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Hazard/Risk Assessment

MEAN EXTINCTION TIME OF POPULATIONS UNDER TOXICANT STRESS AND ECOLOGICAL RISK ASSESSMENT

YOSHINARI TANAKA*†‡ and JUNKO NAKANISHI†
†Institute of Environmental Science and Technology, Yokohama National University, Tokiwadai 79-7, Hodogaya-ku, Yokohama, Kanagawa 240-8501, Japan
‡Japan Science and Technology Corporation, Kawaguchi Center Building 1-8, Honcho 4-chrome, Kawaguchi City 332-0012, Japan

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Abstract—
Population-level effects of chemical pollutants are evaluated in terms of decrements of mean extinction time of populations. Analytical solutions of the mean extinction time based on the diffusion approximation were applied to published chronic ecotoxicological data provided from life table experiments or population growth experiments. Assuming a fairly large population (a million) with environmental fluctuation of an observed magnitude, chemical exposure with a concentration of 10% of LC50 is expected to cause, on average, an extinction risk of 16% reduction in the mean extinction time, which is equivalent to that induced by a 1.2% reduction of the population size (or habitat area). Although the ecological risk assessment based on mean extinction time has many limitations, it may present a possibility for interpreting the ecological risk of chemical pollutants in the context of population vulnerability.

Keywords—Extinction probability Ecological risk assessment Population-level effect Life table Population growth rate

INTRODUCTION

The ultimate goal of ecological risk assessment is to characterize and quantify hazards of pollutant chemicals to ecosystems so that the relative importance among chemicals in terms of hazard to ecosystems is evaluated. In order to quantify the ecological risk, it must be transferred into a universal and ultimate measure of risk.

The extinction probability or mean extinction time (MET) of populations is suggested here as a candidate for the risk unit relevant for such purposes. Further, MET is defined as an expected time to extinction of a population and is interchangeable with an instantaneous rate of extinction (p) of $p = 1/\text{MET}$ if the extinction rate does not depend on time. Since MET has a simple relationship with the extinction probability, many theoretical studies have investigated MET [1–3]. The major merit in estimating extinction risk of populations is the generality of the concept. This measure is commonly utilized for conservation of wildlife [1–3], and the extinction probability or MET may enable comparison between risks due to qualitatively different factors, e.g., destruction of habitat, overhunting, chemical pollution, etc. Other possible normalizations (e.g., population size, reproductive potential) do not measure ecological risks due to various factors with a common unit.

We focus on MET and propose an analytical procedure for estimating extinction risk measured as decrements of MET (referred to as MET risk hereafter) based on experimental data. Calculation of MET risk due to pollutants requires that population parameters necessary for mathematical models be estimated from toxicological and field-exposure data. Intrinsic rate of natural increase (population growth rate) is one of the most important parameters because it represents the net effect of adverse individual-level responses (survival, reproduction, behavior, etc.) to pollutant chemicals on the capability of a population to reproduce [4]. This new effect can originate in any life stage, although the relative contribution of the responses to the intrinsic rate depends on age and the category of responses [5]. Provided that the effect of pollutant chemicals on the intrinsic rate of natural increase is estimated, a MET risk corresponding to the decrement of the intrinsic rate can be estimated with mathematical models [6–9].

The present study reviews the ecological models that are relevant for MET estimation and proposes that life table evaluation is the most relevant toxicity test that can be applied to the theoretical models. Life table evaluation is a chronic test, designed to estimate age-specific fecundity and survival rate for each age class under controlled exposure concentrations of chemicals [10,11]. Since the intrinsic rate of natural increase can be estimated from life table data, the responses in terms of the intrinsic rate to exposure of chemicals are evaluated if several life tables are estimated under different concentrations of chemicals. Published toxicological data based on life table evaluation or on population growth experiments are reviewed and analyzed with a specific dose–response curve model. We employed a quadratic equation for the relationship between exposure concentrations and the intrinsic rate of natural increase.

Although the application of extinction probability models in ecology to ecotoxicological data has several limitations, e.g., scarcity of data, unequal test conditions, restrictive assumptions of the models, etc. [12,13], such a framework may unify the ecological risk assessment of pollutant chemicals and the population vulnerability analysis of conservation biology [1,3,14] and make the comparison of ecological risks resulting from qualitatively different causes (e.g., destruction of habitats, chemical pollution, and overhunting) feasible.

* To whom correspondence may be addressed (tanaka@kan.ynu.ac.jp).

ANALYTICAL MODELS

The MET models and the scaling law

Extinction is induced by several factors, which are theoretically categorized as environmental stochasticity, demographical stochasticity, and random catastrophic events [3,14]. Among these, environmental stochasticity is considered to be the major factor of extinction for moderately large, quasi-equilibrium populations. Most other extinction factors, except for the random catastrophic event, induce extinction only of very small populations. Extinction by environmental stochasticity is induced by occasional reductions of population size due to random fluctuations of environmental factors. The environmental factors include any kind of extrinsic factors that affect survival and reproduction of organisms, e.g., temperature, food level, and predation pressure. Effects of pollutant chemicals directly reduce the potential performance of organisms in survival and reproduction and interact with the extrinsic factors of the environment to induce extinction. For assessing the extinction risk of pollutant chemicals on common plankton species, the extinction scenario by environmental stochasticity is most plausible, provided that the impact of the pollutant to populations is not strong and the target populations for risk assessment are not abruptly endangered.

Theoretical studies on extinction by environmental stochasticity are based on the diffusion approximation for random perturbations of population size. The diffusion approximation is accurate when the population size is large and the fluctuation in population size per generation is small [15]. If ecological risk assessment of pollutant chemicals postulates nearly constant exposure of chemicals under stationary environments, the diffusion approximation is the most realistic assumption. Occasional strong pulse exposure of pollutant chemicals or small population size may need alternative analytical approaches, such as the Leslie matrix simulation [16] and the branching process model [17], especially for site-specific risk assessment. Throughout this paper, we employ the diffusion approximation for calculating the MET risk of pollutants. Our purpose is to present a generic framework of the ecological risk assessment.

The mean extinction time of a population is given by the solution of a diffusion equation in which a population is assumed to grow exponentially and the carrying capacity (maximum population size) is dealt with as a reflecting boundary for population size. External environmental factors (e.g., temperature, food quality and quantity, and predation pressure) are assumed to generate stochastic fluctuations of the population growth rate. The environmental variance of the population growth rate is assumed to be small so that it meets the diffusion process of the population size. Thus, the diffusion model envisages a stationary population, in which the population size fluctuates around the equilibrium size (carrying capacity) and extinction accidentally occurs due to misfortune of repeated bad environments. For populations that are monotonically declining, an alternative mathematical approach based on simulation with a matrix model is often employed. For population vulnerability analysis of endangered species, the latter approach is appropriate. Nonetheless, the diffusion model is more realistic for ecological risk assessment of pollutant chemicals on quasi-equilibrium populations like zooplankton.

We will focus on three analytical models, derived with the diffusion approximation by Lande [7], Foley [8], and Hakoyama and Iwasa [9], to demonstrate major properties of the MET. Two analytical solutions are listed in Table 1. Lande [7] has derived a solution of mean extinction time as a stationary solution of a diffusion equation. His basic assumption is that the population growth rate is density independent as long as the population size is smaller than the carrying capacity and that environmental fluctuation of the population size is small so that the diffusion approximation is realistic. Foley [8] applied the mean persistence time model developed in population genetics to solve the mean extinction time of populations [18]. The basic assumptions of Foley's model are almost the same as those of Lande's model. Hakoyama and Iwasa [9] have analyzed MET with a unique approach. They derived a diffusion equation from the logistic equation and integrated the diffusion equation numerically. From numerical solutions based on various parameter values of r, K, and v (the environmental variance of r), they derived an empirical formula for MET.

Table 1. Mean extinction time models based on diffusion approximation[a]

Model	Mean extinction time
Lande [7]	$\frac{2}{v(2s-1)}\{(K^{2s-1}-1)/(2s-1)-\ln K\}$
Foley [8]	$\frac{1}{2sr}(e^{2s\ln K}-2s\ln K-1)$

[a] $s = r/v$; v = environmental variance of r.

Numerical comparisons of expected METs between the three models with various parameter values of r, K, and v indicate large differences in absolute values of expected METs (data not shown). Nonetheless, there is a fairly consistent tendency among the models that MET decreases with the intrinsic rate of natural increase (Fig. 1).

Among the three models, Lande's gave estimates for MET considerably shorter than the other two models (Fig. 1). Foley [8] conducted some Monte Carlo simulations and showed good accordance with the theoretical predictions. Hakoyama and

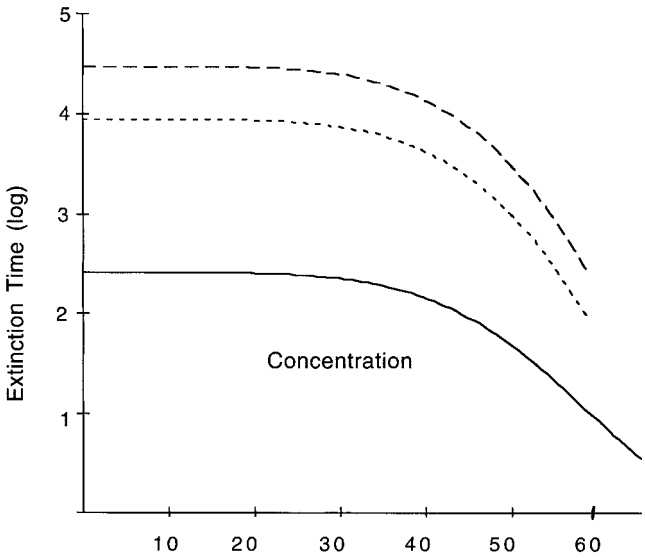

Fig. 1. Comparison among the predictions of the extinction time models of ———, Lande [7], ---------, Foley [8], and – – – –, Hakoyama and Iwasa [9].

Iwasa [9] have also undertaken some simulations to check the robustness of their analytic predictions. Thus, Lande's solution, which predicts MET a couple of orders shorter than those the other two models predict, may give underestimates of the mean extinction time. Nonetheless, the shape of the curve that represents how MET decreases with the intrinsic rate of natural increase (or *s*-value, which is defined as r/v) is similar for all three models (Fig. 1).

Lande [19] reviewed theoretical works on population extinction and proposed a scaling law as a first-order approximation for relationships between MET and demographic and environmental parameters. This law states that MET due to environmental stochasticity is approximately proportional to $K^{2r/v-1}$. Hence, MET increases geometrically with r and is proportionate to powers of K. Transforming both sides of the scaling law into logarithms, we get $\log T = C + (2s - 1)\log K$, where C is a constant.

Some numerical evaluations of the above-mentioned analytical solutions and the scaling law may reveal the relative precision of the scaling law in comparison with the exact solutions of MET (Fig. 2a and b). Figure 2a shows how decrements of log MET ($\Delta \log T$) change in response to decreases of r/v. The predictions of analytical solutions are highly comparable with each other, and the scaling law precisely approximates the analytical solutions. On the other hand, when responses of $\Delta \log T$ to proportional reductions of the equilibrium population size K were considered, the three analytical solutions gave more diverse results than they did when $\Delta \log T$ was compared with r/v (Fig. 2b). The scaling law and Lande's [7] analytical solution gave nearly the same results.

Thus, decrements of MET, in logarithmic form ($\Delta \log T$), corresponding to decrements of the intrinsic rate (Δr) are in fairly good agreement among the exact models, and the scaling law is a good approximation of the exact analytical solutions as a first-order approximation. Since most adverse effects of pollutant chemicals are likely to reduce r rather than K [10, 11, 20, 21], it is feasible to utilize the scaling law for evaluation of the MET risk in terms of $\Delta \log T$. We employed the scaling law for MET analysis because of its relative precision and the simplicity of the mathematical expression.

Provided initial equilibrium values of the population size (carrying capacity) can be hypothesized and the initial population growth rate relative to the environmental variance is large, small changes in s and $\log K$ can be expanded in a bivariate Taylor series around the initial values of s and $\log K$, i.e., $\Delta \log T \cong 2 \log \tilde{K} \times \Delta s + (2\tilde{s} - 1) \times \Delta \log K + 2\Delta s \Delta \log K$, where Δs and $\Delta \log K$ are small deviations from the initial values. Adverse effects of pollutant chemicals on MET may therefore be decomposed into the two fractions Δs and $\Delta \log K$ if they are small. Some ecotoxicological experiments have shown that adverse effects of pollutant chemicals primarily reduce the population growth rate [10,11,20,21], although a few experiments have suggested significant effects on the carrying capacity as well [22–24]. In nature, population size is often limited by conspecific competition resulting from limited food or habitat quality and quantity [25] and, in some cases, by predation or other density-independent effects. Density-perturbation experiments have frequently detected the density dependence [26,27]. If adverse effects of chemicals reduce the carrying capacity at the same proportion as they reduce the intrinsic rate of natural increase, the reduction of $\log K$ is expected to be much smaller than that of r, i.e., $\Delta \log K = \Delta \log(1 - R(x)) \ll \tilde{r}(1 - R(x)) = \Delta r$, where $R(x)$ is the response to exposure concentration x. In addition, in the Taylor series expansion of $\Delta \log T$, the coefficient in the first term is much larger than that in the second term, i.e., $2 \log \tilde{K}/v \gg 2\tilde{s} - 1$ since $\log \tilde{K} \gg \tilde{r} - v/2$; hence, the adverse effects on the carrying capacity are likely to be negligible. Thus, the major population-level effects of pollutant chemicals may reduce the *s*-values. The simplest form of extinction risk due to pollutant chemicals is $\Delta \log T \cong 2 \log \tilde{K} \times \Delta s$. A loss of log MET is roughly estimated by a decrement of the *s*-value, i.e., relative magnitude of the intrinsic population growth rate to the environmental variance in growth rate.

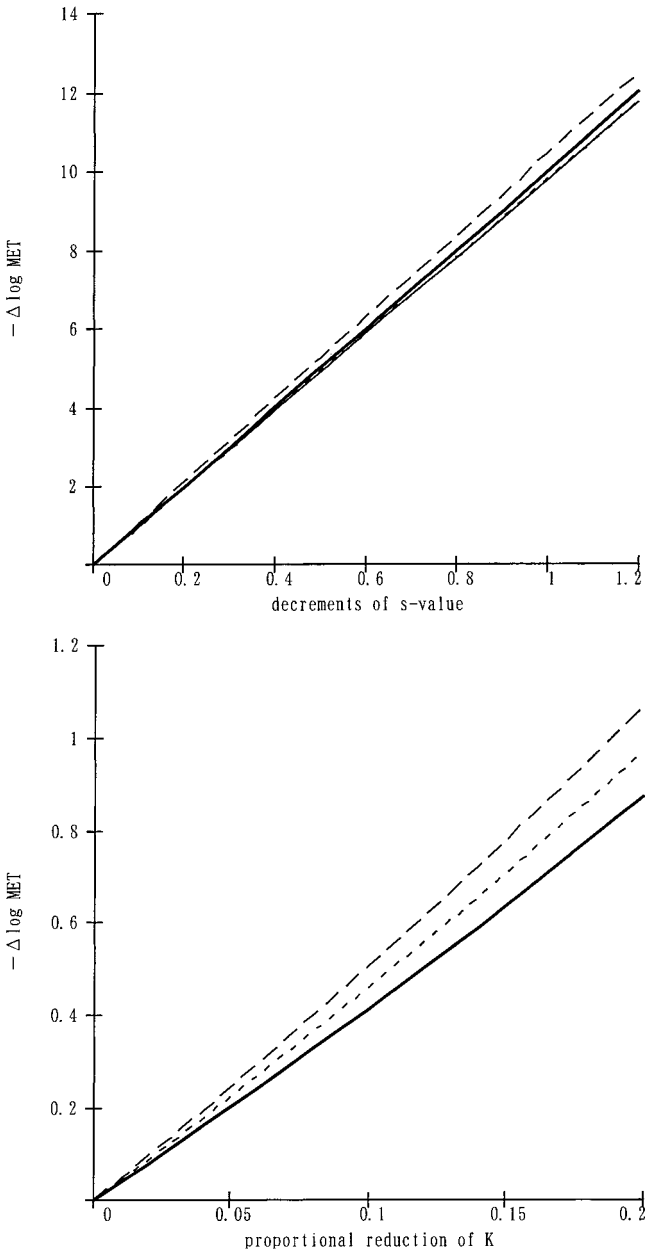

Fig. 2. (**a**) Comparison among models of reductions of mean extinction time, on the logarithmic scale, due to decreases in the magnitude of the intrinsic rate of natural increase relative to the environmental variance, r/v. Initial parameter values were $r_{max} = 3$, $v = 0.6$, $K = 10^5$. ———, Lande [7]; ---------, Foley [8]; – – – –, Hakoyama and Iwasa [9]; bold, solid line, scaling Law. (**b**) Comparison among models of reductions of mean extinction time, on the logarithmic scale, due to decreases in the carrying capacity. Initial parameter values were $r_{max} = 3$, $v = 0.6$, $K = 10^5$.

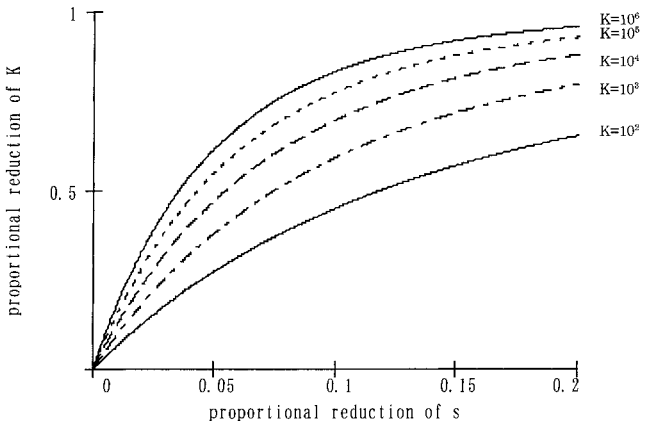

Fig. 3. Lines of equivalent extinction risk from the proportional reduction of the equilibrium population size (carrying capacity) and the proportional reduction of intrinsic rate of natural increase ($s = r/v$) for different carrying capacities.

Setting the left-hand side of the bivariate Taylor series to zero, i.e., $\Delta \log T = 0$, the decrement of the equilibrium population size inducing a MET risk equivalent to that induced by a decrement of s is evaluated from

$$\Delta \log K \cong \Delta s \log \tilde{K} \Big/ \left(\tilde{s} + \Delta s - \frac{1}{2}\right).$$

A MET risk that is caused by a reduction in s or by a reduction in the intrinsic rate of natural increase r can be translated into a corresponding reduction of the initial population size (or the carrying capacity K) of equivalent cost. If reductions of these population parameters are associated with specific risk factors, e.g., chemical pollution reduces r rather than K (or as well as K) while destruction of habitat reduces K rather than r, the qualitatively different ecological risk factors may be quantitatively compared.

Figure 3 plots decrements of log K corresponding to decrements of s-values with an equivalent MET risk. Exposure to a constant level of pollutant chemicals is likely to reduce r and s-values. Fluctuation of exposure concentrations may contribute to the variance v of the population growth rate and further reduce s-values (see Discussion).

Life table evaluation and dose–response function

In order to estimate MET risks, we analyzed chronic toxicity data provided from toxicological experiments that evaluated effects of pollutant chemicals on the intrinsic rate of natural increase (Table 2). The studies listed are not a comprehensive set. Most of the studies listed used life table or population growth experiments to estimate the intrinsic rate of natural increase under several different exposure concentrations of chemicals. Some studies needed reanalysis to estimate r using the Euler–Lotka equation ($1 = \Sigma_t [e^{-rt} m_t l_t]$, where m_t and l_t are age-specific fecundity and longevity, respectively). From the population growth experiments, the intrinsic rate was determined by fitting changes in population size with time to the logistic equation, $dN/dt = rN(1 - N/K)$, or to the exponential function, $dN/dt = rN$, where N is the population size and K is the carrying capacity (maximum population size). The maximum likelihood estimates are found using generalized nonlinear regression (Mathcad Plus 6.0, Math Soft, Seattle, WA, USA).

Each data set provided estimates of r for a control and several exposure concentrations of pollutant chemicals. We assumed that the concentration-r curve is approximated by a quadratic equation, $r(s) = r(0)[1 - (x/\alpha)^2]$, where x is the exposure concentration of a chemical, $r(x)$ is the intrinsic rate of natural increase under exposure concentration x, and α is

Table 2. Predicted reductions in mean extinction time (MET risk) estimated from chronic toxicological data among planktons

Test species	Chemicals	LC50	α[b]	ΔT% (ΔK%)[a]	
				LC50/10	LC50/100
Daphnia pulex	Cadmium	62.0	16.4 [10]	69.4 (6.07)	1.18 (0.062)
Eurytemora affinis	Kepone	40.0	23.1 [11]	22.0 (1.30)	0.25 (0.013)
E. affinis	Dieldrin	23.0	6.1 [20]	69.2 (6.04)	1.17 (0.062)
D. magna	Copper	85.1	111.5 [21]	4.7 (0.25)	0.05 (0.003)
D. magna	Copper	83.4	98.1 [21]	5.8 (0.32)	0.06 (0.003)
Brachionus rubens	PCP	0.2	0.3 [22]	3.6 (0.19)	0.04 (0.002)
B. rubens	4-Chloroaniline	100.0	81.7 [22]	11.7 (0.65)	0.12 (0.007)
B. rubens	4-Nitrophenol	6.3	6.2 [22]	8.2 (0.45)	0.09 (0.005)
D. magna	Disulfiram	12.0	30.5 [23]	1.3 (0.07)	0.01 (0.001)
D. magna	TMTU	75,000	101,500 [23]	4.4 (0.12)	0.05 (0.002)
D. magna	Zineb	89.0	200.8 [23]	1.6 (0.09)	0.02 (0.001)
D. magna	Cadmium	24.0	29.7 [24]	5.3 (0.29)	0.05 (0.003)
D. magna	Cadmium	24.0	57.2 [24]	1.5 (0.08)	0.01 (0.001)
Myopsis bahia	Mercury	3.5	1.46 [34]	36.3 (2.35)	0.45 (0.024)
D. magna	Copper	86.5	150.5 [35]	2.7 (0.14)	0.03 (0.001)
D. pulex	Copper	86.0	84.1 [35]	8.3 (0.46)	0.09 (0.005)
D. parvula	Copper	72.0	63.1 [35]	10.2 (0.57)	0.11 (0.006)
D. ambigua	Copper	67.7	87.3 [35]	4.9 (0.26)	0.05 (0.003)
Lepidodermella squammata	DDT	5.0	4.77 [36]	8.6 (0.47)	0.09 (0.005)
L. squammata	DDT	5.0	3.2 [36]	18.3 (1.06)	0.20 (0.011)
M. bahia	Nickel	508.0	148.6 [37]	62.0 (4.99)	0.96 (0.051)
D. magna	Metals (TU)	1.8	3.4 [38]	2.3 (0.12)	0.02 (0.001)
D. magna	Metals (WQC)	0.6	1.2 [38]	2.1 (0.11)	0.02 (0.001)

[a] $\Delta T\%$ = percent reduction of mean extinction time ($|\Delta T|/T \times 100$); $\Delta K\%$ = percent reduction of equilibrium K values that would cause the same level of extinction risk; parameter values: $K = 10^6$, $r_{max} = 0.3$, and $v = 0.03$; values in parentheses denote the corresponding percent reduction of carrying capacity that would cause the equivalent MET risk to that estimated.

[b] Data source.

a model parameter representing the concentration of a chemical at which r reduces to zero (where the population ceases growing). Thus, α-values represent the magnitude of the toxicity. The quadratic equation can be regarded as a special case of the power function $r(x) = r(0)[1 - (x/\alpha)^{\beta}]$. The parameter β determines the curvature of the response curve. Applying this power function model to all data sets, estimates of β varied greatly between data sets due to the uncertainty of the data (geometric mean, 2.2; standard deviation, 8.4 among the data sets), and extrapolated predictions of responses to very low exposure concentration were too sensitive to variation in the β-values. We estimated the general β-value by fitting $1 - y^{\beta}$, where y is the standardized concentration (x/α), to the entire data set, which was standardized so that $\alpha \rightarrow 1$ and $r\ (0) \rightarrow 1$, as 1.8. On these grounds, we employed the quadratic model ($\beta = 2$) as an extrapolation model for low-dose responses under low-concentration exposure. The reduction of MET, $\Delta \log T$, was numerically evaluated by applying the quadratic model to Δr in $\Delta \log T \cong 2 \log \tilde{K} \times \Delta s$.

Inference and extrapolation using the quadratic model are based on two assumptions, i.e., continuity of the concentration–response curve and the absence of thresholds for effects of pollutants to the intrinsic rate of natural increase. These assumptions, especially the latter, are important in estimating the MET risk at low concentrations of chemicals, i.e., concentrations at which the effect is not statistically significant in toxicological experiments.

The intrinsic rate of natural increase varies between test organisms. A survey on population dynamics of a cladoceran zooplankton (*Diaphanosoma brachyurum*) in Lake Kasumigaura provided estimates around $r = 0.3$ and $v = 0.03$ [28]. The present analysis employed these values. This field estimate of the intrinsic rate of natural increase is compatible with some estimates obtained from laboratory experiments for *Daphnia* [10,11,20,21]. Although r/v-values vary considerably between different higher taxonomic groups, these values may roughly represent cladoceran zooplankton species.

NUMERICAL RESULTS

Toxicant effects on MET and ecological risk assessment

Table 2 shows predicted percent reductions of MET due to chemical exposure to concentrations equivalent to 1/10 and 1/100 of LC50s. The figures in the table denote proportional reduction in percent of MET due to exposure to pollutant chemicals ($|\Delta T|/T \times 100$). These values were calculated from $\Delta \log T \cong 2 \log \tilde{K} \times \Delta s$ and $|\Delta T|/T = 1 - 10^{\Delta \log T}$. The figures in parentheses indicate percent reduction of the carrying capacity (or the equilibrium population size) that would cause reductions in the MET equivalent to those induced by the pollutant chemicals (exposure concentrations of LC50/10 and LC50/100). In response to an exposure of LC50/100, the MET rarely decreased by more than 1% (the mean decreasing rate is 0.22%). The same amount of reduction in the MET would result from a reduction of the carrying capacity only by 0.012% on average if the equilibrium population size (carrying capacity) is 10^6. On the other hand, the exposure of LC50/10 reduced the MET by more than several percent in many cases (the mean decreasing rate is 15.8%). This corresponds to about 1.15% reduction of the carrying capacity on average if the equilibrium population size is 10^6. With a smaller population size, the converted ΔK values are even smaller because a further reduction of population size in a small population entails larger extinction risk than in a large population (Fig. 3).

DISCUSSION

We examined the utility of mean extinction time (MET) for ecological risk assessment of pollutant chemicals. Although estimation of MET reductions due to adverse effects of toxicants has practical limitations (discussed below), its biological or ecological significance is clearer than traditional benchmarks or indices for ecological risk such as maximum acceptable toxicant concentration, which is often defined as the geometric mean of no-observed effect level and lowest observed effect level, and the ecological hazard quotient, which is calculated as a proportion of the environmental exposure concentration (EEC) to the acute toxicity LC50, i.e., EEC/LC50. Another merit of the MET risk analysis is that it may evaluate ecological hazard quantitatively. The extinction risk measured as a decrement of the MET in the logarithmic scale ($\Delta \log T$) is not linearly related to concentrations of chemicals. If α-values are linearly related to LC50s and the intrinsic rate of natural increase follows the quadratic function, the reduction of logarithmic MET is approximated by a quadratic function of exposure concentrations, $\Delta \log T \cong -2h^2k^{-2}r_{\max}v^{-1}\log K$, where k is the regression slope of α to LC50 ($\alpha = k$[LC50]) and h is the relative exposure concentration to LC50. The reduction of logarithmic MET is roughly proportional to the squared relative exposure concentration, h^2, and thus $|\Delta \log T|$ increases more than linearly as the exposure concentration increases. If the ecological hazard is quantified by $\Delta \log T$, the simple proportion of EEC to LC50 may not represent relative magnitudes of ecological hazards. It may underestimate ecological risk caused by high EEC.

The present analysis suggests that exposure to a relatively high concentration (between LC50/100 and LC50/10) induces only a small extinction risk, which is equivalent to a reduction of the population size of less than a few percent. If the reduction in the population size is associated with loss of habitat, this implies that the destruction of habitat has a more profound effect on persistence of populations than does the chemical pollution. Nonetheless, we should recall that we dismissed two important factors in this simplistic analysis. First, real chemical pollution may involve many chemicals and multiple exposure with synergistic interaction among chemicals may inflate the total toxic effect on populations. Second, pollution of aquatic ecosystems by toxic chemicals may result in disturbance of plankton communities rather than in the extinction of a specific plankton species. If the stability of plankton communities is disturbed by adverse effect of chemicals that induce only a weak extinction risk, the MET analysis based on a single species may underestimate real hazards to ecosystems.

Extinction of a population does not necessarily mean species extinction. Many real species may comprise metapopulations consisting of numerous local subpopulations [29]. Even if some subpopulations become extinct due to chemical contamination, immigration of individuals from extant noncontaminated populations rescues the extinct subpopulations [30]. Nonetheless, local extinctions of many subpopulations and gross demographic stresses due to pollutant chemicals deflate the metapopulation dynamics by reducing migration and threaten persistence of a species [30]. In particular, local extinction of a keystone species occupying an influential ecological niche in a food web responsible for stability of a community may induce instability of a community [31,32].

Throughout this paper, we assumed that the major effect of toxicants was to reduce the mean intrinsic rate of natural in-

crease r since many ecotoxicological experiments showed that the reduction of r was the most plausible assumption [10, 11,20,21]. The net effects of toxicants on population growth are readily expressed in terms of r because r is a summary index representing population proliferation [5]. However, toxicant effects may increase the environmental variance of r as well. This inflates the extinction risk because MET is positively associated with the proportion of r to v. If exposure concentration fluctuates in time and the population-level responses follow the quadratic concentration–r curve without time lag, the component in the environmental variance due to the fluctuation of exposure v_{chem} is $v_{chem} \cong (\partial r/\partial x)^2 \mathrm{var}(x)$, where $\partial r/\partial x = -r_{max}(2x/\alpha^2)$ and var(x) is the variance of the exposure concentration in time. Incorporation of the variable exposure concentration is feasible provided that the period of the temporal fluctuation in exposure is longer than the generation time and the magnitude of the fluctuation is not so large as to violate the assumptions of the diffusion approximation. If short-term fluctuation that occurs within a generation is important, bioaccumulation, which is analyzed with the toxicokinetic and residue-based models [33], must be incorporated into the evaluation of the toxicants' effect on the population growth rate.

There are some practical limitations to MET assessment. Predictions of absolute values of MET are considerably different between theoretical models even if they are based on biologically plausible assumptions and the mathematical predictions have yet to be checked by experiments. The present study employed the mathematical models for a limited purpose, i.e., predicting proportional reductions of MET on the logarithmic scale (changes in orders of magnitude of the MET), taking into account the limited precision. It was suggested that different models predict nearly the equivalent values of $\Delta \log T$ from Δr.

Large uncertainties in the MET arise also from the limitations of the data. Only a very small portion of ecotoxicological data is relevant in estimating population-level effect of pollutants. There are considerable taxonomic biases in life table evaluation or population growth experiments. Such biases may be partly resolved by taxonomic extrapolation [13]. However, the taxonomic extrapolation itself entails a large uncertainty [12]. Apparently, we need much more data relevant for estimating population-level effects, especially for species in higher trophic status such as fish in aquatic communities.

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