Ecological Risk Assessment of Chemical Pollutants : Advances in Analyses on Population-Level Effects

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Abstract

Application of the ecological models was attempted to several important aspects in the ecological risk assessment. Since the extinction probability or the mean extinction time is one of the most useful endpoints which are utilized in biological conservation, parallel approaches that convert ecological hazards into the extinction risk are also useful for the ecological risk assessment of chemical pollutants. The present study attempts extension of the PVA (population vulnerability analysis) to examine population-level effects of pollutants in terms of extinction.

Because the intrinsic rate of population growth, r, is one of the most important parameters that determine persistence of a population, I reviewed ecotoxicological data that estimated the population-level effects in terms of r, and examined three mathematical models, i.e., the power function model, Weibull model and the quadratic function model, for describing the concentration-r curves. The power function model provided the best fit to data.

To incorporate the interspecific interactions, e.g., the prey-predator relationship, into the ecological risk assessment is a difficult task. The standard PVA does not directly examine the effects through interspecific interactions. The present study attempted to incorporate population dynamics of food species (planktons) to a target fish species with an extended matrix population model.

Introduction

Estimation of population-level effects of chemical pollutants is a basis of ecological risk assessment (Suter 1993). The population-level effects are associated with reductions in the ability of a population to proliferate and hence extinction of a population. To evaluate the pollutant effects on populations in terms of decreases in "the intrinsic rate of population growth" is especially important because the intrinsic rate of population growth determines the liability of a population to extinction. For protection of a ecosystem, persistence of populations of composite species is necessary.

In quantitative risk evaluation of chemical pollutants, dose-response relationships provide the only source of empirical grounds. Nonetheless, as for the dose-response relationships in terms of the

intrinsic rate of population growth, very few statistical analyses based on mathematical models have been conducted. This study will review ecotoxicity data that estimated pollutant adverse effects upon the intrinsic rate of population growth, and will determine the most likely dose-response function. And the chronic data will be summarized into a couple of index parameters of the model. I also estimated acute-chronic regression slopes in terms of the estimated parameter values and acute LC_{50}s.

Using the derived dose-response function and exposure date of some chemical pollutants I conducted ecological risk assessment based on reductions in mean extinction time (MET analysis).

There is a long history of arguments about two major problems in applying the toxicological data obtained in laboratories to actual hazards by chemicals in nature. First, in nature most chemicals exhibit much lower environmental exposure concentration than experimental exposure concentrations in laboratories. Therefore, extrapolation from experimental data to responses to low concentrations is inevitable. Second, in many real circumstances organisms encounter many chemical pollutants at the same time, and the compound effects may not be explained by additive effects of the single actions of the chemicals. The present study is intending to resolve these problems by introducing an alternative definition of risk, "reductional risk". The reductional risk of a specific risk factor is defined as a decrement of the total risk that is caused from reduction of the risk factor. The total risk means the observable risk level that results from all risk factors. The total risk is conceptually decomposed into infinitesimal risks that are attributed to specific risk factors, the framework is called "the top-down decomposition of reductional risk" in this paper.

There is another complexity concerning the ecological risk assessment. Since real ecosystems consist of many species which are interacting with each other. In many cases hazards by chemical pollutants to ecosystems appear at the level of community through interaction between composite species (Bartell et al. 1992). In the present study, I will combine species interaction of prey-predator relationships with a single species population vulnerability analysis (PVA), which focuses on the medaka fish (*Orizyas latipes*). The interacting species are assumed to be a zooplankton, e.g., *Daphnia* sp, and a phytoplankton, e.g., *Scenedesmus* sp. The present model is not a comprehensive ecosystem model, nonetheless, interaction between species can be partly included in the extinction risk estimation of a focused species.

Extinction Risk Analysis

Mean Extinction Time Analysis (META) with the Diffusion Model

Analytical solutions for mean extinction time (MET) or extinction probability for a specific duration have been investigated by employing the diffusion approximation (Lande 1993; Foley 1994; reviewed by Iwasa 1998; Matsuda 1998; Tanaka 1998). Extinction is induced by several factors, i.e. environmental stochasticity, demographic stochasticity, catastrophic events. Even focusing on a single factor, predictions of MET vary noticeably between theoretical models which are based on different assumptions. Nonetheless, the dependence of MET on demographic and environmental parameters are fairly compatible between models ("scaling law", Lande 1998). The environmental stochasticity is a major factor inducing extinction of relatively large populations. The other three factors govern extinction of small or declining populations, which are essentially at the final phase of extinction. The ecological risk estimation may be based on moderately large populations because most extant populations in nature are not endangered. Therefore, the environmental stochasticity may be the primary factor of extinction when we evaluate extinction risk of chemical pollutants.

According to the scaling law, MET is roughly proportional to a power of population size: $\overline{T} \propto N^{2r/V_e - 1}$, where $\overline{T}$ is MET, N the population size, r the intrinsic rate of population growth,

and V_e the environmental variance of r. Thus MET decreases geometrically with the relative magnitude of the mean population growth rate to the environmental variance of growth rate, r/V_e.

Population Vulnerability Analysis (PVA) with the Matrix Model

Many test species, in particular fishes, are not suitable for complete life cycle experiments mostly due to long life span and large labor needed for rearing the animals for a long period. Partial life cycle test, which examines semi-chronic toxicity for survival and reproduction in each (preferably early) life stage, is the best alternative. The most standard method to simulate population dynamics of such stage-structured populations is Leslie matrix model. The population numbers of each life stage are denoted as a vector **n** of population size, $\mathbf{n}=(n_1\ n_2\ n_3\ n_4\ n_5)^T$ where n_i is population size of the i-th life stage and T is matrix transpose. Population dynamics is expressed as a recurrence equation,

$$\mathbf{n}(t+1) = \mathbf{L}(t)\mathbf{n}(t)$$

where t is time in generations. If only the last life stage reproduces, the projection matrix **L** has elements (vital rates) as follows,

$$\mathbf{L} = \begin{bmatrix} p_1(1-s_1) & 0 & 0 & 0 & f_5 \\ p_1 s_1 & p_2(1-s_2) & 0 & 0 & 0 \\ 0 & p_2 s_2 & p_3(1-s_3) & 0 & 0 \\ 0 & 0 & p_3 s_3 & p_4(1-s_4) & 0 \\ 0 & 0 & 0 & p_4 s_4 & p_5 \end{bmatrix},$$

where p_i is probability of survival per unit time of the i-th stage, s_i is a proportion of survived individuals in the i-th stage to enter the next stage per unit time, and f_i is per capita reproduction of i-th stage. If some elements depend on time, **L** turns out to be time-dependent.

The population dynamics is simulated with the matrix model, and probability of extinction until specific time is directly calculated from the simulations (see the later section).

Population-Level Effects of Pollutants in Terms of the Population Growth Rate

The intrinsic rate of natural increase, r, or the population growth rate is one of the most important benchmark parameters since it determines the persistence or the mean extinction time of populations. The most complete experiments designed for estimating r are the life table evaluation and the population growth experiments (Tanaka 1998). In this section I will review published data on dose-response relationships in terms of r (the data source is listed in Tanaka 1998), and examine mathematical models to describe the responses.

At first I collected 63 concentration-r relationships from 38 publications. I examined three mathematical models for fitness to the data, i.e., the power function model, the Weibull model, and the quadratic function model.

model	equation
power function model	$r(x) = \gamma\left\{1-\left(\frac{x}{\alpha}\right)^{\beta}\right\}$
Weibull model	$r(x) = \gamma\left[2-\exp\left\{\left(\frac{x}{\alpha}\right)^{\beta}\right\}\right]$
quadratic function model	$r(x) = \gamma\left(1-\alpha x-\beta x^2\right)$

The mathematical models were applied to all data sets with the maximum likelihood method, and the fitness to data was compared between the models based on the model selection criterion (MSC). The MSC is defined as

$$MSC = \ln\left[\frac{\sum_{i=1}^{n} w_i(x_i-\bar{x})^2}{\sum_{i=1}^{n} w_i(x_i-E[x_i])^2}\right] - \frac{2p}{n},$$ where x_i is data, $\bar{x}$ mean value, $E[x_i]$ the expected value, p the number of parameters, w_i weighting term, and n the number of data (Newman 1994).

The MSC scores represent the goodness of a specific mathematical model in fitting data. The distribution of MSCs for all data sets with the three models are presented in Figure 1.

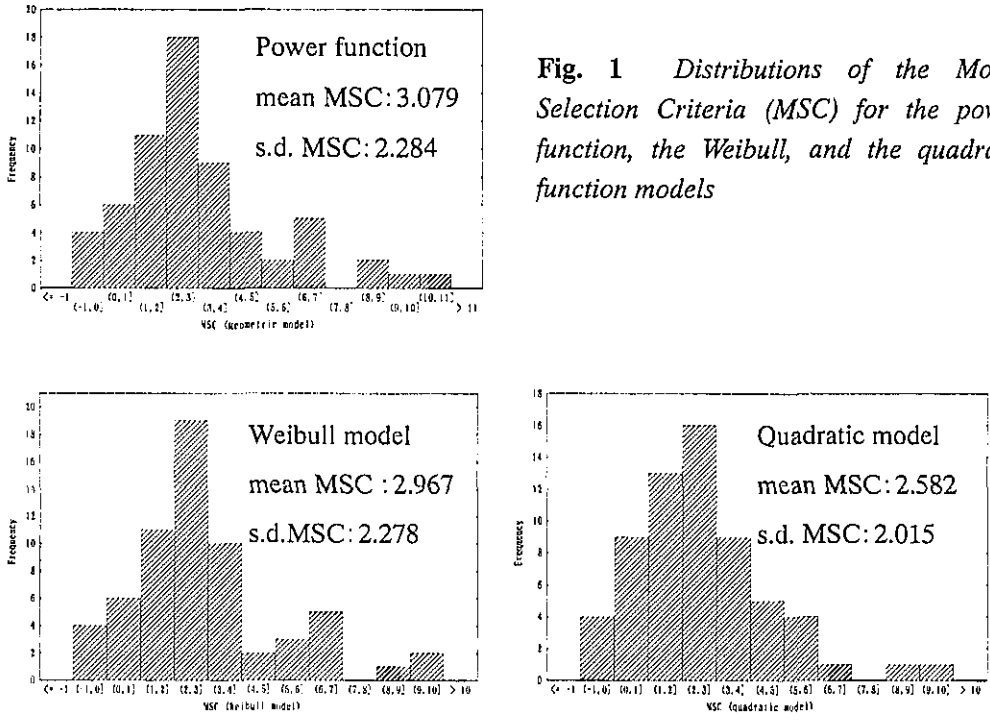


Fig. 1 *Distributions of the Model Selection Criteria (MSC) for the power function, the Weibull, and the quadratic function models*

Among the three models the power function model indicated the best fit, since the power function model produced a larger MSC score than the quadratic model (p<0.01, Wilcoxon-test), and a slightly larger one than the Weibull model (p<0.05, Wilcoxon-test). Thus the analyses will concentrate on the power function model.

The referenced data sets explored responses of various test organisms by various chemicals, and the test conditions may be considerably unequal between experiments. And properties of responses, especially the curvature, may be specific to organisms or chemicals. Nonetheless, it is convenient for risk estimation if there is a general trend in the population-level responses. In the next I roughly estimate the general β-value that approximates the whole data by the following procedure. (1) Exclude data sets that produced MSC scores smaller than 1.5 or larger than 8. (2) All data are

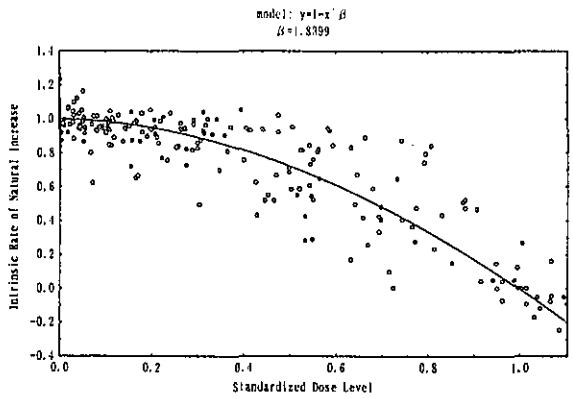


Fig. 2 *Plots of the standardized concentrations and responses.*

From the entire data set, the maximum likelihood β-value was estimated as β =1.84.

standardized with the maximum r and the α-value (exposure concentration at which r reduces to 0). Observed r is transformed to r/γ, and exposure concentration to x/α. (3) Determine the best-fit β-value based on the total data set. (4) Estimate the standard error and the distribution of β with the bootstrap resamplings (500 resamplings of 10 samples).

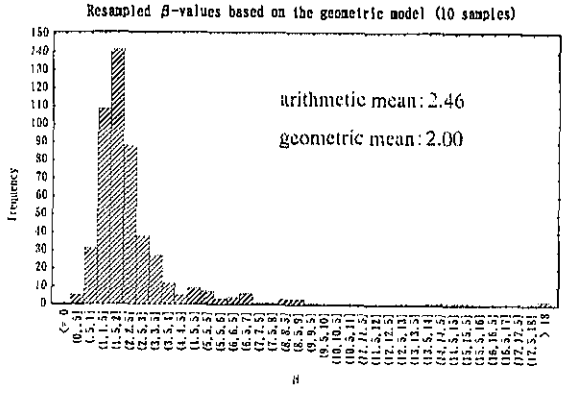


Fig. 3 *The distribution of β-values among resampled subsets of data (group size is 10 and 500 trials of resamplings)*

From the randomly resampled subsets of data, the arithmetic mean and the geometric mean of the β-value was estimated 2.46 and 2.00, respectively.

The standardized dose-r curve and maximum likelihood fitting to the power function model are presented in Fig.2. Both estimates are fairly compatible with the estimate based on the entire data set. The long but slight tail of β in the larger direction may reflect a few very improper resamplings and may result in overestimated β-values. As a whole the best empirical ground supports that the β-value in the power function model is approximately 2.

MET Risk on Populations by Individual Exposure

In this section I will demonstrate MET risk estimations for daphnia populations under the assumption that β-value is approximately 2 for all chemicals and species. Ideally, MET risk estimations need population-level data in terms of r for all chemicals and test organisms. However, only a minor proportion of chemicals and organisms is examined for the population-level responses. Then here I introduce an acute-chronic extrapolation. Among zooplanktons the regression slope of α-values to LC_{50}s is 1.34. The α-values are estimated from LC_{50}s with $\alpha = 1.34[LC_{50}]$.

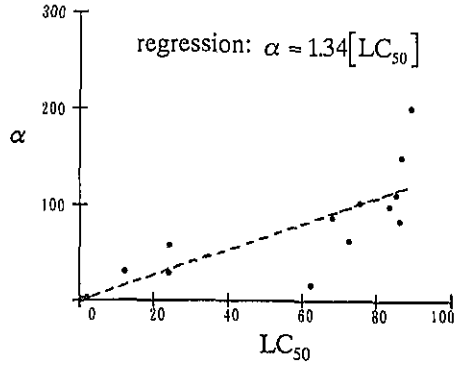


Fig.4. *Regression of α-values to LC_{50}*

From the scaling law, a small reduction of MET in logarithmic scale due to a reduction of r is calculated as $\Delta \log T = 2(\Delta r/v)\log N$, where N is the equilibrium population size. Putting $\beta = 2$ and $\alpha = 1.34[LC_{50}]$ into the power function model, we obtain $\Delta \log T = -1.114h^2(\gamma/v)\log N$, where h represents the exposure concentration relative to LC_{50}. The MET risk estimation by the above equation is based on two simplistic assumptions. First, there is no heterogeneity in the curvature of responses in r (an identical β-value) among chemicals and species. Second, the acute-chronic extrapolation is acceptable. Uncertainties due to the acute-chronic extrapolation and experiments are not taken account. Due to those limitations, the present results of MET analysis should be interpreted cautiously. The MET risks for some chemicals are exemplified in Table 2.

Table 2. *Extinction Risks of Agricultural Chemicals and Surfactants upon Daphnia Populations* (T: mean extinction time)

chemicals	max.conc. (ppb)	Daphnia LC_{50} (ppb)	$\Delta \log T^*$	$\Delta T/T(\%)$
malathon	4.5	13	−1.001E+01	100.00
pyridaphenthion	12	38	−8.332E+00	100.00
LAS	3000	5700	−2.314E+01	100.00
diazinon	2	7.8	−5.493E+00	100.00
nonylphenol	7.1	75	−7.488E−01	82.17
fenocarb	12	320	−1.175E−01	23.70
fenitrothion	0.2	9.2	−3.948E−02	8.691
benthiocarb	7	750	−7.278E−03	1.662
fenthion	0.05	5.5	−6.905E−03	1.577
mefenaset	8	1840	−1.579E−03	0.363
molinate	24	40000	−3.008E−05	0.00693
simetryn	9	27000	−9.283E−06	0.00214
pretyrachlor	6	26500	−4.283E−06	0.00099
butachlor	2	25000	−5.347E−07	0.00012

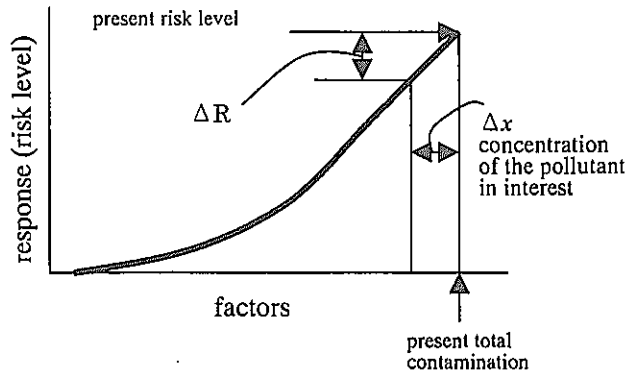
* $K=10^6$, $r_{max}=0.4$, $\nu=0.032$

The Topdown Risk Decomposition and Compound Exposure

In real circumstances, pollution may be caused from a number of chemicals, each of which exists at a very low environmental exposure concentration. The traditional way to estimate the risk of a specific chemical under compound exposure is to calculate the response to the chemical separately and combine all individual responses in order to estimate the total compound response. For example, the response addition assumes that the total response, $R_T(x)$, is calculated from products of residuals of responses to each chemical, $R_T(x)=1-\prod_i(1-R_i(x))$. Each response under low exposure is extrapolated from experimental data which were obtained under much higher exposure concentrations. Nonetheless, addition of the low-dose responses may not be combined to produce the true total effect because each response is predicted under non-contaminated laboratory environments without taking into account interaction between chemicals.

Here I present a hypothesis of compound effects of chemical pollutants and an alternative definition of risk, i.e., the reductional risk. The reductional risk of a pollutant is defined as the decrement of the entire risk due to all factors when the causal pollutant in interest is excluded from the environment. For simplicity, I illustrate it for the case where all pollutants follow an identical dose-response curve and they act interchangeably (Fig. 5).

Fig. 5. *Schematic Drawing of a Reductional Risk of a Pollutant*



From the viewpoint of toxicology, the dose-response relationship of a pollutant in interest is taken at the concentration that would bring about a response corresponding to the observed total risk level rather than at a very low concentration. This is based on the idea that effects of a number of pollutants accumulate rather than they act independently. Different pollutants are assumed to interact with each other with the same magnitude as a pollutant interact with itself (within conspecific molecules). Of course different chemicals may exhibit different curvatures of responses. And the entire response may be a complex function of compound effects of pollutants, $R_T(\boldsymbol{x})$, where $\boldsymbol{x}=(x_1\ x_2\ \ldots\ x_i)$ and x_i is concentration of the i-th pollutant. Nonetheless, if the above assumptions are met, the decrement of risk (the reductional risk, $\Delta R[x_i]$) may be estimated from the differential of the entire response to the exclusion of a pollutant, $\Delta R[x_i]= \partial R_T(\boldsymbol{x})/\partial x_i|_{\boldsymbol{x}}\ \Delta\ x_i$. And if a pollutant interact with other pollutants as well as with the identical pollutant, the differential may be equivalent to that of the response function of the pollutant in interest, $\Delta R[x_i]= \partial R_i(x_i)/\partial x_i|_{\boldsymbol{x}}\ \Delta\ x_i$.

Population Vulnerability Analysis of the Medaka-Plankton System under Chemical Pollution

Another complexity characteristic in the ecological risk assessment arises from interaction between species. The standard population vulnerability analysis (PVA), which focuses on extinction risk of a species, is not designed to detect such indirect effects through interaction between species. Nonetheless, the interspecific interactions can be partly incorporated into the PVA. Although the present model is highly generic and simplistic as regards composition of species, some essential properties of pollutant effects through interspecific interactions may be revealed by such an extended population dynamic model. I analyzed a three-species system, which comprises of a phytoplankton, and a zooplankton, and a small fish populations. The composite species are not exactly specified but I postulate the daphnia (*Daphnia* sp.) as the zooplankton and the medaka (*Oryzias latipes*) as the fish. The three trophic levels are cascaded as the fish predates the zooplankton, which in turn grazes the phytoplankton. And the fish has four life stages, i.e., adults, juveniles, larvae, and eggs. The zooplankton cannot reproduce nor sustain without the phytoplankton, the fish reduces survival rate and reproduction when they cannot predate the zooplankton. The effects of predation and grazing on reproduction of the predator and the grazer saturate as the food species increase (the Holling's type II functional response, May 1974). The effects of pollutants on reproduction of the phytoplankton and the zooplankton are assumed to follow the power function model with $\beta=2$. The responses of survival and reproduction of the fish are subject to the logistic model, $\exp(-\alpha+\beta x)/\{1+\exp(-\alpha+\beta x)\}$, with $\beta=2$. The α-value of the logistic model was determined with $\alpha=\beta\times LC_{50}$.

If we write the population density of the phytoplankton at time t as $n_p(t)$, that of the zooplankton as $n_z(t)$, and those of the four life stages of the fish as $n_{f1}(t)$ (egg), $n_{f2}(t)$ (larva), $n_{f3}(t)$ (juvenile) and $n_{f4}(t)$ (adult), the difference equation of the population dynamics is described as

$$\begin{bmatrix} n_p(t+1) \\ n_z(t+1) \\ n_{f1}(t+1) \\ n_{f2}(t+1) \\ n_{f3}(t+1) \\ n_{f4}(t+1) \end{bmatrix} = \begin{bmatrix} T_{1,1} & 0 & 0 & 0 & 0 & 0 \\ 0 & T_{2,2} & 0 & 0 & 0 & 0 \\ 0 & 0 & T_{3,3} & 0 & 0 & T_{3,6} \\ 0 & 0 & T_{4,3} & T_{4,4} & 0 & 0 \\ 0 & 0 & 0 & T_{5,4} & T_{5,5} & 0 \\ 0 & 0 & 0 & 0 & T_{6,5} & T_{6,6} \end{bmatrix} \cdot \begin{bmatrix} n_p(t) \\ n_z(t) \\ n_{f1}(t) \\ n_{f2}(t) \\ n_{f3}(t) \\ n_{f4}(t) \end{bmatrix}$$

where $T_{1,1} = \exp\left[r_p(1-m_p(x_t))\left\{1-\dfrac{n_p(t)}{K_p(1-m_p(x_t))}\right\}-D_p(t)\right]$, $T_{2,2}=\exp[I_z(t)(1-m_z(x_t))-D_z(t)]$,

$T_{3,3}=p_1(1-s_1)(1-m_{f1}(x_t))$, $T_{4,3}=p_1s_1(1-m_{f1}(x_t))$, $T_{4,4}=p_2(1-s_2)I_{f2}(t)(1-m_{f2}(x_t))$, $T_{5,4}=p_2s_2I_{f2}(t)(1-m_{f2}(x_t))$, $T_{5,5}=p_3(1-s_3)I_{f3}(t)(1-m_{f3}(x_t))$, $T_{6,5}=p_3s_3I_{f3}(t)(1-m_{f3}(x_t))$, $T_{6,6}=p_4I_{f4}(t)(1-m_{f4}(x_t))$ and $T_{3,6}=fI_{egg}(t)$.

The population growth of the phytoplankton and the zooplankton populations are denoted by $T_{1,1}$ and $T_{2,2}$. r and K are the intrinsic rate of population growth and the carrying capacity. I and D denotes the population growth by predating or grazing food species and the population decrease due to predation (see Boxes). The response to chemical pollutants are denoted as $m(x)$ in the whole equation. The survival and growth (transition to the next life stage) of eggs of the fish are denoted by $T_{3,3}$ and $T_{4,3}$, respectively. In the same way, the survival and growth of larvae and juveniles are denoted by $T_{4,4}$, $T_{5,4}$ and $T_{5,5}$, $T_{6,5}$. The adult survival and per capita fecundity are denoted by $T_{6,6}$ and $T_{3,6}$. f is the potential fecundity and I_{egg} denotes effects of foraging.

Box 1. The decrement of population growth rate due to predation or grazing.

$$D_p(t)=\frac{a_1n_z(t)}{1+h_1n_p(t)}$$

$$D_z(t)=\frac{a_2n_f(t)}{1+h_2n_f(t)}$$

a and h are parameters that express saturation of predation.

Here I demonstrate the population dynamics under exposure to diazinon. Toxicological experiments using *D.galeata* and *O.latipes* have provided that α=1.5 ppb for *D.galeata* and LC_{50}s for egg hatch, larval survival, juvenile survival, adult survival are 10 ppm, 5 ppm, 3 ppm and 6 ppm, respectively. The pesticide is not likely to affect phytoplanktons considerably. I tentatively assigned LC_{50}=100 ppm for the phytoplankton. For the environmental exposure data of diazinon, I employed the time-series measurements in Sakura River (NIES 1995). The exposure data ranged from early May to late September (approximately 150 days). In simulation I used the time-series exposure data cyclically without taking into account any phenological changes of ecological parameters except for the environmental exposure concentrations.

Box 2. The effect of predation or grazing on the population growth rate of the exploiting species.

$$I_z(t)=\frac{b_1n_p(t)}{1+h_1n_p(t)}-c_1$$

$$I_{f2}(t)=\frac{b_{f2}n_z(t)}{1+h_2n_z(t)}+c_{f2}$$

$$I_{f3}(t)=\frac{b_{f3}n_z(t)}{1+h_2n_z(t)}+c_{f3}$$

$$I_{f4}(t)=\frac{b_{f4}n_z(t)}{1+h_2n_z(t)}+c_{f4}$$

$$I_{egg}(t)=\frac{b_{egg}n_z(t)}{1+h_2n_z(t)}+c_{egg}$$

bs and hs are parameters that determine the saturating effects of predation. cs are the population growth (or decreasing) rate of the predators when the preys are absent.

The results of simulations are shown in Fig.6. The populations of the three species cyclically fluctuated with the pollutant although they approached stable equilibrium densities without the pollutant. The fluctuations synchronized with the environmental exposure concentration of diazinon. The direct effect of the pollutant is only effective to the zooplankton because the fish and the phytoplankton are tolerable to diazinon. Apparently the effects of the pollutant influenced population dynamics of the fish and the phytoplankton also enormously through interspecific interactions.

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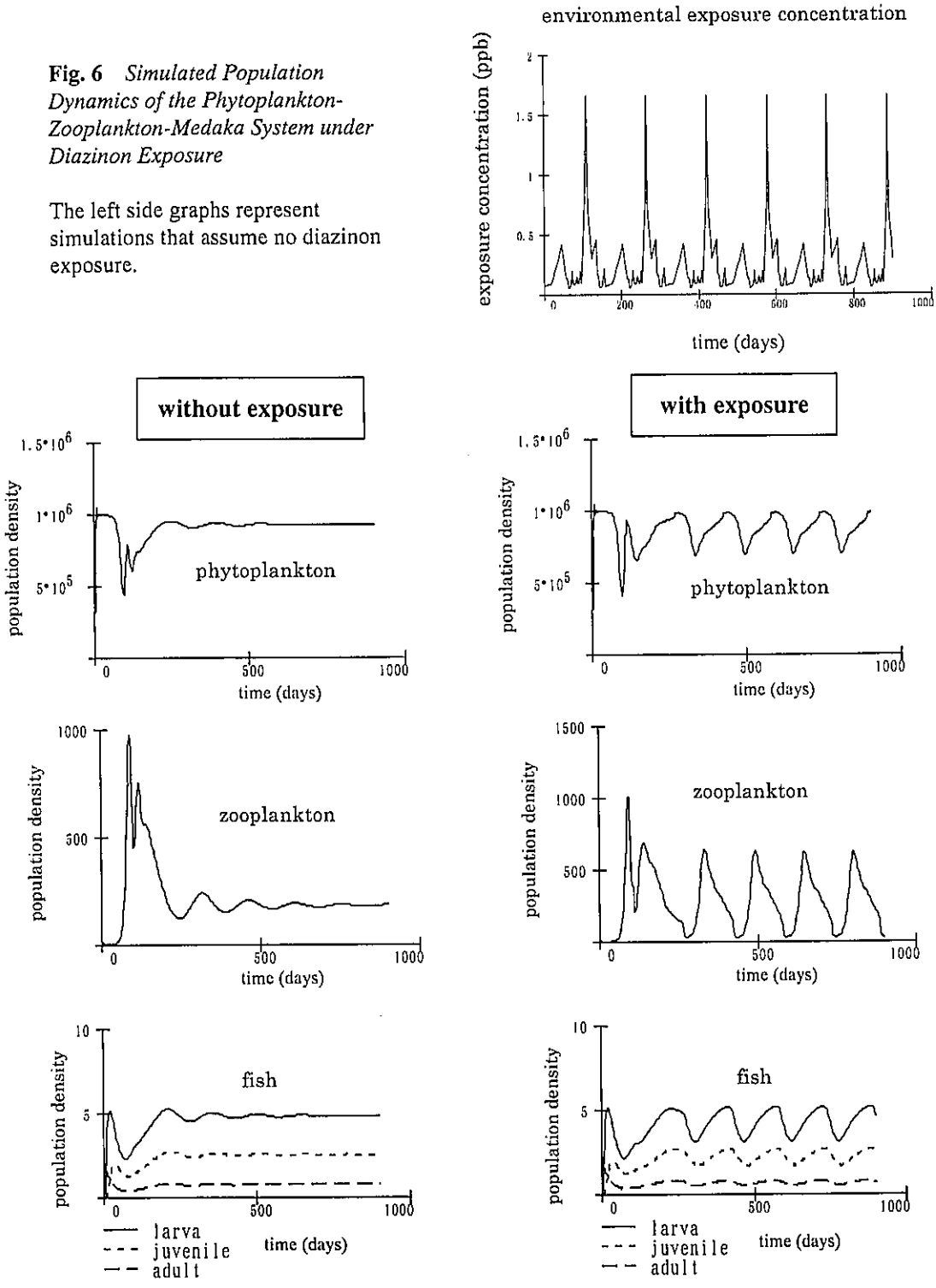

Fig. 6 *Simulated Population Dynamics of the Phytoplankton-Zooplankton-Medaka System under Diazinon Exposure*

The left side graphs represent simulations that assume no diazinon exposure.

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