THE IMPACT OF TOXINS ON COMPETITION DYNAMICS OF THREE SPECIES IN A POLLUTED AQUATIC ENVIRONMENT

YUYUE ZHANG AND JICAI HUANG

School of Mathematics and Statistics
Central China Normal University
Wuhan, Hubei 430079, China

QIHUA HUANG∗

School of Mathematical and Statistical Sciences
Southwest University
Chongqing 400715, China
School of Mathematical and Statistical Sciences
Hubei University of Science and Technology
Xianning 437100, China

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Abstract. Accurately assessing the risks of toxins in polluted ecosystems and finding factors that determine population persistence and extirpation are important from both environmental and conservation perspectives. In this paper, we develop and study a toxin-mediated competition model for three species that live in the same polluted aquatic environment and compete for the same resources. Analytical analysis of positive invariance, existence and stability of equilibria, sensitivity of equilibria to toxin are presented. Bifurcation analysis is used to understand how the environmental toxins, plus distinct vulnerabilities of three species to toxins, affect the competition outcomes. Our results reveal that while high concentrations lead to extirpation of all species, sublethal levels of toxins affect competition outcomes in many counterintuitive ways, which include boosting coexistence of species by reducing the abundance of the predominant species, inducing many different types of bistability and even tristability, generating and reducing population oscillations, and exchanging roles of winner and loser in competition. The findings in this work provide a sound theoretical foundation for understanding and assessing population or community effects of toxicity.

1. Introduction. Pollution in aquatic environments, the release of harmful substances, such as heavy metals, pesticides, and other chemicals, into streams, rivers, lakes, oceans, or other bodies of water, is one of the world’s primary health concerns. Toxins in pollutants pose serious threats to all levels of biological hierarchy,
ranging from cells to organs, organisms, communities, and entire ecosystems. Accu-
rately assessing the risks of toxins in ecosystems and finding factors that determine
population persistence and extirpation are important from both environmental and
conservation perspectives. To protect aquatic ecosystems, a number of researchers
have suggested environmental quality standards and effective control measures for
minimizing pollution in aquatic environments \([2, 3, 6, 12, 26, 30, 32, 36]\).

Environment toxicology, which predicts the environmental and ecological im-
pacts of environmental pollutants, has expanded rapidly during the past several
decades. At the same time, mathematical models play significant roles in chem-
ical risk assessments on a variety of ecological processes. These models include
individual-based models, matrix population models, differential equation models,
etc. A comprehensive review on the selection, realism, relevant, and applicability
of different types of models from the perspective of assessing risks posed by toxic
chemicals can be found in \([1, 24]\). Over the past several decades, some (impulsive)
differential equation models \([9, 10, 11, 18, 20]\) have been developed to study the
dynamical nature of population-toxicant interactions in a polluted aquatic environ-
ment. These models usually involve three state variables that respectively represent
the population biomass, the concentration of toxin in the population, and the con-
centration of toxin in the environment. Based on the fact that the concentration
of toxin in the environment is not significantly affected by the population growth,
mortality, or metabolic process, Huang et al. \([15]\) developed a body-burden de-
pendent population model, which ignores the effect of the population on the toxin
in the environment, to study the impact of environmental toxins on fish popula-
tion dynamics. This is reasonable because the toxin contained in the population
is tiny portion of the total environment. Thus, the concentration of toxin in the
environment was regarded as an important parameter, instead of a state variable.

It is remarkable that the above-mentioned differential equation models are single-
species models, which cannot be used to understand the impact of toxins on species
interactions, nutrient cycling, and trophic transfer of toxins. To investigate how
the transfer of toxins between trophic levels and bioaccumulation process affect
food-web dynamics, Huang et al. \([16]\) developed a toxin-mediated predator-prey
model that tracks the prey and predator densities, as well as the toxin body burden
in each population. The model was used to shed light on the effects of varying
toxin concentration on long-term dynamics of organisms at different trophic levels.
Recently, in order to further study the impact of environmental toxins on population
interactions, Shan and Huang \([25]\) developed a toxin-dependent competition model
for two species that are simultaneously exposed to a polluted aquatic environment.
The model was then utilized to scrutinize how the interplay between direct and
indirect effects of environmental toxins determines competition outcomes of two
species.

The main purpose of this work is to study how environmental toxins affect the
competition dynamics of three species in a polluted aquatic ecosystem. To this
end, we develop a toxin-mediated competition model which extends the two-species
competition model by Shan and Huang \([25]\). Our model is composed of six ordi-
nary differential equations. The first three equations describe the rates of change
of population biomass of three species competing for the same resources, in which
the reproduction and mortality rates are explicit functions of their body burdens.
Body burden, also referred to as tissue residue, is a direct measurement of toxin
concentration in the tissue or organism, instead of in the exposure media \([21]\). The
other three equations are the balance equations for the body burdens of the three species, which describe the accumulation and the elimination of toxin in the organism tissue. The resulting system is difficult to deal with due to its high dimension. To make the model tractable both analytically and numerically, we investigate the toxin-mediated competition dynamics on its slow manifold in terms of geometrical singular perturbation theory. In other words, we simplify the six-dimensional system to a three-dimensional system via a quasi-steady state approximation. In the absence of toxin exposure, our toxin-mediated model reduces to a classical Lotka-Volterra (LV) competition population model, whose dynamics have been well studied (e.g., [13, 17, 35]). We are interested in the following questions: (1) How the classical LV competition dynamics of three species will change when the toxin level in the environment varies from zero to higher levels? (2) How the distinct vulnerabilities of the three species to toxins affect competitive outcomes? We answer these questions through stability theory and bifurcation analysis. Our results reveal that the relatively low level of toxins, plus different vulnerabilities of the three species to toxins, impact competition outcomes in variable counterintuitive ways.

The rest of the paper is organized as follows. In Section 2, we formulate a body burden-dependent competition model for three species that are simultaneously exposed to environmental toxins. In Section 3, we nondimensionalize the competition model, reduce the model to its slow manifold by geometrical singular perturbation theory, and investigate the dynamics of the reduced model. In Section 4, we scrutinize how different toxin levels in the environment and population’s sensitivities to toxin affect the long-term behavior of the three dimensional competition dynamics. Finally, in Section 5, the paper ends with a brief discussion about the ecological interpretations of our results and suggestions about the future research direction.

2. The model formulation. We consider three species, say species 1, species 2, and species 3, which compete for the same resources in an aquatic environment. We use \( x_i(t) \) \((i = 1, 2, 3)\) to represent the concentration of biomass of species at time \( t \). Let \( u_i \) be the concentration of toxin in species \( i \) at time \( t \). Let \( y_i(t) \) be the body burden of species \( i \). See section 2 in [25] for more precise definitions of \( x_i, u_i, \) and \( y_i \).

The mathematical model that describes the toxin-mediated competition dynamics of the three species in a polluted aquatic environment is given by

\[
\frac{dx_i}{dt} = \left(b_i \max\{0, 1 - \beta_i y_i\} - k_i y_i - m_i\right)x_i - c_{ii}x_i^2 - \sum_{j \neq i} c_{ij}x_ix_j, \quad (1a)
\]

\[
\frac{du_i}{dt} = a_i T x_i - c_i u_i - \left(k_i y_i + m_i + c_{ii}x_i + \sum_{j \neq i} c_{ij}x_j\right)u_i, \quad (1b)
\]

\[
y_i = \frac{u_i}{x_i}, \quad i, j = 1, 2, 3, \quad (1c)
\]

where the parameters, \( b_i, \beta_i, k_i, m_i, c_{ij}, a_i, c_i, \) and \( T \), are all positive constants.

Equation (1a) presents the rate of change of the species’ biomass under the influence of the toxin. The term \( b_i \max\{0, 1 - \beta_i y_i\} \) describes the population gain rate that linearly depends on the body burden \( y_i \), where \( b_i \) is the maximum gain rate and \( \beta_i \) denotes the effect coefficient of toxin on the gain rate of population. If there is no toxic effect (i.e., body burden \( y_i = 0 \)), then the gain rate of species biomass is \( b_i \). If the body burden \( y_i \) reaches a threshold level \( 1/\beta_i \), the gain rate of species biomass is 0 (i.e., the species stops reproduction and growth). The term
$k_i y_i + m_i$ means that the population mortality rate linearly depends on the body burden $y_i$, where $k_i$ represents the effect coefficient of toxin on mortality, and $m_i$ is the natural mortality rate. The term $c_{ii} x_i^2$ describes the loss rate of species’ biomass due to intraspecific competition, and $c_{ij} x_i x_j$ $(j \neq i)$ describe the loss rate of species’ biomass due to interspecific competitions, where $c_{ii}$ and $c_{ij}$ are the competition coefficients.

Equation (1b) is a balance equation for the concentration of toxin contained in the species $i$. The term $a_i T x_i$ represents the toxin uptake rate by the population from the environment, which is proportional to both the concentration of toxin in the environment $T$ and the concentration of species biomass (i.e., Law of Mass Action), where $a_i$ is the uptake coefficient. The positive constant $e_i$ is per unit rate of toxin elimination due to metabolic processes of the population. The term $(k_i y_i + m_i + c_{ii} x_i + \sum_{j \neq i} c_{ij} x_j) u_i$ describes the loss of toxin in the population due to mortality (including toxin-dependent mortality and competition mortality). (Note that the two-species competition model studied in [25] ignores the loss of toxin contained in the population due to death from competition.) Finally, equation (1c) defines the body burden — toxin concentration per unit species biomass.

We see from (1a) that the direct influences of toxin on the population growth are realized through the body burdens $y_i$. Using (1a) and (1b), we are able to obtain an equation with respect to the rate of change of $y_i$. This will convert model (1) including nine equations into an equivalent system including six equations. Differentiating (1c) with respect to $t$ and using (1a) and (1b), we obtain

$$\frac{dy_i}{dt} = a_i T - (e_i + b_i \max\{0, 1 - \beta_i y_i\}) y_i.$$ 

Thus, in this work we study the following body burden-dependent competition model for three species:

$$\frac{dx_i}{dt} = b_i \max\{0, 1 - \beta_i y_i\} x_i - (k_i y_i + m_i) x_i - c_{ii} x_i^2 - \sum_{j \neq i} c_{ij} x_i x_j,$$

$$\frac{dy_i}{dt} = a_i T - e_i y_i - b_i \max\{0, 1 - \beta_i y_i\} y_i.$$ 

where $i, j = 1, 2, 3$.

3. **Model analysis.** In reality, the dynamics of toxin uptake and elimination due to metabolism operate on a much faster time scale than the dynamics of population biomass growth. This means that the body burden equations in (2) may approach a quasi-equilibrium state where uptake of toxin and elimination balance out on a fast time scale. To investigate this process mathematically we introduce $\epsilon = b_1/e_1$ where $0 < \epsilon \ll 1$. 


3.1. Nondimensionalization and nonnegativity. To simplify the problem and facilitate analysis, we nondimensionalize system (2) by introducing the following dimensionless quantities:

\[
\bar{x}_i = \frac{c_i}{b_i} x_i, \quad \bar{y}_i = \beta_i y_i \quad \bar{t} = b_1 t, \quad \bar{x}_i = \frac{k_i}{b_1 \beta_i}, \quad \bar{m}_i = \frac{m_i}{b_1}, \quad \bar{c}_{ij} = \frac{b_j c_{ij}}{b_1 c_{jj}} \quad (j \neq i),
\]

\[
\bar{T} = \frac{\beta_1 a_1 T}{\epsilon T}, \quad \bar{\epsilon} = \frac{1}{e_1}, \quad \bar{c}_i = \frac{c_i}{e_1}, \quad \bar{b}_i = \frac{b_i}{b_1}, \quad \bar{a}_i = \frac{\beta_i a_i}{\beta_1 a_1}
\]

Dropping the tildes for notational simplicity, system (2) reduces to

\[
\frac{dx_i}{dt} = (b_i \max\{0, 1 - y_i\} - k_i y_i - m_i) x_i - b_i x_i^2 - \sum_{j \neq i} c_{ij} x_i x_j, \quad (3a)
\]

\[
\epsilon \frac{dy_i}{dt} = a_i T - \epsilon_i y_i - \epsilon b_i \max\{0, 1 - y_i\} y_i, \quad i = 1, 2, 3, \quad (3b)
\]

where \( e_1 = b_1 = a_1 = 1 \).

The following theorem indicates that the solutions of system (3) behave in a biologically reasonable manner. That is, the species biomass \( x_i \) and the body burdens \( y_i \) at any time are always nonnegative and eventually bounded, as long as they are nonnegative at initial time.

**Theorem 3.1.** Any solutions of system (3) with initial conditions \( x_i(0) \geq 0, y_i(0) \geq 0 \) \((i = 1, 2, 3)\) will approach, enter and stay in the region

\[
\Omega = \left\{ (x_1, x_2, x_3, y_1, y_2, y_3) \mid 0 \leq x_i \leq 1, \quad 0 \leq y_i \leq \frac{a_i T}{e_i}, \quad i = 1, 2, 3 \right\}.
\]

**Proof.** The local existence and uniqueness of the solution are guaranteed by the local Lipschitz continuity of system (3). Note that

\[
\left. \frac{dx_i}{dt} \right|_{x_i=0} = 0, \quad \left. \frac{dy_i}{dt} \right|_{y_i=0} = \frac{a_i T}{\epsilon} > 0, \quad i = 1, 2, 3,
\]

so the positive cone of \( \mathbb{R}^6 \) is a positive invariant set of system (3). In this cone, we have

\[
\frac{dx_i}{dt} \leq (b_i - b_i x_i) x_i, \quad i = 1, 2, 3,
\]

so that the standard comparison principle implies that

\[
0 \leq \limsup_{t \to \infty} x_i(t) \leq 1, \quad i = 1, 2, 3.
\]

Similarly, we have

\[
\epsilon \frac{dy_i}{dt} \leq a_i T - \epsilon_i y_i, \quad i = 1, 2, 3,
\]

so that the standard comparison principle implies that

\[
0 \leq \limsup_{t \to \infty} y_i(t) \leq \frac{a_i T}{\epsilon_i}, \quad i = 1, 2, 3.
\]

We define quantities \( p_i := a_i / \epsilon_i, \quad i = 1, 2, 3 \). In terms of the original dimensional parameters, \( p_i \) is given by

\[
p_i = \frac{\beta_i}{\beta_1} \cdot \frac{a_i}{a_1} \cdot \frac{e_1}{\epsilon_i}.
\]

Clearly, \( p_1 = 1 \). For \( i = 2, 3 \), the quantities \( p_i \) compare the sensitivity to toxin of species \( i \) to toxin with that of species 1. More precisely, \( \beta_i > \beta_1, \quad a_i > a_1, \) and
\( e_i < e_1 \) mean that the negative effects of toxin on species \( i \) are stronger than those on species 1. Therefore, if \( p_i > 1 \) \((p_i < 1)\), the negative effects of toxin on species \( i \) are stronger (weaker) than those on species 1. We henceforth refer to \( p_i \) as relative sensitivity.

Throughout this paper, we assume that

\[
m_i < b_i, \quad i = 1, 2, 3, \tag{5}\]

for system (2), these mean that the natural loss rates of the population biomass due to death are less than their maximum gain rates due to growth. For instance, if the condition \( m_1 < b_1 \) is violated, species 1 cannot persist in the environment even without the negative effect of toxin.

We also assume that

\[
T < \min \left\{ \frac{1}{p_1}, \frac{1}{p_2}, \frac{1}{p_3} \right\} = \min \left\{ 1, \frac{1}{p_2}, \frac{1}{p_3} \right\}. \tag{6}\]

Then, from Theorem 3.1, we see that \( 0 < y_i < 1 \). In fact, if \( y_i \geq 1 \), then for any initial value \( x_i(0) \geq 0 \), \( dx_i/dt < 0 \), thus \( \lim_{t \to \infty} x_i(t) = 0 \), and system (3) reduces to a two-species competition model. With assumption (6), system (3) becomes

\[
\frac{dx_i}{dt} = [b_i(1 - y_i) - k_i y_i - m_i] x_i - b_i x_i^2 - \sum_{j \neq i} c_{ij} x_i x_j, \tag{7a}\]

\[
e^\epsilon \frac{dy_i}{dt} = a_i T - e_i y_i - \epsilon b_i(1 - y_i) y_i, \quad i = 1, 2, 3, \tag{7b}\]

where \( e_1 = b_1 = a_1 = 1. \)

3.2. Fast-slow systems and slow manifold. For \( 0 < \epsilon \ll 1 \), system (7) is called a slow system, where \( x_1, x_2, \) and \( x_3 \) are slow variables, while \( y_1, y_2, \) and \( y_3 \) are fast variables. By rescaling the time \( \tau = t/\epsilon \), we will obtain the corresponding fast system which is topological equivalent to the slow system (7) for \( \epsilon \neq 0 \). Letting \( \epsilon \to 0 \) in the fast system, we obtain a set of equilibrium points for this limiting fast system

\[
\mathcal{M}^0 = \left\{ (x_1, x_2, x_3, y_1, y_2, y_3) \in \Omega \mid y_1 = \frac{a_1}{e_1} T, \; y_2 = \frac{a_2}{e_2} T, \; y_3 = \frac{a_3}{e_3} T \right\},
\]

and \( \mathcal{M}^0 \) is called the slow manifold of system (7).

**Theorem 3.2.** The slow manifold \( \mathcal{M}^0 \) is a normally hyperbolic stable manifold which is persistent for \( 0 < \epsilon \ll 1 \). That is, for \( \epsilon > 0 \) small, there exist a stable invariant manifold \( \mathcal{M}^\epsilon \) for system (7), so that \( \mathcal{M}^\epsilon \) is homeomorphic to \( \mathcal{M}^0 \) and \( \mathcal{M}^\epsilon \to \mathcal{M}^0 \) as \( \epsilon \to 0 \).

**Proof.** The eigenvalues of the linearization of the limiting fast system at any point of \( \mathcal{M}^0 \), other than the three zero eigenvalues, are \(-e_1, -e_2, -e_3\). Hence, \( \mathcal{M}^0 \) is a normally hyperbolic manifold. By Fenichel’s Invariant Manifold Theorem (see Fenichel [4], Fenichel [5]), the slow system (7) has an invariant manifold

\[
\mathcal{M}^\epsilon = \left\{ (x_1, x_2, x_3, y_1, y_2, y_3) \in \Omega \mid y_1 = p_1 T + \mathcal{O}(\epsilon), \; y_2 = p_2 T + \mathcal{O}(\epsilon), \; y_3 = p_3 T + \mathcal{O}(\epsilon) \right\}
\]

(where \( p_i = a_i/e_i, i = 1, 2, 3 \)) with respect to the slow flow for \( 0 < \epsilon \ll 1 \), and is homeomorphic to \( \mathcal{M}^0 \). Furthermore, \( \mathcal{M}^\epsilon \) is an attracting manifold, and all orbits of the slow system (7) will approach \( \mathcal{M}^\epsilon \) exponentially fast as \( t \to \infty \).
By Theorem 3.2, $\mathcal{M}^c$ is attracting and invariant, and dynamics of system (7) on $\mathcal{M}^c$ are regular perturbations of the dynamics of the limiting slow system on the three-dimensional slow manifold $\mathcal{M}^0$, i.e.,
\[
\begin{align*}
\frac{dx_1}{dt} &= x_1 \left[ b_1 (1 - p_i T) - k_1 p_i T - m_1 - b_1 x_1 - c_{12} x_2 - c_{13} x_3 \right], \\
\frac{dx_2}{dt} &= x_2 \left[ b_2 (1 - p_2 T) - k_2 p_2 T - m_2 - b_2 x_2 - c_{21} x_1 - c_{23} x_3 \right], \\
\frac{dx_3}{dt} &= x_3 \left[ b_3 (1 - p_3 T) - k_3 p_3 T - m_3 - b_3 x_3 - c_{31} x_1 - c_{32} x_2 \right],
\end{align*}
\]
where the parameters $b_i$, $p_i$, $T$, $k_i$, $m_i$, $c_{ij}$ ($i, j = 1, 2, 3$) are all positive, and $b_1 = p_1 = 1$. So dynamics of system (7) can be fully characterized by the dynamics of system (8). For this reason, we restrict our main analysis to system (8) and examine the effect of environmental toxins on competition dynamics of three species.

### 3.3. Existence and stability of equilibria

#### 3.3.1. Boundary equilibria

By straightforward calculation, we find that for all admissible parameters, system (8) possesses one trivial equilibrium $E_0(0, 0, 0)$ and at most six semi-trivial equilibria:
\[
E_1 \left( \frac{n_1}{b_1}, 0, 0 \right), \ E_2 \left( 0, \frac{n_2}{b_2}, 0 \right), \ E_3 \left( 0, 0, \frac{n_3}{b_3} \right), \ E_{12} \left( \frac{b_2 n_1 - c_{12} n_2}{b_1 b_2 - c_{12} c_{21}}, \frac{b_1 n_2 - c_{21} n_1}{b_1 b_2 - c_{12} c_{21}}, 0 \right), \ E_{13} \left( \frac{b_3 n_1 - c_{13} n_3}{b_1 b_3 - c_{13} c_{31}}, 0, \frac{b_1 n_3 - c_{31} n_1}{b_1 b_3 - c_{13} c_{31}} \right), \ E_{23} \left( 0, \frac{b_2 n_2 - c_{23} n_2}{b_2 b_3 - c_{23} c_{32}}, \frac{b_2 n_2 - c_{23} n_2}{b_2 b_3 - c_{23} c_{32}} \right),
\]
where
\[
n_i = b_i (1 - p_i T) - k_i p_i T - m_i, \ i = 1, 2, 3.
\]
The species $i$-only equilibrium $E_i$ exists if and only if
\[
T < \frac{b_i - m_i}{p_i (b_i + k_i)} := T_i^*, \ i = 1, 2, 3.
\]
The semitrivial equilibrium $E_{ij}$, at which only species $i$ and species $j$ coexist, exists if and only if
\[
T < \min \{T_i^*, T_j^*\} \text{ and } (b_i n_i - c_{ij} n_j) (b_j n_j - c_{ji} n_i) > 0, \ i, j = 1, 2, 3, i < j,
\]
which is equivalent to that the terms $b_i b_j - c_{ij} c_{ji}$, $b_j n_i - c_{ij} n_j$, and $b_j n_j - c_{ji} n_i$ ($i, j = 1, 2, 3, i < j$) have the same sign.

**Remark 1.** $T_i^*$, defined in (10), is the critical environmental toxin level that the species $i$ can persist. If $T > T_i^*$, then for any $x_i(0) \geq 0$, $dx_i/dt < 0$, thus $\lim_{t \to \infty} x_i(t) = 0$, system (8) hence reduces to standard two-dimensional competition Lotka-Volterra (LV) system. It is well known that such a two-dimensional system has no closed orbit, and that the positive equilibrium is globally asymptotically stable if it is locally asymptotically stable.
Next, we consider the local stability of the boundary equilibria, by direct computation, the Jacobian matrix of system (8) at any equilibrium has the form

\[ J = \begin{bmatrix}
  J_{11} & -c_{12}x_1 & -c_{13}x_1 \\
  -c_{21}x_2 & J_{22} & -c_{23}x_2 \\
  -c_{31}x_3 & -c_{32}x_3 & J_{33}
\end{bmatrix}, \tag{12} \]

where \( J_{11} = n_1 - 2b_1x_1 - c_{12}x_2 - c_{13}x_3, \ J_{22} = n_2 - 2b_2x_2 - c_{21}x_1 - c_{23}x_3, \) and \( J_{33} = n_3 - 2b_3x_3 - c_{31}x_1 - c_{32}x_2. \)

**Theorem 3.3.** The extinction equilibrium \( E_0(0, 0, 0), \) at which all species go extinct, is

(i) An unstable node if \( T < \min \{ T_1^*, T_2^*, T_3^* \}; \)

(ii) A saddle if \( \min \{ T_1^*, T_2^*, T_3^* \} < T < \max \{ T_1^*, T_2^*, T_3^* \}; \)

(iii) A stable node if \( T > \max \{ T_1^*, T_2^*, T_3^* \}. \)

**Proof.** From (12), the Jacobian matrix evaluated at \( E_0 \) is

\[ J(E_0) = \begin{bmatrix}
  n_1 & 0 & 0 \\
  0 & n_2 & 0 \\
  0 & 0 & n_3
\end{bmatrix}, \]

and the eigenvalues are \( \lambda_1 = n_1, \lambda_2 = n_2, \lambda_3 = n_3. \) (i) When \( T < \min \{ T_1^*, T_2^*, T_3^* \}, \) all eigenvalues are positive, so \( E_0 \) is an unstable node. (ii) When \( \min \{ T_1^*, T_2^*, T_3^* \} < T < \max \{ T_1^*, T_2^*, T_3^* \}, \) the signs of three eigenvalues are not all the same, \( E_0 \) is a saddle. (iii) When \( T > \max \{ T_1^*, T_2^*, T_3^* \}, \) \( E_0 \) is a stable node since all eigenvalues are negative. \( \square \)

To discuss the local stability of semi-trivial equilibria, for convenience, we introduce the following notations:

\[
\begin{align*}
\gamma_{12} & := b_1n_2 - c_{21}n_1, \quad \gamma_{13} := b_1n_3 - c_{31}n_1, \quad \gamma_{21} := b_2n_1 - c_{12}n_2, \\
\gamma_{23} & := b_2n_3 - c_{32}n_2, \quad \gamma_{31} := b_3n_1 - c_{13}n_3, \quad \gamma_{32} := b_3n_2 - c_{23}n_3, \\
\beta_{12} & := b_1b_2 - c_{12}c_{21}, \quad \beta_{13} := b_1b_3 - c_{13}c_{31}, \quad \beta_{23} := b_2b_3 - c_{23}c_{32}, \\
\Delta_1 & := n_1\beta_{23} - c_{12}\gamma_{23}, \\
\Delta_2 & := n_2\beta_{13} - c_{13}\gamma_{13}, \\
\Delta_3 & := n_3\beta_{12} - c_{23}\gamma_{32},
\end{align*}
\]

from which we can rewrite the semi-trivial equilibrium as

\[ E_{12} \left( \frac{\gamma_{21}}{\beta_{12}}, \frac{\gamma_{12}}{\beta_{12}}, 0 \right), \quad E_{13} \left( 0, \frac{\gamma_{13}}{\beta_{13}}, \frac{\gamma_{31}}{\beta_{13}} \right), \quad E_{23} \left( 0, \frac{\gamma_{32}}{\beta_{23}}, \frac{\gamma_{23}}{\beta_{23}} \right). \]

Then we have the following results on the local stability of the semi-trivial equilibria of system (8).

**Theorem 3.4.** (i) For fixed \( i \in \{ 1, 2, 3 \}, \) the species \( i \)-only equilibrium \( E_i \) is locally asymptotically stable in \( R^3 \) if and only if

\[ \gamma_{ij} < 0, \quad j = 1, 2, 3, \quad j \neq i. \tag{14} \]

Otherwise, \( E_i \) is a saddle.

(ii) For fixed \( i, j \in \{ 1, 2, 3 \}, \ i \neq j, \) the semi-trivial equilibrium \( E_{ij}, \) at which only species \( i \) and species \( j \) coexist, is locally asymptotically stable in \( R^3 \) if and only if

\[ \gamma_{ij} > 0 \quad \text{and} \quad \Delta_h < 0, \quad h = 1, 2, 3, \quad h \neq i, j. \tag{15} \]

Otherwise, \( E_{ij} \) is a saddle. Where \( \gamma_{ij}, \Delta_h \) are given in (13).
Proof. (i) From (12) we can get the Jacobian matrix at \( E_1 \)

\[
J(E_1) = \begin{bmatrix}
-n_1 & -c_{12} \frac{n_1}{b_1} & -c_{13} \frac{n_1}{b_1} \\
0 & \frac{\gamma_{12}}{b_1} & 0 \\
0 & 0 & \frac{\gamma_{13}}{b_1}
\end{bmatrix},
\]

it is easy to know that \( n_1 > 0 \) when \( E_1 \) exists, so it is asymptotically stable if and only if \( \gamma_{12} < 0 \) and \( \gamma_{13} < 0 \). Otherwise, it is a saddle when \( \gamma_{12} > 0 \) or \( \gamma_{13} > 0 \).

Similarly, we can get the conditions of local stability of \( E_2 \) and \( E_3 \).

(ii) From (11) we know that the existence condition of \( E_{12} \) is \( T < \min\{T_1^*, T_2^*\} \) and \( \gamma_{12} \gamma_{21} > 0 \), which is equivalent to that \( \beta_{12}, \gamma_{12} \) and \( \gamma_{21} \) have the same sign.

And from (12) the Jacobian matrix at \( E_{12} \) is

\[
J(E_{12}) = \begin{bmatrix}
-b_1 \frac{\gamma_{21}}{\beta_{12}} & -c_{12} \frac{\gamma_{21}}{\beta_{12}} & -c_{13} \frac{\gamma_{21}}{\beta_{12}} \\
-c_{21} \frac{\gamma_{12}}{\beta_{12}} & -b_2 \frac{\gamma_{12}}{\beta_{12}} & -c_{23} \frac{\gamma_{12}}{\beta_{12}} \\
0 & 0 & \frac{\Delta_3}{\beta_{12}}
\end{bmatrix},
\]

it is easy to see that for the top left 2 × 2 sub-matrix of \( J(E_{12}) \), the trace \(-b_1 \frac{\gamma_{21}}{\beta_{12}} - b_2 \frac{\gamma_{12}}{\beta_{12}} < 0 \) and determinant \( \frac{\gamma_{12}\gamma_{21}}{\beta_{12}} > 0 \) if \( \gamma_{12} > 0 \). And the third eigenvalue is \( \frac{\Delta_3}{\beta_{12}} \).

Combined with the previous analysis, we have that \( E_{12} \) is asymptotically stable if and only if \( \gamma_{12} > 0 \) and \( \Delta_3 < 0 \). Otherwise, the signs of the real parts of three eigenvalues are not all the same when \( \gamma_{12} > 0 \) and \( \Delta_3 > 0 \), or \( \gamma_{12} < 0 \) and \( \Delta_3 \neq 0 \), \( E_{12} \) is a saddle at this moment.

Similarly, we omit the stability analyses of \( E_{13} \) and \( E_{23} \).

\[\Box\]

3.3.2. Interior equilibrium. An interior equilibrium of (8) is a positive solution of the system

\[
\begin{align*}
b_1 x_1 + c_{12} x_2 + c_{13} x_3 &= n_1, \\
c_{21} x_1 + b_2 x_2 + c_{23} x_3 &= n_2, \\
c_{31} x_1 + c_{32} x_2 + b_3 x_3 &= n_3.
\end{align*}
\]

From (16), we see that system (8) possesses a unique interior equilibrium \( E^* = \left( \frac{\Delta_1}{\Delta}, \frac{\Delta_2}{\Delta}, \frac{\Delta_3}{\Delta} \right) \), at which three species coexist, if and only if the following conditions hold:

(i) \( n_i > 0, i = 1, 2, 3 \), which are equivalent to \( T < \min\{T_1^*, T_2^*, T_3^*\} \);

(ii) \( \begin{vmatrix} b_1 & c_{12} & c_{13} \\ c_{21} & b_2 & c_{23} \\ c_{31} & c_{32} & b_3 \end{vmatrix} = \Delta \neq 0 \);

(iii) \( \Delta_i, \Delta, (i = 1, 2, 3) \) have the same sign, where \( \Delta_i \) are defined in (13).

Regarding the local stability of \( E^* \), we have the following results.

**Theorem 3.5.** (i) When \( \Delta < 0 \) and \( \Delta_i < 0 \) for all \( i \in \{1, 2, 3\} \), \( E^* \) is a saddle;

(ii) When \( \Delta > 0 \) and \( \Delta_i > 0 \) for all \( i \in \{1, 2, 3\} \), \( E^* \) is a stable node if \( \rho < 0 \), a saddle if \( \rho > 0 \), and the corresponding Jacobian matrix \( J(E^*) \) of \( E^* \) has a pair of
purely imaginary eigenvalues if \( \rho = 0 \), where

\[
\rho = A_1A_2 - A_3, \quad A_1 = \text{Tr}(J(E^*)), \quad A_3 = \text{Det}(J(E^*)),
\]

\[
A_2 = \frac{1}{\Delta^2}(\beta_12\Delta_1\Delta_2 + \beta_13\Delta_1\Delta_3 + \beta_23\Delta_2\Delta_3).
\]

Proof. (i) From (12) the Jacobian matrix at \( E^* \) is

\[
J(E^*) = \begin{bmatrix}
-b_1\Delta_1 & -c_{12}\Delta_1 & -c_{13}\Delta_1 \\
-c_{21}\Delta_2 & -b_2\Delta_2 & -c_{23}\Delta_2 \\
-c_{31}\Delta_3 & -c_{32}\Delta_3 & -b_3\Delta_3
\end{bmatrix},
\]

and the corresponding characteristic equation is

\[
| I - J(E^*) | = \lambda^3 - A_1\lambda^2 + A_2\lambda - A_3 = 0.
\]

Denote the eigenvalues by \( \lambda_i, i = 1, 2, 3 \), then \( \lambda_1 + \lambda_2 + \lambda_3 = A_1 < 0, \lambda_1\lambda_2\lambda_3 = A_3 > 0 \) if \( \Delta_i < 0 \) holds for all \( i \in \{1, 2, 3\} \), then \( J(E^*) \) has an eigenvalue with negative real part and an eigenvalue with positive real part at this moment, so we can get that \( E^* \) is a saddle.

(ii) The proof of the second part is similar as Proposition 3.3 in [17], so we omit it.

3.4. Asymptotic behaviors and competition outcomes. In this subsection, we present a variety of long-term dynamics that system (8) may exhibit according to Theorems 3.3, 3.4, and 3.5. Inasmuch as the toxin concentration in the environment \( T \) is the most important parameter, we study the long-term dynamics of system (8) based on the following three cases:

(I) \( T > \max\{T_1^*, T_2^*, T_3^*\} \);

(II) \( \min\{T_1^*, T_2^*, T_3^*\} < T < \max\{T_1^*, T_2^*, T_3^*\} \);

(III) \( T < \min\{T_1^*, T_2^*, T_3^*\} \).

Case (I): \( T > \max\{T_1^*, T_2^*, T_3^*\} \). System (8) has a unique equilibrium \( E_0 \), we have the following results.

Theorem 3.6. The extinction equilibrium \( E_0(0,0,0) \), at which all species go extinct, is globally asymptotically stable in the first octant of \( \mathbb{R}^3 \) if \( T > \max\{T_1^*, T_2^*, T_3^*\} \).

Proof. Firstly, by Theorem 3.3, \( E_0 \) is a stable node when \( T > \max\{T_1^*, T_2^*, T_3^*\} \). Moreover, from Theorem 3.1 we know that system (8) has a positive invariant and bounded region, and system (8) has no other equilibrium except \( E_0 \), then all orbits will tend to \( E_0 \), thus \( E_0 \) is globally asymptotically stable if \( T > \max\{T_1^*, T_2^*, T_3^*\} \).

Case (II): \( \min\{T_1^*, T_2^*, T_3^*\} < T < \max\{T_1^*, T_2^*, T_3^*\} \). In this case, from the discussion on the existence of interior equilibrium in subsection 3.3.2, we see that system (8) has no positive equilibrium. Without loss of generality, we assume that \( T_1^* > T_2^* > T_3^* \). Then we have the following results.

Theorem 3.7. (i) When \( T_3^* < T < T_2^* \),

(ia) if \( \gamma_{12} > 0, \gamma_{21} > 0 \), then \( E_{12} \) is globally asymptotically stable in the first octant except the first quadrants of \( x_1 - x_3 \) and \( x_2 - x_3 \) planes (see Fig.1(a));
(ii) When $T < T^*_1$, $E_1$ is globally asymptotically stable in the first octant except the first quadrant of $x_2 - x_3$ plane (see Fig.1(d)).

Proof. (i) Under the fundamental assumption, system (8) has a trivial equilibrium $E_0$, two semi-trivial equilibria $E_1$ and $E_2$. Moreover, for cases (ia) and (ib), there is the other semi-trivial equilibrium $E_{12}$, and there are no other equilibria in all four subcases (ia)-(ib). $E_0$ is a saddle with a one-dimensional stable manifold lying on $x_3$ axis, and a two-dimensional unstable manifold lying on $x_1$, $x_2$ axes, respectively.

For case (ia), from Theorem 3.4, we know that $E_1$ is a saddle with a one-dimensional stable manifold lying on $x_1$ axis, $E_2$ is a saddle with a one-dimensional unstable manifold lying on $x_2$ axis. The orbit in the interior of the first quadrant of $x_1 - x_3$ (or $x_2 - x_3$) plane will converge to $E_1$ (or $E_2$). Moreover, combining the Remark 3.3 and according to Poincaré-Bendixson Theorem, any orbit in the first octant except the first quadrants of $x_1 - x_3$ and $x_2 - x_3$ planes will converge to the stable equilibrium $E_{12}$.

For case (ib), we know that both $E_1$ and $E_2$ are locally asymptotically stable. $E_{12}$ is a saddle with a one-dimensional unstable manifold lying on $x_1 - x_2$ plane and a two-dimensional stable manifold, which divides the first octant into two parts, the orbit with initial value in one part will converge to $E_1$, the orbit with initial value in the other part will converge to $E_2$, and the orbit on the invariant surface will converge to $E_{12}$.

For case (ic), there are only three equilibria $E_0$, $E_1$ and $E_2$ for system (8). $E_1$ is locally asymptotically stable, $E_2$ is a saddle with a one-dimensional unstable manifold lying on $x_1 - x_2$ plane. The orbit with initial value in $x_3$ axis converges to $E_0$ and the other orbit with initial value in the first quadrant of $x_2 - x_3$ plane converges to $E_2$. It is easy to know that the rest of orbit in the first octant will converge to $E_1$.

For case (id), it is similar to case (ic), we omit it.

(ii) In this case, system (8) has a trivial equilibrium $E_0$, which is a saddle, a semi-trivial equilibrium $E_1$, which is locally asymptotically stable, and no other equilibria. The orbit with initial value in the first quadrant of $x_2 - x_3$ plane converges to $E_0$, and the other orbit in the first octant will converge to the stable equilibrium $E_1.$

Case (III): $T < \min\{T^*_1, T^*_2, T^*_3\}$. In this case, the extinction equilibrium $E_0(0,0,0)$ is an unstable node by Theorem 3.3. For the three-dimensional competitive LV system (8), different combinations of signs of $\gamma_{ij}$ and $\Delta_i$ will produce different dynamic results. The theory of Hirsch [13] guarantees the existence of a two-dimensional globally invariant attracting hypersurface $\Sigma$, which is homeomorphic to the unit simplex $\{(x_1, x_2, x_3) \in R^3_+ \mid x_1 + x_2 + x_3 = 1\}$ via radial projection, such that every nontrivial trajectory in $R^3_+ \setminus \{0\}$ is asymptotic to one in $\Sigma$ (see [17, 35]). Zeeman [35] called such a hypersurface $\Sigma$ the carrying simplex.
and showed that the omega limit sets of system (8) are precisely those of the system restricted to the carrying simplex $\Sigma$ (see also [33, 34]). Moreover, Zeeman [35] showed that there are in total 33 classes of nullcline stable LV systems (see Fig.s 6, 7, 8 and 9 in [35]). Based on the classification, she proved that in classes 1-25 all the compact limit sets are equilibria, van den Driessche and Zeeman [28] further proved that classes 32-33 have no nontrivial periodic orbits, and that periodic orbits can occur in each of stable nullcline classes 26-31 (see [8, 14, 19, 22, 23, 29, 31]). Recently, Jiang and Liu [17] described each Zeeman’s class in terms of inequalities on the parameters of three-dimensional competitive LV system (see Appendix A in [17]). Next, we employ the results of Jiang and Liu [17] to our system (8) to consider possible competition outcomes for the case $T < \min\{T_1^*, T_2^*, T_3^*\}$. For convenience, we call the equilibria $E_i$, $i = 1, 2, 3$, at which one species excludes the other two, as strong exclusion equilibrium, the equilibria $E_{ij}$, $i, j = 1, 2, 3, i \neq j$, at which two of them exclude the third species, as weak exclusion equilibrium, and the equilibrium $E^*$, at which three species coexist, as coexistence equilibrium. We then divide the asymptotic dynamics (i.e., the eventual behavior of the species) into the following categories (see Table 1):

(1) One of the strong exclusion equilibria is globally asymptotically stable. That is, only one species survives, the other two die out. Case (1) in Table 1 shows an example.

(2) One of the weak exclusion equilibria is globally asymptotically stable. That is, two species survive, the third one dies out. See case (2) in Table 1.

(3) The coexistence equilibrium $E^*$ is globally asymptotically stable (see case (3a) in Table 1) or system has a stable limit cycle (see case (3b) in Table 1). That is, three species coexist at $E^*$ or they coexist but with abundances of periodic fluctuation under some initial population densities.

(4) Two of semitrivial boundary equilibria are locally asymptotically stable. That is, system (8) has two alternative stable states (bistability), where the competition
Table 1. The asymptotic dynamics on $\Sigma$ of system (8). $\bullet$ signifies an attractive equilibrium on $\Sigma$, $\circ$ signifies a repellent equilibrium on $\Sigma$, the intersection of its hyperbolic manifolds signifies a saddle on $\Sigma$.

| Case | The competition outcome | Phase portrait on $\Sigma$ |
|------|-------------------------|---------------------------|
| 1    | Species 1 excludes the other two species. | ![Diagram](https://example.com/diagram1.png) |
| 2    | Species 1 and 2 coexist, species 3 dies out. | ![Diagram](https://example.com/diagram2.png) |
| 3a   | Three species coexist at equilibrium $E^*$. | ![Diagram](https://example.com/diagram3a.png) |
| 3b   | Three species coexist at a stable limit cycle or species 2 excludes the other two species. | ![Diagram](https://example.com/diagram3b.png) |
| 4a   | Either species 1 excludes the other two species or species 2 excludes the other two species. | ![Diagram](https://example.com/diagram4a.png) |
| 4b   | Either species 1 and 2 coexist, species 3 dies out, or species 1 and 3 coexist, species 2 dies out. | ![Diagram](https://example.com/diagram4b.png) |
| 4c   | Either species 1 excludes the other two species, or species 2 and 3 coexist, species 1 dies out. | ![Diagram](https://example.com/diagram4c.png) |
| 5    | One of species excludes the other two species. | ![Diagram](https://example.com/diagram5.png) |

Outcome depends on initial population densities. This includes the following scenarios: (4a) Two of strong exclusion equilibria are locally asymptotically stable (see case (4a) in Table 1); (4b) Two of weak exclusion equilibria are locally asymptotically stable (see case (4b) in Table 1); (4c) One of strong exclusion equilibria and one of weak exclusion equilibria are locally asymptotically stable (see case (4c) in Table 1).
All three strong exclusion equilibria are locally asymptotically stable. That is, system (8) has three alternative stable states (tristability), only one of species survives at each stable state, and initial population densities will determine which one survives (see case (5) in Table 1).

4. Impact of toxin and species’ tolerance to toxin on competition dynamics. The main purpose of this section is to study how the environment toxin \(T\) and the species’ relative sensitivity to toxin \((p_2, p_3)\) affect the asymptotic dynamics of the competition system (8). To this end, we first compute the rates of change of population abundances at equilibria with respect to \(T\). We then study the bifurcation dynamics of the toxin-dependent system (8) by regarding \(T\) as a bifurcation parameter and letting \(T\) increases from zero to high levels and fixing \(p_2 = p_3 = 1\). Finally, we examine the effects of species’ relative vulnerability to toxin on competition outcomes.

4.1. Dependence of stable population abundance on external toxin. To analyze the sensitivity of asymptotically stable states with respect to the toxin level \(T\), we treat the coordinates of stable equilibria as a function of \(T\) and calculate their rates of change with respect to \(T\).

From section 3.3.1, we know that for fixed \(i \in \{1, 2, 3\}\), when \(T < T_i^*\) and \(\gamma_{ij} < 0, j = 1, 2, 3, j \neq i\), system (8) has a stable strong exclusion equilibrium (species-\(i\) only equilibrium) \(E_i\) with a positive coordinate \(n_i/b_i\), which represents the population abundance of species \(i\). Clearly,

\[
\frac{\partial}{\partial T} \left( \frac{n_i}{b_i} \right) = - \frac{(b_i + k_i)p_i}{b_i} < 0. \tag{17}
\]

This means that when strong competitive exclusion occurs, the abundance of winner decreases as the toxin level \(T\) increases. That is, a high concentration of toxin in the environment is always harmful when only one in three species survives.

We then consider the weak exclusion equilibria. When \(T < \min\{T_1^*, T_2^*\}\), \(\gamma_{12}, \gamma_{21}, \beta_{12} > 0\), and \(\Delta_3 < 0\), system (8) has a stable weak exclusion equilibrium

\[
E_{12} \left( \frac{b_2n_1 - c_{12}n_2}{b_1b_2 - c_{12}c_21}, \frac{b_1n_2 - c_{21}n_1}{b_1b_2 - c_{12}c_21}, 0 \right) := (\bar{x}_1, \bar{x}_2, 0),
\]

where \((\bar{x}_1, \bar{x}_2)\) satisfies the equations

\[
\begin{align*}
b_1(1 - p_1T) - k_1p_1T - m_1 - b_1\bar{x}_1 - c_{12}\bar{x}_2 &= 0, \\
b_2(1 - p_2T) - k_2p_2T - m_2 - b_2\bar{x}_2 - c_{21}\bar{x}_1 &= 0.
\end{align*}
\]

Differentiating the above equations with respect to \(T\), we have

\[
\begin{align*}
-b_1p_1 - k_1p_1 - b_1 \frac{\partial \bar{x}_1}{\partial T} - c_{12} \frac{\partial \bar{x}_2}{\partial T} &= 0, \\
-b_2p_2 - k_2p_2 - b_2 \frac{\partial \bar{x}_2}{\partial T} - c_{21} \frac{\partial \bar{x}_1}{\partial T} &= 0.
\end{align*}
\]

Simple calculation gives

\[
\begin{align*}
\frac{\partial \bar{x}_1}{\partial T} &= \frac{p_2c_{12}(b_2 + k_2) - p_1b_2(b_1 + k_1)}{b_1b_2 - c_{12}c_21}, \\
\frac{\partial \bar{x}_2}{\partial T} &= \frac{p_1c_{21}(b_1 + k_1) - p_2b_1(b_2 + k_2)}{b_1b_2 - c_{12}c_21}. \tag{18}
\end{align*}
\]
Noticing that $b_1 b_2 - c_{12} c_{21} = \beta_{12} > 0$, we find that $\frac{\partial \bar{x}_1}{\partial T} > 0(< 0)$ if $p_2 c_{12}(b_1 + k_2) - p_1 b_2(b_1 + k_1) > 0(< 0)$, and that $\frac{\partial \bar{x}_2}{\partial T} > 0(< 0)$ if $p_1 c_{21}(b_1 + k_1) - p_2 b_1(b_2 + k_2) > 0(< 0)$.

Similarly, we are able to discuss the dependence of weak exclusion equilibria $E_{13}$ and $E_{23}$ on the toxin level $T$.

Finally, for the coexistence equilibrium $E_+^+ \left( \frac{\Delta_1}{\Delta}, \frac{\Delta_2}{\Delta}, \frac{\Delta_3}{\Delta} \right) := (x_1^*, x_2^*, x_3^*)$ (see section 3.3.2 for the conditions on the existence and local stability of $E_+^+$), where $(x_1^*, x_2^*, x_3^*)$ satisfies the equations

\begin{align*}
b_1(1 - p_1 T) - k_1 p_1 T - m_1 - b_1 x_1^* - c_{12} x_2^* - c_{13} x_3^* &= 0, \\
b_2(1 - p_2 T) - k_2 p_2 T - m_2 - b_2 x_2^* - c_{21} x_1^* - c_{23} x_3^* &= 0, \\
b_3(1 - p_3 T) - k_3 p_3 T - m_3 - b_3 x_3^* - c_{31} x_1^* - c_{32} x_2^* &= 0.
\end{align*}

Differentiating the above equations with respect to $T$, we have

\begin{align*}
-b_1 p_1 - k_1 p_1 - b_1 \frac{\partial x_1^*}{\partial T} - b_2 \frac{\partial x_2^*}{\partial T} - c_{12} \frac{\partial x_2^*}{\partial T} - c_{13} \frac{\partial x_3^*}{\partial T} &= 0, \\
-b_2 p_2 - k_2 p_2 - b_2 \frac{\partial x_2^*}{\partial T} - c_{21} \frac{\partial x_1^*}{\partial T} - c_{23} \frac{\partial x_3^*}{\partial T} &= 0, \\
-b_3 p_3 - k_3 p_3 - b_3 \frac{\partial x_3^*}{\partial T} - c_{31} \frac{\partial x_1^*}{\partial T} - c_{32} \frac{\partial x_2^*}{\partial T} &= 0.
\end{align*}

Simple calculation gives

\begin{align*}
\frac{\partial x_1^*}{\partial T} = \frac{D_1}{\Delta}, & \quad \frac{\partial x_2^*}{\partial T} = \frac{D_2}{\Delta}, & \quad \frac{\partial x_3^*}{\partial T} = \frac{D_3}{\Delta},
\end{align*}

where

\begin{align*}
D_1 &= \begin{vmatrix}
-(b_1 + k_1)p_1 & c_{12} & c_{13} \\
-(b_2 + k_2)p_2 & b_2 & c_{23} \\
-(b_3 + k_3)p_3 & c_{32} & b_3 \\
\end{vmatrix}, & \quad D_2 &= \begin{vmatrix}
b_1 & -(b_1 + k_1)p_1 & c_{13} \\
c_{21} & -(b_2 + k_2)p_2 & c_{23} \\
\end{vmatrix}, & \quad D_3 &= \begin{vmatrix}
b_1 & c_{12} & -(b_1 + k_1)p_1 \\
c_{21} & b_2 & -(b_2 + k_2)p_2 \\
c_{31} & c_{32} & -(b_3 + k_3)p_3 \\
\end{vmatrix}.
\end{align*}

Thus, $\frac{\partial x_i^*}{\partial T} > 0(< 0)$ if $D_i$ and $\Delta$ have the same sign (opposite sign).

4.2. Impact of external toxins on competition outcomes. In this subsection, we scrutinize how the environmental toxin affects the competition outcomes by plotting bifurcation diagrams of system (8) with respect to the toxin concentration $T$. To do so, we choose sets of parameters such that when $T = 0$, stable competition outcomes of the classical LV competitive system occur. We then examine how these competition outcomes change as $T$ varies from zero to higher levels.

From (4) we see that the parameters $p_i \ (i = 2, 3)$ compare the sensitivity to toxin of species $i$ to that of species 1. Although different species may have different sensitivities to toxin, in order to concentrate on the impact of the toxin on populations of three species, we first assume that three species have the same tolerance ability for the toxin, i.e., $p_2 = p_3 = 1$. The effects of different relative sensitivities of the three species to the same toxin will be investigated in the next section.

Fig.s 2, 4, and 5 show bifurcation diagrams of the $x_1$-coordinates, $x_2$-coordinates, and $x_3$-coordinates of equilibria of system (8) versus $T$. Fig.3 describes the limit
cycles corresponding to the Hopf bifurcation in Fig.2. These diagrams illustrate that the external toxin affects the asymptotic dynamics of competition system (8) in many different ways. It should be noted that, $E_0$ always exists for all $T \geq 0$, it is unstable when $T < \max\{T_1^*, T_2^*, T_3^*\}$, and stable when $T > \max\{T_1^*, T_2^*, T_3^*\}$. That is, once the toxin level $T$ exceeds the threshold value $\max\{T_1^*, T_2^*, T_3^*\}$, all species go extinct. We are not interested in the existence and stability of $E_0$ and hence omit it in our bifurcation diagrams.

In the bifurcation diagrams, the solid lines signify stable equilibria and dash lines signify unstable equilibria. BP means branch point, at which the existence or stability of the equilibria will be transformed. H means Hopf bifurcation, at which periodic orbits around the coexistence equilibrium $E^*$ will occur when increasing or decreasing toxin at this moment. In particular, we select the following three cases to investigate the effects of external toxins on competition outcomes.

Figure 2. Bifurcation diagrams with respect to toxin level $T$ for the case where species-2 only equilibrium $E_2$ is globally asymptotically stable when $T = 0$. Parameters: $b_1 = 1$, $b_2 = 1.06$, $b_3 = 1.13$, $p_1 = p_2 = p_3 = k_1 = k_2 = k_3 = 1$, $m_1 = 0.44$, $m_2 = 0.5$, $m_3 = 0.56$, $c_{12} = 1.13$, $c_{13} = 1.19$, $c_{21} = 1.06$, $c_{23} = 0.88$, $c_{31} = 0.75$, $c_{32} = 1.24$. Here $T_1^* \approx 0.28$, $T_2^* \approx 0.27$, $T_3^* \approx 0.27$. 
Figure 3. (a) The Hopf bifurcation diagram corresponding to Fig.2; (b) Limit cycle for $T = 0.18069$ in (a); (c) The time series diagram corresponding to (b); (d) The time series diagram of heteroclinic loop for $T = 0.1801$; (e) The phase portrait corresponding to (d). The other parameters are the same as those in Fig.2.

4.2.1. One of the strong exclusion equilibria is globally asymptotically stable when $T = 0$. In Fig.2, species 2 excludes species 1 and species 3 when $T = 0$. As $T$
increases to fall into the interval (0.1801, 0.18071), system (8) has two alternative stable states (bistability): depending on the initial population biomass, either species 2 excludes the other two species or three species coexist but the population levels oscillate periodically around the unstable coexistence equilibrium $E^*$. As $T \in (0.18071, 0.18864)$, another type of bistability occurs: both the strong exclusion equilibrium $E_2$ and the coexistence equilibrium $E^*$ are locally asymptotically stable. As $T$ continues to increase until $T \in (0.18864, 0.21780)$, $E_2$ loses its stability and the coexistence equilibrium $E^*$ becomes globally asymptotically stable. As $T$ increases further ($T > 0.21780$), species 1 and species 3 coexist but species 2 becomes loser (i.e., the weak exclusion equilibrium $E_{13}$ is globally asymptotically stable) until species 1 excludes species 3 (i.e., the strong exclusion equilibrium $E_1$ is globally asymptotically stable) as $T$ increases even further. Finally, all three species go extinct when $T > \max\{T^*_1, T^*_2, T^*_3\}$.

Note that in Fig.2, $T = 0.18071$ is a Hopf bifurcation point, a stable limit cycle occurs on the left side of this point. As $T$ increases and passes through this point, the coexistence equilibrium $E^*$ changes its stability from unstable to stable. In Fig.3, we plot the resulting limit cycle from Hopf bifurcation in $x_1 = T$ plane (Fig.3(a)) and $x_1 - x_2 - x_3$ space (Fig.3(b)). We also draw the curves of periodic solutions in Fig.3(c). As $T$ decreases and passes another critical value 0.1801, this limit cycle expands, disappears and eventually tends a heteroclinic loop forming by three saddles $E_1, E_{13}$ and $E_{12}$ on the carrying simplex, see Fig.3(d) for the time series diagram and Fig.3(e) for the three-dimensional phase portrait.

4.2.2. Two of the strong exclusion equilibria are locally asymptotically stable when $T = 0$. In Fig.4, when $T = 0$, system (8) exhibits bistability: both species 2-only equilibrium $E_2$ and species 3-only equilibrium $E_3$ are locally asymptotically stable. That is, depending upon initial condition, either species 2 or species 3 is the unique winner. As $T$ increases to fall into the interval (0.11979,0.18864), another type of bistability occurs: both strong exclusion equilibrium $E_2$ and the weak exclusion equilibrium $E_{13}$ are locally asymptotically stable. This is because the increased toxin levels in the environment lead to decreased abundances of species 2 and species 3, which releases resources for their competitor — species 1 to survive. As $T$ increases further ($T \in (0.18864,0.23809)$), $E_2$ loses its stability and $E_{13}$ becomes globally asymptotically stable. As $T$ continues to increase, species 1 is left as the only winner since $E_1$ is globally asymptotically stable. In other words, the winner at $T = 0$ (species 2 or species 3) becomes a loser while the loser at $T = 0$ (species 1) becomes winner due to an increase in the toxin level! It is trivial that all three species are extirpated when $T > \max\{T^*_1, T^*_2, T^*_3\}$.

4.2.3. The coexistence equilibrium $E^*$ is globally asymptotically stable when $T = 0$. Fig.5 shows the bifurcation diagrams for the case where three species coexist at positive equilibrium $E^*$ when $T = 0$. Panels (b) and (d) of Fig.5 provide the partial enlarged details in panels (a) and (c), respectively. As we see from Fig.5, the population densities decrease as the toxin level increases. The competition outcomes vary in the following order as $T$ increases gradually: (1) three species coexist at positive equilibrium $E^*$ ($E^*$ is globally asymptotically stable); (2) species 1 and species 2 survive but species 3 dies out ($E_{12}$ is globally asymptotically stable); (3) species 2 excludes the other two species ($E_2$ is globally asymptotically stable); (4) all three species are extirpated.
Figure 4. Bifurcation diagrams with respect to toxin level $T$ for the case where both species 2-only equilibrium $E_2$ and species 3-only equilibrium $E_3$ are locally asymptotically stable when $T = 0$. Parameters: $c_{13} = 1.15$, $c_{23} = 1.19$, $c_{32} = 1.13$, the other parameters and the values of $T_i^*$ ($i = 1, 2, 3$) are the same as those in Fig.2.

The following conclusions can be drawn from Fig.s 2-5: (1) High concentration of toxins in the aquatic environment is harmful to all three species, possibly leading to population extirpation. (2) Intermediate level of toxins may boost coexistence of two or even three species. (3) Intermediate level of toxins may overturn and exchange roles of winner and loser in competition. (4) Intermediate level of toxins may also induce different types of bistabilities of the competition dynamics. In these scenarios, the competition outcome is doomed to strong exclusion, weak exclusion or coexistence, depending on initial population densities. (5) Certain levels of toxin can generate population oscillation (i.e., periodic solutions). On the contrary, the amplitude of population oscillation around the unstable coexistence equilibrium can be reduced until it stabilizes at a coexistence equilibrium as the toxin concentration increases.

4.3. Impact of species’ tolerance to toxin on competition outcomes. In the previous subsection, we concentrate on the effects of environmental toxin on
Figure 5. Bifurcation diagrams with respect to toxin level $T$ for the case where the coexistence equilibrium $E^*$ is globally asymptotically stable when $T = 0$. Part of panel (a) is enlarged by panel (b) and part of panel (c) is enlarged by panel (d). Parameters: $b_1 = 1$, $b_2 = 0.97$, $b_3 = 0.96$, $p_1 = p_2 = p_3 = k_1 = k_2 = k_3 = 1$, $m_1 = 0.52$, $m_2 = 0.47$, $m_3 = 0.53$, $c_{12} = 0.11$, $c_{13} = 0.16$, $c_{21} = 0.16$, $c_{23} = 0.21$, $c_{31} = 0.15$, $c_{32} = 0.22$. Here $T^* = 0.24$, $T^*_2 = 0.25$, $T^*_3 = 0.22$. 
competition outcomes by assuming that all species have the same sensitivity to toxin (i.e., $p_2 = p_3 = 1$). In reality, the tolerance ability for toxin may vary from species to species. Recall that the relative sensitivity parameters ($p_2$ and $p_3$) compare the sensitivity to toxin of species $i$ with that of species 1. In this subsection, we examine the impact of the relative sensitivities on the competition dynamics. To do so, we choose the same model parameters as those in Fig. 2, except the parameters $p_2$ and $p_3$, to depict the bifurcation dynamics of competition system (8) with respect to $T$ (Figs 6 and 7).

![Bifurcation diagrams](image)

**Figure 6.** Bifurcation diagrams with respect to toxin level $T$, where the parameters are the same to those in Fig. 2 except that $p_2 = 0.5$, $p_3 = 0.2$. Here, $T^*_1 = 0.28$, $T^*_2 = 0.54$, $T^*_3 = 1.34$.

Firstly, we let $p_2 = 0.5$, $p_3 = 0.2$, thus $p_1 > p_2 > p_3$, which implies that species 3 has the strongest tolerance ability for toxin and species 1 has the weakest tolerance ability. As shown by Fig. 6, when the aquatic environment is free to toxin (i.e., $T = 0$), the strong exclusion equilibrium $E_2$ is globally asymptotically stable, species 2 overwhelms species 1 and species 3. As the increase of the concentration of toxin, the population abundance of species 2 monotonically decreases, thus more resources are released for its competitors, species 1 and species 3, to survive. This leads to the coexistence of species 2 and species 3 and even the increase of the abundance of species 3 until species 3 becomes the only winner. This is because
the tolerance ability of species 3 for toxin is very strong, it hence benefits from the decreases of its competitor. However, inasmuch as the tolerance ability of species 1 is very weak, it never be able to establish itself although its competitors decrease as $T$ increases.

Secondly, we let $p_2 = 2$, $p_3 = 2.5$, so $p_1 < p_2 < p_3$, which indicates that the tolerance ability of species 1 is the strongest, and that the tolerance ability of species 3 is the weakest. From Fig.7, we see that species 2 excludes species 1 and species 3 when $T = 0$. As $T$ increases, the competition outcomes vary in the following order: (1) species 2 excludes species 1 and species 3; (2) two alternative stable state (bistability) — either species 2 excludes the other two species or three species coexist at stable periodic oscillations; (3) another type of bistability — either species 2 excludes the other two species or three species coexist at the interior equilibrium $E^*$; (4) three species coexist at the interior equilibrium $E^*$; (5) species 1 and species 3 coexist but species 2 dies out; (6) species 1 is the only winner; (7) all species are extirpated. The competition outcomes occur because the abundance of species 2 decreases as the toxin concentration increases, which benefits its competitors (species 1 and species 3). As for the outcome (6), when the toxin concentration is
relatively high, species 1 defeats species 3 inasmuch as species 1 has the strongest
tolerance ability for toxin.

Lastly, we consider the dependence of stable population abundance on external
toxin, which is analyzed in section 4.1. As an example, we look at Fig.7 again
and find that: (1) for low (high) concentration of toxins, as indicated by (17), the
abundance of the only winner, species 2 (species 1), is a decreasing function in $T$
(see black solid line in Fig.7 (b) (Fig.7 (a)); (2) for intermediate level of toxins,
when three species coexist at $E^*(x^*_1, x^*_2, x^*_3)$, from (19) we get that
\[
\frac{\partial x^*_1}{\partial T} \approx 3.33 > 0, \quad \frac{\partial x^*_2}{\partial T} \approx -16.53 < 0, \quad \frac{\partial x^*_3}{\partial T} \approx 11.22 > 0,
\]
that is, as $T$ increases, the stable population abundance of species 2 decreases (see
blue solid line in Fig.7 (b)), but the abundance of species 1 and 3 increase (see
blue solid lines in Fig.7 (a) and (c)) until species 2 is excluded by species 1 and
3 (i.e., the weak exclusion equilibrium $E_{13}$ is stable). For the stable equilibrium
$E_{13} := (\tilde{x}_1, 0, \tilde{x}_3)$, we have that
\[
\frac{\partial \tilde{x}_1}{\partial T} \approx 17.17 > 0, \quad \frac{\partial \tilde{x}_3}{\partial T} \approx -16.11 < 0,
\]
which means that as $T$ increases, the abundance of species 1 increases while the
abundance of species 3 decreases until it is excluded by species 1.

For other situations in sections 4.2 and 4.3, similar analysis shows that the num-
berical results are consistent with the results of sensitivity analysis in section 4.1.
In a word, the dependence of stable population abundance on external toxin signif-
icantly affects the competition outcomes.

5. Discussion. It is evident that the toxins in aquatic environment are obviously
capable of causing various ecological effects and even threaten ecosystem equilib-
rium. Over the last couple decades, a wide range of ecotoxicological technolo-
gies have been developed to assess the impacts of environmental pollutants on
aquatic ecosystems. Meanwhile, there is growing recognition that ecotoxicologi-
gal models are useful tools for understanding and evaluating the effects of toxin on
population dynamics in aquatic environments. Our search of literature shows that
most of existing toxin-dependent population models are single-species models (e.g.,
[7, 9, 10, 11, 15, 27]). These modelling efforts take a variety of approaches to predict
the direct effects of diverse chemical contaminants on organism growth and survival.
However, contaminants can have drastic indirect influences on aquatic ecosystems
by changing competition interactions or predator-prey relationships. In this work,
we developed a toxin-mediated competition model for three species simultaneously
exposed to environmental toxins. Our model incorporate both direct and indirect
toxic effects. While the direct effects may reduce population abundance by de-
creasing birth and increasing mortality, the indirect effects may lead to increased
abundance due to toxin-induced changes in the abundance of competitors.

It should be noted that in some senses, our three-species competition model
(2) extends the two-species toxin-mediated competition model by Shan and Huang
[25], whereas there are two main differences between the two models. In the first
place, model (2) considers the fact that the death from interspecific competition
leads to the loss of toxin in the population, which was overlooked in the two-species
competition model in [25]. In the second place, if there is no toxin or inter-specific
competition, the population growth model of single species in model (2) reduces to
\[
\frac{dx_i}{dt} = (b_i - m_i)x_i - c_{ii}x_i^2, \tag{20}
\]

which is different from the model in [25] in which the population growth is modeled by

\[
\frac{dx_i}{dt} = \frac{\alpha_i x_i}{1 + \gamma_i x_i} - m_i x_i, \tag{21}
\]

In model (20), per capita growth rate is \( \frac{1}{x_i} \cdot \frac{dx_i}{dt} = (b_i - m_i) - c_{ii}x_i := \phi(x) \), a decreasing linear function with respect to population density \( x_i \). But in model (21), per capita growth rate is \( \frac{\alpha_i}{1 + \gamma_i x_i} - m_i := \psi(x) \), a decreasing nonlinear function in \( x_i \). The reason why we choose function \( \phi(x) \), rather than \( \psi(x) \), is because the stability analysis of a three-species competition model with form \( \psi((x) \) is too challenging to carry out.

In order to analytically analyze the model with six equations, we employed a quasi-steady state assumption to reduce the model down to three dimensions. We assumed population metabolism occurs on a faster time scale than population growth dynamics. We analytically investigated the existence and stability of equilibria and obtained the corresponding conditions in terms of the parameter \( T \). These conditions provide the threshold values of external toxin level that lead to population persistence or extirpation. Moreover, by employing the existing theoretical results on the three dimensional LV system [13, 17, 35], we derived possible competition outcomes under the condition that the external toxin concentration is relatively low (i.e., \( T < \min\{T^*_1, T^*_2, T^*_3\} \)). As we discussed in section 3.4, our three-species toxin-mediated competition model exhibits much richer dynamics than two-species model studied in [25].

We numerically analyze how the balance in the classical three-species LV system (without a toxin effect) changes as the concentration of toxins in the environment increases. As shown by Fig.s 2-5, different toxin levels affect competition outcomes in different ways. It turns out that high toxin concentrations lead to population extirpation. Low toxin concentrations do not change the balance in the classical LV system in which the interspecific interactions always have negative effects on all species. But intermediate toxin concentrations can have a positive effect on some disadvantaged species because toxins reduce the abundance of the predominant species, which leaves more resources with its competitors to grow. This indicates that it is possible that certain toxin levels boost coexistence of species and hence keep species diversity in aquatic ecosystems. We also numerically study how the relative sensitivities to toxin of species affect competition outcomes. As we have seen from Fig.s 6 and 7, as the toxin concentration increases, a loser with strong toxin tolerance ability can become a winner, while a winner with weak toxin tolerance ability can become a loser. That is to say, the roles of winner and loser in competition may overturn and exchange because of different relative sensitivities to toxin between species. The counterintuitive results we obtained in section 4 provide some new perspectives and useful guidelines in environmental protection and ecological conservation.

The model formulation of the present investigation could be generalized in several biologically meaningful ways: (1) Model (1) assumes the concentration of toxin in the environment is a constant. This concentration, however, likely vary seasonally or randomly due to a variety of factors, such as industrial activities and total volume of the aquatic environment. (2) For analytic convenience, we assume that
the reproduction and mortality rates of populations depend linearly on the body burdens $y_i$. In practice, these dependence may not be linear and could change from species to species. (3) Our model also assumes that the competition coefficients are constants. However, toxin-induced changes in behaviors of populations may also lead to variation of competition abilities. (4) Model (1) we studied in this work is a non-spatial ordinary differential equation system. It is important to consider the random movement of individuals and the diffusion of toxins in the environment. Extending the current model by incorporating these factors will yield more realistic models and some interesting mathematical problems.

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*E-mail address*: 2548244602@qq.com

*E-mail address*: hjc@mail.ccnu.edu.cn

*E-mail address*: qihua@swu.edu.cn