



Research Article

Mortality Risks and Limits to Population Growth of Fishers

RICK A. SWEITZER,¹ *The Great Basin Institute, 16750 Mt. Rose Highway, Reno, NV 89511, USA*

VIORREL D. POPESCU, *Earth to Ocean Research Group, Simon Fraser University, 8888 University Drive, Burnaby, BC V5A 1S6, Canada; and University of Bucharest, Centre for Environmental Research (CCMESI), 1 N. Balcescu Blvd., Bucharest, Romania*

CRAIG M. THOMPSON, *USDA Forest Service, Pacific Southwest Research Station, 2081 E. Sierra Avenue, Fresno, CA 93710, USA*

KATHRYN L. PURCELL, *USDA Forest Service, Pacific Southwest Research Station, 2081 E. Sierra Avenue, Fresno, CA 93710, USA*

REGINALD H. BARRETT, *Department of Environmental Science, Policy, and Management, University of California, Berkeley, CA 94720, USA*

GRETA M. WENGERT, *Integral Ecology Research Center, Blue Lake, CA 95525, USA*

MOURAD W. GABRIEL, *Integral Ecology Research Center, Blue Lake, CA 95525, USA*

LESLIE W. WOODS, *California Animal Health and Food Safety Laboratory System, Davis, CA 95616, USA*

ABSTRACT Fishers (*Pekania pennanti*) in the west coast states of Washington, Oregon, and California, USA have not recovered from population declines and the United States Fish and Wildlife Service has proposed options for listing them as threatened. Our objectives were to evaluate differences in survival and mortality risk from natural (e.g., predation, disease, injuries, starvation) and human-linked causes (e.g., rodenticide exposure [toxicants], vehicle strikes). We monitored survival of 232 radio-collared fishers at a northern and southern study site in the Sierra National Forest, California. We retrieved fisher carcasses, and used necropsy examinations to determine causes of death. We estimated survival and mortality rates using the Kaplan–Meier estimator and nonparametric cumulative incidence functions, respectively, and integrated risk-specific survival rates into a Leslie matrix to evaluate how population growth (λ) might improve if management can reduce mortality from any of the risks. We determined cause of death for 93 of 121 fishers, and annual survival was 0.69 for all fishers, and 0.72 for female fishers. Mortality rates were 19.5% for predation; 2.5% for disease, injury, and starvation; and 1.9% for toxicant exposure and vehicle strikes. Predation rates were similar between sexes, and relative risk was 51% lower in fall and winter compared to spring and summer. Combined mortality from disease, injuries, starvation, vehicle strikes, and toxicants was 4.4%, and 11 times higher for males than females. We estimated a base λ at the northern site of 0.96, which had the potential to increase to 1.03 or 1.11 if predation were reduced by 25% or 50%, respectively, whereas λ could increase to 0.97 in the absence of deaths from all other risks except predation. Predation was the dominant limiting factor in our study population, and was also the most common mortality risk for fishers in the 3 West Coast states (67%), followed by disease, injury, and starvation (12%) and vehicle strikes (8%). Direct mortality from toxicants appeared limited to fishers in California. Our results identified the need for continued and potential expanded restrictions on habitat disturbance in fisher denning habitats. Research is needed on contact rates between larger predators and fishers, including whether fishers are more vulnerable to attack in open forests or along roads used by coursing predators. © 2015 The Wildlife Society.

KEY WORDS California, cause-specific, disease, fisher, Kaplan–Meier, lambda, mortality, *Pekania pennanti*, predation, roadkill, rodenticides, Sierra Nevada, vehicle strike.

The reasons animals die are of interest because mortality shapes life histories (Gaillard et al. 1998) and because information on cause-specific mortality can identify factors that limit growth of wildlife populations (Larsen et al. 1989, Clapham et al. 1999, Festa-Bianchet et al. 2006). Natural mortality from predation, injuries, starvation, or disease will

occur in all wildlife populations but may increase because of imbalances in abundance of interacting species from harvest, habitat change associated with resource extraction, and the introduction of non-native species or pathogens (Goodrich and Buskirk 1995, Roemer et al. 2001, Pedersen et al. 2007, Clifford et al. 2009, Ritchie and Johnson 2009). Data on baseline levels of natural mortality are seldom available (Wengert et al. 2014), yet additional mortality within a population from human-linked causes such as roadkill or exposure to rodenticides may alter the growth trajectory of a population, thereby threatening persistence whether or not

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¹E-mail: rsweitzerrick@gmail.com

the number of deaths from natural sources has changed over time (Sweitzer et al. 1997, Hayes et al. 2000). For these reasons, quantitative data on the relative importance of natural and human-linked mortality risks can inform mitigation efforts to prevent local and regional extinction (Newmark 1995, Clevenger et al. 2001, Ceballos and Ehrlich 2002).

The fisher (*Pekania pennanti*) is a forest carnivore with a historical distribution in North America spanning the boreal forest zone of Canada, the central and northern Rocky Mountains, the Great Lakes region and northeastern United States, and mountainous areas of western North America from California to British Columbia (Gibilisco 1994). By the 1950s, the range of fishers had been reduced to 43% of its historical extent by targeted and incidental trapping, and loss or degradation of forest habitats from logging, fire management, and development (Lewis et al. 2012). Although fishers have recovered a modest portion of their former range in central and eastern North America (Powell et al. 2003, Lewis et al. 2012), they remain uncommon in western North America (Lofroth et al. 2010), and occupy just 21% of their historical distribution in the United States in Washington, Oregon, and California, an area designated the West Coast Distinct Population Segment (West Coast population segment) by the U.S. Fish and Wildlife Service [USFWS] (2014a).

Within the West Coast population segment, fishers occur in 3 remnant populations in southern Oregon, northern California, and the southern Sierra Nevada in eastern California (Lewis et al. 2012). Although 2 small, reintroduced populations are becoming established on the Olympic Peninsula, Washington and in eastern California after translocations in 2008 and 2009, extensive track and camera trap surveys suggest no evidence that any of the 3 remnant populations are experiencing consistent growth or reoccupying areas where they were once present (Zielinski et al. 2005, 2013; Swiers 2013; Spencer et al. 2015a; Sweitzer et al. 2015a).

Known causes of death, which may be limiting numeric and spatial recovery of fisher populations, include exposure to disease and debilitating injury (Aubry and Raley 2006, Larkin et al. 2011, Keller et al. 2012, Gabriel 2013, Lewis 2014), predator attacks (Truex et al. 1998, Aubry and Raley 2006, Wengert et al. 2014), incidental or targeted fur trapping (Lewis and Zielinski 1996, Koen et al. 2007, Lewis 2014), vehicle strikes (Krohn et al. 1994, York 1996, Chow 2009, Lewis 2014, Spencer et al. 2015a), entrapment in water tanks or other human structures (Folliard 1997, Truex et al. 1998, Davis 2008), and direct consumption and secondary exposure to rodenticides and insecticides (i.e., toxicants) at trespass marijuana grow sites in public wildlands (Gabriel et al. 2012, Thompson et al. 2013). Several reports suggest that fisher survival may vary between sexes or according to season associated with period-specific life-history events (e.g., reproduction, breeding movements, dispersal; Powell and Leonard 1983, Lewis 2014, Sweitzer et al. 2016a), higher energetic costs during winter (Powell 1979), or timing of exposure to toxicants (Thompson et al.

2013, Gabriel et al. 2015). Moreover, important denning and resting habitats used by fishers in western North America (i.e., mature and late-successional mixed conifer forests; Raley et al. 2012, Weir et al. 2012, Zhao et al. 2012, Aubry et al. 2013, Schwartz et al. 2013) appear at elevated risk from large forest fires (Thompson et al. 2011, Kane et al. 2015). Forest fuel reduction being used to reduce risk of wildfire (mechanical removal of understory trees, shrubs, downed logs, and surface fuels; Collins et al. 2011, Scheller et al. 2011) were reported to diminish suitability of resting habitats used by fishers (Truex and Zielinski 2013). Because habitat used by denning fishers in the southern Sierra Nevada region appears similar to habitats used for resting (Purcell et al. 2009, Zhao et al. 2012), there has been concern that forest fuel reduction may degrade habitat required for successful reproduction (Spencer et al. 2015b).

The USFWS recently proposed to list all (Washington to California), or part (Oregon and California) of the West Coast population segment of fishers as threatened under the Endangered Species Act (USFWS 2014b). The listing proposal was supported by agency concerns for loss or degradation of fisher habitat from wildfires and vegetation management as described above, and the cumulative and synergistic effects of toxicants, and other naturally occurring stressors (predation, disease, vehicle strikes) on survival and reproduction of fishers in the region (USFWS 2014a). Quantitative data are lacking on the population-level effects of any of these stressors on fishers in the West Coast population segment (USFWS 2014a). We evaluated cause-specific mortality and population growth for fishers in the southern Sierra Nevada, California, USA, an area where natural and human-associated risks contribute to reduced survival within an isolated population of <500 animals (Spencer et al. 2015a). Our objectives were to estimate and evaluate potential differences in mortality risks to fishers from natural and human-linked stressors (e.g., toxicants, vehicle strikes), and integrate information on risk-factor-specific survival into a deterministic population model to evaluate how fisher population growth might be altered from the current base level if management is able to reduce mortality from dominant risk factors. We acknowledge that direct or indirect exposure to toxicants may introduce sublethal effects on fisher survival, thereby making them more prone to mortality from other causes (e.g., synergistic effects; Thompson et al. 2013, USFWS 2014a). At present, it is not possible to quantify these influences, however, and our analyses were constrained to known-cause mortalities that were identifiable after thorough, comprehensive necropsy examination of fisher carcasses.

STUDY AREA

We conducted our study in the southern Sierra Nevada, California at 2 study sites in the Sierra National Forest from March 2007 to March 2013 (Fig. 1). A northern site was centered in the Bass Lake Ranger District and encompassed part of Yosemite National Park and a southern site was in the High Sierra Ranger District (Fig. 1). Both study sites were topographically complex, and bisected by river and stream

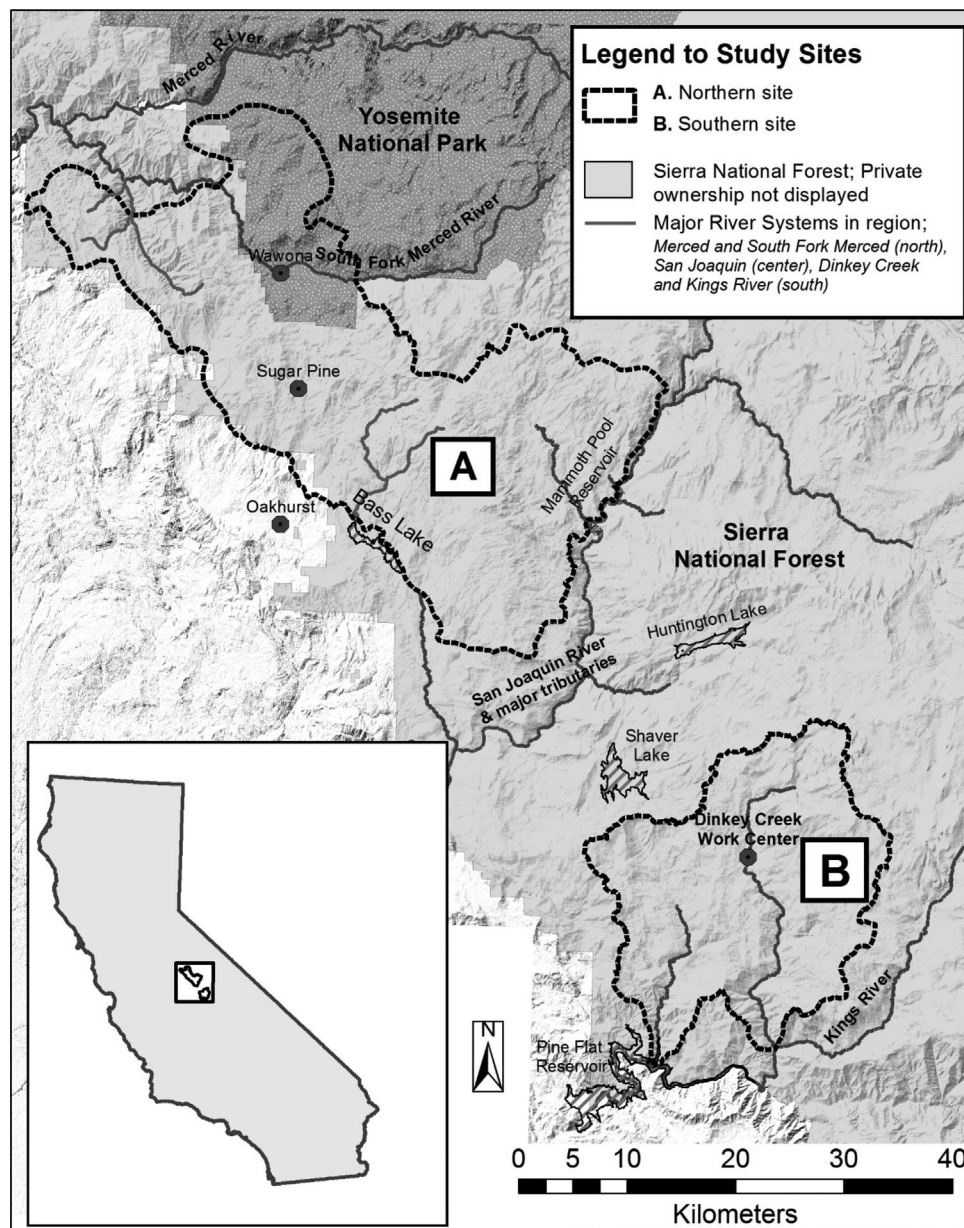


Figure 1. Map of the location of the northern site (A) and southern site (B) study areas where radio-collared fishers were monitored for survival from Mar 2007 to Mar 2014 as part of the Sierra Nevada Adaptive Management and Kings River Fisher Projects in the Sierra National Forest, California, USA.

canyons. The climate at the study sites was Mediterranean with precipitation typically occurring from October to mid-May, and rain during summer to early fall was rare. The long-term mean minimum and maximum annual temperatures at 1,516 m elevation at Fish Camp, near the center of the northern site were -4°C and 8°C , respectively. Land use at both study sites was dominated by public recreation, sightseeing, livestock grazing (excluding Yosemite National Park), and relatively low levels of extractive resource activities (e.g., logging but not within Yosemite National Park; Sweitzer et al. 2016b).

Fishers inhabited the study areas primarily between 1,050 m and 2,100 m elevation (Spencer et al. 2015a). Larger carnivores that attack and kill fisher in the region included bobcat (*Lynx rufus*), mountain lion (*Puma*

concolor), and coyote (*Canis latrans*; Wengert et al. 2014). Potential prey for fishers in the study area (Zielinski et al. 1999) included Douglas squirrel (*Tamiasciurus douglasii*), gray squirrel (*Sciurus griseus*), northern flying squirrel (*Glaucomys sabrinus*), long-eared chipmunk (*Neotamias quadrimaculatus*), and California ground squirrel (*Spermophilus beecheyi*). Primary tree species were incense cedar (*Calocedrus decurrens*), white fir (*Abies concolor*), ponderosa pine (*Pinus ponderosa*), sugar pine (*Pinus lambertiana*), California black oak (*Quercus kelloggii*), mountain dogwood (*Cornus nuttallii*), and white alder (*Alnus rhombifolia*). Giant sequoia (*Sequoiadendron giganteum*) was present but restricted to remnant populations in a few areas. Common shrubs and tree-like shrubs in the study area included willow (*Salix* spp.), whiteleaf manzanita (*Arctostaphylos*

viscida), greenleaf manzanita (*Arctostaphylos patula*), mountain misery (*Chamaebatia foliolosa*), blue elderberry (*Sambucus mexicana*), bush chinquapin (*Chrysolepis sempervirens*), mountain whitethorn (*Ceanothus cordulatus*), Sierra gooseberry (*Ribes roezlii*), and hoary coffeeberry (*Rhamnus tomentella cuspidata*).

METHODS

Trapping and Radio-Telemetry

We used steel mesh traps (model 207; Tomahawk Live Trap Company, Tomahawk, WI) modified to include a wood cubby to capture fishers (Wilbert 1992), fit them with radio-transmitters, and monitor them for survival. Full details of the methods we used to live-trap, handle, and process fishers are described elsewhere (Sweitzer et al. 2015a). Sedated fishers were weighed, classified by sex and age (juvenile [6–11 months], subadult [12–23 months], and adult [≥ 24 months]), marked with a subcutaneous passive integrated transponder (PIT) tag (Biomark, Boise, ID), measured for standard morphological features, and held in the cubby box until recovery and release. We affixed very high frequency radio-collars (Holohil Systems Model MI-2M, Ontario, Canada; Advanced Telemetry Systems Model 1930 or 1940, Isanti, MN) to most fishers, but 12 fishers were fitted with global positioning radio-collars (Telemetry Solutions Mini GPS collar, Concord, CA). All radio-collars had mortality sensors, and we inserted leather breakaway devices in the collar straps to avoid injury or strangulation between recaptures. We applied targeted trapping to re-capture fishers whose radio-collars were shed, and those with batteries that were near or completely expended (Sweitzer et al. 2015a).

We monitored radio-collared fishers from March 2007 through March 2014 on the southern site, and from December 2007 through March 2014 on the northern site to identify locations and determine fate. We used standard methods (Thompson et al. 2012) to relocate and assess survival status of all radio-collared fishers; those at the northern site were relocated 4–6 times/week by fixed-wing aerial radio-telemetry (Cessna 185, Cessna Aircraft Co., Wichita, KS; Piper PA-18 Super cub, Piper Aircraft, Vero Beach, FL), whereas those at the southern site were relocated 1–3 times/week by ground triangulation.

Capture, handling, and all other research protocols at our study sites followed American Society of Mammalogist guidelines (Gannon et al. 2007), and were approved by the Institutional Animal Care and Use Committees of the University of California, Berkeley (Protocol no. R139) and the University of California, Davis (Protocol no. 16302).

Determining Cause of Mortality

Signals from mortality sensors allowed us to detect and investigate inactive signals and recover carcasses as soon after death as practicable. We followed a standardized protocol when collecting samples and documenting circumstances at all mortality sites (Wengert et al. 2013). We recorded images and diagrams for all mortality locations, and used latex gloves when handling and recovering carcasses. Whenever field

signs suggested predator attacks, we collected samples for forensic assessment (e.g., clipped fur around bite wounds, swabs from chewed bones, and around bite wounds; Wengert et al. 2013). We stored carcass remains in sealed plastic bags in a -20°C freezer at each study site until they could be mailed or delivered to the University of California, Davis for necropsy and DNA forensic assessment by pathologists at the University of California Davis Veterinary Medical Teaching Hospital, and California Animal Health and Food Safety Laboratory in Davis.

Pathologists attempted to determine cause of death for individual fishers using all available information including gross necropsy, histology, disease and toxicological tests, predation forensics (DNA from predators), and any other descriptive evidence from mortality sites (Gabriel et al. 2015). When body organs were available, we screened liver tissue for presence of rodenticide residues using liquid chromatography-tandem mass spectrometry; toxicants from positive samples were quantified using high-performance liquid chromatography (Gabriel et al. 2012). When predation was suspected, we described all lesions potentially associated with predator attack. Presence of hemorrhaging indicated wounding before death, and helped to distinguish predator attacks from scavenging. For 16 mortalities, very little tissue remains were present and only molecular analyses were possible. We followed up visible field signs of predator attacks with forensic DNA testing of swabs, hair, and skin tissue around putative bite wounds or chew marks to identify the predator species that may have attacked and killed fishers (Wengert et al. 2013).

Predation was considered the cause of death if hemorrhage was present at bite, claw, or talon wounds, often but not always supported by presence of predator DNA. Mortalities were classified as disease when carcasses exhibited clinically significant infectious (e.g., bacterial, viral, macroparasites) or non-infectious factors (e.g., pneumonia, toxoplasmosis) considered to be the primary cause of death. Fishers that exhibited severe nutritional deficiency with lack of body fat and injuries considered likely to have prevented foraging were classified as starvation or illness. Fishers that died directly from entrapment in human structures, vehicle strikes, or those considered to have died after exposure to toxicants (rodenticide-linked toxicosis) were considered human-linked deaths. Fisher mortalities for which carcass remains were insufficient for actual necropsy or in poor condition with lack of forensic evidence because of severe autolysis, and those for which necropsy results were not available before January 2015 were classified as unknown deaths.

Cause-Specific Survival

We estimated annual survival probabilities using the Kaplan–Meier estimator (Kaplan–Meier survival) modified by Pollock et al. (1989) for staggered-entry of newly captured animals. We defined the fisher population year as 18 March to 17 March based on denning chronology (Sweitzer et al. 2015a). We entered radio-collared fishers into the dataset on the day after capture, and coded them as mortalities on the

estimated or known day of death. We censored missing animals but returned them to the dataset if they were recaptured (Heisey and Patterson 2006). A previous assessment for our study populations indicated that age and site were not important determinants of survival (Sweitzer et al. 2016a), and we combined data for all years for the analyses.

We estimated cause-specific mortality using the nonparametric cumulative incidence function (hereafter incidence function) detailed by Heisey and Patterson (2006), implemented in Program R (R Core Team 2013). We considered 3 competing known-cause risks and 1 unknown death factor (unknown). Known-cause risks were 1) predation, 2) disease, injury, and starvation (natural disease and starvation), and 3) rodenticide-linked toxicosis and vehicle strikes (toxicants and vehicle strikes). We did not attempt to estimate predator-specific mortality risks because predators that attacked and killed fishers were not always determinable from DNA forensics. One adult female and 1 juvenile female fisher failed to survive the capture process to release (Keller et al. 2012, Gabriel et al. 2015). We included these 2 deaths in tabular data on mortalities but excluded them from incidence function analyses because neither animal was monitored by radio-telemetry in the forest after capture.

We generated separate incidence functions for the 365-day year, and for 2 6-month seasons: spring and summer (18 Mar to 15 Sep) and fall and winter (16 Sep to 17 Mar). We used 6-month seasons instead of 4-month or 3-month periods in the risk-factor analyses because of sample constraints when partitioning known-cause mortalities into multiple groups (e.g., sex, site, season). We estimated seasonal survival rates by creating subsets of the data containing exposure days for each fisher in each season (Heisey and Patterson 2006). Output from incidence function analyses included Kaplan–Meier survival estimates for each risk factor, and cumulative hazard rates (mortality rates) for each type of risk. Mortality rates from the incidence functions represented the percent of all fishers, or percent of female fishers that were removed annually, or within each season (Patterson et al. 2013). We used Anderson–Gill proportional hazards models implemented in program R, package survival, function coxph (Therneau and Lumley 2011) to assess the influence of site, sex, and season on risk of death by the known-cause risk factors using methods described by Heisey and Patterson (2006) but modified slightly according to Therneau and Grambsch (2000; Supporting Information, available online at www.wildlifejournals.org). We interpreted risk ratios from the proportional hazards analyses as a percentage reduction in risk ($100 \times [\text{risk ratio} - 1.0]$) for the subset of contrasts that were significant at $P < 0.05$ (Barratt et al. 2004).

Known-Cause Survival and Population Growth Rates

We used known-cause survival rates from the incidence functions to evaluate the relative importance of predation, natural disease and starvation, and toxicants and vehicle strikes on the population growth rate (λ) for the fisher population at the northern site. We focused on the northern

site because there were no toxicants and vehicle strike deaths and very few natural disease and starvation deaths at the southern site, and because demographic rates were available for the northern site only (Sweitzer et al. 2015a); demographic rates for the southern site were proprietary and retained by the Forest Service (R. Green, University of California, Davis, personal communication). We previously developed a Leslie matrix (matrix) to estimate λ for the fisher population at the northern site (Sweitzer et al. 2015a), and used this same matrix formulation with previously reported demographic rates to evaluate how changes in survival due to predation, natural disease and starvation, toxicants and vehicle strikes, and natural disease and starvation + toxicants and vehicle strikes contributed to variation in λ . Full details on the matrix are provided by Sweitzer et al. (2015a), but the model was a pre-birth pulse model that integrated data on survival of juvenile female (P_0), subadult female (P_1), adult female (P_2 – P_7), and fertilities for 3 reproducing ages (24 months, m_1 ; 36–60 months, m_2 ; 72 months, m_3) according to $F_j = m_j P_0$ (Mills 2007), where m_j was the product of the overall proportion of each age female in the population that denned $\times$ litter size $\times$ sex ratio at birth (0.5; Sweitzer et al. 2015a). Juvenile survival was from the birth pulse in year j to immediately prior to the birth pulse in year $j + 1$ (Mills 2007), which was estimated from the product of the weaning rate for known denning females, Kaplan–Meier survival of females with trailing young-of-the-year offspring in summer, and Kaplan–Meier survival of radio-collared juveniles in fall and winter (Sweitzer et al. 2015a). Calculations of juvenile survival included consideration of deaths of adult females with trailing offspring, and deaths of radio-collared juveniles in fall and winter (Sweitzer et al. 2015a).

Our matrix analyses to estimate change in λ associated with each cause-specific mortality risk used Kaplan–Meier survival rates for subadult and adult females that were adjusted upward by reducing or removing the effect of each known-cause risk on survival. Kaplan–Meier survival for subadult and adult females were adjusted using the equation:

$$\text{Female Kaplan–Meier survival} + P(1 - \text{Kaplan–Meier survival})$$

where P was a proportion (0.25, 0.50, 1.0) representing a potential, or hypothetical management-based reduction in the mortality rate for each known-cause mortality risk factor.

Literature Review of Known-Cause Mortalities

We summarized data on cause-specific mortalities for radio-collared fishers that were identified at our 2 study sites and by 5 other fisher projects within the larger West Coast population segment. The 5 other sites for which data were available included the Hoopa Valley, northern California (Gabriel et al. 2015); Klamath River and north coast areas, northern California (Truex et al. 1998); Sequoia National Forest (Truex et al. 1998); southern Oregon (Aubry and Raley 2006); and the Olympic Peninsula, western Washington (Lewis 2014). Our purpose for this review was to combine information on fisher mortalities from other studies with ours, and evaluate the relative importance of

Table 1. Information on radio-collared fishers captured and monitored for survival in 7 population years (18 Mar–17 Mar) at a northern and southern site, Sierra National Forest, California, USA, March 2007 to March 2014.

Site and year	Monitored ^a	New captures ^b		Mortalities ^c		Missing ^d	Radio days ^e
		F	M	F	M		
Northern							
2007–2008	10	3	7		1		586
2008–2009	41	24	10	3	5	2	7,259
2009–2010	51	11	9	8	4	4	9,581
2010–2011	55	10	8	8	5	4	9,453
2011–2012	59	11	10	6	3	11	9,767
2012–2013	52	5	3	5	3	8	11,985
2013–2014	35	1	1	4	8	12	6,894
All year totals	113	65	48	34	29	41	55,525
Southern							
2007–2008	25	13	12	3	3		4,091
2008–2009	41	15	7		3	2	8,377
2009–2010	49	7	6	5	4	6	9,563
2010–2011	48	8	7	8	2	8	10,036
2011–2012	45	9	5	6	3	4	8,408
2012–2013	46	8	6	2	4	2	9,677
2013–2014	53	9	7	11	3	10	10,524
All year totals	119	69	50	35	22	32	60,676

^a No. of individual fishers monitored for 1 day in the population year, or during all years of the study.

^b No. of previously unmarked fishers captured in the population year, or during all years of the study.

^c No. of known fisher mortalities in the population year, or during all years of the study.

^d No. of individual fishers with dropped or failed radio-collars in the population year that were not subsequently recaptured; represents those fishers that were permanently censored in the Kaplan–Meier analyses of survival.

^e No. of days that radio-collared individual fishers were alive and monitored for 1 day within the population year, or during all years of the study.

deaths from multiple natural and anthropogenic causes on fisher populations within the West Coast population segment. We restricted the review to studies for which causes of death for radio-collared fishers were assigned after lab-based necropsy or examination by licensed pathologists or wildlife veterinarians.

RESULTS

We radio-collared 113 fishers at the northern site and 119 fishers at the southern site (Table 1). Fifty-six percent ($n = 63$) and 49% ($n = 58$) of the individual fishers captured and radio-marked died on the northern and southern sites,

Table 2. Known-cause mortalities identified for radio-collared fishers at a northern and southern study site, Sierra National Forest, California, USA from March 2007 to March 2014. Information on deaths of 15 non-collared fishers that were identified and reported to the research teams within the same time period is identified but was not included in analyses.

Cause of death	Northern		Southern		Non-collared fisher deaths (study site association)
	F	M	F	M	
Predation ^a	22	14	22	15	1; kit death base of den tree (northern).
Disease ^b		5	1 ^c		
Starvation or illness ^d	2 ^e	1		1	5; kit deaths at den trees or after rescues (2 northern, 3 southern).
Rodenticide toxicosis	1	5			
Vehicle strike		2			6 (5 northern, 1 southern).
Entrapment ^f				1	1; carcass in water tank (northern).
Rattlesnake bite			1		
Indeterminate ^g	2	3	6	4	2; skeletal remains (northern).
Unknown/no data ^h	4	2	5	2	

^a Includes 1 F that was infected with canine distemper when killed by a coyote (Keller et al. 2012), and 2 F that survived initial predator encounters but died of septicemia when wounds became infected.

^b Includes 3 deaths that were directly associated with canine distemper virus, 2 associated with non-infectious disease (Toxoplasmosis, pneumonia), and 1 from bacterial infection (septicemia).

^c A juvenile F fisher that died during capture was determined infected with canine distemper virus; this death was not included in analyses of known-cause mortality.

^d Fishers with evidence of debilitating injury that prevented foraging, leading to starvation or illness. One death was an animal that succumbed to bacterial infection (septicemia) after an unknown-cause injury.

^e Includes 1 adult F that was near-starvation and ill with septicemia when captured. She died before release, and the death was not included in analyses of known-cause mortality.

^f This animal was entrapped in a polyvinyl chloride (PVC) air sampling tube, and died by starvation and dehydration.

^g Fishers that were decomposed, or those for which neither necropsy or DNA forensics were able to identify cause of death.

^h Fishers for which necropsy results were indeterminate, or not available before 31 January 2015.

respectively (Table 1). Monitoring effort exceeded 55,500 radio days at both sites (Table 1).

Mortalities

We typically recovered carcasses ≤ 2 days after death at the northern site and ≤ 5 days after death at the southern site. We were able to assign a cause of death for 52 radio-collared fishers from the northern site (83% of all northern site deaths), and 41 fishers from the southern site (71% of all southern site deaths; Table 2). Predation was the dominant known-cause mortality risk, which removed more fishers from our study populations than any other environmental risk (Fig. 2). Predation was the primary cause of death at both sites, accounting for 69.2% of 52 known-cause deaths on the northern site, and 90.2% of 41 known-cause deaths on the southern site (Table 2). We identified 6 deaths from disease: 5 on the northern site and 1 on the southern site (Table 2). We identified 6 mortalities from exposure to toxicants and 2 from vehicle strikes, all at the northern site (Table 2). Death from starvation or illness was uncommon, based on just 4 deaths from these risk factors (Table 2). One fisher on the southern site that was recovered from an air sampling tube was considered an entrapment death, but we observed that the animals partially decomposed carcass had evidence of puncture wounds of indeterminate origin. There was a single fisher death on the southern site from a rattlesnake (*Crotalus oreganus*) bite (Table 2). We were unable to identify a definitive cause for 5 mortalities at the northern site and 10 mortalities at the southern site, and necropsy reports were not available for 13 radio-collared fishers that died (Table 2).

Six fisher deaths included indications of contributing mortality factors. The carcasses of 2 fishers that we classified as predation had puncture wounds consistent with predator attacks. The wounds became infected, and both animals died from septicemia days or weeks after being wounded (Table 2). Two northern-site fishers classified as starvation had suffered debilitating injuries from unknown causes sometime before death (Table 2). One fisher on the northern site that died from predation was infected with canine distemper virus at the time of death (Keller et al. 2012).

We also identified deaths of 15 non-collared fishers that we report but which were not used in any analyses. We were aware of the deaths of 6 fisher kits after their mothers died in the denning season (Table 2). Six non-collared fishers died by vehicle strikes, 5 within Yosemite National Park at the northern site, and 1 near Shaver Lake at the southern site. A fisher carcass was found inside a water tank by maintenance workers at the northern site, and cooperators located near skeletal remains of 2 different non-collared fishers at the northern site (Table 2).

Survival and Mortality Rates

Deaths from known and unknown causes contributed to an annual Kaplan–Meier survival rate of 0.69 for all fishers, and 0.72 for female fishers (Table 3). For clarity, we reiterate that these estimates of annual survival did not include the capture-associated deaths of 2 female fishers (Table 2). Known-cause survival rates ranged from 0.98 for toxicants and vehicle strikes to 0.79 for predation (Table 3). Known-

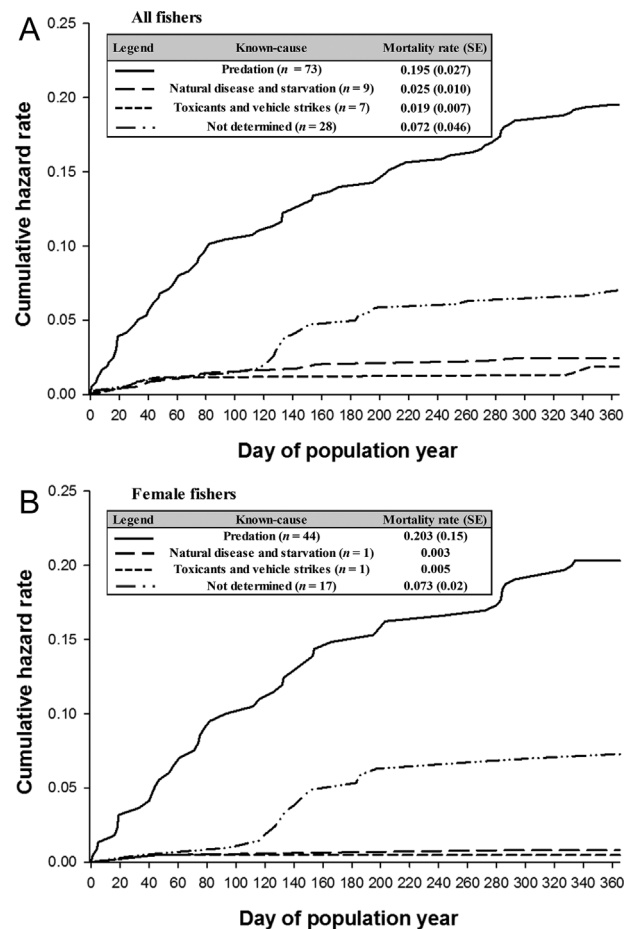


Figure 2. Cumulative probability of mortality (cumulative hazard rates) according to day of the fisher population year (day 1 = 18 Mar) for all fishers (A) and female fishers (B) from 3 classes of known causes of mortality. Causes of death were determined by necropsy examination, and mortalities were grouped for analysis based on evidence of predation (predation), evidence of disease and debilitating injury that contributed to starvation (natural disease and starvation), and evidence that exposure to rodenticide poisons or vehicle strikes produced death (toxicants and vehicle strikes). Mortalities for which a cause of death was not determinable by necropsy, and those for which necropsies were not completed before Jan 2015 were classified as not determined. Numbers of mortalities for each known cause are provided in parentheses. Mortality rates are the cumulative hazard rates for each type of known-cause death on the last day of the population year. Data used for the analysis were from radio-collared fishers that were monitored for survival from Mar 2007 to Mar 2014 as part of the Sierra Nevada Adaptive Management (northern site) and Kings River (southern site) Fisher Projects in the Sierra National Forest, California, USA.

cause survival rates among female fishers ranged from 0.99 for natural disease and starvation + toxicants and vehicle strikes to 0.79 for predation (Table 3).

Known-cause mortality rates among all fishers were highest from predation and lower from the natural disease and starvation category and the toxicants and vehicle strikes category (Fig. 2). Known-cause mortality rates among female fishers were highest for predation and lower for the toxicants and vehicle strikes category and the natural disease and starvation category (Fig. 2). Mortality rates from predation were higher in spring and summer (0.14 ± 0.02 SE) compared to fall and winter (0.07 ± 0.02 SE; Cox proportional hazard $z = -2.91$, $P < 0.01$, risk ratio = 0.49). The

Table 3. Kaplan–Meier estimator survival rates and cause-specific survival rates for multiple known risks encountered by radio-collared fishers at 2 study sites in the Sierra National Forest, California, USA, March 2007 to March 2014. Known-cause risks were grouped into 4 classes and 1 combined class for survival analyses: 1) predation; 2) natural disease and starvation; 3) toxicants and vehicle strikes; 4) unknown; and 5) natural disease and starvation and toxicants and vehicle strikes combined. Annual survival was for the fisher population year, 18 March to 17 March.

Description	Survival rates (SE)		
	All fishers	Female fishers	Adjusted female survival (proportional reduction) ^a
Annual	0.689 (0.024)	0.716 (0.030)	
Predation	0.793 (0.022)	0.789 (0.034)	0.769 (0.25), 0.822 (0.50)
Natural disease and starvation	0.971 (0.009)	0.995 (0.003)	0.721 (1.0)
Toxicants and vehicle strikes	0.978 (0.008)	0.995 (0.005)	0.721 (1.0)
Unknown ^b	0.915 (0.016)	0.916 (0.020)	0.916 (1.0)
Natural disease and starvation and toxicants and vehicle strikes	0.951 (0.008)	0.990 (0.003)	0.726 (1.0)

^a Estimated as annual F survival + $P(1 - \text{known-cause survival})$, where P is the hypothetical proportional reduction identified in parentheses for each known-cause survival rate.

^b Included deaths that were indeterminate, and those for which necropsy reports were not available.

risk ratio for this comparison indicated that relative mortality risk from predation was approximately 51% lower in the fall and winter compared to spring and summer. Mortality rates from predation were similar between female (0.20 ± 0.03 SE) and male fishers (0.18 ± 0.04 SE; Cox proportional hazard $z = -0.11$, $P = 0.91$), and similar between the northern site (0.19 ± 0.04 SE) and the southern site (0.20 ± 0.04 SE; Cox proportional hazard $z = -0.78$, $P = 0.43$). Mortality rates from natural disease and starvation + toxicants and vehicle strikes (combined because of small sample size) were lower for female fishers ($\leq 0.01 \pm 0.01$ SE) than for male fishers (0.10 ± 0.03 SE; Cox proportional hazard $z = 3.34$, $P < 0.01$, risk ratio = 12.35), and higher at the northern site (0.07 ± 0.02 SE) than at the southern site (0.01 ± 0.01 SE; Cox proportional hazard $z = -2.90$, $P < 0.01$, risk ratio = 0.11). Risk ratios from these comparisons indicated that relative mortality risk from natural disease and starvation + toxicants and vehicle strikes was 11.4 times higher for male fishers, and 89% lower at the southern site than at the northern site. Mortality rates from natural disease and starvation + toxicants and vehicle strikes were similar in spring and summer (0.03 ± 0.01 SE) and fall and winter (0.02 ± 0.01 SE; Cox proportional hazard $z = 0.10$, $P = 0.91$).

Cause-Specific Survival and Population Growth Trajectories

Predation was the only risk factor that depressed survival sufficiently to produce a potential negative overall growth trajectory for our study populations (Table 4). Annual female survival was estimated as 0.72, which could potentially improve to 0.77, 0.82, or 0.93 after 25%, 50%, or 100% reductions in predation (Table 4). Annual female survival would increase slightly to 0.73 with 100% reduction in both natural disease and starvation and toxicants and vehicle strikes (Table 3). The base population growth rate, λ , for the northern site population was projected from the matrix as 0.96 (Table 4). Integrating adjusted survival rates into the matrix produced estimates of λ of 1.03 (25% reduced predation), 1.11 (50% reduced predation), 0.97 (100% removal of natural disease and starvation), 0.97 (100%

removal of toxicants and vehicle strikes), and 0.97 (100% removal of natural disease and starvation + toxicants and vehicle strikes; Table 4).

Review of Known-Cause Mortalities

Our review of the available data identified information on 239 radio-collared fishers that died among 7 study sites within the West Coast population segment between 1992 and 2014 (Table 5). Known or likely causes of death were reported for 183 (77%) of the mortalities (Table 5). Predation was the most common cause of death for radio-collared fishers at 5 of the 7 sites, which was linked to 67% of all known-cause deaths (Table 5). Known-cause deaths associated with disease, injury, or starvation were identified at 4 of the 6 study sites, which were linked to 12% of all known-cause deaths (Table 5). Vehicle strikes occurred at 4 of 7 study sites, and contributed to 8% of all known-cause deaths (Table 5). Carcass remains were screened for toxicant residues at 3 research sites in California: the northern and southern site in this study, and the Hoopa Valley, northern California. Thirteen fisher deaths from toxicants were identified, representing 8% of 136 known-cause deaths for the 3 sites (Table 5). Eleven fisher deaths were linked to other types of natural or anthropogenic factors, and 56 deaths were not attributed to a known cause (Table 5).

DISCUSSION

Our study advanced prior research on fishers in western North America by linking data on mortality risks and cause-specific survival to likely growth trajectories for a remnant population that is of conservation concern due to small size (Spencer et al. 2015a). Koen et al. (2007) was the only other study of fishers in North America that used incidence functions to estimate mortality rates for known risks in a fisher population. Mortality rates for fishers in eastern Ontario, Canada were 30% from anthropogenic factors (fur- and nuisance trapping, road kill, other), 18% from natural causes (injury, disease, starvation), and 20% from unknown causes (Koen et al. 2007). Fur-trapping occurred in the Koen et al. (2007) study area, and when fur- and nuisance trapping were excluded, the mortality rate for anthropogenic causes

Table 4. Information on demographic rates, and annual female Kaplan–Meier survival rates (S) used to evaluate how potential management reductions of fisher mortality from predation (predation); disease, injury, and starvation (natural disease and starvation); exposure to anticoagulant rodenticides and vehicle strikes (toxicants and vehicle strikes); or natural disease and starvation and toxicants and vehicle strikes combined may influence population growth, based on a Leslie matrix developed for the northern site in the Sierra National Forest, California. Blank cells indicate that the parameter remained the same as the base rate. The matrix was fully detailed by Sweitzer et al. (2015a), and the data were from radio-collared fishers monitored at the northern site from December 2007 to March 2014.

Demographic rates ^a	Parameter values with S adjusted ^b				
	Base rates	25%, 50% reduced predation	100% reduced natural disease and starvation	100% reduced toxicants and vehicle strikes	100% reduced natural disease and starvation + toxicants and vehicle strikes
Fecundity (female kits) ^c					
Young ad (F_2)	0.544				
Ad (F_3)	0.729				
Mature (F_{4-7})	0.641				
Survival rates for estimating P_0 ^d					
Kit survival in dens (18 Mar–10 Jun)	0.823				
Denning F survival (11 Jun–1 Sep)	0.870	0.890, 0.911			
Fall to winter juvenile survival (30 Sep–17 Mar)	0.790	0.842, 0.895			
Survival rates					
Juvenile (P_0) ^e	0.565	0.671, 0.670			
Subad (P_1) ^f	0.716	0.769, 0.822	0.721	0.721	0.726
Ad (P_{2-7}) ^f	0.716	0.769, 0.822	0.721	0.721	0.726
Fertility rates ^g					
Young ad (F_2)	0.308	0.336, 0.365			
Ad (F_3, F_4, F_5)	0.412	0.450, 0.489			
Mature ad (F_6, F_7, F_8)	0.362	0.396, 0.430			
Leslie matrix λ ^h	0.960	1.032, 1.108	0.965	0.965	0.970

^a Demographic rates (reproductive rates, litter sizes) for fishers at the northern site reported by Sweitzer et al. (2015a).

^b Adjusted F Kaplan–Meier S rates were calculated as F Kaplan–Meier $S + P(1 - \text{known-cause Kaplan–Meier } S)$, where known-cause Kaplan–Meier S was the Kaplan–Meier survival rate for each known-cause mortality risk, and P was the proportional reduction in mortality risk for each known-cause survival rate.

^c Fecundity was the product of reproductive rate $\times$ litter size $\times$ sex ratio, which were reported by Sweitzer et al. (2015a).

^d Rates were calculated using the Kaplan–Meier estimator implemented in Program R for the periods of the year described for each age-class of fisher.

^e Estimated as the product of kit survival in the den season $\times$ denning F survival $\times$ fall to winter juvenile survival.

^f Rates were from Kaplan–Meier survival analyses for F fishers using the nonparametric cumulative incidence function script implemented in R.

^g Estimated according to $F_j = m_j P_0$, where m_j was the product age-specific fecundity $\times$ juvenile survival (P_0).

^h Estimated with a Leslie matrix using the survival rates, P_0, P_1, P_2-P_7 , and fertility rates (F_2-F_8) in each column.

(vehicle strikes and other) was 7%, compared to approximately 2% for human-linked causes (toxicants or vehicle strikes) in our study population. The mortality rate of 18% due to natural risks in eastern Ontario, Canada was slightly lower than the 22% mortality rate from predation, disease, injuries, and starvation at our study sites in California.

Wengert et al. (2014) hypothesized that high levels of predation in the West Coast region are due to human-induced change in forest habitat that facilitates more frequent contact between fishers and other forest carnivores. It is also possible that roads, trails, and early seral and edge habitats function to increase the number of lethal interactions between fishers and larger predators (Spencer et al. 2015a) because bobcats, mountain lions, and coyotes all use roads or edge habitats when transiting their home ranges and searching for prey (Rolley and Warde 1985, Smallwood and Fitzhugh 1995, Atwood et al. 2004).

Death from starvation was uncommon ($n = 3$) and always associated with illness or injury that prevented foraging. Two fishers that succumbed to starvation had suffered debilitating injuries from unknown causes sometime before death

(Table 2). Those 2 deaths occurred in summer or early fall, suggesting that potential prey limitation or higher energetic costs during winter (Powell 1979) were not contributing factors. The single starvation death that occurred during winter was an 11-year-old female with heavily worn teeth, for which the necropsy identified presence of noninfectious disease as a contributing factor. Three other studies previously reported injury-related deaths in western North America (Aubry and Raley 2006, Weir and Corbould 2008, Lewis 2014).

Six radio-collared fishers in our study population died from exposure to diseases that were not associated with old age or prior injury (Table 2). Infectious disease has been a concern for fisher populations in California since exposure to canine distemper virus and other pathogens was first documented 15 years ago (Brown et al. 2008). Canine distemper virus is of particular concern because near catastrophic, population-wide mortality has been reported for multiple species of rare carnivores (e.g., Island fox [*Urocyon littoralis catalinae*]; Timm et al. 2009, black-footed ferret [*Mustela nigripes*]; Williams et al. 1988, Woodroffe 1999). A localized epizootic

Table 5. Review of data on mortalities reported for radio-collared fishers from recent studies in the West Coast region of the United States. Data do not include information on deaths of non-collared fishers that were occasionally identified by biologists at the study sites.

Location ^a	All mortalities, known cause (% total) ^b	Natural deaths			Human-linked deaths			
		Likely predation	Disease, injury, starvation	Drowning, other	Toxicants	Vehicle- strike	Humans, trapping	Unknown
Sierra National Forest, CA								
Northern site ^c	63, 52 (83%)	36	8		6	2		11
Southern site ^c	58, 41 (71%)	37	2	1			1	17
Sequoia National Forest, CA	9, 4 (44%)	3	1					5
E. Klamath, North Coast, CA	13, 10 (77%)	6				2	2	3
Hoopa Valley, northern CA ^c	52, 43 (83%)	23	9		7	3	1	9
Olympic Peninsula, WA	35, 25 (71%)	15		2		7	1	10
Rogue River, OR	9, 8 (89%)	2	3	1			2	1
Totals	239, 183 (77%)	122 (67%)	23 (13%)	4 (2%)	13 (10%) ^d	14 (8%)	7 (4%)	56

^a Sources of data: this study (Sierra National Forest), Truex et al. (1998) (Sequoia National Forest, E. Klamath, North Coast), (Hoopa Valley), Lewis (2014) (Olympic Peninsula), Rogue River).

^b Known-cause deaths for all studies were determined by necropsy, necropsy with associated testing for diseases and rodenticides, or from examination by licensed wildlife veterinarians. Percent total was calculated from known-cause deaths/all mortalities.

^c Studies for which organ tissues were assessed for presence of anticoagulant rodenticides do not include the Olympic Peninsula study because the researchers did not consider tissues samples that were assessed for rodenticide residues representative of toxicant exposure in the region (J. Lewis, Washington Department of Fish and Wildlife, personal communication).

^d Calculated using the data from 3 study sites in California where carcass remains were assessed for exposure to anticoagulant rodenticides.

of canine distemper virus in our study sites in spring 2009 that contributed to deaths of 4 fishers was described by Keller et al. (2012). We note that 1 female fisher (F14) at the northern site was infected with canine distemper when killed by a coyote. Fishers at both of our study sites had evidence of prior exposure to canine distemper that did not kill them (Gabriel 2013). Fisher F14 was an example of a synergistic association between mortality risks because it is possible the animal would have survived had illness not compromised her ability to avoid the predator.

Toxicant-linked deaths have been of high concern among fishers in the West Coast region of the United States since 4 deaths most likely associated with poisons left at trespass marijuana grow sites were reported in 2012 (the deaths occurred in 2009 and 2010; Gabriel et al. 2012, Thompson et al. 2013). Two of the 4 toxicant deaths reported by Gabriel et al. (2012) were radio-collared fishers at our northern site. Subsequent testing of archived tissue revealed that the majority of fishers that died in California from 2007 to 2011 had been exposed to 1 toxicants associated with trespass grow sites (Gabriel et al. 2012). Moreover, in the period from 2009 to 2014, 13 fishers in California died from toxicants: 6 at our northern site, and 7 in the Hoopa Valley in northern California (Gabriel et al. 2015). Eleven of the 13 known toxicant deaths in California were male fishers, and Gabriel et al. (2015) reported that male fishers were 7 times more likely to die from toxicant exposure than from natural disease, and 13 times more likely to die from poisoning than from predation. Thompson et al. (2013) reported reduced survival among female fishers with higher numbers of trespass grow sites within their annual home ranges, but male fishers were not included in the analyses. We hypothesize that male fishers may be more prone to death from toxicants than females because males range over much larger areas than females (Popescu et al. 2014, Sauder and Rachlow 2015),

thereby increasing their exposure to poisons at trespass marijuana grow sites. For example, the mean annual home range size for male and female fishers at the northern site were 86.2 km² and 22.9 km², respectively (Sweitzer et al. 2015b). There is a need for detailed analyses of relationships between fisher survival (both sexes) and exposure to toxicants. One suggested approach would be to explicitly link the number, year, and actual location of trespass marijuana grow sites to concurrent occupied fisher home ranges. A model-based survival analyses could be used to evaluate survival in relation to sex, home range size, and various covariates for toxicant exposure including number of grow sites, types of chemicals known present, and year of grow site activity. These types of analyses may improve our understanding of the apparent sex-bias and local and region scale differences in toxicant deaths among fishers in California (Gabriel et al. 2015).

The importance of fisher deaths by wildlife-vehicle collisions was likely underestimated by the deaths of 2 radio-collared fishers on a state highway at the northern site. The 2 deaths occurred on Highway 41, a busy road that provides access to Yosemite National Park from the south. Between 2008 and 2014, 5 non-collared fishers were killed on Highway 41 within Yosemite National Park (Table 2), but we know that this stretch of highway has been continually problematic for fisher survival because Chow (2009) previously reported 4 vehicle-strike deaths on it. We are aware of 24 roadkill deaths in the West Coast population segment between 1992 and 2014, and fishers elsewhere in the North America commonly die on highways (Krohn et al. 1994, York 1996). Data from cameras used to monitor culverts along Highway 41 at the northern site revealed that fishers used several existing culverts to safely pass beneath the highway, suggesting it may be possible to reduce roadkill deaths by more regular maintenance of damaged or debris-occluded culverts (O'Brien et al. 2013). Moreover,

highway improvement funds could be used for construction of underpass or overpass structures specifically designed for wildlife use (McCollister and Manen 2010, Kintsch and Cramer 2011).

Two fisher deaths in our study area were linked to entrapment in human-structures (Table 2). Truex et al. (1998) and Powell et al. (2013) both reported deaths of radio-collared fishers in water tanks in northern California, whereas Folliard (1997) recovered remains of 8 fishers from a single abandoned water tank in northwestern California. We repeat Folliard's (1997) previous recommendation that abandoned tanks should be covered or modified by inserting branches or poles that will allow trapped fishers opportunity for escape.

The base λ for our northern site population was estimated at 0.96, and we found that hypothetical reduction in predation was the only way to promote improvement in λ to 1.0 (Table 4). Twenty-five percent or 50% reductions in predation might improve λ to 1.03 or 1.11, whereas 100% analytical reduction in fisher deaths from all other known-cause risks would fail to promote an increase in λ above 0.97 (Table 4). However, we also note that a modest 25% reduction in predation would be difficult to achieve because direct predator control would not be readily accepted by the public, state, and federal agencies, or nongovernmental environmental groups because of interest in mammalian carnivores, and because of the potential for disruption of ecosystem processes from predator removal (Reynolds and Tapper 1996, Rominger et al. 2006, Estes et al. 2011). Moreover, we suggest that habitat management might be a viable alternative for reducing contact between fishers and larger carnivores (Spencer et al. 2015b).

Based on our data on known-cause deaths and prior work on the population biology of fishers, we were not surprised that λ in the Sierra National Forest was more sensitive to predation than the other risks. Our estimates of cause-specific mortality (Fig. 2; 0.203 from predation, <0.003 from natural disease and starvation, < 0.005 from toxicants and vehicle strikes 2) can be used to represent differences in annual removal of female fishers from the different risk factors. Fisher deaths from predator attacks removed 67 times (0.203 vs. 0.003) and 41 times (0.203 vs. 0.005) more female fishers from the study sites each year than natural disease and starvation, or toxicants and vehicle strikes, respectively. The high rate at which predation removed females was important because all prior models for fisher populations reported that λ was more sensitive to female survival than any other demographic rate (Spencer et al. 2011, Lewis et al. 2012, Swiers 2013, Sweitzer et al. 2015a).

Although natural mortality from predation was the dominant risk encountered by fishers in our study and in the overall West Coast population segment region (Table 5), we know from prior research on fishers and other carnivores that sub-lethal doses of poisons can weaken, and incapacitate them (Riley et al. 2007, Thompson et al. 2013). Because of the limited serological and tissue tests available (Gabriel et al. 2015), we cannot identify what proportion of deaths from predation, disease, starvation, or vehicle strikes reported in

the West Coast population segment may have resulted when toxicant-stricken fishers were unable to avoid other risks.

MANAGEMENT IMPLICATIONS

Newly implemented policies and law enforcement are likely reducing use of toxicants on national forests (USFWS 2014a), but exposure will continue for an unknown duration and we recommend expedited removal of poisons at trespass grow sites whenever possible. Predation was the most important population-limiting factor in this study, and the dominant mortality risk in the West Coast region. Managers will, therefore, need to consider indirect approaches for reducing predation while research is underway to illuminate the types of habitat conditions that contribute to interspecific contact and vulnerability of fishers to predator attack. Female fishers are often killed near den trees in spring (Sweitzer et al. 2015a), and management prescriptions for minimizing habitat disturbance in known denning areas (U.S. Forest Service 2004) could be extended to areas identified as suitable by models of denning habitat (Spencer et al. 2015b). When mechanical fuel reduction is necessary, silvicultural activities should avoid large reductions in overstory canopy cover and understory structural diversity in high suitability denning areas (Zhao et al. 2012). Reducing predation via management of forest habitat will take time, but remediating current low use, unneeded roads, and skid trails in otherwise intact forest could facilitate reduced contact between fishers and larger carnivores without interfering with recreational use of wildlands.

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