

1 **Estimating spatially variable and density-dependent** 2 **survival using open-population spatial capture-recapture** 3 **models**

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12 **Open Research statement:** code to reproduce the analysis is available on github;

13 <https://github.com/Cyril-Milleret/Public/tree/master/SpatialSurvivalOPSCR>

14 **Abstract:**

15 Open-population spatial capture-recapture (OPSCR) models use the spatial information
16 contained in individual detections collected over multiple consecutive occasions to estimate
17 occasion-specific density, but also demographic parameters. OPSCR models can also estimate
18 spatial variation in vital rates, but such models are neither widely used nor thoroughly tested.

19 We developed a Bayesian OPSCR model that not only accounts for spatial variation in survival
20 using spatial covariates, but also estimates local density-dependent effects on survival within a

21 unified framework. Using simulations, we show that OPSCR models provide sound inferences
22 on the effect of spatial covariates on survival, including multiple competing sources of
23 mortality, each with potentially different spatial determinants. Estimation of local density-

24 dependent survival was possible but required more data due to the greater complexity of the
25 model. Not accounting for spatial heterogeneity in survival led to positive bias in abundance
26 estimates (up to 10% relative bias). We provide a set of features in R package nimbleSCR that
27 allow computationally efficient fitting of Bayesian OPSCR models with spatially varying
28 survival. The ability to make population-level inferences of spatial variation in survival is an

29 essential step towards a fully spatially-explicit OPSCR model that can disentangle the role of
30 multiple spatial drivers on population dynamics.

31 **1. Introduction**

32 Spatial capture recapture (SCR) models are hierarchical models that explicitly use the spatial
33 information contained in repeated individual detections to account for imperfect detection and
34 estimate density (Efford 2004, Borchers and Efford 2008, Royle et al. 2014). Because SCR
35 models are spatially explicit and accommodate various types of data (e.g., physical capture,
36 photographic, non-invasive genetic, acoustic), they are now routinely used to analyze wildlife
37 monitoring data. When data are collected over several consecutive occasions, open-population
38 SCR (OPSCR) models can be used to estimate demographic rates and movement of individuals
39 between occasions in addition to densities (Bischof et al. 2020a). Modelling individual
40 movement between occasions can help distinguish between the different causes of individual
41 disappearances from the population (Gardner et al. 2018). These properties make OPSCR
42 models well-suited for drawing population-level inferences about the drivers of demographic
43 processes.

44 Demographic rates, such as survival, are known to vary in time (Gaillard et al. 2000), with
45 individual attributes (de Valpine et al. 2014), and across space (DeCesare et al. 2014).
46 Temporal and individual variation of demographic parameters can be readily integrated in
47 OPSCR models (Augustine et al. 2019, Bischof et al. 2020a), and the possibility of inferring
48 spatial heterogeneity in survival using OPSCR models has been suggested (Royle et al. 2014)
49 and applied (Chandler et al. 2018). However, the performance of models that estimate spatially-
50 variable survival has not been thoroughly tested and their potential remains under-exploited.
51 Estimation of spatially varying vital rates is a key step in the development of OPSCR models

52 (Royle et al. 2014) as it will lead to a better understanding of the processes driving the spatial
53 distribution of individuals (Pulliam 1988).

54 At their core, OPSCR models account for imperfect detection by using an observation process
55 which assumes that an individual's probability of detection is a function of distance from its
56 activity center (AC) (Borchers and Efford 2008, Royle et al. 2014). The location of individual
57 ACs is a latent quantity and is a representation of the center of the individual's home range.
58 AC locations are a key quantity of OPSCR models as they allow the estimation of density and
59 inter-annual movement. AC locations also provide the spatial information necessary to
60 characterize the environment in which individuals are located, and therefore its influence on
61 survival (Chandler et al. 2018).

62 Density itself can be a key driver of survival (Gaillard et al. 2000). The study of density-
63 dependent survival has often been limited to estimating the average population response to
64 variation in overall population size through time (Bonenfant et al. 2009). However, variation
65 in density is a spatiotemporal process and individuals within the population may not experience
66 the same density. OPSCR models, by estimating spatio-temporal variation in density, offer a
67 unique opportunity to study density-dependence in survival at the local scale while accounting
68 for variation and uncertainty in both density and survival within a unified framework.

69 Here, we present a Bayesian OPSCR model that accounts for spatial variation in survival as a
70 function of spatial covariates (e.g., characteristics of the landscape, resources availability) and
71 density. We model survival using a hazard rate formulation to allow inferences on spatial
72 variation in competing risks of mortality (Ergon et al. 2018). We quantify model performance
73 by simulating OPSCR datasets under a wide range of scenarios. In addition, we quantify the
74 consequence of ignoring spatial heterogeneity in mortality for OPSCR inferences. All
75 functionalities are made available in R package nimbleSCR which provides tools for fitting

76 efficient Bayesian (Markov chain Monte Carlo) MCMC models (de Valpine et al. 2017,
77 NIMBLE Development Team 2019, Bischof et al. 2020b, Turek et al. 2021).

78 **2. Methods**

79 **2.1. OPSCR model**

80 To estimate spatial variation in mortality from live encounter and dead recoveries collected
81 over several consecutive occasions (hereafter “years”), we built a Bayesian hierarchical state-
82 space OPSCR model. The model is composed of four sub-models for 1) density and inter-
83 annual movement, 2) demography, 3) live detections, and 4) dead recoveries. (Royle et al.
84 2014, Bischof et al. 2020a, Milleret et al. 2020, 2021, Dupont et al. 2021). We created two
85 versions of the model. The first version can distinguish between two spatially-variable and
86 competing causes of mortality. For example, it is possible to distinguish between culling and
87 other causes of mortality in the case that all individuals culled are recovered dead (Bischof et
88 al. 2020a). The second model version only considered one cause of mortality. This reflects a
89 realistic limitation because dead recoveries are not always available in OPSCR datasets and
90 distinguishing between multiple causes of death may not be possible.

91 **2.1.1. *Spatial distribution and movement submodel***

92 In SCR models, the location of individuals is represented by their activity centers (ACs) within
93 the spatial domain (S). We used a Bernoulli point process with spatial intensity $\lambda(s)$ to model
94 the distribution of ACs, where s is a vector of spatial coordinates of ACs (Zhang et al. 2020).
95 For $t \geq 1$, the probability density of $s_{i,t}$ is conditional on the Euclidean distance to $s_{i,t-1}$:

$$96 \lambda(s_{i,t} | s_{i,t-1}, \tau) \propto e^{-\frac{\|s_{i,t} - s_{i,t-1}\|^2}{2\tau^2}} \quad \text{eqn 1}$$

97 where τ is the standard deviation of a bivariate normal distribution centered on $s_{i,t-1}$. This
98 represents movement between $t-1$ and t and helps distinguish between mortality and emigration
99 (Gardner et al. 2018).

100 **2.1.2. Demographic submodel**

101 We used a multistate formulation (Lebreton and Pradel 2002) where each individual life history
102 is represented by a succession of up to four discrete states $z_{i,t}$: 1) “unborn” if the individual has
103 not been recruited in the population; 2) “alive” if it is alive; 3) “culled” if it was culled and
104 therefore recovered dead between the start of the previous and current occasion; or 4) “dead”:
105 if it has died but was not recovered dead. We used data augmentation, whereby additional,
106 undetected individuals are available for inclusion in the population at each time step (Royle et
107 al. 2007, Royle and Dorazio 2012).

108 During the first year, individuals can only be designated as “unborn” ($z_{i,1}=1$) or “alive” ($z_{i,1}=2$)
109 so that $z_{i,1} \sim \text{dcat}(1-\gamma_1, \gamma_1, 0, 0)$ where γ_1 represents the probability to be “alive” at time $t=1$.

110 For $t \geq 2$, $z_{i,t}$ is conditional on the state of individual i at $t-1$:

111 • If $z_{i,t-1} = 1$, individual i can be recruited (i.e., transition to state 2) with probability γ_t ,
112 or remain unborn with probability $1 - \gamma_t$, so $z_{i,t} \sim \text{dcat}(1 - \gamma_t, \gamma_t, 0, 0)$.

113 • If $z_{i,t-1} = 2$, individual i can survive with probability ϕ_i and remain $z_{i,t}=2$. If it does not
114 survive, it can either die due to culling and be recovered (transition to $z_{i,t}=3$) with
115 probability h_i , or die from other causes without being recovered (transition to $z_{i,t}=4$)
116 with probability w_i , so that $z_{i,t} \sim \text{dcat}(0, \phi_i, h_i, w_i)$, where $\Phi_i = 1 - h_i - w_i$.

117 • All individuals in dead states ($z_{i,t-1} = 3$ or 4) transition to $z_{i,t-1} = 4$, the absorbing state,
118 with probability 1 , so that $z_{i,t} \sim \text{dcat}(0, 0, 0, 1)$

119 Abundance estimates are obtained by $\hat{N}_t = \sum_{i=1}^M I(z_{i,t} = 2)$, where $I(z_{i,t} = 2)$ is an indicator
120 function to count alive individuals, and M is the number of detected and augmented individuals.

121 **Parameterization with mortality hazard rates** When an individual dies from a specific
122 cause, it is no longer available to die from another cause. Therefore, mortality causes are
123 competing and non-independent (Ergon et al. 2018). Following the recommendation of (Ergon
124 et al. 2018), we parameterized the model using mortality hazard rates instead of mortality
125 probabilities. We expressed survival and mortality probabilities as functions of the culling
126 hazard rate (m_h) and the hazard rate associated with to all other causes of mortality combined
127 (m_w). For simplicity, we assumed that the hazard rate from culling and other causes remained
128 proportional within time intervals:

129
$$\phi_i = \exp \left(- \left(m_{h_i} + m_{w_i} \right) \right) \quad \text{eqn 2}$$

130
$$h_i = (1 - \phi_i) \left(\frac{m_{h_i}}{m_{w_i} + m_{w_i}} \right) \quad \text{eqn 3}$$

131
$$w_i = (1 - \phi_i) \left(\frac{m_{w_i}}{m_{h_i} + m_{w_i}} \right) \quad \text{eqn 4}$$

132 **Spatial and individual variation in mortality** We accounted for spatial variation in cause-
133 specific mortality by modelling mortality hazard rates (m_h and m_w) as functions of a spatial
134 covariate $SpatialCov_{s_i,t}$ at the location of the AC ($s_{i,t}$):

135
$$\log(m_{H_{i,t}}) = \log(m_{0h}) + \beta_h * SpatialCov_{s_i,t} \quad \text{eqn 5}$$

136
$$\log(m_{W_{i,t}}) = \log(m_{0w}) + \beta_w * SpatialCov_{s_i,t} \quad \text{eqn 6}$$

137 Where β_h and β_w are the coefficients of the relationship between the spatial covariate on
138 culling and other mortality, respectively. m_{0h} and m_{0w} represent the intercept for culling and
139 other mortality hazard rate, respectively.

140 **Density dependent survival** At each occasion, local density within any habitat cell r of S (r
141 $= 1, \dots, R$) can be obtained as $d_{r,t} = \sum_{i=1}^M I(s_{i,t} = r, z_{i,t} = 2)$, where $I(s_{i,t} = r, z_{i,t} = 2)$ is an
142 indicator function denoting whether the individual AC falls within cell r and it is alive.

143 By replacing the *SpatialCov* in Eqn.5-6 with the logarithm of the local density $\log(d_{s_i,t,t})$, we
144 can estimate the effect of local density-dependence on individual mortality between occasion
145 (t-1) and t. Note that since (Eqn 5-6) are on the log scale, transformation of $d_{r,t}$, such as $d_{r,t}' =$
146 $f(d_{r,t}) = d_{r,t} + 1$, is necessary to avoid $\widehat{d_{r,t}} = 0$.

147 **2.1.3. Live detection submodel**

148 We used the half-normal function to model detection probability of individuals alive, whereby
149 the probability $p_{i,j,t}$ of detecting individual i at detector j and time t decreases with distance
150 between the location x of detector j and the AC ($s_{i,t}$):

$$151 \quad p_{i,j,t} = p_0 * \exp\left(-\frac{1}{2\sigma^2} \|s_{i,t} - x_j\|^2\right) \quad \text{eqn 7}$$

152 where p_0 is the baseline detection probability, and σ the scale parameter.

153 The detection $y_{i,j,t}$ of alive individual i at detector j and time t , is modelled as the realization of
154 a Bernoulli process conditional on both the “alive” individual state (i.e., $z_{i,t}=2$) and the
155 individual and detector-specific detection probability $p_{i,j,t}$:

$$156 \quad y_{i,j,t} \sim \text{Bernoulli}(p_{i,j,t} * I(z_{i,t} = 2)) \quad \text{eqn 8}$$

157 **2.1.4. Dead recovery model**

158 To model dead recoveries within S , we used a Bernoulli point process with a bivariate normal
159 density model, where:

$$160 \quad \lambda(y.\text{dead}_{i,t} | s_{i,t}, z_{i,t}, \sigma) \propto e^{-\frac{\|y.\text{dead}_{i,t} - s_{i,t}\|^2}{2\sigma^2}} * I(z_{i,t} = 3). \quad \text{eqn 9}$$

161 where $y.\text{dead}_{i,t}$ is the vector of spatial coordinates of the dead recovery locations. The indicator
162 function is used to condition dead recoveries on the individual being culled and recovered dead.
163 The detection probability function represents space use, we therefore assume that σ , the shape

164 parameter of the detection probability function, is identical for live detections and dead
165 recoveries.

166 **2.2. OPSCR model with a single cause of mortality**

167 To provide an example of the OPSCR model where dead recoveries ($y.\text{dead}$) are not available
168 and cause-specific mortality is not estimable, we built an OPSCR model with only three
169 demographic states z . Individuals could be unborn ($z_{i,t} = 1$), alive ($z_{i,t} = 2$), or dead ($z_{i,t} =$
170 3). For $t > 1$, alive individuals ($z_{i,t-1} = 2$) can survive with probability $\phi_{i,t-1}$ and remain $z_{i,t} =$
171 2 or die with probability $(1 - \phi_{i,t-1})$ and transition to $z_{i,t} = 3$, the dead absorbing state. We
172 modelled the effect of density at occasion t on individual survival between occasion t and $t+1$:

173 $\log(m_{i,t}) = \log(m_0) + \beta_d * \log(d_{s_{i,t}} + 1)$ eqn 10

174 $\phi_{i,t} = 1 - \exp(-m_{i,t})$ eqn 11

175 **2.3. Simulations**

176 We conducted simulations to quantify the performance of 1) the OPSCR model in estimating
177 spatial variation in cause-specific mortalities with a deterministic spatial covariate; of 2) the
178 version of the OPSCR model with integrated density-dependent survival. Finally, we tested the
179 consequences of ignoring spatial variation in survival for abundance estimates.

180 **2.3.1. Using deterministic covariate**

181 We created a spatial domain (S) of 28 x 28 distance units (du) subdivided in $R=49$ cells of 4x4
182 du. We centered in S a 16 x 16 detector grid (with a minimum distance of 1 du between
183 detectors). This configuration left a 6 du buffer around the detector grid where individuals
184 cannot be detected alive. We set $p_0=0.1$, $\sigma=2$, $\tau = 3$ and considered five consecutive occasions.
185 This set-up led to an average of 2 (95% quantiles=1.5-2.3) detections per individual detected,
186 and on average 43% (95% quantiles=34%-51%) of individuals alive detected at each occasion

187 (Appendix 1, table 1). We created a spatially autocorrelated covariate following a diagonal
188 gradient (*SpatialCov*, ranging from (-2 to 2); Figure 1A).

189 We set $M=650$ and $N_1=250$ individuals during the first occasion leading to $\gamma_1 = \frac{N_1}{M} = \frac{250}{650}$. For
190 $t>1$, we assumed a constant 0.3 per capita recruitment rate. We set baseline mortality hazard
191 rates to $m_{0_h}=-2.25$ and $m_{0_w}=-1.75$ and created four different scenarios with all combinations
192 of $\beta_h = (1, -1)$ and $\beta_w = (1, -1)$.

193 We repeated the simulation scenarios described above, but with 1) lower population size,
194 $N_1=120$ ($M=370$), and 2) a spatially random covariate $SpatialCov_r \sim Uniform(-1, 1)$
195 (Appendix 1, table 1). We expected estimation performance for such scenarios to be more
196 challenging due to 1) sparser OPSCR data sets (Appendix 1, table 1), and 2) lower level of
197 spatial autocorrelation and overall variation in mortality. In total, we simulated 100 replicated
198 OPSCR data sets from each of the 16 scenarios (Appendix 1, table 1). We used NIMBLE's
199 simulation feature (de Valpine et al. 2017, NIMBLE Development Team 2019) to simulate
200 OPSCR data sets directly from the nimble OPSCR model (Appendix 4-5).

201 **2.3.2. Using density dependent survival**

202 To illustrate how to estimate density-dependent survival, we used the OPSCR model with a
203 single source of mortality. Preliminary analyses showed that, computationally (convergence,
204 mixing), OPSCR models performed more poorly when survival was modeled as dependent on
205 latent density rather than deterministic spatial covariates. covariate. To counter this, we used a
206 larger spatial domain (S) to increase the number of habitat cells (R) and thus provide the model
207 with more variable latent density points to serve as covariate on the mortality hazard rate. This
208 led to habitat of 40x40 distance units (du) subdivided in $R = 64$ cells of dimension 5x5 du and
209 in which we centered a 30 x 30 detector grid. We set $M=650$ and $N_1 = 250$. We set $m_0 = 1.6$

210 and $\beta_d = -1$ to simulate negative density-dependence on survival and simulated 100 replicated
211 OPSCR data sets (Appendix 1, Table 12).

212 **2.3.3. Ignoring spatial heterogeneity in mortality**

213 To evaluate whether ignoring spatial variation in mortality leads to biased parameter estimates,
214 especially abundance (\hat{N}), we fitted all simulated datasets described above with OPSCR models
215 that assumed constant survival across space and time. As in the earlier simulations, we fitted
216 models with two competing risks for the deterministic spatial covariate scenarios and a single
217 cause of mortality for the density dependent scenario.

218 **2.4. Model fitting**

219 We fitted the Bayesian OPSCR models using Markov chain Monte Carlo (MCMC) simulation
220 with NIMBLE in R version 4.1.0 (R Core Team 2021). We used R package nimbleSCR
221 (Bischof et al. 2020b, Turek et al. 2021) which implements the local evaluation approach
222 (Milleret et al. 2019) to increase MCMC efficiency. For each simulation, we ran three chains
223 of 30,000 (60,000 for density-dependent survival) iterations, including a 2000-iteration burn-
224 in. We considered models as converged when the Gelman-Rubin diagnostic (\hat{R} , (Gelman and
225 Rubin 1992)) was < 1.1 for all parameters and by visually inspecting trace plots from a
226 randomly selected subset of simulations. We also computed the prior-posterior distribution
227 overlap, and used overlap $\geq 35\%$ as an indicator of weak identifiability (Gimenez et al. 2009).

228 **2.5. Evaluation of model performance**

229 We summarized the posterior $\hat{\theta}$ for each parameter and each simulation using relative error of
230 the mean posterior ($\frac{\bar{\theta} - \theta}{\theta}$) and relative precision as the coefficient of variation ($CV = \frac{SD(\hat{\theta})}{|\theta|}$),
231 where θ is the true (simulated) parameter value, $\bar{\theta}$ the mean of the posterior, and $SD(\hat{\theta})$ the
232 standard deviation of the posterior. We quantified accuracy of the estimators across many

233 simulations using relative bias of the average of the mean posteriors and the coverage accuracy
234 of 95% credible intervals. The latter was determined as the rate of correct inferences, which is
235 the probability that the 95% credible interval of the parameter estimate contains the true value
236 of that parameter. In addition, we used results from a few of the simulated scenarios with a
237 deterministic covariate (as described above) to quantify the ability of the model to predict the
238 spatial pattern in mortality (see further details in Appendix 3).

239 **3. Results**

240 **3.1. Spatial heterogeneity using a deterministic spatial covariate**

241 All model parameters converged and were identifiable for scenarios with the spatial gradient
242 covariate and large population size ($N_1=250$) (Appendix 1, table 2). The combination of the
243 spatially random covariate and low population size ($N_1=120$) led to the poorest parameter
244 convergence and identifiability (28-57%; Appendix 1, table 2).

245 All model parameters had low bias ($\leq 6\%$ relative bias) for all scenarios with large population
246 size and a gradient covariate (Figure 1, Appendix 1, table 3-6). Accurate estimates were more
247 challenging to obtain for scenarios with low population size and a spatially-random covariate
248 (Appendix 1, table 3-6). For example, relative bias of β_w reached 44% for the scenario with
249 low population size ($N_1=120$), a spatially-random covariate and $\beta_h = -1$; $\beta_w = 1$; (Appendix 1,
250 table 5). For the same scenario, but with a larger population size ($N_1=250$), relative bias for β_w
251 was substantially lower (RB= 2%). Coverage remained relatively high (>90%) for all scenarios
252 (Appendix 1, table 3-6). Across all scenarios, the effect of spatial covariates on mortality cause
253 with no dead recovery information (β_w) was more challenging to estimate, with lower precision
254 (approx. 2 times larger CV) than (β_h) (Fig 1.C, Appendix 1, table 3-6).

255 **3.2. Density-dependent survival**

256 Across the 100 replicated datasets, 86 models reached convergence and showed no
257 identifiability issues (Appendix 2, table 13). We detected a 13% positive relative bias in ϕ_0
258 and β_d but coverage was >92% for all parameters (Figure 2 A, table 14).

259 **3.3. Ignoring spatial heterogeneity in mortality**

260 Apart from not being able to provide robust inferences on mortality, not accounting for spatial
261 heterogeneity in mortality can lead to bias in abundance estimates. The relative bias (up to 10%
262 for our simulated scenarios) and lower coverage were especially pronounced for scenarios with
263 a spatial gradient in mortality (Figure 2 B, Appendix 1, table 7-11).

264 **4. Discussion**

265 We described and tested an OPSCR model that explicitly models and estimates spatial variation
266 in survival. The model is versatile enough to allow survival to be modelled as a function of any
267 spatial covariate, including local density estimated within the same model. Survival is modeled
268 as a function of the location of the AC of both detected and undetected individuals which allows
269 population-level and spatially-explicit inferences. Using simulations, we show that the model
270 produces sound inferences on the role of spatial covariates and density dependence in
271 explaining spatial variation in survival. In addition, the model allows for integrating spatial
272 dead recoveries (Dupont et al. 2021) and estimates multiple competing sources of mortality
273 with potentially different spatial determinants. The model overcomes a challenge faced by
274 other methods, namely to obtain population-level assessment of spatial determinants of
275 variation in survival (Royle et al. 2018).

276 Despite the recognized potential of OPSCR models to estimate spatial variation in vital rates
277 (Royle et al. 2014), few studies have attempted to use them for this purpose (Chandler et al.
278 2018)., Furthermore, we are not aware of any study that has evaluated the performance of
279 OPSCR models that include spatially-explicit vital rates. Our simulations show that sound

280 inferences in the spatial variation of cause-specific mortality can be obtained when survival is
281 modelled as a function of a deterministic spatial covariate, even with relatively small OPSCR
282 datasets (5 occasions, ~50 individuals detected per occasion). OPSCR model performance
283 (convergence, precision) was significantly higher for simulations with spatially autocorrelated
284 survival (gradient) compared with spatially-random survival (Appendix 1, table 3-6).

285 One of the main advantages of SCR models is that they can estimate spatial variation in density.
286 We showed that OPSCR models can simultaneously estimate local density and its effect on
287 survival. This has the advantage that the uncertainty in both the number of individuals alive
288 and their location is propagated when estimating the spatially link with survival. However,
289 density being both a latent variable and a covariate used to explain variation in survival, it is
290 computationally challenging (i.e., due to the increased number of model dependencies) to fit
291 density dependent-survival OPSCR models. In Appendix 5, we showcase how centering the
292 density covariate (d) can improve the mixing of MCMC chains of different model parameters.

293 Future research should focus on building and testing models that estimate spatial variation in
294 other demographic parameters, i.e., recruitment, emigration and immigration, as it is an
295 essential step to fully understand the mechanisms driving spatial heterogeneity in density and
296 therefore population dynamics (Chandler et al. 2018). Meanwhile, it is possible to use a spatial
297 covariate on the intensity parameter in the spatial point process submodel for the ACs to help
298 account for spatial heterogeneity in recruitment (Zhang et al. 2020).

299 The OPSCR model described here can identify spatial variation in mortality and its
300 determinants from spatial capture-recapture data, which are collected by many monitoring
301 programs (Royle et al. 2018). Climatic conditions, resources, human activities, hunting,
302 predation risk, and intra- and inter- specific competition are some examples of the pressures
303 that are inherently spatial and known to impact survival and that could be studied with the

304 model presented here. We also show that estimates of density obtained from OPSCR models
305 can be biased (reached up to 10% in the scenarios tested) when spatial variation in survival is
306 not accounted for. The Bayesian model written in NIMBLE and the set of features available in
307 the nimbleSCR package will allow users to fit efficient and flexible OPSCR models. This
308 development represents an essential step towards a fully spatially-explicit OPSCR model that
309 can disentangle the role of spatial drivers on population dynamics (Chandler et al. 2018).

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315 **Author contribution**

316 CM, RB, PD and SD conceived and designed the study. CM implemented the analysis and
317 wrote the manuscript with contributions from all authors. All authors gave final approval for
318 publication.

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396 **Figure caption**

397 **Figure 1: A)** Maps depicting the simulated spatial variation in cause-specific mortality (h and
398 w) within the spatial domain (S) for the scenario with a gradient covariate $\beta_h=-1$ and $\beta_w=1$
399 (Table 5 Appendix 1). White points represent detectors. Violin plots show the distribution of
400 the relative error (left; points: relative bias) and coefficient of variation (CV; right, points:
401 median) in **B)** occasion-specific abundances, and **C)** cause-specific mortality parameters
402 obtained after fitting the OPSCR model accounting for spatial variation in mortality to 100
403 replicated datasets. Results from scenarios with small ($N_l=120$) and large ($N_l=250$) population
404 size are presented.

405 **Figure 2: A)** Violin plots (points: medians = relative bias) representing the distribution of the
406 relative error in occasion-specific abundances (\hat{N}) and parameters controlling for the effect of
407 density in survival (β_ϕ, ϕ_0). Abundance estimates and associated relative error were obtained
408 by fitting an OPSCR model that did not account for spatial variation in survival (“without”)
409 and a model that accounted for density-dependent survival (“with”) to 100 replicated datasets
410 simulated with a negative effect of density on survival $\beta_\phi= -1$. **B)** Violin plots of distribution
411 of the relative error (left; points: medians= relative bias) and coverage (right) of occasion-
412 specific abundances obtained by fitting an OPSCR model that accounted for spatial variation
413 in mortality (“with) and a model did not (“without”). Results are presented for 100 replicated
414 datasets simulated with a spatial gradient in mortality, $\beta_h=-1$, $\beta_w=1$ and $N_l=250$.

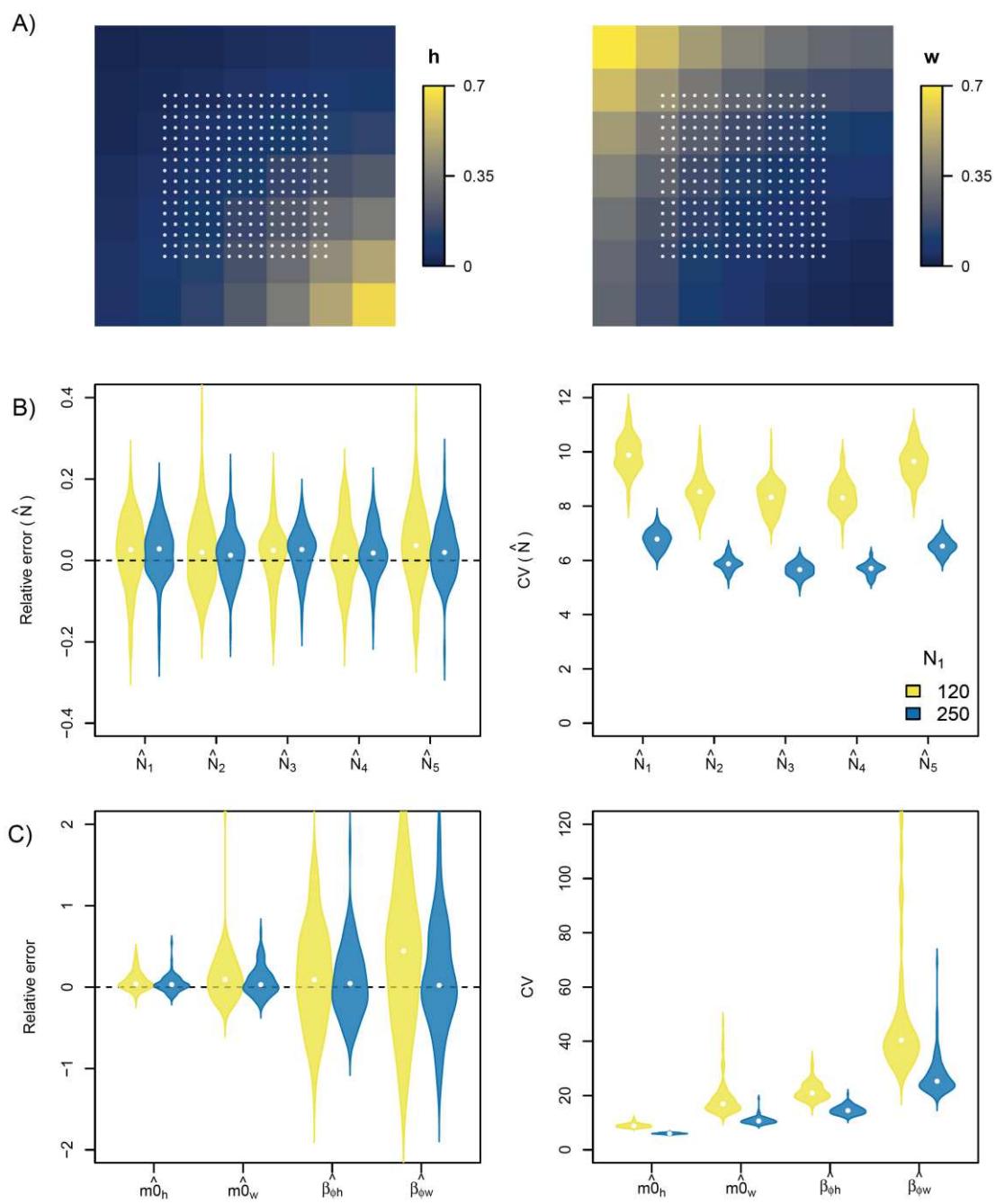
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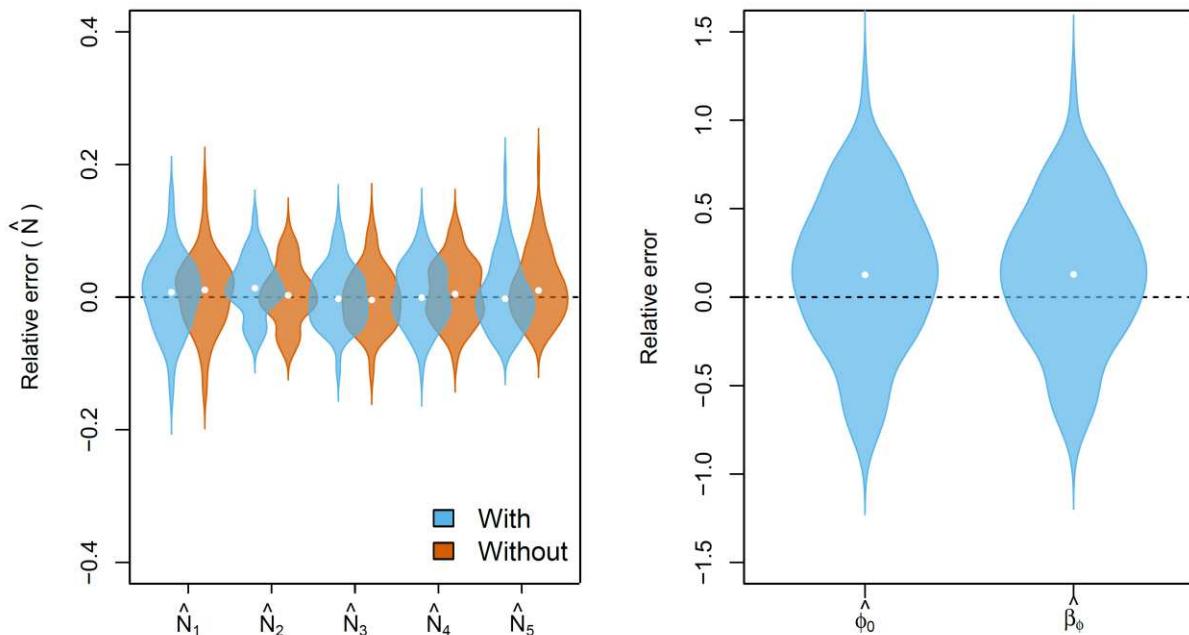
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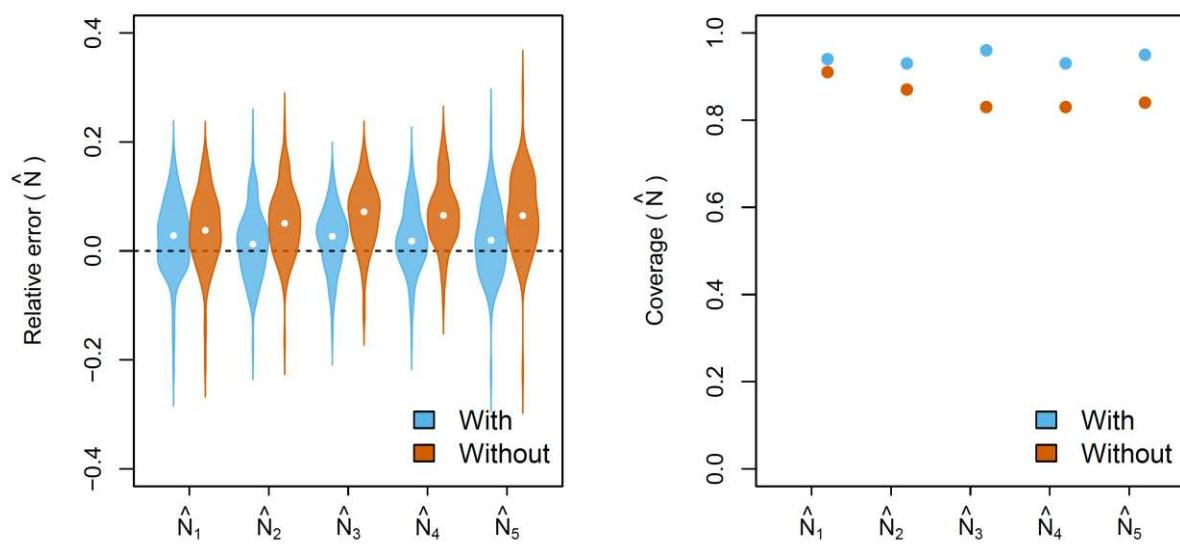
421 **Figure 1**

422 A)



423

424 B)



425

426 **Figure 2**

427