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***The use of models for ecological risk assessment in coastal ecosystems:
Thresholds point of view***

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Executive Summary

The main objective of this work is to assess the different types of models, e.g. population models, ecosystem models, chemical fate and transport models, bioaccumulation models and food web exposure models, from the thresholds perspective; and to study how an integrated fate and effect model should be developed to consider the occurrence of points at which there is an abrupt change in an ecosystem quality produced by a small change in an environmental driver. The main objective is the increased concern about preventing dramatic state changes in ecosystems and our modelling approach should help in determining critical pollutant loads. The focus of this study is on coastal ecosystems which is the main focus of the IP Thresholds of environmental sustainability. The results of this analysis will then be incorporated in Stream 4 (S4: Thresholds of contaminants) where specific studies are being conducted and models developed. Two case examples have been studied: a stage based population model and a continuous food-web model that simulates a mesocosm experiment with the fast addition of a contaminant.

1. Introduction

Ecological Risk Assessment (ERA) can be defined as “the process for systematically evaluating how likely it is that adverse ecological effects may occur as a result of exposure to one or more stressors” (U.S. EPA, 1998).

The idea of adopting ERA as a fundamental component in the management of ecosystems has gained an increased recognition due to the fact that it is impossible to eliminate all environmental effects of human activities and that decisions must be made on the basis of incomplete data and incomplete scientific knowledge (Suter, 1993). Therefore, it is necessary to reach a compromise between acceptable risks levels and the costs of reducing these risks. This becomes even more relevant when different stressors, i.e. physical, chemical and biological, are involved which is typically the case of aquatic ecosystems.

The existence of several spatio-temporal scales when assessing the risks in ecosystems and several levels of ecological organization from organism, population to food web, etc. provokes the existence of a gap between what is feasible to measure and what is interesting to assess. Being organisms the easiest unit to measure and ecosystem the relevant level for protection. It is for this reason that models are frequently used to fill this gap and provide meaningful extrapolation across time, space and biological organization scales (Suter, 1993).

Concerning chemical stressors to aquatic ecosystems, eutrophication and contamination with pollutants have been subject to intensive modelling research during the past decades (Cloern, 2001). Unfortunately, they have been mainly treated separately under the assumption that changes in trophic state cause negligible feedback on the fate of pollutants and their effects, and that toxicity of chemical produced negligible feedback on the physicochemical processes that determined the fate of pollutants (Koelmans *et al.*, 2001). Of course, these assumptions are not always true and it has been seen that heavy metals (Sanders and Cibik, 1988, Kuwabara *et al.*, 1989), chlorinated hydrocarbons as DDT and PCBs (Mosser *et al.*, 1972) and herbicides such as atrazine and diuron inhibit selectively some species of algae promoting population growth of the less-sensitive taxa. Furthermore, nutrient enrichment leads sometimes to enhanced accumulation of contaminants (Gunnarsson and Sköld, 1999) or may cause dilution of contaminants, e.g. nutrient enhanced algal production may cause change in the overall toxicity by increased transformation of contaminants through algal metabolism (Breitburg *et al.*, 1999).

The main objective of this work is to assess the different types of models, e.g. population models, ecosystem models, with and without spatial components, chemical fate and transport models,

bioaccumulation models and food web exposure models, from the thresholds perspective; and to assess how an integrated fate and effect model should be developed to consider the occurrence of points at which there is an abrupt change in an ecosystem quality produced by a small change in an environmental driver. The main objective is the increased concern about preventing dramatic state changes in ecosystems and our modelling approach should help in determining critical pollutant loads. The focus of this study will be on coastal ecosystems which is the main focus of the IP Thresholds of environmental sustainability. The results of this analysis will then be incorporated in Stream 4 (S4: Thresholds of contaminants) where specific studies are being conducted and models developed.

2. Ecological Risk Assessment

In this Section we are going to provide a short overview of Ecological Risk Assessment (ERA). We have followed closely the U.S. EPA (1998) document on “Guidelines for Ecological Risk Assessment”.

Ecological risk assessment has three main phases that are followed by risk management (see fig. 1):

- Problem formulation: In this phase it is necessary to select the assessment endpoints (defined as a formal expression of the environmental value to be protected, Suter, 1989), develop the conceptual model and prepare an analysis plan.
- Analysis: In this phase two principal activities should be carried out which are exposure and ecological effects characterization. The first activity describes the sources of stressors, their environmental distribution and contact with ecological receptors, whereas ecological effects characterization deals with the evaluation of stressor response relationships, e.g. dose response curves for a contaminant.
- Risk characterization: During this phase the ecological risks as well as the confidence degree are evaluated. However, risk assessment results have to be combined with socio-economic and legal aspects before decisions be taken.

Ecological risk assessments can be used to predict future effects or to evaluate effects caused by a past exposure or in a combined way. Afterwards, the next step is risk management. In case of adverse effects measures to mitigate the risk are necessary; this should be carried out in parallel with a monitoring plan to follow ecosystem recovery or to assess if the ecological risk is being reduced.

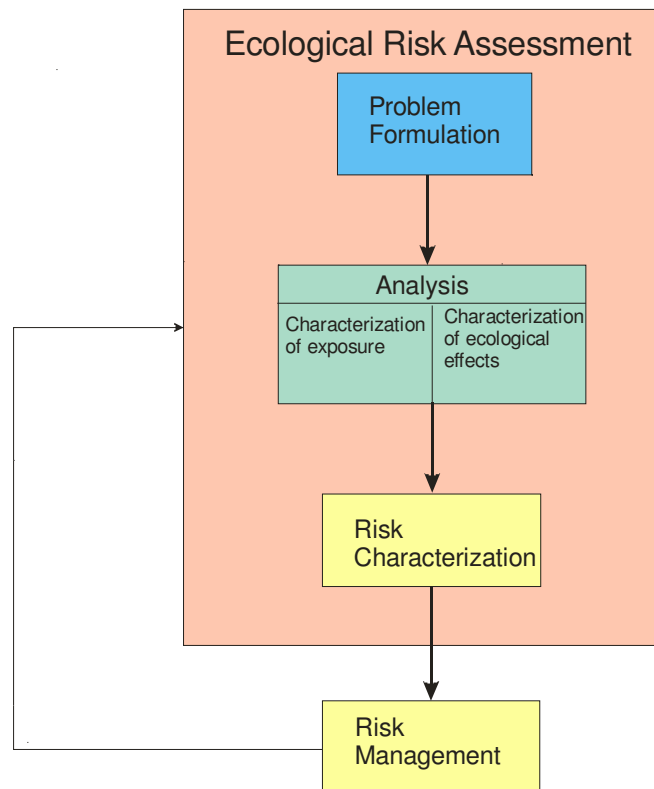


Figure 1. The phases in Ecological Risk Assessment (modified from US EPA 1998).

2.1. Problem formulation

This initial phase provides the foundation for the entire process. During this phase three essential steps are required: Endpoints assessment, conceptual model and analysis plan (U.S. EPA, 1998).

- Endpoints assessment:

An assessment endpoint is a formal expression of the environmental values to be protected (Suter, 1993). In order to define them it is necessary first to identify valued attributes of the environment at risk and to define these attributes in operational terms. Generally speaking, endpoints should have biological as well as social relevance, a clear operational definition and proven susceptibility to the contaminant as well as should be easily measurable.

However, sometimes assessment endpoints and measured endpoints are not the same. Measurement endpoints are generally values from toxicology, e.g. 96-h LC₅₀, or functions as dose-response, whereas assessment endpoints generally refer to characteristics of ecosystems defined over long temporal scales, e.g. fish population in a lake. Therefore, it is necessary to define in any ERA, if assessment and measured endpoints are different, a method to extrapolate from one to the other.

Well selected assessment endpoints may be sensitive to several identified stressors and therefore, provide an opportunity to consider combined effects. In this case it is important that all the effects can be expressed in the same units.

There are two statistical types of measurement endpoints (Suter, 1993): a/ those that prescribe a level of effect by fitting a function relating the measured effects to measurement of exposure (response-dose). Normally only a particular level is used, e.g. LC₅₀, LD₅₀ (median lethal dose), EC₅₀ (median effective concentration), and LC₀₁ (lethal threshold concentration); b/ those that are based on hypothesis testing. In this case, responses at the exposure concentrations are compared with a control (unexposed) to test the null hypothesis that they are the same as the control responses, examples include: NOEC (no observed effect concentration) and LOEC (lowest observed effect concentration).

- Conceptual model:

A conceptual model is a written and/or visual representation of predicted relationships between ecological entities and the stressors to which they may be exposed (U.S. EPA, 1998). In order to develop a conceptual model it is necessary to have information on existing stressors, potential exposure and predicted effects on the assessment endpoints.

Conceptual models have two main components: Risk hypothesis and diagrams (U.S. EPA, 1998). Risk hypothesis are assumptions about potential risk to assessment endpoints, whereas conceptual model diagrams represent visually the risk hypotheses. Conceptual models may miss sometimes important relationships and therefore may misrepresent risks. For this reason ERA processes is normally an iterative process in which the different steps are improved as the information increases.

- Analysis plan:

This stage should identify the problem, establish study boundaries, and determine necessary data quality, quantity and applicability to the problem being evaluated. Is in this part of the process that, risk hypothesis has to be evaluated to determine how they are going to be assessed. The analysis plan should include needed data and gaps with recommendations for new data collection (if needed), hypothesis prioritisation as a function of the level of risk and confidence level expected from management point of view.

This first phase is essential if we want to avoid typical problems in ecological risk assessment such as the absence of clearly defined objectives, the use of endpoints that are difficult to measure and assess, the failure to identify important risks or conclusion of no risk because the endpoint selected was not susceptible to the stressor.

2.2. Analysis phase

The objective of this phase is to determine or be able to predict the ecological responses of the endpoint organisms as a function of the exposure conditions. This is not an easy task since in most of the cases, the source term (rate and the spatial and temporal patterns of release of a chemical) are the major source of uncertainty in ERA.

- Characterization of exposure:

The process of converting a source term into estimates of contact with or doses to the selected endpoints is called exposure assessment (Suter, 1993). It requires that a model of the exposed environment is developed that includes all relevant media that are going to be in contact with the endpoints.

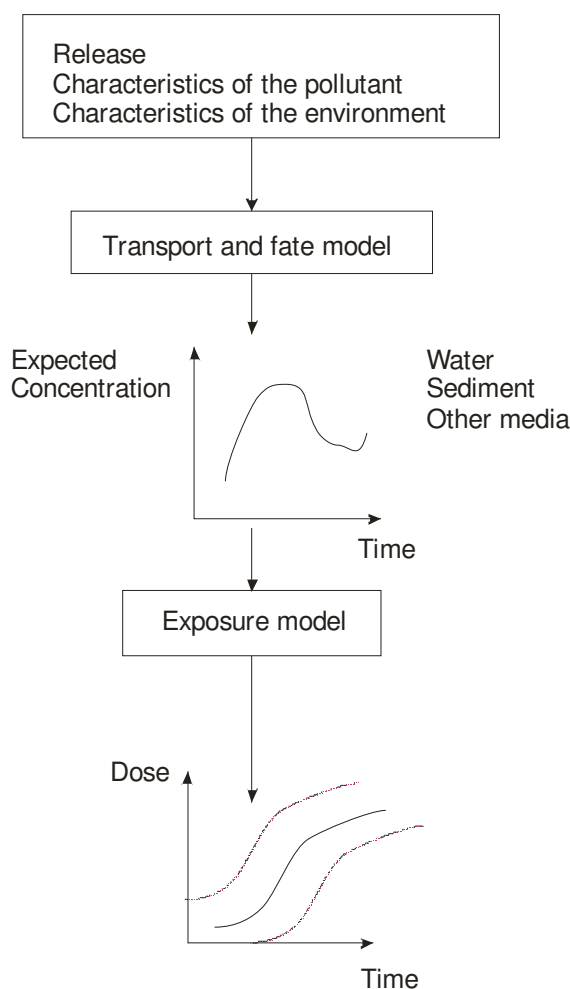


Figure 2. Representation of the process of exposure assessment (modified from Suter, 1993).

The objective on this phase is to move from data on chemical release, taking into account the characteristics of the chemical (stressor) as well as the environment, to the concentration level at which

the selected endpoint(s) are exposed. This is normally called the exposure pathway. An important consideration for chemicals is the assessment of their partitioning in several media which depends mainly on their physico-chemical properties as well as their bioavailability which is a measure of the physicochemical access that a toxicant has to the biological processes of an organism. Furthermore, certain chemicals may be able to bioaccumulate or biomagnificate through the food chain.

For some chemicals, secondary stressors, i.e. metabolites, biodegradation products or decomposition products can alter the conclusions about risk and they may become of greater concern than the primary stressor.

- *Characterization of ecological effects:*

Effects assessment is the process of determining the relationship between exposure to the pollutant and its effects to the assessment endpoint(s) and how they change with varying pollutant levels.

The most simple and used approach, i.e. Dose-Response analysis, consist on evaluating how the response, e.g. mortality, growth, etc., evolves as a function of the dose.

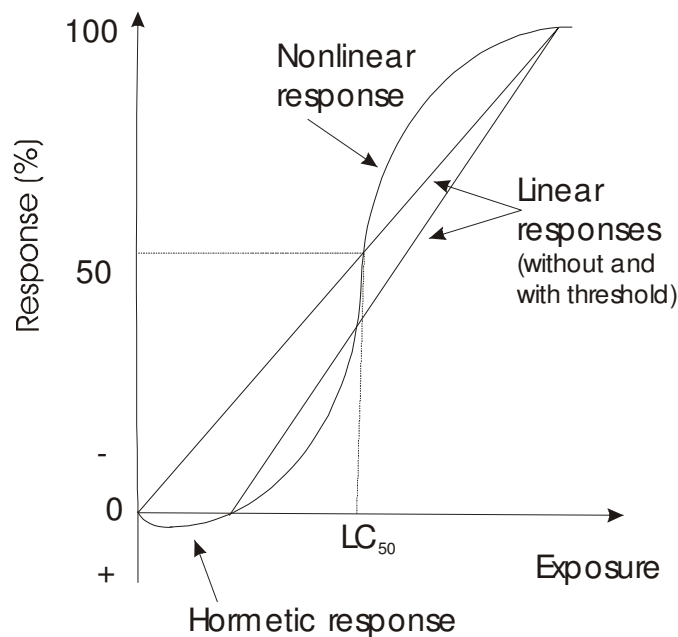


Figure 3. General dose-response functions: a/ linear with and without thresholds and nonlinear with hormesis.

Normally, these effects are represented using the median effects levels which are defined as those observed in 50% of test organism exposed to a pollutant. When these effects are expressed in terms of lethality or mortality, they are known as LC_{50} , if concentrations in diet or water are used, or LD_{50} , if doses (mg/Kg) are used. Other typical median effects are on growth, EC_{50} or ED_{50} . Median effects are associated with a time which is normally 24, 48, 72 or 96 hours.

More complex relationships may be considered. For example, if we are interested at population level rather than using single individuals, it is possible to develop response relationships at this level. For example, Lopes *et al.* (2005) had studied the effects of a pesticide (methiocarb) on the population growth of *Chironomus riparius* by adjusting a discrete population model (Leslie matrix, see Ch. 4) based on the pesticide effects to each stage (eggs, larva, pupa and adults). With this approach they were able to obtain the response of the entire population as a function of the pesticide concentration. Figure 4 show these results. At λ (population growth rate, eigenvalue of the Leslie matrix) values lower than 1, the population will become extinct (see also Ch. 4).

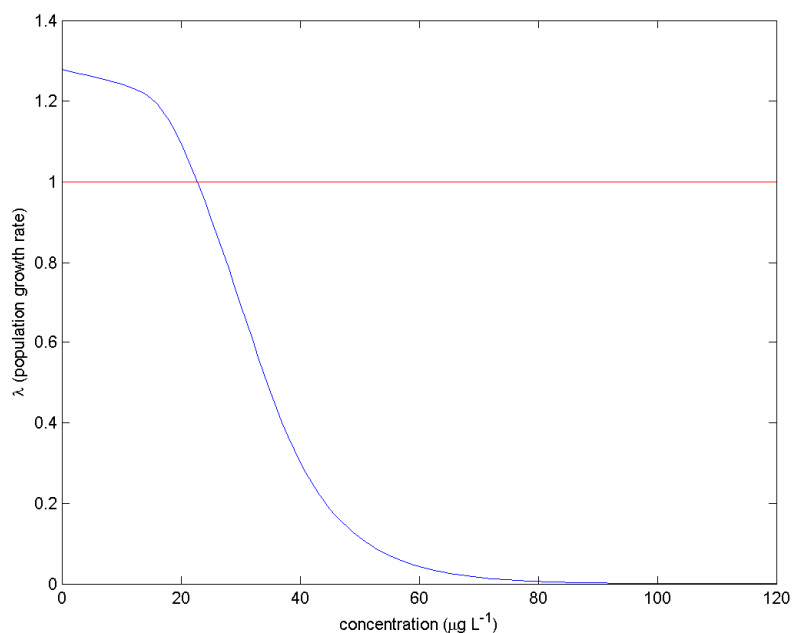


Figure 4. Effects of methiocarb concentrations on population growth rate of *Chironomus riparius* (Lopes *et al.*, 2006).

2.3. Risk characterization

The integration of information concerning sources, exposure and effects is the last step in ERA. This integration should allow to obtain an estimate of the level of effects that will result from the exposure. According to U.S. EPA (1998), it is composed of two steps: Risk estimation and risk description.

- Risk estimation:

When integrating exposure and effects assessment, it is necessary to express the effects using a fourth-dimensional state space (Suter, 1993), whose dimensions (two for exposure and two for effects) are:

- 1/ the concentration of the substance to which organisms are exposed;
- 2/ the duration of the exposure;

3/ the proportion of the community responding;

4/ the severity of the effect.

Whereas in traditional human health risk assessment the important parameter is the duration of the exposure (dose rate x time), normally, in ERA it is more important the duration of the effects to assess how long an ecosystem is degraded. In addition, ecosystem exposed for long periods may adapt to a certain pollutant and biological effects may end before exposure disappear or may continue to occur after the exposure ceases, because there are time lags or other physical processes –e.g. resuspension by storms -in the induction of the effects. Unfortunately, there is no general theory of ecosystem response and recovery that may be used for evaluate such effects.

Additional difficulties arise when answering the severity of the effects. In this case, there are several graded scales such as (Dourson, 1986): no observed effect (NOE), no observed adverse effect, adverse effects (e.g. metabolic and growth), death.

- *Risk description:*

The risk description must consider an evaluation of the lines of evidence for risk as well as their evaluation and the assessment of the significance of the adverse effects on the selected endpoints. This evaluation should be easy to understand for the managers that should take the final decisions.

In this case also an overall degree of confidence in risk estimates should be included in the risk assessment report.

2.4. Risk Management

Even though risk management can be carried out without risk analysis, the effectiveness of it depends necessarily on the successful development of the ERA process. The ideal input from an ERA process to risk managers would be setting criteria for the allowable concentrations of a chemical in air, water, etc. and providing probability functions for unacceptable effects on the endpoint. This allows developing a set of preventive and protective measures that are not over- or under-protective.

However, in addition to the ERA and human health effects risk management should also consider other aspects based on social, political, legal and economic considerations that are not provided by the ERA process as well as other techniques not considered as cost-benefit or risk-benefit analysis.

3. Ecological models and ecologic risk assessment

In a recent survey carried out by Fleeger *et al.* (2003) the effects of contaminants in aquatic ecosystems were divided in direct and indirect effects. Direct effects from the release of a pollutant into aquatic

habitats vary with intensity and duration of exposure and are frequently studied as a part of the estimation of risk of a certain chemical and the establishment of a threshold value for its permissible concentrations. These studies are normally based on laboratory toxicity tests using model species responses to a single contaminant exposure. Direct effects typically reduce organisms' abundance, e.g. increase of mortality, reduction of growth rate or fecundity, etc. However, pollutants may have other effects even on tolerant species by other ecological mechanisms, e.g. direct influences of contaminants on predators can lead to cascading indirect effects on resistant species in other trophic levels by altering competitive interactions and therefore modifying substantially its abundance and dynamical behaviour. Such effects are called indirect (or secondary) contaminant effects (Flegger *et al.*, 2003) and sometimes can be as or more significant than the direct (toxic) effects of a contaminant.

Ecological models have become effective tools in evaluating direct and indirect effects, estimating and to managing ecological risks (Bartell, 1996; Pastorok *et al.*, 2003). In addition, ecological models may be applied to forecast future potential risks or to estimate risks when field experiments cannot be performed, i.e. the release of a new chemical into the environment. They are useful tools for testing alternative hypothesis or to reconstruct past situations where evidence of toxic exposure cannot be demonstrated.

It is evident that there is no general methodology for quantifying ecological risk, since there will be always limitations to the amount of data we can obtain in terms of time, resources, and/or knowledge (Pastorok *et al.*, 2003). For these reasons, mathematical models have often been used in ERA. These models may be divided in two general categories: statistical and mechanistic models (Suter, 1993). Statistical models attempt to extract generalizations from data using statistical techniques. A statistical model has no underlying assumptions to explain the observations in terms of cause-effect relationships, and therefore their use for extrapolation is always questionable. On the contrary, mechanistic models have as its objective to describe quantitatively the relationship between a phenomenon and its causes. In this case parameters in a mechanistic model have a clear definition and could be, in principle, obtained from experiments.

Mathematical models, once properly validated, offer the possibilities to simulate process that would not be possible to measure in reality; to forecast the results of different plausible scenarios; and to extract information about the global dynamic behaviour of the studied system which would be impossible from any field experiment. These are the reasons for which they are used in ERA studies.

Concerning coastal (aquatic ecosystems), the mechanistic models of interest for risk assessment can be divided into several classes (Koelmans *et al.*, 2001):

- Eutrophication model (Cycling of nutrients and the growth of algae)
- Contaminant fate model (describing the fate and distribution of contaminants in the aquatic system)
- Ecosystem model (feeding relationship, prey-predators among the species in the aquatic ecosystem)
- Food chain bioaccumulation model (processes of contaminant uptake, depuration and transformation in aquatic organisms and pollutants transfer through the food web)
- Effect and impact model(s) that link bioaccumulation and/or toxic effects and ecosystem distribution at the level of ecosystem components. This information is necessary to allow risk assessment studies to be undertaken.

These modules should be linked through the following processes:

- Cycling of organic carbon in the water column and the sediment and the association of organic contaminants with organic matter (Eutrophication-Contaminant fate)
- Acute and chronic toxicity of contaminant, impacts upon the functioning of the food chain components and transport and accumulation in the food chain (Contaminant fate-bioaccumulation/effects and impacts)
- Bottom-up versus top-down control of food chain structure (Eutrophication-Food Web)

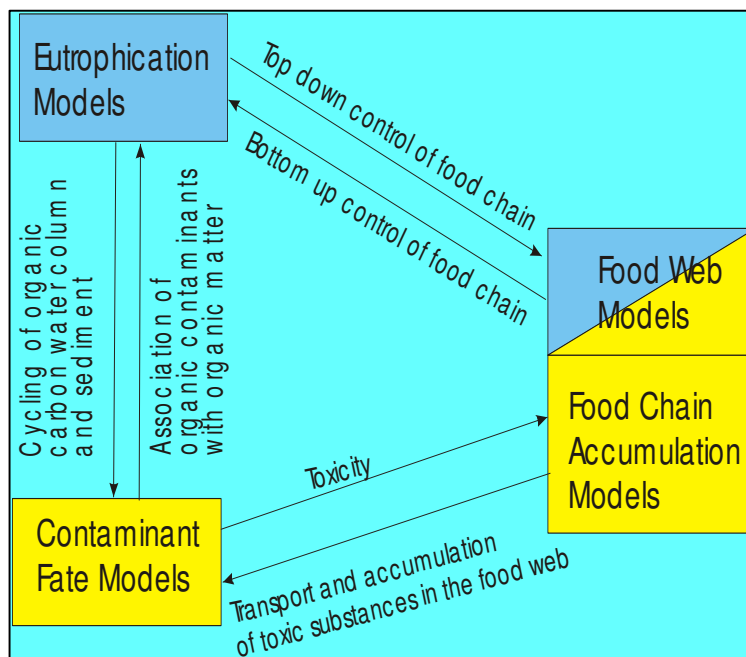


Figure 5. Models for ERA (Koelmans *et al.*, 2001).

3.1. Effect-load-sensitivity analyses – basic concepts: the case of nutrients

Richard Vollenweider presented the first load models for phosphorus for lakes in the late 1960s (Vollenweider, 1968). By means of mass-balance calculations and statistical regressions, he could demonstrate that in many lakes, eutrophication could be reversed by reducing the input of total phosphorus (TP) to the lakes so that the mean lake annual concentrations of TP could be lowered. Since then, many studies have demonstrated where the Vollenweider approach can and cannot be used (Schindler, 1977, 1978; Bierman, 1980; Chapra, 1980). The Vollenweider model (and later versions, such as OECD, 1982), and the analysis behind this load model, constitutes a fundamental base for practically all environmental assessments of eutrophication for lakes. The interesting part, however, is not to predict a concentration of a chemical element like a nutrient, but to predict ecological effects related to nutrients (see fig. 6). It is evident that the concentration of the nutrient can be influenced by emissions from many types of sources, like point sources (domestic sewage, industries and fish farms), atmospheric deposition (to the water surface and the catchment area), internal loading (linked to resuspension, diffusion, etc.) and, in estuaries, inflow from the sea and tributary input, where the characteristics of the catchment, like bedrocks, soils, land use, etc., influence the nutrient concentration in the coastal area.

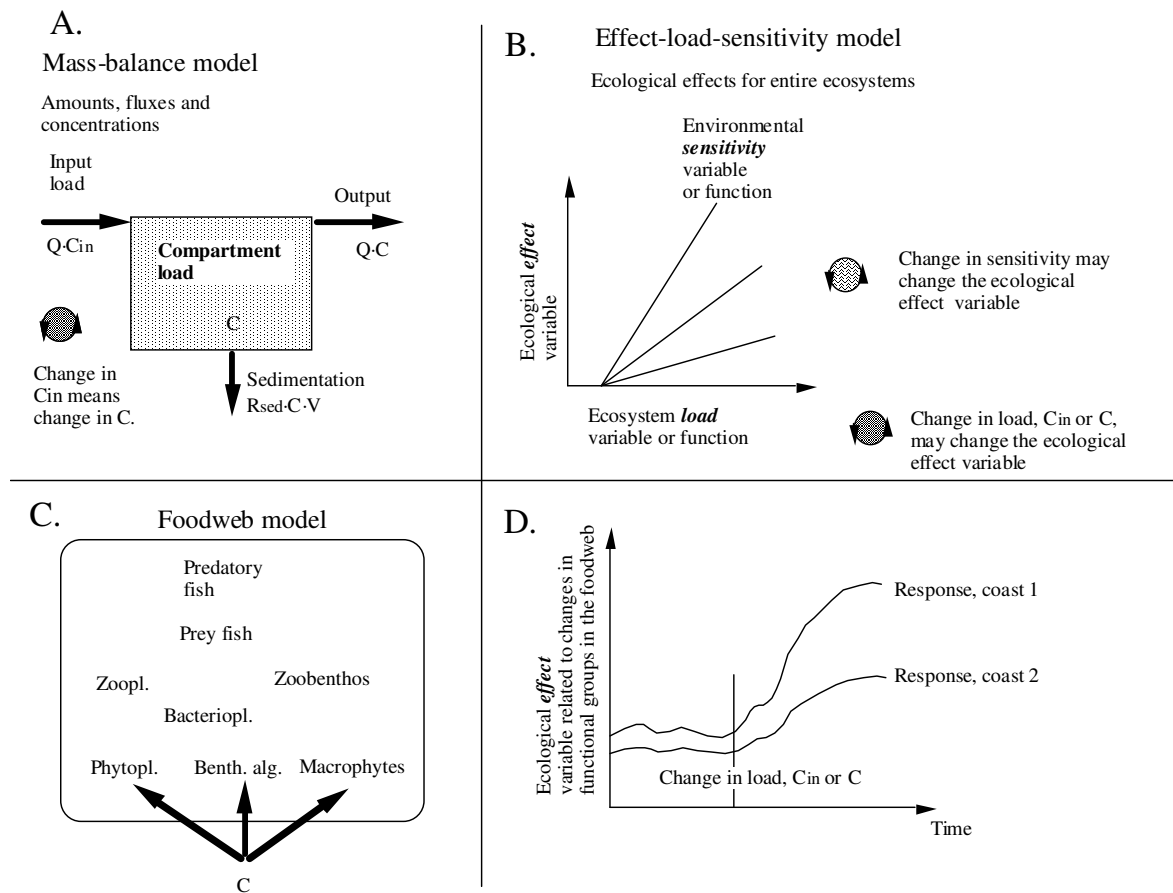


Figure 6. Illustration of the fundamental difference between dynamic, mass-balance models (fig. a) and effect-load-sensitivity models (ELS) based on regressions (b) and ELS-models related to dynamic foodweb models (c) and (d) how changes in the load at a given time may cause different responses in the aquatic foodweb in coastal systems of different size and form (coast 1 compared to coast 2). The wheels indicate that by means of remedial measures one may reduce the load variable in dynamic models and the load and the sensitivity variables in ELS-models. Q = Water discharge ($m^3/time$); C_{in} = concentration of substance in inflow (g/m^3); C = concentration of substance in the system (g/m^3); R_{sed} = sedimentation rate ($1/time$); V = volume (m^3).

Differential equations are often used to quantify fluxes ($g X /yr$), amounts ($g X$) and concentrations ($g X/m^3$) of all types of materials (such as toxins and nutrients), but not generally bioindicators such as the Secchi depth, chlorophyll-a concentrations and the oxygen saturation in the deep-water zone (O_2Sat) or other types of ecosystem effect variables (fig. 7). Regressions based on empirical data are often necessary to relate concentrations of chemicals in water or sediments to OEVs. In theory, both model approaches (see fig. 6a and b) may be used for the effect-load-sensitivity analyses (ELS; see Håkanson, 1999) provided that at least one operationally defined ecological effect variable relevant for the load variable(s) in question is included in the model. Step b in fig. 6 illustrates a regression. Ideally, the effect variable should express the production or biomass of defined functional organisms

(preferably fish at the top trophic level, see fig. 6c), which characterize a given coastal system. Fig. 6d illustrates schematically that two coastal areas are likely to react differently to a change in the load of nutrients to the system. The classical approach (from Vollenweider, 1968) to carry out ELS-analysis is to use dynamic mass-balance models to predict concentrations of nutrients and empirical models (like regressions) to link these concentrations to measured data on the effect variables (see fig. 7). In contexts of coastal management, one must generally for practical and economical reasons seek simpler but relevant operational effect variables than the ideal ones related to production or biomasses of functional groups or species. The mean concentration of a given toxic substance (or substances) in predatory fish, the Secchi depth, the oxygen saturation/concentration and chlorophyll-a concentrations are examples of simple, operational effect variables.

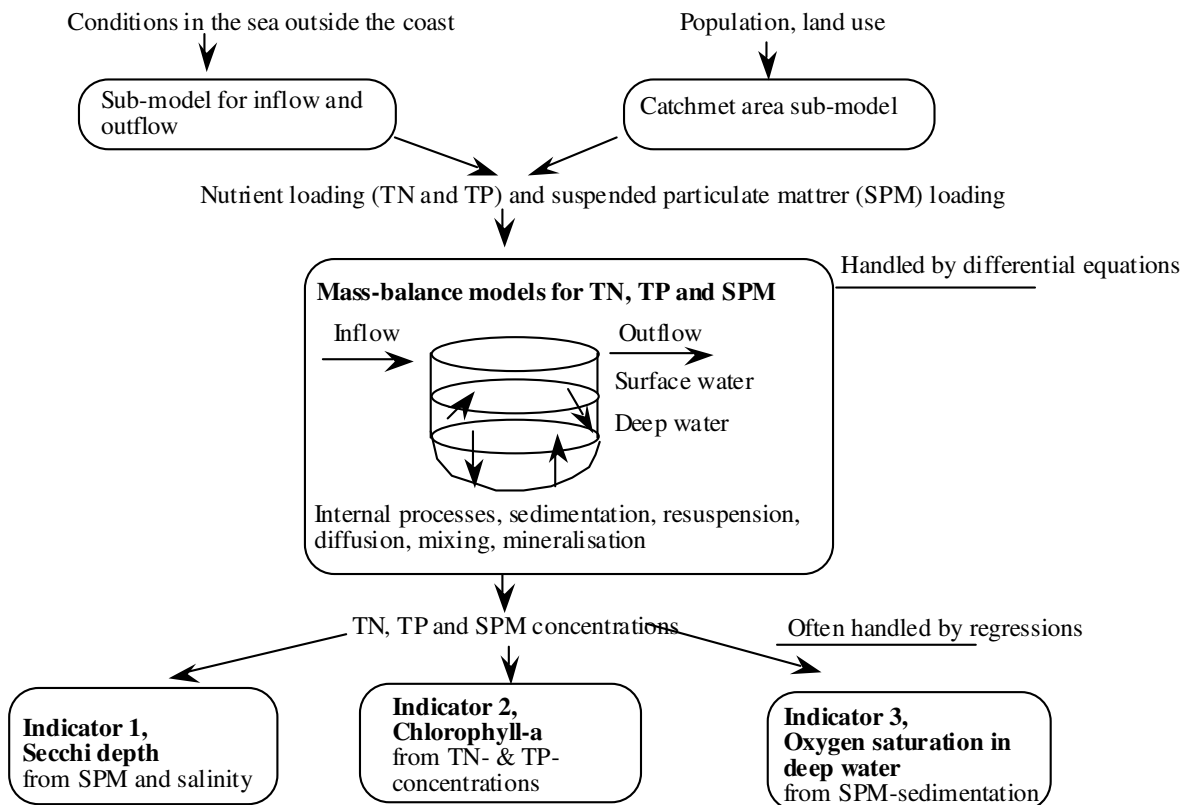


Figure 7. Basic elements in Effect-Load-Sensitivity (ELS) modelling for coastal water eutrophication utilizing mass-balance modelling and regression analyses relating nutrient concentrations to operational effect variables (Secchi depth, chlorophyll-a concentrations and oxygen saturation in the deep-water zone).

Fig. 6b gives the principles of an ELS-model illustrated as a ELS-diagram. Environmental goals (generally set by National Environmental Protection Agencies) should concern the ecological effect

variables and not the load variables, since one and the same load may cause different effects in ecosystems of different sensitivities. From this diagram, important concepts like natural background concentration and critical load can be scientifically defined (see Håkanson, 1999). When no practically useful validated ELS-models are available, there exists ample room for speculations about cause and effect, and about the best strategies to remediate aquatic systems. In contexts of ELS-analyses, the primary interest is not on site-specific conditions ("the sampling bottle"), on the individual, organ or cell level, but at the ecosystem level. That perspective should be of main interest from a management point of view where questions are posed concerning the status of larger water bodies (ecosystems), and the remedial actions that could be used in practice to improve the conditions in such systems.

It should be stressed that ELS-analyses are of fundamental importance in water management and that ELS-models are essential tools to examine consequences of remedial measures that may influence a target effect variable. One can reduce negative ecosystem effects of contaminants by reducing the load to the system or by changing the sensitivity (for example, by changing loading of nutrients to the given coastal area). One cannot generally change the morphometry of the coastal area, but coasts of different size and form will react differently to remedial measures and it is essential to know this so that one can have realistic expectations of the remedial measures for a given coast.

It is generally not possible to derive ELS-models which apply with equal success to all types of ecosystems. Therefore, the operational range, the domain, of the model must be explicitly given to avoid abuse of the model for ecosystems for which it was never intended to be used. If dynamic (time-dependent) ELS-models can meet these requirements, they would generally be preferable to statistical/empirical models because they can provide better understanding of mechanisms and processes.

3.2. Ecological models

Normally an eutrophication model plus a food web model is considered a complete ecological model. Models of aquatic ecosystems can be characterised according to the types of processes they describe, the level of disaggregation and the degree of biological detail. Furthermore from the point of view of the type of equations used it is possible to distinguish between:

A/ Continuous modelling approach: Which is mainly based on Lotka-Volterra type ordinary differential equations, Takeuchi (1996). The Lotka-Volterra equations with diffusion can be described by:

$$\begin{aligned}\frac{dx_i}{dt} &= x_i(r_i + \sum_{j=1}^n a_{ij} \cdot x_j) + D_i(y_i - x_i) \\ \frac{dy_i}{dt} &= y_i(\bar{r}_i + \sum_{j=1}^n \bar{a}_{ij} \cdot y_j) + \bar{D}_i(x_i - y_i)\end{aligned}\tag{1}$$

where r_i is the intrinsic growth rate for i-th species and the $\mathbf{A}=\{a_{ij}\}$ is the interaction matrix in patch X. The D_i are non-negative diffusion constants between the patches for i-th species. The first equation describes growth of i-th species in Patch X and the second in Patch Y. The first term in the right-hand sides in the two equations represent dynamics within patches X and Y respectively, and the remaining term express species dispersal between patches. In this type of approach the system is composed of several subsystems (called patches) described by Lotka-Volterra equations and the species are allowed to disperse among the patches. Except some examples known as diffusive instability or diffusion-induced instability, a diffusion process generally tends to promote stability in ecological systems, Takeuchi (1996). Obviously, this modelling approach is a crude simplification of reality and many attempts have been carried out to improve the Lotka-Volterra equations by introducing different kinds of saturation functions, reproduction, mortality, etc. (Bazykin, 1998).

B/ Discrete stage-based modelling approach: The use of continuous ordinary differential equations ignore population structure by treating all individuals as identical. The existence of demographically important differences among individuals is obvious. Matrix population models (Caswell, 1989) integrate population dynamics and population structure particularly clearly and they are very useful when the life cycle is described in terms of size classes or development stages, rather than age classes. There are fundamentally two types of approaches, the age classified model and the stage classified model. The first one assumes age-specific survival and fertility are sufficient to determine population dynamics. On the other hand, if the vital rates depend on body size, and growth is sufficiently plastic that individuals of the same age may differ appreciably in size, then age will provide little information about the fate of an individual. In the stage-based type of modelling the matrix \mathbf{A} , called Leslie matrix, which describes the transformation of a population from time t to time $t+1$,

$$\mathbf{n}_{t+1} = \mathbf{A} \mathbf{n}_t\tag{2}$$

has the following structure:

$$\mathbf{A} = \begin{bmatrix} P_1 & m_2 & \dots & \dots & m_q \\ G_1 & P_2 & 0 & \dots & 0 \\ 0 & G_2 & P_3 & 0\dots & 0 \\ \dots & \dots & \dots & \dots & \dots \\ 0 & 0 & 0\dots & G_{q-1} & P_q \end{bmatrix} \quad (3)$$

where \mathbf{n}_t is a vector describing the population at each stage at time t , P_i is the probability of surviving and staying in stage i , G_i is the probability of surviving and growing into the next stage, and m_i is the maternity per fish per unit time (d), $i = 1, 2, \dots, q$. Both P_i and G_i are functions of the survival probability, p_i , and the growth probability, γ_i (Caswell, 1989):

$$P_i = p_i(1 - \gamma_i) \quad (4)$$

$$G_i = p_i \cdot \gamma_i \quad (5)$$

where

$$p_i = e^{-z_i} \quad (6)$$

and

$$\gamma_i = \frac{(1 - p_i)p_i^{d_i-1}}{1 - p_i^{d_i}} \quad (7)$$

where z_i is the daily instantaneous mortality rate (IMR) and d_i is the duration (days) within the i -th stage. Incorporation of interaction between species at different stages can be easily done (Cushing, 1998; Zaldívar and Campolongo, 2000).

3.3. Contaminants fate and transport models

The aim of chemical fate and transport models is to estimate the spatio-temporal distribution of a contaminant in the environment. Normally, these models are based on the mass balance approach, i.e. change = inputs – outputs \pm transformations, and consider the evolution of the pollutant and its distribution between several compartments. An example is shown in fig. 8 (Dueri *et al.*, 2005; Carafa *et al.*, 2006) where the main emphasis is on predict the water and sediment concentrations and where historical concentrations in the sediment, atmospheric concentrations and watershed inputs act as forcing to the model.

The term “compartment” refers to each phase in which the chemical may be present, e.g. dissolved and particulate solids in the water, gas and aerosol phase in the atmosphere, as well as to each element in the food chain. However, fate and transport model consider partitioning between these phases but not the effects of contaminants on predator-prey relationships or toxicity on the abundance of species.

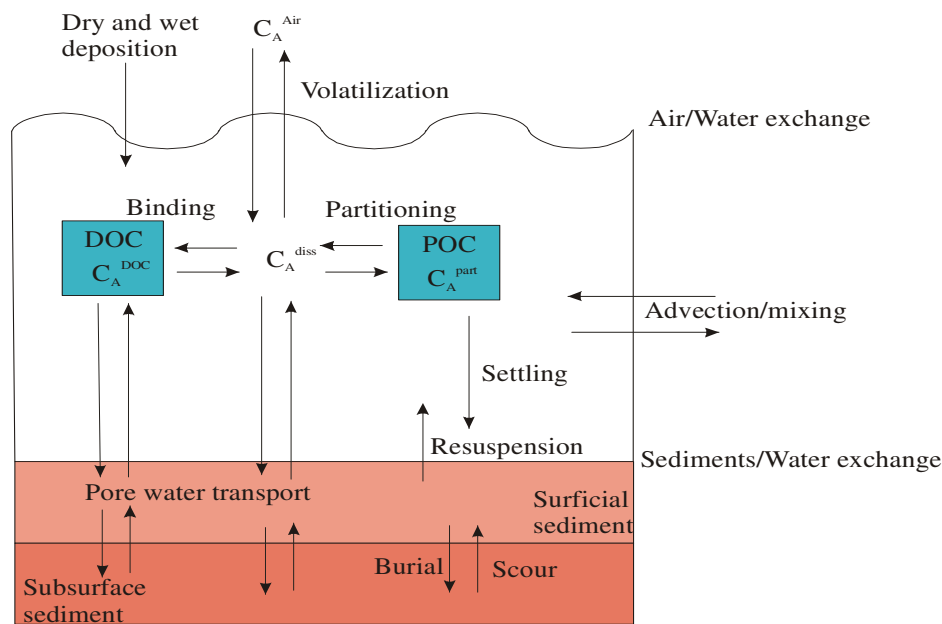


Figure 8. Fate and transport model processes and environmental compartments.

3.4. Food chain bioaccumulation/toxicity model

The main issue normally when introducing contaminants in ecological models is to infer likely the impact on the ecosystem and populations of the toxic effects observed at the individual level. Traditionally, mortality (survival rate) of organisms has been used as the ecological effect of toxicant stress in risk assessment (Bartell, 1996; Lopes *et al.*, 2005, a.o.) but other approaches have been appeared recently. For example, Tanaka (2003) proposed the application of population vulnerability analysis (PVA) which consists on estimating the probability of extinction by evaluating the adverse effects of pollutant chemicals on the intrinsic rate of natural increase, r (Ginzburg *et al.*, 1982).

Bartell (1999) has proposed an iterative process to analyse ecosystem (population) response using toxicological data. This approach is illustrated in fig. 9. In this sense, the risk is evaluated as the probability of detecting a specific change in an annual integrated biomass of a population of interest (O'Neil *et al.*, 1982). In order to carry out this approach, it is necessary to translate single-species toxicity data to elements of an effects matrix which will in turn modify growth rates, mortality, etc. in the modelled processes in the phytoplankton, zooplankton, bacteria, etc. modules contained in the ecological model. This is a necessary step to translate toxicological effects on single population dynamics. Then this combined matrix effects is produced and the total effects at ecosystem level are evaluated. Monte Carlo approach may also be used at this level to assess uncertainty not only in the model equations but also in the environmental factors.

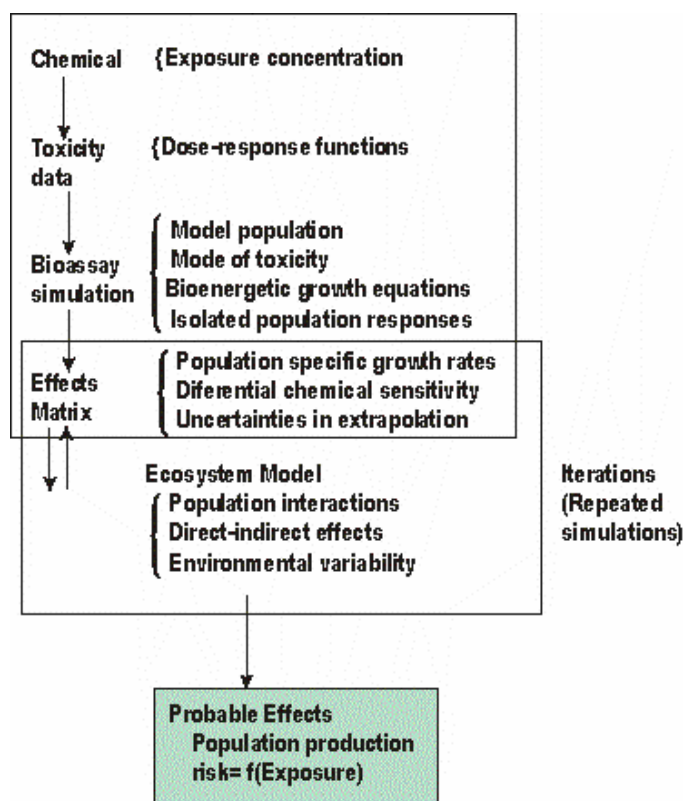


Figure 9. Estimating the ecological risk for a given chemical exposure from toxicity data. The assay simulations produce an effects matrix that modifies growth rates for populations in the food web model. The population effects from several simulations are used in calculating the risk (modified from Bartell, 1999).

3.5. Integrated models

Actually, there are several general models that have been developed to cover several aspects of ERA: ecosystem, fate and effects. Between them: QWASI (Quantitative Water, Air, Sediment Interaction) (Mackay *et al.*, 1983); IFEM (Integrated Fate and Effects Model) (Bartell *et al.*, 1992); the AQUATOX (Park *et al.*, 1995) model, and the GEMCO (Generic Estuary Model for Contaminants) (WL Delft Hydraulics, 2003).

- QWASI

QWASI is a fugacity-based well-mixed compartmental mass balance model also referred as multimedia fate model. These models have been classified according to their complexity in fourth levels: Level I (closed system, equilibrium between compartments, no reaction), Level II (steady-state open system, equilibrium reached at constant emission rate, reaction and advection included), Level III (steady state, compartments not at equilibrium, inter-media transport formulations), Level IV (unsteady state version of Level III). These models have been extensively used in the field of environmental fate assessment

for a large number of chemicals and several intercomparison exercises have been carried out (Cowan *et al.* 1995; Leip and Lammel, 2004, a.o.)

- IFEM Model

This model is the integration of the fates of aromatics model (FOAM) (Bartell *et al.*, 1981) with a general ecological model that takes into account primary producers (phytoplankton, periphyton and rooted macrophytes), and consumers (zooplankton, benthic insects, other benthic invertebrates –e.g. clams-, pelagic omnivorous fish and a benthic detritivorous fish). Figure 10 shows schematic flow diagram of the aquatic ecosystem model, as well as the food web structure and the imposed seasonal patterns of light, temperature and nutrients.

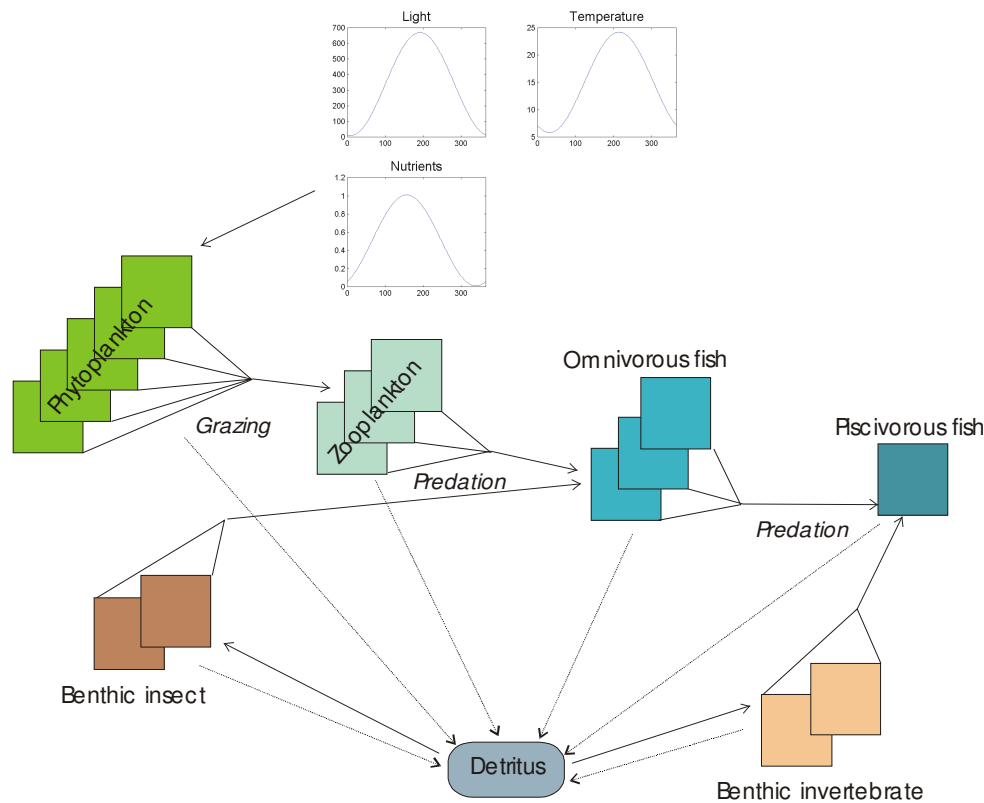


Figure 10. Schematic illustration of the typical aquatic ecosystem showing the food web structure, trophic relations and the imposed seasonal patterns of light, temperature and nutrients (Bartell *et al.*, 1992).

IFEM calculates the uptake, metabolism and effects of accumulated pollutant on aquatic ecosystems. The main chemical processes that are taken into account include: volatilization, photolysis, sorption and desorption. Even though, it was originally developed for PAHs, it has been extended to deal with other families of toxicants (Bartell *et al.* 1999 and 2000).

-AQUATOX

The main objective of AQUATOX (US EPA, 2000) is to provide a realistic estimation of the fate and effects of pollutants in aquatic ecosystems. It has been designed to represent the average daily conditions in a well-mixed aquatic system (0D) or 1D for systems that exhibit stratification. It considers several trophic levels, including attached and planktonic algae and submerged aquatic vegetation, invertebrates, and forage, bottom-feeding and game fish (fig. 11).

The fate model includes partitioning among organisms, suspended and sedimented detritus, suspended and sedimented inorganic sediments and water; volatilization; hydrolysis; photolysis; ionization; and microbial degradation.

The model has been implemented for streams, small rivers, ponds, lakes and reservoirs.

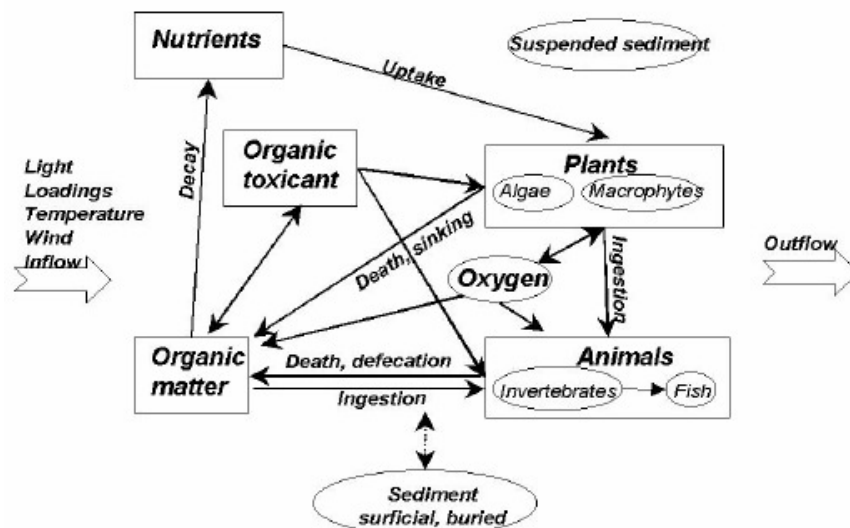


Figure 11. Conceptual model of the ecosystem represented by AQUATOX (US EPA, 2000).

-GEMCO

The GEMCO, born from collaboration between WL Delft Hydraulics (NL), Ifremer (FR) and IVM (NL), was designed to determine the sediment and water concentrations as well as the concentrations and fluxes of contaminants through the different levels in a schematic food web, see fig. 12. Specially, this program was developed to simulate transitional waters and adjacent coastal waters, therefore sediments are taken into account. Estuaries were also classified according to their shape, tidal range and fresh water input in order to derive the salinity distribution. Two types of generic food web have been included: a pelagic and a benthic food web with a pelagic fish (sea bass) and a benthic fish (flat fish) as top predators, respectively.

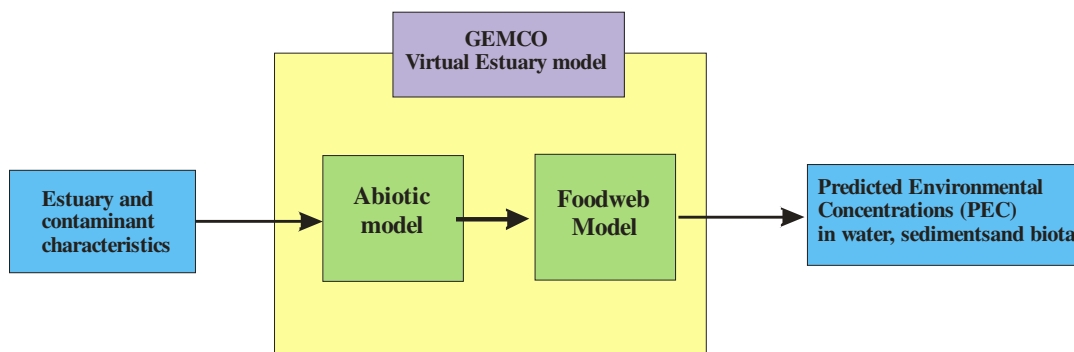


Figure 12. Overview of the GEMCO structure.

As all these models, as input it requires the physico-chemical properties of the contaminant (e.g. molecular mass, organic carbon partition coefficient, Henry coefficient, etc.), the emission data (e.g. concentration in the river, atmospheric load, point sources, etc.) and the estuary characteristics (e.g. river discharge, estuary dimensions, tidal characteristics, water temperature, etc.).

The model was validated in Scheldt and Seine estuaries (Loizeau *et al.*, 2001) using metals (Cd, Cu) and organic pollutants (Atrazine, Lindane and PCBs).

4. Modelling toxic effects in a stage-based population model

As stated previously, one of the main problems in ERA is how to use the data normally obtained from individual responses, such as survival, or growth, to assess the impacts at the level of population or ecosystem. It is clear that the ecological relevance of these data sets is low. However, if these data could be incorporated, through modelling approaches, to population models then we could assess the problems at a more relevant scale.

One solution to this problem is to use discrete ecological models based on a Leslie matrix combined with mathematical modelling of important biological processes. These processes are normally simulated using the theory of Dynamic Energy Budgets (Kooijman, 1993). Furthermore, several Leslie matrices can be combined to develop an ecosystem model (Zaldívar and Campolongo, 2000) and therefore toxic effects introduced at population level can be extended to ecosystem level.

In this report we discuss an approach developed by Lopes *et al.*, 2005 for *C. riparius* which is a non-biting midge with a life-cycle that comprises aquatic stages (eggs, larva and pupae) and aerial ones (adults).

4.1. Population dynamics model of *C. riparius* and response to Methiocarb

In this model the Leslie matrix A , eq. 3, is a 17×17 matrix which coincides with the total duration of the *C. riparius* life cycle. Therefore the probability of surviving and staying in stage i is set to zero, i.e. $P_i = 0$. Furthermore, only adults (latest stage) reproduce and hence, the fecundity for the earlier stages is also set to zero, i.e. $m_2 = \dots = m_{16} = 0$ and $m_{17} = 208.1$ (Lopes *et al.*, 2005).

The probability of surviving and growing into the next stage, G_i , is dependent on the stage and the concentration of the chemical, C ($\mu\text{g L}^{-1}$). In this way results from several bioassays experiments on eggs, larva, pupa and adults can be incorporated correctly to have a population response for a specific chemical (Methiocarb, carbamate pesticide used in agriculture). The equations for each stage can be written as (Lopes *et al.*, 2005):

- Eggs (2 days) and Larval stage 1 (2 days):

$$G_{1-4} = s \frac{1 + \exp(a)}{\exp(a) + \exp(b \cdot C)} \quad (8)$$

with $s=0.836$, $a=8.478$ and $b=0.499$ (Lopes *et al.*, 2005).

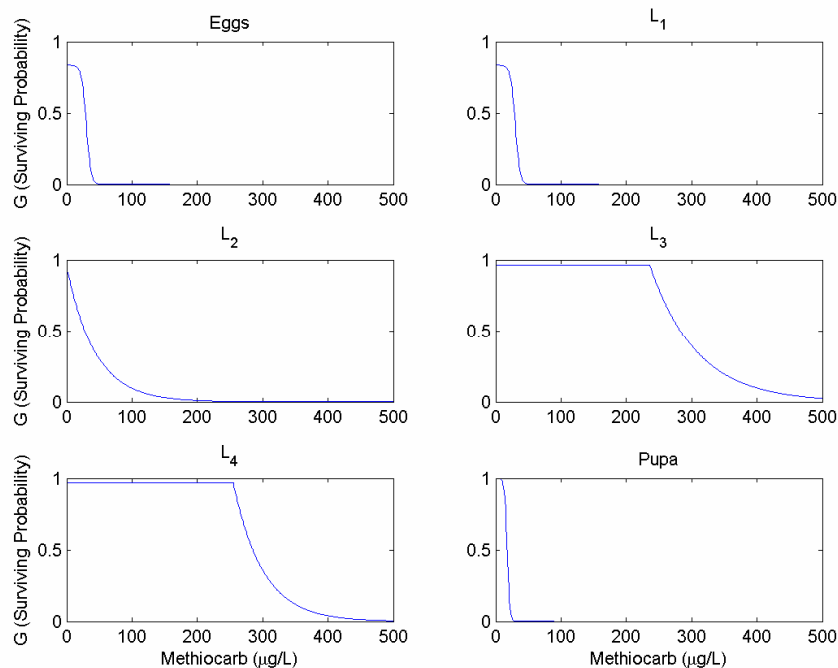


Figure 13. Response of each stage to Methiocarb concentration.

- Larval stages 2 (2days), 3 (3 days) and 4 (6.5 days):

In this case DEBtox models were used. In this approach the uptake of the compound is assumed to be proportional to its concentration in the solution, whereas its elimination proportional to that in the body. This lead to a linear ordinary differential equation that can be solved analytically and by simple manipulation leads to the following expression (assuming that there is a concentration NEC below which there are no effects on the survival of the organism during the bioassays):

If $C < NEC_i$

$$G_i = \exp(-m_i)$$

Else

$$G_i = \exp\left[-m_i + \frac{k_i}{\varepsilon_i} C \exp(-\varepsilon_i \cdot t)[1 - \exp(-\varepsilon_i)] - k_i(C - NEC_i)\right] \quad (9)$$

where m is the natural mortality, k is the killing rate and ε is the elimination rate. Table 1 summarises the parameters estimated for *C. riparius*.

Table 1. Estimated parameters for each stage (Lopes *et al.*, 2005).

Stage	G	m_i	NEC_i	k_i	ε_i
L ₂	G ₅ -G ₆	0.051	$3.73 \cdot 10^{-7}$	0.023	3.474
L ₃	G ₇ -G ₉	0.038	236	0.014	3.171
L ₄	G ₁₀ -G ₁₅	0.033	255	0.022	4.234

- Pupa (0.5 days):

$$G_p = s \frac{1 + \exp(a)}{\exp(a) + \exp(b \cdot C)} \quad (10)$$

with $s=1.0$, $a=8.749$ and $b=0.282$ (Lopes *et al.*, 2005).

As the pupa stage last less than one day, G_{16} is calculated as:

$$G_{16} = G_{15} \cdot G_p \quad (11)$$

Figure 13 shows the effects of methiocarb at each stage. It should be noticed that whereas larval stage L₁ and Pupa are predominantly planktonic, in L₂, L₃ and L₄ they live in the sediments.

The effects of the contaminant at the population level can now be quantified as a function of bioassays data on each stage using the first eigenvalue, λ_l , of the Leslie matrix (Caswell, 1989) also called the population growth rate. If λ_l is lower than one the population will become extinct, greater than one the population will increase exponentially and with a one value it will maintain without growing. As can be seen in fig. 4 in the absence of methiocarb the population growth rate is around 1.28 which corresponds to a daily increase of 28% in absence of predators, i.e. bioassays experiments. As we

increase the concentration of methiocarb this value decreases until a threshold (extinction), in this case a point of non-return, value of approx. $22.72 \mu\text{g L}^{-1}$ where it crosses the one line. Figure 14 shows the simulation at this concentration. As can be seen the population show a cyclic behaviour.

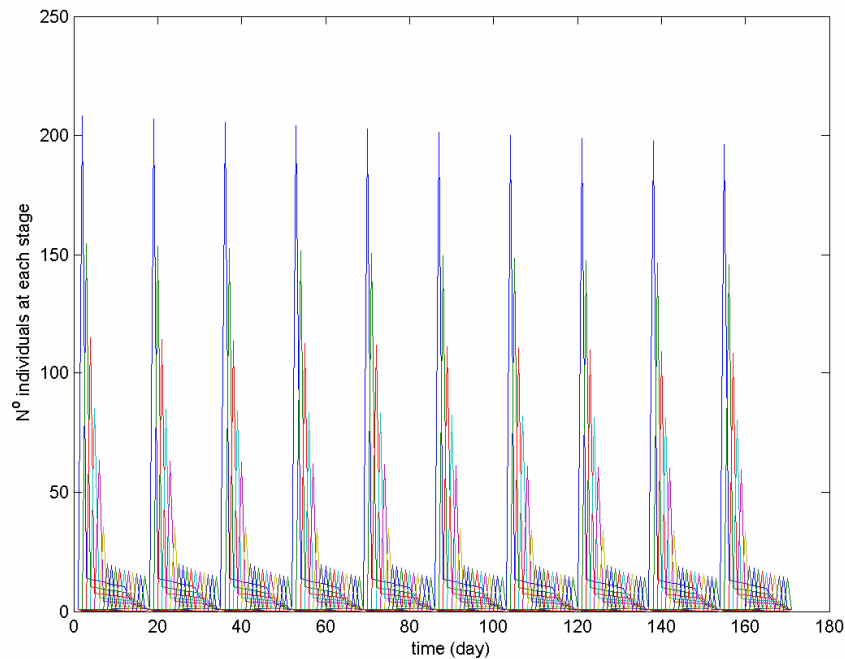


Figure 14. Dynamic simulation of the population of *C. riparius* at each stage with a concentration of methiocarb of $22.72 \mu\text{g L}^{-1}$ which gives a $\lambda \approx 1$.

In this model, the effects of temperature and food availability are not taken into account but they can easily be incorporated as in Zaldívar *et al.* (2003) for clams or as in Smit *et al.* (2006) for *C. volutator* (a marine amphipod used in sediments bioassays).

4.2. Uncertainty analysis in the point of non return

As mentioned above, the value $\lambda = 1$ may be considered as a point of non-return for the *C. riparius* population. This value corresponds to a concentration of $22.72 \mu\text{g L}^{-1}$ of methiocarb. In order to study the uncertainty of this value based on the uncertainty of the experimental results during the analysis of the toxicity experiment for each stage of their life-cycle, we have run approx. 70000 Monte Carlo simulations changing the 16 parameters according with the associated errors (Lopes *et al.*, 2005). The results shown that the point of non return varies from 19.1 to $24.7 \mu\text{g L}^{-1}$ of methiocarb.

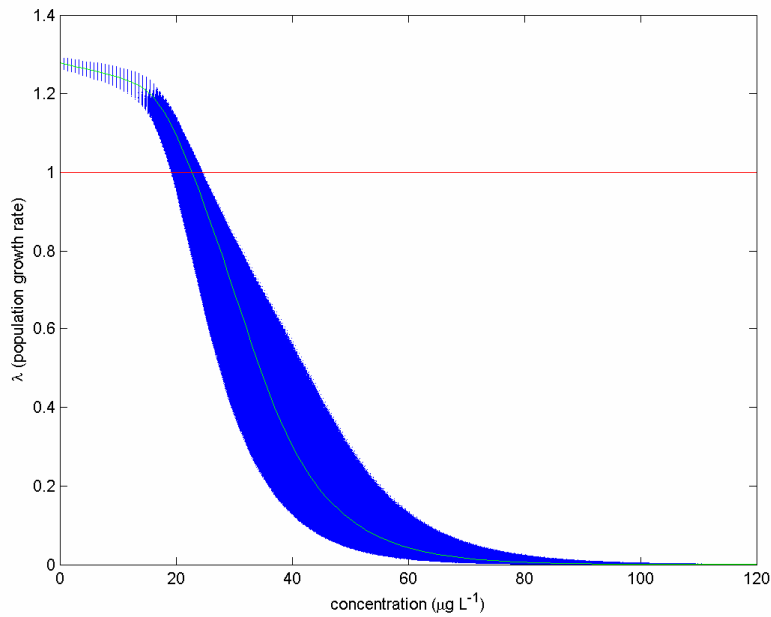


Figure 15. Realisation of 30000 runs on the effects of methiocarb concentrations on population growth rate of *Chironomus riparius* as a function of the uncertainty in the experimental results.

5. Modelling toxic effects in a foodweb model

This model has been developed with the idea of analysing mesocosm experiments that have been conducted by NERI. Therefore, a general model has been adapted for simulating the effects of a pulse of a contaminant on an ecosystem that has zooplankton as the top predator.

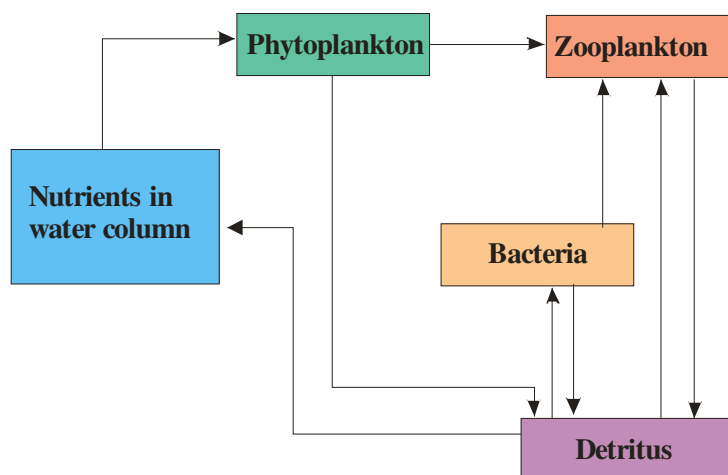


Figure 16. Simplified flow diagram in an aquatic ecosystem (modified from Mosekilde,1996).

5.1. Ecological model

A schematic flow diagram of an aquatic ecosystem is summarised in Fig. 16. Besides the presence of available nutrients in the water column (N), the models normally account for phytoplankton (P), zooplankton (Z). In addition the model contains the so called microbial loop, which accounts for the mineralization of dead organic mater, called detritus (D), performed by the bacteria (B). In the original model (Mosekilde 1996), fish and nutrient in the sediments were also included but for our final objectives, which are to simulate mesocosms experiments (Hjorth *et al.*, 2006) on nutrient and contaminant enrichment, we have not taken into account these compartments. The model assumes that the Redfield molar ratio is conserved, i.e. C:N:P= 106:16: 1.

The ordinary differential equations may be written as:

$$\frac{dP}{dt} = c(t)P\mu_{m,P} \frac{N}{N + K_p} - Z\mu_{m,Z} \frac{P}{P + K_z} \frac{Y_{P,0} + Y_P}{2} - \tau_p P - \rho P \quad (12)$$

$$\frac{dZ}{dt} = Z\mu_{m,Z} \left(\frac{P}{P + K_z} \frac{Y_{P,0} + Y_P}{2} + \frac{B}{B + K_z} \frac{Y_{B,0} + Y_B}{2} + \frac{D}{D + K_z} \frac{Y_{D,0} + Y_D}{2} \right) - \tau_z Z - \rho Z \quad (13)$$

$$\frac{dB}{dt} = B\mu_{m,B} \frac{D}{D + K_B} (1 - \alpha) - Z\mu_{m,Z} \frac{B}{B + K_z} \frac{Y_{B,0} + Y_B}{2} - \tau_B B - \rho B \quad (14)$$

$$\frac{dN}{dt} = \alpha B\mu_{m,B} \frac{D}{D + K_B} - c(t)P\mu_{m,P} \frac{N}{N + K_p} + \rho(C_i - N) \quad (15)$$

$$\frac{dD}{dt} = \tau_p P + \tau_z Z + \tau_B B - Z\mu_{m,Z} \frac{D}{D + K_z} \frac{Y_{D,0} + Y_D}{2} - B\mu_{m,B} \frac{D}{D + K_B} - \beta D - \rho D \quad (16)$$

In this model an annual sine function is used to describe the variation in energy input (light and temperature) to the system:

$$c(t) = \frac{1}{2} \left[\sin \left(2\pi \frac{t - 80}{365} \right) + 1 \right] \quad (17)$$

The specific growth rates follow the Monod equation (Monod, 1950) modified for taking into account the case that there is more than one prey. In this case, the growth rate becomes the sum of n terms of Monod type, one term for each of the n preys species that the considered predator eats. Each of these n terms is weighted so the i -th contribution reads:

$$\mu_{m,i} \frac{S_i}{S_i + K} \frac{Y_{i,0} + Y_i}{2} \quad (18)$$

with $Y_i = S_i / \sum_{i=1}^n S_i$ and $Y_{i,0} = \text{constant}$. Here S_i is the biomass of the i -th prey. $\mu_{m,i}$ and K are the maximal specific growth rate and the half-saturation constant. The parameter $Y_{i,0}$ defines the optimal fraction of the predator's diet deriving from prey of species i , and Y_i measures the actual value of this fraction. Moreover, α is the mineralization part of the bacterial uptake, β the sedimentation rate, γ the mineralization rate, ρ the rate of dilution, C_i the inlet concentration of nutrients. τ is the mortality rate. Y_P , Y_B and Y_D describe the composition of the zooplankton diet.

Figure 17 shows an example of a simulation run covering one year after the model has reached steady state using parameters from Table 2. As can be seen this highly idealised model is able to reproduce the phytoplankton spring bloom immediately followed by an increase in zooplankton population, which in turn depletes the phytoplankton population. A detailed analysis on attractors, bifurcation points and influence of different parameters on the model dynamic behaviour can be found in Mosekilde (1996).

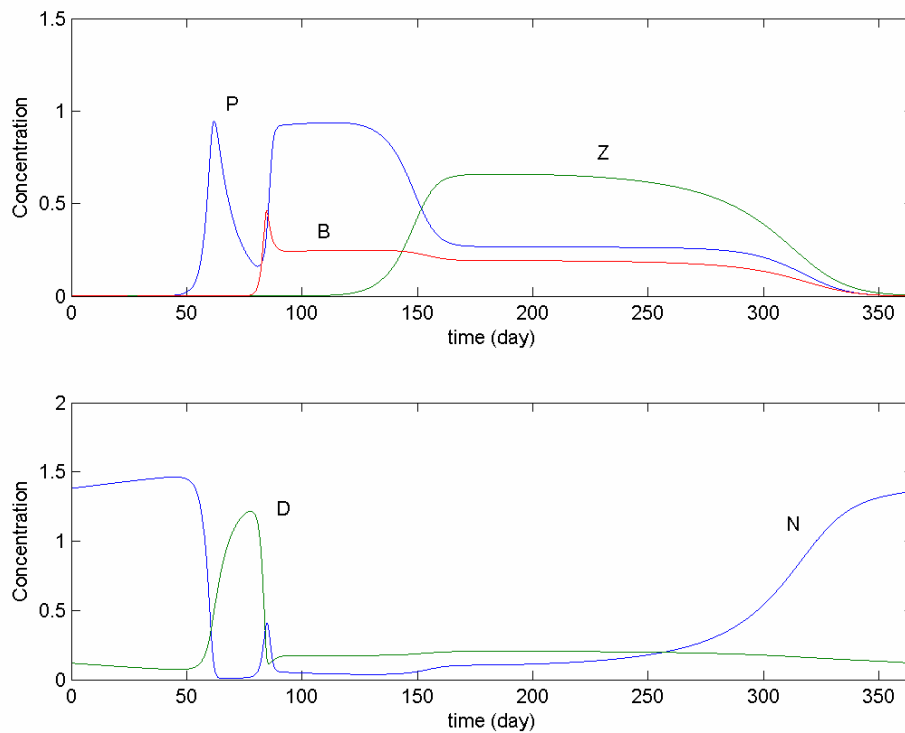


Figure 17. Simulated annual variations of the compartments in the model.

Table 2. Base case parameters for the aquatic ecosystem.

parameter	unit	value	parameter	unit	value
$\mu_{m,P}$	day ⁻¹	2.5	τ_B	day ⁻¹	0.9
$\mu_{m,Z}$	day ⁻¹	0.5	α	-	0.33
$\mu_{m,B}$	day ⁻¹	2.9	β	day ⁻¹	0.009
K_P	mg L ⁻¹	0.6	ρ	day ⁻¹	0.0035
K_Z	mg L ⁻¹	0.6	C_i	mg L ⁻¹	2.0
K_B	mg L ⁻¹	0.2	$Y_{P,0}$	-	0.85
τ_P	day ⁻¹	0.12	$Y_{B,0}$	-	0.05
τ_Z	day ⁻¹	0.14	$Y_{D,0}$	-	0.1

5.2. Fate model

In order to evaluate contaminant exposure in the water column a 0D fate model has been developed, i.e. well-mixed water column, no spatial distribution. The fate model consists of a dynamic mass balance model for calculating pollutant concentrations in the water column. The model is based on Farley *et al.*, (1999) and it has been applied to DDT (Dueri *et al.*, 2005) in lake Maggiore and to s-triazines (Carafa *et al.*, 2006) - coupled with a 3D model- in Sacca di Goro lagoon. As we are interested in simulating mesocosm experiments all the part related to sediments have been cancelled, as well the atmospheric exchanges.

The mass balance equations for the total concentration in the water column can be written as (Farley *et al.*, 1999):

$$V \frac{dC_T}{dt} = Q_{in} \cdot C_{in} - Q_{out} \cdot C_T - w_s \cdot A_s \cdot m \cdot \Gamma + w_u \cdot A_s \cdot m_{sed} \cdot \Gamma_{sed} - A_s \cdot F^{W/S} + A_s \cdot F^{A/W} + A_s \cdot F^{dep} - k \cdot V \cdot C^{diss} - F^{uptake} \quad (19)$$

The first and second term of the *rhs* represent the mass rate of chemical flowing into and out of the system; the third term represents the chemical loss from the water column by settling; the fourth term represents the rate of chemical gain from resuspension; the fifth term represents diffusive exchange between dissolved concentrations in the water column and pore (interstitial) waters; the sixth term represents the transfer of chemical across the air-water interface; the seventh term represents the input by wet and dry deposition; the eighth term represents transformation losses from the water column, e.g. by UV degradation; and the last term represents the contaminant uptake by biota. In this case, there are no inputs and outputs and therefore $Q_{in}=Q_{out}=0$. A pulse of contaminant is simulated by changing the initial conditions at a certain time during the simulation. Furthermore, resuspension, diffusive exchange with the sediments and atmosphere as well as wet and dry deposition have been cancelled.

In this case Pyrene in the water column can be divided into two main components related to their fates and transport routes: dissolved and particulate, i.e. $C_T = C^{diss} + C^{DOC} + C^{part}$. Particulate bound Pyrene is subject to sedimentation. A general approach to describe the particle affinity is by means of the partition coefficient K_d ($m^3 \mu gdw^{-1}$) which is defined as the ratio between the particulate and the dissolved phases as:

$$K_d = \frac{\Gamma}{C^{diss}} \quad (20)$$

whereas C^{diss} is the dissolved concentration ($ng\ m^{-3}$) and Γ ($ng\ \mu g^{-1}$) is the ratio between the particulate concentration (C^{part} , $ng\ m^{-3}$) and the suspended particulate matter concentration in mass dry weight per volume (m , $\mu gdw\ m^{-3}$):

$$\Gamma = \frac{C^{part}}{m} \quad (21)$$

Furthermore, the dissolved phase can be distributed between free chemical and chemical bound to dissolved organic carbon (DOC). Also in this case a partitioning equilibrium relationship is assumed:

$$K_{DOC} = \frac{C^{DOC}/DOC}{C^{diss}} \quad (22)$$

Similar approach can be made for the sediments (Carafa *et al.*, 2006) between dissolved (interstitial or pore water) and particulate concentrations.

It is possible to express the concentration in each phase for the water column and for the sediment, as a function of the total concentration and the equilibrium partition relationships (Farley *et al.*, 1999):

$$C^{diss} = \frac{C_T}{\phi + \phi \cdot K_{DOC} \cdot DOC + K_d \cdot m} \quad (23)$$

$$C^{DOC} = \frac{K_{DOC} \cdot DOC \cdot C_T}{\phi + \phi \cdot K_{DOC} \cdot DOC + K_d \cdot m} \quad (24)$$

$$C^{part} = \frac{K_d \cdot m \cdot C_T}{\phi + \phi \cdot K_{DOC} \cdot DOC + K_d \cdot m} \quad (25)$$

where the water porosity $\phi=1$.

Normally, in fate and transport of organic contaminants models, K_d is considered to be a function of the fraction of organic carbon for suspended solids or sediments, f_{OC} , and the organic-carbon partition coefficient, K_{OC} :

$$K_d = f_{OC} \cdot K_{OC} \quad (26)$$

Values of K_{DOC} can be approximated at K_{OC} values (Karickhoff *et al.*, 1979; Schwarzenbach *et al.*, 2003). According to Chiou *et al.* (1998) for PAHs, K_{OC} can be obtained as:

$$\log K_{OC} = \log K_{ow} - 0.21 \quad (27)$$

Even though air-water and sediment-water exchange, and dry and wet deposition have not been taken into account for the present model, they have been parameterised for Pyrene to be included in the general model under development (Jurado *et al.*, 2006).

Air-Water Exchange

The air-water flux ($F_A^{A/W}$, $\text{ng}\cdot\text{m}^{-2}\cdot\text{s}^{-1}$) assuming equilibrium between the two phases at the interface (i) is given by (Westerterp *et al.*, 1984) as:

$$F_A^{A/W} = \left(\frac{1}{k_G \cdot K_{GL}} + \frac{1}{k_L} \right)^{-1} \left(\frac{C^{Air}}{K_{GL}} - C^{diss} \right) \quad (28)$$

where C^{diss} and C^{Air} are the gas-phase and the dissolved (liquid) concentrations of A (ng/m^3), respectively, K_{GL} is the dimensionless gas-liquid distribution coefficient, $K_{GL} = C_{AG}^i / C_{AL}^i$, which may be calculated from the Henry's law constant using: $K_{GL} = H / (R \cdot T)$, and k_G and k_L are the air-water mass transfer coefficients (m/s).

Mass transfer coefficient: Liquid side

In this work we have used a recent correlation developed by Nightingale *et al.*, (2000) that has been employed to estimate the global dynamics and sinks of organic pollutants by Dachs *et al.* (2002). This correlation can be written as:

$$k_{L_{norm}} = 6.667 \cdot 10^{-7} u_{10} + 1.6944 \cdot 10^{-7} u_{10}^2 \quad (29)$$

where k_L is given in m/s. In order to correct for a different Schmidt number ($Sc = \mu / D \cdot \rho$) one has to modify the value as follows:

$$k_L = k_{L_{norm}} \left(\frac{Sc_A}{600} \right)^{-0.5} \quad (30)$$

The liquid phase diffusion coefficients are calculated as a function of temperature and water viscosity following the correlation proposed by Wilke and Chang (1955) as:

$$D_L (\text{m}^2 / \text{s}) = \alpha_{id} \cdot T / \mu_w \quad (31)$$

with α_{id} equal to $6.78 \cdot 10^{-12}$ for Pyrene.

Mass transfer coefficient: Gas side

Similarly, several correlations, using water vapour laboratory and field experiments, have been developed to estimate the gas phase mass transfer coefficient (m/s). In this case the normalised value refers to the mass transfer coefficient for water, which may be correlated as a function of wind speed like follows (Schwarzenbach *et al.*, 2003):

$$k_G^{H_2O} = 2 \cdot 10^{-3} u_{10} + 3 \cdot 10^{-3} \quad (32)$$

and then

$$k_G = k_G^{H_2O} \left(D_G^A / D_G^{H_2O} \right)^{0.61} \quad (33)$$

where D_G refers to the diffusion coefficients of the chemical and water in the gas phase (air). The gas phase diffusion coefficient as a function of temperature has been calculated using the empirical correlation developed by Fuller *et al.* (1966):

$$D_G (m^2 / s) = \alpha_{gd} \cdot T^{1.75} \quad (34)$$

For the case of water, and pyrene the values are: $1.237 \cdot 10^{-9}$, $2.695 \cdot 10^{-10}$, respectively.

The Henry's law constant

The temperature dependence of the Henry's law constant can be expressed as:

$$\log H = A_H - \frac{B_H}{T} \quad (35)$$

The following values for A_H and B_H has been used: $A_H = 9.17$, $B_H = 2475$, for pyrene (Paasivirta *et al.* 1999).

Dry and wet deposition

The dry deposition flux F^d ($\text{ng} \cdot \text{m}^{-2} \cdot \text{s}^{-1}$) may be estimated from the particle deposition velocity (v_d , m/s) and the micropollutant concentration in the aerosol phase (ng/m^3) as (Swackhamer *et al.*, 1999):

$$F_A^d = C_A^{\text{aerosol}} \cdot v_d \quad (36)$$

Normal values for particle deposition velocity for lakes and coastal areas usually range from $1 \cdot 10^{-3}$ to $8 \cdot 10^{-3}$ m/s (Nho-Kim *et al.*, 2004). In this work we have used $2 \cdot 10^{-3}$ m/s as in Shawn *et al.*, (1997).

Wet deposition, F^w may be calculated (Swackhamer *et al.*, 1999; Van Ry *et al.*, 2000) from the precipitation rate (Pr , m/s) and the micropollutant concentration in rain water (ng/m^3) as:

$$F_A^w = C_A^{\text{rain}} \cdot Pr \quad (37)$$

The sum of both contributions constitutes the term F^{dep} in Eq. (19). Concentrations in air (gas + aerosol phases) were obtained from concentrations in rain using the approach proposed by Jurado *et al.* (2004, 2005).

Sediment deposition: Settling

As summarised by Hawley (1982), observed data collected for settling velocities of lacustrine and marine particles show velocities up to one order of magnitude higher than using the Stokes' velocity. Normally empirical values are used in simulation programs. In this work we have used the value of $3.87 \cdot 10^{-5}$ m/s proposed by Carrer et al. (2000) for Venice lagoon. Using this value and the concentration in the particulate phase, it is possible to calculate the settling flux ($\text{ng} \cdot \text{m}^{-2} \cdot \text{s}^{-1}$) as:

$$F^{sediment.} = w_s \cdot C^{part} \quad (38)$$

Sediments-Water Exchange

Concerning only the sediment-water diffusive transfer coefficient, Di Toro *et al.* (1981) concluded that the main resistance to mass transfer lies in the sediment and that it is possible to write:

$$k_s = 2.2 \cdot 10^{-6} \phi \cdot MW^{-2/3} \quad (39)$$

for k_s in m/s. A typical order of magnitude for k_s is then about $1.2 \cdot 10^{-8}$ – $1.2 \cdot 10^{-7}$ m/s.

Chemical and biological loss rate

The half-life ($t_{0.5}$) is the time required for pollutant concentration to be reduced by half due to physical and chemical processes, including transformation to its degradation products. It is estimated using the assumption of first-order kinetics. Estimations of pyrene half life show a wide range of value, from few days to more than one year, depending on the environmental conditions as well as on the presence/absence of bacteria (Heitkamp *et al.* 1988; Tam *et al.*, 2003; Greenfield and Davis, 2004; a.o.). A value of $k = 1.157 \cdot 10^{-7} (\text{s}^{-1})$ was selected.

Biota uptake

The uptake by biota is proportional to the dissolved fraction and given by the following equation:

$$F^{uptake} = (P \cdot k_u^p + Z \cdot k_u^z + B \cdot k_u^b) V \cdot C^{diss} \quad (40)$$

5.3. Bioaccumulation model

For simplicity, let us assume a certain contaminant. In this case we have taken a PAH, pyrene, because it has been investigated within the Thresholds project. However, the model is quite general and can be extended to other contaminants. The concentration of Pyrene in the dissolved phase is calculated using Eq. (23) after solving the ordinary differential equation (Eq. 19) that gives the total concentration in the water column.

The accumulation and depuration of a contaminant (pyrene), C_i (ng/kg), by the i -th population may be modeled according to the following equations (Dachs *et al.*, 1999; Del Vento and Dachs, 2002; Berrojalbiz *et al.*, 2006).

- For producers (phytoplankton):

$$\frac{dC_p}{dt} = k_u^p \cdot C_{PAH}^{dis} - k_d^p \cdot C_p \quad (41)$$

where k_u ($m^3 \cdot kg^{-1} \cdot d^{-1}$) and k_d (d^{-1}) are the uptake and depuration rate constants.

- For consumers (zooplankton and bacteria):

$$\frac{dC_z}{dt} = k_u^z \cdot C_{PAH}^{dis} + k_g^z \cdot C_p - k_d^z \cdot C_z - k_e^z \cdot C_z - k_m^z \cdot C_z \quad (42)$$

where k_g ($m^3 \cdot kg^{-1} \cdot d^{-1}$), k_e (d^{-1}) and k_m (d^{-1}) are the grazing, egestion and metabolization rate constants. Bacteria feed on detritus. Therefore it is necessary to consider also the concentration in the particulate phase (Detritus, see Eq. 16). The rate constants used in the model are given in Table 3, phytoplankton data was taken from Del Vento and Dachs (2002), whereas zooplankton data was provided in Berrojalbiz *et al.* (2006). No data was found for bacteria.

$$\frac{dC_b}{dt} = k_u^b \cdot C_{PAH}^{dis} + k_g^b \cdot C_d - k_d^b \cdot C_b - k_e^b \cdot C_b - k_m^b \cdot C_b \quad (43)$$

Table 3. Constants used for the bioaccumulation model.

Species	uptake	depuration	grazing	egestion	metabolism
Phytoplankton	40.4	0.58	-	-	-
Zooplankton	219.31	354.56	226.67	0.13	0.44
Bacteria	219.31	354.56	226.67	0.13	0.44

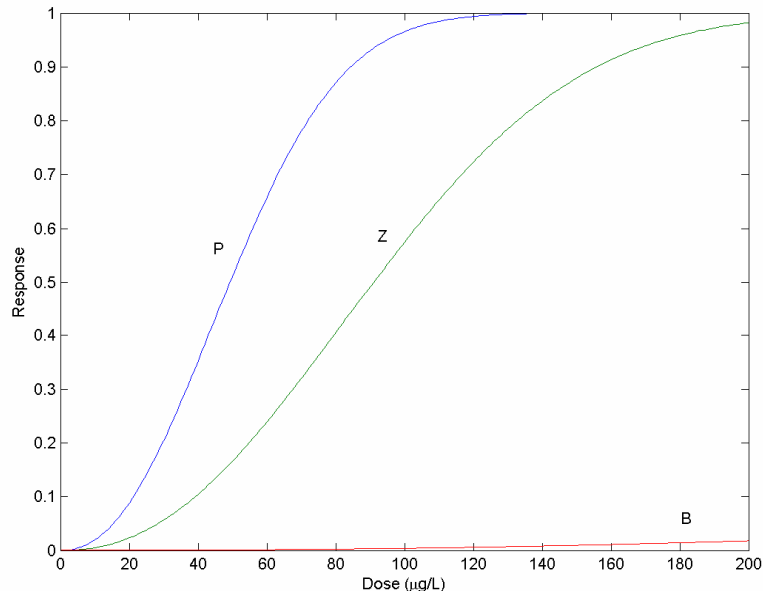


Figure 18. Dose-response curves for phytoplankton (P), zooplankton (Z) and bacteria (B) for pyrene.

5.4. Toxic effects model

Mortality of the different components of the trophic food chain has been modeled based on dose-response data for phytoplankton from Grote *et al.* (2005) and Djomo *et al.* (2004) and from zooplankton from <http://www.pesticideinfo.org/>. No data has been found for bacteria, therefore the toxicity data have been assumed low and comparable with unicellular species in the above mentioned database. In this model the dose-response effects have been simulated using the Weibull equation:

$$f(x) = 1 - \exp[-\exp(\theta_1 + \theta_2 \log_{10} x)] \quad (44)$$

Table 4 shows the fitted parameters, whereas fig. 18 shows the calculated curves for phytoplankton, zooplankton and bacteria.

Table 4. Parameters for the Pyrene dose-response function in the model.

Parameter	Phytoplankton ¹	Zooplankton ²	Bacteria ³
θ_1	-9.072	-10.442	-15.8486
θ_2	5.143	5.143	5.143

¹from Grote *et al.* (2005); ²from <http://www.pesticideinfo.org/> (Daphnia Magna, EC₅₀ 91.02 µg/L); ³No data found

In the model, the mortality of the ecological model is changed as a function of the concentration of contaminant by adding to the mortality rate τ_i , the induced pyrene mortality as given by Eq. (43). In addition this mortality term produces an increase in the detritus fraction and therefore a change in the distribution of the contaminant between dissolved and particulate phases.

5.5. Simulated Results

Due to the fact that we are interested in simulating mesocosm experiments, the model was tested for a pulse addition of pyrene following the experimental conditions described by Hjorth *et al.* (2006) and Dahllöf and Hjorth (2006). In order to let the ecological model reach the final attractor, which is in these conditions a limit cycle, the system was simulated during four years and then a pulse of pyrene was added to the 3 m³ tank at day 0, 80, 160, 240 and 320 of the fourth year. Figures 19-23 show the simulated results for the ecosystem as well as the distribution of contaminants between the species and the fate of pyrene. As can be seen the short term effects change with the time of introduction and are more pronounced in the zooplankton than in the phytoplankton, even though there is a factor of two on EC₅₀. This is a typical example of indirect effects of contaminants. In all the cases, the perturbation does not change the attractor of the system and after a transient phase that can last more than one year, the system returns to the original attractor.

Figure 24 shows in detail the variation of concentrations of contaminant distributed in each species. As can be seen, the concentration in bacteria increases more rapidly due to the fact that they feed on detritus and pyrene tend to be attached to particulate matter. However as particulate matter settles and the first peak of mortality has finished the concentration in bacteria decreases faster than in phytoplankton and zooplankton. However, as said before, we have not been able to find data on bacteria toxicity to pyrene, nor in uptake, depuration etc. rates. Therefore, these first results have not been validated.

The next step will be to fit the model with mesocosm data on pyrene effects and then to validate it by proposing new experiments.

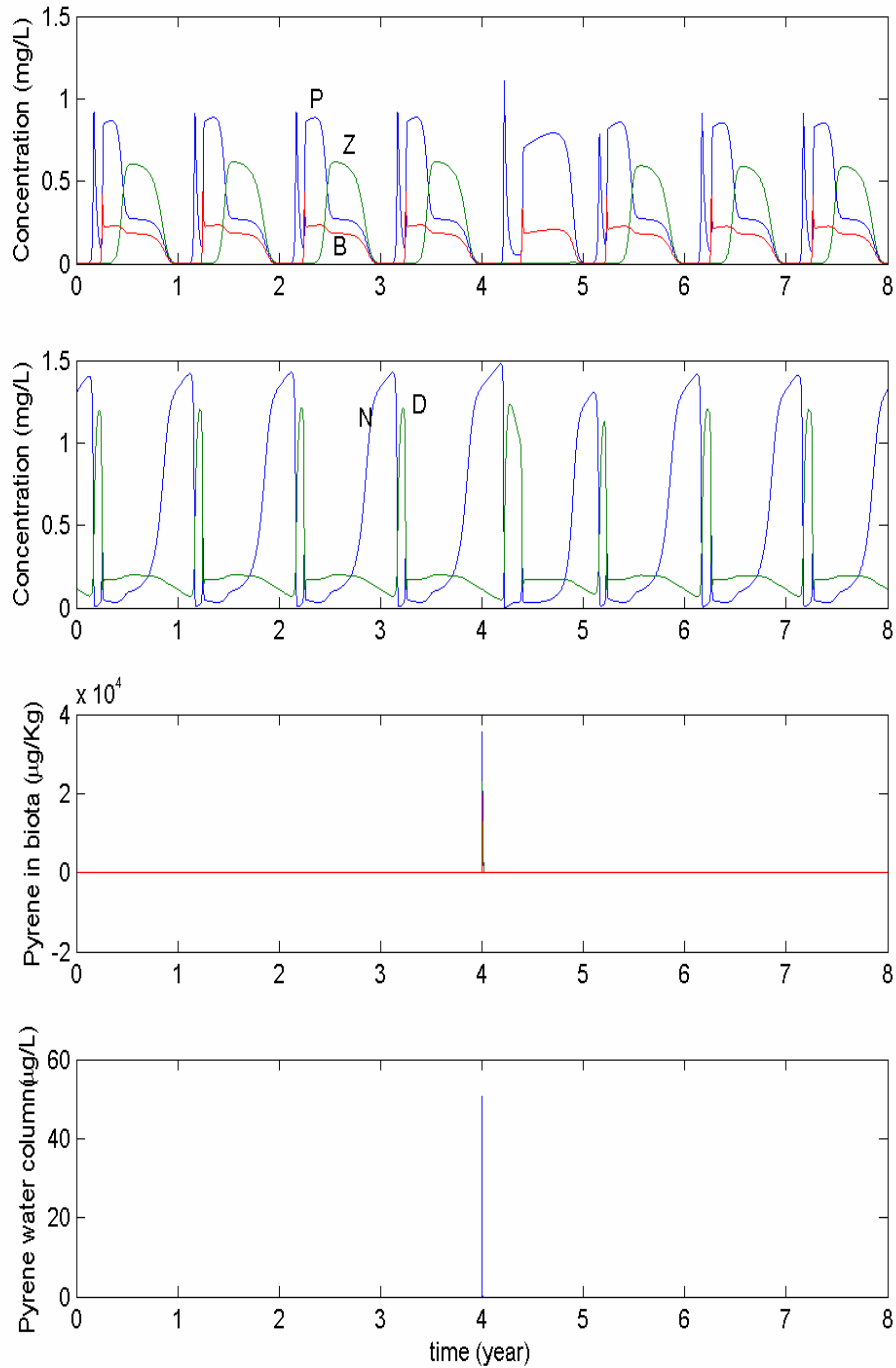


Figure 19. Simulated results of a pulse injection of Pyrene in a mesocosm experiment at the beginning of the fourth year. a/ Phytoplankton (P), zooplankton (Z) and Bacteria (B) biomasses; b/ Nutrient (N) and Detritus (D) concentrations; c/ Contaminant concentrations in Phytoplankton, zooplankton and Bacteria; d/ Total concentration of Pyrene in water.

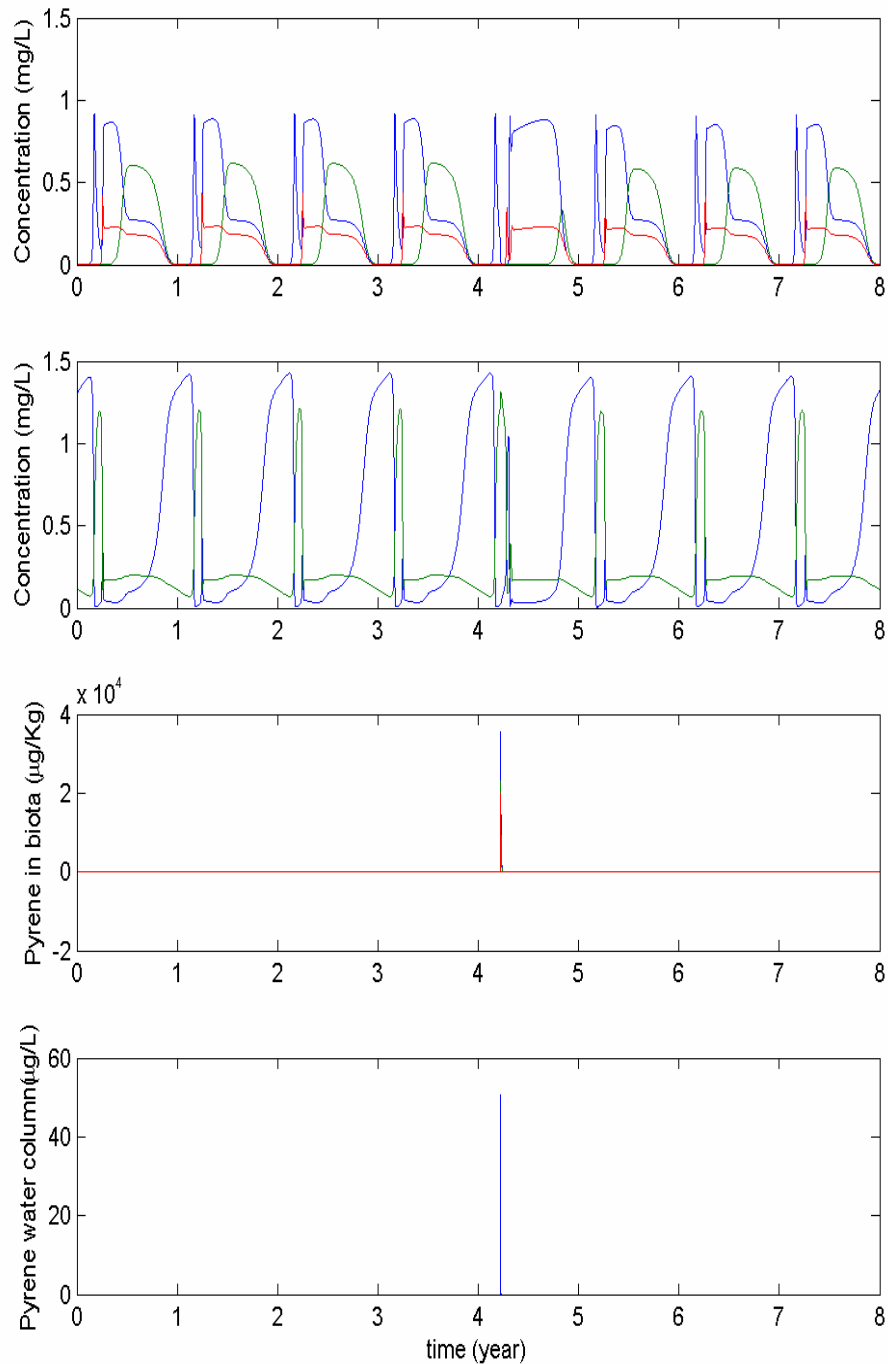


Figure 20. Simulated results of a pulse injection of Pyrene in a mesocosm experiment 80 days after the beginning of the fourth year. a/ Phytoplankton (P), zooplankton (Z) and Bacteria (B) biomasses; b/ Nutrient (N) and Detritus (D) concentrations; c/ Contaminant concentrations in Phytoplankton, zooplankton and Bacteria; d/ Total concentration of Pyrene in water.

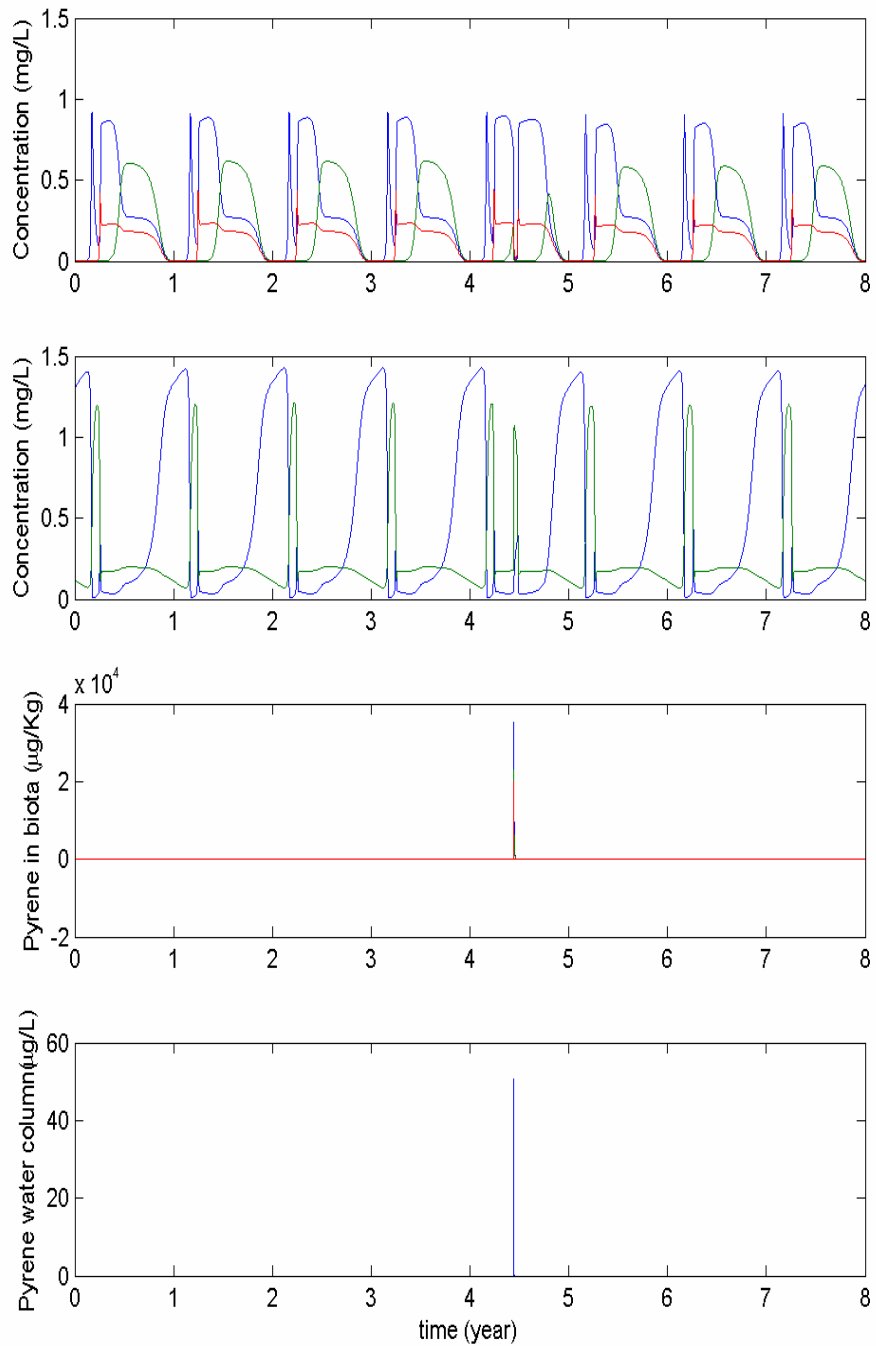


Figure 21. Simulated results of a pulse injection of Pyrene in a mesocosm experiment 160 days after the beginning of the fourth year. a/ Phytoplankton (P), zooplankton (Z) and Bacteria (B) biomasses; b/ Nutrient (N) and Detritus (D) concentrations; c/ Contaminant concentrations in Phytoplankton, zooplankton and Bacteria; d/ Total concentration of Pyrene in water.

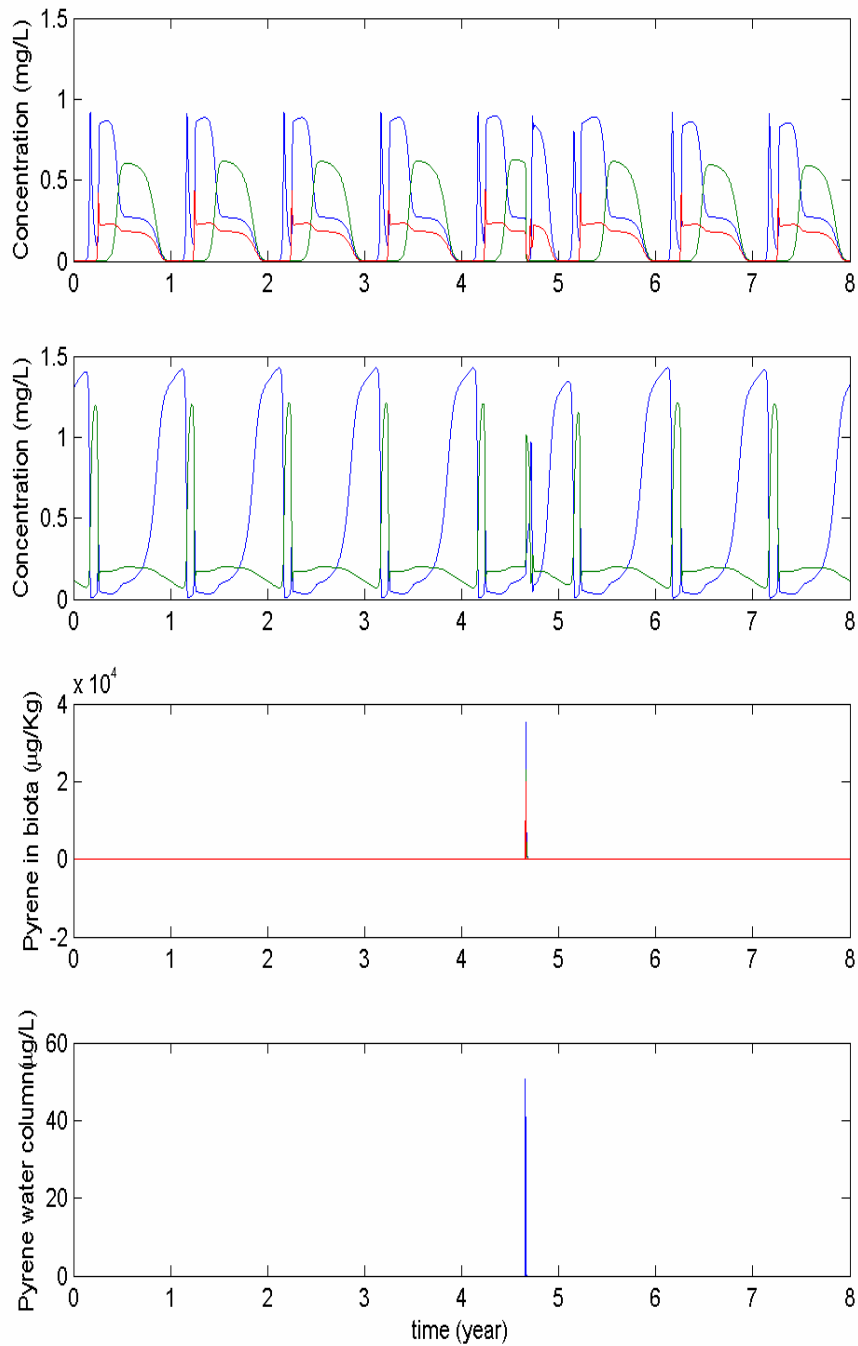


Figure 22. Simulated results of a pulse injection of Pyrene in a mesocosm experiment 240 days after the beginning of the fourth year. a/ Phytoplankton (P), zooplankton (Z) and Bacteria (B) biomasses; b/ Nutrient (N) and Detritus (D) concentrations; c/ Contaminant concentrations in Phytoplankton, zooplankton and Bacteria; d/ Total concentration of Pyrene in water.

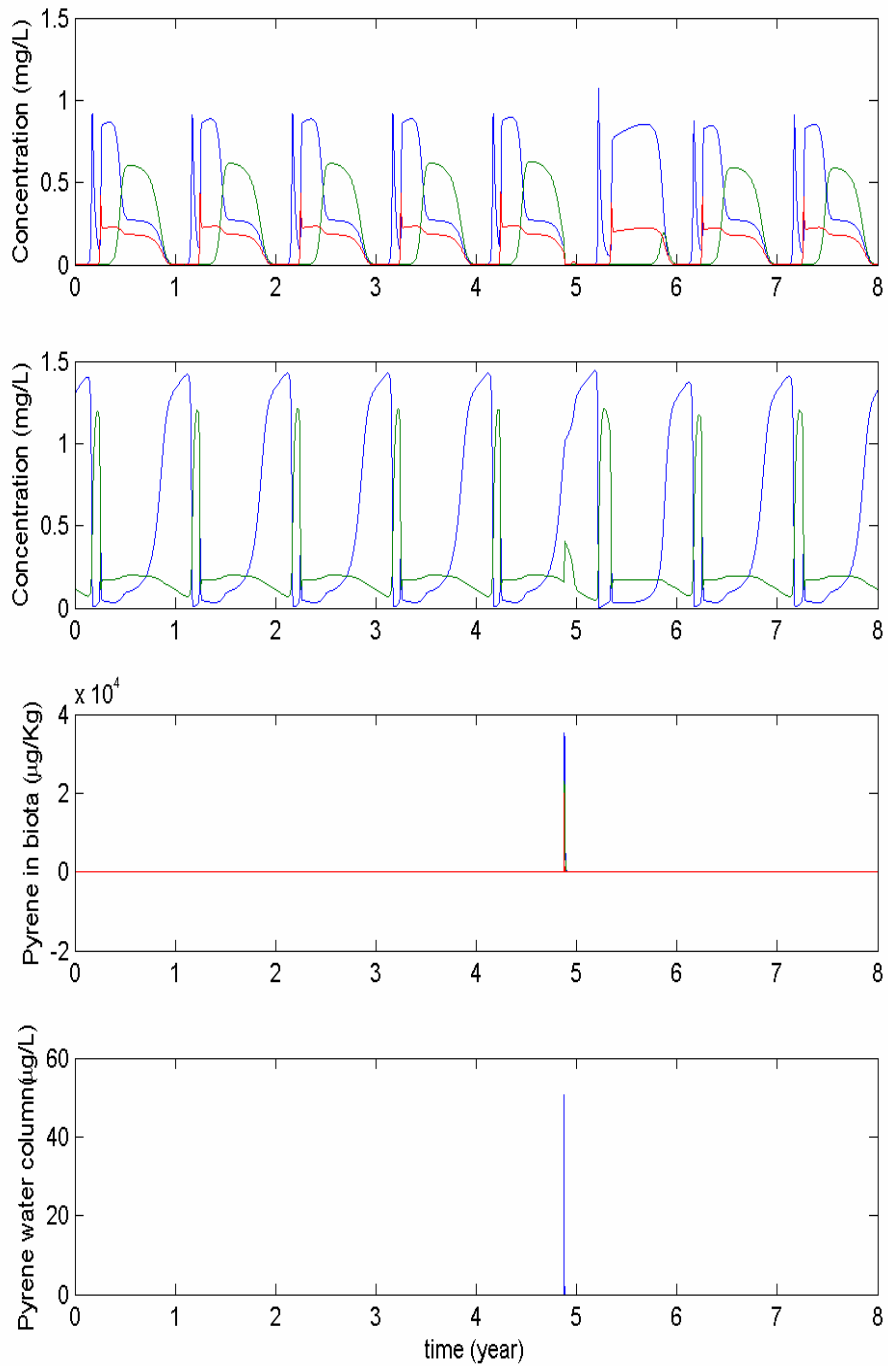


Figure 23. Simulated results of a pulse injection of Pyrene in a mesocosm experiment 320 days after the beginning of the fourth year. a/ Phytoplankton (P), zooplankton (Z) and Bacteria (B) biomasses; b/ Nutrient (N) and Detritus (D) concentrations; c/ Contaminant concentrations in Phytoplankton, zooplankton and Bacteria; d/ Total concentration of Pyrene in water.

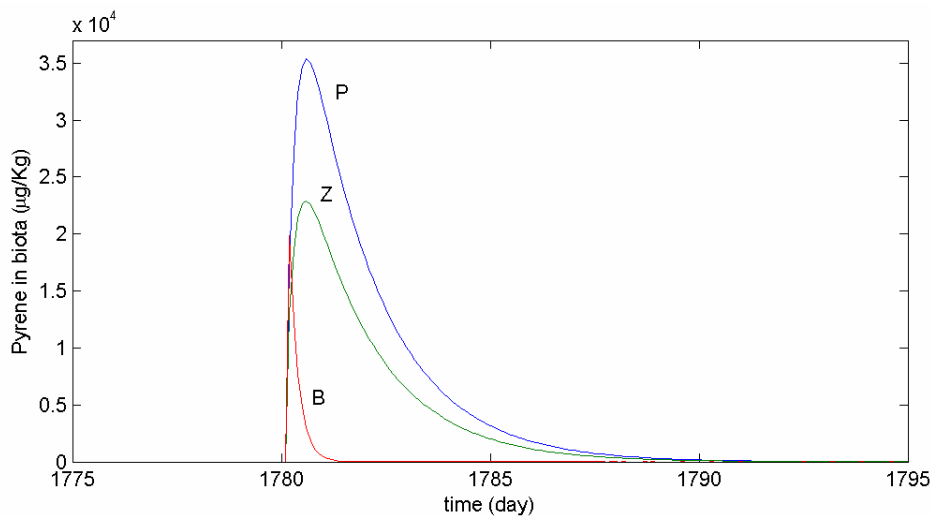


Figure 24. Simulated concentrations of pyrene in phytoplankton (P), zooplankton (Z) and bacteria (B). Detail of fig. 23c.

6. Conclusions

Integrated models are essential tools to move from the effects of toxic chemicals on individual organisms to the ecological significance of these measured effects. This is an important and needed step in ecological risk assessment, since management strategies and measures are taken based on the ecosystem level. Furthermore, models allow to carry out, at the ecosystem level, the uncertainty and sensitivity analysis and are able to assess several aspects related with contaminant release that are not easy to study experimentally.

The introduction of toxic effects in ecological models is more direct in stage-based models since it is only necessary to modify the probabilities of survival to the next stage and/or those of survival in the same stage. These values as it can be seen in Ch. 4 can be expressed directly as a function of the concentration of a contaminant. Furthermore, it is always possible to link several populations and to create a food web stage-based model as in Zaldívar and Campolongo (2000). On the contrary, the introduction of toxic effects on continuous food-web models present several problems, since the values are not easily translated into mortality.

In any case this is a complementary approach to study the complex interactions between ecosystem components and to assess the secondary effects not directly linked with toxicity. This is evident in the food web model where zooplankton is more affected than phytoplankton even though their sensitivity to the toxic compound is lower by a factor of two. This may be explained by the low growth rate values assigned in the model and therefore longer recovering times.

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