Nutrient-limited toxin production and the dynamics of two phytoplankton in culture media: A mathematical model

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A B S T R A C T

In this paper we have proposed and analyzed a simple mathematical model consisting of four variables, viz., nutrient concentration, toxin producing phytoplankton (TPP), non-toxic phytoplankton (NTP), and toxin concentration. Limitation in the concentration of the extracellular nutrient has been incorporated as an environmental stress condition for the plankton population, and the liberation of toxic chemicals has been described by a monotonic function of extracellular nutrient. The model is analyzed and simulated to reproduce the experimental findings of Graneli and Johansson [Graneli, E., Johansson, N., 2003. Increase in the production of allelopathic *Prymnesium parvum* cells grown under N- or P-deficient conditions. Harmful Algae 2, 135–145]. The robustness of the numerical experiments are tested by a formal parameter sensitivity analysis. As the first theoretical model consistent with the experiment of Graneli and Johansson (2003), our results demonstrate that, when nutrient-deficient conditions are favorable for the TPP population to release toxic chemicals, the TPP species control the bloom of other phytoplankton species which are non-toxic. Consistent with the observations made by Graneli and Johansson (2003), our model overcomes the limitation of not incorporating the effect of nutrient-limited toxic production in several other models developed on plankton dynamics.

Keywords:
Toxin producing phytoplankton
Nutrient limitation
Sensitivity analysis

1. Introduction

Some phytoplankton species are known to liberate 'toxic' or 'allelopathic' chemicals harmful for the growth of other algal species (Hallam et al., 1983). Despite extensive research over the last few years the knowledge about the ecological role of algal toxins is limited (Sole et al., 2005). Algal toxins are known to have a negative influence on copepods (Nejsgaard and Solberg, 1996), several herbivorous zooplankton (e.g., Ives, 1985; Huntley et al., 1986; Targett and Ward, 1991) and other algal species (Arlstad, 1991; Myklestad et al., 1995; Windust et al., 1996). An important observation is that, dinoflagellate-released toxins such as okadaic acid (OA) and dinophysistoxin (DTX-1) inhibit the growth of only those microalgae that do not produce toxins, which suggests that toxin production might be a strategy to repress or exclude algal competitors (Windust et al., 1996). It has been claimed that toxin production is a mechanism for controlling the growth of competing algae (Hulot et al., 2004). Conducting a field study followed by analyzing a mathematical model, Roy and Chattopadhyay (2007) have recently shown that toxin allelopathy enhances survival of weak competitors and thereby promotes biodiversity of phytoplankton species.

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Although the ecological role of toxins in the marine environment is significant, it is difficult to predict the strength of toxicity corresponding to a given density of algal cells (Graneli and Johansson, 2003). In some cases high toxicity has been observed with very low algal numbers. However, there are examples in which increased biomass of phytoplankton has not produced an observable toxic effects (Shilo, 1967). The capacity for phytoplankton species to produce toxins depends on different optimal requirements of environmental conditions (Shilo, 1971), and the effects of toxicity are expressed fully only during a growth-limiting condition (Dafni et al., 1972; Johansson and Graneli, 1999). Other experiments suggest that physiological stress factors are more responsible than the nutrient-limitated conditions (Johansson and Graneli, 1996, 1999; Reguera and Oshima, 1990; Bates et al., 1991, 1992, 1996; Igarashi et al., 1996). More specifically, environmental stress factors such as light intensity and the level of salinity have a significant influence on the toxicity (Shilo, 1967; Larsen et al., 1993). Some dinoflagellates such as *Alexandrium tamarense* (Boyer et al., 1985, 1987; Anderson et al., 1990), *Gymnodinium catenatum* (Reguera and Oshima, 1990), and the diatom *Pseudonitzschia multiseries* (Bates et al., 1991, 1992, 1996; Pan et al., 1996) have been observed to increase the release toxin under P-limited conditions. Moreover, some experiments show that the dinoflagellates *Prorocentrum lima* and *Dinophysis acuminata* release more toxin under both N- and P-limited conditions (McLachlan et al., 1994; Sohet et al., 1995; Johansson and Graneli, 1996).

In a laboratory experiment, Graneli and Johansson (2003) investigated the effects of prymnesium toxins on the growth of *Thalassiosira weissflogii*, *Rhodomonas cf. baltica*, and *Prorocentrum minimum*. To the culture media of those three phytoplankton, Graneli and Johansson (2003) added the cell-free filtrate of *P. parvum* cultures, which were grown under a nutrient-sufficient (N or P) or a deficient condition. They found that the filtrate from *P. parvum* cultures grown under nutrient-sufficient condition exhibits no significant effect on the growth of any of the tested species. But under nutrient-deficient conditions, it has a significant negative effect on the growth of those species. Moreover, their analysis found that at the end of the experiments all culture bottles contained excess amounts of NO_3^-, PO_4^{3-}, and SiO_3. They argued that the decrease in cell concentration of each of the three tested species is not due to nutrient limitations, but is caused by the amount of toxin released. Following these results, Graneli and Johansson (2003) suggested that prymnesium toxins play an allelopathic role, and that its production is regulated by the availability of nutrient.

Although important in the context of plankton dynamics, no mechanistic model has been developed yet to theoretically resemble this experimental observation. In the present article, we develop a mathematical model to describe the rate of toxin production by the toxic species and to explore the interaction between two phytoplankton species under laboratory condition similar to those employed by Graneli and Johansson (2003). We present a general analysis of the model and compare the outcome of the model with that of the laboratory experiment performed by Graneli and Johansson (2003). Further through a sensitivity analysis we examine the results of model simulations under different parametric setups. Finally we discuss our results in the context of phytoplankton dynamics in the real world.

2. Mathematical model

Mathematical models of nutrient–phytoplankton–zooplankton (N–P–Z) interaction with different complexity have been constructed and analyzed by many researchers. The mathematical analysis of plankton models goes back to Hallam (1977a,b, 1978) who studied stability and persistence properties of a nutrient controlled plankton models. Arnold (1978, 1980) and Arnold and Voss (1981), also considered N–P–Z models and discussed the existence of limit cycles. Gard (1983) provided a simpler and sharper persistence criterion for a N–P–Z model with general functional responses. Busenberg et al. (1990) studied a N–P–Z model and showed that under certain conditions the coexistence of phytoplankton and zooplankton occurs in an orbitally stable oscillatory mode. Ruan (1993) considered a N–P–Z model with nutrient recycling, periodic nutrient input and periodic washout rate to observe the existence of all components as well as the periodic solutions. Ruan (2001) also considered both instantaneous and delayed nutrient recycling on a N–P–Z model to demonstrate that the delayed nutrient recycling model exhibits more oscillations than the instantaneous nutrient recycling model. Recently Jang and Baglama (2005) investigated a N–P–Z model with both instantaneous and the delayed nutrient recycling, where they used a quadratic term to model zooplankton mortality. With the help of numerical simulation they suggested that delayed nutrient recycling can actually stabilize the nutrient–plankton system and the periodic solution of the system disappeared as zooplanktons mortality rate increases. Jang et al. (2006) studied a N–P–Z model in the presence of toxic chemicals where toxin inhibit the growth of either phytoplankton or zooplankton or both. They observed that the populations undergo cyclic blooms when toxin inhibit the growth of both the populations. However, none of the models developed previously describe the effect of nutrient limitation on toxin production. We develop a model to describe the variations of toxic chemicals release by TPP by considering toxin as an extra variable whose amount depends on nutrient level of the culture media.

Our model consists of four variables: concentration of nutrient levels $N(t)$, toxic phytoplankton population $P_1(t)$, non-toxic phytoplankton population $P_2(t)$ and concentration of toxin present $\theta(t)$ at time t. Assume that there is an external source of nutrients flowing into the system at a constant rate A. TPP (toxin producing phytoplankton) and NTP (non-toxic phytoplankton) populations, rely on nutrient "uptake" for growth and Michaelis–Menten functions $N/(a_1 + N)$ and $N/(a_2 + N)$ are used for modelling the nutrient uptake by toxic and non-toxic phytoplankton, respectively, where a_1, a_2 denote the half saturation constants or Michaelis–Menten constants. The actual uptake rates are also determined by parameters m_1 and m_2, which specify the maximum uptake rates for the toxic and non-toxic phytoplankton, respectively. In addition, TPP and NTP are removed from the water column through mortality and sinking as determined by the parameter δ and γ, respectively. The parameter d represents the rate of

loss of nutrient from the system (DeAngelis, 1992). The parameter r is the mortality rate of NTP due to toxin, d_1 is the washout rate of toxin, and k is the toxin production rate.

The production of toxic chemicals by TPP increases due to insufficiency of nutrient. We model this phenomenon mechanistically by $(kP_1)/(a_1+N)$—a monotonically decreasing function of nutrient level (N). To the best of our knowledge, the mechanism that actually describes how a toxic phytoplankton may affect others is still unknown. There may be several possible ways in which toxic phytoplankton may exert a negative effect on other phytoplankton. We incorporate this effect by an independent mortality term in the equation of NTP growth, due to toxin release by toxic phytoplankton.

All the variables are connected through the following system of differential equations:

$$\left.\begin{aligned}
\frac{dN}{dt} &= A - dN - \frac{m_1 N P_1}{a_1+N} - \frac{m_2 N P_2}{a_2+N},\\
\frac{dP_1}{dt} &= \frac{m_1 N P_1}{a_1+N} - \delta P_1,\\
\frac{dP_2}{dt} &= \frac{m_2 N P_2}{a_2+N} - rP_2\theta - \gamma P_2,\\
\frac{d\theta}{dt} &= \frac{kP_1}{a_1+N} - d_1\theta.
\end{aligned}\right\} \tag{1}$$

System (1) has to be analyzed with the following initial conditions:

$$N(0)>0,\quad P_1(0)>0,\quad P_2(0)>0,\quad \theta(0)>0.$$

3. General stability results

The model system (1) possesses the following equilibria: (i) phytoplankton and toxin free equilibrium $E_0=((A/d),0,0,0)$, (ii) toxic phytoplankton and toxin free equilibrium $E_1=(N^{(1)},0,P_2^{(1)},0)$ where $N^{(1)}=(a_2\gamma)/(m_2-\gamma)$ and $P_2^{(1)}=(A-dN^{(1)})/\gamma$, (iii) non-toxic phytoplankton free equilibrium $E_2=(N^{(2)},P_1^{(2)},0,\theta^{(2)})$ where $N^{(2)}=(a_1\delta)/(m_1-\delta)$, $P_1^{(2)}=(A-dN^{(2)})/\delta$, and $\theta^{(2)}=(kP_1^{(2)})/(d_1(a_1+N^{(2)}))$ (iv) the coexisting (interior) equilibrium $E^*=(N^*,P_1^*,P_2^*,\theta^*)$ where $N^*=(a_1\delta)/(m_1-\delta)$, $P_1^*=(d_1\theta^*(a_1+N^*))/k$, $P_2^*=((a_2+N^*)((A-dN^*)(a_1+N^*)-m_1N^*P_1^*))/((a_1+N^*)m_2N^*)$, and $\theta^*=(m_2N^*-\gamma(a_2+N^*))/(r(a_2+N^*))$.

Equilibrium point E_0 exists for every parametric value, E_1 exists if $m_2>\gamma$ and $N^{(1)}<(A/d)$, E_2 exists if $m_1>\delta$ and $N^{(2)}<(A/d)$, and the interior equilibrium E^* exists if $m_1>\delta$, $m_2N^*>\gamma(a_2+N^*)$ and $(A-dN^*)(a_1+N^*)>m_1N^*P_1^*$.

The local stability of the system (1) around each of the equilibria is obtained by computing the variational matrix corresponding to each equilibrium.

Lemma 1.1. *The system (1) around E_0 is locally asymptotically stable (LAS) if $(m_1N^{(0)})/(a_1+N^{(0)})<\delta$ and $(m_2N^{(0)})/(a_2+N^{(0)})<\gamma$.*

Lemma 1.2. *The system (1) around E_1 is LAS if $(m_1N^{(1)})/(a_1+N^{(1)})<\delta$.*

Lemma 1.3. *The system (1) around E_2 is LAS if $(m_2N^{(2)})/(a_2+N^{(2)})<\gamma+r\theta^{(2)}$.*

Lemma 1.4. *The system (1) around E^* is always unstable.*

Proof. The variational matrix J^* of the system (1) around $E^*(N^*,P_1^*,P_2^*,\theta^*)$ is

$$J^*=\begin{pmatrix}
-d-\frac{a_1m_1P_1^*}{(a_1+N^*)^2}-\frac{a_2m_2P_2^*}{(a_2+N^*)^2} & \frac{a_1m_1P_1^*}{(a_1+N^*)^2} & \frac{a_2m_2P_2^*}{(a_2+N^*)^2} & -\frac{kP_1^*}{(a_1+N^*)^2}\\
-\delta & 0 & 0 & \frac{k}{a_1+N^*}\\
-\gamma-r\theta^* & 0 & 0 & 0\\
0 & 0 & -rP_2^* & -d_1
\end{pmatrix}.$$

The corresponding characteristic equation is given by

$$\lambda^4+D_1\lambda^3+D_2\lambda^2+D_3\lambda+D_4=0 \tag{2}$$

where

$$\left.\begin{aligned}
D_1 &= d+d_1+\frac{a_1m_1P_1^*}{(a_1+N^*)^2}+\frac{a_2m_2P_2^*}{(a_2+N^*)^2}\\
D_2 &= dd_1+\frac{a_1m_1P_1^*(d_1+\delta)}{(a_1+N^*)^2}+\frac{a_2m_2P_2^*(d_1+\gamma+r\theta^*)}{(a_2+N^*)^2}\\
D_3 &= \frac{a_1d_1m_1\delta P_1^*}{(a_1+N^*)^2}+\frac{a_2d_1m_2P_2^*(\gamma+r\theta^*)}{(a_2+N^*)^2}-\frac{krP_1^*P_2^*(\gamma+r\theta^*)}{(a_1+N^*)^2}\\
D_4 &= -\frac{a_1km_1rP_1^*P_2^*(\gamma+r\theta^*)}{(a_1+N^*)^3}.
\end{aligned}\right\} \tag{3}$$

By the Routh–Hurwitz criterion, a set of necessary and sufficient conditions for all the roots of (2) to have negative real part is

(i) $D_1>0$,
(ii) $D_4>0$.
(iii) $D_1D_2-D_3>0$,
(iv) $D_3(D_1D_2-D_3)-D_4D_1^2>0$. □

From (3), it is clear that $D_4<0$. Hence, the interior equilibrium point E^* is unstable for every parametric value.

If the maximal growth rates $(m_1N^{(0)})/(a_1+N^{(0)})$ and $(m_2N^{(0)})/(a_2+N^{(0)})$ of both TPP and NTP populations, respectively, are less than their corresponding natural removal rates δ_1 and γ_1, respectively, and the amount of nutrient is stabilized at $N^{(0)}$, both TPP and NTP populations go to the extinction. If the maximal growth rate $(m_1N^{(1)})/(a_1+N^{(1)})$ of TPP population (when the amount of nutrient is stabilized at $N^{(1)}$) is less than its natural removal rate δ, the NTP population alone will stabilize in a positive steady state. On the other hand, when the amount of nutrient and toxic chemicals are stabilized at $N^{(2)}$ and $\theta^{(2)}$, respectively, NTP population become extinct if its maximal growth rate $(m_2N^{(2)})/(a_2+N^{(2)})$ is less than total loss rate $\gamma+r\theta^{(2)}$. However, because the interior equilibrium is always unstable, corresponding to a set of parameters one of the boundary equilibriums becomes attracting. Thus, the trajectories of the model system (1) eventually go towards that attracting boundary equilibrium. In other words, for a long run the coexistence of both TPP and NTP populations is not possible. However, the laboratory experiment conducted by Graneli and Johansson (2003) continued for only a few days. As we show in the following sections, under a finite time limit, when both the species coexist, our model efficiently supports experimental outcome of the dynamics.

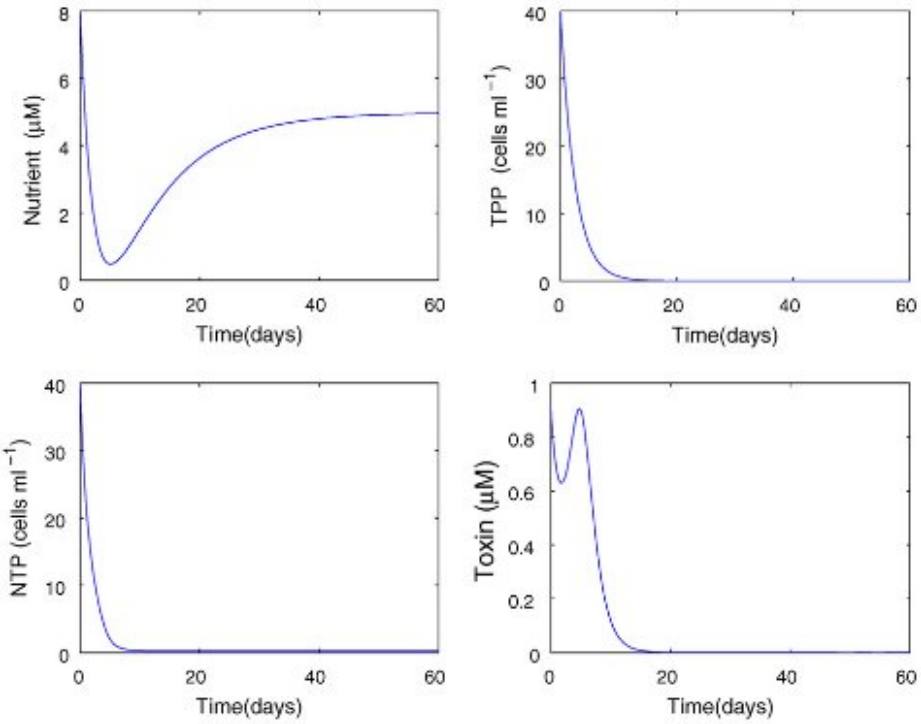

Fig. 1 – The solution with initial condition (8, 40, 40, 0.9) goes to the trivial steady state E_0 where the parameter values are given by $A = 0.5$, $d = 0.1$, $d_1 = 0.8$, $a_1 = 0.07$, $a_2 = 1.0$, $m_1 = 0.1$, $m_2 = 0.01$, $k = 0.07$, $r = 0.8$, $\delta = 0.5$, $\gamma = 0.01$..

3.1. *Numerical examples*

We validate and extrapolate our analytical findings through numerical simulations considering the following hypothetical set of parameter values: $A = 0.5$, $d = 0.1$, $d_1 = 0.8$, $a_1 = 0.07$, $a_2 = 1.0$, $m_1 = 0.1$, $m_2 = 0.01$, $k = 0.07$, $r = 0.8$, $\delta = 0.5$, $\gamma = 0.01$.

With these parameter values, it is easy to verify that $(m_1 N^{(0)})/(a_1 + N^{(0)}) < \delta$ and $(m_2 N^{(0)})/(a_2 + N^{(0)}) < \gamma$. Therefore, the trivial steady state E_0=(5, 0, 0, 0) is locally asymptotically stable (Fig. 1).

If we set $A = 1.0$, $a_2 = 0.2$, $m_1 = 1.0$, $m_2 = 0.7$, $\delta = 1.0$, $\gamma = 0.3$ and the rest of the parameter values are the same as given above, then the inequality $(m_1 N^{(1)})/(a_1 + N^{(1)}) < \delta$ holds so that the system (1) has a steady state $(N^{(1)}, 0, P_2^{(1)}, 0)$=(0.15, 0, 3.28, 0) as shown in Fig. 2.

Now, if we take $A = 40.0$, $a_2 = 0.2 m_1 = 1.0$, $m_2 = 0.7$, $r = 0.1$, $\delta = 0.7$, $\gamma = 0.1$ and the rest of the parameter values are the same as in the first case, then the inequality $(m_1 N^{(2)})/(a_1 + N^{(2)}) < \gamma + r\theta^{(2)}$ holds so that the system (1) has a steady state $(N^{(1)}, P_1^{(2)}, 0, \theta^{(2)}) = (0.16, 57.12, 0, 21.42)$ as shown in Fig. 3.

4. Experimental results vs. model outcome

The time span of the original experiment of Graneli and Johansson (2003) was 3 days, but during the first day the outcome of the cell concentration of non-toxic algae and toxic algae under nutrient sufficient and deficient conditions varied significantly. To capture the experimental outcome of Graneli and Johansson (2003), we fix a set of realistic parameter values tuned with different representative initial conditions. Appropriate initial concentration is chosen to represent nutrient sufficient or deficient environment. We impose the following sets of initial conditions: (40, 40, 40, 0.9) for the nutrient-sufficient experiment and (0.005, 40, 40, 0.9) for the nutrient-deficient experiment. We run our numerical simulation for approximately 20 h and the corresponding dynamics of the system are presented in Fig. 4. The variables for corresponding situations are represented by continuous and dotted line, respectively (Fig. 4, other parameter values given in Table 1).

The model simulation depicts that at low nutrient condition, the amount of release of toxic chemicals become very high whereas it has no significant change at sufficient nutrient condition. Although TPP population shows positive growth rate at both nutrient limited and sufficient conditions, NTP population exhibits positive growth rate only at nutrient-

Table 1 – Parameters and initial conditions for (1)

Name	Description	Value
A	Constant nutrient inflow	40
d	Amount of nutrient loss	0.1
d_1	Washout rate of toxin	0.8
a_1	Half saturation constant	0.07
a_2	Half saturation constant	0.2
m_1	Uptake rate of TPP	1.0
m_2	Uptake rate of NTP	0.7
k	Toxin production rate	0.07
r	Mortality rate of NTP due to toxin	0.8
δ	Percapita natural death rate of TPP	0.5
γ	Percapita natural death rate of NTP	0.01

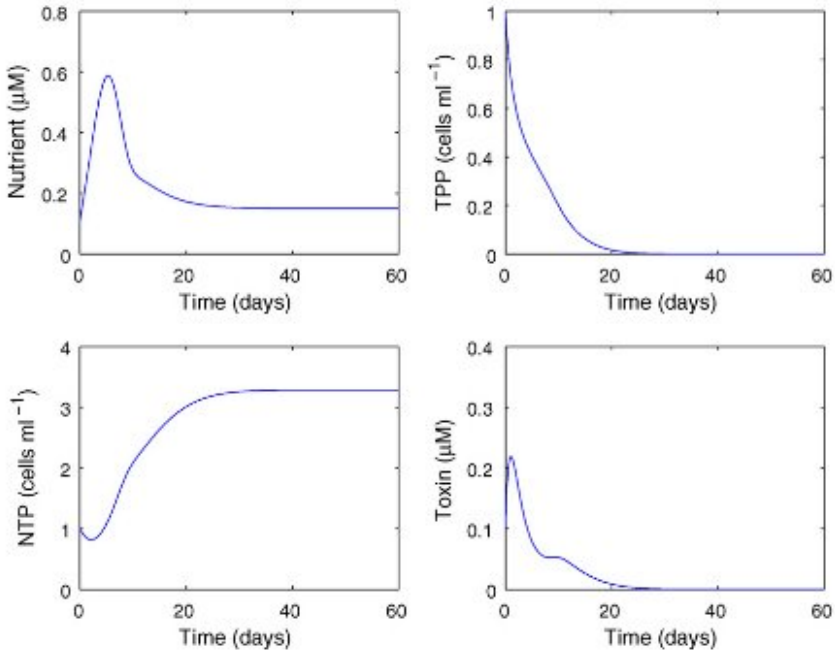

Fig. 2 – The solution with initial condition (0.1, 1, 1, 0.1) goes to the nontrivial steady state E_1 where the parameter values are given by $A = 1.0$, $d = 0.1$, $d_1 = 0.8$, $a_1 = 0.07$, $a_2 = 0.2$, $m_1 = 1.0$, $m_2 = 0.7$, $k = 0.07$, $r = 0.8$, $\delta = 1.0$, $\gamma = 0.3$..

sufficient conditions. However, at low nutrient condition NTP population decreases at a high rate during the first 20 h (Fig. 4). These outcomes resemble the experimental results (Fig. 5) observed by Graneli and Johansson (2003) where they have observed that addition of cell-free filtrates from *P. parvum* cultures grown under nutrient limitations (N or P) had a negative influence on the growth of *T. weissflogii*, *R. cf. baltica* and *P. minimum* (not being able to produce prymnesium toxins) during the first 20 h, resulting in a rapid decrease in cell concentration (Fig. 5). Whereas in contrast, a strain of *Prymnesium patelliferum* known to produce prymnesium toxins was not negatively affected under any conditions.

Moreover, the results shown in Fig. 4 suggests that the huge decrease in cell concentration of NTP population at a low nutrient condition is not due to the insufficiency of nutrient. In fact, a sufficient amount of nutrient has observed after 20 h (Fig. 4(a)), and at the same nutrient condition, TPP population shows a positive growth rate (Fig. 4(b)). Thus, the significant

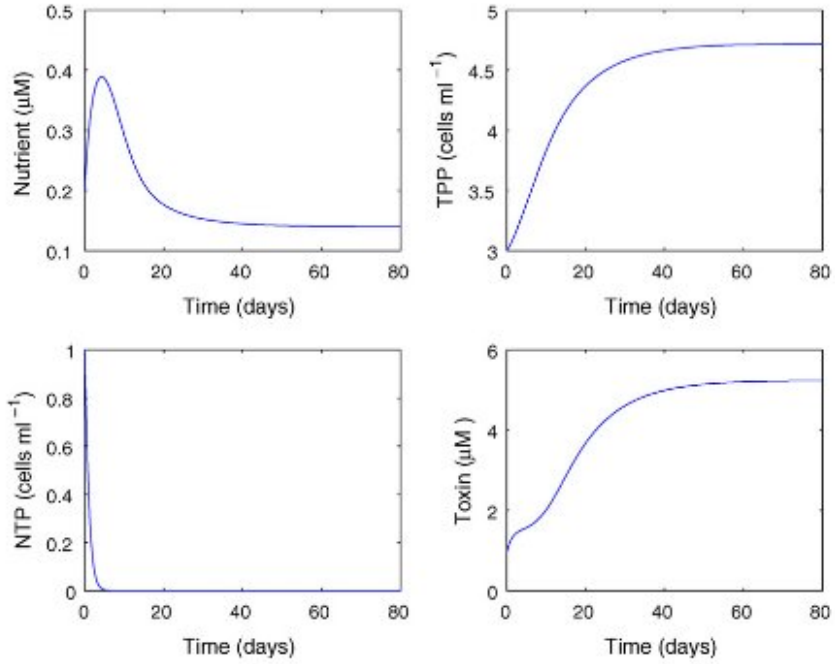

Fig. 3 – The solution with initial condition (0.2, 40, 40, 0.9) goes to the nontrivial steady state E_2 where the parameter values are given by $A = 0.5$, $d = 0.2$, $d_1 = 0.3$, $a_1 = 0.07$, $a_2 = 1.0$, $m_1 = 0.15$, $m_2 = 0.01$, $k = 0.07$, $r = 0.8$, $\delta = 0.1$, $\gamma = 0.01$.

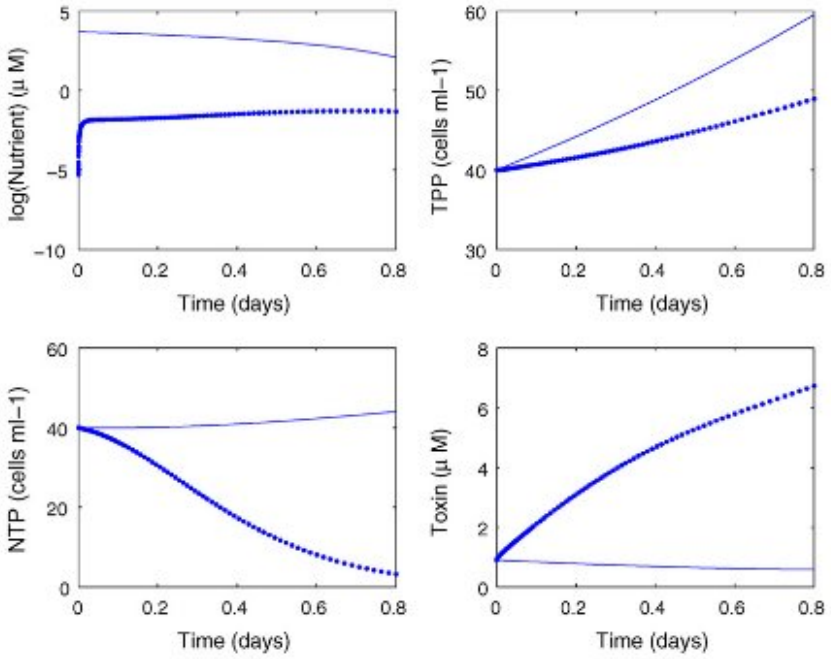

Fig. 4 – Comparison of the model outcome under nutrient sufficient and deficient states. The dotted curve depicts the nutrient-deficient state and continuous curve due to the nutrient-sufficient state. (a) Toxic phytoplankton, (b) non-toxic phytoplankton, and (c) amount of toxin. Parameter values:
$A = 40, d = 0.1, d_1 = 0.8, m_1 = 1.0, m_2 = 0.7, a_1 = 0.07, a_2 = 0.2, k = 0.07, r = 0.8, \delta = 0.5, \gamma = 0.01.$

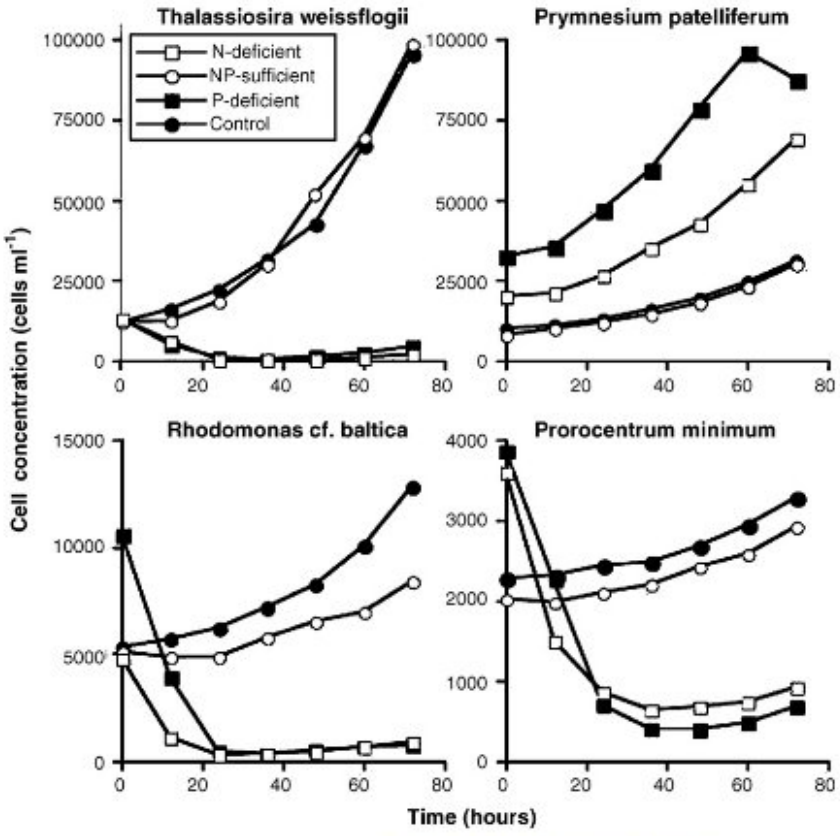

Fig. 5 – Figure taken from Graneli and Johansson (2003).

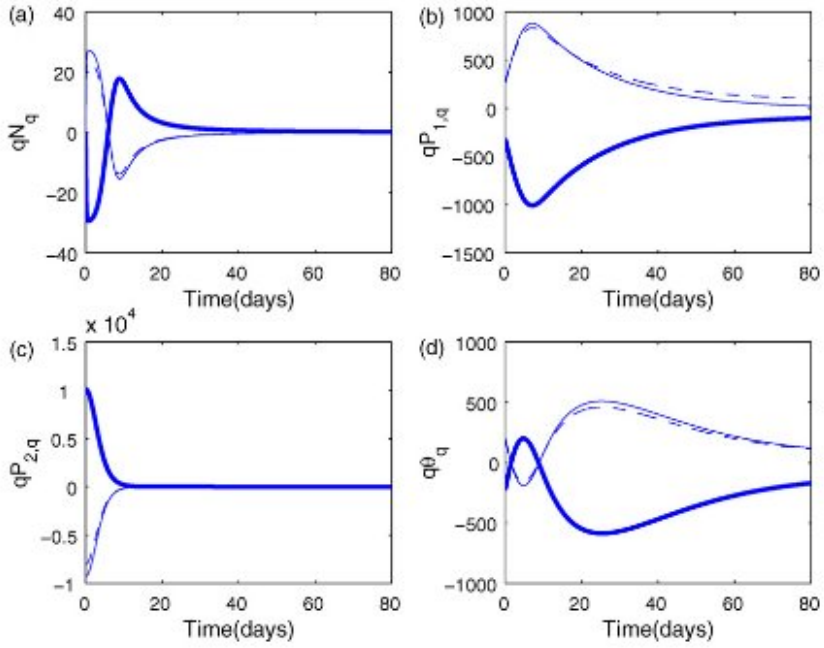

Fig. 6 – Semi-relative sensitivity solutions at nutrient-deficient situation with initial condition (0.005, 40, 40, 0.09); dashed line corresponding to parameter A, continious line corresponding to k and bold line is due to δ.

decrease in NTP population is due to an additional effect of toxin released at an increased rate under a low nutrient condition. Although these toxic chemicals released by TPP inhibit the growth of other phytoplankton, they have no effect on the species releasing the chemicals. This argument is also consistence with the experimental observations (Fig. 5) made by Graneli and Johansson (2003).

Although the output of the numerical simulation of our model qualitatively resembles the experiment of Graneli and Johansson (2003) over the initial shorter time scale of 20 h, our results differs from that experiment after a time scale of 36 h. Graneli and Johansson (2003) observed that after approximately 36 h, cell densities of all the species started to increase, whereas our model outcome shows the extinction of NTP populations. We suggest that the main reason behind this contrast is the way of addition of toxic chemicals into the system. Graneli and Johansson (2003) added cell free filtrates of *P. parvum* into the culture bottles of each species only once, on the first experimental day and observed the corresponding effects. But in our dynamic model we consider the effect of continuously released allelochemicals on NTP populations and as a result NTP population goes to extinction. This argument suggests that repeated addition (as done in Suikkanen. et al., 2004) rather than a single addition of toxic filtrate would lead to an extinction of all the three phytoplankton species not belonging to prymnesium group.

5. Sensitivity of model outcome with experimental setup

The model simulation demonstrates that toxin production increases under low nutrient conditions. Now, to confirm that, the toxin production rate is the main factor for the death of NTP population under a nutrient-deficient condition (also for the whole dynamics of the system), we perform a formal sensitivity analysis. To observe the effect of variation in model parameters on the outcome of the model, the techniques of sensitivity analysis are mathematically more sophisticated than simply observing the output generated by varying a chosen parameter. Here we use a basic differential analysis approach. Illustrations of this technique as well as more mathematically advanced methods can be found in several articles and text books (e.g., Eslami, 1994; Kleiber et al., 1997; Saltelli et al., 2000, to mention a few).

The semi-relative sensitivity solutions with respect to each of the three parameters A, δ, and k corresponding to four state variables are presented in Fig. 6. Each plot depicts the derivative of a state variable with respect to a chosen parameter over the time scale. For a (positive) change in the death rate of TPP (δ), although the amount of nutrient N is low near initial point, it jumps to a high value quickly (just near day 1) (Fig. 6(a)). On the other hand, for a (positive) change in the toxin production rate k (and constant nutrient inflow A), the amount of nutrient N grows to a high value from the initial point (Fig. 6(b)). In an early stage of the simulation the perturbation of k results in a great decrease in NTP population (Fig. 6(c)).

The logarithmic sensitivity solutions [i.e., $(\partial \log(N)/\partial \log(q))(t) = (q/N(t,q))N_q(t,q)$] with respect to each of the three parameters (δ, k, A) for the four state variables and the observed compartment are depicted in Fig. 7. These plots represent a percentage change in the solution induced by positive perturbations of the corresponding parameter. Although in this case the perturbations of k have greatest negative impact on the NTP population, the perturbations of δ have a positive impact on the same population. At around time point $t = 80$ days, the perturbations of k cause a (roughly) 200, 000% change in NTP populations.

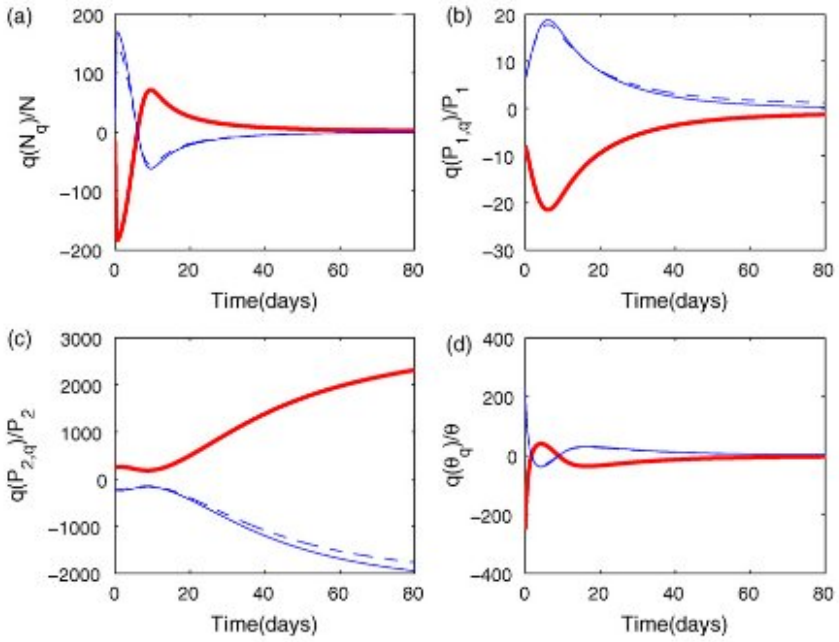

Fig. 7 – Logarithimic sensitivity solutions at nutrient-deficient situation with initial condition (0.005, 40, 40, 0.09); dashed line corresponding to parameter A, continious line corresponding to k and bold line is due to δ.

To observe the sensitivity of the parameters on the dynamics of the system (1) we have varied all the parameters around the values given in Table 1. The only three parameters with significant sensitivities (i.e., that significantly affected the dynamics of the model output) were δ, k and A. However, a minor change of those three parameters have a great impact on the whole dynamics of the system. It is already established that under a nutrient-deficient condition there is a significant increase of toxin release. Here we observe that under limiting nutrient condition, the excess amount of toxin released results in nearly a 200, 000% decrease of NTP population. Thus, at a low nutrient condition, toxin production is an important cause for the rapid decrease of NTP population. These results are consistent with the experimental outcomes of Graneli and Johansson (2003) (Fig. 5).

6. Discussion

In this paper we have developed a simple mathematical model to describe the mechanism of toxin production by an algae (termed TPP) and its effect on those alga that do not produce toxin (NTP). Limitation of extracellular nutrient level has been used as an environmental stress for the phytoplankton, which is potentially responsible for the production of toxic chemical. Under nutrient-limiting conditions TPP increase their release of toxic chemicals, and this phenomenon is described by a monotonic function of nutrient concentration. The model is used to simulated the experimental findings of Graneli and Johansson (2003). We have carried out the local stability analysis of the system around the steady states and performed numerical simulations to produce desired results consistent with the experimental findings. The outcome is compared qualitatively with the experimental observations of Graneli and Johansson (2003). Consistent with that observed in an ideal laboratory condition by Graneli and Johansson (2003), theoretical analysis of our model demonstrates that in nutrient-limited condition enhanced toxin production by a toxic phytoplankton influences the competitive interaction of two species. We produce the robustness of the theoretical results through a formal sensitivity analysis.

Our present study produces the first theoretical model that resembles the important experimental findings by Graneli and Johansson (2003). Several other theoretical models of phytoplankton dynamics (e.g., Arnold, 1978; Ruan, 2001) exist that do not incorporate the effect of nutrient-limited toxin production. Incorporation of such an effect, which is derived even in an experiment, could theoretically alter the present understanding of the species interaction described by other models of phytoplankton systems. The species that liberate toxic chemicals gain an advantage in competition and those chemicals suppresses the bloom formation of other non-toxic algae. However, in the real world, there are several factors that affect the interaction of the species. A natural question arises as to whether these toxic chemicals are as important for bloom termination in the real world. Some of our previous studies with the help of field observation and mathematical modelling have already established that toxin producing phytoplankton acts as a controlling agent for the termination of the planktonic bloom (Chattopadhyay et al., 2002a, 2002b; Sarkar and Chattopadhyay, 2003). However, even in those field-based studies the effect of different nutrient conditions upon the production of toxin was not considered. The present study overcomes that limitation, and our model output depict that the nutrient regulated release of toxins by certain algae can potentially control the growth of other non-toxic phytoplankton.

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Appendix A. Approach for sensitivity analysis

The first step in the sensitivity analysis is to derive the *sensitivity equations* by formally taking derivatives (with respect to a parameter of interest) on both sides of the original equation(s). The solution to this new system (assuming for the moment it is well-posed) contains information regarding the sensitivity of the original system to perturbations in the chosen parameter (around some *a priori* fixed value of that parameter). Hereafter we will refer to the solution to the sensitivity equations as a *sensitivity function*.

Let us denote N_q, $P_{1,q}$, $P_{2,q}$, and θ_q as the sensitivity functions of N, P_1, P_2, and θ with respect to an arbitrary parameter q, that is

$$N_q(t) = \frac{\partial}{\partial q} N(t, q),$$

$$P_{1,q}(t) = \frac{\partial}{\partial q} P_1(t, q),$$

$$P_{2,q}(t) = \frac{\partial}{\partial q} P_2(t, q),$$

$$\theta_q(t) = \frac{\partial}{\partial q} \theta(t, q),$$

for all t. The corresponding sensitivity systems with respect to A, δ, and k are

$$\dot{N}_A(t, A) = 1 - dN_A - \frac{m_1}{a_1 + N} NP_{1,A} - \frac{a_1 m_1}{(a_1 + N)^2} N_A P_1 - \frac{m_2}{a_2 + N} NP_{2,A} - \frac{a_2 m_2}{(a_2 + N)^2} N_A P_2,$$

$$\dot{P}_{1,A}(t, A) = \frac{m_1}{a_1 + N} NP_{1,A} + \frac{a_1 m_1}{(a_1 + N)^2} N_A P_1 - \delta P_{1,A},$$

$$\dot{P}_{2,A}(t, A) = \frac{m_2}{a_2 + N} NP_{2,A} + \frac{a_2 m_2}{(a_2 + N)^2} P_2 N_A - r\theta P_{2,A} - rP_2\theta_A - \gamma P_{2,A},$$

$$\dot{\theta}_A(t, A) = \frac{k}{a_1 + N} P_{1,A} - \frac{k}{(a_1 + N)^2} P_1 N_A - d_1\theta_A,$$

and

$$\dot{N}_\delta(t, \delta) = -dN_\delta - \frac{m_1}{a_1 + N} NP_{1,\delta} - \frac{a_1 m_1}{(a_1 + N)^2} N_\delta P_1 - \frac{m_2}{a_2 + N} NP_{2,\delta} - \frac{a_2 m_2}{(a_2 + N)^2} N_\delta P_2,$$

$$\dot{P}_{1,\delta}(t, \delta) = \frac{m_1}{a_1 + N} NP_{1,\delta} + \frac{a_1 m_1}{(a_1 + N)^2} N_\delta P_1 - \delta P_{1,\delta} - P_1,$$

$$\dot{P}_{2,\delta}(t, \delta) = \frac{m_2}{a_2 + N} NP_{2,\delta} + \frac{a_2 m_2}{(a_2 + N)^2} P_2 N_\delta - r\theta P_{2,\delta} - rP_2\theta_\delta - \gamma P_{2,\delta},$$

$$\dot{\theta}_\delta(t, \delta) = \frac{k}{a_1 + N} P_{1,\delta} - \frac{k}{(a_1 + N)^2} P_1 N_\delta - d_1\theta_\delta,$$

and

$$\dot{N}_k(t, k) = -dN_k - \frac{m_1}{a_1 + N} NP_{1,k} - \frac{a_1 m_1}{(a_1 + N)^2} N_k P_1 - \frac{m_2}{a_2 + N} NP_{2,k} - \frac{a_2 m_2}{(a_2 + N)^2} N_k P_2,$$

$$\dot{P}_{1,k}(t, k) = \frac{m_1}{a_1 + N} NP_{1,k} + \frac{a_1 m_1}{(a_1 + N)^2} N_k P_1 - \delta P_{1,k},$$

$$\dot{P}_{2,k}(t, k) = \frac{m_2}{a_2 + N} NP_{2,k} + \frac{a_2 m_2}{(a_2 + N)^2} P_2 N_k - r\theta P_{2,k} - rP_2\theta_k - \gamma P_{2,k},$$

$$\dot{\theta}_k(t, k) = \frac{k}{a_1 + N} P_{1,k} - \frac{k}{(a_1 + N)^2} P_1 N_k + \frac{1}{a_1 + N} P_1 - d_1\theta_k.$$

When evaluated at time t, the value of the sensitivity function indicates the rate of change in the state with respect to the change in the chosen parameter. Since the parameters differ in their units, the sensitivity functions for different parameters would also have different units, and rendering any comparison is meaningless. To enable a comparison of the effects that parameters with different units have on the solution, we simply multiply by the parameter under consideration, e.g., $((\partial/\partial q)N(t, q), (\partial/\partial q)P_1(t, q), (\partial/\partial q)P_2(t, q), (\partial/\partial q)\theta(t, q)) \cdot q$, which provides information concerning the amount the state will change when that parameter is doubled (i.e., a perturbation on the order of q). This form of the sensitivity function is known as the *semi-relative* or *semilogarithmic* or *unnormalized* sensitivity function (Bortz and Nelson, 2004).

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