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Methods for Studying Cause-Specific Senescence in the Wild

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33 **Summary**

34 **1.** The founding evolutionary theories of ageing indicate that the force of mortality imposed by
35 environmental factors should influence the strength of natural selection against actuarial senescence and
36 its evolution. To rigorously test this idea, field biologists need methods that yield estimates of age-
37 specific mortality according to cause of death.

38 **2.** Here, we present existing methods commonly applied in studies of human health that could be used to
39 accomplish these goals in studies of wild species for which fate can be determined with certainty. We
40 further present a new application of hidden Markov models for capture-reencounter studies of wild
41 animals that can be used to estimate age-specific trajectories of cause-specific mortality when detection
42 is imperfect.

43 **3.** By applying our new hidden Markov model with the E-SURGE and MARK softwares to capture-
44 reencounter datasets for long-lived species, we demonstrate that senescence can be severe for natural
45 causes of mortality in the wild, while being largely nonexistent for anthropogenic causes.

46 **4.** Moreover, we show that conflation of mortality causes in commonly used survival analyses can
47 induce an underestimation of the intensity of senescence and overestimation of mortality for pre-
48 senescent adults. These biases have important implications for both age-structured population modelling
49 used to guide conservation and comparative analyses of senescence across species. Similar to frailty,
50 individual differences in causes of death can generate individual heterogeneity that needs to be
51 accounted for when estimating age-specific mortality patterns.

52 **5.** The proposed hidden Markov method and other competing risk estimators can nevertheless be used to
53 formally account for these confounding effects, and we additionally discuss how our new method can be
54 used to gain insight into the mechanisms that drive variation in ageing across the tree of life.

55 **Key-words:** ageing, capture-reencounter, competing risk analysis, frailty, harvest, heterogeneity, hidden
56 Markov model, predation.

57

58 **Introduction**

59 Assessing the sources of mortality over life and how they shape age-specific mortality trajectories is of
60 paramount importance in ecology, evolution and public health. Biostatisticians have long known that
61 much can be learned by decomposing mortality into its respective causes (Chiang 1968). For example, if
62 an individual smokes, a ‘competing risk analysis’ can help identify how this affects the chance of dying
63 from lung cancer relative to heart disease or other causes (Berkson & Elveback 1960; Chiang 1991).
64 Competing risk analyses can additionally be used to help identify gene loci and gene expressions that are
65 involved in the phenotypic expression of early- and late-life chances of dying from various causes (e.g.
66 Slagboom *et al.* 2000). Cause of death data can thus shed light on the underlying life choices,
67 environmental factors, and genetic mechanisms that shape mortality risks over the life course compared
68 to a common survival analysis that disregards diverse causes of death (Finch 1990). For these reasons,
69 great effort has been devoted to studying cause-specific mortality from the youngest to the oldest age
70 classes in human populations (e.g. Horiuchi & Wilmoth 1997; Horiuchi *et al.* 2003).

71 In wild vertebrates, there is also a long history of studying cause-specific mortality, but with a
72 specific focus on pre-defined single (e.g. Singer *et al.* 1997 on juveniles; Brodie *et al.* 2013 on adults) or
73 multiple (e.g. Dumke & Pils 1973; Nelson & Mech 1986) age classes. Determining the cause-specific
74 drivers of mortality in both juvenile and adult age classes can indeed help focus management efforts
75 aimed at conserving populations (Forrester & Wittmer 2013). Apart from the study of simple age
76 classes, however, empirical studies of age-specific causes of mortality and their consequences on the
77 shape of mortality trajectories in wild organisms are lacking.

78 This is surprising because the original theories on the evolution of senescence (here, the actuarial
79 definition of an increase in mortality and decrease in survival with age) hinge upon concepts of cause-
80 specific mortality (see Box 1). Methods for estimating competing risks of mortality in the wild, and how
81 they change with age, could help evolutionary ecologists identify the mechanisms underlying patterns of

senescence across species and environmental conditions (Jones *et al.* 2008, 2014; Baudisch *et al.* 2013; Nussey *et al.* 2013).

Studying senescence in the wild is further complicated by the fact that individual heterogeneity can have important effects on the estimation of age-specific patterns of mortality and survival (Cam *et al.* 2002; Nussey *et al.* 2008; Péron *et al.* 2010; Aubry *et al.* 2011). In most populations, ‘frail’ individuals readily die, leaving only the more ‘robust’ individuals in a study sample at advanced ages (*sensu* Vaupel *et al.* 1979). When not accounted for, intra-generational viability selection (Endler 1986) among heterogeneous individuals can bias marginal estimates of age-specific mortality (Vaupel & Yashin 1985). At the end of life, cause of death is a component of the phenotype and can therefore be thought of as a type of individual heterogeneity. Although different than individual heterogeneity at the beginning of life, individual variation in fates could also affect the estimation of age-specific mortality but has not previously been considered to our knowledge. We fill this gap by reviewing contemporary competing-risk analyses that can be used to examine age-specific variation within each risk, and additionally provide an original method for estimating age-specific mortality trajectories while accounting for individual differences in mortality causes when detection is imperfect.

We focus on capture-reencounter hidden Markov models that account for imperfect detection and can even accommodate fates that are not observable (i.e. hidden states; Pradel 2005; Gimenez *et al.* 2012), both of which are common to studies of wild organisms (Williams *et al.* 2002). Applying this method to example datasets, we provide a first demonstration that decomposing age-specific mortality into its respective causes can have notable effects on the estimated rate of senescence in the wild. In addition, age profiles of cause-specific mortality provide more explicit targets for associating physiological condition, gene loci, and quantitative gene expressions with senescence in the competing risks they affect most (Nussey *et al.* 2008).

107 **Estimating cause-specific mortality across ages in the wild**

108 *Perfect detection*

109 In plants, sessile organisms, and captive or semi-captive animal populations, cause-of-death data for
110 studied individuals can be collected along with the standard actuarial life table (e.g. Mumby *et al.* 2013).
111 In addition, cause of mortality data is often collected in radio-telemetry and GPS-transponder studies of
112 free-ranging animals (Heisey & Fuller 1985; Tomkiewicz *et al.* 2010). In the past, cost and logistics
113 associated with these technologies have prohibited the large sample sizes needed to examine senescence
114 at advanced ages, but that may change as GPS and associated battery-life technology develop
115 (Tomkiewicz *et al.* 2010).

116 In some cases, the immediate cause of death might be obvious (e.g. vehicle or wind turbine collision,
117 hunting or fishing recoveries of marked individuals, bark beetle kill in trees, etc.), but necropsies and
118 other types of expert assessments on the less obvious causes of death could provide more detailed
119 insight into the array of factors that kill individuals in the wild (e.g. Mar *et al.* 2012). Often times,
120 however, such detail is out of reach and cause of death will have to be collapsed into broader categories.
121 Grouped causes of death (e.g. predation, disease) can nevertheless provide deeper insight into mortality
122 dynamics than an assessment of overall mortality (as demonstrated by the examples below).

123 In studies where observers can at least ascertain whether an individual is alive or not at each census
124 period (i.e. known-fate data), age-specific cause of death data can be analyzed with existing competing-
125 risk statistical models. The general approach is straightforward; instead of specifying a standardized
126 calendar date as the unit of time at which an individual enters and exits the study sample, one must
127 simply substitute ‘age’ as the unit of time (e.g. Aubry *et al.* 2011). Even the popular Cox proportional
128 hazard model (Cox 1972) can be extended to estimate age- and cause-specific mortality (Heisey &
129 Patterson 2006). To address questions related to the rate of senescence, one might prefer to fit
130 parametric relationships between age and mortality rate according to cause of death (e.g. accelerated
131 failure time models; Wei 1992), whereas for questions related to the shape of mortality over life, one

132 might prefer to fit semi-parametric or non-parametric models (e.g. Kaplan-Meier models [1958]). The
133 extensive repertoire of modelling possibilities that are available in traditional survival analysis can for
134 the most part be extended to the study of competing risks (Kleinbaum & Klein 2012).

135 There are nevertheless caveats associated with analyzing cause of death data (see Heisey & Patterson
136 2006). Similar to any statistical analysis with dichotomous variables, specifying too many causes of
137 mortality may limit degrees of freedom, diminishing precision of parameter estimates. In addition,
138 staggered entry of individuals into the study sample is common in studies of wild organisms (i.e. left
139 truncation: the addition of individuals to the study sample after the beginning of the study). However,
140 only a few competing risk methods properly account for staggered entry that affects the at-risk sample in
141 ways that are not due to death or right censoring (Lunn & McNeil 1995; de Wreede *et al.* 2011; Geskus
142 2011). These methods offer a fruitful way forward for examining age-specific competing risks in data
143 that are often augmented with staggered entries to maintain the sample sizes needed to address questions
144 concerning senescence.

145 *The challenge of imperfect detection and unobservable fates in the wild*

146 In non-captive animal populations, live individuals might not be detected during a survey for an array of
147 reasons. In such cases, the standard life table and aforementioned competing-risk methods yield
148 measures of age-specific ‘return rates’ to the observer, as opposed to the desired quantities of survival
149 and mortality. This is problematic because a return rate is a function of three different events: survival,
150 fidelity to the study area (i.e. $1 - \text{emigration}$), and the probability of detection given an individual is
151 alive and on the study area (Martin *et al.* 1995). Failure to account for imperfect detection can thus lead
152 to flawed inference in both ecological (Nichols 1992) and evolutionary studies (Gimenez *et al.* 2008).

153 Fortunately, capture-reencounter (CR) methods can decouple the probabilities that comprise a return
154 rate (Burnham 1993), and are often used to robustly estimate survival in wild populations (Williams *et*
155 *al.* 2002). Because of their properties, CR methods are now commonly used to study senescence in the
156 wild (e.g. Gaillard *et al.* 2004; Péron *et al.* 2010). The nuisance of imperfect detection nevertheless

157 presents a challenge to estimating cause-specific mortality in wild animal populations. Death is never
158 observed for most individuals in a wild animal population, and determination of certain causes of death
159 might be completely ‘unobservable’.

160 All is not lost, however, because modern multistate CR methods make it possible to estimate cause-
161 specific probabilities of mortality when detection is imperfect. The original multistate CR estimators for
162 cause-specific mortality were restricted to situations where each cause of mortality was at least partially
163 observable (Lebreton *et al.* 1999; Schaub & Lebreton 2004; Schaub & Pradel 2004). This approach has
164 since been extended to allow for an additional ‘unobservable’ cause of mortality using a hidden Markov
165 specification of the multistate CR model (hereafter CR HMM; Pradel 2005; Servanty *et al.* 2010;
166 Gimenez *et al.* 2012). The CR HMM for cause-specific mortality may be more generally applicable to
167 the study of marked animals because it does not exclude causes of death that are completely hidden from
168 the observer (e.g. consumption by certain predators), but does nevertheless require the combination of
169 live recaptures and marked-individual recoveries from at least one source of mortality. In practice, the
170 combination of live recapture and dead recovery data are typically represented using a capture history
171 for each individual where, e.g., 0AA0C would represent an individual that was captured and released
172 alive (A) on the second capture occasion, recaptured alive (A) on the third occasion, and then not
173 observed until it was recovered between occasions four and five when it died of cause C (note that letter
174 identifiers could be replaced with numbers). The zeros between non-zeros in a capture history provide
175 critical information about imperfect detection.

176 The individual-based encounter histories are combined into a population-level dataset to estimate the
177 probability of individual i dying from cause k between discrete time step t and $t+1$ ($\mu_{i,t}^k$) using a CR
178 HMM like that shown in Figure 2. By defining dead states as absorbing states, the probabilities of
179 transitioning from a live state (A in Fig. 2) to a dead state (B, C, and O in Fig. 2) naturally become
180 cause-specific mortality probabilities (Gauthier & Lebreton 2008). Of key importance, the transition

181 probabilities are estimated conditionally on state-specific probabilities of detecting each individual i in
 182 state k at time step t ($p_{i,t}^k$) that are simultaneously solved for using either a maximum-likelihood or
 183 Bayesian approach (Lebreton *et al.* 2009). According to the example (Fig. 2), $p_{i,t}^A$ would represent the
 184 probability of recapturing a live individual i at time t . As long as dead recoveries can be attained at a
 185 spatial scale much larger than the study area, fidelity can be subsumed within $p_{i,t}^A$ like in our example
 186 (Schaub & Lebreton 2004), or fidelity can be separately estimated (Burnham 1993). For the dead states
 187 $p_{i,t}^B$ is the probability that an individual i that died of cause B between $t-1$ and t was ‘recovered dead and
 188 reported’ to the observers at time t , and $p_{i,t}^C$ the same detection probability for an individual that died of
 189 cause C. The detection probability must be fixed to 0 for other sources of mortality that are not
 190 observable (state O in Fig. 2). It is nevertheless possible to identify the probability of dying from
 191 collectively unobservable causes ($\mu_{i,t}^O$ in Fig. 2) by borrowing information from the joint live recaptures
 192 and dead recoveries (Servanty *et al.* 2010).

193 With regard to cause-specific mortality, multistate CR models have been used to compare and
 194 identify problematic sources of mortality in declining or managed populations (Schaub & Pradel 2004;
 195 Bischof *et al.* 2009), estimate the strength of natural selection on hunting mortality relative to non-
 196 hunting mortality in harvested populations (Gamelon *et al.* 2011), and to estimate the degree of
 197 compensation or additivity between sources of mortality (Schaub & Lebreton 2004; Servanty *et al.*
 198 2010; Koons *et al.* 2014). To our knowledge, however, complete age trajectories of cause-specific
 199 mortality have never been estimated appropriately while accounting for imperfect detection. When
 200 trying to focus on senescence in natural causes of mortality, past studies of ageing in the wild have
 201 typically right-censored individuals once they were known to have died from anthropogenic causes.
 202 Because fates are not known for all individuals in a CR study, this form of non-random censoring
 203 introduces a source of bias. In the examples below, we use long-term studies of two long-lived

204 vertebrates to illustrate how to overcome these important gaps in our understanding of ageing in the
205 wild. To help others use the CR HMM method for their own, potentially more illuminating studies and
206 questions pertaining to senescence and cause-specific mortality, we provide annotated code for
207 implementing the examples using the two leading CR softwares: E-SURGE and program MARK (see
208 App. 1).

209 **Examples demonstrating the use of CR HMM for estimating age trajectories of cause-specific** 210 **mortality**

211 *Age-specific trajectories of mortality causes in the lesser snow goose*

212 A marked population of lesser snow geese (*Chen caerulescens caerulescens*) has been studied near La
213 Pérouse Bay, Manitoba, Canada since 1969 (58°44' N, 94°28' W; Cooke *et al.* 1995), where recaptures
214 of previously marked individuals are recorded every year during banding drives. In parallel, public
215 hunters across North America submit records of harvested birds with bands to the USGS Bird Banding
216 Laboratory. To estimate age-specific mortality according to cause, we focused on a sample of known-
217 age females banded and released as goslings ($n = 45,914$) with subsequent live recaptures ($n = 1,976$)
218 and hunter recoveries ($n = 5,163$) between 1969 and 2011. Because the human hunter is the predominant
219 predator of adult lesser snow geese (Koons *et al.* 2014), the age-specific level of hunting mortality may
220 modify the strength of natural selection against senescence in both direct and indirect ways (Box 1, Fig.
221 1). Thus, we developed a CR HMM with one alive state (A) and two dead states: a partially observable
222 state of 'died from hunting' (H; i.e. legally hunted) and an unobservable state of 'died from non-hunting'
223 (NH, which includes any unobservable crippling loss). Observed capture histories never contain explicit
224 information about individuals in unobserved states like NH. To develop a CR HMM, one must therefore
225 define the unobserved states of interest and fix the respective detection probabilities to 0 (Pradel 2005).
226 In appendix 1 we demonstrate how to do this for the snow goose example using the RMark package for
227 R (Laake & Rexstad 2012), describe our analysis in more detail, and provide annotated code for the

228 modelling steps. Here, we focus on demonstrating the approach of using a CR HMM to study age-
229 specific mortality according to cause in the wild.

230 Drawing from the long history of research on snow goose survival and previous findings for the
231 same or similar dataset (Francis *et al.* 1992a; Cooch *et al.* 2001; Aubry *et al.* 2013; Koons *et al.* 2014),
232 we developed CR HMMs that account for important sources of age and temporal variation in the
233 detection probabilities, as well as temporal variation in cause-specific mortality probabilities for each of
234 two age classes (hatch-year and after-hatch-year; see App. 1A). To estimate the trajectory of cause-
235 specific mortality at each age x , μ_x^k , we considered the Gompertz, Weibull, and logit-linear functions
236 (Gaillard *et al.* 2004), with alternative ages of onset for senescence (ages 4, 6, 8, 10, 12, 14, or 16; non-
237 statistically significant results from Francis *et al.* 1992b indicate that if there is senescence, the onset
238 may be delayed until ~ age 10). At each modelling step, we used Akaike's Information Criterion
239 adjusted for sample size (AIC_c; Akaike 1973; Burnham & Anderson 2002) to identify the model
240 structure most supported by the data.

241 While controlling for temporal variation, we found strong support for Gompertz senescence in non-
242 hunting mortality past age 14 (loglog link: $\hat{\beta} = 0.119$, 95% CI: 0.083 – 0.154; Fig. 3; $\Delta\text{AIC}_c = 45.2$ for
243 simple age-class effects). This model was more supported than other ages of onset in non-hunting
244 mortality senescence ($\Delta\text{AIC}_c > 0.8$), as well as the Weibull ($\Delta\text{AIC}_c = 0.1$) and logit-linear ageing
245 functions ($\Delta\text{AIC}_c = 11.0$). We detected marginal support for reduced hunting mortality with age (1.1 unit
246 improvement in AIC_c), but the effect was biologically minor and imprecisely estimated ($\hat{\beta} = -0.006$,
247 95% CI: -0.014 – 0.001).

248 Thus, it seems that actuarial senescence is delayed and restricted to non-hunting sources of mortality
249 in lesser snow geese. Studies of senescence in other long-lived avian species have similarly found
250 delayed onsets of aging that begin well past the age of primiparity (which ranges from 2-4 in snow
251 geese; Juillet *et al.* 2012), but also less severe senescence in species that are longer-lived than snow

geese on average (e.g. Pardo *et al.* 2013; Jones *et al.* 2014). Interestingly, previous studies of age-specific demography in snow geese had detected senescence in reproductive success (Rockwell *et al.* 1993) but not in survival (Francis *et al.* 1992b). This may have been due to the lack of a large enough sample of old-age individuals at the time of analysis, conflation of individuals that died from senescent non-hunting sources of mortality with those that died from non-senescent hunting mortality, or both.

Indeed, a multistate CR model without specification of mortality cause (i.e. just live and dead states) offered a relatively poor fit to the same dataset. Although the onset of senescence at age 14 was once again more supported than onset at other ages, the model was 1180 AIC_c units worse than a cause-specific mortality model with two age classes and 1210 AIC_c units worse than the top cause-specific mortality model with senescence past age 14 in non-hunting mortality. In addition, not accounting for cause of mortality led to an underestimation of overall juvenile mortality, overestimation of mortality during pre-senescent adult life, and a 44.5% reduction in the estimated rate of senescence ($\hat{\beta} = 0.066$, 95% CI: 0.035 – 0.098; Fig. 3, compare open and closed circles). In essence, conflation of mortality causes led to a flattening of the estimated boat-shaped mortality curve and forced the bottom of the boat to pop up.

Age-specific trajectories of mortality causes in roe deer

Our second example pertains to a roe deer (*Capreolus capreolus*) population in the enclosed forest (13.6 km²) of the Territoire d'Etude et d'Expérimentation of Trois Fontaines, in eastern France (48°43'N, 54°10'W), that has been intensively monitored using CR methods from 1975 to 2013. Each year since 1985, newborn fawns were captured, sexed, marked, and released after handling (Gaillard *et al.* 1998). Here we focus on the 556 known-age females, of which 217 were recaptured at least once and 41 were deadly injured during handling, victim of car collisions, or recovered and reported by hunters (collectively denoted as human-related mortalities). To control population size, some individuals were removed from the forest and released outside the study area (and right-censored from the dataset). There

276 are no predators of adult roe deer at Trois Fontaines, and thus the age-specific level of human-related
277 mortality may modify the strength of natural selection against senescence in both direct and indirect
278 ways (Box 1, Fig. 1).

279 We estimated natural and human-caused mortality using a CR HMM allowing for the joint analysis
280 of live recaptures and dead recoveries of individuals (Schaub & Pradel 2004; Lebreton *et al.* 2009). Four
281 states were used to describe the fates of each individual: two partially observable states, one for
282 individuals that were alive (A) at time t and another for individuals that had just died from human-
283 related causes (H), and two unobservable states, one for individuals that had just died from natural
284 causes (NH) and an absorbing state for the collection of individuals that were already dead (D). Given
285 these state definitions, the human-related mortality probability (μ^H) corresponded to the transition
286 probability from the state A at time t to state H by time $t+1$, and similarly, the natural mortality
287 probability (μ^{NH}) corresponded to the transition probability from state A at time t to state NH by time
288 $t+1$. Because an individual could not return to state A once dead, we fixed these transitions to 0. To
289 ensure that all probabilities were estimated within the interval $[0, 1]$ and summed to 1, we used a
290 generalized (or multinomial) logit link function (e.g. Choquet *et al.* 2009). A live individual could be
291 recaptured with probability $p_{i,t}$, or not recaptured with probability $1 - p_{i,t}$. Because capture effort varied
292 among years and age classes (Gaillard *et al.* 2003; Choquet *et al.* 2011), we included an interactive
293 effect of time-dependence in $p_{i,t}$ for age class 1 relative to individuals older than 1 year of age. An
294 individual that just died from human causes could be recovered and reported with probability $r_{i,t}$, or not
295 recovered and reported with probability $1 - r_{i,t}$. Because tag recovery protocols were constant over the
296 course of the study, we considered $r_{i,t}$ to be constant over time and across age classes.

297 To estimate cause- and age-specific mortality, we allowed natural and/or human-related mortalities
298 to vary linearly on the generalized logit scale from age 1 or 2 onward based on previous studies (e.g.
299 Gaillard *et al.* 2004). When natural mortality was allowed to vary linearly with age on a generalized

logit scale, human-related mortality was either constrained to be constant, allowed to vary among ‘age classes’, or allowed to vary linearly with age on the generalized logit scale (and vice versa when human-related mortality was a generalized logit-linear function of age). We considered and compared the following age class parameterizations: 0-1, > 1, or 0-1, 1-2, > 2. In addition, according to previous research on roe deer survival (Festa-Bianchet *et al.* 2003), we also tested models that allowed cause-specific mortality probabilities to vary among 5 age classes: fawn summer mortality up to age 1, age 1 to 2, ages 2 to 8, an early senescent category for individuals between age 8 and 13, and a senescent category for older individuals. We then used QAIC to compare the various competing models using the E-SURGE software (Choquet *et al.* 2009). In appendix 1B, we show how to implement CR HMM models for the roe deer example.

The best model indicated senescence in natural mortality from age 2 onwards (generalized logit link $\hat{\beta} = 2.239$, 95% CI: 0.942 – 3.535; Fig. 4). The best model also retained a constant human-related mortality probability from age 1 onwards but a higher mortality in the first year of life after birth (human-related mortality probability $_{0-1} = 0.132$, 95% CI: 0.053 – 0.294; human-related mortality probability $_{1+} = 0.057$, 95% CI: 0.027 – 0.115; Fig. 4). This model performed better than other ages of onset for senescence in natural mortality ($\Delta\text{QAIC} > 2$) and other parameterizations of age effects for human-related mortality ($\Delta\text{QAIC} > 1.5$).

To examine the effect of conflating the causes of mortality on the estimated age-specific mortality trajectory, we developed a multistate CR model without specifying mortality causes similar to that developed for the snow goose example above. For this analysis, three states were considered in E-SURGE: one for individuals that were alive (A) at time t , another for individuals that had newly died (from human-related or natural causes) (ND), and an absorbing state for the collection of individuals that were already dead (D). Given these state definitions, the overall mortality probability corresponded to the transition probability from state A at time t to state ND by time $t+1$. We then cast model structures

324 similar to those described above for the CR HMMs, and although senescence was still detected, the
325 estimated rate of senescence was 48.4% lower than in the CR HMM that accounted for mortality cause
326 ($\hat{\beta} = 1.155$, 95% CI: 0.784 – 1.526, Fig. 4 open circles). Moreover, this model was 7.64 QAIC units
327 worse than the top cause-specific mortality model. Similar to the snow geese, failure to account for the
328 cause of mortality led to an overestimation of mortality in roe deer from age 4 to 10, and
329 underestimation from age 11 to 17 (Fig. 4, compare open and closed circles).

330 **Discussion**

331 Nature provides a vast array of ecological conditions that provide a powerful stage for testing
332 evolutionary theory (*sensu* Hutchinson 1965). This is especially relevant for studies of senescence
333 because the environmental factors that shape the onset and magnitude of senescence in the wild are
334 poorly understood. Using the comparative method, much has recently been learned about the great
335 variety of age-specific mortality trajectories across the tree of life (Jones *et al.* 2008, 2014; Baudisch *et*
336 *al.* 2013; Nussey *et al.* 2013). Here, we presented old and new methods that can improve the estimation
337 of senescence by decomposing age-specific mortality into proximate causes. When applied to specific
338 questions and mechanisms, the presented methods could even be used to gain a deeper understanding of
339 senescence in the wild (or lack thereof).

340 Given the impact that exposure to additional mortality can have on selection pressures affecting
341 senescence (Box 1, Fig. 1), both known-fate and CR HMM methods should be used to compare
342 trajectories of age-specific mortality according to cause of death across populations and species where
343 data are available or can be collected. Moreover, these methods can be used to refine insight into
344 findings that indicate old individuals in the wild are sometimes more susceptible to hunting or predation
345 because of interactions between physiological and ecological processes (e.g. Garrott *et al.* 2002; Carlson
346 *et al.* 2007), pressures of trophy hunting (Coltman *et al.* 2003), or because of social organization that
347 exposes the eldest individuals first (Festa-Bianchet *et al.* 2006). In the case of the human hunter, it has

348 been found in some systems that the ‘human predator’ may simply consume prey in proportion to their
349 occurrence (e.g. abundant prime-aged adult elk; Wright *et al.* 2006). In our examples, we found that the
350 chance of dying from human-related causes was largely age-independent after maturity in both female
351 snow geese and roe deer. A high rate of hunting mortality can nevertheless select for increased early-life
352 allocation to reproduction (Gamelon *et al.* 2011), and future studies should examine how this may in
353 turn affect the rate of actuarial senescence (Fig. 1) as well as senescence in reproductive success
354 (Rockwell *et al.* 1993).

355 Our examples also indicate that some sources of mortality may senesce at a rapid pace while others
356 may not senesce at all. By decoupling human-related from natural causes of mortality with CR HMMs,
357 we were able to estimate the rate of senescence in both natural and overall mortality in snow geese and
358 roe deer (Figs. 3 & 4). Past CR studies that have attempted to make inference about senescence in
359 natural causes of mortality have typically right-censored individuals once they were known to have died
360 from anthropogenic causes, but because fates are not known for all individuals in a CR study, this non-
361 random censoring introduces a source of bias. Our CR HMM is a type of ‘competing risk analysis’ that
362 allows for appropriate estimation of age- and cause-specific mortality probabilities when detection is
363 imperfect. Going forward, methods like ours should be used to examine age trajectories of mortality
364 among competing risks experienced by wild populations. In addition, we have shown that conflating
365 causes of mortality in a traditional CR analysis can lead to an underestimation of the rate of senescence
366 and overestimation of mortality in pre-senescent adults. Both of these biases have important implications
367 for age-structured modelling used to guide conservation and management. Based on simulation, the
368 underestimation of senescence in a traditional CR analysis is most severe when the age trajectories of
369 underlying competing risks are very different (see Table 1A). Like frailty (Vaupel *et al.* 1979),
370 heterogeneity in the eventual causes of death can thus also affect marginal estimates of age-specific
371 mortality. Whether this is also true for known-fate analyses remains to be explored.

372 Examining age trajectories of cause-specific mortality (e.g. in humans, Horiuchi *et al.* 2003) can also
373 provide more explicit targets for associating environmental conditions (e.g. toxin or pathogen exposure),
374 physiological condition, gene loci, and quantitative gene expressions with senescence in the competing
375 risks they affect most (Nussey *et al.* 2008). For example, the CR HMM method presented here can
376 readily be used in longitudinal studies of individual life histories (Clutton-Brock & Sheldon 2010) to
377 examine trade-offs between age-specific allocations to reproduction, the cause of mortality these
378 allocations affect most at given points in the life cycle, and the net impact this has on selection against
379 overall actuarial or reproductive senescence. By honing in on the specific causes of mortality that
380 reproductive allocations affect most, the CR HMM method could be used to help clarify the role of
381 pleiotropic gene expressions (Charmantier *et al.* 2006) and environmental conditions (van Noordwijk &
382 de Jong 1986) that shape trade-offs in the wild.

383 Similar to event-history analysis (Tuma *et al.* 1979), the CR HMM method could be extended to
384 include individual transitions among live states (e.g. epidemiological or morbidity states; Choquet *et al.*
385 2013) over the life course to determine how this affects cause-specific chances of dying at a given age.
386 Such developments would offer an especially promising avenue to gain deeper insight into the
387 mechanistic drivers of ageing for species where cause of death could be categorized according to
388 disease, predation, hunger, and toxicity exposure for example. Another useful extension of our CR
389 HMM method would be to couple it with recently developed capture-reencounter methods for
390 estimating age-specific survival from data collected on unknown-age individuals (Colchero *et al.* 2012;
391 Matechou *et al.* 2013). The rich history of research on snow geese and roe deer allowed us to streamline
392 the age structures considered in our examples, however, this will not always be possible. The use of
393 flexible hazard functions or penalized splines that accommodate an array of both early- and late-life
394 mortality trajectories in the CR HMM framework would allow for powerful comparisons of cause-
395 specific mortality trajectories across species (Gimenez *et al.* 2006; Choquet *et al.* 2011).

396 Although the CR HMMs used in our examples were *a priori* identifiable, not all CR HMM
397 parameterizations will be (see Table 1B). As in our examples (App. 1), accounting for temporal
398 variation and other variables can actually help improve parameter identifiability (Schaub & Lebreton
399 2004; Schaub & Pradel 2004). Future studies should conduct thorough analyses of parameter
400 identifiability to determine the types of CR HMMs and link functions that can and cannot be fit to cause-
401 specific mortality data (Gimenez *et al.* 2003; Table 1). In conclusion, the methods presented here
402 provide a baseline for enhancing methodological developments and advancing the analysis of
403 mechanisms that drive the large variation in ageing observed across the tree of life (Jones *et al.* 2014).

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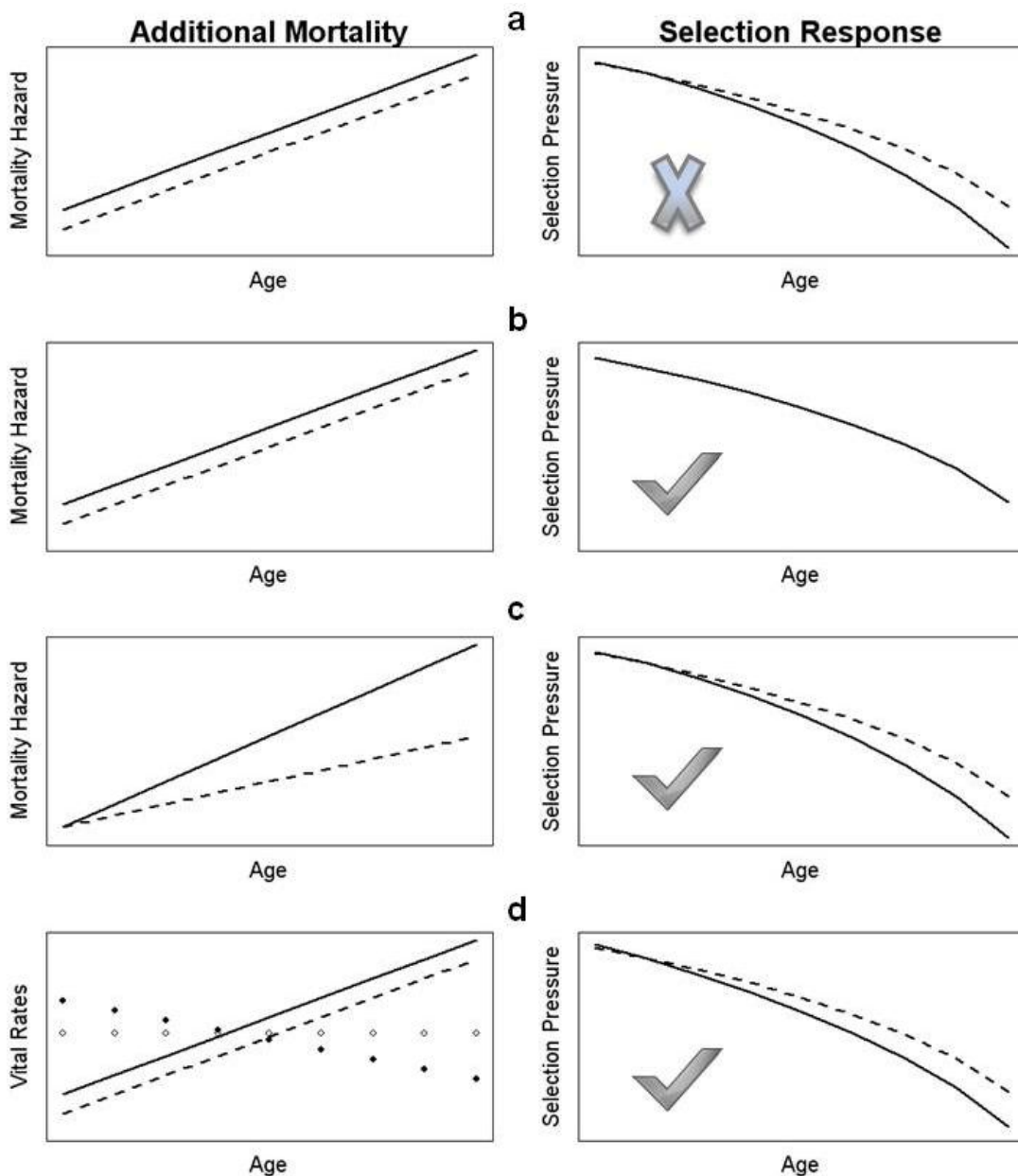
643 Table 1. Simulated logit-linear coefficients for age effects on mortality probabilities in a CR HMM with
644 one alive state, a partially observable state for mortality cause X , an unobservable state for other (O)
645 causes of mortality, and an absorbing state for those already dead ($++ = 0.5$ increase in mortality with
646 age on the real scale, $+$ = 0.25 increase, and 0 no increase; implemented using a continuous age variable
647 on the logit scale with value 0 for age 0, 0.1 for age 1, 0.2 for age 2 and so on). Provided in A) are the
648 estimated coefficients relative to simulated values (95% CI provided within brackets), as well as the
649 estimated age effects for mortality when states X and O are collapsed (conflated) into a single state.
650 Simulations involved the release of newborns in a fashion that maintained 10,000 individuals in the
651 simulated sample at all time steps, $p^X = 0.3$, $p^O = 0.3$, and a common mortality intercept (-1.50).
652 Simulations in B) were similar but included unique parameters for the mortality intercepts of cause X
653 and O (each simulated with a value of -1.50). Differences between simulated and estimated coefficients
654 indicate problems with either partial or complete non-identifiability of model parameters. Interestingly,
655 estimation with a constrained identity link function (not shown) corrected these problems.

Simulated		Estimated				Conflated
β_{age}^X	β_{age}^O	$\hat{\beta}_{age}^X$	$\hat{\beta}_{age}^O$	$\hat{\beta}_{int}^X$	$\hat{\beta}_{int}^O$	$\hat{\beta}_{age}^{conflated}$
A)	++	+	0.50	0.18	-1.48	0.48
			[0.36, 0.65]	[-0.01, 0.36]	[-1.50, -1.47]	[0.40, 0.56]
	+	++	0.17	0.61	-1.50	0.51
			[0.02, 0.31]	[0.46, 0.76]	[-1.52, -1.49)	[0.43, 0.58]
	++	0	0.46	-0.04	-1.50	0.32
			[0.32, 0.59]	[-0.22, 0.14]	[-1.51, -1.48]	[0.25, 0.40]
	0	++	0.07	0.48	-1.50	0.35
			[-0.07, 0.21]	[0.33, 0.62]	[-1.51, -1.48]	[0.28, 0.42]
B)	++	+	0.44	0.34	-2.35	-0.93
			[0.30, 0.58]	[0.15, 0.53]	[-3.80, -0.91]	[-1.49, -0.37]
	+	++	0.13	0.54	-2.70	-0.85
			[0, 0.26]	[0.45, 0.63]	[-3.16, -2.23]	[-0.98, -0.72]
	++	0	0.45	-0.01	-1.57	-1.42
			[0.10, 0.80]	[-1.5, 1.5]	[-5.5, 2.41]	[-5.0, 2.18]
	0	++	0.07	0.47	-1.54	-1.45
			[-0.09, 0.23]	[-0.22, 1.16]	[-5.45, 2.36]	[-5.13, 2.22]

Box 1. The role of cause-specific mortality in evolutionary theories of senescence.

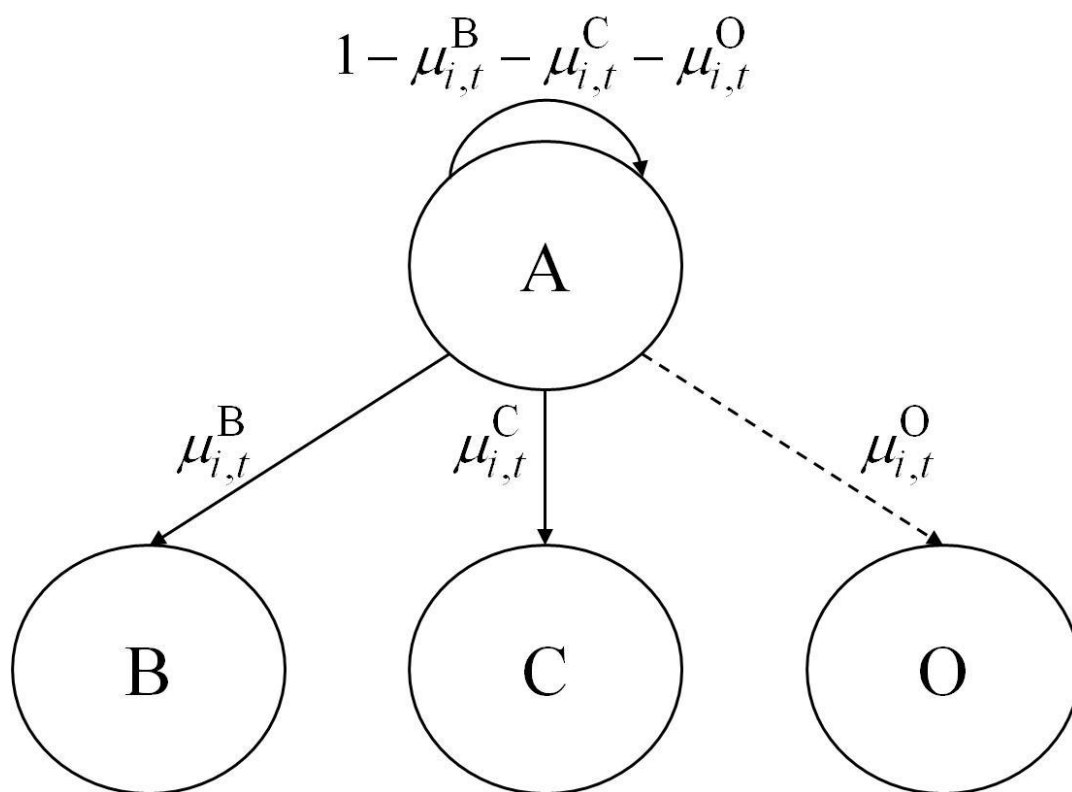
George C. Williams (1957) predicted that according to either Medawar's (1952) Mutation Accumulation (MA) theory or his own Antagonistic Pleiotropy (AP) theory, greater environmentally-driven adult mortality should lead to more rapid senescence. The reasoning being that a reduced chance of making it to old age should either reduce selection on mortality at old age, allowing mutations to accumulate (MA), or reduce the selective advantage of living long relative to investing more in early-life reproduction (AP and the Disposable Soma theory launched by Kirkwood in 1977); thereby allowing more rapid senescence to evolve (Hamilton 1966). Moreover, both Hamilton and Williams' models predicted that senescence should be more apparent in populations exposed to higher levels of mortality (e.g. wild vs. captive populations, harvested vs. protected; see Fig. 1a for a graphical example).

Although not unanimous (Vaupel *et al.* 2004; Ricklefs 2008), a large number of experimental and comparative studies have shown that more rapid senescence tends to occur in populations that are thought to experience higher adult mortality (e.g. Austad 1993; Ricklefs 1998, 2000; Ricklefs & Scheuerlein 2001, 2002; Bryant & Reznick 2004, Reznick *et al.* 2004). However, Abrams (1993) and Caswell (2007) clearly showed that all else being equal, exposure to additional age-independent adult mortality does not affect the strength of selection on age-specific mortality, and therefore cannot affect the evolution of senescence (see Fig 1b and Shokhirev & Johnson (2014) for theoretical evidence that higher predation can lead to both slower or faster life histories depending on the context). That said, if traditionally dichotomized environmental and physiological processes interact (e.g. Williams & Day 2003; Williams *et al.* 2006) to alter age-dependent mortality (e.g. starvation or predation; see Garrott *et al.* 2002; Loe *et al.* 2003; Smith *et al.* 2004; Festa-Bianchet *et al.* 2006; Wright *et al.* 2006; Carlson *et al.* 2007), then the strength of selection acting on age-specific mortality will change (Caswell 2007; see Fig. 1c) and may even allow for the evolution of decreased mortality with age (Vaupel *et al.* 2004; Baudisch 2005). Furthermore, 'all else is rarely equal' in nature; if exposure to additional age-independent adult mortality drives increased allocation to earlier reproduction in order to compensate for increased mortality (as was shown in guppies; Bryant & Reznick 2004; Reznick *et al.* 2004), the strength of selection against actuarial senescence will weaken (Hamilton 1966; see Fig. 1d) and affect the evolutionarily optimal level of allowable senescence (Kirkwood 1977; Wensink *et al.* 2012).



657

658 Box 1, Figure 1. A graphical example of how exposing a population to additional mortality (left panels)
 659 affects the pressure of selection on age-specific mortality hazards (right panels; sensu Hamilton 1966).
 660 The left side of panel 'a' depicts the original prediction that exposing a population already experiencing
 661 senescence (dashed line) to additional age-independent mortality (resulting solid line) will lead to a
 662 decrease in selection pressure on age-specific mortality (right side), which has since been shown to be
 663 false (denoted by the X). Rather, there is no effect on the selection pressure (panel b, lines on the right
 664 side overlap). If a population is instead exposed to additional 'age-dependent' mortality, the selection
 665 pressure on age-specific mortality will indeed decline (panel c), allowing for more rapid senescence to
 666 evolve. Interestingly, if a population that is exposed to additional age-independent mortality responds by
 667 allocating more to early-life reproduction (solid circles) than it had before (open circles) and maintain
 668 the original level of fitness, the selection pressure on age-specific mortality will decline (panel d).



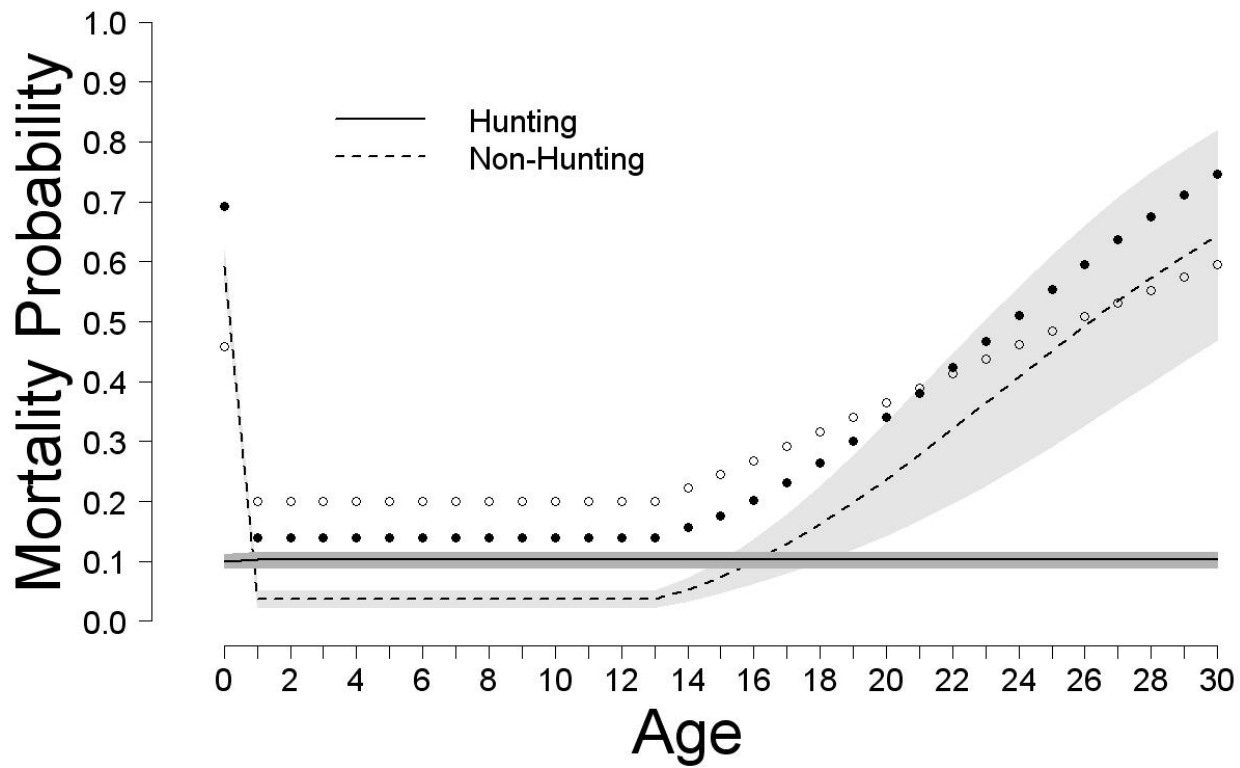
670

671 Figure 2. The observable (solid lines) demographic transitions of remaining alive (A), dying from cause
 672 B or C, and the unobservable (dashed line) transition of dying from other causes (O); where $\mu_{i,t}^k$ denotes
 673 the cause-specific probability of mortality per time step (subscripts are as described in the text).

674

675

676



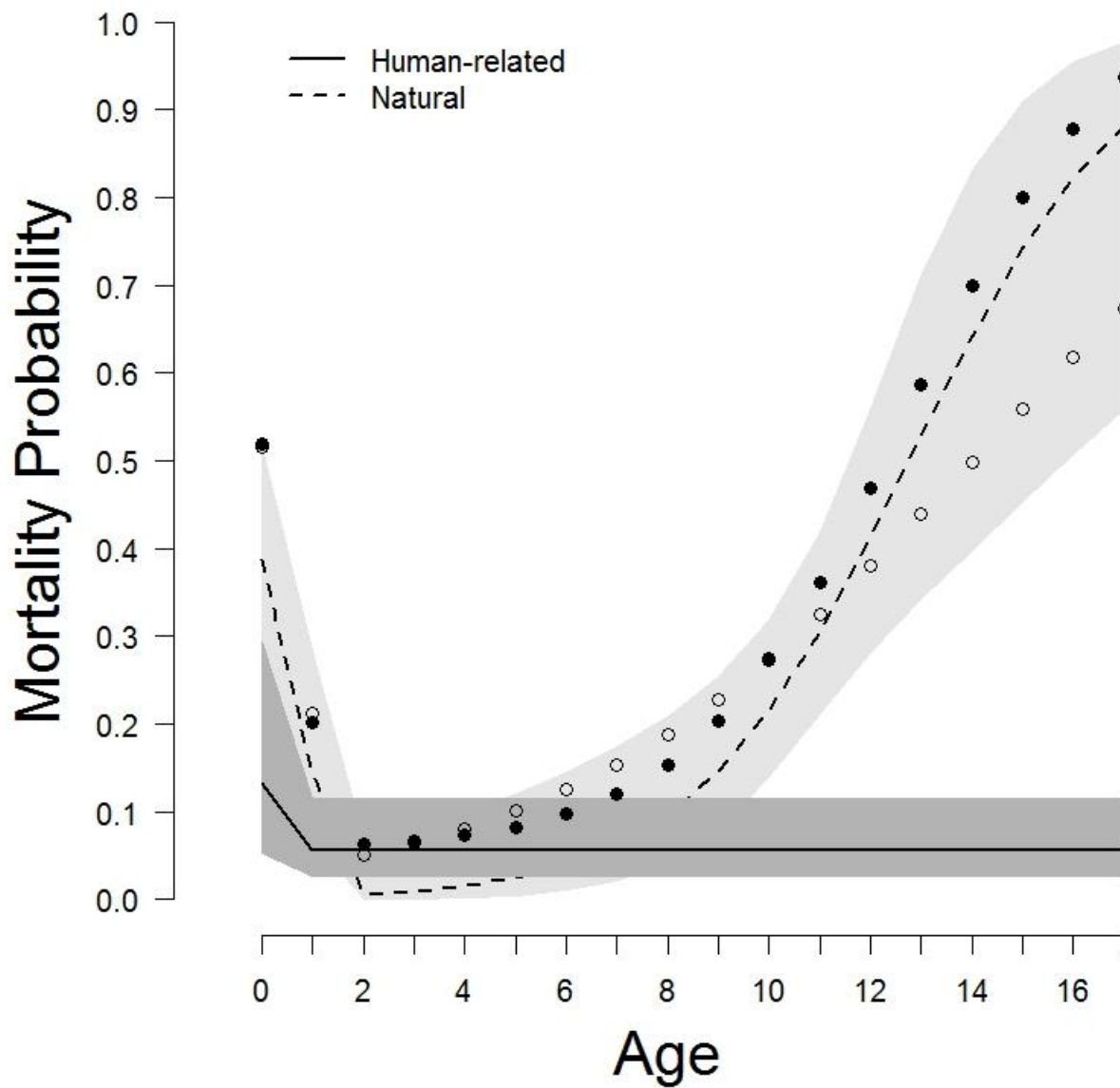
677

678 Figure 3. Trajectories of age-specific mortality probability according to hunting (solid line) and non-
 679 hunting (dashed line) causes for female lesser snow geese at La Pérouse Bay, Canada from 1969 to
 680 2010. Shaded polygons represent 95% confidence bounds. The closed circles represent total mortality
 681 (i.e. the addition of the two causes), and the open circles represent estimates of overall mortality from a
 682 multistate CR analysis where cause was not specified.

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687 Figure 4. Trajectories of age-specific mortality probability for human-related (solid line) and natural
 688 (dashed line) causes in female roe deer at Trois-Fontaines, France, from 1985 to 2013. Shaded polygons
 689 represent 95% confidence bounds. The closed circles represent total mortality (i.e. the addition of the
 690 two causes), and the open circles represent estimates of overall mortality from a multistate CR analysis
 691 where cause was not specified.

692

Appendix 1A. Estimation of age-specific profiles of hunting and non-hunting mortality in lesser snow geese.

For the lesser snow goose analysis we used the RMark package (Laake & Rexstad 2012), which calls program MARK from R (Cooch & White 2012), in part because of complications with the data that sometimes occurred over the many years of study. Specifically, banding did not take place in 1996, 1997, and 2009 (and thus recaptures could not occur), and in 2002 and 2004, the gosling cohort was not marked and released (because of either reproductive failure or the goslings being too small to hold bands at the time of marking; see Aubry *et al.* 2013). Given our desire to fit models that accounted for sources of age-class and time variation in the detection probabilities that were known to be important (see Cooch *et al.* 2001; Koons *et al.* 2014), as well as age- and cause-specific mortality, we needed to fix detection and mortality probabilities to 0 for specific age, time, and state combinations. These complications are relatively straightforward to deal with in a programming language like R (see below). We use Courier font like this to indicate annotated RMark code for analyzing live and dead encounters with a CRHMM (comments in R code follow a # sign).

For RMark to recognize the unobservable NH state, the following two-part trick was needed when importing the data into the RMark environment.

[illegible]

Once the data are read into RMark, the user can create additional explanatory covariates and factors not already attached to the imported data. This can be especially useful when wanting to create dynamic age-class variables or customized temporal covariates (see Laake & Rexstad 2012 for details). As an example, we show how we built a dynamic 5 age-class variable for live recapture probabilities (age 1, 2, 3, 4, and 5+),

```
MS.ddl$p$a1=0
MS.ddl$p$a1[MS.ddl$p$age==1 & MS.ddl$p$stratum==1]=1
MS.ddl$p$a2=0
MS.ddl$p$a2[MS.ddl$p$age==2 & MS.ddl$p$stratum==1]=1
MS.ddl$p$a3=0
MS.ddl$p$a3[MS.ddl$p$age==3 & MS.ddl$p$stratum==1]=1
MS.ddl$p$a4=0
MS.ddl$p$a4[MS.ddl$p$age==4 & MS.ddl$p$stratum==1]=1
MS.ddl$p$a5=0
MS.ddl$p$a5[MS.ddl$p$age>=5 & MS.ddl$p$stratum==1]=1
```

and a variable for activating a parametric aging function at a specified age of onset (here age 14).

```
MS.ddl$Psi$M14Age=0
for (i in 14:30){
  MS.ddl$Psi$M14Age[MS.ddl$Psi$Age==i&MS.ddl$Psi$stratum==1]=i-13
}
MS.ddl$Psi$M14Age[MS.ddl$Psi$Age>=30&MS.ddl$Psi$stratum==1]=30-13
```

As the first important step in analysis, we fixed the survival probabilities for individuals in states A, H, and NH (denoted in the code as strata 1, 2, and 3) to 1, 0, and 0 respectively.

```
S1=as.numeric(row.names(MS.ddl$S[MS.ddl$S$stratum==1,]))
S2=as.numeric(row.names(MS.ddl$S[MS.ddl$S$stratum==2,]))
S3=as.numeric(row.names(MS.ddl$S[MS.ddl$S$stratum==3,]))
S1val=rep(1,length(S1))
S2val=rep(0,length(S2))
S3val=rep(0,length(S3))
```

Next, we fixed the probabilities of transitioning ‘from’ states H and NH to any other state to 0, and therefore the probabilities of ‘remaining’ in states H and NH to 1 (i.e. the dead states were defined as absorbing states). By fixing these parameters, the remaining transition probabilities from state A to H and from state A to NH become the $\mu_{i,t}^k$ (Fig. 1 in main text). In theory, the dead states should be split into ‘newly dead’ and ‘already dead’ states because an individual can only be recovered during the

particular time step that it dies. However, given our parameterizations in RMark, the addition of an ‘already dead’ state is not needed (Gauthier & Lebreton 2008).

```
Psi2=as.numeric(row.names(MS.ddl$Psi[MS.ddl$Psi$stratum==2,]))
Psi3=as.numeric(row.names(MS.ddl$Psi[MS.ddl$Psi$stratum==3,]))
Psi2val=rep(0,length(Psi2))
Psi3val=rep(0,length(Psi3))
```

Shown below are examples of how we fixed a few of the detection probabilities to 0, a key one being that for the NH state (denoted as r3 in the code).

```
pal=as.numeric(row.names(MS.ddl$p[MS.ddl$p$a1==1 &
  MS.ddl$p$stratum==1,]))
p2004.y=as.numeric(row.names(MS.ddl$p[MS.ddl$p$time==2004 &
  MS.ddl$p$stratum==1 & MS.ddl$p$cohort==2002,]))
palval=rep(0,length(pal))
p2004.yval=rep(0,length(p2004.y))

r3=as.numeric(row.names(MS.ddl$p[MS.ddl$p$stratum==3,]))
r3val=rep(0,length(r3))
```

We then show how these specifications (and others not shown here) were implemented into the required or most-supported model structures for survival, transition probabilities (i.e. the cause-specific mortality probabilities), and detection probabilities, with details provided in the commented text.

```
# Survival model specification with S fixed to 1 for state 1 and 0 for
# dead states 2 and 3 as specified above.
Sfix <- list(formula = ~stratum, fixed = list(index = c(S1,S2,S3),
  value = c(S1val,S2val,S3val)))
```

```
# Gompertz age trajectory of adult NH mortality from age 14 onward, and age-
# class differences in H mortality. The variables to2 and to3 denote
# mortality transitions to states H and NH respectively; from2 and from3 are
# specified so that they can be fixed to 0 later in the statement; hy and
# ahy denote age classes for the first year of life and thereafter; the
# M14Age variable specifies the onset of Gompertz senescence as shown above;
# the variables ending in tscr account for temporal variation in the age-
# class and cause-specific mortality probabilities; and the loglog link was
# used to implement Gompertz senescence.
PsigomNH14p <- list(formula = ~ -1 + to2:hy + to2:ahy + to2:hy:hyhtscr
  + to2:ahy:ahyhtscr + to3:hy + to3:ahy + to3:hy:hynhtscr +
  to3:ahy:ahynhtscr + to3:a14p:M14Age + from2 + from3,
  fixed = list(index = c(Psi2,Psi3,Psi1996.g,Psi1997.g,Psi2002.g,
    Psi2004.g,Psi2009.g), value = c(Psi2val,Psi3val,Psi1996.gval,
    Psi1997.gval,Psi2002.gval,Psi2004.gval,Psi2009.gval)), link =
  "loglog")
```

```
# Age-class and time variation in the state-specific detection
# probabilities. The variables str1, str2, and str3 denote strata A,H, and
# NH respectively, with required fixes specified later in the model
# statement; the a1 through a5 age-class variables were defined above; and
# the time variables are described below.
p5spl30rsp135 <- list(formula = ~ -1 + str1:a1 + str1:a2 + str1:a3 +
  str1:a4 + str1:a5 + str1:bs(Time,df=3,degree=3) + str2 +
  str2:bs(Time,df=8,degree=3) + str3, fixed = list(index=c(pal,p1996,
  p1997,p1998.y,p1999.y,p2004.y,p2006.y,p2009,p2011.y,r3,r1997.g,
  r1998.g,r2003.g,r2005.g,r2010.g),value=c(palval,p1996val,p1997val,
  p1998.yval,p1999.yval,p2004.yval,p2006.yval,p2009val,p2011.yval,
  r3val, r1997.gval,r1998.gval,r2003.gval,r2005.gval,r2010.gval)),
  link="logit")
```

These model specifications were then run in RMark using the `mark` function and the simulated annealing algorithm to maximize the potentially multi-modal likelihood:

```
PsigomNH14p <- mark(MS.process,MS.ddl,model.parameters=list(S=Sfix,
  p=p5spl30rsp135,Psi=PsigomNH14p),options="SIMANNEAL")
```

In the model for detection probabilities presented above in the annotated code, we interfaced RMark with the ‘splines’ package in R (see posts on www.phidot.org for details) to implement polynomial B-spline functions for temporal variation, where degree specifies the order of the polynomial function and the difference between df and degree specifies the numbers of inner knots (Hastie 1992). Methods for automatically selecting the amount of smoothing (e.g. Gimenez *et al.* 2006) are not currently available for use in RMark. We additionally fit B-spline functions for age-specific mortality according to cause, but the fit was quite poor, with large confidence intervals.

Appendix 1B. Estimation of age-specific profiles of human-related and natural mortality in roe deer.

All individuals included in the analyses were first captured as fawns (i.e. known-age individuals). Some of them were recaptured alive, some were reported from dying of anthropogenic causes, and others were not detected. A capture history could thus take the following form: 10112, meaning that the individual was first captured and marked, not seen the second year, recaptured the 3rd and the 4th years, and finally killed by a hunter or vehicle and reported in the 5th year. These records were denoted as ‘events’: **0**, not seen; **1**, captured for the first time or recaptured; **2**, killed by human activities and reported. The

corresponding individual states that we considered were: **A**, alive; **H**, individual just died from human causes; **NH**, individual just died from a natural (non-human) cause; **D**, individual already dead.

We then built CR HMM models in several stages using program E-SURGE (Choquet *et al.* 2009). Below, the sum of a row = 1. Consequently, one cell of each row in a parameter matrix will be calculated as the complement of the sum of the others, denoted with a ‘*’ symbol. Cells equal to 0 are denoted with a ‘-’ symbol. An active cell containing a parameter to be estimated receives an arbitrary letter. Note that the same letter in two cells does not mean that the two values are equal (these constraints are made later). The primary symbols for parameters are: μ , mortality probability, and p , detection (or event) probability.

The Gepat interface:

Initial state probabilities

A	H	NH
*	-	-

Transition probabilities: Survival & Cause-Specific Mortality

In the transition probabilities matrix below, the probabilities of an individual transitioning from (rows) being alive at time t (**A_t**) to (columns) either being dead due to human (**H_{t+1}**) or natural (**NH_{t+1}**) causes by time $t+1$ are denoted with a ‘ μ ’ symbol. Individuals dead at time t or ‘already dead’ at time t remain in the state **D** with a probability of 1. Note that this latter state was not explicitly included in the RMark analysis for lesser snow geese above.

	\mathbf{A}_{t+1}	\mathbf{H}_{t+1}	\mathbf{NH}_{t+1}	\mathbf{D}
\mathbf{A}_t	*	μ	μ	-
\mathbf{H}_t	-	-	-	*
\mathbf{NH}_t	-	-	-	*
\mathbf{D}_t	-	-	-	*

Event probabilities: Live Recapture & Dead Recovery

The matrix for event probabilities shown below denotes both live recapture and dead recovery probabilities with a ' p ' symbol (modelled difference in p between the states are designated below). At occasion t , individual states are shown in rows and encounter observations in columns. For example, in the first row individuals not seen (0) but alive (\mathbf{A}) are not recaptured (*), whereas individuals alive (\mathbf{A}) and seen (1) are recaptured with probability p . In the second row, individuals dead from human causes (\mathbf{H}) and not seen (0) are not recovered (*), whereas individuals dead from human-related causes and recovered have a probability p of being observed as such. Individuals dead from natural causes (\mathbf{NH}) are never seen (0), and thus the corresponding detection probability was fixed to 0, as was also done for individuals that were already dead (\mathbf{D} , which was specified as an absorbing state):

	0	1	2
A_t	*	p	-
H_t	*	-	p
NH_t	*	-	-
D_t	*	-	-

The Gemaco interface:

Phrase for initial state: There is no active parameter, just use the keyword ‘i’.

Phrase for transition probabilities: Given our specifications in the ‘Gepat’ step above, this step corresponds to specifying individual variation in cause-specific mortality probabilities (human-related and natural). For instance, in the best model retained for females we considered a parameter specifying a separate human-related mortality probability for age 1, and another for individuals older than 1 year of age. We made similar specifications for natural mortality probability, but additionally specified a generalized logit-linear increase in natural mortality probability from 2 years of age onwards, using the external variable x(1) and the following text in the Gemaco interface (note that age 1 in E-SURGE relates to biological age 0):

‘from(1).to(2).[a(1)+a(2:18)]+from(1).to(3).[a(1)+a(2)+[a(3)&a(4)&a(5)&a(6)&a(7)&a(8)&a(9)&a(10)&a(11)&a(12)&a(13)&a(14)&a(15)&a(16)&a(17)&a(18)]+[a(3)+a(4)+a(5)+a(6)+a(7)+a(8)+a(9)+a(10)+a(11)+a(12)+a(13)+a(14)+a(15)+a(16)+a(17)+a(18)]*x(1)]’.

Phrase for event probabilities: The last step corresponds to specifying the variation in live recapture and dead recovery probabilities. We considered interactive time-dependence in recapture probabilities for the first age class and that for individuals older than 1 year of age, and a constant dead recovery

probability using the following text in the Gemaco interface (where ‘from’ refers to the row, and ‘to’ refers to the column in the event probabilities matrix above):

‘from(1).to(2).[a(1)+a(2:18)].t+from(2).to(3)’.

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