Toxin-allelopathy among phytoplankton species prevents competitive exclusion

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Abstract

Toxic or allelopathic compounds liberated by toxin-producing phytoplankton (TPP) acts as a strong mediator in plankton dynamics. On an analysis of a set of phytoplankton biomass-data that have been collected by our group in the North-West part of the Bay of Bengal, and by analysis of a three-component mathematical model under a constant as well as a stochastic environment, we explore the role of toxin-allelopathy in determining the dynamic behaviour of the competing-phytoplankton species. The overall results, based on analytical and numerical wings, demonstrate that toxin-allelopathy due to the toxin-producing phytoplankton (TPP) promotes a stable coexistence of those competitive phytoplankton that would otherwise exhibit competitive exclusion of the weak species. Our study suggests that TPP might be a potential candidate for maintaining the coexistence and diversity of competing phytoplankton species.

Key words: Phytoplankton, toxin, allelopathy, coexistence, competitive exclusion, paradox of plankton

1 Introduction

The principle of competitive exclusion ensures that the number of competing species cannot exceed the number of distinct resources (Hardin, 1960). Simple competition models and competition experiments in laboratory also suggest that the number of species that co-exist in equilibrium can be greater than the number of limiting resources only if additional mechanisms are involved (Tilman, 1977, 1981; Somer 1985, 1986; Rothhaupt 1988, 1996; Scheffer *et al.*, 1997; Huisman and Weissing, 1999). For instance, temporal variation in the supply of a single resource may allow the coexistence of two species (Stewart & Levin, 1973; Levins, 1979; Armstrong & McGehee, 1980). For two competing prey or parasites, predator or parasite-mediated coexistence is possible provided that the inferior competitor is resistant to exploitation (Levin, 1970; Levin *et al.*, 1977). Sometimes interference competition also promotes stable coexistence of two species on a single resource (e.g., Vance, 1985). Furthermore, in homogeneous environment inhibitory substances such as pesticides, derived from external sources can promote stable coexistence of two species competing for a single resource (Lenski & Hattingh, 1986). Unlike the above biological situations, in view of the competitive exclusion principle the coexistence of a large number of phytoplankton species on a seemingly limited variety of resources in natural waters is remarkable; this is referred to as 'the paradox of the plankton' (Hutchinson, 1961). To explain this paradox, several attempts have been made. Hutchinson (1961) proposed that because of weather-driven fluctuations, plankton communities are not in equilibrium. Authors such as Richerson *et al.* (1970) argued in a fashion similar to Hutchinson (1961) that continuous variation in environmental conditions, due to seasonal cycles and less predictable factors such as weather, offer the most likely solution. On the other hand, theoretical studies predict that competition among different species of phytoplankton can generate oscillations and chaos, which may in turn promote their coexistence (Huisman & Weissing, 1999). However, none of these explanations is universally accepted.

In an aquatic ecosystem, some species of plankton liberate "toxic" or "allelopathic agents" that affect the growth of other micro-algae (Hallam *et al*, 1983; Arzul *et al* 1999). Among marine algae, allelopathy was observed both *in vitro* and *in situ* (e.g Chan *et al.*, 1980; Nielsen *et al.*, 1990; Schmidt and Hansen, 2001, Tillmann and John, 2002; Fistarol *et al.*, 2003, 2004), however, the chemical nature and role of allelopathic compounds remained

poorly understood (Sole *et al.*, 2005). In phytoplankton-zooplankton interactions, toxicity acts as a strong mediator (Kozlowski-Suzuki *et al.*, 2003). Efforts have been made to study the role of toxin inhibition on zooplankton (e.g. Chattopadhyay *et al.*, 2002a, 2002b; Sarkar & Chattopadhyay, 2003). Recently, Roy *et al.* reported that in regulating non-equilibria of a phytoplankton-zooplankton system, toxin inhibition on zooplankton caused by toxin-producing phytoplankton (TPP) acts as a driving force. However, allelopathic interaction among the phytoplankton species has not been included in that study. Among the algae species, toxin-allelopathy is an important chemical-signaling process (see, review by Cembella, 2003). Interactions between two allelopathic-species was studied mathematically by many authors (e.g. Maynard-Smith, 1974; Chattopadhyay, 1996; Mukhopadhyay *et al.*; 1998; Nakamaru & Iwasa, 2000). Schimdt and Hansen (2001) made a laboratory experiment on plankton allelopathy in which 15 species of marine phytoplankton were exposed to suspensions of a toxic alga known as *Crysocomulina polylepis*. Recently, Sole *et al.* (2005) used those experimental data to estimate the allelopathic parameters based on a model proposed by Chattopadhyay (1996). The study of Solé *et al.* (2005) suggests a functional form suitable for quantifying the strength of allelopathic interaction between toxic and non-toxic algae.

However, in the previous studies little attention has been paid to explore the role of allelopathic interaction on the coexistence and persistence of phytoplankton species competing for the same resources. The objective of this article is to investigate the role of toxin-allelopathy in maintaining the coexistence of the competitive-phytoplankton species in the marine ecosystem. On analysis of a set of field-data that we have collected from the North-West coast of the Bay of Bengal, we propose that a possible role of toxic phytoplankton might be responsible for a stable coexistence of the competing phytoplankton. Next we formulate a simple three-component model for describing the interaction among two non-toxic phytoplankton and a toxic phytoplankton. We analyze the model in a deterministic and a stochastic environment, and find suitable bounds on the allelopathic parameters under which a stable coexistence of the competing species is possible. Through numerical experiments, we support our analytical findings and demonstrate the role of toxin allelopathy in maintaining the stable coexistence of those competing phytoplankton that would otherwise exhibit an exclusion of the weak species. The study demonstrates that toxin-allelopathy among phytoplankton species counteracts competitive exclusion.

The organization of the paper is as follows. Section 2 produces a qualitative analysis of the plankton dynamics based on the field observation on non-toxic and toxic phytoplankton. Section 3 proposes a three-component mathematical-model consisting of two-competitive-phytoplankton and a toxic phytoplankton. The model is analyzed to find the criterion for coexistence and persistence of the species. In Section 4, by incorporating stochastic perturbation, the dynamic behaviour is studied under environmental fluctuations. In Section 5 we present numerical experiments to support the analytical results. We discuss the overall results of our study in Section 6.

2 Field Observation

Since 1999, the monitoring and identification (Tomas, 1997) of marine plankton population has been carried out by our group in the North - West coast of the Bay of Bengal (for detail see, Chattopadhyay *et al.*, 2002). A significant number of species of phytoplankton have been identified that produce toxic or inhibitory compounds (Chattopadhyay *et al.*, 2002a, 2002b; Sarkar & Chattopadhyay, 2003). The toxin-producing phytoplankton (TPP) group contains (i) planktonic or benthic micro-algae that produce toxin (e.g., the motile stage of *Alexandrium*, the benthic *Gambierdiscus*), (ii) other toxic dinoflagellates (e.g., *Pfiesteria*), (iii) macroalgae that results in noxious smells (e.g. *Pilayella*), (iv) a few species of Cyanobacteria or blue algae (e.g., *Microcystis*), (v) non-toxic microorganisms that result in hypoxic conditions (e.g. *Chaetoceros, Mesodinium*). For a detailed list of TPP species identified by our group, see Chattopadhyay *et al.*, 2002.

For understanding the interaction between non-toxic and toxic algae at species level, we choose from the list of phytoplankton species that have been identified during the period 2000-2001, a combination three species consisting of two non-toxic and a toxic phytoplankton. We choose those algae that were present at significant biomass throughout the study period. The three species chosen belong to diatom group. The two non-toxic phytoplankton (NTP) are *Coscinodiscus sp* (say species 1, biomass at any time x_1) and *Biddulphia sp* (species 2, biomass x_2); and the toxin-producing phytoplankton (TPP, say species 3, biomass x_3) is *Chaetoceros sp* (as cited by Chattopadhyay *et al.*, 2002). The abundance level of all the species are fluctuating over the time (Figure 1), and throughout

the period of observation the abundance level of *Coscinodiscus sp* is higher than that of *Biddulphia sp*. Now, because all the species interact in a common marine environment, in principle they compete for the common resources available (such as sunlight, dissolved nutrient). In our study region, among the chosen species *Coscinodiscus sp* is the most dominant in biomass throughout the sampling period (see Figure 1). Because the species have been identified in a common sampling and from a common field, it is reasonable to assume that the ecological and biological factors that affect the growth of species are similar for all the species. Moreover, in a general sense a potential role of competitive effect of one species would be to hamper the abundance level of the other species. So, due to the lack of any other experimental data, we may consider the abundances of two non-toxic species (where toxin-allelopathy does not come into play) as a potential indicator of the dominance level of resource competition. Clearly this argument does not hold for a toxic and a non-toxic species. In this sense *Coscinodiscus sp* is a stronger competitor than *Biddulphia sp*. The distribution of the abundance ratio of *Coscinodiscus* to *Biddulphia*, when plotted against the abundance of the toxic phytoplankton, depicts a decreasing trend for higher biomass of toxic phytoplankton (Figure 2). Pearson correlations confirm this trend. The correlation coefficient between the abundance of TPP (in log scale) and the abundance ratio of x_1 to x_2 is ($r = -0.515$), which is significant at 5% level. On the other hand, the total biomass of the two non-toxic algae has a significant positive correlation with the x_1/x_2 coefficient ($r = 0.40$, $P < 0.05$). However, if we include the toxic algae the sum of the biomass of all the species has insignificant correlation ($r = 0.24$, $P > 0.1$) with the x_1/x_2 coefficient. These results suggest that, when the overall biomass of two non-toxic algae (that influences the resource competition) increases, the abundance ratio of the two algae also increases significantly; consequently in the system the pressure of *Coscinodiscus* is enhanced and that of the *Biddulphia sp* is reduced. However, the scenario changes significantly if the presence of a toxic algae is taken into consideration. The abundance ratio x_1/x_2 shows a significantly reverse trend (Figure 2).

To compare the ratio x_1 to x_2 in situations of high and low TPP abundance, we divide the TPP biomass in two categories: 'less than mean value' (say M_1) i.e., when TPP abundance is less than its overall average over the observed time points, and 'greater than or equal to mean value' (say M_2) i.e., when TPP abundance is greater than or equal to its overall average over the observed time points. We say TPP is at low abundance at any

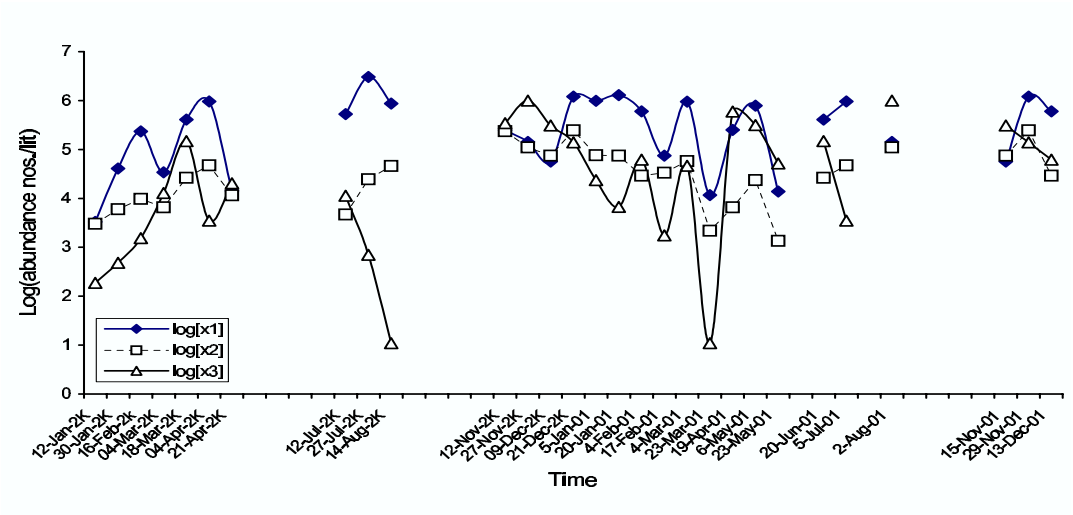

Figure 1: Coexistence of two non-toxic competitive-phytoplankton species with a toxic species. Here, x_1 is the biomass at any time point of the NTP (species 1) *Coscinodiscus sp*, x_2 is that of NTP (species 2) *Biddulphia sp*, and x_3 is the same of TPP (species 3) *Chaetoceros sp*; *Coscinodiscus sp* is the most abundant species and stronger competitor than *Biddulphia sp*. The gaps in the axis of collection represents the time point when the sampling was suspended due to several reasons.

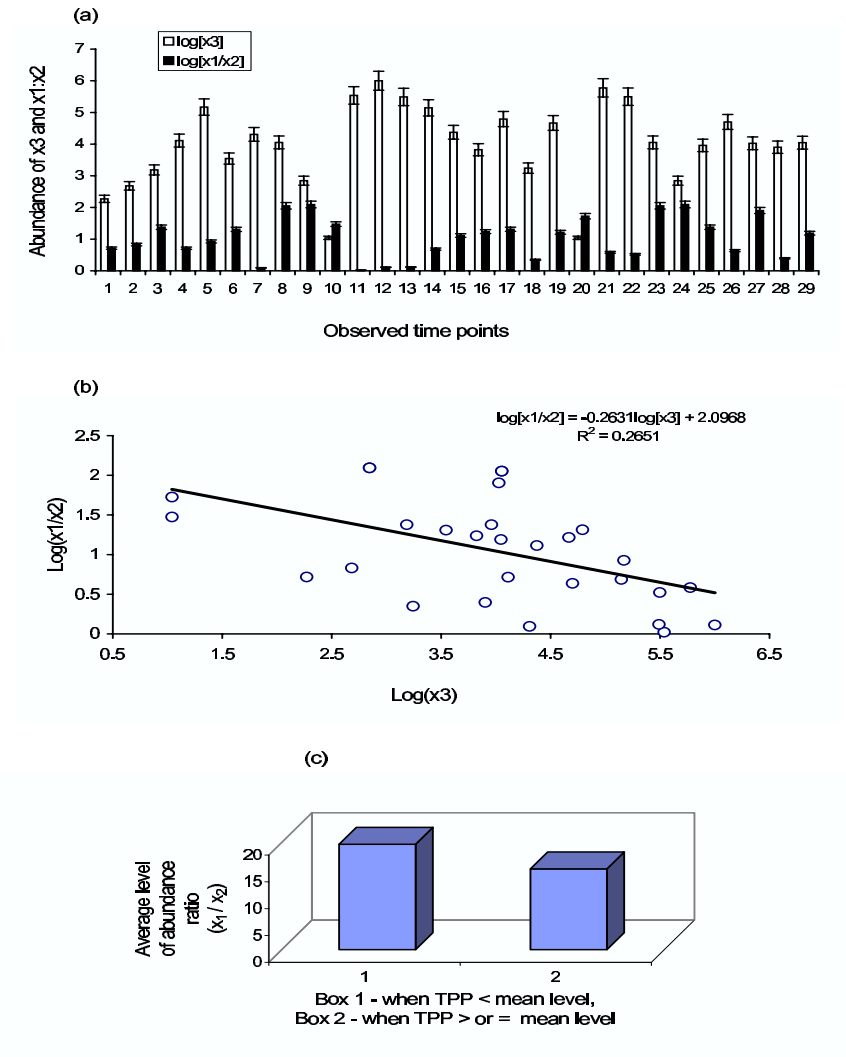

Figure 2: Distribution of the abundance ratio of *Coscinodiscus sp* to *Biddulphia sp* and the abundance of *Chaetoceros sp*. 2(a) Bar diagram depicting the abundance ratio of x_1 to x_2 and abundance of x_3. Log(x_1/x_2) reduces by 25 % when TPP is sufficiently present in the system. 2(b) Abundance ratio of *Coscinodiscus sp* to *Biddulphia sp* against the abundance of *Chaetoceros sp* (in log form) depicts a negative slope of the trend line. 2(c) Levels of average abundance ratios x_1/x_2 corresponding to TPP biomass at 'less than mean level' (M_1) and 'greater than mean level' (M_2). Compared with category M_1, the average level of x_1/x_2 decreases to category M_2 by 25%

time if the TPP biomass belongs to category M_1, and that TPP is at high abundance if TPP biomass belongs to M_2. Now, corresponding to those time points where TPP biomass belongs to M_1, let the average of the x_1/x_2 ratios be m_1. And corresponding to category M_2, let the average of the x_1/x_2 ratios be m_2. The the quantity $\delta_m = \frac{(m_2-m_1)}{m_1} \times 100$ may represent the change in the mean level of the abundance of *Coscinodiscus* to *Biddulphia* when the biomass of *Chaetoceros sp* is sufficiently high compared with when its abundance is low in the system. We find that δ_m is around (-25%), i.e., the mean level of x_1/x_2 reduces by 25% when TPP abundance is high (see, Figure 2(c)). These results suggest that the presence of toxic phytoplankton is favourable for the existence of the weak species in marine ecosystem. Hulot & Huisman (2004) claimed that, because of the toxic compounds released by the TPP, the competitive disadvantage between phytoplankton species is reduced. Our field observation also resembles the claim. By releasing allelopathic chemicals, the toxic species of phytoplankton gain a significant advantage in resource competition. Moreover, these toxic chemicals affect significantly the growths of the other competitors. Due to the presence of toxic chemicals, the species of non-toxic phytoplankton can hardly impose any competitive effect on these allelopathic species. So, the competition coefficient between a toxic and a non-toxic phytoplankton is negligible (Solé et al. 2005). In this way, in a mixed-species environment, the allelopathic species exhibit a passive mutualism towards the weak species, and promote those species to survive in competition (also found in Roy *et al. submitted*). Although not presented here, some other triad of two non-toxic and a toxic species that are present in dominant biomass would also exhibit a similar dynamics (Roy *et al.* submitted). We would like to mention that while using the correlation analysis and linear regression, we have ignored the data autocorrelation, a well-known analysis for a time series. However, because the data series considered is short and discontinuous, it is difficult to use the techniques specific for time series analysis. An entirely different approach for estimation of the missing values by an imputation method called Expectation-Maximization, and analysis of autocorrelation by a Vector Auto-regressive model also supports the results obtained here; the details of this analysis is reported in Roy *et al. submitted*.

Based on these arguments, to explore and display the dynamic behaviour of the competing phytoplankton species taking into account the presence of allelopathic species, in the following sections we propose and analyze a simple mathematical model. The main

objective of the analysis of the model is to find suitable mathematical bounds on the toxin-allelopathy parameters, under a constant environment as well as under a stochastic environment.

3 The Mathematical Model

To develop a mathematical model for describing the interaction among two non-toxic phytoplankton (species 1 with biomass x_1 and species 2 with biomass x_2) and a toxic phytoplankton (species 3 with biomass x_3), we make the following assumptions,
(i) The non-toxic phytoplankton species (species 1 and 2) compete for the same resource following the Lotka-Volterra competition model, where species 1 is the stronger competitor than species 2.
(ii) Allelopathic interactions between a non-toxic and a toxin-producing phytoplankton is described by a nonlinear function suggested by Solé *et al.* (2005).
(iii) Competitive interaction between a non-toxic and a toxic phytoplankton is negligible (Solé *et al.* , 2005)

Based on the above assumptions, the interaction among two non-toxic and a toxic phytoplankton is represented in the following mathematical model:

$$\begin{aligned} \frac{dx_1}{dt} &= x_1 \left(r_1 - \alpha_1 x_1 - \beta_{12} x_2 - \gamma_1 x_1 {x_3}^2\right), \\ \frac{dx_2}{dt} &= x_2 \left(r_2 - \alpha_2 x_2 - \beta_{21} x_1 - \gamma_2 x_2 {x_3}^2\right), \\ \frac{dx_3}{dt} &= x_3 \left(r_3 - \alpha_3 x_3\right), \end{aligned} \tag{1}$$

The model is analyzed under the following initial conditions:

$$x_1(0) > 0,\ x_2(0) > 0,\ x_3(0) > 0. \tag{2}$$

Here, r_i $(i = 1, 2, 3)$ are the specific growth rates of species i, α_i are the coefficients of intraspecific competition, β_{12} and β_{21} are the interspecific competition coefficients between x_1 and x_2, γ_i $(i = 1, 2)$ are the strengths of toxin-allelopathy between toxic and non-toxic phytoplankton.

3.1 Local stability analysis

The model system (1) has the following equilibria:
$E_0\,(0,\,0,\,0)$, $E_1\left(\frac{r_1}{\alpha_1},\,0,\,0\right)$, $E_2\left(0,\,\frac{r_2}{\alpha_2},\,0\right)$, $E_3\left(0,\,0,\,\frac{r_3}{\alpha_3}\right)$,
$E_4\left(\frac{\alpha_2\,r_1-\beta_{12}\,r_2}{\alpha_1\,\alpha_2-\beta_{12}\,\beta_{21}},\,\frac{\alpha_1\,r_2-\beta_{21}\,r_1}{\alpha_1\,\alpha_2-\beta_{12}\,\beta_{21}},\,0\right)$, $E_5\left(\frac{r_1\,\alpha_3^2}{\alpha_1\,\alpha_3^2+\gamma_1\,r_3^2},\,0,\,\frac{r_3}{\alpha_3}\right)$, $E_6\left(0,\,\frac{r_2\,\alpha_3^2}{\alpha_2\,\alpha_3^2+\gamma_2\,r_3^2},\,\frac{r_3}{\alpha_3}\right)$,
and the interior equilibrium $E^*(\,x_1^*,\,x_2^*,\,x_3^*)$,

where,

$$x_1^* = \frac{{\alpha_3}^2 {r_3}^2 r_1\,\gamma_2 + {\alpha_3}^4\,(\alpha_2\,r_1\,-\,r_2\,\beta_{12})}{(\alpha_2\,{\alpha_3}^2+\gamma_2\,{r_3}^2)\;{r_3}^2\,\gamma_1+{\alpha_3}^2\,(-{\alpha_3}^2\beta_{21}\,\beta_{12}+{\alpha_3}^2\alpha_2\,\alpha_1+\gamma_2\,{r_3}^2\alpha_1)}, \tag{3}$$

$${x_2}^* = \frac{{\alpha_3}^2 {r_3}^2 r_2\,\gamma_1 + {\alpha_3}^4\,(r_2\,\alpha_1\,-\,r_1\,\beta_{21})}{(\alpha_2\,{\alpha_3}^2+\gamma_2\,{r_3}^2)\;{r_3}^2\,\gamma_1+{\alpha_3}^2\,(-{\alpha_3}^2\beta_{21}\,\beta_{12}+{\alpha_3}^2\alpha_2\,\alpha_1+\gamma_2\,{r_3}^2\alpha_1)} \tag{4}$$

and

$$x_3^* = \frac{r_3}{\alpha_3}. \tag{5}$$

For any non-negative set of values of the model-parameters, the equilibria E_0, E_1, E_2, E_3, E_5 and E_6 exist. A sufficient condition on the parameters for feasibility of E_4 is

$$\alpha_1\,\alpha_2 \;>\; \beta_{12}\,\beta_{21} \tag{6}$$

The interior equilibrium E^* exists if the following set of inequalities hold

$$\gamma_1 \;>\; \frac{{\alpha_3}^2\,r_1}{{r_3}^2\,r_2}\,\beta_{21}, \tag{7}$$

$$\gamma_2 \;>\; \max\left\{\,\frac{{\alpha_3}^2\,r_2}{{r_3}^2\,r_1}\,\beta_{12},\;\frac{{\alpha_3}^2\;(\beta_{12}\,\beta_{21}\,-\,\alpha_1\,\alpha_2)}{\alpha_1\,{r_3}^2}\,\right\}. \tag{8}$$

We find that the coexistence of the interior equilibrium depends on the strength of the toxin-allelopathy parameters. On generating the community matrix, we perform local-stability analysis (LAS) of the model system (1) around each biologically feasible equilibrium. In the following theorem, we summarize the results of the LAS.

Theorem 3.1: *The boundary equilibria (E_0, E_1, E_2, E_3, E_4, E_5 and E_6) are repellers under the following conditions,*

$$\alpha_1\,+\gamma_1\left(\frac{r_3}{\alpha_3}\right)^2 \;>\; \beta_{21}\,\frac{r_1}{r_2}, \tag{9}$$

$$\alpha_2\,+\gamma_2\left(\frac{r_3}{\alpha_3}\right)^2 \;>\; \beta_{12}\,\frac{r_2}{r_1}. \tag{10}$$

The interior equilibrium point E^* is locally asymptotically stable if the following condition holds

$$\gamma_1 \gamma_2 \geq \left(\frac{\alpha_3}{r_3}\right)^4 \beta_{12} \beta_{21} . \tag{11}$$

(Proof is obvious)

In the absence of the toxic phytoplankton (x_3), the model system (1) reduces to the well known Lotka-Volterra (LV) competition model. It is well established that LV model exhibits competitive exclusion of one or both the competitors if any one or both of the following conditions hold (for detail see, Kot, 2001)

$$\beta_{21} \frac{r_1}{r_2} > \alpha_1 \tag{12}$$

$$\beta_{12} \frac{r_2}{r_1} > \alpha_2 \tag{13}$$

Now we are in a position to compare the inequalities obtained in (9)-(10) and (12)-(13). It follows from Theorem (3.1) that, even if any one or both of the conditions (12)-(13), that are necessary for competitive exclusion in LV model, is satisfied, toxin allelopathy due to TPP, the strength of which satisfies the conditions (9)-(10), promotes coexistence of the competitive phytoplankton species.

In the following section, to study the dynamics of the interacting species under a variable environment, we extend the scope of deterministic model to a stochastic set up.

4 The Stochastic Model

We assume that the stochastic perturbations of the variables around E^* are of white-noise type proportional to the distances of x_1, x_2, x_3 from the values ${x_1}^*, {x_2}^*, x_3^*$ (Beretta *et al.*, 1998). Under this assumption, system (1) takes the following form

$$\begin{aligned} dx_1 &= [\, x_1 \left(r_1 - \alpha_1 x_1 - \beta_{12} x_2 - \gamma_1 x_1 {x_3}^2\right)]\, dt + \sigma_1 (x_1 - {x_1}^*)\, d{\xi_t}^1, \\ dx_2 &= [\, x_2 \left(r_2 - \alpha_2 x_2 - \beta_{21} x_1 - \gamma_2 x_2 {x_3}^2\right)]\, dt + \sigma_2 (x_2 - {x_2}^*)\, d{\xi_t}^2, \\ dx_3 &= [\, x_3 \, (r_3 - \alpha_3 x_3)]\, dt + \sigma_3 (x_3 - x_3^*)\, d{\xi_t}^3. \end{aligned} \tag{14}$$

Here σ_i, $(i = 1, 2, 3)$ are real constants defined as the intensities of the stochasticity, and ${\xi_t}^i = \xi_i(t)$, $(i = 1, 2, 3)$ are independent standard Wiener-process (Gikhman and Skorokhod, 1974, 1975, 1979).

To investigate the robustness of the dynamical behaviour of model (1), stochastic stability of the interior equilibrium E^* is studied using the model (14). System (14) can be represented as an Ito Stochastic Differential Equation of the following type

$$\begin{aligned} dX_t &= f(t, X_t)\, dt + g(t, X_t)\, d\xi_t, \\ X_{t_0} &= X_0,\ t \in [t_0, t_f], \end{aligned} \tag{15}$$

where the solution $\{X_t, t \in [t_0, t_f]\ (t > 0)\}$ is an Ito process, f is the slowly varying continuous component or drift coefficient and g is the rapidly varying continuous random component or diffusion coefficient (Kloeden and Platen, 1995), ξ_t is a multi-dimensional stochastic process having scalar Wiener-process components with increments $\Delta {\xi_t}^j = {\xi_{t+\Delta t}}^j - {\xi_t}^j = \xi_j(t + \Delta t) - \xi_j(t)$, which are independent Gaussian random-variables $N(0, \Delta t)$.

Comparing (14) and (15), we have

$$X_t = (x_1, x_2, x_3)^T,\ \xi_t = ({\xi_t}^1, {\xi_t}^2, {\xi_t}^3)^T, \tag{16}$$

$$f = \begin{pmatrix} x_1\ (r_1 - \alpha_1\, x_1 - \beta_{12}\, x_2 - \gamma_1\, x_1\, {x_3}^2) \\ x_2\ (r_2 - \alpha_2\, x_2 - \beta_{21}\, x_1 - \gamma_2\, x_2\, {x_3}^2) \\ x_3\ (r_3 - \alpha_3\, x_3) \end{pmatrix} \tag{17}$$

and

$$g = \begin{pmatrix} \sigma_1(x_1 - {x_1}^*) & 0 & 0 \\ 0 & \sigma_2(x_2 - {x_2}^*) & 0 \\ 0 & 0 & \sigma_3(x_3 - x_3^*) \end{pmatrix} \tag{18}$$

Since the diffusion matrix (18) depends on the solution $X_t = (x_1, x_2, x_3)^T$, system (14) is said to have multiplicative noise. From the diagonal form of the diffusion matrix (18), system (14) is said to have (multiplicative) diagonal noise.

4.1 Stochastic stability of the interior equilibrium

By defining the variables $u_1 = x_1 - {x_1}^*,\ u_2 = x_2 - {x_2}^*,\ u_3 = x_3 - x_3^*$, the stochastic differential equations (14) can be centered at the interior equilibrium E_*.

To show that system (14) is asymptotically stable in mean square sense (or in probability) we linearize the vector function f around the positive equilibrium E^*. The linearized stochastic differential equations around E^* (using the variational matrix J) take the following form

$$du(t) = f(u(t))\, dt + g(u(t))\, d\xi(t) \tag{19}$$

where $u(t) = (u_1(t), u_2(t), u_3(t))^T$ and

$$f(u(t)) \;=\; \begin{bmatrix} x_1^* \left(-\alpha_1 - \gamma_1\, x_3^{*2}\right) u_1 - x_1^*\, \beta_{12}\, u_2 - 2\, x_1^{*2} \gamma_1\, x_3^*\, u_3 \\ -x_2^*\, \beta_{21}\, u_1 + x_2 \left(-\alpha_2 - \gamma_2\, x_3^{*2}\right) u_2 - 2\, x_2^{*2} \gamma_2\, x_3^*\, u_3 \\ -\alpha_3\, x_3^*\, u_3 \end{bmatrix} \tag{20}$$

$$g(u(t)) \;=\; \begin{pmatrix} \sigma_1\, u_1 & 0 & 0 \\ 0 & \sigma_2\, u_2 & 0 \\ 0 & 0 & \sigma_3\, u_3 \end{pmatrix}, \tag{21}$$

with the parametric conditions for existence of E^* stated in section (3.1). Clearly, the positive equilibrium E^* in equation (19) corresponds to the trivial solution $(u_1, u_2, u_3) = (0, 0, 0)$.

Let us define a set $\Psi = \{(t \geq t_0) \times R^3,\ t_0 \in R^+\}$. Now there exists a function $V \in C_2^0(\Psi)$ such that V is twice continuously differentiable (i.e., a C^2 function) with respect to u and continuous (i.e., C^0) with respect to t. With reference to (19), we define the following function:

$$W(u,t) = \frac{\partial V(u(t),t)}{\partial t} + f^T(u(t))\, \frac{\partial V(u,t)}{\partial u} + \frac{1}{2}\, Tr\, [\, g^T(u(t))\, \frac{\partial^2 V(u,t)}{\partial u^2}\, g(u(t))\,], \tag{22}$$

where $\frac{\partial V}{\partial u} = \left(\frac{\partial V}{\partial u_1}, \frac{\partial V}{\partial u_2}, \frac{\partial V}{\partial u_3}\right)^T$, $\frac{\partial^2 V(u,t)}{\partial u^2} = (\frac{\partial^2 V}{\partial u_j \partial u_i})_{i,j=1,2,3}$ and 'T' stands for transposition. Now, we state the following theorem due to Afanasev *et al.* (1996).

Theorem 4.1: *Suppose there exists a function $V(u,t) \in C_2(\Psi)$ satisfying the inequalities*

$$K_1|u|^p \leq V(u,t) \leq K_2|u|^p, \tag{23}$$

$$W(u,t) \leq -K_3|u|^p, \; K_i > 0, \; p > 0 \; (i = 1,2,3). \tag{24}$$

Then the trivial solution of (19) is exponentially p-stable for $t \geq 0$.

We note that, if $p = 2$ in (23) and (24) then the trivial solution of (19) is exponentially mean-square stable. Furthermore, the trivial solution of (19) is globally asymptotically stable in probability (Afanasev *et al.*, 1996). From the standard stability analysis of the stochastic model (14), we state the following theorem.

Theorem 4.2: *Along with the existence criterion for E^* (condition (7)-(8)) as stated in section (3.1), if the following condition holds*

$$\gamma_1 \gamma_2 > \left(\frac{\alpha_3}{r_3}\right)^4 \left(\frac{\sigma_1^2 \sigma_2^2 \sigma_3^2}{8 r_3}\right) \tag{25}$$

then the trivial solution of system (14) is asymptotically mean-square stable.
(For proof see Appendix)

We recall that, σ_is $(i = 1,2,3)$ represent the intensities (or rapidity) of the environmental fluctuations. The above theorem demonstrates that suitable values of the growth rate of TPP and the intensity of the toxin-allelopathy parameters determine the stochastic stability. Hence similar to deterministic environment, toxin allelopathy might also be a potential candidate for preventing competitive exclusion among the phytoplankton species in stochastic environment. Recalling condition (11) for the stability of coexisting equilibrium in the deterministic model, we find that along with the existence criterion (in section 3.1), if the product of the strengths of toxin allelopathy (i.e., γ_1 and γ_2) is bigger in magnitude than $\max\{\left(\frac{\alpha_3}{r_3}\right)^4 (\beta_{12}\,\beta_{21}), \left(\frac{\alpha_3}{r_3}\right)^4 \left(\frac{\sigma_1^2 \sigma_2^2 \sigma_3^2}{8 r_3}\right)\}$, then the dynamics of the coexisting competitive-phytoplankton species is locally stable, both in constant and fluctuating environment.

5 Numerical experiments

Dynamics when no toxic species present

When the toxic phytoplankton is absent in the system, without any loss of generality, let the two component model have hypothetical parameter values as follows,
$r_1 = 0.6$ day^{-1}, $r_2 = 0.6$ day^{-1}, $\alpha_1 = 0.01$ biomass^{-1} day^{-1}, $\alpha_2 = 0.04$ biomass^{-1} day^{-1}. Because species 1 is assumed to be a stronger competitor than species 2, for the following numerical simulations we take $\beta_{21} > \beta_{12}$. Now the two-component model of the competitive-phytoplankton species in absence of toxic algae is simulated. We fix $\beta_{12} = 0.02$ biomass^{-1} day^{-1}, and vary β_{21}. A suitable range of the competition coefficient β_{21} is found ($0.021 \leq \beta_{21} \leq 0.05$) for which the weak competitor goes to extinction, both in deterministic and stochastic model (Figure 3(a)-3(b)).

Dynamics when toxic species is included

Now we introduce the toxic algae and simulate the three-component model with parameters for the two-component model left unchanged. Suitable values of the toxin-allelopathy parameters are found for which all the species coexist (Figure 3). The coexisting equilibrium is stable under deterministic as well as stochastic set up (Figure 3(c)-(3(d)). A gradual increase in the intensity of allelopathy show that, a reasonably large range of each of those allelopathic parameters is obtainable, even beyond the reported ranges (Solé *et al.*, 2005), for which the weak and the strong species stably coexist with non-zero equilibrium biomass (Figure 4). In other words, this result shows that the stable coexistence of the all the species is robust with respect to the allelopathic effect. Following these arguments, we suggest that, in the presence of a toxic alga, the possibility for a competitive exclusion of the weak species of non-toxic phytoplankton might be overturned.

Dynamics on consideration of competition coefficient between TPP and NTP explicitly

As already mentioned, by releasing allelopathic chemicals that affect the growth of other species, a toxic phytoplankton gains an advantage in competition. Hence the competing effects of non-toxic phytoplankton on the toxic phytoplankton is negligible (Hulot & Huisman, 2004, Roy *et al. submitted*), and can be ignored in modeling the interaction of a non-toxic and toxic algae (Solé *et al.*, 2005). Keeping these observations in mind, in

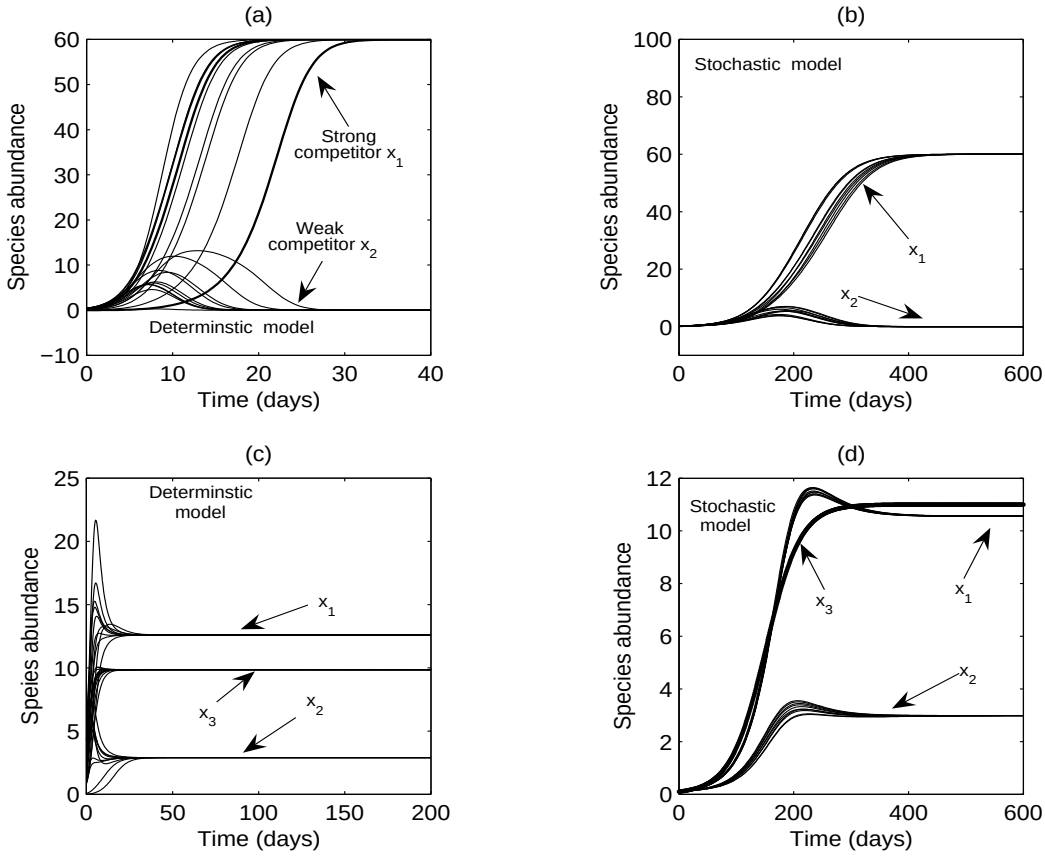

Figure 3: (a) Competitive exclusion of the weak competitor (species 2) in the absence of TPP for the deterministic model (1). The fixed parameters are $r_1 = 0.6\,\text{day}^{-1}$, $r_2 = 0.6\ \text{day}^{-1}$, $\alpha_1 = 0.01\,\text{biomass}^{-1}\,\text{day}^{-1}$, $\alpha_2 = 0.04\,\text{biomass}^{-1}\ \text{day}^{-1}$, $\beta_{12} = 0.02$ $\text{biomass}^{-1}\,\text{day}^{-1}$; for $0.021 \leq \beta_{21} \leq 0.05$, for which species 1 persists but species 2 goes extinct. (b) Competitive exclusion of the weak competitor (species 2) in the absence of TPP for the stochastic model (14), with the parameters fixed as in 3(a) and $\sigma_1 = 0.0004$, $\sigma_2 = 0.0005$. Stable coexistence of the competitive species in presence of the TPP: (c) deterministic stability of the model model system (1); all the species coexists in the same range $0.021 \leq \beta_{21} \leq 0.05$, due to introduction of the TPP with $r_3 = 0.66\,\text{day}^{-1}$, $\alpha_3 = 0.06\,\text{biomass}^{-1}\,\text{day}^{-1}$, $\gamma_1 = 0.00034\,\text{biomass}^{-3}\,\text{day}^{-1}$, $\gamma_2 = 0.00006\,\text{biomass}^{-3}\,\text{day}^{-1}$ and the other parameters fixed as in 3(a); (d) stochastic stability of the model system (14); stable coexistence of all the species even under stochastic perturbation, with intensity of stochasticity $\sigma_1 = 0.00036$, $\sigma_2 = 0.005$, $\sigma_3 = 0.00037$.

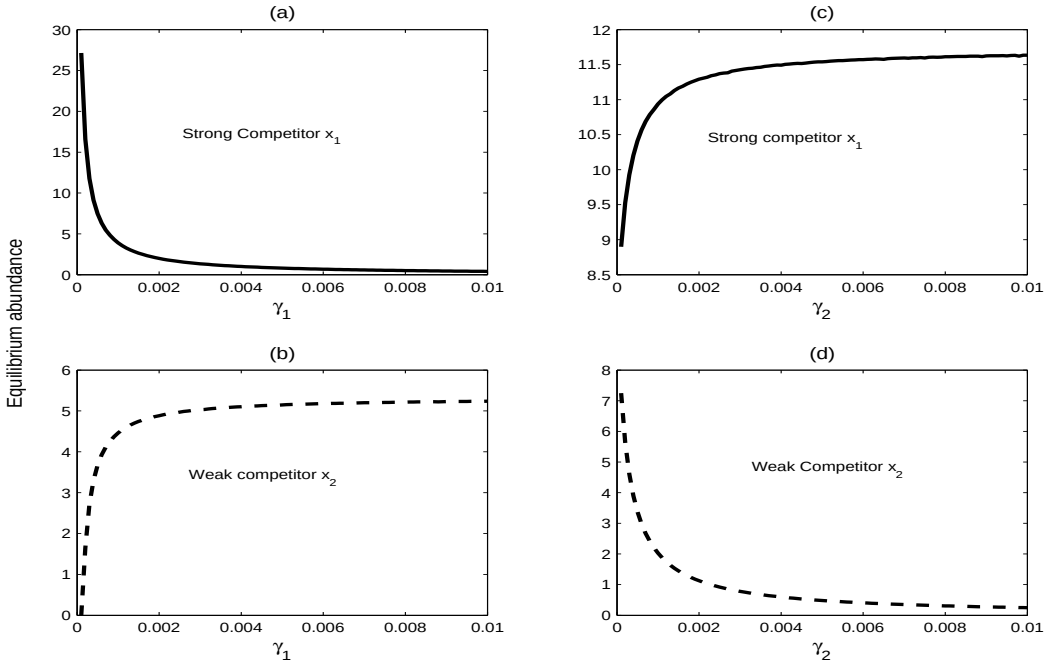

Figure 4: Equilibrium abundance of the strong and weak competitor with variation in the allelopathic intensity. The reported range of allelopathic parameter in Solé *et al.* (2005) is 10^{-6} to 10^{-5}. However, stable coexistence is found for a very large range of γ_1 and γ_2. Left panels (a) & (b) depicts the variation of the positive-equilibrium abundance of x_1 and x_2 for a large range $0 \leq \gamma_1 \leq 0.01$. Right panels (c) & (d) depict the same for a large range $0 \leq \gamma_2 \leq 0.01$.

our three-component model, we have not considered any competition coefficient between a toxic and a non-toxic phytoplankton. However, for the the completeness of our study, and for strengthening the arguments drawn for the stable coexistence of the species, let us now explicitly introduce the effects of weak competition between x_1-x_3 and x_2-x_3, represented by the coefficients β_{13} and β_{23} respectively. Along with the unaltered forms of the first two equations, the third equation of model system (1) now takes the following form,

$$\frac{dx_3}{dt} = x_3 \left(r_3 - \alpha_3 x_3 - \beta_{13} x_1 - \beta_{23} x_2\right). \tag{26}$$

To examine the effect of the competition coefficients β_{13} and β_{23}, the values of which are very low because of the presence of toxin allelopathy, we simulate the new form of the model system (1) with the other parameters kept fixed as in Figure (3). Again, similar to the previous case, the model shows a stable coexistence of all the species (Figure 5). Moreover, provided that the weak-competition coefficients β_{13} and β_{23} are bounded within reasonable ranges, and do not attain high values, positive equilibrium exists and is stable (Figure 6). Because x_2 is considered as the weak competitor that undergoes competitive exclusion in the absence of the toxic species, the stability of the system tolerates much higher range of the parameter β_{23} than that of β_{13} (Figure 6).

6 Discussion

There is no universally-accepted explanation on how a large number of species of phytoplankton co-exist on a limited variety of resources (violating the principle of competitive exclusion). Either the external factors such as weather or oscillation and chaos generated by competition among the species were cited for probable explanations (Hutchinson, 1961; Richerson *et al.*, 1970; Huisman & Weissing, 1999). However, in plankton community, the presence of TPP is remarkable in this context. On an analysis of a set of field-data that we have collected from the North-West coast of the Bay of Bengal, here we propose a possible role of toxin-allelopathy that might be responsible for a stable coexistence of the competing phytoplankton. Analysis of our field data suggests that toxic or allelopathic compounds liberated by TPP may be helpful for reduction of the competition coefficient among phytoplankton species (Section 2, also claimed by Hulot & Huisman, 2004). Based on our field observations and following the study of Solé *et al.* (2005), we have pro-

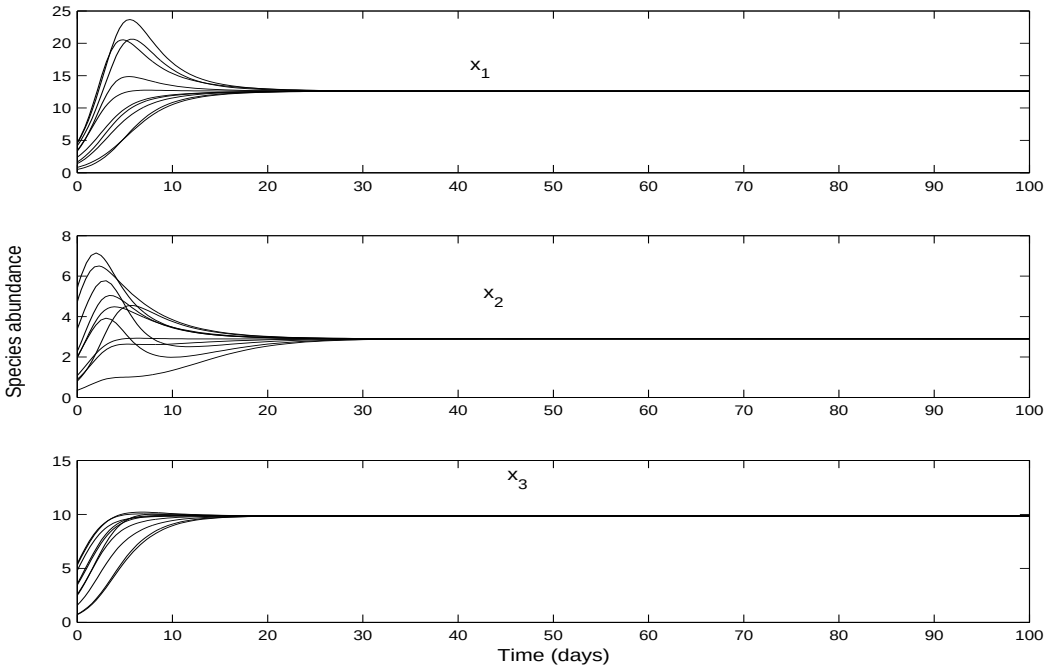

Figure 5: Time series solution of the model system (1), when the weak competition effect of non-toxic species on the toxic species is considered explicitly: $\frac{dx_3}{dt} = x_3\,(r_3 - \alpha_3\,x_3 - \beta_{13}\,x_1 - \beta_{23}\,x_2)$. Stable coexistence of all the species for $\beta_{13} = 0.005$, $\beta_{23} = 0.002$, and other parameters fixed as before.

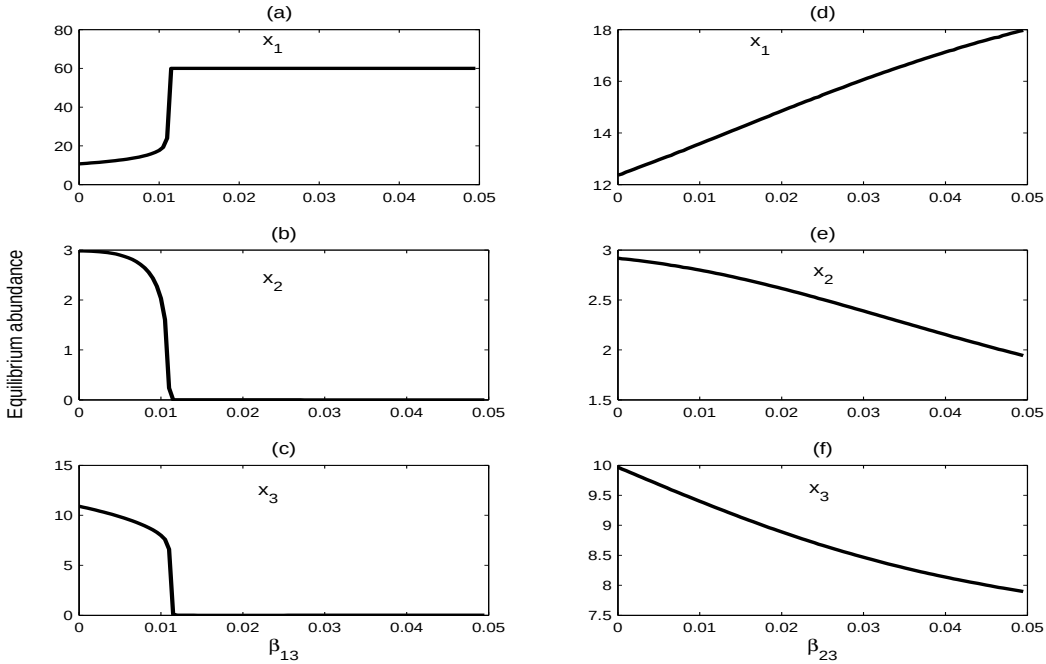

Figure 6: Numerical bounds of the weak competition coefficients β_{13} and β_{23} corresponding to other parameters of the model fixed as in Figure (1). Left panels (a)-(c) depict the variation of equilibrium density of x_1, x_2 and x_3 respectively corresponding to β_{13}. Right panels (d)-(f) depicts the same corresponding to β_{23}. The Figures depict that the stability of the system tolerates much higher range of the parameter β_{23} than that of β_{13}; a result desirable because x_2 is considered as the weak competitor that undergoes competitive exclusion in the absence of the toxic species.

posed and analyzed a three-component mathematical model to describe the interactions among two non-toxic and a toxic phytoplankton. Starting from a simple two-component Lotka-Volterra competition model representing competition between two non-toxic phytoplankton, the three-component model is developed on introducing a third population floor occupied by a toxic phytoplankton. The analysis in Section 3 demonstrates that, the strength of toxin-allelopathy determines the coexistence of the competing-phytoplankton species that would otherwise exhibit a competitive exclusion of the weak competitor. Restrictions on the strength of toxin-allelopathy is found that promotes the coexistence of the phytoplankton species. Moreover, for some ranges of toxin-allelopathy parameters, the dynamics of the competitors is stabilized. The conditions for stability, as we have found, are suitable restrictions on the allelopathic interactions among non-toxic and toxic phytoplankton species, not driven by external factors. The dynamics of the competing-phytoplankton species is also explored in a variable environment. The analysis in Section 4 determines the restrictions on the parameters of toxin-allelopathy that determine the stability of the coexisting equilibrium under stochastic fluctuation. These results establish that the growth rate of toxic phytoplankton and the strength of toxin allelopathy act as potential parameters for determining the dynamic behaviour of the competing phytoplankton species, both in a constant and a fluctuating environment. Finally, for a set of hypothetical parameters of the model system, numerical simulations have been performed. Our results show that the possibility of competitive exclusion among phytoplankton species is overturned because of the presence of toxin-allelopathy. The overall study suggests that, although at a species-level interaction toxin-allelopathy due to a TPP is harmful for the growth of a NTP species, for the competitive interaction of many NTP species the presence of TPP might be favourable for the stable coexistence of those species that would otherwise not coexist. In marine ecosystem where a large number of phytoplankton species coexist, TPP might be a potential candidate that, by releasing chemicals, influence on the competitive interaction among the species, and might promote the survival of the weak species.

Although the general conclusions drawn from our study follow from the analysis of the filed samples and that of mathematical models, we would like to mention some of the limitations of our field study that could be overcome by a number of complimentary studied. Because the data that we have used for our analysis is restricted to a field study, the results of the statistical analysis might associate factors that are not detectable from

a field study without any laboratory experiments. For instance, although the biomass coefficient (x_1/x_2) of the two non-toxic species has a significant negative correlation with the abundance of TPP species, the effect of the abundance of TPP may not be the only cause for reducing the abundance ratio x_1/x_2. There may be several other causes such as different sensitivity of the analyzed algae to the toxin or indirect effects such as zooplankton and TPP relationship, that can be crucial in this context. However, due to lack of experimental evidences along with our field observations, it is physically impossible for us to eliminate such effects. Finally, we suggest that, a number of extensive field studies in multiple locations would be necessary to establish the implications of our study in explaining the diversity of phytoplankton in natural waters.

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Appendix

Proof of Theorem (4.2):

Let us consider the following Lyapunov function

$$V(u(t),t) = \frac{1}{2}\,[\,{u_1}^2 + w_2\,{u_2}^2 + w_3\,{u_3}^2\,] \tag{27}$$

where w_i $(i = 1, 2, 3)$ are real positive-constants to be chosen suitably. It can be easily verified that the inequality (22) holds for $p = 2$. Now,

$$\frac{\partial^2 V}{\partial u^2} = \begin{bmatrix} 1 & 0 & 0 \\ 0 & w_2 & 0 \\ 0 & 0 & w_3 \end{bmatrix} \tag{28}$$

Hence,

$$g^T(u(t))\,\frac{\partial^2 V}{\partial u^2}\,g(u(t)) = \begin{bmatrix} {\sigma_1}^2\,{u_1}^2 & 0 & 0 \\ 0 & w_2\,{\sigma_2}^2\,{u_2}^2 & 0 \\ 0 & 0 & w_3\,{\sigma_3}^2 {u_3}^2 \end{bmatrix}, \tag{29}$$

so,

$$\frac{1}{2} Tr\,[\,g^T(u(t))\,\frac{\partial^2 V}{\partial u^2}\,g(u(t))\,] \;=\; \frac{1}{2}\left(\,{\sigma_1}^2 {u_1}^2 + w_2\,{\sigma_2}^2\,{u_2}^2 + w_3\,{\sigma_3}^2\,{u_3}^2\right). \tag{30}$$

Again

$$\begin{aligned} f^T(u(t))\,\frac{\partial V(u,t)}{\partial u} \;=\; & x_1^*\,\left(-\alpha_1 - \gamma_1\,x_3^{*2}\right){u_1}^2 + x_2^*\,\left(-\alpha_2 - \gamma_2\,x_3^{*2}\right) w_2\,{u_2}^2 - \\ & \alpha_3\,x_3^*\,{u_3}^2 w_3 - 2\,u_3\,x_2^{*2}\gamma_2\,x_3^*\,w_2\,u_2 - 2\,x_1^{*2}\gamma_1\,x_3\,u_3\,u_1 - (x_2^*\,\beta_{21}\,w_2 + x_1^*\,\beta_{12})\,u_1\,u_2 \end{aligned} \tag{31}$$

Therefore,

$$\begin{aligned} W(u(t)) = -[\frac{1}{2}\,w_3\,\left(2\,\alpha_3\,x_3^* - {\sigma_3}^2\right){u_3}^2 + \left(-\frac{1}{2}\,{\sigma_1}^2 + x_1^*\,\left(\alpha_1 + \gamma_1\,x_3^{*2}\right)\right){u_1}^2 + \\ \left(x_2^*\,w_2\,\left(\alpha_2 + \gamma_2\,x_3^{*2}\right) - \frac{1}{2}\,w_2\,{\sigma_2}^2\right){u_2}^2 + (x_2^*\,\beta_{21}\,w_2 + x_1^*\,\beta_{12})\,u_1\,u_2 + \\ 2\,u_3\,x_2^{*2}\gamma_2\,x_3^*\,w_2\,u_2 + 2\,{x_1}^2\gamma_1\,x_3^*\,u_3\,u_1]. \end{aligned} \tag{32}$$

Let the following conditions hold

$${\sigma_1}^2 \;<\; 2\,x_1^*\,\left(\alpha_1 + \gamma_1\,x_3^{*2}\right),\;\; \sigma_2^2 \;<\; 2\,x_2^*\,\left(\alpha_2 + \gamma_2\,x_3^{*2}\right),\;\; \sigma_3^2 \;<\; 2\,\alpha_3\,x_3^* \tag{33}$$

Then $W(u(t))$ can be written in the following form

$$W(u(t)) \;=\; -u^T\,Q\,u \tag{34}$$

where $u \;=\; (u_1, u_2, u_3)^T$ and Q is the following positive definite symmetric-matrix

$$Q \;=\; \begin{bmatrix} -\frac{1}{2}\,{\sigma_1}^2 + x_1^*\,\left(\alpha_1 + \gamma_1\,x_3^{*2}\right) & \frac{1}{2}\,x_2^*\,\beta_{21}\,w_2 + \frac{1}{2}\,x_1^*\,\beta_{12} & x_1^{*2}\gamma_1\,x_3^* \\ \frac{1}{2}\,x_2^*\,\beta_{21}\,w_2 + \frac{1}{2}\,x_1^*\,\beta_{12} & x_2^*\,w_2\,\left(\alpha_2 + \gamma_2\,x_3^{*2}\right) - \frac{1}{2}\,w_2\,{\sigma_2}^2 & x_2^{*2}\gamma_2\,x_3^*\,w_2 \\ x_1^{*2}\gamma_1\,x_3^* & x_2^{*2}\gamma_2\,x_3^*\,w_2 & \frac{1}{2}\,w_3\,\left(2\,\alpha_3\,x_3^* - {\sigma_3}^2\right) \end{bmatrix}$$

Clearly, the eigenvalues of Q namely, $\lambda_1, \lambda_2, \lambda_3$ are real positive quantities. If λ_m denotes the minimum of $\lambda_1, \lambda_2, \lambda_3$, then from (21) we get

$$W(u,t) \leq -\lambda_m |u(t)|^2 \tag{35}$$

Hence the trivial solution of system (14) is asymptotically mean-square stable.

Sufficient conditions for stability of the model system (1) under stochastic fluctuation, i.e., system (14), follow

$$\sigma_1{}^2 \; < \; 2\, x_1^* \left(\alpha_1 + \gamma_1\, x_3^{*2}\right), \tag{36}$$

$$\sigma_2^2 \; < \; 2\, x_2^* \left(\alpha_2 + \gamma_2\, x_3^{*2}\right), \tag{37}$$

$$\sigma_3^2 \; < \; 2\, \alpha_3\, x_3^* \tag{38}$$

Combining the above three conditions and after simplification, we find the following sufficient condition for stochastic stability

$$\gamma_1\, \gamma_2 \; > \; \left(\frac{\alpha_3}{r_3}\right)^4 \left(\frac{\sigma_1^2\, \sigma_2^2\, \sigma_3^2}{8\, r_3}\right)$$

Hence the theorem.