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Methods for studying cause-specific senescence in the wild

David N. Koons[1,2]*, Marlène Gamelon[3], Jean-Michel Gaillard[4], Lise M. Aubry[1,2], Robert F. Rockwell[5], François Klein[6], Rémi Choquet[2] and Olivier Gimenez[2]

[1]*Department of Wildland Resources and the Ecology Center, Utah State University, 5230 Old Main Hill, Logan, UT 84322-5230, USA;* [2]*Centre d'Ecologie Fonctionnelle et Evolutive, Campus CNRS, UMR 5175, 1919 Route de Mende, Montpellier Cedex 5 34293, France;* [3]*Department of Biology, Centre for Biodiversity Dynamics, Norwegian University of Science and Technology, Trondheim N-7491, Norway;* [4]*Laboratoire de Biométrie et Biologie Evolutive, UMR 5558, Université de Lyon, Université Lyon 1, Villeurbanne F-69622, France;* [5]*Division of Vertebrate Zoology, American Museum of Natural History, Central Park West at 79th Street, New York, NY 10024, USA; and* [6]*Office National de la Chasse et de la Faune Sauvage, Centre d'Etude et de Recherche Appliquée Cervidés-Sanglier, 85 bis avenue de Wagram, Paris 75008, France*

Summary

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

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

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

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

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

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

Introduction

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

In wild vertebrates, there is also a long history of studying cause-specific mortality, but with a specific focus on predefined single (e.g. Singer *et al.* 1997 on juveniles; Brodie *et al.* 2013 on adults) or multiple (e.g. Dumke & Pils 1973; Nelson & Mech 1986) age classes. Determining the cause-specific drivers of mortality in both juvenile and adult age classes can indeed

*Correspondence author. E-mail: david.koons@usu.edu

help focus management efforts aimed at conserving populations (Forrester & Wittmer 2013). Apart from the study of simple age classes, however, empirical studies of age-specific causes of mortality and their consequences on the shape of mortality trajectories in wild organisms are lacking.

This is surprising because the original theories on the evolution of senescence (here, the actuarial definition of an increase in mortality and decrease in survival with age) hinge upon concepts of cause-specific mortality (see Box 1). Methods for estimating competing risks of mortality in the wild, and how 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).

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).

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, Manton & Stallard 1979). When not accounted for, intragenerational 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, Nichols & Conroy 2002). Applying this method to example data sets, 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).

Estimating cause-specific mortality across ages in the wild

PERFECT DETECTION

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

In some cases, the immediate cause of death might be obvious (e.g. vehicle or wind turbine collision, hunting or fishing recoveries of marked individuals, bark beetle kill in trees), but necropsies and other types of expert assessments on the less obvious causes of death could provide more detailed insight into the array of factors that kill individuals in the wild (e.g. Mar, Lahdenperä & Lummaa 2012). Often times, however, such detail is out of reach and cause of death will have to be

collapsed into broader categories. Grouped causes of death (e.g. predation, disease) can nevertheless provide deeper insight into mortality dynamics than an assessment of overall mortality (as demonstrated by the examples below).

In studies where observers can at least ascertain whether an individual is alive or not at each census period (i.e. known-fate data), age-specific cause-of-death data can be analysed with existing competing risk statistical models. The general approach is straightforward; instead of specifying a standardized calendar date as the unit of time at which an individual enters and exits the study sample, one must simply substitute 'age' as the unit of time (e.g. Aubry *et al.* 2011). Even the popular Cox proportional hazard model (Cox 1972) can be extended to estimate age- and cause-specific mortality (Heisey & Patterson 2006). To address questions related to the rate of senescence, one might prefer to fit parametric relationships between age and mortality rate according to cause of death (e.g. accelerated failure time models; Wei 1992), whereas for questions related to the shape of mortality over life, one might prefer to fit semiparametric or nonparametric models [e.g. Kaplan & Meier (1958)]. The extensive repertoire of modelling possibilities that are available in traditional survival analysis can for the most part be extended to the study of competing risks (Kleinbaum & Klein 2012).

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

THE CHALLENGE OF IMPERFECT DETECTION AND UNOBSERVABLE FATES IN THE WILD

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

Fortunately, capture-reencounter (CR) methods can decouple the probabilities that comprise a return rate (Burnham 1993) and are often used to robustly estimate survival in wild populations (Williams, Nichols & Conroy 2002). Because of their properties, CR methods are now commonly used to study senescence in the wild (e.g. Gaillard *et al.* 2004; Péron *et al.* 2010). The nuisance of imperfect detection nevertheless presents a challenge to estimating cause-specific mortality in wild animal populations. Death is never observed for most individuals in a wild animal population, and determination of certain causes of death might be completely 'unobservable'.

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

The individual-based encounter histories are combined into a population-level data set to estimate the probability of individual i dying from cause k between discrete time step t and $t + 1$ ($\mu_{i,t}^{k}$) using a CR HMM like that shown in Fig. 2. By defining dead states as absorbing states, the probabilities of transitioning from a live state (A in Fig. 2) to a dead state (B, C, and O in Fig. 2) naturally become cause-specific mortality probabilities (Gauthier & Lebreton 2008). Of key importance, the transition probabilities are estimated conditionally on state-specific probabilities of detecting each individual i in state k at time step t ($p_{i,t}^{k}$) that are simultaneously solved for using either a maximum-likelihood or Bayesian approach (Lebreton *et al.* 2009). According to the example (Fig. 2), $p_{i,t}^{\mathrm{A}}$ would represent the probability of recapturing a live individual i at time t. As long as dead recoveries can be attained at a spatial scale much larger than the study area, fidelity can be subsumed within $p_{i,t}^{\mathrm{A}}$ like in our example (Schaub & Lebreton 2004), or fidelity can be separately estimated (Burnham 1993). For the dead states, $p_{i,t}^{\mathrm{B}}$ is the probability that an individual i that died

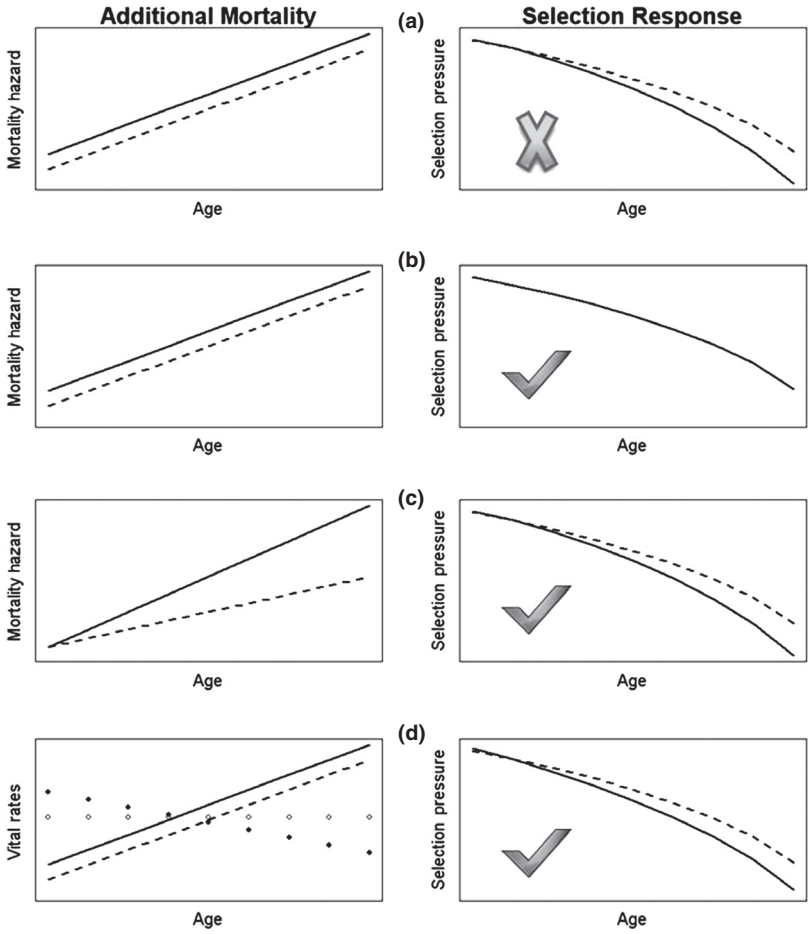

Fig. 1. A graphical example of how exposing a population to additional mortality (left panels) affects the pressure of selection on age-specific mortality hazards (right panels; *sensu* Hamilton 1966). The left side of panel 'a' depicts the original prediction that exposing a population already experiencing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

of cause B between $t-1$ and t was 'recovered dead and reported' to the observers at time t, and $p^{C}_{i,t}$ the same detection probability for an individual that died of cause C. The detection probability must be fixed to 0 for other sources of mortality that are not observable (state O in Fig. 2). It is nevertheless possible to identify the probability of dying from collective unobservable causes ($\mu^{O}_{i,t}$ in Fig. 2) by borrowing information from the joint live recaptures and dead recoveries (Servanty *et al.* 2010).

With regard to cause-specific mortality, multistate CR models have been used to compare and identify problematic sources of mortality in declining or managed populations (Schaub & Pradel 2004; Bischof *et al.* 2009), estimate the strength of natural selection on hunting mortality relative to non-hunting mortality in harvested populations (Gamelon *et al.* 2011) and to estimate the degree of compensation or additivity between sources of mortality (Schaub & Lebreton 2004; Servanty *et al.* 2010; Koons, Rockwell & Aubry 2014). To our knowledge, however, complete age trajectories of cause-specific mortality have never been estimated appropriately while accounting for imperfect detection. When trying to focus on senescence in natural causes of mortality, past studies of ageing in the wild have typically right-censored individuals once they were known to have died from anthropogenic causes. Because fates are not known for all individuals in a CR study, this form of non-random censoring introduces a source of bias. In the examples below, we use long-term studies of two long-lived vertebrates to illustrate how to overcome these important gaps in our understanding of ageing in the wild. To help others use the CR HMM method for their own, potentially more illuminating studies and questions pertaining to senescence and cause-specific mortality, we provide annotated code for implementing the examples using the two leading CR softwares: E-SURGE and program MARK (see Appendix S1).

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

AGE-SPECIFIC TRAJECTORIES OF MORTALITY CAUSES IN THE LESSER SNOW GOOSE

A marked population of lesser snow geese (*Chen caerulescens caerulescens*) has been studied near La Pérouse Bay, Manitoba, Canada, since 1969 (58°44′N, 94°28′W; Cooke, Rockwell & Lank 1995), where recaptures of previously marked individuals are recorded every year during banding drives. In parallel, public hunters across North America submit records of harvested birds with bands to the USGS Bird Banding Laboratory. To estimate age-specific mortality according to cause, we focused on a sample of known-age females banded and released as goslings (n = 45 914) with subsequent live recaptures (n = 1976) and hunter recoveries (n = 5163) between

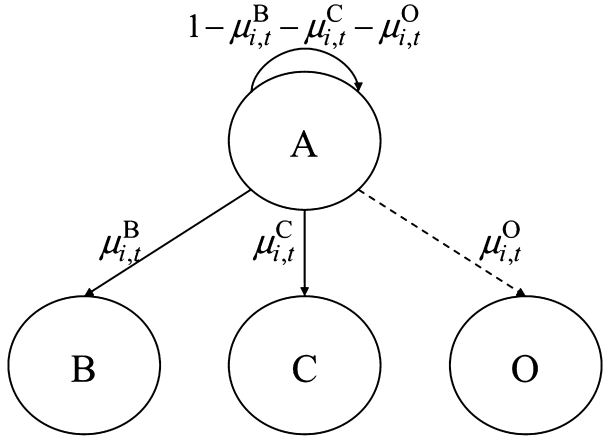

Fig. 2. The observable (solid lines) demographic transitions of remaining alive (A), dying from cause B or C and the unobservable (dashed line) transition of dying from other causes (O); where $\mu^{k}_{i,t}$ denotes the cause-specific probability of mortality per time step (subscripts are as described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

1969 and 2011. Because the human hunter is the predominant predator of adult lesser snow geese (Koons, Rockwell & Aubry 2014), the age-specific level of hunting mortality may modify the strength of natural selection against senescence in both direct and indirect ways (Box 1, Fig. 1). Thus, we developed a CR HMM with one alive state (A) and two dead states: a partially observable state of 'died from hunting' (H; i.e. legally hunted) and an unobservable state of 'died from non-hunting' (NH, which includes any unobservable crippling loss). Observed capture histories never contain explicit information about individuals in unobserved states like NH. To develop a CR HMM, one must therefore define the unobserved states of interest and fix the respective detection probabilities to 0 (Pradel 2005). In Appendix S1, we demonstrate how to do this for the snow goose example using the RMark package for R (Laake & Rexstad 2012), describe our analysis in more detail and provide annotated code for the modelling steps. Here, we focus on demonstrating the approach of using a CR HMM to study age-specific mortality according to cause in the wild.

Drawing from the long history of research on snow goose survival and previous findings for the same or similar data set (Francis *et al*. 1992a; Cooch, Rockwell & Brault 2001; Aubry *et al*. 2013; Koons, Rockwell & Aubry 2014), we developed CR HMMs that account for important sources of age and temporal variation in the detection probabilities, as well as temporal variation in cause-specific mortality probabilities for each of two age classes (hatch-year and after-hatch-year; see Appendix S1). To estimate the trajectory of cause-specific mortality at each age x, μ^{k}_{x}, we considered the Gompertz, Weibull and logit-linear functions (Gaillard *et al*. 2004), with alternative ages of onset for senescence (ages 4, 6, 8, 10, 12, 14 or 16; non-statistically significant results from Francis *et al*. 1992b indicate that if there is senescence, the onset may be delayed until ~ age 10). At each modelling step, we used Akaike's Information Criterion adjusted for sample size (AIC_c; Akaike 1973; Burnham & Anderson 2002) to identify the model structure most supported by the data.

While controlling for temporal variation, we found strong support for Gompertz senescence in non-hunting mortality past age 14 (loglog link:$\hat{\beta}$ = 0·119, 95% CI: 0·083–0·154; Fig. 3; ΔAIC_c = 45·2 for simple age-class effects). This model was more supported than other ages of onset in non-hunting mortality senescence ($\Delta AIC_c > 0{\cdot}8$), as well as the Weibull ($\Delta AIC_c = 0{\cdot}1$) and logit-linear ageing functions ($\Delta AIC_c = 11{\cdot}0$). We detected marginal support for reduced hunting mortality with age (1·1 unit improvement in AIC_c), but the effect was biologically minor and imprecisely estimated ($\hat{\beta} = -0{\cdot}006$, 95% CI: $-0{\cdot}014 - 0{\cdot}001$).

Thus, it seems that actuarial senescence is delayed and restricted to non-hunting sources of mortality in lesser snow geese. Studies of senescence in other long-lived avian species have similarly found delayed onsets of ageing that begin well past the age of primiparity (which ranges from 2 to 4 in snow 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

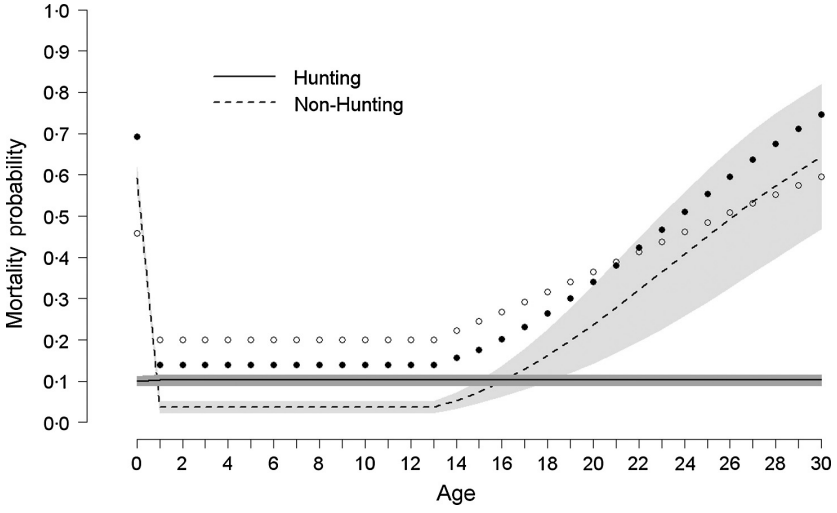

Fig. 3. Trajectories of age-specific mortality probability according to hunting (solid line) and non-hunting (dashed line) causes for female lesser snow geese at La Pérouse Bay, Canada from 1969 to 2010. Shaded polygons represent 95% confidence bounds. The closed circles represent total mortality (i.e.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

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

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

To estimate cause- and age-specific mortality, we allowed natural and/or human-related mortalities to vary linearly on the generalized logit scale from age 1 or 2 onward based on previous studies (e.g. 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, Gaillard & Côté 2003), we also tested models that allowed cause-specific mortality probabilities to vary among five age classes: fawn summer mortality up to age 1, age 1–2, ages 2–8, 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, Rouan & Pradel 2009). In Appendix S2, 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 (ΔQAIC > 2) and other parameterizations of age effects for human-related mortality (ΔQAIC > 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

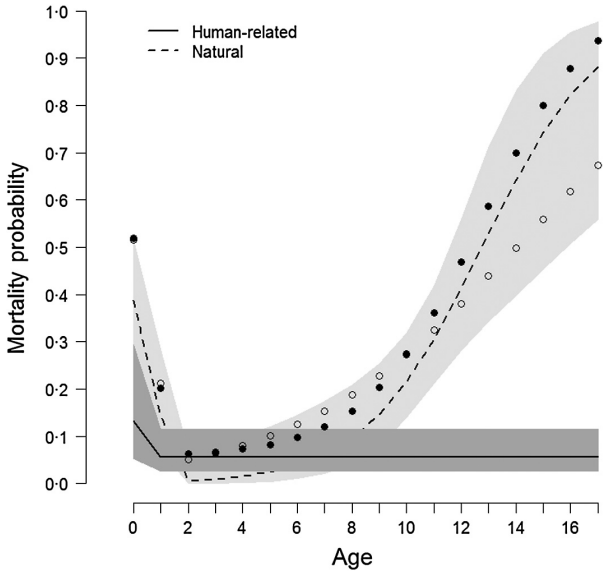

Fig. 4. Trajectories of age-specific mortality probability for human-related (solid line) and natural (dashed line) causes in female roe deer at Trois-Fontaines, France, from 1985 to 2013. Shaded polygons represent 95% confidence bounds. The closed circles represent total mortality (i.e. the addition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

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 similar to those described above for the CR HMMs, and although senescence was still detected, the estimated rate of senescence was 48·4% lower than in the CR HMM that accounted for mortality cause ($\hat{\beta} = 1{\cdot}155$, 95% CI: 0·784–1·526, Fig. 4 open circles). Moreover, this model was 7·64 QAIC units worse than the top cause-specific mortality model. Similar to the snow geese, failure to account for the cause of mortality led to an overestimation of mortality in roe deer from age 4 to 10 and underestimation from age 11 to 17 (Fig. 4, compare open and closed circles).

Discussion

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

Given the impact that exposure to additional mortality can have on selection pressures affecting senescence (Box 1, Fig. 1), both known-fate and CR HMM methods should be used to compare trajectories of age-specific mortality according to cause of death across populations and species where data are available or can be collected. Moreover, these methods can be used to refine insight into findings that indicate old individuals in the wild are sometimes more susceptible to hunting or predation because of interactions between physiological and ecological processes (e.g. Garrott *et al.* 2002; Carlson *et al.* 2007), pressures of trophy hunting (Coltman *et al.* 2003) or because of social organization that exposes the eldest individuals first (Festa-Bianchet *et al.* 2006). In the case of the human hunter, it has been found in some systems that the 'human predator' may simply consume prey in proportion to their occurrence (e.g. abundant prime-aged adult elk; Wright *et al.* 2006). In our examples, we found that the chance of dying from human-related causes was largely age independent after maturity in both female snow geese and roe deer. A high rate of hunting mortality can nevertheless select for increased early-life allocation to reproduction (Gamelon *et al.* 2011), and future studies should examine how this may in turn affect the rate of actuarial senescence (Fig. 1) as well as senescence in reproductive success (Rockwell *et al.* 1993).

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

Examining age trajectories of cause-specific mortality (e.g. in humans, Horiuchi *et al.* 2003) can also provide more explicit targets for associating environmental conditions (e.g. toxin or pathogen exposure), physiological

Table 1. Simulated logit-linear coefficients for age effects on mortality probabilities in a CR HMM with one alive state, a partially observable state for mortality cause X, an unobservable state for other (O) causes of mortality and an absorbing state for those already dead (++ = 0·5 increase in mortality with age on the real scale, + = 0·25 increase and 0 no increase; implemented using a continuous age variable 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 estimated coefficients relative to simulated values (95% CI provided within parentheses), as well as the estimated age effects for mortality when states X and O are collapsed (conflated) into a single state. Simulations involved the release of newborns in a fashion that maintained 10 000 individuals in the simulated sample at all time steps, $p^X = 0{\cdot}3$, $p^O = 0{\cdot}3$ and a common mortality intercept (−1·50). Simulations in B) were similar but included unique parameters for the mortality intercepts of cause X and O (each simulated with a value of −1·50). Differences between simulated and estimated coefficients indicate problems with either partial or complete non-identifiability of model parameters. Interestingly, estimation with a constrained identity link function (not shown) corrected these problems

Simulated		Estimated				
β^X_{age}	β^O_{age}	$\hat{\beta}^X_{age}$	$\hat{\beta}^O_{age}$	$\hat{\beta}^X_{int}$	$\hat{\beta}^O_{int}$	Conflated $\hat{\beta}^{conflated}_{age}$
(A)						
++	+	0·50 [0·36, 0·65]	0·18 [−0·01, 0·36]	−1·48 [−1·50, −1·47]		0·48 [0·40, 0·56]
+	++	0·17 [0·02, 0·31]	0·61 [0·46, 0·76]	−1·50 [−1·52, −1·49)		0·51 [0·43, 0·58]
++	0	0·46 [0·32, 0·59]	−0·04 [−0·22, 0·14]	−1·50 [−1·51, −1·48]		0·32 [0·25, 0·40]
0	++	0·07 [−0·07, 0·21]	0·48 [0·33, 0·62]	−1·50 [−1·51, −1·48]		0·35 [0·28, 0·42]
(B)						
++	+	0·44 [0·30, 0·58]	0·34 [0·15, 0·53]	−2·35 [−3·80, −0·91]	−0·93 [−1·49, −0·37]	–
+	++	0·13 [0, 0·26]	0·54 [0·45, 0·63]	−2·70 [−3·16, −2·23]	−0·85 [−0·98, −0·72]	–
++	0	0·45 [0·10, 0·80]	−0·01 [−1·5,1·5]	−1·57 [−5·5, 2·41]	−1·42 [−5·0, 2·18]	–
0	++	0·07 [−0·09, 0·23]	0·47 [−0·22, 1·16]	−1·54 [−5·45, 2·36]	−1·45 [−5·13, 2·22]	–

condition, gene loci and quantitative gene expressions with senescence in the competing risks they affect most (Nussey *et al.* 2008). For example, the CR HMM method presented here can readily be used in longitudinal studies of individual life histories (Clutton-Brock & Sheldon 2010) to examine trade-offs between age-specific allocations to reproduction, the cause of mortality these allocations affect most at given points in the life cycle, and the net impact this has on selection against overall actuarial or reproductive senescence. By honing in on the specific causes of mortality that reproductive allocations affect most, the CR HMM method could be used to help clarify the role of pleiotropic gene expressions (Charmantier *et al.* 2006) and environmental conditions (Van Noordwijk & de Jong 1986) that shape trade-offs in the wild.

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

Although the CR HMMs used in our examples were *a priori* identifiable, not all CR HMM parameterizations will be (see Table 1B). As in our examples (Appendices S1 and S2), accounting for temporal variation and other variables can actually help improve parameter identifiability (Schaub & Lebreton 2004; Schaub & Pradel 2004). Future studies should conduct thorough analyses of parameter identifiability to determine the types of CR HMMs and link functions that can and cannot be fit to cause-specific mortality data (Gimenez, Choquet & Lebreton 2003; Table 1). In conclusion, the methods presented here provide a baseline for enhancing methodological developments and advancing the analysis of mechanisms that drive the large variation in ageing observed across the tree of life (Jones *et al.* 2014).

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Data accessibility

The roe deer data used in the example are property of the French Office National de la Chasse et de la Faune Sauvage. The snow goose banding and encounter data are made available to the public by the USGS Bird Banding Laboratory at http://www.pwrc.usgs.gov/BBL/homepage/datarequest.cfm.

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Supporting Information

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

Appendix S1. Estimation of age-specific profiles of hunting and non-hunting mortality in lesser snow geese.

Appendix S2. Estimation of age-specific profiles of human-related and natural mortality in roe deer.