Extinction Risk to Bird Populations Caused by DDT Exposure

by

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Abstract

The impact of toxic chemicals on wild animals and plants can be quantified in terms of the enhanced risk of population extinction. To illustrate a method for doing this, we estimated such impact for two bird species: herring gull (*Larus argentatus*) in Long Island, NY, and sparrowhawk (*Accipiter nisus*) in easthern England, when they were exposed to DDT (*p, p*'-dichlorodiphenyltrichloroethane) and its metabolites (called DDTs). The method we used is based on a formula of the mean time to population extinction derived for a stochastic differential equation (the canonical model). The intrinsic rate of natural population growth was estimated from an exponentially growing population, and the intensity of the environmental fluctuation was estimated from the magnitude of population size fluctuation. The effect of exposure to DDTs in reducing the population growth rate was evaluated based on an age-structured population model, by assuming that age-specific fertility is density-dependent and sensitive to DDTs exposure, but age-specific survivorship is not. The results are expressed in terms of the risk equivalent -- the decrease in carrying capacity K that causes the same enhancement of extinction risk as chemical exposure at a given level. The risk equivalent can be used in mitigation banking.

Key Words: extinction risk assessment, DDT, herring gull, sparrowhawk, risk equivalent, mitigation banking

1. Introduction

Recently, human health risk has become widely used to quantify the hazardarous effect of environmental chemicals (Suter 1993; Oka et al. 1997; Nakanishi et al. 1998; Gamo et al. 2000, 2001). However, policy choices based exclusively on the human health risk might be problematical, which can be most clearly demonstrated in the case of DDT (*p, p*'-dichlorodiphenyltrichloroethane). DDT is a very effective pesticide and has no acute toxicity. The half-life of DDT in the environment is long. Once assimilated by organisms, DDT and its metabolites (called DDTs), particularly DDE, remain in the body and are transferred to their predators when they are eaten. The concentration increases as we move up along the food chain (biomagnification). DDE exposure caused the thinning of birds' eggshells and the decline in the bird population in the late 1950s (Newton, 1979). DDT has an endocrine-disrupting effect and was quite possibly responsible for the abnormal maternal behavior of female herring gulls observed in the Great Lakes, resulting in brood desertion (Colborn *et al.* 1996). DDT feminized male sexual organs of birds (Fry and Toone, 1981). These hazardous effects of DDT on wildlife caused many developed countries to ban the use of DDT in the 1970s. On the other hand, DDT is still being used in many countries in which malaria is a serious problem. In the 1990s, three million people suffered from malaria and more than one million people (mostly children) died; among these deaths, approximately 90% occurred in sub-Saharan Africa (WHO, 1999). To control mosquitoes that are vectors of malaria parasites, DDT is a very effective and economical pesticide (e.g., see Mellanby 1992). In short, DDT has no strong acute human health risk and is a very effective pesticide for controlling malaria, while it has a clearly harmful effect on wildlife, particularly on birds.

To evaluate the situation by the risk-benefit analysis, we need to quantify the ecological risk of toxic chemical exposure, which is an endpoint separate from the human health risk. Nakanishi (1995) proposed that ecological risk evaluation be based on the extinction risk of natural populations. Population or species extinction is an irreversible loss, whilst other more commonly used criterion of ecological risks, such as changes in the number of individuals or biomass of organisms in the ecosystem, are often reversible. Hence, extinction is likely to be a situation that most can agree should be avoided, and hence it is desirable to adopt it as the basis

of an endpoint for environmental risk management. For this purpose, we need to calculate the enhanced extinction risk of wildlife populations when exposed to chemicals at a low concentration.

Mathematical and computational techniques to evaluate population extinction risk have been developed in conservation biology, but most of them concern the extinction of endangered populations whose size declines quickly and steadily. These are not very suitable for our purpose. To discuss the enhanced risk of population extinction caused by reduction in habitat size or by the exposure to toxic chemicals, we need to use models with a density-dependent population regulation as well as environmental and demographic stochasticities. Since the time until extinction for sustainable and density-dependent populations is often long, direct computer simulation is not practical for estimating their mean extinction time.

To overcome this difficulty, we use a mathematical formula of the mean extinction time for a simple model of population dynamics (canonical model) derived by Hakoyama & Iwasa (2000). The model is a stochastic differential equation and includes density-dependence as well as environmental and demographic stochasticities; it is the simplest possible model for evaluating the extinction time distribution for the situation herein examined. The model includes only three parameters which are estimated from the available data of demography and ecology of the population. The relative importance of different risk factors can be evaluated by the decrease in the mean extinction time. Hakoyama *et al.* (2000) derived an approximate formula for the reduction in habitat size that enhances extinction risk by the same magnitude as a given decrease in survivorship caused by toxic chemical exposure.

In the present paper, we illustrate Hakoyama and Iwasa's (2000) method, using two examples: a herring gull (*Larus argentatus*) population in Long Island, NY (Nakamaru *et al.* 2001a), which were exposed to DDT in Long Island, NY just before the use of DDT was banned, and a sparrowhawk (*Accipiter nisus*) population in easthern England (Nakamaru *et al.*, 2001b). There are several reasons for choosing these two species. First DDT concentrations in birds, particularly waterfowl and raptors, are greatly enhanced by biomagnification, and they are likely to receive a significant hazard (e.g., see Newton 1979; Newton & Bogan 1974). Second, DDT data on the herring gull and sparrowhawk are relatively easily accessible. Third,

the bird population size is smaller than that of other species, such as fishes or insects, and the average extinction time for stable populations is much shorter and hence the influence of chemicals on the mean extinction time is more likely to be of practical importance than that of more abundant species. Fourth, since raptors are likely to be endangered or threatened, the importance of evaluating the extinction risk of raptors, such as sparrowhawks, is an important focus of conservation biology and is readily justifiable. However, herring gulls were used only to illustrate the method. They are not endangered and, in fact, have been increasing in number in many areas.

In estimating the parameters, such as the effect of DDT on the population growth rate, we use an age-structured population model. We estimate "risk equivalent", or the equivalent loss of habitat which causes the extinction risk by the same magnitude as that of chemical exposure at a given level (see Hakoyama *et al*. 2000; also see Tanaka & Nakanishi 2000). Finally we discuss the application of mitigation banking and the difference both in estimating and in results between two species.

2. Canonical model and effect of toxic chemicals

We consider a simple general model which can be applicable to any sustainable population of plants and animals. The population size fluctuates around a positive level for many years before eventually becoming extinct. Hakoyama & Iwasa (2000) studied the dynamics of population size X at time t and expressed them in terms of a stochastic differential equation (canonical model):

$$\frac{dX}{dt} = rX\left(1 - \frac{X}{K}\right) + \sigma_e \xi_e(t) \circ X + \sigma_d \xi_d(t) \cdot \sqrt{X}, \qquad (1)$$

where the first term indicates the logistic population growth. r is the intrinsic rate of natural population growth and is the exponential population growth rate when the density is low. The population growth rate decreases as the population size approaches carrying capacity K, which is determined by the size and quality of the habitat. The second term is the environmental stochasticity, where σ_e is the intensity of the environmental fluctuation and ξ_e is white noise

with a mean of zero. The third term indicates demographic stochasticity caused by the fluctuation in survivorship and reproductive success that are independent between individuals. σ_d is the intensity of the demographic fluctuation and it is assumed to be one. ξ_d is white noise with a mean of zero. The white and black points in Eq. (1) indicates Stratonovich-calculus and Ito-calulus, respectively (see Hakoyama & Iwasa (2000) for details). Time is measured in units of the number of generations. Hakoyama & Iwasa (2000) derived the average extinction time as

$$T = \frac{2}{\sigma_e^{\,2}} \int_0^K \int_0^\infty e^{-R(y-x)} \left(\frac{y+D}{x+D} \right)^{R(K+D)+1} \frac{1}{(y+D)y}\, dydx, \qquad (2)$$

where $R \equiv \frac{2r}{\sigma_e^{\,2} K}$ and $D \equiv \frac{1}{\sigma_e^{\,2}}$. They also obtained approximate formulas based on regression analysis, but we use Eq. (2) in the present paper.

Using this formula, we can calculate how the mean extinction time is shortened when the parameter is shifted by exposure to toxic chemicals or by reduction in habitat size. We consider a stable population exposed to toxic chemical substances such as DDT. Toxic chemical substances reduce the population growth rate and can be represented by an additional negative term. Equation (1) becomes

$$\begin{aligned} \frac{dX}{dt} &= rX\left(1 - \frac{X}{K}\right) + \sigma_e \xi_e(t) \circ X + \sigma_d \xi_d(t) \cdot \sqrt{X} - \delta X \\ &= r' X\left(1 - \frac{X}{K'}\right) + \sigma_e \xi_e(t) \circ X + \sigma_d \xi_d(t) \cdot \sqrt{X} \end{aligned}, \qquad (3a)$$

with

$$r' = (r - \delta), \; K' = (r - \delta) K / r, \qquad (3b)$$

in which δ is the reduction in the per-capita population growth rate caused by DDT exposure (Hakoyama *et al.*, 2001). Equation (3b) shows that the chemical exposure leads to the decline

in both *r* and *K*. Figure 1 shows the effects of four parameters on the mean extinction time and Table 1 lists the parameters.

3. Estimation of parameters

To evaluate the effect of exposure to the toxic chemicals that reduce both *r* and *K* as in Eq. (3b), we need to know the intrinsic growth rate *r*, intensity of environmental fluctuation σ_e^2, the carrying capacity *K*, and the effect of a unit of toxic chemicals on the population growth rate δ (Figure 1) (Nakamaru *et al.*, 2001a). We obtain the demographic data, such as annual survivorship and fertility of females of different ages, and consider how these are modified with exposure to toxic chemicals. The survivorship of newly born chicks until the first census time is included in breeding success and hence in fertility. Egg production and chick survival are the stages most sensitive to the shortage of food, abnormal parental behavior, parasites, and developmental failures. To simplify the analysis, we assume the following: (1) First, the density-dependence of fertility at different ages is much more pronounced than the density-dependence of age-specific survivorship (see Kadlec & Drury 1968, for herring gulls). This implies that, when the population density increases, crowding and shortage of resources reduce the successful production of chicks much more than the survivorship of individuals older than one year. (2) Second, the exposure to toxic chemicals affects the fertility rather than adult survivorship. This is a good assumption for DDTs, which has no acute toxicity to birds but causes abnormal breeding behavior and eggshell thinning (Newton, 1979). (3) Third, we assume that the sensitivity of age-specific fertility to population density and to chemical exposure is common among females of different ages. Based on these simplifying assumptions, we can calculate four parameters included in the model and estimate the effect of toxic chemicals on the population extinction risk as follows.

3.1. Intrinsic rate of population growth (*r*)

First**,** we note that the Malthusian parameter of a demographic model including age-specific fertilities and mortalities obtained for saturated populations cannot be used for the intrinsic rate of population growth, *r*, because *r* is the growth rate for low-density populations. To overcome this difficulty, we used doubling time for exponentially growing populations that

started from recent invasion of birds. Alternatively, we can estimate *r* from a time series of population size, although the estimate includes a large variance (Hakoyama & Iwasa 2000).

3.2. Decrease in population growth rate caused by DDTs (δ)

We estimated the decrease in the per-capita population growth rate (δ) caused by the exposure to DDTs by the following steps: First, we calculated the fertility of females not exposed to DDTs based on the age-structured matrix model. Second, we estimated the decrease in the fertility casued by DDTs. Then, we obtained the intrinsic growth rate for a population exposed to DDTs, and the decrease in the population growth rate caused by exposure to DDTs. Finally, we converted the population growth rate per year to the population growth rate per generation.

3.2.1. Fertilities of females not exposed to DDTs

Demographic data including age-specific annual survivorship and age-specific fertilities are often available for a well-studied population. An aged-structured matrix model can be used to calculate the exponential population growth rate from these data. However, because of the density dependence, the age-specific fertility of females from a high-density population is lower than that from a low-density population. We can estimate age-specific fertilities in a low-density population from the intrinsic population growth rate, under the assumption that fertilities at different parental ages are multiplied by a common factor for density dependence.

Let $n(t,a)$ be the number of females at '*a*' years old in *t* year. p_a is the annual survival probability from '*a*-1' years old to '*a*' years old. The number of females at '*a*' years old in *t*+1 year is $n(t+1,a) = n(t,a-1) \times p_a$. Let $f(a)$ be the fertility of a female of '*a*' years old. The fertility is the mean number of chicks surviving until the next census time. The number of new born female chicks in *t*+1 year is $n(t+1,0) = \sum_{a=0} f(a) \times n(t,a)$. Then the exponentially population growth rate is given by the Euler-Lotka equation:

$$1 = \sum_{a=0}^{w} e^{-(a+1)r^*} \quad f(a) \quad p_1 p_2 \quad p_a , \qquad (4)$$

where r^* is the intrinsic rate of natural population growth per year if it is not exposed to DDTs, $p_1 p_2 \quad p_a$ is the survivorship, $f(a)$ is the fertility, and w is the maximum life span. Assuming that fertility $f(a)$ is multiplied by a factor for density dependence independent of the female age, we can estimate this factor using eq. (4) and the doubling time of an exponential growing population.

3.2.2. Decrease in fertilities caused by DDTs

Next, we estimated the decrease in the fertility of females caused by exposure to environmental DDTs. Since we assume that the sensitivity of age-specific female fertilities to DDT exposure is independent of age, the fertility of a female of age a is $C \times f(a)$, where C is a factor for the decrease in breeding success caused by DDTs. Then, the intrinsic population growth rate affected by DDTs can be calculated.

Hakoyama & Iwasa's formula (2000) gives the mean time to extinction expressed in terms of the number of generations. By multiplying the mean generation time, we converted the intrinsic rate of population growth per year into that per generation time. The mean generation time is calculated from the following (Pielou, 1969):

$$T_g = \frac{\sum_{a=0}^{w} a \; e^{-(a+1)r^*} \; M \; f(a) \; p_1 p_2 .. p_a}{\sum_{a=0}^{w} e^{-(a+1)r^*} \; M \; f(a) \; p_1 p_2 .. p_a} . \qquad (5)$$

3.3. Intensity of environmental fluctuation (σ_e^2)

Hakoyama & Iwasa (2000) developed a statistical method to estimate the intensity of the environmental fluctuation (σ_e^2) from time series data, noting that the intensity of the environmental fluctuation (σ_e^2) is proportional to the squared coefficient of the variation of the population size:

$$\sigma_e^2 = 2r \, \mathrm{CV}^2 \qquad , \qquad (6)$$

where the squared coefficient of the variation (CV^2) is $Var[X]/E[X]^2$ and r is the intrinsic rate of population growth per generation. However for a short time series, this is likely to seriously underestimate the variance. The values of the CV for many natural populations were reviewed by Pimm *et al.* (1988), who concluded that CV is 0.2 - 0.8. Hence we set CV=0.2 in the following.

3.4. Carrying capacity (K)

We calculated the results for several cases with different levels of carrying capacity K. Here we consider population size X and carrying capacity K measured in terms of the number of breeding females. We let K_0 be the number of females in the population not exposed to DDTs.

3.5. Equivalent loss of carrying capacity

Both exposure to environmental toxic chemicals and habitat size reduction enhance a population extinction risk. Based on the canonical model, land development reduces carrying capacity K. In contrast, the exposure to chemical substances reduces both the intrinsic growth rate, r, and the carrying capacity, K, simultaneously (Eq. (3b)). Hakoyama *et al.* (2000) considered the equivalent loss of the carrying capacity corresponding to the decrease in the mean extinction time as a good measure of risk caused by toxic chemical exposure, and termed it *risk equivalent*. Hakoyama *et al.* (2000) derived an approximate formula for the equivalent loss of carrying capacity (K) with other parameters unchanged: $\Delta \log T \approx \frac{1}{CV^2} \Delta \log K$. Here we directly carried out numerial analysis of Eqs. (2) and (3b) using Mathematica 4.0® and then obtained the relatoionship between equivalent loss of habitat ΔK and δ in the following:

$$f(r,\ K-\Delta K,\ \sigma_e^2) = f(r-\delta,\ K-K\frac{\delta}{r},\ \sigma_e^2) \quad . \qquad (7)$$

where $f(r,\ K,\ \sigma_e^2)$ is given by Eq. (2) (Hakoyama *et al.*, 2001).

Using *risk equivalent*, we can easily compare the ecological risk caused by different risk factors, such as land development and chemical exposure.

4. Case studies

4.1. A herring gull population in Long Island, NY

We summarize the application of the method to a herring gull populatoin (Nakamaru *et al.* 2001a).

We consider populations living in Long Island, NY because demographic and ecological data together with the measurement of DDTs are available. Unfortunately we could not obtain all the data needed for parameter estimation for herring gulls living in the same habitat. Some data from different habitats of the same species or from different species need to be combined.

The intrinsic population growth rate can be estimated from the doubling time of exponentially growing populations in a newly invaded habitat. According to Kadlec & Drury (1968), the doubling time was 15 years for a new colony of herring gulls in New England, USA. From this, the intrinsic population growth rate per year was $r^* = 0.0462$.

Kadlec & Drury (1968) reported the following estimates for annual survivorship (p_a): $p_1 = 0.641$, $p_2 = 0.800$, $p_3 = 0.757$, $p_4 = 0.739$, $p_5 = 0.736$, $p_6 = 0.722$, $p_7 = 0.784$, $p_8 = 0.707$, $p_9 = 0.727$, $p_{10} = 0.785$, $p_{11} = 0.685$, $p_{12} = 0.800$, and $p_a = 0.766$ ($a \geq 13$).

In Eq. (4), the sum was calculated up to the longest life span (w) of 30 years of age (Samuels and Ladino, 1983/84).

Chabrzyk & Coulson (1976) reported the number of fledglings for different maternal ages. Using these data we can estimate the ratio of fertility of the a-years-old females to the fertility of adults (7 years old or older). If the density is low, the fertility should be higher than these values for a saturated population. Let M be the fertility of fully mature females when the population density is low. If females of different ages are equally sensitive to the density, the fertility of females at different ages is multiplied by a common factor M: $f(a) = 0$ $(0 \leq a \leq 4)$, $f(5) = 0.52M$, $f(6) = 0.72M$ and $f(a) = M$ $(a \geq 7)$. For this to be consistent with the intrinsic population growth rate $r^* = 0.0462$ for a population with a low density, we obtained

M = 2.27 from eq. (4). This implies that the fertility in a low-density population is approximately 2.3 times (= 2.27/0.99) higher than that in a saturated population (=0.99).

Next, we estimated the decrease in the fertility of females caused by exposure to environmental DDTs, by assuming that the annual survivorship for individuals greater or equal to 1 year old is rather insensitive to chemical exposure.

Unfortunately, no data is available for herring gull on the relationship between the DDTs concentration in the egg and the survivorship of egg and chick. We hence used the data on black duck *Anas rubripes* (Beyer *et al.*, 1996), assuming that the sensivity of fertility to DDTs is the same between the black duck and the herring gull. The decrease in the chicks' survivorship after hatching is also caused by both abnormal parental behaviour, resulting in frequent brood desertion and the thinning of eggshells. We set C = 1 for 0 ppm. Using the Beyer *et al.* (1996) data, C = 0.61 (=23%/38%) for 46 ppm in egg, and C = 0.24 (=9%/38%) for 144ppm.

In Long Island, NY (Woodwell *et al.*, 1967), the DDTs concentration was 0.00005 ppm in water (C_e). In the herring gulls living there, the DDTs concentration in the body of adults (C_b) was 11.9 ppm, which is enhanced from the DDTs in water by biomagnification factor BMF $= 2.38 \times 10^5$. Here we assume that the DDTs concentration in the egg was the same as that in the adult body.

Combining these pieces of information, we can obtain the regression equation of the intrinsic population growth rate per generation as follows: $r(C_e) = -1.57 \times 10^3 \times C_e + 0.372$, which was obtained from linear regression with the estimate for unexposed data fixed. Hence, the decrease in the population growth rate δ is the difference between $r(0)$ and $r(C_e)$; $\delta(C_e) = 1.57 \times 10^3 \times C_e$. As C_e was 0.00005 ppm in the Long Island in the 1960s, r is 0.294. The positive growth rate means that we can adopt the canonical model when C_e is 0.00005 ppm.

With CV = 0.2, we can estimate the magnitude of environmental fluctuation σ_e^2 as 0.0298 without exposure from DDTs. We estimated the extinction risk for the cases with carrying capacities of K = 100, 10^3, 10^4 and 10^5 measured in terms of the number of breeding females.

Inserting these values into Eq. (2), we obtain Figs. 2A-C. Figure 2A illustrates the decrease of the logarithm of the mean extinction time which is caused by the exposure to DDE in an egg. In this case, the larger the carrying capacity, the larger the decrease. Figure 2B is the relationship between equivalent habitat loss and the decrease of the logarithm of the mean extinction time. This shows how the loss of land development reduces the mean extinction time.

Equation 7 shows that both Figs. 2A and 2B are needed to calculate risk equivalent or an equivalent habitat loss (ΔK). Figure 2C indicates the habitat loss equivalent to DDE exposure, which is estimated using Eq. (7), and is also the result of the combination of Figs. 2A and 2B. This graph has the same tendency as that in Fig. 2A. When the concentration of DDTs is the same as in the 1960s in Long Island, the equivalent loss of habitat is 30.5% for K=100 and CV = 0.2 (σ_e^2=0.0298) (Table 2).

4.2 Case 2: The sparrowhawk population in eastern England

We here summarize application of the model to the sparrowhawk populatoin (Nakamaru *et al.* 2001b).

Unlike for herring gulls, all the data needed to esimate the extinction risk of sparrowhawk living in eastern England are available. We used the same method as in the last section.

Intrinsic rate of natural population growth

We derived the intrinsic rate of population growth r^*=0.4 per year from the number of sightings of the sparrowhawk in eastern England while the population size was recovering (Newton & Wyllie, 1992).

Annual survival rate

Newton & Rothery (1997) concluded that annual survivorship is given by smoothed estimated data:

$$\log\left(p_a / \left(1 - p_a\right)\right) = -0.589 + 0.599\ \left(a - 1\right) - 0.071\ \left(a - 1\right)^2 \quad , \quad (8)$$

where p_a is the probability of annual survivorship in age *a*-1 from *a.*

The relationship between female age and the young per nest

Newton & Rothery (1997) showed the smoothed estimates of the young per nest as $y(a) = 0.578 + 1.069\ a - 0.103\ a^2$, where $y(a)$ is the young per nest when the female age is *a.* This formula, however, overestimates the young per female for ages 1 and 2 at which only a small fraction of females lay eggs. For these ages, the fertility was estimated from the data of the annual production of young ($y(1)$ = 0.32/2 = 0.16, $y(2)$ = 1.20/2 = 0.60), and the fertility for 3 years or older was estimated by the above formula, using the sex ratio of 1:1. Newton (1989) showed that the longest life span of females (*w*) is 10 years. From Eq. (4), we obtain *M* = 9.1.

Damage of DDE to the sparrowhawk

Newton (1986) showed that DDE only causes eggshell thinning, which leads to population decline because of egg breakage. Then, we can obtain the relationship

$$[\%\text{Reduction}] / 100 = -0.51919 + 0.35247 \ln[\text{DDE}] \quad , \quad (9)$$

where 4.36 ppm < [DDE] < 10 ppm. We denote the r.h.s. by RB for the moment.

We assume that the fertility of the population exposed to DDE is $f(a) \times (1 - \text{RB})$, and then inserting these values into Eq. (4), we can obtain the relationship between the DDE in eggs and the intrinsic growth rate per year. Then we converted the growth rate per year to that per generation, and obtained *r* =1.04 - 0.020[DDE]. Thus, we obtain the decrease in the per capita population growth rate as $\delta = 0.020 \times [\text{DDE}]$.

With CV = 0.2, we can estimate the magnitude of environmental fluctuation σ_e^2 as 0.0832.

We estimated the extinction risk for the cases with carrying capacities of K = 100, 10^3, 10^4 and 10^5 measured in terms of the number of breeding females. Figure 3 indicates the equivalent habitat loss ($\Delta K / K$) caused by DDE exposure. When the concentration of DDTs is almost 12 ppm, which is almost the same as that of herring gull ($=0.5\times10^{-4}\times\left(2.38\times10^{5}\right)$) in the 1960s in Long Island, the equivalent loss of habitat is 50.5% for K=100 (Table 2).

The equivalent loss of habitat or the extinction risk equivalent of sparrowhawks obtained was higher than that of herring gulls, although they were of the same order of magnitude (Table 2).

5. Discussion

We now compare the estimation of parameters in the herring gull (Nakamaru *et al*, 2001a) with that in the sparrowhawk. [1] In estimating the extinction risk of the sparrowhawk (Nakamaru *et al*. 2001b), we used only the data from sparrowhawk data in the eastern England, but in calculation for herring gulls, we had to combine data from several different habitats, (For example, we used biomagnification factor in Long Island, NY, and the doubling time in New England, USA.). [2] To estimate the intrinsic rate of natural population growth (r), we used the doubling time of a newly invaded population of herring gulls but the population size recovery after the use of a pesticide HEDO was banned for sparrowhawks. [3] In terms of survivorship, we used the field data for both species. Concerning the decrease of fertility caused by exposure to DDTs, however, we had to use the data from different species (black dack) in the herring gull study, while in the sparrowhawk study, we used the data from the same species. [4] In both the herring gull and sparrowhawk studies, we adopted several values of carrying capacity and a single standard value of CV= 0.2. Since estimate of CV from a relatively short time series is very unreliable, it is desiable to use these as a standard set of parameters in esimating risk equivalent to compare the risk of different toxic chemicals to different species.

In the previous section we also compared the results of two species of birds. The methods of estimating parameters, however, were different as we mentioned in the previous

paragraph. Therefore it is necessary to examine carefully what kinds of data are taken in the field or laboratory when estimating the extinction risk.

The comparison bewteen different local populations might give an estimate of the effect of toxic chemicals on the demongraphic parameters. However, Gilman *et al.* (1977) concluded that the decrease in the fertility and survivorship of herring gulls inhabiting the Great Lakes in the 1970s was caused not only by DDT and DDE but also by PCBs. Since we cannot distinguish the effect of DDE and PCB in fertility reduction, we did not use the comparative method in our extinction risk estimate (Nakamaru *et al.*, 2001a).

Since the canonical model includes only three parameters, we need to estimate four parameters. If we use a more realistic and complicated model including numerous variables and parameters to estimate, the difficulty in obtaining the data for reasonably accurate parameter estimation becomes tremendous, as is common to many examples of ecological modelling. To avoid this problem, we adopted the simplest possible model as the basis of our estimate considering the shortage of data available. If a sufficiently detailed set of data were available, we might be able to use a model with more structures than the canonical model. The extinction risk evaluation using the simplest possible model, such as done in the present paper, is recommendable as a standard method that is most easily applicable.

The mean extinction time evaluated for a sustainable population with density dependence and population fluctuation tends to be very large. Although the value itself has no practical meaning, the change of the mean extinction time caused by a risk factor can provide a useful method for evaluating the magnitude. The change in the mean extinction time itself is not very useful because the estimated value is too large to have practical importance. The change in the logarithm of the mean extinction time caused by the toxic chemical exposure ($\Delta \log T$) remains within a reasonable range of values, but it is still difficult to grasp the importance of risk caused by the chemicals. We believe that the use of "risk equivalent" or the equivalent loss of carrying capacity that causes the same decline in the mean extinction time is the most promising currency for evaluating the magnitude of risk (Hakoyama et al. 2000). This method of calculation may enable evaluation of the vastly different risk processes using the same currency in environmental policy choice. This measure might be used to evaluate the

magnitude of compensation of chemical pollution by enlargement or preservation of the habitats, which is termed mitigation banking (e.g. Tanaka, 1998).

The risk of exposure to toxic chemicals expressed as the equivalent loss of habitat tends to be larger for stable populations with long mean extinction time than for endangered populations (Hakoyama *et al*., 2000) -- it is larger when (1) K is larger, (2) CV is smaller and (3) r is larger. We believe that it is quite natural that the extent of damage in a small population is smaller than that in a larger population, when populations are exposed to the same concentration level of chemicals (e.g. Fig. 2A).

Several mathematical models have been studied to evaluate the hazardous effects of toxic chemical substances on populations of animals and plants. Tanaka & Nakanishi (2000) estimated the decrease in the mean extinction time using experimental *Daphnia* populations exposed to chronically toxic chemicals in the environment. They compared the extinction risk calculated by three mathematical models, namely, those by Lande (1993), Hakoyama & Iwasa (2000), and Foley (1994). They related the experimental measurement of LC_{50} to the enhancement of population extinction risk, and then to the equivalent loss in the carrying capacity. For example, a *Daphnia* population of 10^6 individuals exposed to chemicals at $[LC_{50}]/10$ level is exposed to the same risk as the reduction in carrying capacity by 1.2 %. Tanaka & Nakanishi's estimate differs from ours in several points. First, their work is based on the population growth experiments in the laboratory, while ours is on the estimation of the effect of DDTs on wild populations, the latter requiring compiling data obtained from published literature. Second, Tanaka & Nakanishi based their calculation mostly on Lande's formula (Lande, 1993) that assumes an exponential population growth with a ceiling at carrying capacity, whilst our analysis is based on Hakoyama & Iwasa's (2000) formula based on logistic population growth. This difference in the choice of population growth model causes the difference in how the parameters are modified when the population is exposed to toxic chemicals -- Tanaka & Nakanishi (2000) assumed that the chronically toxic chemicals affects only the population growth rate r, but not the carrying capacity K, while we assumed that the toxic chemicals decrease both the intrinsic rate of population growth r and the carrying capacity K. Both of these choices are consistent with the choice of growth rate function if the hazardous

effects of toxic chemicals are represented as an additional negative term in the population growth rate.

Here, we discussed the extinction of a single isolated population. However most species live in a number of habitats, and these local populations might be connected by migration, forming a metapopulation. The mean time to extinction of the entire metapopulation depends on the magnitude of migration between them, the environmental correlation between local populations, and their spatial configuration (Frank and Wissel, 1998). Even when different populations are completely separated, we need to relate the extinction of a single population to the risk of extinction of the whole species, which should be an important theme of future theoretical study. We may quantify the importance of a particular habitat by evaluating how the mean extinction time of the entire species is shortened when the habitat disappears compared with when it exists.

The calculation in this article is only a first attempt at evaluating the extinction risk to bird populations with toxic chemical exposure, and it includes many shortcomings that should be improved in future studies. Our purpose is only to illustrate the possibility of this method for evaluating the extinction risk to wildlife populations caused by toxic chemical exposure . The data required in order to apply this method to natural populations are not very difficult to obtain, if the data collection is planned beforehand with this application in mind.

In the present paper, we attempted to evaluate the ecological risk of DDT as a risk separate from its human health risk. We are also evaluating the human health risk of malaria. By combining this with the estimate of population extinction risk, we can discuss the rational management of environmental chemicals when there are tradeoffs between human health risk and ecological risk.

6. Acknowledgments

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8. Vitae

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9. Table 1

Parameters	Meaning of each paremeter
C	factor for the decrease in breeding success caused by DDTs
C_e	concentration of DDTs in the environment (concentration in the water in this paper)
C_b	concentration of DDTs in the body
$f(a)$	fertility at 'a' years old
K	carrying capacity
K'	carrying capacity with exposure to DDTs
$K(C_e)$	carrying capacity with exposure DDTs whose concentration in the environment is C_e
M	fertility of fully mature females older than 7 years when the population density is low
p_a	annual survivorship from 'a-1' years old to 'a' years old
r	intrinsic rate of natural population growth
r'	intrinsic rate of natural population growth with exposure to DDTs
$r(C_e)$	intrinsic rate of natural population growth with exposure to DDTs whose concentration in the environment is C_e
t	time
T	mean extinction time
T_g	mean generation time
X	population size
δ	decrease in the per-capita population growth rate caused by the exposure to DDTs
$\delta(C_e)$	decrease in the per-capita population growth rate caused by the exposure to DDTs whose concentration in the environment is C_e
σ_e	intensity of environmental fluctuation
σ_d	intensity of demographic fluctuation
$\xi_e(t)$ and $\xi_d(t)$	white noise with a mean of zero

The explanation of all the parameters.

10. Table 2

The equivalent fraction of reduction of the carrying capacity to the given decrease in *T*. The vertical rows are for four parameters of the canonical model. The columns are for the parameter values of each species: herring gull and sparrowhawk.

	herring gull	sparrowhawk
intrinsic growth rate (r)	**0.372**	**1.04**
environmental fluctuation (σ_e^2) CV = 0.2	**0.0298**	**0.0832**
carrying capacity (K)	**100**	**100**
equivalent loss of habitat (DDE in egg = 12 ppm)	**30.5 %**	**50.5 %**

11. Figure legend

Figure 1

Four parameters in the canonical model concerning the mean extinction time. δ is the reduction in the per-capita population growth rate caused by exposure to DDTs. Chemical exposure leads to a decline in both the intrinsic rate of population growth, r, and the carrying capacity, K. Environmental fluctuation is estimated from the time series data, given as the coefficient of variation of population size, CV. Using Eq. (2), we can obtain the mean extinction time (T). The increment of r and K makes T longer, but a higher CV leads to a shorter T.

Figure 2A

Decrease of the logarithm of T when the herring gull population is exposed to environmental DDE. Horizontal axis is for the DDTs concentration in water (ppm). The small dotted curve is for K =100 expressed by the number of females. The middle-size dotted curve is for $K = 10^3$; the largest broken curve is for $K = 10^4$; and the solid curve is for $K = 10^5$.

Figure 2B

Decrease of the logarithm of T of the herring gull populaiton when the carrying capacity is reduced by land development. Horizontal axis is for the decrease in the carrying capacity. The small and light gray point is for K =100 expressed by the number of females. The small, dark-gray points are for $K = 10^3$; the black points are for $K = 10^4$; and the large gray points are for $K = 10^5$.

Figure 2C

Risk equivalent, or equivalent habitat size loss, of the herring gull population caused by chemical exposure. The horizontal axis is for the concentration of DDTs in water (ppm), and the vertical axis is for the equivalent loss of carrying capacity that causes the same reduction in the mean extinction time as the exposure to the concentration of DDE in the egg. This was

produced by combining Figs. 2A and 2B. Light gray points and the solid curve are for K = 100 (the number of females); light gray points and small dotted curve are for $K = 10^3$; dark gray points and solid curve are for $K = 10^4$; and black points and broken curve are for $K = 10^5$.

Figure 3

Risk equivalent, or the equivalent habitat size loss, of the sparrowhawk population caused by the chemical exposure. The horizontal axis is for the DDE concentration in eggs (ppm); the vertical axis, for the equivalent loss of carrying capacity that causes the same reduction in the mean extinction time as the exposure to the concentration of DDE in the eggs. Light gray points and solid curve are for K = 100 (the number of females); light gray points and small dotted curve are for $K = 10^3$; dark gray and solid curve are for $K = 10^4$; and black points and broken curve are for $K = 10^5$.

Figure 1
Nakamaru *et al*.

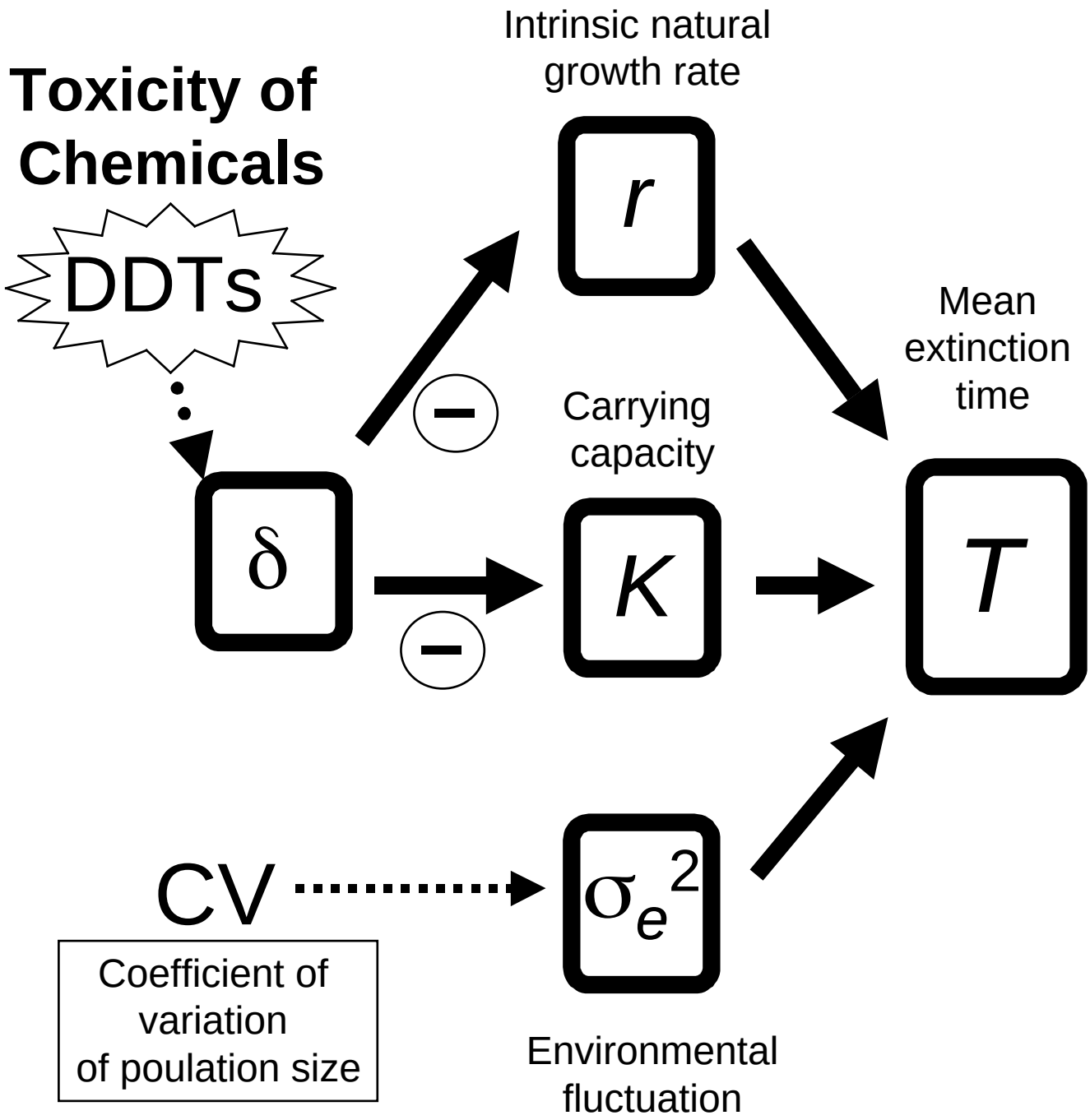
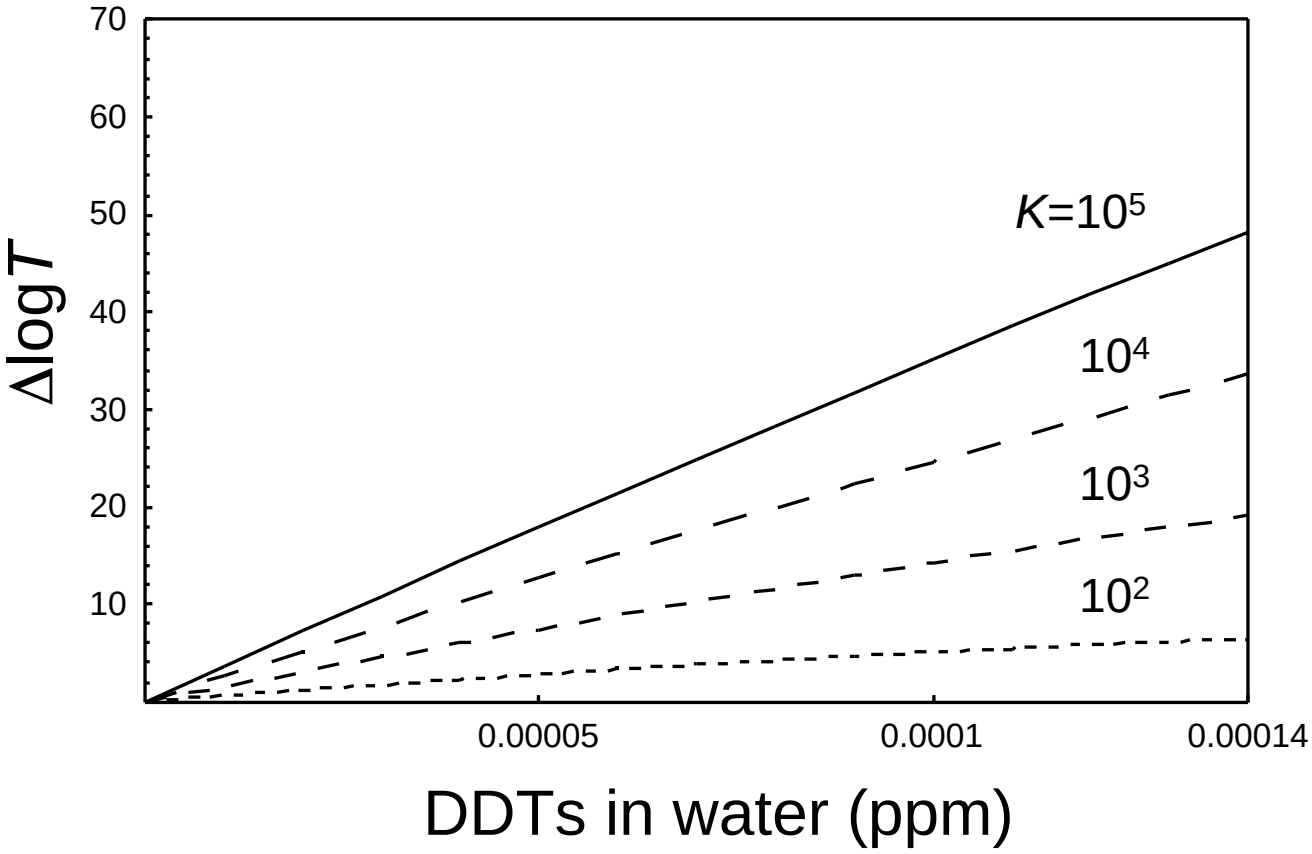


Figure 2A
Nakamaru *et al*.



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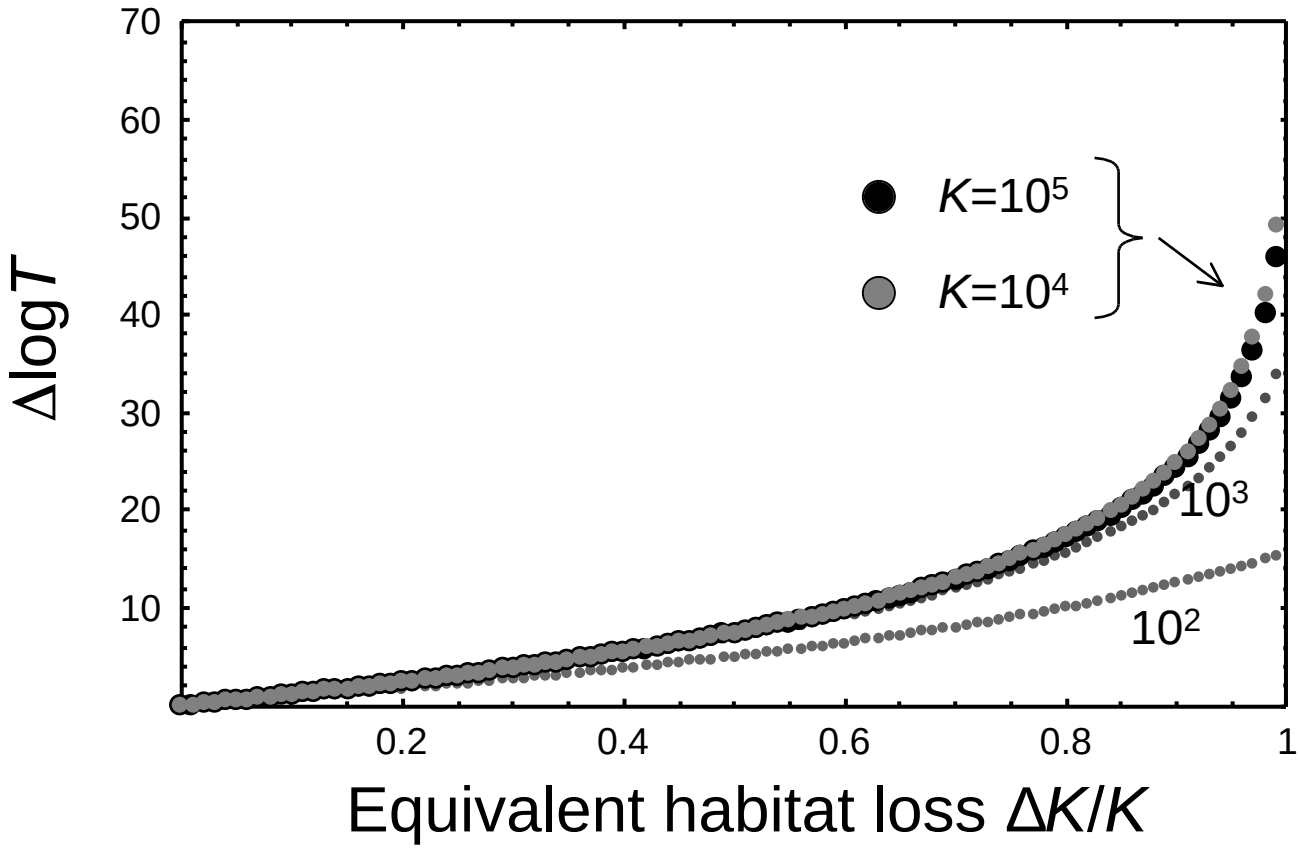
Nakamaru *et al*.



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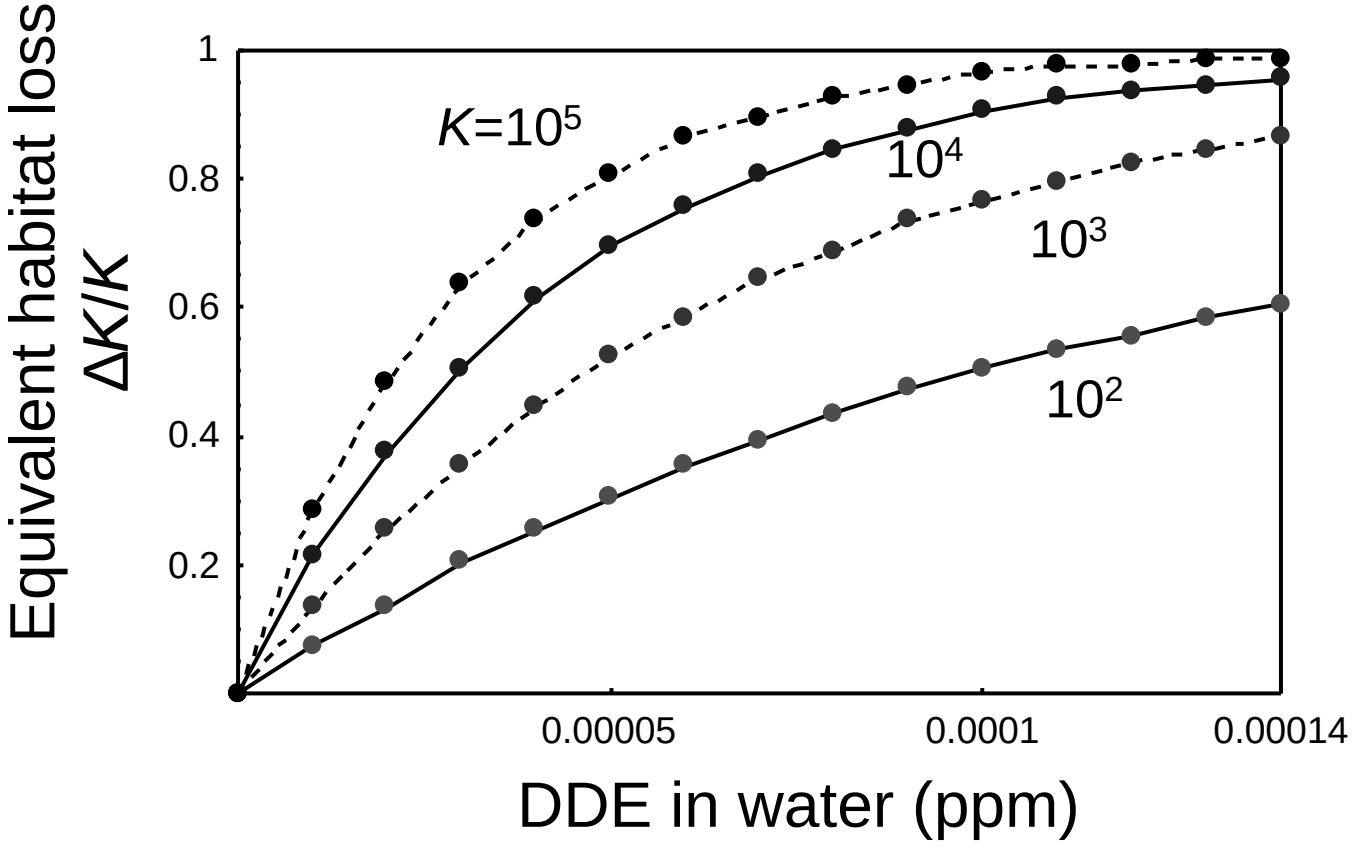

Figure 3
Nakamaru *et al*.