Development of a Toxin-Mediated Predator-Prey Model Applicable to Aquatic Environments in the Athabasca Oil Sands Region

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December 2014

Oil Sands Research and Information Network

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This report may be cited as:

Huang, Q., H. Wang and M.A. Lewis, 2014. Development of a Toxin-Mediated Predator-Prey Model Applicable to Aquatic Environments in the Athabasca Oil Sands Region. Oil Sands Research and Information Network, University of Alberta, School of Energy and the Environment, Edmonton, Alberta. OSRIN Report No. TR-59. 59 pp.

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REPORT SUMMARY

Industrial contaminants are one of the leading causes of pollution worldwide. It has been shown that 13 elements considered priority water pollutants by the US Environmental Protection Agency are present in the Athabasca River and are found in oil sands process-affected water. There are likely natural and anthropogenic sources of these toxins in the receiving environment. To protect ecological environments and aquatic species in Alberta, it is necessary to assess the risk of toxins to aquatic organisms, and find important factors that determine the persistence and extirpation of populations or species.

While previous work has considered the effect of a toxin on the population dynamics of a single trophic level, such as fish, we focus on the impacts of toxins on the population dynamics of aquatic food webs to understand possible outcomes.

Mathematical models have been widely applied to perform chemical risk assessments on all levels of the biological hierarchy, from cells to organs to organisms to populations to entire ecosystems. Here we develop a toxin-mediated predator-prey model that includes population dynamics. We use the model to evaluate the flow of toxins through the aquatic food web into the aquatic ecosystem and study how the transfer of toxins between trophic levels changes the food web dynamics. We analyze the model by studying the existence and stability of steady states and the effect of toxin level in the environment on steady states.

The model is then connected to experimental data via model parameterization. In particular, we consider the toxic effects of methylmercury on rainbow trout (*Oncorhynchus mykiss*) and its prey (small fish or aquatic insects) and obtain an appropriate estimate for each model parameter. The results of model parameterization and model analysis are used to numerically solve the model, and the results of the effect of the methylmercury on the end behavior of rainbow trout and its prey (small fish or aquatic insects) are provided.

From our analysis and numerical exploration of the food web toxin model we found that different toxin concentrations affect organisms at different trophic levels in many different ways. For example, high toxin concentrations in the environment are harmful to both species, and may lead to extirpation of both species. However, low toxin concentrations produce counterintuitive results. That is, contaminant effects on predators can actually lead to increased abundance of the prey.

The existence of limit cycles, where both population levels fluctuate around coexistence equilibrium, is found in most classical predator-prey models. Our findings show that increasing toxin level may reduce and prevent populations from fluctuating when the predator and the prey are exposed simultaneously to a toxin. Unlike most standard predator-prey systems, where populations will eventually tend toward only one stable state, our findings indicate that with a toxic effect, predator-prey systems may lead to multiple possible long-term outcomes. In this scenario, the initial population level will determine the final fate.

ACKNOWLEDGEMENTS

The Oil Sands Research and Information Network (OSRIN), School of Energy and the Environment (SEE), University of Alberta provided funding for this project. We also received suggestions, input and initial direction from Alberta Environment and Sustainable Resource Development (AESRD). We are especially grateful to Carolyn Bampfylde (AESRD) and Chris Powter (OSRIN) for helpful suggestions during preparation of the report.

1 INTRODUCTION

There is increasing global concern over the effects of anthropogenic and natural environmental toxins on ecosystem health. Industrial toxins are one of the leading causes of pollution worldwide. In the United States, for example, the Environmental Protection Agency estimates that up to 50% of the nation's pollution is caused by industry[1]. Industrial toxins may arise as a result of air emissions, water releases, water seepage, air deposition or disposal and leaching of solid waste. Toxins of concern may also be transported through natural systems as a result of weathering or leaching. The US Environmental Protection Agency has designated 126 priority pollutants[2] and the Canadian Council of Ministers of the Environment has a list of priority chemicals of concern for the protection of aquatic life[3]. These priority substances include metals and organic compounds.

The oil sands in Northeastern Alberta represent one of the largest oil deposits in the world. Production from the oil sands has reached 1 million barrels per day, and is predicted to increase five-fold by 2030[4]. It has been shown that 13 elements considered priority water pollutants by the US Environmental Protection Agency are present in the Athabasca River at low concentrations and also can be found in oil sands process-affected water (Kelly et al. 2010).

The Athabasca River bisects the surface mineable area. The river accumulates toxins due to leaching from exposed oil sands seams along the riverbank; in addition, there are likely anthropogenic sources of toxins from seepage or air deposition. However, there has been no release of process-affected water to date.

The combination of natural and anthropogenic sources of toxins present challenges with respect to the protection of local freshwater resources throughout the lower Athabasca River basin. To protect ecological environments and aquatic species in Alberta, it is necessary to assess the risk to aquatic organisms exposed to toxins, and find relevant factors that determine the persistence and extirpation of organisms.

1.1 Model Selection

Over the past several decades, mathematical models have been widely applied to perform chemical risk assessments on all levels of biological hierarchy, from cells to organs to organisms to populations to entire ecosystems. These models include population models (scalar abundance, life history, individual-based, and metapopulation), ecosystem models (food-web, aquatic and terrestrial), landscape models, and toxicity-extrapolation models (Bartell et al. 2003, Galic et al. 2010, Pastorok et al. 2001, 2003). The selection of specific models for addressing an ecological risk issue depends on: the habitat; endpoints and chemicals of interest; the balance between

[1] See http://www.epa.gov/

[2] See http://www.epa.gov/

[3] See http://www.ccme.ca/

[4] See http://www.energy.gov.ab.ca/89.asp

model complexity and availability of data; the degree of site specificity of available models; and, the risk issue (Pastorok et al. 2001). A comprehensive review of the realism, relevance, and applicability of different types of models from the perspective of assessing risks posed by toxic chemicals is provided by Bartell et al. (2003) and Pastorok et al. (2001).

In practice, toxin-dependent individual-based models and matrix population models are widely used to evaluate the ecological significance of observed or predicted effects of toxic chemicals on individual organisms and population dynamics. Literature search results show that relatively few differential equation models have been developed to describe population-toxin interactions (e.g., Freedman and Shukla 1991, Hallam and Clark 1983, Hallam et al. 1983, Luna and Hallam 1987, Thieme 2003, Thomas et al. 1996). These interactions are usually described by a system, which contains components representing the population density, the concentration of toxin in an organism, and the environmental concentration of toxin.

Recently, we developed a toxin-dependent model given by a system of differential equations, to describe the impact of contaminants on fish population dynamics (Huang et al. 2013). Given that the concentration of biomass of the population, in reality, is usually very low, the concentration of toxin in the environment therefore is not affected significantly by death or metabolic processes of the population. In other words, the concentration of toxin in the environment is mainly determined by external conditions, such as the exogenous input of toxin, decomposition by sunlight or hydrolysis. Unlike the above-mentioned population-toxin interaction models, our toxin-dependent model assumes that the population does little or no regulation of toxin in the environment. The concentration of toxin in the environment hence is treated as a parameter. The model was connected to literature-sourced experimental data via model parameterization. In particular, we considered the toxic effects of methylmercury on rainbow trout (*Oncorhynchus mykiss*) and obtained an approximate range or value for each model parameter. The parameter estimates were then used to illustrate the long-term behavior of rainbow trout population. The numerical results provide threshold values of concentration of methylmercury in the environment to maintain the population and prevent extirpation. The findings are consistent with Alberta Provincial surface water quality guidelines (Alberta Environment 1999).

It is significant that all above-mentioned differential equation models are single-species models in which populations were assumed to uptake toxin only from exposure to water. However, it is well recognized that the primary route of toxin uptake in higher-trophic level organisms (predators) is via food ingestion. As one organism eats another, it also eats the pollutants in its prey. The higher up the food chain, the more pollutants that are eaten and stored. The build-up of toxic pollutants is called bioaccumulation (Arnot and Gobas 2004, Mackay and Fraser 2000, Mathew et al. 2008). Bioaccumulation means that the non-linear effects observed in ecosystems cannot often be described or understood through looking at species individually because food web interactions need to be considered (e.g., Kidd et al. 2007).

In this report we evaluate the flow of contaminants through the aquatic food web into the aquatic ecosystem and study how the transfer of contaminants between trophic levels changes the food web dynamics. We do this by extending the single-species toxin dependent model in Huang et

al. (2013) to a toxin-mediated predator-prey interaction. The model consists of four equations. The first and second equations describe the prey and the predator growth rates, respectively, where the birth and death rates are explicit functions of body burdens. The third and fourth equations are the balance equations for the body burden of the two species, which describes the accumulation, the dilution of toxin in the organism tissue, and the transfer of toxin from prey to predator.

If there is no toxin, our toxin-mediated predator-prey model reduces to a classical predator-prey model whose dynamics are well studied. The main objective of this study is to investigate how the balance of the classical predator-prey dynamics will change when the toxin level in the environment varies from zero to a high level.

1.2 Toxin Selection

Under Schedule I of the *Canadian Environmental Protection Act* (Government of Canada 1999), mercury is considered a toxic substance. Mercury may be released into the aquatic environment in states of relatively low toxicity, but will be transformed into a highly toxic state, namely methylmercury. Methylmercury's harmful effects on fish include death, reduced reproduction, slower growth and development, and abnormal behavior (Eisler 1987). Methylmercuy is of special concern, not only because of its toxicity, but also because of its tendency to biomagnify in upper trophic levels of aquatic food webs (Canadian Council of Ministers of the Environment 2003). The Canadian water quality guidelines for methylmercury for the protection of aquatic life were developed based on the CCME protocol (Canadian Council of Ministers of the Environment 1991).

Rapid expansion of crude oil production from the Alberta oil sands has generated widespread concern regarding the potential impact of oil sands development on the Athabasca River and its watersheld. During the processing and upgrading of oil sands, mercury is released through coking, coke combustion, and through the production of wastes and fly ash that contain mercury (Environment Canada 2014). Environmental organizations have claimed tailing ponds leak an unknown volume of tailings wastes that contain mercury and other contaminants of concern directly into the Athabasca River (Price 2008). In recent years, the Alberta government and oil sands companies indicated that oil sands development did not contribute substantial loadings of mercury to the region and that any increases in mercury in the Athabasca River and its tributaries were due to natural erosion of oil sand formations. To investigate further, an assessment of mercury concentrations water of the Athabasca River, its tributaries, the Athabasca Delta and Lake Athabasca was conducted by Radmanovich (2013). The results of the study by Radmanovich (2013) indicate that concentrations of mercury in some snow and waters samples from tributary and Athabasca River sites near development exceed guidelines for the protection of aquatic life, and oil sands upgrading facilities are a sigificant source of mercury within the Athabasca watershed. Li et al. (2014) also discuss the toxicity of mercury and note that concentrations in oil sands process-affected water range from <0.02 µg/L to 0.17 µg/L, which are generally higher than CCME guidelines (0.026 µg/L).

The toxic effects of methylmercury exposure in fish and wildlife species are well documented (reviewed by Sandheinrich and Wiener 2011). The United States Geological Survey (USGS) developed the National Descriptive Model for Mercury in Fish (Wente 2004) to partition variation in methylmercury concentration due to size, species and sample type across space and time. The Canadian Fish Mercury Database includes over 330,000 records representing 104 species of freshwater fish collected from over 5,000 locations across Canada between 1967 and 2010 (Depew et al. 2013).

1.3 Predator and Prey Selection

We use rainbow trout (*Oncorhynchus mykiss*) as our representative predatory fish. This species is commonly used for toxicology studies and is well studied, not only for mercury, but also for the effects of oil sands contaminants (e.g., Gorelick 1990). Therefore there is good toxicological information on rainbow trout that allows us to parameterize our model. Although we treat these as a representative fish, where detailed physiological information is available, this species of trout is not common to the oil sands area. Therefore, the report should not be interpreted as making specific predictions about oil sands toxins on natural rainbow trout populations. Rather it should be interpreted as developing a comprehensive framework for understanding impacts of toxins on multitrophic population dynamics, with the view that future studies can be used to parameterize the model more thoroughly for fish found commonly found in the oil sands area such as walleye (*Sander vitreus*), lake whitefish (*Coregonus clupeaformis*), northern pike (*Esox lucius*) and lake trout (*Salvelinus namaycush*) (Evans and Talbot 2012).

By way of background information on biogeography and status of rainbow trout within Alberta, there are two primary strains, introduced and native Athabasca rainbow trout. Athabasca rainbow trout are considered "at risk" by the General Status of Alberta Wild Species (Alberta Environment and Sustainable Resource Development 2014), due to potential habitat loss, hybridizations with introduced rainbow trout and competition with Eastern brook trout. The Athabasca rainbow trout are distributed throughout the headwaters of the Athabasca River system, with an extent of occurrence of approximately 20,000 km^2 (Rasmussen and Taylor 2009). This range includes the Athabasca River itself, as well as its major tributaries, including the Mcleod, the Wildhay/Berland, the Sakwatamau and the Freeman rivers. The range includes siginificant portions of Jasper National Park (Ward 1974), including the mainstem Athabasca River watersheld downstream of Sunwapta Falls, and the lower reaches of the Snaring, Maligne, Rocky and Snake Indian river systems downstream of majors waterfalls, and the majority of the Miette River watersheld.

Rainbow trout routinely feed on larval, pupal and adult forms of aquatic insects (typically caddisflies, stoneflies, mayflies and aquatic diptera), and small fish up to one-third of their length[5]. Caddisflies are an order, *Trichoptera*, of insects with approximately 12,000 described

[5] See http://www.env.gov.bc.ca/wld/documents/fishfacts/rainbowtrout.pdf

[6] See http://www.uky.edu/Ag/CritterFiles/casefile/insects/caddisflies/caddisflies.htm

species. In most temperate areas, caddisflies complete their life cycles in a single year, from egg to larva, to pupa, then adult. Most caddisfly larvae (caddis worms) live about one year in the aquatic environment before pupating. The adult stage of caddisflies, in most cases, is very short-lived, usually only 1 to 2 weeks. Caddisfly adults live just long enough to mate; they do not eat and focus only on reproduction (Willis and Hendricks 1992). Each adult female can lay up to 800 eggs. Willis and Hendricks (1992), in a study of the life history and production of hydropsychid caddisflies in Mill Creek, Virginia, found that only about 0.5% of the original eggs survived to adulthood. This finding is in close agreement with the findings of Elliott (1981, 1982) that survival to reproduction in *Philopotamus montanus* was 0.4% and survival from egg to imago in *Potamophylax cingulatus* was 1% to 2%.

1.4 Structure of this Report

The rest of this report is organized as follows. In Section 2, we develop the toxin-mediated predator-prey model. In Section 3, we parameterize the model using experimental results in the literature. In Section 4, we reduce the dimensionality of the model using a quasi-steady state approximation. We then analyze the existence and stability of extirpation and coexistence equilibria based on the quasi-steady system. In Section 5, we show possible asymptotic dynamics of the model. In Section 6, we study how toxin level (mercury) in the environment affects the long-term behavior of the populations. In Section 7, we apply the results of model parameterization and numerical simulations to consider the toxic effects of mercury on rainbow trout and its prey (small fish aquatic insects). Finally a brief discussion section completes the report.

2 MODEL FORMULATION

Since we are interested in an aquatic environment, we formulate the model in terms of concentration of population biomass, concentration of toxin in the population, and concentration of toxin in the environment. In this study, we let

$$\begin{aligned}&\text{Concentration of population biomass}\\ &= \frac{\text{total mass of the population}}{\text{volume of the total aquatic environment where the population lives}},\end{aligned}$$

$$\text{Concentration of toxin in the population} = \frac{\text{total mass of toxin contained in the population}}{\text{volume of the total aquatic environment}},$$

$$\text{Concentration of toxin in the environment} = \frac{\text{total mass of toxin in the environment}}{\text{volume of the total aquatic environment}},$$

and

$$\text{Body burden of population} = \frac{\text{total mass of toxin contained in the population}}{\text{total mass of all individuals in the population}}.$$

The state variables of the model are:

- $x = x(t)$, the concentration of prey biomass in g/L at time t;
- $y = y(t)$, the concentration of predator biomass in g/L at time t;
- $U = U(t)$, the concentration of toxin contained in the prey in µg/L at time t;
- $V = V(t)$; the concentration of toxin contained in the predator in µg/L at time t;
- $u = u(t)$, the body burden of the prey in µg/g at time t; and
- $v = v(t)$, the body burden of the predator in µg/g at time t.

A mathematical model that describes the effect of toxin on the predator-prey system is given by

$$\underbrace{\frac{dx}{dt}}_{\substack{\text{rate of change}\\ \text{of the concentration}\\ \text{of prey biomass}}} = \underbrace{b(u,x)x}_{\substack{\text{gain due to}\\ \text{birth and growth}}} - \underbrace{d_1(u)x}_{\substack{\text{loss due}\\ \text{to death}}} - \underbrace{p(x)y}_{\substack{\text{loss due}\\ \text{to predation}}},$$

$$\underbrace{\frac{dy}{dt}}_{\substack{\text{rate of change}\\ \text{of the concentration}\\ \text{of predator biomass}}} = \underbrace{e(v)p(x)y}_{\substack{\text{gain due to}\\ \text{birth and growth}}} - \underbrace{d_2(v)y}_{\substack{\text{loss due}\\ \text{to death}}},$$

$$\underbrace{\frac{dU}{dt}}_{\substack{\text{rate of change}\\ \text{of the concentration}\\ \text{of toxin in the prey}}} = \underbrace{a_1 T x}_{\substack{\text{uptake from}\\ \text{environment}}} - \underbrace{\sigma_1 U}_{\substack{\text{depuration due}\\ \text{to metabolism}}} - \underbrace{d_1(u)U}_{\substack{\text{loss due}\\ \text{to death}}} - \underbrace{p(x)yu}_{\substack{\text{loss due}\\ \text{to predation}}},$$

$$\underbrace{\frac{dV}{dt}}_{\substack{\text{rate of change}\\ \text{of the concentration}\\ \text{of toxin in the predator}}} = \underbrace{a_2 T y}_{\substack{\text{uptake from}\\ \text{environment}}} - \underbrace{\sigma_2 V}_{\substack{\text{depuration due}\\ \text{to metabolism}}} - \underbrace{d_2(v)V}_{\substack{\text{loss due}\\ \text{to death}}} + \underbrace{p(x)yu}_{\substack{\text{gain due to}\\ \text{predation}}},$$

$$\underbrace{u = U/x}_{\text{body burden of prey}}, \qquad \underbrace{v = V/y}_{\text{body burden of predator}}, \tag{1}$$

with appropriate initial conditions, which describe the initial concentrations of prey and predator biomass and toxin concentrations.

The first equation presents a generic description of the growth of prey under the influence of the toxin. The second equation describes the growth of predator under the influence of the toxin. The third and fourth equations are balance equations for the concentrations of the toxin contained in the individuals of prey and predator, respectively.

The function $b(u,x)$ represents the biomass gain rate of the prey due to reproduction and growth; $d_1(u)$ denotes the biomass loss rate of the prey due to death; $d_2(v)$ represents the

biomass loss rate of the predator due to death; $p(x)$ is the predator functional response that specifies the rate at which prey are consumed, per predator, as a function of the prey density; $e(v)$ is the conversion efficiency. We will introduce specific expressions for the functions $b(u,x)$, $d_1(u)$, $d_2(v)$, $p(x)$, and $e(v)$ at the end of this section.

The toxin uptake rates by the population from the environment, a_1Tx and a_2Ty, are modeled according to the *Law of Mass Action* and are proportional to both the concentration of toxin in the environment, T, and the concentration of population biomass. In this model a_1 and a_2 are the uptake coefficients for the prey and the predator, respectively. The positive constants σ_1 and σ_2 are the toxin depuration rates of the prey and the predator, respectively due to metabolic processes. The death of an individual leads to not only a loss of population biomass, but also a loss of population toxin concentration. This leads to the term $-d_1(u)x$ in the first equation and the term $-d_1(u)U$ in the third equation. The predation of prey by predator leads to both a loss of the prey biomass and a gain of the predator biomass; accordingly, it leads to a transfer of toxin from the prey to the predator. This results in the term $-p(x)yu$ in the third equation and the term $p(x)yu$ in the fourth equation.

From the first two equations of the model (1), we notice that the direct influences of toxin on the growth of populations are implemented through their body burdens u and v. This motivates us to write down the equations describing the rate of change of u and the rate of change of v. As we will see, this allows us to study an equivalent system involving four state variables and four equations, instead of the model (1) which includes six state variables and six equations.

From the fifth equation of (1), we have

$$\frac{du}{dt} = \frac{U'}{x} - \frac{U}{x}\frac{x'}{x} \tag{2}$$

Substituting the first equation and the third equation of (1) into (2), we obtain

$$\begin{aligned} \frac{du}{dt} &= a_1T - [\sigma_1 + d_1(u)]u - \frac{p(x)y}{x}u - \left[b(u,x) - d_1(u) - \frac{p(x)y}{x} \right]u \\ &= a_1T - \sigma_1 u - b(u,x)u. \end{aligned} \tag{3}$$

Similar calculations in terms of the last, second and fourth equations of (1) gives

$$v' = \frac{V'}{y} - \frac{V}{y}\frac{y'}{y} = a_2T - \sigma_2 v + p(x)[u - e(v)v] \tag{4}$$

Combining the first two equations of (1) and equations (3) and (4), we have

$$\begin{aligned}
\frac{dx}{dt} &= b(u,x)x - d_1(u)x - p(x)y, \\
\frac{dy}{dt} &= e(v)p(x)y - d_2(v)y, \\
\frac{du}{dt} &= a_1T - \sigma_1 u - b(u,x)u, \\
\frac{dv}{dt} &= a_2T - \sigma_2 v + p(x)[u - e(v)v].
\end{aligned} \tag{5}$$

We now introduce specific forms for the functions $b(u,x)$, $d_1(u)$, $d_2(v)$, $p(x)$, and $e(v)$. Following Huang et al. (2013), we let

$$b(u,x) = \frac{\alpha_1 \max\{0, 1-\alpha_2 u\}}{1+\alpha_3 x} \tag{6}$$

with positive constants α_i $(i = 1,2,3)$. Here the term $\alpha_1/(1+\alpha_3 x)$, which is a decreasing function with respect to prey biomass, represents a density-dependent per unit biomass gain rate. The term $\max\{0,1-\alpha_2 u\}$, which is a fraction between 0 and 1, represents a linear dose response for the gain rate. If there is no toxic effect (body burden $u = 0$), then $\max\{0,1-\alpha_2 u\} = 1$, hence the gain rate of prey biomass is given by $\alpha_1/(1+\alpha_3 x)$. If the body burden u reaches a threshold level $1/\alpha_2$, then the individuals in the prey stop reproduction and growth, hence the gain rate of prey biomass is 0. A derivation of the expression (6) from a resource-consumer model via a time scale argument is presented in Thieme (2003).

In 1992, the committee on toxicology of the National Research Council recommended the use of a power law to study the relationship between toxin concentration and mortality rate since it has been shown to fit the data well (Miller and Janszen 2000). Here, for model analysis, we assume a special case of power law with power one. That is, we assume that mortality rates $d_1(u)$ and $d_2(v)$ linearly depend on their body burdens u and v. Thus, taking natural mortality rates into account, we let

$$d_1(u) = k_1 u + m_1, \quad d_2(u) = k_2 u + m_2 \tag{7}$$

where k_1, m_1, k_2, m_2 are positive constants. The predator functional response describes a predator's per capita feeding rate. Here we use a Type II functional response (Holling 1959) which is more realistic than Type I (e.g., Polis et al. 1989) as it incorporates predator satiation through the assumption that predators have a prey handling time. In this case, the per capita feeding rate of the predator is given by a function of the form

$$p(x) = \frac{\gamma x}{1+\gamma h x} \tag{8}$$

where γ is the encounter rate (or capture efficiency) and h is the handling time.

For convenience, many researchers rewrite the above Type II functional response as

$$p(x) = \frac{\xi x}{\eta + x} \tag{9}$$

where $\xi = 1/h$ and $\eta = 1/(\gamma h)$ (Kot 2001). Since $p(\eta) = \xi/2$, η is referred as the half-saturation constant. In this study, we chose (9) as the expression of the functional response.

We assume that the dependence of the reproduction efficiency of the predator on its body burden v is given by

$$e(v) = \beta_1 \max\{0, 1 - \beta_2 v\} \tag{10}$$

where $0 < \beta_1 < 1$. The term $\max\{0, 1-\beta_2 v\}$ represents a linear dose response for the reproduction efficiency. If there is no toxic effect (body burden $v = 0$), then $\max\{0, 1-\alpha_2 u\} = 1$, hence the reproduction efficiency is β_1. If the body burden v reaches a threshold level $1/\beta_2$, the reproduction efficiency is 0, which means that predators stop reproduction and growth.

Therefore, in this study we propose the following toxin-mediated predator-prey system

$$\begin{aligned}
\underbrace{\frac{dx}{dt}}_{\substack{\text{rate of change}\\ \text{of the concentration}\\ \text{of prey biomass}}} &= \underbrace{\frac{\alpha_1 \max\{0, 1-\alpha_2 u\} x}{1+\alpha_3 x}}_{\substack{\text{gain due to}\\ \text{birth and growth}}} - \underbrace{(k_1 u + m_1) x}_{\substack{\text{loss due}\\ \text{to death}}} - \underbrace{\frac{\xi x y}{\eta + x}}_{\substack{\text{loss due}\\ \text{to predation}}} \\
\underbrace{\frac{dy}{dt}}_{\substack{\text{rate of change}\\ \text{of the concentration}\\ \text{of predator biomass}}} &= \underbrace{\frac{\beta_1 \xi x y \max\{0, 1-\beta_2 v\}}{\eta + x}}_{\substack{\text{gain due}\\ \text{to birth and growth}}} - \underbrace{(k_2 v + m_2) y}_{\substack{\text{loss due}\\ \text{to death}}} \\
\underbrace{\frac{du}{dt}}_{\substack{\text{rate of change}\\ \text{of the body burden}\\ \text{of the prey}}} &= \underbrace{a_1 T}_{\substack{\text{uptake from}\\ \text{environment}}} - \underbrace{\sigma_1 u}_{\substack{\text{depuration due}\\ \text{to metabolism}}} - \underbrace{\frac{\alpha_1 \max\{0, 1-\alpha_2 u\}}{1+\alpha_3 x} u}_{\substack{\text{dilution due to}\\ \text{birth and growth}}} \\
\underbrace{\frac{dv}{dt}}_{\substack{\text{rate of change}\\ \text{of the body burden}\\ \text{of the predator}}} &= \underbrace{a_2 T}_{\substack{\text{uptake from}\\ \text{environment}}} - \underbrace{\sigma_2 v}_{\substack{\text{depuration due}\\ \text{to metabolism}}} + \underbrace{\frac{\xi x}{\eta + x} u}_{\substack{\text{gain due}\\ \text{to predation}}} - \underbrace{\frac{\xi x}{\eta + x} \beta_1 \max\{0, 1-\beta_2 v\} v}_{\substack{\text{dilution due to}\\ \text{birth and growth}}}
\end{aligned} \tag{11}$$

In the absence of a predator, the model (11) reduces to the one species toxin-mediated model studied in Huang et al. (2013).

3 MODEL PARAMETERIZATION

While our toxin-mediated predator-prey model (11) is general, we apply it to consider the effect of a toxin on the dynamics of fish and its prey. In this section, we first describe the parameterization of the model (11) by choosing rainbow trout as the predator and small fish or

aquatic insects as the prey. The results of model parameterization are then used to understand the impact of methylmercury on the dynamics of rainbow trout and its prey (small fish or aquatic insects).

As we mentioned earlier, in the absence of the predator, the toxin-mediated predator-prey system (11) reduces to a one species toxin-dependent model which we developed in Huang et al. (2013). Therein, we considered the toxic effect of methylmercury on rainbow trout (*Oncorhynchus mykiss*) and obtained an approximate range for each model parameter. To parameterize the current model (11), we first apply the parameter estimates in Huang et al. (2013) to those predator-related parameters in the model (11). We then estimate the prey (aquatic insects)-related parameters using literature-derived experimental results.

3.1 Predator (Rainbow Trout)-Related Parameter Estimates

The parameter estimates in Huang et al. (2013) were given by certain ranges (intervals), and here we take the midpoint of the intervals as the corresponding parameter values and obtain:

- $\beta_2 = 33.41$ g/μg,
- $k_2 = 0.00398$ g/μg/day,
- $m_2 = 0.00057$ day^{-1},
- $a_2 = 0.1733$ L/g/day, and
- $\sigma_2 = 0.0062$ day^{-1}.

The carrying capacity of rainbow trout was estimated as $\eta = 0.00091$ g/L, hence we take the half-saturation constant $\eta = 0.000455$ g/L. The maximum growth rate of rainbow trout is estimated as 0.0047 day^{-1}. This corresponds to the term $\frac{\beta_1 \xi x}{\eta + x}$ in (11). For simplicity, letting $\frac{\beta_1 \xi x}{\eta + x} = 0.0047$ as $x \to \infty$, we get $\beta_1 \xi = 0.0047$ day^{-1}. It is commonly assumed that transfer efficiency between trophic level is 0.1 (Bax 1998). Letting $\beta_1 = 0.1$, we obtain $\xi = 0.047$ day^{-1}.

3.2 Prey (Aquatic Insects)-Related Parameter Estimates

It is difficult to find experimental results for one species of aquatic insects to estimate all model paprameters. In what follows, we choose data for several related species to roughly estimate the parameters in the model (11).

We chose caddisflies to estimate maximum reproduction rate α_1 and the natural mortality rate m_1 for the prey. By assuming 1:1 female-male ratio we chose the maximum reproduction rate of caddisflies $\alpha_1 = 800 \times 0.5 \times 0.01/365 = 4/365$ day^{-1}. We take the natural mortality rate $m_1 = 1/365$ day^{-1} by assuming that the average natural life span of caddisfly is 365 days.

We estimate the crowding effect parameter α_3 by using the estimated carrying capacity of aquatic insect populations in the literature (Gilpin and Ayala 1973). The carrying capacities of two species of *Drosophila* were estimated by fitting two analytic models to experimental data. The mean of the carrying capacities of two species, denoted by K, is 0.0011 g/L. We use the carrying capacity, K, to the crowding effect parameter α_3 as follows.

If there is no toxin or predation, the first equation of (11) becomes

$$\frac{dx}{dt} = \left(\frac{\alpha_1}{1+\alpha_3 x} - m_1 \right) x = \frac{(\alpha_1 - m_1)(1 - \frac{x}{K})x}{1+\alpha_3 x} \tag{12}$$

with

$$K = \frac{\alpha_1 - m_1}{m_1 \alpha_3} \tag{13}$$

Notice that $\alpha_1 - m_1$ is always positive from the estimated values for α_1 and m_1. It is not difficult to check that K plays the role of the carrying capacity with the logistic equation. Using the above equation and taking the estimates of α_1 and m_1 into account, we obtain $\alpha_3 = 1{,}330$ L/g.

Next we estimate the toxin-related parameters in the model (11) for aquatic insects. There is much evidence which indicates that aquatic insects are much less sensitive to mercury than rainbow trout. For instance, the maturity percentage and percentage of hatched eggs of the fruit fly are approximately 100% in 10 µg/L of mercury (Table 1 in Abnoos et al. 2013). An aquatic insect, the southern house mosquito (*Culex quinquefasciatus*: Diptera), still has high survivor rate at methylmercury concentrations as high as 1 µg/L (Table 1 in Jensen et al. 2007). However, for aquatic life, the Alberta provincial interim acute guideline is 0.002 μg/L and the chronic guideline is 0.001 µg/L (Alberta Environment 1999). Our findings in earlier work (Huang et al. 2013) estimated that the threshold value of methylmercury for rainbow trout extirpation is around 0.0045 µg/L. The toxic effect on the prey (aquatic insects) can be ignored if the external toxin level is sufficiently low such that the predator (rainbow trout) can survive. Therefore, we chose the parameter $\alpha_2 = 0$, which measures the effect of toxin on reproduction, and the parameter $k_1 = 0$, which measures the effect of toxin on mortality.

The uptake rate constants and depuration rates for mercury by four aquatic insect species (two caddisflies and two mayflies) were estimated by Xie et al. (2009). We chose the uptake coefficient of the prey (aquatic insects) to be the mean of the four uptake rate constants and obtain the estimate: $a_1 = 0.55$ in L/g/day. We chose depuration rate of aquatic insects to be the mean of the four depuration rates and obtain the estimate: $\sigma_1 = 0.12$ in day^{-1}.

We consider two predator-prey scenarios. Firstly, we assume that the small fish prey have the same vital rates and same sensitivity to mercury as the trout, and directly apply the results of model parameterization in Huang et al. (2013) to those prey-related parameters in the

model (11). Secondly, we regard rainbow trout as the predator and aquatic insects as the prey. We use the model parameterization in numerical simulations of model (11) in Sections 5 to 7. We list rainbow trout- and aquatic insect-related parameter estimates in Table 1.

Table 1. Rainbow trout- and aquatic insect-related parameters.

Symbols	Definitions	Estimates values
α_1	Maximum reproduction rate of aquatic insects	4 /365 day^{-1}
α_2	Effect of toxin on the growth of aquatic insects	0
α_3	Crowding effect of aquatic insects	1330 L/g
k_1	Effect of toxin on the mortality of aquatic insects	0
m_1	Natural mortality rate of aquatic insects	1/365 day^{-1}
ξ	Per capita feeding rate	0.047 day^{-1}
η	Half-saturation constant	0.000455 g/L
β_1	Reproduction efficiency of rainbow trout	0.1
β_2	Effect of toxin on the reproduction of rainbow trout	33.41 g/μg
k_2	Effect of toxin on the mortality of rainbow trout	0.00398 g/μg/day
m_2	Natural mortality rate of rainbow trout	0.00057 day^{-1}
a_1	Uptake coefficient for aquatic insects	0.55 L/g/day
σ_1	Depuration rate for aquatic insects	0.12 day^{-1}
a_2	Uptake coefficient for rainbow trout	0.1733 L/g/day
σ_2	Depuration rate for rainbow trout	0.0062 day^{-1}

4 MODEL ANALYSIS

We expect that the dynamics for the depuration due to metabolism of toxin will operate on a much faster time scale than the dynamics of population biomass growth. This means that the body burden equations may approach a quasi-equilibrium state where uptake of toxin and depuration balance out on a fast time scale. To investigate this process mathematically we define $\varepsilon = \alpha_1/\sigma_1$ to be a small parameter. For example, we find that $\varepsilon = 0.091$ from the parameter estimates for aquatic insects and rainbow trout (See Table 1).

4.1 Nondimensionalization and Non-negativity

To simplify the problem and facilitate analysis, we rescale the system (11) by introducing the nondimensional quantities

$$\begin{aligned}
&\tilde{x} = \alpha_3 x, \quad \tilde{y} = \frac{\alpha_3 \xi}{\alpha_1} y, \quad \tilde{u} = \alpha_2 u, \quad \tilde{v} = \beta_2 v, \quad \tilde{t} = \alpha_1 t \\
&\tilde{k}_1 = \frac{k_1}{\alpha_1 \alpha_2}, \quad \tilde{m}_1 = \frac{m_1}{\alpha_1}, \quad \tilde{\eta} = \alpha_3 \eta, \quad \tilde{\beta}_1 = \frac{\beta_1 \xi}{\alpha_1}, \quad \tilde{k}_2 = \frac{k_2}{\alpha_1 \beta_2}, \quad \tilde{m}_2 = \frac{m_2}{\alpha_1}, \\
&\tilde{T} = \frac{\alpha_2 a_1 T}{\sigma_1}, \quad c = \frac{a_2 \beta_2}{a_1 \alpha_2}, \quad \tilde{\sigma}_2 = \frac{\sigma_2}{\sigma_1}, \quad \tilde{\beta}_2 = \frac{\beta_2 \xi}{\alpha_2 \sigma_1}, \quad \varepsilon = \frac{\alpha_1}{\sigma_1}.
\end{aligned} \tag{14}$$

Dropping the tildes for notational simplicity, we rewrite the system (11) in its dimensionless form:

$$\begin{aligned}
\frac{dx}{dt} &= \left(\frac{\max\{0, 1-u\}}{1+x} - k_1 u - m_1 \right) x - \frac{xy}{\eta + x} \\
\frac{dy}{dt} &= \frac{\beta_1 xy \max\{0, 1-v\}}{\eta + x} - (k_2 v + m_2) y \\
\varepsilon \frac{du}{dt} &= T - u - \varepsilon \frac{\max\{0, 1-u\}}{1+x} u \\
\varepsilon \frac{dv}{dt} &= cT - \sigma_2 v + \left(\beta_2 u - \varepsilon \beta_1 \max\{0, 1-v\} v \right) \frac{x}{\eta + x}
\end{aligned} \tag{15}$$

We first show that solutions of system (15) behave in a biologically reasonable manner. That is, the population densities at any time, which are given by the solutions of the model at time t, are always nongeative but not arbitrarily large.

Theorem 4.1 Each component of the solution of system (15) with non-negative initial conditions remain bounded and non-negative for all $t > 0$.

See Appendix 1 for the proof.

4.2 Quasi-Steady System

Because the model (15) is a high dimensional system, the stability analysis of model (15) is challenging. We simplify it to a two-dimensional system via the quasi-steady state approximation. Since ε is introduced as a small parameter, letting $\varepsilon \to 0$ in (15), we have

$$u = T, \quad v = \frac{cT}{\sigma_2} + \frac{\beta_2 T}{\sigma_2} \frac{x}{\eta + x} \tag{16}$$

Substituting (16) into the first and second equations of (15), we obtain the following quasi-steady state system

$$\begin{aligned}\frac{dx}{dt} &= \left(\frac{\max\{0,1-T\}}{1+x} - k_1 T - m_1\right)x - \frac{xy}{\eta + x} \\ \frac{dy}{dt} &= \frac{\beta_1 xy \max\{0,1-\dfrac{cT}{\sigma_2} - \dfrac{\beta_2 T}{\sigma_2}\dfrac{x}{\eta+x}\}}{\eta + x} - \left[k_2\left(\frac{cT}{\sigma_2} + \frac{\beta_2 T}{\sigma_2}\frac{x}{\eta+x}\right) + m_2\right]y\end{aligned} \tag{17}$$

Throughout this paper, we assume that

$$m_1 < 1 \quad \text{and} \quad T < \min\{1, \sigma_2/c\} \tag{18}$$

These mean that the natural loss rate of the prey biomass due to death is less than its maximum gain rate due to birth and growth, and that the environmental toxin levels are low enough that the prey can reproduce and grow. If either of these conditions are violated then the prey cannot persist and both prey and predator are extirpated (Appendix 1).

We also assume that

$$m_2 < \beta_1 \tag{19}$$

This means that the natural loss rate of the predator biomass due to death is less than its maximum gain rate due to birth and growth. If this condition is violated then the predator is extirpated, and the system (17) reduces to a one species model (Appendix 1).

With these assumptions, the nondimensionalized system (17) can be rewritten as

$$\begin{aligned}\frac{dx}{dt} &= f(x) - \varphi(x)y \\ \frac{dy}{dt} &= g(x)y\end{aligned} \tag{20}$$

with

$$f(x) = \left(\frac{1-T}{1+x} - k_1 T - m_1\right)x \tag{21}$$

$$\varphi(x) = \frac{x}{\eta + x} \tag{22}$$

and

$$g(x) = \beta_1 \varphi(x) \max\left\{0, 1 - \frac{cT}{\sigma_2} - \frac{\beta_2 T}{\sigma_2}\varphi(x)\right\} - \frac{k_2 cT}{\sigma_2} - \frac{k_2 \beta_2 T}{\sigma_2}\varphi(x) - m_2 \tag{23}$$

4.2.1 *Existence of Equilibria*

To investigate the long-term behavior of the system (20), we look for the steady states (equilibria) where neither, one or both species survive. These can be found by finding the intersections of prey and predator zero-growth isoclines (or null-clines), where either prey or predator growth rate is zero. We summarize the existence of extirpation and coexistence equilibria and corresponding conditions required in Table 2. The detailed discussion is provided in Appendix 1.

Table 2. The existence of equilibria.

	Mathematical conditions	Equilibria	Biological interpretations
A	$T > T_0^*$	$E_{0,1} = (0,0)$	System only has extirpation equilibrium if the external toxin level is high enough that neither population persists (top left panel of Figure 2)
B	$T < T_0^*$	$E_{0,1}$ and $E_{0,2} = (x_0,0)$	System has extirpation and prey-only equilibria if the toxin level is low enough that prey can survive but predator cannot (top right panel of Figure 2)
C	$\max\{T_2^*, T_3^*\} > \min\{T_0^*, T_1^*\}$ $T < \min\{T_0^*, T_1^*, T_2^*\}$ $\eta < \frac{x_0}{\varphi_1} - x_0$	$E_{0,1}$, $E_{0,2}$, and $E_1 = \left(x_1, \frac{f(x_1)}{\varphi(x_1)}\right)$	A coexistence equilibrium appears if the toxin level and the half-saturation constant are sufficiently low such that both populations can coexist (bottom panel of Figure 2)
D	$\max\{T_2^*, T_3^*\} < \min\{T_0^*, T_1^*\}$ $\max\{T_2^*, T_3^*\} < T < \min\{T_0^*, T_1^*\}$ $\eta < \frac{x_0}{\varphi_2} - x_0$	$E_{0,1}, E_{0,2}$, E_1, and $E_2 = \left(x_2, \frac{f(x_2)}{\varphi(x_2)}\right)$	System has two coexistence equilibria when the toxin level lies within a certain range and the half-saturation constant is sufficiently low such that both populations can coexist (top panel of Figure 4)

In Table 2,

$$T_0^* = \frac{1-m_1}{k_1+1}$$

$$T_1^* = \frac{\beta_1\sigma_2(\beta_1 c + k_2\beta_2 + 2\beta_2 m_2) - \beta_1\sigma_2\sqrt{4\beta_2(\beta_1 c + \beta_2 m_2)(m_2 + k_2)}}{(\beta_1 c - k_2\beta_2)^2}$$

$$T_2^* = \frac{(\beta_1 - m_2)\sigma_2}{\beta_1\beta_2 + \beta_1 c + k_2\beta_2 + k_2 c}$$

$$T_3^* = \frac{\beta_1\sigma_2}{\beta_1 c + k_2\beta_2 + 2\beta_1\beta_2}$$

$$x_0 = (1-T)/(k_1 T + m_1) - 1$$

$$x_{1,2} = \frac{\eta\varphi_{1,2}}{1-\varphi_{1,2}}$$

$$\varphi_{1,2} = \frac{\beta_1(\sigma_2 - cT) - k_2\beta_2 T \mp \sqrt{\Delta}}{2\beta_1\beta_2 T}$$

$$\Delta = [\beta_1(\sigma_2 - cT) - k_2\beta_2 T]^2 - 4\beta_1\beta_2 T(k_2 cT + m_2\sigma_2)$$

As shown in Table 2, the conditions of the existence of equilibria are given by the restriction conditions with respect to the toxin level in the environment T and the half-saturation constant η that is related to the capture efficiency. Note that the quantities $T_0^*, T_1^*, T_2^*, T_3^*$ are not dependent on T and η, if we assume that these quantities are constant, then the magnitudes of toxin concentration in the environment and half-saturation constant determine the number of equilibria.

It is worth mentioning that in row C of Table 2, the condition $T < \min\{T_0^*, T_1^*, T_2^*\}$) guarantees that $x_0 > 0$ and $\varphi_1 < 1$, hence $x_0/\varphi_1 - x_0 > 0$ and the condition $\eta < x_0/\varphi_1 - x_0$ plays a role.

Row D of Table 2 implies that very strict conditions are required to guarantee the existence of another coexistence equilibrium E_2. The first condition, $\max\{T_2^*, T_3^*\} < \min\{T_0^*, T_1^*\}$ allows for the possibility that the toxin level lies within the range ($\max\{T_2^*, T_3^*\} < T < \min\{T_0^*, T_1^*\}$). The condition $T < \min\{T_0^*, T_1^*\}$ guarantees that $x_0 > 0$ and $\varphi_2 > 0$. The condition $T > \max\{T_2^*, T_3^*\}$ guarantees that $\varphi_2 < 1$. Thus, $x_0/\varphi_2 - x_0 > 0$ and it is possible that the last condition $\eta < x_0/\varphi_2 - x_0$ can be realized.

Recall that the half saturation constant $\eta = 1/(\gamma h)$ (see equations (8) and (9)), where γ is the encounter rate (or capture efficiency) and h is the handling time). If we assume that the handling time h is constant, then a small half-saturation constant means a high capture efficiency and a large half-saturation constant corresponds to a low capture efficiency. Thus, the conditions $\eta < x_0/\varphi_1 - x_0$ and $\eta < x_0/\varphi_2 - x_0$ in Table 2 can be interpreted as the predator requiring sufficientlyhigh capture efficiencies.

Figure 1 illustrates several possible null-clines where either the prey or predator growth rate is zero. At the intersection of the prey and predator null-clines, we find equilibria points. In all panels of Figure 1, the conditions $\max\{T_2^*, T_3^*\} < \min\{T_0^*, T_1^*\}$ and $\max\{T_2^*, T_3^*\} < T < \min\{T_0^*, T_1^*\}$ are satisfied, hence the boundary equilibria $E_{0,1}$ and $E_{0,2}$ always exist.

As shown in Figure 1, depending on the values of η which determine the intersections of the null-clines, the system may have zero, one or two coexistence equilibria. In the left panel, $\eta > x_0/\varphi_1 - x_0$, the system has no coexistence equilibrium. In the middle panel, $x_0/\varphi_2 - x_0 < \eta < x_0/\varphi_1 - x_0$, the system has only one coexistence equilibrium E_1. In the right panel, $\eta < x_0/\varphi_2 - x_0$, the system has two coexistence equilibria E_1 and E_2.

4.2.2 *Stability of Equilibria*

To analyze the stability of an equilibrium, we may use the Jacobian matrix if the eigenvalues of the Jacobian evaluated at the equilibrium have nonzero real parts. The Jacobian matrix for system (20) is

$$J = \begin{pmatrix} f'(x) - \varphi'(x)y & -\varphi(x) \\ g'(x)y & g(x) \end{pmatrix} \tag{24}$$

To assess the stability of extirpation equilibrium $E_{0,2}$, we need another quantity measuring the external toxin level, that is,

$$T_4^* := \frac{\beta_1\sigma_2(\beta_1 c + k_2\beta_2 + 2\beta_2 m_2) + \beta_1\sigma_2\sqrt{4\beta_2(\beta_1 c + \beta_2 m_2)(m_2 + k_2)}}{(\beta_1 c - k_2\beta_2)^2} \tag{25}$$

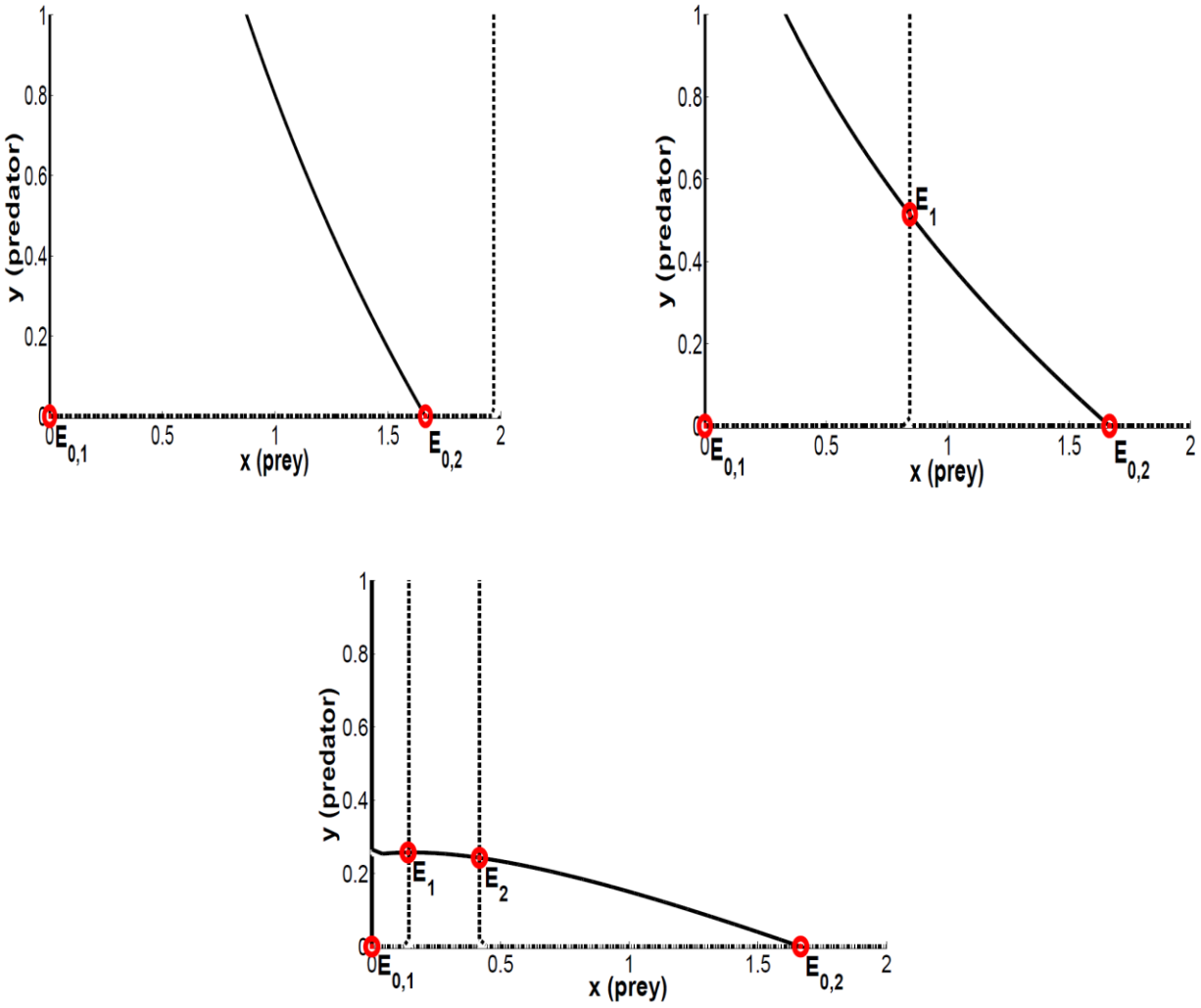

Figure 1. Possible null-clines for system (20) with the parameter values, T = 0.2, $k_1 = 1$, $m_1 = 0.1$, $\beta_1 = 1$, $\beta_2 = 4$, c = 1.5, $\sigma_2 = 1$, $k_2 = 0.2$, $m_2 = 0.02$, and different values of η: (Top left) $\eta = 7$, (Top right) $\eta = 3$, (Bottom) $\eta = 0.5$.
Solid curves are the prey null-clines and dashed lines are the predator null-clines. Circles indicate equilibrium points.

We make the following conclusions regarding the stability of extirpation equilibria. The proof is provided in Appendix 1.

Theorem 4.2 (1) The extirpation equilibrium $E_{0,1} = (0,0)$ is globally asymptotically stable if $T > (1-m_1)/(k_1+1)$. $E_{0,1}$ is an unstable saddle point if $T < (1-m_1)/(k_1+1)$.

(2) The prey-only equilibrium $E_{0,2} = (x_0, 0)$ is locally asymptotically stable if one of the following conditions is satisfied:

1. $T_1^* < T < T_4^*$,
2. $T < T_1^*$ and $\eta > x_0/\varphi_1 - x_0$, and
3. $\max\{T_2^*, T_3^*\} < \min\{T_0^*, T_1^*\}$, $\max\{T_2^*, T_3^*\} < T < \min\{T_0^*, T_1^*\}$, and $\eta < \frac{x_0}{\varphi_2} - x_0$.

The mathematical results in Theorem 4.2 can be interpreted as follows:

1. If the external toxin level is sufficiently high ($T > (1-m_1)/(k_1+1)$), then from Table 2, we see that the extirpation equilibrium $E_{0,1} = (0,0)$ is the only stable state of the system. Hence the solutions of the system must tend towards this equilibrium, which means that both populations are extirpated.
2. In the following scenarios, the prey can survive but the predator cannot:
   a. If the external toxin level lies falls within a certain range ($T_1^* < T < T_4^*$), only the prey can persist because the toxin level is sufficiently low ($T < T_4^*$). However, it is too high ($T > T_1^* < T$) for the predator to persist.
   b. The toxin level is sufficiently low ($T < T_1^*$) such that the prey can persist. The predator is extirpated because the half-saturation constant is too large ($\eta > x_0/\varphi_1 - x_0$)(i.e., the capture efficiency is too low), which leads to a low growth rate of the predator.

For coexistence equilibria, we have the following results. The proof is provided in Appendix 1.

Theorem 4.3 The equilibrium E_1 is locally asymptotically stable if either $\eta \le 1$ or both conditions $0 < \eta < 1$ and $k_1 T + m_1 > \frac{(1-\eta)(1-T)}{(1+x_1)^2}$ are satisfied. The equilibrium E_2 is always an unstable saddle point.

Combining the condition of existence of equilibria (Table 2) and the condition of stability (Theorem 4.3) of coexistence equilibrium E_1, we find that the both populations can coexist in the following two scenarios:

1. Both populations coexist at the equilibrium E_1 if the half-saturation constants lie within a certain range ($1 \le \eta < \frac{x_0}{\varphi_1} - x_0$) (i.e., the capture efficiency lies within a certain range) and the toxin-dependent mortality rate of the prey is sufficiently low ($k_1 T + m_1 < (1-T)(1-\varphi_1)$). This is because of the condition of the existence of E_1,

$\eta < \frac{x_0}{\varphi_1} - x_0$, the condition $\eta \geq 1$ plays a role only when $\frac{x_0}{\varphi_1} - x_0 > 1$, which is equivalent to $k_1 T + m_1 < (1-T)(1-\varphi_1)$.

2. Both species coexist at the equilibrium E_1 if the half-saturation constants are sufficiently small ($0 < \eta < 1$)(i.e., the capture efficiencies are sufficiently high) and the prey has an intermediate mortality rate ($\frac{(1-\eta)(1-T)}{(1+x_1)^2} < k_1 T + m_1 < \frac{1-T}{1+x_1}$). This is because we can equivalently rewrite the condition of the existence of E_1, $\eta < \frac{x_0}{\varphi_1} - x_0$, as $k_1 T + m_1 < \frac{1-T}{1+x_1}$. Thus, from Theorem 4.3, we see that both populations are able to coexist at E_1 if both conditions $0 < \eta < 1$ and $\frac{(1-\eta)(1-T)}{(1+x_1)^2} < k_1 T + m_1 < \frac{1-T}{1+x_1}$ are satisfied.

From the discussion in this section, we know that the quasi-steady system (17) has at most two boundary equilibria $E_{0,1}$(both extirpation) $E_{0,1}$(only prey) and two interior (coexistence) equilibria E_1 and E_2 which depends on the intersections of the null-clines. We also show that the stability of these equilibria can be guaranteed if the toxin level T and half saturation constant η satisfy certain conditions.

5 NUMERICAL OBSERVATIONS OF ASYMPTOTIC DYNAMICS

In this section, we show a variety of long-term asymptotic dynamics that the system (17) may exhibit based on the results of the existence and stability of equilibria. To do so, we plot phase portraits (Figure 2) using the existing open MATLAB program pplane8.m by choosing different parameter values. The phase portraits illustrate different types of eventual behavior of the populations.

As we observe from Figures 2 to 5, the asymptotic dynamics (i.e., the eventual behavior of the populations) can be grouped into six general structures:

- extirpation of both species (extirpation equilibrium $E_{0,1}$ is globally asymptotically stable);
- prey-only extirpation (extirpation equilibrium $E_{0,2}$ is globally asymptotically stable);
- coexistence at an interior equilibrium point (coexistence equilibrium $E_{0,2}$ is globally asymptotically stable);
- coexistence with periodic population oscillations which decrease in amplitude as T increases (system has a globally asymptotically stable limit cycle);

- bistability where both the extirpation equilibrium $E_{0,2}$ and the interior equilibrium point E_1 are locally asymptotically stable; and
- bistability where the system has a stable limit cycle and a stable prey-only equilibrium.

Figure 2 shows several types of global stability:

- The top left panel of Figure 2 shows that both species are extirpated when the toxin level in the environment T is high and leads to high population mortality rates and low population growth rates.
- The top right panel of Figure 2 shows that the prey excludes the predator when half saturation constant η is large. In this scenario, the system has no interior equilibrium (i.e., both species cannot coexist) even though the toxin level T is low.
- In the bottom panel of Figure 2, both species are able to coexist at interior equilibrium because the predation benefits the predator but is not too harmful to the prey when the toxin level T is low.

Figure 3 shows that both species coexist but the population levels oscillate periodically around the unstable interior equilibrium E_1. With limit-cycle oscillations, Figure 3 clearly illustrates that the prey is reduced to extremely low levels yet recovers while the predator biomass remains above a certain level even at the lowest prey population level.

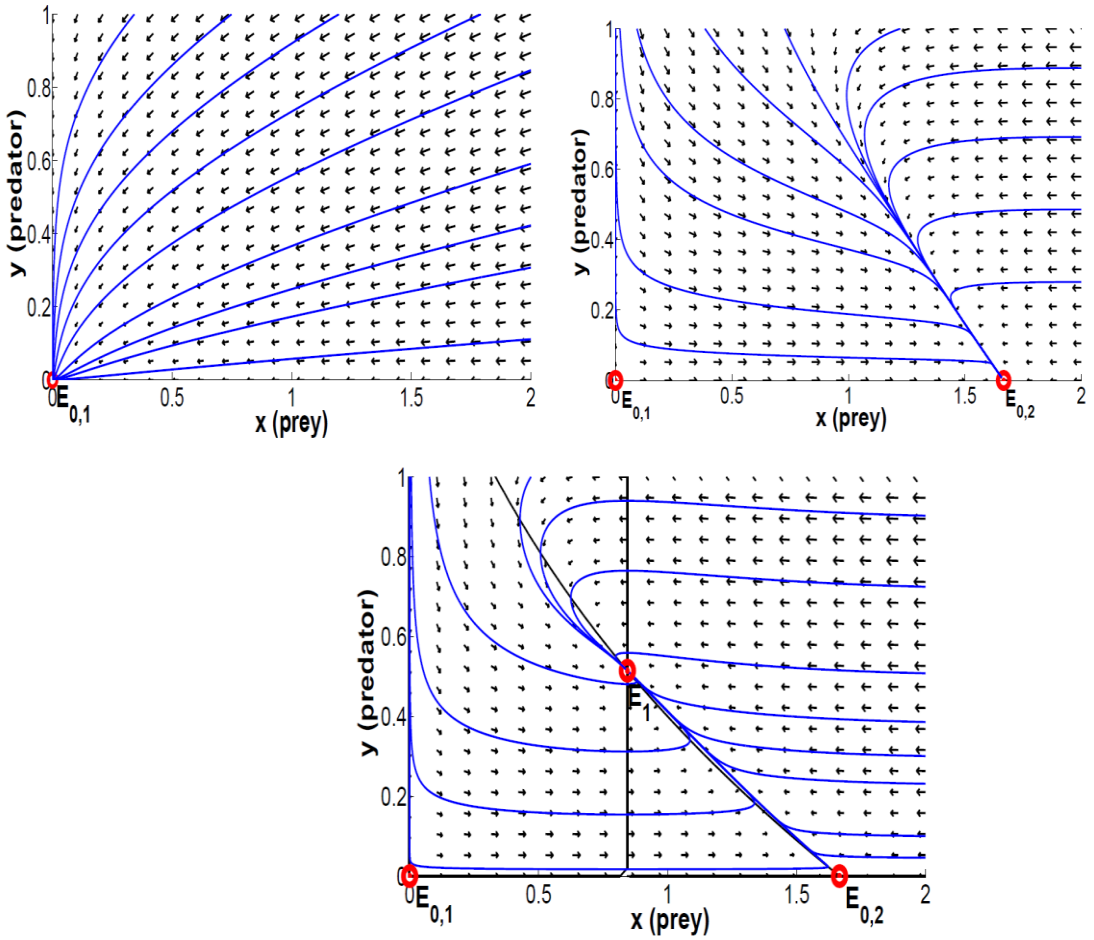

Figure 2. Phase portraits of the predator density versus the prey density with various values of T and η.

Units for prey and predator densities are dimensionless as described in equation (4.1).

In each case, extirpation or coexistence equilibria are globally asymptotically stable (GAS).

(Top left) Extirpation equilibria $E_{0,1}$ = (0, 0) is GAS with T = 0.5 and η = 1.

(Top right) Extirpation equilibria $E_{0,2}$ = (1.67, 0) is GAS with T = 0.2 and η = 7.

(Bottom) Coexistence equilibrium E_1 = (0.84, 0.51) is GAS with T = 0.2 and η = 3.

The other parameters for these three panels are the same as those in Figure 1.

Circles indicate equilibrium points.

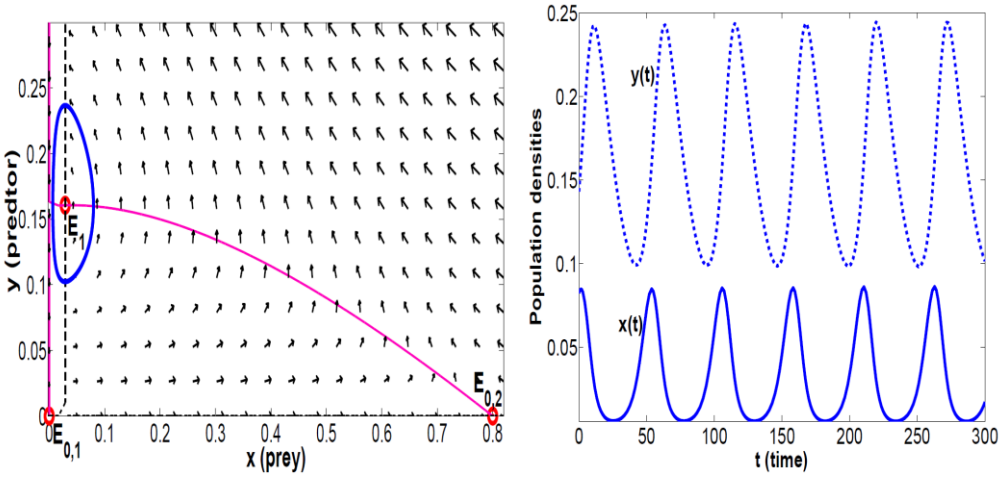

Figure 3. System shows globally stable limit cycle.
(Left) Phase portraits of the predator density over the prey density.
(Right) Solution curves (solid curve represents the prey density and dashed curve is the predator density).
System has only one coexistence equilibrium E_1 = (0.028, 0.16) which is an unstable spiral source, both boundary equilibria $E_{0,1}$ = (0, 0) and $E_{0,2}$ = (0.80, 0) are unstable saddle points.
Circles indicate equilibrium points.
T = 0.1, η = 0.4, m_1 = 0.4, other parameters are the same as those in Figure 1.

In the previous section, we proved that E_2 is always an unstable saddle point when the system has two interior equilibria E_1 and E_2. Figures 4 and 5 show that the system has two alternative stable states (bistability) when both interior equilibria E_1 and E_2 exist. The initial conditions determine which steady state the system will tend towards. The stable manifolds of the unstable interior equilibrium E_2 indicates the edges of the basin of attraction for each steady state. The bistability shown by Figure 4 means that either the prey excludes the predator or both species coexist at the interior equilibrium E_1 depending on the initial population levels.

Figure 5 shows another type of bistability. That is, the system has two alternate stable states: either the prey excludes the predator or both species coexist but with oscillating population levels. When both species coexist but with oscillating population levels, both equilibria E_1 and E_2 are unstable. The system will tend towards a stable limit cycle only when the initial populations fall in a small domain which is the basin of attraction of the limit cycle.

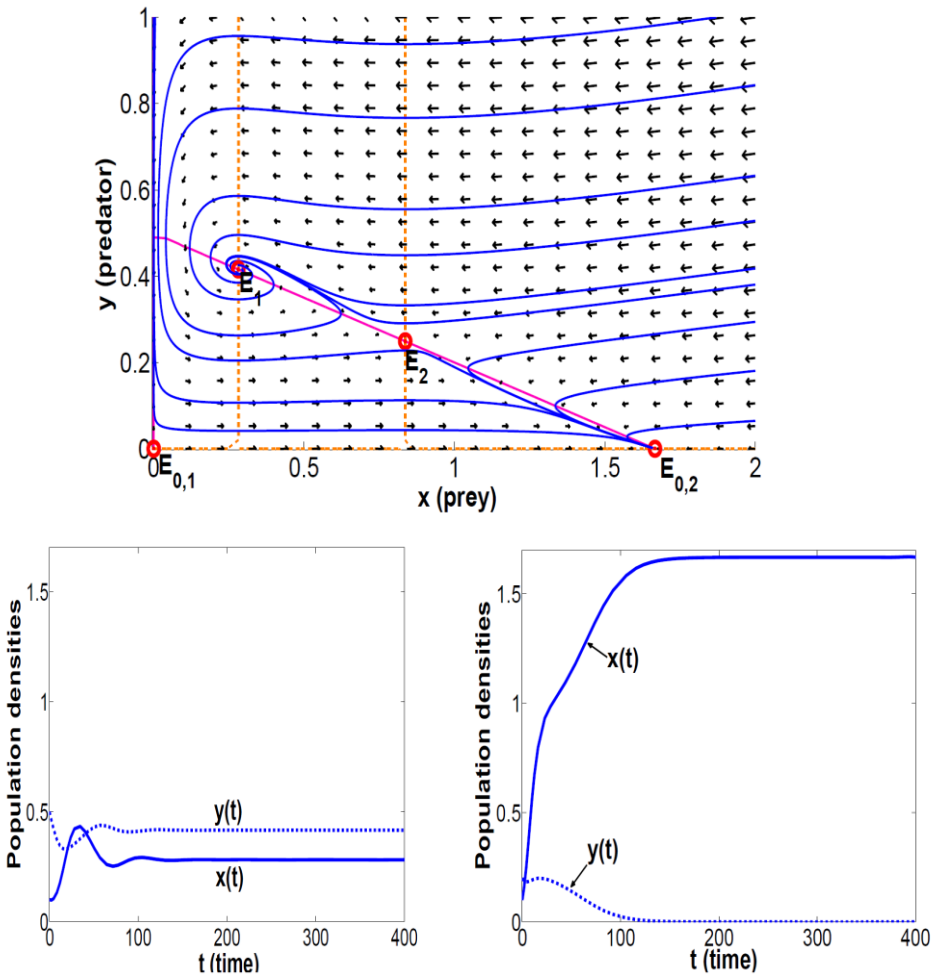

Figure 4. System shows bistability with a locally stable coexistence equilibrium E_1 and a locally stable extirpation equilibrium $E_{0,2}$.

The trajectories either converge to E_1 or converge to $E_{0,2}$. Circles indicate equilibrium points.

(Top row) Phase portraits of the predator density versus the prey density. (Bottom row) Solution curves. The solution with initial condition (0.1,0.2) converges to E_1 (Bottom left). The solution with initial conditions (0.1, 0.5) converges to $E_{0,2}$ (Bottom right). The system has two coexistence equilibria E_1 = (0.28, 0.42) which is stable spiral node and E_2 = (0.84, 0.25) which is an unstable saddle point, boundary equilibria $E_{0,1}$ = (0, 0) is an unstable saddle point, boundary equilibria $E_{0,2}$ = (1.67, 0) is stable node. The parameters: η = 1, other parameters are the same as those in Figure 1.

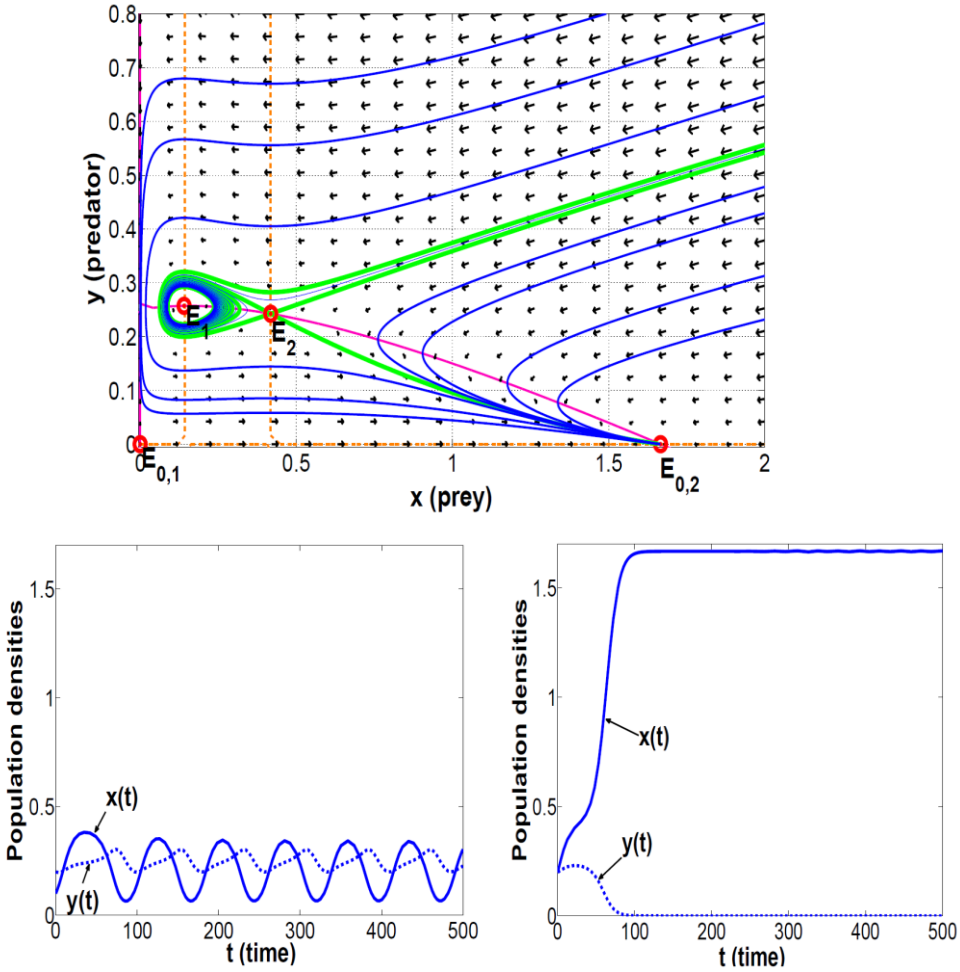

Figure 5. System shows bistability with a locally stable limit cycle and a locally stable extirpation equilibrium $E_{0,2}$.

The stable manifold of the limit cycle and the stable manifold of $E_{0,2}$ are depicted by bold curve. (Top row) Phase portraits of the predator density versus the prey density. Circles indicate equilibrium points. (Bottom row) Solution curves. The solution with initial conditions (0.1, 0.2), which belongs to the stable manifold of the limit cycle, oscillates periodically as shown by the limit cycle (Bottom left). The solution with initial conditions (0.2, 0.2), which is outside the limit cycle, converges to $E_{0,2}$ = (1.67, 0) (Bottom right). The system has two coexistence equilibria E_1 = (0.14, 0.26) which is an unstable spiral source and E_2 = (0.42, 0.24) which is an unstable saddle point. Boundary equilibrium $E_{0,0}$ = (0, 0) is an unstable saddle point, boundary equilibrium $E_{0,2}$ = (1.67, 0) is a stable node. The parameters: η = 0.5, other parameters are the same as those in Figure 1.

6 EFFECT OF TOXIN ON POPULATION DYNAMICS

The goal of this section is to study how the balance of classical predator-prey dynamics will change as the concentration of a toxin increases from zero to higher level. To do so, we first present the results of classical predator-prey dynamics associated with our toxin-mediated predator-prey dynamics. We then plot bifurcation dynamics for the toxin-mediated system by regarding the external toxin level T as a bifurcation parameter. The bifurcation figures will clearly illustrate how the external toxin changes the long-term asymptotic behavior of the prey and the predator.

6.1 A Traditional Predator-Prey System

If there is no toxin ($T = 0$), then system (17) reduces to the following prey-predator system:

$$\begin{aligned} \frac{dx}{dt} &= \left(\frac{1}{1+x} - m_1\right)x - \frac{xy}{\eta + x} \\ \frac{dy}{dt} &= \frac{\beta_1 xy}{\eta + x} - m_2 y \end{aligned} \tag{26}$$

Regarding the existence and stability of the system (26), we have the following results:

Lemma 6.1 Let $0 < m_1 < 1$ and $m_2 < \beta_1$.

(i) The boundary equilibrium $E_{0,2} = (1/m_1 - 1, 0)$ exists. $E_{0,2}$ is globally asymptotically stable if $\eta > \dfrac{(1-m_1)(\beta_1 - m_2)}{m_1 m_2}$.

(ii) If $\eta < \dfrac{(1-m_1)(\beta_1 - m_2)}{m_1 m_2}$, then system (26) has only one interior equilibrium

$$E_1 = \left(\frac{m_2\eta}{\beta_1 - m_2}, \frac{\eta\beta_1[(1-m_1)(\beta_1 - m_2) - m_1 m_2 \eta]}{(\beta_1 - m_2)(\beta_1 - m_2 + m_2\eta)}\right)$$

Assume that the interior equilibrium E_1 exists, then E_1 is globally asymptotically stable if either $\eta \geq 1$ or both the conditions $0 < \eta < 1$ and $\dfrac{(1-\eta)(\beta_1 - m_2)^2}{(\beta_1 - m_2 + m_2\eta)^2} < m_1$ are satisfied.

We interpret the mathematical results in Lemma 6.1 as follows. First of all, the condition $0 < m_1 < 1$ means that the loss rate of the prey biomass due to death is less than its maximum gain rate. If this condition is violated then both populations are extirpated. The condition $m_2 < \beta_1$ means that the loss rate of the predator biomass is less than its maximum gain rate. If this condition is violated then the predator is extirpated and the system (26) reduces to a single species model.

If the half-saturation constant is sufficiently large ($\eta > \frac{(1-m_1)(\beta_1 - m_2)}{m_1 m_2}$)(i.e., the capture efficiency is sufficiently low, assuming that the handling time is constant), then the prey-only equilibrium $E_{0,2}$ is stable, which means that the prey persists and the predator is eventually extirpated.

Combining the conditions of existence and stability of coexistence equilibrium E_1, we find that the both species coexist in the following two scenarios:

1. Both populations coexist at the equilibrium E_1 when the half-saturation constant is relatively large ($1 \le \eta < \frac{(1-m_1)(\beta_1 - m_2)}{m_1 m_2}$)(i.e., the capture efficiency is relatively low) and the mortality rate of the predator is sufficiently low ($m_2 < (1-m_1)\beta_1$). This is because if $1 < \frac{(1-m_1)(\beta_1 - m_2)}{m_1 m_2}$, which is equivalent to $m_2 < (1-m_1)\beta_1$, then both populations are able to coexist if the condition $1 \le \eta < \frac{(1-m_1)(\beta_1 - m_2)}{m_1 m_2}$ is also satisfied.
2. Both populations coexist at the equilibrium E_1 if the half-saturation constant is sufficiently small ($0 < \eta < 1$)(i.e., the capture efficiency is sufficiently high) and the prey has an intermediate mortality rate ($\frac{(1-\eta)(\beta_1 - m_2)^2}{(\beta_1 - m_2 + m_2\eta)^2} < m_1 < \frac{\beta_1 - m_2}{\beta_1 - m_2 + m_2\eta}$). This is because we can equivalently rewrite the condition of the existence of E_1, $\eta < \frac{(1-m_1)(\beta_1 - m_2)}{m_1 m_2}$, as $m_1 < \frac{\beta_1 - m_2}{\beta_1 - m_2 + m_2\eta}$, then both populations are able to coexist at E_1 if both conditions $0 < \eta < 1$ and $\frac{(1-\eta)(\beta_1 - m_2)^2}{(\beta_1 - m_2 + m_2\eta)^2} < m_1 < \frac{\beta_1 - m_2}{\beta_1 - m_2 + m_2\eta}$ are satisfied.

Lemma 6.2 *If* $0 < \eta < 1$ *and* $m_1 < \frac{(1-\eta)(\beta_1 - m_2)^2}{(\beta_1 - m_2 + m_2\eta)^2}$, then the system has a coexistence equilibrium E_1 which is unstable, and the system (26) possesses a unique limit cycle which is stable.

Lemma 6.2 implies that both species coexist, but their densities fluctuate periodically if the half-saturation constant is sufficiently small ($0 < \eta < 1$)(i.e., the capture efficiency is sufficiently high) and the mortality rate of the prey is sufficiently low ($m_1 < \frac{(1-\eta)(\beta_1 - m_2)^2}{(\beta_1 - m_2 + m_2\eta)^2}$).

Similar to most standard predator-prey systems, system (26) possesses three possible globally asymptotically stable states: prey only, coexistence at an equilibrium point, and coexistence at a limit cycle.

6.2 Dependence of Stable Population Density on External Toxin

In what follows, we are concerned with how different toxin concentrations in the aquatic environment affect the predator-prey dynamics. To this end, we turn to the toxin-mediated system (20) and analyze the sensitivity of asymptotically stable states (equilibria) with respect to toxin level T. This sensitivity analysis illustrates how the stable densities of prey and predator vary when the toxin level in the environment increase from zero to a higher level. Mathematically, this can be done by treating stable equilibria (including the stable prey and predator densities) as a function of T, then calculating the rate of change of stable prey and predator densities with respect to T (see Appendix 1).

We know from Table 2 and Theorem 4.2 that system (20) has a stable prey-only equilibrium $E_{0,2} = x_0, 0$ if external toxin levels are sufficiently high. The results of our sensitivity analysis imply that the prey density x_0 decreases as the toxin level T increases. That is, high concentrations of toxin in the environment are always harmful to the prey. If the external toxin levels are sufficiently low, say $T < \min\{T_0^*, T_1^*, T_2^*\}$ (Table 2), and the half-saturation constants satisfy certain conditions (Table 2 and Theorem 4.3), then the system has a stable coexistence equilibrium $E_1 = (x_1, y_1)$. For this scenario, our analysis shows that y_1 is always a decreasing function of T, which means that the toxin is always harmful to the predator, increasing toxin levels lead to decreasing predator density. However, the toxin affects the asymptotic prey density in a different way: x_1 increases as the toxin level T increases from 0 to $\min\{T_0^*, T_1^*, T_2^*\}$ until the system shifts from the stable coexistence state to the prey-only state when the toxin level reaches the threshold value $\min\{T_0^*, T_1^*, T_2^*\}$. This threshold value determines whether a given toxin level is beneficial or harmful to the prey. The bifurcation dynamics shown in Figure 6 in the next subsection illustrates our results of asymptotic analysis regarding the relationship between the stable population density and the toxin level in the environment.

6.3 Bifurcation Dynamics

To further understand the effects of the toxin on predator-prey dynamics, next we plot the bifurcation dynamics of the system with respect to the toxin concentration T. In particular, we chose a set of parameters such that both species coexist at an interior equilibrium or limit cycle when there is no toxin ($T = 0$). We then examine how these stable population densities will vary as T increases from zero to higher concentration.

Figures 6 to 9 illustrate that the toxin concentration in the environment affects the population dynamics in many different ways. In Figure 6, when $T = 0$, both species coexist at interior equilibrium. As T increases but still at a low concentration, the prey benefits since the stable predator density decreases. That is, contaminant effects on predators release the prey from

predation, which lead to increased abundance of the prey. As T continues to increase , the stable prey level decreases and the predator is extirpated. Finally, if we increase T further, both species are extirpated. The results indicate that the low toxin concentration provides a benefit to the prey by reducing the predator abundance.

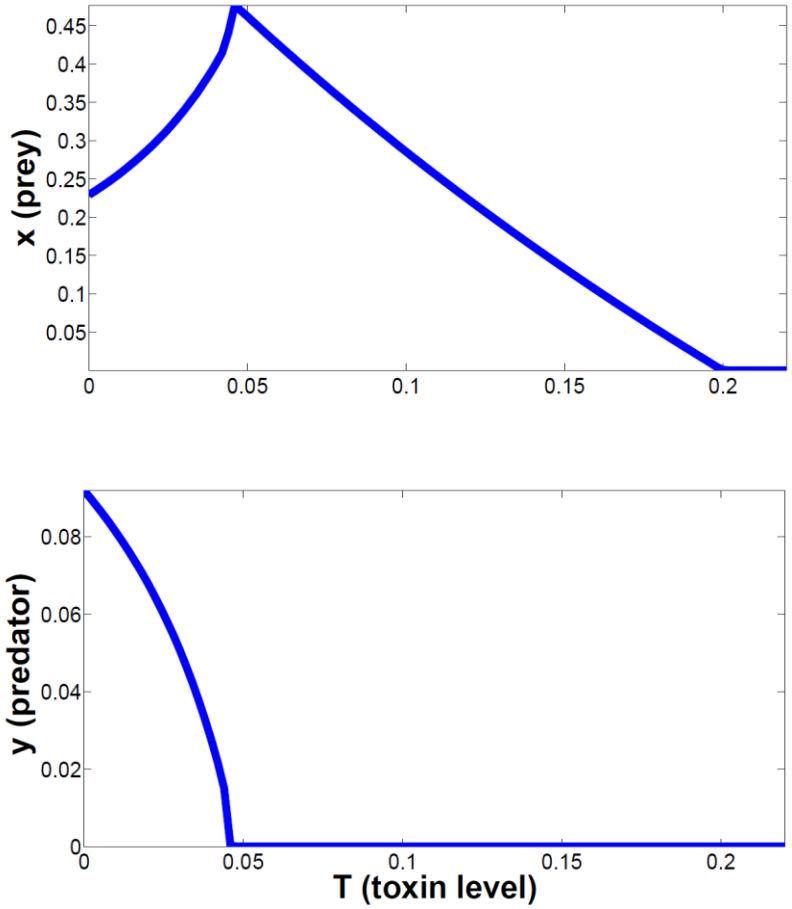

Figure 6. Bifurcation diagram with respect to toxin level T – both species coexist at interior equilibrium.

Parameters: $m_1 = 0.6$, $\eta = 0.2$, $\beta_1 = 0.75$, $m_2 = 0.4$, $k_1 = 1$, $k_2 = 1$, $\sigma_2 = 1$, $\beta_2 = 1$, $c = 1$. Here, $T_0^* = 0.200$, $T_1^* = 0.147$, $T_2^* = 0.2$, $T_3^* = 0.2$ (see Table 2).

In Figure 7, when $T = 0$, both species coexist but oscillate around an unstable interior equilibrium. As T increases but still at low concentration, population densities oscillate but with decreasing amplitudes until they reach a stable state at an interior equilibrium. As we continue to increase T, further similar dynamics to those in Figure 6 are displayed. The unstable asymptotic behavior of the populations can be stabilized by increasing T.

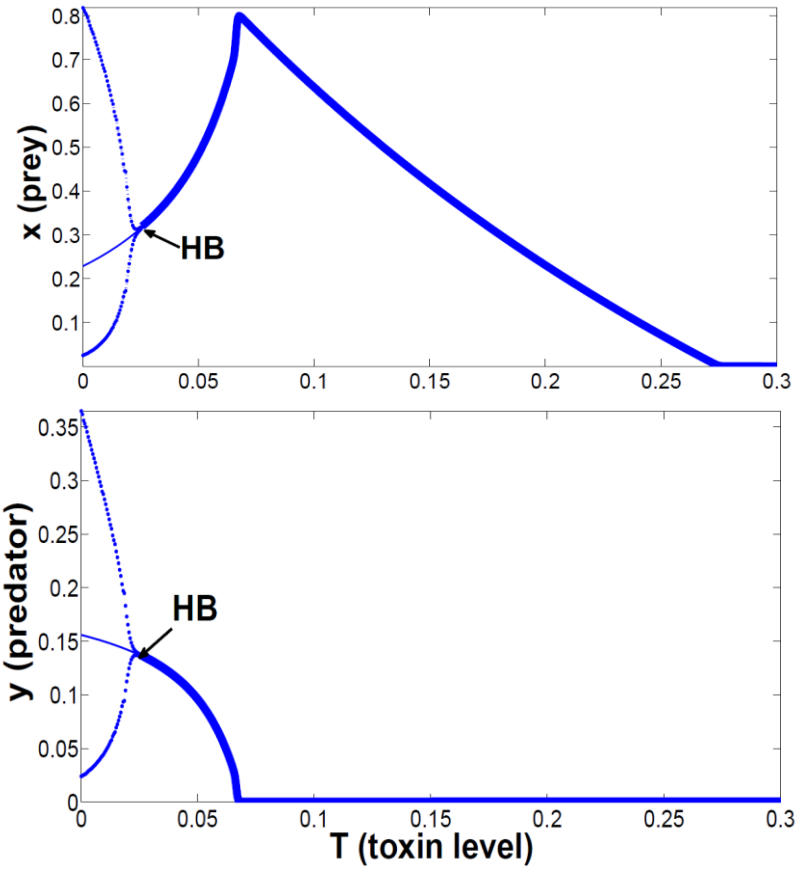

Figure 7. Bifurcation diagram with respect to toxin level – both species coexist but oscillate around an unstable interior equilibrium.
This shows the highest and lowest values of x-coordinates and y-coordinates of stable limit cycles (dot curves), x-coordinates and y-coordinates of unstable coexistence equilibria (thin solid curves), x-coordinates and y-coordinates of stable coexistence equilibria and prey-only equilibria (thick solid curves).
HB: (super-critical) Hopf bifurcation. $m_1 = 0.45$. Other parameters are the same as those in Figure 6.
Here, $T_0^* = 0.275$, $T_1^* = 0.147$, $T_2^* = 0.100$, $T_3^* = 0.231$ (see Table 2).

Figures 8 and 9 show that the populations have alternative stable states over a certain range of T. In Figure 8, the types of stable states vary in order as T increases. When $T = 0$, both species coexist at an interior equilibrium. As T increases but at low concentration, it benefits the prey since the stable predator density decreases. As we continue to increase T, the population densities tend to move to alternative stable states: either to a prey-only state or coexistence at an interior equilibrium. As T increases further, the coexistence state disappears and the population densities tend to a prey-only state. Finally, if we increase T even further, the stable prey density decreases until both species are extirpated.

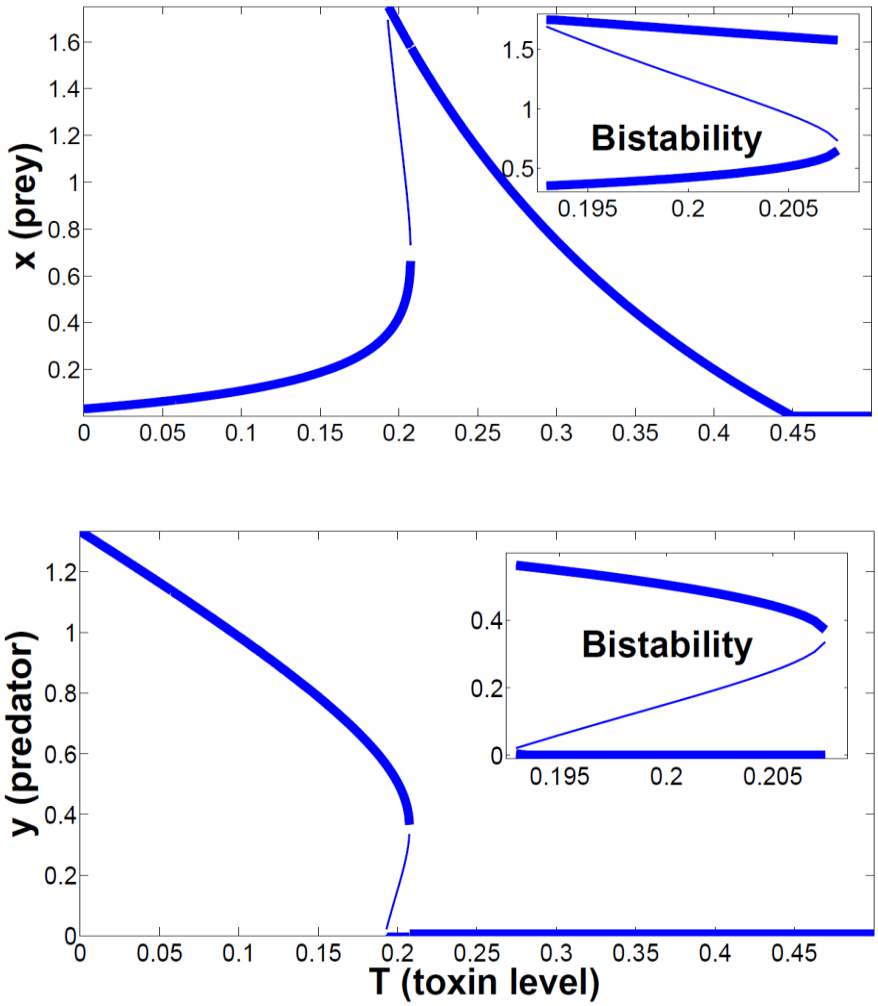

Figure 8. Bifurcation diagram with respect to toxin level T- the system has two coexistence equilibria.
Stable equilibria (thick solid curve). Unstable equilibria (thin solid curve).
Parameters: $\eta = 1.5$, $\beta_1 = 1$, $m_1 = 0.1$, $m_2 = 0.02$, $k_1 = 1, k_2 = 0.2$, $\sigma_2 = 1$, $\beta_2 = 4$, c = 1.5. Here, $T_0^* = 0.450$, $T_1^* = 0.208$, $T_2^* = 0.149$, $T_3^* = 0.097$ (see Table 2).
Note that the condition $\max\{T_2^*, T_3^*\} < \min\{T_0^*, T_1^*\}$ is satisfied, the system has two coexistence equilibria.
When $0.193 < T < 0.208$, the system has bistability, which is highlighted at the top-right corners of the panels.

In Figure 9, both species coexist at a limit cycle when $T = 0$. As T increases, population abundances continue to fluctuate but with decreasing amplitudes until they reach a bistable state: a stable limit cycle and a prey-only equilibrium. As we continue to increase T, the limit cycle disappears and another type of bistability appears: a stable coexistence equilibrium and a prey-

only equilibrium. As T increases further, coexistence state disappears and the population densities tend to prey-only state. Finally, if we increase T even further, the stable prey density decreases until both species are extirpated.

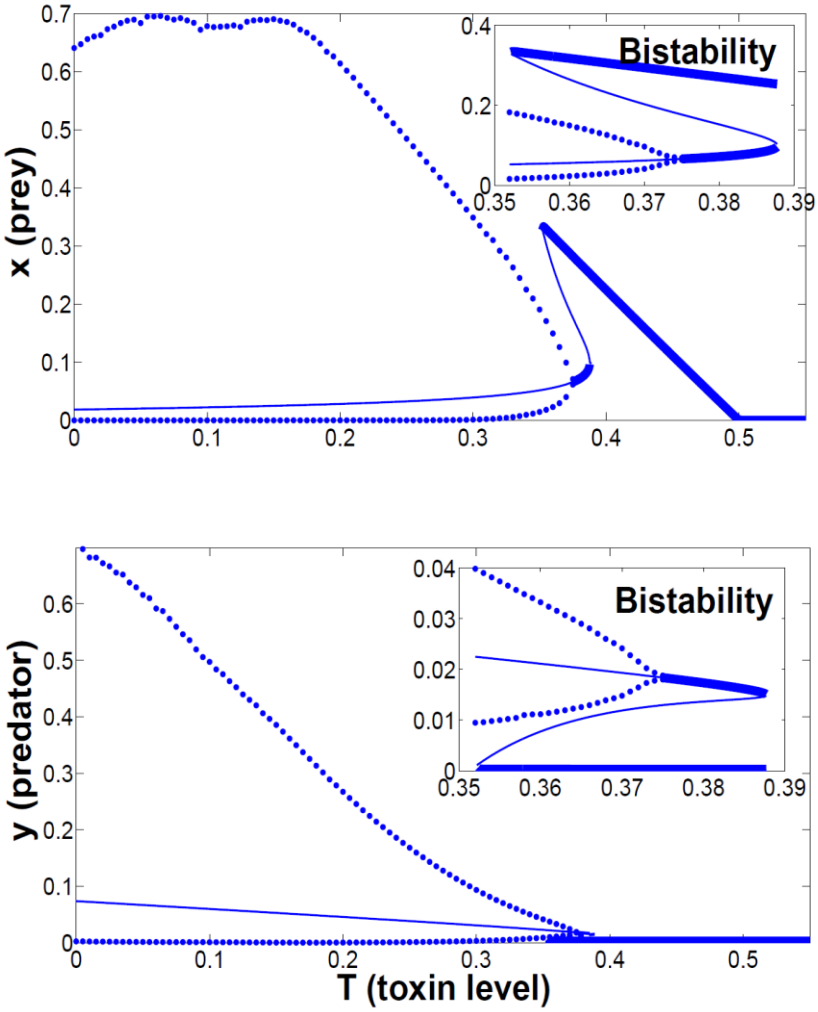

Figure 9. Bifurcation diagram with respect to toxin level T- the system has two typies of bistabilities.
The highest and lowest values of x-coordinates and y-coordinate of stable limit cycles (dot curves), x-coordinates and y-coordinates of unstable coexistence equilibria (thin solid curves), x-coordinates and y-coordinates of stable coexistence equilibria and prey-only equilibria (thick solid curves). Parameters: η =0.12, β_1 = 0.75, $m_1 = 0.45$, m_2 = 0.1, k_1 = 0.1, k_2 = 0.1, σ_2 = 1, β_2 = 2, c = 0.5. Here, $T_0^* = 0.500$, $T_1^* = 0.388$, $T_2^* = 0.306$, $T_2^* = 0.210$ (see Table 2). Note that the condition $\max\{T_2^*, T_3^*\} < \min\{T_0^*, T_1^*\}$ is satisfied, the system has two coexistence equilibria. When 0.352<T <0.388, the system has bistability, which is highlighted at the top-right corners of the panels.

Figures 6 to 9 highlight several key points:

1. High toxin concentration in the environment is harmful to both species – it may lead to extirpation of both species.
2. The population dynamics is counterintuitive when both the prey and the predator are exposed in environment with low concentration of toxin. That is, low toxin concentration benefits the prey because the bioaccumulation of toxin in the predator reduces the predator abundance, which releases its prey from predation.
3. 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. Certain toxin levels may lead to more than one asymptotic population density of either the prey or the predator. In this scenario, the initial population density of the prey or the predator determines its eventual population density.

7 APPLICATION

In what follows, we use the parameter estimates from Section 3 to numerically solve the toxin-mediated predator-prey model (11). Our purpose is to understand how the concentration of a toxin in the environment affects the long-term biomass of rainbow trout and its prey. We make numerical simulations by considering two scenarios.

In the first scenario, we regard the rainbow trout as the predator and small fish as the prey. We plot the stable biomass of prey and predators as the concentration of methylmercury T increases from 0 to 0.09 µg/L in Figure 10. As we see, the bifurcation dynamics are similar to that in Figure 7.

In the second scenario, we regard the rainbow trout as the predator and aquatic insects as the prey. We apply the results of model parameterization from Section 3 to the model (11). We then describe the bifurcation dynamics as the concentration of methylmercury in the environment T changes from 0 to 0.03 μg/L (Figure 11). As shown in Figure 11, the stable predator biomass decreases as the concentration of methylmercury increases which leads to the stable prey biomass increasing until it reaches its environmental carrying capacity.

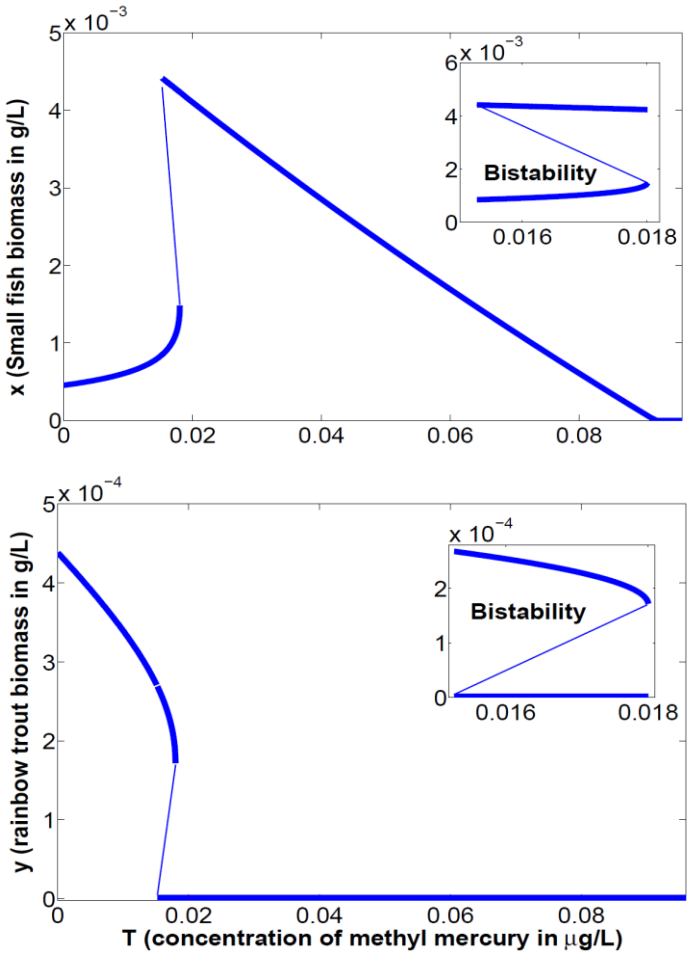

Figure 10. The stable biomass of the prey (small fish) and the predator (rainbow trout) when the concentration of methylmercury in the environment changes from 0 to 0.09 µg/L. x-coordinates and y-coordinates of unstable coexistence equilibria (thin solid curves) x-coordinates and y-coordinates of stable coexistence equilibria and prey-only equilibria (thick solid curves).

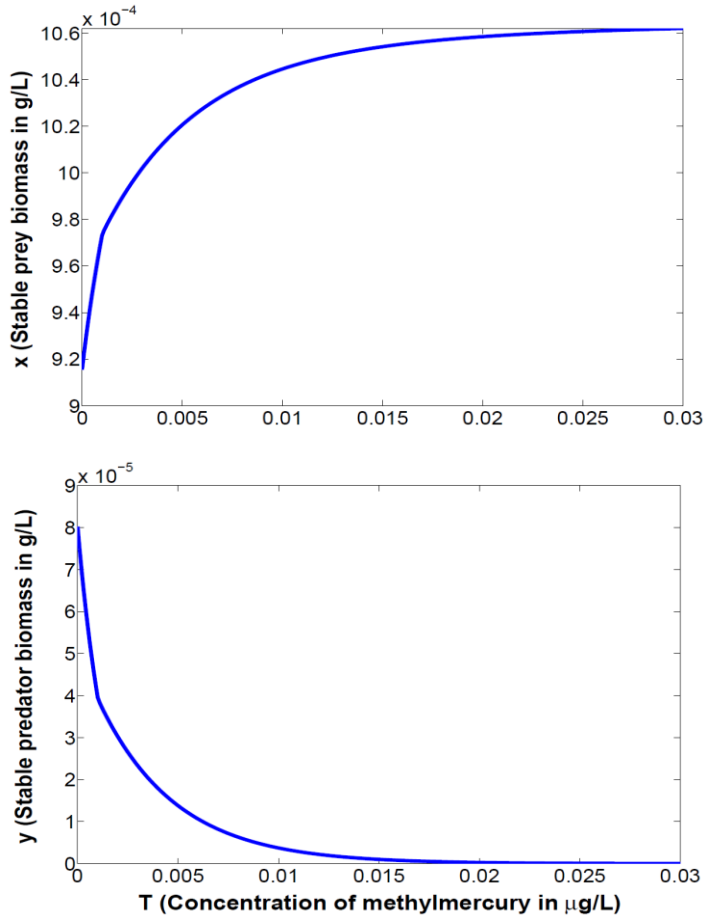

Figure 11. The stable biomass of the prey (aquatic insects) and the predator (rainbow trout) when the concentration of methylmercury in the environment changes from 0 to 0.03 μg/L.

8 CONCLUSIONS AND RECOMMENDATIONS

Contamination by toxic pollutants is a significant problem in water management. The effect of a toxic contaminant can, in principle, be exerted on all levels of the biological hierarchy, from cells to organs to organisms to populations to entire ecosystems. Mathematic models are useful tools for evaluating the ecological significance of observed or predicted effects of toxic chemicals on individual organisms and population dynamics. Most toxin-mediated single-species models assume that populations take up contaminants from water and ignore bioaccumulation (contaminant uptake, excretion, and contaminant transfer through aquatic food chain). These single-species models cannot predict the effects of toxin on species interactions, nutrient cycling, or contaminant flow in aquatic systems.

8.1 Conclusions

In this study, motivated by the fact that many aquatic organisms take up contaminants both from water and from food (their prey), we developed a toxin-mediated predator-prey model that

consists of four differential equations. To facilitate model analysis, we approximate the model with a two dimensional system because population metabolism takes place over a much faster time scale than population growth does. We then analyze the existence and stability of extirpation and coexistence equilibria based on the two dimensional system. The conditions that guarantee the existence and stability of equilibria provide meaningful biological interpretations. For instance, high toxin concentrations in the environment lead to the extirpation of prey and predators, and low toxin concentrations lead to the coexistence of both populations. However, intermediate toxin concentrations result in two alternative stable states: a prey-only equilibrium and coexistence of prey and predators. In this scenario, the initial conditions determine which steady state the populations will tend towards. The results of model analysis are then used to show all possible asymptotic behaviors of the system. To do this, we plot a series of phase portraits to identify possible outcomes. These outcomes suggest that our toxin-mediated system has richer dynamics than traditional predator-prey system due to the existence of two interior equilibria and bistability.

Predator-prey interactions have been one of the central themes in ecology. The dynamics of traditional predator-prey systems have been well documented. The main aim of the present study is to investigate how the balance of a traditional predator-prey system (without a toxin effect) will change when the prey and the predator are exposed in an aquatic environment where toxins may be present at low levels. To this end, treating toxin level T as a bifurcation parameter, we plot many bifurcation dynamics. The results imply that sublethal contaminant effects on predator-prey interactions are counterintuitive. That is, increased toxin level has a positive effect on prey persistence even though it has a negative effect on predator persistence. This is because the bioaccumulation of toxin in the predator reduces the predator abundance, which releases its prey from predation. In addition, our findings indicate that an increasing toxin level reduces the amplitude of population cycle oscillations, which often occur in traditional predator-prey systems. In other words, the toxin has a stabilizing effect on population cycles.

8.2 Recommendations

For model analysis, we mainly focus on local stable analysis of equilibria. As we observed in Section 5.1, the system has several types of phase portraits. We believe that the global dynamics will provide more clear insights into the effects of a toxin on long-term behavior, but this is challenging and is left for future work.

Species in different trophic levels may have different sensitivity to each toxin. We hope to encourage the connection of the model data on fish species common in oil sands areas and to other species and contaminants of interest in other areas. Then the bifurcation analysis in Section 5.2 will provide threshold values of toxin concentration in the environment for the persistence of one or both species. The threshold value for shifting the system from one stable state to another can also be observed. This will help consider acute and chronic guideline developments for target species and chemicals.

Our model assumes that concentration of the toxin in the environment is a constant. In reality, the toxin concentration may vary over time (and space, if sediment and plant uptake and release

are considered) due to a variety of factors. In addition, we have only considered the interaction between one predator species and one prey species. When toxins flow across multiple prey and predators, the outcome can be more complicated. Our model also assumes that the capture rate in the Holling's type II function response is a constant. In practice, contaminant-induced changes in a population's behavior may also lead to abundance changes in prey and predator populations. For example, the dynamics might be very different if predators prefer "toxic" prey (because they are slower, sicker, easier to catch) versus if they avoid toxic prey (because they taste bad or they know that the toxin is bad for them). Further consideration of these factors in the model framework are required to investigate this problem. We expect that the main results we obtained in this study are robust, even though the details will certainly change if we include these factors in the model.

Although our toxin-mediated predator-prey system is developed based on an aquatic environment, the model and the results in this study are applicable to predator-prey systems in terrestrial ecosystems.

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10 GLOSSARY

10.1 Terms

Bifurcation Dynamics

In mathematics, particularly in dynamical systems, bifurcation dynamics show the possible long-term values (equilibria/fixed points or periodic orbits) of a system as a function of a parameter in the system.

Bioaccumulation

The accumulation of substances, such as pesticides, or other organic chemicals in an organism. Bioaccumulation occurs when an organism absorbs a toxic substance at a rate greater than that at which the substance is lost. Thus, the longer the biological half-life of the substance the greater the risk of chronic poisoning, even if environmental levels of the toxin are not very high.

Bistability

In a dynamic system, bistability means that the system has two locally stable equilibrium states.

Body Burden

The accumulated total of chemical toxins in the body. The precise definition is given by a fraction in the beginning of Section 2.

Boundary Equilibrium

It is also called extirpation or extinction equilibrium, at which at least one species is extirpated or goes extinct. In this study, the equilibrium $E_{0,1} = (0,0)$ and $E_{0,2} = (x_0,0)$ are boundary equilibria (see Table 2).

Carrying Capacity

The carrying capacity of a biological species in an environment is the maximum population density of the species that the environment can sustain indefinitely, given the food, habitat, water and other necessities available in the environment.

Coexistence Equilibrium

A stable state at which both species are able to coexist.

Depuration

The process by which toxins are digested and excreted due to the metabolism of individuals.

Differential Equation Model

Differential equation models relate functions of dependent variables with their derivatives. In this paper the derivatives are taken with respect to time.

Extinction

In biology and ecology, extinction is the end of an organism or of a group of organisms (taxon), normally a species.

Extirpation

Local extinction, in which is the condition of a species (or other taxon) that ceases to exist in the chosen geographic area of study, though it still exists elsewhere.

Globally Asymptotically Stable

An equilibrium point x^* is said to be globally asymptotically stable if the solutions with all nonnegative initial conditions will tend towards x^* as $t \to \infty$.

Half-Saturation Constant

The half-saturation constant represents the population density at which half of the maximum per capita feeding rate is reached. In the predator functional response $p(x) = \frac{\xi x}{\eta + x}$ (equation (9)), η is referred as the half-saturation constant, since $p(\eta) = \frac{\xi}{2}$; that is, per capita feeding rate is the half of the maximum rate when the population density $x = \eta$.

Interior Equilibrium

It is also called coexistence equilibrium, a stable state at which both species are able to coexist. In this study, the equilibrium $E_1 = (x_1, y_1)$ and $E_2 = (x_2, y_2)$ are coexistence equilibria (see Table 2).

Isocline and Null-cline

The term isocline derives from the Greek words for "same slope." For a first-order ordinary differential equation $y' = f(x, y)$, a curve with equation $f(x, y) = C$ for some constant C is known as an isocline. An isocline with constant $C = 0$ is called a null-cine.

In population dynamics, a null-cline refers to the set of population densities at which the rate of change, or partial derivative, for one population in a pair of interacting populations is zero.

Lemma

A minor result whose sole purpose is to help in proving a theorem. It is a stepping stone on the path to proving a theorem.

Limit Cycle

In the study of dynamic systems with two-dimensional phase space, a limit cycle is a closed trajectory in phase space having the property that at least one other trajectory spirals into it as time approaches infinity or as time approaches minus infinity.

Locally Asymptotically Stable

An equilibrium point x^* is said to be locally asymptotically stable if x^* is locally stable, and futhermore, all solutions starting near x^* tend towards x^* as $t \to \infty$.

Locally Stable

An equilibrium point x^* is locally stable if all solutions that start near x^* (meaning that the initial conditions are in a neighborhood of x^*) remain near x^* for all time.

Manifold

For the purposes of this paper, a manifold can be considered a surface contained within a higher dimensional space. The full mathematical definition is more complex.

Nondimensionalization

Nondimensionalization is the partial or full removal of units from an equation involving physical quantities by a suitable substitution of variables. This technique can simplify and parameterize problems where measured units are involved. It is closely related to dimensional analysis. Nondimensionalization can also recover characteristic properties of a system. For example, if a system has an equilibrium, or limit cycle, nondimensionalization can recover these values.

Parameter

A constant or variable term in a mathematical function that determines the specific form of the function but not its general nature, as a in $f(x) = ax$, where *a* determines only the slope of the line described by $f(x)$.

Parameterization

Parametrization (also parameterisation) is the process of deciding and defining the parameters necessary for a complete or relevant specification of a model.

Phase Plane

A visual display of certain characteristics of certain kinds of differential equations.

Phase Portrait

A phase portrait is a geometric representation of the trajectories of a dynamic system in the phase plane.

Quasi-Steady State Approximation

The quasi-steady-state approximation is a standard procedure in the study of high dimensional systems where certain state variables change over a faster time scale than other state variables. By introducing a small parameter and making nondimensionalization for the system, then letting the small parameter be zero, one can reduce the dimension of the system.

Saddle Point

A saddle point is an unstable equilibrium point at the intersection of unstable and stable manifolds.

Solution Curve

A solution curve is a graph that describes how the population density changes with respect to time.

State Variable

A state variable is one of the set of variables that are used to describe the mathematical "state" of a dynamic system. Intuitively, the state of a system describes enough about the system to determine its future behavior. Models that consist of coupled first-order differential equations are said to be in state-variable form.

System

A system is a set of interacting or interdependent components forming an integrated whole. It is a set of detailed methods, procedures and routines created to solve a problem.

Theorem

A mathematical statement that is proved using rigorous mathematical reasoning. In a mathematical paper, the term theorem is often reserved for the most important results.

Threshold

The point that must be exceeded to begin producing a given effect or result or to elicit a response.

Toxin

A toxin is a substance poisonous to living cells or organisms.

Toxin-Mediated

In this report, a biological process that is affected by, or controlled by, a toxin in the environment or in an organism.

Uptake

The act of taking in or absorbing, especially into a living organism.

10.2 Acronyms

GAS	Globally Asymptotic Stable
HB	Hopf Bifurcation
OSRIN	Oil Sands Research and Information Network
SEE	School of Energy and the Environment
USGS	US Geological Survey

APPENDIX 1: Supporting Information about the Model

Proof of Theorem 4.1

Proof. Positivity obviously holds for the system (15). Let $z(t) = \beta_1 x(t) + y(t)$ and differentiating z once yields

$$\begin{aligned} \frac{dz}{dt} &= \beta_1\left(\frac{\max\{0,1-u\}}{1+x} - k_1 u - m_1\right)x - \beta_1 \frac{xy}{\eta + x} + \frac{\beta_1 xy \max\{0,1-v\}}{\eta + x} \\ &\quad -(k_2 v + m_2)y \\ &\le \beta_1 - \beta_1 m_1 x - m_2 y \\ &\le \beta_1 - \min\{m_1, m_2\} z. \end{aligned} \tag{27}$$

Hence, we have

$$0 \le z(t) \le \frac{\beta_1}{\min\{m_1, m_2\}} + \left(z(0) - \frac{\beta_1}{\min\{m_1, m_2\}}\right) e^{-\min\{m_1, m_2\}t} \tag{28}$$

which implies

$$\limsup_{t\to\infty} z(t) \le \frac{\beta_1}{\min\{m_1, m_2\}} \qquad \textit{and} \quad z(t) \le \max\left\{\frac{\beta_1}{\min\{m_1, m_2\}}, z(0)\right\} \tag{29}$$

On the other hand, from the third equation of the system (15), we have

$$\varepsilon \frac{du}{dt} \le T - u \tag{30}$$

Similar to the argument on $z(t)$, we can conclude that $u(t)$ is ultimately bounded. Therefore, from the fourth equation of the system (15), we obtain

$$\varepsilon \frac{dv}{dt} \le cT - \sigma_2 v + \beta_2 u \le cT + \beta_2 T - \sigma_2 v \tag{31}$$

which indicates that $v(t)$ is also ultimately bounded.

Argument about the assumptions (18) and (19)

Clearly, if $m_1 \ge 1$, then for any $x \ge 0$ and $T \ge 0$,

$$\frac{\max\{0,1-T\}}{1+x} - k_1 T - m_1 < \frac{1}{1+x} - m_1 < 1 - m_1 < 0$$

thus $\lim_{t\to\infty} x(t) = 0,$ which will lead to $\lim_{t\to\infty} y(t) = 0.$

If $T \ge 1$, then $\max\{0,1-T\} = 0$, thus $\lim_{t\to\infty} x(t) = 0$, $\lim_{t\to\infty} y(t) = 0$.

Similarly, if $cT \ge \sigma_2$, then $\max\{0,1-cT/\sigma_2 - \beta_2 Tx/(\sigma_2(\eta + x))\} = 0$, thus $\lim_{t\to\infty} y(t) = 0.$

If $m_2 > \beta_1$, then for any $x \geq 0$ and $T \geq 0$,

$$\frac{\beta_1 x \max\{0, 1 - \frac{cT}{\sigma_2} - \frac{\beta_2 T}{\sigma_2}\frac{x}{\eta + x}\}}{\eta + x} - k_2\left(\frac{cT}{\sigma_2} + \frac{\beta_2 T}{\sigma_2}\frac{x}{\eta + x}\right) - m_2 < \beta_1 - m_2 < 0$$

thus, $\lim_{t\to\infty} y(t) = 0,$ and the system (17) reduces to a single species model.

Existence of equilibria

The prey x-nullclines are

$$x = 0, \quad y = \frac{f(x)}{\varphi(x)} \tag{32}$$

and the predator y-nullclines are

$$g(x) = 0, \quad y = 0 \tag{33}$$

From the intersections of the nullclines, we find that the system has only one extirpation equilibrium $E_{0,1} = (0,0)$ if $T \geq (1 - m_1)/(k_1 + 1) := T_0^*$, and the system has an extirpation equilibrium $E_{0,1}$ and a prey-only equilibrium $E_{0,2} = (x_0, 0)$ if $T < T_0^*$ with $x_0 = (1 - T)/(k_1 T + m_1) - 1.$

The interior equilibria (coexistence equilibria) can be found by setting

$$g(x) = 0 \quad \text{and} \quad y = \frac{f(x)}{\varphi(x)} > 0 \tag{34}$$

Noticing that $\varphi(x)$ is an increasing positive function on $(0, \infty)$, we require

$$f(x) = \left(\frac{1 - T}{1 + x} - k_1 T - m_1\right)x > 0 \tag{35}$$

that is, $T < T_0^*$ and $0 < x < (1 - T)/(k_1 T + m_1) - 1 := F(\varphi) = x_0$. The second condition is equivalent to

$$\varphi(x) < \varphi(x_0) \tag{36}$$

Also, if $\varphi(x) \geq (\sigma_2 - cT)/\beta_2 T$, then $\max\{0, 1 - cT/\sigma_2 - \beta_2 T\varphi(x)/\sigma_2\} = 0.$ In this case, $g(x) < 0$. Hence, we require

$$\varphi(x) < \frac{\sigma_2 - cT}{\beta_2 T} \tag{37}$$

When (37) holds, function $g(x)$ becomes

$$\begin{aligned} g(x) &= \beta_1\varphi(x)\left(1-\frac{cT}{\sigma_2}-\frac{\beta_2 T}{\sigma_2}\varphi(x)\right)-\frac{k_2cT}{\sigma_2}-\frac{k_2\beta_2 T}{\sigma_2}\varphi(x)-m_2 \\ &= -\frac{1}{\sigma_2}\left[\beta_1\beta_2 T(\varphi(x))^2-(\beta_1(\sigma_2-cT)-k_2\beta_2 T)\varphi(x)+k_2cT+m_2\sigma_2\right] \end{aligned} \tag{38}$$

Therefore, system (20) has coexistence equilibrium if and only if the quadratic equation with respect to $\varphi := \varphi(x)$

$$\beta_1\beta_2 T\varphi^2-[\beta_1(\sigma_2-cT)-k_2\beta_2 T]\varphi+k_2cT+m_2\sigma_2=0 \tag{39}$$

has at least one positive root which satisfies

$$\varphi < \min\left\{\frac{\sigma_2-cT}{\beta_2 T}, \varphi(x_0)\right\} \tag{40}$$

Let

$$\Delta := [\beta_1(\sigma_2-cT)-k_2\beta_2 T]^2-4\beta_1\beta_2 T(k_2cT+m_2\sigma_2) \tag{41}$$

Notice if $\Delta \geq 0$, the quadratic equation (39) has either two positive roots (when $\beta_1(\sigma_2-cT)-k_2\beta_2 T>0$) or two negative roots (when $\beta_1(\sigma_2-cT)-k_2\beta_2 T<0$).

Thus, we require that (39) has two positive roots. We also find that (39) has two positive roots

$$\varphi_{1,2}=\frac{\beta_1(\sigma_2-cT)-k_2\beta_2 T \mp \sqrt{\Delta}}{2\beta_1\beta_2 T} \tag{42}$$

if and only if the following condition holds:

$$\beta_1(\sigma_2-cT)-k_2\beta_2 T > 2\sqrt{\beta_1\beta_2 T(k_2cT+m_2\sigma_2)} \tag{43}$$

Next, we equivalently rewrite the condition (43) into a restriction condition with respect to *T*. Firstly, the condition (43) implies that $\beta_1(\sigma_2-cT)-k_2\beta_2 T>0$, which is equivalent to

$$T < \frac{\beta_1\sigma_2}{\beta_1 c+k_2\beta_2} \tag{44}$$

Secondly, if we introduce a function G with respect to T,

$$G(T)=\beta_1(\sigma_2-cT)-k_2\beta_2 T-2\sqrt{\beta_1\beta_2 T(k_2cT+m_2\sigma_2)} \tag{45}$$

Then G is a decreasing function of T. Solving $G(T)=0$, we can get a threshold value of T, which is

$$T=\frac{\beta_1\sigma_2(\beta_1 c+k_2\beta_2+2\beta_2 m_2)-\beta_1\sigma_2\sqrt{4\beta_2(\beta_1 c+\beta_2 m_2)(m_2+k_2)}}{(\beta_1 c-k_2\beta_2)^2}:=T_1^* \tag{46}$$

Clearly, $G(\beta_1\sigma_2/(\beta_1 c+k_2\beta_2))<0$, since $G(T^*)=0$ and $G(\cdot)$ is a decreasing function, so $T_1^*<\beta_1\sigma_2/(\beta_1 c+k_2\beta_2)$.

Therefore, a combination of (44) and (46) yields that the condition (43) is equivalent to

$$T<T_1^* \tag{47}$$

We now require φ_1 and (or) φ_2 satisfy the condition (40). We notice that

$$\varphi_1<\varphi_2<\frac{\sigma_2-cT}{\beta_2 T} \tag{48}$$

In fact, we have

$$\begin{aligned}\varphi_2<\frac{\sigma_2-cT}{\beta_2 T} &\Leftrightarrow \frac{\beta_1(\sigma_2-cT)-k_2\beta_2 T+\sqrt{\Delta}}{2\beta_1\beta_2 T}<\frac{\sigma_2-cT}{\beta_2 T}\\ &\Leftrightarrow \sqrt{\Delta}<\beta_1(\sigma_2-cT)+k_2\beta_2 T\\ &\Leftrightarrow \Delta<(\beta_1(\sigma_2-cT)+k_2\beta_2 T)^2\\ &\Leftrightarrow -4\beta_1\beta_2 T\sigma_2(k_2+m_2)<0,\end{aligned} \tag{49}$$

which is true. Thus, the existence of coexistence equilibrium depends on whether or not $\varphi_{1,2}<\varphi(x_0)$.

Noticing that $0<\varphi(x_0)=x_0/(\eta+x_0)<1$ and φ_1 and φ_2 do not depend on η (see eq.(9.11)), we can choose an appropriate η such that $\varphi_{1,2}<\varphi(x_0)$ if $\varphi_{1,2}<1$.

From (42), we find that $\varphi_1<1<\varphi_2$ if

$$T<\frac{(\beta_1-m_2)\sigma_2}{\beta_1\beta_2+\beta_1 c+k_2\beta_2+k_2 c}:=T_2^*, \tag{50}$$

and $\varphi_1<\varphi_2<1$ if

$$T>\max\{T_2^*,T_3^*\}, \tag{51}$$

where

$$T_3^*=\frac{\beta_1\sigma_2}{\beta_1 c+k_2\beta_2+2\beta_1\beta_2}. \tag{52}$$

Therefore, if $T < \min\{T_0^*, T_1^*, T_2^*\}$ and $\eta < x_0/\varphi_1 - x_0$, then system (20) has only one coexistence equilibrium $E_1 = (x_1, \frac{f(x_1)}{\varphi(x_1)})$, where x_1 is given by $\varphi_1 = \varphi(x_1)$. More precisely, since $\varphi_1 = x_1/(\eta + x_1)$, we have $x_1 = \eta\varphi_1/(1-\varphi_1)$.

If $T_3^* < \min\{T_0^*, T_1^*\}$, then system (20) has two coexistence equilibrium E_1 and $E_2 = (x_2, \frac{f(x_2)}{\varphi(x_2)})$ with $x_2 = \eta\varphi_2/(1-\varphi_2)$ when $T_3^* < T < \min\{T_0^*, T_1^*\}$ and $\eta < x_0/\varphi_2 - x_0$.

Proof of Theorem 4.2

Proof. At $E_{0,1}$, where both prey and predator are extirpated, the Jacobian is

$$J(E_{0,1}) = \begin{pmatrix} f'(0) & -\varphi(0) \\ g'(0) & g(0) \end{pmatrix} = \begin{pmatrix} 1-T-k_1T-m_1 & 0 \\ 0 & -\frac{k_2cT}{\sigma_2} - m_2 \end{pmatrix} \tag{53}$$

and the eigenvalues are the components on the diagonal,

$$\lambda_1 = 1-T-k_1T-m_1, \quad \lambda_2 = -\frac{k_2cT}{\sigma_2} - m_2 \tag{54}$$

If $k_1T + m_1 + T > 1$, then $E_{0,1}$ is a stable node because both eigenvalues of $J(E_0)$ are negative. Moreover, only the boundary equilibrium $E_{0,1}$ is feasible when $k_1T + m_1 + T > 1$. Because solutions are bounded, solutions must converge to $E_{0,1}$. If $k_1T + m_1 + T < 1$, then $E_{0,1}$ is a saddle point because the two real eigenvalues are of opposite sign.

The Jacobian at $E_{0,2}$, where only the prey survives, is

$$J(E_{0,2}) = \begin{pmatrix} f'(x_0) & -\varphi(x_0) \\ 0 & g(x_0) \end{pmatrix} \tag{55}$$

and the eigenvalues are

$$\lambda_1 = f'(x_0) = \frac{(k_1T + m_1)(T + k_1T + m_1 - 1)}{1-T}, \quad \lambda_2 = g(x_0) \tag{56}$$

The condition of the existence of $E_{0,1}$, $T + k_1T + m_1 < 1$, implies that $\lambda_1 < 0$. Thus, the stability of $E_{0,1}$ can be determined by the sign of eigenvalue of $g(x_0)$. That is, $E_{0,1}$ is a stable (unstable) node (saddle point) if $g(x_0) < (>) 0$.

Next, we investigate the condition $g(x_0) < 0$ further under which $E_{0,1}$ is a stable node. Let $\varphi_0 = x_0/(\eta + x_0)$, using (38) and (39) we find that $g(x_0) < 0$ is equivalent to

$$\beta_1\beta_2 T\varphi_0^2 - [\beta_1(\sigma_2 - cT) - k_2\beta_2 T]\varphi_0 + k_2 cT + m_2\sigma_2 := F(\varphi_0) > 0 \tag{57}$$

Using the same discriminant Δ as in (41), we consider the following cases:

1. If $\Delta < 0$, then $F(\varphi_0)$ represents a parabola which opens upward and does not intersect the φ_0 axis, then $F(\varphi_0) > 0$ for any φ_0. From the discussion about the existence of equilibria (see Existence of equilibria of equilibria), we can easily find that $\Delta < 0$ is equivalent to

$$T_1^* < T < T_4^* \tag{58}$$

where

$$T_4^* := \frac{\beta_1\sigma_2(\beta_1 c + k_2\beta_2 + 2\beta_2 m_2) + \beta_1\sigma_2\sqrt{4\beta_2(\beta_1 c + \beta_2 m_2)(m_2 + k_2)}}{(\beta_1 c - k_2\beta_2)^2} \tag{59}$$

2. If $\Delta > 0$ and $\beta_1(\sigma_2 - cT) - k_2\beta_2 T < 0$ (i.e., $T > \beta_1\sigma_2/(\beta_1 c + k_2\beta_2)$), then $F(\varphi_0)$ represents a parabola which opens upward and intersects negative φ_0 axis, then $F(\varphi_0) > 0$ since $\varphi_0 > 0$. Again, from the discussion about the existence of equilibria, we know that $\Delta > 0$ is equivalent to $T < T_1^*$ and $T_1^* < \beta_1\sigma_2/(\beta_1 c + k_2\beta_2)$. Therefore, the condition $\Delta > 0$ contracts the condition $\beta_1(\sigma_2 - cT) - k_2\beta_2 T < 0$.

3. If $\Delta > 0$ and $T < \beta_1\sigma_2/(\beta_1 c + k_2\beta_2)$ (note that $\Delta > 0$ is equivalent to $T < T_1^*$ and $T_1^* < \beta_1\sigma_2/(\beta_1 c + k_2\beta_2)$), then $F(\varphi_0)$ represents a parabola which opens upward and intersects positive φ_0 axis, $F(\varphi_0) = 0$ has two positive roots φ_1 and φ_2 ($\varphi_1 < \varphi_2$). Thus, $F(\varphi_0) > 0$ when $\varphi_0 < \varphi_1$ (i.e., $\eta > x_0/\varphi_1 - x_0$) or $\varphi_0 > \varphi_2$ (i.e., $\eta < x_0/\varphi_2 - x_0$).

From (1) to (3), we conclude that the prey-only equilibrium $E_{0,2}$ is locally asymptotically stable if one of the following conditions is satisfied:

- $T_1^* < T < T_4^*$,
- $T < T_1^*$ and $\eta > x_0/\varphi_1 - x_0$,
- $T < T_1^*$ and $\eta < x_0/\varphi_2 - x_0$.

Note that the condition plays a role only when the conditions $\max\{T_2^*, T_3^*\} < \min\{T_0^*, T_1^*\}$ and $\max\{T_2^*, T_3^*\} < T < \min\{T_0^*, T_1^*\}$ are satisfied (see subsection 3.1.1).

Proof of Theorem 4.3

Proof. At coexistence equilibria E_i $(i = 1,2)$, where both prey and predator coexist, the Jacobian is

$$J(E_i) = \begin{pmatrix} f'(x_i) - \varphi'(x_i)\dfrac{f(x_i)}{\varphi(x_i)} & -\varphi(x_i) \\ g'(x_i)\dfrac{f(x_i)}{\varphi(x_i)} & 0 \end{pmatrix} \tag{60}$$

and the characteristic equation is

$$\lambda^2 - \left[f'(x_i) - \varphi'(x_i)\frac{f(x_i)}{\varphi(x_i)} \right]\lambda + g'(x_i) f(x_i) = 0 \tag{61}$$

For the equilibrium E_1, simple calculation gives

$$g'(x_1) = -\frac{1}{\sigma_2}[2\beta_1\beta_2 T\varphi(x_1) - \beta_1(\sigma_2 - cT) + k_2\beta_2 T)]\varphi'(x_1) = \frac{\sqrt{\Delta}\varphi'(x_1)}{\sigma_2} > 0 \tag{62}$$

Since the quantities $g'(x_1)$ and $f(x_1)$ are positive, the Routh-Hurwitz criterion guarantees that equilibria E_1 is stable if

$$f'(x_1) - \varphi'(x_1)\frac{f(x_1)}{\varphi(x_1)} < 0 \tag{63}$$

Note that

$$f'(x_1) - \varphi'(x_1)\frac{f(x_1)}{\varphi(x_1)} = \varphi(x_1)\left(\frac{f(x_1)}{\varphi(x_1)}\right) \tag{64}$$

and $\varphi(x_1) > 0$, condition (53) is equivalent to

$$\left(\frac{f(x_1)}{\varphi(x_1)}\right)' < 0 \tag{65}$$

Simple calculation yields

$$\left(\frac{f(x_1)}{\varphi(x_1)}\right)' = \frac{(1-T)(1-\eta)}{(1+x_1)^2} - (k_1 T + m_1) \tag{66}$$

Clearly, if $\eta > 1$, then condition (65) is satisfied, E_1 is stable. If $\eta < 1$, then (65) is equivalent to

$$k_1 T + m_1 < \frac{(1-T)(1-\eta)}{(1+x_1)^2} \tag{67}$$

Therefore, E_1 is stable either $\eta \geq 1$ or both conditions $0 < \eta < 1$ and $k_1 T + m_1 < \frac{(1-T)(1-\eta)}{(1+x_1)^2}$ are satisfied.

Analytical Calculations on the Sensitivity of Stable Population Density to Toxin level

If the system (20) has a stable prey-only equilibrium $E_{0,2} = (x_0, 0)$ with $x_0 = (1-T)/(k_1 T + m_1) - 1$, then the stable prey density decreases as the toxin level T increases because x_0 is a decreasing function of T.

If the condition

$$\left(\frac{f(x_1)}{\varphi(x_1)} \right)' < 0 \tag{68}$$

is satisfied (here $'$ denote the derivative with respect to x), then the system (20) has a stable coexistence equilibrium $E_1 = (x_1, y_1)$ with $x_1 = \frac{\eta \varphi_1}{1-\varphi_1}$ and $y_1 = \frac{f(x_1)}{\varphi(x_1)} := \frac{f(x_1)}{\varphi_1}$, where φ_1 is given by the following quadratic equation (see equation (39)):

$$\beta_1 \beta_2 T \varphi_1^2 - [\beta_1(\sigma_2 - cT) - k_2 \beta_2 T]\varphi_1 + k_2 cT + m_2 \sigma_2 = 0 \tag{69}$$

Differentiating both sides of the above equation with respect to T, we have

$$\beta_1 \beta_2 (\varphi_1^2 + 2T\varphi_1 \frac{\partial \varphi_1}{\partial T}) + (k_2 \beta_2 T + \beta_1 cT - \beta_1 \sigma_2) \frac{\partial \varphi_1}{\partial T} + (k_2 \beta_2 + \beta_1 c)\varphi_1 + k_2 c = 0 \tag{70}$$

Thus,

$$\frac{\partial \varphi_1}{\partial T} = \frac{-(\beta_1 \beta_2 \varphi_1^2 + k_2 \beta_2 \varphi_1 + \beta_1 c \varphi_1 + k_2 c)}{2\beta_1 \beta_2 T \varphi_1 + k_2 \beta_2 T + \beta_1 cT - \beta_1 \sigma_2} \tag{71}$$

Applying equation (41) to the above equation, we have

$$\frac{\partial \varphi_1}{\partial T} = \frac{-(\beta_1 \beta_2 \varphi_1^2 + k_2 c + \varphi_1 k_2 \beta_2 + \varphi_1 \beta_1 c)}{-\sqrt{\Delta}} > 0 \tag{72}$$

Therefore,

$$\frac{\partial x_1}{\partial T} = \frac{\partial x_1}{\partial \varphi_1}\frac{\partial \varphi_1}{\partial T} > 0 \tag{73}$$

since $\partial x_1/\partial \varphi_1 > 0$ is obvious from $x_1 = \eta\varphi_1/(1-\varphi_1)$.

That is, if the system stabilize at the coexistence equilibrium $E_1 = (x_1, y_1)$, then the stable prey density always increases as the toxin level T increases.

From (68) and (73), we obtain

$$\frac{\partial y_1}{\partial T} = \left(\frac{f(x_1)}{\varphi(x_1)}\right)' \frac{\partial x_1}{\partial T} < 0 \tag{74}$$

That is, if the system stabilize at the coexistence equilibrium $E_1 = (x_1, y_1)$, then the stable predator density always decreases as the toxin level T increases.

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