A model for the impact of contaminants on fish population dynamics

Qihua Huang [a,*], Laura Parshotam [b], Hao Wang [a], Caroline Bampfylde [c], Mark A. Lewis [a,d]

[a] Centre for Mathematical Biology, Department of Mathematical and Statistical Sciences, University of Alberta, Edmonton, AB, Canada T6G 2G1
[b] Centre for Computational Science, Department of Chemistry, University College London, WC1H 0AJ, UK
[c] Clean Energy Policy Branch, Alberta Environment and Sustainable Resource Development, Edmonton, AB, Canada T5J 1G4
[d] Department of Biological Sciences, University of Alberta, AB, Canada T6G 2E9

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.

ARTICLE INFO

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,

* Corresponding author. Tel.: +1 7804920147.
  E-mail addresses: qihua@ualberta.ca (Q. Huang),
laura.parshotam.12@ucl.ac.uk (L. Parshotam), hao8@ualberta.ca (H. Wang),
caroline.bampfylde@gov.ab.ca (C. Bampfylde),
mark.lewis@ualberta.ca (M.A. Lewis).

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

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