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Competition for one nutrient with internal storage and toxin mortality

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ABSTRACT

This study presents a mathematical model of two species competing in a chemostat for one resource that is stored internally, and who also compete through allelopathy. Each species produces a toxin to that increases mortality rate of its competitor. The two species system and its single species subsystem follow mass conservation constraints characteristic of chemostat models. Persistence of a single species occurs if the nutrient supply of an empty habitat allows it to acquire a threshold of stored nutrient quota, sufficient to overcome loss to outflow after accounting for the cost of toxin production. For the two-species system, a semitrivial equilibrium with one species resident is unstable to invasion by the missing species according to a similar threshold condition. The invader increases if acquires a stored nutrient quota sufficient to overcome loss to outflow and toxin-induced mortality, after accounting for the cost of the invader's own toxin production. If both semitrivial equilibria for the two-species system are invasible then there is at least one coexistence equilibrium. Numerical analyses indicate another possibility: bistability in which both semitrivial equilibria are stable against invasion. In such a case there is competitive exclusion of one species, whose identity depends on initial conditions. When there is a tradeoff between abilities to compete for the nutrient and to compete through toxicity, the more toxic species can dominate only under nutrient-rich conditions. Bistability under such conditions could contribute to the unpredictability of toxic algal blooms.

1. Introduction

Competition is an important interaction between species and has been studied by generations of ecologists. The development of mechanistic theory addressing competition for resources was an important advance [13,29]. In the classical theory of competition between algal and microbial species for one nutrient resource, there is a simple generic outcome. The species with the lowest break even nutrient concentration which balances growth and loss rates will exclude all other competitors, independent of initial conditions [17]. Such early versions of resource competition theory assumed a direct relationship between the external concentration of nutrients and the population growth of microbes, without any intermediate steps of nutrient storage within cells. The potential complication of internal storage was soon addressed, with results parallel to those of classical theory: there is only one outcome, dominance by the superior nutrient competitor [25,26]. Outcomes such as coexistence of two or more species, or bistability where outcomes depend on initial conditions do not occur when species compete for one nutrient, with or without internal storage, unless additional factors are introduced such as spatiotemporal inhomogeneity (e.g. [9,10,12,18]) or predators and other natural enemies [30].

The production of toxins that act against competing species, known as allelopathy, is another factor that potentially influences competitive dynamics [6]. A common outcome in mathematical models that combine allelopathy and resource competition is that bistability occurs under some conditions, when there is a tradeoff between ability to compete for the nutrient and resistance to the toxin (e.g. [20,23]). Although available theory addresses some potential complications in the dynamics of allelopathy and resource competition [21], internal storage of the nutrient is as yet unexplored. Important examples of where allelopathy might occur involve toxic algae that produce red tides and similar harmful blooms [8,19]. Such algae compete for nutrients such as phosphorus, nitrogen and iron that are stored within cells [22]. Thus the study of phytoplankton ecology involves both competition for nutrients that are stored internally, and competition through allelopathy.

Motivated by this observation, in this study we weave together the two threads of competition by allelopathy, and internal storage of a nutrient for which competition occurs. We analyze a model in which two species compete for a nutrient that each species consumes and stores. Population growth then depends on the amount of stored nutrient. Each species also produces a toxin that induces mortality in the other species, and each species is immune to the effect of the toxin it produces (i.e. toxin acts between species but not within species). The model represents the dynamics of the two species, their amounts of internally stored nutrient, the

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concentrations of the toxins they produce, and the concentration of the growth-limiting nutrient. The interactions among these components are studied in a simple model environment, the well-mixed chemostat [26]. In this habitat, the growth-limiting nutrient is supplied at a constant concentration $R^{(0)}$ flowing in at a constant dilution rate, with a balancing outflow that removes all constituents. The chemostat serves as a simple model of natural habitats such as lakes and estuaries, and is also the basis for many elaborations involving spatial or temporal inhomogeneity, or predators and other natural enemies. But we ignore such complications here and assume the environment is constant, uniform, and lacks other species interacting with the focal competitors.

The organization of the paper is as follows. The mathematical model is described in the next section. In Section 3, we first consider the single population model and state a sharp threshold result distinguishing between washout of the organism from the reactor and persistence of the population. Section 4 is devoted to the study of two competing species model. Necessary conditions are stated for equilibrium coexistence of the two species model. Simulation results are collected in Section 5 where competitive exclusion and bistability (outcomes dependent on initial conditions) are observed. Biological interpretations are presented in Section 6. Section A is the Appendix section. Some technical proofs are collected in this section.

2. The model

The basic model and appropriate hypotheses are stated in this section. Consider two populations competing for a single nutrient in the chemostat. The chemostat is supplied with nutrient at a constant concentration $R^{(0)}$ at dilution rate D. There is a compensating outflow also at rate D of the well-stirred contents of the chemostat. Let $R(t)$ be the nutrient concentration at time t; $N_i(t)$ be the concentrations of species i in the culture vessel respectively, and $P_i(t)$ be the concentration of the inhibitor produced by ith population. For $i = 1,2$, $Q_i(t)$ represents the average amount of stored nutrient per cell of ith population at time t. Then we consider the following system:

$$\begin{cases} \frac{dR}{dt} = (R^{(0)} - R)D - \rho_1(R,Q_1)N_1 - \rho_2(R,Q_2)N_2 + m_{12}(P_2)N_1Q_1 + m_{21}(P_1)N_2Q_2, \\ \frac{dN_1}{dt} = [(1-k_1)\mu_1(Q_1) - m_{12}(P_2) - D]N_1, \\ \frac{dQ_1}{dt} = \rho_1(R,Q_1) - (1-k_1)\mu_1(Q_1)Q_1, \\ \frac{dN_2}{dt} = [(1-k_2)\mu_2(Q_2) - m_{21}(P_1) - D]N_2, \\ \frac{dQ_2}{dt} = \rho_2(R,Q_2) - (1-k_2)\mu_2(Q_2)Q_2, \\ \frac{dP_1}{dt} = k_1\mu_1(Q_1)N_1 - DP_1, \\ \frac{dP_2}{dt} = k_2\mu_2(Q_2)N_2 - DP_2, \\ R(0) \geqslant 0,\ P_i(0) \geqslant 0,\ N_i(0) \geqslant 0,\ Q_i(0) \geqslant Q_{\min,i},\ i = 1,2, \end{cases} \tag{2.1}$$

where $\mu_i(Q_i)$ is the growth rate of species i as a function of cell quota Q_i, $\rho_i(R,Q_i)$ is the per capita nutrient uptake rate, per cell of species i as a function of nutrient concentration R and cell quota Q_i, $Q_{\min,i}$ denotes the threshold cell quota below which no growth of species i occurs. The functions $m_{ij}(P_j)$ describe the mortality effect on species i from the toxin produced by species j. The constant k_i is the fraction of consumption devoted to the production of the inhibitor. Hence, $0 < k_i < 1$. In the first equation of system (2.1), the nutrient content of algal cells killed by inhibitors appears as a positive term, corresponding to the assumption that this nutrient content is recycled instantaneously. Algal toxins that induce mortality often act by disrupting cell membranes and releasing cell contents to the surrounding water [8], so it is reasonable to assume that relatively rapid recycling occurs, even if it is not instantaneous. Assuming instantaneous recycling facilitates the application of a conservation principle to construct limiting systems in Sections 3 and 4.

The functions $m_{ij}(P_j)$ satisfy

$$m_{ij}(P_j) \geqslant 0, \quad m_{ij}(0) = 0 \quad \text{and } m'_{ij}(P_j) > 0 \quad \forall P_j \geqslant 0. \tag{2.2}$$

The growth rate $\mu_i(Q_i)$ takes the forms [3–5]:

$$\mu_i(Q_i) = \mu_{i\infty}\left(1 - \frac{Q_{\min,i}}{Q_i}\right) \tag{2.3}$$

or

$$\mu_i(Q_i) = \mu_{i\infty}\frac{(Q_i - Q_{\min,i})_+}{a_i + (Q_i - Q_{\min,i})_+},$$

where $(Q_i - Q_{\min,i})_+$ is the positive part of $(Q_i - Q_{\min,i})$ and $\mu_{i\infty}$ is the maximal growth rate of the species.

According to [12,22], the uptake rate $\rho_i(R,Q_i)$ takes the form:

$$\begin{aligned} &\rho_i(R,Q_i) = \rho_{\max,i}(Q_i)\frac{R}{K_i + R}, \\ &\rho_{\max,i}(Q_i) = \rho_{\max}^{i\text{high}} - (\rho_{\max}^{i\text{high}} - \rho_{\max}^{i\text{low}})\frac{Q_i - Q_{\min,i}}{Q_{\max,i} - Q_{\min,i}}, \end{aligned} \tag{2.4}$$

where $Q_{\min,i} \leqslant Q_i \leqslant Q_{\max,i}$. Cunningham and Nisbet [3,4] took $\rho_{\max,i}(Q_i)$ to be a constant.

Motivated by these examples, we assume that $\mu_i(Q_i)$ is defined and continuously differentiable for $Q_i \geqslant Q_{\min,i} > 0$ and satisfies

$$\begin{aligned} &\mu_i(Q_i) \geqslant 0, \quad \mu'_i(Q_i) > 0 \quad \text{and is continuous for } Q_i \geqslant Q_{\min,i}, \\ &\mu_i(Q_{\min,i}) = 0. \end{aligned} \tag{2.5}$$

We assume that $\rho_i(R,Q_i)$ is continuously differentiable for $R > 0$ and $Q_i \geqslant Q_{\min,i}$ and satisfies

$$\rho_i(0,Q_i) = 0, \quad \frac{\partial \rho_i}{\partial R} > 0, \quad \frac{\partial \rho_i}{\partial Q_i} \leqslant 0. \tag{2.6}$$

In particular, $\rho_i(R,Q_i) > 0$ when $R > 0$.

To end this section, we briefly mention the following Droop model without toxins studied by Smith and Waltman [26,27]:

$$\begin{cases} \frac{dR}{dt} = (R^{(0)} - R)D - \rho_1(R,Q_1)N_1 - \rho_2(R,Q_2)N_2, \\ \frac{dN_1}{dt} = [\mu_1(Q_1) - D]N_1, \\ \frac{dQ_1}{dt} = \rho_1(R,Q_1) - \mu_1(Q_1)Q_1, \\ \frac{dN_2}{dt} = [\mu_2(Q_2) - D]N_2, \\ \frac{dQ_2}{dt} = \rho_2(R,Q_2) - \mu_2(Q_2)Q_2, \\ R(0) \geqslant 0, \quad N_i(0) \geqslant 0, \quad Q_i(0) \geqslant Q_{\min,i}, \quad i = 1,2. \end{cases} \tag{2.7}$$

Note that if we let $k_1 = k_2 = 0$ and $P_1 = P_2 \equiv 0$ in the system (2.1), it is easy to see that the system (2.1) becomes the one (2.7). In [26,27], Smith and Waltman proved that competitive exclusion holds for the system (2.7), that is, the species that can grow at the lowest nutrient concentration will win the competition.

3. Single population model

In this section, we first consider the single population model corresponding to (2.1), that is,

$$\begin{cases} \frac{dR}{dt} = (R^{(0)} - R)D - \rho(R,Q)N, \\ \frac{dN}{dt} = [(1-k)\mu(Q) - D]N, \\ \frac{dQ}{dt} = \rho(R,Q) - (1-k)\mu(Q)Q, \\ \frac{dP}{dt} = k\mu(Q)N - DP, \\ R(0) \geqslant 0, \quad N(0) \geqslant 0, \quad Q(0) \geqslant Q_{\min}, \quad P(0) \geqslant 0. \end{cases} \tag{3.1}$$

with initial values in the domain

$$\mathbf{X} = \{(R,N,Q,P) \in \mathbb{R}^4_+ : Q \geqslant Q_{\min}\}. \tag{3.2}$$

It is easy to show that $\mathbf{X}$ is positively invariant for the system (3.1).