Mathematical modelling of harmful algal blooms supported by experimental findings☆

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Abstract

In this paper non-toxic phytoplankton, toxin producing phytoplankton and zooplankton (NTP–TPP–Zooplankton) system is proposed and analyzed. It is assumed that the grazing pressure of zooplankton reduce due to presence of toxin producing phytoplankton. It is observed that TPP free steady state is unstable in nature and depicts bloom phenomena, whereas in the presence of TPP the system settles down to the positive state depicting stable phenomena and play a key role in bloom termination. The qualitative behaviour of the system is verified through our field observation and numerical simulation.

Keywords: Toxin producing plankton (TPP); Zooplankton; Bloom; Predational response function; Biological control

1. Introduction

Plankton are the basis of all aquatic food chain. 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 (Duinker and Wefer, 1994). Now the researchers are mainly concentrating on bloom dynamics with special emphasis on harmful algal blooms (HABs).

The dynamics of rapid increase or almost equally decrease of phytoplankton populations is known as 'bloom'. The planktonic blooms may be categorized into spring blooms and red tides. Spring blooms occur seasonally due to changes in temperature or nutrient availability. Red tides are the result of localized outbreaks associated with water temperature (Truscott and Brindley, 1994).

Phytoplankton species which cause fish poisoning (ciguatera) and shellfish-vectored poisoning of humans are clearly harmful (Hallegraeff, 1993). Harmful species can be characterized by three distinct models of negative impact such as 'toxic', noxious and nuisance used either synonymously or to characterize relative degree of impact. There are atleast eight different modes and mechanisms by which harmful phytoplankton species can cause mortality,

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physiological impairment, or other negative in situ effects. These fall into two general types: non chemical effects which lead to starvation or cause harmful mechanical and physical damage, and chemical effects attributable to physical-chemical reactions, phycotoxins, or other metabolites. Among the chemically harmful mechanisms, die-offs due to anoxia or hypoxia following blooms of large, relatively ungrazed species, such as *Ceratium*, are well known (Graneli et al., 1989; Mahoney and Steimle, 1979). *Noctiluca* produces a remarkably simple ichthyotoxin NH_4 (Okaichi and Nishio, 1976). Mortality can occur through direct ingestion(endotoxin)of the harmful species, upon exposure to secreted toxins (exotoxins), or from toxin vectoring through the food web, accompanied by conformational changes in the toxic principles and their potencies (Shimizu, 1989; Yasumoto and Murata, 1993). The impact may be on a directly targeted predator or lead to indiscriminate die-offs and physiological impairment during food-web vectoring. Despite convincing evidence that harmful phytoplankton species occur and bloom, knowledge of what defines a harmful species is qualitative. Such species are still being identified, and the modes and mechanisms of harmful effects, and ecophysiological divergence of harmful taxa from species considered to be non-harmful are still being described.

Quantitative ecological studies to establish in situ impacts of harmful algal species and their blooms should therefore be a high priority of future research. Among the more relevant issues needing investigation are the extent to which such blooms impair or disrupt "equilibrium" trophodynamics and whether they fall within the variance in natural catastrophic events which shape the equilibrium. Such studies are also needed to assess the validity of the spring diatom bloom model as a general model of bloom dynamics in the sea.

Blooms, as a phenomenon, collectively have properties other than biomass, numerical population density, and the potential of the bloom species to inflict harm. These features are also relevant to their definitions, occurrences and in situ trophodynamic consequences. The processes influencing species occurrences, whether harmful or not, may differ significantly from those regulating their blooms. And, at any given time or spatial location, as an inherent aspect of species succession, one or more species are in a state of bloom even though they may not achieve high biomass or high population density. The presence of toxic species does not necessarily lead to a deleterious impact; such effects require a threshold population density level, above which it becomes inimical. Given these considerations, formal definitions of what makes a species or its blooms harmful, toxic, noxious, a nuisance, benign, exceptional, and(or)unusual are neither practical nor helpful, and the application of such descriptors to in situ processes should be discontinued until, or unless, quantitative data in support of such distinctions are available. However, the basic distinction between harmful and non harmful blooms is not only warranted, it is an important first step toward classification of the various types of phytoplankton blooms which undoubtedly occur.

The adverse effect of harmful algal blooms is clear, but the control of such problems is under investigation. A group of researchers is in the favour of viral infection and the other groups are using toxin producing plankton (TPP) for controlling the bloom.

Recent works suggest that viruses are important regulatory factors in marine ecosystems. Lytic viruses directly control population dynamics by viral lysis (Suttle et al., 1990; Bratbak et al., 1995; Brussard et al., 1996). Few mathematical models also suggest that viral infection may be used as a controlling agent for the termination of planktonic blooms (Beltrami and Carroll, 1994; Chattopadhyay et al., 2003).

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. Examples of these diverse models of impact were given by Smayda (1992). Information on the recently discovered, remarkable ambush predatory behavior of the harmful dinoflagellate, *Pfiesteria piscicida* was presented by Burkholder and Glasgow (1995) and Lewitus et al. (1995). Toxin producing plankton (TPP) release toxic chemicals in the water and reduce the grazing pressure of zooplankton. As a result TPP may act as biological control for the termination of planktonic blooms (Chattopadhyay et al., 2002a,b; Sarkar and Chattopadhyay, 2003a).

In our previous works, non toxic phytoplankton population is not considered, but the role of non-toxic phytoplankton population in this regard cannot be ignored. 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. 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 reduce the growth of zooplankton.

To establish the theoretical results we shall use our field observations. We consider the toxin producing plankton species as *Noctiluca scintillans*, non-toxic phytoplankton species as *Coscinodiscus sp.* and zooplankton species as *Paracalanus sp.* (the taxonomical and functional distinctions of all the species are given in Chattopadhyay et al. (2002a), Sarkar and Chattopadhyay (2003b). 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.

2. Role of toxin producing plankton (TPP) in the termination of planktonic blooms

To establish the role of TPP in the termination of plankton blooms Chattopadhyay et al. (2002a) proposed a general mathematical model based on their field observation. They considered the following model:

$$\left.\begin{aligned}\frac{dP}{dt} &= rP\left(1-\frac{P}{K}\right)-\alpha f(P)Z\\ \frac{dZ}{dt} &= \beta f(P)Z-\mu Z-\theta g(P)Z\end{aligned}\right\} \tag{2.1}$$

Here P is the density of TPP population and Z the density of zooplankton population. Let r be the growth rate of toxic phytoplankton and K be the carrying capacity. α (>0) is the rate of predation of zooplankton on TPP population. Further β (>0) denotes the ratio of biomass consumed by zooplankton for its growth and μ (>0) is mortality rate of zooplankton due to natural death as well as for higher predation. θ denotes the rate of toxin liberation by TPP population. Here $f(P)$ represents the predational response function and $g(P)$ represent the distribution of toxic substances.

They observed that when $f(P)$, $g(P)$ are linear and also $f(P)$ is linear, $g(P)$ is Holling type II, the bloom phenomena has not been observed. But if the uptake functions are homographic type whatever be the distribution of toxic substances (linear or homographic), the excitable nature of blooms through periodicity has been observed. It is also noted that when the toxin liberation process is of Holling type III and uptake function is linear, the excitable nature of blooms through periodicity has been observed under some parametric restrictions. Moreover, when the upatake function is of Holling type III and toxin liberation process is linear or Holling type II and the upatake function is of Holling type II and toxin liberation process is Holling type III, then the bloom occur through multistability of positive equilibrium.

Further they also observed that if the growth of phytoplankton species follows logistic law, uptake function is of linear type in the absence of toxic substances, reduction of grazing pressure due to toxic substances is of Holling type II and the distribution of toxic substance is instantaneous, oscillatory succession of planktonic blooms is not observed by this model formulation and also observed that TPP population do not release toxic chemicals always but release only in the presence of dense zooplankton population around it. Moreover, in reality the reduction of grazing pressure due to toxic substance is not instantaneous, but is mediated by some time lag required to release toxic substances by the phytoplankton species. From this point of view Chattopadhyay et al. (2002b) modified the model by introducing discrete time variation in the grazing term. With this assumption model (2.1) takes the following form:

$$\left.\begin{aligned}\frac{dP}{dt} &= P\left(r-\frac{rP}{K}\right)-\alpha PZ\\ \frac{dZ}{dt} &= \beta PZ - cZ - \frac{\theta P(t-\tau)}{\gamma + P(t-\tau)}Z\end{aligned}\right\} \quad (2.2)$$

where τ is the discrete time delay and γ the half saturation constant.

Chattopadhyay et al. (2002b) also derived the conditions for which system (2.2) around E^* enters into supercritical or subcritical bifurcation. From the delay induced model they concluded that enhancement of environmental carrying capacity decreases the critical value of the delay factor to zero. As a result the instability region of the system increases. This observation also exhibits the nature of blooms through periodicity.

In order to understand ecosystem functioning better, we need to understand what determines phytoplankton species composition and succession during blooms, what happens when blooms decay, how contaminants influence bloom dynamics and what is the interplay between nutrient enrichment (through artificial eutrophication) and harmful algal blooms. This forcing appears rather normally due to daily, seasonal or annual cycles, photosynthetically active radiation, temperature, nutrient availability, eutrophication etc. and phytoplankton populations often fluctuate unpredictably in numbers (Evans and Parslow, 1985; Truscott, 1995; Popova et al., 1997; Ryabchenko et al., 1997). From this point of view Sarkar and Chattopadhyay (2003a) again modified and studied the system (2.2) taking into account the additive color noise with proper choice of autocorrelation time of the noise process as the residence time for nutrient concentration. The model equation takes the following form:

$$\left.\begin{aligned}\frac{dP}{dt} &= P(r+\eta(t)-\frac{rP}{K})-\alpha PZ\\ \frac{dZ}{dt} &= \beta PZ - \mu Z - \frac{\theta P(t-\tau)Z}{\gamma + P(t-\tau)}\end{aligned}\right\} \quad (2.3)$$

where the perturbed term $\eta(t)$ follows Ornstein–Uhlenbeck process (Uhlenbeck and Ornstein, 1954).

Moreover their analysis shows that there is a linear relationship between the delay factor and correlation time with a negative slope. The delay factor (τ) of the species is an inherent property and cannot be controlled in other way. Finally they concluded that TPP and control of the rapidity of environmental fluctuation are the key factors in termination of planktonic blooms. It is interesting to to see that whether the conclusion drawn from the above three studies is true or not, if we include non-toxic phytoplankton population into the system. The subsequent investigation to follow has been focussed in this direction.

3. The role of TPP in a non-toxic phytoplankton–toxin producing phytoplankton–zooplankton system

As we have mentioned earlier, that in our previous works non toxic phytoplankton population is not considered, but the role of non-toxic phytoplankton population in this regard cannot be ignored.

In our study we shall try 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 reduce 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 Chattopadhyay et al. (2002a), Sarkar and Chattopadhyay (2003b). 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.

3.1. Field observation and findings of Experimental results

From the above discussion, it is now clear that for coexistence of plankton populations and terminations of blooms, TPP and NTP have major role. To establish this we shall propose a mathematical model and the findings will be supported through our field observations.

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 Chattopadhyay et al. (2002a) and Sarkar and Chattopadhyay (2003b). 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, Figs. 1 and 2).

Moreover, the pattern observed for zooplankton population depicts the termination of bloom phenomenon (see, Figs. 1 and 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% increase in NTP biomass accumulation whereas about 49.7% decrease in zooplankton biomass (see, Table 1).

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

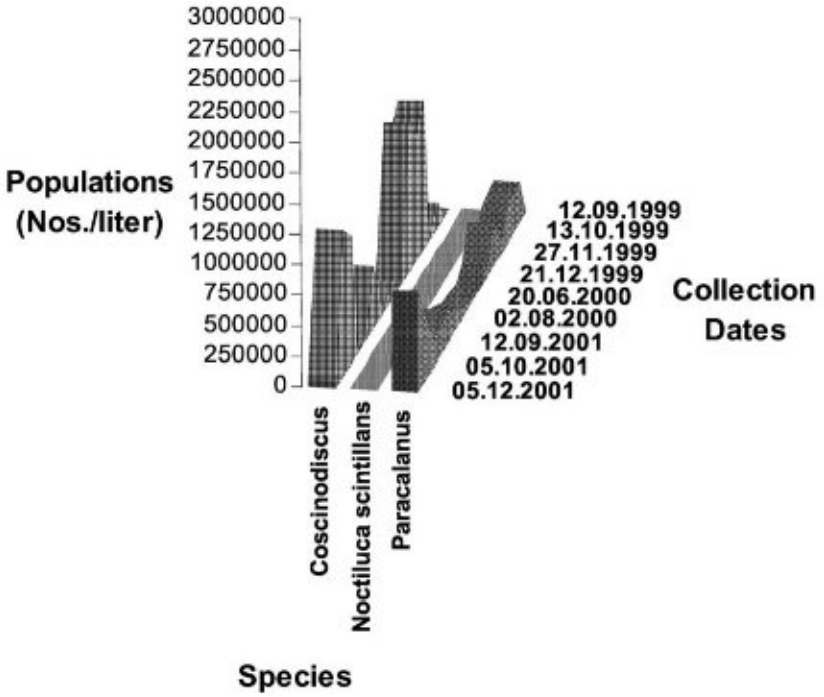

Fig. 1. Biomass distribution in absence of toxin producing phytoplankton.

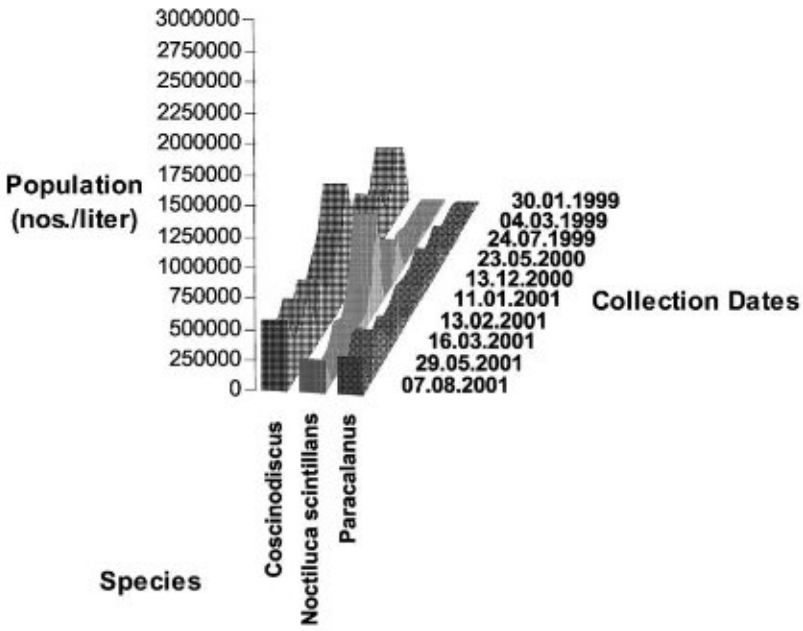

Fig. 2. Biomass distribution in presence of toxin producing phytoplankton.

phytoplankton in the termination of planktonic blooms have been observed.

3.2. 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. NTP and TPP are phytoplankton and have vertical movement only. They only share for the same resource. Here adequate forms would be an overall carrying capacity for both the species. For mathematical simplicity we assume that they are sharing the same carrying capacity (K). Let α and β be the maximum zooplankton ingestion rate and maximum zooplankton conversion rate respectively. Let μ be the death rate of zooplankton. Let s be the growth rate toxic phytoplankton. Let θ be the rate of toxin liberation by the toxic phytoplankton. Let θ_1 be the specific predation rate of zooplankton population on toxic phytoplankton. It is already confirmed by our field observation and different literature that toxic substances released by phytoplankton reduce the grazing pressure of its predator, zooplankton. As the fractional changes in the phytoplankton population per unit time effectively illustrates the impact of predation on the population at any particular time, it is interesting to examine the specific predation rate for the system as the outbreak advances (Truscott and Brindley, 1994). It is well known that at certain times, conditions (adequate temperature, proper light intensity, warmer water and minimal predation pressure) are adequate for planktonic growth. The phytoplankton will continue to bloom until one or more of the key factors prompting phytoplankton growth is no longer available. Keeping the above mentioned facts of phytoplankton–zooplankton population

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

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

in mind, we assume two predational forms for describing the dynamics. When phytoplakton populations do not produce toxin, we assume that the predation rate will follow the simple law of mass action. But as liberation of toxin by TPP reduces the growth of zooplankton, causes substantial mortality of zooplankton and in this period phytoplankton population is not easily accessible, hence a more common and intuitively obvious choice is of the Holling type II functional form to describe the grazing phenomena with γ as half saturation constant. Moreover saturation of grazing function allows the phytoplankton population to escape from grazing pressure of zooplankton and form a tide. This suppression of grazing is usually associated with active hunting behaviour on the part of the predator, as opposed to passively waiting to encounter food and there is a maximum rate of consumption per individual however large the phytoplankton population becomes. Holling type II or type III predational form (Ludwig et al., 1978) is an obvious choice to represent the hunting behaviour of predator. In reality the raptorial behaviour of Copepods is highly complex and exhibit an hunting behaviour (Uye, 1986) and hence type II or type III is an appropriate choice.

The mathematical model is:

$$\begin{aligned}\frac{dP_1}{dt} &= rP_1\left(1-\frac{P_1}{K}\right)-\alpha P_1 Z\\ \frac{dP_2}{dt} &= sP_2\left(1-\frac{P_2}{K}\right)-\frac{\theta P_2 Z}{\gamma+P_2}\\ \frac{dZ}{dt} &= \beta P_1 Z-\mu Z-\frac{\theta_1 P_2 Z}{\gamma+P_2}\end{aligned} \tag{3.1}$$

For sake of simplicity, we put in dimensionless form the model Eq. (3.1) by rescaling the variables on the carrying capacity value K, i.e.,

$$p_1=\frac{P_1}{K},\quad p_2=\frac{P_2}{K},\quad z=\frac{Z}{K} \tag{3.2}$$

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

$$\begin{aligned}\frac{dp_1}{d\tau} &= ap_1(1-p_1)-p_1 z\\ \frac{dp_2}{d\tau} &= bp_2(1-p_2)-\frac{cp_2 z}{1+p_2 d}\\ \frac{dz}{d\tau} &= ep_1 z-fz-\frac{gp_2 z}{1+p_2 d}\end{aligned} \tag{3.3}$$

where

$$a=\frac{r}{\alpha K},\quad b=\frac{s}{\alpha K},\quad c=\frac{\theta}{\alpha\gamma},\quad d=\frac{K}{\gamma},$$
$$e=\frac{\beta}{\alpha},\quad f=\frac{\mu}{K\alpha},\quad g=\frac{\theta_1}{\alpha\gamma} \tag{3.4}$$

System (3.3) has to be analyzed with the following initial conditions:

$$p_1(0)\geq 0,\quad p_2(0)\geq 0,\quad z(0)\geq 0. \tag{3.5}$$

For convenience in the following, time τ is replaced by t as the dimensionless time.

3.3. Some basic results

Boudedness of the system:

Theorem 3.1. *All the solutions of (3.3) are ultimately bounded.*

The proof is obvious.

Equilibria:

The system (3.3) 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 $E_4(f/e,0,a(1-(f/e)))$. The existence criterion of E_4 is $\beta K>\mu$. The positive equilibrium $E^*=(p_1^*,p_2^*,z^*)$, a triply coexisting equilibrium, where $z^*=a(1-p_1^*)$, $p_2^*=(ep_1^*-f)/(g+fd-ep_1^* d)$ and p_1^* satisfies the relation $Ap_1^{*3}+Bp_1^{*2}+Cp_1^*+D=0$, where $A=cae^2d^2$, $B=-cae^2d^2-2ca(g+fd)ed$, $C=-bge(1+d)+2ca(g+fd)ed+ca(g+fd)^2$, $D=bg(g+fd+f)-ca(g+fd)^2$.

Existence of positive interior equilibrium:

Now we observe that the triply coexisting equilibrium E^* exists if

$$L_1<\theta<L_2 \tag{3.6}$$

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

$$M_1 = \frac{f}{e}$$

$$M_2 = \max\left\{1, \frac{g}{ed} + \frac{f}{e}\right\}$$

Eigen value 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 3.1. *The steady state $E_0 = (0, 0, 0)$ of the system (3.3) is a saddle point.*

Lemma 3.2. *There exists a feasible toxic phytoplankton and zooplankton free steady state $E_1 = (1, 0, 0)$ which is unstable saddle.*

Lemma 3.3. *There exists a non-toxic phytoplankton and zooplankton free steady state $E_2 = (0, 1, 0)$ which is unstable saddle.*

Lemma 3.4. *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 3.2. *If the inequality*

$$\frac{\alpha\beta\gamma Ks}{r(\beta K - \mu)} > \theta \tag{3.7}$$

hold then the toxic phytoplankton free steady state $E_4 = (f/e, 0, (a(e - f))/e)$ is unstable

Proof. The variational matrix of the system around steady state $E_4 = (f/e, 0, (a(e - f))/e)$ is

$$V_4 = \begin{pmatrix} \frac{-af}{e} & 0 & \frac{-f}{e} \\ 0 & b - \frac{ca(e-f)}{e} & 0 \\ a(e-f) & \frac{-ga(e-f)}{e} & 0 \end{pmatrix}.$$

Further the eigen values of the variational matrix V_4 are λ_1, λ_2 which are the roots of the equation

$$\lambda^2 + \lambda\frac{af}{e} + \frac{af}{e}(e - f) = 0$$

and $\lambda_3 = b - (ca(e - f)/e)$.

Clearly λ_1 and λ_2 have negative real parts. Now if $\lambda_3 > 0$ i.e. $(\alpha\beta\gamma Ks/r(\beta K - \mu)) > \theta$ then E_4 is unstable. □

Theorem 3.3. *If the inequality*

$$\theta_1 < \frac{\gamma(\beta K - 2\mu)}{K} \tag{3.8}$$

hold then all the three species will persist.

The variational matrix of system (3.3) around the positive equilibrium $E^* = (p_1^*, p_2^*, z^*)$ is

$$V^* = \begin{pmatrix} -ap_1^* & 0 & -p_1^* \\ 0 & -bp_2^* - \frac{cz^*}{(1+p_2^*d)^2} + \frac{cz^*}{1+p_2^*d} & \frac{-cp_2^*}{1+p_2^*d} \\ ez^* & \frac{-gz^*}{(1+p_2^*d)^2} & 0 \end{pmatrix}.$$

To consider the local stability analysis of the triply coexisting 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 ineqality of ξ as

$$\min\left[\frac{be}{da^2}, \frac{fd + gd + fd^2 - g}{ed(d+1)}, \frac{M - \sqrt{(M^2 - 4LN)}}{2L}\right] < \xi < \frac{M + \sqrt{(M^2 - 4LN)}}{2L} \tag{3.9}$$

For positive equilibrium $E^* = (p_1^*, p_2^*, z^*)$, the characteristic equation is

$$\eta^3 + Q_1(\xi)\eta^2 + Q_2(\xi)\eta + Q_3(\xi) = 0$$

where the coefficients $Q_I(\xi)$, $I = 1, 2, 3$ are

$$Q_1(\xi) = ap_1^* + bp_2^* - \frac{cz^*p_2^*d}{(1+p_2^*d)^2}$$

$$Q_2(\xi) = ap_1^*(bp_2^* - \frac{cz^*p_2^*d}{(1+p_2^*d)^2}) - \frac{cgz^*p_2^*}{(1+p_2^*d)^3} + p_1^*ez^*$$

$$Q_3(\xi) = \frac{-ap_1^*cgz^*p_2^*}{(1+p_2^*d)^3} + p_1^*ez^*(bp_2^* - \frac{cz^*p_2^*d}{(1+p_2^*d)^2})$$

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

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

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

where $L = 2e^2 d(d+1)$, $M = ed(3g+3f+3fd+e+ed)$, $N = (g+fd)^2 + f(g+fd) - eg + efd + edg + efd^2$.

Since $d > 1$, it is obvious that $L > 0, M > 0$ and $N > 0$.

Also we observe that $Q_1(\xi) > 0$ and $Q_3(\xi) > 0$ if conditions (3.8) and (3.9) are satisfied.

Now let us define

$$\begin{aligned}\delta^{(2)}(\xi) &= Q_1(\xi)Q_2(\xi) - Q_3(\xi) \\ &= A_1(\xi)\xi\left[a^2\xi - \frac{be}{d}\right] + a\xi A_1^2(\xi) + a\xi^2 ez^* \\ &\quad + \frac{gA_1^2(\xi)}{d(1 + dp_2^*)} + \frac{A_1(\xi)bf}{d}\end{aligned}$$

Obviously $\delta^{(2)}(\xi) > 0$ by conditions (3.8) and (3.9).

Therefore according to Routh–Hurwitz criterion, E^* is locally asymptotically stable.

4. Discussion

In the literature two different approaches- cell-lysis due to viral infection and toxin producing phytoplankton, are being used for the termination of planktonic blooms. In our previous works we have established with the help of our experimental results and mathematical modelling, that toxin producing phytoplankton may be used as a controlling agent for the termination of planktonic blooms. But those studies contain certain flaws as we have omitted the presence of non toxic phytoplankton in such situations.

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 all 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 $E_4(f/e, 0, a(1-(f/e)))$ and which exists provided $\beta K > \mu$. It is also observed that this equilibrium is unstable in nature. But introduction of TPP in the system give rise to the triply coexisting equilibrium E^* which exists provided $L_1 < \theta < L_2$ and $f/e < p_1^* < \max\{1, f/e + g/ed\}$. Our analytical study reveals that the equilibrium level of NTP population increases and is due to the presence of TPP. Further for the zooplankton population we observe that when TPP is absent then the equilibrium value for zooplankton population is $Z^* = a(1-(f/e))$ but when TPP is present then the equilibrium value of Z^* satisfies the inequality $a(1-\max\{1, f/e+g/ed\}) < Z^* < a(1-(f/e))$. Hence presence of TPP decreases the bloom of zooplankton population. It is also observed that the positive interior equilibrium is locally asymptotically stable for certain threshold value of the toxin liberation rate.

The above findings clearly demonstrate the role of TPP in the termination of planktonic blooms. It is to be noted here our experimental findings also reflect the same observations.

To substantiate the analytical findings the following set of hypothetical parameter values have been considered for numerical simulations: $r = 4.5$, $K = 10$, $\alpha = 0.068$, $s = 0.1$, $\theta = 0.02$, $\gamma = 0.5$, $\beta = 0.08$, $\mu = 0.7$, $\theta_1 = 0.005$. For these set of parameter values we observe that TPP free equilibria as $E_4(0.875, 0, 0.827)$ but the triply coexisting equilibrium 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, Fig. 3).

In our previous works (without non-toxic phytoplankton) we observed the positive impact of toxin producing phytoplankton for the termination of planktonic blooms. In this work (in presence of non-toxic phytoplankton) also we arrived at the same conclusion. Thus we may finally conclude that toxin producing phytoplankton may be used as a bio-control agent

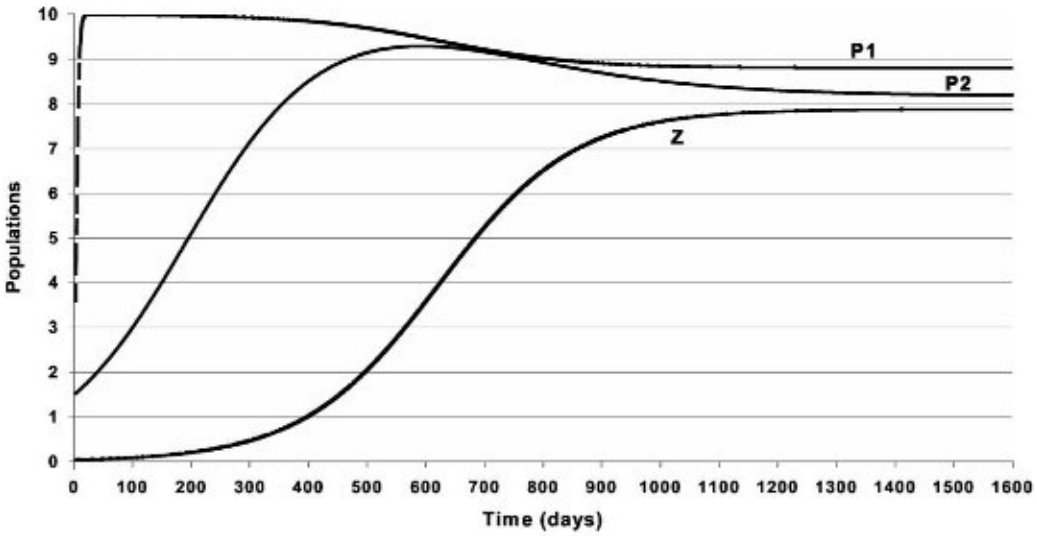

Fig. 3. Stable situation for inclusion of toxin producing phytoplankton depicting coexistence of all the species.

for the HAB's problems. The role of time lag and environmental fluctuations in the NTP, TPP, and zooplankton dynamics may give some interesting results and needs further investigations.

Before ending this paper we like to mention that the reason of occurrence of planktonic blooms and its possible control mechanism is still in infancy, hence the progress of such imporant areas urgently requires special attention both from experimental and mathematical ecologists.

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References

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

Bratbak, G., Levasseur, M., Michand, S., Cantin, G., Fernandez, E., Heldel, M., 1995. Viral activity in relation to *Emiliania huxleyi* blooms: a mechanism of DMSP release? Mar. Ecol. Progr. Ser. 128, 133–142.

Brussaard, C.P.D., Kempers, R.S., Kop, A.J., Riegman, R., Heldel, M., 1996. Virus like particles in a summer bloom of *Emiliania huxleyi* in the North Sea. Aq. Microbial. Ecol. 10, 105–113.

Burkholder, J.M., Glasgow Jr., H.B., 1995. Interactions of a toxic estuarine dinoflagellate with microbial predators and prey. Arch. Protistenkd 145, 177–188.

Chattopadhyay, J., Sarkar, R.R., Mandal, S., 2002a. Toxin producing plankton may act as a biological control for planktonic blooms-field study and mathematical modeling. J. Theor. Biol. 215, 333–344.

Chattopadhyay, J., Sarkar, R.R., El abdllaoui, A., 2002b. A delay differential equation model on harmful algal blooms in the presence of toxic substances. IMA J. Math. Appl. Med. Biol. 19, 137–161.

Chattopadhyay, J., Sarkar, R.R., Pal, S., 2003. Dynamics of nutrient-phytoplankton interaction in the presence of viral infection. BioSystems 68, 5–17.

Duinker, J., Wefer, G., 1994. Das CO_2-Problem und die Rolle des Ozeans. Naturwissenschahten 81, 237–242.

Evans, G.T., Parslow, S., 1985. A model of annual plankton cycles. Biol. Oceanogr. 3, 327–347.

Graneli, E., et al., 1989. From anoxia to fish poisoning: The last ten years of phytoplankton blooms in Swedish marine waters. In: Cosper, E.M., et al. (Eds.), Novel Phytoplankton Blooms, Springer, pp. 407–427.

Hallegraeff, G.M., 1993. A review of harmful algal blooms and the apparent global increase. Phycologia 32, 79–99.

Lewitus, A.J., et al., 1995. Discovery of the "phantom" dinoflagellate in Chesapeake Bay. Estuaries 18, 373–378.

Ludwig, D., et al., 1978. Qualitative analysis of an insect outbreak system: the spruce budworm and forest. J. Anim. Ecol. 47, 315.

Mahoney, J., Steimle, F.W., Jr., 1979. A mass mortality of marine animals associated with a bloom of *Ceratium tripos* in New York Bight. In: Proceedings of the Second International Conference of the Toxic Dinoflagellate Blooms. Elsevier, pp. 225–230.

Okaichi, T., Nishio, S., 1976. Identification of ammonmia as the toxic principle of red tide of *Noctiluca miliaris*. Bull. Plankton Soc. Jpn. 23, 25–30.

Popova, E.E., Fasham, M.J.R., Osipov, A.V., Ryabchenko, V.A., 1997. Chaotic behaviour of an ocean ecosystem model under seasonal forcing. J. Plankton. Res. 19, 1495–1515.

Ryabchenko, V.A., Fasham, M.J.R., Kagan, B.A., Popova, E.E., 1997. What causes short term oscillations in ecosystem models of the ocean mixed layer? J. Mar. Syst. 13, 33–50.

Sarkar, R.R., Chattopadhyay, J., 2003a. Occurrence of planktonic blooms under environmental fluctuations and its possible control mechanism - mathematical models and experimental observations. J. Theor. Biol. 224, 501–516.

Sarkar, R.R., Chattopadhyay, J., 2003b. The role of environmental stochasticity in a toxic phytoplankton–non-toxic phytoplankton–zooplankton system. Environmetrics 14, 775–792.

Shimizu, Y., 1989. Toxicology and pharmacology of red tides: an overview. In: Proceedings of the Firs International Symposium on Red Tides of the Red Tides: Biology, Environmental Science and Toxicology. Elsevier, pp. 17–22.

Smayda, T.J., 1992. Global epidemic of noxious phytoplankton blooms and food chain consequences in large ecosystems. In: Sherman, K.L., et al. (Eds.), Food Chains, Models and Management of Large Marine Ecosystem, pp. 257–307, Westview.

Suttle, C., Charm, A., Cottrell, M., 1990. Infection of phytoplankton by viruses and reduction of primary productivity. Nature 347, 467–469.

Truscott, J.E., 1995. Environmental forcing of simple plankton models. J. Plankton Res. 17, 2207–2232.

Truscott, J.E., Brindley, J., 1994. Ocean plankton populations as excitable media. Bull. Math. Biol. 56, 981–998.

Uhlenbeck, G.E., Ornstein, L.S., 1954. On the theory of Brownian motion. In: Wax, N. (Ed.), Selected Papers on Noise and Stochastic Process. Dover, New York, USA.

Uye, S., 1986. Impact of copepod grazing on the red tide flagellate *Chattonella antiqua*. Mar. Biol. 92, 35.

Yasumoto, T., Murata, M., 1993. Marine toxins. Chem. Rev. 93, 1897–1909.