Role of two toxin-producing plankton and their effect on phytoplankton–zooplankton system – a mathematical study supported by experimental findings

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Abstract

Plankton is the basis of the entire aquatic food chain. Phytoplankton, in particular, occupies the first trophic level. Plankton performs services for the Earth: it serves as food for marine life, gives off oxygen and also absorbs half of the carbon dioxide from the Earth's atmosphere. The dynamics of a rapid (or massive) increase or decrease of plankton populations is an important subject in marine plankton ecology and generally termed as a 'bloom'. Harmful algal blooms (HABs) have adverse effects on human health, fishery, tourism, and the environment. In recent years, considerable scientific attention has been given to HABs. Toxic substances released by harmful plankton play an important role in this context. In this paper, a mathematical model consisting of two harmful phytoplankton and zooplankton system will be discussed. The analytical findings will be verified through our experimental observations which were carried out on the eastern part of Bay of Bengal for the last three years.

Keywords: Two harmful phytoplankton; Zooplankton; Bloom; Competition; Biological control

1. Introduction

Phytoplankton are the source of almost all energy passing through aquatic food webs and comprise some 40% of the total fixed global primary productivity (Falkowski, 1984). A large component of this productivity can be attributed to the occurrence of both seasonal and sporadic algae blooms that form as patches over the ocean's surface in areas of localized nutrient enrichment. Research is now concentrating mainly on bloom dynamics with a special emphasis on harmful algal blooms (HABs). The dynamics of a rapid increase or decrease of phytoplankton populations is known as 'bloom'. Planktonic blooms may be categorized into spring blooms and red tides.

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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 that 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 the relative degree of impact. There are at least eight different modes and mechanisms by which harmful phytoplankton species can cause mortality, physiological impairment, or other negative in situ effects. These fall into two general types: non-chemical effects that 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). *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. 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 that 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 that undoubtedly occur. The adverse effect of harmful algal blooms is clear, but the control of such problems is under investigation.

Recent studies reveal that some times bloom of certain harmful species may lead to the release of both toxins and allelopathic substances. 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), 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 a biological control for the termination of planktonic blooms (see, Chattopadhyay et al., 2002a,b; Sarkar and Chattopadhyay, 2003; Chattopadhyay et al., 2004).

Our previous work has not cosidered two harmful phytoplankton populations, but the role of two harmful phytoplankton population in this regard cannot be ignored. In the present study, we will give special emphasis to the fact that the occurrence of toxin-

producing phytoplankton may not always be harmful but may help to maintain the stable equilibrium in trophodynamics through the coexistence of all the species. The model that we propose has three interacting components consisting of the two harmful phytoplankton and a herbivorous zooplankton with an additional factor that the release of toxic substance by toxic/harmful-phytoplankton species reduce the growth of zooplankton. We use field observations to establish the theoretical results.

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 the TPP population and Z the density of the zooplankton population. The variable 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. The variable θ denotes the rate of toxin liberation by the TPP population. Here $f(P)$ represents the predational response function and $g(P)$ represents the distribution of toxic substances.

They observed that the bloom phenomena can not be observed for two cases: (i) when $f(P)$ and $g(P)$ are both linear and (ii) $f(P)$ is linear but $g(P)$ is Holling type II. But if the uptake functions are homographic whatever 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 the uptake function is linear, the excitable nature of blooms through periodicity has been observed under some parametric restrictions.

Further, they also observed that TPP population did not always release toxic chemical but instead release only in the presence of dense zooplankton population around it. Moreover, in reality the reduction of grazing pressure due to a 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. From the delay-induced model they concluded that the 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 (Popova et al., 1997; Ryabchenko et al., 1997). From this point of view, Sarkar and Chattopadhyay (2003) again modified and studied the delay-induced system taking into account the additive colored noise with the proper choice of autocorrelation time of the noise process as the residence time for nutrient concentration. Finally they concluded that TPP and the control of the rapidity of environmental fluctuation are the key factors in the termination of planktonic blooms. It is interesting to see whether or not conclusion drawn from the above three studies can be verified, if we include more toxic phytoplankton population into the system.

3. The role of two harmful phytoplanktons in a phytoplankton–zooplankton system

In our study, we try to observe the effects of harmful plankton in the dynamics of two harmful phytoplankton–zooplankton interaction. The model

that we propose has three interacting components consisting of the two harmful phytoplankton (HP) and herbivorous zooplankton (Z) with an additional factor that the release of toxic substance by toxic-phytoplankton species reduces the growth of zooplankton.

We consider the toxin-producing plankton species as *Noctiluca scintillans*, harmful phytoplankton species as *Chaetoceros sp.*, and zooplankton species as *Paracalanus sp.* (the taxonomical and functional distinctions of the species *N. scintillans* and *Paracalanus sp.* are given in Chattopadhyay et al., 2002a; Sarkar and Chattopadhyay, 2003), and *Chaetoceros sp.* belongs to the group diatom. It is a non-toxic microorganisms but results in hypoxic conditions and thus, is a harmful algae and reduces the growth of a zooplankton population. Our field study suggests that when any one of the harmful phytoplanktons is absent the equilibrium level of both harmful phytoplanktons and zooplankton are much higher than the value observed when both harmful phytoplanktons are present. This phenomenon depicts the fact that the presence of two harmful phytoplanktons in the system reduces the high abundance of harmful phytoplanktons and the zooplankton population. Further, the biomass distribution observed in our field study demonstrates that the introduction of two harmful phytoplanktons leads to the persistence of all the species through the termination of blooms and can be used as a controlling agent for the stability of marine ecosystem.

3.1. Field observation and findings of experimental results

Monitoring of plankton population was carried out since January, 1999 in the North–West coast of Bay of Bengal. We choose *N. scintillans* and *Chaetoceros sp.* as harmful phytoplankton species and *Paracalanus sp.* as zooplankton for this study. The materials and method of the study have been discussed elaborately in Chattopadhyay et al. (2002a), Sarkar and Chattopadhyay (2003). Here we consider the data of the field observation from March 1999 to February 2001 (33 sample collection dates). To establish our theoretical realization we focussed mainly on the dynamics of the two HP–zooplankton system in the total time frame and we observed the high abundance (may also depicted as a bloom) of harmful phytoplankton species due to the absence of other harmful species. In our total study time, when both the harmful phytoplankton species were present, the peaks of zooplankton population came down in compared to the presence of single harmful phytoplankton (see, Fig. 1).

Moreover, the pattern observed for the zooplankton population depicts the termination of the bloom phenomenon due to the presence of two HP (see, Fig. 1). Again equilibrium analysis is useful in that it identifies the effect of inclusion of some species in real food chain models. To study this we observe the average biomass accumulation over the entire time period of our field

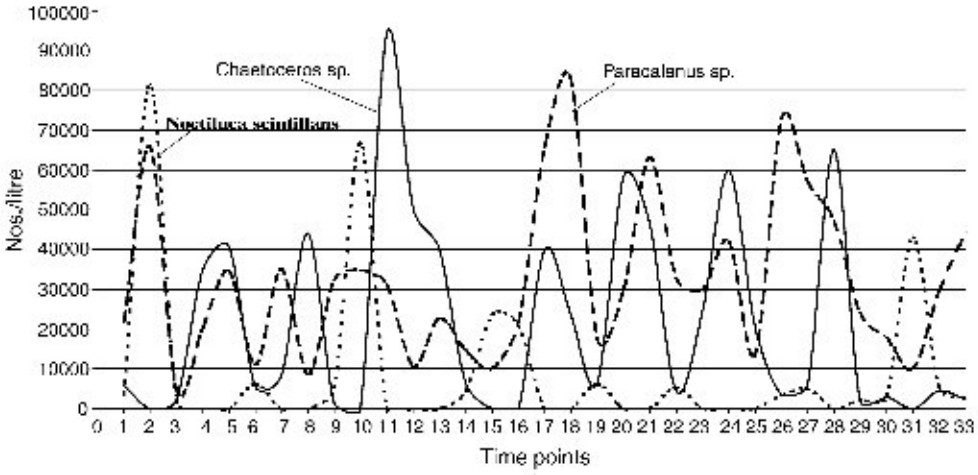

Fig. 1. Biomass distribution of *Chaetoceros sp.*, *Noctiluca scintillans* and *Paracalanus sp.*

Fig. 2. Average biomass of *Chaetoceros sp.* and *Paracalanus sp.*

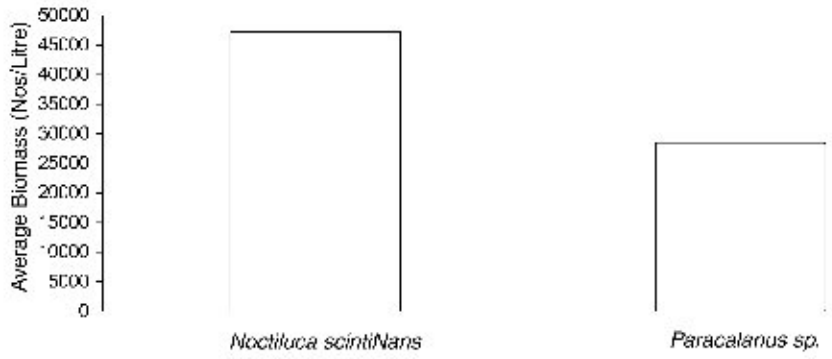

Fig. 3. Average biomass of *Noctiluca scintillans* and *Paracalanus sp.*

study for three different cases: (i) the presence of only *Chaetoceros sp.* and *Paracalanus sp.* (see, Fig. 2), (ii) the presence of only *N. scintillans* and *Paracalanus sp.* (see, Fig. 3) and (iii) the presence of two harmful phytoplankton (both *N. scintillans* and *Chaetoceros sp.*) and zooplankton (*Paracalanus sp.*), i.e., all three species (see, Fig. 4). We observe that there was a considerable decrease in harmful phytoplankton and zooplankton biomass accumulation as compared to the situation when a single harmful phytoplankton was present.

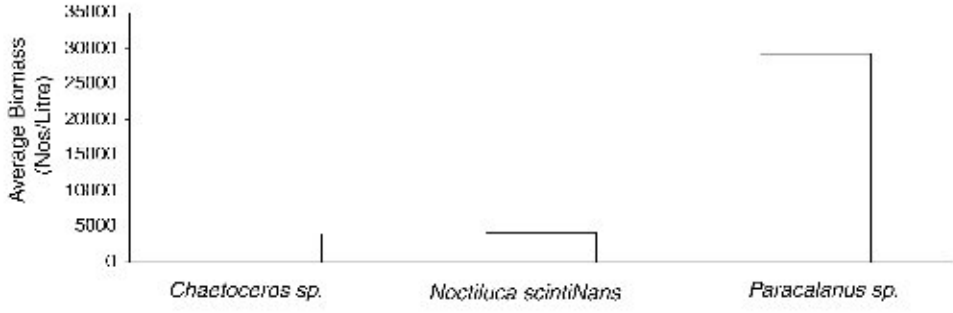

Fig. 4. Average biomass of *Chaetoceros sp.*, *Noctiluca scintillans* and *Paracalanus sp.*

Motivated from the literature and our field observations, a dynamic model consisting of two harmful phytoplankton and zooplankton has been proposed and the role of harmful phytoplankton in the termination of planktonic blooms has been observed.

3.2. The mathematical model

Let $P_1(t)$ and $P_2(t)$ be the concentrations of the harmful phytoplanktons at time t. Let $Z(t)$ be the concentration of zooplankton at time t. Let r_1 and r_2 be the growth rates of the harmful phytoplanktons, respectively. Let K and L be the environmental carrying capacity of the two harmful phytoplanktons. Let m and n be the maximum zooplankton ingestion rates for both the harmful phytoplankton species and m_1 and n_1 be the maximum zooplankton conversion rates respectively. Let μ be the natural death rate of zooplankton. Let α and β be the inhibitory effects of the two competeting harmful phytoplanktons. Let θ_1 and θ_2 be the rates of toxin liberation by the harmful phytoplanktons, respectively, which reduces the growth of zooplankton. Here $f(P_1)$ and $g(P_2)$ represent the distribution of toxic substances for the two phytoplanktons.

The mathematical model is:

$$\begin{aligned}\frac{dP_1}{dt} &= r_1P_1\left(1-\frac{P_1}{K}\right)-\alpha P_1P_2-mP_1Z\\ \frac{dP_2}{dt} &= r_2P_2\left(1-\frac{P_2}{L}\right)-\beta P_1P_2-nP_2Z\\ \frac{dZ}{dt} &= (m_1P_1+n_1P_2)Z-\mu Z\\ &\quad -\theta_1 f(P_1)Z-\theta_2 g(P_2)Z.\end{aligned} \tag{3.1}$$

System (3.1) has to be analyzed with the following initial conditions:

$$P_1(0)\geq 0,\ P_2(0)\geq 0,\ Z(0)\geq 0. \tag{3.2}$$

3.3. Some basic results

3.3.1. Boudedness of the system

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

Proof. We define a function

$$w = P_1+P_2+Z \tag{3.3}$$

The time derivative of (3.3) along the solutions of (3.1) is

$$\begin{aligned}\frac{dw}{dt} &= P_1r_1\left(1-\frac{P_1}{K}\right)-\alpha P_1P_2-mP_1Z\\ &\quad +P_2r_2\left(1-\frac{P_2}{L}\right)-\beta P_1P_2-nP_2Z+m_1P_1Z\\ &\quad +n_1P_2Z-\mu Z-\theta_1 f(P_1)Z-\theta_2 g(P_2)Z\end{aligned}$$

For $q>0$

$$\begin{aligned}\frac{dw}{dt}+qw &\leq P_1r_1\left(1-\frac{P_1}{K}\right)+P_2r_2\left(1-\frac{P_2}{L}\right)\\ &\quad -\mu Z+qP_1+qP_2+qZ\end{aligned} \tag{3.4}$$

If we take q such that $0<q<\mu$ and $M>0$ and $M>\max\{M_1, M_2\}$ where M_1 maximum value of $P_1[r_1(1-\frac{P_1}{K})+q]$, i.e., $M_1=\frac{r_1}{4}+q$ and M_2 maximum value of $P_2[r_2(1-\frac{P_2}{L})+q]$, i.e., $M_2=\frac{r_2}{4}+q$, then

$$\frac{dw}{dt}+qw\leq M \qquad \square$$

Equilibria: The system (3.1) possesses the following equilibria:the plankton free equilibrium $E_0=(0,0,0)$, one harmful phytoplankton and zooplankton free equilibrium $E_1(K,0,0)$, another harmful phytoplankton and zooplankton free equilibrium $E_2(0,L,0)$. A feasible zooplankton free equilibrium $E_3(\frac{Kr_2(\alpha L-r_1)}{\alpha\beta KL-r_1r_2},\frac{Lr_1(\beta K-r_2)}{\alpha\beta KL-r_1r_2},0)$, which exists if $r_1>\alpha L$ and $r_2>\beta K$. A feasible one harmful phytoplankton free equilibrium $E_4(P_1',0,Z')$ where $Z'=\frac{r_1}{m}(1-\frac{P_1'}{K})$ and P_1' satisfies the relation $m_1P_1'-\mu-\theta_1f(P_1')=0$. Another harmful phytoplankton free equilibrium $E_5(0,P_2'',Z'')$ where $Z''=\frac{r_2}{n}(1-\frac{P_2''}{L})$ and P_2'' satisfies the relation $n_1P_2''-\mu-\theta_2g(P_2'')=0$. The positive interior equilibrium is $E^*=(P_1^*,P_2^*,Z^*)$ where $P_2^*=LAP_1^*+LB$, $Z^*=\frac{r_2}{n}[1-AP_1^*-B]-\frac{\beta}{n}P_1^*$ and P_1^* satisfies the relation $m_1P_1^*+n_1LAP_1^*+n_1LB-\mu-\theta_1f(P_1^*)-\theta_2g(LAP_1^*+LB)=0$ where $A=\frac{nr_1-m\beta K}{K(mr_2-n\alpha L)}$ and $B=\frac{mr_2-nr_1}{mr_2-n\alpha L}$.

Now we observe that the positive interior equilibrium E^* exists if the following inequalities hold true: $\min\{\frac{\alpha L}{r_2},\frac{r_1}{r_2}\}<\frac{m}{n}<\frac{r_1}{\beta K}$, $P_1^*<\frac{Kr_2(r_1-\alpha L)}{r_1r_2-\alpha\beta KL}$ and

$$\begin{aligned}&m_1P_1^*+n_1LAP_1^*+n_1LB-\mu-\theta_1f(P_1^*)\\ &-\theta_2g(LAP_1^*+LB)>0\end{aligned} \tag{3.5}$$

3.3.2. Eigenvalue analysis

In this section, local stability analysis of the system around the biological feasible equilibria will be performed. The aim of this part is to find out the suitable mechanism to explain planktonic blooms and possible control.

Lemma 3.1. *The plankton free steady state E_0 of the system (3.1) is a saddle point.*

Lemma 3.2. *There exists a feasible one toxic phytoplankton and zooplankton free steady state E_1 of the system (3.1) which is unstable saddle if $r_2 > \beta K$ (for existence of E^*).*

Lemma 3.3. *There exists another feasible toxic phytoplankton and zooplankton free steady state E_2 of the system (3.1) which is unstable saddle if $r_1 > \alpha L$ (for existence of E^*).*

Lemma 3.4. *If the inequality $m_1 P_1 + n_1 P_2 > \mu + \theta_1 f(P_1) + \theta_2 g(P_2)$ holds then E_3 is a unstable saddle.* (see Appendix A).

Lemma 3.5. *If the inequality $r_2 - \beta P_1' - nZ' > 0$ then E_4 is a unstable saddle.* (see Appendix A).

Lemma 3.6. *One toxic phytoplankton free equilibrium $E_5(0, P_2'', Z'')$ is unstable if $r_1 - \alpha P_2'' - mZ'' > 0$.* (see Appendix A).

Now we shall derive the local behavior of the system (3.1) around the positive interior and obtain the following theorem equilibrium.

Theorem 3.2. *If*

$$\frac{\alpha L}{r_2} < \frac{m}{n} < \frac{r_1}{\beta K} \tag{3.6}$$

and $n_1 > \theta_2 g'(P_2^*)$ and $m_1 > \theta_1 f'(P_1^*)$ then all the three species will persist (see Appendix A).

We have already mentioned that toxin liberation processes play an important role in planktonic dynamics. As the liberation of toxin reduces the growth of zooplankton, it causes substantial mortality of zooplankton and in this period, the TPP population is not easily accessible. Hence, a more common and intuitively obvious choice is of Holling types II or III functional form to describe the grazing phenomenon.

The main objective for taking into consideration different combinations of functional forms is to understand mechanisms of planktonic blooms and their possible control.

4. Toxin-producing phytoplankton–zooplankton model when the distribution of toxic substances are Holling type II functions

Here we consider the distribution of toxic substances as having Holling type II functional form and rewrite the system equation as:

$$\begin{aligned}
\frac{dP_1}{dt} &= r_1 P_1 \left(1 - \frac{P_1}{K}\right) - \alpha P_1 P_2 - m P_1 Z \\
\frac{dP_2}{dt} &= r_2 P_2 \left(1 - \frac{P_2}{L}\right) - \beta P_1 P_2 - n P_2 Z \\
\frac{dZ}{dt} &= (m_1 P_1 + n_1 P_2) Z - \mu Z \\
&\quad - \theta_1 \frac{P_1}{\gamma_1 + P_1} Z - \theta_2 \frac{P_2}{\gamma_2 + P_2} Z.
\end{aligned} \tag{4.1}$$

Here γ_1 and γ_2 denote the half-saturation constants for the two toxin-producing phytoplanktons.

Equilibria: The system (4.1) possesses the following equilibria: the plankton free equilibrium $E_0 = (0, 0, 0)$, one harmful phytoplankton and zooplankton free equilibrium $E_1(K, 0, 0)$. Another harmful phytoplankton and zooplankton free equilibrium $E_2(0, L, 0)$. A feasible zooplankton free equilibrium $E_3(\frac{Kr_2(\alpha L - r_1)}{\alpha\beta KL - r_1 r_2}, \frac{Lr_1(\beta K - r_2)}{\alpha\beta KL - r_1 r_2}, 0)$, which exists if $r_1 > \alpha L$ and $r_2 > \beta K$. A feasible one harmful phytoplankton free equilibrium $E_4(P_1', 0, Z')$ where $Z' = \frac{r_1}{m}(1 - \frac{P_1'}{K})$ and P_1' satisfies the relation $m_1 P_1'^2 + P_1'(m_1\gamma_1 - \mu - \theta_1) - \mu\gamma_1 = 0$. If $m_1\gamma_1 - \mu - \theta_1 > 0 or < 0$, in each case one root of the above equation is positive. Another harmful phytoplankton free equilibrium $E_5(0, P_2'', Z'')$ where $Z'' = \frac{r_2}{n}(1 - \frac{P_2''}{L})$ and P_2'' satisfies the relation $n_1 P_2''^2 + P_2''(n_1\gamma_2 - \mu - \theta_2) - \mu\gamma_2 = 0$. Here also for $n_1\gamma_2 - \mu - \theta_2 > 0 or < 0$, in each case one root of the above equation is positive. The positive interior equilibrium is $E^* = (P_1^*, P_2^*, Z^*)$ where $P_2^* = LAP_1^* + LB$, $Z^* = \frac{r_2}{n}[1 - AP_1^* - B] - \frac{\beta}{n}P_1^*$ and P_1^* satisfies the relation

$$C_1 P_1^{*3} + C_2 P_1^{*2} + C_3 P_1^* + C_4 = 0$$

where

$$C_1 = LA(m_1 + n_1 LA)$$

$$C_2 = (\gamma_2 + LB)(m_1 + n_1 LA) + LA(m_1\gamma_1 + n_1\gamma_1 LA + n_1 LB - \mu - \theta_1) - \theta_2 LA$$

$$C_3 = (\gamma_2 + LB)(m_1\gamma_1 + n_1\gamma_1 LA + n_1 LB - \mu - \theta_1) + LA(n_1\gamma_1 LB - \mu\gamma_1) - (\theta_2 LA\gamma_1 + \theta_2 LB)$$

$$C_4 = (\gamma_2 + LB)(n_1\gamma_1 LB - \mu\gamma_1) - \theta_2 LB\gamma_1$$

The conditions for the existence of a positive interior equilibrium E^* is given in Appendix B.

4.1. Eigenvalue analysis

First consider the plankton free steady state E_0 of the system (4.1).

Clearly the plankton free steady state is unstable (saddle) and there exists a feasible one toxic phytoplankton and zooplankton free steady state E_1.

This steady state is unstable if $r_2 > \beta K$ (for existence of E^*).

Then there exists a feasible another toxic phytoplankton and zooplankton free steady state E_2.

This steady state is unstable if $r_1 > \alpha L$ (for existence of E^*).

Now if $m_1 P_1 + n_1 P_2 > \mu + \frac{\theta_1 P_1}{\gamma_1 + P_1} - \frac{\theta_2 P_2}{\gamma_2 + P_2}$ then E_3 is a unstable saddle (see Appendix B).

If $\frac{K(nr_1 - mr_2)}{nr_1 - m\beta K} < P_1'$ then E_4 is a unstable saddle (see Appendix B).

Now if $\frac{L(mr_2 - nr_1)}{mr_2 - n\alpha L} < P_2''$ then E_5 is an unstable saddle (see Appendix B).

Theorem 4.1. *If*

$$\frac{\alpha L}{r_2} < \frac{m}{n} < \frac{r_1}{\beta K} \tag{4.2}$$

and

$$P_1^* > \min\left\{\sqrt{\frac{\theta_1\gamma_1}{m_1}} - \gamma_1, \frac{1}{LA}\left(\sqrt{\frac{\theta_2\gamma_2}{n_1}} - \gamma_2 - LB\right)\right\} \tag{4.3}$$

then all the three species will persist.

5. Numerical experiments

Now we compare qualitatively the dynamical patterns observed in our field study with our model formulation for different functional forms. For this we substantiate our analytical findings through numerical simulations considering the following hypothetical set of parameter values: $r_1 = 0.55$ day^{-1}, $r_2 = 0.5$ day^{-1}, $K = 20$ nos. ml^{-1}, $L = 22$ nos. ml^{-1}, $\alpha = 0.005$ ml nos.$^{-1}$ day^{-1}, $\beta = 0.004$ ml nos.$^{-1}$ day^{-1}, $m = 0.15$ ml nos.$^{-1}$ day^{-1}, $n = 0.15$ ml nos.$^{-1}$ day^{-1}, $m_1 = 0.09$ ml nos.$^{-1}$ day^{-1}, $n_1 = 0.075$ ml nos.$^{-1}$ day^{-1}, $\mu = 0.09$ day^{-1}, $\theta_1 = 0.06$ ml day^{-1}, $\theta_2 = 0.07$ ml day^{-1}, $\gamma_1 = 0.1$ nos.

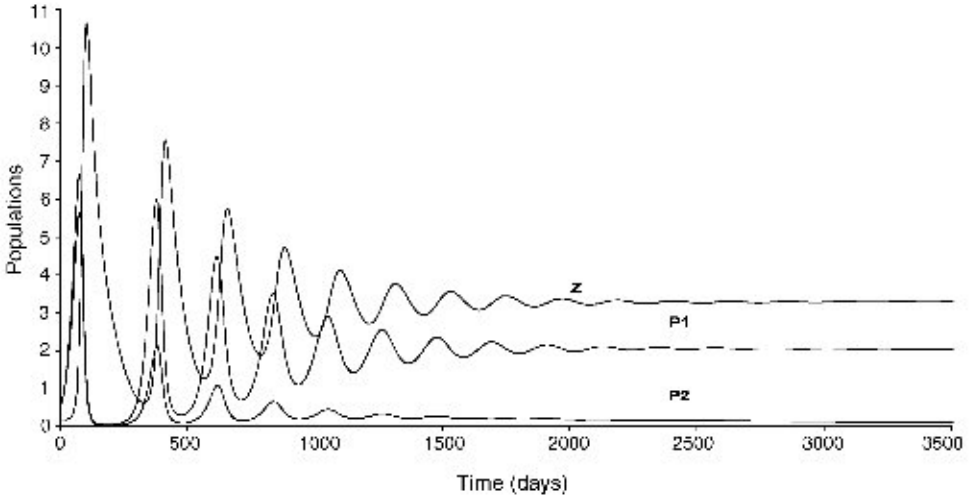

Fig. 5. Stable situation when $f(P_1)$ and $g(P_2)$ are both of Holling type II.

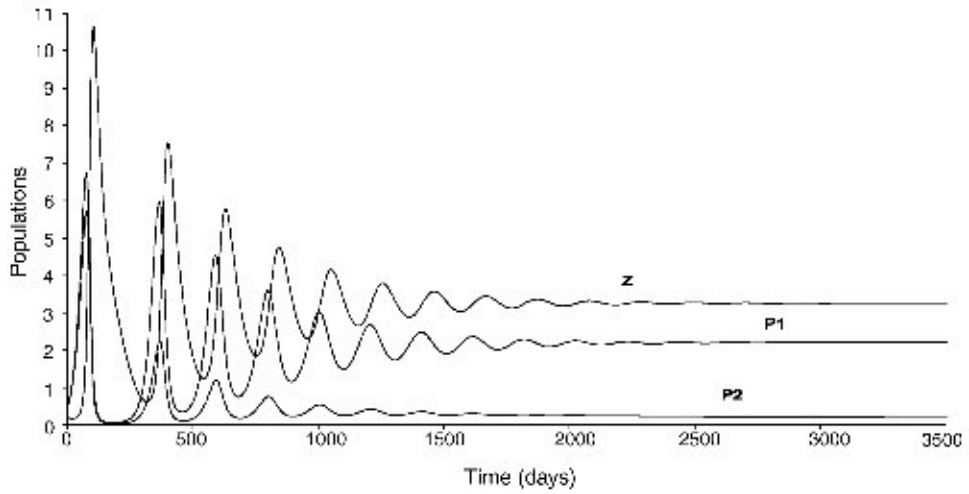

Fig. 6. Stable situation when $f(P_1)$ and $g(P_2)$ are both of Holling type III.

ml^{-1}, $\gamma_2 = 0.12\,nos.ml^{-1}$. Our numerical results show the coexistence of all the species when both $f(P_1)$ and $g(P_2)$ are of Holling type II (a stable situation, see, Fig. 5).

When both $f(P_1)$ and $g(P_2)$ are of Holling type III, i.e., $f(P_1) = \frac{{P_1}^2}{{\gamma_1}^2+{P_1}^2}$ and $g(P_2) = \frac{{P_2}^2}{{\gamma_2}^2+{P_2}^2}$ (in all the cases γ_1 and γ_2 denote the half-saturation constants for the two toxin-producing phytoplanktons), in this case we also observe a stable situation (see, Fig. 6).

We also observe the situation when both $f(P_1)$ and $g(P_2)$ are linear, i.e., $f(P_1) = P_1$ and $g(P_2) = P_2$. In this case we observe a stable situation (see, Fig. 7).

In another case when $f(P_1)$ is Holling type II, i.e., $f(P_1) = \frac{P_1}{\gamma_1+P_1}$ and $g(P_2)$ is Holling type III, i.e., $g(P_2) = \frac{{P_2}^2}{{\gamma_2}^2+{P_2}^2}$, we observe a stable situation (see, Fig. 8).

We have not identified the model parameters directly from our field observations, but the qualitative comparison of our model simulation with the dynamical pattern observed in the field study may give some insight into estimating of the parameter values. The

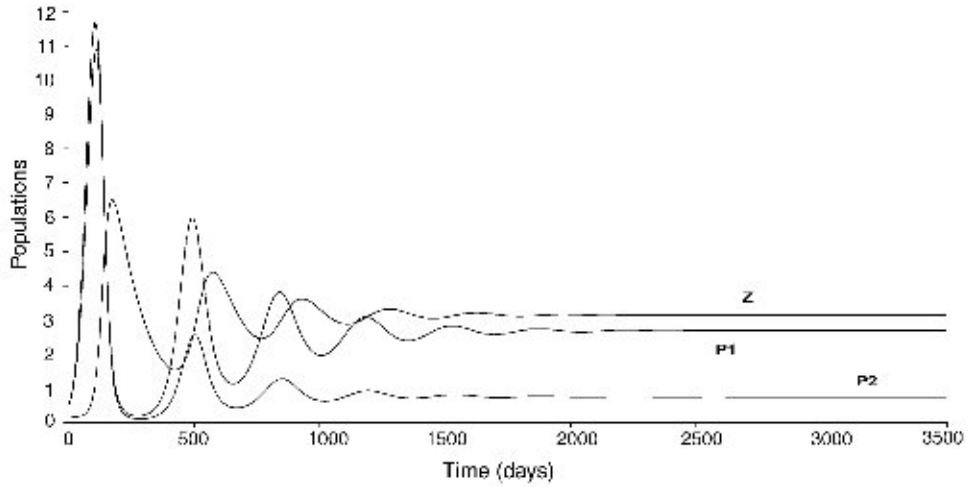

Fig. 7. Stable situation when $f(P_1)$ and $g(P_2)$ are both of Holling type I.

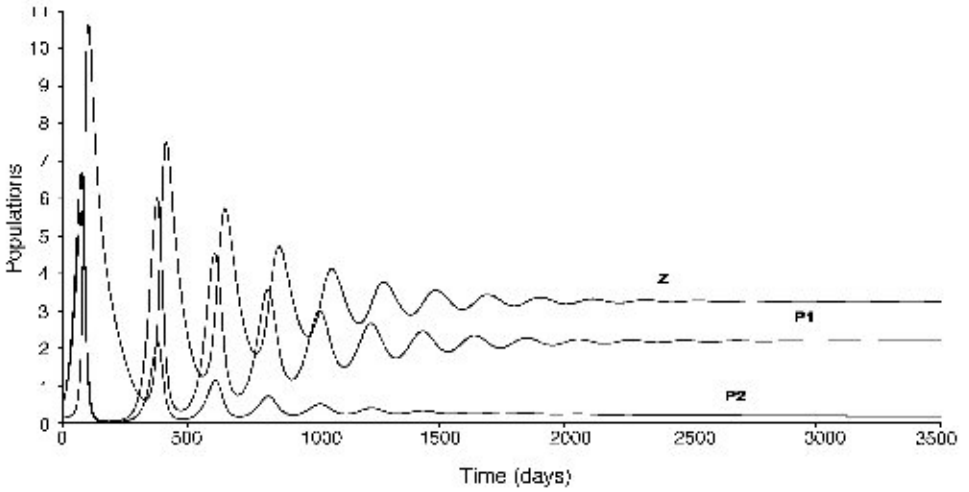

Fig. 8. Stable situation when $f(P_1)$ is of Holling type II and $g(P_2)$ of Holling type III.

above numerical study also help us to estimate the thresholds for which the system possesses asymptotic stability around the interior equilibrium and supports our claim that presence of two harmful phytoplankton has a positive impact for the termination of planktonic blooms.

6. 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. Previous studies have established, with the help of 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 do not contain the presence of two harmful phytoplanktons in such a situation.

In this paper we have proposed and analyzed a three-component model consisting of two harmful phytoplanktons 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 certain conditions are satisfied. Our theoretical as well as numerical 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.

Our analytical and numerical study reveal that the equilibrium levels of HP and zooplankton population decrease and are due to the presence of two HP. Hence, the presence of two HP decreases the bloom of an individual population as well as a zooplankton population. The above findings clearly demonstrate the role of HP in the termination of planktonic blooms. Note that our experimental findings also reflect the same observations.

In the previous studies (without two harmful phytoplankton) we observed the positive impact of toxin-producing phytoplankton for the termination of planktonic blooms. In this work (in presence of two harmful phytoplankton) we also arrived at the same conclusion. Thus, we may finally conclude that harmful phytoplanktons may be used as a bio-control agent for the HAB's problems. The role of time lag and environmental fluctuations in the two harmful phytoplankton–zooplankton dynamics may give some interesting results and needs further investigation.

The reason for the occurrence of planktonic blooms and their possible control mechanism is still its infancy, hence, the progress of such imporant areas urgently requires special attention both from experimental and mathematical ecologists.

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Appendix A

Let $\bar{E} = (\bar{P}_1, \bar{P}_2, \bar{z})$ be any arbitrary equilibrium. Then the variational matrix about $\bar{E}$ is given by

$$\bar{V} = \begin{bmatrix} r_1 - \dfrac{-2r_1\bar{P}_1}{K} - \alpha\bar{P}_2 - m\bar{z} & -\alpha\bar{P}_1 & -m\bar{P}_1 \\ -\beta\bar{P}_2 & r_2 - \dfrac{-2r_2\bar{P}_2}{L} - \beta\bar{P}_1 - n\bar{z} & -n\bar{P}_2 \\ m_1\bar{Z} - \theta_1 f'(\bar{P}_1)\bar{Z} & n_1\bar{Z} - \theta_2 g'(\bar{P}_2)\bar{Z} & m_1\bar{P}_1 + n_1\bar{P}_2 - \mu - \theta_1 f(\bar{P}_1) - \theta_2 g(\bar{P}_2) \end{bmatrix}$$

By computing the variational matrix for the equilibrium E_0 of the system (3.1) we find that the eigenvalues of the variational matrix V_0 are $\lambda_1 = r_1 > 0$, $\lambda_2 = r_2 > 0$, $\lambda_3 = -\mu < 0$.

Clearly the plankton free steady state is unstable (saddle).

Further, the eigenvalues of the variational matrix V_1 of the equilibrium E_1 of the system (3.1) are $\lambda_1 = -r_1 < 0$, $\lambda_2 = r_2 - \beta K$ and $\lambda_3 = m_1 K - \mu - \theta_1 f(K)$. This steady state is unstable if $r_2 > \beta K$ (for existence of E^*).

The eigenvalues of the variational matrix V_2 are $\lambda_1 = -r_2 < 0$, $\lambda_2 = r_1 - \alpha L$ and $\lambda_3 = n_1 L - \mu - \theta_2 g(L)$.

Thus, the steady state E_2 is unstable if $r_1 > \alpha L$ (for existence of E^*).

Further, the eigenvalues of the variational matrix V_3 around the equilibrium E_3 of the system (3.1) are λ_1', λ_2' which are the roots of the equation

$$\lambda^2 + \lambda\left(\frac{r_1 P_1}{K} + \frac{r_2 P_2}{L}\right) + P_1 P_2 \frac{(r_1 r_2 - \alpha\beta KL)}{KL} = 0$$

and $\lambda_3' = m_1 P_1 + n_1 P_2 - \mu - \theta_1 f(P_1) - \theta_2 g(P_2)$. Clearly λ_1' and λ_2' have negative real parts. Now if $\lambda_3' > 0$, i.e., $m_1 P_1 + n_1 P_2 > \mu + \theta_1 f(P_1) + \theta_2 g(P_2)$ then E_3 is an unstable saddle.

In similar manner, the equilibrium $E_4(P_1', 0, Z')$ of the system (3.1) is unstable saddle if $r_2 - \beta P_1' - nZ' > 0$.

The equilibrium $E_5(0, P_2'', Z'')$ of the system (3.1) is unstable if $r_1 - \alpha P_2'' - mZ'' > 0$.

The variational matrix of system (3.1) around the positive equilibrium $E^* = (P_1^*, P_2^*, Z^*)$ is

$$V^* = \begin{bmatrix} \dfrac{-r_1 P_1^*}{K} & -\alpha P_1^* & -m P_1^* \\ -\beta P_2^* & \dfrac{-r_2 P_2^*}{L} & -n P_2^* \\ m_1 Z^* - \theta_1 f'(P_1^*)Z^* & n_1 Z^* - \theta_2 g'(P_2^*)Z^* & 0 \end{bmatrix}$$

For positive equilibrium $E^* = (P_1^*, P_2^*, Z^*)$, the characteristic equation is

$$\lambda^3 + Q_1\lambda^2 + Q_2\lambda + Q_3 = 0$$

where the coefficients Q_I, $I = 1, 2, 3$ are

$$Q_1 = \frac{r_1 P_1^*}{K} + \frac{r_2 P_2^*}{L}$$

$$\begin{aligned} Q_2 = {} & \frac{r_1 r_2 P_1^* P_2^*}{KL} - \alpha\beta P_1^* P_2^* \\ & + (m_1 - \theta_1 f'(P_1^*))m P_1^* Z^* \\ & + (n_1 - \theta_2 g'(P_2^*))n P_2^* Z^* \end{aligned}$$

$$\begin{aligned} Q_3 = {} & \frac{r_1 n P_1^* P_2^* Z^* (n_1 - \theta_2 g'(P_2^*))}{K} \\ & + \frac{r_2 m P_1^* P_2^* Z^* (m_1 - \theta_1 f'(P_1^*))}{L} \\ & - \alpha n P_1^* P_2^* Z^* (m_1 - \theta_1 f'(P_1^*)) \\ & - \beta m P_1^* P_2^* Z^* (n_1 - \theta_2 g'(P_2^*)) \end{aligned}$$

Since $P_1^*, P_2^* > 0$ (from (3.5)) then $Q_1 > 0$. Now let us define

$$\begin{aligned} \delta^{(2)} &= Q_1 Q_2 - Q_3 \\ &= \left(\frac{r_1 P_1^*}{K} + \frac{r_2 P_2^*}{L}\right) P_1^* P_2^* \left(\frac{r_1 r_2}{KL} - \alpha\beta\right) \end{aligned}$$

$$+(n_1-\theta_2 g'(P_2^*))\left(\frac{r_2 n P_2^{*2} Z^*}{L}+\beta m P_1^* P_2^* Z^*\right)+(m_1-\theta_1 f'(P_1^*))\times\left(\frac{r_1 m P_1^{*2} Z^*}{K}+\alpha n P_1^* P_2^* Z^*\right)$$

Now $Q_3 > 0$ and $\delta^{(2)} > 0$ if conditions (3.6) are satisfied.

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

Appendix B

Now we observe that the positive interior equilibrium E^* exists if the following inequalities hold true:

$$N_1 < \gamma_2 < N_2$$

where

$$N_1 = \min\{L_1, L_2\}$$

and

$$N_2 = \frac{LB(\theta_2 - n_1 LB + \mu)}{n_1 LB - \mu}$$

where

$$L_1 = \frac{LA(\mu+\theta_1+\theta_2-m_1\gamma_1 - n_1\gamma_1 LA - 2n_1 LB) - m_1 LB}{m_1 + n_1 LA}$$

and

$$L_2 = \frac{LA\gamma_1(\theta_2 - n_1 LB + \mu) + LB(\mu+\theta_1+\theta_2 - m_1\gamma_1 - n_1\gamma_1 LA - n_1 LB)}{m_1\gamma_1 + n_1\gamma_1 LA + n_1 LB - \mu - \theta_1}$$

Also

$$\min\left\{\frac{\alpha L}{r_2}, \frac{r_1}{r_2}\right\} < \frac{m}{n} < \frac{r_1}{\beta K}$$

$$P_1^* < \frac{Kr_2(r_1 - \alpha L)}{r_1 r_2 - \alpha\beta KL}$$

The eigenvalues of the variational matrix V_0 of the system (4.1) are $\lambda_1 = r_1 > 0$, $\lambda_2 = r_2 > 0$, $\lambda_3 = -\mu < 0$. Clearly the plankton free steady state is unstable (saddle).

Further, the eigenvalues of the variational matrix V_1 around the equilibrium E_1 are $\lambda_1 = -r_1 < 0$, $\lambda_2 = r_2 - \beta K$ and $\lambda_3 = m_1 K - \mu - \frac{\theta_1 K}{\gamma_1 + K}$.

This steady state is unstable if $r_2 > \beta K$ (for existence of E^*).

Further, the eigenvalues of the variational matrix V_2 are $\lambda_1 = -r_2 < 0$, $\lambda_2 = r_1 - \alpha L$ and $\lambda_3 = n_1 L - \mu - \frac{\theta_2 L}{\gamma_2 + L}$. This steady state is unstable if $r_1 > \alpha L$ (for existence of E^*).

Further, the eigenvalues of the variational matrix V_3 are λ_1', λ_2' which are the roots of the equation

$$\lambda^2 + \lambda\left(\frac{r_1 P_1}{K} + \frac{r_2 P_2}{L}\right) + P_1 P_2 \frac{(r_1 r_2 - \alpha\beta KL)}{KL} = 0$$

and $\lambda_3' = m_1 P_1 + n_1 P_2 - \mu - \frac{\theta_1 P_1}{\gamma_1 + P_1} - \frac{\theta_2 P_2}{\gamma_2 + P_2}$. Clearly λ_1' and λ_2' have negative real parts. Now if $\lambda_3' > 0$, i.e., $m_1 P_1 + n_1 P_2 > \mu + \frac{\theta_1 P_1}{\gamma_1 + P_1} - \frac{\theta_2 P_2}{\gamma_2 + P_2}$ then E_3 is an unstable saddle.

Further, the eigenvalues of the variational matrix V_4 are λ_1'', λ_2'' which are the roots of the equation

$$\lambda^2 + \lambda\frac{r_1 P_1'}{K} + m P_1'\left(m_1 Z' - \frac{\theta_1\gamma_1 Z'}{(\gamma_1 + P_1')^2}\right) = 0$$

and $\lambda_3'' = r_2 - \beta P_1' - nZ'$. Clearly λ_1'' and λ_2'' have negative real parts. Now if $\lambda_3'' > 0$, i.e., $\frac{K(nr_1 - mr_2)}{nr_1 - m\beta K} < P_1'$ then E_4 is a unstable saddle.

Further, the eigenvalues of the variational matrix V_5 are λ_1''', λ_2''' which are the roots of the equation

$$\lambda^2 + \lambda\frac{r_2 P_2''}{L} + nP_2''Z''\left(n_1 - \frac{\theta_2\gamma_2}{(\gamma_2 + P_2'')^2}\right) = 0$$

and $\lambda_3''' = r_1 - \alpha P_2'' - mZ''$. Clearly λ_1''' and λ_2''' have negative real parts. Now if $\lambda_3''' > 0$, i.e., $\frac{L(mr_2 - nr_1)}{mr_2 - n\alpha L} < P_2''$ then E_5 is a unstable saddle.

References

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., 2004. Mathematical modelling of harmful algal blooms supported by experimental findings. Ecol. Complexity 1, 225–235.

Falkowski, P.G., 1984. The role of phytoplankton photosynthesis in global biogeochemical cycles. Photosynth. Res. 39, 235–258.

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-Verlag 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.

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

Shimizu, Y., 1989. Toxicology and pharmacology of red tides: an overview. In: Red Tides: Biology, Environmental Science and Toxicology. Proceedings of the First International Symposium on Red Tides. 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. Westview, pp. 257–307.

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

Yasumoto, T., Murata, M., 1993. Marine toxins. Chem. Rev. 93, 897–909.