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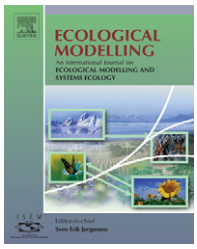

Sublethal toxic effects in a simple aquatic food chain

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ABSTRACT

In this paper, we study the sublethal effect of toxicants on the functioning (biomass production, nutrient recycling) and structure (species composition and complexity) of a simple aquatic ecosystem in a well-mixed environment (chemostat reactor). The modelled ecosystem consists of a nutrient consumed by a prey (*e.g.* bacteria, algae) which, in turn, is consumed by a predator (*e.g.* ciliates, daphnia) population. The dynamic behaviour of this ecosystem is described by a set of ordinary differential equations (ODE)s: one for the nutrient and one for each population. The system is stressed by a toxicant dissolved in the in-flowing water. The transport of the toxicant is modeled using a mass balance formulation leading to an ODE. Bioaccumulation in the prey and predator populations is via uptake from the water, in case of the predator also via consumption of contaminated prey. Mathematically, this process is described by a one-compartment model for the kinetics of the toxicant: uptake (from water and food) and elimination. The toxicant affects individuals which make up populations. In the model the physiological parameters depend on the internal concentration of the toxicant in individuals. Examples of physiological parameters are cost for growth and maintenance, and assimilation efficiency. In this paper, we use bifurcation theory to analyse the long-term dynamics of the models. In this way, the parameter space is divided into regions with qualitatively different asymptotic dynamic behaviour of the system. As logical choice for bifurcation parameters are the input rate of the nutrient and toxicant. The dynamic behaviour of the stressed ecosystem can be much more complicated than that of the unstressed system. For instance, the nutrient–prey–toxicant system can show bi-stability and oscillatory dynamics. Due to the toxic effects a total collapse (both prey and predator population go extinct) of the nutrient–prey–predator–toxicant system can occur after invasion of a predator.

1. Introduction

With the theoretical assessment of consequences of toxicants on the functioning of aquatic ecosystems five steps can be distinguished, see (Calow et al., 1997):

(1) *Ecological theory*: modelling of the biological functioning of the system. For an ecosystem, we need
- a model for an individual life-cycle
- a model of each population using a model of individual behaviour
- a model for the ecosystem using models of populations including their mutual interactions and interaction with the physical environment such as transport of nutrients.

(2) *Environmental chemical theory*: environmental chemistry and geochemistry related models to describe fate of toxicant: transport, distribution and exposure of toxicants.

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(3) *Toxicological theory*: the relationship between exposure to a toxicant, toxicokinetics and behaviour of an individual.
(4) *Ecotoxicological theory*: effect of toxic stress on the individual, population and ecosystem level via bioconcentration (exclusively from water), biomagnification (exclusively from food) and bioaccumulation (from water and food).
(5) *Risk assessment*: using the ecosystem, exposure and effect models to assess community/ecosystem consequences (*e.g.* extinction of one or more populations).

In this paper, we focus our attention on (4) where we use existing models for a simple ecosystem models (1), for the fate of the toxicant (2), and an exposure model (3). How the obtained results can be used for risk assessment (5) is discussed briefly.

The dynamical behaviour of small-scale microbial food chains or aquatic ecosystems, such as a system of nutrient, detritus, phytoplankton, zooplankton and fish, have been studied intensively in the literature (see for instance (DeAngelis, 1992)). Generally each population is modelled by one or a few ODEs. Two ingredients of these systems are *state variables*, such as nutrient, detritus, biomass or energy content, and *parameters*, such as maximum ingestion rate, assimilation efficiency, immigration or emigration rates, reproduction rate, searching rate for food, handling time of prey, maintenance rate and mortality rate. For the long-term dynamics important features of the ecosystem are persistence: the structural composition of the ecosystem, and dynamical behaviour of the ecosystem: the occurrence of a steady state, oscillations or chaos.

In an elementary ecological setting these parameters are species-specific constants or, in the case of diurnal or seasonal forcing, they also depend explicitly on time, for instance light intensity. With stressed system these parameters may depend on external parameters, such as pH, temperature, rainfall. The subject of this paper are the consequences of toxic stress. Then the population parameters depend on the concentration of the toxicant in the water which is a state variable.

Toxicants are emitted and distributed into the ambient water. The transport of the toxicant is modelled by mass-balance equations (ODEs). Exposure of the organisms is by absorption from the water or via consumption of contaminated food. The kinetics of the toxicant in the organism is modelled with a first order one-compartment model where two processes are involved, namely uptake (from water and/or food) and elimination. The rate of these processes depend on the internal and water concentration of the toxicant or contaminated food availability (Kooijman and Bedaux, 1996). For each species–toxicant combination a concentration–effect relationship describes how the toxicant changes population parameters that determine the rate of physiological processes. These parameter changes affect in turn the functioning of the ecosystem (extinction of a population or system destabilising).

In this paper, we analyse the lowest levels of an aquatic ecosystem. The model for the populations (*e.g.* bacteria or algae consumed by ciliates) that compose the ecosystem is a simplified version of the DEB model (Kooijman, 2000). The toxic effects on the population level are described by the DEBtox approach for uni-cellular organisms with a simple life-history namely propagation by binary fission (Kooijman and Bedaux, 1996). The effect module is not based on parameters estimated from descriptive models but on process-based models where physiological parameters depend on the internal toxicant concentration. The possibly affected physiological processes are assimilation, maintenance, growth and mortality. These are the possible modes of action of the toxicant, that is these processes are the targets of the toxic effects. Here, we describe the consequences on the ecosystem behaviour where nutrients and toxicant are supplied and removed at a constant rate in a spatially homogeneous chemostat (Smith and Waltman, 1994).

In Thomann and Connolly (1984); Thomann and Mueller (1987); Thomann (1989); Clark et al. (1990); Gobas (1992); Campfens and MacKay (1997); Traas et al. (2004a, b) bioaccumulation in food webs is also studied. In these papers the transfer of the toxicant through the ecosystem is decoupled from dynamics of the ecosystem by assuming the ecosystem to be in an equilibrium. For the populations, the internal concentration is assumed to be in equilibrium with the ambient concentration, that is the concentration ratio is constant. For the prey where uptake is only from the ambient water this ratio is called the bioconcentration factor (BCF), for the predator where intake of toxicant is also via contaminated prey biomagnification factor (BMF) and combined the bioaccumulation factor (BAF) (Thomann, 1989). Hence, for each trophic level there is an expression that links the two concentrations algebraically and no extra ODE for the internal toxicant concentration is needed while the two exchange rates are replaced by a single BCF parameter. This simplifies the analysis considerably. Besides the BCF, BMF and BAF values, additional information needed is on the dietary preference matrix that determines the feeding relationships between the prey–predator populations in the ecosystem. In this way, ecosystem dynamics and the fate of the toxicant are modelled separately.

This paper is organized as follows. In Section 2, the modelling and analysis approaches are introduced. Here, we use an approach where ecological processes and the fate of the toxicant as well as their interactions are modelled integrated (Koelmans et al., 2001). The model for the nutrient–prey system in the chemostat is formulated in Section 3. The model for the unstressed system predicts simple dynamic behaviour: a stable equilibrium when sufficient nutrient is supplied. In Section 4, we show that under toxic stress, the model predicts bi-stability under suitable environmental conditions. In Section 5, the model for the nutrient–prey–predator system is formulated. Expressions for BCF, BMF and BAF are derived. Two situations are analysed in Section 6. In the first case, both prey (via water) and predator (via water and food) population are affected by the toxicant, for instance a pesticide. In the second case, the toxicant is a bactericide (antibiotics) or algicide (herbicide), where only the prey population is effected. In the latter case, the model predicts that after inoculation of the predator into the nutrient–prey system a collapse of the system is possible whereby both prey and predator population go extinct. In Section 7, we conclude that due to the dynamics of the toxicant there is an extra removal mechanisms from the reactor. Presence of the toxicant influences the growth of the populations which in turn changes the uptake rate of the toxicant by these populations. This feedback mechanism appears