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A METHOD OF ASSESSING ECOLOGICAL RISK TO NIGHT HERON, *NYCTICORAX NYCTICORAX*, POPULATION PERSISTENCE FROM DICHLORODIPHENYLTRICHLOROETHANE EXPOSURE

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Abstract—This paper first introduces a probabilistic method for quantitatively evaluating the effects of chemical pollutants in the environment on a wildlife population, which was applied to assess the ecological risk to night heron (*Nycticorax nycticorax*) population persistence from dichlorodiphenyltrichloroethane (DDE) exposure in Tai Lake, China. Intrinsic rate of population increase (r) calculated with a population age–structured matrix model was used to measure the adverse effect on population. To perform a probabilistic analysis of risk, lost intrinsic rate of population increase (ΔZ) because of DDE exposure was applied to express the exact extent of risk. The result showed that the risk (i.e., the expectancy of ΔZ of the night heron population exposed to DDE in Tai Lake) was 0.0259, indicating a decrease in gross population size of 2.56% every year compared with that of the previous year without DDE exposure.

Keywords—Age-structured matrix model Dichlorodiphenyltrichloroethane Ecological risk assessment Intrinsic increase rate

INTRODUCTION

Contaminants exert adverse effects on all levels of biological organization from molecules to ecosystems. Although ecological risk assessment (ERA) at the levels of molecules, organisms, and individuals has been applied widely in the development of water quality criteria [1], the ERA at the population level has begun to be accepted by the public since it was proposed by Barthouse [2] and emphasized by Clements [3] and Calow [4]. The ERA at the population level can provide a more relevant measure of ecological impact because it is closer to the aims of managers or regulation makers than lower levels [5–9]. The U.S. Environmental Protection Agency (U.S. EPA) research plans for next few years include the modification of the criteria to protect water resources on the basis of risk to the piscivorous birds at the population level [10].

Several methods (e.g., field surveys of organism distribution and abundance; model extrapolation of estimating the extinction probability, biomass, and intrinsic rate of increase) have been proposed for population assessment [9]. The extinction endpoints provide a universal criterion for measuring ecological hazards [11] and have been applied to assess the extinction risk to herring gull populations from DDT exposure [12]. When data are scare for the population size and population fluctuation coefficient, which are necessary for estimation of extinction risk, the intrinsic rate of population increase (r) is considered to be another important parameter [5,6]. The intrinsic rate of increase was listed as an assessment endpoint of population-level effects in the Ecological Committee on the Federal Insecticide, Fungicide, and Rodenticide Act risk assessment methods (http://www.epa.gov/oppefed1/ecorisk/introduction.pdf). Although several applications have been reported to date and a single-point deterministic assessment process is often used to describe the trend of population dynamics under contaminants [7,12], probabilistic analysis is necessary for assessing the effects of chemical exposure in wildlife populations by combining field exposure analysis with the adverse effects on the population.

A persistent lipophilic metabolite from DDT, dichlorodiphenyltrichloroethane (DDE), can cause thinning of bird eggshells and decrease the size of the bird population. Concentrations of DDE in birds, particularly waterfowls and raptors, are greatly enhanced by bioaccumulation. Although DDT was banned at the beginning of the 1980s in China, there are high DDE residues in night heron (*Nycticorax nycticorax*) eggs in Tai Lake [13]. Several papers have reported that night herons are susceptible to persistent organic chemicals, and their reproductive success decreases at relatively low concentrations of DDE compared with other bird species [14–17]. Although the species was once common in the 1930s in New Jersey, USA, they became a threatened species in 1999 (http://www.nj.gov/dep/fgw/ensp/pdf/end-thrtened/bcnightheron.pdf). Night heron is one of the birds that migrates between the Americas and China [18] and has been used as a bioindicator to assess the risk of DDE to piscivorous birds at the individual level [19]. To our knowledge, however, there have been no reports about the risk at the population level, although the data necessary for the estimation of population-level effects—consisting of exposure data [13], toxicological data [14,15,20], and demographic data—are available [21].

In this paper, the magnitude of ΔZ, i.e., the difference between the intrinsic rates of increase under contaminant-free and contaminated conditions, was used as an index to reflect the exact extent of chemical effects at the population level. The hazard relationship between DDE dose (biotic burdens in eggs) and ΔZ was developed with a power function and the use of demographic data of the night heron and its toxicological data of exposure to DDE. Finally, the risk from DDE in night

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Table 1. Life cycle demographic parameters of night heron population used in the model

	Age (years)				
	1	2	3	≥4	Reference
Age-specific mortality (p_i)	0.39	0.69	0.69	0.69	[21]
Eggs per pair (n_i)	0	3	3.28	3.78	[23]
Probability of pairing (m_i)	0	0.422	0.566	0.955	Estimated

heron inhabiting the Tai Lake region (i.e., expectancy of ΔZ) was assessed with probabilistic analysis by combining field exposure analysis with the dose–response relationship between ΔZ and DDE exposure concentration.

METHODOLOGY

Biology of night heron

Most habitats of the night heron are associated with large wetlands such as swamps, streams, rivers, marshes, muddy flats, and the edges of lakes. They feed largely on fish but also eat frogs, crayfish, and even small rodents [19,22,23]. The longevity of the night heron is about 21 years, as reported by Roger et al. [24]. The annual mortality rates of one-year-old and older night herons are 0.61 and 0.31, respectively [21]. Night heron brood once a year, and incubation takes from 24 to 26 d. They often begin to pair at four years of age and sometimes at two or three years (http://www.biology.ualberta.ca/uamz.hp/heron.html). Up to now, there has been no literature about the age at which night herons stop pairing. It has been reported that the breeding success of short-tailed adult shearwaters (*Puffinus tenuirostris*) and herring gulls have no significant decrease with age [25,26]. In this study, we made an assumption that adult night herons keep pairing until death, and a sensitivity analysis for the year that pairing stops on ΔZ was carried out. The number of eggs for one pair at specific ages (n_i) in Tai Lake is three eggs for two-year-olds ($i = 2$), 3.28 eggs for three-year-olds ($i = 3$), and 3.78 eggs for four-year-olds or older ($i \geq 4$) [23].

Because six pairs of four-year-old and older night herons failed to pair out of 206 pairs, the pairing probability ($m_i \geq 4$) for four-year-old and older night herons was estimated to be 95.5% [23]. Although pairing probabilities for two-year-olds and three-year-olds could not be obtained, relative ratios (Ratio_2 and Ratio_3) between the pairing numbers for two-year-olds and three-year-olds and the sum of pairing numbers for four-year-olds and older were 0.303 and 0.257, respectively [23]. Thus, we can estimate the pairing probability for two-year-olds and three-year-olds (m_2 and m_3) according to Equation 1,

$$m_i = \frac{\text{Ratio}_i\left(0.955 \sum_{4}^{21} N_j\right)}{N_i} \tag{1}$$

where i is age (two or three years), N_i is the number of night herons age two or three years, and N_j is the number of night herons age 4 to 21 years. The stable age structure can be calculated as 0.341:0.232:0.497 corresponding to two-year-old:three-year-old:sum of four-year-old and older night herons by combining the following age-structured matrix model and Equation 1 with an iterative method. Thus, the m_2 and m_3 were estimated to be 0.422 and 0.566, respectively, as shown in Table 1.

Age-structured matrix model

The age-structured matrix model is applied to calculate r and the stable age structure [27,28]. The following age-structured matrix model has been applied to describe the dynamics of the night heron population over time.

$$\vec{N}_{t+1} = A \cdot \vec{N}_t \tag{2}$$

$\vec{N}_t$ and $\vec{N}_{t+1}$ are the vectors of age structure at time t and $t + 1$, respectively, and A is the population projection matrix. Equation 2 can be expanded to

$$\begin{pmatrix} N_{\text{egg},t+1} \\ N_{1,t+1} \\ N_{2,t+1} \\ N_{3,t+1} \\ N_{4,t+1} \\ N_{5,t+1} \\ \cdots \\ N_{21,t+1} \end{pmatrix} = \begin{pmatrix} 0 & F_1 & F_2 & F_3 & F_4 & F_5 & \cdots & F_{21} \\ P_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & P_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & P_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & P_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & P_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \cdots & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & P_{21} & 0 \end{pmatrix} \begin{pmatrix} N_{\text{egg},t} \\ N_{1,t} \\ N_{2,t} \\ N_{3,t} \\ N_{4,t} \\ N_{5,t} \\ \cdots \\ N_{21,t} \end{pmatrix}$$

where $N_{i,t+1}$ is the number of night herons of age group i at time $t + 1$, P_i is the probability of individual survival at age i, and F_i is the mean number of female neonatal offspring produced by one female at age i, represented as Equation 3,

$$F_i = \begin{cases} 0 & i = 1 \\ \delta \cdot n_2 \cdot \sigma_{\text{young}} \cdot m_2 & i = 2 \\ \delta \cdot n_3 \cdot \sigma_{\text{young}} \cdot m_3 & i = 3 \\ \delta \cdot n_4 \cdot \sigma_{\text{young}} \cdot m_4 & i \geq 4 \end{cases} \tag{3}$$

where δ is the ratio of neonatal females in neonatal individuals (assumed to be 0.5) and n_i is the number of eggs for one female at age i. In DDE-free environments, survival of young (σ_{young}) was reported to be 95% according to a survey of the literature [21]. The percentages of reduction in young survival (φ_{young}) caused by DDE exposure, summarized in Table 2 [14,15,19], were estimated by regressive Equation 4.

$$\varphi_{\text{young}} = 7.7081 \cdot C_e - 42.68 \qquad r^2 = 0.72 \qquad n = 11 \tag{4}$$

The maximum eigenvalue of matrix $\boldsymbol{A}$ was regarded as the population growth rate (λ) per year, which is the exponent r of the population ($\lambda = e^r$), and the corresponding eigenvector was used as the age structure [27].

Decrease of intrinsic rate of increase caused by DDE

The age-structured matrix model and reduction of young at specific DDE concentrations were combined to calculate the r of the night heron population exposed to DDE. Then, the DDE concentration (C_e) − r curve was fitted by the power function as in Equation 5.

$$r(C_e) = r_0\left[1 - \left(\frac{C_e}{\alpha}\right)^{\beta}\right] \tag{5}$$

Table 2. Exposure–effect relationship for effects of dichlorodiphenyltrichloroethane (DDE) on survival of young and predicted intrinsic rate of natural increase (r) of the night heron; the r at a specific concentration was estimated by an age-structured model that incorporated the effects of DDE on survival of young into the life history parameter F_i according to Equations 3 and 4[a]

Species	C_e (ng/g wet wt)	φ_{young} (%)	r	Reference
Black-crowned night heron	0[b]	0	0.012	[14]
	500	10	−0.005	
	6,000	41	−0.071	
	8,000	28	−0.041	
	500	0	0.012	[15]
	2,500	14	−0.013	
	6,000	18	−0.020	
	10,000	25	−0.035	
	14,000	29	−0.043	
	20,500	29	−0.043	
	37,500	41	−0.071	

[a] C_e = concentration of DDE in eggs; φ_{young} = percent reduction in survival of young; r = intrinsic rate of natural increase.
[b] Reduction of young is assumed to be zero with null exposure.

C_e is the natural logarithm of the concentration (ng/g wet wt of eggs) and its increment variance r, r_0 is the intrinsic rate of increase without DDE exposure in a realistic environment, α is the concentration when $r = 0$, and β is the curvature of responses, indicating the nonlinearity formula [29]. The difference between r_0 and r was defined as the lost intrinsic rate of increase (ΔZ) as in Equation 6 [11].

$$\Delta Z(C_e) = r_0\left(\frac{C_e}{\alpha}\right)^{\beta} \quad (6)$$

Exposure characterization

The DDE concentrations from wet weight measurements in 65 night heron eggs collected from Tai Lake, China, in 2000 were reported in Dong et al. [13] to be in the range of 195.66 to 5,837.29 ng/g wet weight, and the concentration geometric mean (M) and standard deviation (θ) were 1,102.76 ng/g wet weight and 883.24, respectively. The geometric standard deviation (S) of DDE concentration was obtained by the formula $\theta^2 = e^{(\ln S)^2 + 2\ln M}(e^{(\ln S)^2} - 1)$ [30].

Characterizing the risk

Because the magnitude of a toxic effect (ΔZ) is dependent on the toxic exposure level in eggs, we can define the risk for an exposed population as the expectancy of ΔZ, as described by Equation 7.

$$\text{Risk} = \int_0^{+\infty} p(C_e)\Delta Z(C_e)\, dC_e \quad (7)$$

The probability distribution function of DDE concentration in eggs is $p(C_e)$. Finally, the gross population size affected by DDE was assessed excluding density dependency.

RESULTS

Predicted decrease of young survival caused by DDE in Tai Lake

Survival reduction of young night heron in Tai Lake caused by DDE can be estimated by Equation 4 with the use of exposure concentration of DDE in eggs. Survival reduction was estimated to be from no adverse effect to 24.16%, and the mean reduction was 11.32%. The survival of young in Tai Lake determined by a field survey was 76.4% [23] (i.e., the reduction was 23.6%). This result suggests that DDE exposure partly contributes to the survival reduction of night heron in Tai Lake. In fact, other factors, such as raptors and other contaminants, affect the survival reduction of night heron in Tai Lake [31].

Relationships between r *and* ΔZ *with concentration*

Survival of young night heron (σ_{young}) exposed to DDE was incorporated into the population age matrix (Eqn. 3) with the demographic parameters (Table 1) to estimate the r at specific concentrations of DDE (Table 2). The dose (C_e)–response (r) curve shown in Figure 1 was fitted according to Equation 5 by nonlinear regression, and the r_0, α, and β (95% confidence interval [CI]) were 0.016 (−0.020–0.051), 5.96 (3.566–8.353), and 2.93 (−0.325–6.194), respectively, as shown in Equation 8.

$$r(C_e) = 0.016\left[1 - \left(\frac{C_e}{5.96}\right)^{2.93}\right] \quad (8)$$

Corresponding to Equation 8, the hazard relationship between C_e and ΔZ can be obtained by Equation 9.

$$\Delta Z(C_e) = 0.016\left(\frac{C_e}{5.96}\right)^{2.93} \quad (9)$$

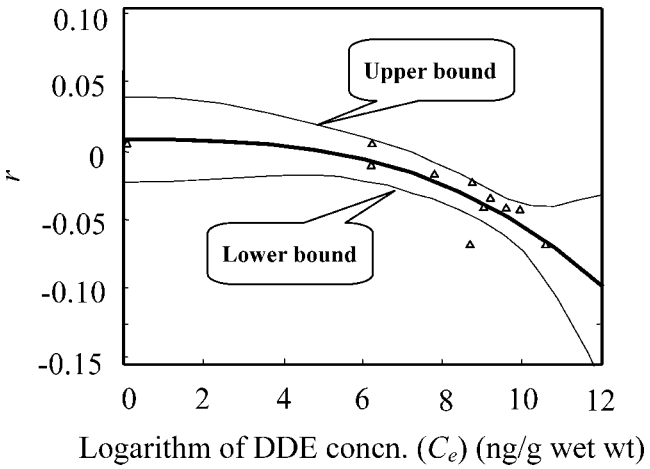


Fig. 1. Regression plot (solid line) of the relationship between predicted intrinsic rate of population increase, r, and dichlorodiphenyltrichloroethane (DDE) concentration in eggs. Dashed lines represent 95% prediction bounds. Plotting symbols (Δ) correspond to predicted r at specific DDE concentrations.

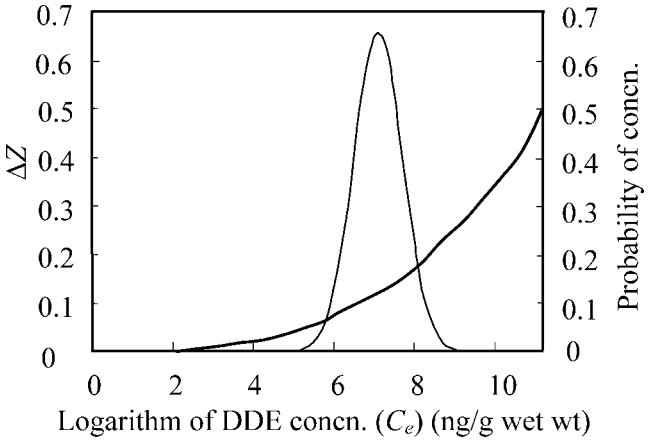


Fig. 2. Reduction of predicted lost intrinsic rate of population increase (ΔZ) with dichlorodiphenyltrichloroethane (DDE) concentration in night heron eggs (C_e, left axis) and natural lognormal probability distribution function of C_e [$p(C_e)$] in Tai Lake, China (right axis). Expectancy of ΔZ is the integral of the product of ΔZ_{C_e} and $p(C_e)$.

Expectancy of ΔZ for the night heron population inhabiting Tai Lake

The lognormal distribution function is frequently used to describe concentrations in the field [32–34], and it was also applied in this study to describe the exposure level, $p(C_e)$, of DDE concentration in night heron eggs in Tai Lake. The geometric mean and geometric standard deviation were 7.01 ng/g wet weight (natural logarithm scale) and 0.606, respectively [13]. To compare these values with the previous risk of night heron from DDE, a Monte Carlo simulation (Crystal Ball pro. 2000, Decisioneering, Denver, CO, USA) was undertaken with a lognormal distribution of the exposure data and a threshold of 1 μg/g (wet wt in eggs) [19] to calculate the risk quotient (RQ). With this approach, 10,000 Monte Carlo simulation runs gave a 56.5% probability of RQ exceeding unity for night heron inhabiting Tai Lake.

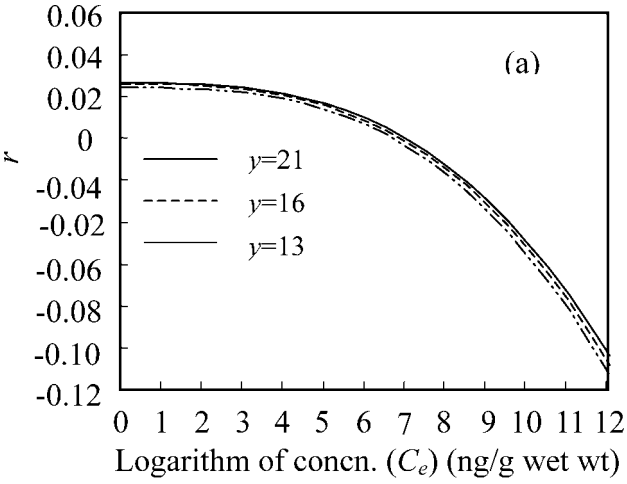
By coupling the $p(C_e)$ and $\Delta Z(C_e)$ as in Equation 7 (shown in Fig. 2), the risk for the night heron population exposed to DDE in Tai Lake was estimated to be about 0.026, and the corresponding r was -0.010. The r_0 (mean ± 95% CI) was compared with r with a one-sample t test, and r was significantly lower than r_0 (two-tailed $p = 0$), suggesting that DDE had an effect on the r for the night heron population inhabiting Tai Lake.

Sensitivity analysis for year that pairing stops on ΔZ

In the process of estimating the expectancy of ΔZ, we made an assumption that adult night herons continue to pair until death because related data were unavailable. Figure 3 shows the effects of the age that pairing stops (stop-pairing age) on r and ΔZ. It was found that setting the stop-pairing age at 11, 16, and 21 years had almost no effects on the ΔZ value (Fig. 3b). On the other hand, when the stop-pairing age was more than 13 years, changes in r were less than 5% with the incremental stop-pairing age compared with a stop-pairing age of 21 years (Fig. 3a). Considering the stop-pairing ages of other birds [25,26], the above assumption is reasonable.

Gross population size versus ΔZ

The gross population size ($\Sigma_1^{21} N_{i,t}$) at time $t + 1$ is dependent on instantaneous r_t, and size at time t can be obtained according to



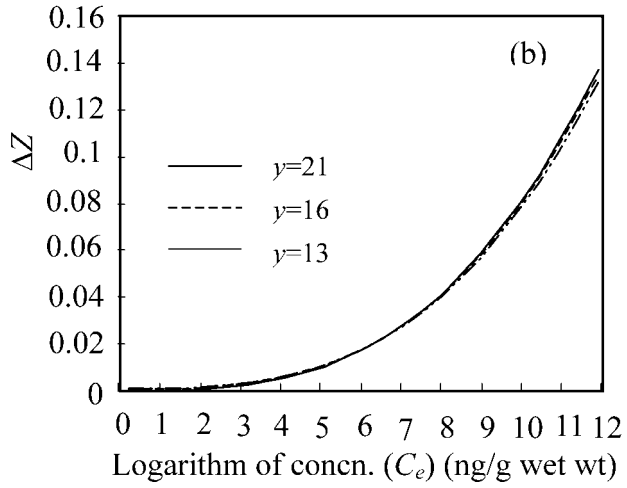


Fig. 3. Changes in concentration of intrinsic rate of population increase, r, (**a**) and lost intrinsic rate of population increase, ΔZ (**b**) corresponding to the different life history parameters (e.g., the year that pairing stops, y).

$$\sum_1^{21} N_{i,t+1} = e^{r_1} \sum_1^{21} N_{i,t}$$

When the population was exposed to DDE in year t, the gross population size at year $t + 1$ could be estimated as

$$\sum_1^{21} N'_{i,t+1} = e^{r'_1} \sum_1^{21} N_{i,t}$$

If no migration occurred and exposure was constant, the reduction of gross population size ($\Delta \Sigma_1^{21} N'_{i,t+1}$) increase with ΔZ can be represented by the equation

$$\Delta \sum_1^{21} N'_{i,t+1} \Big/ \sum_1^{21} N_{i,t+1} = 1 - e^{-\Delta Z}$$

as shown in Figure 4. Recalling that the night heron age-structured matrix model was developed with an internal time step of one year, the risk to night heron predicted over the full range of DDE exposure in Tai Lake can be interpreted as a decrease in population size of 2.56% every year compared with the population size of the previous year without DDE exposure. In other words, approximately five night herons are

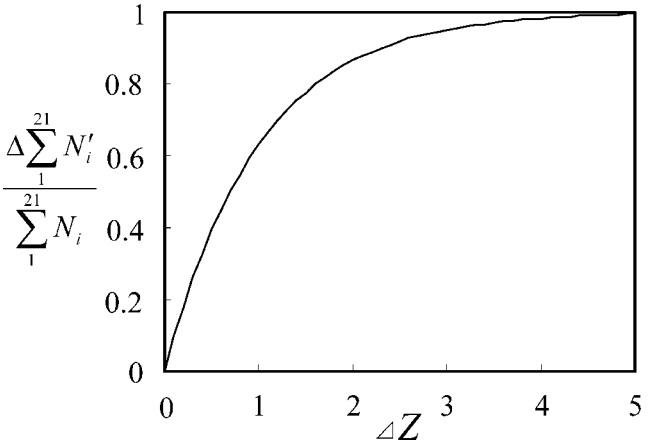


Fig. 4. Potential decrease in population size with an increase in lost intrinsic rate of population increase (ΔZ) without migration. N_i is the number of night herons of age *i*.

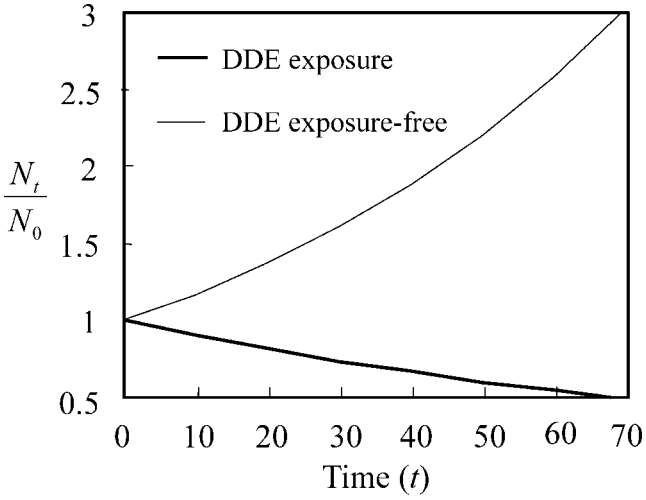


Fig. 5. Comparison of predicted night heron gross population size with time under current dichlorodiphenyltrichloroethane (DDE) exposure (solid line) and DDE-free condition (dashed line). The gross population size was estimated by the equation $N_t/N_0 = e^{rt}$, assuming life history parameters and exposure level were constant.

lost from a population of 100 pairs every year because of DDE exposure.

DISCUSSION

The probability that DDE concentration in night heron eggs inhabiting Tai Lake would exceed the threshold (1 μg/g wet wt) and affect reproductive success was estimated to be 56.5%, which is significantly higher than that (12.4%) in the Mai Po and Inner Deep Bay wetlands, Hong Kong, China [19]. Such a high probability of exceeding the threshold is of concern for the ecological risk of night heron. To assess the ecological effects of toxic chemicals, several endpoints, such as population [35,36], ecosystem, and landscape [37–41], have been proposed. Although effects on the ecosystem and landscape have high ecological relevance, relatively large parameter sets are frequently required [39]. In a trade-off between ecological relevance and tractability [38,39] in this study, we quantified the population-level ecological effects posed by DDE on night heron in Tai Lake with the use of reported data.

To assess the population-level effects of toxic chemicals, the intrinsic rate of increase is considered to be an important index, which is closely related to population size, persistence, and population extinction [42,43]. One method of estimating the intrinsic rate of increase under chemical-free conditions is the use of a time series population size of an increasing population from low density in a low-contamination area. This method has been applied to estimate the intrinsic growth rate of the sparrow hawk [44] and herring gull [12] populations with the regression line of the population size on the population growth rate and the doubling time of a newly established population near the subject site, respectively. In our case, the method was not appropriate for the estimation of r_0 because of a scarcity of a time series population size of night heron in Tai Lake. Consequently, we estimated the intrinsic growth rate by incorporating life history parameters, fertility, and survival under chemical-free conditions into an age matrix model [27]. For the night heron species, the fertility rate is not readily available. In this study, the fertility rate under chemical-free conditions was calculated from lower order fertility parameters, including the ratio of neonatal females, number of eggs, survival of young, and pairing probability, which were obtained directly from the literature and by iterative estimation.

The intrinsic growth rate under chemical pollution is often used as a measure of the population-level effects caused by chemicals, as exemplified by Nakamaru et al. [12] and Murata [45], in which a mean concentration of chemicals (i.e., a single-point deterministic assessment process) was used to estimate the intrinsic growth rate under chemical pollution. It is well known that the concentration of a chemical in the field often occurs with some specific probability distribution [19,32,33]. Thus, a single-point deterministic assessment process would be an under- or overestimation unless the probability of chemical exposure was identified to have a normal distribution. The probabilistic population-level risk assessment used in this study provided a useful method of identifying possible ecological damage.

As a consequence of the probabilistic analysis on the population level effect, the expectancy of ΔZ because of DDE exposure was 0.026; thereby, r was estimated to be -0.010 because r_0 was 0.016. This result showed that although the gross night heron population size under DDE-free conditions would increase by two times after 70 years, the population size of night heron exposed to DDE in Tai Lake would shrink with time and decrease to 50% (as shown in Fig. 5), assuming that there was no immigration of night heron from other habitats and DDE exposure was constant.

Finally, it should be noted that r_0 represents the potential of night heron population increase in the presence of natural environmental stresses other than contamination. These natural stresses include predators, changes in food resources, and bad weather. Hence, the ratio between expectancy of ΔZ and r_0 would be a more promising indicator of the realistic status of a species population, as well as overlapping risks from natural environmental stresses and response of the population to toxic contaminants. The ratio of ΔZ and r_0 for night heron in Tai Lake was estimated to be 1.64, implying that the habitat could not support population persistence because of DDE pollution.

This study is the first to assess the ecological effects of exposure to DDE on night heron populations inhabiting Tai Lake. The site-specific, population-level effects of DDE were estimated quantitatively by probabilistic analysis. The method of combining exposure analysis with this estimation of effect will provide a comprehensive framework for assessing the effects of chemical exposure on a wildlife population.

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REFERENCES

1. Heinz GH. 1989. How lethal are sublethal endpoints? *Environ Toxicol Chem* 8:463–464.
2. Barnthouse LW, Suter GW, Bartell SM, Beauchampp JJ, Gardner RH, Linder E, O'Neill RV, Rosen AE. 1986. User's manual for ecological risk assessment. Publication 2679. Oak Ridge National Laboratory, Oak Ridge, TN, USA.
3. Clements WH, Kiffney PM. 1994. Assessing contaminant effects at higher levels of biological organization. *Environ Toxicol Chem* 13:357–359.
4. Calow P. 1994. Ecotoxicology: What are we trying to protect? *Environ Toxicol Chem* 13:1359.
5. Forbes VE, Peter C. 1999. Is the per capita rate of increase a good measure of population-level effects in ecotoxicology? *Environ Toxicol Chem* 18:1544–1556.
6. Willam KW, John DS. 1997. Comparison of two population-level ecotoxicological endpoints the intrinsic (r_m) and instantaneous (r_i) rates of increase. *Environ Toxicol Chem* 16:1068–1073.
7. Munns WR, Dianne EB, Gleason TR, Salomon K, Bengtson D, Gutjahr-Gobell R. 1997. Evaluation of the effects of dioxin and PCBs on *Fundulus heteroclitus* populations using a modeling approach. *Environ Toxicol Chem* 16:1074–1081.
8. Newman MC. 1995. *Quantitative Methods in Aquatic Ecotoxicology.* CRC, Boca Raton, FL, USA.
9. Suter GWII. 1993. *Ecological Risk Assessment.* CRC, Boca Raton, FL, USA.
10. U.S. Environmental Protection Agency. 2003. Water quality research program multi-year plan office of research and development version, 2003, Update last peer reviewed: October 2001. Washington, DC.
11. Hakoyama H, Iwasa Y, Nakaishi J. 2000. Comparing risk factors for population extinction. *J Theor Biol* 204:327–336.
12. Nakamaru M, Iwasa Y, Nakanishi J. 2002. Extinction risk to herring gull populations from DDT exposure. *Environ Toxicol Chem* 21:195–202.
13. Dong YH, Wang H, An Q, Ruiz X, Fasola M, Zhang YM. 2004. Residues of organochlorinated pesticides in eggs of water birds from Tai Lake in China. *Environ Geochem Health* 26:259–268.
14. Findholt SL, Trost CH. 1979. Organochlorine pollutants, eggshell thickness, and reproductive success of black-crowned night heron in Idaho. *Colon Waterbirds* 8:32–41.
15. Henny CJ, Blus LJ, Krynitsky AJ, Bunck CM. 1984. Current impact of DDE on black-crowned night heron in the intermountain West. *J Wildl Manag* 48:1–13.
16. Blus LJ. 1984. DDE in birds' eggs: Comparison of two methods for estimating critical levels. *Wilson Bull* 96:268–276.
17. Heath RG, Spann JW, Kreitzer JF. 1969. Marked DDE impairment of mallard reproduction in controlled studies. *Nature* 224:47–48.
18. Zhang FY, Yang RL. 1997. *Bird Migration Research of China.* China Forestry Publishing House, Peking.
19. Connell DW, Fung CN, Minh TB, Tanabe S, Lam PKS, Wong BSF, Lam MHW, Wong LC, Wu RS, Richardson BJ. 2003. Risk to breeding success of fish-eating Ardeids due to persistent organic contaminants in Hong Kong: Evidence from organochlorine compounds in eggs. *Water Res* 37:459–467.
20. Henny CJ, Blus LJ, Krynitsky AJ. 1986. Ratiotelemetry loacates wintering grounds of DDE-contaminanted black-crowned night herons. *Wildl Soc Bull* 14:236–241.
21. Hickey JJ. 1952. *Survival Studies of Banded Birds.* U.S. Fish and Wildlife Service, Washington, DC.
22. Zhu X, Yang SD, Zou XP, Chen WZ, Ren JB. 2000. The breeding habits and growth of the nesting black-crowned night heron, *Nycticorax nycticorax*. *Zool Res* 21:58–64.
23. Zhang YM, Zhao DQ, Ruan LZ, Dong YH, Wang H, Fasola M. 2003. Pairing age and reproductive success in black-crowned night herons at Tai Lake, China. *Zool Res* 24:57–59.
24. Roger BC, Klimekiweicz MK, John HK. 1982. Longevity records of North American birds: Gaviidae through Alcidae. *J Field Ornithol* 53:81–124.
25. Wooller RD, Bradley JS, Skira IJ, Serventy DL. 1990. Reprodutive success of short-tailed shearwaters *Puffinus tenuirostris* in relation to their age and breeding experience. *J Anim Ecol* 59: 161–170.
26. Chabrzyk G, Coulson JC. 1976. Survival and recruitment in the herring gull *Larus argentatus*. *J Anim Ecol* 45:187–203.
27. Caswell H. 2001. *Matrix Population Models: Construction, Analysis and Interpretation,* 2nd ed. Sinauer Associates, Sunderland, MA, USA.
28. Daniels RE, Allan JD. 1981. Life table evaluation of chronic exposure to a pesticide. *J Fish Aquat Sci* 38:485–494.
29. Tanaka Y, Nakanishi U. 2001. Model selection and parameterization of the concentration-response functions for population-level effects. *Environ Toxicol Chem* 20:1857–1865.
30. Kenney JF, Keeping ES. 1951. *Mathematics of Statistics,* 2nd ed. Princeton University Press, Princeton, NJ, USA.
31. Zhang YM, Ruan LZ, Dong YH, Gong ZM, Li YD, Wang H, Fasola M. 2000. Breeding biology of night heron (*Nycticorax nycticorax*) and little egret (*Egretta garzetta*) in Tai Lake of Wuxi China. *Zool Res* 21:275–278.
32. Aigner EJ, Leone AD, Falconer RL. 1998. Concentrations and enantiomeric ratios of organochlorine pesticides in soil from the U.S. Corn Belt. *Environ Sci Technol* 32:1162–1168.
33. Gordon SM, Callahan PJ, Nishioka MG, Brinkman MC, O'Rourke MK, Lebowitz MD, Moschandreas MJ. 1999. Residential environmental measurements in the National Human Exposure Assessment Survey (NHEXAS) pilot study in Arizona: Preliminary results for pesticides and VOCs. *J Expos Anal Environ Epidemiol* 9:456–470.
34. Kim SK, Oh JR, Shim WJ, Lee DH, Yim UH, Hong SH, Shin YB, Lee DS. 2002. Geographical distribution and accumulation features of organochlorine residues in bivalves from coastal areas of South Korea. *Mar Pollut Bull* 45:268–279.
35. Ferson S, Ginzburg L, Silvers A. 1989. Extreme event risk analysis for age-structured populations. *Ecol Model* 47:175–187.
36. Burgman M, Akcakaya HR, Loew SS. 1988. The use of extinction models in species conservation. *Biol Conserv* 43:9–25.
37. Pastorok RA. 2003. Introduction: Improving chemical risk assessments through ecological modeling. *Hum Ecol Risk Assess* 9:885–888.
38. Pastorok RA, Akcakaya HR, Regan H, Ferson S, Bartell SM. 2003. Role of ecological modeling in risk assessment. *Hum Ecol Risk Assess* 9:939–972.
39. Pastorok RA, Bartell SM, Ferson S, Ginzburg LR. 2002. *Ecological Modeling in Risk Assessment: Chemical Effects on Populations, Ecosystems, and Landscapes.* Lewis, Boca Raton, FL, USA.
40. Bartell SM, Gardner RH, Oeill RV. 1988. An integrated fates and effects model for estimation of risk in aquatic systems. In *Aquatic Toxicology and Hazard Assessment,* Vol 10. STP 971. American Society for Testing and Materials, Philadelphia, PA, pp 261–274.
41. Burgman MS, Ferson S, Akcakaya HR. 1993. *Risk Assessment in Conservation Biology.* Chapman & Hall, London, UK.
42. Tanaka Y. 2003. Ecological risk assessment of pollutant chemicals: Extinction risk based on population-level effects. *Chemosphere* 53:421–425.
43. Lande R. 1993. Risk of population extinction from demographic and environmental stochastic and random catastrophes. *Am Nat* 142:911–927.
44. Sibly RM, Newton I, Walker CH. 2000. Effects of dieldrin on population growth rates of sparrowhawks 1963–1986. *J Appl Ecol* 37:540–546.
45. Murata M, Iseki N, Masunaga S, Nakanishi J. 2003. Estimation of effects of dioxins and dioxin-like PCBs on wildlife population—A case study on common cormorant. *Chemosphere* 53:337–345.