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The impact of environmental toxins on predator–prey dynamics

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HIGHLIGHTS

- We develop a toxin-dependent predator–prey model.
- We examine how environmental toxin levels alter the balance of the classical predator–prey dynamics.
- We investigate the effect of methylmercury on rainbow trout and its prey.

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ABSTRACT

Predators and prey may be simultaneously exposed to environmental toxins, but one may be more susceptible than the other. To study the effects of environmental toxins on food web dynamics, we develop a toxin-dependent predator–prey model that combines both direct and indirect toxic effects on two trophic levels. The direct effects of toxins typically reduce organism abundance by increasing mortality or reducing fecundity. Such direct effects, therefore, alter both bottom-up food availability and top-down predatory ability. However, the indirect effects, when mediated through predator–prey interactions, may lead to counterintuitive effects. This study investigates how the balance of the classical predator–prey dynamics changes as a function of environmental toxin levels. While high toxin concentrations are shown to be harmful to both species, possibly leading to extirpation of both species, intermediate toxin concentrations may affect predators disproportionately through biomagnification, leading to reduced abundance of predators and increased abundance of the prey. This counterintuitive effect significantly increases biomass at the lower trophic level. Environmental toxins may also reduce population variability by preventing populations from fluctuating around a coexistence equilibrium. Finally, environmental toxins may induce bistable dynamics, in which different initial population levels produce different long-term outcomes. Since our toxin-dependent predator–prey model is general, the theory developed here not only provides a sound foundation for population or community effects of toxicity, but also could be used to help develop management strategies to preserve and restore the integrity of contaminated habitats.

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. 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 (U.S. National Archives and Records Administration, 2013) and the Canadian Council of Ministers of the Environment has a list of priority chemicals of concern for the protection of aquatic life (Canadian Council of Ministers of the Environment, 2003a). These priority substances include metals and organic compounds.

The combination of natural and anthropogenic sources of toxins present challenges with respect to the protection of local freshwater resources. To protect ecological environments and aquatic species, 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. Kooijman and Bedaux (1996) describe how the no-effect concentration can be estimated from data of standardized aquatic toxicity tests: acute and chronic survival, body growth, reproduction, and population growth.

Over the past several decades, mathematical models have been widely applied to perform chemical risk assessments on all levels

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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 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. Despite the nonlinear dynamical nature of population-toxin interactions, our search of the literature shows that relatively few differential equation models have been developed to describe population-toxin interactions (but see Freedman and Shukla, 1991; Hallam and Clark, 1983; Hallam et al., 1983; Luna and Hallam, 1987; Thieme, 2003; Thomas et al., 1996). For those models that do exist, 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). Because the concentration of toxin in the environment is not affected significantly by mortality or metabolic processes of population, our toxin-dependent model focused on the impact of toxin on the population and ignores the influence of the population on the concentration of toxin in the environment. The concentration of toxin in the environment hence was treated as a parameter. The model was connected to literature-sourced experimental data via model parameterization of the toxic effects of methylmercury on rainbow trout (*Oncorhynchus mykiss*). The parameter estimates were then used to illustrate the long-term behavior of rainbow trout population. The numerical results provided threshold values of concentration of methylmercury in the environment to maintain populations and prevent extirpation.

It is significant that all above-mentioned differential equation models are single-species models in which populations are assumed to take up toxin only from exposure to the environment. 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 the pollutants that are eaten and stored. This build-up of toxic pollutants is referred to as bioaccumulation (Arnot and Gobas, 2004; Mackay and Fraser, 2000; Mathew et al., 2008). Bioaccumulation means that the nonlinear effects observed in ecosystems cannot often be described or understood through studying species individually because food web interactions must be considered (e.g., Kidd et al., 2007). A review on bioaccumulation criteria and methods is provided in Gobas et al. (2009). Moreover, Kelly et al. (2007) and Thomann (1989) developed bioaccumulation models.

In this work, we evaluate the flow of contaminants through a simple aquatic food web and study how the transfer of contaminants between trophic levels affects food web dynamics. We do this by extending the single-species toxin-dependent model in Huang et al. (2013) to a predator–prey model with toxin effect. Our 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. Body burden, which is also referred to as tissue residue in McElroy et al. (2010), is a direct measurement of toxin concentrations in the tissue or organism rather than in the exposure media. 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.

This 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 analyze the effect of the methylmercury on the end behavior of rainbow trout and its prey (small fish or aquatic insects). To qualitatively investigate the model, we simplify it to a two-dimensional system via a quasi-steady state approximation. We analyze the quasi-steady system by studying the effect of toxin level in the environment on existence and stability of steady states.

If there is no toxin, our toxin-dependent predator–prey model reduces to a classical predator–prey model, whose dynamics have been well studied. Thus, 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 higher levels. From our analysis and numerical exploration of the food web toxin model we found that toxin concentrations affect organisms at different trophic levels in a variety of 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 a 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 towards only one stable steady 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.

The rest of this paper is organized as follows. In Section 2, we develop a toxin-dependent predator–prey model. In Section 3, we connect the model to experiment data via model parameterization. We apply the results of model parameterization to consider the toxic effects of mercury on rainbow trout and its prey (small fish and aquatic insects). In Section 4, we reduce the dimensionality of the model using a quasi-steady state approximation. We then analyze the existence and stability of extinction 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 in the environment affects the long-term behavior of the populations. Finally, a brief “Discussion” section completes the paper.

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

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