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A model for the impact of contaminants on fish population dynamics

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HIGHLIGHTS

- We formulate a basic toxin-dependent population model.
- We investigate the effect of mercury on the persistence of rainbow trout population.
- Our results are consistent with surface water quality guidance in Alberta.
- Our model can be used to develop the guideline for the protection of aquatic life.

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ABSTRACT

Mathematical models have been widely applied to perform chemical risk assessments on biological populations for a variety of ecotoxicological processes. In this paper, by introducing a dose-dependent mortality rate function, we formulate a toxin-dependent aquatic population model that integrates mortality as toxin effect in addition to considering the effects of toxin on growth and recruitment. The model describes the direct effect of toxin on population by treating the concentration of toxin in the environment as a parameter. The model is more convenient to connect with data than traditional differential equation models that describe the interaction between toxin and population. We analyze the positive invariant region and the stability of boundary and interior steady states. The model is connected to experimental data via model parametrization. In particular, we consider the toxic effects of mercury on rainbow trout (*Oncorhynchus mykiss*) and obtain an appropriate range for each model parameter. The parameter estimates are then used to illustrate the long-time behavior of the population under investigation. The numerical results provide threshold values of toxin concentration in the environment to keep the population from extirpation. The findings are consistent with surface water quality guidelines. It may be appropriate to apply our model to other species and other chemicals of interest to consider guideline development.

1. Introduction

Anthropogenic and natural environmental contaminants are a common problem and a source of concern to ecosystem health. Industrial contaminants may arise as a result of air emissions, water releases, water seepage, air deposition or solid waste. Contaminants of concern may also be transported through natural systems as a result of weathering or leaching. Contaminants such as petroleum hydrocarbons, heavy metals and pesticides can cause toxic effects when released into aquatic environments. The US Environmental Protection Agency (EPA) has designated 126 priority pollutants and the Canadian Council of Ministers of the Environment (CCME) has a list of priority chemicals of concern for the protection of aquatic life. These priority substances include metals and organic compounds. USEPA and CCME have established a series of guidelines aiming to derive ambient water quality criteria for aquatic life for priority chemicals.

The effect of a toxic chemical can, in principle, be exerted on all levels of the biological hierarchy, from cells to organs to organisms to populations to entire ecosystems. Over the past several decades, ecotoxicological models have been applied increasingly to perform chemical risk assessments on a variety of ecological processes. These models include population models (scalar abundance, life history, individual-based, and metapopulation), ecosystem models (food-web,

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aquatic and terrestrial), landscape models, and toxicity-extrapolation models (Bartell et al., 2003; Galic et al., 2010; Pastorok et al., 2003; Pastorok et al.). The selection of specific models for addressing an ecological risk issue depends on the habitat, endpoints, and chemicals of interest, the balance between model complexity and availability of data, the degree of site specificity of available models, and the risk issue (Pastorok et al.,). A comprehensive review on the realism, relevance, and applicability of different types of models from the perspective of assessing risks posed by toxic chemicals is provided in Bartell et al. (2003) and Pastorok et al.

In practice, applying population models to chemical risk assessment is more cost-effective than using ecosystem and landscape models (Pastorok et al., 2003). For instance, toxin-dependent individual-based models and matrix population models are widely used to address the risk issue (Galic et al., 2010). Individual-based models include several key individual-level processes, such as behavior, growth, survival, and reproduction success. However, ignoring population-or higher-level effects and focusing only on individual-level endpoints can lead to inaccurate risk estimates and possible errors in environmental management decisions (Pastorok et al., 2003). The matrix models are often used to describe the fish and wildlife dynamic where survival rate and fecundity are functions of the age or stage to which an organism belongs. However, assessing the effects of toxin on vital rates in fish and wildlife population requires data on life-cycle toxicity testing. More often than not, the available population data is much more incomplete, numerous assumptions must be made to calculate age/stage-specific survival and fecundity (Pastorok et al.).

In this study, we investigate the effect of contaminants dissolved in water on fish population dynamics using a toxin-dependent differential equation model. Literature search results show that relatively few researchers use differential equation models to assess the effects of toxins on population dynamics. In a series of papers, Hallam et al. (1983), Hallam and Clark (1983), and Luna and Hallam (1987) modeled the interaction between toxin in the environment and population by assuming that the growth rate of population density linearly depends upon the toxicant concentration in the population but did not consider the effect of environmental toxin on the population carrying capacity. Freedman and Shukla (1991) and Thomas et al. (1996) therefore modified these models by allowing the carrying capacity to also be dependent on the exogeneous introduction of toxin. A common feature of those models is that the population growth rate is modeled by the logistic equation. We point out that it is not easy to quantitatively analyze the effect of toxin on the carrying capacity of the population. Thieme (2003) proposed a model using the Beverton–Holt equation instead of the logistic equation to describe the growth rate of population. The Beverton–Holt formula allows us to differentiate between the impacts of the toxin on food uptake, food conversion, and biomass gain. However, in his model the toxin concentration in the population only affects the growth rate of the population, but not the mortality rate of the population.

In consideration of the fact that the concentration of toxin in the environment, in reality, is not affected significantly by the metabolic process of population, in this paper, we mainly focus on the effect of toxin on population and ignore the influence of population on the concentration of toxin in the environment. By introducing a dose-dependent mortality rate function, we derive a basic toxin-dependent aquatic population model which extends Thieme's model. In particular, we use the power law to reflect the relationship between toxin concentration per unit population biomass (*body burden*) and population mortality rate. Our model consists of two equations. One equation describes the population growth rate where the birth and death rates are explicit functions of body burden. The other one is the balance equation for the body burden which describes the accumulation and dilution of toxin in the organisms body.

The main objective of this study is to investigate the effect of toxin on the population-level endpoints inferred from individual-level endpoints. To this end, we choose a native, threatened, fish species in North America, rainbow trout, as the study focus, and consider the effect of mercury on its dynamics through increased mortality and reduced reproductive success. Existing data and published studies for rainbow trout and mercury are used to estimate the reasonable range of all model parameters. The estimated parameters are then used to understand the effect of toxin on the long-time behavior of a population and make predictions on the effect of mercury on population stability and persistence.

The rest of the paper is organized as follows. In Section 2, we develop a toxin-dependent aquatic population model. In Section 3, we present a qualitative analysis for the model. We analyze the positive invariant region and investigate the existence and stability of boundary and interior equilibrium. In Section 4, we connect the model to experimental data via model parametrization. In Section 5, the results of model parametrization are used to numerically solve the model, and the results of the effect of the toxin on the end behavior of the population are provided. Finally a brief discussion section completes the paper.

2. Model formulation

The state variables of the model are $x=x(t)$, the concentration of biomass of the population in g/L at time t; $I=I(t)$, the concentration of toxin in μg/L in the population biomass at time t; $E=E(t)$, concentration of toxin in μg/L in the environment at time t; $y=y(t)$, the concentration of toxin per unit population biomass in μg/g at time t (*body burden*).

A model for the interaction between population and toxin is proposed in Thieme (2003) as follows:

$$\begin{cases} \dfrac{dx}{dt} = [\beta(x,y)-\mu]x \\ \dfrac{dI}{dt} = aEx-[\eta+\xi+\mu]I \\ \dfrac{dE}{dt} = -aEx+[\eta+q\mu]I-\theta E+u(t) \\ y = \dfrac{I}{x} \end{cases} \tag{2.1}$$

with appropriate initial conditions.

The first equation presents a generic description of the growth of the population under the influence of the toxin, while the second and third equations are balance equations for the concentration of the toxin contained in the individuals of the population and dissolved in the aquatic environment.

The positive constant μ represents the per unit biomass loss rate of the population due to death, and $\beta(x,y)$ denotes the per unit rate of biomass growth of the population. The toxin uptake rate by the population from the environment, aEx, is modeled according to the *Law of Mass Action* and is proportional to both the concentration of toxin in the environment and the concentration of population biomass. The positive constants η and ξ are per unit rates of toxin egestion and depuration, respectively due to the metabolic processes of the population. $q\in[0,1]$ is a fixed fraction by which the internal toxin is recycled into the environment. The parameter θ denotes the per unit rate of environmental detoxification. The time-dependent function u in the third equation stands for the exogenous input of toxin into the environment.

It is instructive to write down the equation for the body burden y. Since

$$y' = \frac{I'}{x} - y\frac{x'}{x} = aE-[\eta+\xi+\mu]y-y[\beta(x,y)-\mu],$$

we obtain

$$y' = aE - [\eta + \xi + \beta(x,y)]y.$$

We point out 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. For this reason, we regard the concentration of toxin in the environment as a parameter in this study.

In addition, notice that in the model (2.1), the toxin only affects the per unit biomass growth rate, but not the per unit biomass death rate. To amend this, we introduce a dose-dependent function which measures the effect of toxin upon mortality rate and consider the following basic toxin-dependent population model:

$$\begin{cases} \dfrac{dx}{dt} = [\beta(x,y) - \mu(y)]x \\ \dfrac{dy}{dt} = aE - [\sigma + \beta(x,y)]y, \end{cases} \tag{2.2}$$

where $\sigma = \eta + \xi$. We let

$$\beta(x,y) = \frac{\alpha_1[1-\alpha_2 y]_+}{1+\alpha_3 x}, \tag{2.3}$$

with positive constants α_i $(i=1,2,3)$ and $[r]_+ = \max\{0, r\}$ being the positive part of a real number r. A derivation of the above expression 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 the power law to study the relationship between toxin concentration and mortality rate and it has since been shown to fit the data well (Miller and Janszen, 2000). Thus, taking natural mortality rate into account, we let

$$\mu(y) = ky^l + m, \tag{2.4}$$

where k, l, and m are positive constants.

The variables and parameters for the basic models (2.2)–(2.4) which we shall investigate are listed in Table 1.

3. Mathematical analysis

In this section, we assume for convenience that $l=1$, hence the mortality rate function takes the linear form: $\mu(y) = ky + m$.

Table 1
List of variables and parameters.

Symbol	Definition	Unit
$x(t)$	Concentration of biomass at time t	g/L
$y(t)$	Body burden at time t	μg/g
β	Gain rate of the population	day^{-1}
μ	Loss rate of the population	day^{-1}
a	Uptake coefficient	day^{-1}
E	Concentration of toxin in the environment	μg/L
σ	Egestion and depuration rate	day^{-1}
α_1	Maximum growth rate	day^{-1}
α_2	Effect coefficient of toxin on the growth of population	g/μg
α_3	Crowding effect	L/g
k	Coefficient of power function	day^{-1}
l	Exponent of power function	
m	Natural mortality rate	day^{-1}

We rescale the systems (2.2)–(2.4) by setting

$$\tilde{x} = \alpha_3 x, \quad \tilde{y} = \alpha_2 y, \quad \tilde{t} = \alpha_1 t, \quad \tilde{k} = \frac{k}{\alpha_1 \alpha_2}, \quad \tilde{m} = \frac{m}{\alpha_1}, \quad \tilde{a} = \frac{\alpha_2 aE}{\alpha_1},$$

$$\tilde{\sigma} = \frac{\sigma}{\alpha_1}.$$

We drop the tildes for convenience, so that the system (2.2) becomes

$$\begin{cases} \dfrac{dx}{dt} = \left(\dfrac{[1-y]_+}{1+x} - ky - m\right)x \\ \dfrac{dy}{dt} = a - \left(\sigma + \dfrac{[1-y]_+}{1+x}\right)y. \end{cases} \tag{3.1}$$

3.1. Basic results

We shall first show that all solutions initiating in the nonnegative cone are eventually uniformly bounded and enter a certain region as described below.

Theorem 3.1. *The system (3.1) is dissipative with a positively invariant set Ω defined by*

$$\Omega = \left\{(x,y) \in \mathbb{R}^2_+ : 0 < x < \frac{1}{m}, 0 < y < \frac{a}{\sigma}\right\}.$$

Proof. Positivity obviously holds for the system. On the upper boundary of Ω, $y = a/\sigma$, $x \in [0,\ 1/m]$, $dy/dt \le a - \sigma(a/\sigma) = 0$. On the right boundary of Ω, $x = 1/m$, $y \in (0,\ a/\sigma]$, $dx/dt < 1 - m(1/m) = 0$. Therefore, all orbits starting from Ω cannot escape from its boundary. □

The next Theorem shows that the nonexistence of limit cycles when $k \ge 1$.

Theorem 3.2. *If $k \ge 1$, then the system (3.1) has no nontrivial periodic solution.*

Proof. The vector field defined by system (3.1) is locally Lipschitz-continuous in Ω, which guarantees the existence and uniqueness of solutions of system (3.1).

If $a \le \sigma$, the vector field is C^1, Bendixson's Criterion implies the desired conclusion.

However, this vector field is not C^1 if $a > \sigma$, so the classical Dulac's criterion cannot be applied. Fortunately, there is a generalized Dulac's criterion for locally Lipschitz-continuous planar systems

$$\frac{d\mathbf{x}}{dt} = f(\mathbf{x}), \mathbf{x} \in \Omega \subset \mathbb{R}^2. \tag{3.2}$$

If Ω is a simply connected, bounded, open subset of $\mathbb{R}^2$, and there exist a C^1 function $\chi : \Omega \to \mathbb{R}$ and a constant $c > 0$ such that

$$\operatorname{div}(\chi(\mathbf{x})f(\mathbf{x})) \le -c, \quad a.e. \quad \text{in} \quad \Omega,$$

then every compact limit set of (3.2) in Ω consists of equilibria and every compact invariant set of (3.2) in Ω is a set of equilibria and heteroclinic orbits containing no heteroclinic cycles, for instance, see Theorem 9 in Sanchez (2005); here a heteroclinic cycle is a Jordan curve that consists of equilibria and heteroclinic (or homoclinic) orbits of (3.2). Let $\Omega_1 = \{(x,y) \in \mathbb{R}^2_+ : 0 < x < 1/m, 0 < y < 1\}$ and $\Omega_2 = \{(x,y) \in \mathbb{R}^2_+ : 0 < x < 1/m, 1 < y < a/\sigma\}$. We choose $\chi(x,y) = 1$.

Simple computations yield that in region Ω_1,

$$\begin{aligned}\operatorname{div}(\chi(\mathbf{x})f(\mathbf{x})) &= \frac{y(1+2x)}{(1+x)^2}-\frac{x}{(1+x)^2}-ky-m-\sigma\\ &\le (1-k)y-\frac{x}{(1+x)^2}-m-\sigma\\ &\le -m-\sigma.\end{aligned}$$

In region Ω_2,

$$\operatorname{div}(\chi(\mathbf{x})f(\mathbf{x})) = -ky-m-\sigma \le -m-\sigma.$$

Define $c=m+\sigma>0$. The generalized Dulac's criterion can be applied to system (3.1) in Ω and thus the proof is completed. □

3.2. Existence of equilibria

In order to explore the equilibrium, we set the vector field in (3.1) equal to 0 and obtain the equilibrium equations

$$\left(\frac{[1-y]_+}{1+x}-ky-m\right)x=0,$$

$$a-\left(\sigma+\frac{[1-y]_+}{1+x}\right)y=0.$$

From the first equation, we have

$$x=0 \quad \text{or} \quad \frac{[1-y]_+}{1+x}=ky+m.$$

$x=0$ will give rise to so called boundary equilibrium (extinction equilibrium) $(0,y^*)$. Substituting $x=0$ into the second equilibrium equation, we obtain

$$a=(\sigma+[1-y^*]_+)y^* =: \phi(y^*).$$

We notice that $\phi(0)=0$ and $\phi(y)\to\infty$ as $y\to\infty$. So there is always a positive solution to this equation and we will have at least one boundary equilibrium.

Actually, if $a\ge\sigma$, then we have only one boundary equilibrium

$$E_0=\left(0,\frac{a}{\sigma}\right).$$

If $a<\sigma$, then we can solve

$$(\sigma+(1-y^*))y^*=a. \tag{3.3}$$

to get another boundary equilibrium $(0,\ y^*)$ with $y^*<1$. In fact, solving (3.3) yields

$$y^*=\frac{1+\sigma\pm\sqrt{(1+\sigma)^2-4a}}{2},$$

Clearly, if $a<\sigma$, then $(1+\sigma+\sqrt{(1+\sigma)^2-4a}/2)>1$ and $(1+\sigma-\sqrt{(1+\sigma)^2-4a}/2)<1$. That is to say, If $a<\sigma$, we have only one boundary equilibrium

$$E_1=\left(0,\frac{1+\sigma-\sqrt{(1+\sigma)^2-4a}}{2}\right).$$

Let us turn to the alternative $x>0$ and $([1-y]_+)/1+x=ky+m$. This will give rise to a so called interior equilibrium (survival equilibrium) $(x^\star,\ y^\star)$. Substituting $([1-y]_+)/1+x=ky+m$ into the second equilibrium equation leads to

$$a=(\sigma+ky^\star+m)y^\star.$$

Since $y^\star>0$, we get

$$y^\star=\frac{-\sigma-m+\sqrt{(\sigma+m)^2+4ka}}{2k}.$$

Thus, the interior equilibrium exists if and only if $y^\star<1$ and $(1-y^\star/ky^\star+m)-1>0$ are satisfied. This leads to the following conditions:

$$0<m<1, a<\min\left\{k+\sigma+m,\frac{(1-m)(k\sigma+\sigma+m+k)}{(k+1)^2}\right\}.$$

Notice that

$$\frac{(1-m)(k\sigma+\sigma+m+k)}{(k+1)^2}<\frac{(k+\sigma+m)(k+1)}{(k+1)^2}<\frac{(k+\sigma+m)(k+1)^2}{(k+1)^2}=k+\sigma+m.$$

Therefore, the system (3.1) has interior equilibrium

$$E_2=\left(\frac{1-y^\star}{ky^\star+m}-1,y^\star\right),$$

if and only if the following conditions hold:

$$0<m<1, a<\frac{(1-m)(k\sigma+\sigma+m+k)}{(k+1)^2} := R. \tag{3.4}$$

According to the above discussion, depending on the values of m, a, σ, and R. The existence of boundary and interior equilibria and corresponding conditions required are summarized in Table 2.

From Table 2, we see that the extinction equilibria always exist. In particular, if the uptake rate is greater than the elimination rate (the sum of egestion rate and depuration rate), i.e., $a\ge\sigma$, then we have the extinction equilibrium E_0 with body burden $y>1$. If the uptake rate is less than the elimination rate, i.e., $a\ge\sigma$, then we have extinction equilibrium E_0 with body burden $y<1$. The survival equilibrium exists if and only if the natural mortality rate m is less than 1 and the uptake rate is less than the threshold value, R, which is determined by the elimination rate, σ, and the values of k, l, m in the expression of the mortality rate function.

3.3. Stability of equilibria

Theorem 3.3. *The boundary equilibria E_0 or E_1 are globally asymptotically stable if threshold conditions are not satisfied.*

Proof. Since the y coordinates of E_0 is greater than 1, the Jacobian matrix of system (3.1) satisfies

$$J(E_0)=\begin{pmatrix}-k\frac{a}{\sigma}-m & 0\\ 0 & -\sigma\end{pmatrix}.$$

The eigenvalues of $J(E_0)$ are both negative, hence E_0 is locally asymptotically stable.

Since the y coordinates of E_1 is less than 1, the Jacobian matrix evaluated at E_1 satisfies

$$J(E_1)=\begin{pmatrix}1-y^*-ky^*-m & 0\\ (1-y^*)y^* & -\sigma-1+2y^*\end{pmatrix}.$$

Thus, the eigenvalues of $J(E_1)$ are

$$\lambda_1=1-y^*-ky^*-m=1-m-(k+1)\frac{1+\sigma-\sqrt{(1+\sigma)^2-4a}}{2}<0,$$

Table 2
The existence of boundary and interior equilibrium.

Condition	Boundary equilibrium	Interior equilibrium
$m\ge1, a\ge\sigma$	E_0	None
$m\ge1, a<\sigma$	E_1	None
$0<m<1, R<\sigma, a\ge\sigma$	E_0	None
$0<m<1, R<\sigma, R\le a<\sigma$	E_1	None
$0<m<1, R<\sigma, a<R$	E_1	E_2
$0<m<1, R\ge\sigma, a\ge R$	E_0	None
$0<m<1, R\ge\sigma, \sigma\le a<R$	E_0	E_2
$0<m<1, R\ge\sigma, a<\sigma$	E_1	E_2

$$\lambda_2 = -\sigma - 1 + (1+\sigma - \sqrt{(1+\sigma)^2 - 4a}) = -\sqrt{(1+\sigma)^2 - 4a} < 0.$$

Hence, E_1 is locally asymptotically stable.

If $a \geq \sigma$, only the boundary equilibrium E_0 is feasible. Because solutions are bounded, the solutions must converge to E_0.

Similarly, If $a < \sigma$, only the boundary equilibrium E_1 is feasible, E_1 is globally asymptotically stable. □

Theorem 3.4. *Assume that the interior equilibrium E_2 exists, then E_2 is locally asymptotically stable if*

$$(k^2 + 3k)(y^\star)^2 + (2km - 2k + 3m + \sigma)y^\star + m^2 - 2m - \sigma < 0. \tag{3.5}$$

Proof. Since the y coordinates of E_2 is less than 1, the Jacobian matrix evaluated at E_2 satisfies

$$J(E_2) = \begin{pmatrix} \frac{1-y^\star}{(1+x^\star)^2} - ky^\star - m & -\frac{x^\star}{1+x^\star} - kx^\star \\ \frac{(1-y^\star)y^\star}{(1+x^\star)^2} & -\sigma - \frac{1-2y^\star}{1+x^\star} \end{pmatrix}.$$

Noticing that $x^\star = (1-y^\star/ky^\star + m) - 1$, we have

$$\mathrm{Tr}(J(E_2)) = \frac{(ky^\star + m)^2}{1-y^\star} - ky^\star - m - \sigma - \frac{(1-2y^\star)(ky^\star + m)}{1-y^\star},$$

$$\det(J(E_2)) = \frac{(ky^\star + m)(ky^\star + y^\star - 1 + m)(2ky^\star + \sigma + m)}{y^\star - 1}.$$

Since $0 < y^\star < (1 - m/k + 1)$, $\det(J(E_2)) > 0$.

It is easy to check that $\mathrm{Tr}(J(E_2)) < 0$ if (3.5) holds. □

Remark 1. Since $0 < y^\star < (1 - m/k + 1)$, clearly, $\mathrm{Tr}(J(E_2))|_{y^\star = 0} < 0$ and

$$\mathrm{Tr}(J(E_2))|_{y^\star = (1-m)/(k+1)} = -\frac{2m + k + k\sigma + \sigma - 1}{k+1}.$$

Therefore, a simpler sufficient condition on the locally asymptotically stability of E_2 is $2m + k + k\sigma + \sigma > 1$.

4. Parameterization

In this section we describe the parameterization of (2.2)–(2.4). While the model is general, we choose to apply it to a representative species, rainbow trout (*Oncorhynchus mykiss*), and consider the effect of mercury on the fish dynamics. Rainbow trout is found widely throughout the world. However some populations such as the native rainbow trout population in the Athabasca River, Alberta, and rainbow trout found in watersheds west of the Cascade Mountains in the U.S. are threatened (http://albertafishingguide.com/fish/rainbow-trout; http://www.fws.gov/northeast/wssnfh/pdfs/RAINBOW1.pdf). Mercury may be released into the aquatic environment in states of relatively low toxicity, but will be transformed into highly toxic states, namely methylmercury. Mercury's harmful effects on fish include death, reduced reproduction, slower growth and development, and abnormal behavior (Eisler, 1987).

4.1. Functional form for mortality rate $\mu(y)$

To investigate the relationship between the concentration of methylmercury per unit biomass of rainbow trout (body burden) and the mortality rate, we use the data from an experiment in which the percent mortality of rainbow trout injected with methylmercury over a 15 day period was recorded (Hawryshyn and Mackay, 1979). The findings are in agreement with a later evaluation of other studies which found that whole body concentrations of 10–20 μg/g methylmercury could be lethal to fish (Niimi and Kissoon, 1994).

To apply the experimental data from Hawryshyn and Mackay (1979) to estimate constants k and l in (2.4), we introduce a Poisson process to describe survival probability which corresponds to percent mortality of rainbow trout. A Poisson process says that if our time step, h, is sufficiently small then the probability of an event occurring is roughly proportional to h (Grimmett and Stirzaker). Let $p(t)$ be the probability that an individual survives until time t and $p(t+h|t)$ be the probability that an individual which lives until time t will survive until time $t+h$. Then by the Possion process, the probability that an individual dies over the time interval h is

$$1 - p(t+h|t) = \lambda h + o(h).$$

Thus,

$$p(t+h) = p(t)p(t+h|t) = p(t)(1 - \lambda h + o(h))$$

Hence,

$$p(t+h) - p(t) = -\lambda h p(t) + o(h)$$

Dividing through by h and letting $h \to 0$, we get

$$p'(t) = -\lambda p(t),$$

it follows that the probability of survival and mortality are

$$p(t) = e^{-\lambda t}$$

and

$$1 - e^{-\lambda t} := p_0(t),$$

respectively. We set $\lambda = ky^l$ and obtain

$$p_0(t) = 1 - \exp\left\{-ky^l t\right\}.$$

Using the above mortality rate function and employing Matlab routine LSQCURVEFIT to fit the data in Hawryshyn and Mackay (1979), we obtain parameter estimates $k = 0.00398$, $l = 1.489$ with 95% confidence intervals $k \in [0.000864,\ 0.00710]$, $l \in [0.274,\ 2.705]$. The fitting results are plotted in Fig. 1.

As for the natural mortality rate, m, we take $m \in [1/(365 \times 6),\ 1/(365 \times 4)]$ in day^{-1} since the usual life span of rainbow trouts is four to six years (http://www3.northern.edu/natsource/FISH/Rainbo1.htm).

4.2. Maximum gain rate of biomass: α_1

Both reproduction and individual growth result in the gain of population biomass. To measure the maximum gain rate of biomass, we let $\alpha_1 = c\hat{\alpha}_1$, here c is a scaling factor which lies in the range of no growth to immediate growth, for instance, setting

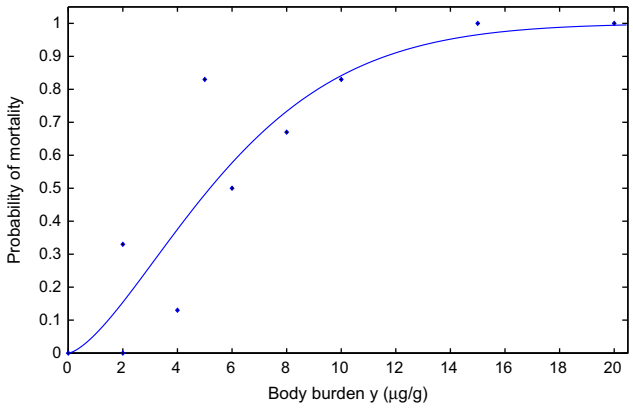

Fig. 1. Comparison of the experimental data from Hawryshyn and Mackay (1979) (where the rainbow trout were exposed in mercury environment for 15 days) with the percent mortality function ($p_0(t) = 1 - \exp\left\{-ky^l t\right\}$) output with estimated parameters.

J_w=weight of juveniles and A_w=weight of adults, we have

$$\frac{J_w}{A_w} \le c \le \frac{A_w}{A_w} = 1.$$

We choose J_w=21.7 g and A_w=553.5 g which are mean weights of juveniles and adults rainbow trout from Anderson et al. (1997) and get $c \in [0.0392, 1]$.

The maximum reproduction rate of fish at low population sizes is estimated in Myers et al. (1999) by employing variance components models and examining a database of over 700 spawner-recruitment series. The estimated results show that the maximum annual reproduction rate, denoted by $\tilde{\alpha}$, over seven species from the Salmonidae family is $\tilde{\alpha} \in [18.9104,\ 38.6456]$, Letting $\exp\{365\hat{\alpha}_1\} = \tilde{\alpha}$, we obtain an intrinsic reproduction rate $\hat{\alpha}_1 = [0.0081,\ 0.010]$ in day^{-1}. To shrink the range of α_1, we take the midpoint of the estimate interval for c and obtain the estimate: $\alpha_1 \in [0.0042,\ 0.0052]$ in day^{-1}.

4.3. Effect of toxin on reproduction and growth: α_2

It is not easy to incorporate the separate effects of the toxin on reproduction and on individual growth into a single parameter. For simplicity, we only consider the effect of toxin on fish reproduction to estimate α_2. The effects of mercury on the reproduction of fish and amphibians have been studied in Birge et al. (1979). Therein, eyed eggs of the rainbow trout were treated from 10 days pre-hatching through 10 days post-hatching, the data about the relationships between the body burden of embryo-larval stages of rainbow trout and the percent survival are recorded.

To use the data from Birge et al. (1979) to estimate α_2, we first assume that the mortality rate of embryo-larval stages (denoted by L) of rainbow trout linearly depends on the body burden, in particular, we have

$$\frac{dL}{dt} = -\overline{\alpha}_2 y L.$$

So

$$L(t) = L(0)e^{-\overline{\alpha}_2 y t}.$$

Letting $L(0)=1$, we define percent survival of embryo-larval stages, denoted by S, as

$$S = e^{-\overline{\alpha}_2 y t}.$$

We fit the data from Birge et al. (1979) to the above survival function (see Fig. 2) and obtain $\overline{\alpha}_2 = 0.495$ day^{-1}.

Note that the term $[1-\alpha_2 y]_+$ in the basic model represents the fraction of successful reproduction which linearly depends on the body burden y. We roughly think of the successful reproduction as an eyed egg being able to survival until it becomes a fry, which is the first time the trout acts like a fish. We assume that it takes T days for an eyed eggs to become a fry. From the life cycle of rainbow trout (Miranda), we find that it takes about 20–80 days for the embryos to develop. Once the egg hatches, it is referred to as an alevin. It will take the alevin two to three weeks to become a fry. Therefore, we choose $T \in [34,\ 101]$ in days. Since the probability that an eyed egg survives until it becomes a fry (i.e., successful reproduction) is

$$e^{-\overline{\alpha}_2 y T} \approx 1 - \overline{\alpha}_2 T y.$$

Letting $\alpha_2 = \overline{\alpha}_2 T$, we obtain $\alpha_2 \in [16.83,\ 49.99]$ in g/μg.

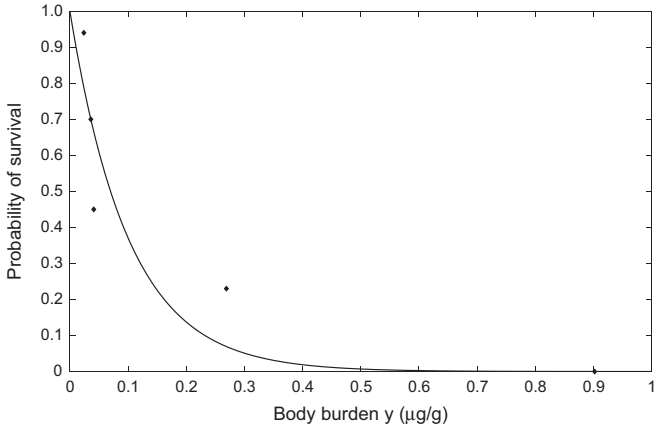

Fig. 2. Comparison of the experimental data from Birge et al. (1979) (where the eyed eggs were exposed in mercury environment for 20 days) with the percent survival function ($S = \exp\{-\overline{\alpha}_2 y t\}$) output with estimated parameters.

4.4. Crowding effect: α_3

If there is no effect of the toxin, the first equation of (2.2) can be written as

$$\frac{dx}{dt} = \left(\frac{\alpha_1}{1+\alpha_3 x} - m\right)x = \frac{(\alpha_1 - m)\left(1 - \frac{x}{K}\right)x}{1+\alpha_3 x}, \tag{4.1}$$

with

$$K = \frac{\alpha_1 - m}{m\alpha_3}. \tag{4.2}$$

Notice that $\alpha_1 - m$ is always positive from the estimated values for α_1 and m. It is not difficult to check that K plays the role of the carrying capacity as with the logistic equation.

In Johnson and Hasler (1954), the authors investigate the level of carrying capacity for a population of rainbow trout living in several small lakes and analyze the factors governing growth and carrying capacity. The carrying capacity of the rainbow trout population was estimated to be approximately 50 pounds per acre. Since the average depth of those lakes is between 5 m and 8 m, simple calculation gives the range of carrying capacity: $k \in [0.000701,\ 0.00112]$ in g/L. Taking the ranges of α_1 and m into account, we obtain that $\alpha_3 \in [4618.5,\ 14701.2]$ in L/g.

The uptake of the methylmercury appears to follow a two-stage processes (Rodgers and Beamish, 1982). A portion of the uptaken methylmercury remains associated with dietary components which are not assimilated and are eliminated rapidly with the feces. The remaining fraction of uptaken methylmercury is absorbed across the gut and incorporated into tissues. This assimilated fraction of uptaken methylmercury is depurated by the kidney, liver and possibly gills. According to the system (2.2), we need to estimate the uptake coefficient a, egestion rate η and depuration rate ξ. The egestion rates are highly positively correlated with daily food ingestion rate which may not be easy to measure (Murtaugh, 1984). For simplicity, quantitatively, we consider the following simpler model related to the second equation of (2.2):

$$\frac{dy}{dt} = \overline{a}E - [\xi + \beta(x,y)]y,$$

where $\overline{a}$ is the assimilation coefficient. In what follows, we will estimate the assimilation coefficient $\overline{a}$ and depuration rate ξ instead of a, η and ξ in the model (2.2).

4.5. Assimilation coefficient: $\overline{a}$

The relationship between concentration of methylmercury in water E (μg/L), methylmercury consumption rate C (ng/L per day), and methylmercury assimilation rate aE (μg/g per day) can be described by the regression equation (Phillips and Buhler, 1978)

$$\overline{a}E = 0.084E + 0.00068C.$$

Furthermore, The relationship between E and C (ng/L per day) can be described by $C = dE$, $d \in [111.43,\ 150.77]$. Simple calculation yield that $\overline{a} \in [0.1598,\ 0.1865]$.

4.6. Depuration rate: ξ

In Rodgers and Beamish (1982), the authors investigate the dynamics of dietary methylmercury in rainbow trout which were fed with diets containing 0, 25, 45, 75, 95 mg Hg kg^{-1} methylmercury for 84 days at meal sizes of 1% and 2% day^{-1} and to satiation. Experimental results show that methylmercury was depurated more rapidly in fish fed with the larger meal size. We take the full range of calculated daily depuration rates for methylmercury by rainbow trout, that is, $\xi \in [0.00516,\ 0.00733]$ in day^{-1}.

5. Numerical results

In this section, the resulting parameter estimates are used to numerically solve the basic models (2.2)–(2.4). Note that the estimated values of parameters are given by certain intervals. In our numerical simulations, we plan to deal with those parameters in the following two ways: (1) We take the midpoint of intervals as the corresponding parameter values and make deterministic simulations. (2) We regard all parameters as triangle distribution random variables where the most probable value are midpoints of the corresponding intervals and run simulations. More precisely, we randomly choose a value for each parameter (in the triangle distribution) which is fixed for each simulation.

5.1. Simulations with fixed parameters

We begin by understanding how the concentration of toxin in the environment affects the population biomass level. We describe the bifurcation dynamics as E changes from 0 to 0.005 μg/L which is represented in Fig. 3. It can be observed that the threshold value of E for the population extinction is around 0.0045 μg/L even though the stable population level becomes very low as E reaches around 0.0011 μg/L.

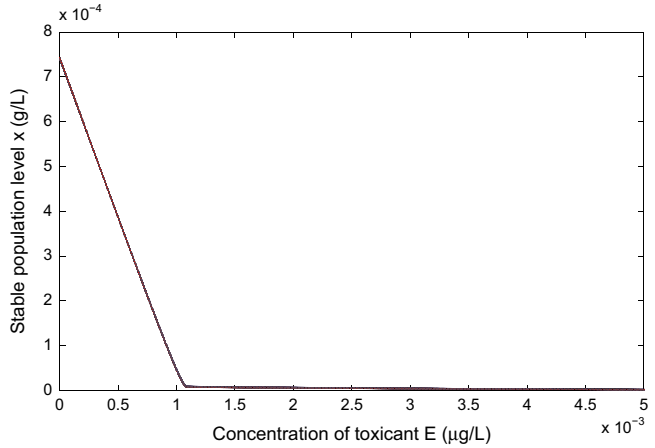

Fig. 3. The stable population level when the bifurcation parameter $E \in [0,\ 0.005]$ in μg/L.

We then compare the levels of population biomass and corresponding body burden under the conditions of three different toxin levels in the environment, $E = 0,0006,\ 0.0012,\ 0.012$ μg/L. Thus, we simulate the model for 20 years (7300 days) and the numerical results are plotted in Fig. 4. In particular, the Fig. 4 shows that the population biomass reaches stable levels when $E = 0.0006$ μg/L. However, the population will become extirpated after about 4380 days (12 years) when $E = 0.012$ in μg/L, and when $E = 0.0012$ μg/L the population will not go extinct after 20 years even though it is very low.

5.2. Simulations with random parameters

We assume that all parameters, except E, which are given by certain intervals are random variables with triangle distributions over the corresponding intervals and simulate the model for 20 years. We run 1000 realizations. The resulting histogram on the probability of population extinction as E changes discretely from 0.001 to 0.005 μg/L is presented in Fig. 5. Clearly, the probability of population extirpation increases from 0 to 1 as the concentration of toxicant in environment increases from 0.001 to 0.005 in μg/L. We point out that these simulation results replenish the deterministic results since the bifurcation diagram (Fig. 3) shows that the population decreases from a low level until it become extirpated when E increases from 0.0011 to 0.0045 μg/L.

These results can be compared to the water quality guidelines for the protection of aquatic life in Alberta. For methylmercury, the acute guideline is 0.002 μg/L and the chronic guideline is 0.001 μg/L (Alberta Environment, 1999). Acute guidelines are ordinarily defined as having no noticeable effect on aquatic life, while chronic guidelines may start to show some effects on the most sensitive species. The model results shows noticeable effects at comparable concentrations to the guidelines.

We also assume that parameter E is a random variable with triangle distribution over the interval [0.001, 0.005] in μg/L and plot the results of 100 realizations in Fig. 6.

6. Discussion

Mathematic models are useful tools for evaluating the ecological significance of observed or predicted effects of toxic chemicals on individual organisms and population dynamics. Few differential equation models have been developed to describe population-toxin interaction. 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. Such models have been investigated via a qualitative approach. In this paper, we assumed 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. We consider the direct influence of toxin on the population vital rates which is implemented through the body burden. Our toxin-dependent population model can be connected to data much more easily than traditional population-toxin interaction models.

Our study is both qualitative and quantitative. The results of qualitative model analysis shows that the extinction equilibrium always exists and is asymptotically stable, and the survival equilibrium exists only if the threshold condition (3.4) holds. Moreover, if the concentration of toxin in the environment (or if the toxin input into the environment) is appropriately controlled, the threshold condition (3.5) is satisfied, the population will persist. On the quantitative side, we connect the model to experimental data via model parametrization. In particular, we consider the toxic effects of mercury on rainbow trout and obtain

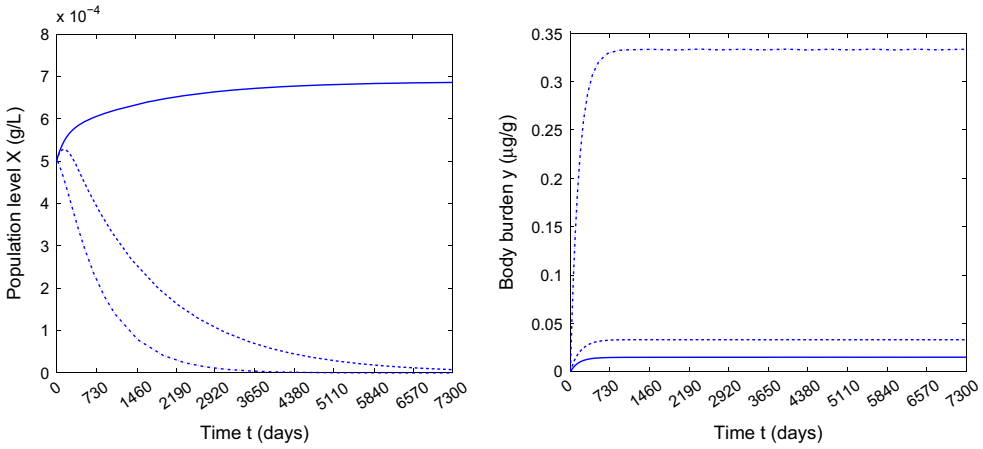

Fig. 4. Comparison of population biomass (left) and body burden (right) between different toxin levels. Solid lines ($E=0.0006$ μg/L), dash lines ($E=0.0012$ μg/L), dash-dot lines ($E=0.012$ μg/L).

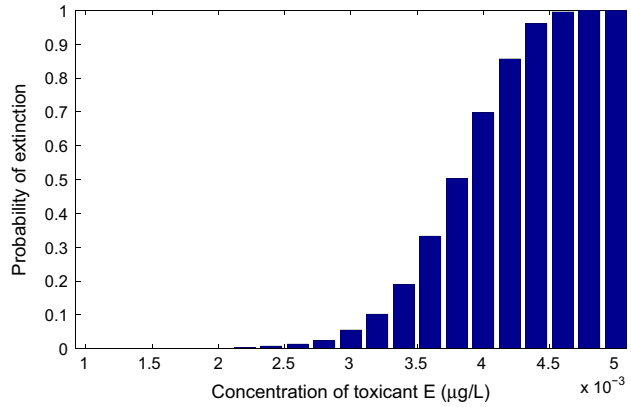

Fig. 5. Probability of extinction for different toxin concentrations in the environment. Simulations are based on 1000 samples.

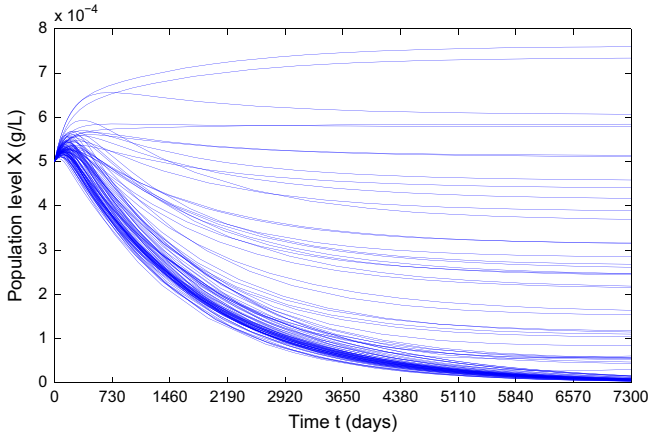

Fig. 6. 100 stochastic realizations of population biomass for 20 years using the stochastic parameters which have triangle distributions over corresponding intervals.

an appropriate range for each parameter. The parameter estimates are then used to illustrate the long-time behavior of the population under investigation. The numerical results provide the threshold value of toxin concentration in the environment to keep the population from extirpation. The findings are consistent (within the same order of magnitude) with surface water quality guideline. This is very promising in such a crude model. This indicates that it may be appropriate to expand the tool to consider other species and other chemicals of interest to consider guideline development.

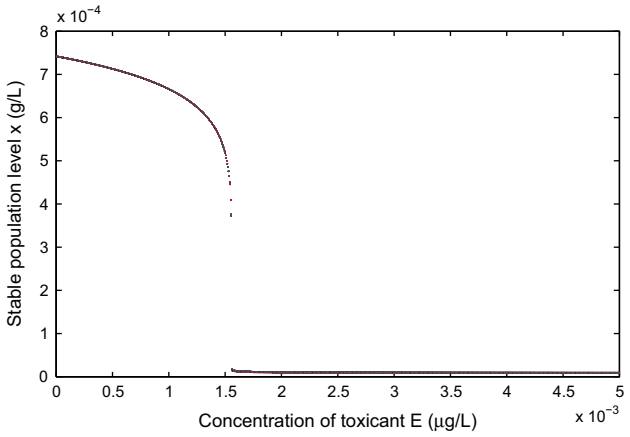

Fig. 7. A special example of bifurcation diagram using nonlinear growth rate function $\beta(x,y)=\alpha_1([1-\alpha_2 y]_+)^{0.5}/(1+\alpha_3 x)$.

The long-term simulations of the model indicate that a stable population level by the end of 20 years almost linearly depends on the bifurcation parameter E, the concentration of toxin in the environment (see Fig. 3). This is due to the fact that the rate of change of the population biomass almost linearly depends upon the body burden y and the body burden linearly depends upon E. As a special example, we let

$$\beta(x,y)=\frac{\alpha_1([1-\alpha_2 y]_+)^{0.5}}{1+\alpha_3 x}.$$

Using the same parameter estimate values we obtain the bifurcation diagram presented in Fig. 7. The big jump in the bifurcation diagram curve indicates that the toxin in the environment has little effect on the persistence of the population as its concentration E is less than the threshold value (around 0.0015 μg/L). However the population will become extirpated after 20 years when E is greater than the threshold value.

Our model extends Thieme's model by introducing a power function to reflect the relationship between body burden and population mortality rate. Considering that a threshold of effect of body burden on the mortality rate may exists, i.e., a sufficiently low toxin concentration is not lethal, one can use the following mortality rate function

$$\mu(y)=k([y-h]_+)^l+m,$$

where k, h, l, m are positive constants. To see the change in the population dynamics resulting from this modification, we use the

lethal rate function, $k([y-h]_+)^l$, to fit the same data as we used in Section 4.1. We obtain parameter estimates: $k=0.00498$, $l=1.406$, $h=0.2745$. By using the above mortality rate function and parameter estimates, and the same values for other parameters as we obtained in the Section 4, we get the same bifurcation diagram as Fig. 3. This implies that the introduction of a threshold h does not change the end behavior of the population. In fact, when the body burden y in Eq. (2.4) is sufficient low, say lower than the above threshold h, simple calculation shows that lethal rate by toxin is very low such that the population mortality rate is mainly determined by natural mortality rate m.

The basic toxin-dependent model (2.2)–(2.4) describes direct population–toxin interactions. As an application of the mathematical model (2.2)–(2.4), we investigate the effect of mercury on the persistence and likelihood of extinction of an rainbow trout (*Oncorhynchus mykiss*) population. We point out that the impact of mercury bioaccumulation on fishing and aquatic life has been well studied. For instance, many laboratory experiments have been conducted to study the effect of mercury on the reproduction, growth and mortality of fish (Birge et al., 1979; Hawryshyn and Mackay, 1979; Niimi and Kissoon, 1994; Rodgers and Beamish, 1982). In a series of papers, Trudel and Rasmussen (1997, 2001, 2006) and Trudel et al. (2000) used a mercury mass balance model which is very similar to the second equation of (2.1) to predict mercury accumulation in fish. The mercury mass model presented in those studies provided a flexible framework for understanding the factors affecting the concentration of mercury in fish. However, they did not consider the effect of mercury on the population dynamics. In this study, we connect the basic population–toxin interaction model to experimental data via model parametrization and investigate the effect of the concentration of mercury per unit population biomass (body burden) on the long-time behavior, such as the persistence and likelihood of extinction, of the rainbow trout population.

There is much to be done for future work. In our model, the function β represents the growth and reproduction combined which limit the applicability of the model. Juvenile individuals have the same concentration of toxin as adults. In order to separate the effects of the toxin on growth and reproduction, and taking it into account the fact that juveniles and adults have different responses to toxins, we will develop a stage-structured model for the population subject to toxins in the environment. In general, the intake of toxin, egestion may depend on age, weight, and/or size. For this reason, a weight/age/size structured model may need to be constructed.

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