population.

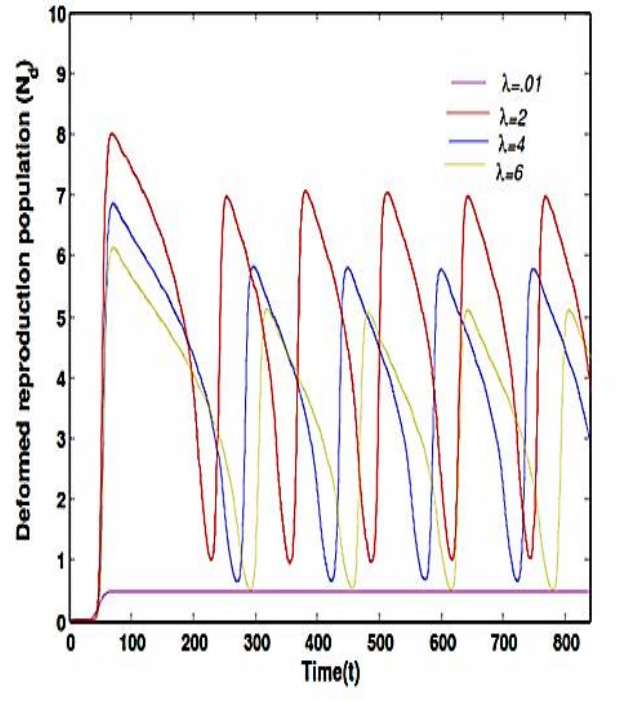

Fig.1: Time series graph for total population

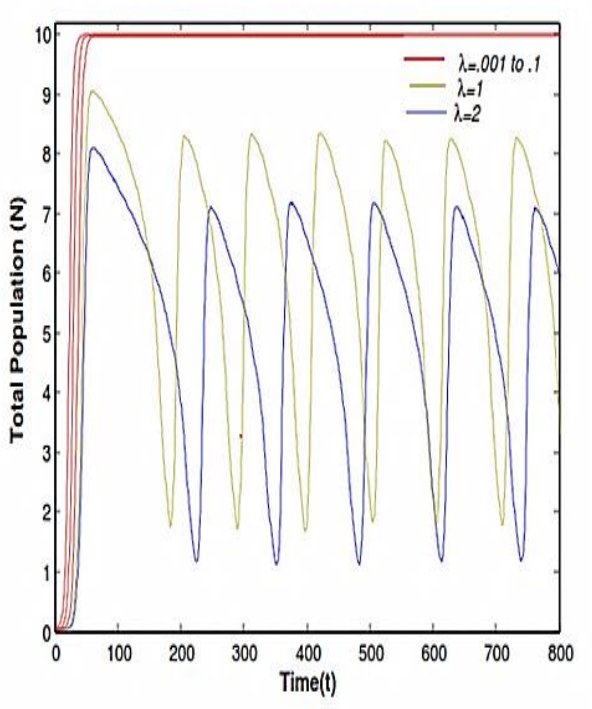

Fig.2: Time series graph of deformed reproduction population.

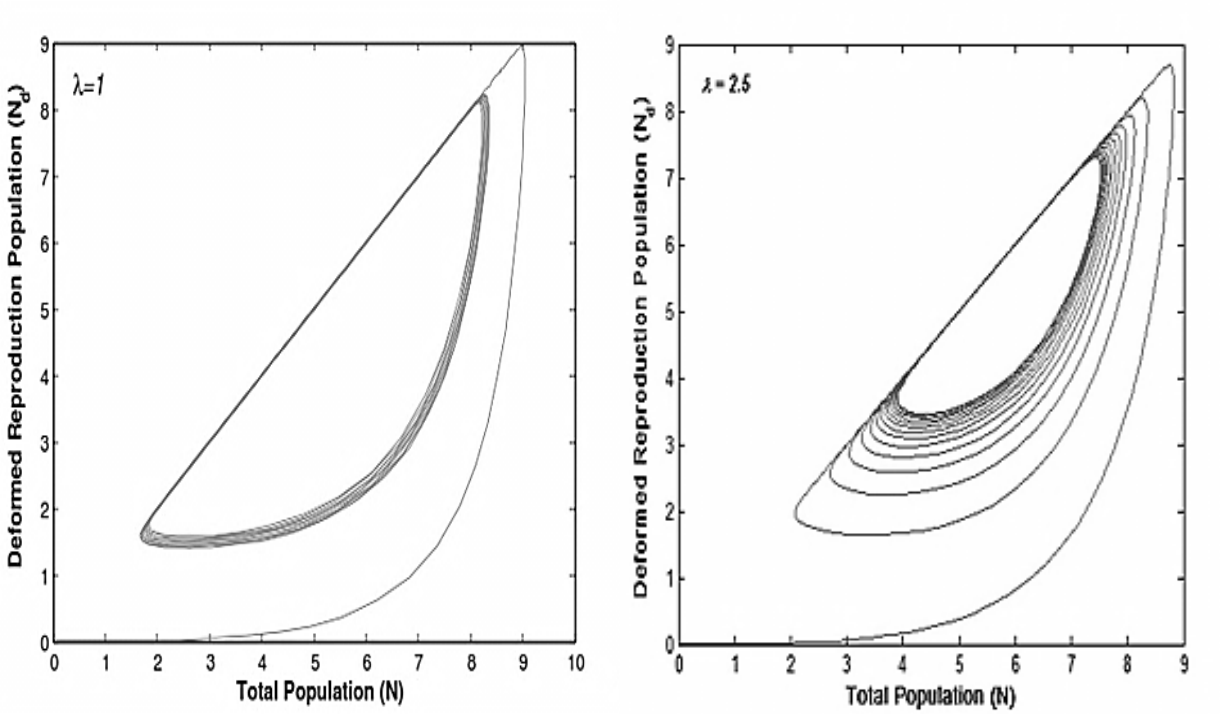

Fig. 3: Phase diagram of N versus N_d w.r.t time (t)

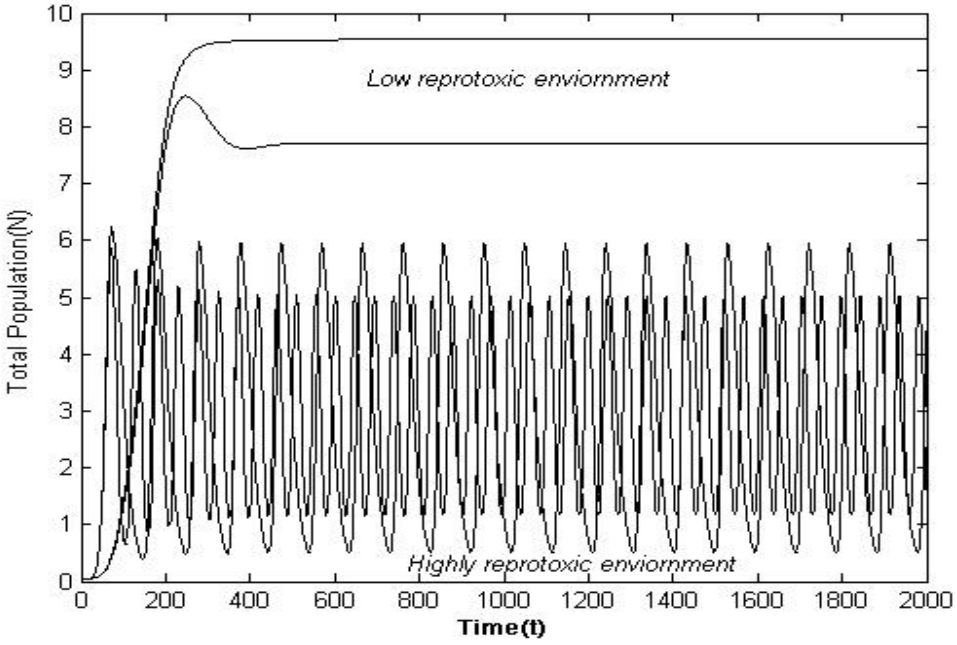

Fig.4. (a): Plot of N versus time (t) for different value of α at $\lambda = 1$ & $\lambda = 9$

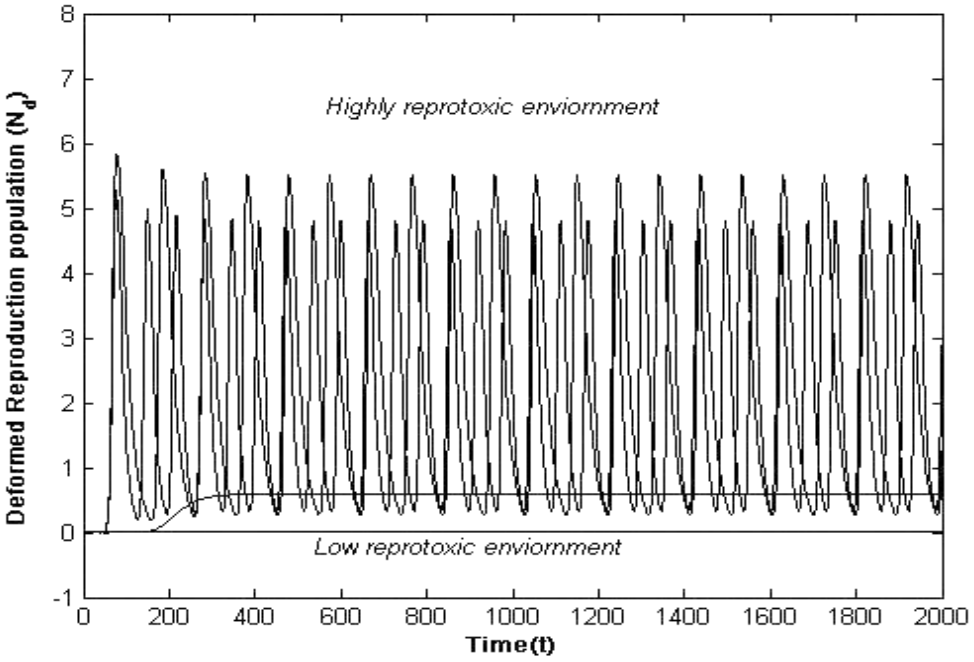

Fig.4. (b): Plot of N versus time (t) for different value of α at $\lambda = 1$ & $\lambda = 9$

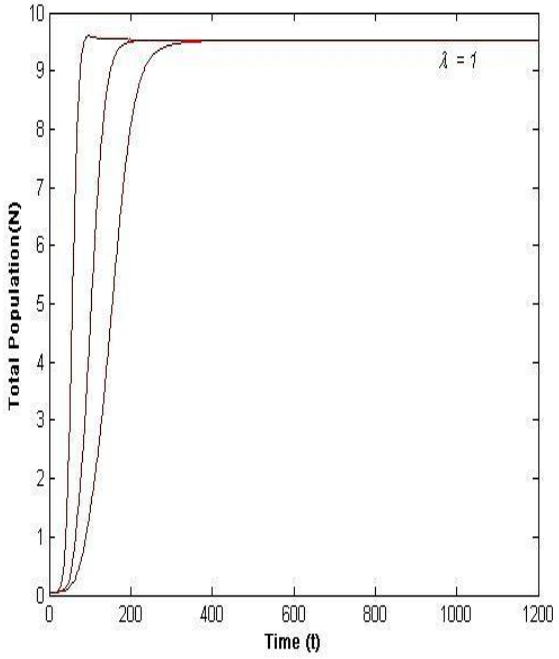

Fig.5: Plot of N versus time (t) for different value of α.

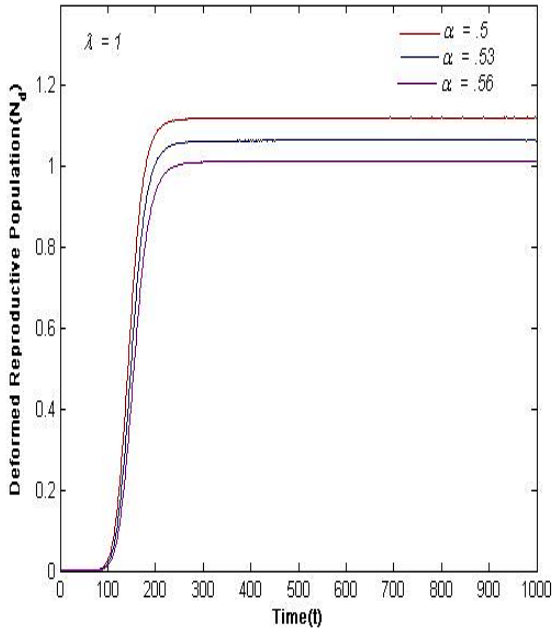

Fig.6: Plot of N_d versus time (t) for different value of α.

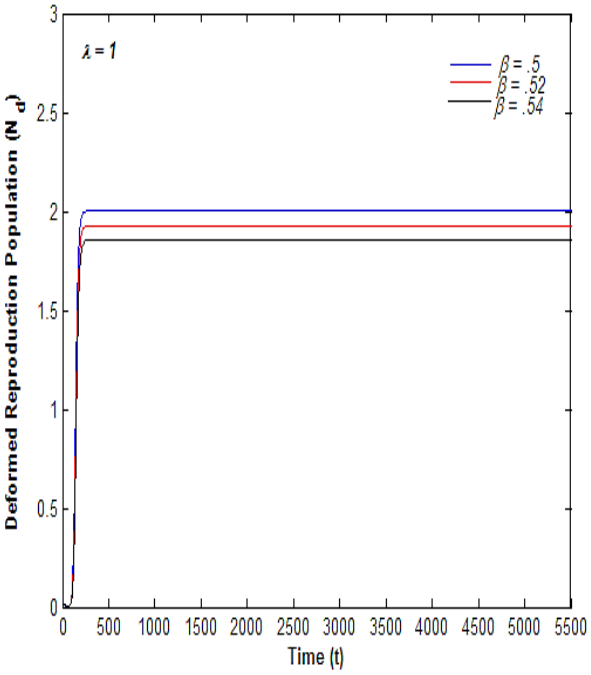

Fig.7: Plot of N_d versus Time (t) for different value of β.

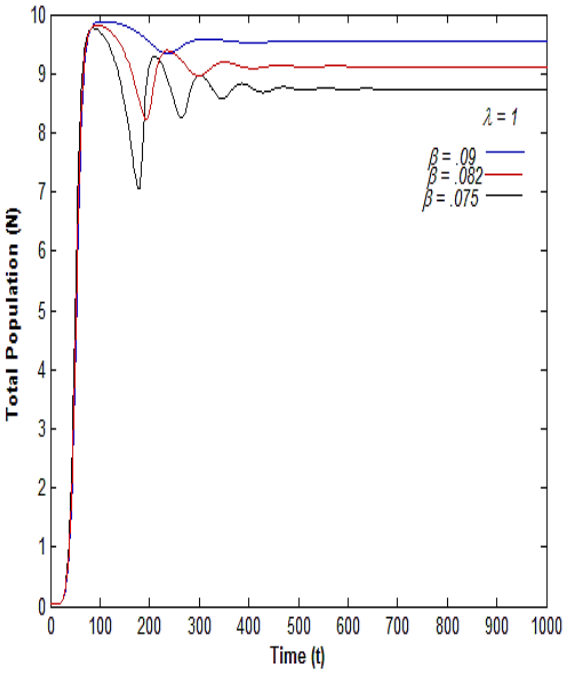

Fig.8: Plot of N versus Time (t) for different value of β.

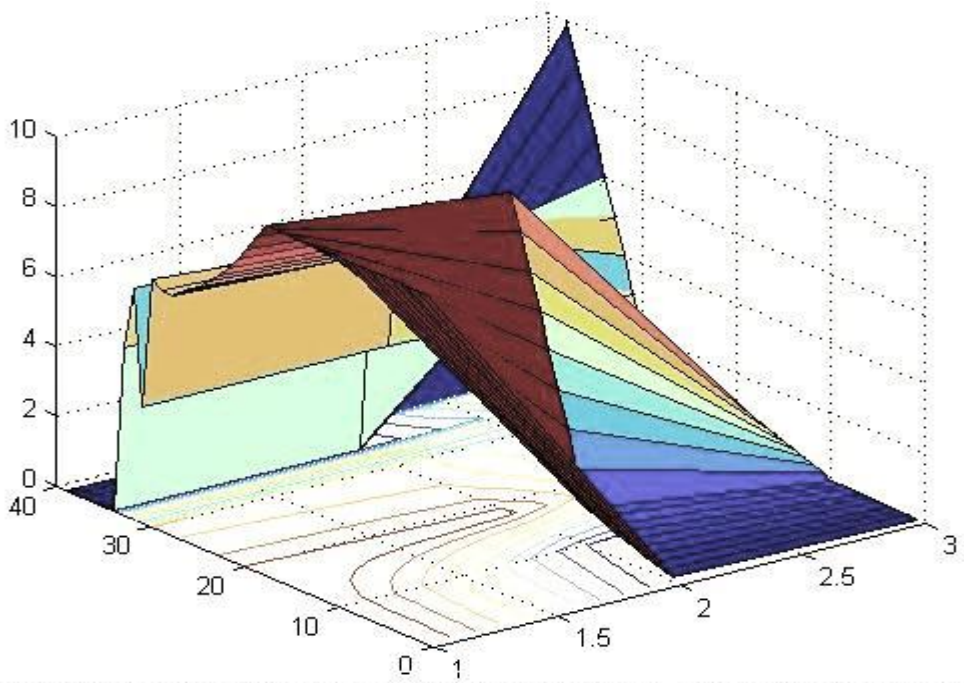

Fig.8.3D surface plot with contour of the proposed system, reproductive toxicant is z-axis.

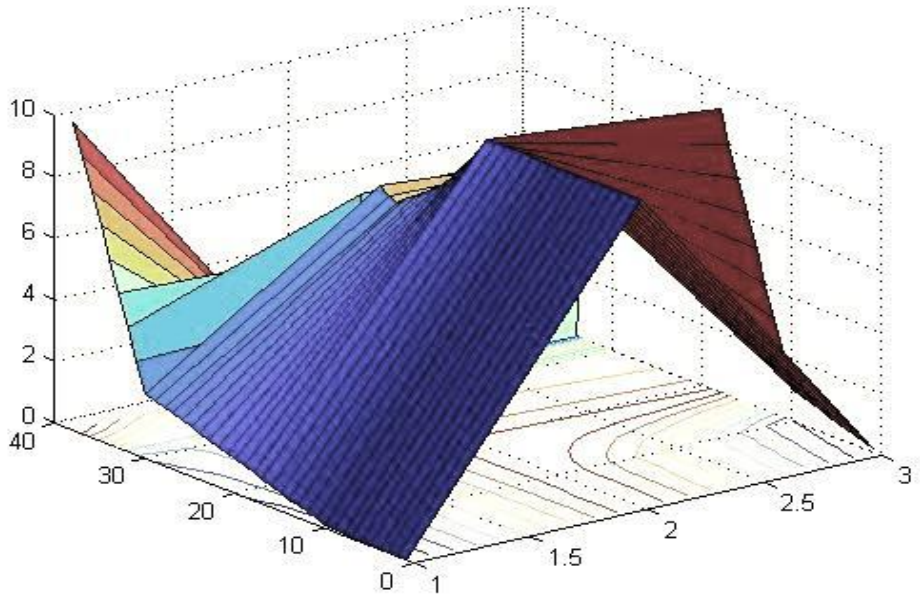

Fig.9.3D surface plot with contour of the proposed system, reproductive toxicant is x-axis

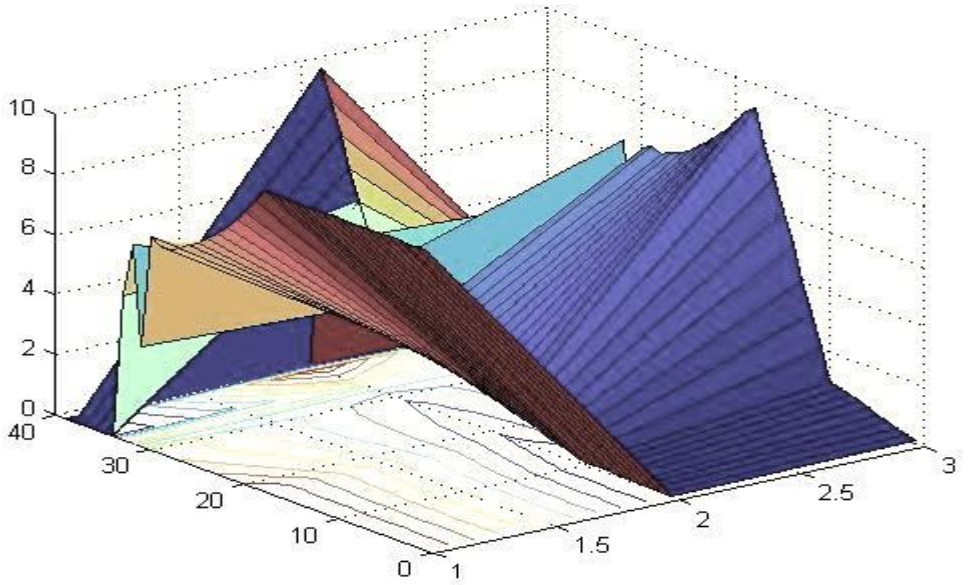

Fig.10. Surface plot with contour of the proposed system, reproductive toxicant is y-axis.

Fig.5 and Fig.6 shows the plot of N and N_d w.r.t time for different values of α. Fig.5 shows that whatever be the value of α, total population always attains the same steady state or equilibrium point. However, in the case of N_d, population-level decreases with rising rate of mortality i.e. equilibrium level of N_d decreases. Since the mortality rate increases due to the death of the

deformed reproduction population affected severely by the highly concentrated reprotoxic environment and is clearly seen in Fig.6. Fig.7 and Fig.8 shows the plot of N_d and N versus time with increasing rate of reproductive toxicant for different value of β. In Fig.7 initially the rate of N_d increases with decreased rate of reproductive toxicant and with decreasing value of β i.e. the level of equilibrium point increases with decreased value of β. However just reverse happen in the case of total population, the level of equilibrium increases with increased value of β. Fig.8, Fig.9 & Fig.10 shows the three-dimensional plot or surfaces with the contour of proposed model (4.3) in which the total population, deformed reproductive population and $\boldsymbol{\lambda}$ are the three variables. Surface plots are diagrams of three-dimensional data rather than showing the individual data points, surface plot shows a functional relationship between a designated dependent variable and two independent variables. Out of these three, two are independent variables and one is dependent variable which is representing into matrix or vector and the new surface color function is denoted by a new matrix. Z-axis specifies the color data also as surface height; therefore the color is proportional to surface height. In this plot, we use a surface with a contour to view mathematical functions over a rectangular region. Here, surface with contour creates colored parametric surfaces specified by total population, deformed reproductive population, and reproductive toxicant, with the color specified by the height of the rectangular grid. It is concluded from the above that both the population decreases as they starts to die with increasing rate of reprotoxic nature of the environment including human population. It also concludes that reproductive toxicant in one species may not be toxic in another (including humans) species because of differences in reproductive or toxicological mechanisms. Within the targeted organ, the reproductive toxicant will get interact to the critical cell or subcellular component disturbing an event important for normal reproductive function. If this toxicant interaction is not repaired, reproduction process will be altered. This reproductive toxicant effect may be specific, very affective on single function of a single cell type.

3. CONCLUSION

This paper includes a nonlinear mathematical model to determine the stability of a system in which the species is living under the effects of reproductive toxicants on the reproductive capability of various species including human beings. Since the species is producing the reproductive toxicant so the species itself gets more affected due to which the fertility rate in the species decreases, decreasing the growth rate of the total population of species. Due to this the reproductive toxicant

various lethal and sub-lethal effects are seen in the reproductive process of the biological species. This is because reproductive toxicant undergoes various mechanism structural similarity or chemically reactivity at or inside the part of reproduction system. It has been found that at positive equilibrium of the model the system is both locally as well as globally stable in a region of attraction. If the rate of reproductive toxicant surpasses the allowable level, the threshold value increases to its maximum value then decreases to attain its minimum value and attains an equilibrium point as time tends to infinity or the system becomes stable. This is because when the deformities rise to their maximum level the mortality rate increases and this decreases the total population density below the carrying capacity. Since the population density is less therefore yielding rate of reproductive toxicant would also be less and in turn its uptake is less by the species due to which the environment again becomes less reprotoxic. Thus, again the growth rate of biological species increases due to the increased rate of fertility in species. From the above, the conclusion can be drawn that the chances of extinction of species in this case are comparatively less as compared to [2] i.e. the system in which the reproductive toxicant is released by the external sources. However, when the emission of reproductive toxicant becomes very high, the situation becomes uncontrollable for the remaining biological species and they may likely to have the chances of extinction or vanishing from the environment.

CONFLICT OF INTERESTS

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

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