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Allelopathic interactions between phytoplankton species: The roles of heterotrophic bacteria and mixing intensity

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ABSTRACT

Toxin-producing phytoplankton species may compensate for competitive disadvantages by secreting chemicals that affect toxin-sensitive phytoplankton species. Heterotrophic bacteria, however, may, in turn, degrade the toxins produced by allelopathic phytoplankton, thus confounding allelopathic interactions between phytoplankton species. Moreover, recent theoretical studies suggest that incomplete mixing of the water column might also modify allelopathic interactions. Here, we analyse a model where phytoplankton species, bacteria, nutrients, and a toxin are linked through material cycling. The model considers a toxin-producing and a toxin-sensitive phytoplankton species and two species of heterotrophic bacteria. The model is analysed for two scenarios: a simple well-mixed aquatic ecosystem is contrasted with an aquatic ecosystem with low mixing intensity. The results show that (1) the winner of competition between toxin-producing phytoplankton and toxin-sensitive phytoplankton species may depend on the species that becomes dominant first, (2) heterotrophic bacteria able to degrade allelopathic toxins will facilitate dominance of toxin-sensitive phytoplankton species, (3) heterotrophic bacteria unable to degrade allelopathic toxins may to some extent counter the facilitating effect of toxin-decomposing bacteria, owing to competition between the bacterial species, and (4) there is a major effect of mixing intensity on these species interactions: when turbulent mixing rates are low, toxin concentrations are less diluted whereas degradation activities of heterotrophic bacteria are more localised. As a result, this model study predicts that weak mixing, especially when combined with the presence of bacteria unable to degrade allelopathic compounds, will favour the development of allelopathic phytoplankton populations.

INTRODUCTION

There is growing awareness that allelopathic interactions between populations might have important consequences for ecosystem functioning. Allelopathy is the release of chemical substances by individuals of a population that have an effect on the individuals of another population. Allelopathic interactions have been reported in various contexts: between bacteria (Chao and Levin 1981), between bacteria and phytoplankton (Cole 1982; Bloor and England 1989; Imai et al. 1995; Imai et al. 2001; Kitaguchi et al. 2001), between phytoplankton and zooplankton (Turner and Tester 1997) and also between calanoid copepods (Folt and Goldman 1981). In particular, allelopathic interactions are commonly observed within phytoplankton communities (Maestrini and Bonin 1981; Mason et al. 1982; Von Elert and Jüttner 1997; Johansson and Granéli 1999; Rengefors and Legrand 2001). Several phytoplankton species produce compounds that inhibit the photosynthetic electron transport of their competitors (Gleason and Paulson 1984; Gleason and Baxa 1986; Gross et al. 1991; Smith and Doan 1999). Other phytoplankton species may inhibit carbon fixation of competing phytoplankton species (Sukenik et al. 2002). There is also an example of a cyanobacterium producing a toxin that paralyzes the motile green alga Chlamydomonas, which leads to fast sedimentation of Chlamydomonas (Kearns and Hunter 2001). As a consequence, allelopathy might have major impacts triggering phytoplankton succession (Lefèvre et al. 1950; Keating 1977; Inderjit and Dakshini 1994).

Recently, Durrett and Levin (1997) developed a model to analyse the dynamics of bacteria producing allelopathic substances. Their model showed that the outcome of competition between a toxin-producing strain and a toxin-sensitive strain depends on mixing intensity. A toxin-producing strain starting with a low initial abundance does not become dominant under homogeneous mixing while it might invade from low densities under weak mixing. In other words, according to the Durrett-Levin model, toxin-producing species can

invade more easily under incomplete mixing than in well-mixed systems. Competition experiments between toxin-producing and toxin-sensitive strains of the bacterium Escherichia coli grown under different mixing regimes support these model predictions (Chao and Levin 1981; Kerr et al. 2002). In addition to the effects of mixing intensity on allelopathic interactions, mixing processes have also a major impact on phytoplankton bloom development (Riley et al. 1949; Koseff et al. 1993; Huisman et al. 1999a; O'Brien et al. 2003) and on the species composition of phytoplankton blooms (Visser et al. 1996; Sherman et al. 1998; Huisman et al. 1999b). A consideration of mixing intensity seems therefore highly relevant for studies on allelopathic interactions in phytoplankton communities.

Heterotrophic bacteria might interfere in allelopathic interactions between phytoplankton species. On the one hand, heterotrophic bacteria may degrade harmful substances and in this way counteract the development of allelopathic phytoplankton populations. On the other hand, some experiments suggest that bacteria living in association with toxin-producing phytoplankton species can produce harmful compounds themselves (Gallacher et al. 1997; Gallacher and Smith 1999; Hold et al. 2001). These bacteria would act in synergy with allelopathic phytoplankton. Therefore, the intriguing role of heterotrophic bacteria in allelopathic interactions between phytoplankton species deserves attention.

Here we develop a model to study the potential impact of heterotrophic bacteria and mixing intensity on allelopathic interactions between phytoplankton species. The model includes a nutrient pool, a toxin-producing phytoplankton species, a toxin-sensitive phytoplankton species, a detritus pool, a toxin pool and two heterotrophic bacteria. One of the bacterial species is able to break down the toxin, whereas the other is not. The role of spatial mixing is analysed by developing two contrasting versions of the model, one version for a well-mixed system and the other version for a system that is not well mixed. We specifically address two questions: (1) To what extent do heterotrophic bacteria capable of degrading

allelopathic compounds influence allelopathic interactions between phytoplankton species? (2) Does mixing intensity influence allelopathic interactions in aquatic ecosystems?

THE MODEL

The model is based on a minimal ecosystem, with primary producers and decomposers. It describes the cycle of a nutrient (Loreau 1998). The model (Fig. 1) consists of a nutrient pool N, two phytoplankton species P_i , a detritus pool D , a toxin pool T and two bacterial species B_k. The two phytoplankton species exploit the nutrient pool and produce detritus. A fraction γ of detritus produced by the toxin-producing phytoplankton species (P_T) is a toxic compound (T) harmful to the toxin-sensitive phytoplankton species (P_S). We refer hereafter to γ as the poisoning rate. We hypothesise a cost to toxin production reflected in a lower competitive ability for nutrients of the toxin-producing phytoplankton species. The harmful effect of the toxin on the toxin-sensitive phytoplankton species is captured by an additional mortality of the toxin-sensitive species proportional to the toxin abundance. In contrast to Durrett and Levin's model (1997) here the toxin dynamics is explicitly described. Detritus is degraded by the two bacterial species. However, only one of these bacteria is able to degrade the toxin. We refer to this bacterial species as the generalist bacterium (B_G) because it is able to degrade both detritus and the toxin. Its niche breadth is broader than that of the other bacterial species, which only degrades detritus and thus will be called the specialist bacterium (B_S). We assume a trade-off between the ability to degrade detritus and the ability to degrade the toxin, which implies that the generalist bacterium has a lower competitive ability for detritus than the specialist bacterium. Owing to bacterial degradation, detritus and the toxin are recycled into inorganic nutrient. The nutrient cycle is open for external nutrient inputs and for nutrient exports from all compartments.

Inspired by the findings of Durrett and Levin (1997), two contrasting versions of the model are considered. We use a system of ordinary differential equations (ODE) to mimic a well-mixed aquatic ecosystem. We use cellular automata to mimic an aquatic ecosystem that is not well mixed.

Complete mixing - the ODE model

The well-mixed version of the model is represented by a system of ordinary differential equations, as follows:

$$\frac{dN}{dt} = I - qN - \sum_i f_{P_i}(N)P_i + \sum_k \chi\rho B_k \tag{1}$$

$$\frac{dP_T}{dt} = \beta_{N,P_T} f_{P_T}(N)P_T - \rho P_T \tag{2}$$

$$\frac{dP_S}{dt} = \beta_{N,P_S} f_{P_S}(N)P_S - \rho P_S - TP_S \tag{3}$$

$$\frac{dD}{dt} = \chi(1-\gamma)\rho P_T + \chi(\rho P_S + TP_S) - \rho D - \sum_k f_{B_k}(D,T)B_k \tag{4}$$

$$\frac{dT}{dt} = \chi\gamma\,\rho P_T - \rho T - f_{B_G}(D,T)B_G \tag{5}$$

$$\frac{dB_S}{dt} = \beta_{D,B_S} f_{B_S}(D)B_S - \rho B_S \tag{6}$$

$$\frac{dB_G}{dt} = \beta_{D,B_G} f_{B_G}(D,T)B_G - \rho B_G \tag{7}$$

Here, I is the constant external input of inorganic nutrient and q the leaching rate of inorganic nutrient, $f_X(Y)$ is the functional response of species X on resource Y. We assume that the functional responses of the two phytoplankton species follow a Monod equation:

$$f_X(N) = \frac{\mu_X N}{K_{NX} + N} \tag{8}$$

where μ_X is the maximum specific growth rate of species X, and K_{NX} is the half-saturation constant of species *X* for nutrient *N*. The functional response of the two bacterial species, $f_{B_k}(D,T)$, is the multi-resource Monod response (O'Neill et al. 1989; Lendenmann and Egli 1998):

$$f_{B_k}(D,T) = \frac{\mu_X (b_{DX} D + b_{TX} T)}{1 + b_{DX} D + b_{TX} T} \tag{9}$$

where μ_X is the maximum specific growth of species X, b_{DX} is the inverse of the half-saturation constant of detritus uptake by species X, and b_{TX} is the inverse of the half-saturation constant of toxin uptake by species X. We assume that the toxin is taken up only by the generalist bacterium, it is not consumed by the specialist bacterium (i.e., b_{TBS} =0). Furthermore, $\beta_{Y,X}$ is the conversion efficiency of resource *Y* into new individuals of *X*, ρ is the turnover rate of the system, and χ is the fraction of nutrient in dead phytoplankton and bacteria that is recycled within the ecosystem. Table 1 gives the values of our parameter set. Numerical simulations of the ODE model are based on a fourth-order Runge-Kutta procedure with a fixed time step of 0.01 day.

Incomplete mixing - The cellular automata model

Incomplete mixing is mimicked by a cellular automata model (Durrett and Levin 1997; Kerr et al. 2002). Here, space is represented by a grid of 100 x 100 cells. The spatial grid has a torus shape, which removes edge effects. The interaction neighbourhood of each grid cell is defined by the four nearest neighbour cells. In a two-dimensional space with integer coordinates, the cell (0,0) interacts with the set of cells {(1,0), (0,1), (-1, 0), (0, -1)}. Each grid cell supports abiotic elements (inorganic nutrient, detritus and toxin) at different concentrations and, for the biotic elements, at most one phytoplankton individual and one bacterial individual. These abiotic and biotic elements represent different "layers" of a grid

cell. The transition of each layer between two time steps is determined by the other layers following the ODE model defined above (Equations 1-9). The transitions are continuous for the abiotic elements and stochastic for the biotic elements. For instance, the dynamics of nutrients in a grid cell (Equations 1 and 8) are specified as follows: between each time step, there is a constant external input of nutrients *I,* a constant loss *qN* of nutrients, an uptake of nutrients $f_{P_i}(N)$ by an individual phytoplankter, if present ($P_i = 1$), and an input of nutrients $\chi\rho B_k$ due to the recycling activities of a heterotrophic bacterium, if present ($B_k = 1$). The dynamics of a toxin-producing phytoplankter (Equation 2) depends on the resource concentration of the grid cell it inhabits. The probability of birth and death (transition rates) are $\beta_{N,P_T} f_{P_T}(N)$ and ρ, respectively. A grid cell interacts with its neighbours through diffusion of abiotic and biotic elements and through birth of biotic elements. At each time step, a proportion (1-*d*) of each abiotic element stays in the cell; the diffusing proportion *d* is shared between the four neighbours. We choose to maximise the homogenisation at each time step by setting *d*=0.8. When a birth event occurs for phytoplankton or bacteria, the offspring is placed in an empty neighbour cell randomly chosen. If an individual of the same functional group (i.e. phytoplankton or bacteria) already occupies the neighbour cell, the event of birth in this cell fails and a new neighbour cell is randomly chosen. In total, four attempts to place an offspring in an empty neighbour cell are made. The diffusion of phytoplankton and bacterial individuals is captured as follows. Each individual may stay in its cell or move to one of its four neighbour cells with the same probability at each time step. If the cell is already occupied by an individual of the same functional group, the motion is cancelled. If an empty cell of the grid is supposed to receive several individuals of the same functional groups from different neighbour cells, then the new resident is randomly chosen. This lottery process allows the same diffusion of individuals irrespective of the density of their population.

Unless specified, the same parameter values were used for both the ODE model and the cellular automata model. Only the initial conditions generally differed between the two models (Table 1).

RESULTS

Complete mixing - the ODE model

Isocline analysis: The ODE model is too complex to allow a complete mathematical analysis. However, quite some understanding of the competitive interactions within a functional group can be obtained by isocline analysis. We plot the zero net growth isoclines, or ZNGIs (Tilman 1982), of competing species as functions of the factors relevant for their growth. The ZNGI of a species identifies the conditions where its birth rate equals its death rate. Above the ZNGI of a species, its death rate exceeds its birth rate and hence its net growth rate is negative. Below its ZNGI, the growth rate is positive because birth rate exceeds death rate. Intersection of the ZNGIs of two competing species defines a potential coexistence equilibrium. The relative position of the impact vectors is an indication of the stability of the equilibrium (Tilman 1982; Leibold 1996; Grover 1997).

Isocline analysis of the phytoplankton species: The ZNGIs of the two phytoplankton species are plotted as a function of the environmental concentrations of the inorganic nutrient and the toxin (Fig. 2A). Because toxin availability has no effect on the toxin-producing phytoplankton species, its ZNGI is a line parallel to the y-axis (toxin concentration) which intercepts with the x-axis (nutrient availability) at the value $N^*_{P_T}$. The growth rate of the toxin-sensitive phytoplankton species is determined by both the inorganic nutrient concentration and the toxin concentration. Therefore its ZNGI is a curve that intercepts with the x-axis (nutrient availability) at the value $N^*_{P_S}$ and increases monotonically with nutrient and toxin availability.

This curved ZNGI shows that increasing nutrient availability may compensate for an increased death rate owing to higher toxin concentrations. The two ZNGIs intersect if the toxin-sensitive species is a better competitor for nutrients than the toxin-producing species ($N^*_{P_S} < N^*_{P_T}$). In this case, there are three equilibria: a coexistence equilibrium at the intersection point, a monoculture equilibrium with the toxin-producing species only, and a monoculture equilibrium with the toxin-sensitive species only. Which equilibrium will be reached, depends on the impact vectors and the position of the supply point. The impact vector of the toxin-sensitive species has a negative horizontal component only, characterising nutrient consumption. The impact vector of the toxin-producing species has a negative horizontal component characterising nutrient consumption and a positive vertical component characterising toxin production (Fig. 2A).

The ZNGIs and the projection of the species impact vectors define four regions in the nutrient-toxin plane. If the supply point lies in the hatched region, neither species can persist. If the supply point lies in the region "Toxin-producing species wins", toxin levels are so high that the toxin-sensitive species is excluded. Hence, only the toxin-producing species persists. If the supply point lies in the region "Toxin-sensitive species wins", nutrient levels are depleted to very low levels, excluding the toxin-producing species. Hence, only the toxin-sensitive species persists. The coexistence equilibrium exists only if the supply point lies in the region "Unstable equilibrium". However, the coexistence equilibrium is indeed unstable. This is because the toxin-sensitive species has a stronger negative effect on nutrient availability than the toxin-producing species whereas the toxin-producing species has a positive effect on toxin availability. As a result, when the toxin-sensitive species becomes dominant first, it will deplete nutrient availability below the minimal nutrient requirements of the toxin-producing species. Conversely, when the toxin-producing species becomes dominant first, it will raise toxin levels above the tolerance limit of the toxin-sensitive species.

Therefore, the two species cannot coexist in equilibrium. Instead, either the toxin-sensitive species or the toxin-producing species will win. The winner depends on the initial conditions. In other words, there is an invasion threshold, such that the toxin-producing species can invade only if its initial abundance exceeds a certain threshold value.

Isocline analysis of the bacterial species: The ZNGIs of the two bacterial species are plotted as a function of the environmental concentrations of detritus and the toxin (Fig. 2B). Because the specialist bacterium does not consume toxin, its ZNGI is a line parallel to the y-axis (toxin availability) which intercepts with the x-axis (detritus availability) at the value D_{B_S}*. The generalist bacterium consumes both resources in a linear substitutable form. Hence, the ZNGI of the general bacterium is a line that intersects the x-axis and y-axis at the points D_{B_G}* and T_{B_G}*, respectively. The ZNGIs of the two bacterial species intersect only if the specialist bacteria are better competitors for detritus than the generalist bacteria. The impact vector of the specialist bacteria has a negative horizontal component only while the impact vector of the generalist bacteria has both a negative horizontal and a negative vertical component.

The isoclines and the projections of the impact vectors define four regions in the detritus-toxin plane (Fig. 2B). If the supply point lies in the hatched region, neither bacterial species can persist. If the supply point lies in the region "Generalist species wins", with a high toxin level, the generalist bacteria competitively exclude the specialist bacteria. If the supply point lies in the region "Specialist species wins", with a high detritus level, the specialist bacteria competitively exclude the generalist bacteria. Coexistence of the two bacterial species is possible only if the supply point lies in the region "Stable equilibrium". Because of the relative positions of the impact vectors, this coexistence equilibrium is indeed stable. Independent of the initial conditions, in this region the system dynamics always lead towards stable coexistence of the specialist and generalist bacteria.

A comparison of the isocline graphs of phytoplankton (Fig. 2A) and bacteria (Fig. 2B) provides some insight of the behavior of the complete system. Let T^*_{phyto} denote the toxin level at the unstable coexistence equilibrium of the phytoplankton, and let T^*_{bact} denote the toxin level at the stable coexistence equilibrium of the bacteria. It is most unlikely that T^*_{phyto} and T^*_{bact} are exactly identical. Hence, equilibrium coexistence of all four species at the same toxin level is highly unlikely. If $T^*_{bact} > T^*_{phyto}$, the equilibrium toxin concentration resulting from bacterial degradation is still high enough to be lethal for toxin-sensitive phytoplankton species. In this case, despite bacterial activity, toxin-producing phytoplankton species may exclude toxin-sensitive species. Conversely, if $T^*_{bact} < T^*_{phyto}$, bacteria are able to reduce the toxin concentration below the toxin threshold lethal for toxin-sensitive phytoplankton, and hence toxin-sensitive phytoplankton is favoured.

Simulation Results: As predicted by the isocline analysis, computer simulations show that either the toxin-sensitive phytoplankton species excludes the toxin-producing phytoplankton species (Fig. 3A), or the toxin-producing phytoplankton species excludes the toxin-sensitive phytoplankton species (Fig. 3B). To investigate this in full detail, we ran numerous simulations in which we systematically varied two parameters: the poisoning rate of the toxin-producing species and the initial abundance of the toxin-producing species (Fig. 3C). This reveals that the toxin-sensitive phytoplankton species always wins if the poisoning rate of the toxin-producing phytoplankton species is lower than a certain threshold value ($\gamma \leq 0.26$ for our parameter settings; in Fig. 3C). Below this threshold value, the toxin production of the toxin-producing species is not efficient enough to compensate for the better nutrient exploitation abilities of the toxin-sensitive species. Above this threshold value, either the toxin-sensitive species wins or the toxin-producing species wins, depending on the initial

conditions (Fig. 3C). The composition of the bacterial community depends on the phytoplankton species that becomes dominant. When the toxin-sensitive phytoplankton species wins, toxins are not produced, and hence the specialist bacteria competitively exclude the generalist bacteria (Fig. 3A). In contrast, when the toxin-producing phytoplankton species wins, the presence of the toxin allows the generalist bacteria to competitively exclude the specialist bacteria (Fig. 3B).

Examination of the transient dynamics leading to the final community composition shows that generalist bacteria have a pivotal role because they break down toxin produced by toxin-producing phytoplankton (Fig. 3A). During the first steps, the abundance of toxin-producing phytoplankton increases; so does the toxin concentration. The toxin peak is followed by a peak of generalist bacteria, which break down the toxin and thereby facilitate the toxin-sensitive phytoplankton species. Depending on the toxin levels finally reached, the toxin-sensitive phytoplankton species may or may not take over. If the toxin-sensitive species excludes the toxin-producing phytoplankton species, the generalist bacteria are excluded as well. Thus, generalist bacteria facilitate toxin-sensitive phytoplankton species, and by doing so generalist bacteria may contribute to their own demise.

What happens when generalist bacteria are absent from the system? The results are dramatically changed. The toxin-producing phytoplankton species become dominant much more easily, as both the poisoning rate and the initial abundance required for dominance of the toxin-producing species are much lower in the absence of generalist bacteria (dotted line in Fig. 4) than in the presence of generalist bacteria (solid line in Fig. 4). The specialist bacteria, in contrast, do not have a major effect on the outcome of phytoplankton competition. In the absence of specialist bacteria (dashed line in Fig. 4), the poisoning rate and the initial abundance required for dominance of the toxin-producing species are almost the same as in the presence of specialist bacteria (solid line in Fig. 4).

In conclusion, the model predicts that, in well-mixed systems, the outcome of the interaction between toxin-producing and toxin-sensitive phytoplankton species depends on the poisoning rate, the initial abundances of the two phytoplankton species, and the activities of specialist and generalist bacteria. The presence of generalist bacteria capable of degrading allelopathic compounds is pivotal, since their degradation activities may lead to the demise of toxin-producing phytoplankton populations. The presence of competing specialist bacteria seems of minor importance for the occurrence of toxin-producing phytoplankton in well-mixed systems.

Incomplete mixing – the cellular automata model

As in the well-mixed system, also under incomplete mixing either the toxin-sensitive phytoplankton species defeats the toxin-producing phytoplankton species (Fig. 5A), or the toxin-producing species defeats the toxin-sensitive species (Fig. 5B). The outcome of competition depends again upon the poisoning rate and the initial abundance of the toxin-producing phytoplankton species (Fig. 6). However, the threshold poisoning rate allowing toxin-producing species to win the competition is two orders of magnitude lower under incomplete mixing than in the well-mixed system (Note the difference in scale of the x-axis in Fig. 4 and Fig. 6). The reason is that under incomplete mixing the toxin produced by the toxin-producing phytoplankton species is only weakly diluted. Therefore, toxin concentrations are locally high and lethal for the individual toxin-sensitive phytoplankter. The toxin-producing phytoplankton species thus wins the competition locally, and from there it might ultimately exclude the toxin-sensitive phytoplankton species throughout the entire space. Furthermore, in contrast to the well-mixed model, generalist bacteria can be competitively excluded by specialist bacteria even when toxin-producing phytoplankton becomes dominant (Fig. 5B). Generalist bacteria survive only at high poisoning rate (Fig.

5C), where they coexist with specialist bacteria. The reason is spatial segregation of resources and consumers: the toxin may accumulate in patches not explored by the generalist bacteria and the specialist bacteria have a competitive advantage in all patches without the toxin. Both aspects favour specialist bacteria, and make it harder for generalist bacteria to survive.

The facilitating role of generalist bacteria for toxin-sensitive phytoplankton appears also under incomplete mixing (Compare the toxin and generalist bacteria in Fig. 5A and B). However, whereas specialist bacteria had little impact in well-mixed systems, specialist bacteria have a major effect on phytoplankton competition under incomplete mixing. More precisely, the parameter region that leads to dominance of toxin-producing phytoplankton species is much larger in the presence than in the absence of specialist bacteria (compare solid line and dashed line in Fig. 6). This can be explained as follows. Under incomplete mixing, the toxin is produced locally by the toxin-producing phytoplankton species, whereas detritus is produced by both phytoplankton species and is thus more widespread. The specialist bacteria are stronger competitors for detritus than the generalists, and therefore outcompete generalist bacteria wherever the toxin is not sufficiently available as a secondary resource for the generalists. As a result, specialist bacteria exclude generalist bacteria. This in turn implies that toxin degradation will be less efficient, so that under incomplete mixing toxin-producing phytoplankton species are more favoured in the presence of specialist bacteria than in the absence of specialist bacteria (Fig. 6).

In conclusion, the cellular automata model predicts that under incomplete mixing a much lower poisoning rate is required for toxin-producing phytoplankton to exclude toxin-sensitive phytoplankton than in a well-mixed system. Similar to well-mixed systems, generalist bacteria capable of decomposing the toxin have an important facilitating role for toxin-sensitive phytoplankton species. However, in contrast to well-mixed systems, under incomplete mixing specialist bacteria can have a major effect as well. They may suppress

toxin degradation activities of generalist bacteria owing to local competition, and via this indirect interaction specialist bacteria unable to degrade the toxin may promote the dominance of toxin-producing phytoplankton species.

DISCUSSION

In this paper we have analysed various model scenarios to gain a better understanding of the mechanisms and environmental conditions favouring allelopathic interactions between phytoplankton species. We will now compare key assumptions and model predictions with available empirical data.

Allelopathy between phytoplankton species and initial conditions

In line with previous studies of Chao and Levin (1981) and Durrett and Levin (1997), our model predicts that the winner of competition between a toxin-producing and a toxin-sensitive species depends on the initial abundances of these species. Toxin-producing species must exceed a threshold abundance before they can take full advantage of the production of allelopathic compounds. Indeed, several studies have observed effects consistent with this prediction. Negative effects of the cyanobacterium Trichormus doliolum on the growth of Anabaena variabilis were observed when inocula of Trichormus represented at least 25% of the total biomass of the mixed culture (Von Elert and Jüttner 1996). Rengefors and Legrand (2001) studied allelopathic effects of Peridinium aciculiferum on Rhodomonas lacustris. The growth of Rhodomonas was significantly poorer in treatments with a ratio of 1:0.2 of Peridinium: Rhodomonas than in controls. However, no significant negative effects on Rhodomonas were found at a ratio of 1:2. Sukenik et al. (2002) observed that the growth rate of Peridinium was stronger inhibited at higher initial population densities of Microcystis. These effects of initial abundances of toxin-producing species are related to the initial

quantity of toxins produced. As for any toxic compound, there is a minimum effective dose. For instance, cyanobacterin, which is a toxin produced by Scytonema hofmanni, inhibits the growth of Synechococcus for an initial concentration of 1.5μg ml^{-1} (Mason et al. 1982). At a lower cyanobacterin concentration of 1 μg/ml, however, Synechococcus growth slows but resumes after 2-3 days (Gleason and Paulson 1984). Owing to this threshold concentration effect, the model predicts that toxin-producing phytoplankton blooms will profit from any physical process that gathers together a high number of toxin-producing cells. These physical processes are, for instance, the convergence of water masses at frontal zones in the oceans which may function as pelagic seed banks (Smayda 2002), the formation of high concentrations of buoyant phytoplankton cells near the water surface at low wind mixing, or the accumulation of resting cells at particular depths in the water column.

Role of heterotrophic bacteria

Toxins produced by phytoplankton are potential resources for decomposers. Our model results suggest that heterotrophic bacteria greatly influence the fate of allelopathic interactions. Bacteria able to decompose toxins may prevent dominance of allelopathic phytoplankton species. Conversely, bacterial degradation activities may be partially modified by the presence of competing heterotrophic bacteria unable to degrade allelopathic compounds. Thus, in practice, the question is to know whether toxins produced by toxin-producing phytoplankton are difficult to be broken down by heterotrophic bacteria. Generally speaking, toxins that are easily decomposed, will not be very effective as allelopathic compounds.

In situations where heterotrophic bacteria able to break down toxins are challenged by better competitors unable to degrade the toxins, toxin decomposers may still have a major transient role by paving the way for toxin-sensitive phytoplankton. However, while

decomposing toxins, these heterotrophic bacteria act against the benefit of their resource providers and thus prepare their own demise. The role of this strategy was described by Nowak and Sigmund (1992) in a game theoretical context as a catalyser essential for starting a reaction: these catalyzers need to be initially present, grow in the intermediate phase, and remain as trace (go extinct in our situation) in the end (Fig. 3A).

Some experimental results support the hypothesis that heterotrophic bacteria play an important role in allelopathic interactions between phytoplankton. Keating (1978) tested the allelopathic effects of some cyanobacteria on diatom species while manipulating the presence of heterotrophic bacteria. Keating was able to rank effects in diatom inhibition. The following rank order was obtained, from strong to weak inhibition: (i) both cyanobacteria and diatoms axenic, (ii) axenic cyanobacteria and bacterized diatoms, (iii) bacterized cyanobacteria and axenic diatoms and (iv) both cyanobacteria and diatoms bacterized. In an enclosure experiment conducted in Heney lake, Quebec, Newhook and Briand (1987) noted a positive effect on diatoms when natural communities were enriched with heterotrophic bacteria. The authors suggested that bacterial degradation of allelopathic substances produced by cyanobacteria freed diatoms from inhibition. These results demonstrate the confounding role of heterotrophic bacteria in allelopathic interactions between phytoplankton species.

Effects of incomplete mixing

The model analysis predicts that allelopathic phytoplankton will become dominant much more easily under incomplete mixing than under complete mixing. Incomplete mixing favours toxin-producing species because the toxins are not as diluted as under complete mixing and because bacterial degradation activity is localised. Toxin levels may thus be locally high and harm toxin-sensitive phytoplankton species.

At the ecosystem level, numerous studies have noticed that harmful algal blooms are favoured by reduced turbulence, stratification, or otherwise calm weather conditions (Visser et al. 1996; Sherman et al. 1998; Huisman et al. 1999b; Smayda 2002). These studies confirm that incomplete mixing may favour harmful phytoplankton. In many of these studies, however, it is not clear whether the toxin produced has an allelopathic effect, since the wide variety of toxins produced by harmful algal blooms may also serve other purposes, like anti-predatory defense (Demott et al. 1991; Turner and Tester 1997).

To the best of our knowledge, among phytoplankton studies, only Keating (1978) suggested that incomplete mixing might be important for the development of allelopathic interactions. Two studies on heterotrophic bacteria, however, directly support the predicted relation between mixing intensity and allelopathic interactions. Chao and Levin (1981) studied competition between a toxin-producing and a toxin-sensitive strain of Escherichia coli in well-mixed liquid cultures and on agar plates. They showed that the toxin-producing strain could exclude the toxin-sensitive strain for much lower initial abundances (up to four orders of magnitude) when grown on agar plates than in well-mixed liquid medium. Kerr et al. (2002) studied competition between three strains of E. coli, a toxin-producing, a toxin-resistant and a toxin-sensitive strain, under different mixing regimes. They tested the prediction that the three strains would be able to coexist under incomplete mixing, in an oscillating fashion analogous to the rock-scissor-paper game (Durrett and Levin 1997; Kerr et al. 2002). Kerr et al. (2002) found that non-equilibrium coexistence of all three strains occurred on agar plates, whereas the resistant strain displaced the toxin-producing and the sensitive strain in well-mixed liquid cultures. Thus, both studies confirm the prediction that bacterial species producing allelopathic compounds are favoured by incomplete mixing. It would be a challenge to test experimentally whether similar patterns are found for phytoplankton as well.

Owing to spatial aspects of competition (Tilman and Kareiva, 1997), our model further predicts that mixing intensity affects the role of heterotrophic bacteria. Bacteria unable to degrade allelopathic compounds have little impact on the chemical warfare between phytoplankton species when mixing is intense. However, these same bacteria may favour toxin-producing phytoplankton when mixing is weak, by competitively displacing bacteria that can degrade allelopathic compounds. To the best of our knowledge, there are thus far no experimental studies that have investigated this complex interaction between mixing intensity, heterotrophic bacteria, and allelopathic phytoplankton.

In conclusion, the model analysed here predicts that weak mixing, especially when combined with the presence of heterotrophic bacteria unable to degrade allelopathic compounds, will favour the development of allelopathic phytoplankton populations. We hope that our model results will stimulate further research on the functional role of algal toxins in an ecological setting.

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Table 1. Variables and parameters used in the models.

Symbols	Meaning	Value		Units
Variables		Initial condition (complete mixing; incomplete mixing)		
N	nutrient	50;	5 per grid cell	quantity of nutrient
P_T	toxin-producing phytoplankton	10;	10% of the grid	number of individuals
P_S	toxin-sensitive phytoplankton	10;	10% of the grid	number of individuals
D	detritus	0;	0	quantity of detritus
T	toxin	0;	0	quantity of toxin
B_S	specialist bacteria	1;	1% of the grid	number of individuals
B_G	generalist bacteria	1;	1% of the grid	number of individuals
Parameters		Parameter value		
I	nutrient input		2	day^{-1}
q	nutrient loss		0.1	day^{-1}
μ_{P_i}	maximum specific growth rate of phytoplankton species i		1	day^{-1}
K_{N,P_T}	half-saturation constant for nutrient-limited growth of toxic phytoplankton		0.2	quantity of nutrient
K_{N,P_S}	half-saturation constant for nutrient-limited growth of sensitive phytoplankton		0.1	quantity of nutrient
β_{N,P_i}	yield of phytoplankton on nutrient		0.3	individuals/ nutrient
γ	poisoning rate		0.2	-
μ_{B_k}	maximum specific growth rate of bacterial species i		1.5	day^{-1}
$K_{D,B_S} = 1/b_{D,B_S}$	half-saturation constant for detritus-limited growth of specialist bacteria		0.3	quantity of detritus
$K_{D,B_G} = 1/b_{D,B_G}$	half-saturation constant for detritus-limited growth of generalist bacteria		0.35	quantity of detritus
$K_{T,B_G} = 1/b_{T,B_G}$	half-saturation constant for toxin-limited growth of generalist bacteria		0.2	quantity of toxin
β_{D_j,B_k}	yield of bacteria on detritus		0.3	individuals/ detritus
ρ	turnover rate		0.2	-
χ	fraction of recycled nutrient		0.9	-

Figure legends

Figure 1.

Diagram of the bacteria-phytoplankton system based on nutrient cycling. Thin arrows represent nutrient fluxes between compartments; the dotted arrow represents the constant external input of inorganic nutrient and bold arrows represent loss of nutrients from the compartment indicated. The dashed line ending with a closed circle represents the poisoning effect of the toxin on the toxin-sensitive phytoplankton species.

Figure 2. Graphical isocline analysis of the algal and bacterial species

(A) Isoclines of the two phytoplankton species in the nutrient-toxin plane. (B) Isoclines of the two bacterial species in the detritus-toxin plane. Closed circles: stable equilibrium points; open circles: unstable equilibrium points. Dashed lines: projection of the impact vectors. Hatch space: none of the species persists.

Figure 3. Complete mixing.

Dynamics of the phytoplankton species, bacteria and toxin for a poisoning rate of (A) $\gamma = 0.20$ and (B) $\gamma = 0.40$. (C) Effects of the poisoning rate γ and the initial abundance of the toxin-producing phytoplankton species on the outcome of the species interactions. Based on a grid of 51 x 101 simulations. Initial abundance of toxin-sensitive phytoplankton species = 10.

Figure 4. Role of heterotrophic bacteria in well-mixed systems.

Effects of the poisoning rate γ and the initial abundance of the toxin-producing phytoplankton species on the outcome of the species interactions. The graph indicates the boundary lines between the parameter region for which the toxin-sensitive species wins and the parameter region for which the toxin-producing species wins. Three different scenarios were evaluated.

Solid line: both bacterial species are present (as in Fig. 3C); dotted line: generalist bacteria are absent; dashed line: specialist bacteria are absent. Initial abundance of toxin-sensitive phytoplankton = 10. Based on 51 x 101 = 5151 simulations with the ODE model.

Figure 5. Incomplete mixing: Dynamics of the phytoplankton species, bacteria and toxin for a poisoning rate of (A) $\gamma = 0.002$, (B) $\gamma = 0.004$ and (C) $\gamma = 0.05$.

Figure 6. Incomplete mixing. Effects of the poisoning rate γ and the initial abundance of the toxin-producing phytoplankton species on the outcome of the species interactions. The graph is as in Fig. 4, but now for incomplete mixing. Solid line: both bacterial species are present (as in Fig. 3C); dotted line: generalist bacteria are absent; dashed line: specialist bacteria are absent. The initial abundance of the toxin-producing phytoplankton is expressed as the percentage of the total amount of grid cells initially occupied. Initial abundance of the toxin-sensitive phytoplankton species = 10%. Based on 15 x 15 = 225 simulations with the cellular automata model.

Figure 1.

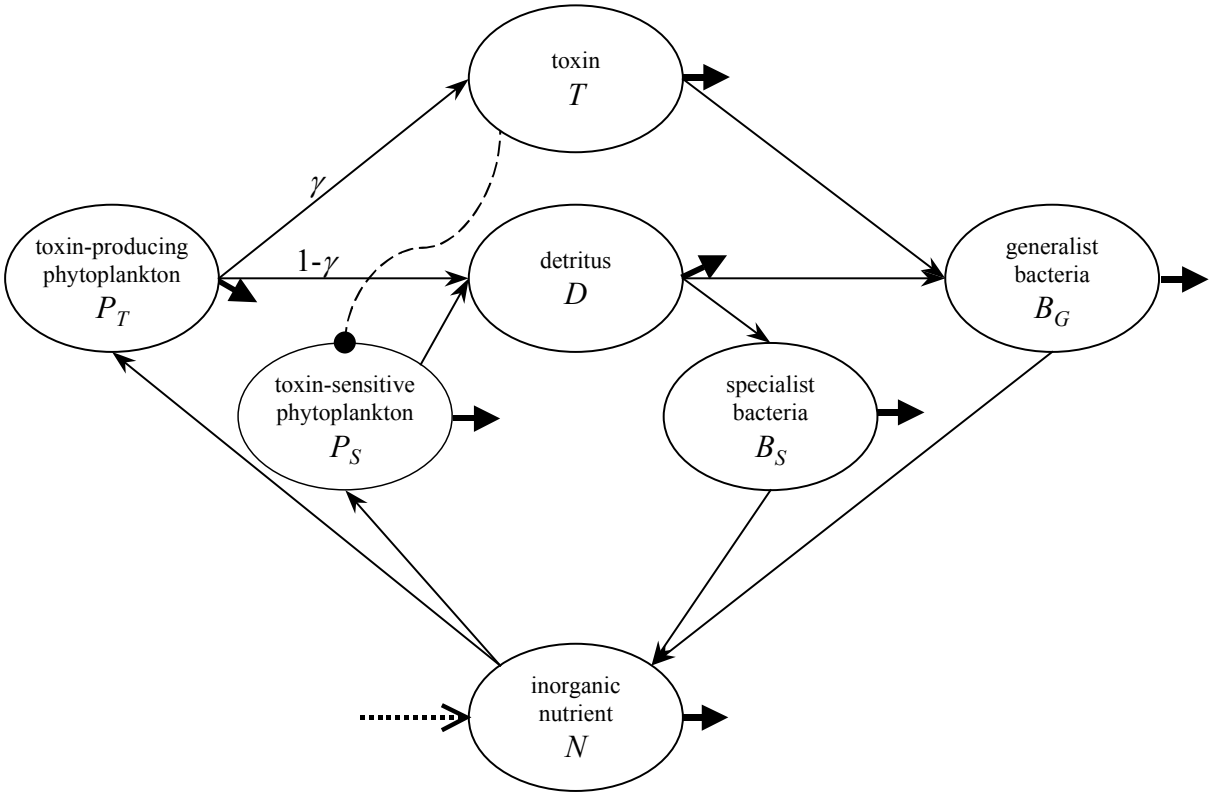

toxin
T
γ
toxin-producing
phytoplankton
P_T
1-γ
detritus
D
generalist
bacteria
B_G
toxin-sensitive
phytoplankton
P_S
specialist
bacteria
B_S
inorganic
nutrient
N

Figure 2.

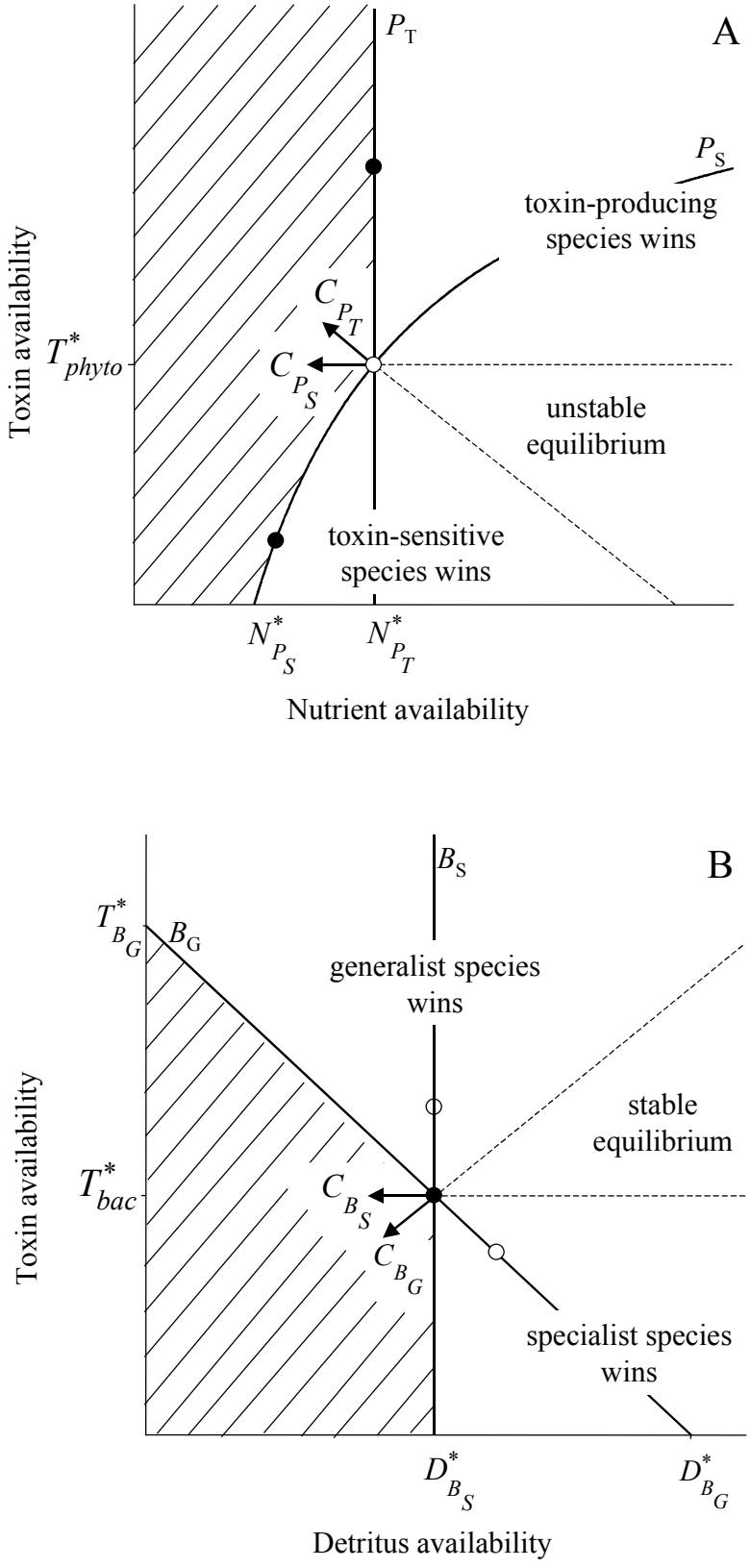

A
P_T
P_S
toxin-producing species wins
C_{P_T}
C_{P_S}
T^*_{phyto}
Toxin availability
unstable equilibrium
toxin-sensitive species wins
$N^*_{P_S}$
$N^*_{P_T}$
Nutrient availability
B
B_S
$T^*_{B_G}$
B_G
generalist species wins
stable equilibrium
T^*_{bac}
C_{B_S}
C_{B_G}
specialist species wins
Toxin availability
$D^*_{B_S}$
$D^*_{B_G}$
Detritus availability

Figure 3.

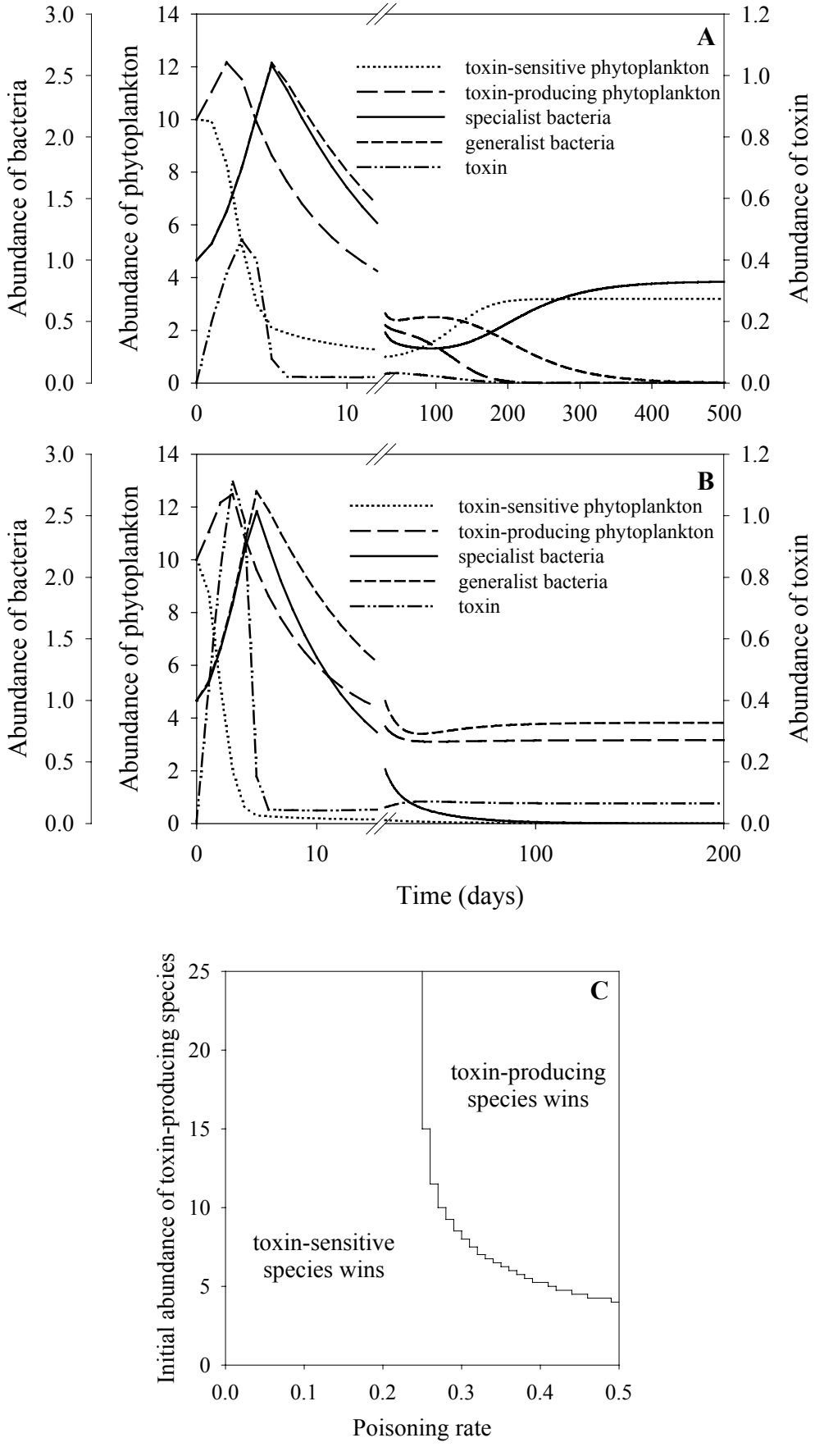

A
B
C
toxin-sensitive phytoplankton
toxin-producing phytoplankton
specialist bacteria
generalist bacteria
toxin
Abundance of bacteria
Abundance of phytoplankton
Abundance of toxin
Time (days)
Initial abundance of toxin-producing species
Poisoning rate
toxin-producing species wins
toxin-sensitive species wins

Figure 4.

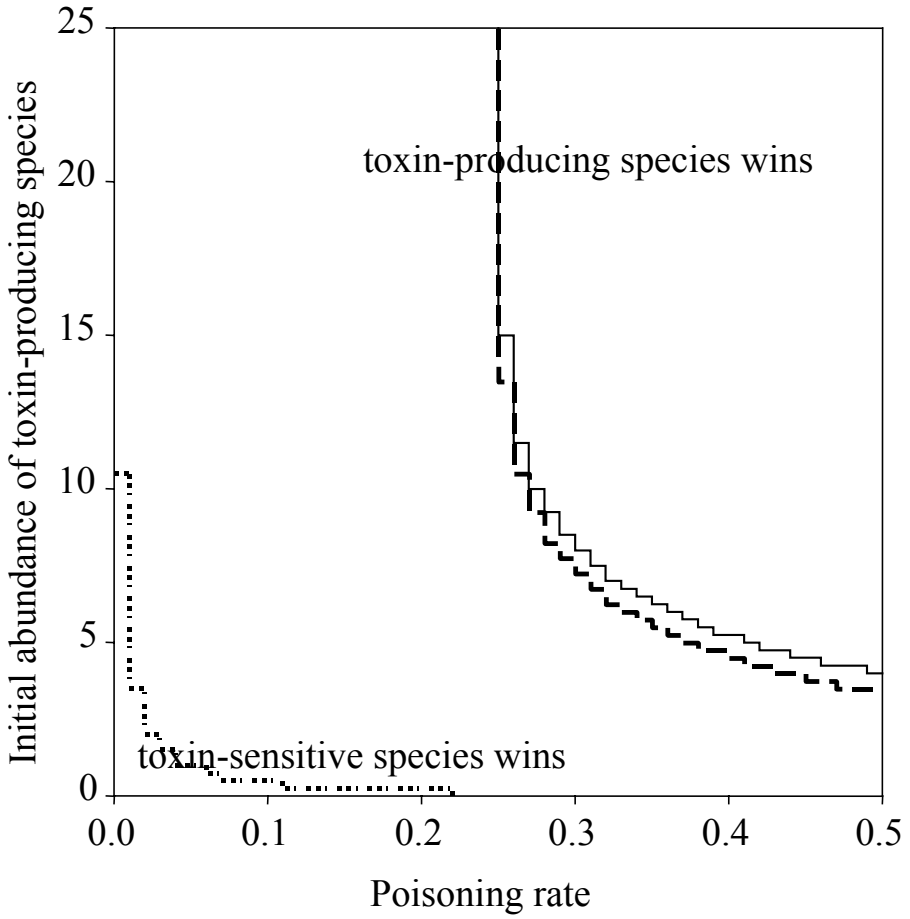

25
20
15
10
5
0
Initial abundance of toxin-producing species
toxin-producing species wins
toxin-sensitive species wins
0.0
0.1
0.2
0.3
0.4
0.5
Poisoning rate

Figure 5

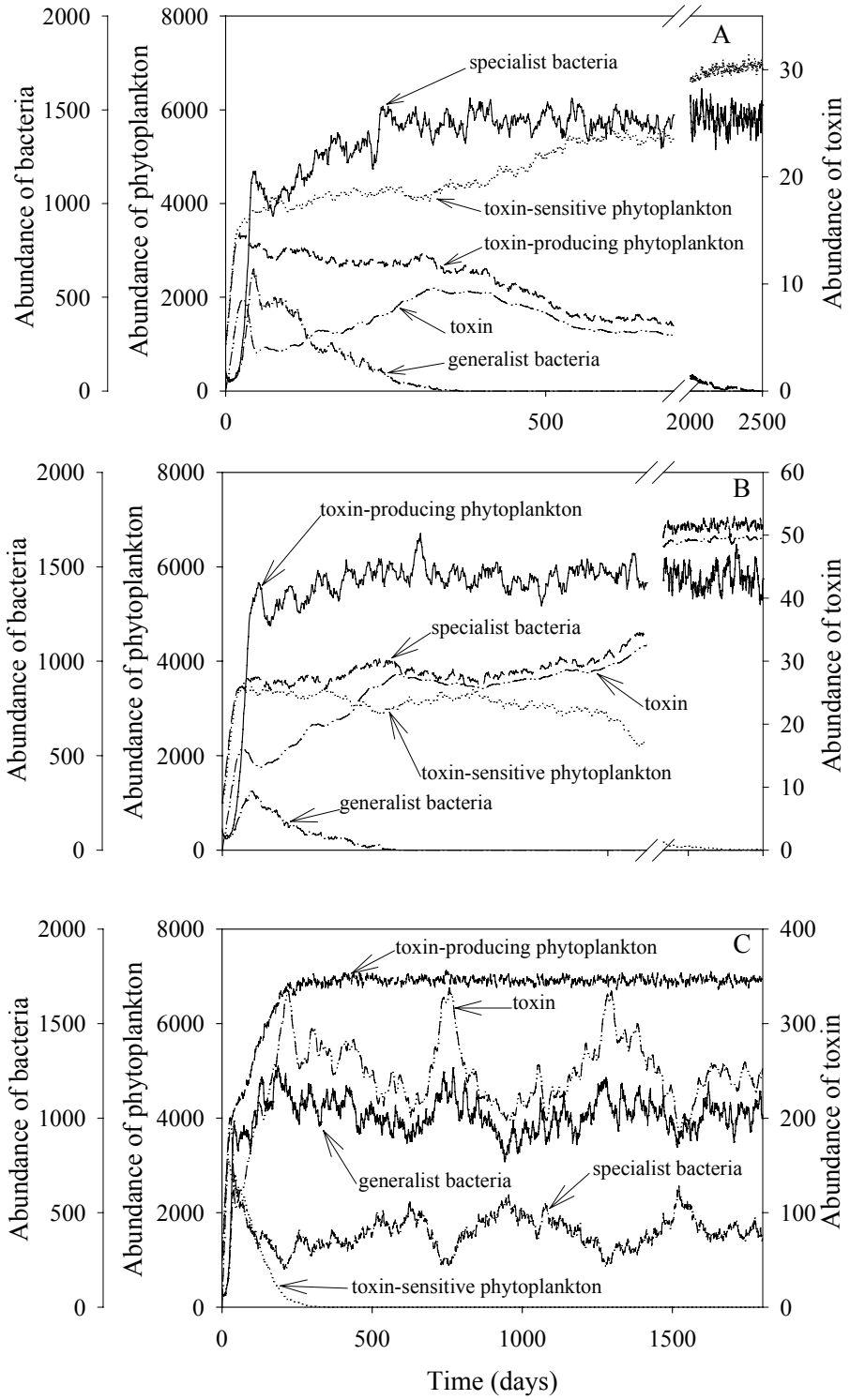
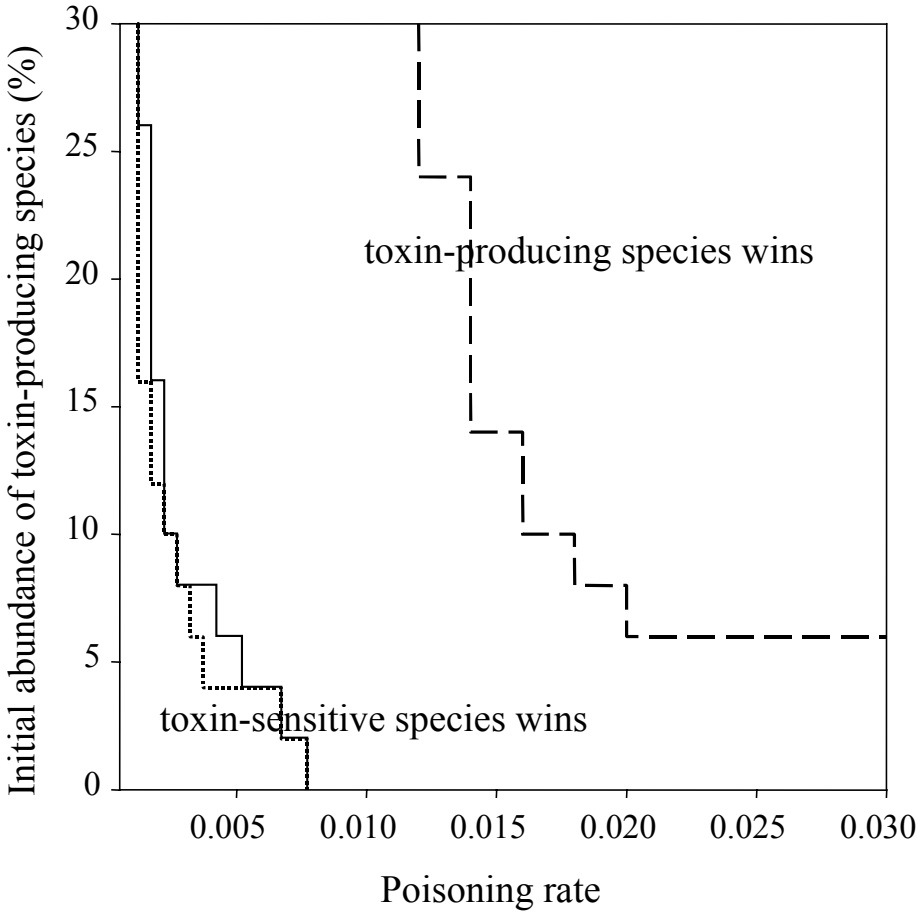

A
specialist bacteria
toxin-sensitive phytoplankton
toxin-producing phytoplankton
toxin
generalist bacteria
B
toxin-producing phytoplankton
specialist bacteria
toxin
toxin-sensitive phytoplankton
generalist bacteria
C
toxin-producing phytoplankton
toxin
generalist bacteria
specialist bacteria
toxin-sensitive phytoplankton
Abundance of bacteria
Abundance of phytoplankton
Abundance of toxin
Time (days)

Figure 6.


Initial abundance of toxin-producing species (%)
30
25
20
15
10
5
0
toxin-producing species wins
toxin-sensitive species wins
0.005
0.010
0.015
0.020
0.025
0.030
Poisoning rate