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Using field data to quantify chemical impacts on wildlife population viability

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Abstract. Environmental pollution is an important driver of biodiversity loss. Yet, to date, the effects of chemical exposure on wildlife populations have been quantified for only a few species, mainly due to a lack of appropriate laboratory data to quantify chemical impacts on vital rates. In this study, we developed a method to quantify the effects of toxicant exposure on wildlife population persistence based on field monitoring data. We established field-based vital-rate-response functions for toxicants, using quantile regression to correct for the influences of confounding factors on the vital rates observed, and combined the response curves with population viability modelling. We then applied the method to quantify the impact of DDE on three bird species: the White-tailed Eagle, Bald Eagle, and Osprey. Population viability was expressed via five population extinction vulnerability metrics: population growth rate \( r_1 \), critical patch size (CPS), minimum viable population size (MVP), probability of population extirpation (PE), and median time to population extirpation (MTE). We found that past DDE exposure concentrations increased population extirpation vulnerabilities of all three bird species. For example, at DDE concentrations of 25 mg/kg wet mass of egg (the maximum historic exposure concentration reported in literature for the Osprey), \( r_1 \) became small (White-tailed Eagle and Osprey) or close to zero (Bald Eagle), the CPS increased up to almost the size of Connecticut (White-tailed Eagle and Osprey) or West Virginia (Bald Eagle), the MVP increased up to approximately 90 (White-tailed Eagle and Osprey) or 180 breeding pairs (Bald Eagle), the PE increased up to almost certain extirpation (Bald Eagle) or only slightly elevated levels (White-tailed Eagle and Osprey) and the MTE became within decades (Bald Eagle) or remained longer than a millennium (White-tailed Eagle and Osprey). Our study provides a method to derive species-specific field-based response curves of toxicant exposure, which can be used to assess population extinction vulnerabilities and obtain critical levels of toxicant exposure based on maximum permissible effect levels. This may help conservation managers to better design appropriate habitat restoration and population recovery measures, such as reducing toxicant levels, increasing the area of suitable habitat or reintroducing individuals.

Key words: Bald Eagle; birds of prey; conservation biology; critical patch size; DDE; extinction risk; Osprey; pollutants; population viability analysis; toxicants; White-tailed Eagle; wildlife.

INTRODUCTION

A key goal in conservation biology is the protection of species to ensure stable and viable populations (Shaffer 1981). This is challenging given the increasing human impact on the environment (Purvis and Hector 2000, Johnson et al. 2017). Environmental pollution has been identified as an important driver of current biodiversity loss and its impact is expected to further increase in the future (Maxwell et al. 2016, Young et al. 2016). For example, synthetic pesticides in agricultural practices are predicted to be increasingly used in the next decades because of climate change (Kattwinkel et al. 2011), potentially leading to large biodiversity losses (Beketov et al. 2013, Hallmann et al. 2014, Dudley et al. 2017). To conserve biodiversity more effectively, it is important to quantify the extinction vulnerability of species and populations due to chemical exposure (De Laender et al. 2014, Forbes et al. 2016).

Population viability analysis (PVA) is commonly used to identify and evaluate threats to populations or species and assess their extinction risk (Akçakaya 2000, Stephens 2016). Using PVA to assess the effects of chemicals on extinction risks is challenging because this requires species-specific data on the reduction of the intrinsic population growth rate due to chemical exposure. Chemical effect data are generally obtained via laboratory experiments. However, test results are often expressed as threshold values or point estimates (such as the no-observed-effect concentration), which do not provide adequate information for assessing population-level viability impacts (Forbes et al. 2016). Appropriate population-level laboratory data are available for a limited number of species only, mainly small invertebrates such as Daphnia brachyurum (Tanaka 2003), and only very few mammals and birds (Sibly et al. 2005, Dalkvist et al. 2009, but see, e.g., Roelofs et al. 2005). In addition, the artificial conditions in laboratory experiments and uncertain correction factors to extrapolate lab to field responses (Hill et al. 1994, Traas et al. 1996, Chapman et al. 1998) have raised criticism of the use of laboratory data to investigate effects of chemical exposure on wildlife populations (Chapman 1995, Power and McCarty 1997). As an alternative, field monitoring data may be used (Blus and Henny 1997). However, this requires an appropriate way to correct for the confounding influences of other environmental factors on the organisms’ response (Cade and Noon 2003, Schipper et al. 2014, van Goethem et al. 2015).
The main objective of this study was to develop and apply a method to quantify the effects of toxicant exposure on wildlife population extinction vulnerability. The method integrates population viability modelling with field-based exposure–response curves that relate vital rates (reproduction and survival success) to toxicant exposure levels. To illustrate the method, we quantified the influence of p,p’-dichlorodiphenyldichloroethylene (DDE), a metabolite of the organochlorine insecticide DDT, on the reproduction success and extirpation vulnerability of the White-tailed Eagle (Haliaeetus albicilla), the Bald Eagle (Haliaeetus leucocephalus), and the Osprey (Pandion haliaetus). DDE has been a major cause of declines of several populations of bird species (Carson 1962, Fry 1995, Schipper et al. 2013). With legislated restrictions on usage of DDT in the 1970s, environmental concentrations decreased substantially, leading to partial recovery of populations (Grier 1982, Best et al. 2010, Dykstra et al. 2010). However, some populations in a few regions did not increase as rapidly, primarily due to the high persistency of DDE (Grier 1982, Dykstra et al. 2010). Over the past 40 yr, several field studies have been conducted to investigate the effects of DDE on populations of the White-tailed Eagle, Bald Eagle, and Osprey, resulting in a large availability of field monitoring data suited to derive exposure–response curves representing toxicant impacts on reproduction. We used these response curves to quantify five population extinction vulnerability metrics in relation to increasing DDE concentrations: the population growth rate, the critical patch size (CPS), the minimum viable population (MVP), the probability of population extirpation (PE), and the median time to population extirpation (MTE). The population growth rate, CPS, and MVP are context-independent measures of population viability in the sense that they can be quantified based on species’ intrinsic life-history characteristics and responses to toxicants (such as DDE) without the need to account for population-specific circumstances (e.g., habitat, resources, and other external pressures). Hence, they can be used as first-tier species-specific vulnerability indicators in absence of population-specific data on for example carrying capacity and current population size. PE and MTE are population-specific viability measures that enable us to explore population viability in relation to toxicant exposure conditional on a given population size and carrying capacity. Apart from quantifying these five metrics in relation to increasing concentrations of DDE for each of the three species, we also collected DDE exposure measurements as reported for specific populations and assessed corresponding extirpation vulnerability.

**METHODS**

**Field-based exposure–response curves**

Toxicant effects on vital rates (reproduction and survival) are usually reported separately (Hendriks and Ens erink 1996, Hendriks et al. 2005) and can be quantified using the Hill equation (Hill 1910)

\[ y = \frac{1}{1 + \left(\frac{C}{C_{50}}\right)^b} \] (1)

in which \( y \) is defined as the response (i.e., reproductive or survival success between 0 and 1), \( C_{50} \) is defined as half the maximal effective concentration or inflection point of the curve (e.g., EC50 for reproductive success and LC50 for survival success), \( C \) is the chemical exposure concentration, and \( b \) is the Hill slope coefficient, which reflects the steepness of the curve. To derive exposure–response curves from field data we use quantile regression, which can be applied to filter out the influences of confounding environmental factors on field observations (Cade and Noon 2003, Iwasaki and Ormerod 2012, van Goethem et al. 2015). Most regression techniques relate changes in the mean of a response variable to one or more explanatory variables. With quantile regression, any part of the distribution of a variable can be used as response (Cade and Noon 2003). Quantile regression based on one of the upper boundaries of the response variable distribution (e.g., the 95th quantile) is expected to show the constraints imposed by the explanatory environmental variable of concern, such as chemical exposure (Cade and Noon 2003, Iwasaki and Ormerod 2012). Therefore, we retrieved the exposure–response curves by fitting sigmoid curves (Eq. 1) through the 95th percentile of field data on vital rates (reproduction and survival) in relation to contaminant exposure.

**Population extinction vulnerability indicators**

**Population growth rate \( (r_1) \).**—By definition, the population growth rate of species in a closed system depends on the reproduction and survival success of the population. The growth rate for a population exposed solely to toxicants can then be obtained by combining the reproduction and survival success with the intrinsic population growth rate \( (r_{max}) \) and generation time \( (\tau_g) \) of a species (Hendriks et al. 2005, Korsman et al. 2012)

\[ r_1(C) = r_{max} + \ln\left(\frac{1}{1 + \left[\frac{EC_{50}}{C}\right]^b}\right)/\tau_g + \ln\left(\frac{1}{1 + \left[\frac{LC_{50}}{C}\right]^b}\right)/\tau_g \] (2)

Similarly, toxicants reduce the carrying capacity \( (K) \) of populations. The toxicant-induced reduction of the carrying capacity of a population can be obtained via (Hakoyama et al. 2000, Nakamura et al. 2003, Hendriks et al. 2005)

\[ K(C) = K(0)\frac{r_1(C)}{r_{max}} \] (3)

In turn, chemical exposure will affect the CPS, MVP, PE, and MTE of species populations via the impact on the population growth rate \( r_1 \) and \( K \).

**Critical patch size \( (CPS) \).**—The CPS is the minimum habitat size required for population persistence. To determine the CPS, we used the demographic model of Pereira et al. (2004), of which predictions have been shown to significantly correlate with IUCN Red List threat status (Pereira et al. 2004, Pereira and Daily 2006, Hilbers et al. 2016b). The model presumes that population extinction is in essence a deterministic event primarily driven by habitat area. Due
to edge effects, the per capita loss of individuals via dispersal into non-suitable habitat increases when habitat area decreases. Local extirpation occurs when the habitat area is so small that the intrinsic population growth rate is not able to compensate for dispersal losses (Skellam 1951). This means that there is a minimum area of native habitat, the CPS, below which a population will decline to extirpation due to individuals dispersing into non-suitable habitat (Pereira et al. 2004, Pereira and Daily 2006).

$$\text{CPS} = \pi \cdot \min_{x\sqrt{2\sigma^2/n}} \leq L \cdot \infty \left\{ \frac{1}{(\sigma - 1)\sqrt{1}} J_1 \left( \frac{1}{(\sigma - 1)^2 \sqrt{L}} \right) + \frac{1}{\sigma \sqrt{2}} K_1 \left( \frac{1}{(\sigma - 1)^2 \sqrt{L}} \right) \right\}^{2}$$

where $\sigma$ is the probability of a species moving outside of a native habitat patch at the habitat border (dimensionless), $r_1$ is the population growth rate inside the native habitat patch (per unit of time), $r_2$ is the population growth rate outside the native habitat patch (per unit of time), $\sigma^2$ is the dispersal variance ($\text{km}^2$ per unit of time), $L$ is the diameter of the circular patch of native habitat (km), $J_0$ and $K_0$ are Bessel functions of the first and second kind of order $n$, respectively, and $J_0$ represents the smallest positive root of the Bessel function $J_0(x)$.

Minimum viable population (MVP).—The MVP is the minimum number of individuals within a population required for a certain probability of persistence over a given time frame. To obtain the MVP, we followed the approach of Brook et al. (2006) and Hilbers et al. (2016) by varying the initial population size until the species population had a 95% probability of surviving for 100 yr. Here, we set the population extinction threshold at 50 individuals to account for stochastic effects other than environmental and demographic stochasticity, such as Allee effects and short-term genetic inbreeding (Franklin 1980, Lande et al. 2003), and used the Ricker-logistic population-dynamic model that is commonly used to describe phenomenological time-series abundance data (Brook et al. 2006):

$$N_{t+1} = N_t \cdot e^{r(C) \cdot (1 - \frac{N_t}{K(C)}) + \sigma_t \cdot \varepsilon_t}$$

where $N_t$ is the population size at time $t$ (in number of individuals), $r(C)$ is the population growth rate (per unit of time, Eq. 2), $K(C)$ is the carrying capacity (in number of individuals, Eq. 3), and $\sigma_t$ is the standard deviation of the population growth rate, thus reflecting the effect of stochasticity on the realized growth rate. The term $\varepsilon_t$ was assumed to represent Gaussian white noise (mean = 0, variance = 1). Following Hilbers et al. (2016), we assumed the initial population size to be equal to the carrying capacity, reflecting a situation in which a healthy population is exposed solely to toxicants, and adopted a ceiling in the Ricker-logistic model whereby the population could only increase up to 10% above carrying capacity.

Probability of population extirpation (PE).—The PE is the probability that a population will go extinct within a given time frame. To calculate PE, we used Eq. 5 to simulate the population for 100 yr starting from a current population size, and recorded the proportion of 1,000 iterations during which the population dropped below the population extinction threshold of 50 individuals. Similarly to MVP, we adopted a ceiling in the Ricker-logistic model whereby the population could only increase up to 10% above carrying capacity.

Median time to population extirpation (MTE).—The MTE is the median time (in years) it takes a population to go extinct. To calculate MTE, we used Eq. 5 to simulate the population starting from a current population size, and recorded the median time of 1,000 iterations at which the population dropped below the population extinction threshold of 50 individuals. We ran the simulations for a maximum time of 1,000 yr and for all simulations where the population still existed after 1,000 yr, the time of population extirpation was set at an arbitrary high value of 100,000 yr. Similarly to MVP and PE, we adopted a ceiling in the Ricker-logistic model whereby the population could only increase up to 10% above carrying capacity.

Application

Model parameterization.—To illustrate the method, we applied it to calculate the impact of DDE on the local extirpation vulnerability of three bird species: the White-tailed Eagle, the Bald Eagle, and the Osprey. Following, e.g., Korsman et al. (2012), Nakamaru et al. (2003), and Schipper et al. (2013), we assumed that exposure to past and current prevailing environmental concentrations of DDE affects the fertility of these species (via abnormal breeding and eggshell thinning) rather than their survivorship. To parameterize Eq. 1, we first gathered field data reported in the literature to quantify the effects of DDE exposure (in mg/kg wet mass of egg) on the reproduction success of the bird species (Data S1). As a proxy for reproduction success, we used the number of fledglings per nest divided by the reported species-specific maximum number of young (569 out of 586 records), or the total number of successfully hatched eggs divided by the total clutch size (17 out of 586 records). In the quantile regression analyses, we weighted each observation (Data S1) based on the number of active nests investigated for reproduction success ($N_{\text{nests}}$) and the number of eggs measured for DDE concentrations ($N_{\text{eggs}}$)

$$\text{Weight Factor} = \frac{1}{N_{\text{nests}} + \frac{1}{N_{\text{eggs}}}}.$$  

Thus, we assigned larger weights to observations that were based on a larger number of active nests and/or a larger number of eggs with DDE measured. To quantify the intrinsic growth rate ($r_{\text{max}}$) of populations in optimal conditions without density limitation and the generation time ($t_g$), needed to translate impacts on reproduction success to
impacts on the population growth rate (Eq. 2), we used species-specific empirical data (Table 1).

The intrinsic growth rate of populations within an unsuitable area ($r_s$), needed to estimate the CPS, was set equal to the natural mortality rate of each species ($\mu$), similar to Pereira et al. (2004). This assumption implies that a species is able to survive yet unable to reproduce in unsuitable areas outside the patch. The mortality rate was derived from literature data on lifespan (Table 1), as the natural mortality rate is the inverse of the average lifespan of a species. The probability of an individual moving outside the native patch $\pi$ was set at 0.5, indicating that a species is equally prone to stay within the patch as to migrate to nonnative areas (following Pereira et al. 2004, Pereira and Daily 2006, Hilbers et al. 2016b). Finally, by assuming Gaussian dispersal, the dispersal variance (in $km^2$) was estimated following Pereira and Daily (2006):

\[
\sigma^2 = \frac{d^2}{(1.81)^2} \times \frac{1}{(1/\mu)}
\]  

(7)

where $1/\mu$ reflects the average life span (in years) and $d_m$ (in km/generation) is the median natal dispersal distance. Both parameters were derived from literature (see Table 1).

We also used species-specific empirical data on the standard deviation of the intrinsic population growth rate ($\sigma_s$) needed to estimate the MVP, PE, and MTE (Table 1). To ensure that $\sigma_s$ reflects environmental stochasticity, we derived empirical $\sigma_s$ values using population growth rate dynamics of populations larger than 50 individuals (Franklin 1980, Lande et al. 2003). To account for demographic stochasticity, we sampled the population size at time $t$ + 1 from a Poisson distribution (Bonsall and Hastings 2004, Melbourne and Hastings 2008). As the current population size and the carrying capacity of populations are context dependent, we used two scenarios in the estimation of PE and MTE, with the carrying capacity without toxicant exposure $K(0)$ set equal to 500 or 5,000 individuals (around the range of values reported in the literature; Fraser et al. 1996, Watts et al. 2008, Krüger et al. 2010, Sulawa et al. 2010, Wahl and Barbraud 2014) and the current population size set equal to the carrying capacity $K(C)$ at the start of the simulations. Similar to MVP, this reflects a situation in which a healthy population is exposed solely to toxicants and thus enables to explore the changes in the response of population viability due to toxicant exposure.

Model simulations.—To account for the uncertainty in the exposure–response relationships and the species-specific demographic parameters, we considered a range of possible reproduction success values per DDE exposure concentration and a range of possible $r_{max}$, $\sigma_s$, $1/\mu$, $d_m$, and $\tau_g$ values. To this end, we constructed confidence intervals around the exposure–response relationships. Furthermore, Student’s $t$ distributions were implemented to reflect the uncertainty in the average of the log-transformed demographic parameters, as derived from literature (Table 1). Then, we calculated $r(1, C)$, $K(C)$, CPS, MVP, PE, and MTE of the three bird species in 1,000 iterations in which the reproduction success for specific DDE concentrations and the demographic parameters were randomly sampled from their intervals and distributions, respectively. We restricted the random sampling so that the average life span was always greater than the generation time of the species, and the median natal dispersal distance, the intrinsic growth rate of populations and its standard deviation were always greater than zero.

To assess the contribution of the uncertainty in the exposure–response relationships and the species-specific demographic parameters to CPS, MVP, PE, and MTE, we calculated Spearman rank correlation coefficients between the population extinction vulnerability indicators and each of the uncertain parameters based on the 1,000 iterations using a DDE range of 0–73 mg/kg wet mass of egg (i.e., the maximum measured DDE concentration in White-tailed Eagle eggs).

Finally, we used the models to quantify the population growth rate, CPS, MVP, PE, and MTE for several populations of the three species, based on the median DDE exposure concentration per population as reported in the literature. We also compared our predicted population growth rate estimates for these populations with observed population growth rates in corresponding regions as derived from literature. To obtain observed population growth rates, we calculated ln($N_{t+1}/N_t$) by using census data (Appendix S1: Table S2) on the population sizes in the year(s) at which DDE exposure concentrations were measured ($N_t$) and the consecutive year(s) ($N_{t+1}$). We then averaged the observed population growth rates over the year(s) at which DDE exposure concentrations were measured to obtain one estimate per population. All analyses were performed using the statistical software environment $R$, version 3.1.3 (R Core Team 2017), in which the “quantreg” package was used for the quantile regression analyses (Koenker 2013).

RESULTS

 exposurere—response relationships

Considerable variation in reproductive success was observed in relation to DDE exposure concentrations, especially for the

| Table 1. Parameters used in the estimation of the population extinction vulnerabilities of the three bird species.† |
|---|---|---|---|---|---|---|
| Species | $r_{max}$ (yr$^{-1}$) | $\sigma_s$ (yr$^{-1}$) | $\text{max}_{\text{young}}$ | $d_m$ (km) | $1/\mu$ (yr) | $\tau_g$ (yr) |
| White-tailed Eagle | (0.12,3,0.06)$\ddagger$ | (0.05,6,0.02)$\ddagger$ | 1.76 | (78.1,6,27.9)$\ddagger$ | (22.8,6,4.3)$\ddagger$ | (14.3,2.3)$\ddagger$ |
| Bald Eagle | (0.15,7,0.08)$\ddagger$ | (0.14,7,0.19)$\ddagger$ | 2.22 | (73.4,8,17.5)$\ddagger$ | (17.9,7,4.4)$\ddagger$ | (10.3,5,3.6)$\ddagger$ |
| Osprey | (0.23,2,0.08)$\ddagger$ | (0.06,6,0.01)$\ddagger$ | 2.00 | (152.1,6,183.0)$\ddagger$ | (22.7,8,2.6)$\ddagger$ | (9.4,3,2.1)$\ddagger$ |

Notes: Parameters are $r_{max}$, the intrinsic population growth rate; $\sigma_s$, the standard deviation of the population growth rate; $\text{max}_{\text{young}}$, the maximum number of young; $d_m$, the median natal dispersal distance; $1/\mu$, the average life span; and $\tau_g$, the generation time.

$\ddagger$See Appendix S1: Table S1 for the reference list.

$\ddagger$Student’s $t$ distribution: $(\text{mean}, \text{degrees of freedom}, \text{standard deviation}).
Bald Eagle and the Osprey, where reproductive success ranged between 0 and 1 up to concentrations of 10 mg/kg wet mass of egg (Fig. 1). Yet, the quantile regression analyses produced highly significant chemical exposure–response relationships for all three bird species. EC$_{50}$ values of the exposure–response curves for DDE overlapped between the three species with 12.0 (95% confidence interval 6.9–29.2) mg/kg wet mass of egg for the Bald Eagle, 14.4 (9.4–35.2) mg/kg wet mass of egg for the Osprey, and 17.3 (11.0–32.8) mg/kg wet mass of egg for the White-tailed Eagle (Table 2). The Hill coefficient, which reflects the steepness of the exposure–response relationships, was different between the three species with $-0.65$ ($-0.53$ to $-0.77$) for White-tailed Eagle, $-0.84$ for the Bald Eagle ($-0.76$ to $-0.93$), and $-2.02$ ($-1.61$ to $-2.05$) for the Osprey.

Population extinction vulnerability indicators

DDE exposure increased the population extinction vulnerability of all three bird species (Fig. 2). For example, at DDE concentrations of 25 mg/kg wet mass of egg (the maximum DDE concentration reported in the literature for the Osprey), population growth rates became small (White-tailed Eagle and Osprey) or close to zero (Bald Eagle), the CPS increased up to almost the size of Connecticut (White-tailed Eagle and Osprey) or West Virginia (Bald Eagle), the MVP increased up to approximately 90 breeding pairs (White-tailed Eagle and Osprey) or 180 breeding pairs (Bald Eagle), the PE increased up to almost certain extirpation (Bald Eagle) or only slightly elevated levels (White-tailed Eagle and Osprey), and the MTE became within decades (Bald Eagle) or remained longer than a millennium (White-tailed Eagle and Osprey).

The uncertainty in the CPS of the White-tailed Eagle was mostly caused by the uncertainty in the intrinsic population growth rate ($r_{max}$), the uncertainty in the CPS of the Bald Eagle was mainly caused by the uncertainty in the DDE exposure–response relationship ($E_{R}$), whereas for the Osprey, the uncertainty in the median dispersal distance ($d_{m}$) was the

![Fig. 1. The exposure–response relationships between reproduction success (dimensionless) and DDE concentration (mg/kg wet mass of egg) for the White-tailed Eagle, Bald Eagle, and Osprey. The relationships for the Bald Eagle and Osprey were extrapolated (transparent lines) to a DDE concentration of 73 mg/kg wet mass of egg (i.e., the maximum measured DDE concentration in White-tailed Eagle eggs) to improve comparison among the species. Points represent the field data in which the size reflects the weighting factor (i.e., the larger the point, the higher the weighting factor), solid lines represent the quantile regression fit and dashed lines represent the confidence interval of the quantile regression fit. EC$_{50}$ is defined in Methods: Field-based exposure–response curves.](image)
TABLE 2. Summary of the exposure–response relationships and the critical levels for DDE based on a number of maximum permissible effect levels: at which the population growth rate is lower than 5% and the MTE is lower than 100 yr.

<table>
<thead>
<tr>
<th>DDE concentrations (in mg/kg wet mass egg)</th>
<th>MVP = 250</th>
<th>PE = 0.05</th>
<th>MTE $= 1000 yr$</th>
<th>$K(0) = 500$</th>
<th>$K(0) = 5000$</th>
</tr>
</thead>
<tbody>
<tr>
<td>20,000 km$^2$, the MVP is 17.3 (11.0/C0)</td>
<td>0.65 (0.53 to 0.77)</td>
<td>24 (11-41)</td>
<td>29 (12-61)</td>
<td>31 (15-41)</td>
<td>37 (18-51)</td>
</tr>
<tr>
<td>0.05</td>
<td>12.0 (6.9/C0)</td>
<td>0.84 (0.76 to 0.93)</td>
<td>16 (6-30)</td>
<td>14 (1-2-60)</td>
<td>35 (18-51)</td>
</tr>
<tr>
<td>Osprey</td>
<td>14.4 (9.4/C0)</td>
<td>$-0.05$ (to -0.75)</td>
<td>23 (11-41)</td>
<td>27 (12-61)</td>
<td>37 (18-51)</td>
</tr>
<tr>
<td>10,000 km$^2$, the MVP is 17.3 (11.0/C0)</td>
<td>0.65 (0.53 to 0.77)</td>
<td>24 (11-41)</td>
<td>29 (12-61)</td>
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</tr>
</tbody>
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Parameters are EC$_{50}$, half the maximal effective concentration or inflection point of the curve for reproductive success; $\beta$, Hill slope coefficient; $r_1$, population growth rate; CPS, critical patch size; MVP, minimum viable population; $K(0)$, carrying capacity without toxicant exposure; PE$_{10}$, probability of extirpation starting at carrying capacity; MTE, time to population extinction starting at carrying capacity; indicators are given in brackets.

Notes: Population-specific application

Based on historically measured exposure concentrations of DDE, the majority of the bird populations were found to have no elevated risk of local extirpation due to past DDE exposure (Table 3). On average, population growth rates remained above 0, CPS were within 10,000 km$^2$, MVP lower than 100 breeders, PE equal to 0, and MTE longer than 1,000 yr. Nevertheless, for the White-tailed Eagle population in the Quarken area of the Gulf of Bothnia (Finland), the population growth rates (median values) were reduced up to a factor of 2.7 compared to values without DDE exposure, CPS (median values) increased up to a factor of 4.0, MVP (median values) increased up to a factor of 1.5, PE (median values) up to 7%, and MTE (median values) decreased down to 736 yr due to DDE exposure.

Comparison with observed population growth rates

For the populations for which census data was available, we found a wider range of historically observed population growth rates for the three bird species than predicted by our models (Fig. 4). Approximately 10% of the observed population growth rates were higher than our estimates, 26% were in between the 10th and 90th percentiles of our estimates, and 65% were below our estimates.

Discussion

Given the potential impact of chemical pollution on biodiversity, there is an urgent need to better quantify the extinction vulnerability of species and populations due to chemical exposure. We provide a simple, coherent method to quantify population extinction vulnerabilities using field monitoring data, which may be used in addition to assessments based on laboratory data or when laboratory data are absent. For example, for the White-tailed Eagle, Bald Eagle, and Osprey, laboratory data on the reduction of the intrinsic population growth rate due to chemical concentrations are lacking, whereas we found 26 field studies investigating reproduction success in relation to DDE. Thus, our method increases the number of species for which population extinction vulnerabilities due to toxicant exposure can be quantified, including threatened or endangered species for which laboratory data are generally not available (Forbes et al. 2016). Moreover, the use of field data obviates the need to correct lab-based effect measurements for differences in exposure conditions between lab and field, such as diet composition and species’ metabolic rates (Traas et al. 1996, Bednarska et al. 2013, Forbes and Calow 2013).

Provided that field measurements are available, our method may be applied to any species population or persistent bioaccumulative toxicant of interest to obtain first estimates of the
population extinction vulnerability due to past, current and
future chemical exposure. For example, our case study
revealed that the White-tailed Eagle population in the
Quarken area of the Gulf of Bothnia (Finland) has been
under elevated threat of local extirpation due to past DDE
exposure (Table 3). Further, our method allows for deriving
critical levels of toxicant exposure based on maximum permissible effect levels. For example, using the toxicant concentrations at which the PE is >5% (as commonly used in ecological risk assessment) would result in critical levels for DDE of 12, 25, and 36 mg/kg wet mass of egg for the Bald Eagle, Osprey, and White-tailed Eagle, respectively, for populations with carrying capacities without toxicant exposure of around 500 individuals (see Table 2 for critical levels for DDE based on other example maximum permissible effect levels). Additionally, for species under influence of toxicant exposure, our method can provide first estimates of the minimum habitat area and population size to be protected. For example, using measured field concentrations of DDE, we found that the Bald Eagle population in Michigan (USA) required at least 17,907 km² of suitable habitat and 166 individuals to be viable (Table 3). As long as exposure levels cannot be reduced, the CPS and MVP metrics can be used as guidelines for increasing habitat area or reintroducing individuals, as was done for example in North America in the 1980s for Bald Eagles exposed to DDE (Sharpe and Garcelon 2005, Sorenson et al. 2017). Using the field-based approach developed in this study to obtain this type of information may help conservation managers to better design appropriate habitat restoration and population recovery measures, such
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<th>CPS (km$^2$)</th>
<th>MVP (no. individuals)</th>
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<th>PE$<em>{E</em>{00}}$ - 50% (-)</th>
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Note: The 10th and 90th percentiles are shown in brackets. The data from Wiemeyer et al. (1987, 1993) and Martin et al. (1993) were directly linked to regressions of median DDE concentration data per region taken from Tables 9 and 4, respectively.
as reducing toxicant levels, increasing the area of suitable habitat and reintroducing individuals.

Nevertheless, our field-based approach has some limitations. Confounding environmental factors that are correlated with the toxicant of interest may result in an overestimation of the impacts on population viability. To overcome this limitation, multiplicative or additive exposure–response relationships could be employed in the method instead (see, e.g., Korsman et al. 2012). Furthermore, for toxic compounds that are not yet released or that dissipate quickly in the environment, hence for which exposure concentrations are not easily obtained from the environment, assessments can be based on laboratory data only.

**Case study results**

We combined population viability analysis and field-based exposure–response curves to quantify the effects of toxicant exposure on the population extinction vulnerability of three bird species. We found highly significant chemical exposure–response relationships for the three bird species, in which increasing DDE concentrations gradually decreased the reproductive success. The reproductive abilities of the White-tailed Eagle and the Bald Eagle were affected at lower concentrations of DDE than the reproductive success of the Osprey, whereas at high concentrations of DDE the reproductive success of the Osprey was relatively low, reflected by similar EC50 values for all species but a steeper slope in the exposure–response curve of the Osprey. The EC50 values of the three species were comparable yet systematically higher than results from Wiemeyer et al. (1993), Elliott et al. (2001), Helander et al. (2002), Henny et al. (2004), and Best et al. (2010). This can be explained by the fact that these field studies used regression techniques that relate changes in the mean of a reproduction success variable to DDE, instead of looking at one of the upper quantiles. Consequently, their EC50 values are likely influenced by confounding environmental factors masking the effect of DDE on reproduction success (Cade and Noon 2003). The quantile regression analysis applied here filters out reductions in reproductive success due to confounding environmental factors by assuming that they are not correlated with DDE but provide
random noise instead. Yet, DDE levels in the environment are likely correlated with other anthropogenic stressors (such as land use and other toxicants), which might cause a systematic bias in the field monitoring data. This implies that the EC50 values and critical levels for DDE specifically might be higher than found in this study.

Our results showed that the range of DDE exposure concentrations encountered in the environment in the past increased the population extinction vulnerability of all three bird species. Although our models were relatively simple, excluding demographic and spatial population structure, our CPS and MVP values for the Bald Eagle were similar to the results of Reed et al. (2003) and Verboom et al. (2014), who reported minimum area requirements of 3,222 km² and MVPs of 193 and 514 individuals, but lower than the MVP estimate of 1,735 individuals of Brook et al. (2006) who used mean population growth rates likely affected by multiple stressors (Hilbers et al. 2016). We could not find CPS and MVP estimates for the White-tailed Eagle and Osprey, except for a generic estimate of 120 reproductive units for the MVP of the White-tailed Eagle and Osprey from Jantke et al. (2011). For PE and MTE, we only found estimates of a PE of <5% (r1 = 0.06) and ~60% (r1 = 0.005) for White-tailed Eagle populations in Germany (Sulawa et al. 2010) and Scotland (Green et al. 1996), respectively, which are comparable to the PE values found in this study at these population growth rates. Similar to Green et al. (1996), Söther et al. (2005), and Sulawa et al. (2010), our results for PE and MTE indicate that increasing the population size and carrying capacity of a population could substantially reduce its vulnerability to extinction (Fig. 2 and Appendix S1: Fig. S1).

The comparison with the observed population growth rates in the field in the past, showed that the predicted population growth rates were in the same order of magnitude as the observed population growth rates in the regions. However, in general, our population growth rate estimates were higher than those observed, especially for the Osprey. This can be explained by the fact that the populations in these regions were likely affected by other environmental factors next to DDE exposure. In addition, the observed population growth rates may deviate from the estimates obtained in this study because of stochastic effects and measurement or observation error in abundance estimates (Brook et al. 2006).

In the field, the highest historically measured population-specific exposure concentrations were found for the White-tailed Eagle, corresponding to relatively larger CPS, MVP, and PE values and lower population growth rate and MTE values compared to the other two bird species. As indicated by van Drooge et al. (2008) and Sivonen (2014), these relatively high exposure concentrations for the White-tailed Eagle compared to the Bald Eagle and Osprey might be explained by a difference in diet composition. Where the Osprey feeds almost exclusively on fish, the White-tailed Eagle, and to a lesser extent the Bald Eagle, also preys upon birds and mammals (Wilman et al. 2014) that may frequently feed in agricultural fields and have been found to have high levels of DDE residues in their systems (van Drooge et al. 2008, Sivonen 2014). Alternatively, the higher field exposure concentrations of DDE for the White-tailed Eagle may reflect that these were measured before or relatively shortly after the ban of DDT in the regions, whereas, in general, the exposure concentrations for the other two species were measured years after the ban of DDT (Table 3) explaining the low median DDE exposure concentrations per population and, in turn, the low risk of local extirpation for the majority of the bird populations.

Acknowledgments

We would like to thank Anders Bignert, John Elliott, Kyle Elliott, Björn Helander, and Donald Tillitt for sharing additional data underlying their publications.

Literature Cited


**Supporting Information**

Additional supporting information may be found online at: http://onlinelibrary.wiley.com/doi/10.1002/eap.1685/full