Mathematical Modelling of Migrated Phytoplankton Species in an Infected and Toxin Producing Phytoplankton-Zooplankton System

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ABSTRACT
In this paper the dynamical behaviour of toxin producing phytoplankton and zooplankton system is investigated. The toxin producing phytoplankton are divided into two groups: susceptible phytoplankton and infected phytoplankton. Conditions of local stability of various equilibrium points are derived. Further it is observed that the range of toxin liberation parameter increases for the coexistence of species with increased number of migratory phytoplankton species.

Keywords
Phytoplankton zooplankton system, Viral infection, Routh Hurwitz criterion, migration.

1. INTRODUCTION
Plankton are single-celled, microscopic organisms upon which almost all aquatic life is based. Phytoplankton, the plant form of plankton, are the primary producers. They are capable of photo-synthesis and stabilize environment by consuming half of the universe carbon dioxide and release huge oxygen. Zooplankton, the animal form of plankton, eat other plankton, which in turn are the basic food source for fish and other aquatic animals. The significant feature associated with many phytoplankton is rapid increase of biomass followed by their rapid decrease after some fixed time period. This kind of rapidly increased phytoplankton density is called bloom, which is of two types. One is seasonal, which occurs because of change in temperature and nutrient level of water depending on season, called 'spring bloom' and the other is localized out break associated with change in water temperature, greater salinity of water column and higher growth rates [1], known as 'red bloom'. In algae bloom of phytoplankton, each alga being short-lived result in a high concentration of dead organic matter which starts to decay. The decaying process consumes dissolved oxygen in the water, resulting in hypoxiation. In the deficiency of dissolved oxygen in the water, animals and plants may die off in mass, which in turn has adverse effects on human health, aquatic population, tourism, fisheries business, water quality and the ecosystem. Algal blooms called 'Harmful Algal Blooms' (HABs) consist of phytoplankton which have negative impact on other organisms causing mass mortality through production of natural toxins, mechanical damage to other organisms, or by other means. For the control of such problems which is under investigation, deep study of plankton system is required.

The growth and bloom of toxin producing phytoplankton species is a complex process. The toxin producing phytoplankton reduces the grazing pressure of zooplankton and may terminate the planktonic bloom. Chattopadhyay et al. [2,3] investigated that toxin producing phytoplankton and toxic substances affect the growth of zooplankton population and phytoplankton zooplankton interaction. Anuj Kumar Sharma et al. [4] have shown that time delay can destabilize the otherwise stable non-zero equilibrium state of a toxin producing phytoplankton, zooplankton and dissolved nutrient system, by inducing Hopf-bifurcation when it crosses a certain threshold value.

Viruses are the most abundant entities in the sea. These play a significant role on the survival, extinction and interaction of planktonic population. Several researchers have investigated the eco-epidemiological systems [5-11]. Beltrami and Carroll [12] investigated that a minute amount of infectious agent can de-stabilize the otherwise stable trophic configuration between a phytoplankton species and its grazer.

Anderson and May [13] concluded the existence of minimum threshold of infection below which the infected population does not persist. Sunita Gakkhar and Kuldeep Negi [14] investigated the dynamical behaviour of toxin producing phytoplankton infected by a viral disease and zooplankton system.

In this paper, a toxin producing phytoplankton zooplankton system is proposed with the assumption that some of the phytoplankton population is infected by a viral infection and there is migration of phytoplankton population at a constant rate. As seen in nature, it is assumed that infected phytoplankton population is more vulnerable to predation and TPP population do not release toxins always but release only in the presence of dense zooplankton population and for this Holling type II functional response has been considered.

2. THE MODEL
Let $P(t)$ and $Z(t)$ be the toxin producing phytoplankton (TPP) and zooplankton population respectively at time t. In the presence of virus, total TPP population is divided into two categories: susceptible phytoplankton population $S(t)$ and infected phytoplankton population $I(t)$ such that

$$P(t) = S(t) + I(t) \qquad (2.1)$$

The susceptible phytoplankton grow logistically. There is a migration of susceptible phytoplankton species at a constant rate. Infection is spread among phytoplankton population only and infection is not genetically inherited. The infected population do not recover or become immune and is not capable of reproducing. However, they can affect the growth

dynamics of the susceptible phytoplankton indirectly, for example by shading. The susceptible phytoplankton becomes infected following simple law of mass action. A simple Lotka-Volterra form of interaction is assumed for phytoplankton zooplankton populations. Using these basic assumptions the dynamics of the system can be governed by the following set of differential equations:

$$\frac{dS}{dt} = rS\left(1-\frac{S+I}{K}\right) - \lambda SI - b_1 SZ + M$$
$$\frac{dI}{dt} = \lambda SI - e_1 IZ - \delta I \qquad (2.2)$$
$$\frac{dZ}{dt} = b_2 SZ + e_2 IZ - dZ - \theta\frac{(S+I)}{(\gamma+S+I)}Z$$

Where *K*, *r* are the carrying capacity and growth rate of phytoplankton population respectively. λ is rate of infection. b_1, e_1 are the rates at which zooplankton predates susceptible and infected phytoplankton respectively. b_2, e_2 are the growth rates of zooplankton due to predation of susceptible, infected phytoplankton respectively. δ is the natural death rate of infected phytoplankton population. *d* is the mortality rate of zooplankton population due to natural death. θ is the rate of toxin liberation by the toxin producing phytoplankton (TPP) population. γ is the half-saturation constant for TPP population.

$S(0)>0$, $I(0)\geq 0$, $Z(0)>0$ are the initial conditions associated with the system *(2.2)*.

3. BOUNDEDNESS OF SOLUTION

Lemma. All the solutions of system *(2.2)* which initiate in R_+^3 are uniformly bounded for suitably chosen positive $\eta \leq \min(\delta, d)$ and $e_1 b_2 \geq b_1 e_2$.

Proof. Let us define

$$W = S + I + \frac{b_1}{b_2}Z \qquad (3.1)$$

Derivative of *(3.1)* w.r.t. time *t*, along the solution of *(2.2)* is given by

$$\frac{dW}{dt} = rS\left(1-\frac{S+I}{K}\right) - \lambda SI - b_1 SZ + M$$
$$+ \lambda SI - e_1 IZ - \delta I + b_1 SZ + \frac{b_1 e_2}{b_2} IZ$$
$$- \frac{db_1}{b_2}Z - \frac{b_1\theta}{b_2}\frac{(S+I)}{(\gamma+S+I)}Z$$

Introducing a positive number η we can write

$$\frac{dW}{dt} + \eta W \leq \frac{K}{4r}(r+\eta)^2 - (\delta-\eta)I - \frac{b_1}{b_2}(d-\eta)Z -$$
$$\left(e_1 - \frac{b_1 e_2}{b_2}\right)IZ + M$$

taking $\eta \leq \min(\delta, d)$ and $e_1 b_2 \geq b_1 e_2$, we get

$$\frac{dW}{dt} + \eta W \leq \frac{K}{4r}(r+\eta)^2 + M$$

Using theory of differential inequality, we obtain

$$0 < W < \frac{K}{4r\eta}(r+\eta)^2\left(1-e^{-\eta t}\right) + \left(W_0 - \frac{M}{\eta}\right)e^{-\eta t}$$

where $W_0 = S_0 + I_0 + \frac{b_1}{b_2}Z_0 > 0$

For $t \to \infty$, we have

$$0 < W < \frac{K}{4r\eta}(r+\eta)^2$$

Hence all the solutions of equations *(2.2)* which initiate in R_+^3 are confined in the region given by

$$\left\{(S,I,Z)\in R_+^3 : W = \frac{K}{4r\eta}(r+\eta)^2 + \varepsilon, \varepsilon > 0\right\}.$$

4. EQUILIBRIUM POINTS

Equilibrium point for the system *(2.2)* are given by

(1) Equilibrium point on the boundary of first octant $E_1 = \left(\frac{rK + \sqrt{r^2K^2 + 4rKM}}{2r}, 0, 0\right)$

(2) Planer equilibrium point on *S-Z* plane is $E_2 = (S', 0, Z')$, where

$$S' = \frac{-(b_2\gamma - d - \theta) + \sqrt{(b_2\gamma - d - \theta)^2 + 4db_2\gamma}}{2b_2}$$

$$Z' = \frac{r}{b}\left(1-\frac{S'}{K}\right) + \frac{M}{S'b_2}$$

For its existence, the necessary and sufficient condition is

$$\theta < \left[b_2\left(K + \frac{M}{r}\right) + b_2\gamma - d\right] - \frac{d\gamma}{K + \frac{M}{r}}$$

In case the phytoplankton is not releasing any toxin, the corresponding equilibrium point $(S_0, 0, Z_0)$ is obtained by

taking $\theta = \gamma = 0$. Thus

$$S_0 = \frac{d}{b_2}, Z_0 = \frac{1}{b_1}\left(r - \frac{rd}{Kb_2} + \frac{Mb_2}{d}\right)$$

Clearly $S' > S_0$ and $Z' < Z_0$. Thus toxin production in phytoplankton increases the equilibrium value of phytoplankton and decreases the equilibrium value of zooplankton.

(3) Planer equilibrium point on *S-I* plane is $E_3 = (\bar{S}, \bar{I}, 0)$, where

$$\bar{S} = \frac{\delta}{\lambda}, \ \bar{I} = \frac{r\delta(K\lambda - \delta) + MK\lambda^2}{\lambda\delta(r + K\lambda)}$$

For existence of E_3, $\bar{I} > 0$, which is possible if $\bar{S} < K + \frac{M}{r}$.

(4) The non-trivial equilibrium point is $E^*(S^*, I^*, Z^*)$ where S^* is the positive root of fourth degree equation:

$$AS^{*4} + BS^{*3} + CS^{*2} + DS^* + E = 0 \quad (4.1)$$

where

$$A = \lambda K(e_1 - b_1)(e_1 r(b_2 - e_2) + \lambda K(e_1 b_2 - e_2 b_1))$$

,

$$B = e_1(r + \lambda K)\begin{bmatrix} e_1(b_2\gamma - d - \theta)(r + \lambda K) + \\ K(b_2 + e_2)(e_1 r + b_1\delta) \end{bmatrix} -$$

$$(e_1 r + b_1\lambda K)\begin{bmatrix} e_1(r + \lambda K)(e_2\gamma - d - \theta) \\ + 2e_2 K(e_1 r + b_1\delta) \end{bmatrix}$$

$$C = e_1(r + \lambda K)\begin{bmatrix} -d\gamma e_1(r + \lambda K) + \\ MKe_1(b_2 + e_2) \\ K(e_2\gamma - d - \theta)(e_1 r + b_1\delta) \end{bmatrix} +$$

$$e_2 K\begin{bmatrix} K(e_1 r + b_1\delta)^2 - \\ 2Me_1(re_1 + b_1\lambda K) \end{bmatrix}$$

$$D = MKe_1\begin{bmatrix} e_1(r + \lambda K)(e_2\gamma - d - \theta) \\ + 2Ke_2(e_1 r + b_1\delta) \end{bmatrix}$$

$$E = M^2K^2e_1^2e_2$$

It is always possible to write *(4.1)* in at least one way, as the product of product of two quadratic forms with real coefficients

$$f(S^*) = (S^{*2} + p_1 S^* + q_1)(S^{*2} + p_2 S^* + q_2) = 0$$

where

$$p_1 + p_2 = \frac{B}{A}$$

$$p_1 p_2 + q_1 + q_2 = \frac{C}{A}$$

$$p_1 q_2 + p_2 q_1 = \frac{D}{A}$$

$$q_1 q_2 = \frac{E}{A}$$

The roots of $f(S^*) = 0$ take the form

$$S^* = \frac{-p_1 \pm \sqrt{p_1^2 - 4q_1}}{2},$$

$$\frac{-p_2 \pm \sqrt{p_2^2 - 4q_2}}{2}$$

The equation in S^* admits at least one positive root in the following cases:

(i) $p_1 < 0, q_1 < 0$

(ii) $p_1 < 0, q_1 < 0, p_1^2 - 4q_1 > 0$

(iii) $p_1 > 0, q_1 < 0$

or

(i) $p_2 < 0, q_2 < 0$

(ii) $p_2 < 0, q_2 < 0, p_2^2 - 4q_2 > 0$

(iii) $p_2 > 0, q_2 < 0$

There will not exist positive S^* in the following cases

(i) $p_1^2 - 4q_1 < 0$

(ii) $p_1 > 0, q_1 > 0, p_1^2 - 4q_1 > 0$

(iii) $p_2^2 - 4q_2 < 0$

(ii) $p_2 > 0, q_2 > 0, p_2^2 - 4q_2 > 0$

I^* is given by:

$$I^* = \frac{K(e_1 r + b_1\delta.)S^* + MKe_1 - (re_1 + b_1 K\lambda)(S^*)^2}{e_1(r + \lambda K)S^*} > 0$$

and

$$Z^* = \frac{\lambda S^* - \delta}{e_1} > 0$$

In case phytoplankton population do not produce toxins, then corresponding equilibrium $\hat{E}=(\hat{S},\hat{I},\hat{Z})$ can be obtained by substituting $\theta=\gamma=0$.

As

$$b_2S^*+e_2I^*-d-\theta\left(\frac{S^*+I^*}{\gamma+S^*+I^*}\right)=0$$

and

$$b_2\hat{S}+e_2\hat{I}-d=0$$

Thus

$$b_2S^*+e_2I^*-d-\theta\left(\frac{S^*+I^*}{\gamma+S^*+I^*}\right)=b_2\hat{S}+e_2\hat{I}-d$$

$$\Rightarrow b_2S^*+e_2I^*>b_2\hat{S}+e_2\hat{I}$$

$$\Rightarrow S^*>\hat{S} \text{ whenever } \hat{I}>I^*$$

and

$I^*>\hat{I}$ whenever $\hat{S}>S^*$.

Further

$$S^*>\hat{S}\Rightarrow Z^*>\hat{Z}$$

and

$$S^*<\hat{S}\Rightarrow Z^*<\hat{Z}$$

5. STABILITY ANALYSIS

We will now discuss the local behaviour of the system around each of the equilibrium points. Variational matrix around the point $E(S,I,Z)$ is given by

$$V(S,I,Z)=\begin{bmatrix} r-\frac{2rS}{K}-\frac{rI}{K} & \frac{-rS}{K}-\lambda S & -b_1S \\ -\lambda I-b_1Z & & \\ \lambda I & \lambda S-e_1Z-\delta & -e_1I \\ b_2Z- & e_2Z- & b_2S+e_2I-d- \\ \frac{\theta\gamma Z}{(\gamma+S+I)^2} & \frac{\theta\gamma Z}{(\gamma+S+I)^2} & \frac{\theta(S+I)}{(\gamma+S+I)} \end{bmatrix}$$

The variational matrix for E_1 is

$$V_1=\begin{bmatrix} r-\frac{2rS_1}{K} & -\frac{rS_1}{K}-\lambda S_1 & -b_1S_1 \\ 0 & \lambda S_1-\delta & 0 \\ 0 & 0 & b_2S_1-d-\frac{\theta S_1}{\gamma+S_1} \end{bmatrix}$$

It has eigen values $\mu_1=-\frac{\sqrt{r^2K^2+4rKM}}{K}$, $\mu_2=\lambda S_1-\delta$ and $\mu_3=b_2S_1-d-\frac{\theta S_1}{\gamma+S_1}$. Thus E_1 is locally asymptotically stable provided $S_1<\frac{\delta}{\lambda}$ and $b_2S_1^2+(b_2\gamma-d-\theta)S_1<d\gamma$. If $S_1<\frac{\delta}{\lambda}$, then E_3 and E^* do not exist. Hence existence of E_3 and E^* implies that E_1 is unstable saddle point.

The variational matrix for E_2 is given by $V_2=$

$$\begin{bmatrix} -\frac{M}{S'}-\frac{rS'}{K} & -\lambda S'-\frac{rS'}{K} & -bS' \\ 0 & \lambda S'-e_1Z'-\delta & 0 \\ \left[b_2-\frac{\gamma\theta}{(\gamma+S')^2}\right]Z' & \left[e_2-\frac{\gamma\theta}{(\gamma+S')^2}\right]Z' & 0 \end{bmatrix}$$

The characteristic equation for E_2 is

$$[\mu-(\lambda S'-e_1Z'-\delta)]\begin{bmatrix} \mu^2+\left(\frac{M}{S'}+\frac{rS'}{K}\right)\mu+ \\ b_1S'Z'\left(b_2-\frac{\gamma\theta}{(\gamma+S')^2}\right) \end{bmatrix}=0$$

Since E_2 exists and $\frac{\gamma}{\gamma+S'}<1$, thus $b_2-\frac{\gamma\theta}{(\gamma+S')^2}>0$. This means that both the roots of the quadratic factor are negative. The third eigen value is $\mu_3=\lambda S'-e_1Z'-\delta=\lambda\left(S'-\frac{\delta}{\lambda}\right)-e_1Z'$.

Thus the third eigen value will be negative if $S'<\frac{\delta}{\lambda}$. Hence E_2 will be locally asymptotically stable. However E_2 can be locally asymptotically stable even if $S'>\frac{\delta}{\lambda}$, provided $Z'>\frac{\lambda S'-\delta}{e_1}>0$ and unstable otherwise.

The variational matrix for E_3 is

$$V_3 = \begin{bmatrix} -\frac{r\bar{S}}{K}-\frac{M}{\bar{S}} & -\frac{r\bar{S}}{K}-\lambda\bar{S} & -b_1\bar{S} \\ \lambda\bar{I} & 0 & -e_1\bar{I} \\ 0 & 0 & b_2\bar{S}+e_2\bar{I}-d-\frac{\theta(\bar{S}+\bar{I})}{(\gamma+\bar{S}+\bar{I})} \end{bmatrix}$$

Its characteristic equation is given by

$$\left(\mu - b_2\bar{S} - e_2\bar{I} + d + \frac{\theta(\bar{S}+\bar{I})}{(\gamma+\bar{S}+\bar{I})}\right)\left[\mu^2 + \left(\frac{r\bar{S}}{K}+\frac{M}{\bar{S}}\right)\mu + \lambda\bar{I}\left(\lambda\bar{S}+\frac{r}{K}\bar{S}\right)\right] = 0$$

The quadratic equation gives negative eigen values. Thus E_3 is locally asymptotically stable if the third eigen value given by $\mu_3 = b_2\bar{S} - e_2\bar{I} + d + \frac{\theta(\bar{S}+\bar{I})}{(\gamma+\bar{S}+\bar{I})}$ is negative, otherwise it will be unstable in the direction of *Z*.

The variational matrix for E^* is given by

$$V^* = \begin{bmatrix} -\left(\frac{rS^*}{K}+\frac{M}{S^*}\right) & -\left(\frac{rS^*}{K}+\lambda S^*\right) & -bS^* \\ \lambda I^* & 0 & -e_1 I^* \\ \left[b_2 Z^* - \frac{\theta\gamma Z^*}{(\gamma+S^*+I^*)^2}\right] & \left[e_2 Z^* - \frac{\theta\gamma Z^*}{(\gamma+S^*+I^*)^2}\right] & 0 \end{bmatrix}$$

The characteristic equation is given by

$$\mu^3 + A_1\mu^2 + A_2\mu + A_3 = 0,$$

where

$$A_1 = \frac{r}{K}S^* + \frac{M}{S^*}$$

$$A_2 = \begin{bmatrix} e_1\left(e_2 - \frac{\theta\gamma}{(\gamma+S^*+I^*)^2}\right)I^*Z^* + \\ b_1\left(b_2 - \frac{\theta\gamma}{(\gamma+S^*+I^*)^2}\right)S^*Z^* + \\ \left(\lambda^2 + \frac{\lambda r}{K}\right)S^*I^* \end{bmatrix}$$

$$A_3 = \left\{\lambda\left[\begin{matrix} b_1\left(e_2 - \frac{\theta\gamma}{(\gamma+S^*+I^*)^2}\right) - \\ e_1\left(b_2 - \frac{\theta\gamma}{(\gamma+S^*+I^*)^2}\right) \end{matrix}\right] + \frac{re_1(e_2-b_2)}{K}\right\}S^*I^*Z^* + \frac{eM}{S^*}\left(e_2 - \frac{\theta\gamma}{(\gamma+S^*+I^*)^2}\right)I^*Z^*$$

Applying Routh Hurwitz criterion, E^* is locally asymptotically stable provided the following conditions are satisfied

$A_1, A_2, A_3 > 0$ and $A_1A_2 > A_3$.

i.e.

$$e_2 - \frac{\theta\gamma}{(\gamma+S^*+I^*)^2} > \frac{e_1}{b_1}\left(b_2 - \frac{\theta\gamma}{(\gamma+S^*+I^*)^2}\right) > 0$$

and

$$b_2 r > (\lambda K + r)\frac{\theta\gamma}{(\gamma+S^*+I^*)^2}.$$

6. CONCLUSIONS

In this paper, the dynamical behaviour of toxin producing phytoplankton and zooplankton system with migration of toxin producing phytoplankton population under the assumption that some of phytoplankton are infected by viral infection, has been considered. Infected phytoplankton are more vulnerable to predation as seen in nature. Conditions of local stability of coexistence of three species are established. It is observed that the range of toxin liberation parameter increases for the coexistence of species with increase in number of migratory phytoplankton species. There is a lot of scope for future research.

Some directions for future research are as below :

1. One can consider the case in which the disease affects the zooplankton population also.
2. The incubation period of the disease can also be considered.
3. One can consider the case of migration of infected phytoplankton population.
4. In this paper it is assumed that the predated phytoplankton are converted into zooplankton instantaneously, although it seems obvious that some delay should be accounted for. One can investigate the role of the delay.

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8. REFERENCES

[1] J.E. Truscott, J. Brindley, Ocean plankton populations as excitable media. Bull. Math. Biol., 56 (1994).

[2] J. Chattopadhyay, R.R. Sarkar, S. Mandal, Toxin producing phytoplankton may act as a biological control for planktonic blooms-field study and mathematical modeling, J. Theor. Biol. 215 (2002) 333-344.

[3] J. Chattopadhyay, R.R. Sarkar, A. El Abdllaoui, A delay differential equation model on harmful algal blooms in the presence of toxic substances, IMA J. Math. Appl. Med. Biol. 19 (2002) 137-161.

[4] A.K. Sharma, A. Sharma, K. Agnihotri, Bifurcation Alalysis of a Plankton Model with Discrete Delay, International journal of Mathematics, Computational Science and Engineering, 8 (2014) 77-86.

[5] J. Chattopadhyay, N. Bairagi, Pelicans at risk in Salton sea- an eco-epidemiological model, J. Ecological Modelling 136 (2001) 103-112.

[6] J. Chattopadhyay, R. R. Sarkar, G. Ghosal, Removal of infected prey prevent limit cycle oscillations in an infected prey-predator system-a mathematical study, J. Ecological Modelling, 156 (2002) 113-121.

[7] Helbert W. Hethcote et. al., A predator-prey model with infected prey, J. Theoretical Population Biology 66 (2004) 259-268.

[8] Pierre Auger et al., Effects of a disease affecting a predator on the dynamics of a predator-prey system, J. Theoretical Biology, 258 (2009) 344-351.

[9] Chiara Tannoia, Emiliano Torre, Ezio Venturino, An incubating diseased-predator ecoepidemic model, J. Biological Physics 38 (2012) 705-720.

[10] Krishna pada Das et al., A predator-prey mathematical model with both the populations affected by diseases, J. Ecological Complexity 8 (2011) 68-80.

[11] Krishna Pada Das, J. Chattopadhyay, A mathematical study of a predator-prey model with disease circulating in the both populations, J. Biomathematics, 8 (2015) 1550015-1 to 1550015-27.

[12] E. Beltrami, T.O. Carroll, 1994. Modelling the role of viral disease in recurrent phytoplankton blooms. J. Math. Biol. 32 (1994) 857–863.

[13] R.M Anderson, R.M. May, Infectious Diseases of Humans, Dynamics and Control. Oxford University Press, Oxford (1991).

[14] S. Gakkhar , K. Negi, A mathematical model for viral infection in toxin producing phytoplankton and zooplankton system, J. Applied Mathematics and Computation 179 (20v06) 301–313.