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POPULATION ECOLOGY – ORIGINAL RESEARCH

Compensatory mortality in a recovering top carnivore: wolves in Wisconsin, USA (1979–2013)

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Abstract

Populations of large terrestrial carnivores are in various stages of recovery worldwide and the question of whether there is compensation in mortality sources is relevant to conservation. Here, we show variation in Wisconsin wolf survival from 1979 to 2013 by jointly estimating the hazard of wolves' radio-telemetry ending (endpoint) and endpoint cause. In previous analyses, wolves lost to radio-telemetry follow-up (collar loss) were censored from analysis, thereby assuming collar loss was unconfounded with mortality. Our approach allowed us to explicitly estimate hazard due to collar loss and did not require censoring these records from analysis. We found mean annual survival was 76% and mean annual causes of mortality were illegal killing (9.4%), natural and unknown causes (9.5%), and other human-caused mortality such as hunting, vehicle collisions and lethal control (5.1%). Illegal killing and natural mortality were highest during winter, causing wolf survival to decrease relative to summer. Mortality was highest during early recovery and lowest during a period of sustained population growth. Wolves again experienced higher risk of human-caused mortality relative to natural mortality as wolves expanded into areas with more human activity. We detected partial compensation in human- and natural-caused mortality since 2004 as the population saturated more available habitat. Prior to 2004, we detected additivity in mortality sources. Assessments of wolf survival and cause of mortality rates and the finding of partial compensation in mortality sources will inform wolf conservation and management efforts by identifying sources and sinks, finding areas of conservation need, and assessing management zone delineation.

Keywords Additive mortality · *Canis lupus* · Cause-specific mortality · Censoring · Survival

Introduction

Understanding annual survival rates and its dependence on interacting causes of mortality in wildlife populations informs researchers and managers about population growth potential, setting of harvest quotas, and effects of age, sex, environmental variables, population responses to management actions, and other covariates (Fuller 1989; Smith et al. 2010). Assessing survival and cause-specific mortality as a function of time and space broadens our understanding of survival in a wildlife population with extensions to identifying sources and sinks, areas of conservation need, management zone delineation, timing of harvest seasons, and periods of reduced survival (Péron et al. 2011; O'Neil et al. 2017; Schwartz et al. 2010). Here, we analyze gray wolf (*Canis lupus*) mortality in Wisconsin from recolonization through the first years of recreational harvest (1979–2013), thereby providing critical information about where and when wolves experience variable hazard rates from different mortality sources. This spatial demographic approach informs conservation and management by detecting whether management zone delineation aligns with hazard risks and if recent changes in mortality hazards indicate compensation

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for additional harvest mortality of one of the most closely monitored wolf populations in the world.

Recolonization of wolves from Minnesota into northwestern Wisconsin began in the mid-1970s, and by 1980 five packs established territories (Wydeven et al. 2009). By 1995, the wolf population increased to > 80 wolves in 21 packs and extended their range into central Wisconsin forests (minimum yearly estimates based on mid-winter counts). Estimated minimum wolf numbers increased to > 800 wolves in 213 packs in 2012 prior to the first wolf hunting season. Recovering wolves occupied high quality habitat first and then progressively occupied more marginal habitat as the population grew (Mladenoff et al. 1995; Mladenoff et al. 2009) coincident with decelerating population growth (Van Deelen 2009). Wolves were delisted from the US endangered species list in 2012, and the Wisconsin legislature mandated a recreational wolf hunting and trapping program (Wydeven et al. 2012) that was implemented during 2012–2014. In December 2014, a Federal Court decision relisted gray wolves in the western Great Lakes region (including Wisconsin) as an endangered species (Humane Society of the United States V. Jewell, 2014 WL 7,237,702; D.D.C. Dec. 19, 2014).

Research into whether human-caused mortality is compensatory or additive with other causes of mortality is controversial and has been identified as an important research need in wolf conservation (Vucetich and Peterson 2004). Analysis of mortality rates from North American wolf populations resulted in conflicting conclusions of compensation, additivity and super-additivity of mortality sources (Adams et al. 2008; Creel and Rotella 2010; Fuller et al. 2003). Rate of intraspecific killing among wolves is lower in exploited wolf populations, potentially suggesting compensatory mechanisms (Cubaynes et al. 2014). Contrarily, wolves are fairly long-lived and recovering over much of their range, making them poor candidates for compensation which is more prominent in short-lived species and populations at carrying capacity (Mech and Boitani 2003; Péron 2013). Development of statistical methods for understanding the spectrum of compensation to additivity while accounting for biases makes assessment of compensation of mortality sources in Wisconsin's wolf population very timely (Péron 2013; Schaub and Lebreton 2004; Servanty et al. 2010).

Survival analysis for Wisconsin's wolf population is also timely because of recent changes in management, variable population growth over the last three decades and across Wisconsin, and the advancement of methods to explicitly estimate all hazards leading to the end of a monitoring record. In most survival analysis, individuals that leave the study (i.e., lose their mark) before death are right censored and this process assumes independence from death (Klein and Moeschberger 2003). This assumption means that censored individuals are no more likely to die than if they remained monitored and in the study. In telemetry-based wildlife survival studies, censoring may occur when individuals are lost to follow-up during the study because of collar failure, dispersal or unknown reasons. For wolves, loss to radio-telemetry follow-up (collar loss, hereafter) could also result from illegal killing and destruction of the radio collar in which case treating as censored is inappropriate because censoring is confounded with death (Liberg et al. 2012; Stenglein et al. 2015c). Under this circumstance where censoring is death, the censoring assumption required for most survival analysis is not met (Klein and Moeschberger 2003). An approach to survival analysis that allows for explicit estimation of the censoring process is especially beneficial for long-lived wildlife species where marked individuals have more time to lose their marks or are lost to follow-up. One such method is a cause-specific hazard approach to survival analysis that separates the analytical task into an overall hazard for the event of interest (i.e., endpoint of a telemetry record) and probabilities that the event of interest occurred due to certain causes (Cross et al. 2015). This framework allows for flexibility in defining causes of interest and timescales, explicitly modeling the censoring process as a cause, inclusion of time-varying covariates for hazard and cause-specific probabilities, and simultaneous estimation of all parameters.

With three decades of radio-telemetry records and the advent of novel survival analysis methods, we have an unprecedented opportunity to understand the survival dynamics of a reestablishing large carnivore population interacting with dynamic sources of human-caused mortality. Using known fate data of 501 radio-collared wolves, we quantify the temporal and spatial dynamics of wolf survival in Wisconsin from 1979–2013. Our event of interest was the time to the end of wolves' radio-telemetry records and we simultaneously estimated censoring and mortality hazards. With annual estimates of survival and cause-specific mortality, we looked for evidence of compensatory mortality.

Our goal was to quantify spatial and temporal variations in cause-specific mortality risks for wolves as their population expanded and management changed, and assess our findings against the following predictions. We expected that the patterns in cause-specific mortality would be different for the two categories of censoring (collar loss and known censoring) because the former could be due to illegal killing which has temporal and spatial trends and the latter we predicted to be more random, and therefore more uniform across space and time. Seasonally, we predicted wolf mortality from illegal killing would be highest in early winter corresponding with Wisconsin's gun deer season when > 600,000 people are afield with weapons (9 days in late November; Stenglein et al. 2015b). Across years, we expected survival to be lowest in early years of recovery when wolves were just

returning to Wisconsin and may have been experiencing an Allee effect (Stenglein and Van Deelen 2016). Spatially, we predicted that wolf survival would be highest in northwestern Wisconsin where wolves have been established the longest. We predicted additivity in mortality sources while the wolf population was reestablishing in Wisconsin and partial compensation in more recent years as the population saturated available habitat (Mladenoff et al. 2009; Van Deelen 2009).

Materials and methods

Study area

Our study area was north and central Wisconsin, USA (1979–2013). Wolves primarily occupied the northern (wolf harvest zones [WHZs] 1, 2, 3, and 4; Fig. 1) and central forest regions (WHZ 5) of Wisconsin (Mladenoff et al. 2009). We limited our analysis to WHZs 1–5 because WHZ 6 had limited wolf activity and was not representative of mortality patterns within primary wolf range.

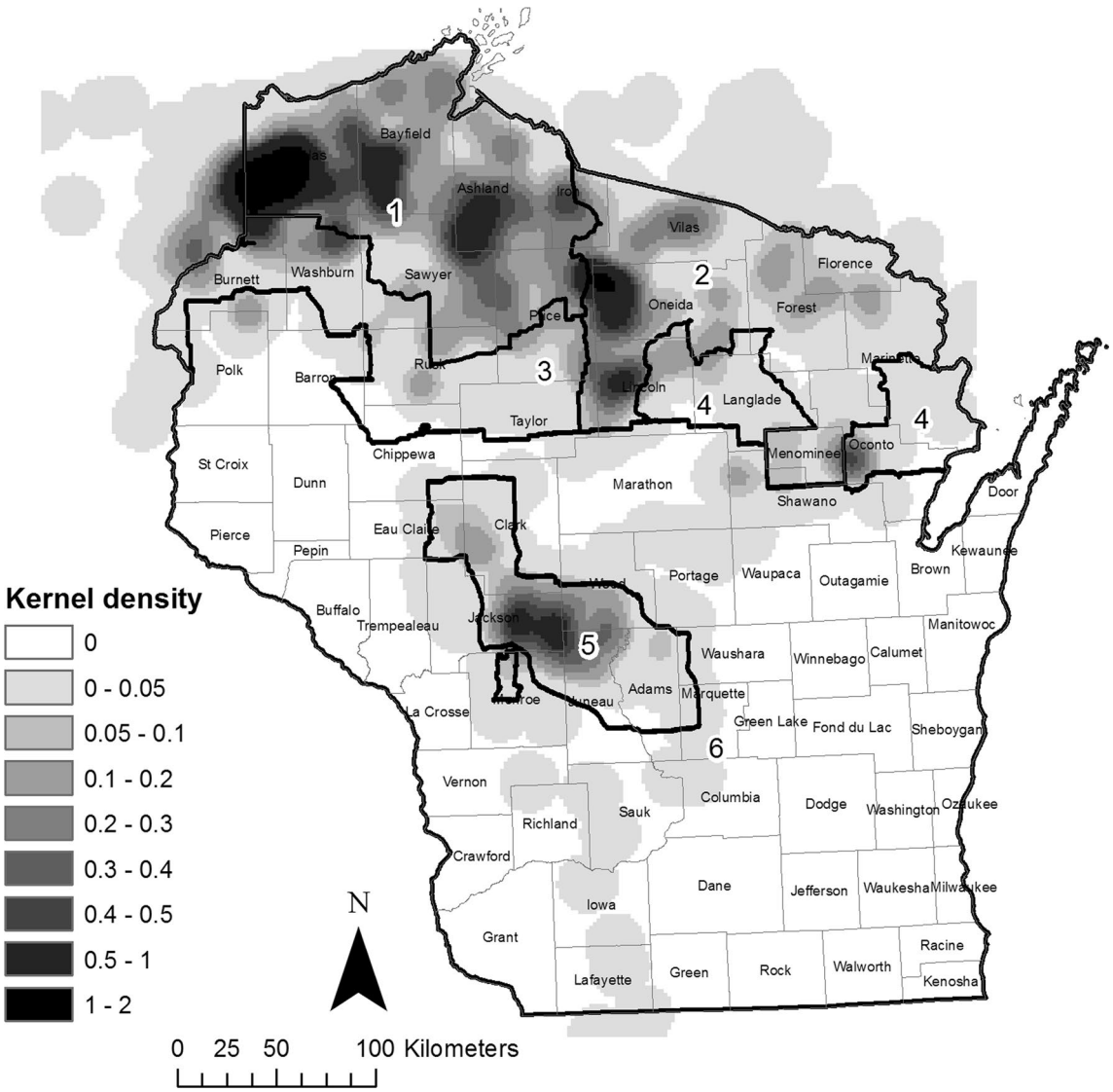

Fig. 1 Wolf Harvest Zones 1–6 (designated in 2012) with counties of Wisconsin, USA, and a kernel density map showing the smoothed radiotelemetry locations of 501 gray wolves (*Canis lupus*) radio-collared and tracked from 1979 to 2013

Dataset

Wisconsin Department of Natural Resources (WDNR) provided radio-telemetry records for wolves. Wolf trapping and handling occurred under the oversight of the WDNR Animal Care and Use Committee (Wydeven et al. 2009). The dataset consisted of > 42,000 weekly locations of 501 wolves captured and tracked (November 1979–December 2013). Wolves were generally radio-collared only if ≥ 4 months old and trapping targeted yearling or adult wolves in wolf pack territories (Wydeven et al. 2009). All applicable institutional and/or national guidelines for the care and use of animals were followed. For each location we compiled week, month, year, coordinate location and outcome. Outcome was 1 for locations that were the last records for individual wolves (hereafter:endpoint) and 0 otherwise. For each endpoint, we recorded a cause classified as: (1) lost to follow-up from unknown causes (collar loss), (2) known collar failure or lost to follow-up during the study and then found dead sometime after its endpoint (known censoring), (3) dead because of illegal killing (illegal kill), (4) dead because of human causes other than illegal killing, including vehicle collision, lethal control action, and harvest (other human), (5) dead because of other causes, including disease, intraspecific strife, and unknown causes (natural and unknown). We did not consider surviving until the end of the study an endpoint. The datasets used in this study are available in the online resources and from the corresponding author on reasonable request.

We summarized data by week and month and kept each wolf's last record during each time period, resulting in 42,504 weekly and 9811 monthly location records and 471 endpoints. We began our monthly location records dataset in May 1980 and omitted records from two wolves captured and found dead in 1979 because of a gap in the dataset with no individuals from November 1979 until May 1980. For the spatial analysis, we used the monthly location records dataset and kept records that were within WHZs 1–5 in Wisconsin. This resulted in 9078 monthly location records and 407 endpoints.

Survival analysis

We used Bayesian hierarchical models to analyze endpoints for radio-collared wolves (see Online Resource 1 for model and R code). We divided our analysis into estimates of overall hazard of endpoints and estimates of cause-specific mortality probabilities (Cross et al. 2015). To estimate overall endpoint hazard, we modeled endpoint cumulative weekly or monthly hazards using a complementary log–log (cloglog) model for grouped survival times (Prentice and Gloeckler 1978). This discrete time proportional hazards model related radio-collared relocation outcomes to smoothed functions of time or space (i.e., penalized splines). We defined the 0 or 1 outcome of whether a wolf reached the end of its radio-telemetry record in each week (temporal analyses) or month (spatial analyses) as y_i where the records $i = 1, 2, \ldots, N$ for N total records were distributed as Bernoulli random variables with means μ_i that related to covariates through the cloglog link. The hazard, h_i, was calculated from μ_i:

$$y_i \sim \text{Bernoulli}\left(\mu_i\right),$$

$$c \log\log\left(\mu_i\right) = \mathbf{X}_i\boldsymbol{\beta} + \mathbf{Z}_i\mathbf{b},$$

$$h_i = -\log(1 - \mu_i).$$

Fixed effects of linear predictors were matrix $\mathbf{X}_i$ with N rows and columns equal to the number of covariates. In our time-varying models, $X_{i,1}$ was equal to one for the intercept and $X_{i,2}$ was the month or year for observation i. In our spatial models, $X_{i,1}$ was equal to one for the intercept,$X_{i,2}$ was the X coordinate, and $X_{i,3}$ was the Y coordinate for observation i. $\boldsymbol{\beta}$ was the vector of estimated fixed effects and we assigned vague priors to $\boldsymbol{\beta}$: $\boldsymbol{\beta} \sim$ normal $(0, 100^2)$.

$\mathbf{Z}_i\mathbf{b}$ was the penalized spline where $\mathbf{Z}_i$ was a distance matrix between the spline covariate ($X_{i,2}$ for time-varying models and $X_{i,2}$ and $X_{i,3}$ for space-varying models) and knots with N rows and columns equal to the number of knots (Crainiceanu et al. 2005). We chose 40 knots for time-varying models and 200 knots for space-varying models. We chose knot locations along annual and monthly time scales as sample quantiles of the time covariate (Crainiceanu et al. 2005). Knot locations in space were determined from a space filling algorithm implemented with 'cover.design' in package Fields (Nychka et al. 2015) and R version 3.1.1 (R development Core Team 2013). We chose the cubic thin plate spline basis for temporal models and bivariate radial spline basis for spatial models (Crainiceanu et al. 2005; Péron et al. 2011). Vectors $\mathbf{b}$ were estimated spline coefficients with length equal to the number of knots. We assigned vague priors to $\mathbf{b}$:

$$\mathbf{b} \sim \text{normal}\left(0, \sigma^2\right),$$

$$\tau = \frac{1}{\sigma^2},$$

$$\tau \sim \text{gamma}\ (0.001, 0.001).$$

Spline fitting performance improves when covariates are not excessively large (Crainiceanu et al. 2005). Both years and coordinates were too large. Therefore, we subtracted 1995 and divided by 12 for the year covariate. For coordinates, we subtracted the minimum coordinates (UTMs) from each X and Y value and divided by 10,000.

For cause-specific probabilities, we took cause-specific endpoints (records when outcome = 1) and related them to covariates through a multinomial logit model. For each

observed endpoint, c_j where the endpoints $j = 1, 2, \ldots, n$ and n total endpoints, we modeled cause-specific probabilities p_{jk} that endpoint j was observed to be due to cause k where causes $k = 1, 2, \ldots, K$ for K total causes, as categorical and $\sum_{k=1}^{K} p_{jk} = 1$:

$$c_j = \text{categorical}(p_{j,1}, p_{j,2}, \ldots, p_{jk}),$$

$$p_{jk} = \frac{e^{\varepsilon_{jk}}}{\sum_{k=1}^{K} e^{\varepsilon_{jk}}},$$

$$\varepsilon_{jk} = \mathbf{U}_j \mathbf{A}_k + \mathbf{V}_j \boldsymbol{\alpha}_k.$$

Through the generalized logit link, we related probabilities p_{jk} to linear predictor ε_{jk}. The fixed effects portion contained data matrix $\mathbf{U}_j$ with n rows and columns equal to the number of covariates. $\mathbf{A}_k$ was the matrix of estimated fixed effects. We used category $k = 1$ as the constraint and set $\mathbf{A}_1 = 0$. We put normal priors on $\mathbf{A}_k$ s for $k = 2, 3, \ldots, K$ with mean 0 and standard deviation 100: $\mathbf{A}_k \sim \text{normal}\ (0, 100^2)$. $\mathbf{V}_j \boldsymbol{\alpha}_k$ was the penalized spline and specified as described for $\mathbf{Z}_i \mathbf{b}$, above.

To get p_{jk} and h_i on the same scale, we created prediction datasets. Our prediction dataset for weekly records within a calendar year was weeks 1, 2… 52 and for monthly records across years was years 1981, 1982,… 2013. We did not include 1980 because of missing data. Our prediction dataset for our spatial model was a grid of locations that covered WHZs 1–5 at one location per 10 km for a total of 561 prediction locations. We denoted all prediction datasets with the variable $t = 1, 2, \ldots, T$ where T is the total number of records. These datasets became covariates $\mathbf{X}_t$ and $\mathbf{U}_t$ and were used to calculate new distance matrices $\mathbf{Z}_t$ and $\mathbf{V}_t$ where $\mathbf{Z}_t = \mathbf{V}_t$. For each prediction dataset, we estimated the hazard, h_t, cause-specific mortality probabilities, p_{tk}, and cause-specific hazard,h_{tk}:

$$c \log\log\left(\mu_t\right) = \mathbf{X}_t \boldsymbol{\beta} + \mathbf{Z}_t \mathbf{b},$$

$$h_t = -\log\left(1 - \mu_t\right),$$

$$p_{tk} = \frac{e^{\varepsilon_{tk}}}{\sum_{k=1}^{K} e^{\varepsilon_{tk}}},$$

$$\varepsilon_{tk} = \mathbf{U}_t \mathbf{A}_k + \mathbf{V}_t \boldsymbol{\alpha}_k,$$

$$h_{tk} = h_t \cdot p_{tk}.$$

The total mortality hazard M_t at time t was the sum of the hazards from the known mortality causes (illegal kill, other human, and natural and unknown) and survival S_t followed the definition of survival for discrete time periods (Klein and Moeschberger 2003). Time period t was weekly for temporal models and monthly for spatial models. Finally, the cause-specific mortality rate m_t was the product of the cause-specific hazards that ended in death ($k = 3, 4, 5$) and survival rate (Heisey and Patterson 2006):

$$M_t = \sum_{k=3}^{5} h_{tk},$$

$$S_t = e^{-\sum_1^t M_t},$$

$$m_t = h_{tk} \cdot S_t.$$

We ran all models with a Gibbs sampler in Program JAGS (Plummer 2003) with library 'rjags' (Plummer 2011) in program R. We ran two chains and discarded the first 5000 iterations as burn-in. Then, we sampled the posteriors for an additional 15,000 iterations. For each model, we assessed convergence using visual inspection of chain mixing and univariate potential scale reduction factors ($\hat{R}$) of survival parameters (Gelman and Rubin 1992). Generally, convergence is adequate when upper 97.5% confidence limits of $\hat{R}$ s are close to 1 and we considered convergence attained if upper 97.5% confidence limits of all $\hat{R}$ s were < 1.1.

Test for compensatory mortality

Accurate calculation of the amount of compensation in mortality sources requires an estimate of bias resulting from the competition of natural- and human-caused mortality on the same population of individuals as the total number of individuals changes over time (Péron 2013; Schaub and Lebreton 2004). This bias can lead to the appearance of partial compensation even when mortality sources are additive (Schaub and Lebreton 2004). To accommodate this bias, researchers need an independent estimate of natural mortality in unexploited populations, so correlation between natural- and human-caused mortality can be estimated. The estimated correlation between natural- and human-caused mortality in exploited populations can be corrected by subtracting the estimated bias (Péron 2013; Schaub and Lebreton 2004; Servanty et al. 2010). We followed Péron (2013) to estimate the compensation-additivity rate, C, where $C = 0$ was complete additivity, $C = 1$ was complete compensation, and values from 0 to 1 indicated partial compensation (see Online Resource 1 for model and R code).

From our inter-annual model, we took annual mean posterior estimates of human-caused, h_t, and natural-caused mortality, n_t, as inputs in a multivariate normal model to estimate correlation coefficient, ρ:

$$\begin{pmatrix} n_t \\ h_t \end{pmatrix} \sim \text{multivariate normal}\left(\begin{pmatrix} \bar{n} \\ \bar{h} \end{pmatrix}, \begin{pmatrix} \sigma_n^2 & \rho \cdot \sigma_n \cdot \sigma_h \\ \rho \cdot \sigma_n \cdot \sigma_h & \sigma_h^2 \end{pmatrix}\right).$$

We used vague priors: $\bar{n} \sim \text{normal}(0, 100^2)$, $\bar{h} \sim \text{normal}(0, 100^2)$, $\rho \sim \text{uniform}(-1, 1)$, $\tau_n \sim \text{gamma}(0.001, 0.001)$, and $\tau_h \sim \text{gamma}(0.001, 0.001)$ where $\sigma_n^2 = 1/\tau_n$ and $\sigma_h^2 = 1/\tau_h$. Our estimate of bias was:

$$b = -\frac{\bar{n}_0}{\sqrt{\bar{n}_0^2 + (1+\bar{h})^2 \cdot \frac{\sigma_{n_0}^2}{\sigma_h^2} + \sigma_{n_0}^2}},$$

where $\bar{n}_0$ and $\sigma_{n_0}^2$ were our estimates of mean and variance of unexploited natural mortality, respectively. We used annual mortality rates from the Isle Royale wolf population (1974–1995) to calculate $\bar{n}_0 = 0.28$ and $\sigma_{n_0}^2 = 0.031$ (Peterson and Page 1988; Peterson et al. 1998). We conducted a sensitivity analysis of C on our choice of $\bar{n}_0$ and $\sigma_{n_0}^2$ by varying $\bar{n}_0$ plus and minus 10% and $\sigma_{n_0}^2$ plus and minus 30%. We calculated our corrected correlation, $\rho_c = \rho - b$, and the rate of compensation-additivity, $C = -\rho_c \cdot \sqrt{\frac{\sigma_n^2}{\sigma_h^2}}$.

We estimated C across all 33 years, and then estimated C separately for the last 10 years (2004–2013) compared to previous years (1981–2003). A 95% credible interval (CrI) on C that overlapped 0 meant we could not reject the hypothesis of additivity between human- and natural-caused mortality sources. We ran models in program JAGS following the same procedures as outlined above, except we ran three chains for 20,000 iterations after discarding the first 20,000 iterations as burn-in.

Results

Dataset

Among all endpoints, 41.4% were collar loss, 10.4% were known censored, 17.2% were illegally killed, 11.0% were killed from other human causes, and 20.0% were killed from natural and unknown causes.

Survival analysis

In all models, upper 97.5% estimates of $\hat{R}$(potential scale reduction factor as a measure of model convergence) were all ≤ 1.1 and we determined adequate convergence. We estimated overall mean annual survival for radio-collared wolves in Wisconsin as 0.759 (SD = 0.019). Overall mean annual mortality was 9.4% (SD = 1.7) from illegal killing, 5.1% (SD = 1.1) from other human causes, and 9.5% (SD = 1.7) from natural and unknown causes. Annually, 21.8% (SD = 2.1) of radio-collared wolves experienced collar loss (unknown censored) and 5.2% (SD = 1.2) were censored (known censored). Weekly hazards varied throughout the year with the highest mortality hazards during winter (Fig. 2). By cause, illegal kill hazards peaked in late November aligning with Wisconsin's nine-day gun deer season as we predicted. Other human mortality peaked in September, and natural and unknown mortality peaked in January (Fig. 3). The hazard of collar loss was highest in February and again in November seeming to correspond most with a combination of the natural and collar loss hazards, partially matching our prediction of highest collar loss hazard in November when illegal killing may increase due to many deer hunters on the landscape with weapons. Censoring hazard was largely independent of month matching our

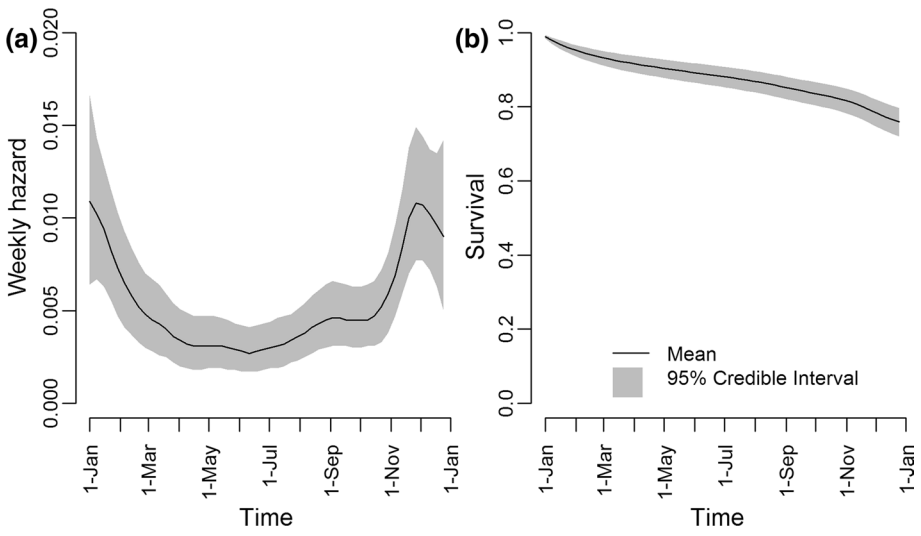

Fig. 2 **a** Estimated weekly hazard of mortality and **b** survival estimates for wolves in Wisconsin, USA from an analysis of radio-tracked wolves, 1979–2013

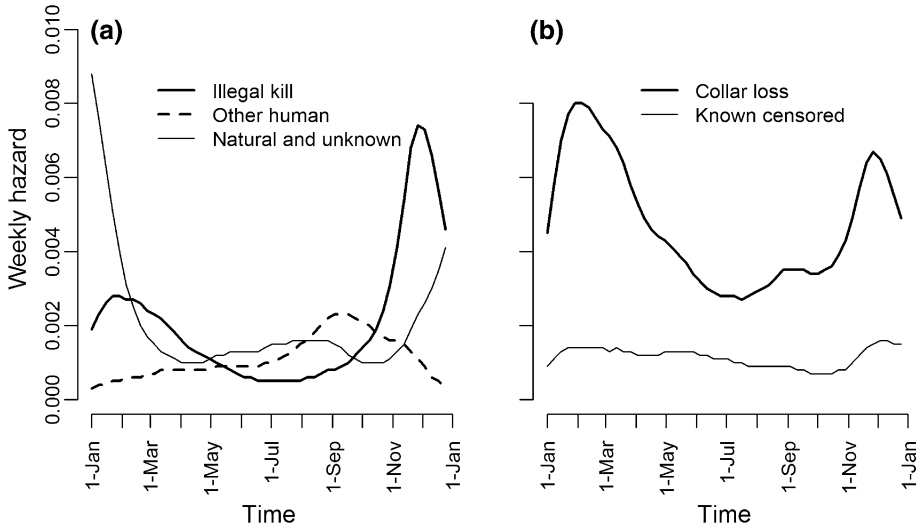

Fig. 3 Estimated weekly hazard from known mortality sources of **a** illegal killing, other human-caused mortality, and natural and unknown mortality, and **b** collar loss and known censoring for wolves in Wisconsin, USA from an analysis of radio-tracked wolves, 1979–2013

prediction that known censoring hazards were more uniform across time and showed a different pattern than collar loss hazards (Fig. 3).

During wolf recovery, total mortality monthly hazard was highest in the early 1980s aligning with our prediction that early recovery had reduced survival potentially due to an Allee effect (Stenglein and Van Deelen 2016). Additional periods of reduced survival were 2003–2005 and 2011–2013, the latter potentially from density dependence in survival as the best habitat became saturated (Fig. 4). Annual survival varied from 0.602 to 0.815 with survival peaking during the late 1990s and 2000s after the population was established and during a time of significant population growth (Fig. 4). Highest monthly hazard of illegal killing occurred in the early 1980s and since 2000. Highest monthly hazard of other human mortality had a small peak in the early 1990s and increased in the 2000s (Fig. 5). Natural mortality hazard was highest in the mid-1980s and early 1990s with another peak in the early 2000s (Fig. 5). Hazard from collar loss was highest in the mid-1980s aligning somewhat with the illegal killing hazard and decreased by mid-1990s. The collar loss hazard decrease over time may be explained by improvements in radio-collaring technology leading to longer battery life on radio-collars, and therefore fewer collar losses from collar failure. Censoring hazard was highest in the late 1980s and early 1990s showing a different pattern from the collar loss hazard, as predicted (Fig. 5).

Estimated annual survival was highest in central WHZ 1 and western WHZ 2 where wolves had been established the longest and aligning with our predictions. Annual survival was lowest and illegal killing hazard the highest in eastern WHZs 2 and 4 and southeastern WHZ 5, showing a dramatic decrease in survival on the edge of Wisconsin's wolf range (Fig. 6). Here standard deviation of annual survival

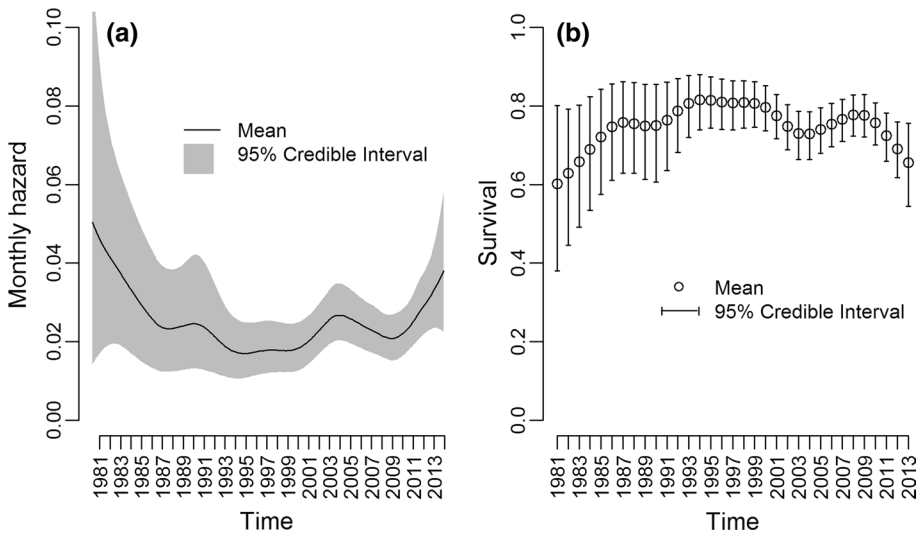

Fig. 4 **a** Estimated monthly hazard of mortality and **b** annual survival estimates for wolves in Wisconsin, USA from analysis of radio-tracked wolves, 1979–2013

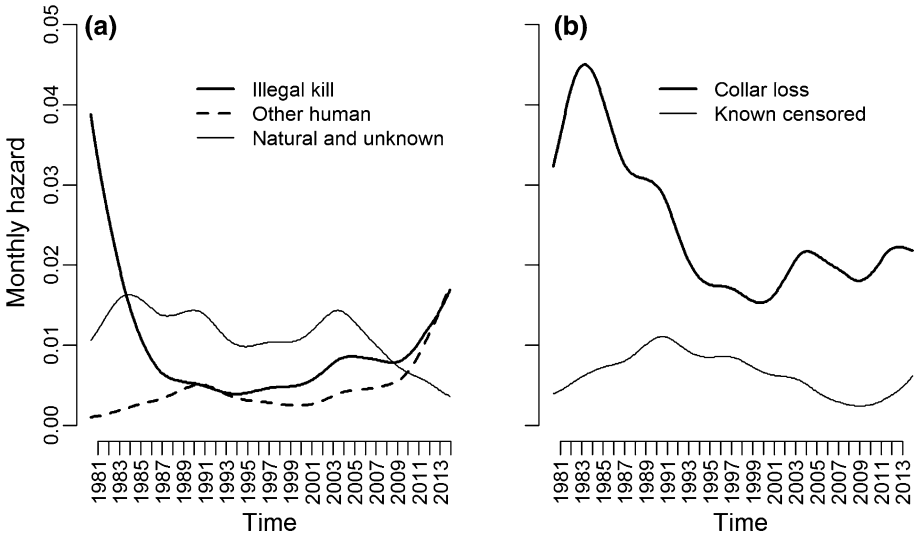

Fig. 5 Estimated monthly hazard from known mortality sources of (**a**) illegal killing, other human-caused mortality, and natural and unknown mortality, and **b** collar loss and known censoring for wolves in Wisconsin, USA from analysis of radio-tracked wolves, 1979–2013

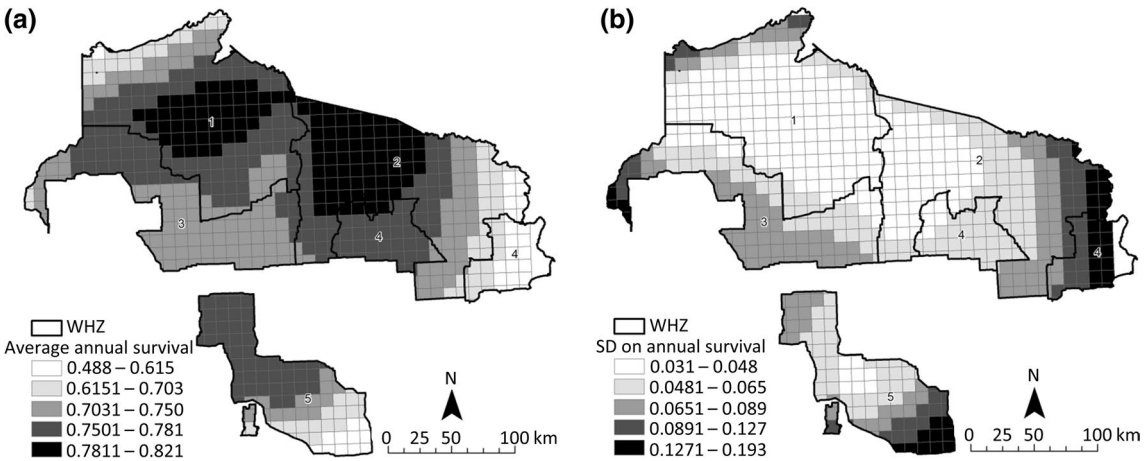

Fig. 6 **a** The annual survival rates of wolves and **b** standard deviations of survival in wolf harvest zones (WHZs) 1–5 of Wisconsin, USA estimated from radio-telemetry locations, 1981–2013

was also highest because there were few telemetry locations (Figs. 1 and 6). Other human mortality hazard was lowest in western WHZ 3 to central WHZ 2 and northern WHZ 5. Natural and unknown mortality hazard was highest in western WHZs 1 and 3 where wolves were entering Wisconsin from a source population in neighboring Minnesota (Fig. 7). Hazard from collar loss was highest in eastern WHZs 2 and 4 and throughout WHZ 5 matching the areas of highest illegal killing hazard and lowest survival overall, and known censoring hazard increased along a northeast to southwest gradient (Fig. 7).

Ratios of human to natural-caused mortality hazards revealed that WHZ 1 and western WHZ 3 have approximately the same amount of human-caused and natural-caused mortality (Fig. 8). As wolves move east, they encounter 2–3 times higher hazard of human-caused to natural-caused mortality into central WHZ 2 and > 5 times higher hazard of human-caused to natural-caused mortality in eastern WHZs 2 and 4 closer to the periphery of their range in Wisconsin (Fig. 8).

Test for compensatory mortality

Complete compensation between human- and natural-caused mortality was not possible because variance in human-caused mortality was greater than variance in natural mortality (Table 1; Péron 2013). Across all years, we detected partial compensation in rates of human- and natural-caused wolf mortality (Table 1, $C = 0.186$ [95% CrI 0.025–0.338]). Therefore, any compensation of human-caused mortality by decreases in natural mortality would be at best partial. When separated into two time periods, we could not reject the hypothesis of additivity from 1981–2003 ($C = -0.141$ [95% CrI − 0.306–0.026]) and there was evidence of partial

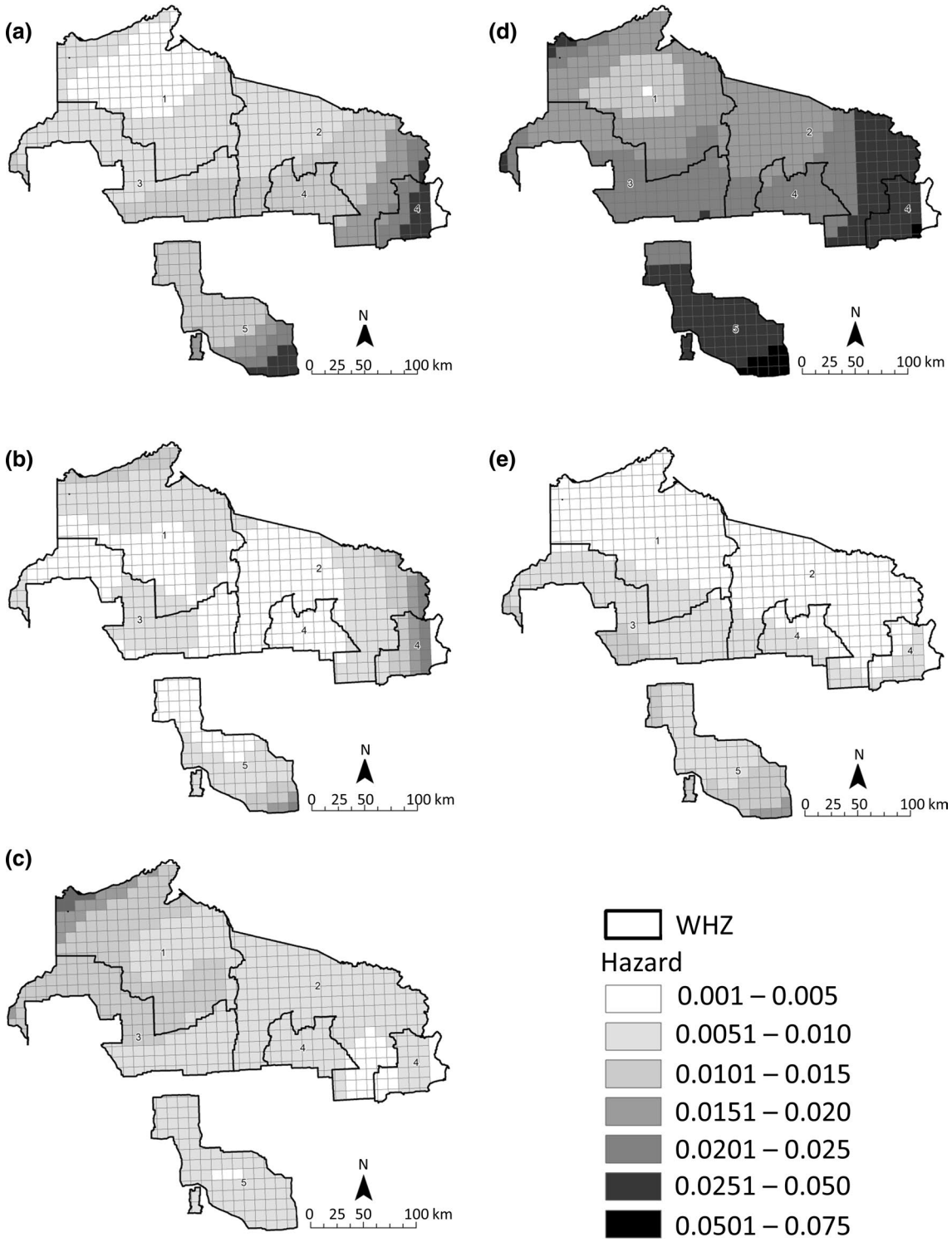

Fig. 7 Estimated monthly hazards of Wisconsin wolves reaching end of radio-telemetry records due to: **a** illegal killing, **b** other human mortality, **c** natural and unknown mortality, **d** collar loss, and **e** known censoring. Monthly hazards were estimated and mapped for wolf harvest zones (WHZs) 1–5 of Wisconsin, USA from radio-telemetry locations, 1981–2013

compensation from 2004–2013, aligning with our predictions (Table 1, $C = 0.351$ [95% CrI 0.087–0.582]). The early years may also show super-additivity concurrent with the population experiencing an Allee effect and later years as the population approaches carrying capacity and saturates the best habitat, we detected partial compensation in

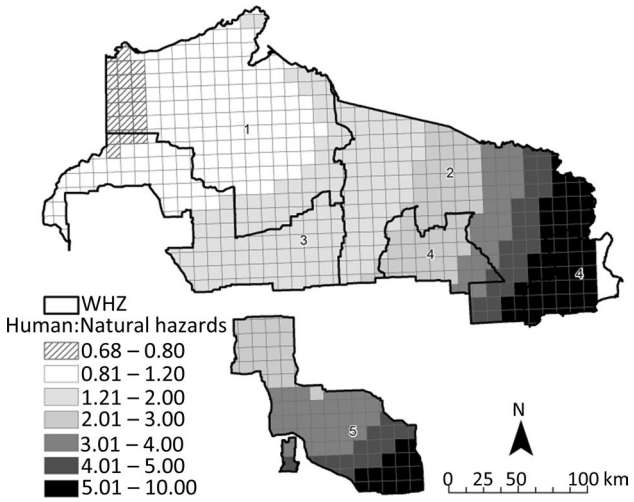

Fig. 8 Wolf harvest zones (WHZs) 1–5 of Wisconsin, USA showing ratios of human mortality hazards (illegal kill plus other human hazards) and natural mortality hazard for wolves as estimated from radiotelemetry locations, 1981–2013

mortality sources. Sensitivity analysis indicated that partial compensation did not change with our choice of mean natural mortality, $\bar{n}_0$, and variance of natural mortality, $\sigma^2_{n_0}$, in an unexploited population (Table 2).

Table 2 Sensitivity analysis for mean posterior estimate of rate of compensatory mortality, C, and 95% credible interval from annual natural- and human-caused rates of mortality in Wisconsin's wolf population, 1981–2013, under different choices of mean natural mortality, $\bar{n}_0$, and variance of natural mortality, $\sigma^2_{n_0}$, in an unexploited population

$\bar{n}_0$	$\sigma^2_{n_o}$		
	0.021	0.031	0.041
0.25	0.180 (0.018, 0.331)	0.192 (0.032, 0.344)	0.198 (0.038, 0.352)
0.28	0.173 (0.011, 0.323)	0.186 (0.025, 0.338)	0.193 (0.033, 0.347)
0.31	0.165 (0.004, 0.314)	0.180 (0.021, 0.332)	0.189 (0.028, 0.342)

Discussion

Our survival analysis quantified cause-specific hazards experienced by radio-collared wolves, relationships between survival and spatial location, and an understanding of compensation in mortality sources in Wisconsin's wolf population and variation over time (1979–2013). We found that radio-collared wolves in Wisconsin had average annual survival of 76% with substantial differences throughout the year, since recolonization, and across the landscape. Because of the small sample size of radio-collared young wolves, our survival estimates are best understood for wolves ≥ 1 year old and within WHZs 1–5.

The annual survival rate estimated for Wisconsin's wolf population was similar to long-term annual survival in the Northern Rocky Mountain and Michigan, USA populations (75%; O'Neil et al. 2017; Smith et al. 2010) and similar to other wolf populations (Fuller et al. 2003). An analysis of radio-collared wolf carcasses in Wisconsin from 1979–2012 estimated a mortality rate of 8–28% (18% ± 10%) with two-thirds due to human causes, which overlapped our mean annual mortality estimate and our finding of ~ 60% of total mortality due to human causes (Treves et al. 2017). Our methods improved on those used by Treves et al. (2017) because we used known fate data from radio-collared wolves instead of wolf carcasses which are a biased sample (Stenglein et al. 2015b), and therefore we were able to get more precise survival and cause of mortality estimates for finer spatial and temporal scales.

Wisconsin wolves' mean hazard of death from illegal killing and natural mortality were equivalent, but had different patterns in time and space. Seasonally, both hazards peaked during winter, leading to overall higher risk of mortality relative to summer. Hazard of illegal kill for wolves peaked first in late November and early December aligning with Wisconsin's firearm deer hunting seasons when people are afield with weapons and often have enhanced visibility because of snow and reduced vegetative cover. Natural mortality hazard for wolves peaked slightly later in December through February, a time when wolves with disease are more likely to die because of physiological stress associated with snow and cold. This finding corroborates an analysis of Wisconsin's wolf carcasses recovered over the same time period that found carcasses with illegal kill and natural mortality

Table 1 Posterior means (and standard deviations) of parameter estimates from analysis of rate of compensation additivity in mortality causes for Wisconsin's gray wolf population over various time periods. See text for parameter names and descriptions

Years	$\bar{n}$	$\bar{h}$	σ_n	σ_h	b	ρ	ρ_c	C
1981–2013	0.116 (0.006)	0.132 (0.011)	0.033 (0.004)	0.063 (0.008)	− 0.115 (0.015)	− 0.466 (0.138)	− 0.351 (0.135)	0.186 (0.079)
1981–2003	0.130 (0.004)	0.114 (0.012)	0.020 (0.003)	0.055 (0.009)	− 0.098 (0.016)	0.280 (0.209)	0.378 (0.213)	− 0.141 (0.084)
2004–2013	0.084 (0.013)	0.176 (0.022)	0.038 (0.010)	0.068 (0.018)	− 0.130 (0.032)	− 0.738 (0.169)	− 0.607 (0.163)	0.351 (0.123)

causes were more common in winter than summer (Stenglein et al. 2015b). Our finding of reduced survival in winter aligns with the Michigan wildlife population (O'Neil et al. 2017), but differs from the northern Rocky Mountain wolf population where researchers found no seasonal difference (Smith et al. 2010) and the western Alps where survival was higher in winter compared to summer (Marucco et al. 2009). This is likely because summer can be a difficult time to find food in more mountainous regions leading to increased risk of natural mortality, but wolves in Wisconsin and Michigan do not encounter food scarcity during summer.

Periods of highest mortality overall occurred during early recolonization into the 1990s when wolf population growth demonstrated an Allee effect (Stenglein and Van Deelen 2016; Van Deelen 2009). Canine parvovirus and high illegal kill rates apparently reduced survival during the 1980s (Wydeven et al. 1995). Hazard from natural mortality stayed consistent from early recolonization into late 2000s when human-caused mortality hazards increased. The rise in other human mortality hazard in recent years reflects establishment of recreational wolf hunting seasons and decreased federal protection of wolves that allowed for increased use of lethal control measures to address livestock depredations (MacFarland and Wiedenhoeft 2013). Lethal control measures were periodically available since 2003, and our use of annual survival estimates does not include the temporal detail needed to understand interplay among illegal killing, lethal control measures, and the political environment (Olson et al. 2015).

Natural mortality hazard was highest where wolves have been reestablished the longest and illegal killing hazard was highest in eastern and southern wolf range where wolves were scarce and packs have not established until recently or at all. There was higher risk of human-caused as compared to natural-caused mortality in the northwest which may indicate portions of wolf range are saturated and displaying density dependence in survival (Mladenoff et al. 2009; Van Deelen 2009). Risk of natural mortality was low to nonexistent at the periphery of wolf range. This lack of uniformity spatially indicates a wolf population still in flux across its range (Stenglein et al. 2015a). Likely, the boundary of wolf range is a dynamic interplay between increasing mortality (this study) and decreasing habitat quality (Mladenoff et al. 1995; Mladenoff et al. 2009).

Spatially variable survival analysis included data from 1981 to 2013, smoothing across all temporal variation found previously. Future work with additional data should perform spatial survival analysis for a temporal subset of records to assess whether there are changes in spatial patterns of survival for different time periods. The results would help to specifically address the influence of legal harvest on spatial patterns in survival because harvest has occurred only in recent years. Additionally, there are few records at the extreme eastern portions of wolf range, so we recommend caution when making inference. We recommend concentrating inference on core areas where there are many records and where wolves have been recolonized the longest. There are many possible extensions of our analysis technique, including use of covariates other than time and spatial location, but our data were sparse when smoothing cause-specific hazards across time and space.

We took a novel approach to estimating wolf survival by not assuming wolves lost to follow-up (collar loss) were independently censored. To model endpoints, it was necessary to separate the model into an overall endpoint hazard and cause-specific probabilities of different events (Cross et al. 2015). This framework has many benefits. In more typical approaches to survival analysis, we would have assumed all radio-collared wolves with collar loss were not dead at the last observation and would have over-estimated the annual survival rate. Second, it is possible to include separate covariates that are estimated independently in model parts. We might assume different circumstances affect whether an endpoint occurred (hazard) versus the discrete cause associated with the event (cause-specific probability). Finally, the model allowed us to incorporate smoothing splines separately for hazard and endpoint probabilities (Crainiceanu et al. 2005; Cross et al. 2015). Smoothing splines were simple to implement and informative for visualizing how cumulative hazard, endpoint probabilities, and cause-specific hazards changed throughout the year, over three decades of wolf recovery, and across Wisconsin.

On average, each year more than half of Wisconsin's radio-collared wolves experienced the end of their record but less than half of those were confirmed deaths. The proportion of radio-collared wolves with collar loss was substantial, and therefore it was important to treat this endpoint separately and not assume these wolves were independently censored. The assumption of independent censoring is used commonly in survival analysis without assessment to the bias that it may impose in survival estimates (Murray 2006). An integrated population model from Wisconsin's wolf population (2003–2011) suggested 4% of wolves with collar loss may actually be dead wolves (Stenglein et al. 2015c). This may imply that recently there is slightly more mortality in the population than is accounted for from calculating survival rates solely from wolves found dead. This additional mortality could result from a portion of wolves with collar loss that were dead, but not recorded as such. However, during all years of recolonization, the percentage of wolves with collar loss that may have been dead wolves was $< 1\%$ (Stenglein et al. 2015c). This implies a survival rate of 75% instead of 76% and indicates that most wolves with collar loss are likely independently censored instead of dead.

Wisconsin's recolonizing wolf population likely experienced little or no compensation in earlier years of

recolonization, and partial compensation in mortality sources since 2003. Additivity in mortality sources in early wolf recovery promotes an understanding that a small population cannot support harvest as an additional source of mortality and remain stable (Anderson and Burnham 1976). Since 2003, the wolf population has expanded its range and numbers and may have reached local saturation in some areas as evidenced by higher natural mortality rates. The finding of partial compensation in mortality sources in later years of wolf recovery reflects theoretical understanding that potential for compensation is higher for populations near carrying capacity (Péron 2013; Sinclair and Pech 1996).

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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