

Modelling interactions of toxicants and density dependence in wildlife populations

Aafke M. Schipper^{1,*}, Harrie W. M. Hendriks^{1,2}, Matthew J. Kauffman³, A. Jan Hendriks¹ and Mark A. J. Huijbregts¹

¹Department of Environmental Science, Radboud University Nijmegen, Institute for Water and Wetland Research, P.O. Box 9010, 6500 GL, Nijmegen, The Netherlands; ²Department of Applied Stochastics, Radboud University Nijmegen, Institute for Mathematics, Astrophysics and Particle Physics, P.O. Box 9010, 6500 GL, Nijmegen, The Netherlands; and ³Department of Zoology and Physiology, Wyoming Cooperative Fish and Wildlife Research Unit, University of Wyoming, Laramie, WY 82071, USA

Summary

1. A major challenge in the conservation of threatened and endangered species is to predict population decline and design appropriate recovery measures. However, anthropogenic impacts on wildlife populations are notoriously difficult to predict due to potentially nonlinear responses and interactions with natural ecological processes like density dependence.

2. Here, we incorporated both density dependence and anthropogenic stressors in a stage-based matrix population model and parameterized it for a density-dependent population of peregrine falcons *Falco peregrinus* exposed to two anthropogenic toxicants [dichlorodiphenyldichloroethylene (DDE) and polybrominated diphenyl ethers (PBDEs)]. Log-logistic exposure–response relationships were used to translate toxicant concentrations in peregrine falcon eggs to effects on fecundity. Density dependence was modelled as the probability of a nonbreeding bird acquiring a breeding territory as a function of the current number of breeders.

3. The equilibrium size of the population, as represented by the number of breeders, responded nonlinearly to increasing toxicant concentrations, showing a gradual decrease followed by a relatively steep decline. Initially, toxicant-induced reductions in population size were mitigated by an alleviation of the density limitation, that is, an increasing probability of territory acquisition. Once population density was no longer limiting, the toxicant impacts were no longer buffered by an increasing proportion of nonbreeders shifting to the breeding stage, resulting in a strong decrease in the equilibrium number of breeders.

4. Median critical exposure concentrations, that is, median toxicant concentrations in eggs corresponding with an equilibrium population size of zero, were 33 and 46 $\mu\text{g g}^{-1}$ fresh weight for DDE and PBDEs, respectively.

5. *Synthesis and applications.* Our modelling results showed that particular life stages of a density-limited population may be relatively insensitive to toxicant impacts until a critical threshold is crossed. In our study population, toxicant-induced changes were observed in the equilibrium number of nonbreeding rather than breeding birds, suggesting that monitoring efforts including both life stages are needed to timely detect population declines. Further, by combining quantitative exposure–response relationships with a wildlife demographic model, we provided a method to quantify critical toxicant thresholds for wildlife population persistence.

Key-words: chemical pollution, DDE, equilibrium population size, *Falco peregrinus*, multiple stressors, PBDEs, population growth rate, population viability

Introduction

A major challenge in the conservation of threatened and endangered species is to predict population decline and

design appropriate recovery measures (Drake & Griffen 2010; Ovaskainen & Meerson 2010). Population models are commonly used to assess population viability under anthropogenic impacts and expected future environmental conditions (Wiese, Robertson & Gaston 2004; Korsman *et al.* 2012). However, the translation from environmentally

*Correspondence author. E-mail: A.Schipper@science.ru.nl

induced changes in individual performance (growth, reproduction, survival) to population-level consequences is not straightforward. Populations may respond nonlinearly to changes in individual performance, and responses to anthropogenic stressors could be obscured by natural ecological processes like density dependence (Forbes, Sibly & Calow 2001; Forbes & Calow 2002; Hodgson & Townley 2004). Organisms already stressed by crowding or food limitation may show greater susceptibility to additional, abiotic stressors (Linke-Gamenick, Forbes & Sibly 1999; Klok 2008; Salice *et al.* 2011). Alternatively, density dependence could diminish the population-level effects of abiotic stressors, as stressor-induced mortality or reduced reproduction may be compensated for by reduced competition for resources (Moe, Stenseth & Smith 2002; Hooper *et al.* 2003; Noel *et al.* 2006; Salice *et al.* 2011). Due to such confounding factors and nonlinear relationships, population responses to anthropogenic stressors are difficult to assess *a priori* (Forbes, Sibly & Calow 2001; Gui & Grant 2008). Thus, there is a need for mechanistic models relating population dynamics to environmental conditions (André, Hadon & Pecl 2010).

In the present study, we incorporated both anthropogenic stressors and density dependence in a stage-based matrix population model. We parameterized the model for the density-dependent population of peregrine falcons *Falco peregrinus* in California exposed to two anthropogenic toxicants: dichlorodiphenyldichloroethylene (DDE) and polybrominated diphenyl ethers (PBDEs). The aim of our study was twofold: (i) to explore interactions between density dependence and toxicant impacts and (ii) to identify critical toxicant exposure concentrations, that is, exposure concentrations corresponding with extirpation of the population. We used logistic exposure–response curves to translate concentrations of DDE and PBDEs in peregrine falcon eggs to reductions in fecundity. Reduced fecundity rates were incorporated in a stage-based matrix population model comprising three life stages commonly recognized in peregrine falcons: juveniles, nonbreeding birds and breeders (Kauffman, Pollock & Walton 2004). Density dependence was accounted for by modelling the probability of a nonbreeding bird acquiring a breeding territory as a function of the current number of breeders (Kauffman, Pollock & Walton 2004). Toxicant impacts were evaluated based on the population growth rate (λ) and equilibrium population size (N_{eq}), which are considered key variables in population ecology and management (Grant 1998; Forbes & Calow 2002; Sibly & Hone 2002).

Materials and methods

STUDY SPECIES AND TOXICANTS

The peregrine falcon is a charismatic raptor species belonging to the falcon family (Falconidae). In the 1950s and 1960s, dramatic declines were observed in peregrine falcon populations world-wide. In North America, declines were primarily attributed

to DDE, a metabolite of the organochlorine pesticide DDT, which affects reproductive success via eggshell thinning (Newton 1998). Populations have been recovering since restrictions on the application of these chemicals, in some regions aided by extensive rehabilitation efforts (Tordoff & Redig 1997; Newton 1998). However, due to the persistence of DDE in the environment, high residual levels of DDE may still pose a threat to peregrine falcon populations (Jurek 1989; Comrack & Logsdon 2008). In addition, recent increasing environmental concentrations of PBDEs have given rise to concern. PBDEs are widely used as industrial flame retardants and are commonly added to plastics, polyurethane foam, synthetic textiles and electronics (Newsome *et al.* 2010). Because PBDEs are not chemically bound to the polymers that contain them, a fraction may escape during production, use, disposal and recycling and enter the environment (Chen & Hale 2010). Due to the long-term use and the persistence of PBDEs, increasing concentrations are found in various aquatic as well as terrestrial species (Law *et al.* 2003). Toxicological studies suggest that PBDEs have a variety of physiological effects on wildlife, including alterations of hormone levels, organ morphology, growth, neurodevelopment and reproductive success (Chen & Hale 2010). PBDE concentrations currently measured in peregrine falcon eggs are among the highest reported for wildlife (Law *et al.* 2003; Chen & Hale 2010). Recent field observations of peregrine falcon reproduction success in relation to PBDE egg concentrations, as well as recent toxicity test results concerning a related species (American kestrel *Falco sparverius*), suggest that environmental PBDE concentrations are high enough to affect peregrine falcon reproduction (Fernie *et al.* 2009; Johansson *et al.* 2009).

MODEL APPROACH

We modelled the peregrine falcon population in California using a stage-based matrix model including three life stages (Kauffman, Pollock & Walton 2004): juvenile birds that are <1 year old (N_j), nonbreeding birds that are at least 1 year old (N_{nb}) and breeding adults that are at least 2 years old (N_b). Because substantial population recovery efforts took place between 1977 and 1999, including the release of approximately 800 juveniles from captive breeding programmes (Kauffman, Pollock & Walton 2004), the model distinguishes between juvenile falcons originating from the wild (N_{jw}) and juvenile falcons originating from captivity (N_{jc}). The breeding population is approximately closed (Jurek 1989; Wootton & Bell 1992); therefore, movements beyond the borders of the state were not accounted for. Numbers of peregrine falcons in a given year N_{t+1} are calculated from the population in the preceding year N_t supplemented by the number of juveniles from captive breeding released at time $t + 1$, as follows:

$$\begin{bmatrix} N_{jw} \\ N_{jc} \\ N_{nb} \\ N_b \end{bmatrix}_{t+1} = \begin{bmatrix} 0 & 0 & S_{nb}F_tP_{b,t} & S_bF_t \\ 0 & 0 & 0 & 0 \\ S_{jw} & S_{jc} & S_{nb}(1 - P_{b,t}) & 0 \\ 0 & 0 & S_{nb}P_{b,t} & S_b \end{bmatrix}_t \cdot \begin{bmatrix} N_{jw} \\ N_{jc} \\ N_{nb} \\ N_b \end{bmatrix}_t + \begin{bmatrix} 0 \\ N_{jc} \\ 0 \\ 0 \end{bmatrix}_{t+1} \quad \text{eqn 1}$$

where S_{jw} , S_{jc} , S_{nb} and S_b denote the survival rates of wild juveniles, juveniles released from captive breeding, nonbreeding birds and breeding adults, respectively, F_t represents the time-varying number of juveniles produced per breeding adult, and $P_{b,t}$ represents the probability that a nonbreeding bird acquires a territory and becomes a breeder at time t . $P_{b,t}$ is dependent on the current

number of breeders at a given time step, following a logistic function (Kauffman, Pollock & Walton 2004):

$$P_{b,t} = \frac{\exp(x_0 + x_1 N_{b,t})}{1 + \exp(x_0 + x_1 N_{b,t})} \quad \text{eqn 2}$$

where $P_{b,t}$ denotes the probability of a nonbreeding bird acquiring a breeding territory at time t , $N_{b,t}$ represents the number of breeders at time t , and x_0 and x_1 are shape-fitting parameters.

As we did not find evidence of lethal effects of environmentally representative DDE or PBDE concentrations on peregrine falcons or other raptors, we assumed effects on survival to be negligible. We quantified the toxicant effects on reproduction based on log-logistic exposure–response curves (Hendriks & Enserink 1996):

$$F_{i,t} = \frac{F_r}{-\ln\left(1 + \left(\frac{C_{i,t}}{EC50_i}\right)^{\beta_i}\right)} \quad \text{eqn 3}$$

where $F_{i,t}$ denotes the fecundity under exposure to toxicant i , F_r represents the reference fecundity, that is, the fecundity under uncontaminated conditions, $C_{i,t}$ represents the exposure concentration of toxicant i at time t , $EC50_i$ denotes the median effect concentration of toxicant i , that is, a concentration resulting in a 50% reduction in the reproduction, and β_i characterizes the slope of the corresponding exposure–response curve. Assuming independent action of DDE and PBDE, we calculated the combined impact of the two toxicants based on response addition (Plackett & Hewlett 1952; Traas *et al.* 2002) according to:

$$F_t = \prod_{i=1}^{i=2} F_{i,t} \quad \text{eqn 4}$$

where F_t denotes the time-varying fecundity under exposure to both toxicants.

MODEL PARAMETERIZATION

Ecological parameters

The ecological parameters in the model (survival rates, probability of territory acquisition) were based on data specific to the peregrine falcon population in California (Table 1). Survival rates of the three life stages (including juveniles from wild and captive origin) were derived from encounter history files of California peregrine falcons from 1977 to 1999 (Kauffman, Frick & Linthicum 2003). The function describing the probability of territory acquisition in relation to the number of breeders (Fig. 1) was parameterized by minimizing the sum of the squared residuals between the numbers of breeding adults modelled and observed from 1982 to 1992. From the late 1970s, systematic surveys of breeding adults have been conducted, involving annual and state-wide monitoring by observers both in helicopters and on the ground (Jurek 1989; SCPBRG 2011). Therefore, the monitoring data of breeding adults used to parameterize the density dependence curve (see Table S1, Supporting Information) are expected to represent a fairly complete account of this approximately closed breeding population. To establish the density dependence curve, we ran our model based on year-specific peregrine falcon fecundity rates observed in California from 1981 to 1991 (Table S1, Supporting Information). Population-specific census data

were used to quantify the initial population size, that is, the numbers of juveniles and breeders in 1980, and the numbers of captive-hatched juveniles introduced throughout the modelling period (Table S1, Supporting Information). As census data regarding nonbreeding birds were not available, we estimated the number of nonbreeding birds in 1980 from the juveniles (from wild and captive origin) surviving from 1975 to 1979.

Toxicant impacts

In order to model the impacts of DDE and PBDEs on fecundity (eqns 3 and 4), we sought a value for the reference fecundity (F_r), that is, the fecundity under uncontaminated conditions, as well as values for the median effect concentrations ($EC50$) and slopes of the exposure–response curves (β). Peregrine falcons commonly produce clutches of 3–4 eggs (Burnham, Sandfort & Belthoff 2003; Comrack & Logsdon 2008). Assuming optimal breeding conditions, we applied a reference fecundity F_r of 1.75 young per breeding individual, that is, 3.5 young per breeding pair. Exposure–response curves (Fig. 2) were established by fitting log-logistic curves through reproduction rates in relation to egg concentrations using maximum-likelihood estimation (see Appendices S1–S3, Supporting Information). Unfortunately, we did not find species-specific data suitable to establish response curves, that is, quantitative data on reproductive success along a gradient of toxicant concentrations in eggs. Because differences in sensitivity between species can be large (Hoffman *et al.* 1998) and tend to increase with taxonomic distance (Raimondo, Mineau & Barron 2007), we searched for data from species as closely related as possible (same genus). For DDE, we used field data on DDE egg residues and reproductive success reported for a population of merlins *Falco columbarius* between 1969 and 1973 (Fox 1979). For PBDEs, we used laboratory data of the reproductive success of American kestrels *F. sparverius* exposed to a penta-BDE mixture (DE-71). DE-71 values reported as dose were converted to egg concentrations based on an absorption fraction of 0.18 (McKernan *et al.* 2009).

MODEL TESTING

We tested our model by simulating the fecundity and population size of peregrine falcons in California from 1980 to 2007 in response to concentrations of DDE and various PBDE congeners measured in California peregrine falcon eggs. For PBDEs, we used the sum of the congeners that constitute DE-71 mixture (La Guardia, Hale & Harvey 2006), because the exposure–response curve for PBDEs was based on DE-71 mixture (see above). In case of multiple egg concentrations per year, the geometric mean concentration was calculated. Subsequently, yearly concentrations were log-transformed and linear trend lines were fitted in order to assess the annual exposure concentrations of DDE and PBDEs from 1980 to 2007 (Fig. 3). Exposure concentrations were converted from lipid weight- to fresh weight-based values based on a species-specific egg lipid fraction (Johansson *et al.* 2009).

To evaluate the performance of our model, we compared model with observed fecundity rates and numbers of breeders (Table S1, Supporting Information). The monitoring data regarding fecundity and breeding bird density were previously used to establish the density dependence curve (see above). However, these data can also be used to test the toxicological component of the model, because the density dependence curve was fitted

Table 1. Ecological and toxicological parameters (most likely values and 90% confidence intervals) used to model impacts of DDE and PBDEs on the density-dependent peregrine falcon population in California

| Model parameter (unit)* | Symbol | Value | 90% CI | | Additional information† |
|---|----------------|----------------|----------------|----------------|---|
| | | | 5% | 95% | |
| Survival rate wild juveniles ($n\ n^{-1}\ year^{-1}$) | S_{fw} | 0.38 | 0.25 | 0.50 | Survival rates are based on 718 encounter history files from California from 1977 through 1999 [1] |
| Survival rate juveniles released from captive breeding ($n\ n^{-1}\ year^{-1}$) | S_{jc} | 0.24 | 0.11 | 0.37 | |
| Survival rate non-breeding birds ($n\ n^{-1}\ year^{-1}$) | S_{nb} | 0.86 | 0.75 | 0.97 | |
| Survival rate breeding adults ($n\ n^{-1}\ year^{-1}$) | S_b | 0.86 | 0.82 | 0.90 | |
| Reference fecundity ($n\ n^{-1}\ year^{-1}$) | F_r | 1.75 | – | – | Based on a commonly observed clutch size of 3–4 eggs [2], assuming maximum breeding success |
| Shape-fitting parameters to describe the probability of territory acquisition (–) | x_0 x_1 | 2.36 –0.014 | 1.10 –0.045 | 10.1 –0.012 | Obtained by minimizing the sum of the squared errors between the simulated number of breeders and the number of breeders observed from 1982 to 1992 |
| Slope of DDE exposure trend ($\mu g\ g^{-1}\ year^{-1}\ lw$) | a | –0.12 | –0.17 | –0.066 | Based on DDE concentrations measured in Californian peregrine falcon eggs ($n = 23$) from 1978 to 1988 [3] |
| Intercept of DDE exposure trend ($\mu g\ g^{-1}\ lw$) | b | 238 | 136 | 340 | |
| Slope of PBDE exposure trend ($\mu g\ g^{-1}\ year^{-1}\ lw$) | a | 0.12 | 0.079 | 0.15 | Based on DE-71 concentrations measured in Californian peregrine falcon eggs ($n = 90$) from 1986 through 2007 [4] |
| Intercept of PBDE exposure trend ($\mu g\ g^{-1}\ lw$) | b | –229 | –303 | –156 | |
| Lipid content of eggs (%) | f_{lipid} | 5.8 | 4.1 | 7.9 | Geometric mean and range of lipid percentages measured in peregrine falcon eggs ($n = 52$) in Sweden from 1987 to 1999 [5] |
| Median effect concentration DDE ($\mu g\ g^{-1}\ fw$) | $EC50$ | 8.4 | 5.4 | 15 | Based on reproduction rates of merlins <i>Falco columbarius</i> in relation to DDE egg concentrations [6] |
| Slope of DDE response curve (–) | β | 1.3 | 0.82 | 2.6 | |
| Median effect concentration PBDE ($\mu g\ g^{-1}\ fw$) | $EC50$ | 6.7 | 3.1 | 45 | Based on reproduction rates of American kestrels <i>Falco sparverius</i> exposed to DE-71 mixture [7] |
| Slope of PBDE response curve (–) | β | 1.8 | 1.2 | 3.3 | |

*fw = fresh weight; lw = lipid weight.

†[1] = Kauffman, Frick & Linthicum (2003); [2] = Burnham *et al.* (2003), Comrack & Logsdon (2008); [3] = Peakall *et al.* (1983), Jarman *et al.* (1993); [4] = Park *et al.* (2009); [5] = Johansson *et al.* (2009); [6] = Fox (1979); [7] = Fernie *et al.* (2009), McKernan *et al.* (2009).

based on population-specific data regarding fecundity, whereas the model performance was evaluated based on fecundity rates calculated from the reference fecundity F_r modified according to the magnitude of the toxicant impacts (eqns 3 and 4).

MODEL APPLICATION

After model parameterization and testing, we applied our model to explore the effects of DDE and PBDEs on our density-dependent study population. We first evaluated the effects on the population growth rate (λ), which is considered a key variable in population ecology (Forbes & Calow 2002; Sibly & Hone 2002). However, if a density-dependent population at equilibrium (i.e. $\lambda = 1$) is exposed to a toxicant, the resulting reduction in population growth rate and hence population size will lessen the suppressing effects of density limitation, which in turn will cause λ to return to 1 and the population to stabilize at a different

equilibrium size (Grant 1998; Forbes, Sibly & Calow 2001). Therefore, we evaluated the toxicant impacts also based on the equilibrium size of the population (N_{eq}). We quantified the equilibrium population size as the number of breeding birds in order to link up with the focus in census efforts. Further, we identified critical concentrations (C_{crit}) of the toxicants, which we defined as concentrations corresponding to $N_{eq} = 0$, that is, extirpation of the population.

To assess the sensitivities of N_{eq} and C_{crit} to uncertainties in the underlying parameters, we performed Monte Carlo simulation (10 000 trials). Probability distributions and the mutual correlations of the parameters determining N_{eq} and C_{crit} are provided in the Supporting Information (Table S2). The contribution of the uncertainty in each parameter to the uncertainty in the outcome was based on rank correlations between the input and output parameters, corrected for mutual correlations among the input parameters (Hamby 1994).

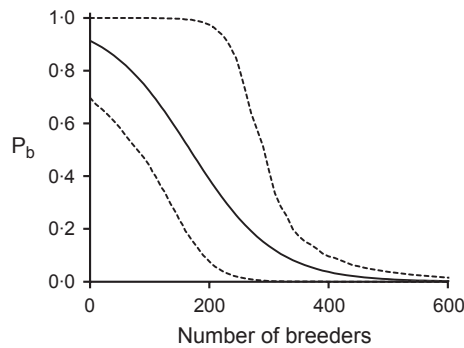


Fig. 1. The probability of territory acquisition (P_b) of peregrine falcons in California as function of the current number of breeders. The solid line represents the most likely curve; dotted lines represent the 90% confidence limits.

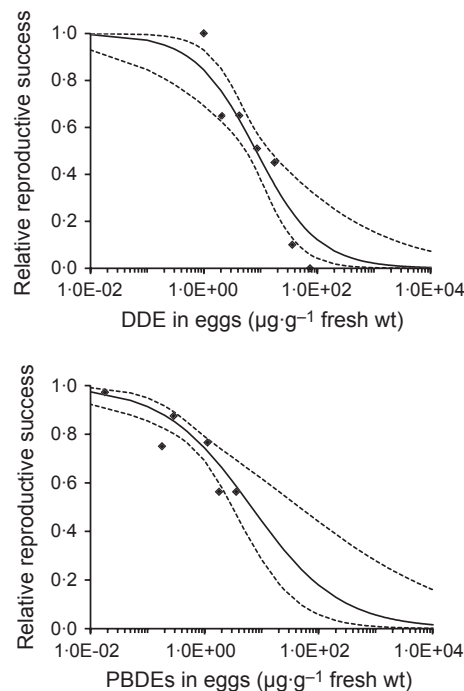


Fig. 2. Exposure–response curves showing the relative reproductive success in relation to the toxicant concentration in eggs for merlin *Falco columbarius* exposed to DDE (top) and American kestrel *Falco sparverius* exposed to PBDEs (bottom). The most likely curves are represented by solid lines, and 90% confidence limits are indicated by dotted lines. Data for DDE are obtained from Fox (1979); data for PBDEs are obtained from Fernie *et al.* (2009) and McKernan *et al.* (2009). PBDEs are represented by DE-71 mixture.

Results

MODEL PERFORMANCE

Increasing trends in peregrine falcon fecundity and numbers of breeding birds observed in California from 1981 to 1997 were well reflected by the model (Figs 4 and 5). These increasing trends mirrored the decrease in the DDE exposure concentrations (Fig. 3), as DDE impacts on

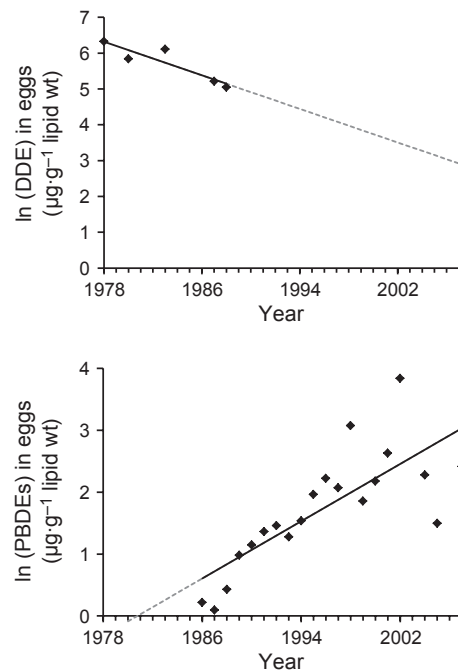


Fig. 3. Concentration trends of DDE ($\mu\text{g g}^{-1}$ lipid weight; top) and PBDEs ($\mu\text{g g}^{-1}$ lipid weight; bottom) in peregrine falcon eggs in California from 1978 to 2007. Data for DDE are obtained from Peakall *et al.* (1983) and Jarman *et al.* (1993); data for PBDEs are obtained from Park *et al.* (2009). PBDEs comprise congeners present in DE-71 mixture.

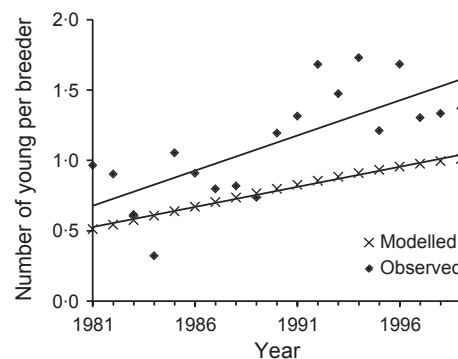


Fig. 4. Fecundity (number of offspring per breeder) of the peregrine falcon population in California from 1981 to 1999. Monitoring data pertain to falcons in rural habitats and are obtained from Kauffman, Frick and Linthicum (2003) (Table S1, Supporting Information). Lines represent linear trends fitted through the data points.

reproduction were larger than PBDE impacts. Yet, fecundity rates and numbers of breeders simulated tended to be somewhat lower than the observations (Figs 4 and 5), suggesting an overestimation of the toxicants' impact.

INTERACTIONS BETWEEN TOXICANT IMPACTS AND DENSITY DEPENDENCE

Population growth rate (λ) was clearly influenced by both toxicant exposure concentrations and population density.

Toxicant impacts on population growth rate lessened with increasing population density (Fig. 6), as population growth at higher densities became increasingly limited by density dependence (Fig. 1). The maximum equilibrium population size (i.e. N_{eq} in absence of toxicant impacts) comprised 386 breeding adults (90% confidence interval 211–506; Fig. 6). This corresponds to 193 breeding pairs, which is slightly more than the 120 to 173 pairs reported as historic breeding population across California (Kauffman, Pollock & Walton 2004). The equilibrium

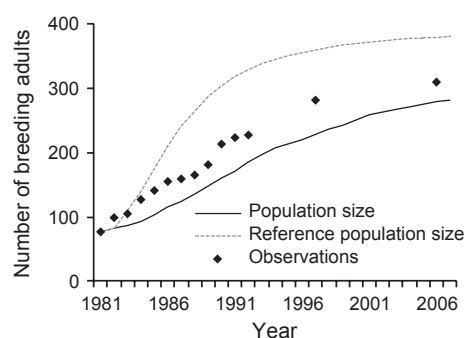


Fig. 5. Numbers of breeding peregrine falcons in California simulated from 1980 to 2007 in response to reproductive effects of DDE and PBDEs (solid line). Simulations without toxicant impacts, that is, based on the reference fecundity F_r , are included for comparison (dotted line). Observations were obtained from Kauffman, Pollock and Walton (2004), Comrack and Logsdon (2008), and the Santa Cruz Predatory Bird Research Group [SCPBRG 2011], see also Table S1, Supporting Information].

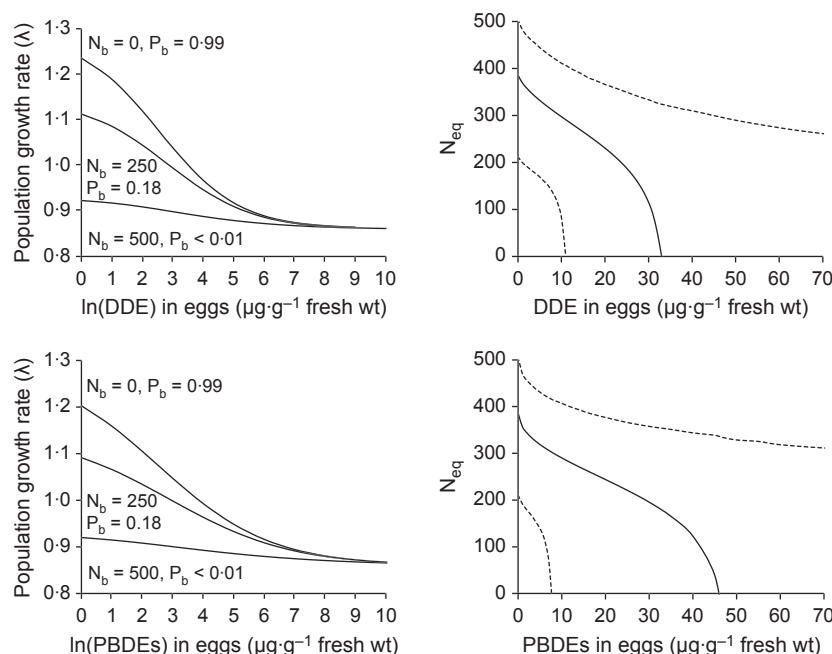


Fig. 6. The intrinsic rate of increase (λ ; left) and the equilibrium population size (N_{eq} ; right) of the peregrine falcon population in California in relation to egg concentrations of DDE ($\mu\text{g g}^{-1}$ fresh weight; top) and PBDEs ($\mu\text{g g}^{-1}$ fresh weight; bottom). N_{eq} represents the number of breeding birds. PBDEs comprise congeners present in DE-71 mixture. N_b = number of breeders; P_b = probability of a non-breeding bird acquiring a breeding territory. Dotted lines represent the 90% confidence limits.

population size responded nonlinearly to increasing toxicant concentrations, showing a gradual decrease followed by a relatively steep decline (Fig. 6).

CRITICAL EXPOSURE CONCENTRATIONS

Median critical exposure concentrations, that is, median concentrations corresponding with $N_{eq} = 0$, were 33 (11–271) and 46 (8–1793) $\mu\text{g g}^{-1}$ fresh weight egg for DDE and PBDEs, respectively, thus being considerably higher than the concentrations resulting in a 50% reduction in the reproduction (Table 1). Critical exposure concentrations were particularly sensitive to uncertainties in the exposure–response curves, followed by uncertainties in the survival rates of juveniles and breeding birds (Table 2; Fig. S1, Supporting Information). The uncertainty in the critical concentration was larger for PBDEs than for DDE, reflecting the larger uncertainty in the PBDE exposure–response curve due to a lack of response data at high concentrations (Fig. 2).

Discussion

MODEL PERFORMANCE

Using a stage-based matrix population model, we modelled the impacts of DDE and PBDEs on the density-dependent peregrine falcon population in California. Because population parameters as well as egg concentrations of DDE and PBDEs have been reported for this

Table 2. Sensitivity of the critical concentrations of DDE and PBDEs to uncertainties in the underlying model parameters. Probability distributions and the mutual correlations of these parameters are provided in the Supporting Information (Table S2)

| Model parameter (unit) | Symbol | Contribution to uncertainty in C_{crit} (%) | |
|---|----------|---|-------|
| | | DDE | PBDEs |
| Median effect concentration ($\mu\text{g g}^{-1}$ fw) | $EC50$ | 16 | 25 |
| Slope of exposure–response curve | β | 26 | 36 |
| Reference fecundity ($\text{n n}^{-1} \text{ year}^{-1}$) | F_r | 9.1 | 5.7 |
| Shape-fitting parameters to describe the probability of territory acquisition | x_0 | 0.52 | 0.12 |
| | x_1 | 0.52 | 0.71 |
| Survival rate wild juveniles ($\text{n n}^{-1} \text{ year}^{-1}$) | S_b | 21 | 15 |
| Survival rate nonbreeding birds ($\text{n n}^{-1} \text{ year}^{-1}$) | S_{pw} | 8.4 | 5.5 |
| Survival rate breeding adults ($\text{n n}^{-1} \text{ year}^{-1}$) | S_{nb} | 18 | 12 |

population (Peakall *et al.* 1983; Jarman *et al.* 1993; Kauffman, Frick & Linthicum 2003; Kauffman, Pollock & Walton 2004; Comrack & Logsdon 2008; Park *et al.* 2009; SCPBRG 2011), we were able to parameterize and test the model based on species- and region-specific data regarding survival, the probability of territory acquisition and toxicant exposure concentrations. Yet, fecundity rates and numbers of breeding adults simulated by our model were somewhat lower than the values observed (Figs 4 and 5), suggesting an overestimation of the toxicants' impact. This seems particularly relevant for DDE, because the exposure–response curve for DDE was based on field data, which may incorporate fecundity reductions due to multiple chemical and nonchemical stressors other than the toxicant of concern (Korsman *et al.* 2012). However, the larger the influence of a stressor, the more reliable the field-based response curve is. The DDE egg concentrations and the reproductive success rates used to establish the exposure–response curve were both strongly related to eggshell thickness, indicating that DDE was the dominant stressor primarily responsible for the effects on reproduction (Fox 1979). The use of toxicity data for species other than peregrine falcons may also have resulted in uncertainties in the toxicological part of the model. In general, the uncertainty associated with extrapolating toxicity data from one species to another increases with taxonomic distance (Raimondo, Mineau & Barron 2007). As both merlin and American kestrel belong to the same genus as the peregrine falcon, the taxonomic distance in this instance is as small as possible. Moreover, effects of DDE on

eggshell thickness are similar for peregrines and merlins (Fyfe *et al.* 1988), indicating that the exposure–response curve is sufficiently representative. Alternatively, the overestimated toxicant impacts could result from uncertainties in the exposure concentrations. Measurements of DDE concentrations were biased towards eggs originating from coastal habitats (Jarman *et al.* 1993), where DDE impacts on peregrine falcons have been larger than elsewhere in California (Wootton & Bell 1992). Furthermore, we had DDE egg concentration measurements available up to 1988 only (Fig. 3). It is possible that DDE residues in eggs have been declining faster than the loglinear temporal trend assumed in our model, which may explain why the increase in the fecundity rates over time was more pronounced for the observations than for our model results (Fig. 4). Given the uncertainties in the exposure concentration trends, our model should not be used to project the toxicant response of the peregrine falcon population in California. However, as the toxicological data for the other falcon species ought to be sufficiently representative, and given that we used population-specific data for the ecological parameters, the model can be profitably used to explore the interactions between toxicant impacts and density limitation.

INTERACTIONS BETWEEN TOXICANT IMPACTS AND DENSITY DEPENDENCE

Our results revealed that toxicant impacts on the peregrine population growth rate differed between high and low population densities (Fig. 6). At low densities, the probability of acquiring a breeding territory was high, implying that the loss of young birds due to toxicant exposure was directly affecting the population growth rate. Contrastingly, at higher densities, the probability of acquiring a breeding territory decreased. Hence, young birds lost due to toxicant exposure were unlikely to breed anyway, resulting in a limited effect on population growth rate (Fig. 6). Thus, the toxicant impacts on population growth rate were buffered at high densities, corresponding with a so-called less-than-additive joint effect of toxicants and density (Forbes, Sibly & Calow 2001). Similar results have been found in previous research pertaining to interactions between density dependence and toxicant impacts, although underlying mechanisms were different (Forbes, Sibly & Linke-Gamenick 2003; Noel *et al.* 2006; Van der Heide *et al.* 2010; Menezes-Oliveira *et al.* 2011). Our results further showed a nonlinear response of the equilibrium number of breeders to increasing toxicant concentrations (Fig. 6). Initially, toxicant-induced reductions in population size were mitigated by an alleviation of the density limitation, that is, an increasing probability of territory acquisition. This was reflected by a clear decrease in the number of nonbreeding birds (Fig. S2). Yet, once the probability of acquiring a breeding territory approached a maximum, toxicant impacts were no longer buffered by an increasing proportion of nonbreeders shifting to the breed-

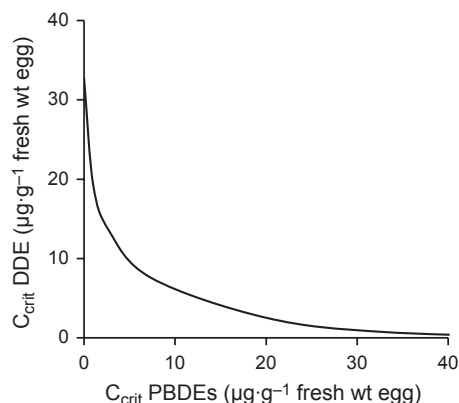


Fig. 7. Critical concentrations of DDEs ($\mu\text{g g}^{-1}$ fresh weight egg) in relation to critical concentrations of PBDEs ($\mu\text{g g}^{-1}$ fresh weight egg), based on the assumption of independent action of the two toxicants (eqn 4). PBDEs comprise congeners present in DE-71 mixture.

ing stage, and a steep decline of the equilibrium number of breeders was observed (Fig. 6, Fig. S2, Supporting Information). A similar nonlinear response of equilibrium population size has been found in a lab experiment exposing springtails *Folsomia candida* to the antiparasitic agent ivermectin (Noel *et al.* 2006). However, a recent modelling study of density-dependent fathead minnow *Pimephales promelas* and brook trout *Salvelinus fontinalis* populations in relation to zinc exposure showed a linear response of equilibrium population size, reflecting the more or less linear response of individual reproduction in the range of zinc concentrations investigated (Hayashi, Kamo & Tanaka 2009). This suggests that these modelled fish populations may have been much less limited by density.

CRITICAL EXPOSURE CONCENTRATIONS

Although the 90% confidence interval of our critical DDE exposure concentration (11–271 $\mu\text{g g}^{-1}$ fresh weight egg) encompassed the critical level of 15–20 $\mu\text{g g}^{-1}$ fresh weight egg that is commonly associated with peregrine falcon population decline (Fyfe *et al.* 1988; Peakall & Kiff 1988), our median critical concentration of 33 $\mu\text{g g}^{-1}$ is relatively high. However, declines in population size may occur well below this level (Fig. S2) and could even result in extirpation before reaching the level corresponding with $N_{\text{eq}} = 0$, as environmental and demographic stochasticity may drive a population to extinction once it has declined below a minimum viable size (Fagan & Holmes 2006). Thus, the critical concentrations identified in our study are liberal and unlikely to provide a sufficiently early warning of significant toxicant impact. An alternative would be to identify the exposure concentration corresponding with the minimum viable population (MVP) size. However, because quantifying the actual MVP is not straightforward and requires a lot of detailed data, a pragmatic solution would be to apply a safety factor to the critical concentrations (Hanson & Stark 2012).

IMPLICATIONS

In general, our modelling results showed that particular life stages of a density-limited population may be relatively insensitive to toxicant impacts until a critical threshold is crossed. In our study population, toxicant-induced changes were observed in the equilibrium number of non-breeding rather than breeding birds (Fig. S2), suggesting that monitoring efforts including both life stages are needed to detect population declines sufficiently early. In addition, we showed how the integration of a matrix population model and quantitative exposure–response relationships facilitates the identification of critical environmental contamination levels influencing population persistence. Our approach is suited to incorporate the influences of multiple simultaneously operating stressors and evaluate how the critical environmental threshold of one stressor changes due to the other stressors present (Fig. 7). Future improvements may include the incorporation of environmental and demographic stochasticity, which may have considerable influence on population persistence particularly if a population is small (Ovaskainen & Meerson 2010; Torang, Ehrlén & Agren 2010). A relatively large amount of data is needed, however, for the model parameterization, including not only data regarding relevant density-dependent demographic rates of the species of concern, like fecundity and survival, but also information to quantify the median response values (e.g. *EC*₅₀) and slope constants of the stressor–response curves. Because these data are not readily available for all stressors and species of interest (Hendriks & Enserink 1996; Korsman *et al.* 2012), the applicability of the model may be limited to well-studied species. However, efforts to integrate ecotoxicology and wildlife demography are particularly relevant in the face of current levels of anthropogenic impacts on biodiversity.

Acknowledgements

This research project was funded by the European Commission under the Seventh Framework Programme on Environment, ENV.2007.3.3.1.1: CADASTER – in silico techniques for hazard, safety, and environmental risk assessment (grant agreement number 2126687). We thank three anonymous reviewers for their thoughtful suggestions to improve this paper. Any mention of trade, product or firm names is for descriptive purposes only and does not imply endorsement by the U.S. Government.

References

- André, J., Haddon, M. & Pecl, G.T. (2010) Modelling climate-change-induced nonlinear thresholds in cephalopod population dynamics. *Global Change Biology*, **16**, 2866–2875.
- Burnham, W., Sandfort, C. & Belthoff, J.R. (2003) Peregrine Falcon eggs: egg size, hatchling sex, and clutch sex ratios. *Condor*, **105**, 327–335.
- Chen, D. & Hale, R.C. (2010) A global review of polybrominated diphenyl ether flame retardant contamination in birds. *Environment International*, **36**, 800–811.
- Comrack, L.A. & Logsdon, R.J. (2008) *Status Review of the American Peregrine Falcon (Falco Peregrinus Anatum)* in California. The Resources Agency, Department of Fish and Game, State of California.

- Drake, J.M. & Griffen, B.D. (2010) Early warning signals of extinction in deteriorating environments. *Nature*, **467**, 456–459.
- Fagan, W.F. & Holmes, E.E. (2006) Quantifying the extinction vortex. *Ecology Letters*, **9**, 51–60.
- Fernie, K.J., Shutt, J.L., Letcher, R.J., Ritchie, I.J. & Bird, D.M. (2009) Environmentally relevant concentrations of DE-71 and HBCD alter eggshell thickness and reproductive success of American kestrels. *Environmental Science & Technology*, **43**, 2124–2130.
- Forbes, V.E. & Calow, P. (2002) Population growth rate as a basis for ecological risk assessment of toxic chemicals. *Philosophical Transactions of the Royal Society of London Series B-Biological Sciences*, **357**, 1299–1306.
- Forbes, V.E., Sibly, R.M. & Calow, P. (2001) Toxicant impacts on density-limited populations: a critical review of theory, practice, and results. *Ecological Applications*, **11**, 1249–1257.
- Forbes, V.E., Sibly, R.M. & Linke-Gamenick, I. (2003) Joint effects of population density and toxicant exposure on population dynamics of *Capitella* sp. I. *Ecological Applications*, **13**, 1094–1103.
- Fox, G.A. (1979) A simple method of predicting DDE contamination and reproductive success of populations of DDE-sensitive species. *Journal of Applied Ecology*, **16**, 737–741.
- Fyfe, R.W., Risebrough, R.W., Monk, J.G., Jarman, W.M., Anderson, D.W., Kiff, L.F., Lincer, J.L., Nisbet, I.C., Walker, W. & Walton, B.J. (1988) DDE, productivity and eggshell thickness relationships in the genus *Falco*. *Peregrine Falcon Populations, their Management and Recovery* (eds T.J. Cade, J.H. Enderson, C.G. Thelander & C.M. White), The Peregrine Fund, Boise, Idaho.
- Grant, A. (1998) Population consequences of chronic toxicity: incorporating density dependence into the analysis of life table response experiments. *Ecological Modelling*, **105**, 325–335.
- Gui, Y. & Grant, A. (2008) Joint effects of density dependence and toxicant exposure on *Drosophila melanogaster* populations. *Ecotoxicology and Environmental Safety*, **70**, 236–243.
- Hamby, D.M. (1994) A review of techniques for parameter sensitivity analysis of environmental models. *Environmental Monitoring and Assessment*, **32**, 135–154.
- Hanson, N. & Stark, J.D. (2012) Comparison of population level and individual level endpoints to evaluate ecological risk of chemicals. *Environmental Science & Technology*, **46**, 5590–5598.
- Hayashi, T.I., Kamo, M. & Tanaka, Y. (2009) Population-level ecological effect assessment: estimating the effect of toxic chemicals on density-dependent populations. *Ecological Research*, **24**, 945–954.
- Hendriks, A.J. & Enserink, E.L. (1996) Modelling response of single-species populations to microcontaminants as a function of species size with examples for waterfleas (*Daphnia magna*) and cormorants (*Phalacrocorax carbo*). *Ecological Modelling*, **88**, 247–262.
- Hodgson, D.J. & Townley, S. (2004) Linking management changes to population dynamic responses: the transfer function of a projection matrix perturbation. *Journal of Applied Ecology*, **41**, 1155–1161.
- Hoffman, D.J., Melancon, M.J., Klein, P.N., Eisemann, J.D. & Spann, J.W. (1998) Comparative developmental toxicity of planar polychlorinated biphenyl congeners in chickens, American kestrels, and common terns. *Environmental Toxicology and Chemistry*, **17**, 747–757.
- Hooper, H.L., Sibly, R.M., Maund, S.J. & Hutchinson, T.H. (2003) The joint effects of larval density and C-14-cypermethrin on the life history and population growth rate of the midge *Chironomus riparius*. *Journal of Applied Ecology*, **40**, 1049–1059.
- Jarman, W.M., Burns, S.A., Chang, R.R., Stephens, R.D., Norstrom, R.J., Simon, M. & Linthicum, J. (1993) Determination of PCDDs, PCDFs, and PCBs in California peregrine falcons (*F. peregrinus*) and their eggs. *Environmental Toxicology and Chemistry*, **12**, 105–114.
- Johansson, A.K., Sellstrom, U., Lindberg, P., Bignert, A. & de Wit, C.A. (2009) Polybrominated diphenyl ether congener patterns, hexabromocyclododecane, and brominated biphenyl 153 in eggs of peregrine falcons (*F. peregrinus*) breeding in Sweden. *Environmental Toxicology and Chemistry*, **28**, 9–17.
- Jurek, R.M. (1989) *Five-Year Status Report American Peregrine Falcon*. Wildlife Management Division, California Department of Fish and Game, Sacramento.
- Kauffman, M.J., Frick, W.F. & Linthicum, J. (2003) Estimation of habitat-specific demography and population growth for peregrine falcons in California. *Ecological Applications*, **13**, 1802–1816.
- Kauffman, M.J., Pollock, J.F. & Walton, B. (2004) Spatial structure, dispersal, and management of a recovering raptor population. *American Naturalist*, **164**, 582–597.
- Klok, C. (2008) Gaining insight in the interaction of zinc and population density with a combined dynamic energy budget and population model. *Environmental Science & Technology*, **42**, 8803–8808.
- Korsman, J.C., Schipper, A.M., Lenders, H.J.R., Foppen, R.P.B. & Hendriks, A.J. (2012) Modelling the impact of toxic and disturbance stress on white-tailed eagle (*Haliaeetus albicilla*) populations. *Ecotoxicology*, **21**, 27–36.
- La Guardia, M.J., Hale, R.C. & Harvey, E. (2006) Detailed polybrominated diphenyl ether (PBDE) congener composition of the widely used penta-, octa-, and deca-PBDE technical flame-retardant mixtures. *Environmental Science & Technology*, **40**, 6247–6254.
- Law, R.J., Alae, M., Allchin, C.R., Boon, J.P., Lebeuf, M., Lepom, P. & Stern, G.A. (2003) Levels and trends of polybrominated diphenylethers and other brominated flame retardants in wildlife. *Environment International*, **29**, 757–770.
- Linke-Gamenick, I., Forbes, V.E. & Sibly, R.M. (1999) Density-dependent effects of a toxicant on life-history traits and population dynamics of a capitellid polychaete. *Marine Ecology-Progress Series*, **184**, 139–148.
- McKernan, M.A., Rattner, B.A., Hale, R.C. & Ottinger, M.A. (2009) Toxicity of polybrominated diphenyl ethers (DE-71) in chicken (*Gallus gallus*), mallard (*Anas platyrhynchos*), and American kestrel (*Falco sparverius*) embryos and hatchlings. *Environmental Toxicology and Chemistry*, **28**, 1007–1017.
- Menezes-Oliveira, V.B., Scott-Fordsmand, J.J., Rocco, A., Soares, A. & Amorim, M.J.B. (2011) Interaction between density and Cu toxicity for *Enchytraeus crypticus* and *Eisenia fetida* reflecting field scenarios. *Science of the Total Environment*, **409**, 3370–3374.
- Moe, S.J., Stenseth, N.C. & Smith, R.H. (2002) Density-dependent compensation in blowfly populations give indirectly positive effects of a toxicant. *Ecology*, **83**, 1597–1603.
- Newsome, S.D., Park, J.S., Henry, B.W., Holden, A., Fogel, M.L., Linthicum, J., Chu, V. & Hooper, K. (2010) Polybrominated diphenyl ether (PBDE) levels in peregrine falcon (*F. peregrinus*) eggs from California correlate with diet and human population density. *Environmental Science & Technology*, **44**, 5248–5255.
- Newton, I. (1998) *Population Limitation in Birds*. Academic Press, London.
- Noel, H.L., Hopkin, S.P., Hutchinson, T.H., Williams, T.D. & Sibly, R.M. (2006) Population growth rate and carrying capacity for spring-tails *Folsomia candida* exposed to ivermectin. *Ecological Applications*, **16**, 656–665.
- Ovaskainen, O. & Meerson, B. (2010) Stochastic models of population extinction. *Trends in Ecology & Evolution*, **25**, 643–652.
- Park, J.S., Holden, A., Chu, V., Kim, M., Rhee, A., Patel, P., Shi, Y.T., Linthicum, J., Walton, B.J., McKeown, K., Jewell, N.P. & Hooper, K. (2009) Time-trends and congener profiles of PBDEs and PCBs in California peregrine falcons (*F. peregrinus*). *Environmental Science & Technology*, **43**, 8744–8751.
- Peakall, D.B. & Kiff, L.F. (1988) DDE contamination in peregrines and American kestrels and its effect on reproduction. *Peregrine Falcon Populations: their Management And Recovery* (eds T.J. Cade, J.H. Enderson, C.G. Thelander & C.M. White), The Peregrine Fund, Boise, Idaho.
- Peakall, D.B., Lew, T.S., Springer, A.M., Walker, W., Risebrough, R.W., Monk, J.G., Jarman, W.M., Walton, B.J., Reynolds, L.M., Fyfe, R.W. & Kiff, L.F. (1983) Determination of the DDE and PCB contents of peregrine falcon eggs - a comparison of whole egg measurements and estimates derived from eggshell membranes. *Archives of Environmental Contamination and Toxicology*, **12**, 523–528.
- Plackett, R.L. & Hewlett, P.S. (1952) Quantal responses to mixtures of poisons. *Journal of the Royal Statistical Society Series B-Statistical Methodology*, **14**, 141–163.
- Raimondo, S., Mineau, P. & Barron, M.G. (2007) Estimation of chemical toxicity to wildlife species using interspecies correlation models. *Environmental Science & Technology*, **41**, 5888–5894.
- Salice, C.J., Rowe, C.L., Pechmann, J.H.K. & Hopkins, W.A. (2011) Multiple stressors and complex life cycles: insights from a population-level assessment of breeding site contamination and terrestrial habitat loss in an amphibian. *Environmental Toxicology and Chemistry*, **30**, 2874–2882.
- SCPCBRG (2011) <http://www2.ucsc.edu/scpcbrg/>. Santa Cruz Predatory Bird Research Group. Santa Cruz.
- Sibly, R.M. & Hone, J. (2002) Population growth rate and its determinants: an overview. *Philosophical Transactions of the Royal Society of London Series B-Biological Sciences*, **357**, 1153–1170.

- Torang, P., Ehrlen, J. & Agren, J. (2010) Linking environmental and demographic data to predict future population viability of a perennial herb. *Oecologia*, **163**, 99–109.
- Tordoff, H.B. & Redig, P.T. (1997) Midwest peregrine falcon demography, 1982–1995. *Journal of Raptor Research*, **31**, 339–346.
- Traas, T.P., Van de Meent, D., Posthuma, L., Hamers, T., Kater, B.J., De Zwart, D. & Aldenberg, T. (2002) The potentially affected fraction as a measure of ecological risk. *Species Sensitivity Distributions in Ecotoxicology* (eds L. Posthuma, G.W.I. Suter & T.P. Traas), pp. 315–344. Lewis Publishers, Boca Raton.
- Van der Heide, T., Van Nes, E.H., Van Katwijk, M.M., Scheffer, M., Hendriks, A.J. & Smolders, A.J.P. (2010) Alternative stable states driven by density-dependent toxicity. *Ecosystems*, **13**, 841–850.
- Wiese, F.K., Robertson, G.J. & Gaston, A.J. (2004) Impacts of chronic marine oil pollution and the murre hunt in Newfoundland on thick-billed murre *Uria lomvia* populations in the eastern Canadian Arctic. *Biological Conservation*, **116**, 205–216.
- Wootton, J.T. & Bell, D.A. (1992) A metapopulation model of the peregrine falcon in California - viability and management strategies. *Ecological Applications*, **2**, 307–321.

Received 15 March 2013; accepted 25 June 2013
Handling Editor: Phil Stephens

Supporting Information

Additional Supporting Information may be found in the online version of this article.

Appendix S1. Maximum likelihood estimation of the logistic concentration-response curves.

Appendix S2. Assessing the uncertainty in the logistic parameters a and b .

Appendix S3. Conversion of the logistic parameters a and b to $EC50$ and β .

Table S1. Fecundity and population census data of the peregrine falcon population in California (1975–2007).

Table S2. Probability distributions of model parameters influencing the response of the peregrine falcon equilibrium population size to exposure concentrations of DDE and PBDEs.

Fig. S1. Uncertainties in the relationship between the equilibrium population size and egg concentrations of DDE of the peregrine falcon population in California.

Fig. S2. The equilibrium size of the peregrine falcon population in California in relation to egg concentrations of DDE and PBDEs.