Time lags can control algal bloom in two harmful phytoplankton–zooplankton system

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Abstract

A mathematical model consisting two harmful phytoplankton and zooplankton with discrete time lags in the mortality of zooplankton due to liberation of toxic substances by harmful phytoplankton has been considered. A stable co-existence of all the species has been observed for no-delay situation. Introduction of single delay in the system cause recurrent algal bloom and a threshold for the delay parameter has been estimated. Further, presence of multiple (two) delays control the oscillatory situation and can be used for termination of planktonic bloom. The analytical results as well as numerical simulations of our study lead to several threshold values for the delay parameters which play important roles in the marine ecological problems. An interesting observation is that multiple delay resolves plankton 'paradox' and establish a positive effect of competitive exclusion principle for stable co-existence of the species.

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Keywords: Harmful phytoplankton; Zooplankton; Single delay; Multiple delays; Oscillations; HAB; Bloom control

1. Introduction

In recent years, there has been considerable scientific attention towards Harmful Algal Bloom (HAB) and its control. Generally high nutrient levels and favorable conditions play a key role in rapid or massive growth of algae and low nutrient concentration, high predational pressure as well as other unfavorable conditions limit their growth, leading to oscillations or recurring bloom in the plankton ecosystem. A frequent outcome of planktonic bloom formation is massive cell lysis and rapid disintegration of large planktonic populations. This is closely followed by an equally rapid increase in bacterial numbers, and in turn by a fast deoxygenation of water, which could be detrimental to aquatic plants and animals. These bloom also reduce the chance of growth for aquatic vegetation. The adverse effect of harmful algal bloom is clear, but...
the control of such problem is under investigation. Hence studies regarding the pattern of bloom and its
control are necessary towards this serious ecological problem. 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 [1]. These fall into two general types: non-chemical effects which lead to star-
vation 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 bloom of large, relatively ungrazed species, such as *Ceratium*, are well
known. 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. 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. 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 signifi-
cantly from those regulating their bloom. The presence of toxic species does not necessarily lead to a del-
eterious impact; such effects require a threshold population density level, above which it becomes inimical.
However, the basic distinction between harmful and non-harmful bloom is not only warranted, but also it is
an important step toward classification of the various types of phytoplankton bloom which undoubtedly
occur.

Recent studies reveal that some times, bloom of certain harmful species leads to release of both toxins and
allelopathic substances, which affect the growth of other microorganisms [2,3]. Among marine algae, allelo-
pathy has been observed both in vitro and in situ [4–8], but the chemical nature and the role of allelopathic
compounds are still not known [9]. Allelopathic substances tend to be directly targeted and may physiologi-
cally impair, stun, repel, induce avoidance reactions, and kill grazers. Examples of these diverse models of
impact were given by [1]. Information on the recently discovered, remarkable ambush predatory behavior
of the harmful dinoflagellate, *Pfiesteria piscicida*, was presented by [10]. Reduction of grazing pressure of zoo-
 plankton due to release of toxic substances by phytoplankton is one of the most vital parameter in this context
[11–13]. Areas rich in some phytoplankton organisms e.g. *Phaeocystis, Coscinodiscus, Rhizosolenia* etc. are
unaccepeted/avoided by zooplankton due to dense concentration of phytoplankton or the production of some
toxic as well as unpleasant factors by them and this phenomena can be well explained by the ‘exclusion’ prin-
ciple [14,15]. Buskey and Stockwell [16] have demonstrated in their field studies that micro and meso zoo-
 plankton population are reduced during the bloom of a chrysophyte *Aureococcus anophagefferens* in the
southern Texas coast. Toxicity may be a strong mediator of zooplankton feeding rate, as shown in both field
studies [17,5,18] and laboratory studies [19–21]. These observations indicate that the toxic substance as well as
toxic phytoplankton plays an important role on the growth of the zooplankton population and have a great
impact on phytoplankton–zooplankton interactions.

Another important issue in this context is the stable co-existence of different species of phytoplankton and
zooplankton. Researchers had pointed out several reasons which can promote the stable co-existence of dif-
ferent competitive species, such as, temporal variation in the supply of a single resource [22,23]; inferior com-
petition leading to resistant to exploitation [24]; effect of inhibitory substances from external resources in
inhomogeneous environment [25]. These phenomena also support that there are additional mechanisms which
help the competing species to co-exist and they are suggested by several theoretical competition models and
experiments in laboratory [26–31]. In view of the competitive exclusion principle, the co-existence of large
number of phytoplankton and zooplankton species in a seemingly limited resources in natural water is
remarkable. Interactions between different allelopathic species of phytoplankton and their stable co-existence
with predators (zooplankton) are also an interesting field to study.

Modeling on plankton communities, specially on harmful algal bloom, co-existence of species, are very
rare in literature. Mathematical models always act as an useful tool to explore the features of the inter-
action between toxin-producing phytoplankton and zooplankton, and predict relationships that may be
looked for in experimental studies. Many models have already been built to simulate zooplankton–phyto-
plankton interaction. The complexity of such models prohibits a simple generic analysis of the effect on the system of predation on zooplankton. The process of production of toxic substances by phytoplankton species is still not clear. The release of toxic substances by phytoplankton may terminate the planktonic bloom, which is not yet well recognized but can not be ignored. Many authors have studied theoretically the interaction between two allelopathic species [32–37]. Recently, Sole et al. [9] have used the data obtained by Schimdt and Hansen [6], who conducted a laboratory experiment on plankton allelopathy in which 15 species of marine plankton were exposed to suspension of a toxic algae known as Cryosculina polyepis, and they have estimated the allelopathic parameters based on a model proposed by Chattopadhyay [33]. However, all the above studies do not explain several key features, such as, allelopathic interactions on the co-existence and persistence of phytoplankton species and its immediate effect on predators, control of recurring harmful algal bloom or oscillations and the effect of time lag required to release toxic substances.

Recently, Sarkar et al. [38] studied the role of two harmful phytoplankton population and observed that the occurrence of toxin-producing phytoplankton may not always be harmful but may help to maintain the stable equilibrium in trophodynamics through the co-existence of all the species. The results were supported by their experimental and mathematical observations. In this stage, we would like to mention that various combinations of predational functional response and toxin liberation process give rise to several interesting dynamics of the system [39]. In this study, we are mainly interested to present a mechanism for planktonic bloom in which the liberation of toxic substance or the effect of toxic phytoplankton is not an instantaneous process but is mediated by some time lag and can be helpful to reduce oscillations in the population and in turn, it is useful to maintain a stable co-existence of the species. The study of biological systems with time delays have been of considerable interest for a long time. There are also several reports that the zooplankton mortality due to the toxic phytoplankton bloom, occurs after some time lapse (see, www.mote.org, www.mds.gum.edu). Our mathematical and field observations [40] also suggest that the abundance of Paracalanus (zooplankton) population reduces after some time lapse of the bloom of toxic phytoplankton Noctiluca scintillans and this allows us some considerable freedom for considering the delay factor in the model construction. The model that we propose has three interacting components consisting of the two competitive 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. Now, to explain the periodic nature of bloom phenomena, we shall assume the mortality of zooplankton species due to toxic liberation by harmful phytoplanktons as a break-even process with discrete delay. The main objective of the article is to investigate the role of toxic allelopathy and corresponding delay parameter in maintaining the co-existence of the competitive phytoplankton species and their predator (zooplankton).

The organization of the paper is the following. In Section 2, we present the mathematical model for two different harmful phytoplankton and zooplankton where the reduction in the growth of zooplankton follows no delay due to the toxin liberation process. The formation of the model has its origin in the existing field experiments and in reported literatures, a brief description of which is given before the construction of the model. Next in Section 3, we modify this model using discrete time lag in the mortality of the predator due to liberation of toxin by one harmful phytoplankton species and finally in Section 4, we observe the situation when there are two time lags in the mortality of predators due to liberation of toxin from two harmful phytoplankton species. A brief numerical simulation study has been discussed in Section 5. The outcome of the analytical and numerical results are discussed in the conclusion along with the importance/significance and future directions of the work.

2. The mathematical model

The current study has been originated from the theoretical as well as experimental results on the interaction of harmful algal bloom and different types of phytoplankton–zooplankton interactions [41,40, 36–39,42]. Motivated from the literature and the field observations [38,42], a dynamic model consisting of two harmful phytoplankton and zooplankton has been proposed and the role of harmful phytoplankton in the termination of planktonic bloom has been observed. In our study, we try to observe the effects of harmful plankton in the dynamics of two harmful phytoplankton–zooplankton interaction with
an additional factor that the release of toxic substance by toxic–phytoplankton species reduces the growth of zooplankton. From the list [38,42] of significant number of toxin phytoplankton and zooplankton species, we chose the toxin-producing plankton species as *N. scintillans* and *Chaetoceros* sp., and zooplankton species as *Paracalanus* sp. (the taxonomical and functional distinctions of the species *N. scintillans* and *Paracalanus* sp. are given in [41,36,38], and *Chaetoceros* sp. belongs to the group diatom). From the field study [37,38,42] we observed that when any one of the harmful phytoplankton is absent, the equilibrium level of both harmful phytoplankton and zooplankton are much higher than the value observed when both harmful phytoplankton are present. This phenomenon depicts the fact that the presence of two harmful phytoplankton in the system reduces the high abundance of harmful phytoplankton and the zooplankton population. Further, the biomass distribution observed in the field study demonstrates that the introduction of two harmful phytoplankton leads to the stable co-existence of all the species through the termination of bloom and can be used as a controlling agent for the stability of marine ecosystem. In the present study we modify the model proposed by Sarkar et al. [37,38] assuming the mortality process of the predator due to toxin liberation by harmful phytoplanktons as a break-even point by discrete delay and investigate the effect of delays to control bloom/oscillations.

Let $P_1(t)$ and $P_2(t)$ be the concentrations of the two harmful phytoplankton and $Z(t)$ be the concentration of zooplankton at time $t$. Let $r_1$ and $r_2$ be the growth rates of the harmful phytoplankton respectively and $k$ be the environmental carrying capacity, which is common for both the plankton species and can be interpreted as competition between the plankton due to limited resource. Let $\beta_1$ and $\beta_2$ be the maximum zooplankton ingestion rates for both the harmful phytoplankton species; $\gamma_1$ and $\gamma_2$ be the maximum zooplankton conversion rates. Let $a$ be the natural death rate of zooplankton. Let $x_1$ and $x_2$ be the inhibitory effects of the two competing harmful phytoplankton; $\theta_1$ and $\theta_2$ be the rates of toxin liberation by the harmful phytoplankton respectively, which reduces the growth of zooplankton. It is not usually possible to know the past history of the release of toxic substances by phytoplankton or the actual form of the delay kernel. So a particular member of the family of kernels is at best an approximation. In our previous study [40] we have observed that, cyclic nature of bloom can not be explained by distributed delay kernels (gamma distribution) for distribution of toxic substance or toxic phytoplankton. The prediction based on the system involving distributed delay illustrates that concentration of toxic substances or toxic phytoplankton, eventually approach equilibrium concentration and hence no periodic solutions are possible. To search excitability (and/or) cyclic nature of bloom in the system we now assume the process of toxic liberation as a break-even point by discrete delay and subsequently denote $\tau_1$, $\tau_2$ as the discrete time lags in the mortality of zooplankton due to liberation of toxic substances by two harmful phytoplankton $P_1$, $P_2$ respectively.

The mathematical model is

\[ \begin{align*}
\frac{dP_1}{dt} &= r_1 P_1 \left(1 - \frac{P_1}{k}\right) - x_1 P_1 P_2 - \beta_1 P_1 Z, \\
\frac{dP_2}{dt} &= r_2 P_2 \left(1 - \frac{P_2}{k}\right) - x_2 P_1 P_2 - \beta_2 P_2 Z, \\
\frac{dZ}{dt} &= (\gamma_1 P_1 + \gamma_2 P_2) Z - aZ - \theta_1 P_1 (t - \tau_1) Z - \theta_2 P_2 (t - \tau_2) Z.
\end{align*} \]

System (1) has to be analyzed with the following initial conditions: $P_1(0) \geq 0$, $P_2(0) \geq 0$, $Z(0) \geq 0$.

In our subsequent analysis we first study the stability analysis for the non-delay model (that is, for $\tau_1 = 0$ and $\tau_2 = 0$) and then continue the study for the delay models.

2.1. Linear stability analysis of the non-delay model

The equilibrium points of system (1) are

1. The trivial equilibrium point: $E_T \equiv (0,0,0)$,
2. The axial equilibrium points: $E_{A_1} \equiv (k,0,0)$ and $E_{A_2} \equiv (0,k,0)$,
3. The boundary equilibrium points:
Lemma 2. There exists two steady states

\[ E_{B_1} = \left\{ \begin{array}{c}
  r_2 k (r_1 - x_1 k) \quad r_1 k (r_2 - k x_2) \\
r_1 r_2 - x_1 x_2 k^2 \quad r_1 r_2 - x_1 x_2 k^2
\end{array} \right\}, \]

\[ E_{B_2} = \left\{ \begin{array}{c}
  0 \quad \frac{r_2}{(\gamma_2 - \theta_2)} \quad r_2 \left( 1 - \frac{a}{k (\gamma_2 - \theta_2)} \right)
\end{array} \right\}, \]

\[ E_{B_3} = \left\{ \begin{array}{c}
  \frac{a}{(\gamma_1 - \theta_1)} \quad 0 \quad \frac{r_1}{k (\gamma_1 - \theta_1)}
\end{array} \right\}. \]

Note. It is easy to check that the boundary equilibrium point \( E_{B_1} \) exist if the inhibitory effects of both the harmful phytoplankton are lower than certain thresholds, that is, if \( x_1 < \frac{a}{k} \), \( x_2 < \frac{a}{k} \). The equilibrium points \( E_{B_2} \) and \( E_{B_3} \) exist if \( \theta_i < \gamma_i - \frac{a}{k} \) \((i = 1, 2)\), that is, if the toxic liberation rate for each harmful phytoplankton does not exceed certain threshold then one phytoplankton and zooplankton exist.

4. The unique interior equilibrium point: \( E^* \equiv (P_1^*, P_2^*, Z^*) \), where

\[ P_1^* = \frac{a (\beta_1 r_2 - x_1 \beta_2 k) + k (\beta_2 r_1 - \beta_1 r_2) (\gamma_2 - \theta_2)}{(\beta_1 r_2 - x_1 \beta_2 k) (\gamma_1 - \theta_1) + (\beta_2 r_1 - x_2 \beta_1 k) (\gamma_2 - \theta_2)}, \]

\[ P_2^* = \frac{a (\beta_2 r_2 - x_1 \beta_2 k) - k (\beta_2 r_1 - \beta_1 r_2) (\gamma_1 - \theta_1)}{(\beta_1 r_2 - x_1 \beta_2 k) (\gamma_1 - \theta_1) + (\beta_2 r_1 - x_2 \beta_1 k) (\gamma_2 - \theta_2)}, \]

\[ Z^* = \frac{k ((r_1 - x_1 k) (\gamma_1 - \theta_1) + (r_2 - x_2 k) (\gamma_2 - \theta_2)) - a (r_1 r_2 - x_1 x_2 k^2)}{k ((\beta_1 r_2 - x_1 \beta_2 k) (\gamma_1 - \theta_1) + (\beta_2 r_1 - x_2 \beta_1 k) (\gamma_2 - \theta_2))}. \]

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

\[ x_1 < \min \left\{ \frac{r_2 \beta_1}{\beta_2 k} + \frac{r_1 \beta_2 - r_2 \beta_2 (\gamma_2 - \theta_2)}{\beta_2 k} \frac{c_1 k}{\gamma_2 - \theta_2} + \frac{c_2 k}{\gamma_2 - \theta_2} \right\}, \]

\[ x_2 < \min \left\{ \frac{r_2 (\gamma_1 - \theta_1)}{\gamma_2 - \theta_2} + \frac{c_2 k}{\gamma_2 - \theta_2} \right\}, \]

\[ \frac{r_2 (\gamma_2 - \theta_2)}{\gamma_2 - \theta_2} + \frac{c_1 k}{\gamma_2 - \theta_2} - \frac{r_1 \beta_2}{\beta_2 k} \frac{c_2 k}{\gamma_2 - \theta_2} - \frac{r_1 \beta_2}{\beta_2 k} \frac{c_1 k}{\gamma_2 - \theta_2} \right\}. \]

provided \( \gamma_1 > \theta_1, \gamma_2 > \theta_2 \).

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 1. The steady state \( E_T = (0, 0, 0) \) of system (1) is an unstable saddle point.

Lemma 2. There exists two steady states \( E_{A_1} = (k, 0, 0) \) and \( E_{A_2} = (0, k, 0) \); which are feasible (one harmful phytoplankton and zooplankton free state) and are unstable saddle.

Lemma 3. There exists a zooplankton free steady state \( E_{B_1} \) which is unstable saddle if \( \frac{a_1}{a_2} < \frac{a_1}{a_2} \).

Lemma 4. There exists two steady states \( E_{B_2} \) and \( E_{B_3} \) which are feasible (one harmful phytoplankton free state only) and are stable.

Note. The behavior of the steady states \( E_{B_2} \) and \( E_{B_3} \) are same as the behavior of the positive interior equilibrium of the system in Chattopadhyay et al. [41] when the functional forms for both predational response and toxin liberation process are taken as Holling type I. But it is interesting to note that if we compare the
equilibrium levels of the species in \( (E_{B1}, E^*) \) and \( (E_{B2}, E^*) \) then the existence condition (2) of the positive interior equilibrium \( E^* \) implies that the equilibrium level of individual harmful phytoplankton population in \( E_{B2} \) and \( E_{B3} \) are much higher than the equilibrium level of the species in \( E^* \). This observation clearly indicate our field observations and we may interpret this situation as planktonic bloom due to high abundance of the species for presence of single harmful phytoplankton. Moreover, existence of \( E^* \) implies that the control of planktonic bloom may be observed through reduction of high abundance of the species due to co-existence of two harmful phytoplankton.

Next, we perform a stability study of the interior equilibrium point \( E^* \). The characteristic equation of the community matrix corresponding to the linearized version of (1) at \( E^* \) is

\[
\lambda^3 + A\lambda^2 + B\lambda + C = 0,
\]

where \( A = \frac{r_1 P_1}{k} + \frac{r_2 P_2}{k} \) (> 0), \( B = \left[\frac{r_1 P_1}{k} - \alpha_1 x_2\right] P_1 P_2 + \beta_2 (\gamma_2 - \theta_2) P_2^2 Z^* + \beta_1 (\gamma_1 - \theta_1) P_1 Z^* \), and \( C = \left[\frac{r_3 P_3}{k} (\gamma_2 - \theta_2) + \frac{r_2 P_2}{k} (\gamma_1 - \theta_1) - \alpha_2 (\gamma_2 - \theta_2) - \alpha_1 (\gamma_1 - \theta_1)\right] P_2^2 Z^* \).

Now we observe that \( C \) is positive from the existence condition (2) and \( AB - C \) is positive if \( \alpha_1 < \frac{\beta_1}{k}, \alpha_2 < \frac{\beta_2}{k} \) and \( \gamma_1 > \theta_1, \gamma_2 > \theta_2 \). Thus from the Routh–Hurwitz criteria for the stability of the system (1) around the positive interior equilibrium \( E^* \), we obtain the following theorem:

**Theorem 1.** System (1) is locally asymptotically stable around the positive interior equilibrium \( E^* \) if the existence condition (2) hold and the inequalities \( \alpha_1 < \frac{\beta_1}{k}, \alpha_2 < \frac{\beta_2}{k} \) are satisfied.

**Note.** From the stability analysis of the positive interior equilibrium \( E^* \), we observe that the system (1) is stable if the rates of inhibition due to competition between two harmful phytoplankton are less than certain upper thresholds. Moreover, all the three species persist if there is a competition between the harmful phytoplanktons and the thresholds so obtained may be used as controlling agent for termination of planktonic bloom.

Now, to observe the effects of time lag on the system we modify the system introducing, first, single delay in the mortality of zooplankton due to toxin liberation process for each harmful phytoplankton, and, second, two delays simultaneously in the mortality of zooplankton due to liberation of toxin by both the harmful phytoplankton.

### 3. Models with single delay

We first analyze the ODE model (1) with one delay \( \tau_1 \) (delayed mortality of zooplankton due to toxin production of \( P_1 \)). The corresponding model is

\[
\frac{dP_1}{dt} = r_1 P_1 \left(1 - \frac{P_1}{k}\right) - \alpha_1 P_1 P_2 - \beta_1 P_1 Z, \\
\frac{dP_2}{dt} = r_2 P_2 \left(1 - \frac{P_2}{k}\right) - \alpha_2 P_1 P_2 - \beta_2 P_2 Z, \\
\frac{dZ}{dt} = (\gamma_1 P_1 + \gamma_2 P_2) Z - aZ - \theta_1 P_1 (t - \tau_1) Z - \theta_2 P_2 Z.
\]

Linearizing the system about the interior equilibrium point we obtain

\[
\frac{du}{dt} = a_{11} u + a_{12} v + a_{13} w, \\
\frac{dv}{dt} = a_{21} u + a_{22} v + a_{23} w, \\
\frac{dw}{dt} = \gamma_1 Z^* u + (\gamma_2 - \theta_2) Z^* v - \theta_1 Z^* u (t - \tau_1),
\]
where

\[ u = P_1 - P_1^*; \quad v = P_2 - P_2^*; \quad w = Z - Z^* , \]

\[ a_i = -\frac{r_i P_i^*}{k} \quad (i = 1, 2); \quad a_{12} = -\alpha_1 P_1^*; \quad a_{13} = -\beta_1 P_1^* , \]

\[ a_{21} = -\alpha_2 P_2^*; \quad a_{23} = -\beta_2 P_2^* . \]

The characteristic equation for the linearized system around the point \((P_1^*, P_2^*, Z^*)\) is given by

\[ Q_1(\lambda) + Q_2(\lambda)e^{-\lambda t_1} = 0 , \tag{5} \]

where

\[ Q_1(\lambda) = \lambda^3 + p_1 \lambda^2 + p_2 \lambda + p_3 , \]

\[ Q_2(\lambda) = q_2 \lambda + q_3 , \]

\[ p_1 = \frac{r_1 P_1^* + r_2 P_2^*}{k} (> 0) , \]

\[ p_2 = \frac{r_1 r_2}{k^2} - \alpha_1 \alpha_2 P_1^* P_2^* + Z^* \left\{ \beta_1 P_1^* \gamma_1 + \beta_2 P_2^* (\gamma_2 - \theta_2) \right\} , \]

\[ p_3 = P_1^* P_2^* Z^* \left\{ \gamma_1 \left( \frac{r_2 \beta_1}{k} - \alpha_1 \beta_2 \right) + (\gamma_2 - \theta_2) \left( \frac{r_1 \beta_2}{k} - \alpha_2 \beta_1 \right) \right\} , \]

\[ q_1 = 0 , \]

\[ q_2 = -\beta_1 \theta_1 P_1^* Z^* , \]

\[ q_3 = -\theta_1 (\beta_1 r_2/k - \alpha_1 \beta_2) P_1^* P_2^* Z^* . \]

**Theorem 2.** If \(a_1 < \omega_0^2 < a_2\), where

\[ a_1 = \frac{p_1 q_2 + p_2 q_3 + \sqrt{(p_1 q_2 + p_2 q_3)^2 - 4(p_1 + q_2)}}{2(p_1 + q_2)} , \quad a_2 = \frac{p_1 q_2 + p_2 q_3 + \sqrt{(p_1 q_2 + p_2 q_3)^2 + 4(p_1 + q_2)}}{2(p_1 + q_2)} \]

and \(\omega_0\) is the positive root of

\[ \omega^6 + A_1 \omega^4 + A_2 \omega^2 + A_3 = 0 , \]

then the system will undergo a stability change for an infinite number of values of \(\tau_1\) say, \(\tau_1^*\), where

\[ A_1 = p_1^2 - 2p_2 - q_1^2 , \]

\[ A_2 = p_2^2 - q_2^2 - 2p_1 q_3 , \]

\[ A_3 = p_3^2 - q_3^2 = (p_3 + q_3)(p_3 - q_3) , \]

and

\[ \tau_1^* = \frac{1}{\omega_0} \arccos \left[ \frac{(p_1 \omega^2 - p_3)q_3 \omega^2 + (\omega^3 - p_2 \omega)(q_2 \omega)}{(q_3 \omega)^2 + (q_2 \omega)^2} \right] + \frac{2n\pi}{\omega_0} . \tag{6} \]

**Proof.** For a stability change the characteristic equation should have a purely imaginary root. Substituting \(\lambda = i\omega\) in (5) and separating real and imaginary parts, we obtain the following system of transcendental equations:

\[ p_1 \omega^2 - p_3 = q_3 \cos(\omega \tau_1) + q_2 \omega \sin(\omega \tau_1) , \]

\[ \omega^3 - p_2 \omega = q_2 \omega \cos(\omega \tau_1) - q_3 \sin(\omega \tau_1) . \tag{7} \]

Squaring and adding the equations in (7) we get

\[ q_1^2 + q_2^2 \omega^2 = (p_1 \omega^2 - p_3)^2 + (\omega^3 - p_2 \omega)^2 , \]

\[ q_3^2 + q_2^2 \omega^2 = (p_1 \omega^2 - p_3)^2 + (\omega^3 - p_2 \omega)^2 . \]
which implies,
\[ \omega^6 + A_1\omega^4 + A_2\omega^2 + A_3 = 0. \]  
(8)

Eliminating \( \sin \omega \tau_1 \) from (7) we get

\[ \cos(\omega \tau_1) = \frac{(p_1\omega^2 - p_3)q_3\omega + (\omega^3 - p_2\omega)q_2\omega}{(q_3\omega)^2 + (q_2\omega)^2}. \]

But for real \( \tau_1 \), \( |\cos \omega \tau_1| < 1 \). This condition holds for a positive root of Eq. (8), say, \( \omega_0 \), if \( \omega_0 \) lies within the range given in the Theorem 2. The corresponding \( \tau_1^\ast_n \) is given by (6). Therefore for these values of \( \tau_1n \), the system undergoes a stability change. \( \Box \)

Similarly, it can be shown that if we consider the toxication delay for the phytoplankton species \( P_2 \) only, the system will undergo a stability change for \( \tau_2 = \tau_2^\ast_n \), where

\[ \tau_2^\ast_n = \frac{1}{\omega_1} \arccos \left[ \frac{(m_1\omega_1^2 - m_3)n_3\omega_1^2 + (\omega_1^3 - m_2\omega_1)(n_2\omega_1)}{(n_3\omega_1^2)^2 + (n_2\omega_1)^2} \right] + \frac{2\pi n}{\omega_1}, \]  
(9)

where

\[ m_1 = \frac{r_1P_1^\ast}{k} + \frac{r_2P_2^\ast}{k} (> 0), \]
\[ m_2 = \left( \frac{r_1r_2}{k^2} - \alpha_1\alpha_2 \right)P_1^\ast P_2^\ast + Z^\ast \left\{ \beta_2P_2^\ast \gamma_2 + \beta_1P_1^\ast (\gamma_1 - \theta_1) \right\}, \]
\[ m_3 = P_1^\ast P_2^\ast Z^\ast \left\{ \gamma_2 \left( \frac{r_1\beta_2}{k} - \alpha_2\beta_1 \right) + (\gamma_1 - \theta_1) \left( \frac{r_2\beta_1}{k} - \alpha_1\beta_2 \right) \right\}, \]
\[ n_1 = 0, \]
\[ n_2 = -\beta_2\theta_2 P_2^\ast Z^\ast, \]
\[ n_3 = -\theta_2(\beta_2r_1/k - \alpha_2\beta_1)P_1^\ast P_2^\ast Z^\ast, \]

and \( \pm i\omega_1 \) are the purely imaginary roots of the corresponding characteristic Eq. (5).

Note. The difference between \( \tau_1^\ast_n \) and \( \tau_2^\ast_n \) mainly depends on the rates of toxication of the two phytoplankton species. The above study depicts the recurrent occurrence of bloom phenomenon in the presence of single delay and helps us to estimate the critical value for the delay parameter to control the oscillations. Next, we will try to observe the effect of two delays in the system and subsequently will try to estimate the threshold values for both the delays to control bloom.

4. Model with multiple delays

We consider the model with delayed mortality due to toxin liberation by both the harmful species. The corresponding linearized system is

\[
\begin{align*}
\frac{du}{dt} &= a_{11}u + a_{12}v + a_{13}w, \\
\frac{dv}{dt} &= a_{21}u + a_{22}v + a_{23}w, \\
\frac{dw}{dt} &= \gamma_1 Z^* u + \gamma_2 Z^* v - \theta_1 Z^* u(t - \tau_1) - \theta_2 Z^* v(t - \tau_2).
\end{align*}

(10)

Let us now try to estimate the length of the delay which preserves the stability of the system. We consider system (10) and the space of real-valued continuous functions defined on \((\tau, \infty)\), where \( \tau = \min\{\tau_1, \tau_2\} \) and satisfying the initial conditions.
Let $\tilde{u}(P)$, $\tilde{v}(P)$ and $\tilde{w}(P)$ be the Laplace transforms of $u(t)$, $v(t)$ and $w(t)$ respectively. Taking Laplace transform of (10) we have

\begin{equation}
(P - a_{11})\tilde{u} = a_{12}\tilde{v} + a_{13}\tilde{w} + u(0),
\end{equation}

\begin{equation}
(P - a_{22})\tilde{v} = a_{21}\tilde{u} + a_{23}\tilde{w} + v(0),
\end{equation}

\begin{equation}
P\tilde{w} = \gamma_1 Z^*\tilde{u} + \gamma_2 Z^*\tilde{v} - \theta_1 Z^*e^{-P\tau_1}(\tilde{u} + K_1(P)) - \theta_2 Z^*e^{-P\tau_2}(\tilde{v} + K_2(P)) + w(0),
\end{equation}

where $K_1(P) = \int_{\tau_1}^{P} e^{-P\tau}u(\tau)\,d\tau$ and $K_2(P) = \int_{\tau_2}^{P} e^{-P\tau}v(\tau)\,d\tau$.

Rearranging we get

\begin{equation}
H(P) = (P^3 + AP^2 + B_2P + C_2 + D_2Pe^{-P\tau_1} + E_2Pe^{-P\tau_2} + F_2e^{-P\tau_1} + G_2e^{-P\tau_2})\tilde{u}
\end{equation}

\begin{equation}
= -(a_{12}a_{23} - a_{13}a_{22})\{\theta_1 Z^*e^{-P\tau_1}K_1(P) + \theta_2 Z^*e^{-P\tau_2}K_2(P)\}
\end{equation}

\begin{equation}
= \left[ \frac{1}{a_{23}}(a_{23}a_{12} - a_{22}a_{13})P + \frac{a_{13}}{a_{23}}(P^2 - Pa_{22} - a_{23}\gamma_2 Z^* + a_{23}\theta_2 Z^*e^{-P\tau_2}) \right] + (P^2 - Pa_{22} - a_{23}\gamma_2 Z^* + a_{23}\theta_2 Z^*e^{-P\tau_2})u(0) + \{a_{13}(P - a_{22}) + a_{23}a_{12}\}w(0),
\end{equation}

where

\begin{equation}
B_2 = \frac{r_1r_2P_1P_2^2}{K^2} - \alpha_1\alpha_2P_1P_2 + \beta_1\gamma_1P_1Z^* + \beta_2\gamma_2P_2Z^*,
\end{equation}

\begin{equation}
C_2 = P_1P_2Z^*\left[ \frac{r_1\beta_2\gamma_2}{k} + \frac{r_2\beta_1\gamma_1}{k} - \alpha_1\gamma_1\beta_2 - \alpha_2\gamma_2\beta_1 \right],
\end{equation}

\begin{equation}
D_2 = -\beta_1\gamma_1 P_1Z^*; \quad E_2 = -\beta_2\gamma_2 P_2Z^*,
\end{equation}

\begin{equation}
F_2 = P_1P_2Z^*\left[ \frac{r_2\beta_1\gamma_1}{k} - \alpha_2\beta_1 \right],
\end{equation}

\begin{equation}
G_2 = P_1P_2Z^*\left[ \frac{r_1\beta_2\gamma_2}{k} + \alpha_1\beta_2 \right].
\end{equation}

The condition for stability of the system is

\begin{equation}
\text{Im}(H(i\mu_0)) > 0,
\end{equation}

\begin{equation}
\text{Re}(H(i\mu_0)) = 0,
\end{equation}

where $\mu_0$ is the smallest positive root of $\text{Re}(H(i\mu)) = 0$.

Now,

\begin{equation}
H(i\mu) = -i\mu^3 - A\mu^2 + B_2i\mu + C_2 + iD_2\mu(\cos\mu\tau_1 - i\sin\mu\tau_1) + iE_2\mu(\cos\mu\tau_2 - i\sin\mu\tau_2)
\end{equation}

\begin{equation}
+ F_2(\cos\mu\tau_1 + i\sin\mu\tau_1) + G_2(\cos\mu\tau_2 + i\sin\mu\tau_2).
\end{equation}

So from (14) we have

\begin{equation}
\mu_0^3 - B_2\mu_0 - D_2\cos\mu_0\tau_1 + F_2\sin\mu_0\tau_1 - E_2\mu_0\cos\mu_0\tau_2 + G_2\sin\mu_0\tau_2 < 0,
\end{equation}

\begin{equation}
\mu_0^2 - C_2 = D_2\mu_0\sin\mu_0\tau_1 + F_2\cos\mu_0\tau_1 + E_2\mu_0\sin\mu_0\tau_2 + G_2\cos\mu_0\tau_2.
\end{equation}

Next, we want to find an upper bound for $\mu$ (say, $\mu_+$), independent of $\tau_1, \tau_2$ such that (14) holds for all $\mu$, $0 \leq \mu \leq \mu_+$ and hence in particular for $\mu_0$, when $\mu_0 \leq \mu_+$.

Maximizing

\begin{equation}
A\mu^2 - C_2 = D_2\mu\sin\mu\tau_1 + F_2\cos\mu\tau_1 + E_2\mu\sin\mu\tau_2 + G_2\cos\mu\tau_2,
\end{equation}

with $|\sin\mu\tau_1| \leq 1$, $|\cos\mu\tau_1| \leq 1$, $|\sin\mu\tau_2| \leq 1$, $|\cos\mu\tau_2| \leq 1$, we get

\begin{equation}
A\mu^2 - D_2\mu - (C_2 + |F_2|) - |E_2|\mu - |G_2| = 0.
\end{equation}
If \( \mu_+ \) is a positive root of (16), then
\[
\mu_+ = \frac{1}{2A} \left[ (|D_2| + |E_2|) + \sqrt{(|D_2| + |E_2|)^2 + 4A(C_2 + |E_2| + |G_2|)} \right].
\]

Obviously, \( \mu_+ \geq \mu_0 \). So we can find an upper bound of \( \mu \) for which (14) holds. Now for stability of the system
\[
\begin{align*}
|D_2|\mu_+^2 \tau_1 + |E_2|\mu_+^2 \tau_2 + |F_2| + |G_2| + \frac{1}{2}A|D_2|\mu_+^2 \tau_1^2 + \frac{1}{2}A|E_2|\mu_+^2 \tau_2^2 + A|F_2|\tau_1 + A|G_2|\tau_2 \\
< A(B_2 + D_2 + E_2) - C_2 \equiv \Gamma_1.
\end{align*}
\]

But
\[
\begin{align*}
\frac{1}{2}A|D_2|\mu_+^2 \tau_1^2 + (|D_2|\mu_+^2 + A|F_2|)\tau_1 - \frac{\Gamma_1}{2} + |F_2| + \frac{1}{2}A|E_2|\mu_+^2 \tau_2^2 + (|E_2|\mu_+^2 + A|G_2|)\tau_2 + |G_2| - \frac{\Gamma_1}{2} \\
< \frac{1}{2}A|D_2|\mu_+^2 \tau_1^2 + (|D_2|\mu_+^2 + A|F_2|)\tau_1 + |F_2| - \frac{\Gamma_1}{2} + \frac{1}{2}A|E_2|\mu_+^2 \tau_2^2 + (|E_2|\mu_+^2 + A|G_2|)\tau_2 + |G_2| - \frac{\Gamma_1}{2}.
\end{align*}
\]

So if \( \tau_1^{d} \) and \( \tau_2^{d} \) are large for large values of \( |F_2| \) and \( |G_2| \) which in turn depend on the different system parameters.

5. Numerical results

Now, we compare qualitatively the dynamical patterns observed in our field study with our model formulation. For this we substantiate our analytical findings through numerical simulations considering the parameter values from the literature [37]: \( r_1 = 2.5, k = 20, \Delta_1 = .01, \beta_1 = .6, r_2 = 2.3, \Delta_2 = .02, \beta_2 = .35, \gamma_1 = .04, \gamma_2 = .3, \theta_1 = .06, \theta_2 = .07, a = .1 \). For this set of parameter values we observe that one harmful phytoplankton free equilibrium as \( E_d(0.29,0.411) \), the other as \( E_0(0.43,4.09) \) and the positive interior equilibrium is \( E_0(0.15,0.21,4.03) \). This clearly demonstrate the termination of planktonic bloom through reduction of equilibrium sizes due to presence of two harmful phytoplankton. Our numerical results also show the co-existence of all the species and supports our observation from the field studies [41,36–38]. This also justifies our choice for this particular parameter set. We will now try to find the effect of single and multiple delays on the dynamics of the system.

With the same set of parameters, we find that \( A_1 < 0, A_2 < 0 \) and \( A_3 > 0 \), then by Descartes rule of signs there exists two positive roots \( \omega_1 = (-0.490976) \) and \( \omega_2 = (-0.527829) \), satisfying (8). According to Theorem 3.1 [43], the stability of \( E^* \) changes a finite number of times as \( \tau \) is increased and eventually becomes unstable for sufficiently large values of \( \tau \).

We use MATLAB dde23 to solve the nonlinear delay differential equation. We first see the effect of a single delay in toxic release (for the term \( \theta_1P_1(t - \tau_1)Z \)). Fig. 1A gives the stable dynamics for both harmful phytoplankton and zooplankton population, for \( \tau_1 = 5.0 \), initial condition being \( P_1(0) = 0.25, P_2(0) = 0.2, Z(0) = 4 \). Fig. 1B shows periodic solutions for all the three populations for \( \tau_1 = 7.43 \), and for this value of \( \tau_1 \) the system shows stable limit cycle (Fig. 1C). Similar dynamics is observed in Fig. 2A–C (for the term \( \theta_2P_2(t - \tau_2)Z \)). The values of \( \tau_2 \) are respectively 5.0 and 6.73. We now consider the case when both the delays are present. Without taking the values arbitrarily, we fixed the value of \( \tau_1 = 7.43 \) (in this case we observed oscillations in the system depicting recurrent bloom situation) and gradually increase the value of \( \tau_2 \). Fig. 3A shows stable dynamics for \( \tau_1, \tau_2 = (7.43,4) \). The system shows periodic solutions for \( \tau_1, \tau_2 = (7.43,5.28) \). Similar dynamics are noticed when the values of \( \tau_1, \tau_2 \) are \( (4,6.73) \) and \( (5.0,6.73) \) respectively (see Fig. 4A–C).
Fig. 1. The time evolution for two harmful phytoplankton and one zooplankton population with single delay. (A) Stable dynamics of all three populations for $s_1 = 5.0$. (B) Oscillations in all three populations for $s_1 = 7.43$. (C) Stable limit cycle is observed at $s_1 = 7.43$. The parameter set and initial conditions are given in the text.

Fig. 2. The time evolution for two harmful phytoplankton and one zooplankton population with single delay. (A) Stable dynamics is observed in all the three populations for $s_2 = 5.0$. (B) Oscillations in all three populations for $s_2 = 6.73$. (C) Stable limit cycle is observed at $s_2 = 6.73$. The parameter set and initial conditions are given in the text.
Fig. 3. The time evolution for two harmful phytoplankton and one zooplankton population with two delays. (A) Stable dynamics is observed in all the three populations for \((\tau_1, \tau_2) = (7.43, 4)\). (B) The system shows periodic solutions for \((\tau_1, \tau_2) = (7.43, 5.28)\). (C) Stable limit cycle is observed at \((\tau_1, \tau_2) = (7.43, 5.28)\). The parameter set and initial conditions are given in the text.

Fig. 4. The time evolution for two harmful phytoplankton and one zooplankton population with two delays. (A) Stable dynamics for all the three populations for \((\tau_1, \tau_2) = (4, 6.73)\). (B) The system shows periodic solutions for \((\tau_1, \tau_2) = (5.00, 6.73)\). (C) Stable limit cycle is observed at \((\tau_1, \tau_2) = (5.00, 6.73)\). The parameter set and initial conditions are given in the text.
Though 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 about the estimation of the parameter values. The 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 positive impact for termination of planktonic bloom. It is interesting to note that introduction of single delay in the system leads to oscillations depicting the recurrent bloom situation. Moreover, time lag due to toxin liberation by both the harmful species may be helpful to control such oscillatory behavior. Our numerical results provide certain threshold values for the delay parameters \( (r_1, r_2) \) for which we can maintain a stable situation for all the species and can control bloom dynamics. These threshold values can be obtained from our analytical expressions by substituting the parameter values and provide an useful insight to control planktonic bloom.

6. Conclusion

In the previous studies, researchers have established with the help of experimental results and mathematical modeling, that toxin-producing phytoplankton may be used as a controlling agent for the termination of planktonic bloom. But those studies do not contain the presence of two harmful phytoplankton in such situation. Moreover, the effects of time lags due to liberation of toxic substances can not be ignored in this context.

In this paper we have proposed and analyzed a three component model consisting of two harmful phytoplankton and zooplankton. We have studied the stability behavior of the system around the feasible steady states. 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 co-existence of all the three species.

Further, we observe that when there is a single time lag present in the mortality of zooplankton due to toxin liberation by one harmful phytoplankton, then, for certain critical value for the delay parameter recurrent bloom occur and similar phenomenon is observed for the other harmful phytoplankton (HP). This phenomenon can be interpreted as a ‘paradox’ in marine ecology where delayed mortality of the predator seems to enhance predator population and reduce prey population, but here it leads to oscillations with high abundance of prey population. This is interesting because competitive exclusion principle then plays a major role and co-existence (though oscillatory in nature) of several prey–predator population are possible. But presence of multiple (two) time lags in the mortality of zooplankton due to toxin liberation by both the harmful phytoplankton reduce the oscillations in the system and further stable co-existence is possible. This gives a strong base to the competitive exclusion principle and resolves the plankton ‘paradox’. Our analytical and numerical study reveal that control of recurrent bloom in the plankton ecosystem is possible due to the presence of two HP. The above findings clearly demonstrate the role of time lag in the mortality of zooplankton due to toxin liberation by two HP and also in the termination of planktonic bloom. It is to be noted here 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 bloom. In this work (in presence of two harmful phytoplankton) we also arrived at the same conclusion. Thus we may finally conclude that harmful phytoplankton may be used as a bio-control agent for the Harmful Algal Bloom (HAB) problems. The role of environmental fluctuations in the two harmful phytoplankton–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 bloom and its possible control mechanism is still in infancy, hence the progress of such important areas urgently requires special attention both from experimental and mathematical ecologists.

Acknowledgement

R.R. Sarkar acknowledges support from the Department of Science and Technology, Ministry of Science and Technology, Government of India (Grant No. SR/FTP/MS-06/2005).
References


