Ecological Risk of DDT - A Case of Biological Concentration of Herring Gull in Long Island.

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Abstract

We propose a new method to evaluate the extinction risk of natural populations enhanced by exposure to toxic chemicals. As an illustrating example, we calculate the extinction risk of Herring Gull (*Larus Argentatus*) in Long Island, NY caused by DDT (p, p'-Dichlorodiphenyltrichloroethane) and its metabolites (called Σ DDT). As a top predator, Herring Gull suffers severely by the biologically concentrated Σ DDT. The canonical model is adopted to evaluate the mean extinction time. The intrinsic rate of growth r is estimated from the doubling time of a fast growing population; the intensity of environmental fluctuation σ_e^2 is from the magnitude of population size fluctuation. Based on an age-structured matrix model with biological concentration factor and the decrease in fertility caused by the exposure to Σ DDT, we can estimate the decrease in the mean extinction time of Herring Gull population for different concentration of Σ DDT in the environment. Risk equivalent, defined as the fraction of habitat loss that causes the same decrease in the mean extinction time as a given exposure to toxic chemicals is very useful in evaluating the ecological risk and in management of toxic chemicals.

1. Introduction

DDT (p, p'-Dichlorodiphenyltrichloroethane) was invented by P. Mueller in 1938. It has been used widely as an very effective pesticide, because of the absence of strong acute toxicity. On the other hand it has a harmful effect to wild life.

Since DDT has a long half life (about 100 years), it stays in the environment and becomes biologically concentrated through the food chain. For example, in Long Island, New York, the Σ DDT concentration was 0.00005 ppm in water, 0.04 ppm in plankton, 0.17 - 2.07 ppm in fish, 1.07-75.5 ppm in birds (Woodwell *et al.*, 1967). The thickness of bird egg shells was reduced and the population sizes declined (Newton, 1979).

Enhanced risk of cancer, demonstrated experimentally for rats is regarded as a potential problem, but for humans there is no statistically significant relationship between DDT exposure and cancer rate. Recently DDT became noted as an endocrine-disrupting chemical (EDC). For example, female Herring gulls in the Grate Lakes, USA, showed enhanced rate of brood desertion. Abnormal parental behavior like this is suspected to be caused by DDT (Colborn *et al.*, 1996). DDT feminized male sexual organs of animals, because p, p'-DDT works as anti-androgen and o, p'-DDT is operated as estrogen. DDT has been banned in developed countries especially because of potential damages to ecosystem. The usage of DDT was banned in USA in 1972. In Japan DDT was widely used as an effective pesticide against domestic ectoparasites after the Second World War, but the production was banned in 1969 and the usage was also banned in 1971.

On the other hand, in many developing countries in South Asia, Africa and South America, malaria is a very serious problem. In these regions, DDT has been used as an effective pesticide against mosquitoes carrying malaria. In 1990s, three million people suffer malaria and more than one million people (most are children) die, among which more 90 % occurs in Sub-Saharan Africa. The case of Sri Lanka illustrates the effect of DDT. In 1934 - 35, there was 1,500,000 malaria patients within 6,000,000, the total population of Sri Lanka. Within seven months, 100,000 patients died of malaria. In 1945 the pre-eradication campaign started and DDT was sprayed all over Sri Lanka until 1946. As the result, the mortality of infants dropped from 141 per thousand to 87, and that of adults dropped down from 15.5 per thousand to 6.5. The national mortality decreased from 22.7 per 1000 to 12.6. In 1948 when the eradication campaign started there were 2,800,000 patients but 17 patients in 1963. However the use of DDT was banned in 1964. In 1969, the number of patients became to the same level as before (2,500,000). Sri Lanka used the alternative chemical, parathion which was not residue in the environment, instead of DDT. However parathion has strong acute toxicity and many employee splaying parathion died. This sort of accident is rare for DDT. Hence DDT is considered as an effective pesticide controlling Malaria and is kept being used in many developing countries in which Malaria is serious health problem.

Evaluating the risk of DDT gives an very good example in which a trade-off between the risk to human health and the risk to ecosystem is of an issue.

In this paper, we report an attempt to evaluate the ecological risk of the exposure to DDT to a bird population, in terms of the enhancement of population extinction risk. We has been developing the method to evaluate the enhanced extinction risk by toxic chemicals and the management of toxic chemicals (Hakoyama & Iwasa, 2000; Iwasa *et al.*, 2000), as explained in the last chapter by Iwasa *et al.*

2. Ecological risk assessment

DDT is famous for biological concentration. Since birds are on the top of the food chain and the concentration of DDT in birds were $10^{5\text{-}6}$ times as high as that in water. To simplify the effect of biological concentration, we use biological concentration factor (BCF), defined as the ration of the concentration in the body of animals and plants (C_b) to that in the environment (water in the present case) (C_e).

The loss of a growth rate exposed by the chemical substances does note always bring the population to immediate extinction but it keeps the population at the lower level and shorten the mean extinction time. To evaluate the effect of toxic chemical exposure to the enhanced risk of population extinction, Hakoyama and Iwasa developed a method of estimating mean extinction time based on a population model with density-dependent regulation with environmental stochasticity. Hakoyama *et al.* (2000) developed a formula converting the extinction risk caused by the exposure to toxic chemicals at a low concentration and the loss of habitat, as explained in the last chapter. We here adopt this method.

3 The calculation of the ecological risk

We use populations of Herring Gull in Long Island, NY, USA. The reasons are: (1) Relatively many data are available on birds exposed by ΣDDT; (2) BCF of birds is high; (3) The bird population size is small (compared to say fish or insects) and hence the average extinction time is short and show clear effect by the exposure to ΣDDT. (4) Waterfowls like Herring Gull and raptors are the top of the food chain, the effect of ΣDDT in these birds are clear. ΣDDT data on Herring Gull are available.

3.1. Intrinsic growth rate (r)

The intrinsic growth rate is obtained using the growth rate at which the very small population increases very rapidly. Note that the rate of population growth does not give a proper estimate for r. Kadles & Drury (1968) showed that the doubling time of new colonies in New England, USA, was 15 years. The doubling time means that the time interval to double the population size when the density is low. Then we can obtain the intrinsic growth rate per year (r^*) from it; $r^* = 0.0462$.

3.2. Magnitude of environmental fluctuation (σ_e^2)

The magnitude of fluctuation in population size is closely related to the intensity of the environmental fluctuation (σ_e^2). According to Hakoyama & Iwasa (in review), we have

$$CV^2 = \sigma_e^2 / 2r, \qquad (1)$$

where the squared coefficient of variation (CV^2) is $Var[X]/E[X]^2$ and r is the intrinsic growth rate per generation. We estimate the CV, using the field data like Pimm *et al.* (1988) where CV is 0.2 - 0.8. Kadles & Drury (1968) had rough time-series data of Herring Gull and we calculated CV= 0.02 - 0.15.

3.3. Carrying capacity (K)

We calculated the risk for several different values of carrying capacity for Herring Gull . The carrying capacity (K_0) is defined as the number of females in the population which is not exposed by DDT.

3.4. The decrease of the population growth rate (α) exposed by DDT

Equation (4) in Iwasa *et al.* (2000) showed the effect of α. In this session we'll estimate the decrease of the growth rate (α) exposed by ΣDDT, usin the biological concentration of ΣDDT and the effect of egg survivorship caused by ΣDDT.

3.4.1. Biological concentration factor (BCF)

At first we put the data on Long Island into Eq. (1). Woodwell *et al.* (1967) noted that C_e = 0.00005 ppm and C_b = 11.9 ppm. Then we can get BFC = 2.38×10^5.

3.4.2. Age-structured matrix model

In evaluating the decline of the per-generation growth rate, we consider the quickly increasing population in which no density dependent population regulation is at work, and then evaluate the growth rate under the exposure to ΣDDT. Since DDT affects fertility but not adult survivorship, we use the age-structured matrix model. We use the survivorship from Kadles & Drury (1968), and then we assume that the survivorship after fledging are not influenced by ΣDDT. Therefore we use the data from Kadles & Drury (1968) as the survival probability (p_a) from 'a-1' years old to 'a' years old.

The fertility of the population without DDT should be higher than the ones observed in a saturated population because of the absence of density dependent process. Chabrzyk & Coulson (1976) showed the data on the number of fledged which a-years-old

female had. These values are very low because this population is considered saturated and reaching the equilibrium.

If the density is low, we expect that the fertility is higher. We assume that the dependence on the mother's age is the same as in Chabrzyk & Coulson (1976). $f(a)$ the fertility rate per 'a' years old female is:

$$f(a) = \begin{cases} 0 & (0 \le a \le 4) \\ M \times 0.51/0.99 \approx 0.52M & (a = 5) \\ M \times 0.71/0.99 \approx 0.72M & (a = 6) \\ M & (a \ge 7) \end{cases}$$

The intrinsic growth rate r is a solution satisfying the Euler-Lotka equation,

$$1 = \sum_{a=0}^{w} e^{-(a+1)r^*} \cdot f(a) \cdot l_a , \qquad (2)$$

where l_a the survival rate from 0 years old to 'a' years old (= $p_1 \times \cdots \times p_a$). The longest life span (w) is assumed to be 30 years old (Samuels & Ladino, 1983/84). For the fertility rates to be consistent with the intrinsic growth rate $r^* = 0.0462$, we obtain M = 2.27, after calculating Eq. (2).

3.4.3 The decrease in fertility caused by DDT concentration in the body

Second, we calculate the decrease in the intrinsic rate by ΣDDT. Our assumptions are: [1] the adult survival rates (pa) are not affected by ΣDDT. [2] The mortality of brood are affected by ΣDDT. We denote C a factor less than 1, indicating the harmful effect of ΣDDT and $f(a)$ should be multiplied by C.

Unfortunately no Herring Gull data is available concerning the relationship between the DDT concentration in eggs and the survivorship from the egg to 3-week brood. We use the Black Duck data (Beyer *et al.*, 1996). We assume that the decrease of survivorship until 3-weeks after hatching includes the effect of both strange behavior (abnormal parental behavior) and the eggshell thinning. Using the data from Beyer *et al.* (1996). When we set the survival rate in 0 ppm C = 1,

$$C = \begin{cases} 1 & 0 \text{ ppm} \\ 23/38 \approx 0.61 & 46 \text{ ppm} \\ 9/38 \approx 0.24 & 144 \text{ ppm} \end{cases} . \qquad (3)$$

Using Eq. (2) with Eq. (3), we can obtain the intrinsic growth rates per year

We have to change into the intrinsic growth rates per generation because the canonical model in Hakoyama & Iwasa has the demographic stochasticity. Then we calculate the average generation time (T_g).

The average generation time is obtained as T_g = 8.05 years. We assume that DDT do note affect the survival rate p_a. By multiplying T_g, the intrinsic growth rates per year are converted to the rates per generation (r). For simplicity, we assumed that the concentration in adult bodies (C_b) are the same as one in eggs. From Eq. (1), $C_b = BCF / C_e$ then we can get the relationship between C_e and r:

ΣDDT residues 0 ppm in eggs	r^* = 0.372	
ΣDDT residues 46 ppm in eggs	r = -0.0236	(4)
ΣDDT residues 144 ppm in eggs	r = -0.550	

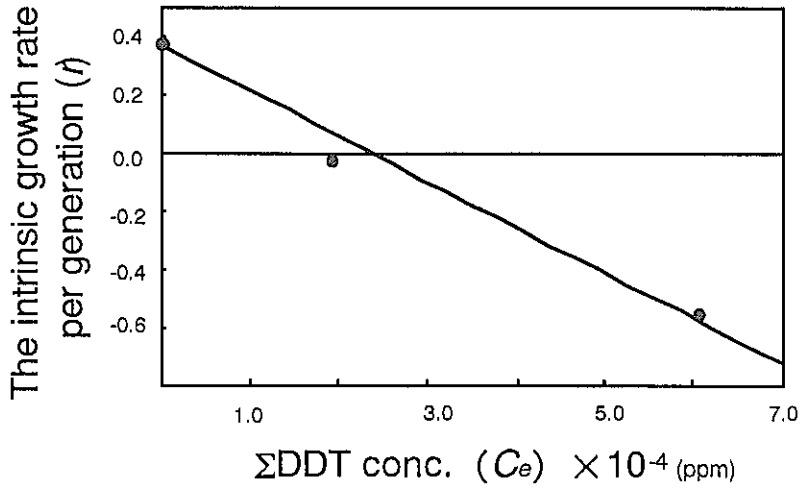

Figure 1; This graph indicates the relationship between ΣDDT concentration in the environment (C_e) and the intrinsic growth rate per generation (r). The solid line comes from Eq. (5) and the black circles are from Eq. (4).

The linear regression line is

$$r(C_e) = -1.57 \times 10^3 \times C_e + 0.372 \quad . \tag{5}$$

Figure 1 indicates this equation. When the ΣDDT concentration (C_e) in water in Long Island is 0.00005 ppm, r is 0.294.

4. The extinction risk represented as the decrease in the average extinction time.

4.1. The mean extinction time without ΣDDT exposure

From Eq. 2 in Iwasa *et al.* (2000), we can obtain the average extinction time. Hakoyama & Iwasa obtained the following regression formula:

$$\log T = -\log\left(\frac{r}{0.1}\right) + \left(1.12073\left(\frac{0.1\sigma_e^2}{r}\right)^{0.318121} - 0.0267559\right)\log\left(\frac{rK}{0.1}\right)^{\left(-9.70471\left(\frac{0.1\sigma_e^2}{r}\right)^{0.176337} + 8.07769\right)} + \left(-1.93776\left(\frac{0.1\sigma_e^2}{r}\right)^{0.113793} + 2.56977\right) \quad (10)$$

where $10^{-4} r \le \sigma_e^2 \le r$ and $1/r \le K \le 100/r$. We use these values to calculate the average extinction time: CV = 0.02, 0.2, 0.5, K_0 = 50, 100, 250. Figure 2 shows the average extinction time of Herring Gull without ΣDDT exposure.

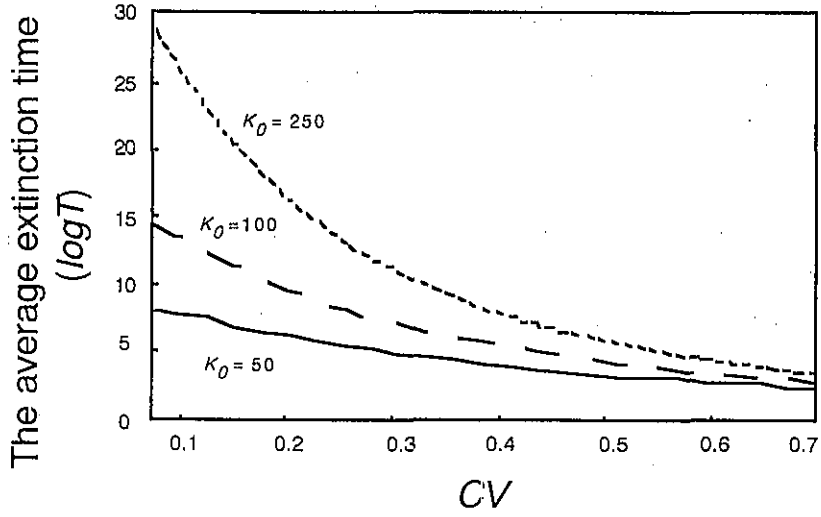

Figure 2; The average extinction time when Herring Gull population isn't exposed by ΣDDT.

4.2. The equivalent loss of habitat causing the same decrease in the extinction time.

The decrease in the mean extinction time, or its logarithm or the inverse are not very useful in expressing the magnitude of risk caused by toxic chemical exposure. Hakoyama *et al.* (in review) calculated the loss of the habitat (K) which was the same as the decrease of the average extinction time.

$$\Delta \log T \approx \frac{1}{CV^2}\Delta \log K \quad (11)$$

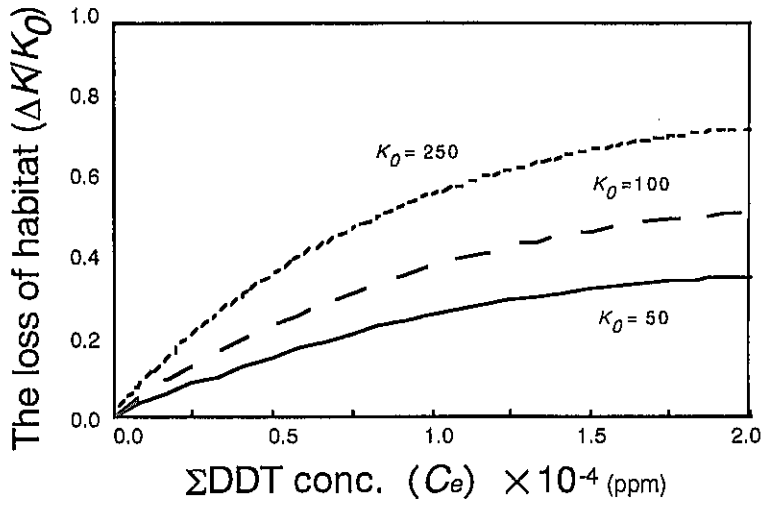

Figure 3; The graph shows the relationship between ΣDDT concentration in the environment (C_e) and the loss of habitat ($\Delta K / K_0$).

Figure 3 shows that the loss rate is larger as the initial habitat size is larger. In Long Island, the loss is estimated as 15% when CV = 0.2 and K_0 = 50 (K_0 is assumed to be the number of females), 23% in K_0=100, 34% in K_0=250. These results notices that the loss of habitat is not twice as large even if the habitat is twice.

We believe that this concept of "risk equivalent" may be very useful to represent the magnitude of extinction risk.

5. References

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