# Ecological risk assessment: a new method of extinction risk assessment and its application to a freshwater fish (*Carassius auratus* subsp.)

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**Abstract**

We propose a new method to evaluate the ecological risk in terms of the increase in extinction rate of natural populations of animals and plants. The potential increase in the extinction risk by exposure to toxic chemicals and habitat loss is calculated for a freshwater fish, Japanese crucian carp (*Carassius auratus* subsp) in Lake Biwa and in Lake Suwa. We start with a stochastic differential equation model (logistic growth with environmental and demographic stochasticites). We estimated three parameters (growth rate $r$, carrying capacity $K$, and environmental noise $\sigma_e^2$) from a time series data of population size using maximum likelihood method. In Lake Biwa, growth rate and environmental noise of crucian carp are estimated as $r = 0.0173959$ and $\sigma_e = 0.0664468$, respectively. Toxic exposure causes the decrease in survivorship per generation by $\alpha$, which cannot be larger than $r$ ($0 < \alpha < r$). Extinction risk increases rapidly with $\alpha$. Habitat reduction (decrease in $K$) also causes the enhancement of extinction probability, but it is not as effective until the fraction of remaining habitat area becomes very small. In contrast, toxic chemical exposure may cause fast reduction in $\log T_K$ for a small concentration, if dose-effect relationship (Tanaka and Nakanishi 1988) is nonlinear ($\beta < 1$). We developed a method to calculate the reduction in habitat area that causes the same extinction risk as a give level of toxic chemical exposure.

## 1. Introduction

The extinction risk of natural populations of animals and plants serves a basis for quantifying ecological risk (Nakanishi 1995). The importance of various risk factors can be evaluated in terms of the enhancement of extinction risk, or the reduction in the mean extinction time of natural populations. If we combine this with the measurement in laboratory experiments of toxicity of chemicals and the estimate of their spatial spread and accumulation, we can calculate the risk of population extinction enhanced by the toxic chemical exposure.

In conservation biology, the extinction risk is measured by constructing computer simulation models. However many parameters included in realistic simulation models are often difficult to estimate. Especially the magnitude and correlation of temporal fluctuation of parameters are very important in determining the extinction risk, and yet are often not available for field populations. To overcome this difficulty we have developed a new method of extinction risk estimate based on model aggregation, as explained in Iwasa's chapter of this proceeding. We choose a simple case (canonical model) with logistic population growth with environmental and demographic stochasticities, and use this as the standard for all the other cases. We derived the mean extinction time as a function of three parameters (intrinsic growth rate $r$, carrying capacity $K$, and environmental stochasticity $\sigma_e^2$). For a given population, we can estimate these from a time series data, and then calculate the mean extinction time.

The same method can be used to simplify a complex model with many variable into a simple model with only three parameters. We generate a time series data from the complex model, obtain three parameters of the canonical model by fitting (maximum likelihood estimate). Then we can estimate the mean extinction time from the formula. We examined this aggregation method for a population including two subpopulations connected by migration (see Iwasa's chapter).

In addition, this method provides a common currency in diverse risk factors, such as habitat size reduction, habitat fragmentation, toxic chemical release, recurrent spread of epidemics, invasion of competitors, genetic deterioration. In terms of the shortening of "population longevity", we can compare for example the reduction of habitat size and the impact of toxic chemicals. We can ask what is the magnitude of habitat area reduction that causes the same magnitude of threat to a population as a given level of toxic chemical exposure.

In this paper, we combine the formula of the mean extinction time with the impact of toxic chemicals causing reduction in the intrinsic growth rate obtained from laboratory measurements. Then we compare the habitat area reduction with the exposure to toxic chemicals in the field in terms of their enhancement of population extinction risk. As an illustrating example we will use a freshwater fish, Japanese crucian carp in Lake Biwa, from which a relatively long time series of fishery data is available, allowing an estimate of the magnitude of environmental fluctuation.

## 2. Model

The dynamics of population size $X$ with time $t$ are expressed in terms of stochastic differential equation (canonical model):

$$\frac{dX}{dt} = rX\left(1-\frac{X}{K}\right) + \sigma_e \xi_e(t) \circ X + \xi_d(t) \bullet \sqrt{X}, \qquad (r, K, \text{and } \sigma_e > 0), \qquad (1)$$

where $r$ is growth rate, and $K$ is carrying capacity, $\xi_e(t)$ is white noise, and $\sigma_e$ is the intensity of the environmental fluctuation (see Iwasa's paper in this proceedings for detail). The average extinction time $T_K$ following Eq. (1) can be calculated as a double integral (see Eq. (2) of Iwasa's paper). To apply the model to field populations, we need three parameters. These can be estimated from a time-series data of population size by maximum likelihood method, and the accuracy of estimation depend on whether estimate of $r$ is available (see Iwasa's paper).

## 3. Risk Assessment in a case study of freshwater fish

### 3.1 *Fisheries data and estimate of parameters, r and* $\sigma_e^2$

We here illustrate our method of extinction risk assessment by using application to local populations of Japanese crucian carp (*Carassius auratus* subsp.) in Lake Biwa. Japanese crucian carp is the wild type of gold fish, and widely distributed in the freshwater of Japan (Kobayasi 1982). Japanese crucian carp reproduces every year and its maximum life is about 10 years. Three subspecies of crucian carp inhabit in Lake Biwa. This popular fish is suitable for case study, because a relatively long time-series of fisheries data is available. Using fisheries of crucian carp in Lake Biwa and the effort of fishery (number of fisherboat in Siga-prefecture) from 1955 to 1995, we calculated the catch per unit effort (CPUE) (Fig. 1; data from "Fisheries statistics of Japan 1955 - 1996"). CPUE, indicating a relative value of population size, stayed near its mean value for as long as 41 years, with fluctuation, although it shows a tendency of decline after 1988 (Fig. 1). Carrying capacity of Lake Biwa is unknown, but it is clearly very large (more than $10^6$ individuals). Then we can estimate growth rate $r$ and environmental noise $\sigma_e^2$ per generation according to estimating strategy as indicated by the method [3] in Iwasa's chapter. Assuming that generation time is 5 years from maximum life, intrinsic growth rate and environmental noise estimated by the maximum likelihood method are $r = 0.0173959$ and $\sigma_e = 0.0664468$.

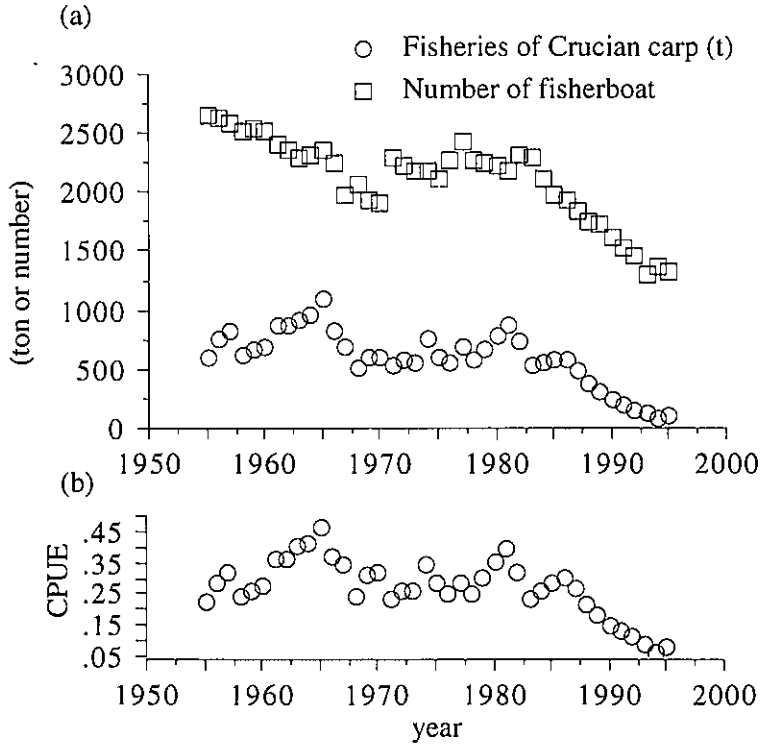


Fig. 1 Change of fisheries of crucian carp in Lake Biwa, number of fisherboat in Siga prefecture, and CPUE (fisheries of crucian carp / fisherboat).

### 3.2 *Risk of toxic chemicals*

When a population is exposed to toxic chemical substances in the environment, the effect causes a constant decrease in the survival rate per generation, denoted by $\alpha$. Change in the population size per generation is

$$\begin{aligned}\frac{dx}{dt} &= rx\left(1-\frac{x}{K}\right)+\sigma_e\xi_e(t)\circ x+\xi_d(t)\circ\sqrt{x}-\alpha x \\ &= \tilde{r}x\left(1-\frac{x}{\tilde{K}}\right)+\sigma_e\xi_e(t)\circ x+\xi_d(t)\circ\sqrt{x},\end{aligned} \qquad (2)$$

where $\tilde{r}=r-\alpha, \tilde{K}=K-K\frac{\alpha}{r}$. Therefore, the decrease in the survival rate per generation caused by exposure to toxic chemicals leads to the decline in both $r$ and $K$ of the canonical model (Eq. 1). The mean extinction time becomes

$$T_k\left(r-\alpha, K-\frac{\alpha K}{r}, \sigma_e^2\right), \qquad (3)$$

where $T_K$ is the formula obtained for the canonical model (Eq. (2) in Iwasa's chapter).

Clearly, the decrease in the survivorship per generation $\alpha$, cannot be larger than the intrinsic growth rate $r$, and the mean extinction time is zero when $r = \alpha$. This means that a species with a small growth rate $r$ is easier to go extinct than a species with large $r$ by the exposure to toxic chemical substances that decrease the survivorship if their sensitivity $\alpha$ is the same.

Figure 2 shows the relationship between the decrease in the survival rate per generation $\alpha$ and mean extinction time $T_K$. Mean extinction time decreases quickly as $\alpha$ increases from zero to $r$ and the way $\log T_K$ decrease with $\alpha$ is close to linear. The decrease in mean extinction time is larger for a large population ($K = 10^6$) than a small population ($K = 10^2$). This means that toxic chemicals are very effective in threatening large populations that is otherwise quite stable.

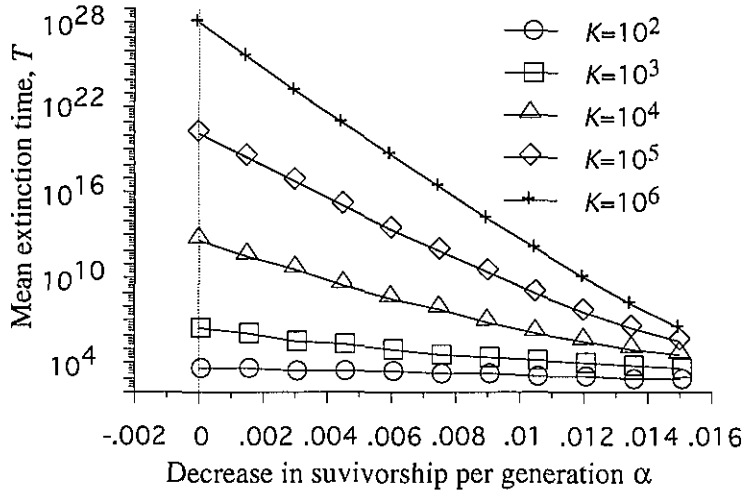


Fig. 2 Relationship between decrease in survivorship per generation and mean extinction time.

Tanaka and Nakanishi (1998) reviewed results of laboratory measurements on the relationship between $r$ and toxic chemical concentration, and summarized them as the following equation

$$r(z) = r_{\max}\left[1 - \left(\frac{z}{z_0}\right)^{\beta}\right]. \tag{4}$$

where $z$ is the concentration of toxic chemicals, $r_{\max}$ is the maximum growth rate, and $\beta$ is a constant for nonlinearity. The reduction in survival rate per generation $\alpha$ is

$$\alpha = r_{\max} g \left( \frac{z}{z_0} \right)^{\beta} \tag{5}$$

where $g$ is the generation time. By combining Eqs. (3) and (5), we can estimate the impact of toxic chemicals to the decrease in the mean extinction time.

The relationship of the decrease in the logarithm of mean extinction time and the reduction in the survivorship $\alpha$ is close to linear (Fig. 2). However the survivorship $\alpha$ decrease with the dose $z$ depends on critically the nonlinearly; i.e. $\beta$ in Eq. (5) may not be 1. Tanaka and Nakanishi (1998) showed that $\beta$ varies greatly between toxic chemicals and between organisms. For the cases of small $\beta$ ($\beta < 1$), small dose can cause a sharp decline in survivorship but further increase in dose does not cause much additional effect. In contrast the cases with a large $\beta$ ($\beta > 1$), toxicity starts above a certain threshold level. The relation between the enhancement of extinction risk and the amount of chemicals hence depends strongly on $\beta$.

### 3.3 *Habitat size reduction causing equivalent size as toxic chemicals*

On the other hand, reduction of habitat size, or the decrease in carrying capacity $K$, also causes the increase of population extinction risk. Figure 3 shows the relationship between habitat loss and mean extinction time $T_k$. Mean extinction time decreases with habitat loss, but initially gradually and then rapidly decrease to zero between 90 and 100 %. This pattern is quite different from the pattern of risk of toxic chemicals in Fig. 2.

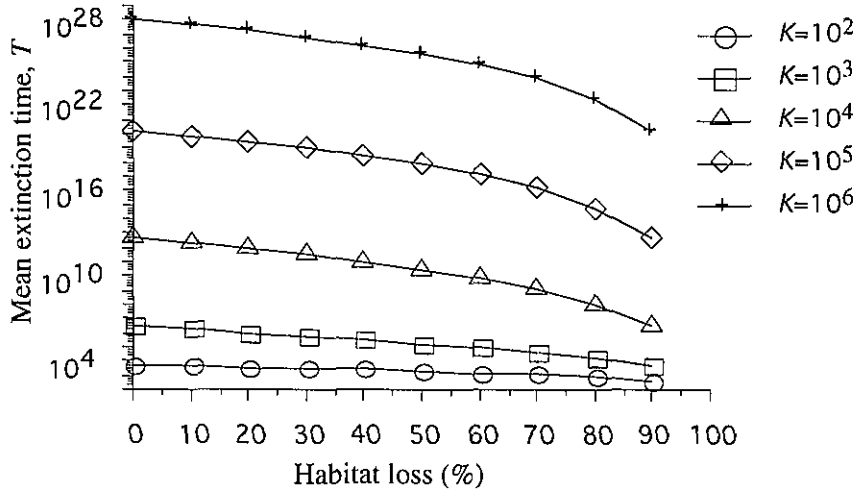


Fig. 3 The relationship between habitat loss and mean extinction time.

Both toxic chemical exposure and habitat loss promote population extinction. We can compare the magnitude of their impacts on extinction risk. If habitat size reduction and toxic chemical exposure cause the same extinction risk, we have

$$T_K\left(r, K10^{-\Delta \log K}, {\sigma_e}^2\right) = T_K\left(r-\alpha, K-\frac{\alpha}{r}K, {\sigma_e}^2\right), \tag{6}$$

where $\Delta \log K$ is the magnitude of habitat size reduction expressed in term of the decrease in the logarithm of $K$. Because there is a nice linear relationship between $\log T_k$ and $\log K$ (see Eq. (3) in Iwasa's chapter), small change in $\log T_k$ caused by the increase in $\log K$ is

$$\Delta \log T_k = -\frac{2r}{{\sigma_e}^2}\Delta \log K \cong -\frac{1}{\mathrm{CV}^2}\Delta \log K \tag{7}$$

where $\mathrm{CV} = \frac{\sqrt{\mathrm{Var}(\mathrm{X})}}{\overline{\mathrm{X}}}$ ($\frac{{\sigma_e}^2}{2r}$ is close to $\mathrm{CV}^2$ from Eq. (4) of Iwasa's chapter). Then, if $\alpha$ is small (small impact), we can derive the relationship between $\Delta \log K$ and $\alpha$ from Eq. (6),

$$\Delta \log K = \frac{\alpha}{r}\left(1+\frac{{\sigma_e}^2}{2}\frac{\partial}{\partial r}\log T_K\right). \tag{8}$$

In the case of Crucian carp in Lake Biwa, Eq. (8) is calculated as

$$\Delta \log K = \frac{\alpha}{0.0173959}(\log K - 0.93627). \tag{9}$$

There linear relationship between $\alpha$ and $\Delta \log K$, demonstrates that a small increase in $\alpha$ corresponds to a relatively large $\Delta \log K$ (Fig. 4). This means that a small decline in the

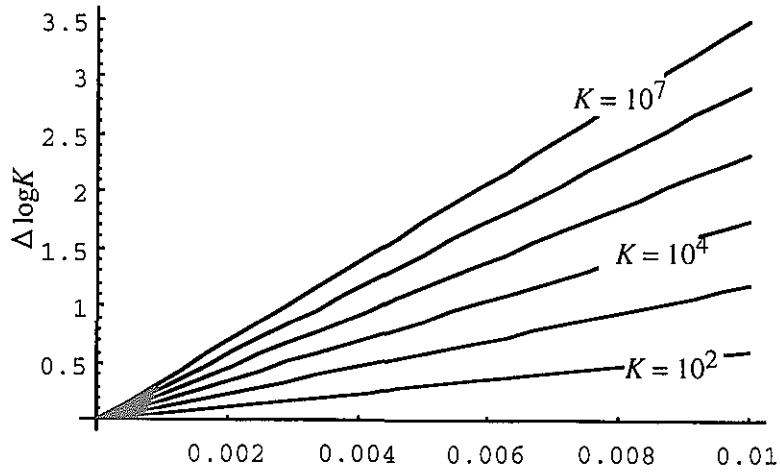


Fig. 4 Relationship between the decrease of survivorship per generation and the reduction of carrying capacity $\Delta \log K$.

survivorship caused by toxic chemical exposure leads to an extinction risk that would be equivalent to a considerable size reduction in habitat area.

We also have studied another population of crucian carp in Lake Suwa, which is smaller in size than Lake Biwa. These are attempt to obtain the size-structure, age-structure and the population dynamics (Fig. 5). According to preliminary study, the population is composed of sexual type and asexually reproducing type (gynogenesis) (Fig. 5). Coexistence of sexual and asexual types give a very interesting question of species interaction, which require the study of male mate preference in laboratory (Hakoyama and Ighchi 1998) and the differential susceptibility to parasitism in natural conditions. These gives a system to assess extinction risk in the presence of closely related competitors.

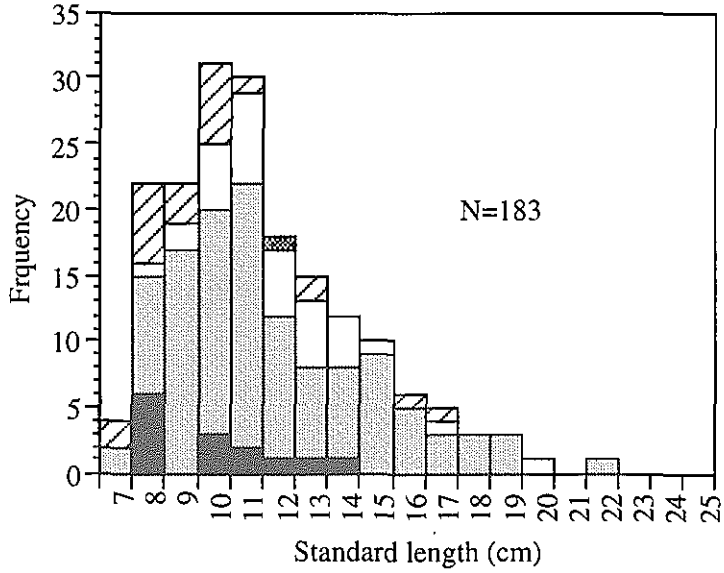


Fig 5 Size structure of Japanese crucian carp of Lake Suwa (Hakoyama et. al. unpublished data). Black column is 2n male, gray column is 2n female (sexual), open column is 3n female (asexual), and lined column is unidentified female.

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