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Death from anthropogenic causes is partially compensatory in recovering wolf populations

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ABSTRACT

There is substantial interest in how mortality rates affect animal populations, but mechanisms explaining when and under what circumstances particular causes of death incur demographic responses are far from clear. In theory, small or expanding populations should experience additive mortality from anthropogenic causes of death, but whether such effects are homogenous across a population or expressed only in certain high-risk individuals is open for debate. We used competing risks models to analyze mortality patterns among radio-collared wolves (*Canis lupus*, $n = 711$) from three populations in northwestern United States (1982–2004), and evaluated the degree to which anthropogenic mortality was additive vs. compensatory to natural demographic processes. Almost 80% ($n = 320$) of wolves dying of known fates were killed by anthropogenic causes (legal control, illegal killing, harvest in Canada, vehicle collision), and additive effects of anthropogenic mortality were most pronounced in northwestern Montana where wolf exposure to humans and livestock was high compared to either the Greater Yellowstone Area or central Idaho, where anthropogenic risk was lower. In contrast, risk from natural hazards was lower in northwestern Montana than in the other areas, implying some degree of compensatory mortality from anthropogenic risk. Animals recruited to the study following human–wolf conflict had markedly higher anthropogenic risk than those recruited for standard monitoring purposes, and juvenile wolves as well as dispersers, succumbed to higher anthropogenic risk. Multivariate models revealed that increasing wolf population density promoted higher anthropogenic risk and reduced natural risk, indicating that partially-compensatory effects of anthropogenic mortality actually became increasingly additive with population density. The observed compensatory mortality and hazard heterogeneity in our study implies that demographic responses to mortality risk may be complex and more subtle than previously thought; the density-dependent effect of anthropogenic mortality portends a stabilizing influence of humans on recovering wolf populations. We conclude that future assessment of the role of anthropogenic mortality should include individual-based hazard estimation as a complement to traditional population-level approaches.

1. Introduction

There exists longstanding interest in patterns of survival and mortality governing animal populations, and how the specific cause and rate of death events can influence numerical change and population persistence (Burnham and Anderson, 1984; Cooley et al., 2009). However, our understanding of the processes by which sources of mortality incur demographic change, or the mechanisms allowing mortality agents to incur differential effects on individuals, remains surprisingly limited. To-date, work undertaken in this area has centered on the influence of human harvest

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on population-level mortality, with three main hypotheses emerging to explain observed mortality patterns. First, harvest mortality may operate strictly additively to natural mortality such that total mortality increases density-independently and correspondingly with changes in either mortality source (Burnham and Anderson, 1984). Second, populations may respond to increased harvest through compensatory decline in natural mortality, thereby leading to consistent total mortality irrespective of harvest rate or population density (Burnham et al., 1984). A more realistic variant of the above models invokes partial compensation depending on factors such as population density, harvest intensity, demographic groups subject to mortality, and adaptive responses manifest through altered life history (Boyce et al., 1999). The partial compensation model is distinguished by a total mortality rate that increases with harvest, but not in direct proportion to harvest mortality.

Although the above models are helpful in considering the range of potential effects of harvest mortality on animal populations, to date none has received consistent and widespread empirical support (see Johnson et al., 2002; Pöysä et al., 2004). This limitation is related to the reliance on indirect means of mortality estimation (i.e., apparent survival via mark-recapture studies) and assessment of population response when evaluating harvest impacts (e.g., Nichols et al., 1984; Schaub and Lebreton, 2004). Further, most empirical studies of harvest responses are conducted using short-lived recruitment-driven waterfowl or gamebird species, where limited individual heterogeneity is assumed (e.g., Ellison, 1991; Smith and Reynolds, 1992). This is an unrealistic scenario for many species having complex life cycles and age-variable mortality (Conroy and Krementz, 1990), and highlights the need to complement population-level studies of mortality with those focusing on individual hazards. Also, because most populations are exposed to risk from several causes of death, each potentially having a different functional role on individuals and populations, research focusing exclusively on harvest provides limited insight in terms of more general questions related to mortality risk determinants. Indeed, because human harvest tends to be density-dependent whereas other sources of mortality can be density-dependent or density-independent (e.g., see Sinclair et al., 2008), we may predict fundamentally different effects across the range of potential causes of death (see Sinclair and Pech, 1996). It follows that our current understanding of the role of mortality in animal population ecology may in fact be incomplete and even biased, especially among populations that are not exposed to legal harvest or those occurring at densities below levels of resource limitation and density-dependent mortality.

Quantitative analysis of competing mortality risks in ecology has languished despite the availability and relevance of sophisticated methods developed in epidemiology and actuarial sciences (see Therneau and Grambsch, 2000; Collett, 2003). Such methods are logically well-suited for addressing questions pertaining to additive vs. compensatory mortality in animals (Heisey and Patterson, 2006). We applied proportional risks models to three recovering wolf (*Canis lupus*) populations in the western United States, to assess the relationship between proximate cause of death and cause-specific mortality risk. The wolf populations we studied experienced low levels of human harvest and were subject to known fates and precise death timing during the study, thereby distinguishing them from most other systems where additive vs. compensatory processes have been studied. Further, wolves have a survival-driven life history compared to the recruitment-driven strategy of most harvested species, thereby making them less prone to compensatory processes especially at population densities below carrying capacity (e.g., Conroy and Krementz, 1990; Boyce et al., 1999). Accordingly, we predicted that anthropogenic hazard due to control actions or poaching would be additive to natural mortality and that the magnitude of anthropogenic mortality risk should be similar across demographic groups because such hazard should be largely non-discriminatory; it follows that the additive role of anthropogenic risk should be inversely density-dependent (depensatory) as populations become increasingly regulated by natural (density-dependent) factors. Surprisingly, our results largely fail to support the above predictions and thereby challenge traditional population-level assessment of cause-specific mortality by highlighting the need to include analyses of individual risk when assessing additive vs. compensatory mortality processes.

2. Methods

2.1. Study area

We studied wolves in three protected populations in the western US (see Oakleaf et al., 2006, Fig. 1). Each study area included core protected habitat where wolves had low risk of anthropogenic mortality surrounded by broader areas where exposure to humans was higher (US Fish and Wildlife Service et al., 2008). The northwestern Montana (MT) study area included Bob Marshall, Great Bear and Scapegoat wilderness areas to the west and south of Glacier National Park, the Greater Yellowstone Area (GYA) comprised Yellowstone National Park and Grand Teton National Park as well as surrounding areas, whereas the central Idaho area (ID) included Frank Church River of No-Return, Selway–Bitterroot and Gospel Hump wilderness areas (US Fish and Wildlife Service, 1994; Bangs et al., 1998). The majority of wolf pack territories occurred within study areas (e.g., 2006: 98.3% of territories within study areas, $n = 172$ packs; 2008: 98.4%, $n = 190$ packs, Oakleaf, unpublished data), allowing us to consider that, as defined, the study areas were appropriate spatial units for wolf population density estimation (see below). Throughout the study, wolf density was low in MT compared to density in the remainder of the region (Bangs, unpublished data).

2.2. Field methods

In MT, wolves were radio-collared starting in 1982 and leghold trapping constituted the main method for recruiting animals to the study. In GYA and ID, wolves were initially transplanted from Alberta in 1995 and British Columbia in 1996 and were released wearing radio-collars; those added to the study through the years were recruited mostly via helicopter darting. Pups <6 months usually were too small to be radio-collared, and in general only 1–2 wolves per pack were equipped with collars at any given time. Overall, the composition of animals recruited to the study was comparable by gender (% female: GYA: 49.3%, $n = 299$; ID: 43.9%, $n = 219$; MT: 56.3%, $n = 193$), adult age class (% adult: GYA: 28.1%, $n = 299$; ID: 32.7%, $n = 219$; MT: 31.7%, $n = 193$), and alpha status (% alpha: GYA: 9.3%, $n = 299$; ID: 14.1%, $n = 219$; MT: 11.5%, $n = 193$). There were more pups recruited in GYA (% pups: GYA: 51.5%, $n = 299$; ID: 30.0%, $n = 219$; MT: 37.4%, $n = 193$) and more dispersers in ID (% dispersers: GYA: 7.3%, $n = 299$; ID: 22.0%, $n = 299$; MT: 7.7%, $n = 193$); these demographic attributes were addressed explicitly in the context of competing risks analyses (see below). See Smith et al. (in press) for additional details on capture efforts and subject composition in the three study areas. Wolf population size increased continuously following release in GYA and ID whereas numbers in MT were largely stationary by the end of our study (2004) but continued to increase thereafter (Fig. 2).

All radio-collars were VHF and included a mortality switch that allowed assessment of survival time and cause of death. Wolf monitoring intensity averaged <14 days, and because dead animals were recovered and cause of death was determined promptly from

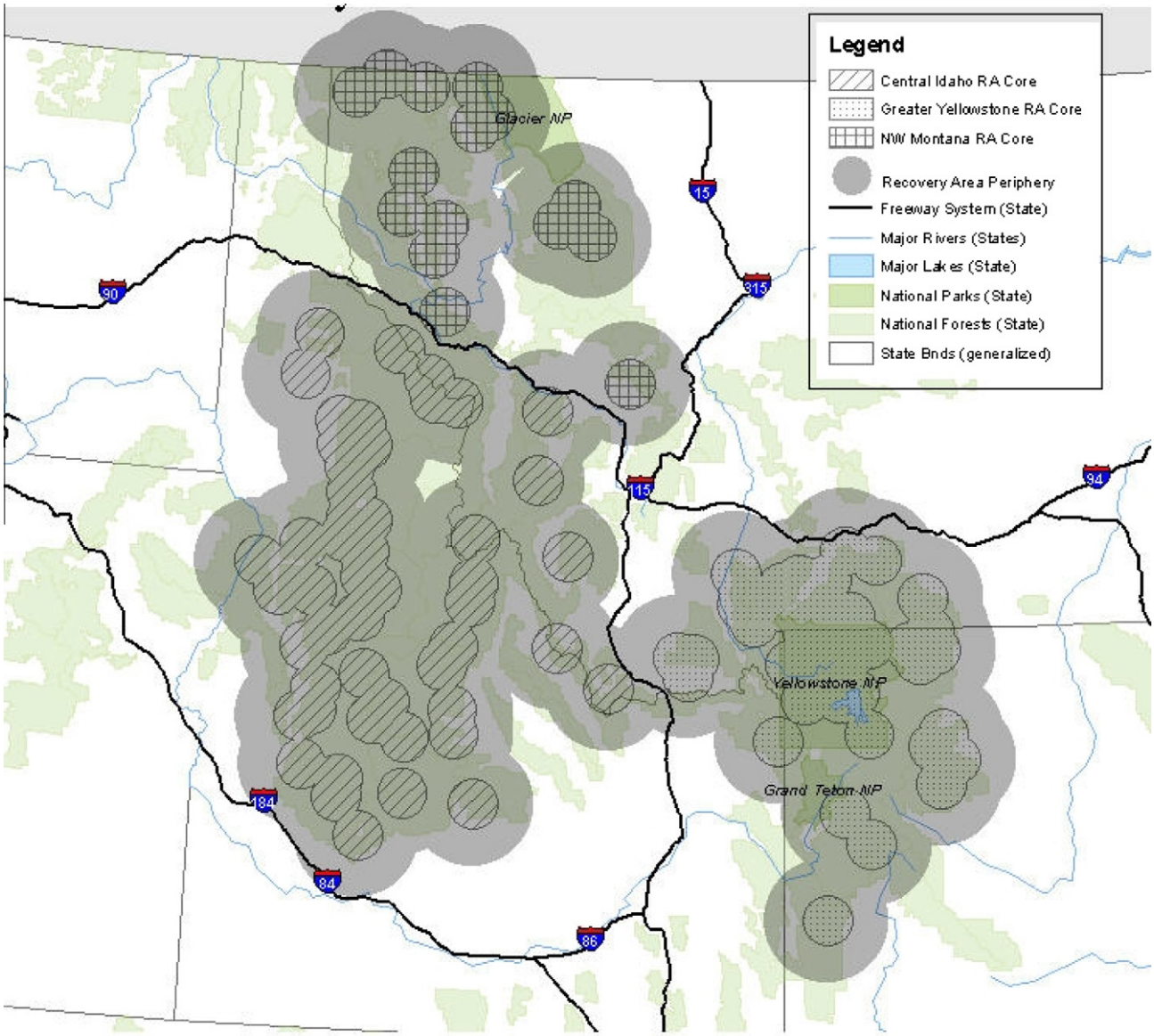

Fig. 1. Locations of primary wolf packs in western United States (1995–2004). Hatched circles represent pack locations in 2004 (final year of present study) and the grey area represents the designated recovery area at that time. For additional detail, see Oakleaf et al. (2006).

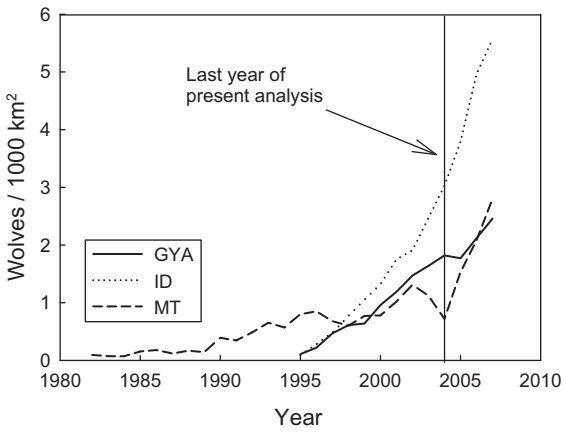

Fig. 2. Population density of wolves in three occupation areas in western United States (1982–2007). Population estimates are from US Fish and Wildlife Service et al. (2008).

evidence at the site, survival data were sufficiently precise to apply continuous-time analysis methods (see Murray, 2006). We pooled deaths from human control, illegal activities (e.g., shooting), legal harvest in Canada (MT only), vehicle collisions, and other human-related causes into 'anthropogenic' deaths, whereas those due to old age, disease or natural accidents, intraspecific strife and interspecific killing were considered 'natural'. Both sources of mortality should act in a largely density-independent manner in low-expanding populations, and thus anthropogenic mortality should be additive. Additional mortalities were due to 'unknown' causes, which included animals having insufficient evidence at the mortality site to allow precise cause of death assessment. Wolves that either died of capture-related causes or whose radio-signal was lost (e.g., transmitter failure, collar loss, emigration from the study area) were right-censored at their final monitoring date. Wolves that survived until the end of the study (December 31, 2004) were right-censored. Note that acquisition and collation of our extensive dataset presented significant logistical challenges such that data post-2004 were not available for the

present analysis. However, our dataset probably constitutes the most extensive to-date for assessment of cause-specific hazard determinants, and inclusion of more recent data was not necessary to address the hypothesis of additive risk at low population density.

2.3. Competing risk analysis

We used a competing risk framework to assess cause-specific hazard, based on the data augmentation method proposed by Lunn and McNeil (1995). Cause-specific risk analysis is an extension of standard survival analysis except that the survival function now 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 t from cause k. Because risk types are mutually exclusive, cause-specific mortality probabilities sum to the total mortality probability, $M(t)$;

$$M(t) = \sum_{k=1}^{Q} M_k(t),$$

where Q is the total number of causes of death. The data augmentation approach for cause-specific mortality analysis takes advantage of the additivity of hazard functions, and the dataset is duplicated Q times (one for each k) and a dummy code assigns each risk set to a specific k. Within each risk set, failure (death) is identified only for the appropriate risk type (Lunn and McNeil, 1995).

Overall, in our study unknown mortalities were the least common of the three broader causes of death and only comprised 11.8% ($n = 363$) of total mortalities. We sought to compare hazards from anthropogenic vs. natural causes of death (i.e., $Q = 2$), but could not assign 'unknown' mortalities to a specific risk type and therefore needed to ascertain that animals succumbing to unknown causes were representative of other sources of mortality. We conducted diagnostic tests similar to those described by Murray (2006) for detecting informative censoring; this analysis failed to identify any of seven independent variables (sex, adult status, yearling status, pup status, dispersal status, % private land in home range, cattle density in home range) that could differentiate unknown from other mortalities (Murray, unpublished). We also conducted exploratory competing risks analysis that revealed distinct hazard ratios between each risk set (anthropogenic: 4.15 (3.33, 5.17) (95% CI); natural: 0.52 (0.40, 0.67); unknown: 0.27 (0.19, 0.36)). When the analysis was restricted to two schemes for $Q = 2$ (i.e., pooled anthropogenic and unknown deaths vs. natural deaths; pooled natural and unknown deaths vs. anthropogenic deaths), 95% CI failed to overlap among hazard ratios including the unknown risk type, whereas when unknown deaths were excluded altogether hazards for the new risk type covariate overlapped with pooled rates (see below). This implied that unknown mortality could not safely be pooled with either risk set and that such mortality likely included animals dying of both natural and anthropogenic causes (see Collett, 2003). Accordingly, our analysis of anthropogenic vs. natural risk was implemented by coding RISKTYPE for $Q = 2$, with mortalities from unknown causes being censored. However, it is important to note that companion analyses using $Q = 3$ failed to reveal qualitatively different results from those presented herein (Murray, unpublished), implying that our findings for $Q = 2$ are robust to mortality classification scheme.

Competing risk analysis can be conducted using standard Cox proportional hazards modeling (or its counting process analogue for discontinuous timelines, Andersen–Gill (AG) modeling, see Therneau and Grambsch, 2000). It is simplest to consider a cause-specific hazard function ($h_i(t)$) for individual i to be associated with a covariate vector $x_I = (x_{i1}, x_{i2}, \ldots, x_{ip})$, with baseline hazard ($h_0(t)$) corresponding to the hazard function of an individual with covariate vector $x_I = (0, 0, \ldots, 0)$. Hazard functions are unspecified, but $h_i(t)$ and $h_j(t)$ differ only because $x_{i1} = x_{j1} + 1$, such that the hazard ratio, $h_i(t)/h_j(t) = \exp(\beta_1)$ is time-independent and $h_i(t)$ and $h_j(t)$ are proportional through time and differ only multiplicatively. Thus, the approach is semi-parametric because it only assumes hazard proportionality. Through evaluating covariate interaction with risk type, the method serves to compare cause-specific hazard, and by extension, additive vs. compensatory mortality (Heisey and Patterson, 2006).

The assumption of proportional hazards is crucial to hazard model fit, and model validity was assessed by calculating Schoenfeld residuals and computing a chi-square test to assess correlation (Therneau and Grambsch, 2000). We tested for hazard proportionality using the RISKTYPE variable; a univariate model with RISKTYPE satisfied the proportional hazards assumption ($\chi^2_1 = 0.38$, $P = 0.54$) implying that proper model structure should include RISKTYPE as covariate rather than stratum (i.e., see Method A, Lunn and McNeil, 1995). Our previous analysis (Smith et al., in press) showed that the method by which wolves were recruited to the study influenced hazard, with animals recruited as part of standard monitoring efforts (representative sampling) having lower mortality risk than those recruited following problems with humans (targeted sampling). Overall, the majority of animals in each study area were recruited via standard monitoring efforts (% recruitment via monitoring: GYA: 88.9%, $n = 299$; ID: 79.7%, $n = 219$; MT: 72.1%, $n = 193$). RECRUITMENT was constant through time when RISKTYPE also was considered ($\chi^2_2 = 0.44$, $P = 0.80$). After having first quantified the role of recruitment on risk, we segregate later analyses according to RECRUITMENT in recognition of differences in the composition of either group. Proportional hazards tests were conducted for all models but not reported unless significant. All hazard models were clustered by individuals, and based on previous analyses (Smith et al., in press) subjects were appropriately considered as independent.

2.4. Additive vs. compensatory mortality

Our previous analysis revealed higher overall mortality risk among wolves in the MT study area (Smith et al., in press), presumably due to higher exposure to anthropogenic sources of mortality. To assess the prediction that anthropogenic hazard is higher in MT, we used a dummy variable to isolate MT from other recovery areas; interaction between MT and RISKTYPE evaluated differential influence of this area on cause-specific hazard. We assessed the prediction that total mortality would be higher where protection was low by looking for main effects of MT on overall hazard. Possible compensatory effects of anthropogenic mortality on natural mortality rates were determined by decomposing the MT * RISKTYPE interaction specifically for natural mortality risk. Where appropriate, we restrict the sample of animals from the MT recovery area to 1995–2004 to be consistent with timelines from the other study areas.

We further evaluated additive risk by including covariates in competing risk models that decomposed RISKTYPE through interactions with other variables (with each covariate and RISKTYPE also included as main effects; see Lunn and McNeil, 1995). Again, main covariate effects assessed change in total mortality whereas decomposed interaction terms represented cause-specific risks. We selected covariates *a priori* on the basis of their potential additivity relative to anthropogenic hazards (see Smith et al., in press). The covariates addressed wolf demographic status (ADULT, YEARLING, PUP), behaviour (DISPERSER), and home range attributes (PRIVATE, CATTLE); each variable was considered time-dependent (but not time-varying) and updated seasonally or annually (Table 1). The PRIVATE and CATTLE variables were restricted to a subset of animals ($n = 297$; 41.8% of total sample) with known home

Table 1
Description of independent variables used in competing risks analysis of wolves in northwestern United States (1982–2004).

Variable	Description and coding system	Coding	Time-dependent	Time scale
RECRUITMENT	Representative or targeted method by which animal recruited to study	Dummy (1 = representative)	No	–
MT	Northwestern Montana recovery area	Dummy (1 = Montana)	No	–
ADULT	Age class variable representing adult wolves (2+ years)	Dummy (1 = adult)	Yes	Annual
PUP	Age class variable representing pups	Dummy (1 = pup)	Yes	Annual
YEARLING	Age class variable representing yearling wolves	Dummy (1 = yearling)	Yes	Annual
DISPERSER	Dispersal status of wolf	Dummy (1 = disperser)	Yes	Seasonal
PRIVATE[a]	Private ownership of land in wolf home range	Numerical (%)	Yes	Annual
CATTLE[a,b]	Cattle density in wolf home range	Numerical (no./km^2)	Yes	Annual
YEAR[b]	Year when wolf monitored	Numerical	Yes	Annual
DENSITY[b]	Density of wolves in recovery area	Numerical (no./1000 km^2)	Yes	Annual

[a] Available only for a subsample of subjects.
[b] Also includes separate variable representing quadratic relationship ($x + x^2$).

ranges where habitat and anthropogenic attributes could be estimated (see Oakleaf et al., 2006). See Smith et al. (in press) for additional details on covariate choice and classification scheme.

We considered year of study (TIME) to address potential temporal shifts in mortality risk, whereas WOLFNO evaluated correlates between mortality risk and annual wolf population estimate in each area (US Fish and Wildlife Service et al., 2008). The WOLFDENS variable scaled population estimates for each recovery area to density estimates based on the fixed size of wolf occupation areas (GYA: 183,000 km^2; ID: 82,300 km^2; MT: 82,700 km^2). Note that exploratory analysis using quadratic forms of scalar parameters failed to improve model fit, and inclusion of season also failed to provide additional explanatory power.

Our main objective was to test specific hypotheses of wolf mortality risk rather than develop parsimonious models of wolf hazard (see Smith et al., in press), so we tested variables in simple tests with RISKTYPE and used *P*-values and 95% confidence intervals (CI) to assess significance. However, it is understood that covariates may interact to reveal complex patterns in risk and we also developed multivariate hazard models using standard model selection procedures including Akaike's information criterion corrected for sample size (AIC_c), AIC_c differences (Δ_i), and AIC_c weights (w_i) (Burnham and Anderson, 2002). Variables were considered as candidates in model selection exercises using cause of death (RISKTYPE) as a main-effects block after they had passed standard diagnostic tests for multi-collinearity (Belsley et al., 1980). We use model-averaged hazard ratios and their unconditional standard errors, as well as the cumulative weight of evidence for a given variable $\sum(w_i)$, to assess the strength of association with cause-specific mortality risk. Initial sets of candidate models were developed excluding PRIVATE and CATTLE (these variables were complete for a subsample of wolves); once the best-fit model was identified, restricted variables were introduced to determine whether they added explanatory power (see Smith et al., in press). Two-way interaction terms with the decomposed RISKTYPE variable were examined, where appropriate.

2.5. Population-level assessment of additivity

We used Heisey–Fuller estimates of annual survival and cause-specific mortality (Heisey and Fuller, 1985) and cumulative incidence functions (Heisey and Patterson, 2006) to determine population-level patterns in risk. Our use of Heisey–Fuller and standard cumulative incidence (not adjusted for left-truncation, see Heisey and Patterson, 2006) was justified by the lack of seasonality in wolf mortality risk as well as strong concordance between wolf risk and a parametric hazard model with underlying Weibull distribution (shape parameter estimate: 1.014 (0.9164, 1.1141)), implying constant hazards (see Murray, 2006).

Population-level determinants of anthropogenic mortality were examined via linear regression of the arcsin-squareroot of annual anthropogenic mortality rate vs. the dummy variable representing MT. We also considered variables representing annual wolf population density estimate, year, and two-way interaction terms between variables. We further tested the hypothesis of additive risk via regression of annual survival vs. annual anthropogenic mortality rate; if the regression slope (±95% CI) of arcsin-squareroot values overlapped −1.0, anthropogenic mortality was fully additive, if the slope = 0 anthropogenic mortality was fully compensatory, whereas intermediate slope corresponded to partial compensation. To correct for serial autocorrelation, we calculated 95% CI by bootstrapping over 2000 iterations, but we did not consider explicitly regression covariance between survival and mortality rates and thus these results are considered as exploratory.

3. Results

3.1. Wolf cause of death

During 1982–2004, we monitored cause-specific mortality for 711 radio-collared wolves across the three recovery areas. Animals monitored during 1982–1994 were exclusively from MT, whereas those tracked during 1995–2004 also included individuals from GYA and ID. Most wolf deaths were attributed directly to anthropogenic causes (67.5%, $n = 363$), with fewer deaths due to natural (20.7%), and unknown (11.8%) causes (Fig. 3). Notably, in each study area the majority of anthropogenic mortality was due to intentional killing of wolves by humans (Table 2). Overall, causes of death were remarkably consistent between recovery areas, although notable exceptions included: (i) less illegal killing in GYA, (ii) legal harvest only for wolves occurring in MT, (iii) more mortality from natural causes in GYA, and (iv) more mortality from unknown factors in MT (Table 2).

3.2. Recruitment method and mortality risk

The hazard ratio for RISKTYPE was 3.2667 (2.5211, 4.2327, 95% CI; $z = 8.96$, $P < 0.001$), indicating an overall higher risk from anthropogenic causes. For comparison, overall number of mortalities attributable to anthropogenic causes was 3.83 times higher

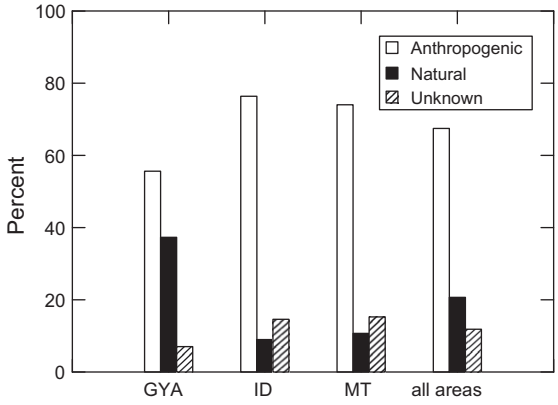

Fig. 3. Fates of 363 radio-collared wolves found dead in western United States (1982–2004).

Table 2
Fates (%) of 711 radio-collared wolves monitored for cause-specific mortality in the western United States (1982–2004). Sample size is in parentheses.

Fate	GYA	ID	MT	All areas
Anthropogenic mortality				
Legal control	16.7 (50)	16.0 (35)	11.9 (23)	15.2 (108)
Illegal killing	5.7 (17)	14.2 (31)	10.2 (39)	12.2 (87)
Legal harvest (Canada)	0 (0)	0 (0)	10.4 (20)	2.8 (20)
Vehicle collision	3.3 (10)	1.4 (3)	2.6 (5)	2.5 (18)
Other (anthropogenic)	0.7 (2)	0 (0)	5.2 (10)	1.7 (12)
Natural mortality				
Natural causes	9.7 (29)	1.8 (4)	5.2 (10)	6.0 (43)
Intraspecific strife	5.4 (16)	1.4 (3)	1.6 (3)	3.1 (22)
Interspecific strife	2.7 (8)	0.5 (1)	0.5 (1)	1.4 (10)
Unknown mortality				
Unknown	3.3 (10)	5.9 (13)	10.4 (20)	6.0 (43)
Censored				
Fate unknown	21.7 (65)	26.5 (58)	20.7 (40)	22.9 (163)
Alive (end of study)	30.8 (92)	32.4 (71)	11.4 (22)	26.0 (185)

than for natural causes (Fig. 3). Inclusion of the RISKTYPE × RECRUITMENT interaction term (0.3846 (0.1869, 0.7916); z = 2.59, P = 0.009) indicated inconsistent differences in hazard type based on recruitment method. In fact, when the interaction term was decomposed to formally assess cause-specific mortality, we detected markedly higher risk for animals dying of anthropogenic causes having been recruited through targeted sampling (2.8327 (2.0160, 3.9803); z = 6.00, P < 0.001) but similar hazard irrespective of recruitment method for those dying of natural causes (1.0895 (0.5462, 2.1734); z = 0.24, P = 0.81; Fig. 4). Because of the overwhelming influence of RECRUITMENT on cause-specific hazards, subsequent analyses are segregated by recruitment method.

3.3. Additive vs. compensatory mortality

Fig. 5 shows higher total mortality among animals from MT; this finding was confirmed by the significant main effect of variable MT in competing risk models for both representative (1.3919 (1.0384, 1.8660); z = 2.21, P = 0.027) and targeted (1.9975 (1.1415, 3.4952); z = 2.42, P = 0.015) animals. The effect of MT was comparable when restricted to 1995–2004 (representative: 1.5325 (1.0769, 2.1807); z = 2.37, P = 0.018; targeted: 1.5694 (0.8774, 2.8072); z = 1.52, P = 0.13), implying that risk in Montana was consistent prior to vs. after 1995. To test our prediction that anthropogenic mortality was additive, we considered the interaction between MT and RISKTYPE. Model results reveal that for the representative sample risk of death due to anthropogenic causes was higher than in the remaining areas (1982–2004: 1.8634 (1.3405, 2.5904); z = 3.70, P < 0.001; 1995–2004: 2.0097 (1.3608, 2.9681); z = 3.51, P < 0.001), whereas for natural causes mortality rate in MT actually was lower (1982–2004: 0.4827, (0.2384, 0.9773); z = 2.02, P = 0.043; 1995–2004: 0.6120 (0.2611, 1.4344); z = 1.13, P = 0.26). In comparison, natural mortality rates were markedly high in GYA whereas those in ID were substantially lower (Fig. 5). For targeted subjects, mortality risk from anthropogenic causes was higher for MT (1982–2004: 1.9261 (1.0708, 3.4646); z = 2.19, P = 0.029; 1995–2004: 1.6977 (0.9222, 3.1252); z = 1.52, P = 0.013), whereas that from natural causes was not lower, although variability was high (1982–2004: 2.5681 (0.7105, 9.2831); z = 1.44, P = 0.15; 1995–2004: 0.5433 (0.0683, 4.3215); z = 0.58, P = 0.56). Overall, 40.3% (n = 193) of wolves monitored in MT succumbed to anthropomorphic causes of death compared to 26.4% (n = 299) and 31.6% (n = 219) for GYA and ID, respectively (Table 2). Thus, anthropogenic mortality risk was largely additive when considered across recovery areas, but to a lesser degree also was compensatory because of the decreased hazard from natural causes in the representative MT sample.

Population-level assessment provided largely consistent findings to those described above. For the representative sample, anthropogenic mortality rate was higher in MT than in other areas (regression coefficient: 0.127 (0.020, 0.235); t_{38} = 2.40, P = 0.022; R^2_{adj}=0.110), but was not related to wolf density or year (all P > 0.26). Annual rates of anthropogenic mortality were consistently <30% for the representative sample of wolves in GYA and

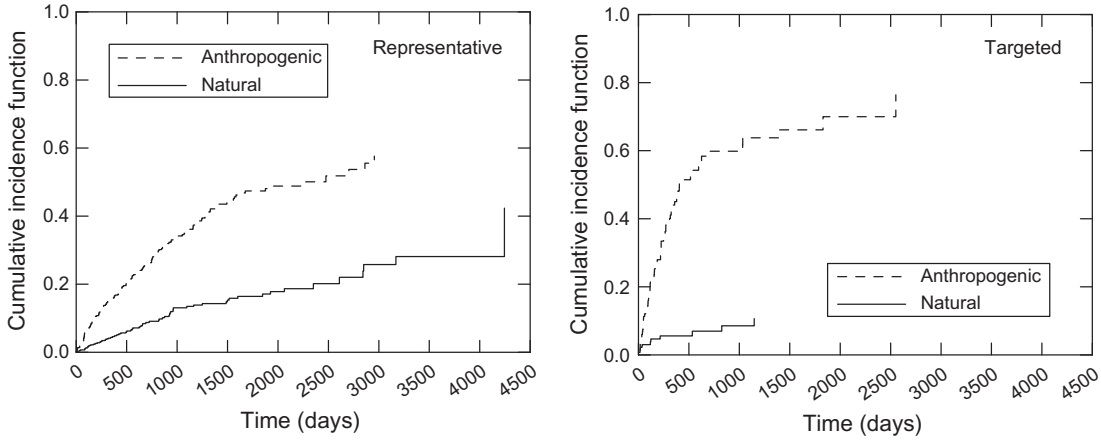

Fig. 4. Cumulative incidence function for 711 radio-collared wolves in western United States (1982–2004).

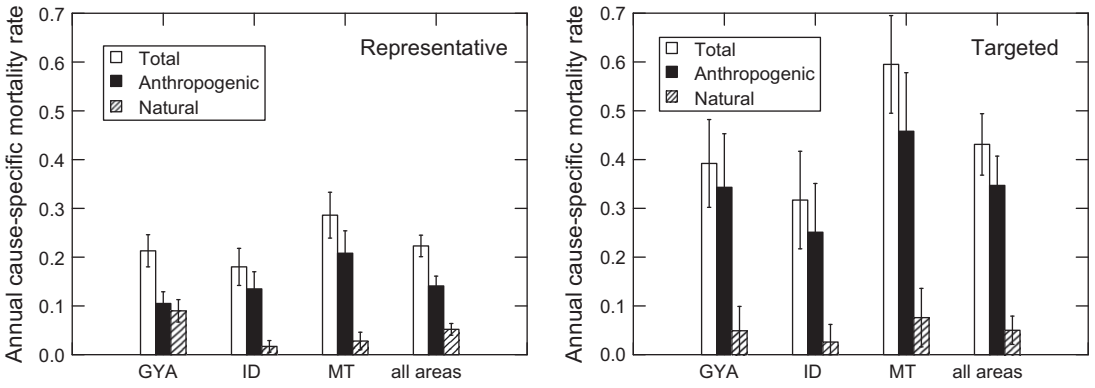

Fig. 5. Annual mortality rates (±95% CI) for 711 radio-collared wolves monitored for survival and cause of death in western United States (1982–2004).

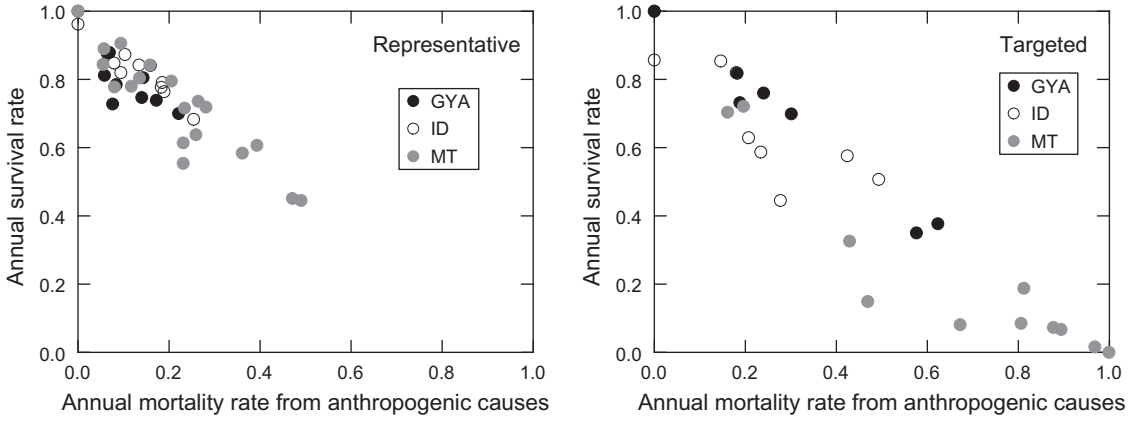

Fig. 6. Back-transformed annual survival rate and anthropogenic mortality rate for 711 wolves in western United States (1986–2004).

ID, whereas for MT this threshold was breached during 4 of 19 years (Fig. 6). For the targeted sample, mortality rate was higher in MT (regression coefficient: 0.481 (0.234, 0.729); $t_{27} = 4.00$, $P < 0.001$; R^2_{adj}=0.356) but unrelated to wolf density or year (all $P > 0.44$). For the targeted sample, the 30% threshold was breached in all areas but exceedances were especially prominent in MT (Fig. 6). At the population-level anthropogenic mortality was largely additive, with the regression slope for annual survival vs. annual anthropogenic mortality rate being closer to 1 than zero (representative slope: −0.833 (−0.836, −0.830), $t_{38} = 13.54$, $P < 0.001$; $R^2_{adj} = 0.717$; targeted slope: −0.824 (−0.829, −0.819), $t_{27} = 5.48$, $P < 0.001$; $R^2_{adj} = 0.518$; Fig. 6). These findings reinforce both the primacy but non-exclusivity of additive mortality from anthropogenic causes, as well as increased risk in MT and among targeted animals.

3.4. Wolf hazard determinants

We assessed mortality risk additivity vs. compensation according to variables listed in Table 1. For animals recruited through representative sampling, total hazard increased with disperser status (2.0815 (1.4527, 2.9824); $z = 4.00$, $P < 0.001$), private ownership (1.0248 (1.0169, 1.0328); $z = 6.22$, $P < 0.001$), and cattle density (1.1783 (1.1100, 1.2508); $z = 5.38$, $P < 0.001$) but not with the remaining variables (all $P > 0.11$). When the above risk was decomposed into cause-specific components, we noted similar hazard for both adult and yearling wolves (all $P > 0.39$). However, for pups hazards were higher from anthropogenic ($z = 2.18$, $P = 0.029$) but not from natural causes ($z = 0.53$, $P = 0.59$), and hazards for dispersers were higher from anthropogenic ($z = 3.87$, $P = 0.001$) but not from natural ($z = 1.56$, $P = 0.12$) causes (Table 3). Private ownership increased risk due to anthropogenic effects; each 1% increase in private ownership in the home range lead to a ∼4% increase in the hazard ratio ($z = 7.39$, $P < 0.001$; Table 3), whereas ownership had no influence on natural risk ($z = 1.63$, $P = 0.10$). We also found that cattle density increased anthropogenic ($z = 6.99$, $P < 0.001$) but not natural hazard ($z = 0.95$, $P = 0.34$). Thus, the main and anthropogenic-only effects of DISPERSER, PRIVATE, and CATTLE were interpreted as evidence of additive hazards in those groups, whereas higher anthropogenic-only mortality among PUPS was supportive of weakly-additive risk.

For the targeted sample, main effects revealed that total mortality increased with yearling (2.0225 (1.2552, 3.2587); $z = 2.89$, $P = 0.004$) and decreased with adult status (0.5411 (0.3406, 0.8596); $z = 2.60$, $P = 0.009$), all other ($P > 0.40$). In terms of anthropogenic mortality, hazard among targeted adults was lower ($z = 3.07$, $P = 0.002$) whereas that among targeted yearlings was higher ($z = 2.42$, $P = 0.016$) than other age groups. This finding contrasts with equivocal risk among the same age groups for representative wolves dying from anthropogenic mortality (Table 3), and is illustrative of selection against yearlings in the targeted group. Hazard was similar among remaining covariates with respect to anthropogenic causes (all $P > 0.084$). Although for natural mortality most covariates were not significant, private ownership ($z = 4.90$, $P < 0.001$) and cattle abundance ($z = 3.61$, $P < 0.001$) actually were negatively correlated to natural hazard (Table 3). This result provides further support for the apparent compensatory nature of anthropogenic mortality among some high-risk animals.

Table 3
Cause-specific hazard ratios (±95% CI) for wolves monitored for mortality in northwestern United States (1982–2004). Hazard ratios were obtained from separate models developed for each listed variable and sampling group (representative, targeted), and included formal segregation of causes of death through interaction terms, while blocking for risk set (see Section 2.4). Demographic and abundance variables were measured for 711 wolves, whereas spatial variables (PRIVATE, CATTLE) were restricted to a subset of 297 wolves whose home range was measured.

Variable	Representative		Targeted	
	Anthropogenic	Natural	Anthropogenic	Natural
ADULT	0.9554 (0.6933, 1.3166)	0.8516 (0.5129, 1.4138)	0.5451 (0.3333, 0.8914)*	0.5138 (0.1432, 1.8427)
YEARLING	1.0260 (0.7019, 1.5001)	0.7461 (0.3821, 1.4572)	2.1898 (1.3278, 3.6113)*	1.1144 (0.2812, 4.4171)
PUP	1.7262 (1.0571, 2.8189)*	0.7285 (0.2278, 2.3295)	0.8394 (0.3682, 1.9136)	3.2378 (0.7986, 13.1268)
DISPERSER	2.2161 (1.4810, 3.3161)*	1.7338 (0.8669, 3.4676)	1.2920 (0.7211, 2.3150)	0.4850 (0.0679, 3.4565)
PRIVATE	1.0324 (1.0237, 1.1041)*	0.9682 (0.9313, 1.0065)	1.0102 (0.9986, 1.0220)	0.7093 (0.6183, 0.8138)*
CATTLE	1.2509 (1.1748, 1.3319)*	0.8638 (0.6386, 1.1685)	1.0168 (0.8801, 1.1748)	0.3736 (0.2189, 0.6375)*
YEAR	0.9814 (0.9469, 1.0172)	1.0700 (1.0046, 1.1396)*	0.8855 (0.8153, 0.9619)*	0.7784 (0.6122, 0.9897)*
NUMBERS	0.9993 (0.9998, 1.0005)	1.0009 (0.9993, 1.0025)	0.9992 (0.9969, 1.0020)	0.9978 (0.9900, 1.0056)
DENSITY	0.9995 (0.8164, 1.2238)	0.9824 (0.7587, 1.2719)	0.9362 (0.6319, 1.3870)	0.4512 (0.0816, 2.4953)

* $P < 0.05$.

3.5. Density-dependent mortality risk

For animals recruited via representative sampling, hazard from both total and anthropogenic-only causes did not change through time ($P > 0.31$), although mortality risk specifically from natural causes actually increased with time (1.0700 (1.0046, 1.1396); $z = 2.10$, $P = 0.036$; Table 3). For animals recruited via targeted sampling, total (0.8678 (0.8029, 0.9380); $z = 3.57$, $P < 0.000$), anthropogenic (0.8855 (0.8153, 0.9619); $z = 2.88$, $P = 0.004$) and natural (0.7784 (0.6122, 0.9897); $z = 2.04$, $P = 0.041$) mortality risk each declined during the study. Assessment of relationships between mortality risk and either NUMBERS or DENSITY were not associated with wolf hazards for either the representative or targeted sample ($P > 0.27$; Table 3). Thus, although there was some degree of change in overall risk through time, relative importance of additive and compensatory mortality appeared largely consistent when examined using simpler models.

We further examined the role of wolf density on hazards by developing multivariate models of cause-specific risk. Because NUMBERS and DENSITY were closely associated, we excluded the former variable and found that the set listed in Table 2 (as well as RISKTYPE, MT) conformed to acceptable standards of colinearity (mean variance inflation factor (VIF) = 2.14; individual VIF < 3.94; condition number = 41.3). For the representative sample, we identified eight candidate models and eight variables that met our inclusion criteria (individual variables $P < 0.10$; $\Delta_i < 10$). The best-fit model had moderate backing ($w_i = 0.499$), and model-averaged hazards indicated higher and lower hazards in MT due to anthropogenic and natural causes, respectively (Table 4). Risk of death from anthropogenic causes was higher among pups, risk of death from both causes was higher among dispersers, and risk from natural causes increased with time; these results were largely consistent with those reported in Table 3. Multivariate models also revealed increasing risk of anthropogenic mortality with wolf density, whereas risks from natural mortality actually declined with density (Table 4 and Fig. 7), presumably through compensatory processes. The main effect of the DENSITY variable was significant when substituted for its interactive terms in the best-fit multivariate model (1.2259 (1.0283, 1.4616); $z = 2.27$, $P = 0.002$), indicating positive effects of density on total risk. This result implies that for the representative group, anthropogenic mortality was density-dependent and natural mortality was depensatory when considered in concert with other variables. Notably, the contrasting relationship between anthropogenic vs. natural hazard vis-a-vis wolf density further argues in favour of compensatory responses. Interaction terms between MT and other variables were excluded from candidate models ($\Delta_i > 11.7$), and spatial variables (PRIVATE, CATTLE) failed to improve model fit ($\Delta_i > 15.2$).

For the targeted sample, six models and six variables met our inclusion criteria. The best-fit model had moderate support ($w_i = 0.456$) and included higher anthropogenic mortality in MT and among yearlings, and lower mortality from either cause of death with time (Table 4). Mortality from anthropogenic causes increased with wolf density, although natural risk remained constant (Fig. 7). Neither interaction terms with MT nor spatial variables added explanatory power to hazard models ($\Delta_i > 18.4$).

Table 4
Model-averaged cause-specific hazard ratios (unconditional 95% CI) and cumulative weight ($\sum(w_i)$) for variables included in multivariate proportional risks models for wolves in northwestern United States (1982–2004). Hazard ratios were estimated for significant ($P < 0.10$) variables and the candidate set was restricted to models with $\Delta_i < 10$.

Variable	Anthropogenic		Natural	
	Hazard	$\sum(w_i)$	Hazard	$\sum(w_i)$
Representative				
MT	2.5619 (1.5232, 3.6006)	1.0	0.4645 (0.0733, 0.8558)	0.535
PUP	1.9166 (0.9822, 2.8510)	0.962	–	
DISPERSER	2.7637 (1.5729, 3.9544)	1.0	1.8639 (0.5619, 3.1658)	0.226
YEAR	–		1.1621 (1.0437, 1.2805)	0.964
DENSITY	1.3850 (1.0708, 1.6992)	1.0	0.5132 (0.2876, 0.7388)	0.930
Targeted				
MT	2.1328 (0.8773, 3.3884)	0.930	–	
YEARLING		–	2.3094 (1.1379, 3.4808)	1.0
PUP	3.0742 (−0.8243, 6.9727)	0.476	–	
YEAR	0.7788 (0.6388, 0.9189)	0.967	0.7781 (0.5950, 0.9612)	0.967
DENSITY	2.8377 (1.3825, 4.2928)	0.967	–	

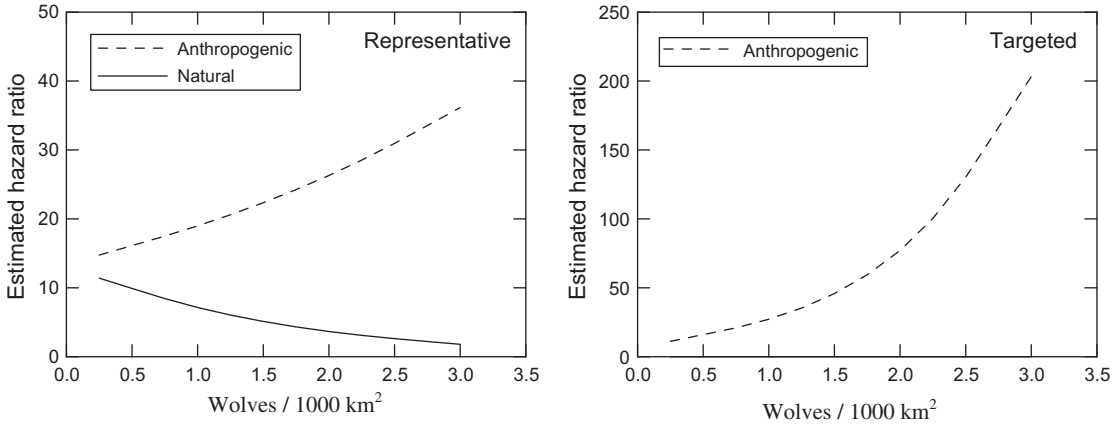

Fig. 7. Estimated hazard ratio (±95% CI) relative to wolf density for 711 wolves in northwestern United States (1982–2004). The relationship was fit using model-averaged hazard ratios from Table 4.

4. Discussion

Our results showed that in expanding wolf populations: (i) anthropogenic mortality is largely additive to natural mortality but compensatory processes also are significant when high vs. low risk individuals are considered explicitly; (ii) anthropogenic mortality is density-dependent whereas natural mortality may be depensatory; and (iii) proportional risk analyses can reveal patterns of differential hazard that are not evident from traditional population-level assessment of cause-specific mortality rates. These findings challenge basic models of cause-specific mortality derived from harvested and recruitment-driven species (e.g., Burnham and Anderson, 1984; Conroy and Krementz, 1990; Boyce et al., 1999), and highlight the need to assess additive vs. compensatory mortality by complementing traditional population-level approaches with analyses of proportional risk of individual animals.

4.1. Anthropogenic effects and wolf population change

In MT, many wolves occurred in proximity to humans and livestock which lead to higher anthropogenic mortality risk. In MT, human-related deaths included not only legal control and illegal killing, but also legal harvest of animals co-occurring in nearby Canada (10.4% of all wolves in MT, $n = 193$). Previous research suggests that ~30% annual human harvest should be sustainable in stationary wolf populations where harvest mortality is the predominant anthropogenic cause of death (Keith, 1983; Fuller et al., 2003; Adams et al., 2008). This implies that anthropogenic mortality rates above this threshold should be additive and destabilizing for most populations, although the point of transition to fully additive effects may be higher for increasing populations that are weakly-harvested. We observed that the 30% anthropogenic mortality threshold was rarely breached for representative wolves (and only in MT), whereas for targeted animals the threshold was exceeded in all areas (but especially in MT). However, because <20% of the total population in each area was comprised of targeted animals, strongly additive effects of anthropogenic mortality in that group do not necessarily translate to marked demographic impact to the larger wolf population. Indeed, wolf populations in GYA and ID increased rapidly during 1995–2004 (ln-transformed annual rate of change GYA: $\mu = 0.308$, $\sigma^2 = 0.062$; ID: $\mu = 0.386$, $\sigma^2 = 0.089$). Although the languishing numerical trend in MT during 1985–2004 ($\mu = 0.080$, $\sigma^2 = 0.114$) was not predicted, it is consistent with the noted high risk from anthropogenic causes in that particular area. Therefore, population growth in MT doubtless was curtailed by largely additive effects of anthropogenic mortality.

We presumed that anthropogenic mortality would be fully additive based on our basic understanding of the likely response to such risk in expanding populations. Instead, evidence points to partially-compensatory processes that were especially apparent in MT and that could be detected at both the population- and individual-level of analysis. Evidently, anthropogenic and natural risks were higher and lower, respectively, among individuals occurring in unprotected or otherwise marginal habitat, implying that the assumption that expanding populations should be fully exposed to additive effects of anthropogenic mortality is suspect. In our study high anthropogenic risk was partially offset by reduced risk from factors such as intraspecific strife and disease, which should be more prevalent in natural landscapes. Accordingly, animals in marginal habitat normally may have lower natural hazard although the extent of natural risk reduction in the absence of anthropogenic effects is unclear. Our results highlight that for wolf populations occupying human-dominated landscapes, high rates of anthropogenic mortality clearly disrupt natural regulatory processes. However, it is notable that wolf populations have a high propensity for demographic response to perturbation (Keith, 1983; Fuller et al., 2003), implying that lesser compensatory responses may occur in other survival-driven species faced with similar levels of anthropogenic mortality when in human-influenced landscapes.

4.2. Risk heterogeneity and density-dependent responses

Compensatory processes should act more subtly on populations than do additive mechanisms and thus are more difficult to quantify. Although population-level analysis revealed some degree of partial compensation acting on wolves especially in MT, the full extent of compensatory processes were only exposed following analysis of individual risks. Differential hazard among individuals, as detected in our simpler models showing higher total and anthropogenic hazard among targeted animals, dispersers, and juveniles, supports the additive mortality model for those particular groups. In contrast, a compensatory process may explain lower natural hazard for targeted animals with home ranges having high private ownership and increased cattle density. Combined, heterogenous demographic responses such as these can directly influence population structure especially among long-lived species with complex life cycles. For example, additive effects of anthropogenic mortality on juvenile wolves can translate to reduced group relatedness and

cohesion (Rutledge et al., 2010) and limitations to social learning (e.g., Nel, 1999), whereas increased mortality among dispersers can impede genetic rescue of distal populations (Haight et al., 1998). Similar responses are inferred from other long-lived carnivore populations where risk of harvest mortality is focused on specific demographic groups (e.g., Wielgus and Bunnell, 2000; Cooley et al., 2009). Mechanisms underlying these subtleties are not easily detected or quantified using the traditional approach of comparing harvest rate to population size, but are especially relevant to species with heterogeneity in individual mortality risk, such as those either with complex social behaviour or having isolated populations in the midst of recovery.

Our finding that anthropogenic hazard is density-dependent whereas natural hazard is depensatory in the representative sample has broad implications to wolf population carrying capacity and recovery. First, it is notable that density effects were only discernible in multivariate hazard models including variable MT, thereby revealing a complex interaction between local wolf population density and mortality risk determinants that was not detectible in population-level analysis. Density-dependent anthropogenic hazard may reflect changes occurring subsequent to expansion of wolves in our study region, including increased dispersal into marginal habitat as prime habitat becomes saturated and higher anthropogenic risk in compromised landscapes (Smith et al., in press). If applied to the broader population including representative animals, this phenomenon is not inconsistent with the observed compensatory natural mortality among targeted animals in proximity to humans or livestock.

Although we failed to find that anthropogenic risk would become increasingly compensatory as the wolf population increased and became regulated by density-dependent factors (see Conroy and Krementz, 1990), density-dependent hazard rates may stabilize population growth. Closed populations can experience weak density-dependent mortality when numbers are below levels where resource limitation influences natality and population size; as prime habitat becomes saturated and animals disperse into marginal areas, density-dependent anthropogenic mortality may become increasingly-important in regulating numbers (Fig. 8). Indeed, leading causes of wolf death during our study (legal control, illegal killing) probably do not operate in a strictly density-dependent manner at low-intermediate wolf densities but rather should become fully regulating as numbers reach a critical threshold determined by habitat availability and human tolerance. This could portend challenges in terms of population management in recovery areas especially if density ultimately reaches levels regulated by new or increasingly-important density-dependent constraints. In fact, this phenomenon may partly explain the recent (post-2004) lower rate of wolf population increase in GYA (Fig. 2), although concomitant numerical increase in ID and MT is difficult to explain in this context and may relate to recent changes in patterns of immigration rather than differential survival (Bangs, unpublished). Regardless, these shifting dynamics highlight the need to manage increasing anthropogenic mortality risk as populations expand and individuals emigrate from core to more marginal areas.

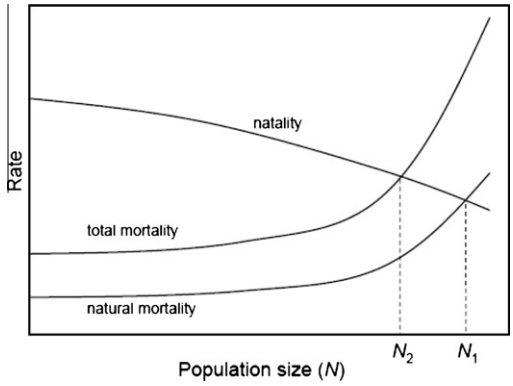

Fig. 8. Population growth as a function of density-dependent mortality and natality. Natural mortality occurs in the absence of anthropogenic mortality, whereas the difference between natural mortality and total mortality represents additive effects of anthropogenic mortality. Anthropogenic mortality is functionally density-dependent at high population density, and reduces equilibrium population density from N_1 to N_2. Adapted from Boyce et al. (1999).

In conclusion, previous work documenting additive mortality processes has focused on population-level effects of human harvest (e.g., Burnham and Anderson, 1984; Boyce et al., 1999), and in general there has not been an earnest effort to discern demographic mechanisms underlying additive hazards under high risk of anthropogenic mortality. Our analysis highlights the need to include assessment of individual risks in such investigation as this approach allowed us to disentangle cause-specific hazards in greater detail than what has been revealed previously. Proportional risks methods currently are underused in ecology but hold promise for radio-telemetry studies where survival time and cause of death are known, especially where anthropogenic hazard plays a premium role on population viability (e.g., Forbes and Theberge, 1996; Woodroffe and Frank, 2005). Thus, as we strive to gain a better mechanistic understanding of the interplay between anthropogenic and natural mortality processes, proportional risks methods should receive increased profile.

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References

Adams, L.G., Stephenson, R.O., Dale, B.W., Ahgook, R.T., Demma, D.J., 2008. Population dynamics and harvest characteristics of wolves in the Central Brooks Range, Alaska. Wildlife Monographs 170, 25.

Bangs, E.E., Fritts, S.H., Smith, D.W., Murphy, K.M., Mack, C.M., Niemeyer, C.C., 1998. Status of gray wolf restoration in Montana, Idaho, and Wyoming. Wildlife Society Bulletin 26, 785–798.

Belsley, D.A., Kuh, E., Welsch, R.E., 1980. Regression Diagnostics: Identifying Influential Data and Sources of Collinearity. John Wiley & Sons, New York.

Boyce, M.S., Sinclair, A.R.E., White, G.C., 1999. Seasonal compensation of predation and harvesting. Oikos 87, 419–426.

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 Inference. A Practical Information – Theoretic Approach. Springer-Verlag, New York, USA.

Burnham, K.P., White, G.C., Anderson, D.R., 1984. Estimating the effect of hunting on annual survival rates of adult mallards. The Journal of Wildlife Management 48, 350–361.

Collett, D., 2003. Modeling Survival Data in Medical Research. Chapman and Hall, New York, USA.

Conroy, M.J., Krementz, D.G., 1990. A review of the evidence for the effects of hunting on American black duck populations. In: Transactions of the North American Wildlife and Natural Resources Conference, vol. 55, pp. 511–517.

Cooley, H.S., Wielgus, R.B., Koehler, G.M., Robinson, H.S., Maletzke, B.T., 2009. Does hunting regulate cougar populations? A test of the compensatory mortality hypothesis. Ecology 90, 2913–2921.

Ellison, L.N., 1991. Shooting and compensatory mortality in tetraonids. Ornis Scandinavia 22, 229–240.

Forbes, G.J., Theberge, J.B., 1996. Cross-boundary management of Algonquin Park wolves. Conservation Biology 10, 1091–1097.

Fuller, T.K., Mech, L.D., Cochrane, J.F., 2003. Wolf population dynamics. In: Mech, L.D., Boitani, L. (Eds.), Wolves, Behavior, Ecology, and Conservation. University of Chicago Press, Chicago, pp. 161–191.

Haight, R.G., Mladenoff, D.J., Wydevan, A.P., 1998. Modeling disjunct gray wolf populations in semi-wild landscapes. Conservation Biology 12, 879–888.

Heisey, D.M., Fuller, T.K., 1985. Evaluation of survival and cause-specific mortality rate using telemetry data. Journal of Wildlife Management 49, 668–674.

Heisey, D.M., Patterson, B.R., 2006. A review of methods to estimate cause-specific mortality in presence of competing risks. Journal of Wildlife Management 70, 1544–1555.

Johnson, F.A., Kendall, W.L., Dubovsky, J.A., 2002. Conditions and limitations on learning in the adaptive management of mallard harvests. Wildlife Society Bulletin 30, 176–185.

Keith, L.B., 1983. Population dynamics of wolves. In: Carbyn, L.N. (Ed.), Wolves of Canada and Alaska: Their Status, Biology, and Management. Canadian Wildlife Service No. 45, Edmonton, AB, pp. 66–77.

Lunn, M., McNeil, D., 1995. Applying Cox regression to competing risks. Biometrics 51, 524–532.

Murray, D.L., 2006. On improving telemetry-based survival estimation. Journal of Wildlife Management 70, 1530–1543.

Nel, J.A.J., 1999. Social learning in canidae: an ecological perspective. In: Box, H.O., Gibson, K.R. (Eds.), Mammalian Social Learning. Cambridge University Press, Cambridge, UK, pp. 259–278.

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 the North American Wildlife and Natural Resources Conference, vol. 49, pp. 535–554.

Oakleaf, J.K., Murray, D.L., Bangs, E.E., Mack, C.M., Smith, D.W., Fontaine, J.A., Oakleaf, J.R., Jiminez, M.D., Meier, T.J., Niemeyer, C.C., 2006. Habitat selection by recolonizing wolves in the northwestern United States. Journal of Wildlife Management 70, 554–563.

Pöysä, H., Elmberg, J., Nummi, P., Sjöberg, G.G., Sjöberg, K., 2004. Ecological basis of sustainable harvesting: is the prevailing paradigm of compensatory mortality still valid? Oikos 104, 612–615.

Rutledge, L.Y., Mills, K., Loveless, K.M., Murray, D.L., Patterson, B.R., White, B.N., 2010. Protection from harvesting restores family pack structure of Eastern wolves in Algonquin Provincial Park. Biological Conservation 143, 332–339.

Schaub, M., Lebreton, J.-D., 2004. Testing the additive versus the compensatory hypothesis of mortality from ring recovery data using a random effects model. Animal Biodiversity Conservation 27, 73–85.

Sinclair, A.R.E., Pech, R.P., 1996. Density dependence, stochasticity, compensation, and predator regulation. Oikos 75, 164–173.

Sinclair, A.R.E., Fryxell, J.M., Caughley, G., 2008. Wildlife Ecology and Management. Blackwell Science, Molden, MA. 488p.

Smith, D.W., Bangs, E.E., Mack, C., Oakleaf, J., Fontaine, J., Boyd, D., Jiminez, M., Pletscher, D., Niemeyer, C., Meier, T.J., Stahler, D., Asher, V., Murray, D.L., in press. Survival of colonizing wolves in the northern Rocky Mountains of the United States 1982–2004. Journal of Wildlife Management.

Smith, G., Reynolds, R., 1992. Hunting and mallard survival. Journal of Wildlife Management 56, 306–316.

Therneau, T.M., Grambsch, P.M., 2000. Modeling Survival Data: Extending the Cox Model. Springer-Verlag, New York, USA.

US Fish and Wildlife Service, 1994. The Reintroduction of Gray Wolves to Yellowstone National Park and Central Idaho. Final Environmental Impact Statement, Denver, Colorado, 608p.

US Fish and Wildlife Service, Nez Perce Tribe, National Park Service, Montana Fish, Wildlife & Parks, Idaho Fish and Game, USDA Wildlife Services, 2008. In: Sime, C.A., Bangs, E.E. (Eds.), Rocky Mountain Wolf Recovery 2007 Annual Report. USFWS, Ecological Services, 585 Shepard Way, Helena, MT, 130 p.

Wielgus, R.B., Bunnell, F.L., 2000. Possible negative effects of adult male mortality on female grizzly bear reproduction. Biological Conservation 93, 145–154.

Woodroffe, R., Frank, L.G., 2005. Lethal control of African lions (*Panthera leo*): local and regional population impacts. Animal Conservation 8, 91–98.