Estimation of Effect of Dioxins on Wildlife Population
― A Case Study of Common Cormorant Population ―

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Abstract

We show a method for quantitatively evaluating the effect of chemical pollutants in the environment on a wildlife population. We expressed the effect of dioxins in Tokyo Bay sediment on the common cormorant (*Phalacrocorax carbo*) population in two ways. One way is the ratio of the change in intrinsic growth rate, and another way is the ratio of the change in population size.

1. Introduction

Dioxins which composed of polychlorinated dibenzo-*p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs) and coplanar polychlorinated biphenyls (co-PCBs) , are highly toxic and persistent lipophilic substances, and are of interest because they accumulate in body of organisms mainly by the process of dietary uptake. It has been reported that wildlife in Japan have been contaminated by dioxins and that the concentration levels in fish-eating birds are especially high (Environmental Agency, 1999,2000; Hasegawa *et al*. 1999; Iseki *et al*. 2000, 2001a,b; Guruge *et al*. 2000). Due to the high social concern regarding the risks posed by dioxins, environmental monitoring and human health risk analyses have been conducted by Japanese regulatory agencies. However, an evaluation of ecological impacts of dioxins has not carried out so far. Thus, we attempted to quantify the effect of dioxins on wildlife and focus on the population-level effect of dioxins. In this modeling study, dioxins effects on common cormorant intrinsic growth rate and population size were evaluated utilizing population dynamics theory and field data. Intrinsic growth rate that integrates fertility rate and survival rate, is regarded as a suitable measure of responses to toxicants because it is a biologically relevant parameter (Forbes and Calow, 1999).

2. Selection of Target Species

Common cormorant population in Shinobazu pond was selected as the target species in this study. The reasons for selecting are as follows.

First, fish-eating birds such as common cormorant are suitable biomarkers of lipophilic, persistent organochlorine compounds (Fox *et al*., 1991a, 1991b). Common cormorant are terminal predator in the aquatic ecosystem and their diet is composed almost entirely of fish, so lipophilic chemicals such as dioxins are highly bioaccumulated in their bodies and eggs. Besides common cormorant has become the target species in the investigation of dioxin bioaccumulation carried out by Japanese Environment Agency from fiscal year 1999 (Environment Agency, 2000).

Second, the theory of population ecology is applicable to this population. During the period from 1971 to the mid-80s, it was reported that Shinobazu Pond had the only one colony of common cormorant in the Kanto region (Ueno Zoological Gardens, 1992; Narusue *et al*., 1997, Ishida *et al*., 2000). This suggested that the population can be treated as an isolated population without immigration and emigration during the period between 1971 and mid-80s.

Third, the data necessary for the estimation of population-level effect are available, because toxicological and demographical studies on the target or similar species have been extensively conducted. Exposure data such as body burden, toxicological data such as dose-response relationship, and demographic data such as survival or fertility rate, are necessary. Residue level of dioxins of common cormorant was investigated by Iseki *et al*. (2000, 2001a,b). Data on the dose-response relationship between TEQ in double-crested cormorant (*Phalacrocorax auritis*) egg and egg mortality rate are available (Tillitt *et al*.,1992 ; Ludwig *et al*.,1996) . Regarding demographic data, this population had been the subject of detailed investigation regarding population number and reproductive outcome after 1973 (Fukuda, 1980,1981,1997; Ueno Zoological Garden,1992).

3. Procedure for Estimation of Population-Level Effect

It is reasonable to assume that the population observed between 1971 and mid-80s was exposed to dioxins. It is unknown how the population would grow under dioxin-free conditions. We estimated the intrinsic growth rate under the condition without exposure to dioxins (r) , using the procedure shown in Fig.1. First, we estimated the intrinsic growth rate under the presence of dioxins from the observed

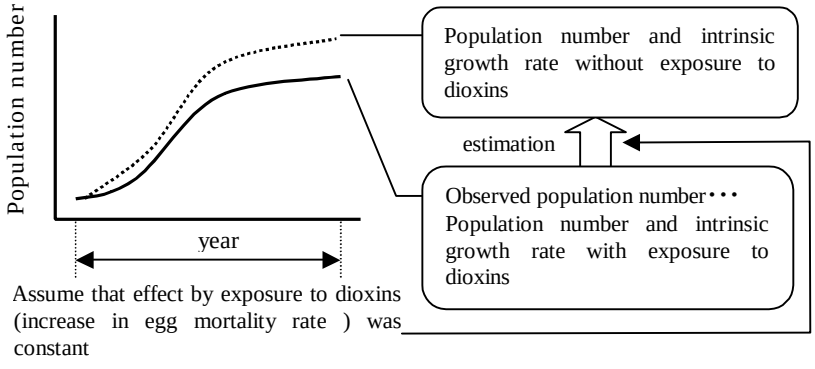

Fig. **1** Schema of Estimation of population-level effect

population number in 1974-1986, then we calculated the intrinsic growth rate under the absence of dioxins. The estimated residue revel in egg by exposure to dioxins in that period was quoted from Iseki *et al*. in the previous chapter. It is assumed that egg mortality rate due to exposure to dioxins was constant in that period, and we calculated the intrinsic growth rate under dioxin-free conditions. The procedure of the calculation was shown in Fig.2 .

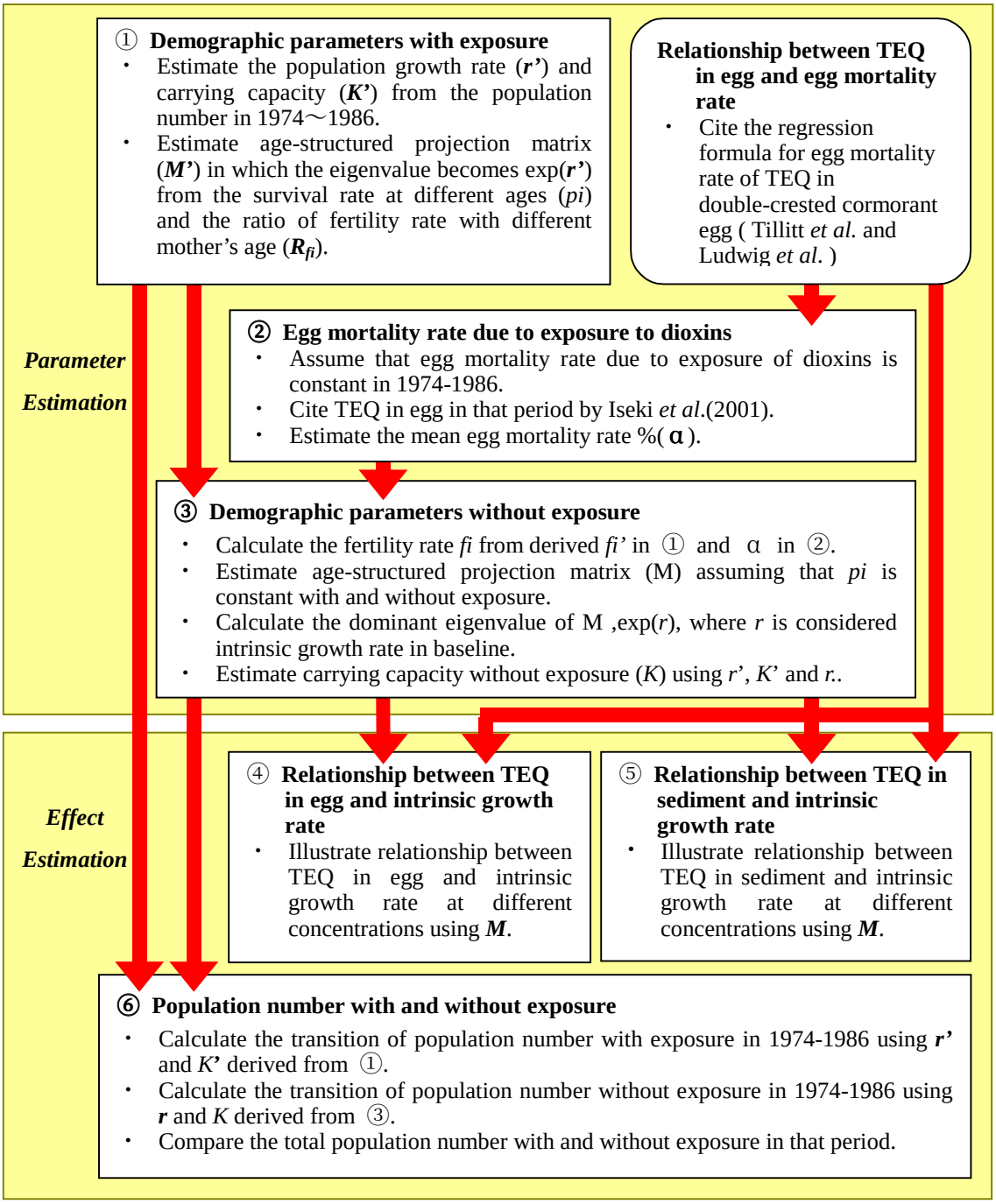

Fig. 2 Procedure for Estimation of Population-Level Effect

4. Calculation

4.1 *Population parameters with exposure to dioxins*

We estimated intrinsic growth rate (*r*'), carrying capacity (K') and age-structured projection matrix (*M*') with exposure by following methods.

Intrinsic growth rate (*r*') and carrying capacity (K') with exposure to dioxins were estimated based on data of the population number [N(t)] at Shinobazu Pond in 1974-1986 (The data were read from the figure.1 pp.66 in Ueno Zoological Garden, 1992) using logistic difference equation [Eq.(1)]. The parameter optimizations were performed using the commercial mathematical software (Mathcad 2000 Professional). The estimated *r*' and K' were 0.360 and 958, respectively. The population multiplication rate ($\boldsymbol{\lambda}$ ') is 1.44 [$\boldsymbol{\lambda}$ '= exp(*r*')]. This population multiplication rate is close to the value of the annual increasing rate of the nest of double-crested cormorant reported in the Great Lakes area between 1970 and 1991. The reported average annual increasing rate were in 1.402 (Lake Ontario), 1.335 (Lake Superior), 1.282 (Lake Huron) and 1.193 (Lake Erie) (Weseloh, 1995).

$$N(t+1) = N(t) \cdot \exp\left[r(1 - N(t)/K)\right] \tag{1}$$

Next, the parameters of age-structured projection matrix were determined using the following methods. Population numbers of each age in year 't' are denoted by vector ***n****(t)* of population size, $\boldsymbol{n}(t) = (N_{1,t}\ N_{2,t}\ N_{3,t}\ldots N_{i,t}\ldots N_{\boldsymbol{\omega},t})^T$, where $N_{i,t}$ is population size of *i*-year-old female in year 't' and *T* is matrix transpose. Population dynamics is expressed as Eq. (2) and Eq. (3) (Caswell, 1989). And the dominant eigenvalue of this matrix is population multiplication rate ($\boldsymbol{\lambda}$ '). f_i' is the fertility rate of *i*-years-old female, p_i is the survival rate at age *i* to *i*+1. $\boldsymbol{\omega}$ is the maximum longevity , 17 years old (Fukuda, 1997).

$$\overrightarrow{n(t+1)} = M' \cdot \overrightarrow{n(t)} \tag{2}$$

$$\begin{bmatrix} N_{1,t+1} \\ N_{2,t+1} \\ \vdots \\ N_{\omega-1,t+1} \\ N_{\omega,t+1} \end{bmatrix} = \begin{bmatrix} f_1' & f_2' & \cdots & f_{\omega-1}' & f_\omega' \\ p_1 & 0 & \cdots & 0 & 0 \\ 0 & p_2 & \ddots & \vdots & \vdots \\ \vdots & 0 & \ddots & 0 & 0 \\ 0 & \cdots & 0 & p_{\omega-1} & 0 \end{bmatrix} \cdot \begin{bmatrix} N_{1,t} \\ N_{2,t} \\ \vdots \\ N_{\omega-1,t} \\ N_{\omega,t} \end{bmatrix} \tag{3}$$

The survival rate at age (p_i) was estimated using the data of the survival rate at different ages in Shinobazu Pond from Fukuda (1981), p_1 =0.847, $p_{2\geqq}$ =0.864. The fertility rate (f_i') is expressed $fi' = R_{fi} \times F'$, where R_{fi} is the ratio of the fertility rate of *i*-years-old female (the fertility rate of mature female is one.). For R_{fi} we referred to the data from Fukuda (1997; The data were read from the fig.1.). We assumed that fertility rate from ages 6-13 is constant, R_{f1}=0.32,

R_{f2}=0.43, R_{f3}=0.75, R_{f4}=0.86, R_{f5}=0.97, $R_{f6\text{-}13}$=1, $R_{f14\text{-}17}$=0. We obtained F'=1.07 in which the dominant eigenvalue of age-structured projection matrix (***M'***) becomes λ'.

4.2 ***Egg mortality due to exposure of dioxins***

The egg mortality rate due to exposure to dioxins (α) was estimated assuming that α was constant in 1974-1986.

The total TEQ in egg (TEQegg) was quoted from the data from Iseki *et al*.(2001b). TEQegg estimated from three sets of concentration data in sediment (1972-1977, 1977-1981, 1981-1986) (Yao *et al*. 2000), were 270, 240, 210 WHO-TEQ(bird) pg/g wet wt.basis, respectively (Iseki *et al*.2001b). In our study, the average of three TEQegg values, 240, was assumed as the representative of TEQegg in 1974-1986.

Next, the TEQegg was substituted into x of Eq.(4) (Tillitt *et al*., 1992) and Eq.(5) (Ludwig *et al*., 1996). Eq.(4) and Eq.(5) describe relationship between egg mortality rate (y %) and TEQ in double-crested cormorant egg (x ;H4ΠE bioassay-derived TEQ pg/g wet wt.basis). Here, we assumed that TEQ in common cormorant egg could be applied to these relationships. Then, we obtained the mean egg mortality rate in 1974-1986, α=29% [applying Eq.(4)], and 21% [applying Eq.(5)]

$$y = 0.067 \cdot x + 13.1 \quad (r^2 = 0.703) \tag{4}$$

$$y = 0.0502 \cdot x + 8.61 \quad (r^2 = 0.906) \tag{5}$$

4.3 ***Population parameters without exposure to dioxins***

The intrinsic growth rate (r), the age-structured projection matrix (M) and the carrying capacity (K) without exposure to dioxins were estimated using the results obtained in the previous section 4.1 and 4.2.

The fertility rate with exposure f_i' derived in the previous section (4.1) is expressed in Eq.(6), assuming that reproduction and survival rate at fledgling to 1-year-old are not affected by exposure to dioxins. f_i is the fertility rate of i-years-old female without exposure. ε is the egg mortality rate without exposure. Egg mortality rate under dioxin-free conditions (ε) is not zero, so y-intercept in Eq.(4) or (5) was assigned to ε.Under the assumption described above, f_i was calculated using Eq.(6).

$$f_i = f_i' \times (100 - \varepsilon)/(100 - \alpha) \tag{6}$$

Assuming the constant survival rate p_i and using the calculated f_i, the age-structured projection matrix (***M***), population multiplication rate λ;the dominant eigenvalue of ***M*** , and the intrinsic growth rate ($r = \ln \lambda$) without exposure were estimated. The calculated r, the intrinsic growth rate without exposure to dioxins, was 0.424 or 0.404 applying Eq.(4) or Eq.(5),

respectively.

Carrying capacity without exposure K was estimated by the following method. Eq.(1) can be transformed in Eq.(7) replacing r/K with β. β is intra-specific competition constant, so the right end in Eq.(7) implies that a reduction of the intrinsic growth rate leads to a proportional decline of r/β (namely K) （Hendriks and Enserink, 1996）. Thus, K is estimated from this relationship （$r'/K' = r/K$） using calculated r', K' and r.

$$N(t+1) = N(t)\cdot\exp[r(1-N(t)/K)] = N(t)\cdot\exp\left[r\left(1-\frac{N(t)}{r/\beta}\right)\right] \qquad (7)$$

5. Results

5.1 *Relationship between TEQ in egg and intrinsic growth rate*

Fig.3 illustrates the ratio of the change in intrinsic growth rate, which is an index of population-level effect, with the increase in egg mortality rate posed by the increase in TEQ in egg.

The results indicate that it was estimated that the intrinsic growth rate became 86% or 90 % of the baseline at the mean TEQ level in egg collected in Tokyo in 1998.

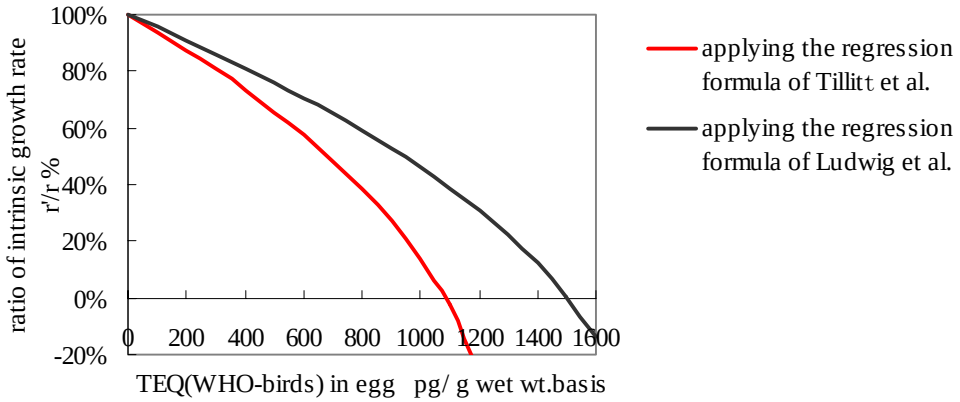

Fig.3 Relationship between TEQ in egg and ratio of intrinsic growth rate

5.2 *Relationship between TEQ in sediment and intrinsic growth rate*

Fig.4 illustrates changes in intrinsic growth rate with the increase in egg mortality rate posed by the increase in TEQ in sediment. TEQ in sediment is generally expressed in WHO-TEF for mammal.

TEQ in egg was unable to calculate directly from TEQ in sediment, because TEQ in egg were derived from the integration of each calculated congener concentration. Therefore, TEQ in egg was calculated from TEQ in sediment using the composition ratio of congeners obtained from recent data (1991-1993) of Yao et al. (2000).

For example, the results indicate that intrinsic growth rate became 87% or 91 % of the baseline at TEQ level in sediment in 1991-1992.

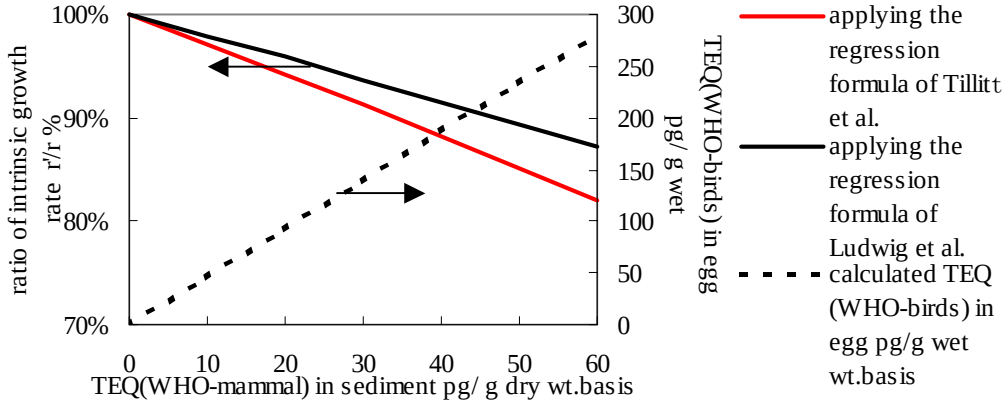

Fig.4 Relationship between TEQ in sediment and ratio of intrinsic growth rate

5.3 *Ratio of the change in population size with and without exposure to dioxins*

We estimated the ratio of the change in population size for the cases with and without exposure to dioxins. Fig.5 shows changes in population size obtained by the following calculation.

Dioxin-caused egg mortality may reduce population number. The observed population number (N') was considered the outcome of such reduction. The change of the population number with dioxin exposure (N') in 1974-1986 was calculated by substituting obtained r'=0.360 and K'=958 into Eq.(1). On the other hand, the potential population number under dioxin-free condition (N) was calculated by substituting obtained r = 0.424 and K=1129, or r = 0.404 and K=1074 into Eq.(1) (r and K have two values applying Eq.(4) or (5) , respectively.). The initial value of the population number N(0) was set at 125 in both cases, which was half of the observed value assuming that male-to-female ratio was one.

Comparing the total population number in that period with and without exposure to dioxins, we estimated ratio of population number with and without exposure to dioxins, $\Sigma N'(t) / \Sigma N(t)$ is 80% [applying Eq.(4)] or 86% [applying Eq.(5)] .

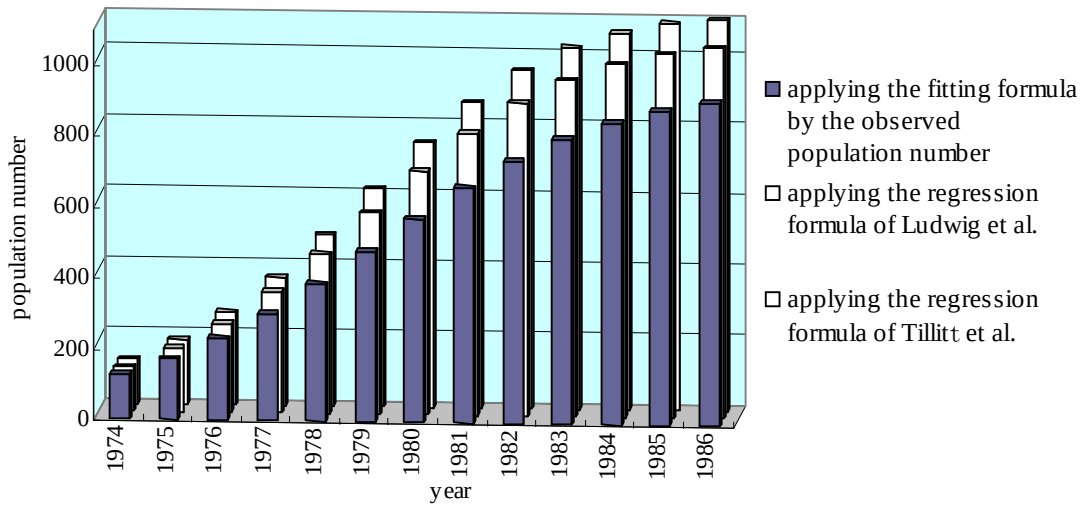

Fig.5 Population number in 1974-1986 in Shinobazu Pond

6. *Acknowledgments*

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