The Journal of Wildlife Management 9999:1–10; 2011; DOI: 10.1002/jwmg.310

Research Article

Variations in Elk Aggregation Patterns Across a Range of Elk Population Sizes at Wall Creek, Montana

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ABSTRACT In the Greater Yellowstone Ecosystem, growing concern over increasing rates of brucellosis seroprevalence in wildlife has challenged wildlife managers to develop strategies for minimizing the potential for pathogen exchange within and between wildlife populations. Recent evidence suggests that increases in elk seroprevalence may be associated with increasing elk densities and/or increasing size of elk aggregations. However, the interactions between elk population density, landscape factors, and elk aggregation patterns are not well-understood, making appropriate management responses challenging. Using a unique, long-term elk aggregation dataset collected across a wide range of elk population sizes, we investigated relationships between elk population size, landscape factors, and elk aggregation responses (group size and group density) with goals of clarifying how changes in elk population size may affect elk aggregation patterns. Overall, landscape attributes and weather had a stronger influence on elk aggregation patterns than factors such as elk population size that are within management control. We found little evidence that elk population size affected mean elk group sizes, but we did find evidence that the size and density of the largest elk aggregations increased as elk population size increased. We also found some evidence that group densities increased following the establishment of wolves. However, across the relatively wide range of elk population sizes observed in this study, only modest changes in elk group density were observed, suggesting that dramatic reductions in population sizes would be necessary to produce measureable reductions in elk group density to affect frequency-dependent transmission. Management actions designed to lower disease transmission are likely to negatively affect other objectives related to elk management and conservation. We therefore suggest that a first step in managing disease transmission risk is agreement among stakeholders interested in elk management of all objectives related to elk management, including acknowledgment that disease transmission is undesirable. © 2011 The Wildlife Society.

KEY WORDS *Brucella abortus*, brucellosis, *Cervus elaphus*, elk, Greater Yellowstone Ecosystem, group size.

Ungulate aggregation patterns are of interest to ecologists and wildlife managers because aggregation behaviors may affect ungulate-plant interactions (Gude et al. 2006), predation dynamics (Hebblewhite and Pletscher 2002), hunter success, crop depredation, as well as the risk of disease transmission (Joly and Messier 2004, Cross et al. 2010*a*). In the Greater Yellowstone Ecosystem (GYE), ungulate aggregation patterns are of particular interest because free-ranging elk and bison are hosts for *Brucella abortus*, the pathogen that causes brucellosis (Olsen 2010). Brucellosis is a chronic bacterial disease that may be transmitted between livestock and wildlife when individuals investigate or feed near infected fetuses, placentas, or birthing fluids (Cheville et al. 1998). The potential for free-ranging wildlife to transmit the disease to livestock has generated considerable controversy between environmentalists, ranchers, and natural resource managers (Beinen and Tabor 2006, Kilpatrick et al. 2009, Proffitt et al. 2010*a*). Recent evidence suggests that at a broad scale, increases in brucellosis seroprevalence are correlated with increases in free-ranging elk densities (Cross et al. 2010*b*). However, the relationship between host density and transmission is not well understood (Maichak et al. 2009). Studies have documented constant brucellosis seroprevalence rates across a wide range of population sizes (Dobson and Meagher 1996, Joly and Messier 2004, Cross et al. 2007), suggesting the relationship may be non-linear. This uncertainty in the relationship between population density and transmission makes defining appropriate management responses difficult.

Variations in the relationship between host density and pathogen transmission may be complicated in elk because elk typically show a skewed group size distribution with many

Received: 24 May 2011; Accepted: 24 September 2011

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small groups and relatively few, if any large groups (Gower et al. 2009, Cross et al. 2010*a*). Disease dynamics and seroprevalence rates may, however, be driven by elk interactions in large, dense aggregations rather than population size or density over large areas (Keeling and Rohani 2008, Cross et al. 2010*b*). If large groups are the most influential driver of disease dynamics, then management actions aimed at broadly reducing elk densities to reduce the size and frequency of large elk aggregations may or may not be an effective strategy. Management strategies to affect brucellosis or other disease dynamics may be more effectively directed at affecting elk group sizes or group densities. However, the effect of elk population sizes and other potential drivers of elk group sizes and densities have received little quantitative attention, limiting development of possible actions to affect elk grouping behaviors. To better inform appropriate management strategies aimed at reducing elk aggregations and pathogen transmission, a better understanding of the relationships between elk population size, elk group size, and elk-grouping density is needed.

We evaluated factors affecting elk group size and group densities on an elk winter range in southwestern Montana from 1987 to 2010. During the course of this study, elk population size ranged from approximately 1,000–3,000 elk, providing the unique opportunity to investigate how elk population size affects elk group size and group density. Identifying long-term changes in elk aggregation patterns may provide insights into potential disease risk consequences of increasing elk populations. Further, quantifying the degree that landscape attributes, predation risk, and population size affect elk aggregations may clarify the potential role of these factors in affecting elk-to-elk disease transmission risk.

STUDY AREA

The Wall Creek elk winter range was located in the western Madison Valley in southwestern Montana. The core of the winter range was located within the Wall Creek Wildlife Management Area (WMA), an area purchased by Montana Fish, Wildlife, and Parks (MFWP) in 1960 to protect the core of the Wall Creek elk winter range. During winter, human activity within Wall Creek WMA was restricted to administrative personnel, and all roads were closed to vehicle traffic. Wall Creek WMA was grazed under a rest-rotational system by approximately 700 cattle from 1 May to 30 September (Shamhart et al., in press). Typical of elk winter ranges throughout the west, the elk winter range expanded into private lands adjacent to the WMA that were used for livestock production.

The winter range covered an area of approximately 100 km^2. Elevation ranged from 1,700 m to 2,400 m. Approximately 75% of the winter range was grasslands, surrounded by higher elevation sagebrush-steppe and coniferous forests (e.g., *Pinus contorta*, *Pinus flexilis*, *Abies lasiocarpa*, *Picea engelmannii*, and *Pseudotsuga menziesii)*. Bluebunch wheatgrass (*Pseudoroegneria spicata*), prairie junegrass (*Koeleria pyramidata*), Sandberg bluegrass (*Poa secunda*), and threadleaf sedge (*Carex filifolia*) were the dominant grass species. Crested wheatgrass (*Agropyron desertorum*) and smooth brome (*Bromus inermis*) were also present in some areas. Green and rubber rabbitbrush (*Chrysothamnus viscidiflorus*, *C. nauseosus*) and silver sagebrush (*Artemisia cana*) were the most common shrubs. The winter climate in the valley was characterized by long, cold winters and strongly influenced by winds. The valley was heavily windswept during winter, often leaving the open, low-elevation benches and higher elevation ridges largely snow-free (Gude et al. 2006).

The area served as a winter range for a migratory herd of 1,200–3,000 elk. The Wall Creek elk herd ranged from a low of 1,186 animals in 1991 to a high of 3,108 elk in 2004. Elk may shed infectious *Brucella abortus* bacteria during late term abortion or parturition events, which generally occur from February through mid-June. Wolves began moving through the area in 2002 and a single pack of 3 wolves became established in the area during winter 2007–2008. The same pack of wolves, numbering 3–8 animals, used the area through Fall 2010, when they were removed because of conflicts with livestock. No human hunting of elk occurred within the study area during the winter study period.

METHODS

From 1987 to 2010, we conducted elk surveys across the entire winter range every 7–14 days from 1 December until 15 May. Exact survey dates varied among years. We conducted surveys from 4 designated areas along the boundary of the WMA and from a nearby highway using a spotting scope. We recorded elk group locations and the number of elk per group on a topographic map. A single observer (F. K.) conducted more than 95% of all surveys throughout the study. We digitized elk distribution data into a geographic information system (GIS), and calculated elk grouping density from the number of elk per group and the area of the group extent polygon calculated in the GIS.

We evaluated 5 factors affecting elk aggregation responses (group size and group density): elk herd size, vegetation cover type, winter severity, season, and wolf period (Creel and Winnie 2005, Gude et al. 2006, Gower et al. 2009, Proffitt et al. 2009). We did not consider hunter access as a potential covariate affecting elk aggregation (Cross et al. 2010*a*) because hunting seasons were not concurrent with our data collection. We used the 2001 national land cover dataset (Homer et al. 2004) to broadly classify vegetation type as grassland, shrubland, forested area, and other (rock, water, etc). We assigned cover type at the centroid of each elk group polygon. We used the cumulative winter snow water equivalence (SWE) as a metric of winter severity. This metric integrates the depth and density of snowpack into a measure of the amount of water contained within the snowpack, and was measured daily at the Beaver Creek, Montana station snowpack telemetry site approximately 44 km from the Wall Creek winter range. We defined winter severity as the sum of daily SWE values from 1 December to 30 May each year. We evaluated season as a dichotomous covariate contrasting observations collected during December–March (winter) and observations collected during April–May (spring). We considered April as the start of the spring season because in

21 of the 24 years of data collection, field notes indicated that the winter range was primarily snow-free by 1 April. We estimated elk population size based on the maximum number of elk counted on the winter range in a given sampling period (i.e., day) each season. We evaluated the interactive effects of winter severity and vegetation type to represent the hypotheses that elk aggregation responses in different vegetation types varied with snowpack (Proffitt et al. 2009). We defined 2 time periods corresponding to different levels of wolf predation risk: pre-wolf and colonizing wolf period (1988–2007) and established wolf period (2008–2010). Wolves from the nearby East Madison Valley pack were first observed using the Wall Creek winter range during the winter of 2002–2003. A member of this pack was collared during the winters of 2006 and 2007 and global positioning system (GPS) location data indicated only minimal wolf activity in the Wall Creek area (MFWP, unpublished data). Therefore, we consider the colonizing wolf period a period of minimal wolf predation risk. During the established wolf period, 3–8 wolves comprising a single pack used the Wall Creek winter range as part of their core winter territory (Sime et al. 2010).

Prior to developing our a priori model list, we screened covariates for correlations and excluded pairs with Pearson's correlation coefficients $|r| \geq 0.7$ and variance inflation factors >5 from entering the same model. We developed 7 a priori models representing potential effects of covariates on elk aggregation responses (group size and group density). We used a linear modeling approach to evaluate competing hypotheses regarding variations in elk aggregation responses (R Development Core Team 2008). We natural log-transformed group size and group density to meet assumptions of normality. We used Akaike's Information Criterion corrected for small sample sizes (AIC_c) to rank competing models and Akaike model weights (w_i) to address model-selection uncertainty. Because the upper ends of the group size and density distributions may be particularly influential risk factors related to the spread of brucellosis or other diseases (Cross et al. 2010*a*), we used a quantile regression model to determine if the upper ends of the group size and group density distributions increased as elk population size increased (R Development Core Team 2008).

We estimated brucellosis seroprevalence in adult female elk in the Wall Creek herd during 2-time periods that corresponded to separate periods of serology data collection and screening, 1983–1993 and 2005–2010. We collected blood samples opportunistically from hunter harvested (2005–2010) and research captured (1983–1993) adult female elk and screened blood serum for *Brucella abortus* antibodies. Hunter harvest occurred from September–November and research captures occurred in January–March. From 1983–1993, we estimated brucellosis seroprevalence from the standard plate agglutination, *B. abortus* antigen rapid card (card), rivanol precipitation (Riv), complement fixation (CFT), and buffered acidified plate antigen (BAPA) tests (Veterinary Diagnostic Laboratory, Bozeman, MT) tests and classified according to the United States Department of Agriculture's brucellosis eradication uniform methods and rules. Certain strains of bacteria including *E. coli*, *Salmonella*, and *Yersinia enterocolitica* O:9 may cross-react in serologic tests designed for *B. abortus*, leading to false positive results. Therefore, all samples collected from 2005–2010 that tested seropositive in the standard tests were screened using the Western immunoblot test to determine if antibodies were due to a cross reaction with *Yersinia enterocolitica* O:9 (Edmonds et al. 1999). We evaluated 5 models representing potential effects of elk population size, size of the largest elk groups, density of the largest elk groups, and the frequency of large groups (i.e., ≥300 animals; Cross et al. 2010*a*) on annual variations in seroprevalence. Because of strong correlations ($r > 0.5$) between predictor variables and a low sample size, we evaluated only simple, 1-predictor models. We used a generalized linear modeling approach to evaluate competing models in Program R using a binomial likelihood and a logit link function (R Development Core Team 2008). To assess overall model fit, we conducted the le Cessie-van Houwelingen goodness-of-fit test on our top ranked model (le Cessie and van Houwelingen 1991, Hosmer et al. 1997). This test is designed to assess goodness-of-fit for models with continuous covariates and binary responses based on non-parametric kernel methods.

RESULTS

We observed 4,503 elk groups during 528 sampling days. The average duration between sampling events was 6 days. Group size ranged from 1 to 2,199 (median = 40). We censored 139 groups of a single animal from the group density analysis because density could not be calculated for a single animal group. Group density ranged from 18–49,955 elk per km^2 (median = 682). Seasonally, mean group size ranged from a low of 104 in April to a high of 174 in January (Fig. 1A) and mean group density ranged from a low of 898 elk/km^2 in April to a high of 1,391 elk/km^2 in January (Fig. 1B). Annually, mean group size ranged from a low of 112 in 1993 to a high of 216 in 1998 (Fig. 1C) and mean group density ranged from a low of 733 elk/km^2 in 1991 to a high of 1,979 elk/km^2 in 2010 (Fig. 1D). The estimated elk population size was approximately 1,200 elk during 1987–1992 and steadily increased to approximately of 3,000 animals during 2004–2007. From 2007 to 2010, the estimated elk population size decreased to approximately 2,000 elk. Elk group observations occurred most commonly in the grassland ($n = 1{,}523$) and shrubland ($n = 2{,}729$) cover types, and less frequently in the forested ($n = 167$) and other ($n = 118$) cover types. Winter severity, measured as cumulative SWE values, ranged from 31.6 m to 106.5 m and averaged 57 m. During winter, we collected 3,256 group observations and 1,247 during spring.

The most supported model explaining variations in elk group sizes contained the covariates vegetation cover type, winter severity, season, elk population size, and wolf period (Table 1). However, model explanatory power was low ($R_{adj}^2 = 0.08$), and the realized effect of model covariates on elk group sizes were minimal. Habitat type had the largest effect on elk group sizes, followed by winter severity, season, and wolf period. Group sizes were predicted to be largest in

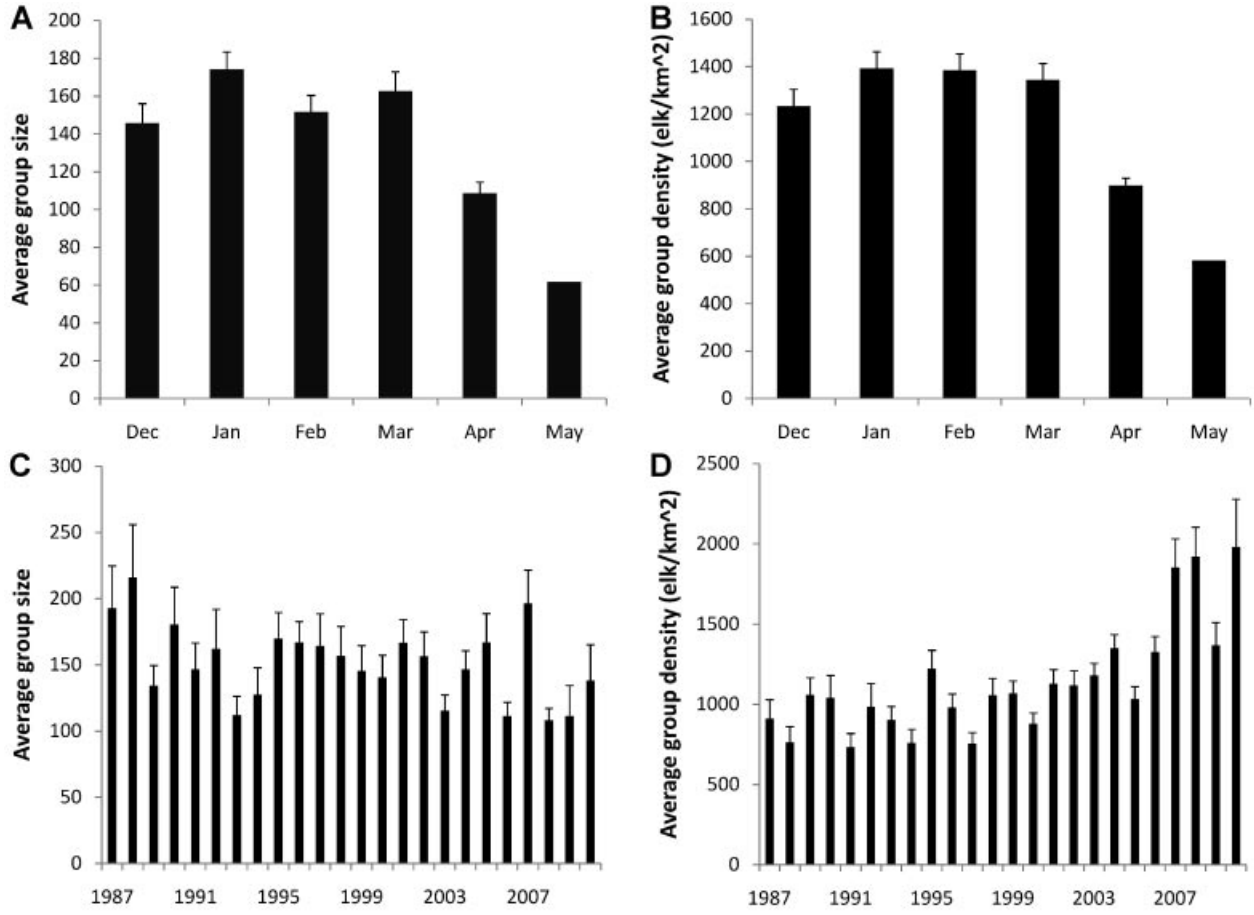

Figure 1. Seasonal (Panel A,B) and annual variations (Panel C,D) in average elk group size and group density at the Wall Creek, Montana winter range, 1987–2010. Error bars represent 1 standard error.

the grassland areas and lowest in the forested areas (compared to the base category grassland; $\hat{\beta}_{\text{forest}} = -2.1$, 95% CI $= -2.5, -1.9$; $\hat{\beta}_{\text{shrub}} = -0.79$, 95% CI $= -0.90, -0.68$; $\hat{\beta}_{\text{other}} = -0.70$, 95% CI $= -1.0, -0.4$). The predicted mean group size varied from 76 (95% CI $= 69, 83$) in grasslands, to 34 (95% CI $= 31, 37$) in shrublands, to 9 (95% CI $= 7, 11$) in forested areas, and 38 (95% CI $= 28, 51$) in other areas (estimates created for winter, pre-wolf period with average population size and snowpack). Group size was smaller as elk population size increased, although the magnitude of this effect was small ($\hat{\beta}_{\text{elk}} = -0.00025$, 95% CI $= -0.00035, -0.00015$). For example, in grasslands, post-wolf, during winter, and under average snowpack conditions, the predicted mean group size ranged from 80 (95% CI $= 66, 98$) at a population size of 1,200 to 52 (95% CI $= 44, 61$) at a population size of 3,000. Group size was smaller in the spring than winter ($\hat{\beta}_{\text{season}} = -0.2$, 95% CI $= -0.33, -0.10$), although the difference between predicted group size in the winter (76, 95% CI $= 69, 83$) and spring (61, 95% CI $= 54, 69$) was small (estimates created for pre-wolf period and with average population size and snowpack). Group size decreased during the post-wolf establishment period ($\hat{\beta}_{\text{wolves}} = -0.19$, 95% CI $= -0.33, -0.04$), although the effect of wolf establishment on group size was minimal (Fig. 2). Group size increased as winter severity increased ($\hat{\beta}_{\text{snow}} = 0.000045$, 95% CI $= 0.00001, 0.000079$; Fig. 3). We found evidence that the upper end of the group size distribution increased as elk population size increased. The quantile regression model indicated that the 50th and 95th percentile of group size did not vary with elk population size, but the 99th percentile increased as elk population size increased (Fig. 4A).

Table 1. Model selection results for models examining the effects of elk population size and landscape attributes on variation in elk group size at the Wall Creek, Montana elk winter range from 1987 to 2010. Covariates included vegetative cover (Veg), winter severity (Snow), season (Season), estimated elk population size (PopSize), and wolf period (wolves). All models are presented along with the number of parameters (K), the difference in second-order Akaike Information Criterion value relative to the smallest value in the model set (ΔAIC_c), and the Akaike weight (w_i). The AIC_c score of the top model was 17,550.

Model structure	K	ΔAIC_c	w_i
Veg + snow + season + popsize + wolves	9	0.0	0.89
Veg + snow + season + popsize	8	4.1	0.11
Veg + popsize	6	16.2	0.00
Veg + snow + season + wolves	8	22.4	0.00
Veg + season	6	30.1	0.00
Veg + snow + season	7	30.5	0.00
Veg + snow	6	40.1	0.00

The most supported model explaining variations in elk group density contained the covariates vegetation cover type, winter severity, season, elk population size, and wolf period (Table 2; $R_{\text{adj}}^2 = 0.11$). The actual effects of cova-

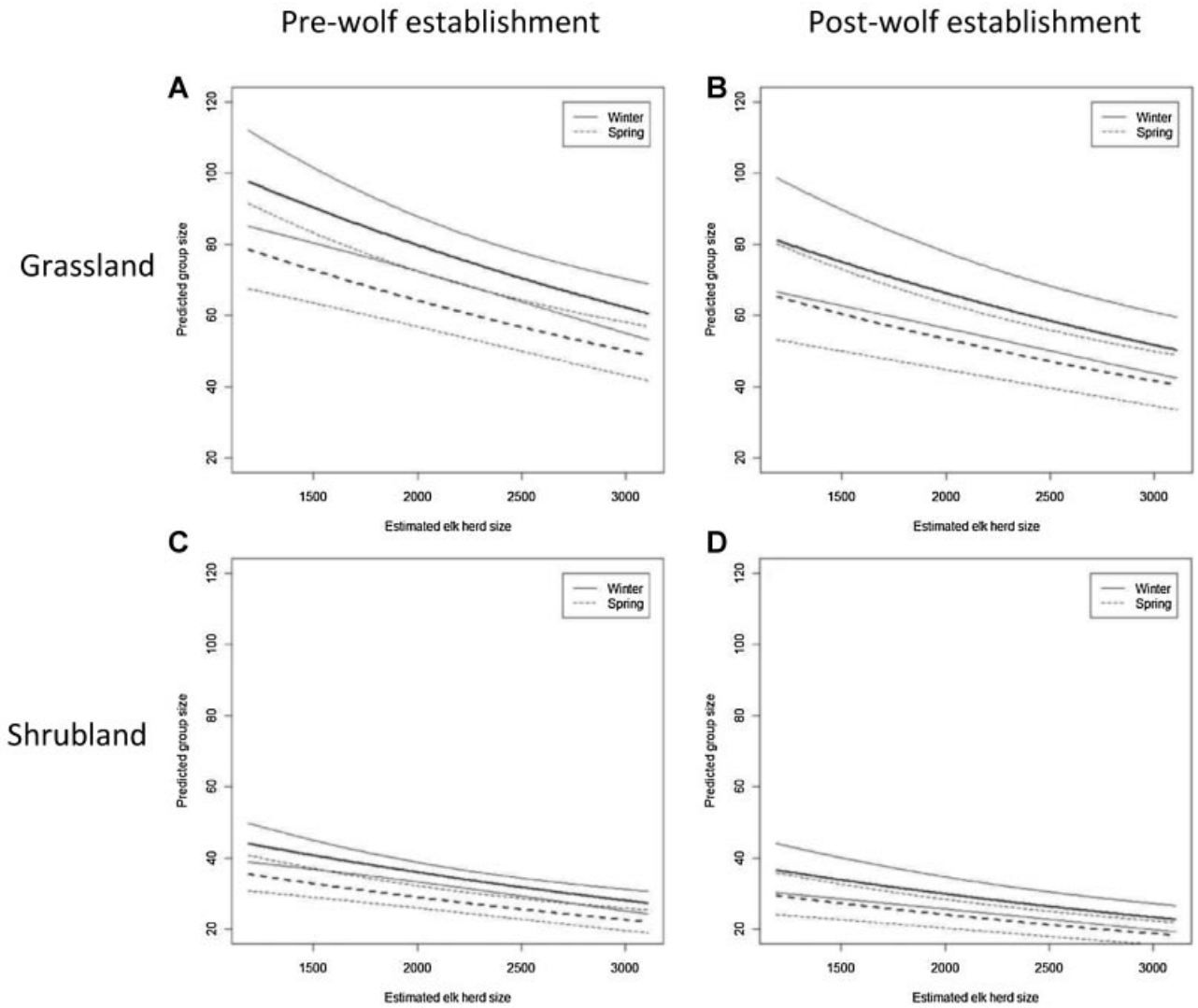

Figure 2. The predicted elk group size during the winter and spring season across the observed range of elk herd sizes at Wall Creek, Montana in grasslands (Panels A,B) and shrublands (Panels C,D) during the pre-wolf (Panels A,C) and post-wolf (Panels B,D) establishment periods. Bold lines represent mean predictions and thin lines represent 95% confidence intervals.

riates on elk group densities was minimal, but habitat type had the largest effect of the covariates we considered. Group density was greatest in the grasslands and least in the forested areas (compared to the base category grassland; $\hat{\beta}_{\text{forest}} = -1.00$, 95% CI $= -1.18, -0.82$; $\hat{\beta}_{\text{shurb}} = -0.45$, 95% CI $= -0.52, -0.39$; $\hat{\beta}_{\text{other}} = -0.33$, 95% CI $= -0.53, -0.13$). The predicted mean group density varied from 954 elk/km^2 (95% CI = 900, 1,012) in grasslands, to 606 elk/km^2 (95% CI = 579, 634) in shrublands, to 352 (95% CI = 296, 419) in forested areas, and 684 (95% CI = 563, 831) in other areas (estimates created for winter, pre-wolf period with average population size and snowpack). Group density decreased in the spring ($\hat{\beta}_{\text{season}} = -0.32$, 95% CI $= -0.39, -0.25$). The predicted mean group density for grassland groups was 954 elk/km^2 (95% CI = 900, 1,012) during winter and 693 elk/km^2 (95% CI = 642, 747) during spring. Group density did not vary with winter severity. Group density increased as population size increased and increased during the post-wolf establishment period ($\hat{\beta}_{\text{wolf}} = 0.36$, 95% CI $= 0.27, 0.45$; Fig. 5). We found evidence that the upper end of the group density distribution increased as elk population size increased. The quantile regression model indicated that the 50th, 95th, and 99th percentile of group density increased as elk population size increased (Fig. 4B).

We estimated brucellosis seroprevalence in adult female elk in the Wall Creek herd at 1.2% in 1983–1993 ($n = 174$) and 0–30% in 2005–2010 ($n = 35$). From 2005–2010, brucellosis seroprevalence using standard testing methods was 30.0%. However, supplemental Western blot testing suggested that each of the positive seroreactors was due to cross-reactions. Seroprevalence estimated from 2005–2010 using Western blot was 0% ($n = 30$). The most supported model explaining variations in seroprevalence (based on standard testing) included the covariate elk population size (Table 3; $w_i = 0.79$, $\hat{\beta}_{\text{PopSize}} = 0.0018$, 95% CI $= 0.0008, 0.0028$). The goodness-of-fit test supported the null hypothesis that the top model fit the data ($P = 0.40$) and the model and the

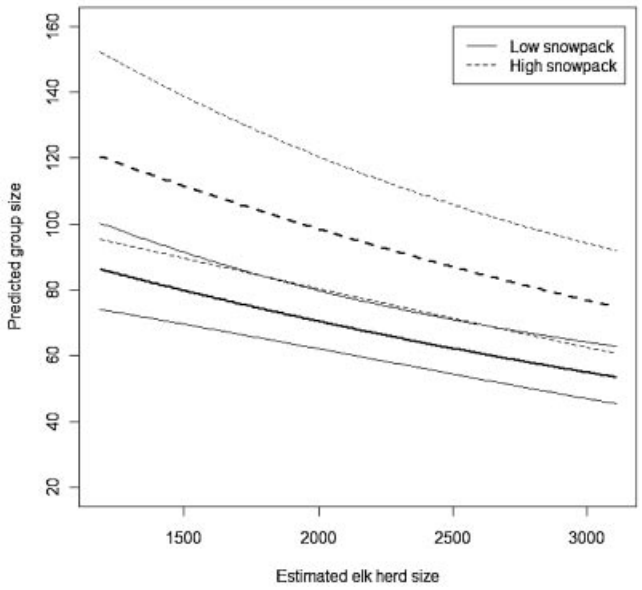

Figure 3. The predicted elk group size during low snowpack and high snowpack conditions across the observed range of elk herd sizes at Wall Creek, Montana (estimates created for grasslands, during winter and the pre-wolf establishment period using the minimum and maximum winter severity values observed during 1987–2010). Bold lines represent mean predictions and thin lines represent 95% confidence intervals.

Nagelkerke R^2 index was 0.26. The predicted seroprevalence ranged from 2.5% (95% CI = 0.7, 8) at a population size of 1,200 elk to 65% (95% CI = 3, 100) at a population size of 3,000 elk. The second most supported model included the covariate 99th quantile of group density, and predicted that seroprevalence increased as the density of the largest elk groups increased ($w_i = 0.18$, $\hat{\beta}_{\text{PopSize}} = 0.0004$, 95% CI = 0.0002, 0.0007). The goodness-of-fit test supported the null hypothesis that the second ranked model fit the data ($P = 0.06$) and the model and the Nagelkerke R^2 index was 0.21.

DISCUSSION

Mean elk group sizes and group densities were more strongly influenced by seasonal and landscape attributes than by elk population size. Similar to other elk grouping studies, groups were largest in grassland areas and smallest in forested areas (Gude et al. 2006, Gower et al. 2009, Proffitt et al. 2009), and we found the highest density groups in grasslands. In contrast to other studies, group sizes increased as snowpack increased (Gower et al. 2009), potentially because on the Wall Creek winter range higher snowpack may have moved elk into the lower elevation open, grassland areas where elk tend to aggregate in larger groups. Grouping density was not related to snowpack. Mean group size decreased with increasing elk population size, although the magnitude of this change across a range of years when elk population size more than doubled was minimal. This result contrasts with our predictions and results of similar studies documenting increases in group size associated with increasing elk population density (Hebblewhite and Pletscher 2002, Cross et al. 2010*a*). Although mean group size did not vary dramatically with elk population size, quantile regression results indicated that the 99th percentile of the elk group size distribution did increase as elk population size increased. Further, the 99th quantile of the elk density distribution increases as elk population size increased. If these largest, high density elk aggregations create the highest risk of elk-to-elk disease transmission risk (Cross et al. 2010*a*), then the increases in the size of large groups and density with increasing elk population size does suggest that elk-to-elk contact rates and the risk of disease transmission increases as elk population size increases.

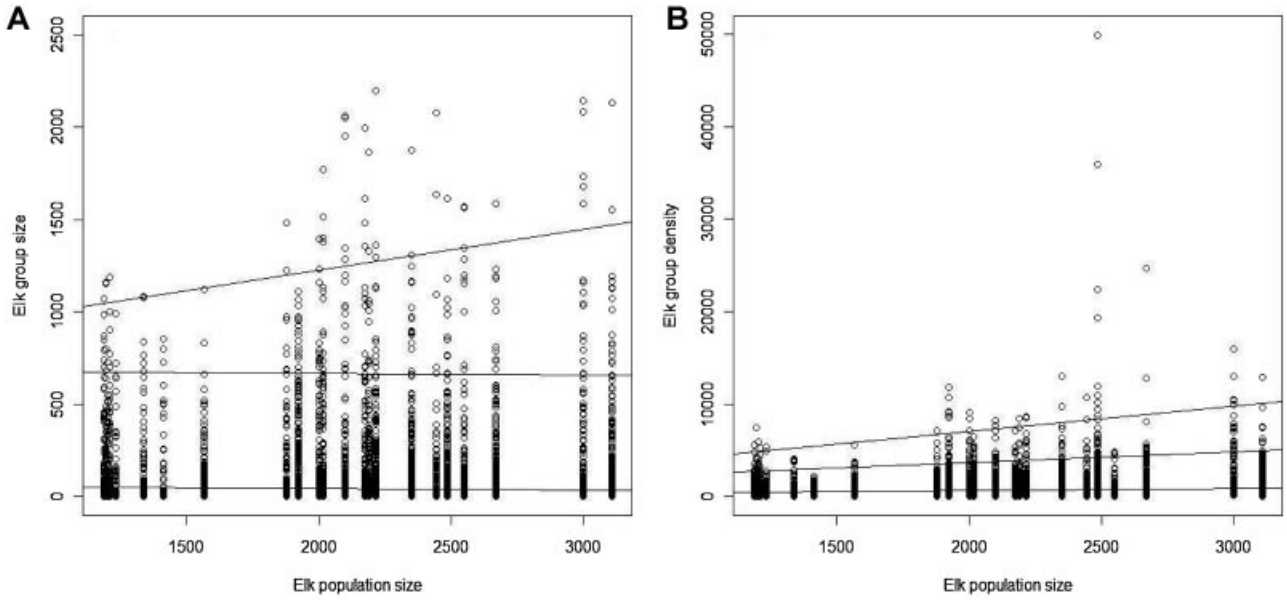

Figure 4. Elk group size (Panel A) and group density (Panel B) across the range of elk population sizes during 1987–2010 at the Wall Creek, Montana winter range. Open circles represent elk group observations. Solid lines represent the quantile regressions of the 50th, 95th, and 99th percentiles.

Table 2. Model selection results for models examining the effects of elk population size and landscape attributes on variation in elk group density at the Wall Creek, Montana elk winter range from 1987 to 2010. Covariates included vegetative cover (Veg), winter severity (Snow), season (Season), estimated elk population size (Popsize), and wolf period (wolves). All models are presented along with the number of parameters (K), the difference in second-order Akaike Information Criterion value relative to the smallest value in the model set (ΔAIC_c), and the Akaike weight (w_i). The AIC_c score of the top model was 12,762.

Model structure	K	ΔAIC_c	w_i
Veg + snow + season + popsize + wolves	9	0.0	1.0
Veg + snow + season + popsize	8	56.6	0.0
Veg + snow + season + wolves	8	84.2	0.0
Veg + popsize	6	128.5	0.0
Veg + snow + season	7	163.0	0.0
Veg + season	6	167.1	0.0
Veg + snow	6	245.1	0.0

The establishment of wolves in the Wall Creek area during the last 3 years of this study did not appear to affect elk group sizes, but we did find evidence that elk group density increased during the post-wolf establishment period. Grouping may benefit prey by diluting predation risk or reducing individual vigilance requirements needed to detect predators (Hamilton 1971, Bertram 1978, Pulliam and Caraco 1984, Roberts 1996). We found that wolf establishment in the Wall Creek system had little impact on elk group sizes, although similar studies have documented increases in elk group size following establishment of wolves (Gower et al. 2009). Instead of increasing or decreasing group sizes following the establishment of wolves in the area, we found that elk maintained similar group sizes but increased grouping density. The observed increases in elk grouping density, but not group size, may reflect individuals' efforts to enhance spread of information throughout the group or group defensive strategies to minimize risk of separation and attack. Increasing elk group density may increase the number or likelihood of animals contacting infected birthing materials and potentially increase brucellosis transmission risk (Maichak et al. 2009). Although elk aggregation responses to wolf establishment observed here were relatively slight, in

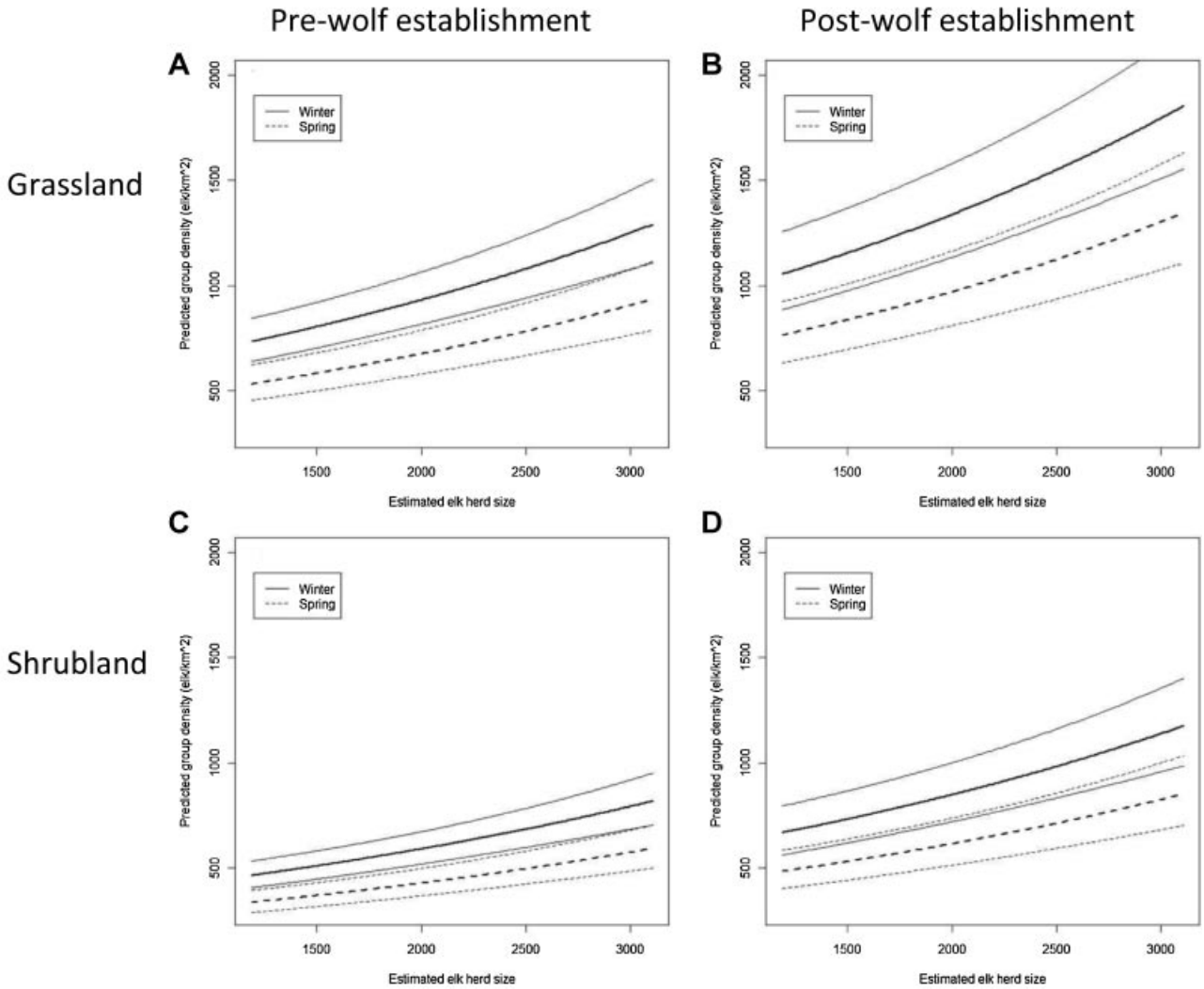

Figure 5. The predicted elk group density during the winter and spring season across the observed range of elk herd sizes at Wall Creek, Montana in grasslands (Panels A,B) and shrublands (Panels C,D) during the pre-wolf (Panels A,C) and post-wolf (Panels B,D) establishment periods. Bold lines represent mean predictions and thin lines represent 95% confidence intervals.

Table 3. Model selection results for models examining the effects of elk population size and aggregation patterns on variation in elk exposure to brucellosis at the Wall Creek, Montana elk winter range from 1987 to 2010. Covariates included elk population size (Popsize), the 99th quantile of group size per year (99Q_GS), the 99th quantile of group density per year (99Q_GD), and the frequency of large group observations (Freq). All models are presented along with the number of parameters (K), the difference in second-order Akaike Information Criterion value relative to the smallest value in the model set (ΔAIC_c), and the Akaike weight (w_i). The AIC_c score of the top model was 64.80.

Model structure	K	ΔAIC_c	w_i
Popsize	2	0.26	0.79
99Q_GD	2	3.19	0.18
Freq	2	7.38	0.02
99Q_GS	2	13.98	0.00
Null	1	14.07	0.00

systems with higher predator:prey ratios, wolf effects on elk aggregation patterns could be more pronounced.

We found that seasonal and landscape attributes had a relatively greater impact on elk aggregation patterns than elk population management or predation risk. However, we found that brucellosis seroprevalence may have increased or remained stable depending on the testing criteria used to define positive cases as population size increased. Our 2005–2010 estimate of seroprevalence may have been underestimated if samples collected in fall are less likely to test seropositive than late winter samples (Cross et al. 2010*b*), further suggesting that seroprevalence increased during the study period. Although we found some evidence of increasing seroprevalence with elk population size, uncertainty in interpretations and accuracy of serology results make these findings inconclusive, and the major drivers of seroprevalence are unknown. A standard, reliable definition of identifying positive brucellosis cases in both live-captured and harvested animals must be developed in order to resolve the drivers of brucellosis seroprevelence, because disease dynamics are markedly different in density dependent versus frequency dependent transmitted diseases (Swinton et al. 2001). The effectiveness of management actions to limit brucellosis spread also therefore will depend on the major drivers of brucellosis infection rates.

Density dependent disease transmission risk models predict transmission rates are a function of host density (Swinton et al. 2001), and recently Cross et al. (2010*a*, *b*) found support for a positive relationship between elk density at the hunting district level and increases in brucellosis seroprevalence in free-ranging elk populations. We found increases in elk group density associated with increasing elk population sizes. Additionally, we found that the upper end of the elk group size and density distributions increased as elk population size increased. These results support the hypothesis that increases in herd size may result in increasing elk group densities, which increase frequency-dependent disease transmission risk. Therefore, increases in brucellosis seroprevalence correlated to increases in elk population sizes do not necessarily support the notion that brucellosis is transmitted in a density-dependent fashion. However, across the relatively wide range of elk population sizes observed in this study, only modest changes in elk group density were observed, suggesting that dramatic reductions in population sizes, which are not likely socially acceptable, would be necessary to produce measureable reductions in elk group density to affect frequency-dependent transmission. This is similar to the conclusions of Cross et al. (2010*b*), in that reductions in elk density to affect density-dependent transmission were also weakly supported management strategies.

Our results suggest that actions aimed at managing disease transmission by reducing elk densities will have minimal effects on elk aggregations and the risk of elk-to-elk contact during the brucellosis transmission risk period. Therefore, density-dependent disease transmission management strategies, such as reducing population sizes, may not affect frequency-dependent transmission. With regards to frequency-dependent disease transmission in elk, our results suggest that elk aggregation patterns are widely variable; our models explained very little of the observed variation in elk group size or density in this dataset. Therefore, either elk aggregation patterns are not predictably variable, such that they cannot be purposefully managed, or we did not consider the primary drivers of elk aggregation patterns in our models. Further, the largest effects on elk aggregation that we documented were seasonal and landscape attributes, which are largely out of management control. Elk aggregation patterns were strongly affected by vegetation cover type and season, with the largest and highest density groups being observed in grassland areas during winter.

We also found evidence that elk group densities increased slightly following wolf colonization. If this is a cause–effect relationship, elk group densities may be decreased by managing wolf numbers. This type of management action, however, will likely not be palatable to all stakeholders. In other elk herds, human hunting pressure has been shown to have dramatic effects on the size of elk groups (Gude et al. 2006, Proffitt et al. 2009). Cross et al. (2010*a*) also hypothesized that lands closed to hunter access resulted in large elk congregations, and others have found elk selection for areas with limited hunter access (Proffitt et al. 2010*b*). We were unable to quantify the impact of human hunting on elk aggregation patterns in this dataset because our study area was closed to hunting during the study period. Similarly, we were unable to evaluate other factors known to affect elk aggregation patterns, such as supplemental feeding (Peek et al. 2002), because they were not present in our study area. Analyses in herds where these other potential drivers are present, or among-herd comparisons, are required to quantify the extent to which they influence elk aggregation or brucellosis seroprevalence patterns. To the extent that hunting pressure, supplemental feeding, or other drivers of elk aggregation actually affect elk aggregation patterns, they also may offer potential management targets to affect frequency-dependent transmission (e.g., Maichak et al. 2009). However, similar to the potential management targets we identified, some potential actions designed to affect elk aggregations and therefore frequency-dependent transmission of disease will require trade-offs, such as increasing hunter numbers or eliminating supplemental feeding.

Although diseases such as chronic wasting disease are not yet present in GYE ungulates, understanding aggregation patterns is also relevant to transmission dynamics of other diseases. To prioritize management actions aimed at reducing elk aggregations and densities across GYE, a better understanding of spatial variations in elk aggregation patterns and disease prevalence rates among herds is needed. If aggregation patterns are largely driven by landscape attributes, elk herds occupying winter ranges characterized by more open, grassland habitats are predicted to have larger, denser elk aggregations, and management actions aimed at reducing elk-to-elk contact during the transmission risk period should be focused in these areas.

MANAGEMENT IMPLICATIONS

Our results provide some evidence that reductions in elk population sizes or wolf populations may lead to modest reductions in elk group densities, thereby limiting frequency dependent disease transmission. Although our results indicate that elk group sizes are affected primarily by landscape attributes, weather, and season, which are out of management control, other studies indicate that other factors, such as human hunters and supplemental feeding strategies can be used to manipulate elk group sizes in order to affect disease transmission. In all of these cases, management actions designed to lower disease transmission are likely to negatively affect other objectives related to elk management and conservation. Specifically, some interests may desire high elk population sizes, desire wolf presence, not want human hunting pressure on a particular landscape or landholding, and/or want supplemental feedgrounds for several reasons unrelated to wildlife disease. We therefore suggest that a first step in managing disease transmission risk is agreement among those interested in elk management of all objectives related to elk management, including acknowledgment that disease transmission is undesirable. Wildlife managers should provide education on wildlife disease management strategies and the consequences of these strategies, and work with the public towards mutually acceptable elk management policies.

ACKNOWLEDGMENTS

Funding was provided by revenues from the sale of Montana hunting and fishing licenses and matching Federal Aid in Wildlife Restoration grants to Montana Fish, Wildlife, and Parks, as well as funding from the United States Department of Agriculture, Animal and Plant Health Inspection Service for brucellosis serology. We thank J. Cunningham for providing comments that improved this manuscript and N. Anderson and K. Aune for providing brucellosis data. Two reviewers and Associate Editor G. Hall provided helpful comments that improved this manuscript.

LITERATURE CITED

Beinen, L., and G. Tabor. 2006. Applying an ecosystem approach to brucellosis control: can an old conflict between wildlife and agriculture be successfully managed? Frontiers in Ecology and the Environment 4:319–327.

Bertram, B. C. R. 1978. Living in groups: predators and prey. Pages 64–96 *in* J. R. Krebs and N. B. Davies, editors. Behavioral ecology: an evolutionary approach. Blackwell Scientific Publications, Oxford, UK.

Cheville, N. F., D. R. McCullough, and L. R. Paulson. 1998. Brucellosis in the Greater Yellowstone Area. National Academies Press, Washington D.C., USA.

Creel, S., and J. A. Winnie. 2005. Responses of elk herd size to fine-scale spatial and temporal variation in the risk of predation by wolves. Animal Behaviour 69:1181–1189.

Cross, P. C., W. H. Edwards, B. M. Scurlock, E. J. Maichak, and J. D. Rogerson. 2007. Effects of management and climate on elk brucellosis in the greater Yellowstone Ecosystem. Ecological Applications 17:957–964.

Cross, P. C., E. K. Cole, A. P. Dobson, W. H. Edwards, K. L. Hamlin, G. Luikart, A. D. Middletown, B. M. Scurlock, and P. J. White. 2010*a*. Probable causes of increasing brucellosis in free-ranging elk of the Greater Yellowstone Ecosystem. Ecological Applications 20:278–288.

Cross, P. C., D. Heisey, B. Scurlock, W. Edwrads, M. Ebinger, and A. Brennan. 2010*b*. Mapping brucellosis increases relative to elk density using hierarchical Bayesian models. PLOSOne 5:1–9.

Dobson A. P., and M. Meagher. 1996. The population dynamics of brucellosis in the Yellowstone National Park. Ecology 77:1026–1036.

Edmonds, M. D., F. M. Ward, T. M. O'Hara, and P. H. Elzer. 1999. Use of Western immunoblot analysis for testing moose serum for *Brucella suis* biovar 4 specific antibodies. Journal of Wildlife Diseases 35:591–595.

Gower, C. N., R. A. Garrott, P. J. White, S. Cherry, and N. Yoccoz. 2009. Elk group size and predation: a flexible strategy when faced with variable risk. Pages 401–422 *in* R. A. Garrott, P. J. White, and F. G. R. Watson, editors. The ecology of large mammals in central Yellowstone: sixteen years of integrated field studies. Academic Press, San Diego, California, USA.

Gude, J. A., R. A. Garrott, J. J. Borkowski, and F. King. 2006. Prey risk allocation in a grazing ecosystem. Ecological Applications 16:285–298.

Hamilton, W. D. 1971. Geometry for the selfish group. Journal of Theoretical Ecology 31:295–311.

Hebblewhite, M., and D. Pletscher. 2002. Effects of elk group size on predation by wolves. Canadian Journal of Zoology 80:800–809.

Homer, C., C. Huang, L. Yang, B. Wylie, and M. Coan. 2004. Development of a 2001 National Landcover Database for the United States. Photogrammetric Engineering and Remote Sensing 70:829–840.

Hosmer, D. W., T. Hosmer, S. le Cessie, and S. Lemeshow. 1997. A comparison of goodness-of-fit tests for the logistic regression model. Statistics in Medicine 16:965–980.

Joly, D. O., and F. Messier. 2004. Factors affecting apparent prevalence of tuberculosis and brucellosis in wood bison. Journal of Animal Ecology 73:623–631.

Keeling, M. J., and P. Rohani. 2008. Modeling infection disease in humans and animals. Princeton University Press, Princeton, New Jersy, USA.

Kilpatrick, A. M., C. M. Gillin, and P. Daszak. 2009. Wildlife-livestock conflict: the risk of pathogen transmission from bison to cattle outside Yellowstone National Park. Journal of Applied Ecology 46:476–485.

le Cessie, S., and J. C. van Houwelingen. 1991. A goodness-of-fit test for binary regression models, based on smoothing methods. Biometrics 47:1267–1282.

Maichak, E. J., B. M. Scurlock, J. D. Rogerson, L. L. Meadows, A. E. Barbknecht, W. H. Edwards, and P. C. Cross. 2009. Effects of management, behavior, and scavenging on risk of brucellosis transmission in elk of western Wyoming. Journal of Wildlife Diseases 45:398–410.

Olsen, S. C. 2010. Brucellosis in the United States: role and significance of wildlife reservoirs. Vaccine 28S:F73–F76.

Peek, J. M., K. T. Schmidt, M. J. Dorrance, and B. L. Smith. 2002. Supplemental feeding and farming of elk. Pages 617–647 *in* D. E. Toweill and J. W. Thomas, editors. North American elk: ecology and management. Smithsonian Institution Press, Washington, D.C., USA.

Proffitt, K. M., J. Grigg, R. A. Garrott, and K. L. Hamlin. 2009. Contrasting effects of wolves and human hunters on elk behavioral responses to predation risk. Journal of Wildlife Management 73:345–356.

Proffitt, K. M., J. A. Gude, K. L. Hamlin, R. A. Garrott, J. A. Cunningham, and J. Grigg. 2010*a*. Elk distribution and spatial overlap with livestock during the brucellosis transmission risk period. Journal of Applied Ecology 48:471–478.

Proffitt, K. M., J. Grigg, R. A. Garrott, K. L. Hamlin, J. Cunningham, J. A. Gude, and C. Jourdonnais. 2010*b*. Changes in elk resource selection and

distributions associated with a late season elk hunt. Journal of Wildlife Management 74:210–218.

Pulliam, H. R., and T. Caraco. 1984. Living in groups: is there an optimal group size? Pages 122–147 *in* J. R. Krebs and N. B. Davies, editors. Behavioral ecology: an evolutionary approach. Second edition. Blackwell Scientific Publications, Oxford, UK.

R Development Core Team. 2008. R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. <http://www.R-project.org>.

Roberts, G. 1996. Why individual vigilance declines as group size increases. Animal Behaviour 31:947–948.

Shamhart, J., F. King, and K. M. Proffitt. in press. Effects of a rest-rotation grazing system on wintering elk distributions at Wall Creek, Montana. Rangelands Ecology and Management.

Sime, C. A., V. Asher, L. Bradley, K. Laudon, N. Lance, M. Ross, and J. Steuber. 2010. Montana gray wolf conservation and management 2009 annual report. Montana Fish, Wildlife & Parks, Helena, Montana, USA.

Swinton, J., M. E. J. Woolhouse, M. E. Begon, A. P. Dobson, E. Ferroglio, B. T. Grenfell, V. Guberti, R. S. Hails, J. A. P. Heesterbeek, A. Lavassa, M. G. Roberts, P. J. White, and K. Wilson. 2001. Microparasite transmission and persistence. Pages 83–101 *in* P. J. Hudson, A. Rizzoli, B. T. Grenfell, H. Heesterbeek, and A. P. Dobson, editors. The ecology of wildlife diseases. Oxford University Press, Oxford, UK.

Associate Editor: Graham Hall.