Nutrient-plankton interaction with a toxin in a variable input nutrient environment

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Abstract. A simple model of phytoplankton-zooplankton interaction with a periodic input nutrient is presented. The model is then used to study a nutrient-plankton interaction with a toxic substance that inhibits the growth rate of phytoplankton. The effects of the toxin upon the existence, magnitude, and stability of the periodic solutions are discussed.

Keywords: plankton populations, toxin, inhibitor, uniform persistence

1 Introduction

Phytoplankton are microscopic plants that live in the ocean, sea or lake. Through photosynthesis, phytoplankton are responsible for much of the oxygen present in the Earth's atmosphere. They convert inorganic materials into new organic compounds by the process of photosynthesis [14]. Hence the stocks of these tiny planktonic algae play a significant role for marine reserves and fishery management. In terms of numbers, the most important groups of phytoplankton are the diatoms, cyanobacteria and dinoflagellates, although many other groups of algae are also very populated.

Pollution of freshwater and marine systems by anthropogenic sources has become a concern over the last several decades. Organic (e.g. triazine herbicides) [1, 16, 17, 18, 25, 26] and inorganic compounds (e.g. heavy metals) [6, 13, 17, 18, 19, 23, 24] both may have harmful effects to the organsims. For example, samples taken from the inner harbor of the Waukegan area, located in Lake County, Illinois, on the west shore of Lake Michigan, have shown that photosynthesis of the green algae *Selenastrum capricornutum* is inhibited due to pollutants originating from industrial and recreational sources.

This study investigates the possible effects of toxic substances upon nutrient-phytoplankton-zooplankton interaction. In the early 1980s, Hansen and Hubbell [10] used antibiotic, nalidixic acid, to examine competition of two strains of *E. coli*. One strain was sensitive and the other was resistant to the inhibitor. This resistance of the population is due to a chromosomal mutation and it does not result in detoxification of the antibiotic. However, resistance by bacteria to antibiotics and heavy metals frequently comes from the acquisition of an extrachromosomal element that encodes an enzyme, which converts the inhibitor into a less toxic form. This reduces the intracellular toxic concentration and enables the survival of bacteria that produce the enzyme. It also results in a significant reduction of the inhibitor in the environment [2, 15]. This biological phenomenon motivates our plankton-toxin model proposed here.

The model consist of a single limiting nutrient, two plankton populations and the inhibitor, where the inhibitor may include agents such as pesticides or heavy metals. The phytoplankton feeds on nutrient and zooplankton grazes on phytoplankton. The zooplankton absorbs the inhibitor without effect, while phytoplankton's uptake rate and consequently its growth rate are inhibited due to the presence of an external inhibitor. Although our models are simplified systems, it is a first step in understanding complex interaction between the first two trophic levels and pollution. More complicated plankton system such as multiple nutrients can be found in Grover [8].

We first propose a simple plankton model with a periodic input nutrient concentration and summarize its dynamical consequences. We then examine the effect of toxin upon the existence, magnitude, and stability of the periodic solutions. Criteria for coexistence of both plankton populations are also discussed. However, comparisons between more complex dynamical behavior will only be numerically simulated. A base nutrient-plankton model of closed ecosystem is presented in the next section. Section 3 studies the model when phytoplankton is inhibited by the toxin. Numerical examples will be provided to illustrate complexity of the interaction. The final section provides a brief summary and discussion.

2 The nutrient-phytoplankton-zooplankton model

In this section we shall introduce a base model which will be used to study the effects of toxin upon plankton interaction later. For simplicity, it is assumed that the organisms and the nutrient are uniformly distributed over the space. Let $N(t)$, $P(t)$, and $Z(t)$ denote the nutrient concentration, the phytoplankton population, and zooplankton population at time t, respectively. For convenience, the two plankton levels are modeled in terms of nutrient content and therefore their units are nitrogen or nitrate per unit volume. We let δ and ϵ denote the per capita natural death rate of phytoplankton and zooplankton respectively. The phytoplankton's nutrient uptake rate is denoted by f, while g is the zooplankton's grazing rate. Since plankton populations are measured in terms of nutrient concentration, f and g are functions of nutrient concentration. Both functions have the standard monotonic assumptions as the classical Michaelis-Menton kinetics, Ivlev, and Holling type III functional responses given below:

(H1) $f, g \in C^1[0,\infty), f(0) = g(0) = 0, f'(x), g'(x) > 0$ for $x \geq 0$ and $\lim\limits_{x\to\infty} f(x) = \lim\limits_{x\to\infty} g(x) = 1$.

Parameter m is the maximal nutrient uptake rate of phytoplankton and c denotes the maximal zooplankton ingestion rate, where β and α are the fraction of zooplankton grazing conversion and phytoplankton nutrient conversion, respectively. In natural nutrient-plankton systems, waters flowing into the system bring input of fluxes of nutrients and outflows also carry out nutrients [3, 7, 20, 21]. Unlike the study in [11], we assume that the input nutrient concentration $N^*(t)$ is varied periodically around N^0 with $N^0(t) = N^0 + ae(t)$, where $N^0 > 0$, $0 < a < N^0$, and $e(t)$ is τ-periodic with mean value zero and $|e(t)| \leq 1$ for $t \geq 0$. It is assumed that the rate of waters flowing in and out of the system is a constant and denoted by D. Both plankton populations are also assumed to be flowing out of the system with the same constant washout rate D.

Nutrients are consumed by the phytoplankton, which in turn is grazed upon by the herbivorous zooplankton. Consequently, there are minus terms $-mf(N)P$ and $-cg(P)Z$ in the equations for $\dot{N}$ and $\dot{P}$, respectively. For simplicity, we assume that the system under study is closed and hence there are positive feedback terms δP, ϵZ, $(1-\alpha)mf(N)P$, and $(1-\beta)cg(P)Z$ appeared in the equation for $\dot{N}$. Our model with the above biological assumptions can be written as the following three dimensional nonautonomous ordinary differential equations.

$$\begin{aligned} \dot{N} &= D(N^0(t)-N) - mf(N)P + \delta P + \epsilon Z + (1-\beta)cg(P)Z \\ &\quad +(1-\alpha)mf(N)P \\ \dot{P} &= [\alpha mf(N) - \delta - D]P - cg(P)Z \\ \dot{Z} &= [\beta cg(P) - \epsilon - D]Z \\ &\quad N(0), P(0), Z(0) \geq 0, \end{aligned} \tag{2.1}$$

where $0 < \alpha, \beta \leq 1$, and $D, N^0, m, c, \epsilon, \delta > 0$.

Notice the scalar periodic equation

$$\begin{aligned} \dot{N} &= D(N^0(t) - N) \\ &\quad N(0) \geq 0 \end{aligned} \tag{2.2}$$

has a unique positive τ-periodic solution

$$N^*(t) = \frac{De^{-Dt}}{e^{Dt}-1} \int_t^{t+\tau} e^{Dr}[N^0 + ae(t)]dr$$

and solutions $N(t)$ of (2.2) can be written as $N(t) = N^*(t) + (N(0) - N^*(0))e^{-Dt}$ for all t. Hence solutions are asymptotic to the periodic solution $N^*(t)$. Since $\dot{N}|_{N=0} \geq DN^0(t) \geq 0$, $\dot{P}|_{P=0} = \dot{Z}|_{Z=0} = 0$, solutions of (2.1) remain nonnegative.

Let $U = N^*(t) - N - P - Z$. Then $\dot{U} = -DU$ and hence solutions of (2.1) are bounded. Moreover, system (2.1) can be rewritten as

$$\begin{aligned} \dot{U} &= -DU \\ \dot{P} &= [\alpha mf(N^*(t) - U - P - Z) - \delta - D]P - cg(P)Z \\ \dot{Z} &= [\beta cg(P) - \epsilon - D]Z. \end{aligned} \tag{2.3}$$

Since the ω-limit set of (2.3) lies on the set $U = 0$, (2.1) has the following limiting system:

$$\begin{aligned} \dot{P} &= [\alpha mf(N^*(t) - P - Z) - \delta - D]P - cg(P)Z \\ \dot{Z} &= [\beta cg(P) - \epsilon - D]Z \\ &\quad P(0), Z(0) \geq 0, P(0) + Z(0) \leq N^*(0). \end{aligned} \tag{2.4}$$

As $N(t) + P(t) + Z(t) = N^*(t)$ for $t \geq 0$ on the ω-limit set and solutions of (2.1) remain nonnegative, we see that $P(t) + Z(t) \leq N^*(t)$ for $t \geq 0$, i.e., system (2.4) is well-defined.

Let

$$\Gamma = \{(P, Z) \in R_+^2 : P + Z \leq N^*(0)\}.$$

System (2.4) has a trivial solution $(0, 0)$ for all parameter values. The Jacobian derivative of the Poincaré map induced by system (2.4) evaluated at $(0, 0)$ is given by $\Phi_0(t)$, where $\Phi_0(t)$ is the fundamental matrix solution of $\dot{X} = J_0 X$ with

$$J_0 = \begin{pmatrix} \alpha m f(N^*(t)) - \delta - D & 0 \\ 0 & -\epsilon - D \end{pmatrix}. \tag{2.5}$$

Let

$$\sigma_0 = \frac{1}{\tau} \int_0^\tau [\alpha m f(N^*(t)) - \delta - D] dt.$$

Theorem 2.1 *If $\sigma_0 < 0$, then solutions of (2.4) satisfy $\lim_{t\to\infty} P(t) = \lim_{t\to\infty} Z(t) = 0$.*

Proof. We may assume $P(0) > 0$. Since $\dot{P} \leq [\alpha m f(N^*(t)) - \delta - D]P$ for $t \geq 0$, consider the following equation

$$\dot{x} = [\alpha m f(N^*(t)) - \delta - D]x$$

with $x(0) = P(0)$. The solution can be written explicitly as

$$\begin{aligned} x(t) &= x(0) e^{\int_0^t [\alpha m f(N^*(r)) - \delta - D] dr} \\ &= x(0) e^{\int_0^{t_0} [\alpha m f(N^*(r)) - \delta - D] dr} e^{\int_{t_0}^{n\tau + t_0} [\alpha m f(N^*(r)) - \delta - D] dr} \\ &= x(0) e^{\int_0^{t_0} [\alpha m f(N^*(r)) - \delta - D] dr} e^{\int_0^{n\tau} [\alpha m f(N^*(r)) - \delta - D] dr} \end{aligned}$$

for some $0 \leq t_0 < \tau$ and $n > 0$, where t_0 and n depend on t. Notice $t \to \infty$ if and only if $n \to \infty$. Hence $\lim_{t\to\infty} x(t) = 0$ as $\sigma_0 < 0$. As a result, $\lim_{t\to\infty} P(t) = 0$. Therefore for any $\eta > 0$, there exists $t_1 > 0$ such that $P(t) < \eta$ for $t \geq t_1$. We choose $\eta > 0$ such that $\beta c g(\eta) < \epsilon + D$. It follows from the equation for $\dot{Z}$ in (2.4) that $\lim_{t\to\infty} Z(t) = 0$ and this completes the proof. ■

Suppose now $\sigma_0 > 0$. Consider the linear periodic system

$$\dot{X} = J_0 X \tag{2.6}$$

where J_0 is given in (2.5) and X is a row vector. Let $\Phi(t)$ be the fundamental matrix solution of the linear system (2.6) with $\Phi(0) = I$, the identity matrix. Then the Floquet multipliers of $(0, 0)$ are the eigenvalues of $\Phi(\tau)$ [5]. Since

$$\Phi(\tau) = \begin{pmatrix} e^{\int_0^\tau [\alpha m f(N^*(t)) - \delta - D]dt} & 0 \\ 0 & e^{-(\epsilon+D)\tau} \end{pmatrix} \tag{2.7}$$

and $\sigma_0 > 0$, we see that $(0, 0)$ is unstable.

Theorem 2.2 *Suppose* $\sigma_0 > 0$. *Then (2.4) has a unique* τ*-periodic solution* $(\bar{P}(t), 0)$ *with* $\bar{P}(t) > 0$. *Moreover, solutions of (2.4) with* $P(0) > 0$ *and* $Z(0) = 0$ *converge to* $(\bar{P}(t), 0)$ *asymptotically.*

Proof. Since $Z(t) = 0$ for $t > 0$ if $Z(0) = 0$, we consider the following equation

$$\begin{aligned} \dot{P} &= [\alpha m f(N^*(t) - P) - \delta - D]P \\ & \quad 0 \le P(0) \le N^*(0). \end{aligned} \tag{2.8}$$

Let $T_0 : [0, N^*(0)] \to [0, N^*(0)]$ denote the Poincaré map induced by equation (2.8), i.e., $T_0(P_0) = P(\tau, P_0)$, where $P(t, P_0)$ is the solution of (2.8) with $P(0) = P_0$.

Notice $T_0(0) = 0$, $T_0(N^*(0)) < N^*(0)$ and $\dot{T}_0 = \dfrac{\partial(\tau, P_0)}{\partial P_0} = v(\tau)$, where $v(t)$ satisfies

$$\begin{aligned} \dot{P} &= [\alpha m f(N^*(t) - P) - \delta - D - \alpha m f'(N^*(t) - P)P]v \\ v(0) &= 1. \end{aligned}$$

Therefore, $\dot{T}_0 > 0$, and in particular when $P_0 = 0$ we have

$$v(\tau) = e^{\int_0^\tau [\alpha m f(N^*(t)) - \delta - D)]dt}.$$

Thus $\dot{T}_0(0) > 1$, and the map T_0 has a unique positive fixed point $\bar{p}$, $\bar{p} < N^*(0)$, which corresponds to a unique positive τ-periodic solution $\bar{P}(t)$ for equation (2.8). Since T_0 is monotone increasing, it can be easily shown that $\lim_{n\to\infty} T_0^n(p) = \bar{p}$ for $0 < p \le N^*(0)$. Consequently, solutions of (2.8) with $P(0) > 0$ satisfy $\lim_{t\to\infty}(P(t) - \bar{P}(t)) = 0$. The proof is then complete. ■

Let

$$\sigma_1 = \frac{1}{\tau}\int_0^\tau [\beta c g(\bar{P}(t)) - \epsilon - D]dt.$$

Theorem 2.3 *Let* $\sigma_0 > 0$ *and* $\sigma_1 < 0$*. Then solutions of (2.4) with* $P(0) > 0$ *satisfy* $\lim_{t\to\infty}(P(t) - \bar{P}(t)) = \lim_{t\to\infty} Z(t) = 0$.

Proof. We claim that $\lim_{t\to\infty} Z(t) = 0$. Since $\dot{P} \leq [\alpha m f(N^*(t) - P) - \delta - D]P$ for all $t \geq 0$, consider the following equation

$$\begin{aligned} \dot{x} &= [\alpha m f(N^*(t) - x) - \delta - D]x \\ & \quad x(0) = P(0). \end{aligned} \tag{2.9}$$

Observe that $P(t) \leq x(t)$ for $t \geq 0$. Since $x(t) \to \bar{P}(t)$ as $t \to \infty$ by Theorem 2.2, $\liminf_{t\to\infty}(x(t) - \bar{P}(t)) = 0$. Hence for any $\eta > 0$ given, there exists $t_0 > 0$ such that $x(t) \leq \bar{P}(t) + \eta$ for $t \geq t_0$. As a result, $P(t) \leq \bar{P}(t) + \eta$ for $t \geq t_0$. By our assumption we can choose $\eta > 0$ such that

$$\int_0^\tau [\beta c g(\bar{P}(t) + \eta) - \epsilon - D]dt < 0.$$

Consequently, $\dot{Z} \leq [\beta c g(\bar{P}(t) + \eta) - \epsilon - D]Z$ for $t \geq t_0$ implies $\lim_{t\to\infty} Z(t) = 0$.

It remains to show that $\lim_{t\to\infty}(\bar{P}(t) - P(t)) = 0$. Consider the Poincaré map T induced by system (2.4), $T(P_0, Z_0) = (P(\tau), Z(\tau))$, where $(P(t), Z(t))$ is the solution of (2.4) with initial condition (P_0, Z_0). Since $\lim_{t\to\infty} Z(t) = 0$, $\lim_{n\to\infty} T^n(P_0, Z_0)$ lies on the P-axis. Moreover, $T^n(P_0, 0) = (T_0^n P_0, 0)$, where T_0 is the Poincaré map associated with equation (2.8). Since T_0 has a unique positive fixed point $\bar{p}$ which is moreover globally asymptotically stable for T_0 in $(0, N^*(0)]$, it follows that $T^n(P_0, 0)$ converges to the fixed point $(\bar{p}, 0)$. Therefore the periodic solution $(\bar{P}(t), 0)$ is globally asymptotically stable. ■

Suppose now $\sigma_0, \sigma_1 > 0$. Then the floquet multipliers are the eigenvalues of $\Phi_1(\tau)$, where $\Phi_1(t)$ is the fundamental matrix solution of $\dot{X} = J_1 X$, where

$$J_1 = \begin{pmatrix} J_{11} & -\alpha m f'(N^*(t) - \bar{P}(t))\bar{P}(t) - c g(\bar{P}(t)) \\ 0 & \beta c g(\bar{P}(t)) - \epsilon - D \end{pmatrix}, \tag{2.10}$$

and

$$J_{11} = \alpha m f(N^*(t) - \bar{P}(t)) - \delta - D - \alpha m f'(N^*(t) - \bar{P}(t))\bar{P}(t).$$

It follows that the periodic solution $(\bar{P}(t), 0)$ is unstable as $\sigma_1 > 0$. Similar to the arguments used in [11] we can prove that both populations can coexist by using the concepts of uniform persistence.

Theorem 2.4 *If $\sigma_0, \sigma_1 > 0$, then system (2.1) is uniformly persistent.*

Proof. We first apply Theorem 3.1 of Butler and Waltman [4] to show uniform persistence of the limiting system (2.4). Let $\mathcal{F}$ be the flow generated by system (2.4) and $\partial\mathcal{F}$ be $\mathcal{F}$ restricted to the boundary Γ. We need to verify that $\partial\mathcal{F}$ is isolated and acyclic. Let $M_0 = \{(0,0)\}$ and $M_1 = \{(\bar{P}(t), 0) : 0 \leq t \leq \tau\}$. Then the invariant set of $\partial\mathcal{F}$ is $\{M_0, M_1\}$. It is clear that $\partial\mathcal{F}$ is acyclic as M_0 and M_1 are globally attracting on the positive Z-axis and P-axis respectively and thus no subset of $\{M_0, M_1\}$ can form a cycle.

It remains to prove that each M_i is isolated for $\partial\mathcal{F}$ and for $\mathcal{F}$ respectively, for $i = 0, 1$. We only verify that M_0 is isolated for $\mathcal{F}$ as the remaining assertion can be argued similarly. Let $c_0 = \max_{0 \leq P \leq N^*(0)} g'(P)$. By our assumption we can choose $\rho > 0$ such that

$$\frac{1}{\tau}\int_0^\tau [\alpha m f(N^*(t) - \rho) - \delta - D - c c_0 \rho] dt > 0. \tag{2.11}$$

Let $\mathcal{N} = \{(P, Z) \in \Gamma : d((P, Z), M_0) < \rho\}$, where d is the Euclidean metric on R^2. We show that $\mathcal{N}$ is an isolated neighborhood of M_0 in Γ.

If this is not true, then there exists an invariant set V in Γ such that $M_0 \subset V \subset \mathcal{N}$ and $V \backslash M_0 \neq \emptyset$. Notice we can find $P(0), Z(0) > 0$ such that $(P(0), Z(0)) \in V \backslash M_0$. On the other hand, $V \subset \mathcal{N}$ implies

$$\begin{aligned} \frac{\dot{P}}{P} &= \alpha m f(N^*(t) - P - Z) - \delta - D - \frac{c g(P)}{P} Z \\ &\geq \alpha m f(N^*(t) - \rho) - \delta - D - c c_0 \rho. \end{aligned}$$

Hence

$$P(t) \geq P(0) e^{\int_0^t [\alpha m f(N^*(s) - \rho) - \delta - D - c_0 c \rho] ds}$$

and we have $\lim\limits_{t\to\infty} P(t) = \infty$ by inequality (2.11). This is impossible as solutions of (2.4) are bounded. Therefore M_0 must be isolated in $\partial\mathcal{F}$. Furthermore, let $\overset{\circ}{\Gamma}$ denote the interior of Γ and $W_i^+(M_i)$ be the stable manifold of M_i, $i = 0, 1$. It follows from the Floquet multipliers of M_i that $W^+(M_i)$ is disjoint from $\overset{\circ}{\Gamma}$ for $i = 0, 1$. Therefore (2.4) is uniformly persistent by [4].

We now rewrite system (2.4) as $\dot{Y} = F(Y,t)$ and system (2.3) as $\dot{X} = F(X,t) + R(X,t)$. Therefore there exists $C = D \max_{0 \le t \le \tau} N*(t)$ such that $|R(X,t)| \le Ce^{-Dt}$ for $t \ge 0$ for all solution $X(t)$ of system (2.3). As a result, Lemma A.4 of Hale and Somolinos [9] implies that the asymptotic behavior of (2.3) and (2.4) are the same. Since systems (2.1) and (2.3) are equivalent, we can conclude that system (2.1) is uniformly persistent. ■

In summary, if the average maximal growth rate $\frac{1}{\tau}\int_0^\tau [\alpha m f(N^*(t)) - \delta - D]dt$ of phytoplankton is less than the total removal rate $\delta + D$, then phytoplankton population goes extinct and so does the zooplankton. If the average maximal growth rate of phytoplankton exceeds its total removal rate then the phytoplankton population can stabilize in a positive periodic solution fashion, $\bar{P}(t)$, in the absence of zooplankton. Consequently, zooplankton population becomes extinct if its average maximal growth rate $\displaystyle\frac{1}{\tau}\int_0^\tau [\beta c g(\bar{P}(t)) - \epsilon - D]dt$ when phytoplankton is stabilized, is less than its total removal rate $\epsilon + D$, and both populations can coexist if these average maximal growth rates are greater than the total removal rates.

3 A nutrient-plankton-toxin model with inhibition of the phytoplankton

Motivated by the discussion in Section 1, in this section we will consider the situation when toxic substance has a negative effect on the phytoplankton. Specifically, the uptake rate and consequently the growth rate of phytoplankton is inhibited by the presence of the toxin, but zooplankton can consume the substance without any effect. An example from the field for this scenario would be a marine planktonic community comprising mainly diatoms and herbivorous copepods in a low silicate, elevated copper environment [22]. In this case copper will harm only diatoms and not crustaceans. At low concentrations the herbicide triazine also affects primary producers directly by inhibiting photosynthesis, while effects on subsequent trophic levels only would be indirect [22]. Our goal is to study toxic effects on the nutrient-plankton system by investigating simple solutions and asymptotic dynamics analytically whenever it is possible.

Let $S(t)$ denote the toxic concentration at time t. In addition to the nutrient concentration, it is assumed that the toxin is continuously pouring into the system with constant input concentration S^0 and the same constant input rate D as the nutrient. It is assumed that zooplankton can uptake the substance without any effect while phytoplankton's uptake rate of nutrient is decreased by a function $h(S)$ depending on the toxin level S. Zooplankton's toxin uptake rate is denoted by u. Functions h and u are assumed to satisfy the following assumptions.

(H2) $h \in C^1[0,\infty)$, $h(0)=1$, $h'(x)<0$ and $h(x)>0$ for all $x \geq 0$.

(H3) $u \in C^1[0,\infty), u(0)=0, u'(x)>0$ for $x \geq 0$ and $\lim\limits_{x\to\infty} u(x)=1$.

Let $b>0$ denote the maximum zooplankton toxin uptake rate. Similar to the previous model we assume the ecosystem under study is closed. With the above biological assumptions, the plankton-toxin interaction is given below.

$$\begin{aligned}
\dot{N} &= D(N^0(t)-N) - mf(N)h(S)P + \delta P + \epsilon Z + (1-\beta)cg(P)Z \\
&\quad +(1-\alpha)mf(N)h(S)P \\
\dot{P} &= [\alpha mf(N)h(S) - \delta - D]P - cg(P)Z \\
\dot{Z} &= [\beta cg(P) - \epsilon - D]Z \\
\dot{S} &= D(S^0 - S) - bu(S)Z \\
&\quad N(0), P(0), Z(0), S(0) \geq 0,
\end{aligned} \tag{3.1}$$

where $0<\alpha,\beta \leq 1$ and $D, N^0, S^0, m, b, c, \epsilon, \delta > 0$.

Since $\dot{S} \leq D(S^0-S)$ for $t \geq 0$, $\limsup\limits_{t\to\infty} S(t) \leq S^0$. Consequently, using the same argument as we did for system (2.1), it can be easily seen that solutions of (3.1) remain nonnegative and are bounded. Moreover, system (3.1) has the following limiting system

$$\begin{aligned}
\dot{P} &= [\alpha mf(N^*(t)-P-Z)h(S) - \delta - D]P - cg(P)Z \\
\dot{Z} &= [\beta cg(P) - \epsilon - D]Z \\
\dot{S} &= D(S^0 - S) - bu(S)Z \\
&\quad P(0), Z(0), S(0) \geq 0, P(0)+Z(0) \leq N^*(0).
\end{aligned} \tag{3.2}$$

Notice that system (3.2) is well defined as $P(t)+Z(t) \leq N^*(t)$ for $t \geq 0$ for all solutions of (3.2) with $P(0)+Z(0) \leq N^*(0)$. Clearly there always exists a trivial solution $(0,0,S^0)$ for (3.2). Let

$$\sigma_0 = \frac{1}{\tau}\int_0^\tau [\alpha mf(N^*(t)) - \delta - D]dt$$

and

$$\rho_0 = \frac{1}{\tau}\int_0^\tau [\alpha m f(N^*(t))h(S^0) - \delta - D]dt.$$

Then

$$\rho_0 < \sigma_0.$$

It is straightforward to show that $(0, 0, S^0)$ is locally stable if $\rho_0 < 0$. Similar to section 2, we can show that solutions of system (3.2) asymptotically approach $(0, 0, S^0)$ if $\sigma_0 < 0$, a stronger condition than $\rho_0 < 0$.

Proposition 3.1 *If* $\sigma_0 < 0$*, then solutions of (3.2) satisfy* $\lim_{t\to\infty} P(t) = \lim_{t\to\infty} Z(t) = 0$ *and* $\lim_{t\to\infty} S(t) = S^0$.

For the autonomous case [12], numerical simulations demonstrated the existence of an attracting interior steady state when $\alpha m f(N^0)h(S^0) < \delta + D$ and $\alpha m f(N^0) > \delta + D$. Therefore, it is strongly suspected that complicated dynamical behavior can occur for system (3.2) when $\sigma_0 > 0$ and $\rho_0 < 0$. We next use numerical examples to demonstrate complexity of the model.

Let $N^0(t) = 10 + 5\sin(\frac{\pi t}{10})$, $f(x) = \frac{x}{2+x}$, $g(x) = \frac{x}{1+x}$, $h(s) = e^{-bs}$ and $u(s) = \frac{s}{6+s}$. Parameters used are $D = 0.07$, $\delta = 0.04$, $\epsilon = 0.01$, $c = 0.3$, $m = 5$, $\alpha = 0.9$, $\beta = 0.4$, $b = 1.5$ and $S^0 = 4$. In this case $\sigma_0 = 3.6369$ and $\rho_0 = -0.1007$. Therefore according to our earlier analysis that trivial solution $(0, 0, S^0)$ is locally stable. Simulations showing the existence of a positive periodic solution which is locally stable. Figure 1 provides two solutions that converge to a positive periodic solution. Figure 2 plots the trivial periodic solution $(0, 0, S^0)$ and a solution (dashed line) with initial condition $(1, 1, 5)$ that converges to the trivial periodic solution. Therefore when $\sigma_0 > 0$ and $\rho_0 < 0$ the model exhibits a locally stable positive periodic solution even when the trivial solution is locally stable.

Proposition 3.2 *If* $\rho_0 > 0$*, then (3.2) has a unique* τ*-periodic solution of the form* $(\hat{P}(t), 0, S^0)$*, where* $\hat{P}(t) > 0$*, and solutions of (3.2) with* $Z(0) = 0$ *satisfy* $\lim_{t\to\infty}(P(t) - \hat{P}(t)) = \lim_{t\to\infty} Z(t) = 0$ *and* $\lim_{t\to\infty} S(t) = S^0$.

Proof. Since $Z(t) = 0$ for $t > 0$ if $Z(0) = 0$, it is enough to consider the

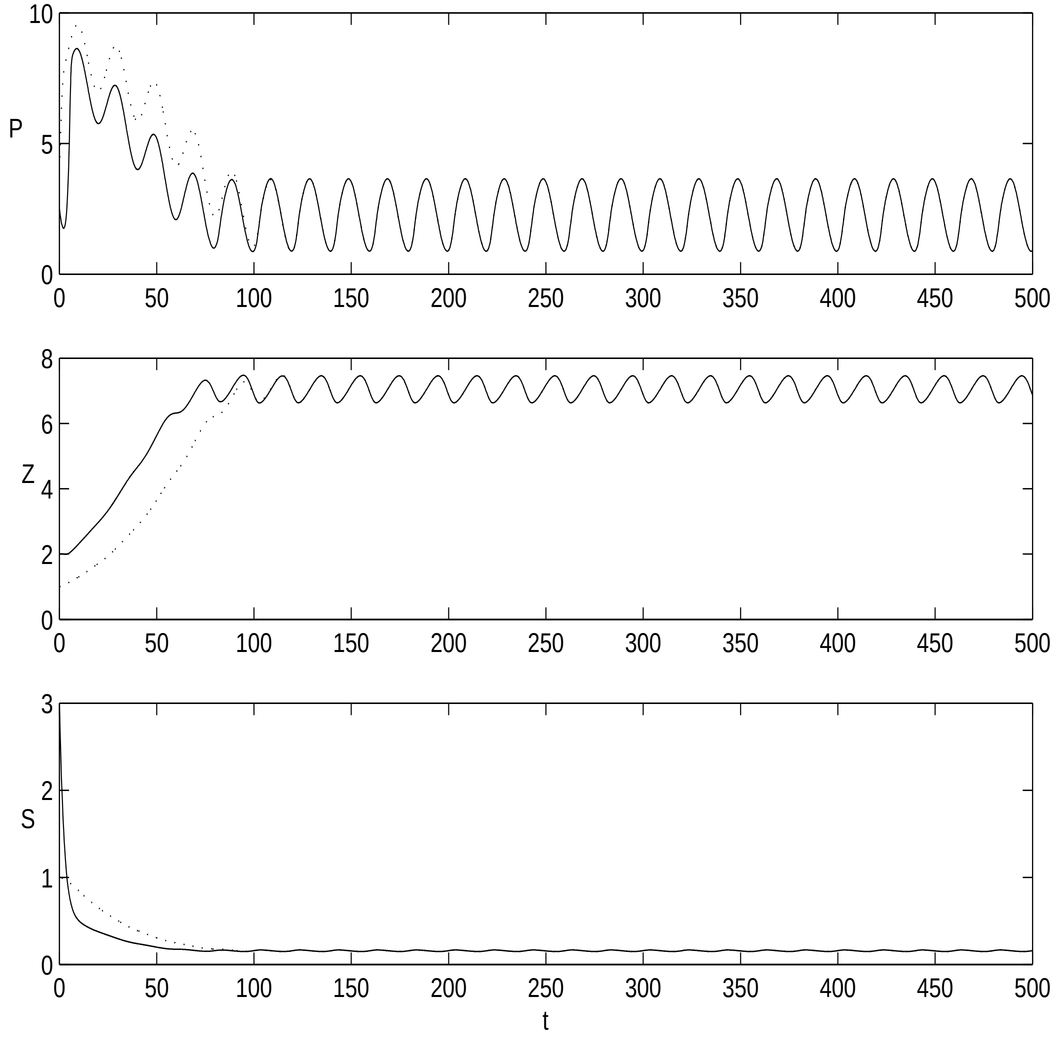

Figure 1: Both solutions asymptotically converge to the positive periodic solution. Initial conditions used are $(2.5, 2, 3)$ for solid curve and $(4, 1, 1)$ for dashed curve.

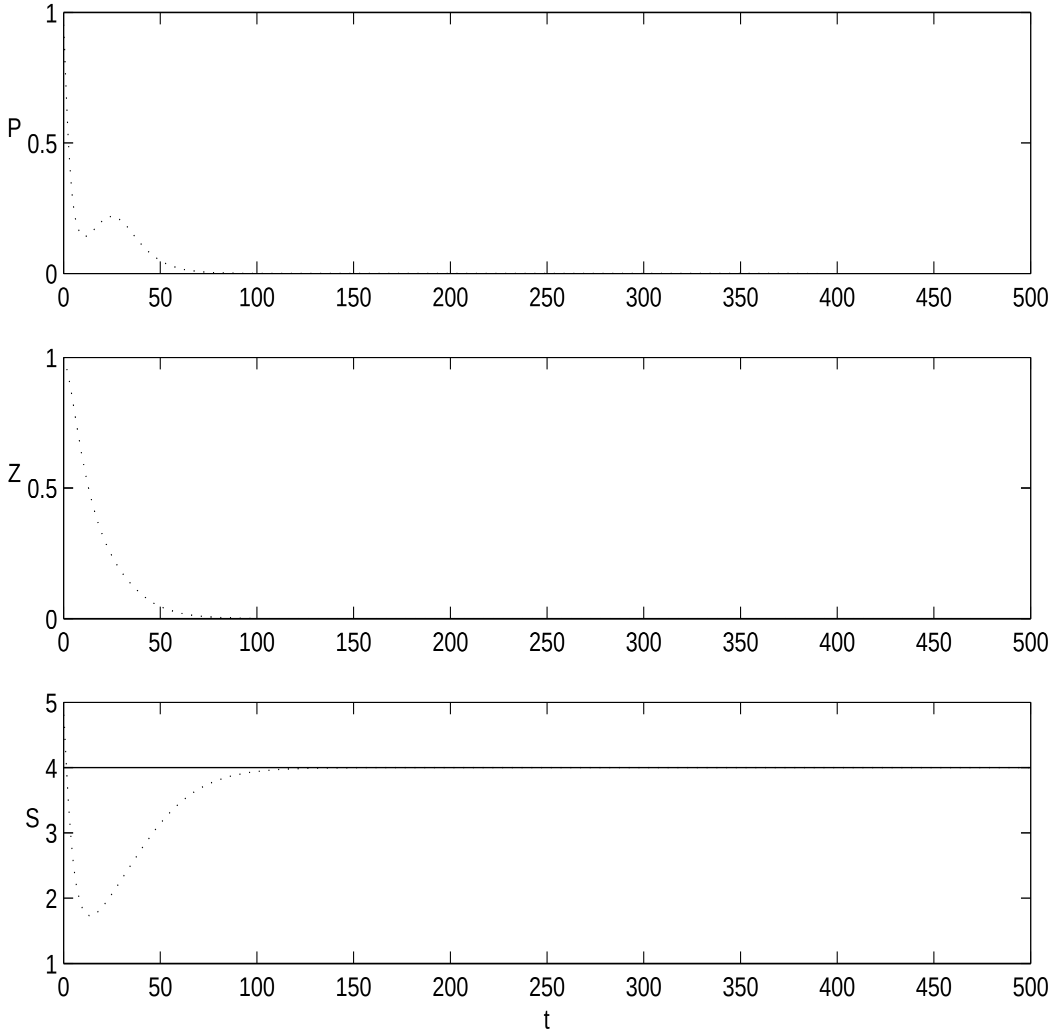

Figure 2: The figure plots the trivial solution $(0, 0, S^0)$. Another solution (dashed curve) using initial condition $(1, 1, 5)$ converges to the trivial solution.

following system

$$\begin{aligned} \dot{P} &= [\alpha m f(N^*(t) - P)h(S) - \delta - D]P \\ \dot{S} &= D(S^0 - S) \\ & \quad P(0), S(0) \geq 0, P(0) \leq N^*(0). \end{aligned} \tag{3.3}$$

As $\dot{S}$ can be decoupled from P, we see that $\lim_{t\to\infty} S(t) = S^0$. Hence for any $\eta > 0$ there exists $t_0 > 0$ such that $S^0 - \eta < S(t) < S^0 + \eta$ for $t \geq t_0$. It is clear that $\frac{1}{\tau}\int_0^\tau [\alpha m f(N^*(t))h(S^0 - \eta) - \delta - D]dt > 0$. We choose $\eta > 0$ such that

$$\frac{1}{\tau}\int_0^\tau [\alpha m f(N^*(t))h(S^0 + \eta) - \delta - D]dt > 0.$$

Notice

$$\alpha m f(N^*(t){-}P)h(S^0{+}\eta){-}\delta{-}D]P \leq \dot{P} \leq [\alpha m f(N^*(t){-}P)h(S^0{-}\eta){-}\delta{-}D]P$$

for all $t \geq t_0$.

Considering

$$\begin{aligned} \dot{x} &= [\alpha m f(N^*(t) - x)h(S^0 - \eta) - \delta - D]x \\ & \quad x(0) = P(t_0) \leq N^*(0), \end{aligned} \tag{3.4}$$

and

$$\begin{aligned} \dot{y} &= [\alpha m f(N^*(t) - y)h(S^0 + \eta) - \delta - D]y \\ & \quad y(0) = P(t_0) \leq N^*(0). \end{aligned} \tag{3.5}$$

Let T_1 and T_2 be the Poincaré maps induced by equations (3.4) and (3.5) respectively, i.e., $T_1 : [0, N^*(0)] \to [0, N^*(0)]$ by $T_1(x_0) = x(\tau, x_0)$, where $x(t, x_0)$ is the solution of (3.4) with initial condition x_0, and T_2 is defined similarly. It follows that $T_i(0) = 0$, $\dot{T}_i > 0$, $T_i(N^*(0)) < N^*(0)$, and $\dot{T}_i(0) > 1$ for $i = 1, 2$. Thus the map T_i has a unique positive fixed point $\hat{p}^i_\eta$, $\hat{p}^i_\eta < N^*(0)$ and solutions with positive initial conditions under forward iterations of T_i all converge to $\hat{p}^i_\eta$ for $i = 1, 2$. Consequently, solutions of (3.4) and (3.5) converge to $\hat{P}^i_\eta$, where $\hat{P}^i_\eta(t)$ is the corresponding positive τ-periodic solution of (3.4) and (3.5), respectively. On the other hand $\hat{p}^i_\eta \to \hat{p}$ as $\eta \to 0^+$ for $i = 1, 2$, where $\hat{p}$ is the unique positive fixed point for the Poincaré map induced by the equation

$$\begin{aligned} \dot{P} &= [\alpha m f(N^*(t) - P)h(S^0) - \delta - D]P \\ & \quad 0 \leq P(0) \leq N^*(0). \end{aligned} \tag{3.6}$$

Notice system (3.2) has a unique τ-periodic solution $(\hat{P}(t), 0, S^0)$. Since $y(t) \leq P(t) \leq x(t)$ for all $t \geq t_0$, we see that $\lim_{t\to\infty}(P(t) - \hat{P}(t)) = 0$ and the proof is complete. ■

Let $\rho_0 > 0$ so that (3.2) has the τ-periodic solution $(\hat{P}(t), 0, S^0)$. Define

$$\rho_1 = \frac{1}{\tau}\int_0^\tau [\beta c g(\hat{P}(t)) - \delta - D]dt.$$

It is clear that $(\hat{P}(t), 0, S^0)$ is locally stable if $\rho_1 < 0$. Similar to the analysis in section 2, we are unable to reach the conclusion as whether (3.2) has a positive τ-periodic solution when $\rho_1 > 0$. We next numerically simulate the model. We adopt the same functionals as we did for the previous two plots but with somewhat different parameter values: $\alpha = 0.15$, $\beta = 0.35$ and $S^0 = 1$. In this case $\rho_0 = 0.5145 > 0$ and $\rho_1 = -0.0199 < 0$. The system has a periodic solution $(\hat{P}, 0, S^0)$ which is locally stable. Figure 3 plots three solutions with quite different behavior. The top curve using initial condition $(1, 0, 1)$ converges to the periodic solution $(\hat{P}, 0, S^0)$. The other two solutions using initial conditions $(2.5, 2, 3)$ and $(4, 1, 1)$, respectively. Therefore the system has a complicated dynamical behavior.

On the other hand, if $\rho_0 > 0$ and $\rho_1 > 0$, then apply a similar argument as in Theorem 2.4 we can show that system (3.1) is uniformly persistent.

Theorem 3.2 *If* $\rho_1 > 0$ *and* $\rho_2 > 0$*, then system (3.1) is uniformly persistent.*

We next use the same functionals as for previous graphs and choose the following parameter values: $D = 0.02$, $\delta = 0.04$, $\epsilon = 0.01$, $c = 0.3$, $m = 5$, $\alpha = 0.15$, $\beta = 0.35$, $b = 1.5$ and $S^0 = 1$. Then $\sigma_0 = 0.5650$, $\rho_0 = 0.0794$ and $\rho_1 = 0.0344$ and system (3.1) is uniformly persistent according to Theorem 3.2. The following figure provides two plots with initial condition $(2.5, 2, 3)$ for solid curve and $(1, 0.1, 1)$ for dashed curve. Although solutions are oscillating, both plankton populations survived.

4 Discussion

Nutrient-phytoplankton-zooplankton models are proposed to study the effects of pollutants upon the nutrient-plankton interaction. For simplicity, the nutrient-plankton interaction is assumed to be a closed ecological system. The input nutrient concentration motivated by the seasonal and day/night

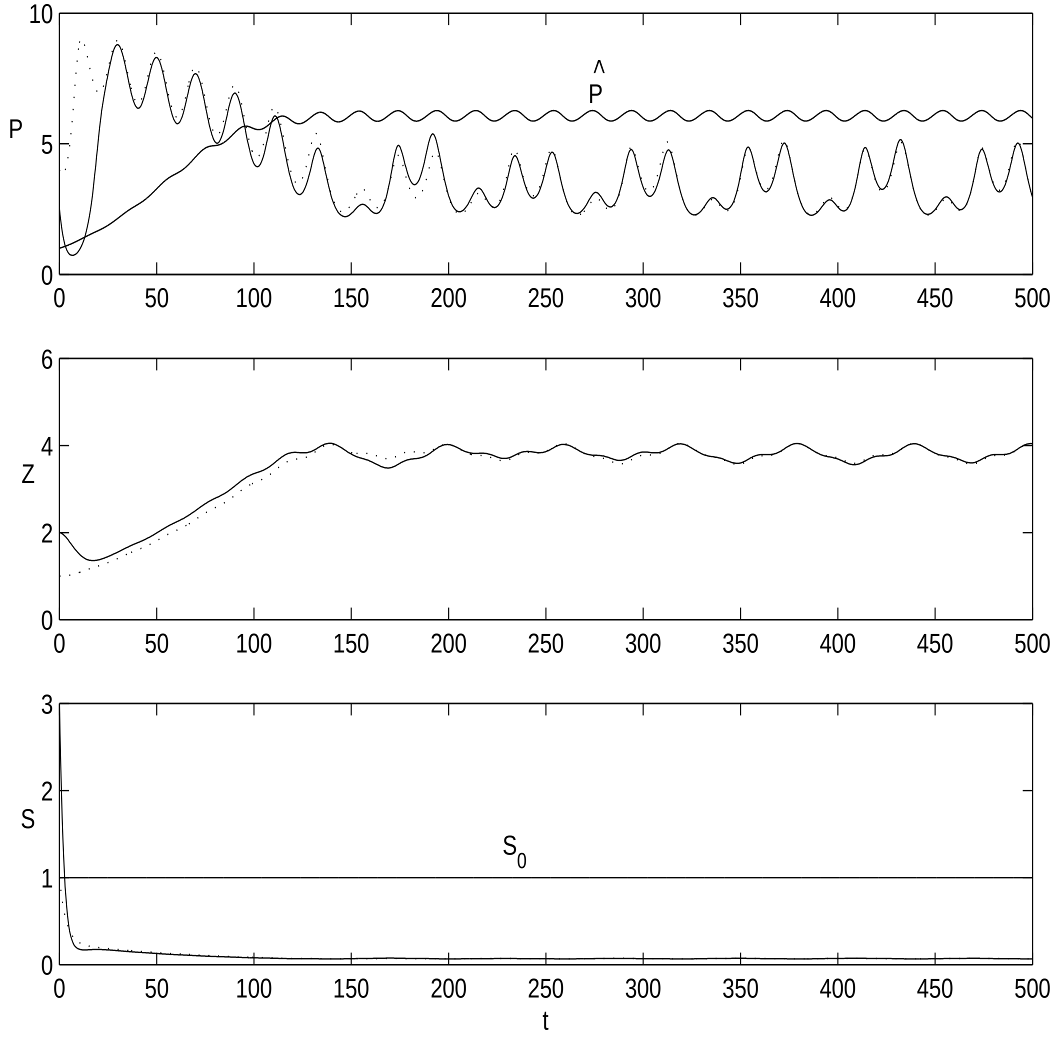

Figure 3: The figure plots three solutions. One solution with initial condition $(1, 0, 1)$ which converges to the periodic solution $(\hat{P}, 0, S^0)$. Another solution (solid curve) using initial condition $(2.5, 2, 3)$ and the other solution (dotted curve) using initial condition $(4, 1, 1)$.

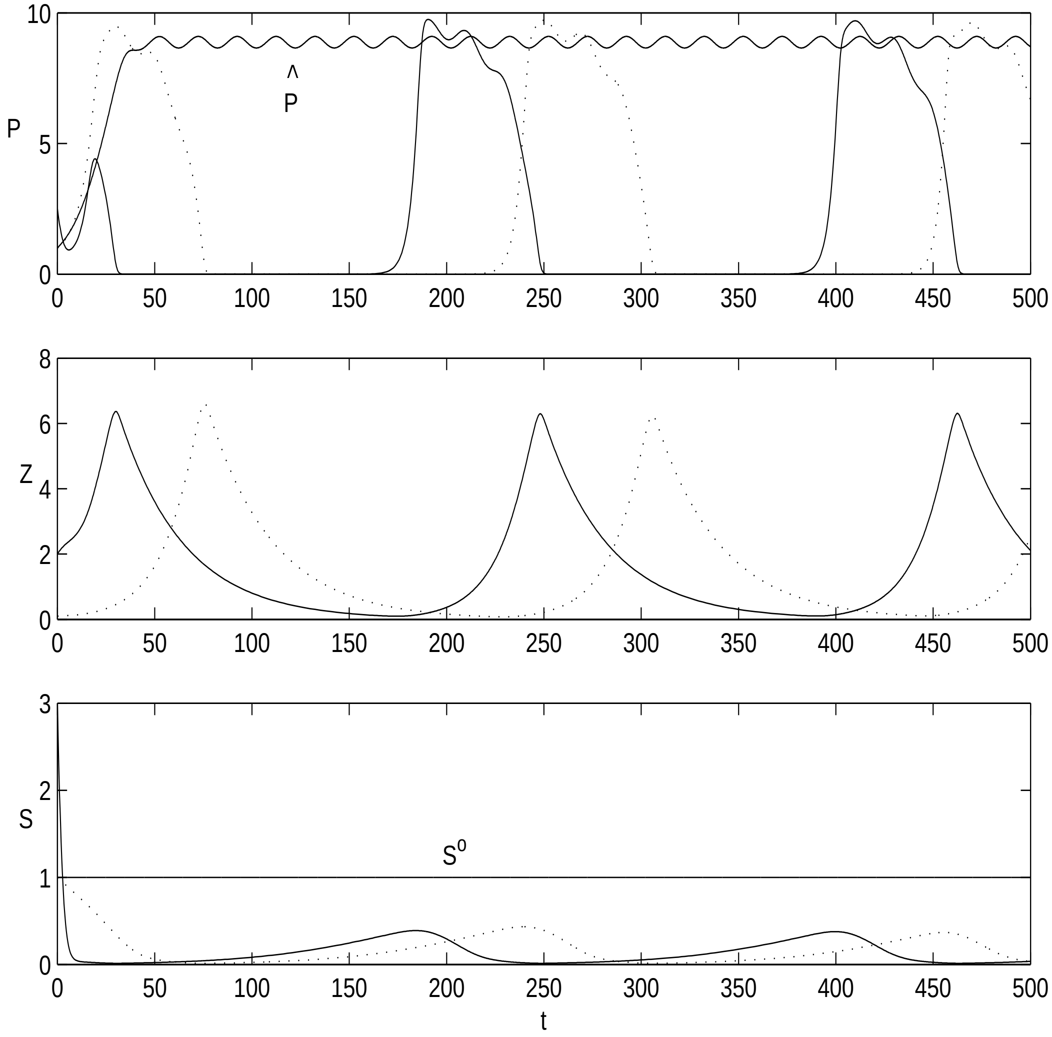

Figure 4: The figure plots two solutions. One solution (solid curve) with initial condition $(2.5, 2, 3)$ and the other solution (dashed curve) using initial condition $(1, 0.1, 1)$.

cycles is assumed to be input periodically. However, the toxin is continuously input to the system with a constant input concentration. There exist population thresholds for the model without the toxin. Both population can coexist if the lumped parameters σ_0 and σ_a are positive. When $\sigma_0 < 0$, then both populations go to extinction. Only phytoplankton can survive if $\sigma_0 > 0$ and $\sigma_1 < 0$.

The introduction of an inhibited substance can alter the dynamical behavior of the plankton interaction unpredictably. The survival and/or extinction of the populations are initial condition dependent. Unlike the model without the toxin, phytoplankton may survive even if $\rho_0 < 0$ which is counter-intuitive as the growth rate of the phytoplankton is diminished due to the toxic substance. Therefore it needs a more delicate ecological study to understand the interaction, especially in the area when phytoplankton population is small but with large concentration of nutrient. It would be interesting to compare the minimum and maximum values of these periodic solutions to the model without the toxin. What happens when the inhibition occurring in the upper trophic level is also worth of pursuing. We leave these questions as another research project to study.

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