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Research Article

Modeling Risk of Pneumonia Epizootics in Bighorn Sheep

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ABSTRACT Pneumonia epizootics are a major challenge for management of bighorn sheep (*Ovis canadensis*) affecting persistence of herds, satisfaction of stakeholders, and allocations of resources by management agencies. Risk factors associated with the disease are poorly understood, making pneumonia epizootics hard to predict; such epizootics are thus managed reactively rather than proactively. We developed a model for herds in Montana that identifies risk factors and addresses biological questions about risk. Using Bayesian logistic regression with repeated measures, we found that private land, weed control using domestic sheep or goats, pneumonia history, and herd density were positively associated with risk of pneumonia epizootics in 43 herds that experienced 22 epizootics out of 637 herd-years from 1979–2013. We defined an area of high risk for pathogen exposure as the area of each herd distribution plus a 14.5-km buffer from that boundary. Within this area, the odds of a pneumonia epizootic increased by >1.5 times per additional unit of private land (unit is the standardized % of private land where global $\bar{x} = 25.58\%$ and SD = 14.53%). Odds were >3.3 times greater if domestic sheep or goats were used for weed control in a herd's area of high risk. If a herd or its neighbors within the area of high risk had a history of a pneumonia epizootic, odds of a subsequent pneumonia epizootic were >10 times greater. Risk greatly increased when herds were at high density, with nearly 15 times greater odds of a pneumonia epizootic compared to when herds were at low density. Odds of a pneumonia epizootic also appeared to decrease following increased spring precipitation (odds = 0.41 per unit increase, global $\bar{x} = 100.18\%$ and SD = 26.97%). Risk was not associated with number of federal sheep and goat allotments, proximity to nearest herds of bighorn sheep, ratio of rams to ewes, percentage of average winter precipitation, or whether herds were of native versus mixed or reintroduced origin. We conclude that factors associated with risk of pneumonia epizootics are complex and may not always be from the most obvious sources. The ability to identify high-risk herds will help biologists and managers determine where to focus management efforts and the risk factors that most affect each herd, facilitating more effective, proactive management. © 2015 The Wildlife Society.

KEY WORDS bighorn sheep, decision curve analysis, disease, Montana, *Ovis canadensis*, pneumonia, risk model.

Pneumonia epizootics present an important challenge for effective management of bighorn sheep (*Ovis canadensis*; Gross et al. 2000, Cahn et al. 2011, Wehausen et al. 2011, Cassirer et al. 2013, Plowright et al. 2013). Once pneumonia pathogens are introduced to a population of bighorn sheep, initial all-age mortality can exceed 80% (Enk et al. 2001, Montana Fish, Wildlife and Parks [MFWP] 2010). The pathogens also may become endemic, resulting in pneumonia outbreaks that can cycle for years to decades (Enk et al. 2001, Cassirer and Sinclair 2007, Cassirer et al. 2013, Plowright et al. 2013). Of critical concern, lamb recruitment often remains chronically low for many years following an epizootic, which further threatens a herd's long-term persistence, particularly if pre-epizootic abundance was low, mortality rates were high, or other stochastic events (e.g., environmental or demographic) occur that further suppress or push the herd to extinction (Woodroffe 1999,

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Singer et al. 2000*c*, Cassirer and Sinclair 2007, Cassirer et al. 2013, Plowright et al. 2013). Herds may require extensive management to recover, including removal of diseased individuals (Edwards et al. 2010), augmentation from other herds (MFWP 2010), or reintroductions (Singer et al. 2000*b*). Despite great outlays of time and expense in attempt to restore herds after a pneumonia epizootic, they may never fully recover to pre-epizootic abundance and health (e.g., Enk et al. 2001, MFWP 2010, Cassirer et al. 2013, Plowright et al. 2013).

Identifying causes and influences of pneumonia epizootics has been the goal of extensive study; the etiology remains poorly understood, however, and the need for further research is commonly cited (Monello et al. 2001; Cassaigne et al. 2010; Miller et al. 2011, 2012). Presence of certain pathogens such as *Mycoplasma ovipneumoniae* and *Mannheimia haemolytica* are likely indicative of risk (Miller et al. 2011; Besser et al. 2012*a*, *b*, 2013; Shanthalingam et al. 2014). After decades of research, however, relationships between the various known and hypothesized risk factors affecting transmission, spread, and susceptibility of the pathogens that lead to pneumonia remain unclear. A single risk factor associated with all pneumonia epizootics has not yet been found, if it exists (Miller et al. 2012). Elucidation of risk factors and novel management tools for this complicated, much-debated management challenge and serious threat to persistence of herds of bighorn sheep are much needed.

The central role of domestic sheep and goats in exposure to pathogens is well documented; pathogen transmission from domestic to bighorn sheep is the only supported hypothesis in experimental trials (Wehausen et al. 2011). Healthy captive bighorn sheep sicken and die when penned with domestic sheep (Foreyt and Jessup 1982, Onderka and Wishart 1988, Foreyt 1989, Lawrence et al. 2010) or after accidental contact with domestic sheep (Foreyt and Jessup 1982). Analysis of pathogens in epizootics of free-ranging bighorn sheep also supports the hypothesis that pathogens are transmitted between Old World Caprinae species and immunologically naïve bighorn sheep (Besser et al. 2012*b*, 2013). Proximity of bighorn sheep to grazing allotments with domestic sheep is associated with increased susceptibility to pneumonia (Monello et al. 2001) and decreased persistence of the herd over time (Singer et al. 2000*b*, 2001; Epps et al. 2004; Clifford et al. 2009; Carpenter et al. 2014). Contact with feral goats also appears to result in exposure to pathogens (Rudolph et al. 2003). Contact with sheep or goats on commercial and hobby farms or when sheep or goats are used for weed control (i.e., targeted grazing to manage noxious weeds) may result in exposure to pathogens (Miller et al. 2011, 2012; Wild Sheep Working Group 2012). Evidence also suggests herds of bighorn sheep are likely more interconnected than previously thought (Singer et al. 2000*a*, DeCesare and Pletscher 2006), and that proximity among herds may increase risk of exposure to pneumonia pathogens through such connectivity (Onderka and Wishart 1984, George et al. 2008, Edwards et al. 2010, Besser et al. 2013).

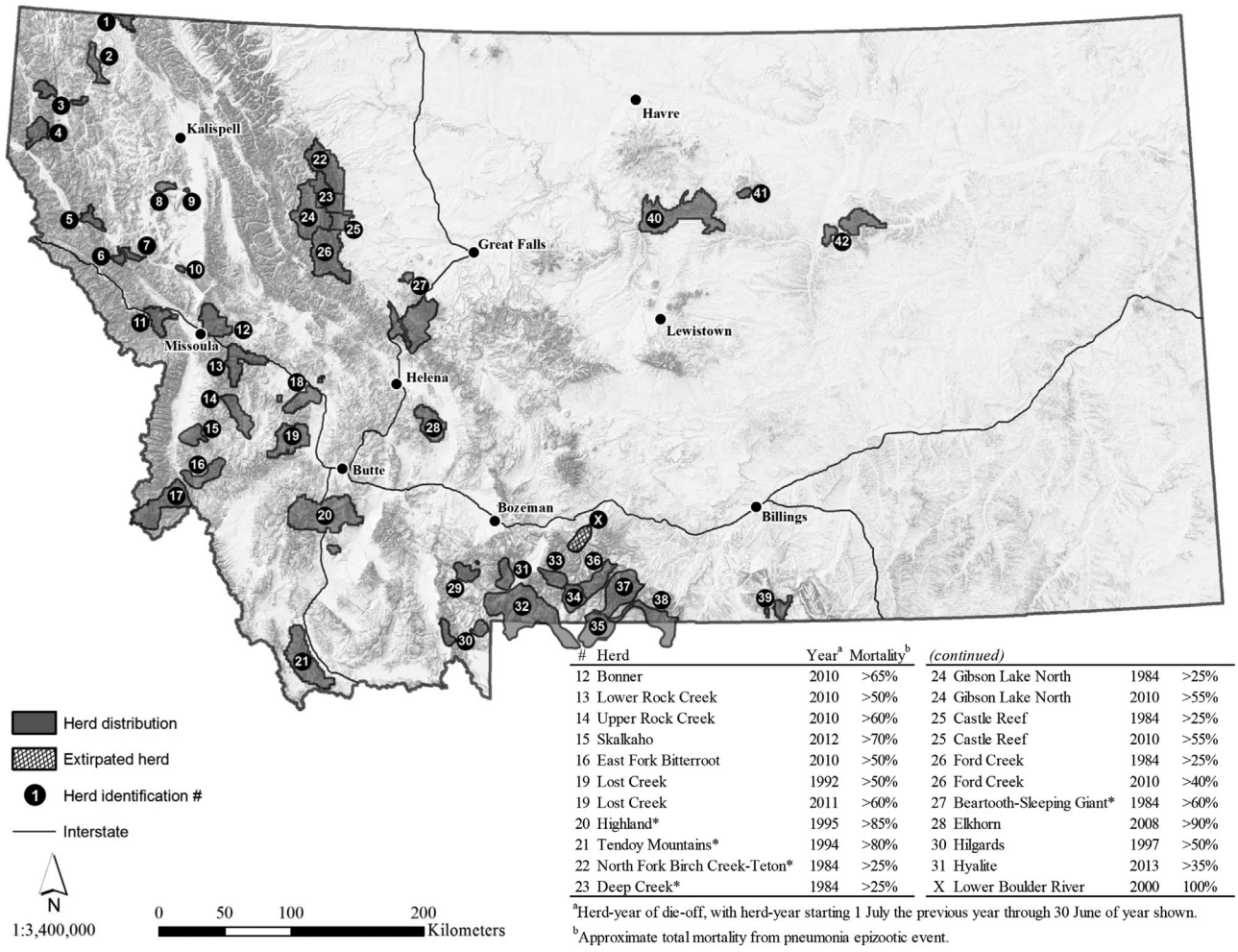
Conditions other than comingling between bighorn sheep and domestic sheep or goats may be associated with spread of and susceptibility to pneumonia pathogens, because comingling does not always quickly lead to pneumonia epizootics and some epizootics occur without known or confirmed contact (e.g., Onderka and Wishart 1984, George et al. 2008, Edwards et al. 2010). Rams have a greater tendency than ewes to make long movements (Singer et al. 2000*b*, DeCesare and Pletscher 2006, O'Brien et al. 2014), probably more so at relatively high densities (Singer et al. 2000*a*, Monello et al. 2001). Such movements increase their risk of contacting domestic sheep or other infected herds and spreading pathogens upon return to their own herds (Onderka and Wishart 1984, George et al. 2008, Besser et al. 2013). High densities of bighorn sheep may also result in high rates of contact between individuals, increasing the rate of spread of pathogens (Monello et al. 2001, Lafferty and Gerber 2002, Clifford et al. 2009). Disease processes can also be influenced by complex environmental interactions, including those that may place stress on the health and immune response of animals (Scott 1988, Wobeser 2006). Harsh winters have been associated with disease events (Monello et al. 2001, MFWP 2010), and pneumonia incidence increases in the fall and winter (Cassirer and Sinclair 2007). Harsher winter conditions may stress animals by affecting energy budgets or reducing access to adequate forage (Goodson et al. 1991, Butler et al. 2013). Low precipitation has been linked to lower lamb survival (Portier et al. 1998) and to herd extinctions (Epps et al. 2004), perhaps because dry growing seasons might increase susceptibility to disease through decreased forage quality (Enk et al. 2001, Monello et al. 2001). Herds that are augmented or reintroduced appear to be at higher risk of pneumonia than native herds, perhaps because of factors associated with reintroduction, the source herd, or the possibility that sites where herds were previously extirpated are more risky for pneumonia than where herds have not died out (Monello et al. 2001, Rudolph et al. 2007, Plowright et al. 2013).

Several models have been developed to simulate impacts of pneumonia from exposure to allotments, distance to domestic sheep, or contact with nearby infected herds of bighorn sheep and to predict population size, mortality rates, or herd persistence in relation to pneumonia (Gross et al. 2000, Clifford et al. 2009, Cassaigne et al. 2010, Cahn et al. 2011, Carpenter et al. 2014). Recent models also estimate the overall probability of transitioning between healthy and all-age, lamb-only, or adult-only pneumonia (Cassirer et al. 2013) and immune response by modeling how pneumonia exposure affects an individual's risk of dying from pneumonia (Plowright et al. 2013). Another recent model estimates probability of contact between individual bighorn sheep and allotments with domestic sheep and goats (O'Brien et al. 2014). Several models simulate the effect of management actions, primarily focused on changing management of grazing allotments (Clifford et al. 2009, Cahn et al. 2011, Carpenter et al. 2014) as well as modifying habitat, colonization of new patches, or impacts of stochastic events (Gross et al. 2000). These models predict the consequences of epizootics, but none predict risk of epizootics for

individual herds (but see Clifford et al. [2009] and Carpenter et al. [2014]).

Despite the breadth of previous studies on pneumonia in bighorn sheep, state wildlife agencies generally do not have a clear understanding of risk factors contributing to epizootics in herds they manage, how data available to them might be associated with such risk factors, or how these data might be used to predict epizootics. Agencies need risk assessment models to help prioritize herds and allocate limited resources to proactively manage risk of disease (Mitchell et al. 2013). Such a model should capture variability across the range of environmental conditions in which managed herds exist; models developed under more limited spatial or temporal extents may have little predictive power. Without such models, management of pneumonia epizootics in bighorn sheep has historically been reactive, resulting in crisis management rather than proactive prevention (Woodroffe 1999).

To begin addressing this issue, Mitchell et al. (2013) developed a preliminary pneumonia risk model and proactive decision model for bighorn sheep in Montana. The goal of the risk model was to predict the likelihood of pneumonia epizootics for herds managed by Montana Fish, Wildlife and Parks (MFWP). The predictions were then used to inform the decision model designed to facilitate proactive management decisions given the objectives and constraints of managers. Their risk model was based only on expert opinion of biologists and managers and did not attempt to empirically quantify risk factors associated with pneumonia epizootics. Our objective, therefore, was to develop an empirical risk model of pneumonia epizootics using readily available data that we hypothesized could contribute to epizootics in bighorn sheep, based on previous work. Our model was designed to facilitate making herd-specific predictions and decisions regarding epizootic risk as part of comprehensive statewide management of bighorn sheep herds in Montana (Fig. 1). We used decision curve analysis (Vickers and Elkin 2006, Steyerberg et al. 2010) to evaluate the capacity of our model to inform such decisions. This analysis allowed us to assess our model's relative capacity for separating high-risk herds from low-risk herds and the relative merits of using reactive or proactive management of all herds in the absence of a predictive model.



#	Herd	Year[a]	Mortality[b]
12	Bonner	2010	>65%
13	Lower Rock Creek	2010	>50%
14	Upper Rock Creek	2010	>60%
15	Skalkaho	2012	>70%
16	East Fork Bitterroot	2010	>50%
19	Lost Creek	1992	>50%
19	Lost Creek	2011	>60%
20	Highland*	1995	>85%
21	Tendoy Mountains*	1994	>80%
22	North Fork Birch Creek-Teton*	1984	>25%
23	Deep Creek*	1984	>25%

(continued)

24	Gibson Lake North	1984	>25%
24	Gibson Lake North	2010	>55%
25	Castle Reef	1984	>25%
25	Castle Reef	2010	>55%
26	Ford Creek	1984	>25%
26	Ford Creek	2010	>40%
27	Beartooth-Sleeping Giant*	1984	>60%
28	Elkhorn	2008	>90%
30	Hilgards	1997	>50%
31	Hyalite	2013	>35%
X	Lower Boulder River	2000	100%

[a]Herd-year of die-off, with herd-year starting 1 July the previous year through 30 June of year shown.
[b]Approximate total mortality from pneumonia epizootic event.

Figure 1. Locations of 43 herds of bighorn sheep with 22 pneumonia epizootic events with ≥25% mortality between 1979 and 2013, which we used to develop a pneumonia risk model for Montana. We excluded several additional epizootics from our analysis. Numbers correspond to risk estimates in Table 5 and to the table for epizootics within the map, where a * after the herd name indicates that we excluded post-epizootic herd-years from analysis because the herd received transplants, confounding signs of recovery.

STUDY AREA

Populations of bighorn sheep are found in western Montana and in portions of the Missouri Breaks in central Montana (Fig. 1). Habitat characteristics vary widely across these regions. Elevations range from 600 m to 4,000 m (MFWP 2010). Northwestern Montana is characterized by dense forests and generally rugged and mountainous terrain with a climate typical of the Pacific Northwest. Southwestern Montana is characterized by rolling foothills and rugged mountains, with heavier snow cover on western aspects, rain shadows on eastern aspects, and shrubs and bunchgrasses leading to conifers and alpine vegetation at increasing elevations. West-central Montana is characterized by low rolling hills and rugged mountain canyons, with a transitional mix of climate characteristics typical of southwestern and eastern Montana. South-central Montana includes sheer mountain canyons and rolling hills with shrub desert, montane forest, intermountain grasslands, alpine plateaus, and widely varying climates. The Missouri Breaks is semiarid with flat or rolling benchlands, rugged badlands, riparian areas, and ponderosa pine (*Pinus ponderosa*) savannahs. Federal sheep and goat grazing allotments have been distributed throughout Montana for the past 3 decades except in the northwestern region. Weed control with domestic sheep and goats has occurred throughout the state, as have commercial and hobby farms on private lands that can include domestic sheep and goats.

METHODS

Survey Data For Bighorn Sheep

We developed a disease risk model using survey and management data for 43 of 52 bighorn sheep herds in Montana from 1979 to 2013 (9 herds were not consistently monitored). We selected 1979 as the preliminary year because data from monitoring surveys and pneumonia epizootics were rare prior to that time. We defined a herd as a group of bighorn sheep that generally form a spatially and demographically distinct group (Wells and Richmond 1995). Not all 43 herds were extant in all years; 9 were established after 1979, 1 of which was extirpated after a pneumonia epizootic. Survey data included air and ground observations of bighorn sheep counts, age classifications, and sex classifications collected at intervals that varied from intermittent to annual, depending on the herd. These observations were primarily collected by MFWP (>90% of all years surveyed). Additional observations were collected jointly between MFWP and the Confederated Salish and Kootenai Tribes (CSKT; <3%), by the CSKT (<2%), or in association with the United States Fish and Wildlife Service (<4%), Bureau of Land Management (BLM; <1%), or the University of Montana (<1%; Fralick 1984).

We defined herd-year as 1 July to 30 June following MFWP's definition for a management year, which encompasses a complete reproductive cycle from breeding through lambing. We defined a pneumonia epizootic as a die-off with ≥25% mortality (Young 1994) caused by pneumonia ($n = 22$; Fig. 1) based on data and expertise from herd biologists and disease specialists at the MFWP Wildlife Laboratory. We included mortalities due to culling of symptomatic bighorn sheep during verified pneumonia events (Edwards et al. 2010). Pneumonia was generally confirmed by necropsy and histological examination of lung tissue, culture, and/or pathology reports ($n = 18$). One die-off was attributed to pneumonia based on biologist knowledge and information presented in Enk et al. (2001). When carcasses or biological samples were unavailable from an epizootic event ($n = 3$), pneumonia was determined based on other evidence (drops of ≥25% in survey numbers, numerous reports of symptomatic individuals, reports of carcasses, and detection of *Mycoplasma ovipneumoniae* in survivors the year following the die-offs; Brent Lonner, MFWP, unpublished data). For each herd experiencing a pneumonia epizootic ($n = 18$), we excluded the 3 following herd-years from analysis because most herds continued to experience noticeable mortality rates in the few years immediately following the preliminary epizootic year (MFWP 2010). We also excluded all herd-years following a pneumonia epizootic if a herd was augmented with animals from other herds because the need for augmentation meant that the herd was not recovering well, and the addition of animals confounded mortality rates and signs of recovery from the epizootic ($n = 5$ herds). We excluded herd-years where die-offs were caused by winter storms ($n = 1$) or unknown factors ($n = 2$). As with the 3 herd-years after pneumonia epizootics, we excluded the 3 herd-years following die-offs caused by unknown factors because they may have been pneumonia epizootic events.

Conceivably, pneumonia epizootics could have gone undetected between 1979 and 2013. To address this possibility and separate years with pneumonia epizootics from those without, we calculated percentage change in survey counts between consecutive herd-years for each herd. We classified herd-years as free of pneumonia epizootics by the following criteria: 1) for herds surveyed annually, the herd had grown, declined <25%, or declined ≥25% followed by ≥200% growth the next year; 2) when surveys occurred every 2 years, the herd grew between surveys; and 3) when surveys occurred every 3 years, the herd grew by ≥200% between surveys. When calculating percentage change, we excluded harvested animals, documented vehicle mortalities, and additions and removals due to transplantation to analyze unexplained change only. Out of 1,333 herd-years available, we used 637 ($\bar{x} = 14.8$ herd-yr per herd, SD = 8.65, range = 1–34) for analysis including the 22 herd-years with pneumonia epizootics. Largely because of a lack of survey data, we excluded remaining herd-years from analysis because of uncertainty of whether herd-years could safely be classified as free of epizootics.

Risk Factor Covariates

We selected 10 covariates we hypothesized were predictive of pneumonia epizootics in Montana and for which sufficient data were available. Many covariates were spatial, based on herd distributions, so we obtained agency records and elicited

expert opinion of agency biologists to delineate approximate boundaries of distributions of herds in each herd-year (Conroy and Peterson 2013). We categorized each covariate as a potential risk factor we hypothesized could primarily contribute to 1) risk of exposure to pathogens, 2) risk of spread of pathogens, or 3) susceptibility to pneumonia epizootics (Mitchell et al. 2013).

Risk of exposure to pathogens.—We hypothesized 5 covariates were positively related to risk of pathogen transmission: proximity to number of domestic sheep and goat allotments (Singer et al. 2000*b*, 2001; Monello et al. 2001; Epps et al. 2004; Clifford et al. 2009), amount of private land (Miller et al. 2011, 2012; Wild Sheep Working Group 2012), use of domestic sheep and goats for weed control (Miller et al. 2012, Wild Sheep Working Group 2012), a history of a pneumonia epizootic in the herd or its neighbors (Onderka and Wishart 1984, George et al. 2008, Edwards et al. 2010, Besser et al. 2013), and close proximity to other herds (Onderka and Wishart 1984, Singer et al. 2000*a*, George et al. 2008, Edwards et al. 2010, Besser et al. 2013). We hypothesized that amount of private land would be representative of risk from hobby or commercial farms with domestic sheep or goats, for which data were not available. For each herd, we estimated an area of high risk for pathogen exposure (distribution of the herd plus a 14.5-km buffer from that perimeter; Wild Sheep Working Group 2012) using a geographical information system (GIS; ArcMap 10.1, Environmental Systems Research Institute, Inc., Redlands, CA). For the first 4 covariates, we modeled risk of pathogen exposure within each area of high risk using 1) number of federally managed sheep and goat allotments, 2) percentage of private land, 3) knowledge of the wildlife biologist responsible for the herd regarding the use of domestic sheep or goats for weed control, and 4) history of a pneumonia epizootic in the herd in a previous herd-year, or a current or previous pneumonia epizootic in a neighboring herd within the area of high risk. We calculated average proximity to the 3 closest herds for the covariate of herd proximity.

We interviewed personnel and consulted records of federal and state agencies to gather data on allotments, private land, weed control, neighbor risk, and herd proximity (Table 1). For data on allotments, we interviewed agency personnel and obtained BLM allotment bills from 1988 onward from the Rangeland Administration System (RAS). We obtained associated geospatial data on allotments from each agency and determined the number of allotment boundaries intersected by each area of high risk using a GIS ($\bar{x} = 0.54$, SD = 1.32 for 565 herd-yr with allotment data). For private land, we obtained land ownership data and calculated the amount of private land within each area of high risk using a GIS ($\bar{x} = 25.58$, SD = 14.53%). We obtained weed control data through elicitation of expert opinion of agency biologists (13.97% of herd-yr had known weed control; Conroy and Peterson 2013). We obtained neighbor risk and herd proximity data through agency records and elicitation of expert opinion of agency biologists. For neighbor risk, when a herd experienced a pneumonia epizootic we assumed neighboring herds were at risk for that and subsequent herd-years. We also assumed a recurring risk to the initial herd in all subsequent herd-years (19.31% of herd-yr had neighbor risk). For herd proximity, we calculated the shortest distance to the perimeters of the distributions of the nearest 3 bighorn sheep herds using a GIS and then calculated the average of those distances (global $\bar{x} = 22.65$ km, SD = 24.27 km). We considered distributions from all herds (including the 9 in Montana excluded from our primary analysis and several herds in British Columbia, Idaho, and Wyoming) for our covariates of neighbor risk if they were within the area of high risk and herd proximity if they were 1 of the 3 closest herds to any of our 43 primary herds.

Risk of spread of pathogens.—We hypothesized high ram: ewe ratios represented increased risk of rams wandering,

Table 1. Data types and associated agencies we collected covariate data from to model risk of pneumonia epizootics for 43 herds of bighorn sheep in Montana from 1979–2013. Numbers represent the approximate percentage of data associated with each agency out of all herd-years with data for that covariate, unless otherwise indicated. Where applicable, we included additional herds beyond our 43 primary herds if they were within 14.5-km of our primary herds or were 1 of the 3 closest herds. Agencies were Montana Fish, Wildlife and Parks (MFWP), United States Fish and Wildlife Service (USFWS), Bureau of Land Management (BLM), United States Forest Service (USFS), National Park Service (NPS), Confederated Salish and Kootenai Tribes (CSKT), Chippewa Cree Tribe (CCT), British Columbia Fish and Wildlife Branch (BCFW), Idaho Fish and Game (IDFG), and Wyoming Game and Fish (WGFD). Blank cells indicate data were not associated with these agencies.

Data	MFWP	USFWS	BLM	USFS	NPS	CSKT	CCT	BCFW	IDFG	WGFD
Allotments[a]		0[b]	68	32						
Private land			100							
Weed control	94	5				1				
Neighbor risk[c]	75	2			5	4		4	5	5
Herd proximity[d]	72	2			5	3	2	5	7	5
Ram:ewe ratios	93	6				1				
Density	94	5				1				
Herd origin	94	5				1				

[a] Of unique allotments ≤14.5 km of herd distributions ($n = 47$), % associated with each agency.
[b] No allotments on USFWS land were ≤14.5 km of herd distributions.
[c] Of all herds ≤14.5 km from 43 primary herds ($n = 56$, including 13 non-primary herds), % associated with each agency.
[d] Of all herds that were 1 of 3 closest to 43 primary herds ($n = 61$, including 18 non-primary herds), % associated with each agency. Sum >100 is due to rounding.

encountering, and spreading pathogens (Onderka and Wishart 1984, Singer et al. 2000*a*, Monello et al. 2001, George et al. 2008, Besser et al. 2013), and that higher relative density increased risk through greater rates of spread of pathogens (Monello et al. 2001, Lafferty and Gerber 2002, Clifford et al. 2009). We obtained herd survey data from the Montana Bighorn Sheep Conservation Strategy (MFWP 2010) and directly from biologists (Table 1). For ram:ewe ratios ($\bar{x} = 0.65$, SD = 0.39), we excluded ratios from analysis where <80% of observed animals were classified by sex, recorded ratios did not match adults counted, or <1 ram or ewe was counted ($n = 50$ excluded ratios associated with included herd-yr). To estimate herd density in each year, we divided the total number of animals counted by the area of the herd's distribution. We then calculated average density, yearly percentage of average density, and the range in percentage of average density for each herd. We assigned each herd's density estimate into 3 equally sized bins of low, medium, and high based on the percentage of average density relative to their 1979–2013 range. Thus, each set of cut-offs were herd-specific, based on historical densities of each herd ($\bar{x}$ cut-off for low density ≤92.15% of average, SD = 13.15; $\bar{x}$ cut-off for medium density ≤151.11% of average, SD = 31.02; 43.80% herd-yr had low density, 36.42% medium, and 19.78% high). When density estimates were not available for years without pneumonia epizootics, we excluded those herd-years from analysis ($n = 65$ of excluded herd-yr). When density estimates were unavailable for years with pneumonia epizootics ($n = 3$), we used the most recent density estimate prior to the epizootic ($n = 2$), or estimated density based on reports of percent declines ($n = 1$). We used a 1-year lag for both covariates because surveys were usually done in spring and thus represented the minimum number of animals likely to be present at the start of the following herd-year.

Susceptibility to pneumonia epizootics.—We hypothesized that relatively harsh winters contributed to susceptibility to pneumonia epizootics by draining energy budgets (Goodson et al. 1991, Monello et al. 2001, Butler et al. 2013). We used percentage of 30-year normal precipitation to represent winter severity. We hypothesized that relatively dry springs contributed to susceptibility to pneumonia epizootics by decreasing forage quality (Portier et al. 1998, Enk et al. 2001, Monello et al. 2001, Epps et al. 2004) and used percentage of 30-year normal precipitation to represent dry spring conditions. Lastly, we hypothesized that mixed (i.e., native herds augmented with animals from other populations) or non-native (reintroduced) herds had increased susceptibility to pneumonia epizootics because these sites might be more risky if conditions that contributed to a previous herd reduction or extirpation persisted in the area (Monello et al. 2001). For winter and spring precipitation, we calculated percentage of normal precipitation using a GIS to determine monthly PRISM precipitation values and 1980–2010 Normals (PRISM Climate Group, Corvallis, Oregon) in each delineated herd distribution (winter $\bar{x} = 98.68\%$, SD = 30.16%; spring $\bar{x} = 100.18\%$, SD = 26.97%). Similar to Butler et al. (2013) but because spring lambing season began in April in some herds, we considered winter to be 1 November–31 March, and spring 1 April–30 June. We used a 1-year lag for both effects to capture the influence of the most recent winter and spring on the next herd-year (Portier et al. 1998, Butler et al. 2013). For herd origin, we obtained agency transplant records (Table 1) to determine in each herd-year if herds were native (21.82% of herd-yr), mixed (20.25%), or reintroduced (57.93%).

Development of Risk Model

Analysis of competing models.—We developed 30 a priori models to test how our hypothesized risk factors predicted pneumonia epizootics. We analyzed the models in a Bayesian framework to allow for modeling of missing values and associated uncertainty and to simplify the use of herd-level random effects due to repeated measurements (Kéry 2010). We centered and scaled covariate data and tested for correlations between continuous covariates; we did not include covariates with >40% correlation in the same model (Dormann et al. 2013). We used JAGS (Version 3.3.0, http://mcmc-jags.sourceforge.net, accessed 14 Mar 2013) called through R (Version 2.13.1, www.r-project.org, accessed 10 Sep 2011) using the package R2jags (Version 0.02-17, http://CRAN.R-project.org/package = R2jags, accessed 14 Mar 2013) to run the logistic regression models (Hosmer and Lemeshow 2000) from these data, with repeated measures and a random effect for herd (Gelman and Hill 2007, Royle and Dorazio 2008, Kéry 2010). We used vague, uniform priors for all parameters (Link et al. 2002). We modeled missing values for ram:ewe ratios ($n = 84$) and number of domestic sheep and goat allotments ($n = 72$) by setting priors equal to the herd mean where available or the global mean otherwise. We ran 100,000 Markov chain Monte Carlo (MCMC) iterations with 3 chains, discarding the first 25,000 iterations as burn-in (Link et al. 2002). We evaluated convergence of the MCMC simulation with the Gelman and Rubin convergence diagnostic ($\hat{R}$; Brooks and Gelman 1998) and visual inspection of the posteriors and chains for mixing (Link and Barker 2010) to ensure convergence for accurate estimates of parameters.

We identified top models based on Deviance Information Criterion (DIC; Spiegelhalter et al. 2002). We excluded models >10 ΔDIC from further consideration. We considered covariates within each model to be fully supported if the 95% credibility interval posterior densities (CRIs; Kéry 2010) did not include 0. Where 95% CRIs included 0, we identified the broadest CRI that would exclude 0 to investigate uncertainty of the covariate.

We used a spreadsheet to calculate probability of a pneumonia epizootic for each herd using the parameter estimates from the top models and covariate data from each herd. The risk model provided probability of a pneumonia epizootic in any given year. We calculated probability of ≥1 epizootic occurring in the next 10 years as $(1 - (1 - \Pr(\text{Epizootic}_{1\text{-yr}}))^{10})$ (Mood et al. 1974).

Assessment of model fit and usefulness.—We used decision curve analysis (DCA; Vickers and Elkin 2006, Steyerberg et al. 2010) to compare net benefits of the top models (<10

Table 2. Parameter estimates of supported a priori models of risk of pneumonia epizootics for 43 herds of bighorn sheep in Montana from 1979–2013. We do not present models with change in Deviance Information Criterion (ΔDIC) >10. Within the distribution of each herd plus a 14.5-km buffer from that perimeter, private land = percentage of private land, weed control = whether the herd biologist knew of the use of domestic sheep or goats for weed control, and neighbor risk = whether the herd or a neighboring herd had a pneumonia epizootic previously. Density = the number of individuals counted divided by the area of each herd's distribution, assigned into 1 of 3 equally sized bins of low, medium (md), and high (hi) density relative to the herd's 1979–2013 percentage of average. Herd effect is the among-herd variation for the herd-level random effect.

			Credibility interval	
	Mean	**SD**	**0.025**	**0.975**
Best model				
β_0 Intercept	−6.269	0.761	−7.931	−4.911
β_1 Private land	0.433	0.239	−0.028	0.910
β_2 Weed control	1.210	0.547	0.115	2.261
β_3 Neighbor risk	2.331	0.524	1.332	3.392
β_4 Density (md)	1.660	0.728	0.309	3.180
β_5 Density (hi)	2.699	0.742	1.332	4.259
Herd effect	0.242	0.131	0.143	0.609
Deviance	153.624	4.125	146.679	162.973
Second model (ΔDIC = 6.9)				
β_0 Intercept	−5.705	0.709	−7.246	−4.445
β_1 Neighbor risk	2.184	0.488	1.244	3.164
β_2 Density (md)	1.535	0.731	0.200	3.085
β_3 Density (hi)	2.548	0.731	1.206	4.090
Herd effect	0.249	0.147	0.143	0.666
Deviance	161.519	3.874	154.019	169.736

ΔDIC) to estimate fit of each model to the data and usefulness of the model. This method allowed assessment of whether the top models were useful compared to totally reactive (i.e., treat all herds as low risk) or totally proactive (i.e., treat all herds as high risk) management of all herds, and the relative consequences of wrong predictions, which is important because a false negative prediction is arguably more harmful for conservation and public enjoyment of bighorn sheep than a false positive prediction. For each model, risk of a pneumonia epizootic could be classified as high if it exceeded a pre-defined threshold probability (p_t). We evaluated a range of p_t (0 to the value of the max. predicted probability of pneumonia epizootic for the 637 herd-yr from each model) for which we calculated sensitivity, specificity, and net benefits,

$$\text{net benefit}_{\text{model}} = (\text{true positive count}/n) - (\text{false positive count}/n) \times p_t/(1-p_t)$$

to estimate and summarize performance and advantages of the model, where $n = 637$. Weighting by the ratio $p_t/(1-p_t)$ accounts for the harm of false positive predictions to harm of false negative predictions at each p_t. For each model, we plotted decision curves of the net benefits across values of p_t to identify the best model that tended to have higher net benefits than the others.

Finally, we determined if the best model was more useful than abandoning the model and instead managing all herds as low risk, which is a management option in absence of a predictive model. We calculated the model advantage across the range of p_t over the option of assuming all herds are low risk as:

$$\text{net increase in true positives} = \text{net benefit}_{\text{model}} \times 100$$

This measure of the model's usefulness calculates the increase in true positives with no increase in false positive per 100 estimates compared to treating all herds as low risk. Similarly, the model advantage across the range of p_t over the option of assuming all herds are high risk is:

$$\text{net reduction in false positives} = \frac{(\text{net benefit}_{\text{model}} - \text{net benefit}_{\text{all high}}) \times 100}{p_t/(1-p_t)}$$

The net reduction in false positives is the reduction of false positives per 100 estimates provided by the risk model without increasing the number of false negatives compared to abandoning the model and treating all herds as high risk. Here, net $\text{benefit}_{\text{all high}}$ is calculated with the net $\text{benefit}_{\text{model}}$ formula except true positive count is the total number of pneumonia epizootic cases (22) and false positive count the total non-pneumonia epizootic cases (615).

Second generation model.—We developed an a posteriori, second generation model by calculating the inclusion probability of each covariate. Inclusion probabilities resulted from introducing a Bernoulli distributed indicator variable with probability equal to 0.5 (Ntzoufras 2009). We ran 3 chains for 500,000 iterations, discarding the first 125,000 iterations as burn-in (Link et al. 2002). We calculated the proportion of times each indicator variable assumed a value of 1 and identified covariates with inclusion probabilities >0.15 (similar to Ntzoufras 2009). We then evaluated a new second generation model with these covariates using the techniques described above for analysis of competing models.

RESULTS

Development of Risk Model

The top-ranked model included private land, weed control, neighbor risk, and density (Table 2). The posterior density CRIs excluded 0 except for private land (95%

CRI, $-0.03 \leq x \leq 0.91$), but a 93% CRI for private land excluded 0 ($0.01 \leq x \leq 0.87$). The second best model included neighbor risk and density ($\Delta DIC = 6.9$). Smooth unimodal posteriors, history plots (Link and Barker 2010), and $\hat{R}$ values of <1.1 indicated convergence (Brooks and Gelman 1998). All other models had $\Delta DIC > 10$, so we excluded them from further consideration.

The top-ranked model was superior to the second-ranked model based on sensitivity, specificity, and net benefits. Sensitivity and specificity were simultaneously maximized for the top model at a p_t of 0.0312, achieving 81.8% sensitivity, 80.2% specificity, and a correct overall classification rate of 80.2% (Fig. 2). Sensitivity and specificity for the second best model were simultaneously maximized at a p_t of 0.0288 with 81.8% sensitivity, 75.3% specificity, and 75.5% correct overall classification rate. We selected the top model as the final risk model because it had a higher overall net benefit than the second model across most p_t's (Fig. 3).

Based on DCA, over a wide range of p_t the final risk model was superior to the 2 alternative options of treating all herds reactively or proactively in absence of a predictive model. The risk model's decision curve had higher net benefits than the decision curve for the alternative of treating all herds as high risk at a p_t of approximately $\geq$0.001 (Fig. 3). The risk model's decision curve was also higher than the decision curve for treating all as low risk at a p_t of approximately $\leq$0.389. Between 0.001–0.389, the risk model would therefore provide both a net reduction in false positive estimates over assuming all herds are high risk and a net increase in true positives over assuming all herds are low risk. Using the risk model with any p_t between these levels would be better than fully reactive management or the alternative of total proactive management of all herds, considering limited resources. It is therefore useful as a model for predicting risk of pneumonia epizootics at any p_t within this range. The model would yield fewer false negative predictions at low values of p_t and fewer false positive predictions at high values of p_t (Table 3).

Effect Sizes for Top Model

Parameters in the risk model provide estimated effects of each risk factor on probability of a pneumonia epizootic. Holding other parameters constant, the odds of a pneumonia epizootic increased 1.54 (95% CRI, $0.97 \leq x \leq 2.48$) times per additional unit of private land within the area of high risk (global $\bar{x} = 25.58\%$, SD = 14.53%). Herds where domestic sheep or goats were known to be used to control weeds within the area of high risk that year had 3.35 (95% CRI, $1.12 \leq x \leq 9.59$) times greater odds of a pneumonia epizootic than those without. Odds of a pneumonia epizootic were 10.29 (95% CRI, $3.79 \leq x \leq 29.73$) times greater for herds if they or their neighbors in the area of high risk previously experienced a pneumonia epizootic. Herds at medium or high density had odds of a pneumonia epizootic 5.26 (95% CRI, $1.36 \leq x \leq 24.05$) and 14.86 (95% CRI, $3.79 \leq x \leq 70.74$) times greater, respectively, than when they were at low density. Altogether, a herd with no private land, weed control, or neighbor risk and with low density was estimated to have 0.0009 (95% CRI, $0.0001 \leq x \leq 0.0045$) probability of a pneumonia epizootic during any year and represents the least risky extreme. On the most risky extreme, a herd in an area of high risk with 100% private land, weed control,

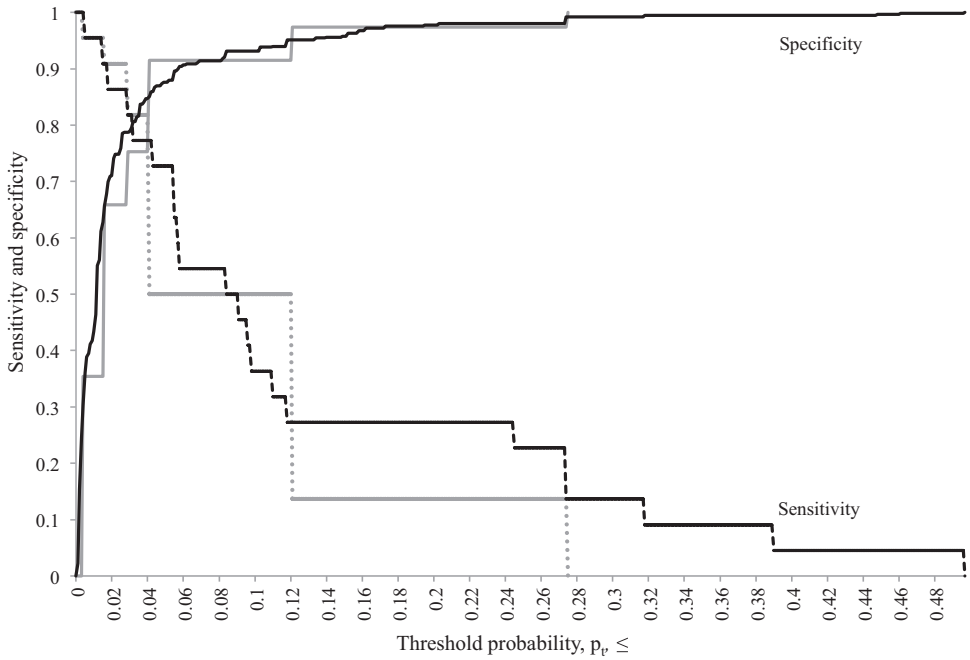


Figure 2. Sensitivity (dashed lines) and specificity (solid lines) at various threshold probabilities (p_t's) for 2 pneumonia risk models developed using data from 1979–2013 for bighorn sheep in Montana. The top-ranked model (black lines) had a higher sensitivity and specificity than the second-ranked model (gray lines): at $p_t = 0.0312$ sensitivity and specificity were simultaneously maximized with 81.8% sensitivity and 80.2% specificity compared to the second-ranked model which had the same sensitivity and 75.3% specificity at $p_t = 0.0288$.

Figure 3. Decision curves for 2 final a priori models considered for selection as a pneumonia risk model for bighorn sheep in Montana. The most supported model (black line) outperformed the second-best model (gray line) over much of the threshold probability (p_t) range based on the higher net benefit overall. We selected the most supported model for the risk model. Using the risk model would be superior to treating all herds as high risk (dotted line; i.e., indiscriminate proactive management of all herds) at any threshold probability at any p_t of approximately $\geq$0.001, and better than treating no herds as high risk (dashed line at net benefit = 0; i.e., reactive management of all herds) at any p_t approximately $\leq$0.389.

Table 3. Comparison of net benefits and advantages for our pneumonia risk model for 43 herds of bighorn sheep in Montana from 1979–2013. Risk of a pneumonia epizootic is classified as high if it exceeds a pre-defined threshold probability (p_t), and low otherwise. The net benefit at each threshold estimates the advantage of the model and can aid selection in p_t for more conservative or liberal estimation based on tolerance of false positives versus false negatives.

			Net benefit		Advantage of model	
$p_t \leq$	Sensitivity	Specificity	Risk model	Treat all[a]	Increase in TP[b]	Decrease in FP[c]
0.000	1.000	0.000	0.035	0.035	3.454	0.000
0.004	1.000	0.302	0.032	0.031	3.183	29.199
0.008	0.955	0.411	0.028	0.027	2.838	20.251
0.012	0.955	0.551	0.028	0.023	2.770	40.293
0.016	0.909	0.657	0.026	0.019	2.601	44.113
0.020	0.864	0.711	0.024	0.015	2.412	45.526
0.024	0.864	0.748	0.024	0.011	2.384	53.061
0.028	0.864	0.787	0.024	0.007	2.390	59.632
0.032	0.773	0.807	0.021	0.003	2.051	54.121
0.036	0.773	0.837	0.021	−0.002	2.083	59.829
0.040	0.773	0.847	0.021	−0.006	2.054	62.951
0.050	0.727	0.876	0.019	−0.016	1.884	66.719
0.060	0.545	0.907	0.013	−0.027	1.313	63.004
0.070	0.545	0.914	0.013	−0.038	1.258	67.369
0.080	0.545	0.914	0.012	−0.049	1.160	70.173
0.090	0.500	0.932	0.011	−0.061	1.075	72.493
0.100	0.364	0.932	0.005	−0.073	0.523	70.173
0.200	0.273	0.977	0.004	−0.207	0.392	84.301
0.300	0.136	0.992	0.001	−0.379	0.135	88.802

[a] Net benefits for treat all herds as high risk, a management alternative in absence of using our risk model to predict and separate high from low risk herds.
[b] Increase in true positives per 100 estimates without increase in false positives compared to treating all herds as low risk.
[c] Reduction in false positives per 100 estimates without increase in false negatives compared to treating all herds as high risk.

neighbor risk, and high density was estimated to have 0.8992 (95% CRI, $0.4256 \leq x \leq 0.9910$) annual probability of a pneumonia epizootic.

Second Generation Model

Inclusion probabilities were >0.15 for private land, weed control, neighbor risk, and density, which aligns with the top model we developed a priori. A fifth and final covariate with >0.15 inclusion probability was spring precipitation. An a posteriori model with these 5 covariates had a DIC of 4 lower than that of our original best model, indicating greater support for the new model. Parameter estimates of the original 4 risk factors were very similar (Tables 2 and 4).

Spring precipitation was negatively correlated with probability of a pneumonia epizootic the next herd-year (starting 1 Jul). Holding other parameters constant, odds of a pneumonia epizootic were 0.41 (95% CRI, $0.20 \leq x \leq 0.78$) times that of years of average spring precipitation per standardized unit increase ($\bar{x} = 100.18\%$, SD = 26.97%). Thus, each increase of 27% from average precipitation was associated with less than half the odds of a pneumonia epizootic compared to years with average spring precipitation. Conversely, for each unit decrease in spring rainfall, risk of a pneumonia epizootic more than doubled.

DISCUSSION

Historically, state wildlife agencies have managed pneumonia epizootics in bighorn sheep largely reactively because they have not had the ability to predict epizootics. Existing models related to pneumonia in bighorn sheep focus largely on predicting consequences of epizootics (e.g., mortality rates and population persistence). Our model was designed to predict the risk of pneumonia epizootics before they happen, which no other model has directly done before (although see Clifford et al. [2009] and Carpenter et al. [2014] for models of disease transmission from allotments). If probability of epizootics cannot be predicted, herds cannot be separated by high and low risk to proactively prevent pneumonia epizootics. Proactively treating all herds as high risk would likely be prohibitively expensive, resulting in the general reactive management status quo.

A more proactive approach integrating wildlife health with wildlife conservation would lead to more effective conservation and management of wildlife populations (Deem et al. 2001). For more proactive management of pneumonia epizootics in bighorn sheep, agencies need risk assessment tools to better understand risk factors that contribute to pneumonia epizootics. They also need to know how to use available data to predict pneumonia epizootics. Models based on more limited temporal and spatial extents may make more precise estimates on such scales, but lose generality across larger ones. A general model that combines information from herds across a state would aid in prediction of risk at the necessary scale for state wildlife agencies to make decisions on how to allocate resources for proactive management. Accordingly, we analyzed epizootic histories and potential risk factors for 43 herds across Montana from 1979 to 2013 to create a statewide risk model for pneumonia.

Risk Factors

Risk of pneumonia epizootics was positively associated with greater amount of private land, weed control with domestic sheep and goats, history of a pneumonia epizootic in a herd or a nearby herd, and higher density. Based on our second generation model, risk also appeared to be associated with spring precipitation. Risk was not associated with number of allotments, herd proximity, ram:ewe ratios, winter precipitation, or herd origin, nor did a single risk factor affect all pneumonia epizootics based on our multivariate model. Although the existence of a single risk factor that we did not evaluate cannot be ruled out, our results agree with the findings of Miller et al. (2012) in their review of hypothesized risk factors of die-offs in bighorn sheep. They failed to find evidence of a single etiological agent and concluded that predictive models of epizootics are needed based on the likely complexity of the etiology of such outbreaks.

Risk of exposure to pathogens.—As we hypothesized, greater percentage of private land in and near areas used by herds

Table 4. Parameter estimates of the second generation model for risk of pneumonia epizootics for 43 herds of bighorn sheep in Montana from 1979 to 2013. The Deviance Information Criterion (DIC) of our second generation model was 4 lower than that of our top-ranked a priori model. Within the distribution of each herd plus a 14.5-km buffer from that perimeter, private land = percentage of private land, weed control = whether the herd biologist knew of the use of domestic sheep or goats for weed control, and neighbor risk = whether the herd or a neighboring herd had a pneumonia epizootic previously. Density = the number of individuals counted divided by the area of each herd's distribution, assigned into 1 of 3 equally sized bins of low, medium (md), and high (hi) density relative to the herd's 1979–2013 percentage of average. Spring = the percentage of average 1 April–30 June precipitation in the herd distribution compared to the average from 1980 to 2010. Herd effect is the among-herd variation for the herd-level random effect.

			Credibility interval	
	Mean	SD	0.025	0.975
Second generation model				
β_0 Intercept	−6.856	0.935	−8.925	−5.288
β_1 Private land	0.487	0.256	−0.002	1.005
β_2 Weed control	1.300	0.577	0.144	2.409
β_3 Neighbor risk	2.474	0.549	1.426	3.583
β_4 Density(md)	1.876	0.809	0.447	3.633
β_5 Density(hi)	3.066	0.843	1.577	4.884
β_6 Spring	−0.882	0.342	−1.587	−0.244
Herd effect	0.250	0.149	0.143	0.676
Deviance	147.583	4.593	139.739	157.825

of bighorn sheep was associated with increased risk of pneumonia epizootics by >1.5-fold per additional unit of private land. Risk associated with contact with domestic livestock on private land has not previously been quantified and tends to be neglected (Miller et al. 2011, 2012), perhaps because data on locations of hobby and commercial farms are generally unavailable and would be highly fluid through time. Exposure to sheep or goats may occur on farms on private lands, whereas exposure on public lands likely occurs primarily on allotments, for which data exist and which agencies can more directly manage. Although risk due to private land was slightly uncertain (the 95% CRI contained 0, however the 93% CRI did not), these results provide the first empirical support for the suggestions of Miller et al. (2011, 2012) and the Wild Sheep Working Group (2012) that risk of exposure to pathogens on private land should receive more focus and concern. The uncertainty of this parameter at the 95% CRI is likely due to the probably low correlation between private land and farms with domestic sheep and goats, because not every parcel of private land contains domestic Caprinae species. Were data available, the effect of commercial and hobby farms could likely be estimated more precisely, yet the readily available percentage of private land was still predictive of risk. Examples of management actions to reduce risk associated with private land might include public education on separation of bighorn sheep and domestic sheep and goats, removal of wandering bighorn sheep in proximity to farms with domestic sheep or goats (Mitchell et al. 2013), or purchasing conservation easements (Sells 2014). We note that the association between private land and pneumonia epizootics could also be related to high human densities or human disturbance (e.g., development) on some areas of private land. Such disturbances could increase stress and potentially predispose herds to pneumonia epizootics.

Our hypothesis that risk of pneumonia epizootics increases when domestic sheep and goats are used for weed control in or near areas occupied by bighorn sheep herds was supported, with a >3.3-fold increase in risk compared to areas or years without known weed control using domestic Caprinae species. To our knowledge, our results are the first to support the suggestion by Miller et al. (2012) and the Wild Sheep Working Group (2012) that such operations increase risk of pathogen exposure. Potential management actions to mitigate this risk include public education about separation between bighorn sheep and domestic sheep and goats (Mitchell et al. 2013), managing timing of grazing to avoid temporal overlap with bighorn sheep, or using other methods to control weeds that do not involve domestic sheep or goats (Sells 2014).

As we hypothesized, risk of pneumonia epizootics increased for a herd when that herd or a nearby herd within 14.5 km had a history of a pneumonia epizootic. Increased risk for a herd after an epizootic is intuitive. Evidence suggests that pathogens become endemic and may cycle for years to decades within herds (Enk et al. 2001, Cassirer and Sinclair 2007, Cassirer et al. 2013). Further evidence suggests that whereas ewes may develop temporary protective immunity, this may wane after exposure to pathogens and does not effectively transfer to lambs, leading to ongoing outbreaks of pneumonia (Plowright et al. 2013). Additionally, Plowright et al. (2013) found that translocated, naïve adults appear to be at particularly high risk of dying from pneumonia. We hypothesized that other naïve individuals in nearby herds may be at a similar risk of contracting pneumonia. Whereas the exposure and spread of pathogens to nearby herds has been hypothesized to contribute to epizootics (Onderka and Wishart 1984, George et al. 2008, Edwards et al. 2010), this risk has not been quantified or received as much focus as other hypothesized risk factors. We found that a pneumonia epizootic was associated with >10-fold risk of pneumonia epizootics for all herds within 14.5 km. Cassirer et al. (2013) reported a slight but uncertain increase in probability of pneumonia for neighboring populations located <20 km apart if a neighbor had any pneumonia mortalities that or the previous year. The reason for this difference may be attributable to an inclusion of short timeframes with all cases of pneumonia as opposed to our use of longer timeframes with high-mortality epizootics. We included histories of epizootics from 1979 to the end of the study given the evidence that pathogens can cycle for decades (Enk et al. 2001, Cassirer et al. 2013). We included only high-mortality epizootics because we hypothesized that pneumonia widely spread in a herd would be linked to more potential exposure between herds (Onderka and Wishart 1984, George et al. 2008, Edwards et al. 2010, Besser et al. 2013), compared to limited cases of pneumonia that may result in less exposure between herds. Thus, across broad temporal and spatial scales, we conclude that pneumonia epizootics have long-term consequences for herds experiencing epizootics and for neighboring herds as well. Potential actions that may reduce this risk could include creating lethal removal zones between infected and naïve herds, culling symptomatic individuals, and avoiding establishing new herds close to those with epizootic histories (Sells 2014). Additionally, we note that past epizootics in or near a herd could be predictive of future epizootics because of shared or recurring conditions in an area besides pathogens (e.g., environmental factors) that could make herds more susceptible to pneumonia epizootics.

Our other hypothesis that proximity to other herds, measured by Euclidean distance, increased risk of pathogen exposure was not supported. The global mean for average proximity to the 3 closest herds (22.65 km, SD = 24.27 km) was >1.56 times farther and highly variable compared to the maximum distance for those herds we considered neighbors (within 14.5 km). Although bighorn sheep are known to move distances comparable to our mean herd proximity (e.g., O'Brien et al. [2014] reported that >10% of rams forayed ≥21.7 km from core herd home ranges each summer), this does not mean they will necessarily come in contact with other herds. By not accounting for barriers to movement, Euclidean distance may misrepresent distances that bighorn sheep would actually travel between herds, particularly at greater distances. Additionally, average distance to the 3 closest herds did not account for epizootic histories, whereas

our identified risk factor of neighboring herds with epizootic histories did. The hypothesis Cassirer et al. (2013) tested for distance to nearest herd with recent cases of pneumonia also allowed for herds at much greater distances ($\leq$70 km) and did not have support. Risk therefore appears to be associated with relatively close neighboring herds with histories of pneumonia epizootics, not to Euclidean distance to herds in general.

Proximity to greater number of allotments was not predictive of pneumonia epizootics, contrary to results reported by other researchers. Monello et al. (2001) reported that herds with pneumonia were closer to domestic sheep allotments than were herds without pneumonia. In their analysis, they included allotments at much greater distances compared to our area of high risk. Clifford et al. (2009) estimated risk of pathogen transmission was higher where strong overlap existed between allotments and known bighorn sheep movements. Our result is counter-intuitive because pneumonia in bighorn sheep is strongly associated with exposure to domestic sheep and goats (Wehausen et al. 2011), which is presumably more likely on allotments. In Montana, however, mean number of allotments within 14.5 km of herds was only 0.54 per herd-year (SD = 1.32). Of herd-years with $\geq$1 allotment (n = 134), mean number was 2.29 allotments (SD = 1.83, max. = 14). Only 14 of the 43 herds were within 14.5 km of allotments with sheep or goats for at least 1 year between 1979 and 2013; of these herds, only 4 had pneumonia epizootics. Simple presence or absence of allotments within 14.5 km was not predictive of epizootics upon further investigation, either. For herds that are close to allotments, exposure may further depend on numerous factors unique to each allotment, including how they are managed (e.g., timing of grazing, management of strays). It may also depend on the degree of actual overlap between species as suggested by Clifford et al. (2009), for which we had no data commensurate with the large spatial and temporal scales at which we worked. We suggest further, more detailed evaluation of how allotments might contribute to risk of pneumonia epizootics is needed before discarding allotments as a potentially predictive risk factor for future models.

Risk of spread of pathogens.—Our hypothesis that relative density within a herd is associated with increased risk of a pneumonia epizootic was supported, lending empirical support to the hypotheses of other researchers (Miller et al. 1991, Monello et al. 2001, Clifford et al. 2009). Risk of a pneumonia epizootic increased >5-fold when herds were at medium density and nearly 15-fold when herds were at high density compared to when they were at low density. Substantial herd variation (e.g., habitat quality and estimated area used by each herd) yielded incomparable absolute densities between herds, so we defined density as relatively low, medium, or high. More analysis on density would be useful in the future, including what absolute values might lead to higher risk of pneumonia epizootics, or if group aggregation size is predictive. Density is a component of risk that has previously received little attention because the positive association between risk of pneumonia and higher densities had not been quantified. The association between higher herd density and risk may appear to contradict the idea that herds of larger population size should be less threatened by extirpation than smaller herds (Woodroffe 1999, Singer et al. 2001, Cassaigne et al. 2010). Rather than reducing herd size only, expanding the distribution of an existing herd (e.g., through habitat improvements that attract animals to new areas or, potentially, short-distance transplant operations to unoccupied areas nearby) would also reduce density by increasing the total area that a herd occupies (Sells 2014).

Ram:ewe ratios were not associated with increased risk. We chose these ratios to represent the likelihood that rams would wander in search of breeding opportunities, thus potentially encountering and spreading pathogens. Our results suggest that rams may not be as important vectors of pathogens in their herds as we hypothesized. Rams are known to make long movements (Singer et al. 2000*b*, DeCesare and Pletscher 2006, O'Brien et al. 2014), probably even more so at relatively high densities (Singer et al. 2000*a*, Monello et al. 2001). To increase risk of pneumonia for its herd, however, a wandering ram would have to become infected, survive long enough to come in contact with other herd members, and successfully transmit pathogens. These odds may be independent of ram:ewe ratios alone. Historically, MFWP often removed wandering rams when discovered comingling with domestic sheep or goats, and this management effort may have further reduced risk from wandering rams in specific cases. Additionally, not all age classes of rams may be at greater risk of wandering. The ratio of young rams in a herd may be more predictive of this potential source of risk of spread of pathogens, but these data were only occasionally collected over the years we analyzed.

Susceptibility to pneumonia epizootics.—We used percentage of normal spring (Apr–Jun) precipitation to represent the hypothesized impact of decreased forage quality on susceptibility to pneumonia epizootics but found no relationship during analysis of our a priori models. This suggested that forage quality might not affect risk of pneumonia epizootics, or that percentage of normal spring precipitation may not be a suitable index to forage quality because it does not account for other environmental factors that also affect forage quality (e.g., timing of precipitation and temperature). We think it more likely, however, that this covariate did not have support because no a priori model included it alongside the other identified risk factors. Based on our a posteriori, second generation model, spring precipitation appeared predictive of pneumonia epizootics. Odds of a pneumonia epizootic were reduced by a factor of 0.41 times per unit of spring precipitation beyond average in the previous spring ($\bar{x}$ = 100.18%, SD = 26.97%). Monello et al. (2001) also noted qualitative evidence for a relationship between summer and fall pneumonia outbreaks and lower than average precipitation. The second generation model could be used to predict risk of pneumonia epizootics instead of our a priori risk model; the effect sizes of the other 4 risk factors were comparable, with a largest difference in any parameter estimate of <0.4 (Tables 2 and 4).

We selected percentage of normal winter precipitation to represent the hypothesized impact of harsh winters on susceptibility to pneumonia epizootics because of increased energy expenditures but found no relationship. This result suggests that harsh winters do not increase risk of pneumonia epizootics, consistent with similar results of Monello et al. (2001). Alternatively, percentage of normal winter precipitation may not have been a suitable index for the effects of harsh winters on energy budgets of bighorn sheep because it did not account for patterns and timing of winter precipitation. These factors could be important components of winter severity but related data were unavailable at the scale of our analysis.

Herds in Montana of mixed or reintroduced origin did not have higher risk of pneumonia epizootics than native herds. This finding contrasts with those of Monello et al. (2001) who evaluated a subset of herds throughout North America and hypothesized that sites of previous herd extirpations could continue to be risky for pneumonia based on characteristics of the site itself. If this were the case, reintroduced herds at sites of historical herd extirpations in Montana could have comparable risk to native herds. This

Table 5. Estimates for risk of pneumonia epizootics as of 2012 for 42 herds of bighorn sheep in Montana, calculated with the pneumonia risk model we developed. The 10-year risk is the probability of ≥1 pneumonia epizootic occurring in 10 years if levels of risk factors remain unchanged. Map ID # corresponds to Figure 1. Within the distribution of each herd plus a 14.5-km buffer from that perimeter, private land = percentage of private land, weed control = whether the herd biologist knew of the use of domestic sheep or goats for weed control, and neighbor risk = whether the herd or a neighboring herd had a pneumonia epizootic previously. Density = the number of individuals counted divided by the area of each herd's distribution, assigned into 1 of 3 equally sized bins of low, medium, and high density relative to the herd's 1979–2013 percentage of average. Where density estimates were unavailable for 2012, we used the most recent density before that year.

		Risk factors				Pr(Epizootic)	
Map ID #	Herd name	Private land (%)	Weed control	Neighbor risk	Density	1 yr (2012)	10 yr (beginning 2012)
1	Ten Lakes	21.25	No	Yes	High	0.203	0.897
2	Koocanusa	6.08	No	No	Low	0.001	0.011
3	Kootenai Falls	25.75	No	No	Low	0.002	0.019
4	Berray Mountain	15.06	No	No	Low	0.001	0.014
5	Thompson Falls	34.96	No	No	Low	0.002	0.025
6	Cut-off	30.04	No	No	High	0.031	0.271
7	Perma-Paradise	32.20	No	No	Medium	0.012	0.114
8	Hog Heaven	57.43	No	No	Low	0.005	0.048
9	Wildhorse Island	39.32	No	No	High	0.041	0.340
10	Bison Range	47.81	No	No	High	0.052	0.412
11	Petty Creek	36.79	No	No	High	0.038	0.320
12	Bonner	46.27	Yes	Yes	Low	0.108	0.681
13	Lower Rock Creek	39.75	Yes	Yes	Low	0.091	0.613
14	Upper Rock Creek	29.33	No	Yes	Low	0.021	0.194
15	Skalkaho[a]	34.29	Yes	No	High	0.109	0.685
16	East Fork Bitterroot	10.60	Yes	Yes	Low	0.040	0.336
17	Painted Rocks	6.03	Yes	Yes	Medium	0.161	0.827
18	Garrison	54.37	Yes	Yes	Low	0.134	0.761
19	Lost Creek	35.73	Yes	Yes	Low	0.081	0.571
20	Highland	35.14	No	Yes	Low	0.025	0.226
21	Tendoy Mountains	26.14	No	Yes	Low	0.019	0.178
22	North Fork Birch Creek-Teton	27.24	No	Yes	Low	0.020	0.183
23	Deep Creek	26.66	No	Yes	Low	0.020	0.181
24	Gibson Lake North	6.04	No	Yes	Low	0.011	0.103
25	Castle Reef	34.46	No	Yes	Medium	0.118	0.714
26	Ford Creek	21.81	No	Yes	Medium	0.084	0.584
27	Beartooth-Sleeping Giant	74.67	Yes	Yes	Low	0.220	0.917
28	Elkhorn	51.41	No	Yes	Low	0.040	0.338
29	Spanish Peaks	28.83	No	No	Medium	0.011	0.103
30	Hilgards	14.58	No	Yes	High	0.173	0.850
31	Hyalite[b]	26.86	No	No	Low	0.002	0.019
32	Upper Yellowstone	9.26	No	Yes	High	0.151	0.806
33	Mill Creek	17.63	Yes	No	Medium	0.026	0.229
34	Monument Peak	0.31	No	No	High	0.013	0.123
35	East Yellowstone	0.75	No	No	High	0.013	0.125
36	Stillwater	8.53	No	No	High	0.017	0.155
37	West Rosebud	16.28	No	No	High	0.021	0.190
38	Hellroaring	9.27	Yes	No	Low	0.004	0.038
39	Pryor Mountains	14.26	Yes	No	Low	0.005	0.044
40	Missouri River Breaks	44.91	Yes	No	High	0.144	0.788
41	Little Rockies	31.18	No	No	Low	0.002	0.022
42	Middle Missouri Breaks	24.57	No	No	Low	0.002	0.018

[a] Had epizootic in 2012 and is now positive for neighbor risk, increasing $Pr(Epizootic_{10\text{-}yr})$ after 2012.
[b] Had epizootic in 2013 and is now positive for neighbor risk, increasing $Pr(Epizootic_{10\text{-}yr})$ after 2013.

could be true because MFWP has tried to avoid reintroducing herds near areas with domestic sheep. Alternatively, whereas we defined epizootics as events with ≥25% mortality, Monello et al. (2001) defined all detected pneumonia events as epizootics including those with <10% mortality. A difference in risk for native versus reintroduced herds may have been more pronounced if reintroduced herds were more likely to experience low-mortality pneumonia events. Reintroduced herds might also have been monitored more closely, providing the ability to better detect low-mortality events.

Overall Model

Availability of certain data limited our ability to analyze additional hypothesized risk factors. Most important was the paucity of pathogen data. Presence of *Mycoplasma ovipneumoniae* or *Mannheimia haemolytica* may be important in predicting risk if sufficient data, understanding, and tests for disease agents were available. Although Montana had over 60 herd-years of *Mycoplasma ovipneumoniae* data and nearly 100 herd-years of *Mannheimia haemolytica* data, more intensive, consistent efforts with larger sample sizes would have been needed for our analysis because so many herd-years were still lacking in data. Also, traditional culture-based methods for *Mycoplasma ovipneumoniae* (Besser et al. 2008) and *Mannheimia haemolytica* (Shanthalingam et al. 2014) appear to miss many positive results compared to new culture-independent methods that detect genetic signatures of the pathogen. This suggests that analysis of these data for our study could lead to misleading and erroneous predictions; therefore, we excluded them from analysis. In addition to pathogen data, body condition data such as body fat levels, parasite loads, mineral levels, or blood parameters may also be of potential value in a future risk model (Mitchell et al. 2013).

Evaluating our model's capacity to predict future epizootics in Montana, or those occurring in other states, offers an opportunity to evaluate and improve the model. It would also constitute a test of the hypothesized relationships posed by our model and its covariates, providing an opportunity to learn more about risk factors for pneumonia epizootics. Our evaluation of 10 hypothesized risk factors clarified the importance of poorly understood risk factors in Montana to better predict risk. These risk factors could differ in their relative importance for herds in places unlike Montana. To maximize usefulness of the model, we recommend that potential variation in risk factors should be tested and calibrated to local conditions as part of an adaptive approach to disease management. Alternative risk factors may also be important in other areas and a subject for future research toward development of predictive models elsewhere. The evidence, based on our second generation model, that spring precipitation is predictive of pneumonia epizootics deserves further attention in future work.

The scope and scale of our study required data collected from numerous biologists, literature sources, and other agency personnel. Because misclassification of pneumonia epizootics could reduce precision, we excluded herd-years for which we were not reasonably certain were free of pneumonia epizootics. Accuracy and precision of spatially related covariates would be compromised if biologists were unable to delineate approximate distributions of herds, so we excluded herds without sufficient spatial data due to limited herd histories or biologist knowledge.

The statistically rare nature of pneumonia epizootic events makes their prediction challenging. Pneumonia epizootics occurred in 22 out of 637 (3.45%) of the herd-years we analyzed. A statistical model based on such data has the potential to incorrectly predict epizootics (i.e., false positives) more often than correctly. Our use of decision curve analysis helped evaluate the extent to which managers can rely on our risk model to make accurate predictions, given the number of pneumonia epizootic events we observed. This relatively new analysis determines the net benefits of using a predictive model for making decisions (i.e., its usefulness; Vickers and Elkin 2006, Steyerberg et al. 2010). This assessment first allowed us to conclude that our top model was more useful than our second model. It also allowed us to evaluate whether using our model to make a decision was more useful than using no model at all. If no model such as ours existed, the status quo decision would generally be reactive management (i.e., treat all herds as low risk) because herds cannot be distinguished by risk level and proactive management of all herds would almost certainly be too costly. To be useful, our predictive model should provide more correct classifications than either alternative in absence of the model.

Decision curve analysis showed that our model is expected to be more useful than the status quo. For example, at a threshold probability of 0.028, our model is expected to provide a net increase in true positive detections of 2.390 per 100 herd-years compared to total reactive management. It would also provide a net reduction in false positive detections of 59.632 per 100 herd-years compared to total proactive management, meaning our model would reduce false positive predictions by 60% over completely proactive management. Thus, many more correct classifications will be provided by our model compared to fully reactive management or fully proactive management of all herds. This ability to reliably differentiate herds by risk level will assist managers in making decisions on where to direct appropriate, potentially costly proactive actions.

An important advantage of DCA is that tolerance for false positive versus false negative predictions can be accounted for by selecting different threshold probabilities. Individual managers will have different risk tolerances when making decisions. Some managers will be more risk averse given the severe implications of pneumonia epizootics. More risk-averse managers could select a lower threshold probability to separate high from low risk herds. Other managers may be more risk tolerant if management actions would be too costly, in which case they could then select a higher threshold probability.

MANAGEMENT IMPLICATIONS

Our model can be used to estimate risk (Table 5), compare and prioritize herds for proactive management, and simulate how potential alternative actions may reduce risk. The model

is not only useful for predicting risk for existing herds, but for estimating future risk for new transplant herds as well. Our approach and results are unique because of the extensive spatial and temporal scales used to develop the risk model and make it valuable for herd-specific decisions as part of regional or statewide management of bighorn sheep in Montana. Used to inform decisions in a structured decision making framework (Mitchell et al. 2013), the model can be used to estimate herd-specific recommendations that best meet agency objectives given each herd's predicted risk. Importantly, sophisticated software is not required; a simple spreadsheet can be used to calculate risk using the parameter estimates from the risk model (Table 2). A spreadsheet for a decision model similar to that shown in Mitchell et al. (2013) would help managers use the risk model to inform decisions. Use of both models will lead to a unified, transparent, and consistent approach to making proactive management decisions given the regional or statewide scale, while simultaneously remaining highly specific to each herd's estimated risk and each manager's goals.

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LITERATURE CITED

Besser, T. E., E. F. Cassirer, M. A. Highland, P. Wolff, A. Justice-Allen, K. Mansfield, M. A. Davis, and W. Foreyt. 2013. Bighorn sheep pneumonia: sorting out the cause of a polymicrobial disease. Preventative Veterinary Medicine 108:85–93.

Besser, T. E., E. F. Cassirer, K. A. Potter, J. VanderSchalie, A. Fischer, D. P. Knowles, D. R. Herndon, F. R. Rurangirwa, G. C. Weiser, and S. Srikumaran. 2008. Association of *Mycoplasma ovipneumoniae* infection with population-limiting respiratory disease in free-ranging Rocky Mountain bighorn sheep (*Ovis canadensis canadensis*). Journal of Clinical Microbiology 46:423–430.

Besser, T. E., E. F. Cassirer, C. Yamada, K. A. Potter, C. Herndon, W. J. Foreyt, D. P. Knowles, and S. Srikumaran. 2012*a*. Survival of bighorn sheep (*Ovis canadensis*) commingled with domestic sheep (*Ovis aries*) in the absence of *Mycoplasma ovipneumoniae*. Journal of Wildlife Diseases 48:168–172.

Besser, T. E., M. A. Highland, K. Baker, E. F. Cassirer, N. J. Anderson, J. M. Ramsey, K. Mansfield, D. L. Bruning, P. Wolff, J. B. Smith, and J. A. Jenks. 2012*b*. Causes of pneumonia epizootics among bighorn sheep, western United States, 2008–2010. Emerging Infectious Diseases 18:406–414.

Brooks, S. P., and A. Gelman. 1998. General methods for monitoring convergence of iterative simulations. Journal of Computational and Graphical Statistics 7:434–455.

Butler, C. J., R. A. Garrott, and J. J. Rotella. 2013. Correlates of recruitment in Montana bighorn sheep populations. http://www.wildsheepworkinggroup.com/app/download/ 7236242856/Correlates + of + Recruitment + in + Montana + Bighorn + Sheep + Populations-FINAL.pdf. Accessed 5 Dec 2013. .

Cahn, M. L., M. M. Conner, O. J. Schmitz, T. R. Stephenson, J. D. Wehausen, and H. E. Johnson. 2011. Disease, population viability, and recovery of endangered Sierra Nevada bighorn sheep. Journal of Wildlife Management 75:1753–1766.

Carpenter, T. E., V. L. Coggins, C. McCarthy, C. S. O'Brien, J. M. O'Brien, and T. J. Schommer. 2014. A spatial risk assessment of bighorn sheep extirpation by grazing domestic sheep on public lands. Preventative Veterinary Medicine 114:3–10.

Cassaigne, G., R. A. Medellín, and J. A. Guasco. 2010. Mortality during epizootics in bighorn sheep: effects of initial population size and cause. Journal of Wildlife Diseases 46:763–771.

Cassirer, E. F., R. K. Plowright, K. R. Manlove, P. C. Cross, A. P. Dobson, K. A. Potter, and P. J. Hudson. 2013. Spatio-temporal dynamics of pneumonia in bighorn sheep. Journal of Animal Ecology 82:518–528.

Cassirer, E. F., and A. R. E. Sinclair. 2007. Dynamics of pneumonia in a bighorn sheep metapopulation. Journal of Wildlife Management 71:1080–1088.

Clifford, D. L., B. A. Schumaker, T. R. Stephenson, V. C. Bleich, M. L. Cahn, B. J. Gonzales, W. M. Boyce, and J. A. K. Mazet. 2009. Assessing disease risk at the wildlife-livestock interface: a study of Sierra Nevada bighorn sheep. Biological Conservation 142:2559–2568.

Conroy, M. J., and J. T. Peterson. 2013. Decision-making in natural resource management: a structured, adaptive approach. Wiley-Blackwell, Oxford, United Kingdom.

DeCesare, N. J., and D. H. Pletscher. 2006. Movements, connectivity, and resource selection of Rocky Mountain bighorn sheep. Journal of Mammalogy 87:531–538.

Deem, S. L., W. B. Karesh, and W. Weisman.. 2001. Putting theory into practice: wildlife health in conservation. Conservation Biology 15:1224–1233.

Dormann, C. F., J. Elith, S. Bacher, C. Buchmann, G. Carl, G. Carré, J. R. G. Marquéz, B. Gruber, B. Lafourcade, P. J. Leitão, T. Münkemüller, C. McClean, P. E. Osborne, B. Reineking, B. Schröder, A. K. Skidmore, D. Zurell, and S. Lautenbach.. 2013. Collinearity: a review of methods to deal with it and a simulation study evaluating their performance. Ecography 36:27–46.

Edwards, V. L., J. Ramsey, C. Jourdonnais, R. Vinkey, M. Thompson, N. Anderson, T. Carlsen and C. Anderson.. 2010. Situational agency response to four bighorn sheep die-offs in western Montana. Biennial Symposium of the Northern Wild Sheep and Goat Council. 29–50. http://media.nwsgc.org/proceedings/MWSGC-2010/Edwards%20et%2029-50.pdf. Accessed 17 Jun 2013. .

Enk, T. A., H. D. Picton, and J. S. Williams. 2001. Factors limiting a bighorn sheep population in Montana following a dieoff. Northwest Science 75:280–291.

Epps, C. W., D. R. McCullough, J. D. Wehausen, V. C. Bleich, and J. L. Rechel. 2004. Effects of climate change on population persistence of desert-dwelling mountain sheep in California. Conservation Biology 18:102–113.

Foreyt, W. J. 1989. Fatal *Pasteurella haemolytica* pneumonia in bighorn sheep after direct contact with clinically normal domestic sheep. American Journal of Veterinary Research 50:341–344.

Foreyt, W. J., and D. A. Jessup. 1982. Fatal pneumonia of bighorn sheep following association with domestic sheep. Journal of Wildlife Diseases 18:163–168.

Fralick, G. L. 1984. A senior thesis study of the bighorn sheep of the Petty Creek Drainage. Senior thesis, University of Montana, Missoula, USA.

Gelman, A., and J. Hill. 2007. Data analysis using regression and multilevel/hierarchical models. Cambridge University Press, Cambridge, United Kingdom.

George, J. L., D. J. Martin, P. M. Lukacs, and M. W. Miller. 2008. Epidemic pasteurellosis in a bighorn sheep population coinciding with the appearance of a domestic sheep. Journal of Wildlife Diseases 44:388–403.

Goodson, N. J., D. R. Stevens, and J. A. Bailey. 1991. Effects of snow on foraging ecology and nutrition of bighorn sheep. Journal of Wildlife Management 55:214–222.

Gross, J. E., F. J. Singer, and M. E. Moses. 2000. Effects of disease, dispersal, and area on bighorn sheep restoration. Restoration Ecology 8(4S):25–37.
Hosmer, D. W., and S. Lemeshow. 2000. Applied logistic regression, Second edition. John Wiley & Sons, Hoboken, New Jersey, USA.
Kéry, M. 2010. Introduction to WinBUGS for ecologists: a Bayesian approach to regression, ANOVA, mixed models and related analyses, First edition. Academic Press, Burlington, Massachusetts, USA.
Lafferty, K. D., and L. R. Gerber. 2002. Good medicine for conservation biology: the intersection of epidemiology and conservation theory. Conservation Biology 16:593–604.
Lawrence, P. K., S. Shanthalingam, R. P. Dassanayake, R. Subramaniam, C. N. Herndon, D. P. Knowles, F. R. Rurangirwa, W. J. Foreyt, G. Wayman, A. M. Marciel, S. K. Highlander, and S. Srikumaran.. 2010. Transmission of *Mannheimia haemolytica* from domestic sheep (*Ovis aries*) to bighorn sheep (*Ovis canadensis*): unequivocal demonstration with green fluorescent protein-tagged organisms. Journal of Wildlife Diseases 46:706–717.
Link, W. A., and R. J. Barker. 2010. Bayesian inference with ecological applications. Academic Press, London, United Kingdom.
Link, W. A., E. Cam, J. D. Nichols, and E. G. Cooch. 2002. Of bugs and birds: Markov chain Monte Carlo for hierarchical modeling in wildlife research. Journal of Wildlife Management 66:277–291.
Miller, D. S., E. Hoberg, G. Weiser, K. Aune, M. Atkinson, and C. Kimberling. 2012. A review of hypothesized determinants associated with bighorn sheep (*Ovis canadensis*) die-offs. Veterinary Medicine International. http://www.hindawi.com/journals/vmi/2012/ 796527/. Accessed 3 Apr 2012.
Miller, D. S., G. C. Weiser, K. Aune, B. Roeder, M. Atkinson, N. Anderson, T. J. Roffe, K. A. Keating, P. L. Chapman, C. Kimberling, J. Rhyan, and P. R. Clarke. 2011. Shared bacterial and viral respiratory agents in bighorn sheep (Ovis canadensis), domestic sheep (Ovis aries), and goats (Capra hircus) in Montana. Veterinary Medicine International. http://www.hindawi.com/journals/vmi/2011/162520/. Accessed 2 Dec.
Miller, M. W., N. T. Hobbs, and E. S. Williams. 1991. Spontaneous pasteurellosis in captive Rocky Mountain bighorn sheep (*Ovis canadensis canadensis*): clinical, laboratory, and epizootiological observations. Journal of Wildlife Diseases 27:534–542.
Mitchell, M. S., J. A. Gude, N. J. Anderson, J. M. Ramsey, M. J. Thompson, M. G. Sullivan, V. L. Edwards, C. N. Gower, J. F. Cochrane, E. R. Irwin, and T. Walshe. 2013. Using structured decision making to manage disease risk for Montana wildlife. Wildlife Society Bulletin 37:107–114.
Monello, R. J., D. L. Murray, and E. F. Cassirer. 2001. Ecological correlates of pneumonia epizootics in bighorn sheep herds. Canadian Journal of Zoology 79:1423–1432.
Montana, F., and P. Wildlife. 2010. Montana bighorn sheep conservation strategy. Montana Fish, Wildlife and Parks, Helena, USA. http://fwp.mt.gov/fwpDoc.html?id=39746. Accessed 5 Sep 2011. .
Mood, A. M., F. A. Graybill, and D. C. Boes. 1974. Introduction to the theory of statistics. McGraw-Hill International, Singapore.
Ntzoufras, I. 2009. Bayesian modeling using WinBUGS. John Wiley & Sons, Hoboken, New Jersey, USA.
O'Brien, J. M., C. S. O'Brien, C. McCarthy, and T. E. Carpenter. 2014. Incorporating foray behavior into models estimating contact risk between bighorn sheep and areas occupied by domestic sheep. Wildlife Society Bulletin 38:321–331.
Onderka, D. K., and W. D. Wishart. 1984. A major bighorn sheep die-off from pneumonia in southern Alberta. Biennial Symposium of the Northern Wild Sheep and Goat Council 4:356–363.
Onderka, D. K., and W. D. Wishart. 1988. Experimental contact transmission of *Pasteurella haemolytica* from clinically normal domestic sheep causing pneumonia in Rocky Mountain bighorn sheep. Journal of Wildlife Diseases 24:663–667.
Plowright, R. K., K. Manlove, E. F. Cassirer, P. C. Cross, T. E. Besser, and P. J. Hudson. 2013. Use of exposure history to identify patterns of immunity to pneumonia in bighorn sheep (*Ovis canadensis*). PLoS ONE 8: (4):e61919. http://www.plosone.org/article/info%3Adoi %2F10.1371% 2Fjournal.pone.0061919. Accessed 5 Dec 2013.
Portier, C., M. Festa-Bianchet, J.-M. Gaillard, J. T. Jorgenson, and N. G. Yoccoz. 1998. Effects of density and weather on survival of bighorn sheep lambs (*Ovis canadensis*). Journal of Zoology 245:271–278.
Royle, J. A., and R. M. Dorazio. 2008. Hierarchical modeling and inference in ecology: the analysis of data from populations, metapopulations and communities. Elsevier, London, United Kingdom.
Rudolph, K. M., D. L. Hunter, W. J. Foreyt, E. F. Cassirer, R. B. Rimler, and A. C. S. Ward. 2003. Sharing of *Pasteurella* spp. between free-ranging bighorn sheep and feral goats. Journal of Wildlife Diseases 39:897–903.
Rudolph, K. M., D. L. Hunter, R. B. Rimler, E. F. Cassirer, W. J. Foreyt, W. J. DeLong, G. C. Weiser, and A. C. S. Ward. 2007. Microorganisms associated with a pneumonic epizootic in Rocky Mountain bighorn sheep (*Ovis canadensis canadensis*). Journal of Zoo and Wildlife Medicine 38:548–558.
Scott, M. E. 1988. The impact of infection and disease on animal populations: implications for conservation biology. Conservation Biology 2:40–56.
Sells, S. N. 2014. Proactive management of pneumonia epizootics in bighorn sheep in Montana. Thesis, University of Montana, Missoula, USA.
Singer, F. J., M. E. Moses, S. Bellew, and W. Sloan. 2000*a*. Correlates to colonizations of new patches by translocated populations of bighorn sheep. Restoration Ecology 8(4S):66–74.
Singer, F. J., C. M. Papouchis, and K. K. Symonds. 2000*b*. Translocations as a tool for restoring populations of bighorn sheep. Restoration Ecology 8(4S):6–13.
Singer, F. J., E. Williams, M. W. Miller, and L. C. Zeigenfuss. 2000*c*. Population growth, fecundity, and survivorship in recovering populations of bighorn sheep. Restoration Ecology 8(4S):75–84.
Singer, F. J., L. C. Zeigenfuss, and L. Spicer. 2001. Role of patch size, disease, and movement in rapid extinction of bighorn sheep. Conservation Biology 15:1347–1354.
Shanthalingam, S., A. Goldy, J. Bavananthasivam, R. Subramaniam, S. A. Batra, A. Kugadas, B. Raghavan, R. P. Dassanayake, J. E. Jennings-Gaines, H. J. Killion, W. H. Edwards, J. M. Ramsey, N. J. Anderson, P. L. Wolff, K. Mansfield, D. Bruning and S. Srikumaran. 2014. PCR assay detects Mannheimia haemolytica in culture-negative pneumonic lung tissues of bighorn sheep (Ovis canadensis) from outbreaks in the western USA 2009–2010. Journal of Wildlife Diseases 50:1–10.
Spiegelhalter, D. J., N. G. Best, B. P. Carlin, and A. Van Der Linde. 2002. Bayesian measures of model complexity and fit. Journal of the Royal Statistical Society. Series B 64:583–639.
Steyerberg, E. W., A. J. Vickers, N. R. Cook, T. Gerds, M. Gonen, N. Obuchowski, M. J. Pencina, and M. W. Kattan. 2010. Assessing the performance of prediction models: a framework for some traditional and novel measures. Epidemiology 21:128–138.
Vickers, A. J., and E. B. Elkin. 2006. Decision curve analysis: a novel method for evaluating prediction models. Medical Decision Making 26:565–574.
Wehausen, J. D., S. T. Kelley, and R. R. Ramey. 2011. Domestic sheep, bighorn sheep, and respiratory disease: a review of the experimental evidence. California Fish and Game 97:7–24.
Wells J. V., and M. E. Richmond. 1995. Populations, metapopulations, and species populations: what are they and who should care. Wildlife Society Bulletin 23:458–462.
Wild Sheep Working Group., 2012. Recommendations for domestic sheep and goat management in wild sheep habitat. Western Association of Fish and Wildlife Agencies, http://www. wildsheepworkinggroup.com/resources/publications/. Accessed 17 Jun 2013. .
Wobeser, G.A. 2006. Essentials of disease in wild animals. Blackwell, Ames, Iowa, USA.
Woodroffe, R. 1999. Managing disease threats to wild mammals. Animal Conservation 2:185–193.
Young, T. P. 1994. Natural die-offs of large mammals: implications for conservation. Conservation Biology 8:410–418.

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