Ecological Risk Assessment of Pollutant Chemicals: Extinction Risk Based on Population-Level Effects

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Abstract

The extinction probability or the mean extinction time is one of the most useful endpoints that are utilized in conservation biology. A parallel approach is advocated for the ecological risk assessment of chemical pollutants. The presented framework estimates extinction probability induced by pollutant chemicals in order to evaluate ecological hazards of pollution. The analytical framework, which is based on stochastic population dynamics theory, is briefly explained. The extinction risk estimation is feasible if ecotoxicological data concerning pollutant effects on population growth rate of organisms (the intrinsic rate of natural increase), and if environmental exposure concentration is provided. Tentative risk estimation was practiced for some agrochemicals and surfactants on zooplankton populations (*Daphnia*) as target organisms.

Introduction

Estimation of population-level effects of chemical pollutants is a basis of ecological risk assessment. Extinction risk of populations or species is one of the most universal criteria for measuring ecological hazard by qualitatively different factors, e.g., destruction of habitats, over-hunting and chemical pollution (Soule 1986; Suter 1993; Burgman et al. 1993; Caughley and Gunn, 1996). To discuss on the relevance of the extinction risk estimation in the context of ecological risk assessment is not the scope of this paper (but see Tanaka and Nakanishi [2000], Iwasa [2001]). Nonetheless, we should state that the present analysis is based on the perspective that application of the population vulnerability analysis (PVA), which is based on estimation of extinction probability, developed in conservation biology to the ecological risk assessment is one of the best ways to introduce ecology or conservation biology into environmental toxicology.

To evaluate the adverse effects of pollutant chemicals on population extinction, effects on the intrinsic rate of natural increase, r, must be estimated because r determines the probability of population extinction (Lande 1993; Foley 1994; Hakoyama and Iwasa 2000).

In quantitative risk evaluation of chemical pollutants, dose-response relationships should be based on fine empirical grounds. Some recent studies have stressed the importance of intrinsic rate of natural increase as an endpoint response to exposure by pollutant chemicals, and examined the relationships between the responses in r and acute effects (Calow et al. 1997; Walthall and Stark 1997; Forbes and Calow 1999; Roex et al. 2000). Nonetheless, as for the dose-response relationships in terms of r, very few statistical analyses based on mathematical models have been conducted (Tanaka

and Nakanishi 2001). A previous study has reviewed ecotoxicological data that estimated adverse effects of pollutant chemicals on *r*, and concluded that the power function model was one of the most relevant dose-response functions and the power was approximately 1.84 (Tanaka and Nakanishi 2001). We 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 pollutant chemicals, an ecological risk assessment is presented based on increments of extinction probability, which is equivalent to instantaneous rate of extinction and is approximately equal to the inverse of mean extinction time.

Analytical Methods

Mean Extinction Time and the Stochastic Model of Populations

The present framework is based on the analytical solutions for mean extinction time (MET) of the stochastic population dynamics model with the diffusion approximation (Lande 1993; Foley 1994; Hakoyama and Iwasa 2000; reviewed by Iwasa 2001). Under this approximation populations of organisms are subject to random Brownian motions due to several stochastic factors, i.e., environmental stochasticity, demographic stochasticity, and catastrophic events (e.g., forest fires and outbreaks of diseases). Even focusing on a single common factor, predictions of MET vary noticeably between theoretical models that are based on different mathematical assumptions. Nonetheless, the dependence of MET on demographic and environmental parameters are fairly compatible between models (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. Ecological risk of pollutant chemicals may be more appropriately estimated for moderately large populations than for endangered small populations because most extant populations subject to adverse effects of pollutants in nature are not endangered. Therefore, the environmental stochasticity may be the primary factor of extinction when we evaluate extinction risk due to pollutant chemicals at very low environmental exposure concentrations.

As a first-order approximation, 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 (carrying capacity), r the intrinsic rate of natural increase, 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. These relationships are not exactly deduced from the analytical solutions, but approximate well the scaling relationships between model parameters and mean extinction time (Lande 1998).

The adverse effects of pollutant chemicals are assumed to primarily reduce r, and the other parameters are supposed to be kept constant. The adverse effects may actually reduce the carrying capacity (maximum population size) as well (e.g., Enserink et al. 1991). Nonetheless, the mean extinction time and the extinction probability are much less sensitive to N than to r if the adverse effects of pollutant chemicals reduce r and N at the same rate. The decrement of MET in the logarithmic scale due to a small reduction of r is calculated as $\Delta \log T = 2(\Delta r/V_e)\log N$, where Δr is the change in r (a minus number as long as r decreases).

As a dose-response function for r, we employed a power function model since this model was one of the best models describing responses in r to exposure by pollutant chemicals (Tanaka and Nakanishi 2001; see below). The power function model depicts

a monotonically decreasing curve with an arbitrary curvature: $r(x) = r_{\max}\left[1-(x/\alpha)^{\beta}\right]$, where x is exposure concentration, $r_{\max}$ is the maximum r without exposure by chemicals, α and β are parameters (Tanaka and Nakanishi 2000, 2001). Respectively, the two parameters, α and β, are associated with the magnitude of toxicity and the curvature of responses in r to exposure concentration, x. The parameter α corresponds to the concentration at which r reduces to 0.

Extinction probability

The extinction probability, i.e., the instantaneous rate of extinction, may be a better unit of extinction risk assessment than the mean extinction time since the extinction probability is likely to linearly measure the hazard to populations. On the contrary, a ten-fold reduction of mean extinction time does not necessarily represent a ten-fold hazard for persistence of populations, rather a much less hazard.

Fortunately, it is relatively easy to transform a reduction of mean extinction time into an increase of extinction probability because it is approximately equal to the inverse of the mean extinction time if the instantaneous rate of extinction does not change with time.

If the mean extinction time expected before exposure is T_0 and that after exposure is T^*, the change of MET in the logarithmic scale is $\Delta\log T = \log T^* - \log T_0 = \log(T^*/T_0)$. The change in the extinction probability p due to exposure is $\Delta p = \Delta(1/T) = (1/T_0)(T_0/T^* - 1)$. Thus, substituting $T_0/T^* = 10^{-\Delta\log T}$ into Δp gives

$$\Delta p = p_0\left(10^{-\Delta\log T} - 1\right), \qquad (1)$$

where p_0 is the background extinction probability without exposure ($1/T_0$). If we

employ the power function model for the responses in r, the change in r due to exposure by a chemical with concentration x is $\Delta r = -(x/\alpha)^{\beta} r_{\max}$. Thus, MET is specified as

$$\Delta \log T = -(x/\alpha)^{\beta} (2r_{\max}/V_e) \log N \ . \qquad (2)$$

Putting equation (2) into equation (1), the increment of extinction probability can be estimated if the background extinction probability, p_0, is known. It is a difficult task to determine the background extinction probability. Nonetheless, if we accept an assumption, it can be specified as follow. Here it is assumed that the extinction probability becomes unity when r reduces to 0 (the exposure concentration is α) so that $\Delta p = 1 - p_0$ if $x = \alpha$,

where $p_0 = 10^{-(2r_{\max}/V_e)\log N}$ (3)

The background extinction risk varies with different target species because all of the composite parameters, r_m, V_e and N, depend on species. In the following risk estimation, we employ 10^{-6} as a background extinction probability. A couple of weak justifications are merely presented for employing this value. According to a population ecological study on a zooplankton (*Daphnia brachyurum*), the mean intrinsic rate of natural increase and the variance of the intrinsic rate were estimated as $r = 0.014$ and $V_e = 0.031$ (Hanazato and Yasuno 1985). Hence, $2\ r_m/V_e$ is approximately unity. The population size of 10^6 for typical wild lives is a likely value. According to fossil records life spans of many extinct species were about a million or ten million years (May et al. 1995). Thus the extinction rate per year is about 10^{-6} or 10^{-7} for typical species.

Population-Level Effects of Pollutant Chemicals on r

The intrinsic rate of natural increase, r, or the population growth rate is one of the most

important ecological parameters since it determines the persistence or the mean extinction time of populations as discussed above. The most complete experiments designed for estimating *r* are the life table evaluation and the population growth experiments (e.g., Winner et al. 1977; Allan and Daniels 1982; Gentile et al. 1983). Tanaka and Nakanishi (2001) have reviewed published data of dose-response relationships in terms of *r*, and examined relevance of some mathematical models to describe the responses. Three dose-response models, i.e., the power function model, the Weibull model, and the quadratic function model, were analyzed with 63 concentration-*r* relationships from 38 publications. The power function model indicated the best fit among the three models because the power function model produced a larger model selection criterion (Newman 1995) than the quadratic model ($p<0.01$, Wilcoxon-test), and a slightly larger one than the Weibull model ($p<0.05$, Wilcoxon-test).

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 the responses in *r* depict a common shape. Tanaka and Nakanishi (2001) have roughly estimated the general β, which approximated the standardized total data set, as 1.84. To find the general β all data were standardized with the maximum *r* and α (both *r* and α were scaled to unity). And the best-fit β was estimated by fitting the power function to the total (standardized) data set. Bootstrap resamplings from the standardized total data set produced a variance of β, which was compatible with the variance between actual data sets. This strongly suggests that the variation of β between data sets (experiments with different test

species or chemicals) can be mostly explained by random sampling errors.

Acute-Chronic Extrapolation

The extinction risk estimation need population-level data in terms of *r* for all chemicals and test organisms. However, only a minor proportion of chemicals and organisms of all possible combinations have been examined for the population-level responses. One way to supplement the scarcity of data is to utilize the acute-chronic extrapolation (Suter 1993; Forbes and Calow 1999; Roex et al. 2000). Recently, Roex et al. (2000) have rigorously analyzed toxicity data on *r* and compared those with acute toxicity data. They concluded that the acute-chronic ratio based on *r* was 14.4 on average but seriously influenced by species sensitivity rather than mode of action of chemicals.

Applying the power function model, the concentration-*r* relationships were summarized by two parameters α and β. We employed the general β (1.84), which was estimated from the entire data set under the assumption that chemical and species specific curvatures of responses in *r* were negligible. The other parameter α was estimated from the acute-chronic extrapolation. Among zooplanktons the regression of α to LC_{50} in the logarithmic scale was estimated as $\ln(\alpha) = 0.843\ln(x) + 0.446$ (The data source was provided by Tanaka and Nakanishi [2001]). We used this regression equation to predict α from LC_{50}.

Results

Tentative extinction risk estimations are exemplified for some agrochemicals and surfactants on *Daphnia* populations (Table 1). Only for a few chemicals among the listed ones the life table toxicity was examined (e.g., dodecilbenzene sulfonate [LAS_{12}]:

Tanaka and Nakanishi [2001b]). The acute-chronic extrapolation based on the above regression equation was applied to all of the chemicals including ones which life table toxicities were known so that uncertainties due to extrapolation were equalized between chemicals. A relevant analytical method to balance the uncertainties due to extrapolation and make the extinction risk to reflect deficiency of data has yet been explored.

The exposure concentrations were assumed to be constant and equal to the maximum environmental exposure concentrations that had been ever reported in Japan.

[Table 1 about here]

The estimated extinction risks (increments of extinction probability) are listed in a descending order in Table 1. Evidently, the extinction risk and the ecological hazard quotient (EHQ), which is a simple ratio of environmental exposure concentration to acute LC_{50}, had the same order. Nonetheless, the two quotients did not descend at the same rate. The extinction risk decreased at a much higher rate than the EHQ as the ranking order dropped. This implies that the ecological risk indicated by the extinction probability is much more concentrated into the most hazardous chemicals than that indicated by the EHQ.

Discussion

The basic framework for extinction risk estimation of pollutant chemicals has been presented and exemplified by a tentative application to agrochemicals with *Daphnia* populations as target species. In the context of ecological risk assessment (c.f., Power

and Dams 1997), the extinction risk estimation has some advantages that are lacking in other methods. First, it holds ecological principles although some simplifications are inevitable. The estimation of extinction probability (or time) is based on the ecological model that simulates population dynamics and extinction. The population-level effects of pollutant chemicals are completely embedded in a model parameter (the intrinsic rate of natural increase). Another merit of the extinction probability is that it may entail a probabilistic risk assessment since the extinction risk itself is a probabilistic concept. This is followed by possibilities that uncertainties of toxicity data and stochastic distribution of exposure concentrations will be included in the risk estimation.

In addition, one of the most important merits of the extinction risk estimation is the potential feasibility to compare ecological risks due to qualitatively different factors. Wildlife is endangered by many factors, e.g., destruction of habitats, over-hunting, introduction of exotic species, and pollution. Since the extinction risk estimation is the outcome of the population vulnerability analysis (PVA), which is the major analytical tool in the conservation biology, all of the above-mentioned risk factors can be examined with a common framework. This enables comparison of hazards due to different factors, e.g., destruction of habitats *vs*. pollution by chemicals.

Meanwhile, the present form of extinction risk analysis has some shortcomings. Validation of predictions is essential for improvement of ecological risk assessment. However, experimental tests or field observations of predicted extinction risk are hard to practice. The scarcity or complete lack of empirical validation of analytical frameworks may seriously restrict future elaboration of the extinction risk estimation.

Impact on ecosystems should be evaluated on the community level because species that compose a community or an ecosystem exhibit heterogeneous sensitivities to

chemicals. And indirect effects via interaction between species, e.g., prey-predator relationship, may bring about an important part of ecological hazard by chemical pollution (Bartell et al. 1992). The extinction risk analysis on a single population does not examine such indirect effects. Nonetheless, multi-species extension of the extinction risk estimation on a single species may present an analytical tool to estimate hazard to a community as a whole. Future work is needed.

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Vitae

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Table 1. Extinction risk estimation for some agrochemicals and surfactants on *Daphnia* populations.

Chemicals	MEEC[a]	LC50	Δp[b]	EHQ[c]
LAS_{12}[d]	3000	5700	4.00×10^{-4}	26.3
Pyridaphenthion	12	38	7.35×10^{-7}	15.8
Malathon	4.5	13	5.91×10^{-7}	17.2
Diazinon	2	7.8	2.58×10^{-7}	12.8
Nonylphenol	7.1	75	7.59×10^{-8}	4.67
Fenocarb	12	320	2.09×10^{-8}	1.88
Fenitrothion	0.2	9.2	2.58×10^{-9}	1.09
Benthiocarb	7	750	2.04×10^{-9}	0.467
Mefenaset	8	1840	6.46×10^{-10}	0.217
Fenthion	0.05	5.5	4.41×10^{-10}	0.455
Molinate	24	40000	3.86×10^{-11}	0.03
Simetryn	9	27000	1.18×10^{-11}	0.017
Pretyrachlor	6	26500	6.33×10^{-12}	0.011
Butachlor	2	25000	8.38×10^{-13}	0.004

[a] Maximum environmental exposure concentration (μg/L).

[b] Increased extinction probability by exposure.

[c] Ecological hazard quotient (MEEC/LC50).

[d] Linear alkylbenzene sulfonate (dodecilbenzene sulfonate).