Extinction Probability and the Ecological Risk Assessment

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Abstract

Ecological risk measured by populational extinction probability is calculated with extinction time models based on the diffusion theory and ecotoxicological experiments. The ecotoxicological data relevant for calculating extinction risk are mostly provided by life table evaluation and population growth experiments. Data are reviewed and analyzed for 47 combinations of test organisms and chemicals. It was suggested that the extinction risk due to exposure of ten percents of LC_{50} reduces mean extinction time by several percents or more while that due to one percent of LC_{50} reduces mean extinction time only by less than one percent.

1. Introduction

Estimation of extinction probability or mean extinction time may be relevant to the ecological risk assessment of chemical pollutants. This measure is commonly used in conservation biology and environmental science (Soule 1987; Caughley and Gunn 1996). With the extinction probability one can compare risks due to qualitatively different causes, e.g. destruction of habitats, over-hunting, chemical pollution, etc.

For calculating extinction risk of populations due to pollutants, population parameters that the mathematical models require for prediction of extinction probability must be estimated based on toxicological data and exposure analysis. Intrinsic rate of natural increase is the most important parameter because it is the end-product of all life history traits or behavioral characters that determines the capability of populations to propagate.

The present study reviews the ecological models that are relevant for extinction risk evaluation, and proposes that the life table evaluation is the most relevant toxicity test for applying the theoretical models. The life table evaluation consists of several life tables and reproduction tables describing age-specific fecundity and survival rate until each age class (Bertram and Hart 1979; Allan and Daniels 1982). Since intrinsic rates of natural increase can be determined from life tables, the intrinsic rates can be estimated as a response to exposure of chemicals if several life

table data are taken for different concentrations of the chemicals.

Provided that the effect of chemical pollutants to the intrinsic rate of increase is estimated, extinction risk corresponding to the decrements of the intrinsic rate can be calculated by mathematical models of extinction probability or time (Lande 1988, 1993; Foley 1994; Hakoyama and Iwasa [unpublished]). Published toxicological data by the life table evaluation are analyzed using a quadratic equation for the relationship between concentration and the intrinsic rate. Extinction risk is evaluated by decrements of mean extinction time due to exposure of pollutants.

2. Analytical Methods

2.1 *Mean Extinction Time Models*

Theoretical studies on extinction are based on application of the diffusion process and the branching process in the probability theory. The diffusion process is more suitable to stable environments where equilibrium population size is large and environmental fluctuation is small. Thus for calculating extinction risk of endangered species the branching process may be more suitable while for calculating that of a large stable population the diffusion process may be more suitable (Fig. 1). For ecological risk assessment of chemical pollutants in nearly stationary environments, e.g. lakes and forests, the diffusion approximation is the most realistic assumption. Throughout this paper we apply the diffusion process for calculating extinction risk of pollutants.

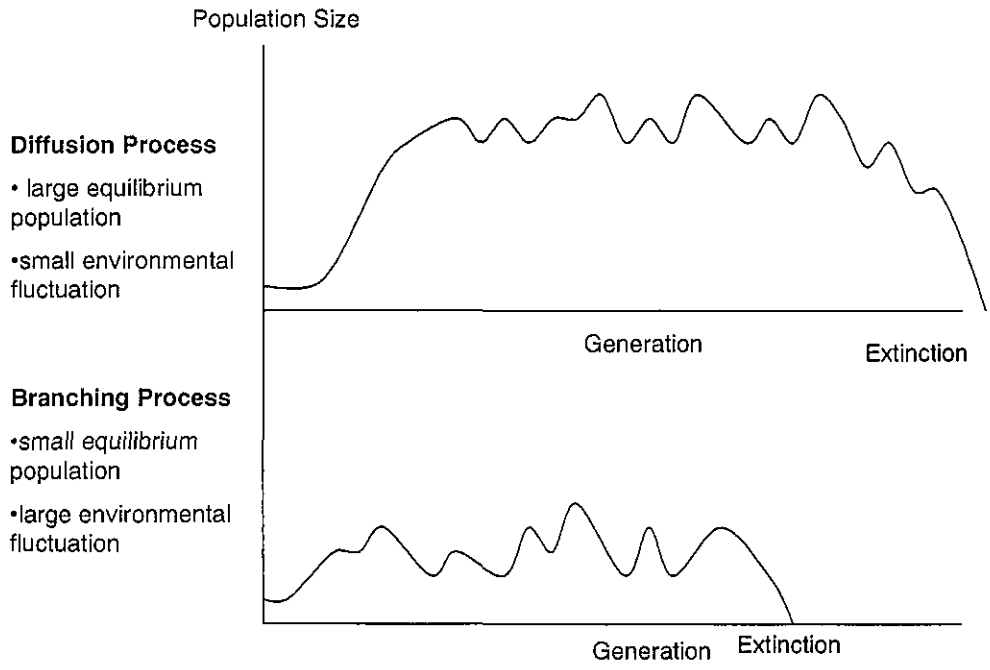

Fig.1. Schematic drawing of populational extinction subject to the diffusion process and the branching process

The mean extinction time of a population due to diffusion process is given by a solution of a

diffusion equation in which directional deviations of population size are expressed as the exponential growth: $dN/dt = rN + \varepsilon$ (N: population size, r: intrinsic rate of natural increase, ε: environmental fluctuation) and a reflecting boundary is assumed to be the carrying capacity K. Environmental variation of population growth rate due to stochastic fluctuations of external environmental factors (e.g. temperature, food quality and quantity, and predation pressure) is expressed by ε. The following paragraphs describe representative theoretical studies on this line.

1) *Lande's model*

Lande (1993) has derived a solution of mean extinction time as a stationary solution of a diffusion equation. His basic assumptions are density-independence of population growth except for neighborhood of the carrying capacity and small environmental fluctuation of population size so that the diffusion approximation is realistic. The mean extinction time T is

$$T = \frac{2}{vc}\left(\frac{K^c - 1}{c} - \ln K\right),$$

where K is the carrying capacity (maximum population size), v is the environmental variance of the growth rate, and $c = 2r/v - 1$.

2) *Foley's model*

Foley (1994) applied the analytical method for mean persistence time of populations developed in population genetics to mean extinction time of populations. The analytical solution for the mean extinction time is

$$T = \frac{1}{2sr}\left[e^{2sk}\left(1 - e^{-2sn_0}\right) - 2sn_0\right],$$

where n_0 is the initial population size in the logarithmic scale, k is the carrying capacity in the logarithmic scale, and $s = r/v$. The basic assumptions of the Foley's model are parallel to those of the Lande's model.

3) *Hakoyama and Iwasa's model*

Hakoyama and Iwasa (personal communication) have analyzed the mean extinction time of populations with a unique approach. They derived a diffusion equation from the logistic equation and solved the diffusion equation numerically. From numerical solutions based on various parameter values of intrinsic rate of increase (r), carrying capacity (K), and environmental variance (v), they estimated an empirical expression predicting the mean extinction time. The estimated expression of mean extinction time is

$$\log T = \left\{\frac{6.370}{\left(\sqrt{\frac{3}{r}}v\right)^{2.134}} + \frac{0.0365\sqrt{r}}{\sqrt{v}}\right\}\log\frac{rK}{3} - \frac{0.263r}{v} + \frac{2.907\sqrt{r}}{\sqrt{v}} - \log r + 0.0589$$

The expression is complicated and hardly intuitive. However, the assumptions are less limited in that the population growth is assumed to be density-dependent.

Comparison between the three models with various parameter values of r, K and ν may give some insight about precision of those models. Results are shown in Fig. 2. The major properties inferred from the figure are large differences in mean extinction times expected from the three models, and a fairly consistent tendency among the three models of mean extinction time in the logarithmic scale decreasing with the intrinsic rate of natural increase.

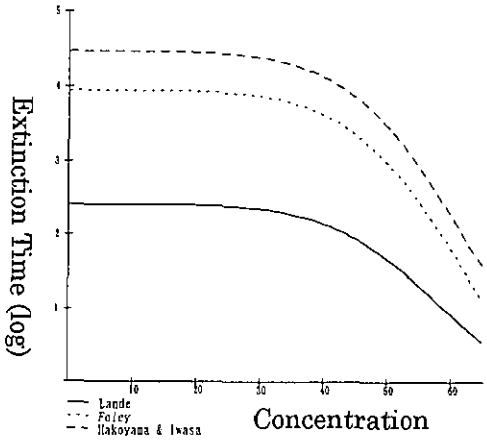

Fig. 2. Extinction time models

Among the three models Lande's model gave estimates of mean extinction times shorter than the other two models. Foley (1994) practiced some Monte Carlo simulations and showed good compatibility with the theoretical predictions. Hakoyama and Iwasa (personal communication) have also executed several simulations to check the robustness of their expression. Thus Lande's solution, which predicts extinction times a couple of orders shorter than those the other two models predict, may give underestimates of the mean extinction time.

Nonetheless, the rough tendency of the mean extinction time in the logarithmic scale to decrease as the intrinsic rate of natural increase decreases is fairly common among the models. Decrements of mean extinction time in logarithm ($\Delta \log T$) corresponding to decrements of the intrinsic rate (Δr) are in a fairly good agreement among the models. Therefore, it is considered to be feasible to evaluate extinction risk in terms of the decrements of mean extinction time in the logarithmic scale, in other words, proportional reduction in the mean extinction time.

Figure 3 shows interactions between decreases of the intrinsic rate of natural increase (or concentrations of chemicals) and carrying capacity to reduce mean extinction time. The mean extinction time decreases exponentially with K values (the vertical axis draws a log scale). The concentration- extinction time curves are nearly the same shape with different K values. This suggests that chemical pollution and a factor decreasing K values (e.g. destruction of habitats) affect the mean extinction time roughly independently. Decrements of log [extinction time] are nearly constant regardless of K values, suggesting that extinction risk measured as proportional reductions of extinction time does not depend on the assumption of equilibrium population size.

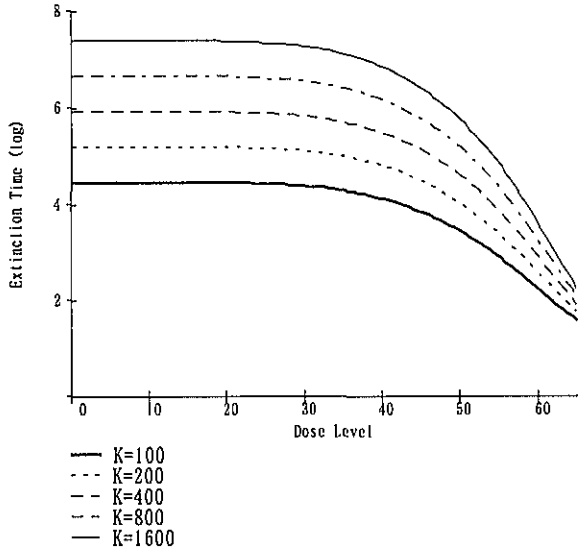

Fig. 3. Mean extinction time as a function of chemical exposure with various carrying capacities

2.2 *Life Table Evaluation and Dose-Response Function*

Ecotoxicological studies that estimated effects of chemical pollutants to organisms in terms of intrinsic rate of natural increase by life table evaluation or population growth experiments are listed in Table 1. However, the list is not perfect. Most of the listed studies estimated intrinsic rate of population increase for several different concentrations of chemicals by life tables or population growth experiments. Some studies need calculations using the Euler-Lotka equation $(1 = \sum_t \left(e^{-rt} m_t l_t\right))$ for estimating the intrinsic rate. From population growth experiments the intrinsic rate is determined by fitting data of population size across time to the logistic equation $(\frac{dN}{dt} = rN\left(1 - \frac{N}{K}\right))$ or exponential function $(\frac{dN}{dt} = rN)$. The fitting to functions are practiced by "generalized regression" of MathCadPlus (MathSoft).

Table 1 α- and β-values in the power function estimated from dose-r data

chemical	species	α (microgram/l)	β	reference
endosulfan	D.magna	2470	0.516	23)
chromium	Daphnia obtusa	109.55	13.23	20)
mercury	Mysidopsis bahia	1.58	2.59	26)
copper	D.magna	120.91	3	14)
copper	D.pulex	68.15	5.06	14)
copper	D.parvula	60	6.25	14)
copper	D.ambigua	68.47	5.41	14)
gamma radiation	D.pulex	69.96	1.99	29)

acid	D.pulex	2.792	2.67	36)
DDT(lab.culture)	Lepidodermella squammata	4.227	0.478	28)
DDT(conj.fiedl)	Lepidodermella squammata	0.69	0.288	28)
dieldrin	E.affinis	5.097	9.16	8)
dieldrin	D.pulex	201.59	29.6	8)
nickel	Mysidopsis bahia	116.42	1.19	25)
kepone	Eurytemora affinis	23.45	1.3	9)
copper (algal food)	D.magna	101.15	5.19	11)
copper (trout food)	D.magna	115.26	1.2	11)
fenvalerate	D.galeata mendotae	0.057	4.92	21)
cadmium	D.pulex	13.14	0.585	19)
metals (TU)	D.magna	3.4	2.98	22)
metals (WQC)	D.magna	1.178	1.42	22)
PCP	Brachionus rubens	0.227	6.192	27)
phenol	Brachionus rubens	44.702	0.846	27)
4-chloroaniline	Brachionus rubens	84.98	1.186	27)
4-nitrophenol	Brachionus rubens	14.92	0.329	27)
gamma radiation	D.pulex	397.4	8.661	30)
4-nitrophenol	D.magna	24110	3.073	12)
4-nitrophenol	D.magna	12090	9.83	12)
disulfiram	D.magna	24.6	3.37	35)
TMTU	D.magna	100060	2.78	35)
zineb	D.magna	221.13	1.18	35)
cadmium	D.magna	3.194	1.93	13)
cadmium	D.magna	30.98	4.82	13)
cadmium	D.magna	192.26	0.614	13)
cadmium	Chlorella pyrenoidosa	10520	0.652	13)
cadmium	Ctenodrilus serratus	4810	1.395	33)
chromium	Ctenodrilus serratus	646	2.365	33)
copper	Ctenodrilus serratus	250	1.617	33)
lead	Ctenodrilus serratus	3253	1.77	33)
mercury	Ctenodrilus serratus	100	1.33	33)
zinc	Ctenodrilus serratus	5266	3.19	33)
cadmium	Ophryotrocha diadema	3491	0.522	33)
chromium	Ophryotrocha diadema	675	1.476	33)
copper	Ophryotrocha diadema	183	1.51	33)
lead	Ophryotrocha diadema	451	0.164	33)
mercury	Ophryotrocha diadema	101	1.14	33)
zinc	Ophryotrocha diadema	936	1.451	33)

Each data set consists of estimates of intrinsic rate for a control and several concentrations of exposed chemicals. Here it is assumed that the concentration-intrinsic rate curve are approximated by a quadratic equation which follows, $r(x) = r_{\max}\left\{1-\left(\frac{x}{\alpha}\right)^2\right\}$, where x is

concentration of chemicals, r_{max} is the maximum intrinsic rate of natural increase (natural increase without exposure of chemicals), and α is a parameter representing the concentration of chemicals at which the intrinsic rate reduces to 0. Hence the α values express magnitude of toxicity of chemicals. The reason why the quadratic equation is chosen is that the best fitting of a more general power function, $r(x) = r_{max}\left\{1-\left(\frac{x}{\alpha}\right)^{\beta}\right\}$, to individual data sets produced a mean β value to be nearly 2. And the β values varied largely between data sets probably due to uncertainty of data. Then a fixed value of β (=2) may give the most accurate expression of dose-response curves.

Inferences or extrapolation with the quadratic equation representing dose-response curves are based on two assumptions; (1) continuity of the concentration-response curve, and (2) absence of thresholds for effects of pollutants to the intrinsic rate of natural increase. These assumptions, especially the latter, are important for estimating extinction risk due to low concentrations of chemicals at which the effects are not statistically significant in toxicological experiments.

Concentration-extinction time curves are numerically evaluated by substituting the quadratic concentration-intrinsic rate curves into the intrinsic rate of natural increase of the extinction time models. The intrinsic rate of natural increase varies between test organisms. In the present analysis the maximum intrinsic rate is standardized as 3, and the effects of pollutants to the intrinsic rate are measured by proportional reduction in the intrinsic rate. The reason why $r_{max} = 3$ is chosen is that Hakoyama and Iwasa's analysis assumes that the intrinsic rate is around 3.

As indicated from the models, predicted mean extinction times depend on relative magnitudes of r to v (environmental variation of r). The environmental variation of population growth rate is largely different between species and populations, and only a few field data are relevant to estimate r/v because it needs long-term field surveys. In the present analysis it is not feasible to take into account the species-specific differences of the environmental variation of population growth rate. As a hypothetical value of r/v, we employed r/v =1.2 for all test species.

3. Results

3.1 *Correlation between Effects of Chemical Pollutants on the Intrinsic Rate of Natural Increase and Acute Toxicity*

There is a significant correlation between LC_{50} and the α values in the quadratic concentration-r function for each data set. Some LC_{50} values were estimated by the same studies that estimated effects on intrinsic rates, but others were estimated by other independent works. Both LC_{50} values and α values are transformed into the logarithmic scale. The LC_{50} values and the α values are highly correlated (correlation coefficient: 0.86). The linear regression of α values to LC_{50} values was estimated as 0.86. It is suggested that the α values are predictable from LC_{50} to a certain

extent.

3.2 *Toxicant Effects on Mean Extinction Time*

Table 2 shows decrements of mean extinction time by exposure of certain concentrations of chemicals. In the table extinction risk due to concentration equivalent to 1/10 and 1/100 of LC_{50} are evaluated. The numbers in the table denote relative magnitudes of mean extinction time with exposure to that without exposure. For example 0.98 means that the exposure of chemical pollutant reduces the mean extinction time by two percents.

The extinction risk is different between species and chemicals. However, there are some general trends. Due to the exposure of 1/100 of LC_{50} the extinction time decreases at one percent in many cases. The mean decreasing rate by the (1/100) LC_{50} exposure is 0.022. This corresponds a decrease in the carrying capacity only by one percent. On the other hand, the exposure of 1/10 of LC_{50} decreases the mean extinction time by 10 or 20 percents in many cases. The mean decreasing rate by the (1/10) LC_{50} exposure is 0.288. This corresponds to about 18.2 percent reduction of the carrying capacity.

4. Discussion

It is widely recognized that chronic toxicity experiments are required in addition to acute tests for ecological risk assessment (Barnthouse and Suter 1986). The definition of "chronic toxicity" is not established and varies among researchers. Nonetheless, the general properties of chronic tests are 1) long duration of exposure, 2) various responses other than the adult short-term mortality, and 3) several life stages including early life stages. These properties of chronic tests intend to detect effects on survival of organisms' populations. In general, chronic toxicity is revealed at lower concentrations than acute toxicity because chronic tests include the most sensitive life stage and/or response. For example, for fish species chemical exposure to larval or juvenile fish is much more toxic than that to adult fish.

One of the most outspread method to utilize the chronic data for ecological risk assessment is to determine a benchmark for critical concentration of safety. The maximum acceptable toxicant concentration (MATC), which is the mean value of LOEC (lowest observed effect concentration) and NOEC (no observed effect concentration) is the most representative benchmark. However, both LOEC and NOEC are not more than statistical measures and do not have strong biological relevance.

Table 2. Results of extinction risk due to certain levels of chemical exposure

chemicals	species	LC50	alpha	$\Delta \log_{10} T$ (ΔK[%]) [LC50/10]	[LC50/100]
mercury	Mysidopsis bahia	3.5	1.46	0.356 (35.0)	0.9886 (0.435)
copper	Daphnia magna	86.5	150.5	0.942 (2.46)	0.9994 (0.025)
copper	D. pulex	86.0	84.1	0.828 (7.57)	0.9981 (0.079)
copper	D. parvula	72.0	63.1	0.791 (9.33)	0.9977 (0.098)
copper	D. ambigua	67.7	87.3	0.897 (4.43)	0.9989 (0.045)
DDT	Lepidodermella squammata (lab.culture)	5.0	4.77	0.820 (7.95)	0.9980 (0.083)
DDT	Lepidodermella squammata	5.0	3.21	0.646 (16.7)	0.9954 (0.183)
dieldrin	E. affinis	23.0	6.09	0.081 (64.9)	0.9750 (1.07)
nickel	M. bahia	508	148.6	0.126 (57.8)	0.9795 (0.878)
Kepone	E. affinis	40.0	23.1	0.582 (20.2)	0.9954 (0.226)
copper	D. magna (with algae)	85.1	111.5	0.899 (4.30)	0.9989 (0.044)
copper	D. magna	83.4	98.1	0.877 (5.30)	0.9987 (0.055)
cadmium	D. pulex	62.0	16.4	0.081 (65.0)	0.9750 (1.072)
metals (TU)	D. magna	1.8	3.42	0.951 (2.07)	0.9995 (0.021)
metals (WQC)	D. magna	0.62	1.18	0.951 (2.07)	0.9995 (0.021)
PCP	Brachionus rubens	0.16	0.313	0.953 (1.96)	0.9995 (0.02)
phenol	B. rubens	600	34.7	$1X10^{-7}$ (99.85)	0.5848 (20.0)
4-chloroaniline	B. rubens	100	81.7	0.764 (10.66)	0.9977 (0.113)
4-nitrophenol	B. rubens	6.3	6.23	0.832 (7.41)	0.9982 (0.077)
disulfiram	D. magna	12.0	30.5	0.973 (1.16)	0.9997 (0.012)
TMTU	D. magna	75000	101500	0.906 (4.03)	0.9990 (0.041)
zineb	D. magna	89.0	200.8	0.966 (1.47)	0.9997 (0.015)
cadmium	D. magna (semistatic water)	24.0	29.7	0.889 (4.79)	0.9988 (0.049)
cadmium	D. magna (intermittent flow)	24.0	57.2	0.968 (1.32)	0.9997 (0.013)
			mean	0.712 (18.24)	0.9781 (1.028)

Based on such benchmarks we may be able to make a qualitative conclusion on whether or not a specific environment is polluted by specific chemicals. However, in order to assess relative hazard that an environment suffers from specific chemicals we need more quantitative methods to analyze ecological risk. If we able to transfer hazard by chemical pollution into a more general criteria such as extinction probability (or time), we may quantitatively compare risks due to qualitatively different sources of environmental degradation (e.g. destruction of habitats and over-hunting). In conservation plans, wild species are classified into endangered groups according to a few criteria that are strongly associated with extinction risk.

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6. Reference

Allan, J. D. and Daniels R. E.: *Marine Biology* **66**, 179-184 (1982).
Barnthouse, L. W. and Suter II G. W. eds. : *User's Manual for Ecological Risk Assessment.* ORNL-6251 (Oak Ridge National Laboratory, Oak Ridge, TN, 1986).
Bertram, P. E. and Hart H. A.: *Environmental Pollution* **19**, 295-305 (1979).
Caughley, G. and Gunn A.: *Conservation Biology in Theory and Practice* (Blackwell Science, Cambridge, 1996).
Coniglio, L. and Baudo R.: *Hydrobiologia* **188**, 407-410 (1989).
Day, K. and Kaushik N. K.: *Environmental Pollution* **44**, 13-26 (1987).
Daniels, R. E. and Allan J. D.: *Can.J.Fish.Aqua.Sci.* **38**, 485-494 (1981).
Enserink, E. L., Maas-Diepeveen J. L. and Van Leeuwen C. J.: *Wat.Res.* **6**, 679-687 (1991).
Fernandez-Casalderrey,A., Ferrando M. D. and Andreu-Moliner E.: *Comp.Biochem.Physiol.* **106**, 437-441 (1993).
Gentile, J. H., Gentile S. M., Hairston, Jr. N. G., and Sullivan B. K.: *Hydrobiologia.* **93**, 179-187 (1982).
Gentile, J. H., Gentile S. M. , Hoffman G., Heltshe J. F. and Hairston, Jr. N.: *Env.Toxicol.Chem.* **2**, 61-68 (1983).
Halbach, U., Siebert M., Westermayer M. and Wissel C.: *Ecotoxicol.Env.Saf.* 7, 481-513 (1983).
Foley, P.: *Conservation Biology* **8**, 124-137 (1994).
Francis, P. C., Grothe D. W. and Scheuring J. C.: *Bull. Environ. Contam. Toxicol.* **36**, 730-737 (1986).
Hummon, W. D.: *Amer.Mid.Natur.* **92**, 327-339 (1973).
Lande, R.: *Science* **241**, 1455-1460 (1988).
Lande, R.: *Amer.Natur.* **142**, 911-927 (1993).
Marshall, J. S.: *Ecology* **43**, 598-607 (1962).
Marshall, J. S.: *Ecology* **47**, 561-571 (1966).
Marshall, J. S.: *J.Fish.Res.Board Can.* **35**, 461-469 (1978).
Reish, D. J. and Carr R. S.: *Marine Pollution Bulletin* **9**, 24-27 (1978).
Soule, M. E.: *Viable Populations for Conservation* (Cambridge University Press, Cambridge, England, 1987).
Van Leeuwen, C. J., Moberts F. and Niebeek G.: *Aquatic Toxicology* 7, 165-175 (1985a).
Van Leeuwen, C. J., Luttmer W. J. andGriffioen P. S.: *Ecotoxicol.Env.Saf.* **9**, 26-39 (1985b).
Walton, W. E., Compton S. M., Allan J. D. and Daniels R. E.: *Can.J.Zool.* **60**, 573-579 (1982).
Winner, R. W., Keeling T. , Yeager R. and Farrell M. P.: *Freshwater Biology* 7, 343-349 (1977).
Winner, R. W. and Farrell M. P.: *J.Fish.Res.Board Can.* **33**, 1685-1691 (1976).