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# Role of toxin producing phytoplankton for coexistence of planktonic ecosystem

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# Role of toxin producing phytoplankton for coexistence of planktonic ecosystem

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Abstract: Effect of toxin producing plankton and its control is an intriguing problem in marine plankton ecology. In this paper we have proposed a three component model consisting of a non-toxic phytoplankton (NTP), toxin producing phytoplankton (TPP) and zooplankton (Z), where the growth of zooplankton species reduce due to toxic chemicals released by phytoplankton species. The nature of the dynamics of the system is observed around the positive equilibrium. Conditions for co-existence or extinction of populations are derived. It is observed that the three components persist if the predation rate of zooplankton population on toxic phytoplankton is bounded in certain regions. It is observed that when toxin is liberated by the toxin producing phytoplankton then stability zone increases. To validate the analytical results, numerical experiments and field collected sample observations are also presented. Our analytical as well as numerical study reveals the essential mathematical features regarding the role of TPP in phytoplankton-zooplankton interaction.

Key-Words: Toxin producing plankton (TPP), Zooplankton, Bloom, Predational response function, Biological control

# 1. Introduction

Plankton are the basis of all aquatic food chain and phytoplankton in particular occupy the first trophic level. Phytoplankton do huge services for our earth: food for marine life, oxygen for human life and also absorb half of the carbon dioxide which may be contributing to global warming [1]. There has been global increase in harmful plankton in last two decades ( [2],[3]) and considerable scientific attention towards harmful plankton has been paid in recent years.

The adverse effects of harmful plankton species on human health, commercial fisheries, subsistence fisheries, recreational fisheries, tourism and coastal recreation, ecosystem and environment are well established. Nevertheless, despite the attention towards this issue, the nature of harmful plankton and its possible control mechanism are not yet well established and required special attention. Hence the experimental as well as mathematical modelling is necessary in this field.

Recent studies reveal that some times bloom of certain harmful species leads to release of both toxins and allelopathic substances. Allelopathic substances are distinguished from phycotoxins in being secondary metabolites; both can co-occur within a given harmful species. Allelopathic substances tend to be directly targeted and may physiologically impair, stun, repel, induce avoidance reactions, and kill grazers. Information on the recently discovered, remarkable ambush predatory behavior of the harmful dinoflagellate, *Pfiesteria piscicida* was presented by [4],[5].

In the present study we will give special emphasis to the fact that occurrence of toxin producing phytoplankton may not always harmful but it may help to maintain the stable equilibrium in trophodynamics through coexistence of all the species. In the present study we shall put our emphasis to observe the effects of toxin producing plankton in the dynamics of non-toxic phytoplankton-toxic phytoplankton- zooplankton interaction. The model that we propose has three interacting components consisting of the non-toxic phytoplankton (NTP), herbivorous zooplankton (Z) and toxin producing phytoplankton (TPP) with an additional factor that the release of toxic substance by toxic-phytoplankton species reduces the growth of zooplankton.

To establish the theoretical results we shall use our field observations. We consider the toxin producing plankton (TPP) species as *Noctiluca scintillans*, non-toxic phytoplankton (NTP) species as *Coscinodiscus sp.* and zooplankton species as *Paracalanus sp.* (the taxonomical and functional distinctions of all the species are given in [6],[7]). Our field study suggests that when TPP is absent the equilibrium level of NTP is much lower than the value observed when TPP is present. Moreover the equilibrium level of zooplankton decreases when TPP is present. This phenomenon depicts the fact that the presence of TPP in the system enhances the production of NTP and reduces the zooplankton grazing pressure. Further, the biomass distribution observed in our field study demonstrate that introduction of TPP leads to the persistence of all the species through reduction of blooms and can be used as controlling agent for stability of marine ecosystem.

The remainder of this paper is organized as follows: In Section 2, we have discussed the experiment and findings of experimental results. In Section 3, we have proposed a mathematical model consisting of non-toxic phytoplankton, toxic- phytoplankton and zooplankton. In section 4, we have studied some basic results such as boundedness, local stability of the equilibria, persistence, etc. of the system. Section 5 contains the general discussion of the paper.

#### 2. Experiment and findings of Experimental results

Monitoring of plankton population was carried out since 1999 in the North-West coast of Bay of Bengal. As we are interested to report the effect of artificial eutrophication on the Non-toxic phytoplankton, toxin producing plankton (TPP) and zooplankton population with the help of field observation. We choose *Noctiluca scintillans* as TPP, non-toxic phytoplankton species (NTP) as *Coscinodiscus sp.* and *Paracalanus sp.* Zooplankton for this study. The materials and method of the study have been discussed elaborately in [6],[7]. In this paper we will consider the data of the field observation from March, 1999 to January, 2002 (30 sample collection dates). To establish our theoretical realization we shall mainly focus on the dynamics of NTP-TPP-Zooplankton system in

different time frames. A series of data have been observed to explore the possible types of response that might be exhibited by NTP, TPP and Zooplankton. We observed that in our total study time when TPP is absent then the possible patterns of NTP is much lower than the pattern observed in presence of TPP (see, Fig 1 and Fig 2).







Figure 2: Biomass distribution in presence of Toxin Producing Phytoplankton

Moreover, the pattern observed for zooplankton population depicts the phenomenon (see, Fig 1 and Fig 2). Again equilibrium analysis is useful in that if identifies the effect of

inclusion of some species in real food chain models. To study this we observe the total biomass accumulation over the whole time period of our field study. We observe that there is about 18.2 percent increase in NTP biomass accumulation whereas about 49.7 percent decrease in zooplankton biomass (see, Table 1).

Total Biomass accumulation of NTP when TPP is absent	Total Biomass accumulation of NTP when TPP is present	Percentage increase of NTP population	Total Biomass accumulation of Zooplankton when TPP is absent	Total Biomass accumulation of Zooplankton when TPP is present	Percentage decrease of Zooplankton population
7093662	8672027	18.2%	4025436	2023230	49.7%

Table 1: Comparison of biomass accumulation in the presence and absence of TPP

Motivated from the literature and our field observations, a dynamic model consisting of non-toxic phytoplankton, toxin producing phytoplankton and zooplankton has been proposed and the role of toxic phytoplankton in the termination of planktonic blooms have been observed.

# 3. The mathematical model

Let  $P_1(t)$  be the concentration of the non-toxic phytoplankton at time t. Let  $P_2(t)$  and Z(t) be the concentration of toxic phytoplankton population and zooplankton respectively at time t. Let r be the growth rate of non-toxic phytoplankton and K be the carrying capacity. Let  $\alpha$  and  $\beta$  be the maximum zooplankton ingestion rate and maximum zooplankton conversion rate respectively. Let  $\mu$  be the death rate of zooplankton. Let s be the growth rate toxic phytoplankton. Let  $\theta$  be the rate of toxin liberation by the toxic phytoplankton. Let  $\theta_1$  be the specific predation rate of zooplankton population on toxic phytoplankton.

The mathematical model is:

$$\frac{dP_1}{dt} = rP_1\left(1 - \frac{P_1}{K}\right) - \alpha P_1 Z$$

$$\frac{dP_2}{dt} = sP_2\left(1 - \frac{P_2}{K}\right) - \frac{\theta P_2 Z}{\gamma + P_2}$$

$$\frac{dZ}{dt} = \beta P_1 Z - \mu Z - \frac{\theta_1 P_2 Z}{\gamma + P_2}$$
(1)

For sake of simplicity, we put in dimensionless form the model equations (1) by rescaling the variables on the carrying capacity value K, i.e.,

$$p_1 = \frac{P_1}{K}, p_2 = \frac{P_2}{K}, p_3 = \frac{P_3}{K}$$
(2)

and then using as dimensionless time,  $\tau = \alpha K t$ , we get the following dimensionless equations

$$\frac{dp_1}{d\tau} = ap_1(1-p_1) - p_1 z$$

$$\frac{dp_2}{d\tau} = bp_2(1-p_2) - \frac{cp_2 z}{1+p_2 d}$$

$$\frac{dz}{d\tau} = ep_1 z - fz - \frac{gp_2 z}{1+p_2 d}$$
(3)

where 
$$a = \frac{r}{\alpha K}, b = \frac{s}{\alpha K}, c = \frac{\theta}{\alpha \gamma}, d = \frac{K}{\gamma}, e = \frac{\beta}{\alpha}, f = \frac{\mu}{K\alpha}, g = \frac{\theta_1}{\alpha \gamma}$$
 (4)

System (3) has to be analyzed with the following initial conditions:  $p_1(0) \ge 0, p_2(0) \ge 0, z(0) \ge 0$  (5)

For convenience in the following, time  $\tau$  is replaced by t as the dimensionless time.

## 4. Some basic results

#### 4.1. Boundedness of the System:-

Theorem 1: All the solutions of (3) are ultimately bounded

The proof is obvious.

### 4.2. Equilibria:

The system (3) possesses the following equilibria:~ the plankton free equilibrium  $E_0=(0,0,0)$ , the toxic phytoplankton and zooplankton free equilibrium  $E_1(1, 0,0)$ , non-toxic phytoplankton and zooplankton free equilibrium  $E_2(0, 1,0)$ , a feasible zooplankton free equilibrium  $E_3(1,1,0)$ , a feasible toxic-phytoplankton free equilibrium  $E_4(\frac{f}{e}, 0, a(1-\frac{f}{e}))$ . The existence criterion of  $E_4$  is  $\beta K > \mu$ . The positive equilibrium  $E^* = (p_1^*, p_2^*, z^*)$  where  $z^* = a(1-p_1^*), p_2^* = \frac{ep_1^* - f}{g + fd - ep_1^*d}$  and  $p_1^*$  satisfies the relation  $Ap_1^{*3} + Bp_1^{*2} + Cp_1^* + D = 0$ , where  $A = cae^2d^2, B = -cae^2d^2 - 2ca(g + fd)ed, C = -bge(1+d) + 2ca(g + fd)ed + ca(g + fd)^2, D = bg(g + fd + f) - ca(g + fd)^2$ .

### 4.3. Existence of Positive Interior Equilibrium:

Now we observe that the positive interior equilibrium  $E^*$  exists if  $L_1 < \theta < L_2$  (6)

where 
$$L_1 = \frac{s\theta_1\beta(\gamma+K)\alpha\gamma}{(2\beta K+\theta_1+\mu)r(\theta_1+\mu)}, L_2 = \frac{s\theta_1\alpha\gamma(K\theta_1+\mu K+\mu\gamma)}{Kr(\theta_1+\mu)^2}$$
  
and  $M_1 < p_1^* < M_2$  where  $M_1 = \frac{f}{e}, M_2 = \max\{1, \frac{g}{ed} + \frac{f}{e}\}$ .

#### 4.4. Eigenvalue analysis to establish local asymptotic stability:

By computing the variational matrix around the respective biological feasible equilibria, one can easily deduce the following lemmas:-

**Lemma 1**. The steady state  $E_0=(0,0,0)$  of the system (3) is a saddle point.

**Lemma 2**. There exists a feasible toxic phytoplankton and zooplankton free steady state  $E_1=(1,0,0)$  which is unstable saddle.

**Lemma 3**. There exists a non-toxic phytoplankton and zooplankton free steady state  $E_2=(0,1,0)$  which is unstable saddle.

**Lemma 4.** There exists a zooplankton free steady state  $E_3=(1,1,0)$  which is unstable saddle in the direction orthogonal to  $p_1 - p_2$  coordinate plane.

**Theorem 2:** If the inequality 
$$\frac{\alpha\beta\gamma Ks}{r(\beta K - \mu)} > \theta$$
 (7)

hold then the toxic phytoplankton free steady state  $E_4(\frac{f}{e}, 0, a(1 - \frac{f}{e}))$  is unstable.

**Theorem 3:** If the inequality 
$$\theta_1 < \frac{\gamma(\beta K - 2\mu)}{K}$$
 (8)

hold then all the three species will persist.

The variational matrix of system (3) around the positive equilibrium  $E^* = (p_1^*, p_2^*, z^*)$ 

$$V^{*} = \begin{bmatrix} -ap_{1}^{*} & 0 & -p_{1}^{*} \\ 0 & -bp_{2}^{*} - \frac{cz^{*}}{(1+p_{2}^{*}d)^{2}} + \frac{cz^{*}}{(1+p_{2}^{*}d)} & \frac{-cp_{2}^{*}}{(1+p_{2}^{*}d)} \\ ez^{*} & \frac{-gz^{*}}{(1+p_{2}^{*}d)^{2}} & 0 \end{bmatrix}$$

To consider the local stability analysis of the positive equilibrium  $E^*$  as  $\theta_1 < \frac{\gamma(\beta K - 2\mu)}{K}$ , we recall that the stability properties of  $E^*$  depend on the toxic phytoplankton  $p_1^*$ , which we shall rename as  $\xi = p_1^*$  and further we obtain the following inequality of  $\xi$  as

$$\min[\frac{be}{a^2d}, \frac{fd + gd + fd^2 - g}{ed(d+1)}, \frac{M - \sqrt{M^2 - 4LN}}{2L}] < \xi < \frac{M + \sqrt{M^2 - 4LN}}{2L}$$
(9)

For positive equilibrium  $E^* = (p_1^*, p_2^*, z^*)$ , the characteristic equation is  $x^3 + Q_1(5)x^2 + Q_2(5)x + Q_2(5) = 0$ , where the coefficients  $Q_1(5)x = 12.3$ 

$$\eta^{*} + Q_{1}(\xi)\eta^{2} + Q_{2}(\xi)\eta + Q_{3}(\xi) = 0, \text{ where the coefficients } Q_{i}(\xi), i = 1, 2, 3 \text{ are}$$

$$Q_{1}(\xi) = ap_{1}^{*} + bp_{2}^{*} - \frac{cz^{*}p_{2}^{*}d}{(1+p_{2}^{*}d)^{2}}, Q_{2}(\xi) = ap_{1}^{*}(bp_{2}^{*} - \frac{cz^{*}p_{2}^{*}d}{(1+p_{2}^{*}d)^{2}}) - \frac{gcz^{*}p_{2}^{*}}{(1+p_{2}^{*}d)^{3}} + p_{1}^{*}ez^{*},$$

$$Q_{3}(\xi) = p_{1}^{*}ez^{*}(bp_{2}^{*} - \frac{cz^{*}p_{2}^{*}d}{(1+p_{2}^{*}d)^{2}}) - \frac{ap_{1}^{*}gcz^{*}p_{2}^{*}}{(1+p_{2}^{*}d)^{3}}$$

Denote 
$$A_1(\xi) = bp_2^* - \frac{cz^* p_2^* d}{(1+p_2^* d)^2}$$
. Applying  $b(1-p_2^*) = \frac{cz^*}{1+p_2^* d}$ ,  $ep_1^* - f = \frac{gp_2^*}{1+p_2^* d}$ 

We get  $Q_1(\xi) = a\xi + A_1(\xi), Q_2(\xi) = a\xi A_1(\xi) - \frac{cgz^*p_2^*}{(1+p_2^*d)^3} + p_1^*ez^*$ 

Now 
$$Q_3(\xi) = \frac{ab\xi(e\xi - f)}{(1 + p_2^*d)(g + fd - ed\xi)^2} [-L\xi^2 + M\xi - N]$$
 where  $L = 2de^2(d + 1)$ ,

 $M = ed(3g + 3f + 3fd + e + ed), N = (g + fd)^2 + f(g + fd) - eg + efd + edg + efd^2.$ Since d>1, it is obvious that L>0,M>0 and N>0.

Also we observe that  $Q_1(\xi) > 0, Q_3(\xi) > 0$  if conditions (8) and (9) are satisfied. Now let us define

$$\delta^{2}(\xi) = Q_{1}(\xi)Q_{2}(\xi) - Q_{3}(\xi) = A_{1}(\xi)\xi[a^{2}\xi - \frac{be}{d}] + a\xi A_{1}^{2}(\xi) + a\xi^{2}ez^{*} + \frac{gA_{1}^{2}(\xi)}{d(1+dp_{2}^{*})} + \frac{A_{1}(\xi)bf}{d}$$

Obviously  $\delta^2(\xi) > 0$  by conditions (8) and (9).

Therefore according to Routh-Hurwitz criterion  $E^*$  is locally asymptotically stable.

### 5. Discussion

In this paper we have proposed and analyzed a three component model consisting of NTP, TPP and zooplankton. We have studied the stability behavior of the system around the feasible steady states. We proved that the three components persist if the predation rate of zooplankton population on toxic phytoplankton is bounded in certain regions. Our theoretical results show that for a certain threshold of the system parameters, the system possesses asymptotic stability around the positive interior equilibrium depicting the coexistence of all the three species.

Moreover we observe that when TPP is absent then the system possesses the Equilibrium

 $E_4(\frac{f}{a}, 0, a(1-\frac{f}{a}))$  and this exist provided  $\beta K > \mu$ . But introduction of TPP in the system give the equilibrium  $E^*$  which exists provided  $L_1 < \theta < L_2$  and  $M_1 < p_1^* < M_2$ where  $M_1 = \frac{f}{e}$ ,  $M_2 = \max\{1, \frac{g}{ed} + \frac{f}{e}\}$ . It is clear from an analytical study that the level of equilibrium value for NTP increases due to presence of TPP. Further for the zooplankton population we observe that when TPP is absent then the equilibrium value for zooplankton population is  $z^* = a(1 - \frac{f}{a})$  but when TPP is present then the equilibrium value of satisfies the inequality  $a(1 - \max\{1, \frac{g}{ed} + \frac{f}{e}\}) < z^* < a(1 - \frac{f}{e})$ . Hence presence of TPP decreases the bloom of zooplankton population. The above of the observations clearly demonstrate the influence of TPP in the equilibrium level of NTP and zooplankton population, which also supports our field observation. To substantiate the analytical findings, a set of hypothetical parameter values have been considered for numerical simulation as  $r = 4.5, K = 10, \alpha = 0.068, s = 0.1, \theta = 0.02, \gamma = 0.5, \beta = 0.08, \mu = 0.7, \theta_1 = 0.005$ . For these set of parameter values we observe that the TPP free equilibria as  $E_4(0.875,0,0.827)$  but the triply coexisting equilibria is  $E^* = (p_1^*, p_2^*, z^*)$  where  $0.875 < p_1^* < 1$ and  $0 < z^* < 0.827$ .Our numerical results also shows the coexistence of all the species ( a stable situation, see figure 3).



Figure 3: Stable situation for inclusion of toxin producing phytoplankton depicting coexistence of all the species

#### References

[1] Duinker, J. and Wefer, G. Das CO<sub>2</sub>-Problem und die Rolle des Ozeans. *Naturwissenschahten*, **81** (1994), pp 237-242.

[2] Smayda, T.J.: Global epidemic of noxious phytoplankton blooms and food chain consequences in large ecosystems. In: Food chains, models and management of large marine ecosystem (K.L.Sherman et al., eds), pp.257-307, Westview, 1992.

[3] Hallegraeff G.M.: A review of harmful algal blooms and the apparent global increase. *Phycologia* **32** (1993), pp 79-99.

[4] Burkholder, J.M. and Glasgow, H.B. Jr.:Interactions of a toxic estuarine dinoflagellate with microbial predators and prey. *Arch. Protistenkd*, **145** (1995), pp 177-188.

[5] Lewitus, A.J. and others : Discovery of the "phantom" dinoflagellate in Chesapeake Bay. *Estuaries*, **18** (1995), pp 373-378.

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

[7] Sarkar, R.R. and Chattopadhyay, J., Occurrence of planktonic blooms under environmental fluctuations and its possible control mechanism - mathematical models and experimental observations. *J. Theor. Biol.*, **224** (2002), pp 501-516.