The coevolution of two phytoplankton species on a single resource: Allelopathy as a pseudo-mixotrophy

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1. Introduction

In aquatic systems the phytoplankton community evolves under the influence of numerous top-down and bottom-up effects, as well as that due to environmental fluctuations. The resultant of all these effects leads to an extreme diversity of phytoplankton species under a limited variety of resources (Hutchinson, 1961; review by Scheffer et al., 2003). It is generally accepted from the principle of competitive exclusion, that the number of species coexisting at equilibrium cannot exceed the number of limiting ‘factors’ (Hardin, 1960). In theoretical ecology, several mechanisms, based on resource partitioning, different forms of predation, biomass fluctuations and environmental factors, have been found for the coexistence of species (Chesson, 2000). In particular, for the phytoplankton community, since the resource-competition theories do not support a stable coexistence (Tilman, 1982), the extreme diversity of the species is generally understood through a non-equilibrium dynamics. Top down effects, resource fluctuation, complexity of species interaction and influence of physical forcing (such as mixing, advection) provide mechanisms for driving this non equilibrium—a detailed account of these mechanisms can be found in the reviews by Scheffer et al. (2003) and by Roy and Chattopadhyay (2007a). It is important to note that, without the top-down effects and the physical forces, none of the existing simple resource-competition models demonstrates a stable coexistence of two phytoplankton species under a single limiting resource.

In the context of functioning of aquatic systems, the release of chemical substances by the individuals of a species affecting the members of another species – known as allelopathy – has a significant role. Allelopathy has been reported in phytoplankton communities at least before three decades (e.g., Maestrini and Bonin, 1981; Mason et al., 1982). Since then there exists an accumulating evidence for the potential significance of allelopathy in phytoplankton interaction (Cembella, 2003; Hulot and Huisman, 2004; Solé et al., 2005). A number of laboratory experiments (Arzul et al., 1999; Granelli and Johansson, 2003; Fistarol et al., 2004; Schatz et al., 2005) as well as field studies (Rengefors and Legrand, 2001; Schagerl et al., 2002) have established that the toxic chemicals released by the group of toxin-producing phytoplankton (TPP) potentially act as allelopathic agents in the phytoplankton community. de Freitas and Fredrickson (1978) and Levin (1988) have introduced the effect of allelopathy in resource competition through mechanistic models. Moreover, several mechanistic models have been analyzed to understand...
the effect of inhibition, either from external or from internal sources (review, Hsu and Waltman, 2004). It has been shown mechanistically that toxin production might be helpful in some cases for coexistence: e.g., when the toxin production is a plasmid-encoded trait (Hsu and Waltman, 2004), or when toxin production is regulated in relation to competitor density, as might happen through quorum sensing mechanisms (Brasleton and Waltman, 2001). Recent results suggest that, by modulating the top-down effects of grazer zooplankton, the inhibitory effects of TPP can drive the planktonic non-equilibrium (Roy et al., 2006). Further, the effects of such ‘toxin-allelopathy’ of a TPP, present as a third species, can successfully overturn the competitive exclusion of two non-toxic phytoplankton undergoing a Lotka–Volterra interaction (Roy and Chattopadhyay, 2007b). However, concentrating on purely resource-competition models, it is fair to say that the works so far have suggested that some ‘particular’ properties are required to make coexistence possible, and that even the simplest mechanistic models of allelopathy have a strong tendency to exclusion.

In microbial ecology, the mixotrophic species are known to have a complicated role in competitive interaction as well as in food-web interaction (Davidson, 1996). The species of mixotrophic algae are an important component of phytoplankton communities in natural waters (e.g., Wiedner and Nixdorf, 1998). By switching to phagotrophy these species can sustain their growth when they are mixed out of euphotic zone (Bird and Kalff, 1989). In conditions of low radiation, temperature, salinity, pH, and situations when algal species are unlikely to meet their carbon requirement for photosynthesis, the species are known to survive through mixotrophy (Hammer, 2003; Hammer and Pitchford, 2006). Thus, mixotrophy contributes to the coexistence of algal species under limiting-resource conditions. However, recent studies have suggested that some algal species (e.g., Prymnesium) can be toxin producer as well as mixotrophic, and in such scenario they show a ‘kill and eat’ behaviour (Tilmann, 2003). Although, toxin production has been recognized as a distinctive feature of many algal species, not many species has yet been identified as a two-in-one package of mixotrophy and allelopathy.

Based on the growing body of evidences on plankton allelopathy, and the increasing interest on the mixotrophic interaction, here I address the question as to whether the allelopathy alone can act a potential factor for the competitive coevolution of two phytoplankton under a single resource. If so, is there any theoretical connection between the effect of a pure allelopathy and the well-known effect of mixotrophy on the survival or coexistence of microbial species. Further, is the coexistence dynamics driven by allelopathy stable? And under this context, how does allelopathy of toxin producers contribute to the resource-species relationship, at least in an ideal situation where the top-down effects do not come into play? To address these issues, I developed and analyze a mathematical model describing the competition for a single nutrient between a non-toxic phytoplankton and a toxin-producing phytoplankton. The results are discussed in the contexts of understanding the key roles of allelopathy and their similarity with mixotrophy on competitive exclusion and phytoplankton diversity.

2. Nutrient competition model under allelopathy

Nutrient competition models of phytoplankton are well studied in several contexts. Starting from a well-known resource-competition model, I construct a mathematical model for describing the interaction between a non-toxic (species 1 with biomass $P_1(t)$) and a toxic phytoplankton (species 2 with biomass $P_2(t)$) under a single nutrient (with concentration $N(t)$). The structure of the model without the effect of allelopathy is similar to that of a standard resource competition model used by many authors (such as Huisman and Weissing, 1999). Species 2 being toxic, the competitive interaction of the two species is affected significantly by its allelopathic ability.

The characteristic and the mode of action of the allelopathic chemicals in marine ecosystems is generally poorly understood and is still under investigation (Cembella, 2003). In general the presence of external and internal inhibition has been explored in a number of mathematical models, and the representations of the inhibitory effects have been taken both explicitly and implicitly (review, Hsu and Waltman, 2004). Based on the studies conducted previously, it is fair to say that there is no universally accepted formulation of the functional form for describing the allelopathic effect of one algae on another (Solé et al., 2005; Hammer and Pitchford, 2006). The complexity of allelopathic effect is generally studied using some nonlinear function, either directly affecting the growth rate or generating an extra mortality to the target species (e.g., Grover, 1997; Hsu and Waltman, 2004; Hammer and Pitchford, 2006). Maynard-Smith (1974) has proposed theoretically an implicit representation of the allelopathic effect by introducing a nonlinear term in a two species competition model. The non-linear allelopathic term is proportional to the product of the concentration of the toxic species and the square of the concentration of the target species (Maynard-Smith, 1974). However, only very few studies (e.g., Uchida et al., 1999) have directly implemented the theoretical representations of plankton allelopathy to either an experimental or a field data. The experimental data of several phytoplankton species conducted by Schmidt and Hansen (2001) has been used by Solé et al. (2005) to validate the allelopathic function proposed by Maynard-Smith (1974). In general this function gives a reasonable qualitative agreement with the experimental data, expect for low concentration of toxic species during the pre-proliferation states (Solé et al., 2005). This limitation of the Maynard-Smith (1974) function has been overcome by introducing a quadratic term for the concentration of both toxic and non-toxic species (Solé et al., 2005). Following these studies, for developing the present model I employ an implicit allelopathic effect (such as Hammer and Pitchford, 2006), and describe it by the modified Maynard-Smith (1974) function developed by Solé et al. (2005).

Therefore, the time evolution of the biomass of a non-toxic and a toxic phytoplankton competing for a single nutrient can be represented as follows:

$$\frac{dN}{dt} = \text{net nutrient input} - \text{uptake by species 1}$$
$$\quad \quad = \text{uptake by species 2}$$
$$\quad \quad = d \left( N_0 - N \right) - \frac{\mu_1 P_1 N}{K_1 + N} - \frac{\mu_2 P_2 N}{K_2 + N}$$
$$\equiv \phi_0 \left( N(t), P_1(t), P_2(t) \right).$$

(1)

$$\frac{dP_1}{dt} = \text{growth} - \text{loss} - \text{loss due to toxin-allelopathy},$$
$$\quad \quad = \frac{\mu_1 P_1 N}{K_1 + N} - m_1 P_1 - 2 \gamma P_2^2$$
$$\equiv \phi_1 \left( N(t), P_1(t), P_2(t) \right).$$

(2)

$$\frac{dP_2}{dt} = \text{growth} - \text{loss},$$
$$\quad \quad = - \frac{\mu_2 P_2 N}{K_2 + N} - m_2 P_2$$
$$\equiv \phi_2 \left( N(t), P_1(t), P_2(t) \right).$$

(3)

A description of the model parameters with their units and a possible range of magnitudes considered for the analysis is given in Table 1.
Table 1
Parameters, their meanings and ranges considered. Species 1 is a non-allelopathic species, and species 2 is an allelopathic species. The default values of the parameter are fixed from the ranges of the respective parameters reported in the following literature: Edward (1997), Scheffer et al. (1997) and Huisman et al. (2006).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Meaning</th>
<th>Unit</th>
<th>Default values</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\mu_1$</td>
<td>Maximum growth rate of species 1 ($P_1$)</td>
<td>Day$^{-1}$</td>
<td>1.0</td>
</tr>
<tr>
<td>$\mu_2$</td>
<td>Maximum growth rate of species 2 ($P_2$)</td>
<td>Day$^{-1}$</td>
<td>1.1</td>
</tr>
<tr>
<td>$K_1$</td>
<td>Half-saturation constant for species 1</td>
<td>$gl^{-1}$</td>
<td>0.6</td>
</tr>
<tr>
<td>$K_2$</td>
<td>Half-saturation constant for species 2</td>
<td>$gl^{-1}$</td>
<td>1.5</td>
</tr>
<tr>
<td>$m_1$</td>
<td>Loss term for species 1</td>
<td>Day$^{-1}$</td>
<td>0.012</td>
</tr>
<tr>
<td>$m_2$</td>
<td>Loss term for species 1</td>
<td>Day$^{-1}$</td>
<td>0.01</td>
</tr>
<tr>
<td>$d$</td>
<td>Rate of nutrient delivery</td>
<td>Day$^{-1}$</td>
<td>0.25</td>
</tr>
<tr>
<td>$N_0$</td>
<td>Input nutrient concentration</td>
<td>$gl^{-1}$</td>
<td>0.11</td>
</tr>
<tr>
<td>$\gamma$</td>
<td>Allelopathy parameter</td>
<td>Cell$^{-1}$day$^{-1}$</td>
<td>0.02</td>
</tr>
<tr>
<td>$\alpha_1$</td>
<td>Nutrient content of a $P_1$ cell</td>
<td>$gcell^{-1}$</td>
<td>$5 \times 10^{-6}$</td>
</tr>
<tr>
<td>$\alpha_2$</td>
<td>Nutrient content of a $P_2$ cell</td>
<td>$gcell^{-1}$</td>
<td>$1 \times 10^{-6}$</td>
</tr>
<tr>
<td>$\eta$</td>
<td>Recycling efficiency</td>
<td>Dimensionless</td>
<td>0.5</td>
</tr>
</tbody>
</table>

2.1. Analysis and results

Due to the competitive exclusion principle (Hardin, 1960), the above model in the absence of allelopathy ($\gamma = 0$) supports the persistence of only one species. The question as to which species will win in the competition, when $\gamma = 0$, depends on the half-saturation constants $K_1$ and $K_2$. For example, if $K_1 > K_2$ species 2 wins, and if $K_2 > K_1$ species 1 wins. When $\gamma \neq 0$, by imposing a non-linear mortality to species 1, the toxin-allelopathy favors species 2 in nutrient competition. So, it is reasonable for the analysis to assume that, in the absence of an allelopathic effect (i.e., when $\gamma = 0$) the toxin-producing alga $P_2$ will lose in nutrient competition to toxin-sensitive species $P_1$, which is assured if $K_2 > K_1$.

Now, for $\gamma \neq 0$, the model system has the following equilibria: (i) the species-free equilibrium $E_0(N_0, 0, 0)$, (ii) non-allelopathic monospecies equilibria $E_1(N_1, P_1, 0)$, where $N_1 = (m_1 K_1)/(\mu_1 - m_1)$, $P_1 = (d/N_1 \mu_1)(N_0 - N_1)(K_1 + N_1)$; (iii) toxic monospecies equilibrium $E_2(N_2, 0, P_2)$, where $N_2 = (m_2 K_2)/(\mu_2 - m_2)$, $P_2 = (d/N_2 \mu_2)(N_0 - N_2)(K_2 + N_2)$; and (iv) the multi-species or interior equilibrium $E^*(N^*, P^*_1, P^*_2)$. The interior equilibrium $E^*(N^*, P^*_1, P^*_2)$ is given by the following Eqs. (4)–(6),

$$N^* = (m_2 K_2)/(\mu_2 - m_2),$$

$$\gamma P_1'(P_1^*)^2 = A,$$

$$C_1 P_1^* + C_2 P_2^* = C_3,$$

with,

$$A = [K_2 m_2(\mu_1 - m_1) + K_1 m_1(\mu_2 - m_2)]/[K_1 (\mu_2 - m_2) + m_2 K_2],$$

$$C_1 = (\mu_1 m_1 K_2)/(K_1 (\mu_2 - m_2) + m_2 K_2),$$

$$C_2 = (\mu_2 N^*)/(K_2 + N^*) = m_2,$$

$$C_3 = d [N_0 - (m_2 K_2)/(\mu_2 - m_2)].$$

The trivial equilibrium $E_0$ is unstable if either $N_0 > N_1$ or $N_0 > N_2$. The boundary equilibrium $E_1$ exists if (i) $\mu_1 > m_1$, and (ii) $N_0 > N_1$, and is unstable if $N_1 > N_2$. Similarly, the other boundary equilibrium $E_2$ exists if (i) $\mu_2 > m_2$, and (ii) $N_0 > N_2$, and is unstable if $N_2 > N_1$.

The multi-species (coexisting) equilibrium $E^*$ exists if along with the following conditions,

$$\mu_2 > m_2,$$

$$N_2 > N_1,$$

the values of $P_1^*$ and $P_2^*$ satisfy the Eqs. (5) and (6). Clearly, when the interior equilibrium $E_2$ is unstable and $E_1$ is stable. Given that the parameters of the model satisfy the conditions (11) and (12), the existence of one or more positive interior equilibrium/equilibria depends on the value of the allelopathy parameter $\gamma$. Depending on the magnitude of $\gamma$, the curve (5) in the $P_1$–$P_2$ plane may or may not meet the straight line (6) (Fig. 1). When (5) and (6) do not meet, there is no coexisting equilibrium. The trajectories in this case always move towards one of the boundary equilibria thereby making coexistence impossible.

On the other hand, for a given critical value of $\gamma$ ($\gamma^*$, say) the straight line (6) touches the curve (5) (Fig. 1), and thus in the $N$–$P_1$–$P_2$ plane a unique multi-species equilibrium exists. The critical magnitude $\gamma^*$ calculated using the Eqs. (5) and (6) is given by the following equation (see Appendix for details),

$$\gamma^* = \frac{27}{4} \frac{C_1 C_2^2}{C_3} = A.$$

The unique point in the $P_1$–$P_2$ plane corresponding to $\gamma^*$, at which the straight line (6) touches (i.e., becomes a tangent on) the curve (5) is given by $\left( \frac{C_1}{3C_3}, \frac{2C_2}{3C_3} \right)$, and the corresponding unique coexisting equilibrium in given by $E^* \equiv \left( N^*, \frac{C_1}{3C_3}, \frac{C_2}{3C_3} \right)$.

However, for the allelopathy parameter $\gamma > \gamma^*$, the straight line (6) cuts the curve (5) in two points (Fig. 1), say, $P_1'$, $P_2'$ and $(P_1'', P_2'')$, and accordingly in the phase space two positive interior
equilibria, namely, $E_1^* \equiv (N^*, P_1^*, P_2^*)$ and $E_2^* \equiv (N^*, P_1^*, P_2^*)$ emerge. It can be proved by local stability analysis that, one of the equilibria ($E_1^*$) is locally stable, whereas the other one ($E_2^*$) is an unstable saddle (see, Appendix for details). In other words, at the critical magnitude of $\gamma = \gamma^c$ the model system undergoes a saddle-node bifurcation, where the unstable and the stable steady states collapse (Fig. 2).

2.2. Mapping the zone of coexistence

When the magnitude of $\gamma$ exceeds the critical value $\gamma^c$, in the $N-P_1-P_2$ phase space two steady states emerge—one of which is locally stable and the other an unstable saddle. As mentioned before, in this condition the boundary equilibrium $E_1$ is unstable and $E_1$ is stable. Thus, depending on the starting point, the model trajectories may or may not reach the attracting steady state. One way to visualize this situation is to map the entire starting points into the $P_1-P_2$ plane (Fig. 3). Starting from all possible initial combinations, when the asymptotic dynamics are mapped into the phase space, they divide the $P_1-P_2$ plane by a separatrix into two non-overlapping regions (Fig. 3, the shaded and non-shaded region divided the separatrix). If an initial combination of $(P_1, P_2)$ belongs to the region in which $(P_1^*, P_2)$ lies (the shaded region in Fig. 3), the time evolution of the trajectories move towards the stable equilibrium $E_1^* \equiv (N^*, P_1^*, P_2^*)$, and since the boundary equilibrium $E_2$ is unstable, this leads to a stable coexistence of the two phytoplankton species (Fig. 4(a)). On the other hand, if an initial combination of $(P_1, P_2)$ belongs to the region in which $(P_1^*, P_2^*)$ lies (as it's a saddle point), and since the boundary equilibrium $E_1$ is stable, this leads to the extinction of $P_2$ (Fig. 4(b)). The switching between two stable equilibrium states (namely, a stable coexistence and a stable persistence of species 1 alone), depending on the initial conditions, may be considered a further example of the well known ecological phenomenon of ‘alternative stable states’ (e.g., May, 1977; Tilman, 1982). Thus, under the influence of allelopathy coupled with a suitable initial condition, two phytoplankton species exhibit a stable coexistence on a single resource, thereby coevolves over a long time scale in almost non-fluctuating biomass (Figs. 3 and 4).

It is worth reiterating that the criteria (11) and (12), derived for the existence of an interior equilibrium, immediately assure that the toxic mono-species equilibrium ($E_2$) is unstable and non-toxic mono-species equilibrium ($E_1$) is locally stable. This is the reason why the non-coexistence region mapped (in Fig. 3) represents the elimination of the toxic species only. However, the resource competition theory (Tilman, 1982) suggests that species commonly limited by a single resource obey the so-called $R^*$ (the resource level at which a species is just able to persist) rule, and the species with the lowest $R^*$ value is the winner in competition (Tilman, 1982; Chesson, 2000). The resource levels $N_1$ and $N_2$ derived here represent Tilman's $R^*$ values of species 1 and species 2 respectively, and the criterion given in (12) suggests that species 1 is competitively superior in this case. This is the biological reason for the elimination of toxic species (species 2) below the separatrix in Fig. 3.

These analyses suggest that a suitable intensity of toxin-allelopathy has the potential to ensure the stable coexistence of more species than the number of limiting resources. Further, a suitable initial combination of the competing species is an important criterion for predicting the long-term behavior of the species—a similar conclusion was drawn on filamentous cyanobacteria by Scheffer et al. (1997). Thus, the coevolution of phytoplankton species in a homogeneous media might be attributed to the regulation by allelopathic effect and the dependence on life-history.

3. Effect of nutrient recycling

The model of the previous Section lacks the effect of recycling of the limiting nutrient. The recycled nutrient contribute to the pool of growth-limiting nutrient. The model can easily be modified to incorporate the effect of nutrient recycled from a phytoplankton cell on its death. The mortality of phytoplankton $P_1$ is a combination of its natural loss and the loss due to the effect of allelopathy. On the other hand, the mortality of phytoplankton $P_2$ is only due to its natural loss. The extra mortality caused by allelopathy produces an additional amount of nutrient recycled from the non-toxic species. By definition the amount of this nutrient recycled is dependent on the rate at which the non-toxic species is killed. For simplicity, it is assumed that the efficiency of nutrient recycled from a dead phytoplankton cell is constant over time, say, $\eta > 0$. On including nutrient recycling, the model system (1)–(3) is extended as follows:
Time evolution of the two phytoplankton under a single resource. For an initial combination of \((P_1, P_2)\) lying in the shaded region of Fig. 3, a stable coexistence of \(P_1\) and \(P_2\) under \(N\) in almost non-fluctuating biomass. For an initial combination of \((P_1, P_2)\) lying in the non-shaded region of Fig. 3, \(P_2\) goes to extinction making the coexistence impossible in the long run.

\[
\frac{dN}{dt} = \text{net nutrient input} - \text{uptake by } P_1 \\
= d \left( N_0 - N \right) - \frac{\mu_1 \alpha_1 P_1 N}{K_1 + N} - \frac{\mu_2 \alpha_2 P_2 N}{K_2 + N} + \eta \alpha_1 \left( m_1 P_1 + \gamma P_1^2 P_2^2 \right) + \eta \alpha_2 m_2 P_2 \\
= \phi_0 \left( N(t), P_1(t), P_2(t) \right),
\]

(14)

\[
\frac{dP_1}{dt} = \text{growth} - \text{loss} - \text{loss due to allelopathy}, \\
= \frac{\mu_1 P_1 N}{K_1 + N} - m_1 P_1 - \gamma P_1^2 P_2^2 \\
= \phi_1 \left( N(t), P_1(t), P_2(t) \right),
\]

(15)

\[
\frac{dP_2}{dt} = \text{growth} - \text{loss} \\
= \frac{\mu_2 P_2 N}{K_2 + N} - m_2 P_2 \\
= \phi_2 \left( N(t), P_1(t), P_2(t) \right)
\]

(16)

where \(\alpha_1\) and \(\alpha_2\) are the nutrient contents of a single cell of \(P_1\) and \(P_2\), respectively.

Adopting an approach similar to the previous Section, it can be proved that the dynamics of the system around the steady states are qualitatively similar to those without nutrient recycling (i.e., \(\eta = 0\)). In the phase space there is either a single coexisting equilibrium or two equilibria depending on whether or not the magnitude of the allelopathy parameter \(\gamma\) equals or exceeds a modified critical value \(\gamma^*\) given by the following equation,

\[
\gamma^* = \frac{27 \bar{C}_1 \bar{C}_2^2}{4 C_3^3} - \bar{A},
\]

(17)

where,

\[
\bar{C}_1 = \alpha_1 \left[ C_1 - (m_1 + A) \eta \right]
\]

(18)

\[
\bar{C}_2 = \alpha_2 \left[ C_2 - m_2 \eta \right],
\]

(19)

and the values of \(C_1\), \(C_2\), \(C_3\) and \(A\) are given in Eqs. (7)–(10). The unique equilibrium corresponding to \(\gamma^*\) is given by \([N^*, (C_1^*), (C_2^*), (C_3^*)]\), where the magnitude of \(N^*\) is the same as Eq. (4). Similar to the previous Section, for \(\gamma > \gamma^*\), two interior equilibria exists one of which is locally stable and the other is a saddle, and the time evolution of the trajectories in the phase space depends on the initial condition. Thus, the incorporation of the effect of nutrient recycling in the model system modifies only the critical values of the parameter bounds, however, the qualitative dynamics remain unaffected. A description on how the critical magnitude of allelopathy varies due to different half-saturation constants and the recycling efficiency is given in the following.

### 3.1. Critical value of allelopathy as a function of the half-saturation of uptake and the recycling efficiency

Eq. (17) suggests that different efficiencies (\(\eta\)) of nutrient recycling change the critical magnitude of the allelopathy parameter. The critical value of \(\gamma\) is a decreasing function of the recycling efficiency (Fig. 5). Further, corresponding to a feasible value of \(\eta\) a range of values of \(\gamma\) is always obtainable for which one of the two coexisting equilibria is locally stable (Fig. 5). Thus, when the effect of nutrient recycling of the dead phytoplankton cells (with an efficiency \(\eta\)) is considered, a suitable magnitude of the allelopathy parameter can ensure the stable coexistence of two phytoplankton under a single resource.

As mentioned before, the competitive ability of the two phytoplankton in the absence allelopathic effect is determined by the half-saturation constants \(K_1\) and \(K_2\), and in the present set up \(K_2 > K_1\). When expressed as a function of \(K_2\) and the recycling efficiency \(\eta\), the critical allelopathy \(\gamma^*\) has a higher magnitude if \(K_2\) increases and \(\eta\) decreases (Fig. 6). So, for a realistic combination
of $K_2$ and $\eta$, a feasible magnitude of $\gamma^c$ is obtainable for ensuring the stable coexistence.

These analyses suggest that the competitive advantage a toxin-producing species gain due to allelopathy is significant for its survival. The release of allelopathic chemicals may be a potential adaptive strategy for a phytoplankton species weak in resource competition under a nutrient deficient condition. Under a resource-competition scenario, toxin-allelopathic effect in a homogeneous environment is a significant factor for ensuring the coexistence of multiple species on a single resource.

4. Discussion

One of the important demonstrations of this study is the stable coexistence of two phytoplankton under a single resource without the influence of any top-down effect and environmental factors. The novel mechanism that leads to such situation is the critical effect of allelopathy due to one of the species that releases toxic chemicals. The model used here is developed based on a standard chemostat model of resource competition, where allelopathic effect is introduced as a phenomenological, density-dependent mortality term. In other words, the model is mechanistic in its description of exploitative competition, but phenomenological in its description of other density-dependent mortality due to allelopathy. Theoretically this nonlinear function, developed from experimental data by Solé et al. (2005), represents a density-dependent mortality for the non-allelopathic species. It is noteworthy that in a different context the phenomenological terms have been previously added to a chemostat model (Amarasekere, 2002). However, the difference in the present model is that the density-dependent term is not a standard Lotka–Volterra term, which is proportional to the product of the two competitors’ densities, but is instead a fourth-order term proportional to the squares of their densities. This difference in model formulation from that of Amarasekere (2002) enables the model to focus specifically to the resource competition scenario under an implicit effect of allelopathy (such as Hammer and Pitchford, 2006).

Under suitable parametric conditions derived, the mortality-function due to allelopathy is capable of breaking the symmetry of the simple resource-competition model, which otherwise supports the persistence of only one (strong) competitor. A critical lower bound of allelopathy has been derived as a function of the parameters of the conventional resource-competition model — which ensures a coexisting equilibrium. Thus, in a nutrient-phytoplankton interaction the allelopathy of a toxic species with suitable intensity appears to be a potential stabilizing factor that boosts the coevolution of two phytoplankton on a single limiting nutrient. In other words, the allelopathy due to toxic species successfully acts as a potential self-limiting factor for the stable coexistence of the phytoplankton species in homogeneous media.

A number of studies conducted previously has established that the mixotrophic interactions in microbial population play a crucial role in species succession (e.g., Davidson, 1996; Jost et al., 2004; Hammer and Pitchford, 2006). For example, in course of nutrient competition, the individuals of mixotrophic protists are known to graze on their fellow individuals thereby modulating the trophic dynamics (Davidson, 1996). Similarly, when the autotroph and mixotroph compete for shared nutrient, the mixotrophic grazing on the autotroph reduces the pressure of competition (Jost et al., 2004). A recent laboratory experiment suggests that mixotrophy acts as a nutrient supplement contributing to the persistence of Prymnesium parvum (Hammer and Grover, 2008). Thus, during a competitive interaction the mixotrophy gives a beneficial effect to the growth of the mixotrophic species. Although the biology of mixotrophy and allelopathy apparently looks different, there is an intriguing similarity between the ultimate effects of these two mechanisms.

The mode of functioning of allelopathy due to a toxin-liberating phytoplankton, although not directly grazing, is meant to kill the cells of other phytoplankton (which has been modeled here by an established negative non-linear function). In the simple resource competition scenario (model (1)–(3)), killing one’s competitor increases the availability of nutrient from the removal of another consumer, allowing the nutrient concentration to rise marginally. But, when the effect of recycling is incorporated (model (14)–(16)), the scenario changes significantly. The recycling process makes some of the dead competitor’s nutrient quota available for uptake. The recycling of the competitors killed by allelopathy generates an extra amount of the nutrient available for uptake. By this process allelopathic killing provides a positive feedback for the increase of the growth limiting resource, thereby causes for the reduction of the competition pressure. Through this feedback loop the allelopathic effect gives crucial benefit to the
growth rate of the algae. The analysis here demonstrates that such functioning potentially regulates/modulates the dynamics of the competing species of a common trophic level. Responsible for a density-dependent mortality of the competitor causing a pivotal benefit to the growth process through an indirect feed-back loop, the action allelopathy of toxic species can be considered as a ‘pseudo-mixotrophy’. Its effect in the two-species interaction, as demonstrated here, suggests that this pseudo-mixotrophy in the form of allelopathy among the phytoplankton promotes species succession though a stable coexistence, and thus contributes in maintaining the species diversity.

The stabilizing mechanism demonstrated for a two-species — one-resource system in the presence of allelopathy as pseudo-mixotrophy raises an important question as to how much diversity in the natural water is ensured under this influence alone. Some kind of relationship between allelopathy and diversity has become a popular hypothesis in microbial ecology (Lenski and Riley, 2002). The results of recent studies have shown that the chemical warfare in general increases bio-diversity in microbial realm (reviewed by Lenski and Riley, 2002). The present study is relevant in this context. The result of the analysis with two species may be extended to many species case. One of the related studies (Roy, 2008) recently reports a deterministic relationship between the abundance of the potential toxic-producing species and the species diversity of the nontoxic phytoplankton in a natural water. This study suggests that the abundance-diversity relationship actually represent a unimodal pathway through which the abundance of toxic species regulates the diversity of phytoplankton. However, further theoretical and experimental studies would give more information about the role of pseudo-mixotrophy in species succession and on the dynamics of microbial food webs.

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Appendix

A.1. Calculation of \( \gamma^C \)

For the interior equilibrium \( E^N(P^*, P^*_2) \), the steady value of nutrient \( (N^*) \) is determined explicitly by Eq. (4), and \( P^*_1 \) and \( P^*_2 \) are the solution of following equations in \( P_1 \) and \( P_2 \)

\[
\gamma P_1, P^*_2 = 0, \quad C_1 P_1 + C_2 P_2 = C_3. \tag{20}
\]

In the \( P_1-P_2 \) plane, the slope of the straight line \( (21) = (-\frac{C_1}{C_2}) \), where the values of \( C_1 \) and \( C_2 \) are given by Eqs. (8) and (9). The slope of the curve (20) at any point \((P_1, P_2)\) in the \( P_1-P_2 \) plane is \( (-\frac{C_1}{C_2}) \).

At the critical point \((P^*_1, P^*_2)\) in the \( P_1-P_2 \) plane, where the straight line \( 20 \) is a tangent to the curve \( 21 \), these two slopes are equal in magnitude, i.e., \( -\frac{C_1}{C_2} = -\frac{C_1}{C_2} \). This gives, \( P^*_2 = \frac{2C_1}{C_2} P^*_1 \). Using this relation in Eq. (20), it is found that \( P^*_1 = \frac{C_2}{3C_1} \), and thus \( P^*_2 = \frac{2C_1}{3C_2} \).

The values of \( P^*_1 \) and \( P^*_2 \) satisfy the Eq. (20) for a critical values of \( \gamma \) given by \( \gamma^C = \frac{22C_1 C_2^2}{4C_1^3} \), with \( A \) given in (7). A similar calculation follows for the model system (14)–(16).

A.2. Local stability of equilibria

The variational matrix of the model system (1)–(3) at an equilibrium \((N, P_1, P_2)\)

\[
\begin{pmatrix}
-d - \frac{\mu_2 P_1 K_1}{K_1 + N} - \frac{\mu_2 P_2 K_2}{K_2 + N} & \frac{\mu_2 P_1 K_1}{(K_1 + N)^2} \frac{\mu_2 P_2 K_2}{(K_2 + N)^2} & - \frac{\mu_2 K_1}{(K_1 + N)^2} - \frac{\mu_2 K_2}{(K_2 + N)^2} \\
\frac{\mu_1 K_1 P_1}{K_1 + N} & - \frac{N P_1}{(K_1 + N)^2} - m_1 - 2 \gamma P_1 P_2 \gamma & - \frac{N P_2}{(K_2 + N)^2} - m_2 \\
0 & -2 \gamma P_1 P_2 \gamma & - \frac{\mu_2 K_2}{(K_2 + N)^2} - m_2
\end{pmatrix}
\]

The stability of an equilibrium is determined by the sign of the real part of eigenvalues of the above variational matrix calculated at that equilibrium. Following the notations of the equilibria used in the text, the eigenvalues of \( E \) at \( E_0(N, 0, 0) \) are found to be \((-d, \gamma, \mu_2)\). Thus at least one eigenvalue is positive and therefore \( E_0 \) is an unstable saddle if either \( N_0 > N_1 \) or \( N_0 > N_2 \), where \( N_1 = \frac{\mu_1}{\mu_2 - m_1} \) and \( N_2 = \frac{\mu_2}{\mu_1 - m_2} \) given in the text. Now, corresponding to the boundary equilibria \( E_1(N_1, 0, 0) \) one eigenvalue of \( E \) always is \((-\gamma, \gamma, \mu_2) \). Thus this eigenvalue is positive and therefore \( E_1 \) is an unstable saddle if \( N_1 > N_2 \). Similarly, corresponding to the boundary equilibria \( E_2(N_2, 0, P_2) \) one eigenvalue of \( E \) always is \((-\gamma, \gamma, \mu_2) \); thus this eigenvalue is positive and therefore \( E_2 \) is unstable if \( N_2 > N_1 \).

Under suitable conditions explained in the text, two interior equilibria exist; and corresponding to the parameter set in Table 1, these two interior equilibria are given by \( E_1(0.0138, 0.1122870863, 2.154198535) \) and \( E_2(0.0138, 0.6836977721, 0.8730086566) \). The eigenvalues of the variational matrix \( (22) \) calculated numerically at \( E_1(0.0138, 0.1122870863, 2.154198535) \) are given by \((-1.970070893, -0.01655272987, -0.03798471086) \); all three eigenvalues are negative thus \( E_1(0.0138, 0.1122870863, 2.154198535) \) is locally asymptotically stable. On the other hand, the eigenvalues calculated at \( E_2(0.0138, 0.6836977721, 0.8730086566) \) are given by \((-1.951730969, -0.0197471039, 0.2890362252) \); two eigenvalues are negative but the third one is positive, thus \( E_2(0.0138, 0.1122870863, 2.154198535) \) is an unstable saddle. A similar calculation follows for the model system (14)–(16).

References