Biological Conservation 182 (2015) 187–196

Contents lists available at ScienceDirect

Biological Conservation

journal homepage: www.elsevier.com/locate/biocon

Is anthropogenic cougar mortality compensated by changes in natural mortality in Utah? Insight from long-term studies

Michael L. Wolfe [a], David N. Koons [a,b], David C. Stoner [a], Patricia Terletzky [a], Eric M. Gese [c], David M. Choate [d], Lise M. Aubry [a,b,*]

[a] Department of Wildland Resources, 5230 Old Main Hill, Utah State University, Logan, UT 84322, USA
[b] The Ecology Center, 5230 Old Main Hill, Utah State University, Logan, UT 84322, USA
[c] U.S. Department of Agriculture, Wildlife Services, National Wildlife Research Center, Department of Wildland Resources, Utah State University, Logan, UT 84322, USA
[d] School of Life Sciences, University of Nevada, 4505 Maryland Parkway, Las Vegas, NV 89154, USA

ARTICLE INFO

Article history:
Received 16 September 2014
Received in revised form 3 December 2014
Accepted 9 December 2014
Available online 31 December 2014

Keywords:
Additive mortality
Carnivore
Compensatory mortality
Competing risks
Cox proportional hazard
Harvest

ABSTRACT

Understanding the interplay between exploitation and natural mortality is essential to guiding sustainable conservation of wildlife. Exploitation of carnivores by humans has long been thought to result in compensatory reductions of natural mortality among survivors. If rates of human exploitation exceed natural mortality, however, such actions will 'add' to overall mortality and could imperil the sustainability of such actions. We applied competing risk analyses to ⩾16 years of data for heavily harvested and semi-protected cougar populations in Utah to test the additive and compensatory mortality hypotheses, while accounting for parameter uncertainty. We additionally tested for presence of the two primary mechanisms by which compensatory mortality can arise: density dependence and individual heterogeneity in mortality risks. Despite an opportunity for compensation in the heavily harvested population, we could not reject the additive mortality hypothesis when uncertainty in parameter estimates was accounted for. In the semi-protected population, however, we detected evidence for partial compensation of increased anthropogenic exploitation via reductions in natural mortality. As may be common in carnivore studies, we found that ignoring uncertainty in estimates of cause-specific mortality systematically led to biased conclusions regarding additive and compensatory mortality hypotheses. Efforts should be made to address and minimize this uncertainty in demographic studies of carnivores in order to avoid flawed management recommendations. To attain the necessary sample sizes for making sound inference, this may require that the spatial extent of management units be extended for territorial species with large home-range requirements.

© 2014 Elsevier Ltd. All rights reserved.

1. Introduction

The interaction between predation and other causes of mortality has long been of interest to ecologists and is of central importance to the sustainable management of wild species (Caughley, 1977). The concept of compensatory mortality with respect to wildlife management emerged largely as an extension of ideas advanced by Errington and Hamerstrom (1935). The core of his hypothesis was that in populations existing above a seasonal carrying capacity ("security threshold"), mortality from human exploitation (or predation) simply removes a "doomed surplus" that would otherwise perish from other natural causes. Populations existing below this threshold of security were thought to be protected from over-exploitation via density dependent processes and the ability to increase in number (Errington and Hamerstrom, 1935). This hypothesis, and its more intuitive alternative that predation and exploitation 'add' to other sources of mortality, have since generated a large body of research (e.g. Anderson and Burnham, 1981; Burnham and Anderson, 1984; Nichols et al., 1984; Bartmann et al., 1992; Sinclair and Pech, 1996; Boyce et al., 1999). Beyond the simple dichotomy of compensatory versus additive mortality, these concepts have developed into a spectrum of alternative hypotheses including partial compensation, over-additivity, and overcompensation (Fig. 1; Servanty et al., 2010).

Although exploitation and predation can be compensated at the population level via subsequent, density-dependent improvements

* Corresponding author at: Department of Wildland Resources and the Ecology center, 5230 Old Main Hill, Utah State University, Logan, UT 84322, USA. Tel.: +1 435 770 338.

E-mail address: lise.aubry@usu.edu (L.M. Aubry).

http://dx.doi.org/10.1016/j.biocon.2014.12.008
0006-3207/© 2014 Elsevier Ltd. All rights reserved.

in reproductive success or the balance between immigration and emigration, the additive and compensatory 'mortality' hypotheses refer specifically to the effects of exploitation or predation on levels of cause-specific mortality (Anderson and Burnham, 1976). This dichotomy is important for guiding research, and for understanding the sustainability of hunting and other forms of exploitation (Runge and Johnson, 2002). As such, the spectrum of additive and compensatory mortality hypotheses *per se* have become the subject of active carnivore management research (e.g. Sparkman et al., 2011; Creel and Rotella, 2010; Murray et al., 2010; Robinson et al., 2014), but could benefit from insight on a number of issues being addressed in other taxa.

For example, exploitation in carnivores is commonly thought to lower the seasonal density of a population, thereby freeing up resources and relaxing competition among survivors (e.g. Knowlton et al., 1999) with the underlying rationale being that for every life taken, a life is saved (Boyce et al., 1999). These findings have served as a virtual *sine qua non* for carnivore management through the 1970s and 1980s (Frank and Woodroffe, 2001; Festa-Bianchet, 2003). But compensatory mortality can also occur when exploitation simply changes the cause of death for 'frail' individuals that would have likely died from other causes (i.e. the notion that certain individuals are intrinsically predisposed to a higher risk of death than others; Lebreton, 2005; Péron, 2013). Indeed, a variety of genetic, maternal and environmental factors can produce individual heterogeneity in survival abilities (Wilson and Nussey, 2010), as well as susceptibility to either wild predators or humans (e.g. Koons et al., 2014a, b). In fact, density dependence in mortality has to reach almost unrealistically high levels to allow for complete compensation of exploitative mortality, whereas compensatory mortality can more easily occur via intra-annual selection on the distribution of individual heterogeneity in survival abilities (Lebreton, 2005). This latter mechanism has been identified as an important determinant of partial compensation in geese (e.g. Lindberg et al., 2013). Regardless of the mechanism, compensatory mortality can broaden the spectrum of sustainable harvest, but also hinder a manager's ability to control a population via harvest management.

Anderson and Burnham (1976) formalized statistical approaches to distinguishing between additive and compensatory harvest mortality (Fig. 1). In commonly used discrete time Capture-Reencounter (CR) models, however, estimates of hunting and non-hunting mortality are intricately linked through sampling covariance. In addition, a process bias can arise because the hunting season is often nested between discrete sampling occasions, affecting the sample of individuals at risk of non-hunting mortality in subsequent seasons (Lebreton, 2005; Péron, 2013; Cooch et al., 2014). The issues of sampling covariance and process bias can both obscure the underlying relationship between the net rates of hunting and non-hunting mortality (e.g. Nichols and Hines, 1987). Modern CR models with random effects can be used to account for the issue of sampling covariance (e.g. Schaub et al., 2004; Sedinger et al., 2010; Koons et al., 2014a, b), but the implications of process bias in CR studies of additive and compensatory mortality can only be examined through sensitivity analyses (Servanty et al., 2010; Péron, 2013).

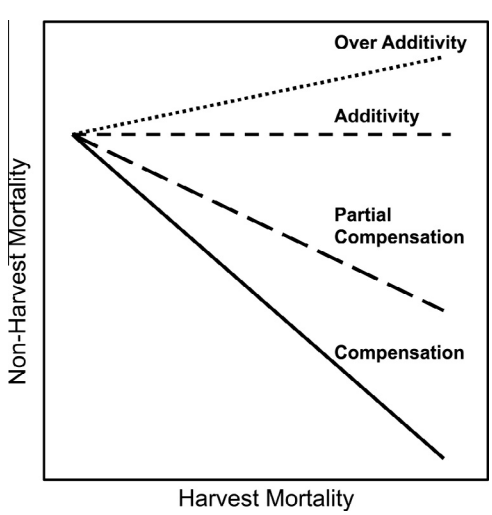

Fig. 1. Possible functional relationships between non-harvest and harvest mortality, such as total compensation (sold line), partial compensation (long dash line), additivity (short dash line), and over-additivity (dotted line).

Radio-telemetry data are well suited to attaining unbiased estimates of net source-specific mortality rates using known-fate competing risk analyses that immediately censor individuals from the at-risk sample once they die of a given cause (Heisey and Fuller, 1985; Heisey and Patterson, 2006). Such data and methods allow for robust inference into the compensatory and additive mortality hypotheses (e.g. Sandercock et al., 2011). There are nevertheless sampling variances and uncertainty in the estimated net rates of mortality that need to be acknowledged when testing these hypotheses with competing risk models. Moreover, when substituting spatial replicates for temporal replicates in tests of the additive and compensatory mortality hypotheses (e.g. Robinson et al., 2014), care must be taken to control for equivalence of environmental conditions, which could otherwise confound insight into the underlying relationship between competing risks of mortality. These issues underscore the need for a better understanding of the relationship between anthropogenic mortality (i.e. harvest, road kill, agency removals, and poaching) and other sources of natural mortality in carnivores such as disease, intra-specific strife, death from injuries sustained during prey capture, and starvation (Quigley and Hornocker, 2009).

The history of cougars (*Puma concolor*) in North America offers a particularly interesting example for testing the additive and compensatory mortality hypotheses in a carnivore. Cougars were once persecuted as a "bountied predator" because of their predation on domestic livestock and indigenous ungulates (Gill, 2009). Beginning in the mid 1960's, however, most states elevated the species' status to that of a protected game animal. Cougars are now managed primarily through regulated sport hunting to maintain viable populations (Keefover-Ring, 2005; Anderson et al., 2009), and reduce impacts of predation on their principal prey species, mainly mule deer (*Odocoileus hemionus*), elk (*Cervus elaphus*), as well as other high profile prey species such as bighorn sheep (*Ovis canadensis*) (Pierce and Bleich, 2003). Levels of cougar exploitation nevertheless vary widely among management jurisdictions, and may not be sustainable in some areas (Cooley et al., 2011). Management agencies often face the difficulty of opposing demands for more effective cougar control to protect human safety, big game populations, and domestic livestock, as well as the demand for additional cougar-hunting opportunities by sportsmen and outfitters and even societal demands for outright protection from exploitation (Lindzey et al., 1992, 1994). Identification of the degree of compensatory mortality is thus important for guiding the management of cougar exploitation.

Here we examine whether anthropogenic cougar mortality is compensated by changes in natural mortality while accounting for the uncertainties mentioned above by analyzing longitudinal data from two cougar populations that have been intensively monitored in Utah over 17 and 16 years, respectively. The first study population inhabits the Monroe Mountains, a remote location in south-central Utah where cougars are subjected to varying levels and sources of exploitation. The second is a semi-protected population in the Oquirrh Mountains, on the periphery of Salt Lake City and exposed to lower and different forms of anthropogenic

mortality (Stoner et al., 2006). This natural experiment offers the unique opportunity to assess whether compensatory mortality is the mechanism at play when harvest is (i.e. Monroe population), or is not (i.e. Oquirrh population) the main driver of overall mortality, in two locations that are subjected to differences in land ownership and associated levels of human access.

2. Methods

2.1. Study areas and harvest regimes

2.1.1. Oquirrh Mountains: protected population, near urban location

The Oquirrh-Traverse Mountains (hereafter the Oquirrhs) are located in north-central Utah on the eastern edge of the Great Basin (40.5°N, 112.2°W; Fig. 2). The Oquirrhs measure >950 km^2, but we focused fieldwork on 500 km^2 encompassing the northeastern slope on properties owned and managed by the Utah Army National Guard (Camp Williams) and the Kennecott Utah Copper Corporation. The site was bounded on the north by the Great Salt Lake and on the east by the Salt Lake Valley. Approximately 55% of the entire mountain range is under the jurisdiction of the Bureau of Land Management (BLM), with the remainder privately held by individuals, grazing associations, the Utah National Guard, and mining companies. The study area was situated within the Utah Division of Wildlife Resources (UDWR) Oquirrh-Stansbury Wildlife Management Unit, but private properties were closed to the public and cougar hunting was prohibited. Although radio-instrumented cougars leaving those properties were legally protected within the management unit, they were susceptible to harvest and other causes of death within the vicinity of the management unit, which was encompassed within our study area. In this sense the population was "quasi-protected." Human density adjoining the study area varied from 232/100 km^2 in rural Tooele County to 47,259/100 km^2 in urban Salt Lake County (Stoner et al., 2006).

2.1.2. Monroe Mountains: exploited population, rural location

The Monroe Mountains comprise part of the Sevier Plateau in the Southern Mountains eco-region of south-central Utah (38.5°N, 112°W; Fig. 2). The study site measured ~1300 km^2, and formed the central unit of the Fishlake National Forest. Other landholders included the BLM, the state of Utah, and various private interests. The study site was within the UDWR Monroe Mountains Wildlife Management Unit, where deer, elk, and cougars were managed for sustainable hunting opportunities. Other carnivores present included bobcats (*Lynx rufus*) and coyotes (*Canis latrans*), which were both subject to trapping pressure. Resource use included livestock grazing (cattle, sheep), logging, fossil fuel exploration, and off-highway vehicle recreation. Human densities around the site varied from 73 to 382/100 km^2 (Stoner et al., 2006), with most of the population distributed among small agricultural communities in the Sevier Valley on the northwestern boundary of the study site. Additional information on the study sites can be found in Appendix A.

2.2. Data collection

2.2.1. Capture, marking, and radio-telemetry

From January 1996 to June 2012, we conducted intensive capture efforts during winter (December to April). We used hounds to trail cougars of all age classes. Pursuit and immobilization techniques are detailed in Stoner et al. (2006). We aged cougars using the tooth-wear criteria of Ashman et al. (1983) and the regressions of Laundré and Hernández (2002) for estimating kitten ages. Regardless of age, all animals captured were tattooed and all sub-adults (1.0–2.5 years) and adults (>2.5 years) were equipped with VHF radio-collars (Advanced Telemetry Solutions, Isanti, MN). Cougar locations were acquired at least once per month from aerial or ground telemetry (Mech, 1983). Each year, 3–4 cougars were fitted with a global positioning system (GPS) collar (Televilt Simplex or LoTeck 4400S) that acquired a location every 3 h. Kittens were marked with an ear transmitter, ear tag, or a drop-off radio-collar ($n = 33$). We considered sub-adults as either yearling kittens still accompanying their mother, or transients initiating or in the act of dispersal. Animal capture and handling procedures were conducted in accordance with Utah State University Institutional Animal Care and Use Committee standards (approval no. 937-R).

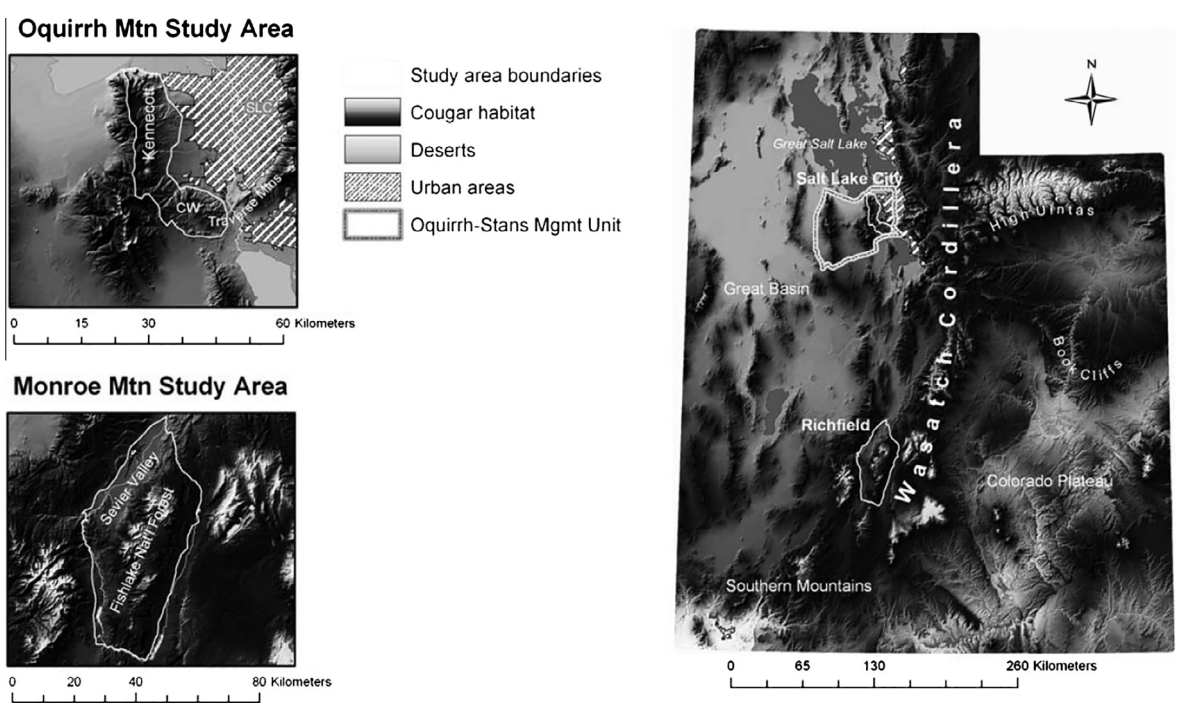

Fig. 2. The Oquirrh (north) and Monroe (south) Mountain study locations in Utah.

Data analysis (see below) was restricted to the radio-telemetry information collected between the 1st of January 1996 and 31st of June 2012 in the Monroe study area, and between the 1st of January 1997 and 31st of June 2012 in the Oquirrh-Stansbury study area. Agency removal of problematic individuals, poaching, road kill, legal harvest, intra-specific strife, sickness, starvation, infection, and injury (including death at capture, or resulting from injuries sustained while capturing prey) were all possible causes of death. Causes of mortality were determined through visual inspection and necropsy of carcasses. When cause of death could not be determined in the field, the carcass was submitted to the Utah Veterinary Diagnostics Lab for detailed analysis. Precision of mortality dates varied; death dates were known to within one day for GPS-collared and hunter-harvested individuals, whereas dates for animals wearing conventional VHF radio-collars were estimated using the midpoint between the last live signal and the detection date of the first mortality signal (up to ±15 days; Sandercock et al., 2011).

2.3. Statistical analyses

2.3.1. Drivers of variation in survival

Classical survival models used in human demography (e.g. Kleinbaum and Klein, 2005) are appropriate for estimating survival trajectories when individual fates are known, which is often the case in radio-telemetry studies of wildlife (e.g. Murray et al., 2010). Various extensions to the non-parametric Kaplan–Meier (Kaplan and Meier, 1958) estimator, such as the semi-parametric Cox Proportional Hazard model (CPH; Cox, 1972), further allow identification of the measurable (observed) covariates associated with patterns in survival trajectories. We used CPH models because they do not require assumptions about the shape of the underlying mortality hazard (a.k.a., the force of mortality) over life. Rather, each covariate within the model is assumed to act multiplicatively (proportionally) on the time-specific baseline mortality hazard and across covariate levels (Bradburn et al., 2003), such as $h_i(t) = h_0(t) \times \exp(\beta_i X_i)$ where h_0 refers to the baseline hazard (i.e. the hazard's value when all covariate values are null), and X denotes a vector of covariates such as $X = (X_1, X_2, \ldots, X_i)$, and t denotes time (in our case, time elapsed since marking; Murray and Patterson, 2006).

Using CPH models, we examined support for hypothesized drivers of variability in survival, such as sex, location, and age (sub-adults and adults), where sub-adults encompassed both individuals that made it to their first birthday, as well as individuals that were marked and released for the first time between ages 1 and 2; data for kittens were excluded from the analysis. Exact age was not always known, which is why we focused on age categories rather than true age *per se*. Because harvest is disproportionately focused on males, we would expect female survival to be superior to that of male survival, especially within the Monroe study area where harvest pressure is far greater than that on the Oquirrh-Stansbury management unit.

To address whether or not density dependence could potentially serve as a mechanism for compensatory mortality, we tested for the effects of estimated population density on survival. As described in Choate et al., 2006, we derived reference population estimates from the combination of captured animals marked or equipped with radio-collars, unmarked hunter- or depredation-killed animals, and evidence based on intensive tracking efforts of unmarked individuals. Specifically, these assessments were conducted between 1996 and 2012, which served as the minimum abundance index covariate at time t in CPH analyses of mortality from t to $t + 1$ (app. B). We considered single, additive, and interactive effects of the aforementioned variables on survival chances over time using the 'coxph' function (R library 'survival'; Therneau and Grambsch, 2000) available in R (version 2.15.0, Development Core Team, 2012).

A variety of genetic, maternal and environmental factors can lead to variation in survival abilities among individuals of the same population (Wilson and Nussey, 2010). When difficult or impossible to measure directly, these unobserved differences in survival abilities across individuals (commonly called 'frailty'; Vaupel and Yashin, 1985) lead to underlying changes in the composition of a sample population. When 'frailty' is related to both succumbing to natural causes of mortality and anthropogenic causes, these intra-generational selective forces can also lead to compensatory mortality (or partial compensation). For example, hunting might simply change the cause of death for a frail individual, but not change the overall rate of mortality when such individuals are also more likely to die from another cause (Lebreton, 2005). Therefore, we used frailty models to estimate the amount of unobserved individual heterogeneity in survival chances not accounted for by covariates in the best performing CPH model (Aubry et al., 2011). To define such a model we used the 'coxme' package in R (Therneau, 2012), which allows one to fit a CPH model containing mixed (fixed and random) effects, and assumes a Gaussian distribution for the random effects.

Model selection of fixed effects was based on Akaike's Information Criterion (Akaike, 1973) corrected for sample size (AICc) and associated metrics such as differences in AICc values between competing models and AICc weights (ΔAICc, w_i respectively; Burnham and Anderson, 2002). To test the validity of using CPH models, we used the 'coxzph' procedure (R library 'survival'; Therneau and Grambsch, 2000) to assess whether each covariate modality within each CPH model acted proportionally on the mortality hazard (Therneau and Grambsch, 2000). If so, the p-value associated with each covariate's proportionality test should be >0.05. We also assessed the statistical precision of each estimated regression coefficient (age class, location, sex) by verifying whether associated 90% confidence intervals for $\exp(\beta)$ (i.e. mortality coefficients) overlapped 0, and by reporting associated p-values.

2.3.2. Cause-specific mortality in the hunted and semi-protected study populations

Competing risk analysis is an extension of classic survival analysis except that the survival function considers a pair of random variables, T, the survival time, and K, the cause of death; cause-specific mortality is the joint probability of death before time t from cause k (Heisey and Patterson, 2006; e.g. Murray et al., 2010). Note that because cause-specific mortality probabilities are mutually exclusive, they sum to the total mortality probability. Conditional on the variables identified as affecting survival from above, we examined differences in cause-specific mortality between these variables using a competing risk framework ('csm' function, R library 'wild1', Sargeant, 2011) as described in Heisey and Patterson (2006). We additionally used Cumulative Incidence Functions 'CIF' (akin to cumulative mortality risk or cumulative hazard) to visualize patterns of cause-specific mortality over time.

2.3.3. Testing compensatory and additive mortality hypotheses

To test the additive and compensatory mortality hypotheses, we defined the cougar year to begin immediately after the end of the harvest season (1st of March 1996 and 1st of March 1997 for the Monroe and Oquirrh-Stansbury study sites, respectively) to avoid having to cut the harvest season in half. We then collapsed causes of mortality into harvest and non-harvest, or anthropogenic and natural categories, and estimated cause-specific mortality probabilities on an annual basis using the same methods as described above. Next, we used linear regression ('lm' function available from R library 'stats'; Chambers, 1992) to examine the relationship between cause-specific annual mortalities for either

(i) human harvest versus all other causes of death, or (ii) overall anthropogenic mortality versus natural mortality after applying an arcsin-square-root transformation to each set of mortality estimates (Murray et al., 2010). We applied our analyses to each location separately, but had to exclude sex from the analysis because of limited sample size (see results) within years. When imprecision (e.g. 90% CI) in the estimated slope of relationship between annual cause-specific mortality probabilities overlaps zero, one would conclude that, given the data, there is insufficient evidence to reject the null hypothesis of fully additive effects of exploitation on overall mortality (Lebreton, 2005). On the other hand, if estimated slopes are negative, one would conclude that at least some compensation of exploitation occurs via changes in non-anthropogenic causes of mortality (Fig. 1).

Fortunately, we did not need to address possible sources of bias induced by substituting spatial replicates (e.g. habitat differences) for temporal replicates in our analyses because the studies provide some of the longest time series of data for estimating cause-specific mortality in felids. Separate tests were therefore performed for each study area with differing environmental conditions (see descriptions above). However, a crucial step that is often overlooked in tests of the additive and compensatory mortality hypotheses is the need to account for sampling error when estimating uncertainty in the relationship between competing risks of mortality. Often times, point estimates of cause-specific mortality probabilities are regressed against each other, and the standard error in the estimated slope coefficient is used to assess uncertainty in the relationship between competing risks of mortality. This practice is not valid because cause-specific mortality probabilities are estimates, not data, and the respective degrees of uncertainty in these estimates are often ignored in carnivore studies. Ideally, one would use a bootstrapping approach (Efron and Tibshirani, 1994) to account for uncertainty in each parameter associated with the study sample.

For example, one could iteratively fit thousands of regressions to parameter estimates from each boot-strapped sample to more rigorously measure uncertainty of tests regarding the additive or compensatory hypotheses (see Murray et al., 2010). Alternatively, one could fit a complicated hierarchical model to the data in order to separate sampling covariance from process covariance (e.g. Koons et al., 2014a, b). Despite the longevity of both studies, annual sample sizes in each study area were too small to attain robust results from a bootstrapping procedure (Efron and Tibshirani, 1994). As an alternative, we used a Monte Carlo simulation approach (Kroese et al., 2011) to sample 1000 observations from within the estimated bounds of error in annual cause-specific mortality probabilities using beta distributions, an appropriate distribution for probabilities (i.e. numbers bounded between 0 and 1). For each iteration, we estimated both the intercept and slope of the relationship between transformations of annual cause-specific mortality probabilities. This allowed us to estimate the mean slope of relationship between competing risks of mortality, as well as confidence intervals that fully acknowledged the associated uncertainty in annual estimates of cause-specific mortality probabilities.

3. Results

Across study sites, overall sample size included 148 individuals (82 females and 66 males) in the Monroe study area and 87 individuals (55 females and 32 males) in the Oquirrh-Stansbury study area. The fate of most individuals that died was known, with the exception of 11 cases. Overall, we were able to determine the cause of death for 156 cougars; an additional 79 cougars were right-censored either because they were still alive at the end of the study or because they emigrated from the respective study areas.

3.1. Drivers of variability in survival

The best performing CPH model of variation in cougar survival retained the effects of sex and location, as well as an interaction between the two variables (Table 2). When interpreting a CPH model, covariate levels are always compared to a baseline hazard, here representing the sample of females belonging to the Monroe study population ($\beta = 0$, $\exp(\beta) = 1$). Males had a much lower survival probability on average than females and a higher mortality rate (Fig. 3; $\exp(\beta_{\text{male,Monroe}}) = 3.932$, s.e. = 0.200, $p < 0.001$), but this result was somewhat tempered within the Oquirrh study site (Fig. 3; $\exp(\beta_{\text{male,Oquirrh}}) = 0.481$, s.e. = 0.355, $p = 0.039$). A less parsimonious model included an additional effect of population density on survival, but this model led to a higher AICc (Table 2) and the effect was not statistically significant ($\exp(\beta_{\text{density}}) = 1.037$, s.e. = 0.153, $p = 0.81$). *P*-values associated with each covariate's proportionality test were >0.05, indicating no sign of departure from the proportionality assumption underlying CPH models. Using frailty models to estimate the amount of unobserved individual heterogeneity in survival that was not accounted for by the covariates, we found that the best performing model structure, with and without frailty, yielded similar results with estimated coefficients that were <0.0001 units different and indicated relatively little random variation in survival among individuals (s.d. = 0.0199).

3.2. Cause-specific mortality

For the sample of individuals that died during the study period, 84% of cougars from the Monroe study area died from anthropogenic causes, mainly harvest (73.6% of all documented fatalities). Within the sample of cougars that succumbed to anthropogenic causes of death, 61.2% were males. In contrast, anthropogenic causes only accounted for 44.0% of fatalities in the Oquirrh study area, where natural mortality was the main cause of death (56%), specifically disease and intra-specific strife (24% and 22% respectively). Of the 34% of individuals harvested, 62.5% were males. Of the 56% individuals dying a natural death in the Oquirrh study site, 63.3% were females. However, fatality frequencies provide biased insight into cause-specific mortality because they do not account for right censoring, staggered entry, and exposure time to various risks of death.

Our more robust cause-specific mortality analyses indicated that Monroe males experienced the highest levels of annual harvest mortality and Oquirrh females the lowest. Annual rates of non-harvest mortality were highest for Oquirrh males and lowest for Monroe males and females (Table 1). Similar patterns were observed when comparing anthropogenic versus natural

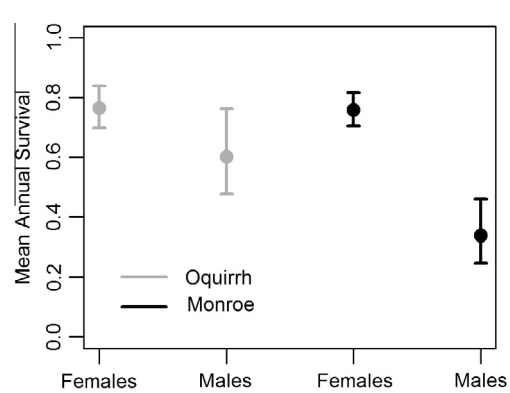

Fig. 3. Mean annual survival estimates for female and male cougars in the Oquirrh and Monroe study populations. Estimates were attainted from the best performing CPH model (Table 2).

mortality among sexes and locations (Table 1). Although the total rate of mean annual mortality was similar between locations for females, males in the Monroe study area experienced higher overall mortality than males in the Oquirrh study area (Fig. 3 and Table 1) because of the high hunting pressure (Appendices C1 and C2).

3.3. Compensatory versus additive mortality

Without accounting for uncertainty in mortality estimates, one might conclude that years with high harvest mortality were partially compensated by reductions in non-harvest mortality for the Monroe population (Fig. 4, top-left, slope parameter = −0.33, $p < 0.001$, $R^2 = 0.28$), and that a similar partially compensatory relationship occurred between anthropogenic and natural mortality (Fig. 4, bottom-left, slope parameter = −0.28, $p = 0.04$, $R^2 = 0.26$). In the Oquirrh population, one might conclude nearly complete compensatory relationships between harvest and non-harvest mortality (Fig. 4, top-right, slope parameter = −0.91, $p = 0.03$, $R^2 = 0.35$), and between anthropogenic and natural mortality (Fig. 4, top-right, slope parameter = −1.03, $p = 0.006$, $R^2 = 0.5$). Such conclusions would however be inappropriate because of the potentially large type II error associated with ignoring sampling error in the respective mortality estimates (see Section 2).

After accounting for such uncertainty using a Monte Carlo simulation approach, results pertaining to the functional relationships between competing risks of cougar mortality were largely inconclusive (Fig. 5). Despite an estimated negative relationship between harvest and non-harvest mortality for the Monroe study area, the estimate was imprecise and of lesser magnitude (Fig. 5 top-left panel) relative to the analysis that ignored uncertainty in mortality estimates (Fig. 4 top-left panel). Similar results were attained when examining the relationship between anthropogenic and natural mortality (Figs. 4 and 5 bottom-left panels). In both cases, 90% confidence intervals estimated from Monte Carlo simulations largely overlap 0, indicating that there was insufficient evidence to reject the null hypothesis of fully additive effects of exploitation on overall mortality (Lebreton, 2005). We were nevertheless able to detect partially compensatory relationships between harvest and non-harvest mortality at the Oquirrh study site (Fig. 5 top-right panel), and between anthropogenic and natural mortality (Fig. 5 bottom-right panel). However, the intensities of these estimated relationships were much less severe (Fig. 5 right panels) compared to the fully compensatory relationships attained when not accounting for uncertainty in mortality estimates (Fig. 4 right panels).

4. Discussion

There is a long tradition of studying the relationship between exploitation and natural mortality in game species (starting with Errington and Hamerstrom, 1935) because of the relevance of such studies to wildlife management (e.g. Burnham and Anderson, 1984). As expected, we found that anthropogenic sources were the leading drivers of mortality in the Monroe population, with hunting being the leading cause of death (harvest mortality rate = 0.304 ± 0.051), especially in males (0.532 ± 0.098). Despite room for compensation between exploitation and natural mortality in the Monroe study area, we could not reject the additive mortality hypothesis when uncertainty in parameter estimates was accounted for. In the Oquirrh study area, the relative proportion of mortalities was almost equally divided between anthropogenic (0.173 ± 0.054) and natural mortality (0.209 ± 0.061). When ignoring uncertainty in parameters estimates, rates of anthropogenic exploitation were fully compensated by changes in natural mortality. When accounting for parameter uncertainty, however, the estimated relationship was either additive or only partially compensatory. Importantly, the data suggested that neither density nor unobserved heterogeneity (i.e. frailty) significantly contributed to changes in cougar survival and mortality in either study population, which could have clouded our conclusions regarding functional relationships between competing mortality risks (Koons et al., 2014a, b). Our most important finding was the observed uncertainty in the functional relationship between exploitation and natural mortality in both populations, which systematically led to biased conclusions when not accounted for, and could lead to the improper management of harvested carnivores.

Studies of compensation and additive mortality are typically focused on ungulate prey species to assess whether cougar predation is additive or not to other sources of mortality (e.g. Boyce, 1989; Bartmann et al., 1992; Bowyer et al., 2005; Hurley et al., 2011). Recently, a number of studies have attempted to address

Table 2
Cox proportional hazard model selection based on ΔAICc and AICc weights (w_i), where "+" and "∗" denote additive and interactive models, respectively, and *np* denotes the number of parameters.

Models	*np*	AICc	ΔAICc	w_i
Sex ∗ pop	3	1777.067	0	0.394
Sex ∗ pop + density	4	1779.037	1.97	0.147
Sex + pop	2	1779.414	2.347	0.122
Sex ∗ pop + pop ∗ density	5	1780.014	2.947	0.090
Sex + pop + age	3	1780.014	2.947	0.090
Sex ∗ age	3	1781.312	4.245	0.047
Sex	1	1781.366	4.299	0.046
Sex + age	2	1782.056	4.989	0.032
Sex + pop + age + density	4	1782.107	5.04	0.032
Age + pop	2	1815.894	38.827	0.000
Age	1	1816.741	39.674	0.000
Age ∗ pop	3	1817.012	39.945	0.000
Pop	1	1819.985	42.918	0.000
No effect	0	1820.609	43.542	0.000
Density	1	1821.69	44.623	0.000
Pop ∗ density	3	1821.808	44.741	0.000

Table 1
Sex-specific mean annual mortality estimates for the Oquirrh-Stansbury and Monroe populations. Estimates and 95% confidence intervals are presented for harvest mortality and all other causes of death, and for anthropogenic versus natural causes of death.

	Monroe females	95% Confidence interval	Monroe males	95% Confidence interval
Harvest mortality	0.1727	0.1231–0.2223	0.5327	0.4342–0.6311
Non-harvest mortality	0.1628	0.1139–0.2117	0.1679	0.0787–0.2572
Anthropogenic mortality	0.2613	0.2024–0.3202	0.6062	0.5117–0.7007
Natural mortality	0.0744	0.0373–0.1115	0.0957	0.0218–0.1696
	Oquirrh-Stansbury females	95% Confidence interval	Oquirrh-Stansbury males	95% Confidence interval
Harvest mortality	0.0677	0.0312–0.1042	0.2028	0.0966–0.3090
Non-harvest mortality	0.2644	0.1929–0.3358	0.2893	0.1622–0.4163
Anthropogenic mortality	0.1418	0.0825–0.2010	0.2165	0.1043–0.3288
Natural mortality	0.1868	0.1180–0.2556	0.2768	0.1475–0.4061

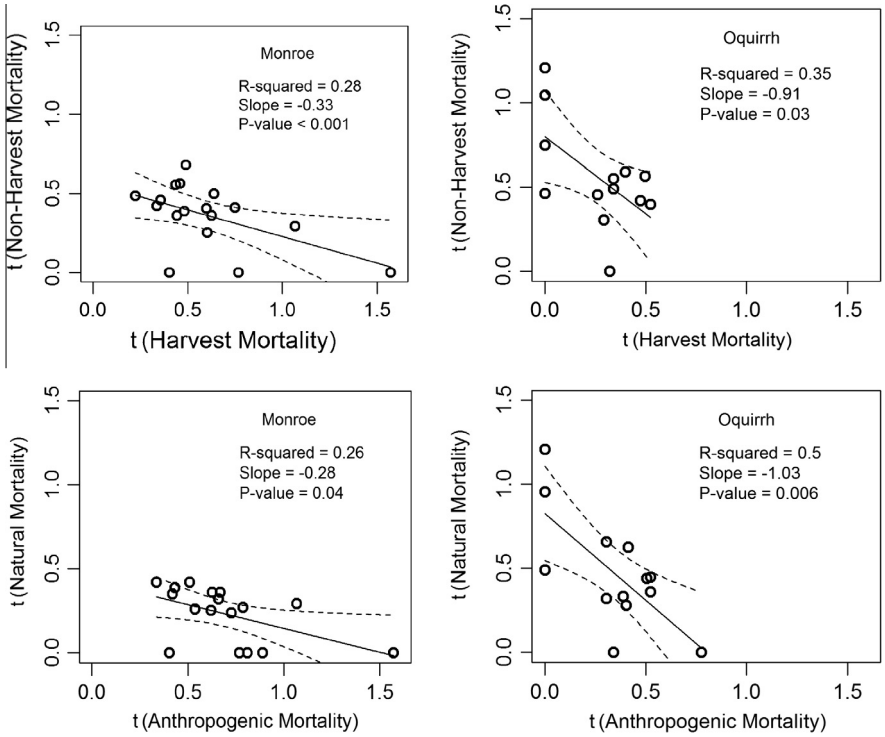

Fig. 4. Functional relationships between competing risks of mortality for the Monroe and Oquirrh Mountain study populations when uncertainty in annual estimates of cause-specific mortality was not accounted for, yielding inappropriately tight 90% confidence intervals (dashed lines) and potentially excessive type II error ('*t*' denotes the arcsine square root transformation).

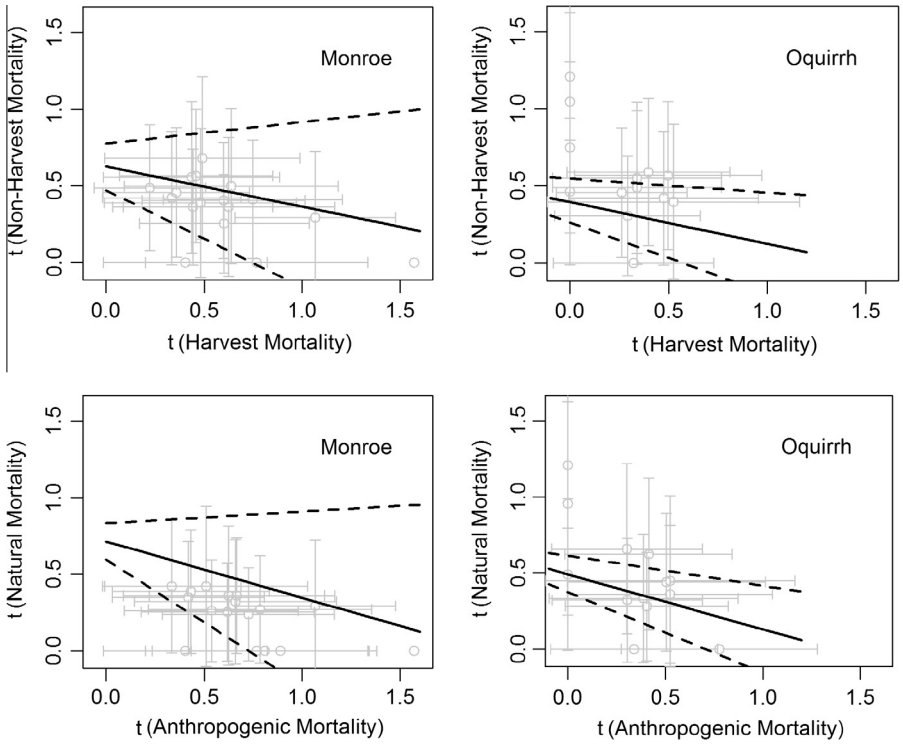

Fig. 5. Functional relationships between competing risks of mortality after uncertainty in mortality estimates was accounted for. Sets of mortality estimates were transformed using an arcsine square root transformation as symbolized by '*t*' (e.g. *t*(non-harvest mortality). Measures of uncertainty in annual estimates of cause-specific mortality analysis are shown by the error bars denoting 90% confidence intervals, and 90% confidence intervals for the estimated relationships between competing risks of mortality are denoted by the dashed lines, which were attained trough Monte Carlo simulations.

the same question by assessing the human–carnivore interaction as a predator–prey relationship (Cooley et al., 2009; Creel and Rotella, 2010; Sparkman et al., 2011; Robinson et al., 2014); yet, only one of these studies has addressed the problem of sampling variance in competing mortality risks (Murray et al., 2010). By adopting a similar approach to that of Murray et al. (2010) in cougars, a carnivore with a completely different social structure (i.e. solitary versus pack-living species), we were unable to reach definitive conclusions regarding the additive and compensatory mortality hypotheses when accounting for sampling variance in cause-specific mortality estimates.

Given parameter uncertainty (Lebreton, 2005), we found that lower rates of harvest allowed for partial compensation between anthropogenic and natural mortality in the quasi-protected Oquirrh study site where hunting pressure was less intense, which has also been concluded in other carnivore studies (e.g. Sterling et al., 1983; Sparkman et al., 2011). This population is situated within an area of high human population growth and residential development, and is therefore characterized by habitat loss and fragmentation (Stoner et al., 2006, 2013a), which can predispose individuals to increased anthropogenic threats. Given the role of highways as a mortality agent within the female segment of this population (10.7%), on top of harvest (17.9%), managers should account for additional causes of anthropogenic death (Quigley and Hornocker, 2009), and progressive land-use planning and public education in the future could be important to the conservation of cougars and their prey in this rapidly developing landscape (UDWR, 2011).

We could not reject the null hypothesis of additivity in the Monroe population once parameter uncertainty was accounted for, a finding that has been supported by other carnivore studies (Cooley et al., 2009; Creel and Rotella, 2010; Robinson et al., 2014). Indeed, Cooley et al. (2009) observed that although immigration compensated for hunting mortalities, they found like us that compensation via other vital rates (e.g. natural mortality) was not present. Recovery from harvest could depend on nearby source populations; justifying the need for cougar management at the scale of meta-populations, rather than single populations. Similarly, Robinson et al. (2014) estimated that total survival of adults and juveniles linearly declined as hunting mortality increased, and further observed that non-harvest mortality (i.e., illegal, natural, depredation, vehicle, and unknown) was lower in a non-hunted population when compared to a hunted population, with both findings supporting the additive mortality hypothesis. Although neither study accounted for uncertainty in parameter estimates, we echo the finding that "the compensatory mortality hypothesis may not be appropriate for modeling hunter harvest of cougars and other large carnivores that exhibit long-distance dispersal" (Cooley et al., 2009).

As expected in the heavily hunted Monroe population, a high proportion of cougars died of anthropogenic causes, especially harvest. A metapopulation approach to cougar management is often cited as the most promising since immigration and emigration may have the power to replenish harvested cougar populations (Stoner et al., 2006, 2013b; Cooley et al., 2009; Robinson et al., 2008, 2014). Immigration in response to vacated home ranges freed via the removal of harvested individuals could likely provide some additional grounds for population-level compensation (Cooley et al., 2009; Stoner et al., 2013a). Previous work indicated that the Monroe population exhibited sink-like mortality (i.e. low productivity and high immigration rates), whereby despite low kitten production, new animals immigrated every winter, primarily sub-adult males (Stoner et al., 2006). We caution that given the potential for additive mortality at high harvest rates in the Monroe population, over-exploitation may override the potential for the replenishment of this heavily harvested sink population, especially when considering that most of the potential source populations surrounding the site are exploited at similar levels (Stoner et al., 2013b). More recent data on cougar movement in and out of the study site would need to be collected to quantify influx (see Stoner et al., 2006, for data collected up to 2004) and the role immigration could play in maintaining stable dynamics in the Monroe population (e.g. Sweanor et al., 2000; Stoner et al., 2013b). Harvest mortality can also be compensated at the population level by density-dependent increases in reproduction (i.e. compensatory natality) (Boyce et al., 1999; Turgeon and Kramer, 2012; Robinson et al., 2014). Unlike similar studies on monogamous carnivores (Knowlton, 1972; Frank and Woodroffe, 2001), however, previous research on the Monroe study site indicated that compensatory reproduction did not occur with increased harvest pressure; conversely, per-capita reproductive output declined as harvest rates increased (Stoner et al., 2006). While social canids are able to reproductively compensate for increased exploitation, solitary felids are restricted by reproductive capacity and longer-term dependence of offspring.

When used as predictors of life history traits, fluctuating uncertainty in indices of abundance (i.e. sampling error) will attenuate the estimated slope of relationship toward 0, and thus conclusions of no effect. Thus, the estimated lack of density dependence in survival may have been due to a lack of density-dependence, or our inability to detect the effect given the index of density. A number of other recently developed estimators might also be useful for estimating the abundance of large carnivores such as cougars; e.g., mark-resight (McClintock and White, 2012) and genetic capture-reencounter extensions to the Lincoln estimator (Lukacs and Burnham, 2005), but these are often cost prohibitive or require capturing and handling a large number of individuals.

5. Conclusions

For species that occur in low densities and have large spatial requirements such as cougars, the sample sizes needed to adequately test the compensatory and additive mortality hypotheses cannot be attained at the spatial scale of hunting units commonly used in the West. If managers want to reach unambiguous conclusions regarding the degree to which harvest mortality is compensatory or additive, the spatial scale of harvest prescriptions may have to be calculated across larger regions than those currently in favor, and should preferentially encompass metapopulations rather than isolated populations, so that estimates of emigration and immigration can be attained. Further, relationships between competing mortality risks have proven difficult to estimate in long-lived carnivores due to challenges in determining known fates of large samples of cougars (Quigley and Hornocker, 2009). The functional form of these relationships, however, is fundamental to our understanding of carnivore population dynamics, and if appropriately quantified, could considerably improve sustainable management of cougars. Despite best attempts, the difficulty in capturing numerically rare and behaviorally elusive species with an affinity for rugged terrain resulted in small to moderate sample sizes of marked individuals each year (Stoner et al., 2006). Given the paucity of equivalently long and thorough carnivore demographic studies, we suspect that this constraint is likely widespread in the carnivore literature (but see Murray et al., 2010). Uncertainty is a natural part of all biological studies and should be acknowledged as such (Møller and Jennions, 2011). Considering our findings, we advocate that carnivore studies be designed to address and minimize this uncertainty. Both bootstrapping and Monte Carlo situations could be considered to address issues of uncertainty. Herein, we use Monte Carlo simulations because of

their versatility and possibility to sample from a statistical distribution rather than to rely on an empirical distribution (Caswell, 2001). For large sample size, the bootstrap is preferred, but for small to moderate sample size (our case), the bootstrap can yield biased estimates of uncertainty. Neither approach, however, accounts for sampling 'covariation' in the competing risks. Most likely, the use of a competing risk analysis that continually updates the 'at-risk' sample should minimize effects of sampling covariation on estimated compensation between competing risks (Cooch et al., 2014). In the future, Bayesian analyses could more directly account for both sampling variation and covariation when testing the additive and compensatory mortality hypotheses (Koons et al., 2014a, b). Collection of longitudinal data on large carnivores, over broader spatial scales, may also help overcome such limitations. Such has been the case in wildlife management of other taxa for decades, where the development of state-of-the-art statistical models to account for such uncertainty (e.g. Burnham and Anderson, 1984; Nichols et al., 1984; Schaub et al., 2004; Schaub, 2009; Servanty et al., 2010), has likely been driven by the availability of large samples of harvested individuals.

5.1. Management implications

Agencies responsible for cougar management face ongoing social and political pressure to implement harvest prescriptions that range from aggressively targeting predation impacts on native and domestic ungulate species, to offering near-complete protection, or providing trophy hunting opportunities (Anderson et al., 2009; Hurley et al., 2011). Given that we could not reject the additive mortality hypothesis at the Monroe study site, we recommend a conservative management approach be adopted to preclude potential over-harvest in future years. Similarly in the Oquirrh study site, even though exploitation was partially compensated by changes in natural mortality, land-use change is the prevailing factor potentially affecting this population, and as such, it is exposed to anthropogenic pressures beyond sport harvest, and therefore we suggest that managers monitor all anthropogenic causes of death. Further, our results underscore the value of long-term data sets and suggest the possibility of expanding the scope of such comparisons to additional management units. Because the Oquirrh-Stansbury and Monroe management units were subjected to contrasting mortality regimes, results herein could potentially be expanded to additional management units with similar environmental, jurisdictional, or land-use characteristics.

Acknowledgements

We would like to thank K. Bunnell, B. Blackwell, B. Bates, C. McLaughlin, J. Shivik, A. Clark, and T. Becker from the Utah Division of Wildlife Resources; K. Rasmussen from the Fishlake National Forest; J. Crane, D. Johnson, and R. Dunton from the Utah Army National Guard; and to W. Adams, and A. Neville from the Kennecott Utah Copper Corporation. This work relies entirely on the dedication and hard work of the following outstanding houndsmen, pilots and technicians: C. Mecham, M. Mecham, C. Shaffer, C. Hunt, S. Biggs, and D. Mitchell, D. Ranglack, and C. Juran without whom data collection would not have been possible. Special thanks to R. Skirpstunas for post-mortem examinations. Funding and logistical was support provided by the Utah Division of Wildlife Resources and the United State Department of Agriculture, Wildlife Services, and the National Wildlife Research Center. DS was supported by a NASA Biodiversity and Ecological Forecasting grant (no. NNH10ZDA001N) during the writing phase of this project.

Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.biocon.2014.12.008.

References

Akaike, H., 1973. Information theory and an extension of the maximum likelihood principle. In: Petran, B.N., Csaki, D.F. (Eds.), International Symposium on Information Theory. Akadéniai Kiadi, Budapest, Hungary, pp. 267–281.
Anderson, D.R., Burnham, K.P., 1976. Mathematical models for nonparametric inferences from line transect data. Biometrics, 325–336.
Anderson, D.R., Burnham, K.P., 1981. Bobwhite population responses to exploitation: two problems. J. Wildl. Manage. 45, 1052–1054.
Anderson, C.R., Lindzey, F.G., Knopff, K.H., Jalkotzy, M.G., Boyce, M.S., 2009. Cougar management in North America. In: Hornocker, M., Negri, S. (Eds.), Cougar: Ecology and Conservation. The University of Chicago Press, Chicago, Illinois, USA, pp. 41–54.
Ashman, D.L., Christensen, G.C., Hess, M.L., Tsukamoto, G.K., Wickersham, M.S., 1983. The mountain lion in Nevada. Nevada Department of Wildlife, Carson City, USA.
Aubry, L.M., Cam, E., Koons, D.N., Monnat, J.Y., Pavard, S., 2011. Drivers of age-specific survival in a long-lived seabird: contributions of observed and hidden sources of heterogeneity. J. Anim. Ecol. 80 (2), 375–383.
Bartmann, R.M., White, G.C., Carpenter, L.H., 1992. Compensatory mortality in a Colorado mule deer population. Wildl. Monogr. 121, 1–39.
Bowyer, R.T., Person, D.K., Pierce, B.M., 2005. Detecting top down versus bottom up regulation of ungulates by large carnivores: implications for conservation of biodiversity. In: Ray, J.C., Redford, K.H., Steneck, R.S., Berger, J. (Eds.), Large Carnivores and the Conservation of Biodiversity. Island, Covelo, California, USA, pp. 342–361.
Boyce, M.S., 1989. The Jackson Elk Herd: Intensive Wildlife Management in North America. Cambridge University Press, Cambridge, UK.
Boyce, M.S., Sinclair, A.R.E., White, G.C., 1999. Seasonal compensation of predation and harvesting. Oikos 87, 419–426.
Bradburn, M.G., Clark, T.G., Love, S.B., Altman, D.G., 2003. Survival analysis Part II: multivariate data analysis: an introduction to concepts and methods. Br. J. Cancer 89, 431–436.
Burnham, K.P., Anderson, D.R., 1984. Tests of compensatory vs. additive hypotheses of mortality in mallards. Ecology 65, 105–112.
Burnham, K.P., Anderson, D.R., 2002. Model Selection and Multimodel Inference: A Practical Information-theoretic Approach, second ed. Springer-Verlag, New York, USA.
Caswell, H., 2001. Matrix Population Models: Construction, Analysis and Interpretation, second ed. Sinauer Associates, Sunderland, Massachusetts.
Caughley, G., 1977. Analysis of Vertebrate Populations. John Wiley and Sons, New York, New York, USA.
Chambers, J.M., 1992. In: Chambers, J.M., Hastie, T.J. (Eds.), Statistical Models in S. Wadsworth and Brooks/Cole, California, USA, pp. 249–307.
Choate, David M., Wolfe, Michael L., Stoner, D.C., 2006. Evaluation of cougar population estimators in Utah. Wildl. Soc. Bull. 34 (3), 782–799.
Cooch, E.G., Guillemain, M., Boomer, G.S., Lebreton, J.-D., Nichols, J.D., 2014. Effects of harvest on waterfowl populations. Wildfowl (Special Issue No. 4), 220–276.
Cooley, H.S., Wielgus, R.B., Koehler, G.M., Maletzke, B.T., 2009. Does hunting regulate cougar populations? A test of the compensatory mortality hypothesis. Ecology 90, 2913–2921.
Cooley, H.S., Bunnell, K.D., Stoner, D.C., Wolfe, M.L., 2011. Population management: cougar hunting. In: Jenks, J.A. (Ed.), Managing Cougars in North America. Jack H. Berryman Institute, Logan, Utah, USA, pp. 111–133.
Cox, D.R., 1972. Regression models and life-tables. J. Roy. Stat. Soc. Biol. Ser. 34 (2), 187–220.
Creel, S., Rotella, J., 2010. Meta-analysis of relationships between human offtake, total mortality and population dynamics of gray wolves (*Canis lupus*). PLoS One 5 (9), e12918.
Efron, B., Tibshirani, R.J., 1994. An Introduction to the Bootstrap, vol. 57. CRC Press.
Errington, Paul L., Hamerstrom Jr., F.N., 1935. Bob-white winter survival on experimentally shot and unshot areas. Iowa State College J. Sci. 9, 625–639.
Festa-Bianchet, M., 2003. Exploitative wildlife management as a selective pressure for the life-history evolution of large mammals. In: Festa-Bianchet, M., Apollonio, M. (Eds.), Animal Behavior and Wildlife Conservation. Island Press, pp. 191–208.
Frank, L.G., Woodroffe, R., 2001. Behaviour of carnivores in exploited and controlled populations. Conserv. Biol. Ser. Cambridge, 419–442.
Gill, R.B., 2009. To save a mountain lion: evolving philosophy of nature and cougars. In: Hornocker, M., Negri, S. (Eds.), Cougar: Ecology and Conservation. The University of Chicago Press, Chicago, Illinois, USA, pp. 5–16.
Heisey, D.M., Fuller, T.K., 1985. Evaluation of survival and cause-specific mortality rate using telemetry data. J. Wildl. Manage. 49, 668–674.
Heisey, D.M., Patterson, B.R., 2006. A review of methods to estimate cause-specific mortality in presence of competing risks. J. Wildl. Manage. 70, 1544–1555.
Hurley, M. et al., 2011. Demographic response of mule deer to experimental reduction of coyotes and mountain lions in Southeastern Idaho. Wildl. Monogr. 178, 1–33.

Kaplan, E.L., Meier, P., 1958. Nonparametric estimation from incomplete observations. J. Am. Stat. Assoc. 53, 457–481.
Keefover-Ring, W.J., 2005. Mountain lions, myths and media: a critical reevaluation of the Beast in the Garden. Environ. Law 35, 1083–1103.
Kleinbaum, D.G., Klein, M., 2005. Survival Analysis: A Self-learning Approach, second ed. Springer, New York, USA.
Knowlton, F.F., 1972. Preliminary interpretations of coyote population mechanics with some management implications. J. Wildl. Manage., 369–382.
Knowlton, F.F., Gese, E.M., Jaeger, M.M., 1999. Coyote depredation control: an interface between biology and management. J. Range Manage., 398–412.
Koons, D.N., Rockwell, R.F., Aubry, L.M., 2014a. Effects of exploitation on an overabundant species: the lesser snow goose predicament. J. Anim. Ecol. 83 (2), 365–374.
Koons, D.N., Gamelon, M., Gaillard, J.M., Aubry, L.M., Rockwell, R.F., Gimenez, O., 2014b. Methods for studying cause-specific senescence in the wild. Meth. Ecol. and Evol. 5 (9), 924–993.
Kroese, D.P., Taimre, T., Botev, Z.I., 2011. Handbook of Monte Carlo Methods, vol. 706. John Wiley and Sons.
Laundré, J.W., Hernández, L., 2002. Growth curve models and age estimation of young cougars in the northern Great Basin. J. Wildl. Manage. 66, 849–858.
Lebreton, J.-D., 2005. Dynamical and statistical models for exploited populations. Aust. N.Z. J. Stat. 47 (1), 49–63.
Lindberg, Mark S., Sedinger, James S., Lebreton, Jean-Dominique, 2013. Individual heterogeneity in Black Brant survival and recruitment with implications for harvest dynamics. Ecol. Evol. 3 (12), 4045–4056.
Lindzey, F.G., Van Sickle, M.D., Laing, S.P., Mecham, C.S., 1992. Cougar population response to manipulation in southern Utah. Wildl. Soc. Bull. 20, 224–227.
Lindzey, F.G., Vansickle, W.D., Ackerman, B.B., Barnhurst, D., Hemker, T.P., Laing, S.P., 1994. Cougar population dynamics in southern Utah. J. Wildl. Manage. 58, 619–623.
Lukacs, P.M., Burnham, K.P., 2005. Review of capture–recapture methods applicable to noninvasive genetic sampling. Mol. Ecol. 14 (13), 3909–3919.
McClintock, B.T., White, G.C., 2012. From NOREMARK to MARK: software for estimating demographic parameters using mark-resight methodology. J. Ornithol. 152 (2), 641–650.
Mech, L.D., 1983. Handbook of Animal Radio-tracking. University of Minnesota Press, USA.
Møller, A.P., Jennions, M.D., 2011. Testing and adjusting for publication bias. Trends Ecol. Evol. 16 (10), 580–586.
Murray, D.L., Patterson, B.R., 2006. Wildlife survival estimation: recent advances and future directions. J. Wildl. Manage. 70, 1499–1503.
Murray, D.L., Smith, D.W., Bangs, E.E., Mack, C., Oakleaf, J.K., Fontaine, J., Boyd, D., Jiminez, M., Niemeyer, C., Meier, T.J., Stahler, D., Holyan, J., Asher, V.J., 2010. Death from anthropogenic causes is partially compensatory in recovering wolf populations. Biol. Conserv. 143, 2514–2524.
Nichols, J.D., Hines, J.E., 1987. Population Ecology of the Mallard: Winter Distribution Patterns and Survival Rates of Winter-banded Mallards. US Department of the Interior, Fish and Wildlife Service.
Nichols, J.D., Conroy, M.J., Anderson, D.R., Burnham, K.P., 1984. Compensatory mortality in waterfowl populations: a review of the evidence and implications for research and management. In: Transactions of North American Wildlife and Natural Resources Conference, vol. 49, pp. 535–554.
Péron, G., 2013. Compensation and additivity of anthropogenic mortality: life-history effects and review of methods. J. Anim. Ecol. 82 (2), 408–417.
Pierce, B.M., Bleich, V.C., 2003. Mountain lion. In: Feldhamer, G.A., Thompson, B.C., Chapman, J.A. (Eds.), Wild Mammals of North America: Biology, Management, and Conservation, second ed. The Johns Hopkins University, Baltimore, Maryland, USA, pp. 744–757.
Quigley, H., Hornocker, M., 2009. Cougar population dynamics. In: Hornocker, M., Negri, S. (Eds.), Cougar: Ecology and Conservation. The University of Chicago Press, Chicago, Illinois, USA, pp. 59–75.
R Development Core Team, 2012. R: A Language and Environment for Statistical Computing. R Foundation for Statistical Computing, Vienna, Austria.
Robinson, H.S., Wielgus, R.B., Cooley, H.S., Cooley, S.W., 2008. Sink populations in carnivore management: cougar demography and immigration in a hunted population. Ecol. Appl. 18 (4), 1028–1037.
Robinson, H.S., Desimone, R., Hartway, C., Gude, J.A., Thompson, M.J., Mitchell, M.S., Hebblewhite, M., 2014. A test of the compensatory mortality hypothesis in mountain lions: a management experiment in West-Central Montana. J. Wildl. Manage. 78 (5), 791–807.
Runge, M.C., Johnson, F.A., 2002. The importance of functional form in optimal control solutions of problems in population dynamics. Ecology 83 (5), 1357–1371.
Sandercock, B.K., Nilsen, E.B., Brøseth, H., Pedersen, H.C., 2011. Is hunting mortality additive or compensatory to natural mortality? Effects of experimental harvest on the survival and cause-specific mortality of Willow Ptarmigan. J. Anim. Ecol. 80, 244–258.
Sargeant, G.A., 2011. Wild1: R Tools for Wildlife Research and Management.
Schaub, M., 2009. Evaluation of bias, precision and accuracy of mortality cause proportion estimators from ring recovery data. In: Modeling Demographic Processes in Marked Populations. Springer, USA, pp. 1081–1097.
Schaub, M., Pradel, R., Lebreton, J.-D., 2004. Is the reintroduced white stork (*Ciconia ciconia*) population in Switzerland self-sustainable? Biol. Conserv. 119 (1), 105–114.
Sedinger, J.S., White, G.C., Espinosa, S., Partee, E.T., Braun, C.E., 2010. Assessing compensatory versus additive harvest mortality: an example using greater sage-grouse. J. Wildl. Manage. 74, 326–332.
Servanty, S., Choquet, R., Baubet, E., Brandt, S., Gaillard, J.-M., Schaub, M., Toïgo, C., Lebreton, J.-D., Buoro, M., Gimenez, O., 2010. Assessing whether mortality is additive using marked animals: a Bayesian state-space modeling approach. Ecology 91, 1916–1923.
Sinclair, A.R.E., Pech, R.P., 1996. Density dependence, stochasticity, compensation, and predator regulation. Oikos 75, 164–173.
Sparkman, Amanda M., Waits, Lisette P., Murray, Dennis L., 2011. Social and demographic effects of anthropogenic mortality: a test of the compensatory mortality hypothesis in the red wolf. PLoS One 6 (6), e20868.
Sterling, B., Conley, W., Conley, M.R., 1983. Simulations of demographic compensation in coyote populations. J. Wildl. Manage., 1177–1181.
Stoner, D.C., Wolfe, M.L., Choate, D.M., 2006. Cougar exploitation levels in Utah: implications for demographic structure, population recovery, and metapopulation dynamics. J. Wildl. Manage. 70 (6), 1588–1600.
Stoner, D.C., Wolfe, M.L., Mecham, C., Mecham, M.B., Durham, S.L., Choate, D.M., 2013a. Dispersal behaviour of a polygynous carnivore: do cougars Puma concolor follow source-sink predictions? Wildl. Biol. 19, 289–301.
Stoner, D.C., Wolfe, M.L., Reith, W.R., Bunnell, K.D., Durham, S.L., Stoner, L.A., 2013b. De facto refugia, ecological traps and the biogeography of anthropogenic cougar mortality in Utah. Divers. Distrib. 19, 1114–1124.
Sweanor, L.L., Logan, K.A., Hornocker, M.G., 2000. Cougar dispersal patterns, metapopulation dynamics, and conservation. Conserv. Biol. 14 (3), 798–808.
Therneau, T., 2012. Mixed Effects Cox Models. R vignette for coxme package. <http://cran.r-project.org/web/packages/coxme/vignettes/coxme.pdf>.
Therneau, T., Grambsch, P., 2000. Modeling Survival Data: Extending the Cox Model. Springer-Verlag, New York, USA.
Turgeon, Katrine, Kramer, Donald L., 2012. Compensatory immigration depends on adjacent population size and habitat quality but not on landscape connectivity. J. Anim. Ecol. 81 (6), 1161–1170.
Utah Division of Wildlife Resources, 2011. Utah Cougar Management Plan V.2.1. Utah Division of Wildlife Resources, Salt Lake City, Utah, pp. 1–27.
Vaupel, J.W., Yashin, A.I., 1985. Heterogeneity's ruses: some surprising effects of selection on population dynamics. Am. Stat. 39, 176–185.
Wilson, A.J., Nussey, D.H., 2010. What is individual quality? An evolutionary perspective. Trends Ecol. Evol. 25 (4), 207–214.