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Intraspecific Variation in Prey Susceptibility Mediates the Consumptive Effect of Predation: A Case Study of Yellowstone Elk and Wolves

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INTRASPECIFIC VARIATION IN PREY SUSCEPTIBILITY MEDIATES THE CONSUMPTIVE EFFECT OF PREDATION: A CASE STUDY OF YELLOWSTONE ELK AND WOLVES

by

Lacy M. Smith

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ABSTRACT

Intraspecific Variation in Prey Susceptibility Mediates the Consumptive Effect of Predation: A Case Study of Yellowstone Elk and Wolves

by

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Predators have the potential to limit the abundance of their prey populations via their consumption of prey. Little is known, however, about how individual heterogeneity in prey susceptibility to predation mediates the consumptive effect of predators. The objective of my dissertation is to improve understanding of how such heterogeneity shapes the consumptive effect of a large predator on prey survival and population dynamics. I used data from northern Yellowstone National Park and adjacent Montana to evaluate 1) how predation by wolves (*Canis lupus*) influences age-specific mortality of adult female elk (*Cervus canadensis*; Chapter 2), 2) how the age-specific susceptibility of adult female elk to wolf predation changes under abiotic and biotic environmental conditions (Chapter 3), and 3) how wolf predation contributes to elk population dynamics over a 17-year period (Chapter 4). In Chapter 2, I show that old female elk (i.e., >14 years old) had a higher probability of being killed by wolves than dying from other

causes of mortality and that wolf predation of older elk was more additive than predation of younger (2-14 years old) female elk. In Chapter 3, I show that adult female elk had a higher probability of being killed by a wolf at younger ages during harsh environmental conditions (e.g., heavy snow) than they did in more mild conditions, although the survival of 2-9 year-old individuals was generally unaffected by the environmental conditions I analyzed. In Chapter 4, I show that mortality of adult (2-14 years old) female elk had the largest influence on elk population dynamics than did other demographic parameters, primarily due to non-wolf causes of mortality. The results from chapters 2-4 suggest that wolf predation reduced elk age-specific survival probability, primarily of the oldest individuals, which lowered the age of onset of actuarial senescence and contributed to the decrease in elk abundance. On average, these older individuals represented a minority of the population, and contributed the least to population growth rate. These results highlight the importance of accounting for stage-specific differences in prey susceptibility to predation when estimating the consumptive effect of a predator.

(186 pages)

PUBLIC ABSTRACT

Intraspecific Variation in Prey Susceptibility Mediates the Consumptive Effect of Predation: A Case Study of Yellowstone Elk and Wolves

Lacy M. Smith

The reintroduction of wolves (*Canis lupus*) to Yellowstone National Park starting in 1995 is an important case study for understanding the consequences of predation on a prey population. Simulation studies conducted prior to and shortly after wolf reintroduction predicted that wolf predation of elk (*Cervus canadensis*) would have a modest influence on elk abundance. Predation of elk by wolves has been well documented and elk have remained the primary prey for wolves despite a decline in elk abundance. I used two quantitative approaches to estimate the influence of wolf predation on adult female elk survival and abundance in northern Yellowstone and adjacent Montana during 2000-2017. My results suggest that, while wolves did kill adult female elk aged 2-14 years old, these elk generally had high survival. Elk were more likely to be killed by wolves as they aged. Wolf predation of adult female elk was primarily restricted to older individuals that generally comprised a small proportion of the total elk population. Harsh environmental conditions, such as heavy snow, increased mortality of adult elk, but elk aged 2-9 years old retained high survival regardless of the environmental conditions. The observed decline in elk abundance across the 17-year study was primarily due to mortality of 2-14 year-old elk that died due to causes

unrelated to wolves, including malnutrition, harvest, and other predators. I could not estimate the full impact of wolves on female elk abundance because of the lack of data on elk calf and yearling mortality. However, wolves likely had a smaller impact on the elk population than did non-wolf causes of elk mortality. These findings clarify how the impact of predation on a prey population may be limited by the age of the prey that are consumed and the relative importance of the predated individuals to the population (i.e., their reproductive potential).

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CHAPTER 1

INTRODUCTION

The consumption of prey by predators is a key process in community ecology and a mechanism by which predators suppress prey abundance. Classic theory about predator-prey interactions (e.g., Lotka-Volterra models) concerns the coupled abundance of a predator and their primary prey (Gotelli 2008). Understanding of predator-prey relationships has continued to advance via empirical and theoretical studies that estimate the impact of a predator on prey survival and abundance. Ecologists have recently drawn attention to the importance of individual-level heterogeneity (i.e. differences between individuals) for understanding predator-prey interactions (Pettorelli *et al.* 2015; Schmitz 2017). Within a prey population, individual-level heterogeneity is manifested as variation in susceptibility to predation. Variation in susceptibility to predation is important because it may alter the consumptive effect of a predator, e.g., by limiting predation to a subset of the prey population, by restricting predation to individuals that contribute relatively little to population growth rate, by removing individuals that are likely to die in the absence of predation, and by increasing prey mortality at late ages and thereby altering patterns of actuarial senescence.

HETEROGENEOUS SUSCEPTIBILITY TO PREDATION

Within a prey population, variation in susceptibility to predation among individuals is due to traits such as body size (Gosler *et al.* 1995), body condition (Murray 2002), coloration (Karpestam *et al.* 2016), and behavior (Hebblewhite & Merrill 2009; Barbosa *et al.* 2018; Moiron *et al.* 2020). An important question with respect to the role

of individual heterogeneity in predator-prey interactions is whether individual susceptibility to predation is age-invariant or whether susceptibility varies with age (Pettorelli *et al.* 2015). Shifts between ontogenetic stages or sizes that occur with increasing age can change an individual's susceptibility to predation (Paine 1976). Predators of large-bodied, dangerous prey often can only kill the youngest and oldest individuals or those in poor body condition (a trait often associated with age) (Mukherjee & Heithaus 2013). However, the distribution of susceptibility across ages may change through time because an individual's susceptibility to predation is likely a combination of their traits (e.g., body condition) and the environmental conditions they experience (Ng'weno *et al.* 2019; Moran *et al.* 2020; Sommer & Schmitz 2020). For example, older individuals may have reduced survival, especially at high density and in harsh winter conditions (Coulson *et al.* 2001). In addition, predators may switch the stage classes they select depending on environmental conditions (Wilmers *et al.* 2020). Yet few studies have examined how environmental conditions actually influence age-specific predation in the wild (but see Garrott *et al.* 2003, 2009; Furness & Reznick 2017; Moorad *et al.* 2019).

Individual variation in susceptibility to predation is often ignored or only accounted for across broad stage classes (e.g., juveniles and adults) despite evidence of increasing susceptibility within adult stages (DelGiudice *et al.* 2006). Individual variation in susceptibility within the adult stage may be important for estimating the impact of predation on prey population size if susceptible individuals comprise a subset of all adults and/or contribute little to population growth rate because of lower reproductive value and minimal representation in the population age structure.

POPULATION STAGE STRUCTURE

Prey populations with a demographic stage structure, in which susceptibility to predation varies by stage or age, contain a subset of stages that are resistant to the predator (Pettorelli *et al.* 2011). These resistant stages represent refugia for the prey population, possibly limiting the consumptive effect of the predator (Miller & Rudolf 2011; Nilsson *et al.* 2018). Prey that are killed across all life stages are more likely to be limited by predation than prey that are only killed during specific life stages (Roos *et al.* 2018). A resistant stage also helps stabilize predator-prey dynamics (Hastings 1983; Abrams & Walters 1996; Nilsson *et al.* 2018). The distinction between resistant and susceptible individuals has a long history in disease research because prevalence and infection rates change with age (Ahmad *et al.* 2001). Yet, individual-level heterogeneity is often omitted from studies of predator-prey interactions due to data limitations, despite widespread evidence of variation in susceptibility to predation, when evaluating the consumptive effect of predators (Pettorelli *et al.* 2015).

Understanding variation in susceptibility to predation by prey stage is also important because, in stage-structured populations, survival at each stage has a different impact on population growth rate (Caswell 2001). If individual susceptibility to predation depends on traits associated with age or stage, then the impact of predation on prey population growth rate depends on the importance of the susceptible individuals to the prey population. Therefore, a predator that kills only the oldest individuals should exert comparatively less consumptive force on a prey population compared to a predator that kills younger individuals with higher reproductive value (Hoy *et al.* 2015). There is also evidence that the importance of age-specific survival, relative to other vital rates, for

population dynamics can change through time (Koons *et al.* 2017), suggesting that the impact of predation on prey population dynamics may also change through time. Despite the importance of predator-caused mortality by stage class, few studies have assessed the influence of predator-caused mortality on prey population growth rate (Nilsen *et al.* 2009; Gervasi *et al.* 2012; Marescot *et al.* 2015).

Further, populations are often assumed to reach a stable stage distribution after a period of initially transient dynamics. If a prey population has a stable stage distribution, then the consumptive effect of a predator should be fixed through time. However, evidence suggests that population stage structure may not be stable through time (Clutton-Brock & Coulson 2002; Hoy *et al.* 2020). With ongoing changes in the environment due to climate, invasive species, habitat alteration, predator reestablishment, or wildlife and habitat management regimes, it may be unreasonable to expect that a population will reach and maintain a stable stage distribution (Tuljapurkar 1990). Therefore, the consumptive effect of a predator, including the degree of additive predation, is likely to change through time with changes in prey stage structure. However, little is known about associations between fluctuating prey stage structure and the impact of predation on prey.

ADDITIVE AND COMPENSATORY PREDATION

The magnitude of a predator's consumptive effect depends on the extent that predation is additive to other sources of prey mortality, removing individuals that would not have died in the absence of the predator. Compensatory predation substitutes for other causes of mortality, thereby exerting comparatively less impact on prey populations, as the "doomed surplus" would have died anyway (Errington 1946). The degree to which

predation is additive can vary across predator species (Griffin *et al.* 2011) and prey life stages (Payton *et al.* 2020). Some studies identify differences in additive predation between juvenile and adult stages, the ontogenetic shift for which stage refugia is defined (Miller & Rudolf 2011). Little is known, however, about the potential for additive predation to vary among adults despite predation risk often increasing as adults age and physically senesce. If predation is not uniformly additive across adults with varying susceptibility, then the extent that predation is additive across the adult population may depend on the frequency of susceptible adults within the population.

ACTUARIAL SENESCENCE

Age-specific survival may differ between causes of mortality (e.g., different predator species, or predator-caused mortality compared to mortality from non-predator causes) if susceptibility to each cause depends on a different degree of physiological deterioration. Therefore, one cause of mortality may select for more rapid actuarial senescence than other causes (Koons *et al.* 2014). Actuarial senescence is defined as an increase in mortality with increasing age and is a demographic outcome of an individual's physiology (Kirkwood 2015). A predator may drive more rapid actuarial senescence of the prey population when older individuals are more susceptible to predation than younger individuals (DelGiudice *et al.* 2002). Environmental hazards may alter patterns of actuarial senescence when older individuals are more susceptible to mortality due to their physiological condition (Williams & Day 2003). However, there is a lack evidence in the wild of the influence of age-selective predation in combination with environmental conditions on actuarial senescence relative to other causes of mortality.

KNOWLEDGE GAPS

There remain key gaps in our understanding of predator-prey interactions because studies generally do not account for individual heterogeneity and how the prey population is structured by individual heterogeneity, temporal variation in prey susceptibility, or variation in additive predation across stages of the prey population. First, variation in the inherent susceptibility of individuals to predation within the adult stage class is likely to have consequences for the consumptive effect of predators on prey population size because A) the proportion of the adult population susceptible to predation may be temporally dynamic; B) additive predation may vary by age; and C) the importance of adult survival to population growth rate should vary by age. Second, environmental conditions may change an individual's susceptibility and the proportion of the adult population susceptible to predation. The impact of predation on prey actuarial senescence and population size may therefore depend on which individuals are susceptible and the impact of these individuals on the population growth rate. Accounting for these integral aspects of predator-prey interactions will improve estimates of the impact of predation on prey survival and abundance, furthering our understanding of the consumptive effects of predators.

COMPETING-RISK MORTALITY

Competing-risk mortality provides an ideal framework to quantify the relative role of predation on the age-related survival patterns of a prey species (Heisey & Patterson 2006). The availability of GPS data provides an opportunity to determine date of mortality and, in many cases, assign a specific cause of mortality to each individual. When predation can be repeatedly assigned as the cause of death, competing-risk

mortality can distinguish between predator-specific mortality and other sources of mortality. Therefore, competing-risk mortality methods can be used to quantify the impact of a predator on a prey population because it provides an estimate of an age-specific vital rate that influences how populations respond to predation and environmental change over time.

DISSERTATION DATA CHAPTERS

To evaluate the consumptive effect of a top predator on a primary prey population within a large-scale, free-living system, I estimated the impact of wolf (*Canis lupus*) predation on adult female elk (*Cervus canadensis*) survival and population dynamics in northern Yellowstone National Park and adjacent Montana. I primarily focused on adult female elk survival because understanding the fate of adult females is important given their strong effect on population growth rate relative to males (Gaillard *et al.* 2000; Bonenfant *et al.* 2009) and the availability of long-term data. While calf survival was monitored from 2003-2005 (Barber-Meyer *et al.* 2008), adult survival was monitored from 2000 to 2008 and 2011 to 2017. This longer time frame is important because it includes peak wolf abundance as well as a reduced and stationary wolf abundance. This period also coincides with a decline in elk abundance as well as a slight increase in abundance in more recent years. I include year-round data because, while wolves are more proficient at killing adult female elk during winter and spring (Metz *et al.* 2012), elk die year-round and their survival may be influenced by the environmental conditions they experience across the year.

In chapter two, I provide the first comprehensive assessment of the influence of wolf predation on adult female elk survival in northern Yellowstone over a 17-year

period after wolf reestablishment. Earlier estimates of elk survival were conducted with lifetable, harvest, and radio-collar data restricted to pre-wolf or early post-wolf time periods and were generally limited to prime-aged individuals (Houston 1982; Vore 1990; Eberhardt 2002; White & Garrott 2005; Evans *et al.* 2006; Hamlin *et al.* 2009; Brodie *et al.* 2013). In contrast, I estimated survival and wolf-caused mortality risk of elk by each year of age (i.e., 2-24 years old) using radio-collared data in a competing-risk mortality framework. Further, I determined whether predation was additive or compensatory across two stage classes and how additive predation across the adult female population varied through time based on changes in population age structure.

In chapter three, I estimated the influence of biotic and abiotic environmental conditions (e.g., weather, wolf abundance) on age-specific survival and cause-specific mortality of adult female elk. I assessed how wolf predation and environmental conditions influence three key parameters underlying actuarial senescence: age at onset of senescence, shape of actuarial senescence (i.e., how steeply mortality increases with age), and mean life expectancy. To the best of my knowledge, these results are the first to demonstrate the impact of environmental conditions on predator-caused mortality across individual ages of adult prey.

In chapter four, I examined how female elk vital rates and age structure vary through time and influence variation in realized population growth rate over a 17-year period after wolf reestablishment. To obtain annual vital rate estimates, I combined all available sources of information on northern Yellowstone elk demography in an integrated population model. The consumptive effect of wolves on northern Yellowstone elk, particularly in relation to other predators including humans, cougars, and bears, is

debated. A strong consumptive effect of wolves assumes that wolves kill individuals that are important for elk population growth rate. Therefore, I decomposed adult elk survival into cause-specific mortality to estimate the contribution of wolf predation by stage class of adult elk to variation in elk population growth rate compared to other causes of mortality. This framework allowed me to compare how mortality of four stage classes influenced elk population dynamics, and how wolf predation influenced elk population dynamics relative to other sources of mortality for two adult stage classes of elk.

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CHAPTER 2

PREY STAGE STRUCTURE MEDIATES THE CONSUMPTIVE EFFECT OF A STAGE-SELECTIVE PREDATOR[1]

ABSTRACT

It is well established that pathogen-caused mortality depends on the fraction of the population that is susceptible to the pathogen, yet a similar understanding of predator-caused mortality is not well-developed. Although additive predation is commonly estimated for adult prey, little is known about how the fluctuating abundance of individuals resistant to predation due to their stage or size class ("stage refugia") alters the consumptive effect of a predator. I used data of wolves hunting elk in Yellowstone National Park to demonstrate that young adult female elk (2-14 years old) were resistant to wolf predation, whereas old adult female elk (>14 years old) were susceptible to wolf predation. Rather than a doomed surplus resulting in compensatory mortality, predation added to other sources of mortality for old adult females, whereas evidence suggested partial compensation for young adult females. These results demonstrate that variation in prey stage structure with respect to the relative frequency of susceptible and resistant individuals can mediate the consumptive effect of a stage-selective predator.

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INTRODUCTION

Community dynamics are classically predicted under the assumption that a population within a species is homogeneous (Nakazawa 2015), but individual-level heterogeneity may increase coexistence and stability of community dynamics (Miller & Rudolf 2011). It is well established that shifts between ontogenetic stages or sizes can change an individual's susceptibility to predation (Paine 1976), rendering a portion of the prey population resistant to a particular predator (Pettorelli *et al.* 2011). A resistant stage is important in prey populations because it can stabilize predator-prey dynamics (Hastings 1983; Abrams & Walters 1996; Nilsson *et al.* 2018). The distinction between resistant and susceptible individuals has a long history in disease research as it drives the rate of disease transmission and mortality. Predation can be considered in a similar light, whereby not all contacts between a predator and prey are likely to result in a predation event because some prey individuals are more resistant than others. Similar to epidemiological studies, in systems with stage-selective predators, predation may need to be standardized by prey age or stage to account for this variation in susceptibility (Ahmad *et al.* 2001). Although there is widespread evidence of variation in susceptibility to predation (Pettorelli *et al.* 2015), individual-level heterogeneity is often omitted from studies of predator-prey interactions due to data limitations.

Demographic stage structure, an important component of individual heterogeneity, provides stage refugia when individuals decrease their susceptibility to predation at certain life history stages, creating a subset of the population that is resistant to predation (Miller & Rudolf 2011; Nilsson *et al.* 2018). When stage structure is considered, classic consumer-resource models assume stable stage distributions (Rudolf

& Rasmussen 2013b), despite evidence for dynamic stage structures in nature (Hoy *et al.* 2020). If the proportion of a population occupying a stage refuge is time-variant, the impact of predation should change dynamically. The functional role of predators within communities is known to change based on the stage structure of their populations (Rudolf & Rasmussen 2013a,b). However, little is known about how fluctuations in prey stage structure influence the consumptive effects of predators, especially in free-living vertebrate systems.

The hypothesis that apex predators exert strong consumptive effects is defined by the extent that predation is additive to other sources of prey mortality (i.e., removing individuals that would not have died in the absence of the predator). Compensatory predation exerts comparatively less impact on prey populations, as the "doomed surplus" would have died from another cause of mortality in the absence of the predator (Errington 1946). While some studies distinguish between juvenile and adult stages, which is the ontogenetic shift for which stage refugia is typically defined (Miller & Rudolf 2011), it may be equally important to distinguish between young and old adults because predation risk often increases as individuals age and physically senesce. Despite the ubiquity of individual-level heterogeneity in susceptibility to predation (Pettorelli *et al.* 2015), field studies of predator-prey interactions often ignore the presence and proportion of the population in a stage refuge or only consider a subset (e.g., juveniles but not senescent adults). However, if predation is not equally additive across individuals with varying susceptibility, then combining susceptible and resistant individuals could bias inferences about the strength of consumptive effects especially if resistant and susceptible individuals have different reproductive values.

I tested for differences in the degree to which wolf (*Canis lupus*) predation added to or replaced other sources of mortality between prime-aged and senescent stages of adult elk (*Cervus canadensis*) using a long-term study in Yellowstone National Park and adjacent Montana. Here, wolf predation on elk is concentrated on calves (< 1-year-olds) and old adults (Smith *et al.* 2004; Wright *et al.* 2006; Metz *et al.* 2012). The reported effect of wolf predation on Yellowstone elk has ranged from primarily compensatory (Vucetich *et al.* 2005) to substantially additive (White *et al.* 2003; White & Garrott 2005). An analysis of more than 1,000 radio-collared adult female elk in 16 western North American populations exposed to wolves, including 194 elk in Yellowstone, found that wolf predation was additive (Brodie *et al.* 2013). However, these studies overlooked differences between susceptible and resistant adults.

Conceivably, predation is additive for young-adult females because they have high survival rates in the absence of wolves and at population levels well below carrying capacity (White *et al.* 2003; White & Garrott 2005). Wolf predation may be compensatory for old females because of reduced body condition and higher rates of mortality from other causes (Vucetich *et al.* 2005). Alternatively, wolf predation may be additive for old females because they represent a large and consistent majority of the adult female elk killed by wolves (Wright *et al.* 2006; MacNulty *et al.* 2020). As in other ungulate species, understanding the fate of adult females is paramount given their strong effects on population growth relative to males and juveniles (Gaillard *et al.* 2000; Bonenfant *et al.* 2009; Eacker *et al.* 2017), making them the most important segment of a population for measuring consumptive effects of predation.

MATERIALS AND METHODS

Study area

This study focused on the area encompassing the winter and summer ranges of the northern Yellowstone elk population (Houston 1982). The winter range is a 1520 km^2 area along the northern border of Yellowstone National Park encompassing low-elevation (1500-2600 m) grasslands and shrub steppes surrounding the Yellowstone River and its tributaries (Lemke *et al.* 1998). Approximately 65% (995 km^2) of the winter range is located within the park, and the remaining 35% (525 km^2) extends north of the park boundary. The summer range includes the majority of Yellowstone National Park and adjacent high-elevation areas (elevation range 2206-3091 m across all summer ranges) (Craighead *et al.* 1972; White *et al.* 2010). Elk were subjected to regulated harvest outside the park. Elk abundance decreased from 17,609 in winter 2000/2001 to 6,872 in winter 2016/2017 (MacNulty *et al.* 2020).

Wolves were reintroduced to Yellowstone in 1995-1997 (Bangs & Fritts 1996) and their distribution is concentrated in northern Yellowstone inside the park (Cassidy *et al.* 2020), ranging between 19 and 98 individuals (Smith *et al.* 2020). Wolf abundance in Montana adjacent to the Park ranged between 0 and 23 (Kohl 2019). Elk are the primary prey of wolves in Yellowstone, comprising over 80% of their diet during summer and spring and 94% or more of their diet in winter (Metz *et al.* 2020). Besides elk, wolves also consumed bison (*Bison bison*), deer (*Odocoileus spp.*), bighorn sheep (*Ovis canadensis*), moose (*Alces alces*), and pronghorn (*Antilocapra Americana*) (Metz *et al.* 2012, 2020). In addition to wolves, cougars (*Puma concolor*) are the other main top predator that kill elk of all age classes in the study area (Ruth *et al.* 2019), while grizzly

bears (*Ursus arctos*), black bears (*U. americanus*), and coyotes (*C. latrans*) predominantly prey on elk calf neonates (Barber-Meyer *et al.* 2008).

Data collection

This study includes data obtained from radio-collared elk, harvested elk, and wolf-killed, uncollared elk. Female elk (> 1 year old) were live-captured and radio-collared during 2000-2017. Yellowstone personnel determined their age at capture and, if they subsequently died, their cause of mortality. I also obtained ages of elk harvested during winter hunts in Montana between 1996-2009 from Montana Fish, Wildlife, and Parks. During field surveys from 1995-2016, Yellowstone personnel recovered elk carcasses (uncollared) and determined age and cause of mortality. Details on field methods are outlined in the sections that follow.

Aging elk

The age of live-captured and dead (harvest, recovered carcasses) adult female elk was determined using cementum analysis of an extracted incisor or upper canine (Hamlin *et al.* 2000). Cementum analysis was conducted by Matson's Laboratory (Manhattan, MT, USA). Birth year equaled the difference between the year of tooth extraction and the estimated age. I assigned each elk a birthdate of 1 June of their birth year, rounded to the nearest month based on the mean birth date of elk (27-29 May) (Barber-Meyer *et al.* 2008). I calculated age-at-death as the difference between birth year and death year. Each year in the following analyses corresponded to an elk year, from 1 June to 31 May.

Carcass data

Yellowstone personnel surveyed potential wolf-kill sites from 1995 to 2016 for carcasses of non-collared elk by monitoring locations of collared wolves. At least one wolf per pack was outfitted with either a very high frequency (VHF) or Global Positioning System (GPS) collar and additional survey methods are provided in Metz *et al.* 2011. Potential wolf-kill sites were visited by ground crews from 15 November through 14 December, 1 March through 30 March, and additionally from June through August for a subset of years (2004-2016). During the two winter study periods, observers monitoring wolf behavior also made direct observations of wolves killing elk. Additional carcasses were found opportunistically by ground or aircraft crews throughout the rest of the year. At each carcass Yellowstone personnel extracted a tooth for age estimation. For those mortalities not directly observed, Yellowstone personnel assessed whether the time of death corresponded with wolf GPS locations (see Metz *et al.* 2011) and whether wolves had likely made the kill based on evidence at the carcass site (see Mech *et al.* 2001). Using ages of 616 wolf-killed females aged two years and older, I estimated the mean age of adult female elk killed by wolves and the age distribution of wolf kills. Sampling of harvested elk carcasses is described under age structure.

Radio-collar data

Yellowstone personnel monitored the survival of 281 radio-collared female elk. From 2000 to 2003, 2005 to 2006, and 2011 to 2017, female elk (> 1 year old) were captured using net guns from helicopters (Hawkins and Powers, Greybull, Wyoming, USA; Leading Edge Aviation, Clarkson, WA, USA) or ground-darting and fitted with VHF or GPS collars. GPS collars included Telonics (Mesa, Arizona, USA), Advanced

Telemetry Systems Inc. (Isanti, Minnesota, USA), and Vectronic Aerospace GmbH (Berlin, Germany). Elk were captured and handled in accordance with applicable guidelines from the American Society of Mammalogists (Sikes 2016) and approved by the National Park Service Institutional Animal Care and Use Committee. VHF-collared elk were tracked with ground-based and aerial radio telemetry one to four times per month. GPS collars were programmed to collect locations at 1-6-hour intervals depending on the season, collar type, and other study objectives. The tracking period started immediately after capture in 2000 and ended in 2008 due to logistical issues (note that tracking extended two years past the 2006 captures). Both captures and tracking resumed in 2011 and continued through May of 2017. The status (alive/censored/dead) and location of each elk were tracked until the collar failed or was removed, or until the elk died. When possible, failing collars were replaced.

VHF and GPS collars were equipped with a mortality sensor, and Yellowstone personnel conducted field necropsies of dead elk to determine cause of death based on the carcass condition and location, blood trails, and evidence of predators including tracks, scat, carcass caching, bed sites, and wounds (Evans *et al.* 2006). Each elk was assigned a date of death based on timing of mortality signal or condition of the carcass. When months elapsed between the most recent resighting and a mortality, I assigned a death date halfway between when the mortality signal was heard and when the elk was last sighted (i.e., the midpoint rule).

A cause of death was recorded if inspection of the carcass in the field provided sufficient evidence to determine predator-specific or a non-predator cause of death. Hunters returned collars to the National Park Service from collared elk they harvested. I

classified all non-wolf and non-human caused mortalities as 'other-caused mortality' because the analysis focused on the effect of wolf predation on elk survival (Table 2-1). Cause of death was unknown for 21 non-human caused mortalities. I used the frequency of known wolf-caused mortalities occurring inside (80% of 51) and outside (47% of 19) wolf pack boundaries to classify these unknown mortalities as either wolf-caused or other-caused according to whether the unknown mortalities occurred inside (N = 10) or outside (N = 11) wolf pack boundaries. Each mortality was located with respect to wolf pack boundaries of the corresponding year of death. I used wolf pack boundaries estimated with minimum convex polygons of wolf tracking data and provided by the Yellowstone Wolf Project (available at https://www.nps.gov/yell/learn/nature/wolf-reports.htm). I randomly assigned wolves as the cause of death for 80% of 10 unknown mortalities inside wolf pack boundaries (N = 8) and 47% of 11 unknown mortalities outside wolf pack boundaries (N = 5). I classified the remaining 8 unknown mortalities as other-caused.

Data analysis

Age structure

I described the age structure of the adult female elk population using data from a prior reconstruction analysis of the same elk population (Hoy *et al.* 2020). This analysis used dead-recovery data from hunter-harvested elk between 1996-2009 (N=10,133) as well as elk that died of natural causes and were detected during ground and aerial surveys between 1995-2015 (N=3,078) (e.g., Fryxell *et al.* 1988, 1999). For each elk, Hoy *et al.* (2020) obtained age-of-death and year-of-death data to calculate the minimum number of

elk alive in each age per year. The minimum total number alive (regardless of adult age) each year was estimated from uncorrected annual aerial counts (Lemke *et al.* 1998). Reconstruction analyses are sensitive to the lifespan of the species; therefore, the analysis did not extend beyond 2009 because of the large number of individuals still alive in recent years. For further details see Hoy *et al.* (2020). For elk aged 2 years and older, I estimated the proportion of elk of each age in the population across the 15-year period (1995-2009).

Cause-specific mortality by age

To determine the effect of wolf predation on elk survival according to elk age, I used the elk survival data in a competing risk mortality analysis. In this analysis, the cause-specific mortality is a joint probability of dying before a given time and by a given cause, where cause-specific mortality probabilities are mutually exclusive (Heisey & Patterson 2006; Wolfe *et al.* 2015). I fit a fully parametric, continuous-time multistate model with two mortality states (wolves and other) and a Weibull distribution (R package Flexsurv) (Jackson 2016).

In the multistate model, elk transitioned from an alive state to one mortality state or remained alive (Fig. 2-1). Elk that were harvested (N=19) or hit by vehicles (N=2) were censored upon death to focus on non-human causes of mortality (N=91); thus, I analyzed competing risk mortality in the absence of human-caused mortality. Transitions were not allowed between mortality states or from a mortality state to the alive state. I estimated the instantaneous rate of transition (transition intensity) for elk survival and the transition to each mortality state. I constructed the model with elk age as the time scale to estimate the age-specific probability of mortality by cause. Yearling data were excluded

from analysis because there were only six elk marked as yearlings; thus, I focused on elk ≥ 2 years-old (inclusive of the captured yearlings once they became two). Using these results, I also derived elk survival and mortality probabilities between each age (spanning 1 June through 31 May; e.g., the probability of survival to age 5 given that an individual survived to age 4; $S_{a \rightarrow a+1} = S_{a+1}/S_a$) and estimated corresponding standard errors using the delta method (Seber 1982). The analysis included elk that lived through a three-year monitoring gap (2008-2010) as well as elk that went missing and were later found dead. Elk that died during the monitoring gap were treated as alive and censored from the analysis at the start of the monitoring gap. I excluded the gap years from the analysis for elk that lived through the monitoring gap (i.e., they were right-censored and then re-entered).

Impact of wolf-caused mortality on annual survival

For annual cause-specific mortality rates, I used the collared elk data to estimate cumulative incidence functions in a competing risks framework (csm function in R package wild1; Heisey & Patterson 2006). I estimated unique, annual survival and cause-specific mortality probabilities by 'age class', with a breakpoint between resistant elk and susceptible elk determined from the other analyses described above (see Results), as well as all ages combined. Sample size was insufficient to include more than two age classes in the analysis. I estimated annual survival and mortality probabilities for 2000-2003, 2005-2007, and 2011-2016. The years of the monitoring gap were excluded because I did not have date of death and cause of death data for elk that died during that period, and year 2004 was excluded because no collared elk in the resistant age class died that year.

I assessed the impact of annual wolf-caused mortality on annual overall survival using linear models and a corrected slope (model slope divided by intercept) of the relationship between the two probabilities (Brodie *et al.* 2013). Slopes less than -1 indicated additive predation, slopes equal to 0 indicated compensatory predation, and slopes between -1 and 0 indicated the proportion of predation that was additive (i.e., partially additive predation). To account for estimated uncertainty in the annual survival and wolf-caused mortality probabilities, I fit regressions to Monte Carlo realizations within the range of uncertainty for each annual probability (Wolfe *et al.* 2015). I constructed a beta distribution using moment matching for each survival and wolf-caused mortality probability and then sampled 1,000 realizations from each distribution per year. On each Monte Carlo iteration, I fit a (corrected) slope of the relationship between the simulated survival and wolf-caused mortality probabilities, resulting in a total of 1,000 slope estimates. I then assessed whether the 95% confidence intervals of the simulated slopes overlapped -1 and 0, respectively, to determine whether I could reject the additive or compensatory hypotheses of predation. I tested for additive predation in the 'resistant' and 'susceptible' age classes to determine if the degree to which predation was additive varied by elk stage.

I then stage-standardized these slope estimates to account for potential and known variation in the age structure of the elk population. These standardized slopes (n=1,000) were calculated as,

$$\beta_{standardized} = \beta_s * P_s + \beta_r * P_r \quad (1)$$

where β is the slope estimate and P is the proportion of susceptible (s) and resistant (r) elk. The values of P_s and P_r were fixed for all 1,000 slope estimates. To compare across

a range of susceptible elk in the population, I repeated this calculation based on a standard population of 0 to 50% susceptible individuals in 0.05 increments. I calculated the mean and 95% CI for each set of 1,000 slopes. These estimates allowed us to determine how additive predation across the adult population could vary with changes in the age structure (e.g., a population with a small proportion of old individuals compared to a population with a higher proportion of old individuals).

In addition, I standardized slopes based on the age structure estimates of the annual proportion of elk in the adult female population exceeding 14 years-old (susceptible elk) from 1995 to 2009 (Hoy *et al.* 2020). Thus, I calculated annual estimates of additive predation across the population according to empirically estimated population age structure.

RESULTS

Age structure and selective predation

Across a 15-year period (1995-2009), the age distribution of adult (≥ 2-year-old) females in the northern Yellowstone elk population was skewed towards younger individuals (Fig. 2-2a). The median age was 6 years-old (range 2 – 26; mean 7.2) and 93% of individuals were 14 years old or younger. During this same period, the age distribution of wolf-killed females was skewed towards older individuals (Fig. 2-2a). Among radio-collared and uncollared females killed by wolves (1995-2009), the median age was 15 years (range: 2 – 26) and 59.2% were older than 14 years. Among radio-collared females killed by wolves (2000-2016), the median age was 15.6 years (range: 6 – 24) and 55% were older than 14 years (Fig. 2-2a).

The age structure of the elk population varied from 1995 to 2009, with a higher frequency of younger individuals in 1995 and a higher frequency of older individuals in 2009 (Figs 2-2b, 2-2c). This overall trend was comprised of two distinct waves of aging caused by reduced recruitment during the late-1990's and early 2000's. By contrast, the age distribution of all wolf-killed females (collared and uncollared) varied little across the same period (Fig. 2-2c) and through 2016 (Fig. 2-3). Increasing overlap in the age distributions of elk killed by wolves and elk in the population at large indicates that the adult female elk population was increasingly susceptible to wolf predation from 1995 to 2009.

Cause-specific mortality by age

Of the 281 radio-collared adult females, 63 were killed by wolves and 28 were killed by other causes, including malnutrition and other predators (Table 2-1). I right-censored 21 elk with human-caused mortality (harvest or vehicle strike) at the time of mortality. Elk 2-8 years-old maintained high survival (≥ 0.95) despite exposure to wolves (Fig. 2-4). The instantaneous risk of wolf-caused mortality was zero (95% CI: 0.00-0.01) for 2-5 year-olds, 0.01 to 0.05 (95% CI: 0.00-0.09) for 6-9 year-olds, 0.08 to 0.29 (95% CI: 0.04-0.38) for 10-14 year-olds and 0.37 to 0.70 (95% CI: 0.26-0.78) for 15-24 year-olds (see Table 2-2 for model parameter estimates). The plateau in mortality probability (Fig. 2-4) may be due to low sample size of elk over 20 years old. Elk had a greater than 0.80 probability of surviving between consecutive ages until they reached 15 years old (Table 2-3). Annual survival probability during the late teenage years remained high, but declined from 0.47 to 0.27 after age 20 (Table 2-3). During their twenties, elk had a 0.34

to 0.45 annual probability of being killed by a wolf and 0.19 to 0.28 annual probability of being killed by another cause (Table 2-3).

Elk not killed by wolves or other causes (i.e., those individuals censored at end of study period, at time of harvest, or when collar failed or individual went missing) ranged in age from 2 to 23 years-old. The median life expectancy of elk, given that they lived to two years, was 17.5 years (95% CI: 16.7 – 18.2 years). If 2-9 year-old elk are largely resistant to wolves, the results indicate that the average adult female elk was only susceptible to wolves (mortality risk > 0.05) for 8.5 years, or 55% of her adult life.

Strength of additive wolf predation

Based on the foregoing evidence for the median age of elk killed by wolves, I used age 14 as the breakpoint between resistant elk (2-14 years) and susceptible elk (>14 years). Annual rates of wolf-caused mortality were higher and more variable for susceptible elk compared to resistant elk (Fig. 2-5, Table 2-4).

After accounting for uncertainty using Monte Carlo sampling, the degree to which wolf predation added to (or compensated for) other sources of mortality in affecting survival differed between resistant and susceptible stages of elk. The mean simulated slope of the relationship between wolf-caused mortality and annual survival for resistant elk was -0.45 (Fig. 2-6a), compared to -0.74 for susceptible elk (Fig. 2-6b), suggesting that a greater proportion of wolf predation was additive for susceptible elk compared to resistant elk. Moreover, the upper confidence interval of the slope did not overlap zero for susceptible elk (CI: -1.45, -0.01; Fig. 2-6b), indicating that compensatory predation was statistically unlikely for this age class, whereas it was statistically plausible for the younger, resistant age class (CI: -1.27, 0.05; Fig. 2-6a).

The mean slope across all elk ages (-0.66; CI: -1.32, -0.15; Fig. 2-6c) indicated that wolf predation was generally additive across all elk ages combined. Given a standard population of 10% susceptible elk (Raithel *et al.* 2007), the mean slope was -0.48 and the upper confidence interval of the slope excluded zero (CI: -1.21, -0.01; Fig. 2-6d). With a standard population of 40% susceptible elk, the mean slope was -0.56 and the upper confidence interval of the slope excluded zero (CI: -1.09, -0.11; Fig. 2-6d).

Adjusting the frequency of susceptible elk in the population from 0 to 50%, decreased the mean slope from -0.45 (CI: -1.27, 0.05) to -0.59 (CI: -1.20, -0.11), indicating an increased degree of additive predation in the population with more susceptible elk (Fig. 2-6e). There was more evidence of additive predation (slope 95% CI excluding 0) once susceptible elk comprised ≥ 10% of the population (Fig. 2-6e).

The proportion of elk older than 14 years-old in the population was fairly constant from the late 1990's to the early 2000's at < 0.09, and subsequently increased to 0.26 in 2009 (Fig. 2-7a). Given the estimated annual age structure of the adult female population from 1995 to 2009 and the associated mean slopes, additive predation was increasingly likely from 2003 to 2009 (Fig. 2-7b).

DISCUSSION

The consumptive effect of predation depends on the stage structure of the prey population when one or more stages are resistant to predation and serve as stage refugia (Miller & Rudolf 2011). Here, I demonstrated that heterogeneity in predation risk across stages can mediate the consumptive effect of a predator and that ignoring such heterogeneity and the frequency of susceptible prey can bias inferences about the consumptive effect. Moreover, I found that the degree to which predation is additive

depends on the susceptibility (stage class) of the prey, with more additive predation of susceptible than resistant prey. Further, these results highlight how temporal variation in the frequency of susceptible prey alters the consumptive effect of predation through time.

Adult female elk were largely resistant to wolf predation until their mid-teens when susceptibility increased. When I did not distinguish between susceptible and resistant elk, the results concurred with Brodie *et al.* (2013), suggesting that wolf predation was additive when adult female stages were conflated. Instead, the stage-specific analysis revealed that wolf predation was more additive for susceptible elk than it was for resistant elk. Thus, not controlling for stage heterogeneity in predation susceptibility (Fig. 2-6c) concealed how predation was partially compensated by other sources of mortality in resistant elk. Previous conclusions that wolf predation of Yellowstone elk was additive (White *et al.* 2003; White & Garrott 2005) or compensatory (Vucetich *et al.* 2005) neither considered the continuum of partial additivity or stage structure nor estimated the relationship between elk survival and wolf-caused mortality.

White and Garrott (2005) suggested that wolf predation of young adult (resistant) elk was strongly additive because of high survival rates in the absence of harvest and predation and an elk population that may have been below carrying capacity. However, all organisms, regardless of prevailing population density, are subject to potential compensatory mortality, so long as the focal source of mortality remains less than all other sources combined (Burnham & Anderson 1984). Few resistant elk were killed by wolves, likely because wolves were unable to overcome their antipredator defenses such as confrontation, grouping, and flight (MacNulty *et al.* 2007; Mech *et al.* 2015). Given their success in hunting northern Yellowstone adult elk (males and females) is less than

10% (MacNulty *et al.* 2012), wolves must target elk with a reduced capacity for defense. Likewise, cougars target younger mule deer if they are diseased (Krumm *et al.* 2009) as well as older bighorn sheep (Festa-Bianchet *et al.* 2006). I speculate that prime-aged elk killed by wolves may have also suffered from conditions (e.g., injuries, poor nutritional condition) that increased their vulnerability to mortality in general, further resulting in wolf predation towards the compensatory end of the spectrum.

The finding that wolves largely had an additive effect on the survival of susceptible elk contrasts with the "doomed surplus" hypothesis (Errington 1956), whereby predators remove excess prey that would have died from other causes (e.g., starvation), thus having a compensatory effect on overall survival. I do acknowledge that the old stage-class is broad and that the effect of wolf predation is likely less additive for 20-year-old elk than it is for 15-year-old elk. Predation on the oldest elk (i.e., > 20 years old) is potentially compensatory because there is a decreased survival probability between subsequent ages (Table 2-3). Unfortunately, I lack adequate data to separate elk into narrower stage classes to determine if predation becomes more compensatory at the oldest ages. Nonetheless, old elk experienced additive wolf predation when it was severe enough to overcome rates of dying from other causes (which it was in most years of the radio-collar study).

Old elk likely contribute the least to population growth because of reproductive senescence and rarity in the population (i.e., low reproductive value). Elk 15 years and older do not contribute substantially to recruitment relative to younger elk (Wright *et al.* 2006; Raithel *et al.* 2007). Predation of individuals with low reproductive value may moderate the impact predators have on prey populations (Wright *et al.* 2006; Hoy *et al.*

2015). However, prey with lower reproductive value are more important to the population when they comprise a relatively large proportion of the population (sensu the definition of fitness sensitivities to vital rates; Caswell 2001). Thus, the impact of predation may also depend on the frequency of susceptible prey.

My findings suggest that the effect of wolf predation is strongest on individuals that often occur in low frequency. When additive predation is primarily limited to a small subset of prey, the predator's consumptive effect should be weak. In disease research, populations often have high survival and reproductive success when few individuals are susceptible (Beldomenico & Begon 2009). Likewise, predators may have a limited consumptive effect when a large proportion of the prey population is resistant. Resistant elk comprised the majority of the female population, but their proportion decreased through time with complementary increases in susceptible individuals.

The level of additive predation across the prey population should fluctuate with changes in the frequency of susceptible prey. My results demonstrate that observed changes in elk stage structure may correspond to variation in the proportion of the prey population that is subject to additive predation. Such changes in stage structure may be driven by recruitment pulses. In long-lived species, reduced recruitment shifts populations to an older stage structure (Wheeler *et al.* 2003; Browne & Hecnar 2007). Given heavy predation of elk calves by wolves, cougars, and grizzly and black bears in Yellowstone (Smith *et al.* 2004; Barber-Meyer *et al.* 2008; Ruth *et al.* 2019), predation may contribute to the pattern of elk recruitment and drive changes in future stage structure. Heavy calf predation or other lapse in recruitment in a given year or series of years will increase the relative frequency of susceptible elk > 14 years in the future (Fig.

2-2b), leading to a higher proportion of additive wolf predation in later years. As such, the early-life impacts of predators on prey recruitment could also affect late-life mortality dynamics and the net impact of predator consumptive effects.

Human harvest of wild populations may also contribute to changes in stage structure. Selective harvest of large (old) fish, coral, and male ungulates shifts populations to a younger size (age) structure (Bianchi *et al.* 2000; Tsounis *et al.* 2006; Monteith *et al.* 2013). Harvest of female elk is concentrated on elk that are younger (median 9 years-old) than those that wolves kill (Evans *et al.* 2006; Wright *et al.* 2006), and variation in female harvest may contribute to fluctuations in stage structure via removal of younger individuals with higher reproductive potential. To manage for a wolf-resistant elk population, managers should aim for a younger adult stage structure by minimizing harvest and increasing survival of prime-aged females, yearlings, and calves.

Conclusion

My study highlights the importance of distinguishing between susceptible and resistant individuals when estimating the consumptive effect of predation, particularly because the level of additive predation can differ by susceptibility. In disease research, populations are routinely age-standardized for analysis because disease is age-dependent and the underlying population age structure influences disease dynamics (Ahmad *et al.* 2001). But prior research on free-living predator-prey systems has not accounted for individual heterogeneity in predation risk (reviewed by Pettorelli *et al.* 2015) and combining susceptible and resistant individuals is prone to bias inferences in the absence of standardization. Accounting for the frequency of susceptible prey is particularly important because their frequency may shift through time and space depending on life

history or prior harvest and predation pressure. My results highlight the fallacy of a dichotomy of predation as either additive or compensatory because the proportion of predation that is additive should change temporally and spatially when populations are not at a stable stage distribution.

An important insight of this study is that the magnitude of a predator's consumptive effect appears to fluctuate with long-term changes in the population frequency of susceptible prey individuals. Predators may have a limited consumptive effect on prey populations if susceptible prey are infrequent, despite additive predation of these individuals. While not all susceptible prey are "doomed," their early demise due to predation, rather than living longer before dying by another cause of mortality, may be of minimal consequence to the population. But as susceptible prey comprise an increasing proportion of a population undergoing transient change in the stage structure (Caswell 2007), their early demise may decrease population growth. Accounting for individual heterogeneity in predation risk is therefore critical for understanding the community-level consequences of predator-prey interactions (Miller & Rudolf 2011).

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TABLES AND FIGURES

Table 2-1 Causes of mortality for radio-collared adult female elk in northern Yellowstone and adjacent Montana, 2000-2016 (winters 2000/2001 to through 2016/2017). Elk with human-caused mortality (harvest or vehicle strike) were censored from mortality analyses at time of death.

			Other Natural-Caused				
	Wolf	Human-Caused	Cougar	Grizzly Bear	Malnutrition	Unknown Non-Predation	Total
2000	4	5	1	0	0	0	10
2001	3	4	0	0	0	0	7
2002	4	1	0	0	3	1	9
2003	7	2	0	1	0	3	13
2004	3	1	0	0	0	1	5
2005	2	1	0	0	1	0	4
2006	2	0	0	0	0	2	4
2007	7	0	0	0	1	0	8
2011	6	0	0	0	0	1	7
2012	5	0	1	0	0	0	6
2013	7	0	0	0	0	1	8
2014	3	2	1	0	1	1	8
2015	5	3	1	0	0	5	14
2016	5	2	0	0	1	1	9
Total	63	21	4	1	7	16	112
%	0.56	0.19	0.04	0.01	0.06	0.14	-

Table 2-2 Parameter estimates for competing risk Weibull model of adult female elk survival in northern Yellowstone and adjacent Montana. The Weibull shape and scale parameters are used to derive survival estimates. Transition refers to the transition from alive to one of two possible mortality states: wolf predation or other causes and is a covariate on both the shape (shape(transition)) and scale (transition) parameters.

Parameter	Estimate	L 95% CI	U 95% CI
shape	3.288	1.91	5.67
scale	15.15	13.33	17.22
transition	0.15	0.06	0.23
shape(transition)	0.32	-0.05	0.69

Table 2-3 Interval survival and cause-specific mortality probabilities for adult female elk in northern Yellowstone and adjacent Montana, 2000-2016.

Age	Survival Probability	95% CI	Wolf-Caused Mortality Probability	95% CI	Other-Caused Mortality Probability	95% CI
2 to 3	1.00	(1.00, 1.00)	0.00	(-0.20, 0.21)	0.00	(-0.20, 0.20)
3 to 4	1.00	(1.00, 1.00)	0.00	(-0.20, 0.20)	0.00	(-0.20, 0.20)
4 to 5	1.00	(0.99, 1.00)	0.00	(-0.20, 0.20)	0.00	(-0.20, 0.20)
5 to 6	1.00	(0.99, 1.00)	0.00	(-0.19, 0.20)	0.00	(-0.19, 0.19)
6 to 7	0.99	(0.98, 1.00)	0.01	(-0.18, 0.19)	0.00	(-0.19, 0.19)
7 to 8	0.99	(0.96, 1.01)	0.01	(-0.16, 0.19)	0.00	(-0.17, 0.18)
8 to 9	0.98	(0.94, 1.01)	0.02	(-0.15, 0.18)	0.00	(-0.16, 0.17)
9 to 10	0.96	(0.92, 1.01)	0.03	(-0.12, 0.18)	0.01	(-0.14, 0.15)
10 to 11	0.95	(0.88, 1.02)	0.04	(-0.10, 0.18)	0.01	(-0.12, 0.14)
11 to 12	0.93	(0.83, 1.03)	0.06	(-0.08, 0.19)	0.02	(-0.10, 0.13)
12 to 13	0.90	(0.78, 1.03)	0.07	(-0.06, 0.21)	0.02	(-0.08, 0.13)
13 to 14	0.87	(0.72, 1.02)	0.09	(-0.05, 0.24)	0.04	(-0.06, 0.13)
14 to 15	0.83	(0.65, 1.02)	0.12	(-0.03, 0.27)	0.05	(-0.05, 0.15)
15 to 16	0.78	(0.57, 1.00)	0.15	(-0.02, 0.31)	0.07	(-0.04, 0.18)
16 to 17	0.73	(0.49, 0.97)	0.18	(0.00, 0.36)	0.09	(-0.03, 0.21)
17 to 18	0.67	(0.40, 0.94)	0.21	(0.02, 0.40)	0.12	(-0.01, 0.25)
18 to 19	0.60	(0.31, 0.90)	0.24	(0.03, 0.45)	0.15	(0.00, 0.30)
19 to 20	0.53	(0.19, 0.88)	0.28	(0.05, 0.50)	0.19	(0.02, 0.36)
20 to 21	0.46	(0.07, 0.85)	0.31	(0.06, 0.55)	0.23	(0.04, 0.43)
21 to 22	0.39	(-0.02, 0.79)	0.34	(0.09, 0.58)	0.28	(0.07, 0.48)
22 to 23	0.32	(-0.07, 0.70)	0.36	(0.14, 0.59)	0.32	(0.12, 0.52)
23 to 24	0.25	(-0.08, 0.58)	0.39	(0.20, 0.57)	0.37	(0.19, 0.55)

Table 2-4 Annual estimates of adult female elk survival, wolf-caused mortality, and other-caused mortality in northern Yellowstone and adjacent Montana. There was a gap in monitoring from 2008 to 2010 and no mortality of prime-aged elk in 2004 so these years were excluded from analysis. Percent of the sample was calculated based on elk that remained alive each year, whereas sample size (N) includes elk that remained alive and died each year.

	elk ≤ 14 years old							elk > 14 years old						
year	wolf-caused mortality	wolf-caused mortality SE	other-caused mortality	other-caused mortality SE	survival	N	% of sample	wolf-caused mortality	wolf-caused mortality SE	other-caused mortality	other-caused mortality SE	survival	N	% of sample
2000	0.06	0.04	0.00	0.00	0.94	47	0.82	0.15	0.10	0.08	0.07	0.77	13	0.18
2001	0.06	0.04	0.00	0.00	0.94	50	0.79	0.09	0.09	0.00	0.00	0.91	14	0.21
2002	0.05	0.03	0.02	0.02	0.93	54	0.84	0.20	0.13	0.29	0.14	0.51	15	0.16
2003	0.14	0.06	0.03	0.03	0.83	37	0.82	0.17	0.11	0.28	0.14	0.56	12	0.18
2005	0.04	0.03	0.00	0.00	0.96	55	0.85	0.00	0.00	0.13	0.12	0.88	10	0.15
2006	0.05	0.04	0.00	0.00	0.95	41	0.72	0.00	0.00	0.12	0.08	0.88	17	0.28
2007	0.31	0.11	0.10	0.05	0.84	26	0.59	0.47	0.20	0.07	0.07	0.46	23	0.41
2011	0.09	0.06	0.00	0.00	0.91	33	0.62	0.27	0.12	0.06	0.06	0.67	24	0.38
2012	0.06	0.04	0.02	0.02	0.92	44	0.68	0.14	0.08	0.00	0.00	0.86	22	0.32
2013	0.03	0.02	0.00	0.00	0.97	58	0.81	0.27	0.10	0.05	0.05	0.68	19	0.19
2014	0.02	0.02	0.00	0.00	0.98	62	0.73	0.09	0.06	0.12	0.07	0.79	27	0.27
2015	0.03	0.02	0.00	0.00	0.97	76	0.82	0.15	0.08	0.28	0.10	0.57	25	0.18
2016	0.03	0.02	0.01	0.01	0.96	74	0.78	0.15	0.08	0.04	0.04	0.80	24	0.22

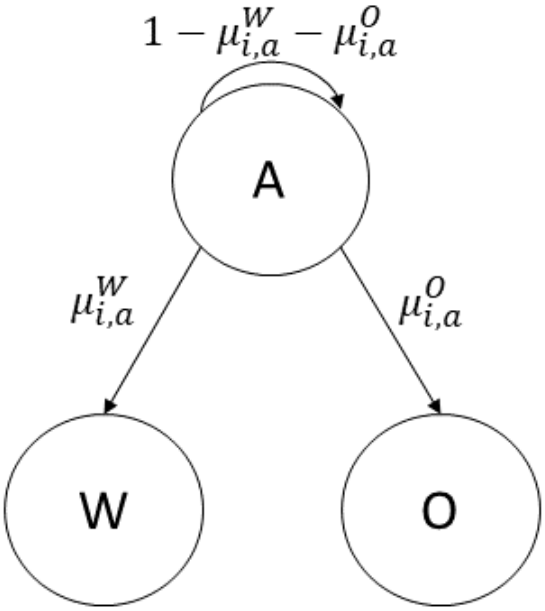

Figure 2-1 The demographic transitions of female elk (≥ 2 years old) in norther Yellowstone and adjacent Montana remaining alive (A), being killed by a wolf (W), or dying from a non-wolf, non-human cause (O). $\mu^{k}_{i,a}$ denotes the cause-specific probability of mortality per individual *i* at age *a* (2 to 24 years old).

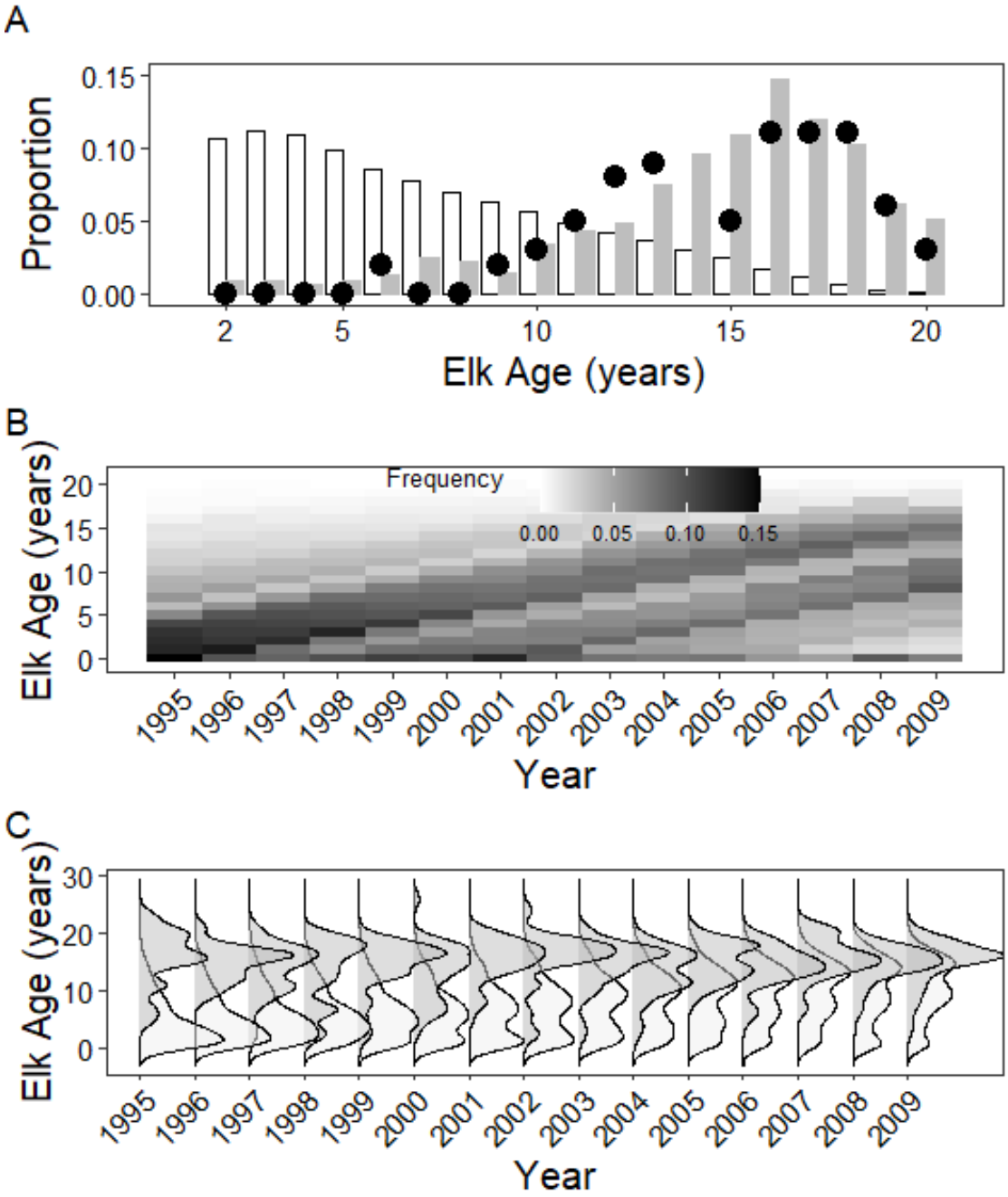

Figure 2-2 Comparison of the adult (≥ 2 years-old) female elk age distribution summarized from hunter harvest and recovered carcass data in northern Yellowstone and adjacent Montana (A, white bars; 1995-2009), wolf-killed carcasses (radio-collared and uncollared) in northern Yellowstone (A, grey bars; 1995-2009), and mortalities of radio-collared elk (A, black dots; 2000-2016). The frequency of female elk by age in northern Yellowstone from 1995 to 2009 (B) indicated an increasingly older age structure through time. Annual density of female elk in the population in northern Yellowstone (white) and of wolf-killed female elk carcasses (radio-collared and uncollared; grey; C).

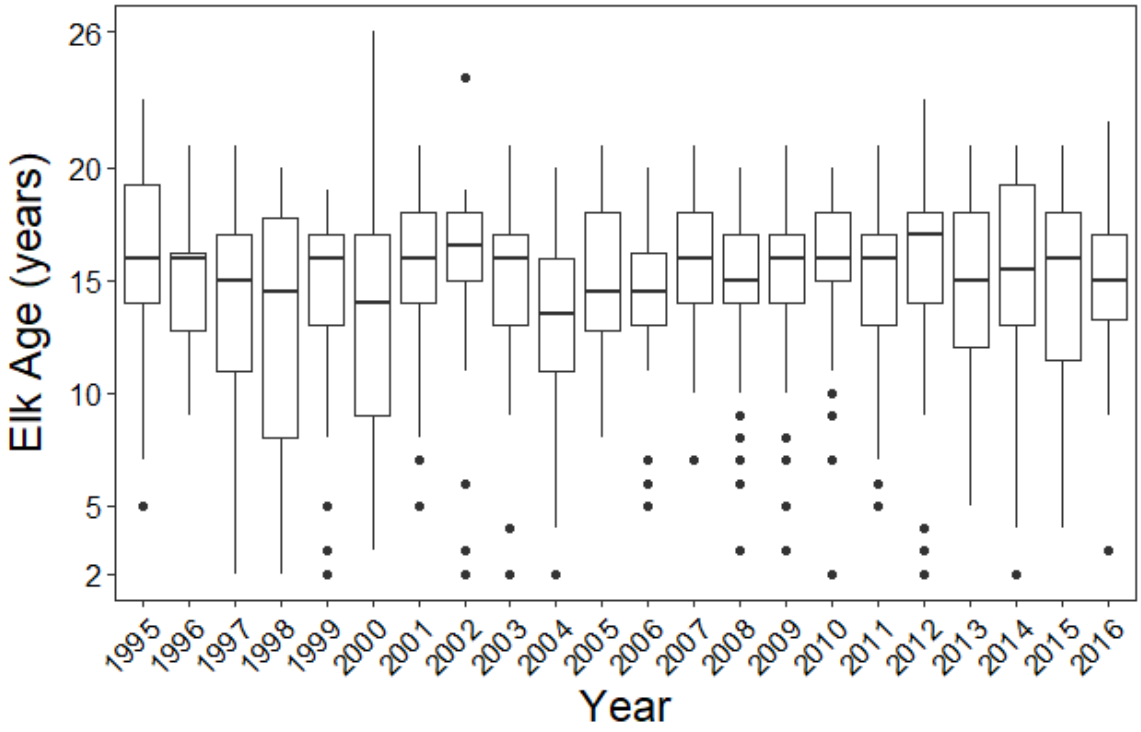

Figure 2-3 Annual median age of wolf-killed adult (≥ 2 years-old) female elk carcasses (radio-collared and uncollared) in northern Yellowstone and adjacent Montana. Data displayed from 1995 to 2009 are the same data used in Figure 2-2c; here I extend data out to 2016.

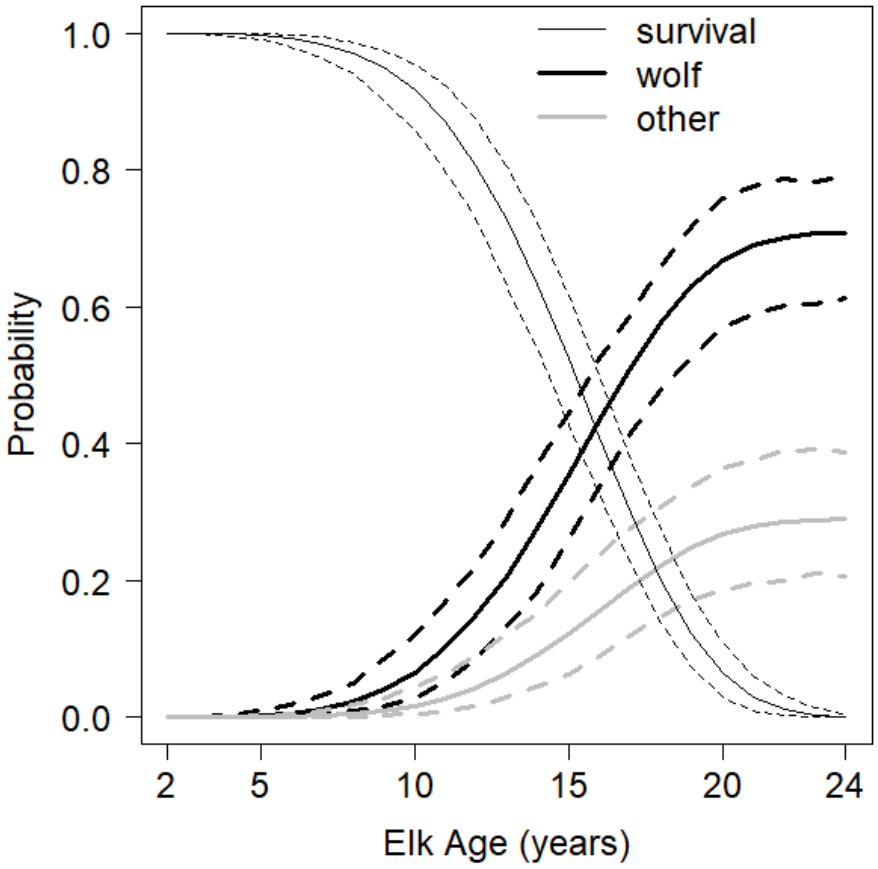

Figure 2-4 Instantaneous probability of survival (thin black line), wolf-caused mortality (thick black line), and other-caused mortality (non-wolf and non-human; thick grey line) of adult female elk aged 2 through 24 years old in northern Yellowstone and adjacent Montana. Dashed lines indicate associated 95% confidence intervals.

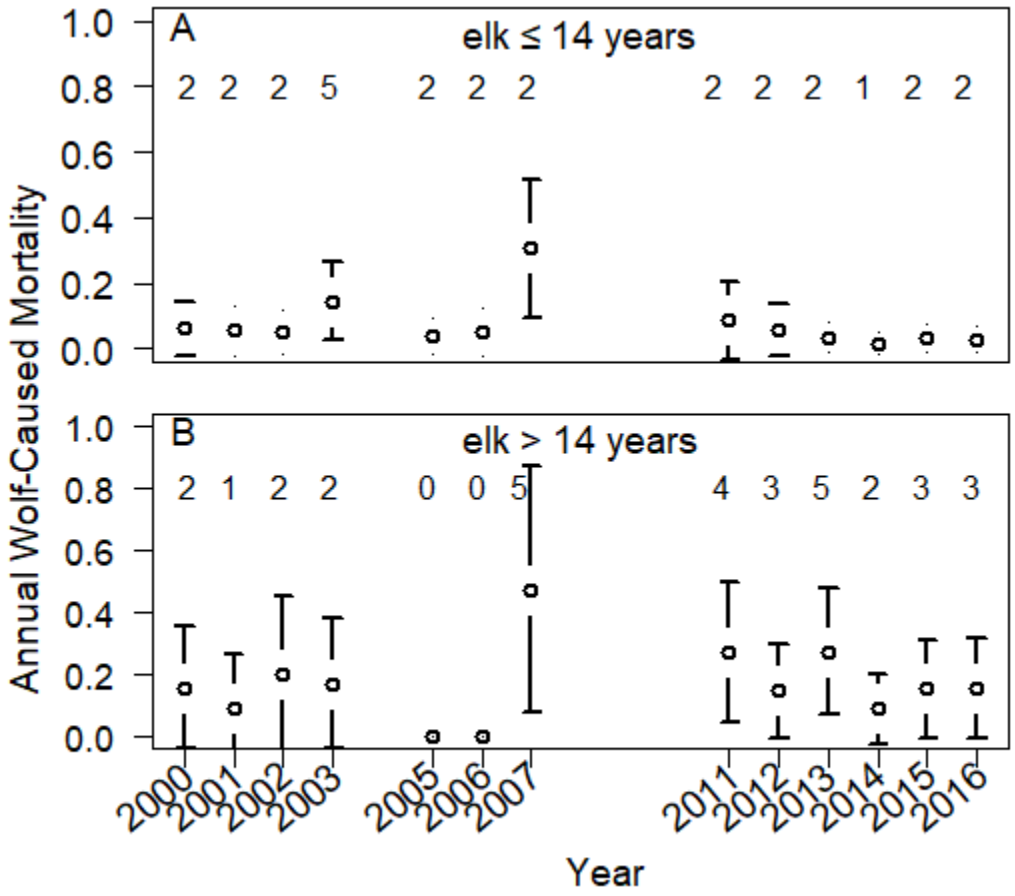

Figure 2-5 Annual wolf-caused mortality estimates and 95% CIs for collared young female elk (2 to 14 years old; A) and old female elk (> 14 years old; B) in northern Yellowstone and adjacent Montana. There was a gap in monitoring from 2008 to 2010 and no mortality of young elk in 2004 so these years were excluded from analysis. Numbers indicate the number of each age class killed by wolves each year.

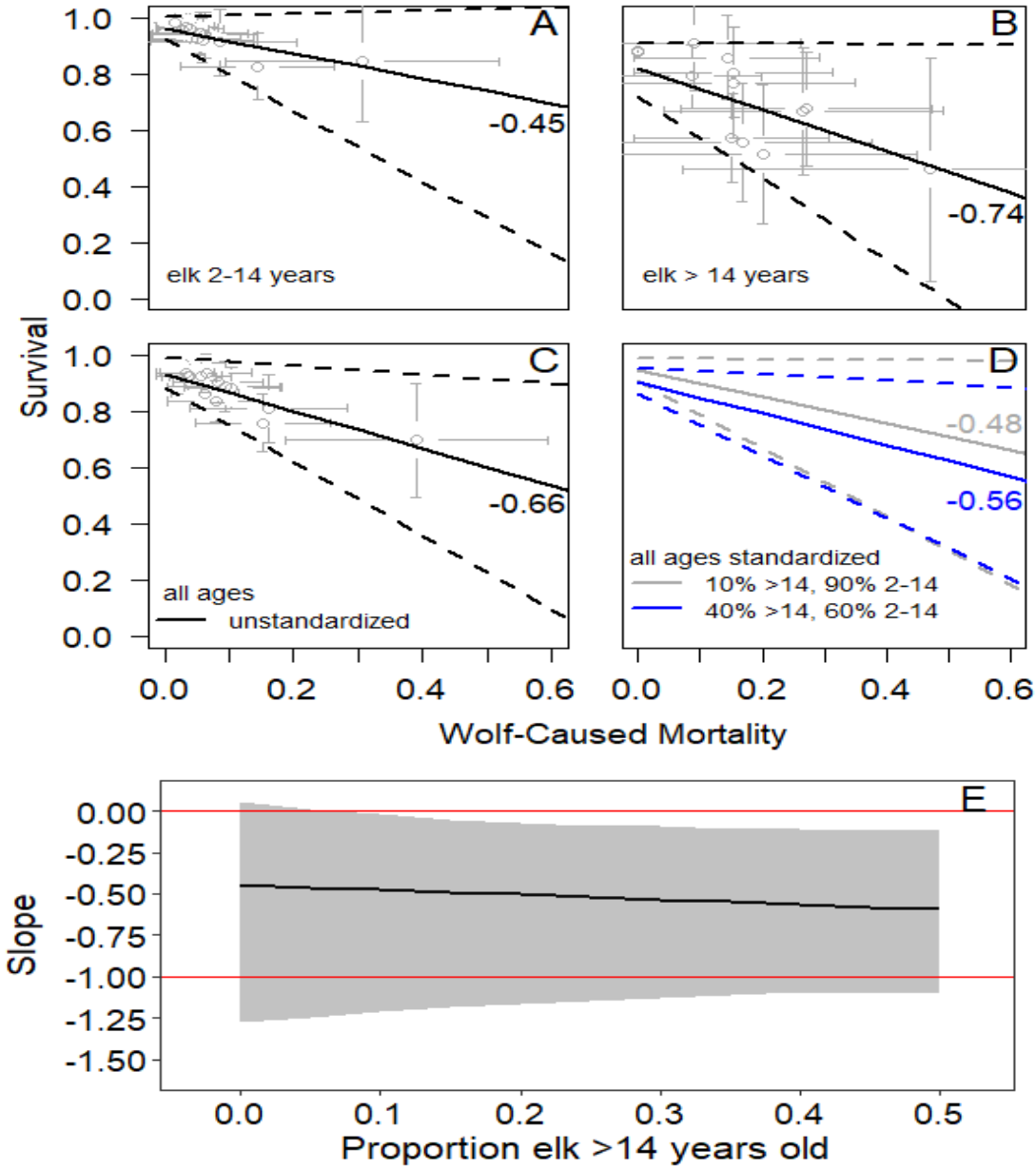

Figure 2-6 Relationship between annual wolf-caused mortality and survival probabilities of adult female elk in northern Yellowstone and adjacent Montana aged 2-14 years old (A), >14 years old (B), 2-24 years-old (C), and standardized by 10 or 40% of the population being comprised of elk > 14 years old (D) after accounting for uncertainty in mortality estimates. Points are annual rates with error bars denoting 95% confidence intervals. Also shown is the relationship between annual wolf-caused mortality and survival probabilities standardized by 0 to 40% of the population being comprised of elk >14 years-old, after accounting for uncertainty in mortality estimates (E). Solid lines are mean corrected slopes and dashed lines (A-D) and shading (E) are associated 95% confidence intervals, both obtained from Monte Carlo simulations. Slopes closer to -1 indicate additive wolf predation while slopes closer to 0 indicate compensatory predation.

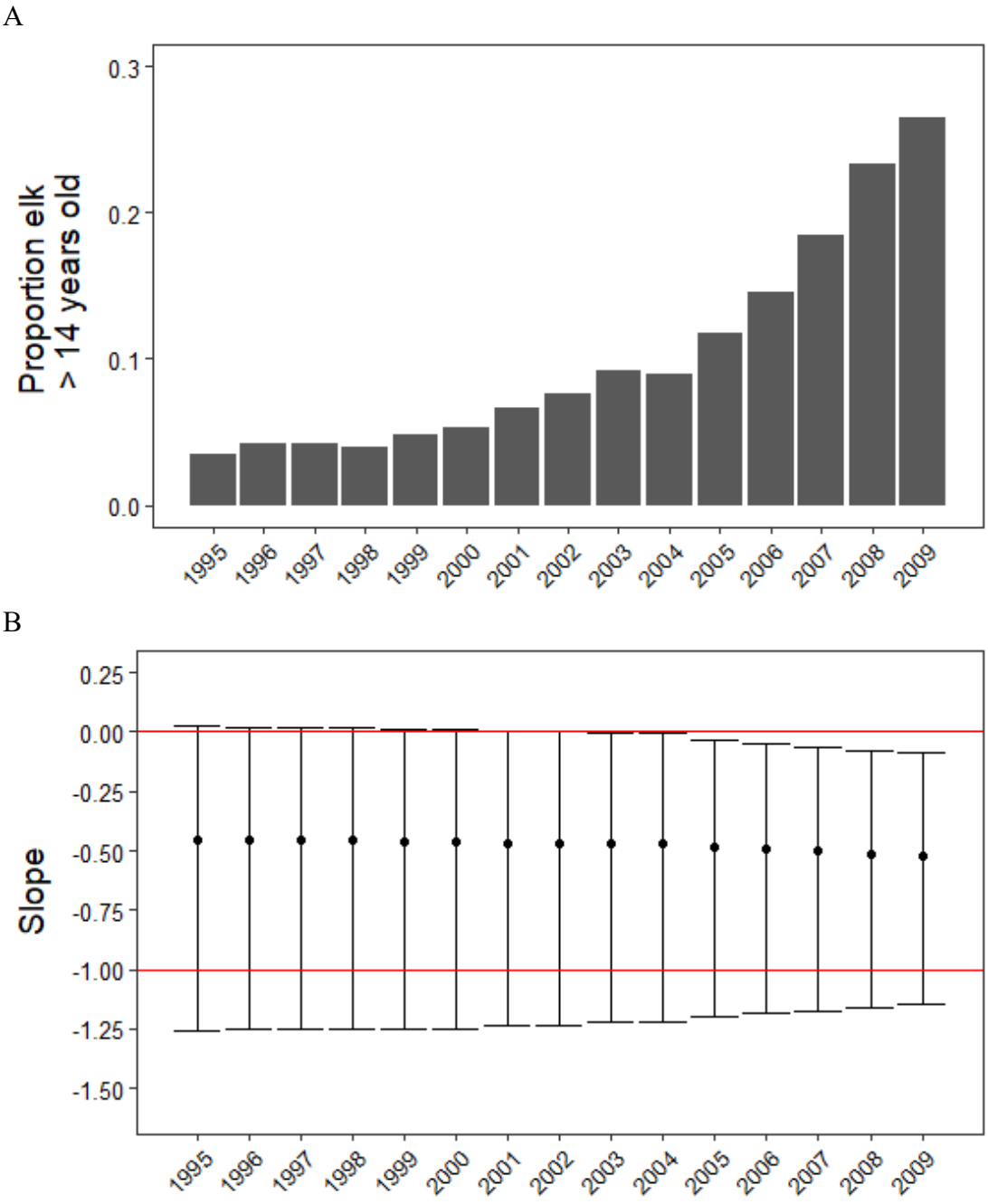

Figure 2-7 Annual proportion of adult female elk older than 14 years in northern Yellowstone and adjacent Montana based on age reconstruction from 1995 to 2009 (A). Annual relationships between wolf-caused mortality and survival probabilities standardized by the annual proportion of elk older than 14 years from 1995 to 2009 (B). Points are mean corrected slope estimates and lines are associated 95% confidence intervals, both obtained from Monte Carlo simulations. Confidence intervals that do not cross zero indicate lack of evidence for the compensatory predation hypothesis across the population.

CHAPTER 3

PREDATION AND ABIOTIC CONDITIONS SHAPE ACTUARIAL SENESCENCE OF A LONG-LIVED UNGULATE[2]

ABSTRACT

It is well established that mammals experience decreased survival with increasing age (actuarial senescence), but we understand little about how different sources of mortality and environmental conditions shape patterns of senescence. I used long-term data of radio-collared female elk (*Cervus canadensis*) in northern Yellowstone National Park and adjacent Montana to test the predictions that harsh environmental conditions and intense predation pressure decrease the age at onset of senescence, increase the intensity of actuarial senescence, and decrease the mean life expectancy. I used parametric survival and multi-state competing-risk models to estimate age-specific survival and cause-specific mortality, respectively. Dry conditions over three years, high snow water equivalent, and high wolf abundance led to an earlier age at onset of senescence. Wolf-caused mortality was the dominant mediator of senescence, and the age-specific wolf mortality hazard increased with snowy conditions and high wolf abundance. Despite increased senescent mortality in harsh conditions, there remained a subset of 'prime-aged' elk (e.g., 2-9 years old) that were generally unaffected by changes in the environment, indicating a limit to the extent that current environmental conditions may alter patterns of senescence.

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[2] Smith, L.M., Koons, D.N., Smith, D.W., Stahler, D.R., & MacNulty, D.R.

INTRODUCTION

Senescence, the cellular and physiological deterioration of an organism with age, leads to declines in reproduction and survival with age in many bird and mammal species (Jones *et al.* 2008; Nussey *et al.* 2013; Gaillard *et al.* 2017). Theory suggests that the evolution of senescence is driven by mortality that occurs as an interaction between an individual's age, physiological condition, and the environment it experiences (Abrams 1993; Williams & Day 2003; Moorad *et al.* 2019), with increased mortality at late ages leading to the evolution of senescence (Caswell 2007; Caswell & Shyu 2017). Actuarial senescence, defined as an increase in mortality and a decrease in survival with age, is a demographic outcome of an individual's physiology (Kirkwood 2015). Environmental hazards may alter patterns of actuarial senescence when older individuals are more susceptible to mortality due to their physiological condition (Williams & Day 2003). For example, older snail kites (*Rostrhamus sociabilis plumbeus*) have reduced survival during droughts (Reichert *et al.* 2010) and older Soay sheep (*Ovis aries*) have reduced survival at high density and in harsh winter conditions (Coulson *et al.* 2001). Despite widespread evidence of actuarial senescence (Nussey *et al.* 2013; Gaillard *et al.* 2017), only a few studies have examined how environmental conditions actually influence age-specific survival in the wild (Garrott *et al.* 2003, 2009; Moorad *et al.* 2019).

One cause of mortality may select for more rapid actuarial senescence (i.e., mortality increasing at a fast rate with age) than other causes if susceptibility to that cause depends on a higher degree of physiological deterioration (Koons *et al.* 2014). For example, compared to younger adults, older adults of white-tailed deer (*Odocoileus virginianus*) and elk (*Cervus canadensis*) have a greater risk of being killed by wolves

(*Canis lupus*) than dying of other causes (DelGiudice *et al.* 2002; Chapter 2). This difference suggests that predation of older prey may result in more rapid actuarial senescence, and prey populations may display slower actuarial senescence in the absence of or reduction in predation pressure. Further, physiologically-weakened individuals may die earlier than they would otherwise when exposed to predators or harsh abiotic conditions (Coulson *et al.* 2001; Ricklefs 2008). However, we lack evidence in the wild of the influence of selective predation on actuarial senescence relative to other sources of mortality. This deficiency is due in part to the difficulty of studying cause-specific mortality in wild populations, and of attaining the sample sizes needed to examine actuarial senescence at older ages (Koons *et al.* 2014).

Adult female ungulates usually have a relatively high and constant rate of survival until their body deteriorates at older ages, after which their chance of dying from all types of natural mortality increases rapidly (Loison *et al.* 1999; Gaillard *et al.* 2000b). They have 'planned senescence' pre-determined by tooth height, which wears down over the course of their life via grazing on plants containing silica and other granular compounds (Carranza *et al.* 2004). Worn teeth, in turn, contribute to physiological weakness from inefficient foraging, and thus, body mass declines at old ages (Skogland 1988), and vulnerability to predation and pathogens likely increases (Garrott *et al.* 2002; Ricklefs 2008). Wolves are age-selective predators (Wright *et al.* 2006) that may drive actuarial senescence of ungulate prey by 'adding' to the mortality induced by other causes (i.e., not compensating) in old individuals (e.g., elk >14 years old in Yellowstone National Park; Chapter 2) and thereby altering the intensity of age-dependent mortality.

To test the influence of abiotic and biotic environmental conditions on the actuarial senescence of elk in Yellowstone National Park and adjacent Montana, I used long-term data on the cause-specific mortalities of known-age adult females after wolf reintroduction. This dataset, combined with long-term monitoring of wolves, provides a unique opportunity to examine actuarial senescence of a long-lived species. I tested whether wolf abundance, alone or combined with either density-dependence, alternative wolf prey (*Bison bison*), or abiotic factors altered the age at onset of actuarial senescence (point of inflection of survival curve), the shape of actuarial senescence (i.e., how steeply mortality increases with age; Wrycza *et al.* 2015), mean life expectancy, and cause-specific mortality. I predicted increased wolf abundance, harsh abiotic conditions, and high densities of elk would lead to more rapid actuarial senescence and a shorter life expectancy, with heightened risk of wolf-caused mortality for older elk. In contrast, I predicted that increased bison abundance would lead to delayed actuarial senescence and a longer life expectancy of elk by providing wolves with an alternative food source (Tallian *et al.* 2017; Metz *et al.* 2020a), thereby reducing predation pressure on old elk. Here, I demonstrate how patterns of actuarial senescence of a wild, long-lived ungulate vary across environmental conditions and different causes of mortality.

MATERIALS AND METHODS

Study Area

This study encompassed the winter and summer ranges of the northern Yellowstone elk herd (Houston 1982). The winter range (1520 km^2) comprises low-elevation (1500-2600 m) grasslands and shrub steppes around the Yellowstone River and

its tributaries along the northern border of Yellowstone National Park and adjacent Montana (Lemke *et al.* 1998). Approximately 65% (995 km^2) of the winter range is located within the park, and the remaining 35% (525 km^2) extends north of the park boundary. The summer range includes the majority of Yellowstone and high-elevation areas outside the park to the north (elevation range 2206-3091 m across all summer ranges; (Craighead *et al.* 1972; White *et al.* 2010). Wolves were reintroduced to Yellowstone in 1995-1997 (Bangs & Fritts 1996). The northern Yellowstone elk herd and wolf reintroduction are described in detail in Smith *et al*. (2004, 2020). In addition to wolves, cougars (*Puma concolor*) are the other main top predator that kill elk of all age classes in the study area (Ruth *et al.* 2019), while grizzly bears (*Ursus arctos*), black bears (*U. americanus*), and coyotes (*C. latrans*) predominantly prey on elk calves (Barber-Meyer *et al.* 2008).

Data collection

Yellowstone personnel monitored the survival of 281 radio-collared female elk. From 2000 to 2003, 2005 to 2006, and 2011 to 2017, female elk (> 1 year old) were captured using net guns from helicopters (Hawkins and Powers, Greybull, Wyoming, USA; Leading Edge Aviation, Lewiston, Idaho, USA) or ground-darting and fitted with VHF or GPS collars. GPS collars included Telonics (Telonics, Mesa, Arizona, USA), Advanced Telemetry Systems Inc. (Isanti, Minnesota, USA), and Vectronic Aerospace (Vectronic Aerospace GmbH, Berlin, Germany). Elk were captured and handled in accordance with applicable guidelines from the American Society of Mammalogists (Sikes 2016) and approved by the National Park Service Institutional Animal Care and Use Committees. Yellowstone personnel tracked VHF-collared elk with ground-based

and aerial radio telemetry one to four times per month depending on weather and staff availability. GPS collars were programmed to collect locations at 1-6-hour intervals depending on the season, collar type, and other study objectives. The tracking period started immediately after capture in 2000 and ended in 2008 due to logistical constraints (note that tracking continued for two years after the 2006 captures). Both captures and tracking resumed in 2011 and continued through May of 2017. Yellowstone personnel tracked the status (alive/censored/dead) and location of each elk until the collar failed or was removed, or until the elk died. When possible, failing collars were replaced on the same individuals.

All collars were equipped with a mortality sensor, and Yellowstone personnel conducted field necropsies of dead elk to determine cause of death based on the carcass condition and location, blood trails, and evidence of predators including tracks, scat, bed sites, and wounds (Evans *et al.* 2006). Each elk was assigned a date of death based on timing of mortality signal or condition of the carcass. When months elapsed between the most recent resighting and a mortality, I assigned a death date halfway between when the mortality signal was heard and when the elk was last sighted (i.e., the midpoint rule; N=17). Yellowstone personnel recorded a cause of death if there was sufficient evidence to determine predator species or a non-predator cause of death upon site visit. Hunters returned collars to the National Park Service from collared elk they harvested.

I combined all non-wolf causes of mortality into a separate category ('other-caused mortality') to isolate the effect of wolf predation on elk survival and due to data limitations for additional categories. Other causes of mortality included cougar and grizzly bear predation, malnutrition, winterkill, and unknown causes of mortality that

excluded predation. I did not expect any of the covariates to influence human-caused mortalities and I was primarily interested in the impact of covariates on natural causes of mortality. Therefore, I right-censored elk that were harvested (N = 19) or hit by vehicles (N = 2) upon their date of death rather than include them in the "other" mortality state. Cause of death was unknown for 21 non-human caused mortalities. I used the frequency of known wolf-caused mortalities occurring inside (80% of 51) and outside (47% of 19) wolf pack boundaries to classify these unknown mortalities as either wolf-caused or other-caused according to whether the unknown mortalities occurred inside (N = 10) or outside (N = 11) wolf pack boundaries. Each mortality was located with respect to wolf pack boundaries of the corresponding year of death. I used wolf pack boundaries estimated with minimum convex polygons of wolf tracking data and provided by the Yellowstone Wolf Project (available at https://www.nps.gov/yell/learn/nature/wolf-reports.htm). I randomly assigned wolves as the cause of death for 80% of 10 unknown mortalities inside wolf pack boundaries (N = 8) and 47% of 11 unknown mortalities outside wolf pack boundaries (N = 5). I classified the remaining 8 unknown mortalities as other-caused.

Explanatory variables

Elk, bison, and wolf abundance

I included elk abundance in the analysis to evaluate whether conspecific density influenced survival. Elk were counted by Yellowstone personnel annually using 3-4 fixed-wing aircraft flying simultaneously in non-overlapping regions between December and March of 2000 to 2016. I used estimates of elk abundance adjusted for sightability

based on the group sizes of observed elk (Tallian *et al.* 2017; Fig. 3-1). Elk abundance decreased from 17,609 in winter 2000/2001 to 6,872 in winter 2016/2017 (MacNulty *et al.* 2020).

I included bison abundance in the analysis because winter-killed bison carcasses, calves, and bulls that die during the fall rut provide alternative food to wolves (Metz *et al.* 2020a). Yellowstone personnel counted the number of bison annually each summer and winter from 2000 to 2017 (Geremia *et al.* 2017), while distinguishing between northern and central Yellowstone. I used the abundance of bison in northern Yellowstone during winter (2000-2016) and the parkwide abundance of bison during summer (2000 to 2017) to coincide with the seasonal spatial distribution of elk. Estimated bison abundance increased from 550 in 2000 to 2,098 in 2016 in northern Yellowstone and from 2,708 in 2000 to 4,816 in 2017 parkwide (Fig. 3-1).

I included wolf abundance in the analysis because elk are the primary prey of wolves in Yellowstone (Metz *et al.* 2012, 2020a). Yellowstone personnel counted wolves annually across Yellowstone during their mid-November to mid-December and March wolf study periods (Smith *et al.* 2004). I used northern Yellowstone wolf abundance during the winter (November – April; Kohl *et al.* 2018) and parkwide wolf abundance during the summer (May – October) to coincide with the seasonal spatial distribution of elk. Wolf abundance varied between 83 and 172 individuals since 2000, but remained between 70 and 94 for the last six years of the study (Smith *et al.* 2020; Fig. 3-1). I also estimated the number of adult wolves of prime hunting age (2-6 and 3-5 years old) because wolf hunting ability senesces with age (MacNulty *et al.* 2009). A large proportion of the wolf population is comprised of known-age individuals because 30% to

50% of 9-month-old pups are radio-collared each year (Smith *et al.* 2004; MacNulty *et al.* 2009). I first calculated the number of wolves by age each year from the ages of radio-collared wolves. I then calculated the proportion of total known-age wolves that were in the 2-6- and 3-5-year-old age classes. I considered two age classes of wolves because ages 2 to 6 are typically considered prime ages (Hoy *et al.* 2020) and I also wanted to consider a more conservative age range. I multiplied the proportions of wolves in the two age classes by total adult wolf abundance to obtain an estimate of wolf abundance by prime hunting age. Therefore, the analysis included three covariates of wolf abundance (total wolf abundance, abundance of 2-6 and 3-5 year-olds) and two covariates of the proportion of wolf abundance comprised of prime hunting age wolves (proportion of 2-6 and 3-5 year-olds). I included both the abundance and proportion of prime hunting age wolves to consider both the numerical impact and a form of frequency-dependence of these individuals.

Winter snowpack

Winter snowpack influences the mortality risk of ungulates by reducing availability of forage and increasing wolf hunting success (DelGiudice *et al.* 2002; Metz *et al.* 2012). I used spatially-explicit (100-m resolution) estimates of snow water equivalent (hereafter 'snow') for Yellowstone (Wockner *et al.* 2006) as a proxy for the winter conditions elk experienced. I identified elk winter and summer ranges by the spatial extent of their telemetry/GPS locations using the Aggregate Points tool in ArcMap 10.3.1 (Esri, 2015). I extracted weekly snow estimates for the winter and summer ranges and averaged across their spatial extent. I then averaged across weeks to obtain monthly estimates.

Drought severity

Drought conditions during spring and summer may influence the availability of quality forage for an ungulate, and in turn their body condition (Cook *et al.* 2004b), which can affect the risks of predation (Funston & Mills 2006) or starvation (Young 1994). I used the spatially-explicit, monthly standardized precipitation evapotranspiration index (SPEI) to test the effect of short- and long-term drought conditions on elk survival. The SPEI advances the standardized precipitation index by accounting for temperature changes through evaporative demand with a water budget (Abatzoglou *et al.* 2017). Monthly data calculated for 1-month, 1-year, 3-year, and 5-year time intervals with 4-km spatial resolution were obtained for the U.S. through the West Wide Drought Tracker website (wrcc.dri.edu/wwdt; Abatzoglou *et al.* 2017). Each dataset occurs at a monthly scale but the 1- or multi-year intervals used data from the preceding 12, 36, or 60 months to provide an estimate of longer-term drought. I considered these different time scales for drought because of the potential influence of prolonged dryness on vegetation. I first extracted estimates for elk winter and summer ranges and then averaged across the spatial extent.

Data analysis

First, I tested the influence of biotic and abiotic factors on survival and senescence by conflating all non-human causes of mortality and estimating elk survival probabilities with a parametric survival model and a Weibull distribution commonly used for mortality that accelerates with advancing age (R package Flexsurv; Jackson 2016). I constructed the model with elk age as the time scale to parametrically estimate age-specific survival. I focused on elk ≥ 2 years-old because only six individuals were

marked as yearlings. Individual follow-up times were terminated when an individual died, went missing, or at the end of the study period (May 2017). The analysis included elk that lived through a three-year monitoring gap (2008-2010) as well as elk that went missing and were later found dead. I excluded the gap years from the analysis for elk that lived through the monitoring gap (i.e., they were right-censored and then re-entered) because it can bias the survival results by including information for individuals that survived while excluding information for those that were never observed again (Bart & Robson 1982; Bunck *et al.* 1995). Moreover, elk that died during the monitoring gap were treated as alive and censored from the analysis at the start of the monitoring gap.

Second, I modeled survival with four drought covariates (i.e., drought at 1-month, 1-year, 3-year, and 5-year lagged time scales) to determine the most appropriate temporal duration of drought effects on elk survival, and five wolf covariates to determine the best supported wolf abundance metric. I used Akaike's Information Criterion adjusted for sample size (AIC_c) to rank statistical support for each model, given the data. The covariate in the top model for each of these variables was advanced to the candidate set of models that considered the full suite of covariates.

I developed the candidate model set by considering the separate effects of elk abundance, bison abundance, drought, and snow, in addition to a combined weather effect (i.e., drought and snow). I included wolf abundance in every model because of the predominance of wolf-killed elk in the sample and because elk are the primary prey of wolves in Yellowstone (Metz *et al.* 2012). I assigned elk winter abundance to each month spanning from the prior June through May after the count. For example, I assigned elk abundance from the winter of 2015-2016 to June 2015 through May 2016.

Limitations in the spread of data across covariate pairs precluded use of interactions (i.e., high-low combinations, and vice versa, often did not occur during the study). All covariates were standardized ($(x - \bar{x})/ sd$) prior to analysis because of differences in scale of raw values; therefore, a covariate value of 0 is the average value. I tested the collinearity of covariate combinations in the final model set and none exceeded ± 0.46.

Third, I repeated the analysis in a competing-risk mortality framework with the covariates that were most supported in the survival analysis (Figure 3-2). Here, the cause-specific mortality was a joint probability of dying before a given time and by a given cause, where cause-specific mortality probabilities were mutually exclusive (Heisey & Patterson 2006; Wolfe *et al.* 2015). I fit a fully parametric, continuous-time multistate model with two mortality states (wolves and other) and a Weibull distribution to the data using the Flexsurv package (Jackson 2016) in R (3.5.1). Transitions were not possible between mortality states or from a mortality state to the alive state. I estimated the instantaneous rate of transition (transition intensity) for elk survival and the transition to each mortality state. The effect of each covariate or covariate group was applied separately to each mortality state, and I assumed that wolf abundance would not influence the non-wolf causes of mortality. I selected the model with the lowest AICc score per cause of mortality in the final tier of model selection.

Shape and pace of aging

I compared how different levels of the covariate (e.g. quantiles of wolf abundance) influenced actuarial senescence. I distinguished between the shape of senescence (how steeply survival changes with age) and life expectancy (how fast mortality progresses) to classify patterns of senescence (Baudisch 2011; Wrycza *et al.*

2015). I assessed the shape of senescence (i.e., changes to the shape of the survival curve) using a shape index (Wrycza *et al.* 2015) based on the probability of surviving to the mean age at death. I chose this measure for its ease of calculation. In addition, an analysis by Wrycza *et al.* (2015) with a small sample size using different shape measures did not produce statistically significant differences in the results, suggesting that the choice of shape index may not strongly influence the results. Shape of senescence describes how mildly or steeply survival decreases towards the end of the lifespan for an average individual within the population and is independent of time (Wrycza *et al.* 2015). The index is calculated as

$$S(l) = 1 - H(e_0) = 1 + \log(l(e_0)), \quad (1)$$

where S(l) is the shape index number for survival function l, $H(e_0)$ is the cumulative hazard at the mean life expectancy e_0. I compared the shape index across 0.1 intervals of covariate quantiles to determine how covariates altered the shape of senescence. Larger index values indicate a steeper decrease in survival at old ages, and thus stronger actuarial senescence.

I calculated mean life expectancy (e_0) past age two as the integral of the survival curve from age 2 to infinity. This estimate is the average length of time an elk is expected to live given that she has already survived two years. I also compared life expectancies across covariate quantiles to determine how life expectancy was affected by levels of the covariate. I calculated the shape index and mean life expectancy for covariates in the top survival models.

Onset of senescence

I estimated the age at onset of actuarial senescence as the inflection point of the survival curve when mortality accelerates. I calculated the second derivative of the estimated age-specific survival curves using the 'deriv' function (package stats) in R. I considered the first age at which the second derivative reached its local maximum value as the age at onset (Jones *et al.* 2008; Aubry *et al.* 2009; Lemaître *et al.* 2020). For survival models, I considered quantiles of one covariate while holding the other covariate(s) at the respective mean values to estimate the influence of explanatory variables on the age at onset of senescence. Specifically, I estimated the age at onset of senescence for the 0, 50^{th}, and 90^{th} percentiles of covariates.

RESULTS

I estimated elk survival and cause-specific mortality based on 281 radio-collared adult females (≥ 2 yrs. old), of which 91 died of non-human causes, including 63 due to wolves. At the time of capture, age of elk ranged from 2 to 22.5 years. The minimum and maximum age at death was 6 and 24 years old, respectively. Wolf-killed elk ranged in age from 6 to 24 years old (mean 15.4). According to the null competing-risk model, the instantaneous risk of wolf-caused mortality was 0.00-0.10 for 2-10 year-olds and 0.70-0.72 for 20-24 year-olds, whereas the instantaneous risk of other-caused mortality was 0.00-0.03 for 2-10 year-olds and 0.27-0.29 for 20-24 year-olds. The second derivative of the estimated age-specific survival indicated that the age at onset of actuarial senescence was 11 years old.

Survival models

Among the considered drought and wolf variables, three-year drought and total wolf abundance had the lowest AIC_c scores (SPEI 3yr in Table 3-1a and wolf abundance in Table 3-1b), and were thus included in the candidate set of survival models. The top-ranked survival model included 3-year drought and wolf abundance (SPEI 3yr + wolf in Table 3-1c; for parameter estimates see Table 3-2). The second-ranked model included the effects of the top-ranked model plus snow water equivalent (hereafter 'snow'; i.e., the top model was nested within the second-ranked model). Though the addition of snow to the second-ranked model led to a slightly higher AIC_c score ($\Delta AIC_c = 0.04$), I do not interpret the effect of snow on age-specific survival as being uninformative because the estimated effect was biologically strong, estimated precisely, and the estimated effect of snow was similar in the simpler, third-ranked model (Table 3-1c, Table 3-2). As such, I focus my inference on the estimated effects from the second-ranked model that collectively join those from model rankings one and three.

Age-interval survival probability and age at onset of senescence decreased with increasing snow, dryness, and wolf abundance (Figs 3-3a, 3-3b, 3-3c). Age-interval survival probability remained stable for elk < 8 years old under increasingly snowy and dry conditions (Figs 3-4a, 3-4b) and under higher wolf abundance (Fig. 3-4c).

Survival at late ages (shape index value) decreased more steeply with increasingly dry conditions, with wolf abundance and snow held at their means (Table 3-3; Fig. 3-3b). Likewise, survival at late ages decreased more steeply with increasing wolf abundance, with drought and snow held at their means (Table 3-3; Fig. 3-3c). An increase in wolf abundance had a stronger influence on the shape of the survival curve (larger shape index

value) than did an increase in drought. In contrast, survival at late ages decreased less steeply with increasing snow, with drought and wolf abundance held at their means (Table 3-3; Fig. 3-3a). Life expectancy decreased with increasing drought, increasing wolf abundance, and increasing snow (Table 3-3).

Wolf-caused mortality

I retained the top three survival models for consideration in the analysis of competing risks (Table 3-1d). For wolf-caused mortality, however, a model restricted to the effects of snow and wolf abundance had the lowest AIC_c score (Table 3-1d; for parameter estimates see Table 3-4a) and was the only model with a lower AIC_c score than the null model ($\Delta AIC_c = 2.9$). The 95% confidence intervals of snow and wolf abundance scale parameters did not overlap zero, suggesting an important influence of snow and wolves despite the ranking of the null model (Table 3-4a). The 95% confidence intervals of the parameters in the lowest-ranking model (Table 3-1d) did somewhat overlap zero (Table 3-4a).

Estimates from the top model indicated that heavy snow conditions (as represented by snow water equivalent) increased the wolf-caused mortality hazard for most ages of elk compared to snow-free conditions (Fig. 3-5a), but the effect was greatest for teenage elk (Fig. 3-5b). However, across levels of snow, the wolf-caused mortality hazard was relatively stable for elk < 10 years-old, but past average snow, the mortality hazard of elk >20 years old declined (Fig. 3-5b).

Maximum wolf abundance increased the wolf-caused mortality hazard for teenaged elk compared to minimum wolf abundance, but the hazard was near-zero for elk < 10 years-old regardless of wolf abundance (Fig. 3-5c). Across levels of wolf

abundance, the wolf-caused mortality hazard for elk > 12 years-old had the greatest increase, whereas the increase in hazard by wolf abundance was less substantial for elk 10-12 years-old (Fig. 3-5d).

Other-caused mortality

I similarly retained the top three survival models for consideration in the analysis of other-caused mortality, but I removed the wolf covariate from all models for reasons explained in the Methods (Table 3-1e). Interestingly, the competing-risk model with a sole effect of drought had the lowest AIC_c score among the models considered (SPEI 3yr in Table 3-1e; for parameter estimates see Table 3-4b), serving as a complement to the effects of snow and wolf abundance on wolf-caused mortality, and helping explain why all three variables affected age-specific survival. The 95% confidence intervals of the drought scale parameter did not overlap zero, suggesting an important influence of drought despite the close ranking of the null model (Table 3-4a). In contrast, the 95% confidence intervals of snow in the lowest-ranking models (Table 3-1e) did overlap zero (Table 3-4b).

Dry conditions increased the other-caused mortality hazard for teenaged elk compared to wet conditions (Fig. 3-6a). Increasing dryness increased the mortality hazard for elk > 14 years old, although the increase was most substantial for elk > 18 years old (Fig. 3-6b). Of the 28 other-caused mortalities in the study, 5 were due to non-wolf predators, 7 were due to malnutrition, and 16 were due to unknown, non-predation causes of mortality.

DISCUSSION

My findings advance knowledge of actuarial senescence in mammals (Nussey *et al.* 2013; Gaillard *et al.* 2017; Galliard & Lemaitre 2020) by evaluating the role of environmental conditions on overall survival and cause-specific mortality. First, the results showed that increased predator abundance and harsher winter conditions increased prey actuarial senescence via predation mortality, resulting in reduced life expectancy and an earlier onset of actuarial senescence. Second, drier 3-year conditions were negatively related with age-specific survival; although, I had no evidence of a possible negative effect of drought on predation mortality (Table 3-1d). Drought therefore influenced elk senescence primarily through other causes of natural mortality. Third, the results showed that predation by a primary predator had a stronger impact on actuarial senescence (higher prey mortality at late ages) than did other sources of mortality combined. Fourth, changes in environmental conditions primarily influenced the mortality of individuals > 8 years old, supporting the idea that environmental conditions interact with age, and likely physiological deterioration with age, to influence senescence patterns (Williams & Day 2003; Moorad *et al.* 2019).

Elk survival decreased and wolf-caused mortality increased with increasing wolf abundance. Similarly, Brodie *et al.* (2013) found that elk survival decreased in the presence of wolves but they did not demonstrate how age-specific survival shifted with wolf abundance. Elk are the primary prey of wolves in Yellowstone and the wolf predation rate of elk peaked during maximal wolf abundance (Metz *et al.* 2020b). The results suggest that a sustained high abundance of wolves would produce early onset of actuarial senescence and a shorter life expectancy compared to average and low wolf

abundance. When sustained across generations, such selection pressures could cause directional evolution of actuarial senescence in elk. However, wolf abundance (N) peaked in Yellowstone in 2003, hovered around N=100 from 2008 onward, and might not return to that peak because of inter-pack aggression, disease, and a lower elk abundance, collectively yielding time-variant selection on senescence patterns caused by wolf predation (Smith *et al.* 2020).

Increasing monthly snow levels decreased elk survival and increased wolf-caused mortality. This result is not surprising given that winter severity has previously been shown to decrease survival of elk (Garrott *et al.* 2003, 2009) and other ungulates (Coulson *et al.* 2001; DelGiudice *et al.* 2002, 2006). Similar to my findings, snowpack had no influence on the survival of elk aged 1 to 9 years old, except at the greatest snow levels in central Yellowstone (Garrott *et al.* 2009). However, these findings are novel in that they quantify the impact of snow on wolf-caused mortality. Wolves kill younger adult ungulates (DelGiudice *et al.* 2002) and focus on female elk (Wilmers *et al.* 2020) when snow is deep. Likewise, I found that elk were more susceptible to wolf predation at younger ages in snowy conditions. Thus, a series of winters with heavy snowpack could decrease age at onset of actuarial senescence and life expectancy compared to average conditions. Conversely, a decrease in snowpack, as predicted for Yellowstone due to climate warming (Tercek *et al.* 2015), may delay wolf-driven actuarial senescence by forcing wolves to kill only the weakest (and likely oldest) individuals. The slight decrease of the wolf-caused mortality hazard for elk older than 20 years old with more snow may be due to the increasing susceptibility of younger (10-16 years old), but not prime-aged, elk to predation, the rarity of old individuals (Hoy *et al.* 2020), or a learned behavioral

avoidance of areas with deeper snow. However, the result may also be an artifact of a smaller sample size of the oldest individuals.

Elk survival also decreased with increasingly dry conditions. Dry conditions may limit forage availability and reduce body mass, with demographic consequences for survival (Gaillard *et al.* 2000a), reproduction (Cook *et al.* 2004a; Tollefson *et al.* 2010), and population growth rate (Duncan *et al.* 2012; López-Montoya *et al.* 2017). Adult ungulate survival (Owen-Smith *et al.* 2005) and abundance (Ogutu & Owen-Smith 2005) declines during dry conditions, but age-specific changes in survival have hitherto been unknown. Yellowstone's climate has warmed over 1982 – 2015 and the park was in a drought from 2000 – 2016 (Notaro *et al.* 2019). If these dry conditions continue or worsen, the results suggest earlier age at onset of senescence and shortened life expectancy. The lack of an influence of drought on wolf-caused mortality may be due the importance of snow for wolf hunting success (Huggard 1993) and drought-stressed individuals may have been more vulnerable to wolves in deep snow. These findings suggest that wolves are not able to kill all old elk on the landscape, and the survival of those individuals that avoid wolf predation is more strongly influenced by drought than by snow.

I found no influence of elk or bison abundance on elk survival. Prior to wolf reintroduction, adult elk survival was also independent of elk density (Coughenour & Singer 1996). This finding concurs with other studies that suggest that adult ungulate survival, particularly of prime-aged individuals, is density-independent (Gaillard *et al.* 1998, 2000b; Coulson *et al.* 2001). While density effects on senescent ungulates have been understudied (Bonenfant *et al.* 2009), only one of four species had density-

dependent survival of older individuals (Coulson *et al.* 2001; Festa-Bianchet *et al.* 2003). In Yellowstone, wolves kill bison calves and scavenge on winter-killed bison carcasses (Metz *et al.* 2012; Tallian *et al.* 2017) but there is no evidence of classic prey switching (Tallian *et al.* 2017). The result suggests that the increase in bison biomass in the Yellowstone wolf diet (Metz *et al.* 2020a) is not sufficient or has not occurred across a long enough period to influence age-specific patterns of elk survival.

Older ungulates are generally more sensitive to environmental conditions than prime-aged adults (Gaillard *et al.* 2000b). Consistent with other ungulate studies (Loison *et al.* 1999; Gaillard *et al.* 2000b), my findings support the idea of a broad 'prime-age' class (a demographic refuge; Miller & Rudolf 2011) that is robust to wolf predation and environmental factors affecting body condition, and in turn robust to senescence. The age class estimate concurs with prior classifications of ages 2-9 years old as prime-aged (Garrott *et al.* 2003; Wright *et al.* 2006; Raithel *et al.* 2007; Hebblewhite & Merrill 2011). There is likely a limit to the possible timing and shapes of senescence at the time scale and range of environmental conditions observed.

Environmental conditions can change the risk of predation, thereby possibly altering patterns of actuarial senescence in the wild. In central Yellowstone, high snowpack forced elk to forage in geothermal areas where they had a high rate of fluoride and silica consumption, which compromised normal tooth matrix formation of juvenile elk and accelerated tooth wear and age at onset of actuarial senescence compared to elk in northern Yellowstone (Garrott *et al.* 2002). This decreased life expectancy increased the proportion of the population in the senescent age class, and thereby increased the proportion of elk susceptible to wolf predation, with likely impacts on population

dynamics (Garrott *et al.* 2002). Climate warming may eventually make it more difficult for wolves to kill elk in winter by reducing snowpack and increasing forage availability for elk, as well as potentially increasing calf recruitment (Proffitt *et al.* 2014). However, a reduced snowpack combined with drier summer conditions may reduce overall elk forage availability and alter elk body condition. The implications of these changing climatic conditions for both predator and prey demography is an important avenue for future research.

Conclusion

I demonstrated the combined roles that predation and abiotic environmental conditions play in shaping both the onset and shape of actuarial senescence in a wild population of long-lived elk. When climatic conditions are harsh, prey survival at late ages declines rapidly, and age at onset of senescence and life expectancy also decline, yet prime-aged individuals retained high survival. High survival of prime-aged individuals, despite harsh environmental conditions, suggests that this subset of an age-structured population can potentially buffer the population during challenging years and may influence how the population responds to changing climate (Gaillard & Yoccoz 2003; Pardo *et al.* 2013). Understanding the length of prime versus senescent stages may provide an indication of how vulnerable the population is to environmental conditions (Bleu *et al.* 2015), as well as how the population may be influenced by stage-selective predation.

I provide evidence that a predator can alter patterns of actuarial senescence when they selectively prey on older adults over younger adults. In addition, I found that the influence of predation on age-specific mortality of prey depends on the predator's

abundance. Prey populations may have a longer life expectancy and slower rate of senescence when exposed to reduced predation pressure than they would when exposed to higher predation pressure (Gaillard *et al.* 2017). I did not test for differences in actuarial senescence between two populations exposed to different predator abundances, but the results indicate that predation pressure sustained at higher and lower levels would result in different patterns of actuarial senescence. The potential co-evolution of senescence patterns between predator and prey species remains unexplored. There is value in maintaining long-term monitoring of known-age individuals to gain deeper insight into the influence of environmental conditions on survival, cause-specific mortality, and senescence patterns (Clutton-Brock & Sheldon 2010). Future work should also aim to estimate the heritability of survival-related traits as well as genotype-phenotype associations that can advance our understanding of eco-evolutionary dynamics shaping senescence in the wild (Coulson *et al.* 2011).

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TABLES AND FIGURES

Table 3-1 Models for the influence of drought (standardized precipitation evapotranspiration index, SPEI) and wolves on survival of adult female elk (≥ 2 years old) in northern Yellowstone and adjacent Montana (a, b). The drought and wolf covariates from their respective top models were included in the final survival model set (c). Candidate models for the effect of wolf abundance (wolf), bison abundance (bison), elk abundance (elk), snow water equivalent (SWE), and drought (SPEI) on survival of adult female elk (c). Candidate models for the competing-risk wolf-caused mortality analysis (d) included the top three models from the survival analysis (c). Candidate models for the competing-risk other-caused mortality analysis (e) included the drought and SWE covariates from the top three models of the survival analysis (c).

Analysis	Model	AICc	ΔAICc
(a) Survival – Drought*	SPEI 3yr	441.18	0.00
	SPEI 5yr	445.69	4.51
	SPEI 1month	455.37	14.19
	SPEI 1yr	456.02	14.83
	null	456.58	15.40
(b) Survival - Wolf	Wolf abundance	443.51	0.00
	Prime-aged wolf abundance (2-6 yrs old)	448.90	5.39
	Prime-aged wolf abundance (3-5 yrs old)	454.93	11.42
	null	456.58	13.07
	Proportion of prime-aged wolves (2-6 yrs old)	457.08	13.58
	Proportion of prime-aged wolves (3-5 yrs old)	458.83	15.32
(c) Survival	SPEI 3yr + wolf	437.16	0.00
	SPEI 3yr + wolf + SWE	437.20	0.04
	SWE + wolf	439.39	2.23
	wolf	443.51	6.34
	wolf + bison	443.52	6.36
	elk + wolf	445.54	8.38
	null	456.58	19.42
(d) Competing Risks: Wolf-caused Mortality	SWE + wolf	334.23	0.00
	null	337.13	2.90
	SPEI 3yr + SWE + wolf	337.60	3.37
	SPEI 3yr + wolf	338.39	4.16
(e) Competing Risks: Other-caused Mortality	SPEI 3yr	177.00	0.00
	null	178.26	1.26
	SPEI 3yr + SWE	180.83	3.83
	SWE	181.75	4.75

*SPEI data corresponds to 1 month, 1-year, 3-year, and 5-year monthly timescales.

Table 3-2 Parameter estimates from survival analyses of adult female elk (≥ 2 years old) in northern Yellowstone and adjacent Montana. The Weibull parameterization used in R package Flexsurvreg (Jackson 2016) is consistent with dweibull in R (refer to package documentation for details).

Model	Parameter	Data Mean	Estimate	L95%	U95%	SE
SPEI 3yr + wolf	shape	NA	4.95	4.09	5.99	0.48
	scale	NA	16.22	15.42	17.06	0.42
	SPEI 3yr	-0.02	0.07	0.02	0.12	0.02
	wolf	-0.26	-0.04	-0.10	0.01	0.03
	shape(SPEI 3yr)	-0.02	0.01	-0.16	0.18	0.09
	shape(wolf)	-0.26	0.15	-0.05	0.35	0.10
SPEI 3yr + wolf + SWE	shape	NA	4.95	4.06	6.04	0.50
	scale	NA	15.96	14.94	17.04	0.54
	SPEI 3yr	-0.02	0.06	0.00	0.11	0.03
	wolf	-0.26	-0.09	-0.17	-0.01	0.04
	SWE	0.05	-0.11	-0.23	0.01	0.06
	shape(SPEI 3yr)	-0.02	-0.01	-0.18	0.17	0.09
	shape(wolf)	-0.26	0.01	-0.25	0.27	0.13
	shape(SWE)	0.05	-0.27	-0.50	-0.03	0.12
SWE + wolf	shape	NA	4.81	3.95	5.86	0.48
	scale	NA	15.63	14.60	16.73	0.54
	SWE	0.05	-0.16	-0.27	-0.05	0.06
	wolf	-0.26	-0.13	-0.21	-0.06	0.04
	shape(SWE)	0.05	-0.36	-0.54	-0.17	0.10
	shape(wolf)	-0.26	-0.01	-0.24	0.21	0.12

Table 3-3 Shape indices and life expectancies of adult female elk (≥ 2 years old) in northern Yellowstone and adjacent Montana based on the second-ranked survival model with drought (standardized precipitation evapotranspiration index), wolf abundance, and snow water equivalent (SWE). I calculated values for drought and wolf abundance with quantiles at 0.1 intervals. Values in parentheses are wolf abundance at the given quantile. SWE quantiles 0.1 and 0.2 are not shown because they also referred to no snow. A higher shape index value equals a faster increase in mortality at late ages (Wrycza *et al.* 2015).

	Quantile	Life expectancy at age 2	Shape Index
3-yr Drought	0.0 (wet)	14.72	0.65
	0.1	13.96	0.66
	0.2	13.27	0.67
	0.3	12.96	0.68
	0.4	12.75	0.68
	0.5	12.60	0.68
	0.6	12.40	0.69
	0.7	12.27	0.69
	0.8	11.98	0.70
	0.9	11.54	0.71
	1.0 (dry)	11.16	0.71
Wolf (Abundance)	0.0 (34)	14.80	0.65
	0.1 (38)	14.59	0.65
	0.2 (48)	14.07	0.66
	0.3 (65)	13.28	0.67
	0.4 (73)	12.89	0.68
	0.5 (80)	12.59	0.68
	0.6 (84)	12.37	0.69
	0.7 (93)	11.98	0.70
	0.8 (102)	11.63	0.70
	0.9 (108)	11.36	0.71
	1.0 (142)	10.02	0.74
Snow Water Equivalent	0.0 (no snow)	14.38	0.72
	0.3	14.11	0.72
	0.4	13.81	0.71
	0.5	13.15	0.70
	0.6	12.44	0.68
	0.7	11.97	0.67
	0.8	11.41	0.65
	0.9	10.69	0.63
	1.0 (heavy snow)	7.63	0.20

Table 3-4 Parameter estimates from the top model for competing-risk mortality analyses of adult female elk (≥ 2 years old) in northern Yellowstone and adjacent Montana. The Weibull parameterization used in R package Flexsurvreg (Jackson 2016) is consistent with dweibull in R (refer to package documentation for details).

Cause of Mortality	Model	Parameter	Data Mean	Estimate	L95%	U95%	SE
(a) Wolf	SWE + wolf	shape	NA	4.57	3.63	5.75	0.54
		scale	NA	16.01	14.92	17.18	0.58
		wolf	-0.26	-0.10	-0.18	-0.02	0.04
		SWE	0.05	-0.12	-0.23	-0.01	0.06
		shape(wolf)	-0.26	-0.03	-0.28	0.23	0.13
		shape(SWE)	0.05	-0.23	-0.44	-0.01	0.11
	SPEI 3yr + wolf + SWE	shape	NA	4.62	3.67	5.83	0.55
		scale	NA	16.10	15.00	17.30	0.57
		SPEI 3yr	-0.02	0.03	-0.04	0.10	0.03
		wolf	-0.26	-0.09	-0.17	0.00	0.04
		SWE	0.05	-0.11	-0.21	0.00	0.05
		shape(SPEI 3yr)	-0.02	0.00	-0.21	0.21	0.11
		shape(wolf)	-0.26	-0.03	-0.29	0.24	0.14
		shape(SWE)	0.05	-0.21	-0.44	0.01	0.11
	SPEI 3yr + wolf	shape	NA	4.49	3.56	5.66	0.53
		scale	NA	16.26	15.24	17.34	0.54
		SPEI 3yr	-0.02	0.03	-0.04	0.09	0.03
		wolf	-0.26	-0.06	-0.13	0.01	0.04
		shape(SPEI 3yr)	-0.02	-0.06	-0.27	0.14	0.10
		shape(wolf)	-0.26	0.01	-0.25	0.27	0.13
(b) Other	SPEI 3yr	shape	NA	6.26	4.64	8.45	0.96
		scale	NA	19.51	18.29	20.80	0.64
		SPEI 3yr	-0.02	0.07	0.01	0.14	0.03
		shape(SPEI 3yr)	-0.02	0.00	-0.28	0.29	0.15
	SWE	shape	NA	6.08	4.52	8.17	0.92
		scale	NA	19.34	18.17	20.59	0.62
		SWE	0.05	-0.03	-0.11	0.05	0.04
		shape(SWE)	0.05	-0.15	-0.57	0.27	0.21

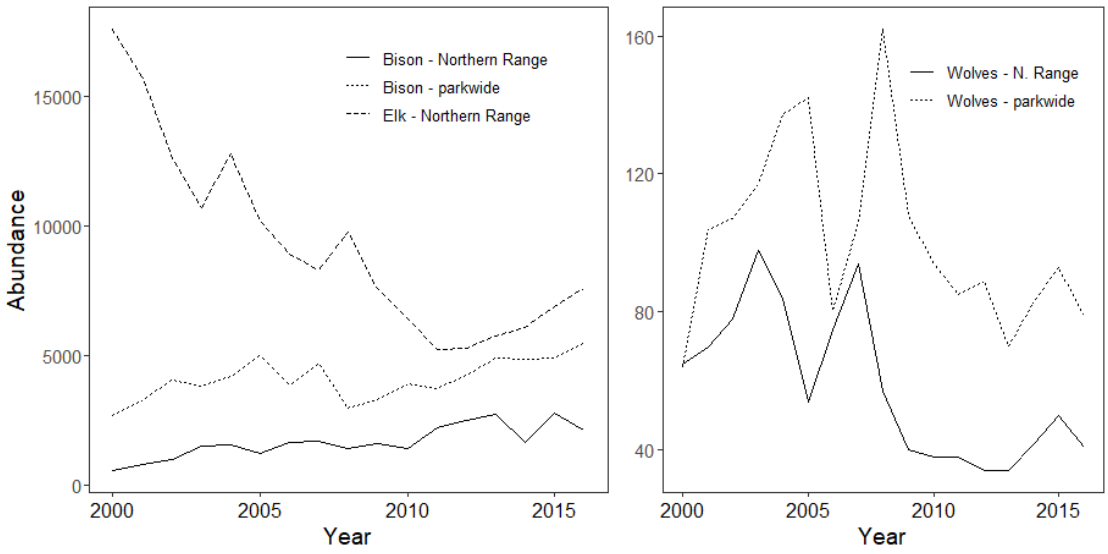


Figure 3-1 The abundance of elk, bison, and wolves in Yellowstone National Park from 2000 to 2016. Northern Range refers to the wintering range of the elk population, which extends from northern Yellowstone into Montana. Northern Range wolf abundance was comprised of winter estimates while parkwide wolf abundance was comprised of summer estimates.

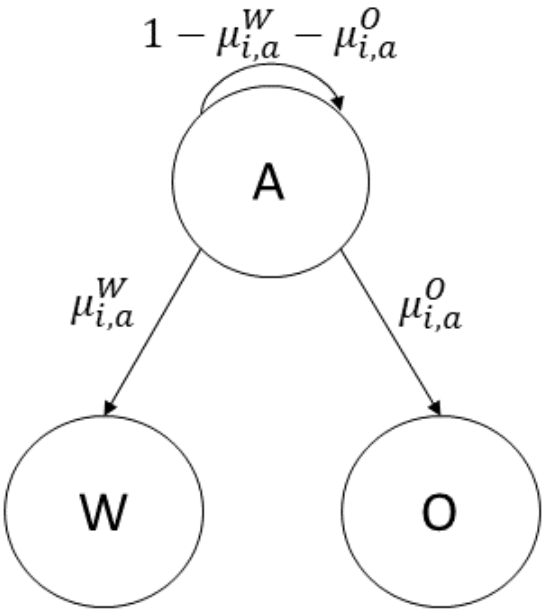


Figure 3-2 The demographic transitions of female elk (≥ 2 years old) in northern Yellowstone and adjacent Montana remaining alive (A), being killed by a wolf (W), or dying from a non-wolf, non-human cause (O). $\mu^{k}_{i,a}$ denotes the cause-specific probability of mortality per individual *i* at age *a* (2 to 24 years old).

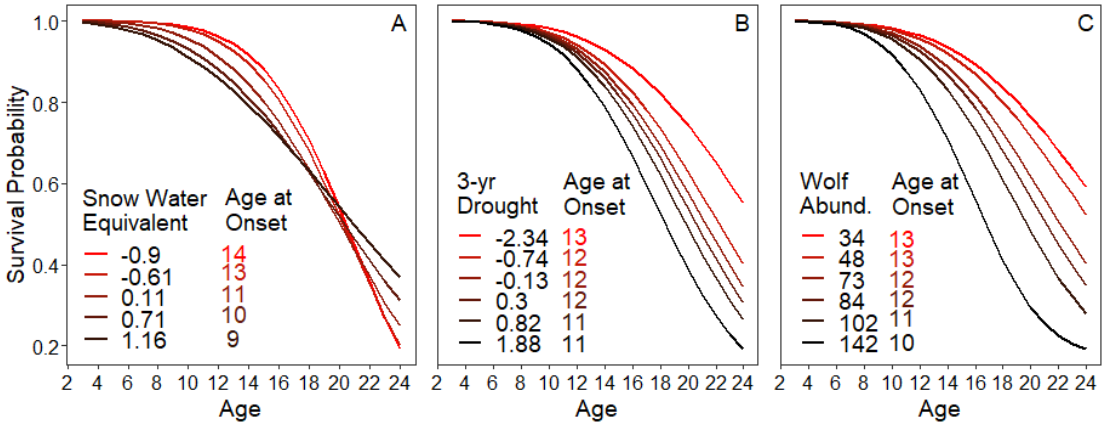


Figure 3-3 The probability of adult female elk surviving between subsequent ages from age 2 onward (e.g., 2 to 3, 3 to 4, etc.) in northern Yellowstone and adjacent Montana decreased as monthly snow water equivalent increased (A), as 3-year drought (standardized precipitation evapotranspiration index, SPEI; a monthly estimate that incorporates the previous 36 months where an increasing value indicates increasing dryness) increased (B), and as wolf abundance increased (C). In each panel, the other covariates are held at their respective means. Ages at onset of senescence are listed next to their associated covariate level.

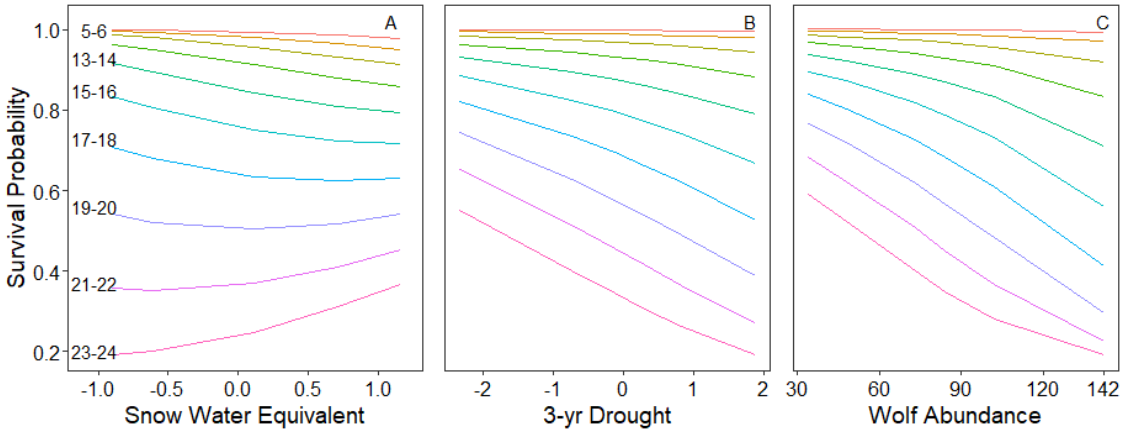


Figure 3-4 The probability of age-specific adult female elk survival from 5 years old and older in northern Yellowstone and adjacent Montana decreased as monthly snow water equivalent increased (A), as monthly 3-year drought (standardized precipitation evapotranspiration index, SPEI) increased (B), and as wolf abundance increased (B). Ages 2 through 4 are not shown because they overlap with age 5 (high survival across all covariate levels). Snow increased from -0.9 (no snow) to 5.10 during the study period, but here I present up to 1.16 (90th percentile) because of data limitations at higher snow levels.

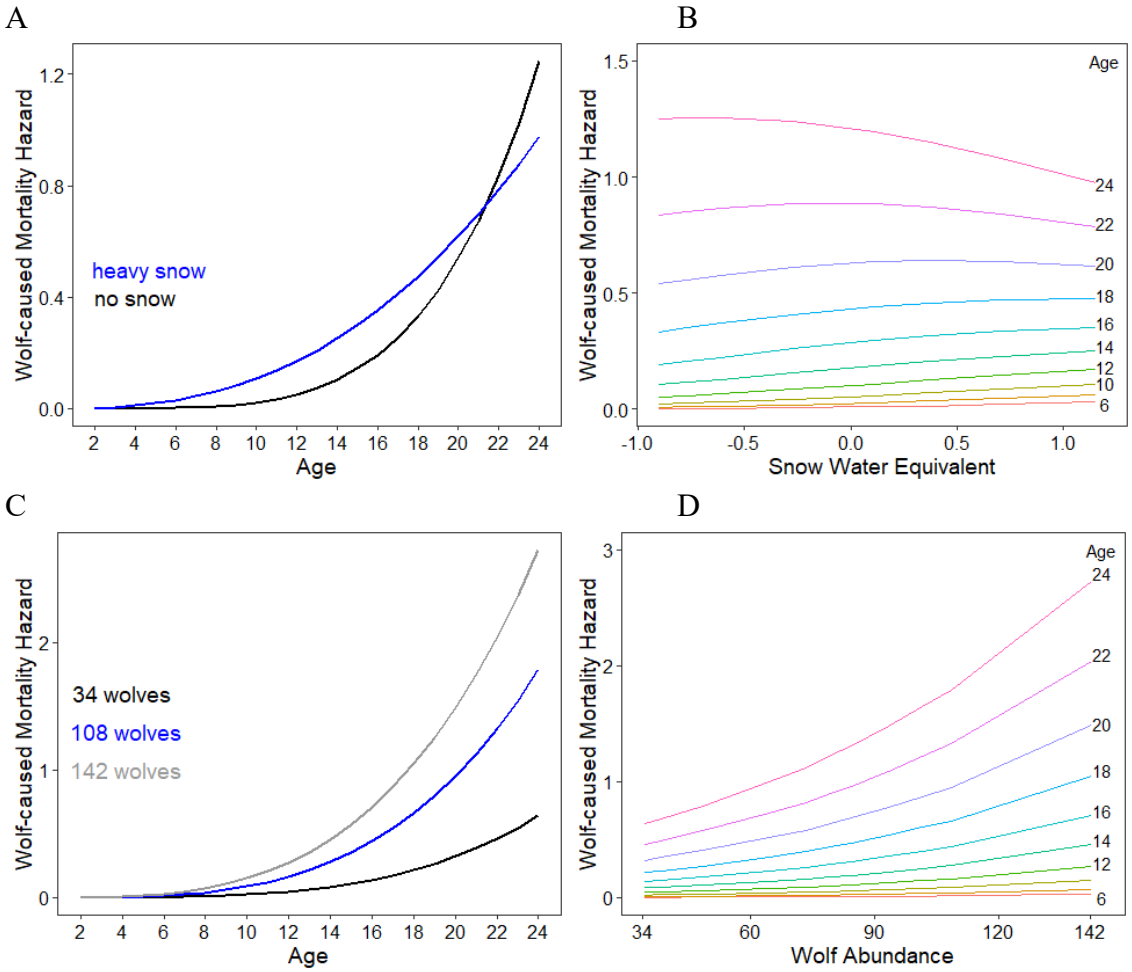
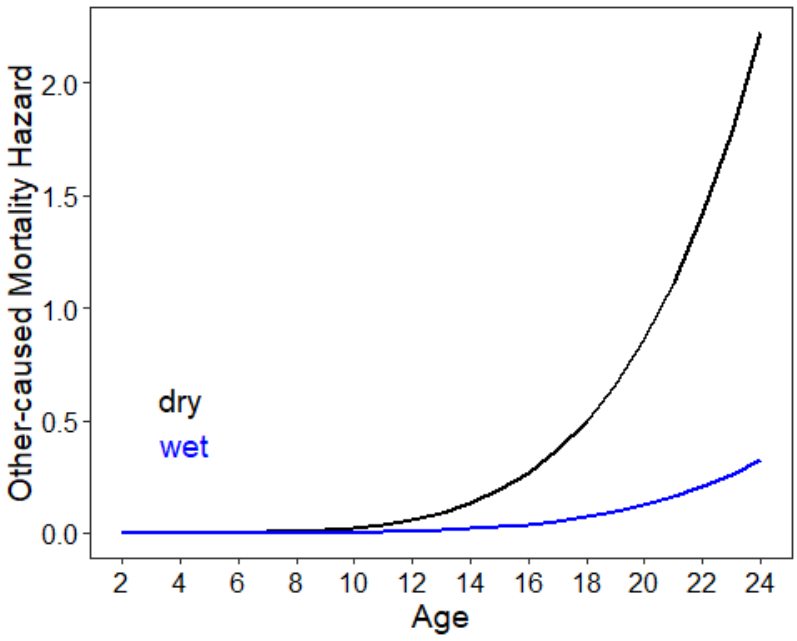


Figure 3-5 The wolf-caused mortality hazard of adult female elk (≥ 2 years old) in northern Yellowstone and adjacent Montana based on the top-ranked model with monthly snow water equivalent at the 90th percentile (1.16) and no snow (-0.90) estimates during the study period with wolf abundance held at its mean (A). The wolf-caused mortality hazard across monthly snow water equivalent for elk aged 6 to 24 years old, in two-year increments (B). The wolf-caused mortality hazard with wolf abundance at the minimum (34), 90th percentile (108), and maximum (142) estimates during the study period while snow water equivalent is held at its mean (C). The wolf-caused mortality hazard across wolf abundance for elk aged 6 to 24 years old, in two-year increments (D).

A



B

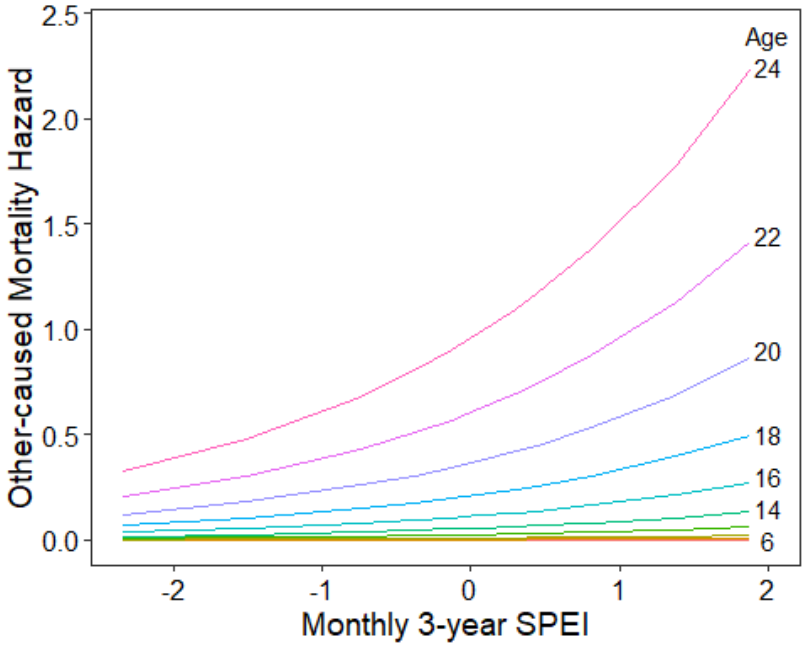


Figure 3-6 The other-caused mortality hazard of adult female elk (≥ 2 years old) in northern Yellowstone and adjacent Montana based on the top-ranked model with monthly 3-year drought (standardized precipitation evapotranspiration index, SPEI) at the minimum and maximum estimates during the study period while wolf abundance is held at its mean (A). The other-caused mortality hazard across monthly 3-year drought estimates from wet (negative) to dry (positive) for elk aged 6 to 24 years old (B).

CHAPTER 4

PREY STAGE REFUGIA LIMIT THE EFFECTS OF WOLF PREDATION ON ELK POPULATION DYNAMICS[3]

ABSTRACT

Predation risk can vary across prey life-history stages, yet demographic stage structure is often ignored when estimating the consumptive effect of a predator. The impact of a predator on prey population growth rate depends on the importance of the stage classes they most readily consume. I integrated data for female elk abundance and vital rates in Yellowstone National Park and adjacent Montana in the first transient life table response experiment to measure the contribution of stage refugia to prey population growth rates. Mortality of prime-aged (2-14 years old) female elk was the most important driver of changes in elk population growth rate from 2000-2016. The relative contribution of prime-aged mortality was 0.63, compared to 0.19 for calves, 0.13 for old adults (>14 years old), and 0.07 for yearlings. Pregnancy and population age structure had limited contributions (range -0.05 to 0.003 across all stage classes). A decrease in prime-aged mortality between the early and late periods was also the most important driver of a switch from a declining population growth rate in 2000-2005 to an increasing population growth rate in 2011-2016. Other, non-wolf-caused mortality of elk had double the contribution of wolf-caused mortality across the study and triple the contribution of wolf-

[3] Smith, L.M., Koons, D.N., Smith, D.W., Stahler, D.R., & MacNulty, D.R.

caused mortality between the two time periods to elk population dynamics. Harvest of prime-aged elk was likely the primary mortality cause within non-wolf mortality driving these findings because harvest decreased substantially from 2000-2016 and constituted 82% of non-wolf mortality of prime-aged, radio-collared elk. However, the total impact of wolf predation on elk population dynamics depends on the extent that wolves contributed to calf and yearling mortality, for which I lacked data. If the contributions of calf and yearling mortality were entirely due to wolves, then the wolf contribution (0.53) would exceed the contribution of other causes of mortality (0.48). However, it is likely that the wolf contribution is actually less than the contribution of other causes of mortality because of the numerous other predator species that also kill calves (e.g., bears and cougars). My results provide a unique demonstration of how a primary predator can have a secondary influence on prey population dynamics when it cannot frequently consume resistant prey individuals ('stage refugia') that contribute the most to prey population growth.

INTRODUCTION

The consumption of prey by predators is a key process in community ecology and a mechanism by which predators may suppress prey abundance and stoke an evolutionary arms race. It is well understood that there is variation in predation risk across prey life-history stages, and that predators can preferentially select particular stages of prey (Paine 1976; Pettorelli *et al.* 2011; Mukherjee & Heithaus 2013). Studies of predator-prey interactions often constrain prey stages to juveniles and adults because of data limitations. Yet, predators may distinguish between younger and older adults (DelGiudice *et al.* 2002, 2006; Wright *et al.* 2006), particularly because older adults are

more likely to be in poor body condition as they physically senesce, thereby creating a stage refugia within the younger adult stage (Miller & Rudolph 2011). Accounting for demographic stage structure may therefore be important for estimating the consumptive effect of a predator if the selected stages differ in their contribution to prey population dynamics compared to refuge stages (Marescot *et al.* 2015).

Demographic parameters (e.g., survival, reproduction, and age structure) can differentially contribute to population growth rate (Caswell 2001), and therefore the consumptive effect of a predator depends on the importance of prey stages to overall population dynamics. Predation of a life stage important for population growth rate will exert a comparatively stronger consumptive force on a prey population compared to predation of a life stage that has less impact on population growth rate (Koons *et al.* 2014; Hoy *et al.* 2015). Little is known about how the overall consumptive effect is partitioned across prey stages because few studies have assessed the impact of heterogeneous predation across prey life-history stages on prey population growth rates (but see Nilsen *et al.* 2009; Gervasi *et al.* 2012; Marescot *et al.* 2015). Furthermore, the influence of a stage-specific vital rate on population dynamics, relative to other vital rates, can change through time (Koons *et al.* 2017), and thus the consumptive effect of a predator may be temporally dynamic.

In Yellowstone National Park and adjacent areas of Montana, elk (*Cervus canadensis*) are the primary prey of wolves (*Canis lupus)*, though they are also killed by other predators, including humans outside of the park (Vucetich *et al.* 2005; Barber-Meyer *et al.* 2008; Ruth *et al.* 2019). There is substantial evidence that wolves remove large numbers of elk, their predation is stage-selective, and the degree to which their

predation is additive varies by stage class (whereby ‘stage’ refers to broad ‘age classes’ hereafter; Smith *et al.* 2004; Metz *et al.* 2012; Chapter 2). In the first decade after wolf reintroduction, human harvest and abiotic conditions were more responsible for a decline in the elk population than wolf predation (Vucetich *et al.* 2005; Varley & Boyce 2006; Wright *et al.* 2006; Eberhardt *et al.* 2007; MacNulty *et al.* 2020). However, no study has yet to account for stage-specific predation of adult elk by wolves and its impact on elk population dynamics in more recent years. Here, I use transient life table response experiments (LTRE) to assess the contribution of stage-specific wolf predation of adult elk (2-14 year old and > 14 years old) to elk population dynamics relative to other causes of mortality in adult elk, calf and yearling mortality, fecundity, and population stage structure. Transient LTREs were recently developed to accommodate non-stationary environments when assessing the influence of change in demographic parameters on past population dynamics (Koons *et al.* 2016, 2017). To my knowledge, this is the first study to use transient LTREs to estimate the contributions of cause-specific mortality and stage refugia to population dynamics.

To test the influence of stage-specific wolf predation and other causes of mortality on the population growth rate of elk in northern Yellowstone National Park and adjacent Montana beyond the park boundary, I integrated long-term data on the abundance and vital rates of female elk after the reintroduction of wolves in 1995 (June 2000 to May 2017). Given earlier findings that wolves select the oldest individuals (Smith *et al.* 2004; Wright *et al.* 2006; Metz *et al.* 2012, Hoy *et al.* 2021), which generally occur in low frequency in the population (Chapter 2) and have low reproductive rates (Wright *et al.* 2006; MacNulty *et al.* 2020), and that wolf predation of younger adults is infrequent and

partially compensatory (Chapter 2), I predicted that wolf predation of adult female elk has contributed little to elk population dynamics in northern Yellowstone compared to other causes of adult female mortality (e.g., malnutrition, harvest, cougars) and other vital rates (e.g., fecundity). Alternatively, wolf predation may have a stronger contribution to adult survival than other causes of mortality because the study period coincided with a reduction in female harvest and an increase in wolf abundance (MacNulty *et al.* 2020). This study sheds light on how stage-specific predation by an apex predator can influence prey population dynamics.

MATERIALS AND METHODS

Study area

The study encompassed the winter and summer ranges of the northern Yellowstone elk population (Houston 1982). The winter range (1520 km^2) comprises low-elevation (1500-2600 m) grasslands and shrub steppes around the Yellowstone River and its tributaries along the northern border of Yellowstone National Park and adjacent Montana (Lemke *et al.* 1998). Approximately 65% (995 km^2) of the winter range is located within the park, and the remaining 35% (525 km^2) extends north of the park boundary. The summer range includes the majority of Yellowstone and high-elevation areas outside the park to the north (elevation range 2206-3091 m across all summer ranges; Craighead *et al.* 1972; White *et al.* 2010). Wolves were reintroduced to Yellowstone in 1995-1997 (Bangs & Fritts 1996). The northern Yellowstone elk herd and wolf reintroduction are described in detail in Smith *et al*. (2004). Northern Yellowstone also supports populations of cougar (*Puma concolor*), black bear (*Ursus americanus*),

and grizzly bear (*U. arctos*) that prey on elk (Barber-Meyer *et al.* 2008, Ruth *et al.* 2019). Elk are harvested on the portion of the winter range that extends beyond the park boundary into Montana.

Data sources

Aerial count data

Elk count data were collected annually by the National Park Service and Montana, Fish, Wildlife, and Parks between December and March 2000 to 2017. Though counts were conducted in the winters of 2005/2006 and 2013/2014, they were considered unreliable because of weather conditions and pilot availability. Flight dates were variable through time due to flight conditions, and staff and plane availability. Elk were counted from 3-4 fixed-wing aircraft flying simultaneously in non-overlapping regions. Estimated counts were obtained for 2005/2006 and 2013/2014 via a state-space model and all counts were adjusted for sightability based on the group size of observed elk (Singer & Garton 1994; Tallian *et al.* 2017b). I multiplied the annual count data by the proportion of the population that was female (see *Sex and age structure data*) to convert each annual count to a female-only count.

Survival data

The National Park Service monitored the survival of 281 radio-collared female elk. From 2000 to 2003, 2005 to 2006, and 2011 to 2017, female elk (> 1 year old) were captured using net guns from helicopters (Hawkins and Powers, Greybull, Wyoming, USA; Leading Edge Aviation, Lewiston, Idaho, USA) or ground-darting and fitted with VHF or GPS collars. GPS collars included Telonics (Telonics, Mesa, Arizona, USA),

Advanced Telemetry Systems Inc. (Isanti, Minnesota, USA), and Vectronic Aerospace (Vectronic Aerospace GmbH, Berlin, Germany). Elk were captured and handled in accordance with applicable guidelines from the American Society of Mammalogists (Sikes 2016) and approved by the National Park Service Institutional Animal Care and Use Committees. Birth year of elk was determined with cementum analysis of an extracted vestigial canine tooth (Hamlin *et al.* 2000; Matson's Laboratory, Milltown, MT, USA). VHF-collared elk were tracked with ground-based and aerial radio telemetry one to four times per month depending on weather and staff availability. GPS collars were programmed to collect locations at 1-6-hour intervals depending on the season, collar type, and other study objectives. The tracking period started immediately after capture in 2000 and ended in 2008 due to logistical issues. Tracking resumed in 2011 and continued through May of 2017. The status (alive/censored/dead) and location of each elk was tracked until the collar failed or was removed, or until the elk died. When possible, failing collars were replaced. Elk that survived the monitoring gap were censored from the analysis for those years and re-entered when monitoring resumed.

All collars were equipped with a mortality sensor, and the National Park Service conducted field necropsies of dead elk to determine cause of death based on the carcass condition and location, blood trails, and evidence of predators including tracks, scat, bed sites, and wounds (Evans *et al.* 2006). Each elk was assigned a date of death based on timing of mortality signal or condition of the carcass. When months elapsed between the most recent resighting and a mortality, I assigned a death date halfway between when the mortality signal was heard and when the elk was last sighted (n=17 individuals). A cause of death was recorded if there was sufficient evidence to determine predator species or a

non-predator cause of death upon site visit. Hunters returned collars to the National Park Service from harvested collared elk. The number of collared elk killed by cause are shown in Tables 4-1 and 4-2.

I combined all non-wolf causes of mortality (malnutrition/winterkill, harvest, cougar, grizzly bear) into a separate category ('other-caused mortality') to isolate the effect of wolf predation on elk survival and due to data limitations for additional categories. Different from previous chapters, I included harvested elk in the other category but excluded two elk that died in vehicle collisions. I did not have an adequate sample size for harvested elk to formally include them as a separate cause of mortality. Cause of death was unknown for 21 mortalities. I used the frequency of known wolf-caused mortalities occurring inside (80% of 51) and outside (47% of 19) wolf pack boundaries to classify these unknown mortalities as either wolf-caused or other-caused according to whether the unknown mortalities occurred inside (N = 10) or outside (N = 11) wolf pack boundaries. Each mortality was located with respect to wolf pack boundaries of the corresponding year of death. I used wolf pack boundaries estimated with minimum convex polygons of wolf tracking data and provided by the Yellowstone Wolf Project (available at https://www.nps.gov/yell/learn/nature/wolf-reports.htm). I randomly assigned wolves as the cause of death for 80% of 10 unknown mortalities inside wolf pack boundaries (N = 8) and 47% of 11 unknown mortalities outside wolf pack boundaries (N = 5). I classified the remaining 8 unknown mortalities as other-caused.

Pregnancy data

A blood serum sample was collected from captured elk and tested with the pregnancy-specific protein B (PSPB) assay for pregnancy status, which is a standard method for nonlethal pregnancy assessment in elk (BioTracking, Moscow Idaho, USA; Sasser et al. 1986; Noyes et al. 1997). The National Park Service obtained the pregnancy status of 256 females aged 2 to >20 years old. I assumed that all elk testing positive for pregnancy gave birth to a single live fetus at an equal sex ratio. Therefore, I assumed that stage-specific birth rate was equal to stage-specific pregnancy rate. Pregnancy data were not available for all stages of elk in 2003, 2006-2009 and for elk >14 years old in 2013.

Sex and age structure data

Montana Fish, Wildlife, and Parks (MFWP) conducted an annual aerial classification survey by helicopter separate from the fixed-wing aerial count survey to estimate calf:cow and bull:cow ratios. MFWP biologists counted a subset of the visible elk population by age and sex category, including calf, cow, bull, and spike, providing information about the broad age and sex structure of the population. Here I used the calf and cow classifications for the female segment of the population to provide information about calf survival from the time of birth until the survey, and to assess possible discrepancies between the pregnancy data and realized birth rates (e.g., due to abortions) that would also influence the calf:cow ratios. I used this data to convert the annual count estimates to female-only counts based on the proportion of females observed, after combing cows with 50% of the calves.

Female elk were primarily harvested during the Gardiner late hunt (January – February), with only a few females harvested during the autumn general hunt (White *et*

al. 2003). All hunters were required to report to check stations, where MFWP biologists obtained teeth of harvested female elk on an annual basis from 2000 to 2008. Age-at-harvest was estimated by cementum analysis of a canine vestigial tooth (Hamlin *et al.* 2000; Matson's Laboratory, Milltown, MT, USA). Harvest was spread across ages each year, and hunters did not avoid a particular age or age range of female elk. I assumed that harvest data represented the underlying age structure of the population and that female elk were similarly susceptible to harvest across ages. I used the age structure of the harvest data to inform the annual estimates of abundance each age of elk.

Data analysis

I developed an integrated population model (IPM; Besbeas *et al.* 2002; Schaub & Abadi 2011) to explicitly link the available sources of elk data mentioned above to estimate annual vital rates according to stage class (see below for definitions of these classes) and stage-specific abundances (as well as total abundance) over the study period of June 2000 – May 2017. The use of an IPM was crucial for estimating annual calf and yearling survival because I lacked direct survival and cause-of-mortality data over the study period for these developmental phases of life, and the IPM framework allowed me to use information from the annual count and classification surveys in combination with published calf survival estimates from a limited time period and other locations to inform these vital rates. The IPM similarly provided more robust estimates of pregnancy rates compared to estimates from the limited dataset on its own, enhancing information about this important component of recruitment to the population.

Correlation between wolf and other causes of adult female mortality

The extent to which wolf predation of adult female elk is additive varies by elk stage class (Chapter 2); therefore, I estimated the correlation between wolf and other causes of mortality to inform the estimates of wolf-caused mortality based on the degree to which wolf predation was additive in each stage class. I used the collared elk survival data to estimate cumulative incidence functions in a competing risks framework (csm function in R package wild1, Heisey & Patterson 2006). I estimated annual wolf-caused mortality and survival probabilities by 'stage class', based on established adult ages of female elk previously determined to be 'resistant' (2-14 year olds) and 'susceptible' (>14 year olds) to wolf predation (see Chapter 2) and because data limitations prohibited annual estimates by each adult age. Data were available to estimate probabilities for 2000-2003, 2005-2007, and 2011-2016. I converted wolf-caused and other-caused mortality probabilities to hazards (Ergon *et al.* 2018) and calculated their correlation. I excluded years without wolf-caused mortality. This correlation was later used to inform adult elk survival in the process model (see *Priors* below).

Process model

I used a female-only, pre-birth pulse matrix projection model (Caswell 2001) to define the structured process of population dynamics between 2000 and 2016 for the northern herd of elk in Yellowstone. I focused on females only because in ungulate populations, they have a strong impact on population growth relative to males (Gaillard *et al.* 2000; Bonenfant *et al.* 2009; Eacker *et al.* 2017), and they are therefore the most important segment of a population for estimating the consumptive effect of predation.

Each year was modeled annually from 1 June through 31 May (i.e., the 'elk year') based on the median birthdate of elk calves in Yellowstone, and when referring to years, for example, 2016 refers to the elk year June 2016 through May 2017.

I structured the matrix population model with each individual age (1-20) to avoid estimating transitions within and between stage classes (as would be necessary using a stage-based projection model) and because I lacked adequate data to estimate annual age-specific vital rates. I considered age 20 as a final, absorbing age class that included elk ≥ 20 years old because of the rarity of these individuals. The process model was therefore represented by a 20 x 20 matrix

$$\begin{bmatrix} N_1 \\ N_2 \\ N_3 \\ \dots \\ N_{19} \\ N_{20} \end{bmatrix}_t = \begin{bmatrix} 0 & 0.5 * f_2 * S_0 & 0.5 * f_{3\text{-}14} * S_0 & \dots & 0.5 * f_{>14} * S_0 & 0.5 * f_{>14} * S_0 \\ S_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & S_{2-14} & 0 & 0 & 0 & 0 \\ 0 & 0 & S_{2-14} & \dots & 0 & 0 \\ 0 & 0 & 0 & 0 & S_{>14} & S_{>14} \end{bmatrix}_{t-1} * \begin{bmatrix} N_1 \\ N_2 \\ N_3 \\ \dots \\ N_{19} \\ N_{20} \end{bmatrix}_{t-1} \quad (1)$$

where N_a denotes age-specific abundance, 0.5 the multiplier to track only female offspring in the female-based model under the assumption of an equal sex ratio at birth, and f_{sc} and S_{sc} denotes the pregnancy and survival probabilities, respectively, for stage classes (sc): calf (subscript 0), yearling (subscript 1), primiparous adult (subscript 2), young adult (subscript 3-14), and old adult (subscript >14), from year t-1 to t. Colloquial 'yearling' pregnancy refers to elk that are ~1.75 years of age at the time of testing and who will turn 2 years old at the time of giving birth for the first time (in line with time steps of the matrix). Hence, I use the subscript 2 to denote the age of primiparity as opposed to the age of pregnancy. Note that all vital rates in Eqn 1 were allowed to vary over time, and I denote time on the matrix rather than each vital rate for brevity.

I used a binomial distribution to model demographic stochasticity in the number of yearlings in year *t* as a function of calf survival (S_0), adult pregnancy (f_{sc}), and the number of adult females by stage class (N_{sc}) in year *t-1*

$$N_{1,sc,t} \sim binomial\ (f_{sc,t-1} S_{0,t-1}, N_{sc,t-1}) \quad (2)$$

The total number of female yearlings (N_1) in year *t* is the sum of the yearlings produced by each stage class of mother ($N_{1,sc,t}$), which was then adjusted for a 0.50 sex ratio.

I also used a binomial distribution to model demographic stochasticity in the number of adult elk ($N_{2,\ldots,}N_{19}$) in year *t* as a function of stage-class survival (S_{sc}) and abundance of the preceding age (a; $N_{1,\ldots,}N_{18}$) in year *t-1*.

$$N_{a+1,t} \sim binomial\ \left(S_{sc,t-1}, N_{a,t-1}\right). \quad (3)$$

Similarly, the binomial distribution for the abundance of elk in the final age class (N_{20}) in year *t* was a function of the oldest stage-class survival ($S_{>14}$) and abundance of the current and preceding ages (N_{19}, N_{20}) in year *t-1*.

$$N_{20,t} \sim binomial\ \left(S_{>14,t-1}, N_{19,t-1} + N_{20,t-1}\right) \quad (4)$$

Data likelihoods

The vital rates in the matrix population model were estimated directly by the likelihoods for survival and pregnancy that follow. The likelihood for the annual aerial count informs the sum of the age-specific abundances from the process model (Eq. 1) Additional sources of data on elk population age structure were combined with the matrix model in the IPM framework to inform the relative proportions of each age estimated in the matrix projection model, which also indirectly informs estimation of the vital rates.

Aerial count

I used a Poisson distributed likelihood to relate the annual female elk count (y; treated as data) to the latent total abundance of female elk (N_{tot}) in year t predicted from the process model in Eqn 1.

$$y_t \sim Poisson(N_{tot,t}) \quad (5)$$

Survival

I used the elk radio-collar data to inform annual cause-specific hazards for wolf predation (w) and other (o) causes of mortality, and overall survival (S). I constrained age-specific variation in the mortality and survival parameters using stage classes of young elk (2-14 years old) and old elk (>14 years old) due to data limitations, and because wolves tended to kill elk over age 14 more than younger elk (Chapter 2). I modeled the individual failure times of individuals (D_i) in each year (t) with a proportional hazards Weibull distribution and accounted for left-truncation with a staggered-entry design. The Weibull likelihood includes a shape parameter (ω) and a scale parameter (Λ),

$$D_{t,i,m} \sim Weibull(\omega_m, \Lambda_{t,i,m}) \quad (6)$$

for each cause of mortality (m). I modeled Λ as a function of elk stage class with a correlated random year effect (η) based on the covariance between annual wolf predation and other causes of mortality for each young and old stage classes (see *Correlation between wolf and other causes of mortality*). The cumulative hazard is defined as $H_t = \Lambda_t x^{\omega}$ for each cause of mortality and each stage class. The survival function across all causes of mortality is defined as $S_t = \exp(-(H_{w,t} + H_{o,t}))$ for each stage class. I derived

annual survival by setting fine-scale time (x) in the cumulative hazard equation to 365 days.

I modeled yearling survival by multiplying an intermediate parameter (ψ) by the survival probability for 2-14 year olds to constrain yearling survival to be less than the young adult survival because I did not have data on yearling survival (Lubow & Smith 2004; Raithel *et al.* 2007). The intermediate parameter was modeled with an informative prior distribution as beta(20.84, 2.76) based on mean annual survival (0.883) and process variance (0.0042) for yearling elk from the Raithel *et al.* (2007) meta-analysis of elk across the Western U.S. I doubled the process variance from the meta-analysis to reduce the prior information given to the intermediate parameter.

Pregnancy

I estimated the annual fecundity rate (f) according to the stage classes defined in the previous section. I assumed a litter size and fetal survival rate of 1, making pregnancy the modeled parameter controlling fecundity (i.e., the production of new offspring; though see my earlier comment about the ability of the classification data to adjust estimates of f in the IPM). I modeled the number of pregnant individuals (P) in the sample of each adult stage class (subscript sc; 2 year olds, 3-14 year-olds, and >14 year olds) using a binomially distributed likelihood with annual fecundity rate (f) and n_{sc} sampled individuals in each year t.

$$P_{sc,t} \sim binomial(f_{sc,t}, n_{sc,t}) \quad (7)$$

Harvest

I used a multinomial likelihood to relate the sample number of each age harvested ($k_{a,t}$) in year *t* to the estimated latent proportional abundance of each age ($Nprop_{a,t} = N_{a,t} / Ntot_t$) predicted by the process model in Eqn 1 and the total number harvested (b) in year *t* (note that as described above in *Data sources*, total annual harvest removal data were available for only the first nine years of the model timespan).

$$k_{a,t} \sim multinomial(Nprop_{a,t}, b_t) \quad (8)$$

Calf:cow

I also used a binomially distributed likelihood to relate the number of female calves (c) observed each spring (at approximately 9 months old, with an assumed 50:50 sex ratio) to the estimated latent proportion of yearlings each year ($Nprop_{1,t} = N_{1,t} / Ntot_t$) predicted by the process model in Eqn 1 (assuming that little mortality occurs between 9 and 12 months of age) and the total number of female elk (e) observed in year *t* during the classification surveys.

$$c_t \sim binomial(Nprop_{1,t}, e_t) \quad (9)$$

Priors

*A*ge-specific elk abundance (N_a) in the first year was assigned a Norm($Ninit_a$, 1.0^{-5}) distribution truncated at zero, where $Ninit_a$ was based on the corrected abundance of elk (Tallian *et al.* 2017b) adjusted by the proportion of elk that were female and the proportion of each age (1-20+) as estimated by Hoy *et al.* (2020). I rounded the initial abundances to conform to the requirement of the binomial distribution used to model demographic stochasticity in each $N_{a,t}$.

I used vague priors for the Weibull shape (ω) and scale (Λ) parameters for adult survival because the available prior information (Houston 1982; Eberhardt 2002; White et al. 2003; White & Garrott 2005; Evans *et al.* 2006; Hamlin *et al.* 2009) did not meet the criteria of the study (i.e., post-wolf reintroduction, independent of the data used here, and a competing-risk framework). Thus, I assigned each cause-specific shape parameter an exp(0.01) distribution. The cause-specific scale parameters included three hyper-parameters on the logit scale, which functionally allowed the cause-specific scale parameters to differ between the two stage classes. I assigned alpha (α) a vague Norm(0, 1) distribution, I set β to zero for the old age class (>14 years old) and assigned a vague Norm(0, 1) distribution for the young stage class (2-14 years old). However, I informed the scale parameters with the correlated random effects (η; refer to previous section *Correlation between wolf and other causes of mortality*), which I assigned a mNorm(0, σ_{ac}) distribution by each stage class. σ is the variance-covariance matrix for wolf and other causes of mortality by stage class (*sc*). To inform the correlation coefficient for young elk, I assigned a Norm(-0.26, 0.08) distribution on the hazard scale and for old elk, I assigned a Norm(-0.36, 0.04) distribution, both truncated at 1 and -1. For each stage class, I assigned σ a unif(0, 5) distribution.

To estimate the cause-specific mortalities used in the correlations, I constructed a beta distribution using moment matching for each annual wolf-caused mortality and survival probability and then sampled 1,000 realizations from each distribution per year. I used these Monte Carlo realizations from within the range of uncertainty for each annual probability (Wolfe *et al.* 2015) to account for uncertainty in the annual estimates. On each Monte Carlo iteration, I fit a corrected slope (model slope divided by intercept;

Brodie *et al.* 2013) of the relationship between the simulated survival and wolf-caused mortality probabilities, resulting in a total of 1,000 slope and intercept estimates.

I used the relationship between wolf mortality and survival to recalculate 1,000 estimates of survival, thereby eliminating pairs of simulated wolf mortality and survival estimates where wolf mortality was greater than survival. For every simulation, I calculated other-caused mortality as $1 - wolf\ mortality - survival$. I excluded pairs of simulations where the sum of survival and wolf mortality exceeded one and therefore other mortality was negative.

For calf survival, I modeled an informative prior for calf survival at each time step as beta($S_{0,t}$| 0.27,0.96) based on the mean annual survival (0.22) reported in Barber-Meyer *et al.* (2008) for northern Yellowstone. I used the process variance (0.04) for calf survival reported in the Raithel *et al.* (2007) elk meta-analysis because process variance was not available in Barber-Meyer *et al.* (2008). I doubled the process variance from the meta-analysis to reduce the prior information given to the parameter. I modeled the prior estimates on the logit scale with the temporal random effect (φ), where I assigned φ a Norm(0,υ) distribution, $\upsilon = \kappa^{-2}$, and I assigned κ a gamma(8.15, 37.03) distribution with parameters moment-matched from the aforementioned estimates of calf survival.

I modeled a vague prior for adult pregnancy as beta(1,1). I did not have data on yearling pregnancy. Thus, I assigned yearling pregnancy an informative beta(p_2|7.78,31.52) distribution based on the pregnancy rate and process variance reported in Raithel *et al.* (2007) for elk in the western United States. I did not use information from Raithel *et al.* (2007) for adult pregnancy because the wide temporal and spatial distribution over which the meta-analysis encompassed may inappropriately

influence the posterior estimates in my IPM for Yellowstone. I lacked pregnancy data for 6 years of the study period for young adults and 7 years for old adults. I therefore modeled temporal variation in pregnancy using a temporal random effect on the logit scale (ε) for its shrinkage estimation properties, where I assigned ε a Norm(0, τ) distribution, $\tau = \varsigma^{-2}$, and I assigned ς a unif(0, 5) distribution.

I combined the likelihoods of the five datasets to improve estimates of vital rates for which data were missing for all or some years. A directed acyclic diagram for the model is provided in Fig. 4-1. I defined the full posterior and joint distributions as

$$Pr\begin{bmatrix} N_{tot,t}, Nprop_{a,t}, N_{a,2000}, \omega_m, \alpha_m, \\ \Lambda_{i,m,t}, \beta_{m,t}, \eta_{sc,t}, \sigma_{sc}, \\ f_{sc,t}, p_{sc}, \varepsilon_t, \varsigma, S_{0,t}, \\ calf, \varphi_t, \kappa, S_{1,t}, yearl, \psi, S_{2-14,t} \end{bmatrix} y_t, D_{i,m,t}, P_{sc,t}, n_{sc,t}, k_{a,t}, b_t, c_t, e_t, \ Ninit_{a,2000}], \quad (10)$$

where *a* is age 1 to 20 years old, *m* is wolf or other cause of mortality, *sc* is stage class, *i* is individual collared elk, and *t* is year.

Model fitting

I fit the model using Bayesian methods with JAGS (Plummer 2017) via the jagsUI package in R version 4.0.3 (R Core Team 2017). Parameter posterior distributions were estimated using Markov chain Monte Carlo methods. I ran three chains for each parameter and examined trace plots to determine that an adequate burn-in period was reached. I sampled from the posterior distributions with 4,000,000 iterations, discarded the first 3,000,000 as burn-in, and retained every 100th sample for a total of 10,000 samples from each chain (30,000 samples total). Thinning was necessary to reduce the

processing time for the life table response experiments. All parameters were checked for convergence.

Transient life table response experiments

Following Koons *et al.* (2017), I conducted a transient life table response experiment (LTRE) to determine the contribution of all estimated vital rates to temporal variation in realized population growth rates for female elk in northern Yellowstone during the study period. Unlike classical life-stage simulation analysis and LTRE, the transient LTRE does not assume a stationary environment nor a stable age distribution, and can decompose the contributions of vital rates and age structure to realized population growth rates (Koons *et al.* 2016). Contributions from any vital rate can also be decomposed into its lower-level components. For example, survival can be decomposed into cause-specific mortality to assess the impact of a given predator on a prey population's growth rate. Thus, transient LTREs constructed with cause-specific mortality can provide novel insight into a predator's past consumptive effects on the prey population.

The transient LTRE is based on a structured population model such as $\mathbf{n}_{t+1} = \mathbf{A}_t\mathbf{n}_t$, where $\mathbf{A}_t$ is the projection matrix containing age-specific vital rates of elk in year t, and $\mathbf{n}_t$ denotes a vector of female elk abundance by age (1-20; Eqn 1). To include cause-specific mortality, survival transitions in $\mathbf{A}_t$ were parameterized as a function of wolf- and other-caused mortality hazards. I defined realized population growth rate as $\lambda_t = N_{t+1}/N_t = \| \mathbf{A}_t\mathbf{n}_t \| / \| \mathbf{n}_t \|$ over the time interval $[t, t+1]$, where $\| \ \|$ is the sum of the vector elements. From this formula I calculated the sensitivity with respect to change in each demographic parameter ($\partial\lambda_t / \partial\theta_{i,t}$), where $\boldsymbol{\theta}_t$ denotes a vector of the parameters of $\mathbf{A}_t$ and the

proportional values of $\mathbf{n}_t$. These sensitivities are implemented in the transient LTRE to estimate the contribution of variability in each vital rate and component of age structure (θ_i) to the temporal variance of λ_t.

$$contribution_{\theta_i}^{var(\lambda_t)} \approx \sum_j cov\left(\theta_{i,t}, \theta_{j,t}\right) \frac{\partial \lambda_t}{\partial \theta_{i,t}} \frac{\partial \lambda_t}{\partial \theta_{j,t}} \Big|_{\bar{\theta}} \quad (13)$$

Rather than adult survival, I used the cause-specific cumulative hazard estimates by adult stage class in the calculations of LTRE contributions. This change allowed me to compare the contribution of wolf predation to the contribution of other sources of adult female mortality. However, I could not estimate the contribution of wolf predation on calves or yearlings because of the lack of annual cause-specific mortality for these stage classes. I then estimated the overall contribution of each cause of mortality by summing their respective age-class contributions. Similarly, I estimated the contribution of total mortality for each stage class irrespective of cause of mortality. I excluded inference for the years 2008, 2009, and 2010 because data limitations for adult survival and mortality resulted in large variances and poor convergence of vital rate estimates for those years (i.e., the telemetry gap years). I scaled the median contribution values to sum to one. A larger contribution value indicates a stronger influence of temporal variation in that demographic parameter to temporal variation in realized population growth rates.

The LTRE requires variation in the demographic parameters to estimate their contribution. If a parameter were fixed through time (with variance equal to zero), then its LTRE contribution would be zero. For some populations, this situation could occur if the exact same number of individuals were harvested each year and harvest mortality was a separate demographic parameter in the model. For some analyses, this situation could occur if data was not available for a demographic parameter and a single value was used

across years. However, I obtained annual estimates for all demographic parameters by implementing an IPM to estimate annual demographic parameters.

I conducted a secondary transient LTRE to decompose the contribution of vital rates to change in the geometric mean rate of population growth ($\Delta\log\lambda_g$) between two time periods of equal duration (Koons *et al.* 2016, 2017). This analysis estimates the direct effect of changing vital rates (**A**), and the indirect effect of vital rates via changes in age structure ($\hat{\boldsymbol{n}}$), to change in the geometric mean rate of population growth over specified time periods. I compared the early years of the study period ('elk years' 2000-2005; subscript a), when wolf abundance was at its peak, to the late years ('elk years' 2011-2016; subscript b) when wolf abundance was lower. The transient LTRE decomposition of $\Delta\log\lambda_g$ is

$$contribution_{\theta_i}^{\Delta log\lambda_g} \approx \left(log\mu_{i,b} - log\mu_{i,a}\right)\left(\bar{e}_{\mu_i}^{\boldsymbol{A}} + \bar{e}_{\mu_i}^{\hat{\boldsymbol{n}}}\right) + (log\sigma_{i,b} - log\sigma_{i,a})(\bar{e}_{\sigma_i}^{\boldsymbol{A}} + \bar{e}_{\sigma_i}^{\hat{\boldsymbol{n}}}) \quad (10)$$

where μ is the mean of vital rate i over time period a or b, σ the standard deviation for time period a or b, and $\bar{e}$ the real-time elasticity for a reference population with mean of per time step vital rates between the two time periods (Koons *et al.* 2016, 2017).

Demographic parameters with the largest contributions are interpreted as the primary parameters influencing the change in population growth rate. A mortality contribution with a positive value would indicate that reduced mortality was responsible for the change in growth rate between the two periods.

RESULTS

Integrated population model

The model achieved convergence for all estimated parameters ($\hat{R} < 1.05$ and trace plots indicating mixing among the MCMC chains), except for those years with data limitations for adult survival (2008, 2009, and 2010). The model provided estimates of elk abundance in northern Yellowstone from June 2000 to May 2017 ('elk years' 2000 to 2016) that tracked the population trends observed in annual aerial counts (Fig. 4-2A). The abundance of elk 2-14 years old generally followed the trend of overall abundance, declining until 2012 and then increasing modestly. The abundances of less common elk >14 years old increased through 2008, consistent with Hoy *et al.* (2020), decreased from 2009 to 2012, and then slightly increased to 2016 (Fig. 4-2A). The annual calf abundance fluctuated through time (Fig. 4-2A). The proportion of adult stage classes varied through time (Fig. 4-2B), whereby the proportion of elk 2-14 years-old was slightly lower during 2013-2016 compared to the early 2000s. In contrast, the proportion of elk >14 years old was slightly higher in 2014-2016 compared to the early 2000s. The proportion of yearling elk fluctuated across the study period.

Annual elk survival probabilities were highest for elk yearlings and 2-14 years old, followed by elk >14 years old, and calves (Fig. 4-3A). Adult survival of both stage classes dipped in 2002 and 2004, but survival of 2-14 year-old elk remained relatively high and fairly stable over the last few years of the study (2012-2016, Fig. 4-3A) when the population began to increase in abundance (Fig. 4-2A). Calf survival was higher in the later years (2012-2016) than in the early years (2000-2006) and seemed to track survival of 2-14 year-old elk (Fig. 4-2A).

Annual adult cause-specific cumulative hazards were higher for elk >14 years old compared to elk 2-14 years old (Figs 4-3C, 4-3D). Cumulative hazards of wolf predation (Fig. 4-3D) were greater than the cumulative hazards of other-caused mortality (Fig. 4-3C) for elk >14 years old for most years of the study (notable exceptions in 2002 and 2004-2005). Annual adult elk pregnancy was highest for elk 3-14 years old, followed by elk >14 years old, and elk 2 years old (Fig. 4-3B). Pregnancy of all stage classes was fairly stable through time, but imprecisely estimated (Fig. 4-3B).

Transient life table response experiments

The transient LTREs combined a) the sensitivity of realized population growth rate to equivalent, infinitesimal changes in both vital rates and age structure with b), the temporal process variance in these demographic parameters to estimate the contribution of variation in each demographic parameter to overall temporal variance in realized elk population growth rates (0.0074; 95% CI: 0.0066, 0.0083; Table 4-3). Across all ages, the relative contribution of mortality was 1.01 (i.e., scaled so that contributions sum to 1; Fig. 4-4A). Fluctuations in 2-14 year-old elk mortality contributed the most to temporal variation in realized population growth rates (0.63; Table 4-3; Fig. 4-4B). Calf mortality had the next greatest contribution (0.19), followed by mortality of elk >14 years old (0.13; Table 4-3 Fig. 4-4B). Components of population structure (-0.01), yearling mortality (0.07), and pregnancy (0.004) at all stages contributed the least (Table 4-3). Fluctuations in other, non-wolf causes of adult mortality, which included hunting, contributed more to variation in population growth rate (0.48) than did wolf-caused adult mortality (0.28; Table 4-3; Fig. 4-4B).

I could not formally estimate the total wolf contribution without annual cause-specific mortality estimates of calves and yearlings. I could, however, consider a heuristic approach to outline possibilities for the wolf contribution. Approximately 16% of the elk calves killed by a known cause in Barber-Meyer *et al.* (2008) were killed by wolves (excluding calves where wolves were present with other predators but including calves that died of an unknown non-predator cause). If 16% of the calf mortality contribution was due to wolves, then 0.03 of the contribution would come from wolves and the remaining 0.16 would come from other causes. Under such a scenario, the total wolf contribution across calves and adults would be 0.31. For comparison, if all of the calf mortality contribution was hypothetically due to wolves, then the wolf contribution would be 0.47. Including the entire yearling contribution, the wolf contribution would be 0.38 under the former estimate and 0.53 under the latter estimate.

If the entire contribution of calf and yearling mortality was hypothetically due to wolves, then wolf predation (0.53) would exceed the contribution of other causes of mortality (0.48). This scenario is unlikely given that other predator species also kill calves (Barber-Meyer *et al.* 2008, Ruth *et al.* 2019). Therefore, the total contribution due to wolves might not exceed the contribution due to other-caused mortality. However, if Barber-Meyer *et al.* (2008) underestimated wolf predation of calves (MacNulty *et al.* 2020) and if a portion of the yearling contribution is due to wolves, then the wolf contribution is likely larger than 0.31. These estimates are merely a best guess based on the limited data available for cause-specific sources of mortality over time for elk calves and yearlings.

More 2-14 year-old collared elk were killed by wolves than by harvest or by all other causes combined during the study (Table 4-1). However, harvest comprised 82% of the non-wolf caused mortality of 2-14 year-old elk, and the number of harvested elk was higher in the early years compared to the late years (Table 4-1). Wolf predation had a smaller LTRE contribution on 2-14 year-old mortality than did other causes (Table 4-3) because wolf predation was temporally stable while other-caused mortality fluctuated through time with a declining trend that was largely due to reduced harvest in later years (Table 4-1).

More old elk >14 years were killed by wolves than by other causes of mortality (Table 4-2). Only one was killed by harvest (Table 4-2); therefore, the contribution of other mortality was not likely to be driven by harvest for this stage class. About half of the other mortalities were due to unknown, non-wolf causes (Table 4-2), so the primary cause of mortality driving the contribution of other-caused mortality is unknown.

The female elk population declined between 2000 and 2005 ($\log\lambda_g$ = -0.062, 90% BCI -0.066, -0.057) and increased between 2011 and 2016 ($\log\lambda_g$ = 0.052, 90% BCI 0.047, 0.058; Fig. 4-2A). When I applied the transient LTRE to the difference in geometric mean population growth rate between time periods (Eq 14), reduced mortality of 2-14 year-old elk (Figs 4-3C, 4-3D) was the primary driver of the switch from a declining population in the early years to an increasing population in the later years (Fig. 4-5). The direct effects of reduced mortality (**A**) of 2-14 year-old elk due to non-wolf causes of mortality in the later years had a larger contribution than that due to wolf predation (Fig. 4-5). The direct effects of reduced mortality of calves and adults >14 years old due to other causes had the next largest contributions (Fig. 4-5). Indirect effects

of the vital rates via changes in the proportionate abundance ($\hat{\boldsymbol{n}}$) of the stage classes had little contribution (Fig. 4-5).

If the direct effects of calf and yearling mortality were all due to other, non-wolf causes of mortality, the total contribution of wolf mortality of adult elk to the difference in geometric mean population growth rate between time periods would be approximately 16% of the direct effects of other causes of mortality. By contrast, if the direct effects of calf and yearling mortality were all due to wolves, the total contribution of direct effects of wolf mortality would be approximately 70% of the direct effects due to other causes of mortality of adult elk. Together, these results indicate that while the total contribution due to wolves between the two time periods is unknown, it was less than the contribution due to other causes of mortality.

DISCUSSION

This study is the first to demonstrate how transient LTREs can be used to estimate the consumptive effect of a primary predator on its prey population. Temporal variation in prime-aged (2-14 years old) adult female mortality was the primary driver of changes in elk population growth rate from June 2000 to May 2017 ('elk years' 2000-2016), especially via other, non-wolf causes of mortality. The next largest contributors were calf mortality and old (>14 years old) adult female mortality. Fecundity and population stage structure contributed little to elk population growth rate.

Temporal variation in other-caused mortality of prime-aged elk contributed more to elk population dynamics than did wolf predation of prime-aged elk. Prime-aged elk have higher pregnancy rates than elk >14 years old that are unaffected by elk or predator density (Proffitt *et al.* 2014), they are infrequently killed by wolves or die of natural

causes (Evans *et al.* 2006; Wright *et al.* 2006; Chapter 2; Chapter 3), but they comprised 64-85% of harvested elk during the first nine years of the study period. In contrast, temporal variation in wolf predation of old elk contributed more to elk population dynamics than did other-caused mortality, but the contribution of either cause of mortality was small for this stage class, in part because old individuals have low reproductive value and thus population growth rate is relatively insensitive to changes in the vital rates of old, senescent elk.

The total contribution of wolves is unknown because of the lack of data to decouple calf and yearling mortality into wolf and other causes of mortality. The relative contribution of wolves combined across adult elk age classes comprised only 0.28 of the temporal variation in elk population growth rate, after accounting for the degree to which predation was additive. Only if 79% or more of the calf and yearling mortality contribution was due to wolves would the wolf contribution equal or exceed that of the contribution due to other causes of mortality (Table 4-3). Even under this extreme scenario, the results demonstrate that wolves were not the primary driver of changes in the realized population growth rate of northern Yellowstone elk during 2000-2016.

Prior to wolf reintroduction, models predicted that the elk population would decline 5-30% in the presence of wolves, based on elk as their primary prey (Boyce 1993; Mack & Singer 1993). Studies conducted a decade after wolf reintroduction found little influence of wolf predation on elk population decline (Vucetich *et al.* 2005; Wright *et al.* 2006; Eberhardt *et al.* 2007) or a wolf influence that was less than harvest (Varley & Boyce 2006). Recent studies suggested that wolves had minimal influence on elk abundance until the second decade after wolf reintroduction (MacNulty *et al.* 2020, Metz

et al. 2020). Wolves generally select elk calves and older cows (Metz *et al.* 2012; Hoy *et al.* 2021), stage classes that contributed less to population growth rate than did prime-aged elk (Table 4-3). However, my results suggest a potentially greater contribution of wolves from 2000-2016 than earlier studies, perhaps because wolves were responsible for 56% of the radio-collared, prime-aged elk that died (Table 4-1).

The transient LTRE estimates of contributions to the difference in population growth rates between two time periods suggested that reductions in non-wolf caused mortality across adult elk was the primary factor contributing to a switch from a declining population to an increasing population (Fig. 4-5). Wolf predation of radio-collared, prime-aged elk remained fairly stable throughout the study, despite lower wolf abundance in the later years (Smith *et al.* 2020). The LTRE estimates include both vital rate sensitivity and process covariance. Therefore, the model estimated a contribution (0.21 relative contribution; Table 4-3) of wolf-caused mortality of prime-aged elk despite its low temporal variability. But the greater temporal variation in other-caused mortality (particularly harvest) led to a greater contribution of other-caused mortality than wolf-caused mortality for prime-aged elk. It is important to evaluate the LTRE results in light of the temporal variance in population growth rate. In the absence of harvest (perhaps in the future), wolf predation of prime-aged elk could become the dominant contributor even if there is little change in elk population dynamics. Although, that scenario may also depend on the degree to which wolf predation is additive.

The IPM accounted for the degree to which wolf predation of adult elk was additive when estimating annual, cause-specific hazards. Additive predation removes healthy individuals while compensatory predation removes the “doomed surplus” that

would have died in the absence of predation (Errington 1946). The degree to which predation is additive can vary across predator species (Griffin *et al.* 2011), as well as across prey life stages (Payton *et al.* 2020). The wolf contribution from old elk mortality was about a third of the contribution of wolf predation to prime-aged mortality despite wolf predation being more additive for old elk than for prime-aged elk (Chapter 2). This finding suggests that additive wolf predation of old elk is of little consequence to elk population dynamics, even if there is variation in the degree to which predation is additive across the broad stage class (e.g., more additive for elk closer to age 14 than for elk over 20 years old) or across time (Chapter 2). Wolf predation of prime-aged elk was split approximately halfway between additive and compensatory mortality (Chapter 2). The portion that was additive was substantial enough to contribute to elk population dynamics (approximately 1/5 of the total contribution; Table 4-3).

Potential for multi-predator influence

Harvest of female elk during the study was gradually curtailed between 2000 and 2005, with substantially fewer female elk harvested in 2006 through 2008 and only tribal and youth hunts in later years. I could not test the contribution of harvest separate from other (non-wolf) causes of mortality, but the contribution of other mortality for prime-aged elk was double the contribution of wolf predation for prime-aged elk. The high proportion of other mortality due to harvest in prime-aged elk and the variation in harvest through the study period (Table 4-1) suggests that the contribution of other-caused mortality in prime-aged elk may have been due to relaxed harvest more than other (non-wolf) predator sources of mortality. Prior to wolf reintroduction, harvest accounted for 47% of the observed variation in elk population growth rate (Vucetich *et al.* 2005).

Harvest rate peaked in the years prior to wolf reintroduction and influenced the elk population decline that occurred between 1995 and 2004 (Vucetich *et al.* 2005). Reduced harvest (Table 4-1) likely improved adult survival between 2011 and 2016 and spurred the resulting increase in population growth rate. The LTRE results suggest a potentially strong role of harvest, via other-caused mortality, on elk population dynamics, despite predation by wolves and other species.

Cougar density in northern Yellowstone doubled between pre-wolf (1987-1993) and post-wolf reintroduction (1998-2004) (Ruth *et al.* 2019) and recent (2014-2017) cougar density is similar to the post-wolf reintroduction estimates (Anton 2020). Elk (both male and female) comprised 74% of Yellowstone cougar diet between 1998 and 2004, of which calves comprised 54% and cow elk 37% of known-age kills (Ruth *et al.* 2019). The adult mortality data contained few cases of cougar predation, possibly because elk were primarily captured and radio-collared outside the traditional hunting domain of cougars (rugged, forested terrain) in open, flat habitats where wolves hunt. Alternatively, the few cases were because cougars killed less than five percent of adult female elk annually between 1998 and 2004 (Ruth *et al.* 2019). The results suggest that cougar predation of adult female elk contributed little to elk population change, relative to wolves and harvest.

Cougars increased their predation of elk calves after wolf reintroduction, annually killing 10-60% of the calf population during 1998-2004 (Ruth *et al.* 2019). If this estimate occurred across the study period, it would suggest that Barber-Meyer *et al.* (2008) underestimated cougar predation of calves and that the influence of cougar predation could be somewhat more substantial. In Montana, reduced cougar abundance

led to an increase in elk recruitment and elk population growth rate (Proffitt *et al.* 2020). Cougar predation of neonates is partially compensatory (Griffin *et al.* 2011) and the nutritional condition of calves killed during winter in Yellowstone suggested that 23-30% of winter cougar-killed calves were compensatory predation (Ruth *et al.* 2019). I did not estimate the contribution of cougar predation to elk dynamics because I lacked adequate data to partition calf mortality across years into cause-specific sources. However, cougar predation likely did not exceed the net contribution of wolves and harvest because the contribution of calf survival was less than one-third the contribution of prime-age adult survival.

Grizzly bear density in Yellowstone increased between 2003 and 2012 (Bjornlie *et al.* 2014). There were an estimated 150-278 black bears in the Northern Range from 2017 to 2018 (Bowersock 2020), but there are no estimates of temporal changes in black bear density during the study period. Bear predation was the dominant source of calf mortality during 2003-2005 (Barber-Meyer *et al.* 2008), and a prior study in northern Yellowstone found that calf survival decreased as grizzly bear abundance increased (Proffitt *et al.* 2014). The proportion of calf mortality due to bears may have increased over the study period given the increase in grizzly bear abundance and decreases in wolf abundance and harvest. Any potential bear contribution to elk dynamics is limited to calf predation because bears rarely kill adult elk, instead scavenging on winter-killed, cougar-killed, or wolf-killed carcasses (Ballard *et al.* 2003; Mech & Peterson 2003; Stahler *et al.* 2020). Bear predation likely did not exceed the contribution of wolves and harvest because the contribution of calf survival was less than one-third the contribution of prime-age adult survival.

The presence of more than one elk predator species, and their relative abundance, likely influences the distribution of impact across predator species on elk calf mortality and elk population dynamics. For example, kleptoparasitism of wolf-killed elk carcasses by grizzly bears reduced wolf kill rate of elk (Tallian *et al.* 2017a). Further, cougars kill more calves in the absence of bears and wolves (Griffin *et al.* 2011; Lehman *et al.* 2018) and they lose carcasses to wolves and bears, requiring them to kill more frequently (Ruth *et al.* 2019). These findings suggest that, in the absence or reduced abundance of grizzly bears, wolf predation may increase while cougar predation may decrease. In addition, understanding the extent to which predation is additive across predator species is important for assessing their consumptive effects. Yet it is unknown to what degree compensatory predation may shift to additive predation, or vice versa, with changes in relative predator abundance. The complex competitive interactions that occur between predator species in Yellowstone likely fluctuate with changes in relative predator abundance and thereby influence the relative contribution of each species to changes in elk population dynamics.

Data limitations

The lack of annual calf mortality data may influence how the LTRE contribution is divided among vital rates because true calf mortalities may differ from the IPM estimates had they been informed directly by mortality data rather than a heavy reliance on the other data sets in the IPM. Several studies have demonstrated that ungulate population growth rate is driven by variation in calf mortality (Raithel *et al.* 2007; Marescot *et al.* 2015; Lehman *et al.* 2018). In Montana, increased elk recruitment corresponded to increased population growth rate (Proffitt *et al.* 2020). By using an IPM

framework, I updated the informed prior estimate of calf mortality (2003-2005; Barber-Meyer *et al.* 2008) with data from annual population counts and classification surveys. These estimates of annual calf mortality are not as precise and accurate as they would be if data were directly available for calf mortality on an annual basis. Therefore, the true contribution of calf mortality may differ somewhat from what was estimated.

I could not estimate the total contribution due to wolves because of the lack of annual cause-specific mortality data for calves and yearlings. Therefore, the wolf contribution to adult mortality is likely an underestimate of the total wolf contribution. It is unknown if the observed proportion of calf mortality by predator species during 2003-2005 (Barber-Meyer *et al.* 2008) was consistent throughout the study period or if calf predation by each predator varied through time with changes in predator abundance. The latter seems more plausible. There is also some evidence that the calf mortality reported by Barber-Meyer *et al.* (2008) underestimated calf predation by wolves and cougars because of the limited sample size of radio-collared calves entering the winter season and spatial mismatches between these calves and predators during winter (MacNulty *et al.* 2020). Despite these limitations, this study highlights the important influence of non-wolf sources of mortality, such as hunter harvest, affecting prime-aged adults.

Further, this study found that almost all of the contribution was due to mortality, while fecundity had near zero contribution. The fecundity estimates were imprecise, likely due to a small annual sample size and low power to detect annual changes. It is also important to note that when parameter sample sizes are small, the random effects in the model will ‘shrink’ annual estimates to the mean. Further, small estimates of process variance do not necessarily mean that it is small in nature. A small sample size limits the

model's ability to detect and properly estimate process variance in a parameter. The IPM framework helped improve the fecundity estimates compared to using the single dataset on its own, but because the fecundity estimates had little detectable temporal variation, they had little contribution to variation in population growth rate.

In other ungulate populations, fecundity has more annual variation than does adult survival (Gaillard *et al.* 2000). In retrospective analyses, prime-aged adult ungulate survival generally contributes little to population dynamics because although it has high sensitivity and elasticity, it is usually buffered against environmental variability (Gaillard *et al.* 1998, 2000; Gaillard & Yoccoz 2003). In this study, prime-aged adult survival had more annual variation than fecundity. The fluctuations in prime-aged survival, combined with high sensitivity, led to its dominant contributions to population dynamics. Mortality in prime-aged elk was generally quite low but the population growth rate was highly sensitive to changes in the mortality of this stage class and the modest fluctuations combined with sensitivity was enough to make it the dominant vital rate.

Conclusion

I provide an important step forward in understanding the role of wolf predation on elk population dynamics in northern Yellowstone (MacNulty *et al.* 2020). By estimating the contribution of cause-specific mortality, I quantified the impact of wolf predation across adult elk. Further, I accounted for the extent that wolf predation was an additive cause of mortality. I found that the contribution of non-wolf adult elk mortality exceeded the contribution of wolf-caused adult mortality (Table 4-3). My results are only in agreement with previous findings of the relative importance of harvest versus wolf predation for elk population dynamics (Vucetich *et al.* 2005; Wright *et al.* 2006;

Eberhardt *et al.* 2007) if the relative contritbution of wolves to calf and yearling mortality is somewhat small. However, if all calf and yearling mortalities were due to wolves, the contribution of wolf predation would be slightly greater than the contribution of other causes (0.53 vs 0.48; Table 4-3). The true contribution of wolves is likely less than this extreme because of the large diversity of calf predators (Barber-Meyer *et al.* 2008). I made substantial progress in estimating the impact of wolf predation on elk population dynamics but the lack of annual cause-specific mortality data for calves and yearlings thwarts a complete understanding of the consumptive effect of wolves.

My findings suggest that elimination or limitation of female elk harvest will benefit elk population growth rate. Maintaining the current restrictions on adult female harvest will likely be necessary to support a stable or increasing population in the future. Whereas, increased hunter harvest of female elk has the potential to reverse the population trajectory from an increasing population to a declining population.

The primary insight from this study is that a stage-selective predator species can have a smaller impact on prey population dynamics, compared to other causes of mortality, when prey individuals occupying the "stage refugia" contribute the most to population dynamics. Few studies have assessed the contribution of temporal variation in vital rates and age structure to realized population growth rate (Koons *et al.* 2016, 2017; Maldonado-Chaparro *et al.* 2018; Taylor *et al.* 2018; Fay *et al.* 2019; Layton-Matthews *et al.* 2019; Paquet *et al.* 2019; Nuijten *et al.* 2020) and none have evaluated the contribution of stage-specific predation. However, consideration of temporal variation in predation is important for estimating the consumptive effect of a predator. Decomposing

mortality into cause-specific sources in transient LTREs provides an important advance in our understanding of the contribution of predation to prey population dynamics.

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TABLES AND FIGURES

Table 4-1 Causes of mortality for radio-collared adult female elk (2-14 years old) in northern Yellowstone and adjacent Montana, 2000-2016 (winters 2000/2001 through 2016/2017).

Year	Cougar	Grizzly Bear	Harvest	Malnutrition	Unknown, Non-wolf	Total Non-wolf	Wolf	Total
2000	0	0	5	0	0	5	2	7
2001	0	0	4	0	0	0	2	6
2002	0	0	1	1	0	2	2	4
2003	0	0	2	0	1	3	5	8
2004	0	0	1	0	0	1	0	1
2005	0	0	1	0	0	1	2	3
2006	0	0	0	0	0	0	2	2
2007	0	0	0	0	0	0	2	2
2011	0	0	0	0	0	0	2	2
2012	1	0	0	0	0	0	2	3
2013	0	0	0	0	0	0	2	2
2014	0	0	1	0	0	1	1	2
2015	0	0	2	0	0	2	2	4
2016	0	0	1	0	1	2	2	4
Total	1	0	18	1	2	22	28	50
%	0.02	0.00	0.36	0.02	0.04	0.44	0.56	-

Table 4-2 Causes of mortality for radio-collared adult female elk (>14 years old) in northern Yellowstone and adjacent Montana, 2000-2016 (winters 2000/2001 through 2016/2017).

Year	Cougar	Grizzly Bear	Harvest	Malnutrition	Unknown, Non-wolf	Total Non-wolf	Wolf	Total
2000	1	0	0	0	0	1	2	3
2001	0	0	0	0	0	0	1	1
2002	0	0	0	2	1	3	2	5
2003	0	1	0	0	2	3	2	5
2004	0	0	0	0	1	1	3	4
2005	0	0	0	1	0	1	0	1
2006	0	0	0	0	2	2	0	2
2007	0	0	0	1	0	1	5	6
2011	0	0	0	0	1	1	4	5
2012	0	0	0	0	0	0	3	3
2013	0	0	0	0	1	1	5	6
2014	1	0	0	1	1	3	2	5
2015	1	0	1	0	5	7	3	10
2016	0	0	0	1	0	1	3	4
Total	3	1	1	6	14	25	35	60
%	0.05	0.02	0.02	0.10	0.23	0.42	0.58	-

Table 4-3 The estimated sensitivities of realized population growth rate to change in each modeled vital rate, process variances in the vital rates, and transient life table response experiment (LTRE) contributions (evaluated at median values with 95% Bayesian credible intervals) of the vital rates to variation in realized population growth rates for the northern Yellowstone and adjacent Montana elk population from 2000 to 2016 (winters 2000/2001 through 2016/2017, excluding 2008-2010 because of the gap in survival and mortality data). Also shown are the LTRE contributions on the relative scale (scaled to sum to 1), based on the median contribution.

Vital Rate	Median Sensitivity	Process Coefficient of Variance	Median Contribution	Relative Contribution
Pregnancy: yearling	0.010 (0.010, 0.011)	0.000	0.0000 (0.0000, 0.0000)	0.000
Pregnancy: 2-14 yrs	0.092 (0.086, 0.10)	0.000	0.0000 (-0.0001, 0.0002)	0.003
Pregnancy: >14 yrs	0.012 (0.010, 0.015)	0.000	0.0000 (0.0000, 0.0001)	0.001
Mortality: Calf	0.357 (0.335, 0.378)	0.004	0.0014 (0.0010, 0.0019)	0.186
Mortality: yearling	0.087 (0.084, 0.089)	0.004	0.0005 (0.0004, 0.0006)	0.068
Wolf Mort: 2-14 yrs	-0.717 (-0.73, -0.702)	0.001	0.0014 (0.0004, 0.0038)	0.207
Other Mort: 2-14 yrs	-0.717 (-0.73, -0.702)	0.003	0.0031 (0.0008, 0.0045)	0.423
Wolf Mort: >14 yrs	-0.108 (-0.124, -0.095)	0.009	0.0005 (0.0000, 0.0012)	0.073
Other Mort: >14 yrs	-0.108 (-0.124, -0.095)	0.005	0.0004 (0.0000, 0.0009)	0.054
Abundance of yearlings	0.00000 (0.00000, 0.00000)	0.001	0.0001 (0.0001, 0.0001)	0.012
Abundance of 2-14 year-olds	0.00004 (0.00003, 0.00004)	0.001	-0.0003 (-0.0004, -0.0003)	-0.045
Abundance of >14 year-olds	-0.00010 (-0.00014, -0.00007)	0.001	0.0001 (0.0000, 0.0003)	0.019

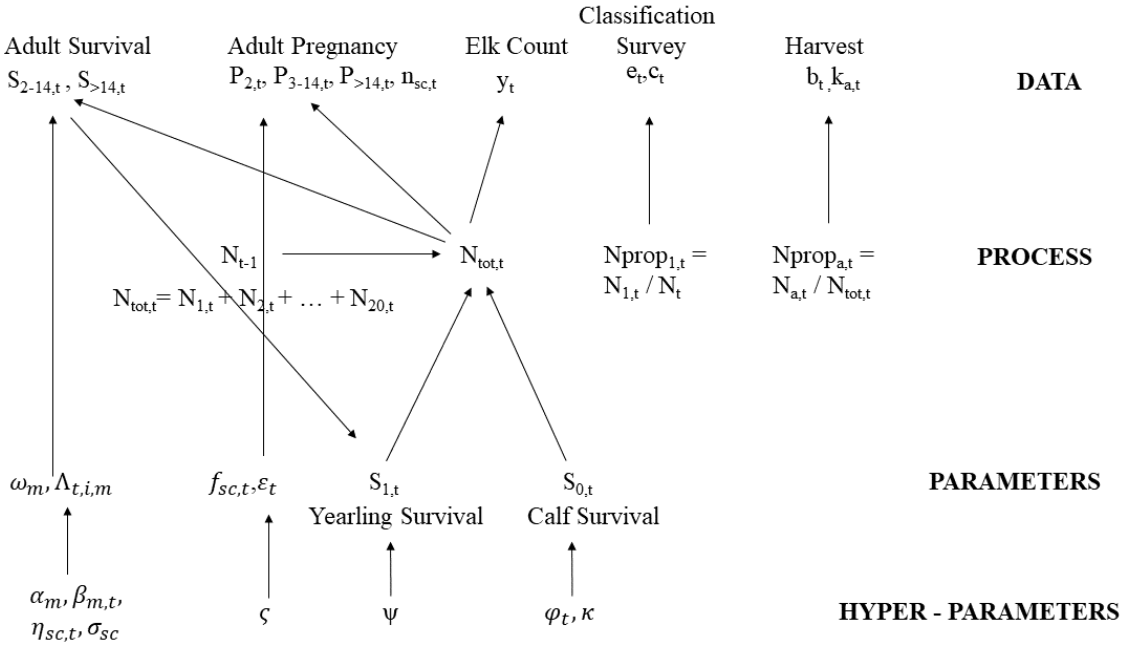
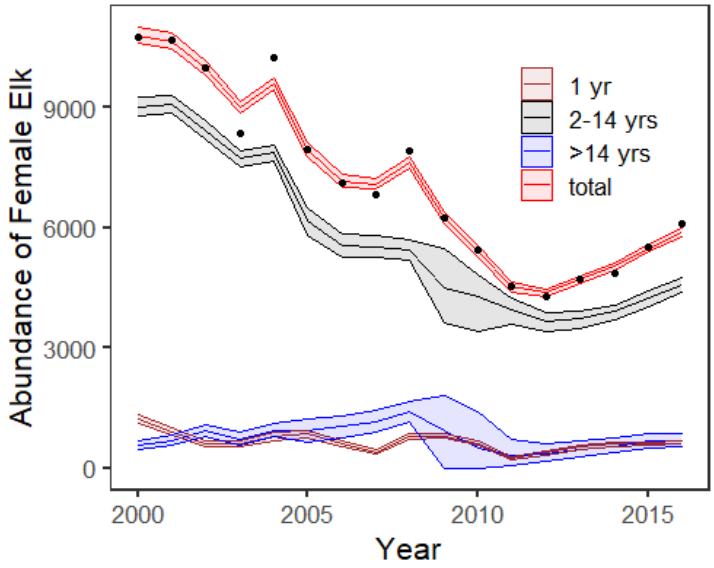


Figure 4-1 Directed acyclic diagram showing relationships in an integrated population model for elk in northern Yellowstone and adjacent Montana. See text for definitions and additional details.

A



B

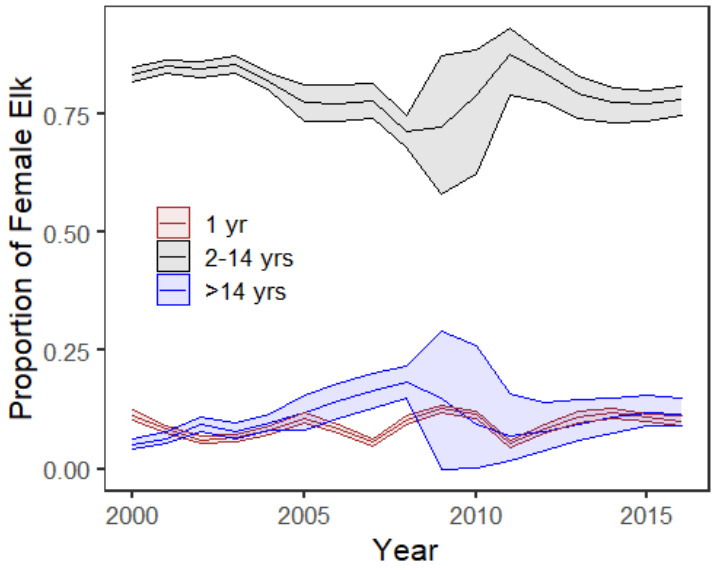


Figure 4-2 Estimates of female elk abundance by stage class in northern Yellowstone and adjacent Montana between 2000 and 2016 (winters 2000/2001 through 2016/2017) based on the integrated population model (A; center lines with 95% Bayesian credible intervals denoted by the colored shading and upper and lower lines). Calf abundance was not estimated in the model. Annual count data of the female elk population are shown as black circles. Proportion of stage classes based on the integrated population model (B).

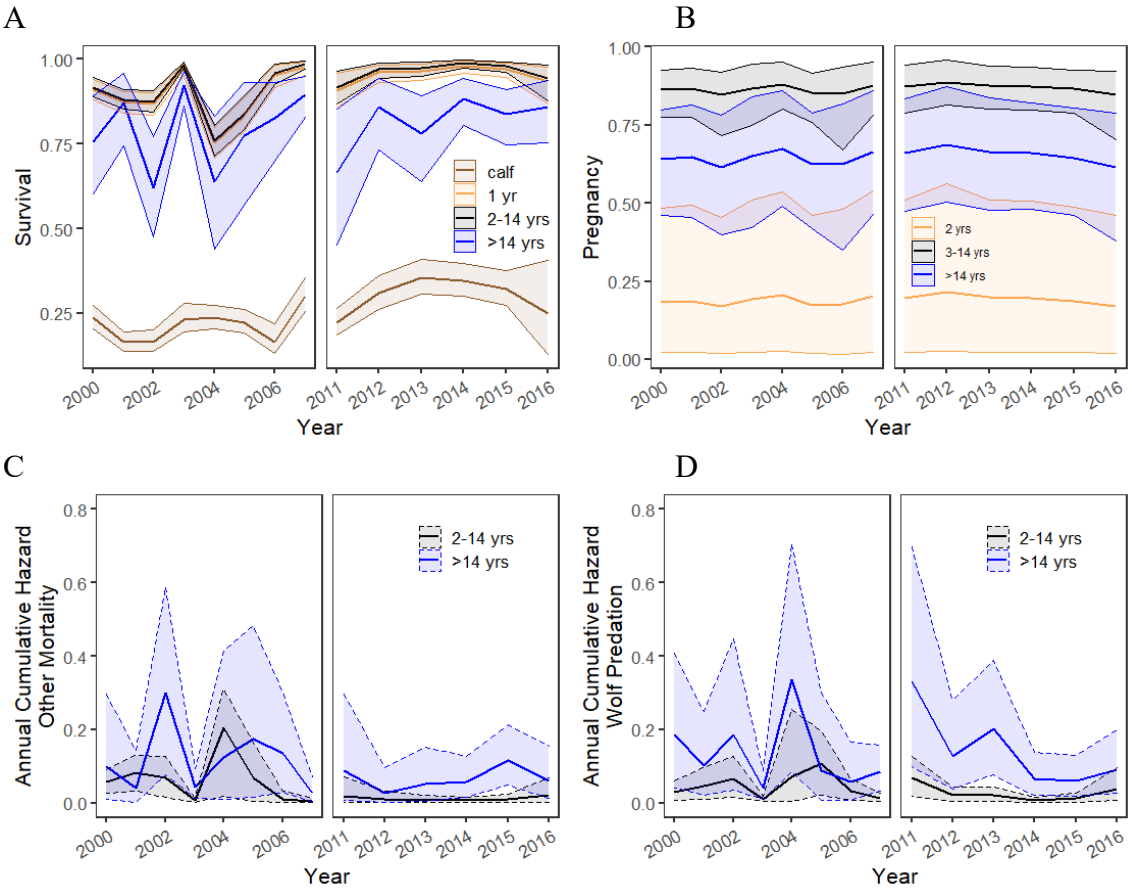
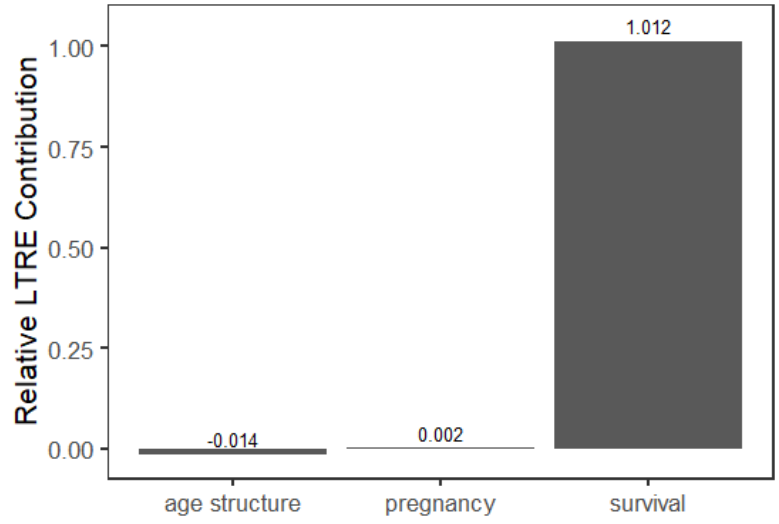


Figure 4-3 Estimates of annual female elk survival (A), pregnancy (B), cumulative hazard of other mortality (C), and cumulative hazard of wolf predation (D) by stage class in northern Yellowstone and adjacent Montana between 2000 and 2016 (winters 2000/2001 through 2016/2017, excluding 2008-2010 because of the gap in survival and mortality data) based on the integrated population model (center lines with 95% Bayesian credible intervals denoted by the colored shading and upper and lower lines).

A



B

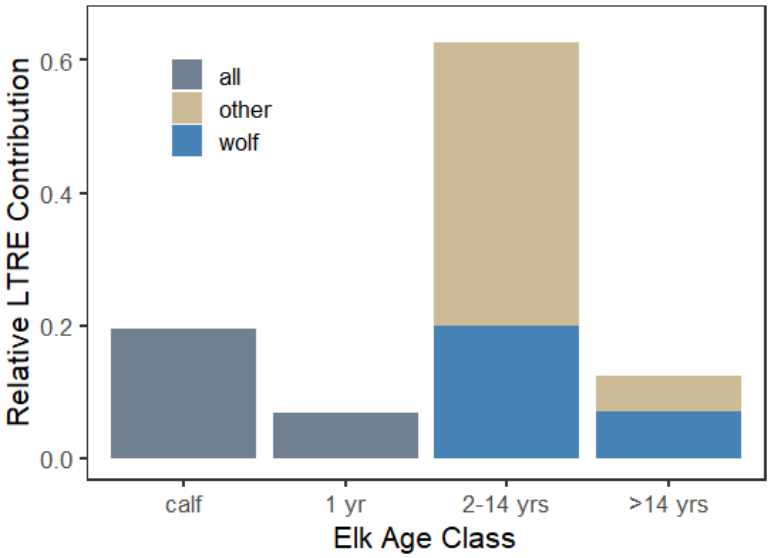


Figure 4-4 The transient life table response experiment relative contributions of age structure, pregnancy, and survival across all stage classes to temporal variation in realized population growth rates for female elk in northern Yellowstone and adjacent Montana between 2000 and 2016 (winters 2000/2001 through 2016/2017, excluding 2008-2010 because of the gap in survival and mortality data; A). The relative contribution of mortality by stage class and cause, Table 2 (B).

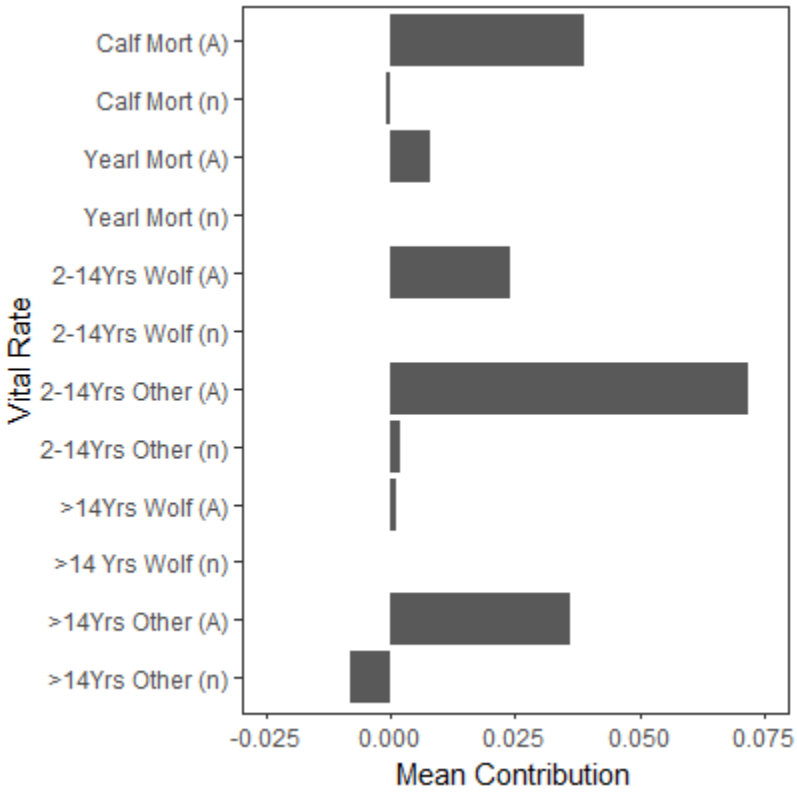


Figure 4-5 The direct contribution of cause-specific mortality by stage class (denoted (A)) and indirect effects of the vital rates via changes in the proportionate abundance of the stage class (denoted (n)) to change in the geometric mean rate of population growth ($\Delta \log \lambda_g$) of elk in northern Yellowstone and adjacent Montana between 2000-2005 and 2011-2016 ($\Delta\lambda$ 0.114; 90% CI: 0.106, 0.121).

CHAPTER 5

CONCLUSIONS

Apex predators can have a strong top-down impact on their prey populations (Ripple *et al.* 2014). However, the consumptive effect of a predator depends on prey life history and the degree to which predation adds to or replaces other sources of mortality. The preceding chapters attempted to address the impact of wolf predation on adult female elk survival and population dynamics in northern Yellowstone National Park and adjacent Montana. In chapter two, I addressed how the age-specific survival of adult female elk varied by cause-specific mortality and the degree to which wolf predation was additive across younger and older stage classes of adult elk. In chapter three, I demonstrated how the probability that an adult female elk was killed by a wolf varied by environmental conditions, leading to a shorter life expectancy and earlier onset of actuarial senescence during harsh (e.g., dry, snowy) conditions. In chapter four, I used a population modeling approach to estimate the contribution of wolf predation, relative to other causes of mortality, to the temporal variance in female elk population growth rate over a 17-year period after wolves were reestablished in Yellowstone National Park.

I made two advances regarding the effect of predation by a selective predator in chapter two. First, I demonstrated that adult, female elk survival was high during their pre-teenaged years and that the probability of being killed by a wolf was substantially greater than dying from other (non-harvest) causes once elk reached their teens and into their twenties. I used this distinction between elk that were susceptible (>14 years old) to wolf predation and those that were resistant (2-14 years old) to determine if the degree to

which predation was additive varied by susceptibility. This distinction is important because prior studies have assumed equal susceptibility to predation across stage classes when estimating additive predation of adults (e.g., Brodie *et al.* 2013). I showed that wolf predation was more compensatory for elk resistant to wolf predation than for elk that were susceptible. This finding counters the argument that predation of old prey should be compensatory because at old ages individuals are likely to die from a variety of causes. While that concept may still hold true for the oldest elk, many teenaged elk were not yet weak enough to have been killed by natural, non-wolf causes. However, younger elk are harder to kill (MacNulty *et al.* 2007, 2012; Mech *et al.* 2015) and those killed by wolves may have suffered from conditions that increased their susceptibility to mortality in general.

The second key advance related to how the relative frequency of prey susceptible to predation varied through time, which led to changes in the degree to which wolf predation was additive across the elk population each year. This finding is important because it shows how the assumption of a stable stage distribution, and thus a constant proportion of susceptible prey, can bias estimation of the consumptive effect of a predator when additive predation varies by stage class. Further, the frequency of prey for which predation is additive can moderate the consumptive effect of predation when those individuals are rare.

Together, these results clarify the influence of wolf predation on adult female elk survival in northern Yellowstone and adjacent Montana. The degree to which wolf predation was additive across the elk population likely increased through the early 2000's as the population aged (Hoy *et al.* 2020). More broadly, these results demonstrate the

importance of distinguishing between individuals that are susceptible and resistant to a predator when studying predator-prey interactions. Further, accounting for the temporal frequency of susceptible prey when evaluating predation is often overlooked yet critical because their frequency may shift through time and space depending on life history, changes to a harvest regime, predator populations, or other environmental pressures, or due to a lag effect from prior harvest and predation pressure.

In chapter three, I extended my analysis of elk survival and cause-specific mortality to consider the effects of environmental conditions (e.g., weather, predator abundance) on the age-specific mortality of adult female elk. This analysis was unique in that it was the first to examine the combined role of predation and abiotic environmental conditions in shaping both the onset and shape (i.e., how steeply survival changes with age) of actuarial senescence and cause-specific mortality patterns. I demonstrated that both the age at onset of senescence and life expectancy of female elk declined with increasing snowpack, long-term drought, and wolf abundance. Survival also declined under these conditions, but the decline was substantially sharper for teenaged and older elk, the stage class primarily targeted by wolves (Wright *et al.* 2006; Metz *et al.* 2012, Hoy *et al.* 2021). I showed that survival of elk before their teenage years was relatively high despite increasing wolf abundance. Only when wolf abundance was at its maximum (N=142) did survival of 6-10 year-old elk decrease more rapidly compared to lower levels of wolf abundance. My results highlight the importance of evaluating prey ages, predator abundance, and other environmental conditions to understand prey survival.

I further demonstrated that wolf predation was the primary driver of actuarial senescence in female elk. In general, adult female elk were more susceptible to wolf

predation at younger ages under harsh conditions (e.g., heavy snowpack and high wolf abundance) than they were under more moderate conditions. This finding is important because it suggests that the degree to which predation is additive can increase not only with an older age structure, but also with environmental conditions that increase age-specific susceptibility to predation. These results also suggest that severe conditions reduce the age-threshold at which individuals become susceptible to predation, thus increasing the proportion of the population exposed to additive predation. Young adult elk (e.g., < 9 years old) maintained high survival, despite harsh conditions, and therefore, persisted as a demographic stage refuge (Miller & Rudolf 2011) that may increase population resilience to environmental challenges.

In chapter four, I evaluated the contribution of wolf-caused mortality of adult female elk to changes in elk abundance relative to other causes of adult female mortality. I included harvest mortality in the other causes of mortality to evaluate the impact of all elk mortality. This study combined my cause-specific mortality analysis of adult female elk (chapter 2) with available datasets on female elk abundance, population age structure, and pregnancy to provide the first comprehensive population model for northern Yellowstone elk. I demonstrated that the influence of wolf-caused mortality on adult female elk population dynamics was less than the influence of non-wolf sources of adult female mortality. Mortality of adult female elk (2-14 years old) was the primary driver of change in the population growth rate. I demonstrated that non-wolf causes of adult female mortality (including harvest, other predators, and malnutrition) combined had a greater contribution than wolf predation of adult female elk to temporal variance in realized population growth rate. Harvest of prime-aged elk was likely the primary mortality cause

within non-wolf mortality driving this finding because harvest decreased substantially from 2000-2016 and constituted most of the non-wolf mortality of prime-aged, radio-collared elk. The lack of annual, cause-specific mortality data on calves and yearlings yielded an incomplete estimate of the contribution of wolf predation to elk population dynamics. The total wolf contribution would exceed the contribution of other causes of mortality if the contributions of calf and yearling mortality were entirely due to wolves. However, it is likely that the wolf contribution is less than the contribution of other causes of mortality because of the numerous other predator species that also kill calves (e.g., bears and cougars).

Chapter four highlights the importance of accounting for variation in susceptibility to predation across prey stage classes because the impact of a predator on prey population dynamics depends on the relative importance of the prey stage class they consume. My study demonstrated how the impact of predation across stages of prey can be estimated by considering cause-specific mortality. These results are important because they demonstrate how predation influenced prey population dynamics relative to other causes of mortality while accounting for the degree to which predation was additive. The results can also help guide management practices (e.g., limiting female harvest) or be used to assess if prior management actions were effective in achieving their goals.

IMPLICATIONS FOR YELLOWSTONE

The overall goal of this dissertation was to estimate the impact of wolf predation on female elk survival and population dynamics in northern Yellowstone National Park and adjacent Montana. Yellowstone provides a unique opportunity to assess the impact of a predator on a prey population in greater detail than is usually achieved for free-ranging

terrestrial vertebrates because of the substantial high-quality, long-term data available for these species. Analyses of elk population counts a decade after wolf reintroduction suggested that wolf predation was of little consequence to the elk population, and harvest and drought were primarily responsible for the population decline (Vucetich *et al.* 2005; Eberhardt *et al.* 2007). A decrease in other-caused mortality of prime-aged elk between the early and late portion of the study was the most important driver of a switch from a declining population growth rate in 2000-2005 to an increasing population growth rate in 2011-2016. Harvest of prime-aged elk was likely the primary mortality cause within non-wolf mortality driving these findings because harvest decreased substantially from 2000-2016 and constituted 82% of non-wolf mortality of prime-aged, radio-collared elk. However, wolf predation was potentially an important contributor to changes in elk population dynamics, with a relative contribution between 0.28 and 0.53, depending on how much of the calf and yearling contribution was due to wolves.

In Yellowstone, wolves are one of several predator species (e.g., humans, bears, cougars) that kill elk (Barber-Meyer *et al.* 2008; Ruth *et al.* 2019). Most prey species also have more than one predator and predators may partition shared prey across prey life history stages (Miller & Rudolf 2011). It is important to consider how the consumptive effect of predation is divided among predator species that may kill different prey stages to understand the impact of predation in a multi-predator system on prey population dynamics. However, I lacked sufficient mortalities of adult elk by harvest and cougars to separate these predators from non-predation causes of mortality (i.e., malnutrition, winter kill). Further, the lack of research on black bears and coyotes, studies of limited temporal

duration on cougars, and limited estimates of cause-specific mortality of calves has hindered an informed, multi-predator perspective on elk population dynamics.

A better understanding of predator-specific mortality across stage classes could potentially be more important during periods without harvest. For example, the relative contribution of adult survival to population dynamics may decrease if prime-aged adult survival becomes stationary through time with high survival in the absence of harvest. In such a scenario, the contribution of calf survival may increase and the contribution of bear predation to elk population dynamics could potentially increase relative to that of wolf predation if bear predation of calves is substantially higher than that of wolves. The diversity of calf predators in Yellowstone will continue to impact the elk population, even if mortality of calves remains a relatively smaller contributor to population dynamics. Wildlife managers will need to continue to monitor adult cause-specific mortality as well as start monitoring calf and yearling cause-specific mortality if they want to understand which stage class and predator has the most influence on population dynamics over any period of time or to assess the impact of a harvest regime.

Harvest of large numbers of prime-aged elk in the late 1990s combined with calf predation—due to increasing populations of calf predators—likely reduced elk recruitment (MacNulty *et al.* 2020) and influenced population age structure (Hoy *et al.* 2020). Any future harvest plan should account for declines in elk abundance that shift the age structure to include more older, less reproductive individuals. For example, reduced survival of prime-aged elk (e.g., increased harvest) or reduced recruitment (e.g., increased calf predation that might occur because of increased predator abundance) would likely lead to an older population age structure in the future. Limited harvest of

adult female elk in the future would likely be necessary to promote elk population growth because most harvested elk are likely to be prime-aged as they comprise the majority of the population and are important for population growth.

IMPLICATIONS FOR PREDATOR-PREY INTERACTIONS

I demonstrated the importance of incorporating individual heterogeneity in susceptibility to predation in studies of predator-prey interactions. The inclusion of individual susceptibility is important because 1) susceptibility to predation can vary within a broad prey stage class (adults); 2) the degree to which predation is additive varies between individuals that are susceptible and individuals that are resistant to predation; 3) how the prey population is structured by susceptible individuals is temporally dynamic and therefore, the level of additive predation across the population is also temporally dynamic; 4) individual susceptibility to predation can change under different environmental conditions; 5) individual susceptibility to predation at late ages allows a predator to drive prey actuarial senescence; and 6) the impact of a predator on prey population dynamics depends on how predation contributes to changes in prey population growth rate across each stage class of prey, relative to other sources of mortality.

I identified variation in additive predation by level of prey susceptibility to predation across the adult stage class, which, to my knowledge, previously had not been done in other predator-prey studies. This finding is important because it suggests that studies that do not distinguish prey by susceptibility may over- or under-estimate the level of additive predation. In addition, accounting for the frequency of susceptible prey is important because their frequency may shift through time and space depending on life

history, changes to a harvest regime and predator populations, or prior harvest and predation pressure, thereby causing changes to the level of additive predation through time. This distinction of the level of additive predation by prey susceptibility and their frequency is an important contribution to the scientific literature because it helps explain why the consumptive effect of a predator may be limited and temporally dynamic.

Further, I found that a subset of the prey population may retain high survival and avoid predation despite temporal variation in predator abundance and abiotic environmental conditions, thus serving as a demographic stage refuge for the population (Miller & Rudolf 2011). However, individuals older than prime-age had increased mortality and predation under higher predator abundance and harsh abiotic conditions, suggesting that predation in tandem with harsh conditions can drive actuarial senescence of prey. Environmental conditions can therefore shift the proportion of susceptible prey in a population, in this case by lowering the age at which individuals are susceptible. Such a shift has the potential to alter the impact of predation on the prey population by allowing predators to consume individuals that might be more important to prey population growth rate. For example, if wolves hypothetically killed more prime-aged elk during years of heavy snow compared to years of mild snow, and population growth rate declined during the snowy years and increased during the mild years, then there may be a large contribution from wolf predation of prime-aged elk driving the change in elk population growth rate between the two periods. These findings are important because they contribute to the growing knowledge of actuarial senescence in mammals (Nussey *et al.* 2013; Gaillard *et al.* 2017) and advance our limited knowledge of the impact of

predators and abiotic environmental conditions on mammal age-specific survival and cause-specific mortality.

Finally, few studies have assessed the contribution of temporal variation in vital rates and age structure to realized population growth rate (Koons *et al.* 2016, 2017; Maldonado-Chaparro *et al.* 2018; Taylor *et al.* 2018; Fay *et al.* 2019; Layton-Matthews *et al.* 2019; Paquet *et al.* 2019; Nuijten *et al.* 2020) and none, to my knowledge, have evaluated the contribution of stage-specific predation. Thus, I provide the first estimate of the contribution of predation across adult stages of prey. My results demonstrate how the contribution of predation can be estimated across prey stage classes while also accounting for the temporal variation in vital rates and population age structure that occurred over the time period of interest. Low variation in high survival of prime-aged individuals is expected to buffer populations against harsh environmental conditions (Gaillard & Yoccoz 2003; Pardo *et al.* 2013), but I showed how temporal variation in prime-aged survival was the greatest contributor to population dynamics and it was dominated by non-wolf causes of mortality. Wolf predation, when focused on juveniles and old prey, may therefore provide a smaller contribution to prey population dynamics than non-wolf causes of mortality. Thus, individual susceptibility to predation has the potential to limit the consumptive effect of a predator.

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Paquet, M., Arlt, D., Knape, J., Low, M., Forslund, P. & Pärt, T. (2019). Quantifying the links between land use and population growth rate in a declining farmland bird. *Ecol. Evol.*, 9, 868–879.

Pardo, D., Barbraud, C. & Weimerskirch, H. (2013). What shall I do now ? State-dependent variations of life-history traits with aging in Wandering Albatrosses, 474–487.

Ripple, W.J., Estes, J.A., Beschta, R.L., Wilmers, C.C., Ritchie, E.G., Hebblewhite, M., *et al.* (2014). Status and ecological effects of the world's largest carnivores. *Science*, 80, 343

Ruth, T.K., Buotte, P.C. & Hornocker, M.G. (2019). *Yellowstone cougars: ecology before and during wolf reestablishment*. University Press, Boulder, Colorado.

Taylor, L.U., Woodworth, B.K., Sandercock, B.K. & Wheelwright, N.T. (2018). Demographic drivers of collapse in an island population of Tree Swallows. *Condor*, 120, 828–841.

Vucetich, J.A., Smith, D.W. & Stahler, D.R. (2005). Influence of harvest, climate and wolf predation on Yellowstone elk, 1961-2004. *Oikos*, 2, 259–270.

Wright, G.J., Peterson, R.O., Smith, D.W. & Lemke, T.O. (2006). Selection of northern

Yellowstone elk by gray wolves and hunters. *J. Wildl. Manage.*, 70, 1070–1078.

CURRICULUM VITAE

Lacy M. Smith
(December 2021)

Education

2021	Ph.D. in Ecology – Defended 14 December 2020 Advisors: Daniel MacNulty & David Koons	Utah State University
2011	A.S. in Geographic Information Systems	American River College
2008	B.S. in Wildlife, Fish, & Conservation Biology Minor in Global and International Studies	University of CA, Davis

Professional Experience

2013 – 2015 *Biologist and Project Manager*
San Francisco Bay Estuary Field Station, Western Ecological Research Center, U.S. Geological Survey, Vallejo, CA

2008 – 2013 *Biological Science Technician*
San Francisco Bay Estuary Field Station, Western Ecological Research Center, U.S. Geological Survey, Vallejo, CA

Peer-Reviewed Publications

Journal Articles

2021 Hill, N.J., **L.M. Smith**, S.B. Muzaffar, J.L. Nagel, D.J. Prosser, J.D. Sullivan, K.A. Spragens, C.A. DeMattos, C.C. DeMattos, L. El Sayed, K. Erciyas-Yavuz, C.T. Davis, J. Jones, Z. Kis, R.O. Donis, S.H. Newman, and J.Y. Takekawa. Crossroads of highly pathogenic H5N1: overlap between wild and domestic birds in the Black Sea-Mediterranean impacts global transmission. Virus Evolution 7, veaa093.

2018 Zheng, R., **L.M. Smith**, D.J. Prosser, J.Y. Takekawa, S.H. Newman, J.D. Sullivan, L. Ze, and B. Yan. Investigating home range, movement pattern, and habitat selection of bar-headed geese during breeding season at Qinghai Lake, China. Animals 8, 182.

2016 Choi, C., J.Y. Takekawa, D.J. Prosser, **L.M. Smith**, C.R. Ely, A.D. Fox, L. Cao, X. Wang, N. Batbayar, T. Natsagdorj, and X. Xiao. Chewing lice (Phthiraptera) of swan geese (*Anser cygnoides*): new host-parasite associations. Korean Journal of Parasitology 54: 685-691.

2016 Choi, C., K. Lee, N.D. Poyarkov, J.Y. Park, H. Lee, J.Y. Takekawa, **L.M. Smith**, C.R. Ely, X. Wang, L. Cao, A.D. Fox, O. Goroshko, N. Batbayar, D.J. Prosser, and X. Xiao. Low survival rates of swan geese (*Anser cygnoides*) estimated from neck-collar resighting and telemetry. Waterbirds 39: 277-286.

2014 Brand, L.A., J.Y. Takekawa, J. Shinn, T. Graham, K. Buffington, K.B. Gustafson, **L.M. Smith**, S. Spring, and K. Miles. Effects of wetland management on carrying capacity of diving ducks and shorebirds in a coastal estuary. Waterbirds 37: 52-67.

2013 Schwartz, M.W., **L.M. Smith**, and Z.L. Steel. Conservation investment for rare plants in urban environments. PLoS ONE 8: e83809.

2012 Brand, L.A., **L.M. Smith**, J.Y. Takekawa, N.D. Athearn, K. Taylor, D. Schoellhamer, G. Shellenbarger, and R. Spenst. Trajectory of early tidal marsh restoration: elevation, sedimentation and colonization of breached salt ponds in the northern San Francisco Bay. Ecological Engineering 42: 19-29.

2012 Muzaffar, S.B., N.J. Hill, J.Y. Takekawa, W.M. Perry, **L.M. Smith**, and W.M. Boyce. Role of bird movements in the epidemiology of West Nile and avian influenza virus. Human-Wildlife Interactions 6: 65-81.

Book Chapter

2020 MacNulty, D.M., D.R. Stahler, T. Wyman, J. Ruprecht, **L.M. Smith**, M.T. Kohl, and D.W. Smith. Population dynamics of northern Yellowstone elk after wolf reintroduction. *In* Yellowstone wolves: science and discovery in the world's first national park. Eds. D. W. Smith, D. R. Stahler, and D. R. MacNulty. *University of Chicago Press*, pp. 184-204.

Oral Conference Presentations

2017 **L.M. Smith**, D.N. Koons, D.W. Smith, D. Stahler, P.J. White, D.R. MacNulty. Survival expectations in a wolf-elk system: How selective predation and the environment shift elk senescence. Ecological Society of America – 102nd Annual meeting, Portland, OR.

2017 **L.M. Smith**, D.N. Koons, D.W. Smith, D. Stahler, P.J. White, D.R. MacNulty. Survival expectations in a wolf-elk system: How selective predation and the environment shift elk senescence. 12th International Mammalogical Congress, Perth, Australia.

2015 **Smith, L.M.**, S.E.W. De La Cruz, S. M. Moskal, J. Yee, C. Strong, J. Krause, N. Washburn, and J.Y. Takekawa. Managing pond habitat for waterbirds: evaluating waterbird habitat use in the largest tidal marsh restoration project on the west coast of the U.S. The Wildlife Society Annual Conference, Winnipeg, Manitoba, Canada.

2015 **Smith, L.M.**, S.M. Moskal, J.Y. Takekawa, S.E.W. De La Cruz, J. Krause, and R. Spenst. Shorebird response to varying salinity and water depth in an experimental design in salt pond management. Bay Area Conservation Symposium, University of California, Berkeley, CA.

2015 **Smith, L.M.**, S.M. Moskal, J.Y. Takekawa, S.E.W. De La Cruz, J. Krause, and R. Spenst. Shorebird response to varying salinity and water depth in an experimental design in salt pond management. The Wildlife Society Western Section Annual Conference, Santa Rosa, CA.

2014 **Smith, L.M.**, S.M. Moskal, J.Y. Takekawa, S.E.W. De La Cruz, J. Krause, and R. Spenst. Shorebird response to varying salinity and water depth in an experimental design in salt pond management. Bay-Delta Science Conference, Sacramento, CA.

2013 **Smith, L.M.**, J.Y. Takekawa, S. Moskal, T.R. Graham, E. Mruz, C. Strong, and K. Taylor. Transitional habitat: the value of breached former salt ponds for migratory waterbirds. The Wildlife Society Western Section Annual Conference, Sacramento, CA.

2012 **Smith, L.M.**, L.A. Brand, J.Y. Takekawa, N.D. Athearn, K. Taylor, G.G. Shellenbarger, D.H. Schoellhamer, and R. Spenst. Elevation, sedimentation, and colonization of breached salt ponds in the northern San Francisco Bay. Bay-Delta Science Conference, Sacramento, CA.

2012 **Smith, L.M.** and J.Y. Takekawa. Status and distribution of Rallidae in coastal estuaries from tidal marshes to mangrove forests. Coastal and Estuarine Research Federation, Mar del Plata, Argentina.

Poster Presentations

2015 **Smith, L.M.**, S.E.W. De La Cruz, T.R. Graham, S.M. Moskal, C. Strong, and J. Krause. Shorebird response to experimental variations in salinity and depth of managed ponds. 12th Biennial State of the San Francisco Estuary Conference, Oakland, CA.

2010 **Smith**, **L.M.,** L.A. Brand, J.Y. Takekawa, and N.D. Athearn. Bathymetric surveys of former salt ponds 3, 4, and 5 of the Napa-Sonoma Marshes, Northern San Francisco Bay, CA. Poster Presentation. Bay-Delta Science Conference, Sacramento, CA.

2009 Athearn, N.D., J.M. Shinn, **L.M. Smith**, J.Y. Takekawa, K.D. Henderson, G.G. Shellenbarger, and C.M. Strong. Factors affecting bird abundance and habitat quality at managed salt ponds in San Francisco Bay. Poster Presentation. The Wildlife Society 16th Annual Conference, Monterey, CA.

Funding

2020 Utah State University, Ecology Center, Finishing-Up Funds, $2000.

2017 National Science Foundation, Graduate Research Fellowship Program, $138,000.

Utah State University, Dept. of Wildland Resources, Student Travel Grant, $400.

Utah State University, Graduate Studies, Student Travel Grant, $400.

2014 Alameda County Resource Conservation District, An applied study of the Eden Landing Pond E12 and E13 project habitat enhancement. L.M. Smith, S.E.W. De La Cruz, and J.Y. Takekawa. $166,578.

2014 U.S. Fish and Wildlife Service, Refuges Inventory and Monitoring Program. Science support for salt pond restoration and management FY15: waterbird monitoring analyses. S.E.W. De La Cruz, L.M. Smith, and J. Yee. $79,560.

2014 U.S. Army Corps of Engineers. Post-construction monitoring of the Napa-Sonoma Marshes wetland restoration project ponds 6-8, 2018-2023. S.E.W. De La Cruz, I. Woo, and L.M. Smith. $1,075,000.

2013 U.S. Army Corps of Engineers. Post-construction monitoring of the Napa-Sonoma Marshes wetland restoration project ponds 6-8, 2016-2017. J.Y. Takekawa, I. Woo, and L.M. Smith. $456,378.

2013 U.S. Army Corps of Engineers. Construction monitoring of the Napa-Sonoma Marshes wetland restoration project ponds 6-8. J.Y. Takekawa, I. Woo, and L.M. Smith. $683,025.

Teaching Experience

Lecturer

2018 Plant & Animal Populations (WILD 3810) Utah State University

Instructor of record for upper-division undergraduate course on population ecology. Responsibilities included two 50-minute lectures and one two-hour computer laboratory per week, writing and grading quizzes and exams, and office hours. I mentored one Teaching Assistant. Class size of 46 students.