Received: 6 November 2018 | Accepted: 4 May 2019

DOI: 10.1111/1365-2664.13448

RESEARCH ARTICLE

Journal of Applied Ecology | BRITISH ECOLOGICAL SOCIETY

The sensitivity of seabird populations to density-dependence, environmental stochasticity and anthropogenic mortality

Julie A. O. Miller[1] | Robert W. Furness[2] | Mark Trinder[2] | Jason Matthiopoulos[1]

[1]Institute of Biodiversity, Animal Health and Comparative Medicine, University of Glasgow, Glasgow, UK

[2]MacArthur Green, Glasgow, UK

Correspondence
Julie A. O. Miller
Email: j.miller.5@research.gla.ac.uk

Funding information
NERC CASE

Handling Editor: Ailsa McKenzie

Abstract

1. The balance between economic growth and wildlife conservation is a priority for many governments. Enhancing realism in assessment of population-level impacts of anthropogenic mortality can help achieve this balance. Population Viability Analysis (PVA) is commonly applied to investigate population vulnerability, but outcomes of PVA are sensitive to formulations of density-dependence, environmental stochasticity and life history. Current practice in marine assessments is to use precautionary models that assume no compensation from density-dependence or rescue-effects via "re-seeding" from other colonies. However, if we could empirically quantify regulatory population processes, the responses of populations to additional anthropogenic mortality may be assessed with more realism in PVA.
2. Using Bayesian state-space models fitted to population time series from three sympatric seabird populations, selected for varied life histories, we inferred the extent to which their dynamics are driven by environmental stochasticity and density-dependence.
3. Based on these inferences, we conducted an exhaustive PVA across credible parameterizations for intrinsic and extrinsic population regulation, simulated as a closed and re-seeded system. Scenarios of anthropogenic mortality, along a sliding scale of precaution, were applied both proportionally and as a fixed quota using Potential Biological Removal (PBR).
4. Baseline results from fitting revealed clear environmental regulation in two of our three species. Crucially, we found that for our empirically derived, realistic model parameterizations there are risks of decline to real populations even under very precautionary mortality scenarios. We find that PBR is dubious in application as a sustainable tool for population assessment when we account for regulation. Closed versus re-seeded models showed a large divergence in outcomes, with sharper declines in closed simulations. Fixed-quota mortality typically induced greater population declines comparative to proportional mortality, subject to regulation and re-seeding.

5. *Synthesis and applications*. Practitioners using arbitrary formulations of population regulation risk over-precaution (economic constraint) or under-precaution (endangering populations). The demands of increased economic development and preservation of wildlife require that methodologies apply techniques that confer reality and rigour to assessment. The current practice of employing models lacking density-dependence and empirical environmental information imposes limitations in the efficacy of estimating impacts. Here, we provide a method to quantify the conditions that predominantly regulate a population and exacerbate the risk of decline from anthropogenic mortality. It is in the interests of both developers and conservationists to apply methods in population impact assessments that capture realism in the processes driving population dynamics.

KEYWORDS
density-dependence, environmental stochasticity, marine renewables, population dynamics, rescue-effect

1 | INTRODUCTION

Globally, many species are facing decline and potential extinction from anthropogenic activities (Murphy & Romanuk, 2014; Ripple et al., 2016). Seabird populations are at risk from additional anthropogenic mortality from by-catch, oiling, harvesting and marine renewables (Croxall et al., 2012). The UK coastline hosts internationally significant numbers of seabirds, especially during the breeding season, and several of these species have been assessed as potentially at risk from offshore wind installations (Furness, Wade, & Masden, 2013). The marine renewables industry (Platteeuw et al., 2017) is seeking expansion, incentivized, in part, by internationally agreed sustainable-energy targets (Marques, Fuinhas, & Manso, 2010). Poorly planned developments may cause impacts such as additional mortality from collision strikes (Johnston, Cook, Wright, Humphreys, & Burton, 2014). Industry, especially in countries bearing a legal requirement for environmental assessment (European Commission, 2011), has a responsibility to minimize its impact on wildlife. Assessment approaches vary, but generally contain an appraisal of risk to populations from impacts of additional mortality (Buckland, Goudie, & Borchers, 2000), following the precautionary principle (Sands & Peel, 2012), supported by EU directives (European Commission, 2009).

Determining the consequences of additional mortality to a population requires an estimate of population size, an understanding of life history, estimates of demographic rates and how these are affected by regulating influences such as density-dependence and environmental stochasticity (Lande, Engen, & Saether, 2003). At the point of assessment and more generally in population ecology, there are inherent uncertainties associated with these parameters (Clark, 2003). Assessments of inadequately studied populations may use proxy values from populations of the same or similar species for productivity or survival rates (Kindsvater et al., 2018). In addition, a lack of empirical estimation of connectivity (immigration or emigration) often leads practitioners to use closed system models (Hanski, 1998). Empirical estimates of population regulation across spatio-temporal scales are also deficient. The shape and magnitude of density-dependence is difficult to quantify in wild populations despite its recognized importance for population dynamics (Bonenfant et al., 2009; Knape & deValpine, 2012). It is also challenging to derive quantitative estimates for extrinsic regulation from environmental stochasticity, especially since it may be confounded with intrinsic regulation in short time series (Engen, Bakke, & Islam, 1998). Understanding how population persistence is affected by variable combinations of density-dependence and environmental regulation is unknown (Saether, Engen, Lande, Both, & Visser, 2002). To increase the credibility of predicted outcomes from population assessments it is essential to represent biological reality whilst capturing uncertainty in estimates of understudied processes (Saunders, Cuthbert, & Zipkin, 2018). Failure to faithfully reflect accuracy and precision in predictions leads to one of two detrimental outcomes for human-wildlife conflict; over-precaution due to an inflated estimate of risk may curtail economically important activities, whilst undetected risks to population viability, could set sensitive populations on a path to extinction.

Reflecting knowledge gaps in connectivity and the ability to capture population regulation, UK seabird populations are modelled in an environmental impact assessment (EIA) assuming no immigration and, often, no density-dependence. The risk of additional mortality to a seabird population from collision is estimated typically across a period of 25 years, the operational life span of a wind farm, and evaluated as to how this estimate will affect the population. Population viability analysis (PVA) (Boyce, 1992) and Potential Biological Removal (PBR) (Wade, 1998), are among the methods used to evaluate anthropogenic additional mortality on seabird populations under assessment. PVA is an application of projection matrix modelling. PBR, designed initially

for marine mammal assessment, is a simple harvest assessment calculating the maximum number of animals that may be sustainably removed from a given population. In EIA, PVA usually contains some estimate of demographic rates, with more recent assessments attempting to include environmental stochasticity by implementing the demographic rates as stochastic processes. Although some models may include a form of density-dependence, there is ongoing debate about the practical justification for its inclusion (Green, Langston, McCluskie, Sutherland, & Wilson, 2016; Horswill, O'Brien, & Robinson, 2016). In general, PVA has been shown to be sensitive to estimates of parameters, affecting predicted outcomes (Cook & Robinson, 2016; Reed et al., 2002). PBR requires minimal demographic input, save for an adult survival estimate and the age at first breeding. There is an assumption of compensatory density-dependence included in the model background (Niel & Lebreton, 2005). The PBR calculation returns a threshold value of additional mortality to a population above in which a decline is expected, it contains no stochastic component and no population projection. PBR has been criticized as a tool for EIA as it fails to quantify potentially serious population consequences which can occur below this threshold (Green et al., 2016; O'Brien, Cook, & Robinson, 2017).

Environmental signals affecting population dynamics can be explored using time-series data (Frederiksen, Mavor, & Wanless, 2007; Leirs et al., 1997), and density-dependence signals may also be extracted from population time-series (Brook & Bradshaw, 2006; Lande et al., 2002). For realistic assessment, we should aim to derive empirical values for the strength of density-dependence and the magnitude of environmental stochasticity from such data. Exploring these values in a PVA would provide multiple intrinsic and extrinsic regulatory scenarios against which additional mortality may be assessed. Furthermore, modelling the populations with a rescue-effect (where immigration prevents extinction, Brown & Kodric-Brown, 1977) could help us assess the sensitivity of outcomes to the assumption of closed populations. We therefore aimed to assess how several levels of anthropogenic mortality can impact real seabird populations (both closed and open to a rescue-effect), operating under empirically derived estimates of environmental stochasticity and density-dependence.

We used Bayesian methods to fit a state-space population model to historical data from sympatric populations of three well-studied UK seabird species with divergent life histories: Northern gannet *Morus bassanus* L., black-legged kittiwake *Rissa tridactyla* L. and common guillemot *Uria aalge* Pontoppidan. This provided posterior credible intervals for density-dependence and environmental stochasticity for each population. These intervals defined parameter ranges for the strength of density-dependence and environmental regulation, which we explored systematically using our population model, generating predictions of population viability across the entire space of plausible combinations of intrinsic versus extrinsic regulation. To assess risk from additional mortality we utilized PBR as a tool providing a range of values across a precautionary gradient, allowing a general approach to visualizing 25-year mortality impact. Populations were modelled both under assumed closedness and under the possibility of annual rescue-effects from immigration.

2 | MATERIALS AND METHODS

2.1 | Demographic Model

High-quality demographic rate and population abundance data were obtained for the sympatric island populations of Bass Rock (gannet) and the Isle of May (kittiwake and guillemot), Scotland (Appendix S1(A1)).

All modelling and analysis was undertaken in R (R Core Team, 2016). A stochastic stage-structured matrix model was developed for each species. The dimensions of the annual transition matrix were based on each species' age-at-maturity. For example, the deterministic version of our model, using rates, for a species with one juvenile and three sub-adult classes would take the form:

$$\begin{pmatrix} n_{1,t+1} \\ n_{2,t+1} \\ n_{3,t+1} \\ n_{4,t+1} \\ n_{5,t+1} \end{pmatrix} = \begin{pmatrix} 0 & 0 & 0 & 0 & B_t \\ S_{j,t} & 0 & 0 & 0 & 0 \\ 0 & S_{1,t} & 0 & 0 & 0 \\ 0 & 0 & S_{2,t} & 0 & 0 \\ 0 & 0 & 0 & S_{3,t} & S_{a,t} \end{pmatrix} \begin{pmatrix} n_{1,t} \\ n_{2,t} \\ n_{3,t} \\ n_{4,t} \\ n_{5,t} \end{pmatrix} \quad (1)$$

where B and S represent the vital rates of productivity and survival, and the subscripts j, 1, 2, 3 and a on the vital rates denote juvenile, sub adult and adult age classes respectively.

As a female-only model, productivity was adjusted (Appendix S1(A2)) under the assumption of a 1:1 sex-ratio. In the stochastic model, the number of chicks fledged and number of animals of each age class surviving were modelled as binomial processes, for all species, including kittiwake (Appendix S1(A3)).

Generally, seabirds are characterized as long-lived animals that exhibit delayed reproduction, low fecundity linked to ecological constraints of resource patchiness and there is evidence of compensatory density-dependence in several studies (Bried & Jouventin, 2002; Horswill et al., 2016; Appendix S1(A4)). There is evidence across all species that adults forego breeding when extrinsic and intrinsic conditions are sub-optimal to maintain condition (Oro & Martinez-Abrain, 2004; Weimerskirch, 2001). The model therefore estimates the strength of compensatory density-dependence in productivity alone, under the assumption that this would be the demographic rate most likely to be affected.

We do not attempt in this model to account for a potential "floater" population of adults that have the potential to breed but have not.

The probability of fledging female chicks at each time step B_t was modelled as a logit function of a linear predictor b_t. The logit transformation was used to ensure that B_t remained bounded within the 0-to-1 range. The linear predictor took the form,

$$b_t = b_0 - \phi n_{t-1} + \varepsilon_t \quad (2)$$

The terms on the right-hand side of Eqn (2) correspond to an intercept b_0, the effect of density (n_{t-1}) on productivity, measured as a

penalty to the intercept by a coefficient (ϕ), and the effect of an environmental perturbation term (ε_t) comprising a time series of identically distributed Gaussian terms $\varepsilon_t : N(0, \sigma)$. The standard deviation (σ) of this term, was used as our metric for the magnitude of environmental stochasticity acting on the populations. The intercept represented intrinsic productivity, in the absence of density-dependence and environmental perturbations and was derived from female-only reported maximal values of B (Supplementary Information) as the inverse logit transformation:

$$b_0 = \log \frac{B}{1-B} \tag{3}$$

Environmental variation can affect survival at all life stages (Frederiksen, Daunt, Harris, & Wanless, 2008; Jenouvrier, Barbraud, Cazelles, & Weimerskirch, 2005; Lewis, Elston, Daunt, Cheney, & Thompson, 2009; Weimerskirch, 2001) and we assume that its impact is synchronous to the impact on productivity. In the model, the same fluctuations in environmental stochasticity are therefore also applied to survival, using a scaling term (p) (expressing the relative importance of environmental stochasticity for breeding and the survival rates of different life stages). This scalar was calculated from available time series of demographic data, specific to each of our study populations (Appendix S1(A5)). Survival for each age class ($S_{*,t}$) was modelled as a logit of baseline survival (s_{*0}) and environmental stochasticity ($p_* \varepsilon_t$). For example, in the case of juveniles, the linear predictor took the form:

$$s_{j,t} = s_{j,0} + p_j \varepsilon_t \tag{4}$$

The population's initial age and sex structure was unknown, so as a plausible start (assuming equal sex-ratio), we used the population's stable age distribution, derived from the dominant eigenvector (Caswell, 2001) of the density-independent projection matrix. This distribution was then scaled using the population starting size to derive numbers for each age class (Appendix S1(A6)).

2.2 | State space model fitting to derive regulation

The above population model was fitted to the observations of historical population trend data for each species at each colony using the program JAGS (Plummer, 2003) interfaced with R via the runjags package (Denwood, 2016). There were missing data from years between counts in all the colonies except kittiwake. The datasets for each species were: kittiwake: years = 24; estimates across years = 24; gannet: years = 29; estimates across years = 5; guillemot: years = 44; estimates across years = 39. Observation error was included in the model by allowing for a 1% sampling error in colony counts.

To estimate the regulatory parameters of interest: strength of density-dependence (ϕ) and strength of environmental stochasticity (σ), minimally informative priors were used for the models (Appendix S1(A7)).

The density-dependence parameter ϕ (Eqn 3) was assigned a gamma distribution, restricting density-dependence to a compensatory form. During model fitting, the MCMC chains explored extremely low values for density-dependence in some species. To facilitate computation in these cases we imposed a floor-truncation to this prior at the value 10^{-11}. Our model estimated the strength of environmental stochasticity as the precision ($\tau = \sigma^{-2}$) of the stochastic process ε_t. This was assigned a gamma prior (constraining σ^2 to positive values). Models were run until satisfactory convergence, based on the Gelman-Rubin diagnostic, PSRF (Brooks & Gelman, 1997), effective sample sizes for all model parameters and visual inspection of traceplots (Kass, Carlin, Gelman, & Neal, 1998). The ranges of values for the strength of density-dependence and environmental stochasticity were given by the upper and lower 95% credible intervals of the posteriors for ϕ, σ.

2.3 | Mortality sensitivity: PVA

Populations were projected in a PVA analysis using the demographic matrix model outlined in 2.1 with ranges of values for density-dependence and environmental stochasticity taken from the posterior credible intervals (2.2) obtained from model-fitting. We divided each interval into 51 increments, producing 2,601 pairwise combinations of values for density-dependence and environmental stochasticity. For each species, every combination was examined via population projections from our PVA model. Firstly, to gain a broader overview of the dynamics of these models and to allow comparisons between species, the estimated posterior values for density-dependence and environmental stochasticity were extended beyond their credible values and projected in PVA with no additional mortality. To assess what impact mortality had on populations under different regulation, populations were again projected under each pairwise combination. After a settling-in period of 200 years (Appendix S1(A8)), mortality treatment was applied with the PBR derived from the starting population size at the 200-year mark. Severity of mortality was manipulated by the recovery factor term (FR) in the equation (Appendix S1 (A9)). The PBR was applied annually as a fixed number, and as a proportion removed annually based on the starting population size at 200 years. In all cases mortality was applied across all age classes in proportion to their annual size. Mortality was applied over a 25-year period, a typical windfarm life span. The mean population size μ_1 across this period, was then calculated and compared to the 25-year population mean μ_0 directly prior to the introduction of anthropogenic mortality. Impact (I) was calculated as a proportional change in population thus:

$$I = \frac{\mu_0 - \mu_1}{\mu_0} \tag{5}$$

To further explore the effects of mortality in this system we simulated the PVA analysis under a traditional "closed" system, where we first assumed no connectivity in the populations and

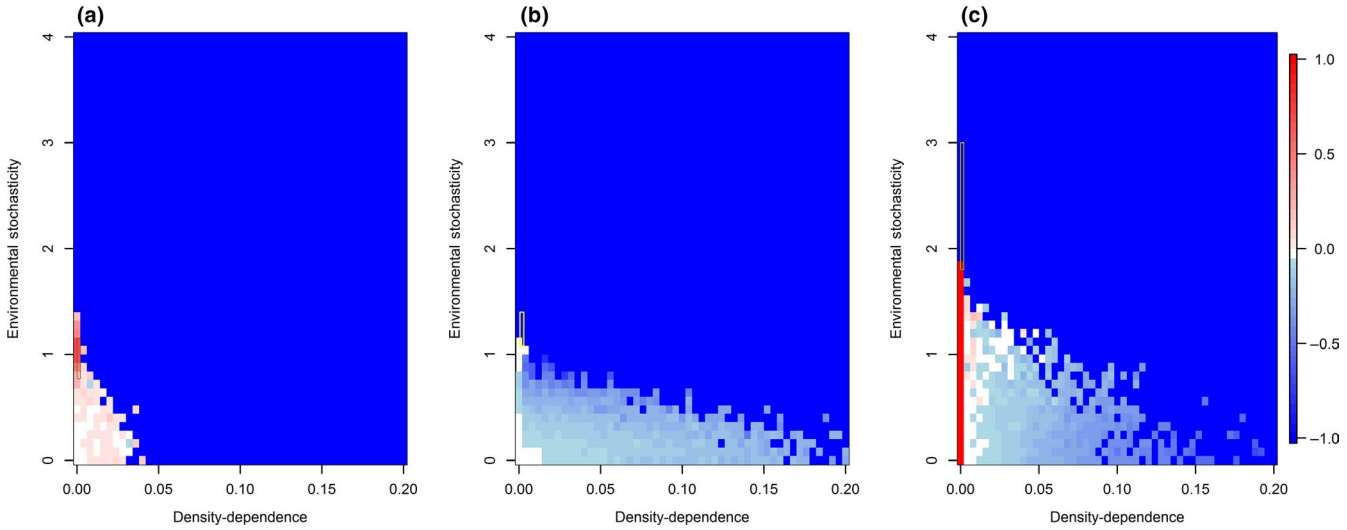

FIGURE 1 Baseline population change when density-dependence (DD) varies between 1e-11 and 0.2 and environmental stochasticity (ES) varies between 0 and 4. Panels a, b and c represent gannet, kittiwake and guillemot, respectively, with boxes indicating the posterior boundaries of DD and ES derived from fitting and used in subsequent mortality projections. Pale colours are near-zero changes, pink through to red indicate proportional increases. Light blue indicates small declines through to darker blues indicating greater decline

then allowed an annual rescue-effect by the introduction of a female, re-seeding the population and preventing extinction, but not avoiding declines.

3 | RESULTS

3.1 | Baseline population change beyond boundaries of empirically derived density-dependence and environmental stochasticity

With no additional mortality, each species displayed declines and eventual extinctions under higher strengths of density-dependence and higher environmental stochasticity (Figure 1). All species produced similar qualitative patterns to increased regulation but varied in their response and sensitivity to regulation at lower values. The boxes overlaying plots in Figure 1 indicate the fit-derived estimates for density-dependence and environmental stochasticity (Appendix S1(A11)). The boxes vary in their shape and limits across each of the species, but all are estimated near the lower limits of the full range of density-dependence explored, where colony persistence is unaffected by density regulation.

3.2 | Mortality treatment results: Gannet

The gannet baseline, with no mortality treatment, showed no clear pattern of population change regulated by environmental stochasticity, strength of density-dependence or any combination of these. Both closed and re-seeded baseline projections were found to have increased across the 25-year period on average (Figure 2, panels 1A, 2A and 1F, 2F). Under a proportional mortality there was no difference in impact between the closed and re-seeded projections. Density-dependence contributed to decline under increased proportional mortality in both (declines seen from RHS to LHS along x-axis), with the full proportional impact causing loss across much of the projected populations (mean −24%; Figure 2, panels 1A–1J). In the fixed mortality analyses results varied between the closed and re-seeded simulations. Increasing mortality resulted in higher vulnerability predominantly under higher environmental stochasticity in the closed analyses (decline seen in populations nearer the upper limit of the y-axis (Figure 2 panels 2A–2E)) with decline more prominent under higher density-dependence in the re-seeded versions (Figure 2, panels 2F–2J). Closed, fixed take simulations with the highest mortality experienced extinction across density-dependence and environmental stochasticity combinations. In contrast re-seeded simulations under the same conditions averaged −39% change (Figure 2, panels 2E and 2J).

3.3 | Mortality treatment results kittiwake

The kittiwake closed baseline simulations indicated extinction and therefore regulation by environmental stochasticity to all projections above a clear threshold (Figure 3, panels 1A and 2A). Projections below this level showed increases but no pattern was seen from density-dependence. The re-seeded baseline (Figure 3, panels 1F and 2F) showed increases in the areas previously declining or extinct in the closed scenarios. Closed baseline scenarios showed a mean population drop across the simulated space, whilst the re-seeded baseline found a mean increase in population change across projections (Figure 3, panels 1A, 2A and 1F, 2F). In the closed simulations, as mortality treatment was applied, both proportional (Figure 3 panels 1A–1E) and fixed (Figure 3, panels 2A–2E) declined, with the fixed declining to a greater extent than the proportional projections. The re-seeded simulations were found to also decline as mortality treatment was increased, the decline was more gradual than their closed

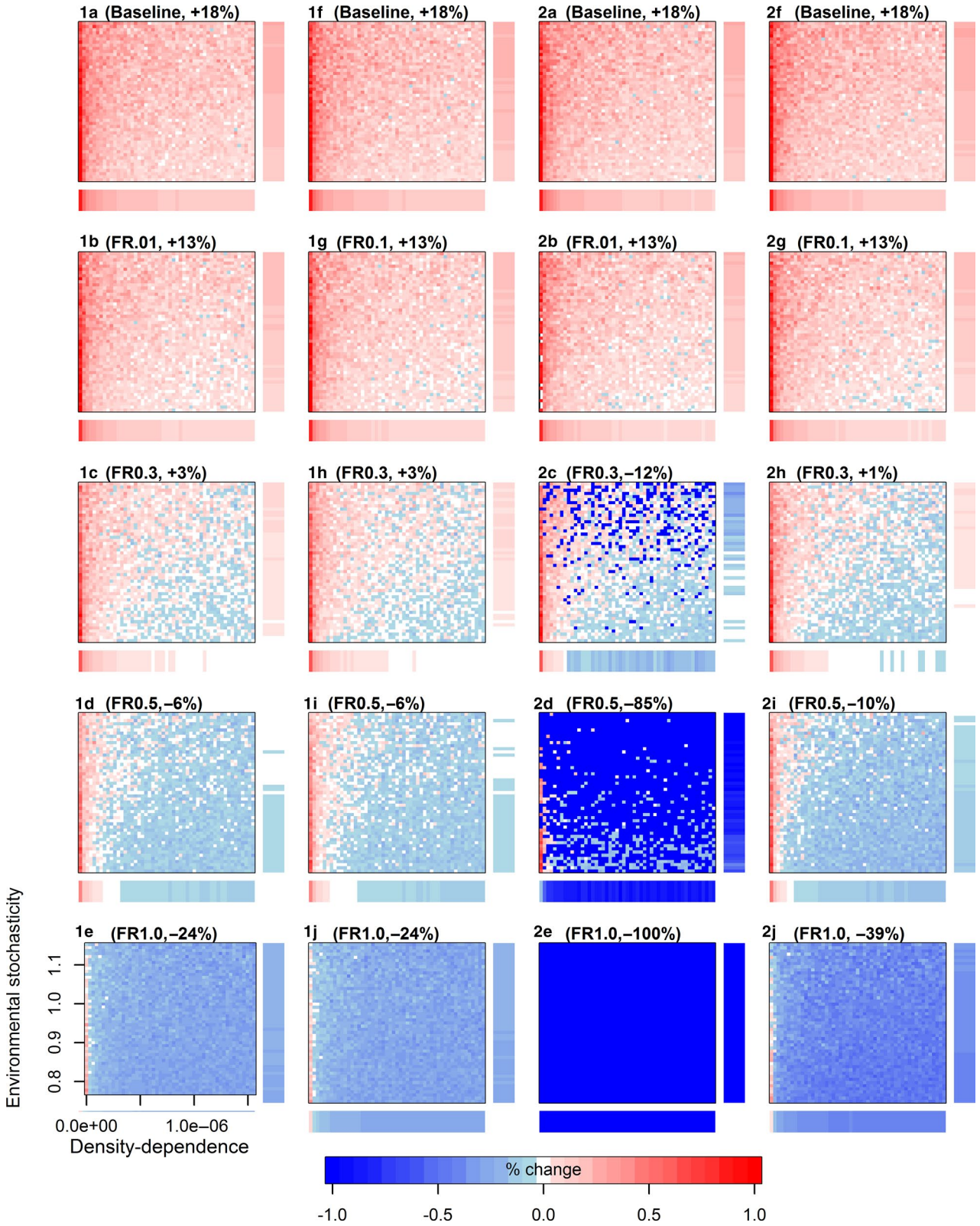

FIGURE 2 Gannet population projections under estimates of strength of density-dependence (*x*-axis) and environmental stochasticity (*y*-axis). Closed proportional removal (1A–1E) and re-seeded (1F–1J). Closed fixed removal (2A–2E) and re-seeded (2F–2J). A-J represent different mortality treatments, reporting the corresponding impact (mean population change (%) pre- and post-mortality) across each plot. The bands to the right and below each plot represent mean % change across rows and columns. A & F—no additional mortality (baseline); B &G—additional mortality with PBR FR of 0.1; C & H—additional mortality with PBR FR of 0.3; D & I—additional mortality with PBR FR of 0.5, E & J—additional mortality with PBR FR of 1.0. Coloration indicates impact. Blues indicate population decline, white indicate zero change and pinks through to red indicate population increases, with darkest reds being any proportional increase of 1.0 or greater

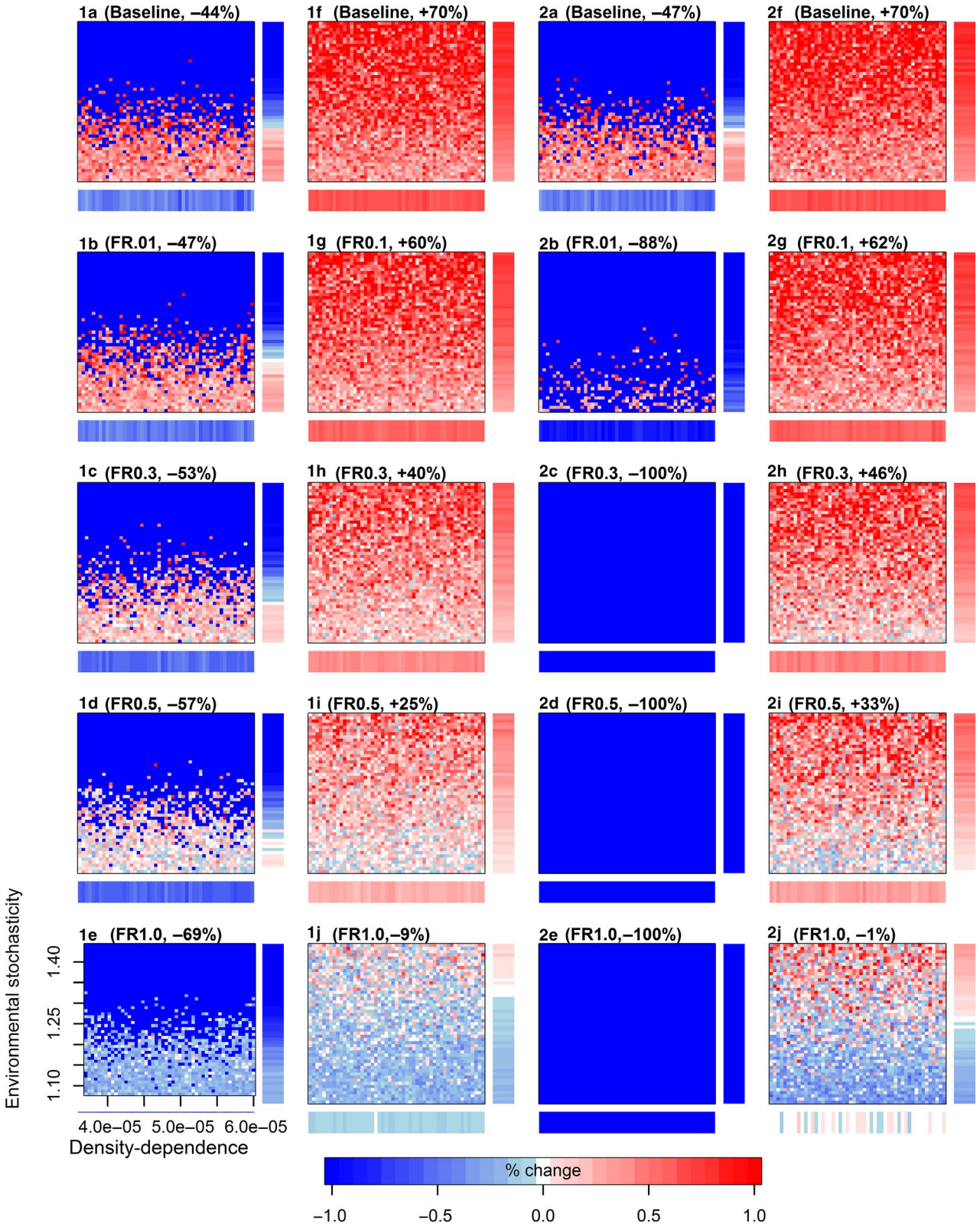

FIGURE 3 Kittiwake population projections under estimates of strength of density-dependence (*x*-axis) and environmental stochasticity (*y*-axis). Closed proportional removal (1A–1E) and re-seeded (1F–1J). Closed fixed removal (2A–2E) and re-seeded (2F–2J). A-J represent different mortality treatments reporting the corresponding impact (mean population change (%) pre- and post-mortality) across each plot. The bands to the right and below each plot represent mean % change across rows and columns. A & F—no additional mortality (baseline); B &G—additional mortality with PBR FR of 0.1; C & H—additional mortality with PBR FR of 0.3; D & I—additional mortality with PBR FR of 0.5, E & J—additional mortality with PBR FR of 1.0. Coloration indicates impact. Blues indicate population decline, white indicate zero change and pinks through to red indicate population increases, with darkest reds being any proportional increase of 1.0 or greater

counterparts (Figure 3, panels A–J). Different magnitudes of decline were seen between the proportional and fixed re-seeded simulations (Figure 3, panels G–J). As mortality increased, those populations facing a proportional mortality reduced more than those under a fixed mortality. Overwhelmingly, re-seeded simulations maintained positive growth (dark red pixels) under high environmental stochasticity, whilst those simulations under increased mortality and lower environmental stochasticity were found to decline (blue pixels; Figure 3, panels F–J).

3.4 | Mortality treatment results guillemot

The guillemot closed baseline projections indicated clear extinction and therefore regulation by environmental stochasticity to all projections above ~"2.2" strength. The re-seeded populations however reversed this effect, with increases shown (Figure 4, panels 1A, 2A, 1F and 2F). In the closed simulations all mortality treatments reported extinctions, with mean values higher than the baseline. However, the risk of decline as mortality increased was reduced in the proportional harvests comparative to the fixed harvest rate (Figure 4, panels 1B–1E, 2B–2E). The re-seeded simulations were found to also decline as mortality treatment was increased (Figure 4, panels G–J), the decline was more gradual than their closed counterparts. In the re-seeded simulations a very different pattern was observed than in the closed. Here, under lower mortality those populations experiencing neither intermediate environmental conditions experienced decline (Figure 4, panels F–I). As mortality increased, this pattern enhanced, capturing more of the middle band of environmental stochasticity as vulnerable to decline and eventually at the highest proportional mortality causing decline even in those projections with lower environmental stochasticity (Figure 4, panels 1G–1J). There is also a very slight hint at density-dependent risk to decline from the bottom right-hand side of the highest mortality (Figure 4 plot 1J and 2J) comparative to those projections with lower density-dependence on the bottom left-hand side of the same plots.

4 | DISCUSSION

Population persistence is a function of quantifiable effects (e.g. mortality from collisions at wind farms), population dynamics (life history, strength of density-dependence, connectivity) and random effects (variable environmental conditions) and these may conspire to cause declines in seemingly stable populations. Quantifying internal and external processes and associated population change are still fundamental problems in population biology and conservation (Elton, 1924; Matthiopoulos et al., 2015; Turchin, 1995).

In our study, exploring extended ranges for the strength of density-dependence and environmental stochasticity allowed a visual comparison of vulnerability between our study species. All three-study species presented extinctions under higher strength of density-dependence (penalized fecundity) and high environmental stochasticity (high unpredictability), but the shape of these effects varied depending on each species' demography. The gannet and guillemot time series we used for model fitting were intermittent. These lower sample sizes and the necessary data-imputation carried out by our model, extended the time necessary for convergence and generated broader credible intervals than we might expect with more data-rich populations (Appendix S1(A11)). Hence, our broader ranges of scenarios examined along the environmental stochasticity axis, for these populations, are also the result of greater observational uncertainty.

Under the explicit assumptions of population structure in these simulations, i.e. closedness, and in the absence of additional mortality, the baseline windows derived from the model fitting show kittiwake and guillemot straddle a well-defined threshold separating proliferation and extinction- mediated by environmental stochasticity. Notably, the gannets are not operating close to this threshold, possibly reflecting the fact that although current population growth is slowing down, the Bass Rock population has been increasing and gannets are arguably more robust to environmental perturbations due their life-history (Garthe, Montevecchi, & Davoren, 2011; Montevecchi, Benvenuti, Garthe, Davoren, & Fifield, 2009; Wanless, Murray, & Harris, 2005). When we allowed rescue-effects, the picture changed for kittiwakes and guillemots. There is a clear indication, particularly for kittiwake, that population re-seeding can offset detrimental environmental stochasticity. The results are similar albeit more stochastic for guillemot where re-seeded simulations also offset the magnitude of decline under certain mortality treatments.

This work highlights several findings of interest to conservation strategists and population assessments. Firstly, the PBR calculation, is questionable in its theoretical sustainability of the population. Results here show clearly that for all species there were some stark reductions from the baseline mean population size under mortality estimated using PBR, exacerbated by regulatory processes. This was seen in both the closed and re-seeded simulations of each mortality type (fixed or proportional), where population baselines were not maintained. Whilst perhaps some lower levels of PBR applied mortality may be considered "sustainable", a switch in regulation may further reduce a population, potentially affecting sustainability. PBR has come under criticism from conservationists and industry advisors as to its appropriateness in assessment and is now broadly considered unsuitable for seabird assessment in the U.K. EIA process. The results here agree with recent work suggesting that it should not be utilized in seabird population management (Green et al., 2016; O'Brien et al., 2017).

Secondly, there is no permissible value of decline to a population under assessment from industry impact. In renewable energy impact assessment, mortality is usually applied as a proportion thereby treating the impact as a per capita effect. Intuitively, we expect a fixed mortality to have a stronger effect on a declining population and a weaker effect on an increasing population. In most of our treatments the proportional application of mortality conveyed a reduction in mean decline comparable to the fixed mortality. However, our results also indicated different patterns in regulation and risk when the same population baseline was

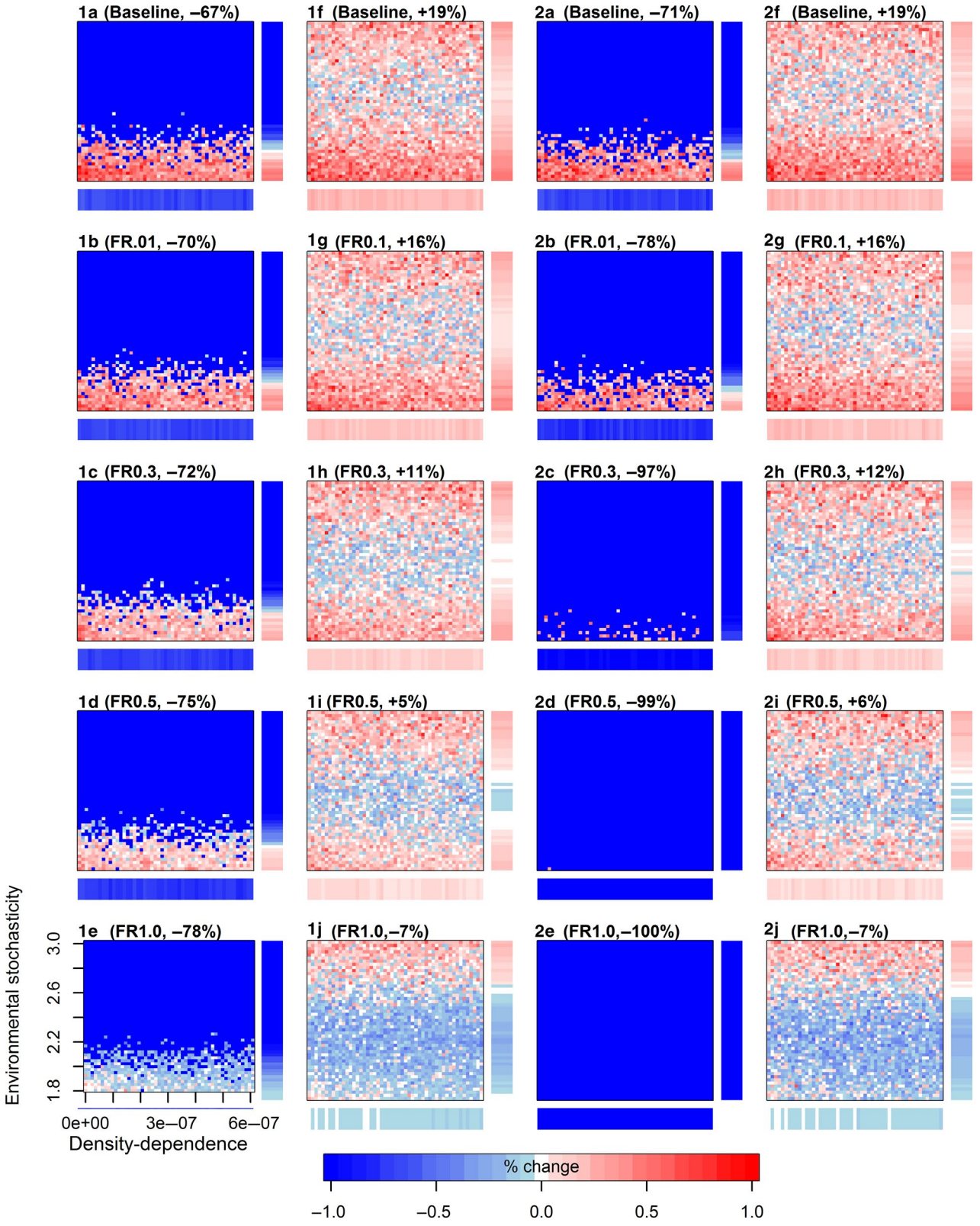

FIGURE 4 Guillemot population projections underestimates of strength of density-dependence (*x*-axis) and environmental stochasticity (*y*-axis). Closed proportional removal (1A–1E) and re-seeded (1F–1J). Closed fixed removal (2a–2e) and re-seeded (2F–2J). A-J represent different mortality treatments reporting the corresponding impact (mean population change (%) pre- and post-mortality) across each plot. The bands to the right and below each plot represent mean % change across rows and columns. A & F—no additional mortality (baseline); B &G—additional mortality with PBR FR of 0.1; C & H—additional mortality with PBR FR of 0.3; D & I—additional mortality with PBR FR of 0.5, E & J—additional mortality with PBR FR of 1.0. Coloration indicates impact. Blues indicate population decline, white indicate zero change and pinks through to red indicate population increases, with darkest reds being any proportional increase of 1.0 or greater.

subjected to a proportional or fixed mortality. For example, in the kittiwake re-seeded models, risk of decline from mortality was greater to those projections under lower environmental stochasticity compared to the closed projections; the decline from baseline was marginally greater under a proportional mortality than a fixed when re-seeded. This suggests that regulation from environmental stochasticity can mediate between mortality estimate and population size to either enhance the buffering effects of the re-seeding to decline or effectively absorb the buffering re-seed. In such cases the fixed calculation may be more conservative in application than a proportional, dependent on starting population size, period of mortality and subsequent environmental stochasticity. The closed simulations showed that environmental conditions and a fixed mortality will result in extinctions. This suggests that the population dynamics, starting population size, time-series trend and regulation is crucial to consider when applying a mortality treatment. For example, the gannets at Bass Rock, an increasing population thought to slowly be reaching their carrying capacity (k) (Murray, Harris, & Wanless, 2015) reflected no difference in results between the closed and re-seeded simulations and a proportional take. Given the inherent positive growth of this population, re-seeding the population has very little influence on the risk of extinction. Density-dependent regulation was clear in patterns from multiple mortality treatments applied to the gannets; this does suggest that, particularly in the case of the gannets that they are likely to be sensitive to regulation from their conspecifics, such that mortality and higher strengths of density-dependence (penalized maximal fecundity) work together to confer decline. Our re-seeded models for guillemot and kittiwake returned a band of decline at intermediate and lower environmental stochasticity respectively. This suggests that the guillemot, in intermediate levels of environmental stochasticity are either being simulated with successive "bad years", lowering k and exacerbating density-dependence penalties to fecundity (reflected as a decline in % change between the means we are comparing across time-scales) or hit by successive "good years" mediated by density-dependence and show no change. For the kittiwake example of more decline in lower fluctuations of environmental stochasticity, two scenarios may be unfolding. In higher fluctuations of successive "good years" of environmental stochasticity, density-dependence becomes irrelevant and is over-ridden by radical growth or in higher levels of negative environmental perturbations, re-seeding forces a recovery, biasing our proxy for impact (this % change).

With a paucity of reliable connectivity estimates between populations of the same species, closed system modelling is the preferred precautionary approach to population assessment. There is clear evidence of connectivity through immigration and emigration in many seabird populations (Horswill & Robinson, 2015) and low rates of either may impact on colony population growth. Our basic theoretical re-seeded models followed classic metapopulation theory where local extinctions have the potential for local increases in the face of connectivity for our kittiwake and guillemot (Hanski & Gilpin, 1997). Our results show a clear reduction in sensitivity to decline for many projections under this basic rescue-effect, but what it does not reveal is the true spatial population dynamics of connectivity between colonies of each species. For example, the risk of decline and extinction of a colony, we can hypothesize, will be different for those acting as sinks rather than sources within a metapopulation structure (Sanz-Aguilar, Igual, Tavecchia, Genovart, & Oro, 2016). It would be of benefit to industry and conservation objectives alike to encourage research empirically and theoretically exploring connectivity estimates to examine this dynamic further in the context of regulation and risk.

Thirdly, the ongoing debate around the inclusion or exclusion of density-dependence in population assessment modelling derives from the stance that density-independent models are a more precautionary approach. Our results indicate that risk of decline was enhanced by the penalty effect on fecundity at higher strengths of density-dependence under certain conditions upon application of even our lowest mortality. Density-dependence was included as a compensatory mechanism in the models. However, there is a growing literature suggesting other forms of density-dependence may be appropriate to explore in future adaptations of these models, particularly in the case of species such as kittiwake, where severely reduced population sizes can experience enhanced predation, accelerating declines (Horswill & Robinson, 2015). Here we explore the effect of direct mortality, but guillemot may suffer displacement (Furness et al., 2013). This may manifest a different pattern in impact under regulation, the ecological mechanisms of which are not captured here, but we provide a baseline method against which such effects may also be assessed.

5 | CONCLUSIONS

Seabird populations are dynamic systems, regulated by extrinsic and intrinsic influences. Insights from this work unveil vulnerability in different seabird populations from these influences even in the absence of additional anthropogenic mortality. Assessment in wildlife systems is rarely able to derive estimates for the strength of intrinsic and extrinsic regulation reducing our confidence in PVA predictions. Clearly, as our results show, unpredictability in the environment is key to population viability and subsequent carrying capacity (Lande, 1993) affecting density-dependent responses on fecundity. We understand that extreme weather events may be more frequent and that climate change is affecting processes like the synchronicity of breeding attempts with prey availability and changing the accessibility of prey spatially. Our approach captures information contained in demography to allow us to theorize about the risk of decline in future, reflecting how our populations may respond to unknown conditions and highlighting how regulation is affected by mortality and the sensitivity of projections to mortality treatment. We show that this method can be applicable to data-limited populations, a benefit of a Bayesian approach. Populations lacking time series for example would produce larger credible intervals, capturing more uncertainty. Care must be taken in these instances to describe the

data-limitation, rather than assume a potential greater effect signal from the process estimated. Complementing current literature, this work determines that PBR is not a rigorous tool for seabird assessment and furthermore we recommend it not be used to facilitate EIA for seabirds. Here we demonstrate that population decline, together with regulation may confer opposing outcomes of risk depending on the population dynamics and the type of mortality applied. Our results suggest that populations experiencing higher environmental perturbations, such as unpredictable foraging sources, require enhanced protection from additional mortality. The responses to the rescue-effect in our analyses were interesting to note between our study species and their underlying dynamics. Further exploration of the connectivity in these systems would be beneficial. For example, our assumption that colonies experiencing more environmental dynamism might be more vulnerable is confounded by the knowledge gaps in the mechanisms of connectivity i.e. to what extent is connectivity driven by extrinsic or intrinsic factors and what is the availability of immigrants for species, such as the kittiwake, where multiple colonies are in decline? We should stress that, in the absence of empirical rates of connectivity, precaution remains with the assumption of a closed-system. Resolution of human-wildlife conflict happens at the space between over-precaution and recklessness. This can only be found and navigated with sound quantitative knowledge of the certainties and uncertainties in the systems involved. Adding realism in impact assessment can only improve upon mediating positive outcomes in both conservation, environmental and economic goals.

ACKNOWLEDGEMENTS

This work was supported by funding from NERC CASE partnership grant: in partnership with MacArthur Green. The Authors thank everyone involved with gathering the datasets they used, particularly Mark Newell and Francis Daunt for facilitating data acquisition. We thank Cat Horswill and the Matthiopoulos lab for useful discussion. We also thank our referees and editors at the Journal of Applied Ecology for constructive feedback.

AUTHORS' CONTRIBUTIONS

R.W.F. and M.T. formulated the original idea, with J.M. and J.A.O.M. developing the idea and approach. J.A.O.M. analysed the data and drafted the original manuscript with all authors providing further editorial advice and final approval for publication.

DATA AVAILABILITY STATEMENT

Data and the full reproducible models are available via the Dryad Digital Repository (Miller, Furness, Trinder, & Matthiopoulos, 2019) https://doi.org/10.5061/dryad.mh4vh5v .

ORCID


Julie A. O. Miller https://orcid.org/0000-0002-4786-7619

REFERENCES

Bonenfant, C., Gaillard, J. M., Coulson, T., Festa-Bianchet, M., Loison, A., Garel, M., … DuToit, J. (2009). Empirical evidence of density-dependence in populations of large herbivores. *Advances in Ecological Research*, *41*, 313–357.

Boyce, M. S. (1992). Population viability analysis. *Annual Review of Ecology and Systematics*, *23*(1), 481–497. https://doi.org/10.1146/annurev.es.23.110192.002405

Bried, J., & Jouventin, P. (2002). Site and mate choice in seabirds: An evolutionary approach. *Biology of Marine Birds*, *435*, 263–305.

Brook, B. W., & Bradshaw, C. J. A. (2006). Strength of evidence for density dependence in abundance time series of 1198 species. *Ecology*, *87*(6), 1445–1451. https://doi.org/10.1890/0012-9658(2006)87[1445:SOEFDD]2.0.CO;2

Brooks, S. P., & Gelman, A. (1997). General Methods for Monitoring Convergence of Iterative Simulations. *Journal of Computational and Graphical Statistics*, *7*, 434–455.

Brown, J. H., & Kodric-Brown, A. (1977). Turnover rates in insular biogeography: Effect of immigration on extinction. *Ecology*, *58*, 445–449. https://doi.org/10.2307/1935620

Buckland, S. T., Goudie, I. B. J., & Borchers, D. L. (2000). Wildlife population assessment: Past developments and future directions. *Biometrics*, *56*(1), 1–12. https://doi.org/10.1111/j.0006-341X.2000.00001.x

Caswell, H. (2001). *Matrix population models*. Sunderland, MA: Sinauer Assoc. Inc.

Clark, J. S. (2003). Uncertainty and variability in demography and population growth: A hierarchical approach. *Ecology*, *84*(6), 1370–1381. https://doi.org/10.1890/0012-9658(2003)084[1370:UAVIDA]2.0.CO;2

Cook, A. S. C. P., & Robinson, R. A. (2016). Testing sensitivity of metrics of seabird population response to offshore wind farm effects, JNCC Report 553, ISSN 0963–8901.

Croxall, J. P., Butchart, S. H., Lascelles, B. E. N., Stattersfield, A. J., Sullivan, B. E. N., Symes, A., & Taylor, P. (2012). Seabird conservation status, threats and priority actions: A global assessment. *Bird Conservation International*, *22*(1), 1–34. https://doi.org/10.1017/S0959270912000020

Denwood, M. J. (2016). runjags: An R package providing interface utilities, model templates, parallel computing methods and additional distributions for MCMC models in JAGS. *Journal of Statistical Software*, *71*(9), 1–25. https://doi.org/10.18637/jss.v071.i09

Elton, C. S. (1924). Periodic fluctuations in the numbers of animals: Their causes and effects. *British Journal of Experimental Biology*, *2*, 119–163.

Engen, S., Bakke, Ø., & Islam, A. (1998). Demographic and environmental stochasticity-concepts and definitions. *Biometrics*, *54*(3), 840–846. https://doi.org/10.2307/2533838

European Commission. (2009). European Commission Directive 2009/147/EC of the European Parliament and the Council of 30 November 2009. *Official Journal of the European Union L*, *20* (7).

European Commission. (2011). *EU guidance on wind energy development in accordance with the EU nature legislation*. Luxembourg: Publications Office of the European Union.

Frederiksen, M., Daunt, F., Harris, M. P., & Wanless, S. (2008). The demographic impact of extreme events: Stochastic weather drives survival and population dynamics in a long-lived seabird. *Journal of Animal Ecology*, *77*(5), 1020–1029. https://doi.org/10.1111/j.1365-2656.2008.01422.x

Frederiksen, M., Mavor, R. A., & Wanless, S. (2007). Seabirds as environmental indicators: The advantages of combining data sets. *Marine Ecology Progress Series*, *352*, 205–211. https://doi.org/10.3354/meps07071

Furness, R. W., Wade, H. M., & Masden, E. A. (2013). Assessing vulnerability of marine bird populations to offshore wind farms. *Journal of*

Environmental Management, *119*, 56–66. https://doi.org/10.1016/j.jenvman.2013.01.025

Garthe, S., Montevecchi, W. A., & Davoren, G. K. (2011). Inter-annual changes in prey fields trigger different foraging tactics in a large marine predator. *Limnology and Oceanography*, *56*, 802–812. https://doi.org/10.4319/lo.2011.56.3.0802

Green, R. E., Langston, R. H., McCluskie, A., Sutherland, R., & Wilson, J. D. (2016). Lack of sound science in assessing wind farm impacts on seabirds. *Journal of Applied Ecology*, *53*(6), 1635–1641. https://doi.org/10.1111/1365-2664.12731

Hanski, I. (1998). Metapopulation dynamics. *Nature*, *396*(6706), 41–49. https://doi.org/10.1038/23876

Hanski, I., & Gilpin, M. E. (1997). *Metapopulation biology. Ecology, genetics, and evolution*. Cambridge, MA: Academic Press Limited.

Horswill, C., O'Brien, S. H., & Robinson, R. A. (2016). Density dependence and marine bird populations: Are wind farm assessments precautionary? *Journal of Applied Ecology*, *54*(5), 1406–1414.

Horswill, C., & Robinson, R. A. (2015). Review of seabird demographic rates and density dependence. *JNCC Report*, no. 552.

Jenouvrier, S., Barbraud, C., Cazelles, B., & Weimerskirch, H. (2005). Modelling population dynamics of seabirds: Importance of the effects of climate fluctuations on breeding proportions. *Oikos*, *108*(3), 511–522. https://doi.org/10.1111/j.0030-1299.2005.13351.x

Johnston, A., Cook, A. S., Wright, L. J., Humphreys, E. M., & Burton, N. H. (2014). Modelling flight heights of marine birds to more accurately assess collision risk with offshore wind turbines. *Journal of Applied Ecology*, *51*(1), 31–41. https://doi.org/10.1111/1365-2664.12191

Kass, R. E., Carlin, B. P., Gelman, A., & Neal, R. M. (1998). Markov chain Monte Carlo in practice: A roundtable discussion. *The American Statistician*, *52*(2), 93–100.

Kindsvater, H. K., Dulvy, N. K., Horswill, C., Juan-Jordá, M. J., Mangel, M., & Matthiopoulos, J. (2018). Overcoming the data crisis in biodiversity conservation. *Trends in Ecology & Evolution*, *33*(9), 676–688. https://doi.org/10.1016/j.tree.2018.06.004

Knape, J., & deValpine, P. (2012). Are patterns of density dependence in the Global Population Dynamics Database driven by uncertainty about population abundance? *Ecology Letters*, *15*(1), 17–23.

Lande, R. (1993). Risks of population extinction from demographic and environmental stochasticity and random catastrophes. *The American Naturalist*, *142*(6), 911–927. https://doi.org/10.1086/285580

Lande, R., Engen, S., & Saether, B. E. (2003). *Stochastic population dynamics in ecology and conservation*. Oxford University Press on Demand.

Lande, R., Engen, S., Sæther, B. E., Filli, F., Matthysen, E., & Weimerskirch, H. (2002). Estimating density dependence from population time series using demographic theory and life-history data. *The American Naturalist*, *159*(4), 321–337. https://doi.org/10.1086/338988

Leirs, H., Stenseth, N. C., Nichols, J. D., Hines, J. E., Verhagen, R., & Verheyen, W. (1997). Stochastic seasonality and nonlinear density-dependent factors regulate population size in an African rodent. *Nature*, *389*, 176–180. https://doi.org/10.1038/38271

Lewis, S., Elston, D. A., Daunt, F., Cheney, B., & Thompson, P. M. (2009). Effects of extrinsic and intrinsic factors on breeding success in a long lived seabird. *Oikos*, *118*(4), 521–528. https://doi.org/10.1111/j.1600-0706.2008.17308.x

Marques, A. C., Fuinhas, J. A., & Manso, J. P. (2010). Motivations driving renewable energy in European countries: A panel data approach. *Energy Policy*, *38*(11), 6877–6885. https://doi.org/10.1016/j.enpol.2010.07.003

Matthiopoulos, J., Fieberg, J., Aarts, G., Beyer, H. L., Morales, J. M., & Haydon, D. T. (2015). Establishing the link between habitat selection and animal population dynamics. *Ecological Monographs*, *85*(3), 413–436. https://doi.org/10.1890/14-2244.1

Miller, J. A. O., Furness, R. W., Trinder, M., & Matthiopoulos, J. (2019). Data from: The sensitivity of seabird populations to density-dependence, environmental stochasticity and anthropogenic mortality. Dryad Digital Repository, https://doi.org/10.5061/dryad.mh4vh5v

Montevecchi, W. A., Benvenuti, S., Garthe, S., Davoren, G. K., & Fifield, D. (2009). Flexible foraging tactics by a large opportunistic seabird preying on forage-and large pelagic fishes. *Marine Ecology Progress Series*, *385*, 295–306. https://doi.org/10.3354/meps08006

Murphy, G. E., & Romanuk, T. N. (2014). A meta-analysis of declines in local species richness from human disturbances. *Ecology and Evolution*, *4*(1), 91–103. https://doi.org/10.1002/ece3.909

Murray, S., Harris, M. P., & Wanless, S. (2015). The status of the gannet in Scotland in 2013–14. *Scottish Birds*, *35*(1), 3–18.

Niel, C., & Lebreton, J. D. (2005). Using demographic invariants to detect overharvested bird populations from incomplete data. *Conservation Biology*, *19*(3), 826–835. https://doi.org/10.1111/j.1523-1739.2005.00310.x

O'Brien, S. H., Cook, A. S. C. P., & Robinson, R. A. (2017). Implicit assumptions underlying simple harvest models of marine bird populations can mislead environmental management decisions. *Journal of Environmental Management*, *201*, 163–171. https://doi.org/10.1016/j.jenvman.2017.06.037

Oro, D., and Martinez-Abrain, A. (2004). Ecology and behaviour of seabirds. C. M. Duarte (Ed.). *The Encyclopedia of Life Support Systems (EOLSS)*. Oxford, UK: Eolss Publishers UNESCO.

Platteeuw, M., Bakker, J., van den Bosch, I., Erkman, A., Graafland, M., Lubbe, S., & Warnas, M. (2017). A Framework for Assessing Ecological and Cumulative Effects (FAECE) of offshore wind farms on birds, bats and marine mammals in the Southern North Sea J. Köppel (Ed.). In *Wind energy and wildlife interactions* (pp. 219–237). Cham, Switzerland: Springer International Publishing.

Plummer, M. (2003). A program for analysis of Bayesian graphical models using Gibbs sampling. *Proceedings of the 3rd International Workshop on Distributed Statistical Computing*.

R Core Team. (2016). *R: A language and environment for statistical computing*. Vienna, Austria: R Foundation for Statistical Computing. Retrieved from https://www.R-project.org/

Reed, J. M., Mills, L. S., Dunning, J. B., Menges, E. S., McKelvey, K. S., Frye, R., … Miller, P. (2002). Emerging issues in population viability analysis. *Conservation Biology*, *16*(1), 7–19. https://doi.org/10.1046/j.1523-1739.2002.99419.x

Ripple, W. J., Chapron, G., López-Bao, J. V., Durant, S. M., Macdonald, D. W., Lindsey, P. A., … Zhang, L. I. (2016). Saving the world's terrestrial megafauna. *BioScience*, *66*(10), 807–812. https://doi.org/10.1093/biosci/biw092

Saether, B.-E., Engen, S., Lande, R., Both, C., & Visser, M. E. (2002). Density dependence and stochastic variation in a newly established population of a small songbird. *Oikos*, *99*, 331–337. https://doi.org/10.1034/j.1600-0706.2002.990214.x

Sands, P., & Peel, J. (2012). *Principles of international environmental law*. Cambridge University Press.

Sanz-Aguilar, A., Igual, J. M., Tavecchia, G., Genovart, M., & Oro, D. (2016). When immigration mask threats: The rescue effect of a Scopoli's shearwater colony in the Western Mediterranean as a case study. *Biological Conservation*, *198*, 33–36. https://doi.org/10.1016/j.biocon.2016.03.034

Saunders, S. P., Cuthbert, F. J., & Zipkin, E. F. (2018). Evaluating population viability and efficacy of conservation management using integrated population models. *Journal of Applied Ecology*, *55*, 1–13. https://doi.org/10.1111/1365-2664.13080

Turchin, P. (1995). Population regulation: Old arguments and a new synthesis. *Population Dynamics: New Approaches and Synthesis*, 19–40.

Wade, P. R. (1998). Calculating limits to the allowable human-caused mortality of cetaceans and pinnipeds. *Marine Mammal Science*, *14*(1), 1–37. https://doi.org/10.1016/j.biocon.2011.04.0142005

Wanless, S., Murray, S., & Harris, M. P. (2005). The status of northern gannet in Britain & Ireland in 2003/04. *British Birds*, *98*, 280–294.

Weimerskirch, H. (2001). Seabird demography and its relationship with the marine environment. In E. A. Schreiber, & J. Burger (Eds.), *Biology of marine birds* (pp. 115–136). Boca Raton, FL: CRC Press.

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

How to cite this article: Miller JAO, Furness RW, Trinder M, Matthiopoulos J. The sensitivity of seabird populations to density-dependence, environmental stochasticity and anthropogenic mortality. *J Appl Ecol*. 2019;56:2118–2130. https://doi.org/10.1111/1365-2664.13448