

# Impact of harvesting on infected and toxin producing phytoplankton-zooplankton model

Kulbhushan Agnihotri<sup>1</sup> and Harpreet Kaur<sup>2,3</sup>

<sup>1</sup> Department of Applied Sciences & Humanities,

Shaheed Bhagat Singh State Technical Campus, Ferozepur, Punjab, India

<sup>2</sup> Research Scholar, I.K. Gujral Punjab Technical University, Kapurthala, Punjab, India

<sup>3</sup> Department of Applied Sciences, Lala Lajpat Rai Instt. of Engg. and Tech., Moga, Punjab, India

**Abstract** This paper aims to study the role of harvesting of phytoplankton population on the dynamical behaviour of toxin producing phytoplankton and zooplankton system. The toxin producing phytoplankton population is divided into two groups: susceptible phytoplankton population and infected phytoplankton population. Conditions of local stability of various equilibrium points are derived. Further it is observed that harvesting helps to reduce the outbreak of disease in phytoplankton population.

**Keywords:** harvesting, infection, local stability, toxin producing phytoplankton, zooplankton

## I. Introduction

Ecology and epidemiology are the research fields which are treated separately. But there are some common features between these fields and interacting species may suffer from various diseases and thus merging these fields may help to study the dynamics of such systems. Eco-epidemiology is the branch of science which studies both the ecological and epidemiological issues simultaneously.

Plankton are single-celled, microscopic organisms upon which almost all aquatic life is based. Phytoplankton, the plant form of plankton, are the primary producers, 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: **Spring bloom** and **Red bloom**. Spring bloom is seasonal and it occurs because of change in temperature and nutrient level of water depending on season. Red bloom is localized outbreak associated with change in water temperature and with greater salinity of water column and higher growth rates [1]. 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 and cause mass mortality of animals and plants. This mass mortality 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 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] have 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, interaction of planktonic population. Several researchers have investigated the eco-epidemiological systems [5]-[13]. Sunita Gakkhar and Kuldeep Negi [14] investigated the dynamical behaviour of toxin producing phytoplankton infected by a viral disease and zooplankton system.

Reasonable harvesting policy is one of the important and interesting problems in ecology and economics. The exploitation of biological resources and harvesting of population species are commonly practiced in fishery, forestry, agriculture and wildlife management. Harvesting has sometimes been considered as a stabilizing factor [15], a destabilizing factor [16] or even an oscillation-inducing factor [17].

In this paper, we propose a toxin producing phytoplankton zooplankton system with the assumption that some of the phytoplankton population is infected by a viral infection and there is harvesting of susceptible and infected phytoplankton population. It is assumed that infected phytoplankton population is more vulnerable to predation.

## II. The model

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

$$P(t) = S(t) + I(t) \quad (1)$$

The susceptible phytoplankton grow logistically. Infection is spread among phytoplankton population only. 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. The phytoplankton liberates toxins instantaneously. The effect of toxin liberation decreases the growth of zooplankton according to Holling Type I functional response. There is harvesting of phytoplankton population. Using these basic assumptions the dynamics of the system can be governed by the following set of differential equations:



$$\begin{aligned} \frac{dS}{dt} &= rS\left(1 - \frac{S+I}{K}\right) - cSI - bSZ - q_1ES \\ \frac{dI}{dt} &= cSI - eIZ - \delta I - q_2EI \\ \frac{dZ}{dt} &= gSZ + hIZ - dZ - \theta(S+I)Z \end{aligned} \tag{2.2}$$

Where  $K, r$  are the carrying capacity and growth rate of phytoplankton population respectively.  $c$  is rate of infection.  $b, e$  are the rates at which zooplankton predate susceptible and infected phytoplankton respectively.  $g, h$  are the growth rates of zooplankton due to predation of susceptible, infected phytoplankton respectively.  $\delta$  is the natural death rate of infected phytoplankton population.  $d$  is mortality rate of zooplankton population due to natural death.  $\theta$  is the rate of toxin liberation by the toxin producing phytoplankton (TPP) population.  $\gamma$  is the half-saturation constant for TPP population.  $q_1, q_2$  are the catchability coefficients of susceptible and infected phytoplankton populations, respectively.  $E$  is the combined external effort devoted to non-selective harvesting of susceptible and infected phytoplankton population by the external harvester. All the parameters are assumed to be positive.

$S(0) > 0, I(0) \geq 0, Z(0) > 0$  are the initial conditions associated with the system (1).

**III. Boundedness of solution**

**Lemma.** All the solutions of system (1) which initiate in  $R_+^3$  are uniformly bounded for suitably chosen positive  $\eta \leq \min\{\delta + q_2E, d\}$  and  $eg \geq bh$ .

**Proof.** Let us define

$$W = S + I + \frac{b}{g}Z \tag{2}$$

Derivative of (2) w.r.t. time  $t$ , along the solution of (1) is given by

$$\frac{dW}{dt} = rS\left(1 - \frac{S+I}{K}\right) - cSI - bSZ - q_1ES + cSI - eIZ - \delta I - q_2ES + bSZ + \frac{bh}{g}IZ - \frac{bd}{g}Z - \frac{b\theta}{g}(S+I)Z$$

Introducing a positive number  $\eta$  we can write

$$\frac{dW}{dt} + \eta W \leq \frac{K}{4r}(r + \eta - q_1E)^2 - (\delta - \eta + q_2E) - \frac{b}{g}(d - \eta)Z - \left(e - \frac{bh}{g}\right)IZ$$

taking a positive number  $\eta \leq \min\{\delta + q_2E, d\}$  and  $eg \geq bh$ , we get

$$\frac{dW}{dt} + \eta W \leq \frac{K}{4r}(r + \eta - q_1E)^2$$



Using theory of differential inequality, we obtain

$$0 < W < \frac{K}{4r\eta} (r + \eta - q_1 E)^2 (1 - e^{-\eta t}) + W_0 e^{-\eta t}$$

where  $W_0 = S_0 + I_0 + \frac{b}{g} Z_0 > 0$

For  $t \rightarrow \infty$ , we have

$$0 < W(S, I, Z) < \frac{K}{4r\eta} (r + \eta - q_1 E)^2$$

Thus all the solutions of equations (1) that initiate in  $R_+^3$  are confined in the region given by

$$\left\{ (S, I, Z) \in R_+^3 : W = \frac{K}{4r\eta} (r + \eta - q_1 E)^2 + \varepsilon, \varepsilon > 0 \right\}$$

**IV. Equilibrium points**

Equilibrium point for the system (1) are given by

(1) Trivial Equilibrium point  $E_0 = (0,0,0)$ .

(2) Equilibrium point on the boundary of the first octant  $E_1 = \left( K \left( 1 - \frac{q_1 E}{r} \right), 0, 0 \right)$ , where  $E < \frac{r}{q_1}$ .

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

$$\bar{S} = \frac{\delta + q_2 E}{c}, \bar{I} = \frac{r(Kc - (\delta + q_2 E)) - q_1 EKc}{c(r + Kc)}, \text{ where } Kc > \frac{q_1 EKc}{r} + \delta + q_2 E.$$

(4) Another equilibrium point  $E_3(S', 0, Z')$  on the S-Z plane is obtained as

$$S' = \frac{d}{g - \theta}, Z' = \frac{K(r - q_1 E)(g - \theta) - rd}{bK(g - \theta)}$$

$$g > \theta, K(r - q_1 E)(g - \theta) > rd$$

(5) The non-trivial equilibrium point  $E^*(S^*, I^*, Z^*)$ , where

$$K(h - \theta)[e(r - q_1 E) + b(\delta + q_2 E)]$$

$$S^* = \frac{-ed(r + Kc)}{(h - \theta)(re + bKc) - e(g - \theta)(r + cK)} > 0$$



$$I^* = \frac{d - (g - \theta)S^*}{h - \theta} > 0 \text{ and } Z^* = \frac{cS^* - \delta - q_2E}{e} > 0$$

$$\frac{\delta + q_2E}{c} < S^* < \frac{d}{g - \theta}$$

**V. Spread of disease**

The basic reproductive ratio for the system (2.2) is given by  $R_0 = \frac{cS}{\delta + q_2E}$ . This ratio gives the number of new infected cases arising from the introduction of unit infected phytoplankton species into the susceptible phytoplankton population. From this ratio we can find that whether the infection will spread or die off. The infection will persist is  $R_0 > 1$ , otherwise the infection will die out. From the above calculated value of  $R_0$ , it is observed that the basic reproductive ratio is directly proportional to the host density and is inversely proportional to the harvesting. Thus harvesting enhances the critical host density required for the onset of disease and this critical value increases with the effort  $E$ . Thus when harvesting process is employed, the system becomes able to support higher number of susceptible phytoplankton population. Thus harvesting process helps to reduce the outbreak of disease.

**VI. Stability analysis**

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

$$V(S, I, Z) = \begin{bmatrix} X & \frac{-rS}{K} - cS & -bS \\ cI & cS - eZ - \delta - q_2E & -eI \\ (g - \theta)Z & (h - \theta)Z & Y \end{bmatrix}$$

where

$$X = r - \frac{2rS}{K} - \frac{rI}{K} - cI - bZ - q_1E$$

$$Y = gS + hI - d - \theta(S + I).$$

The variational matrix for  $E_0$  is

$$V_0 = \begin{bmatrix} r - q_1E & 0 & 0 \\ 0 & -\delta - q_2E & 0 \\ 0 & 0 & -d \end{bmatrix}$$



The eigenvalues for  $V_0$  are  $r - q_1E, -\delta - q_2E, -d$ , which show that  $E_0$  will be stable if  $E$ , the effort level satisfies

$E > \frac{r}{q_1}$ , otherwise  $E_0$  will be a point and stable in I-Z direction but unstable in S direction.

The variational matrix for  $E_1$  is

$$V_1 = \begin{bmatrix} r - \frac{2r}{K}X_1 & -\left(\frac{r}{K} + c\right)X_1 & -bX_1 \\ 0 & cX_1 - \delta - q_2E & 0 \\ 0 & 0 & -d - (\theta - g)X_1 \end{bmatrix}$$

Where  $X_1 = K\left(1 - \frac{q_1E}{r}\right)$ . Eigen values are  $\mu_1 = -r + 2q_1E$ ,  $\mu_2 = cX_1 - (\delta + q_2E)$  and  $\mu_3 = -d - (\theta - g)X_1$ .

Therefore  $E_1$  is locally asymptotically stable provided  $E < \frac{r}{2q_1}$ ,  $cX_1 < \delta + q_2E$  and  $\theta - g > 0$ . Also if

$\theta - g > 0$  then  $E_3$  and  $E^*$  do not exist. Therefore existence of  $E_3$  and  $E^*$  implies that  $E_1$  is unstable.

The variational matrix for  $E_2$  is

$$V_2 = \begin{bmatrix} -\frac{r\bar{S}}{K} & -\frac{r\bar{S}}{K} - c\bar{S} & -b\bar{S} \\ c\bar{I} & 0 & -e\bar{I} \\ 0 & 0 & \bar{Y} \end{bmatrix}$$

where  $\bar{Y} = g\bar{S} + h\bar{I} - d - \theta(\bar{S} + \bar{I})$ .

And the characteristic equation is given by

$$(\lambda - \bar{Y})\left[\lambda^2 + \left(\frac{r\bar{S}}{K}\right)\lambda + c\left(c + \frac{r}{K}\right)\bar{S}\bar{I}\right] = 0$$

The quadratic equation gives negative eigenvalues. The third eigen value is  $\lambda_3 = \bar{Y}$ . Hence  $E_2$  is locally asymptotically stable for  $\lambda_3 < 0$ , and unstable in the direction of Z if  $\lambda_3 > 0$ .

The variational matrix for  $E_3$  is

$$V_3 = \begin{bmatrix} -\frac{rS'}{K} & -cS' - \frac{rS'}{K} & -bS' \\ 0 & cS' - eZ' - \delta - q_2E & 0 \\ (g - \theta)Z' & (h - \theta)Z' & 0 \end{bmatrix}$$

The and the corresponding characteristic equation is



$$[\lambda - (cS' - eZ' - \delta - q_2E)] \times$$

$$\left[ \lambda^2 + \left( \frac{rS'}{K} \right) \lambda + b(g - \theta)S'Z' \right] = 0$$

As  $g > \theta$ , the roots the roots of the quadratic factor are negative. The third eigen value  $\lambda_3 = cS' - eZ' - \delta - q_2E$  will be negative if  $S' < \frac{\delta + q_2E}{c}$  and then the equilibrium point  $E_3$  will be locally asymptotically stable and under this

condition  $E^*$  does not exist. However for  $S' > \frac{\delta + q_2E}{c}$ ,  $E_3$  can be locally asymptotically stable provided  $Z' > \frac{cS' - \delta - q_2E}{e} > 0$ .

The variational matrix for  $E^*$  is

$$V^* = \begin{bmatrix} -\left(\frac{rS^*}{K}\right) & -\left(\frac{rS^*}{K} + cS^*\right) & -bS^* \\ cI^* & 0 & -eI^* \\ (g - \theta)Z^* & (h - \theta)Z^* & 0 \end{bmatrix}$$

The characteristic equation is given by

$$\lambda^3 + A_1\lambda^2 + A_2\lambda + A_3 = 0$$

Where,  $A_1 = \frac{r}{K}S^*$ ,  $A_2 = e(h - \theta)I^*Z^* + b(g - \theta)S^*Z^* + \left(c^2 + \frac{cr}{K}\right)S^*I^*$  and

$$A_3 = \left[ \frac{re}{K}(h - g) + c\{b(h - \theta) - e(g - \theta)\} \right] S^*I^*Z^*$$

By applying Routh Hurwitz criterion,  $E^*$  is locally asymptotically stable provided the following conditions are satisfied:

$$A_1, A_2, A_3 > 0 \text{ and } A_1A_2 > A_3$$

i.e.

$$g - \theta > 0, b(h - \theta) > e(g - \theta), \frac{gr}{K} > \left(c + \frac{r}{K}\right)\theta$$

$$0 < \frac{cgK}{r + cK} < g - \theta < \frac{b(h - g)}{e - b}.$$



## VII. Conclusions

In this paper, we have investigated the effect of harvesting of phytoplankton population on the dynamical behaviour of toxin producing phytoplankton and zooplankton system. It is assumed that some of phytoplankton species are infected by viral diseases and thus dividing the total phytoplankton population into two parts, namely susceptible phytoplankton population and infected phytoplankton population. The Infected phytoplankton are more vulnerable to predation. The resulting three dimensional mathematical model has been studied for local stability of various equilibrium points. It is observed that harvesting helps to reduce the outbreak of disease in phytoplankton species.

## Acknowledgment

The authors are thankful to the anonymous reviewers for their suggestions to improve the quality of the paper. We are also thankful to the editor for his/her helpful comments. Further authors acknowledge the I.K. Gujral Punjab Technical University, Kapurthala, Punjab for providing research support.

## 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 Analysis 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*. Wiley online Library 1992, Vol 28.
- [14] S. Gakkhar , K. Negi, A mathematical model for viral infection in toxin producing phytoplankton and zooplankton system, *J. Applied Mathematics and Computation* 179 (2006) 301–313.
- [15] B.S. Goh, G. Leitmann, T.L. Vincent, Optimal control of a prey–predator system, *Math. Biosci.* 190 (1974) 263.
- [16] J.P. Cohn, Saving the Salton Sea, *Bioscience* 50 (4) (2000) 295.
- [17] N. Jonzen, E. Ranta, P. Lundberg, V. Kaitala, H. Linden, Harvesting induced fluctuations?, *Wildlife Biol* 9 (2003) 59.
- [18] J. L´opez-Pujol, *The Importance of Biological Interactions in The Study of Biodiversity*. InTech., Croatia, 2011.
- [19] J. N. Kapur, *Mathematical modelling*. New Age International, 1988.
- [20] N. Eldredge, *Life on Earth: AG. ABC-CLIO*, 2002, vol. 1.
- [21] A. Stevens, “Predation, herbivory, and parasitism,” *Nature Education Knowledge*, vol. 3, pp. 1–6, 2012.
- [22] E. Venturino, “Epidemics in predator-prey models: disease in the predators”, *Mathematical Medicine and Biology*, vol. 19, no. 3, pp. 185–205, 2002.
- [23] K. P. Haderl and H. Freedman, “Predator-prey populations with parasitic infection,” *Journal of mathematical biology*, vol. 27, no. 6, pp. 609–631, 1989.
- [24] F. M. Hilker and K. Schmitz, “Disease-induced stabilization of predator-prey oscillations,” *Journal of Theoretical Biology*, vol. 255, no. 3, pp. 299–306, 2008.
- [25] G. T. Evans and J. S. Parslow, “A model of annual plankton cycles,” *Biological Oceanography*, vol. 3, no. 3, pp. 327–347, 1985.
- [26] S. Busenberg, S. K. Kumar, P. Austin, and G. Wake, “The dynamics of a model of a plankton-nutrient interaction,” *Bulletin of Mathematical Biology*, vol. 52, no. 5, pp. 677–696, 1990.
- [27] A. Huppert, B. Blasius, and L. Stone, “A model of phytoplankton blooms,” *The American Naturalist*, vol. 159, no. 2, pp. 156–171, 2002.
- [28] T. G. Hallam, “Structural sensitivity of grazing formulations in nutrient controlled plankton models,” *Journal of Mathematical Biology*, vol. 5, no. 3, pp. 269–280, 1978.
- [29] A. M. Edwards and J. Brindley, “Oscillatory behaviour in a three-component plankton population model,” *Dynamics and stability of Systems*, vol. 11, no. 4, pp. 347–370, 1996.
- [30] J. Chattopadhyay, R. R. Sarkar, and S. Pal, “Dynamics of nutrient-phytoplankton interaction in the presence of viral infection,” *Bio-systems*, vol. 68, no. 1, pp. 5–17, 2003.
- [31] S. Pal, S. Chatterjee, and J. Chattopadhyay, “Role of toxin and nutrient for the occurrence and termination of plankton bloom-results drawn from field observations and a mathematical model,” *Bio Systems*, vol. 90, no. 1, pp. 87–100, 2007.
- [32] S. Khare, O. Misra, and J. Dhar, “Role of toxin producing phytoplankton on a plankton ecosystem,” *Nonlinear Analysis: Hybrid Systems*, vol. 4, no. 3, pp. 496–502, 2010.
- [33] S. Chakraborty, S. Roy, and J. Chattopadhyay, “Nutrient-limited toxin production and the dynamics of two phytoplankton in culture media: a mathematical model,” *Ecological Modelling*, vol. 213, no. 2, pp. 191–201, 2008.