ASSESSING THE POTENTIAL EFFECTS OF TREPONEME ASSOCIATED HOOF DISEASE (TAHD) ON ELK POPULATION DYNAMICS IN SOUTHWEST WASHINGTON

PROJECT UPDATE
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INTRODUCTION

Various hoof diseases have been reported worldwide in numerous free-ranging ungulates, including elk (*Cervus elaphus*; Murie 1930, Gray et al. 2001, Thorne et al. 2002), mule deer (*Odocoileus hemionus*; Wobeser et al. 1975), white-tailed deer (*O. virginianus;* Sleeman et al. 2009), moose (*Alces;* Flynn et al. 1977, Clauss et al. 2009), fallow deer (*Dama*; Lavin et al. 2004), reindeer (*Rangifer tarandus*; Handeland et al. 2010), roe deer (*Capreolus*; Handeland and Vikøren 2005), and mouflon (*Ovis gmelini musimon*; Volmer et al. 2008). Reports of elk in southwestern Washington with evidence of lameness or various hoof abnormalities were historically sporadic and infrequent. In early 2008, however, the number and geographic extent of elk displaying evidence of an apparently novel hoof disease significantly increased (Mansfield et al. 2011, WDFW unpublished data).

The emergence of this disease in southwest Washington elk herds is unique in that bacteria in the genus *Treponema*, (aka "treponemes"), never previously associated with hoof diseases in any free-ranging ungulate, have been identified as causal (Clegg et al. 2015). Treponemes are strongly associated with similar diseases of domestic livestock: bovine digital dermatitis of cattle (Evans et al. 2009), contagious ovine digital dermatitis (CODD) of domestic sheep (Sayers 2009), and a CODD-like disease of domestic goats (Sullivan et al. 2015).

Elk affected by treponeme-associated hoof disease (TAHD) often have severely overgrown and deformed hooves with sole ulcers and sloughed hoof walls (Han and Mansfield 2014). TAHD can occur in multiple limbs and can affect all age and sex classes (Clegg et al. 2015). The severity of clinical signs, coupled with the seemingly rapid expansion of impacted areas, have generated a great deal of concern for the Washington Department of Fish and Wildlife (WDFW), other resource management agencies, hunters, tribes, and local citizens. In response to these concerns, WDFW continues to work with several specialists to better understand the etiology of TAHD. In addition, WDFW established a Hoof Disease Technical Advisory Group (HDTAG) and a Hoof Disease Public Working Group (HDPWG). The HDTAG has guided the diagnostic effort, identified research needs, and provided review and input to management options. The HDPWG has provided input to management and research options and serves as a venue for WDFW to share information with the public. However, it is difficult to assess what implications TAHD will have for the management of affected elk herds because the effects of TAHD on elk vital rates (e.g., survival, reproduction, etc.) are unknown.

It is reasonable to assume that elk with advanced stages of TAHD have a decreased probability of survival because their infirmities may predispose them to predation, harvest, severe weather events, or other types of disease (Bender et al. 2008). For example, mule deer with chronic wasting disease (CWD), prior to developing obvious clinical signs, have been shown to be more vulnerable to predation (Miller et al. 2008, Krumm et al. 2009), vehicle collisions (Krumm et al. 2005), and possibly harvest (Conner et al. 2000). This is an important consideration because the growth rate of large ungulate populations, such as elk, is highly sensitive to changes in adult female survival (Nelson and Peek 1982, Eberhardt 2002) and strongly correlated with the production and survival of juveniles (Gaillard et al. 2000; *see also* Smith and Anderson 1998, Raithel et al. 2007). When adult female and juvenile survival are concurrently reduced, populations would be expected to decline (Gaillard et al. 2000; *see also* Bender et al. 2007, McCorquodale et al. 2014). Consequently, if TAHD reduces the survival of adult females and calves, it has the potential to have a negative effect on the population dynamics of impacted elk herds.

Although McCorquodale et al. (2014) monitored 16 adult female elk that had varying degrees of presumed TAHD (i.e., they had varying degrees of hoof deformities, but no lab samples were collected and tested) inferences from their work are limited. Twelve of 16 affected elk they monitored survived ≥ 1 year and of those that did not survive ≥ 1 year, all were harvest-related mortalities. In addition, 3 of 4 elk that were fitted with VHF collars that had a battery life of several years survived until radio contact was lost 3-4 years after they were captured. Anecdotally, this indicates that if TAHD negatively affects the natural survival of elk, it may take several years before it does so. We need to improve our understanding of how quickly TAHD progresses and if, and when, it may begin to predispose affected elk to mortality.

TAHD may also have the potential to affect the population dynamics of impacted elk herds because of its effect on the energy dynamics of female elk. The nutritional condition of female ungulates can influence age at first breeding (Cook et al. 2004), timing of estrus and subsequent birth date (Andersen and Linnell 1998, Cook et al. 2004, Bishop et al. 2009), probability of conception (Cook et al. 2004, Cook et al. 2013), fetal development and survival (Verme 1969, Ozoga and Verme 1982), birth weight (Verme and Ullrey 1984, Keech et al. 2000, Lomas and Bender 2007), milk yield or composition (Landete-Castillejos et al. 2003, Tollefson 2007), and subsequent growth and survival of juveniles (Clutton-Brock et al. 1982, Bishop et al. 2009). For example, elk from the Mount St. Helens elk herd area (MSH) and other coastal regions of Washington are

characterized by pregnancy rates for prime-aged females that are consistently depressed [Kuttel 1975 (74%), Smith 1980 (61%), Cook et al. 2013 (68-100%), McCorquodale et al. 2014 (71%)] because marginal nutrition limits the level of condition female elk are able to achieve during the summer-autumn period (Cook et al. 2013). Due to the additional energetic requirements for mounting an immune response and for tissue repair (Deming 2009), TAHD may further limit the ability of affected elk to improve their condition during the summer-autumn period and therefore has the potential to reduce overall pregnancy rates even further, which could reduce demographic vigor.

Some have attributed recent declines in the MSH elk herd to TAHD because the monitored portions of the MSH herd declined by 30-35% over a 4-year period (2009–2013; McCorquodale et al. 2014) that coincided with an increase in the prevalence and distribution of the disease (WDFW, unpublished data). However, this period of population decline also occurred concurrently with a directed effort by WDFW to reduce the elk population through substantial increases in antlerless harvest because of evidence that the MSH elk herd was above ecological carrying capacity (WDFW 2006, McCorquodale et al. 2014). Moreover, density independent severe winter weather that occurred in 2012 likely contributed to the documented decline (McCorquodale et al. 2014). Because these three events overlapped temporally and elk with presumed TAHD represented <15% of the adult females that were monitored, McCorquodale et al. (2014) were not able to conclude whether or not TAHD was a contributing factor in observed declines.

The number of elk that have TAHD and the effects of TAHD on elk vital rates, collectively, will determine what the long-term implications of TAHD are for the viability, and subsequent management, of impacted elk herds (Wobeser 2007). Consequently, our primary research goals are to quantify how TAHD may affect the survival, pregnancy rates, productivity, and nutritional condition of adult female elk. Our specific study objectives include:

1. *Estimate the effects of TAHD on survival of adult (≥ 2 years old) female elk.*
2. *Determine cause-specific mortality rates for adult female elk that have TAHD.*
3. *Estimate the effects of TAHD on the pregnancy rates of adult female elk.*
4. *Estimate the effects of TAHD on elk productivity (i.e., survivorship of calves).*

5. *Estimate the effects of TAHD on the level of condition (i.e., IFBF) adult female elk are able to achieve in autumn.*

6. *Increase our understanding of how TAHD progresses in individual elk, and whether affected elk may recover from the disease.*

STUDY AREA

Our study area consists of 5 Game Management Units (GMUs) that, collectively, represent the core range of the MSH herd (Figure 1). The primary reasons we focused our work in this area are: 1) it occurs within the TAHD endemic area; 2) it decreases the probability of stochastic variation in the data independent of TAHD; and 3) it is the same study area of McCorquodale et al. (2014). Having the same study area as McCorquodale et al. (2014) afforded us the opportunity to put more emphasis on monitoring elk affected by TAHD because we could potentially use their findings for non-affected elk, 2009–2012, as baseline estimates of survival for elk independent of the disease.

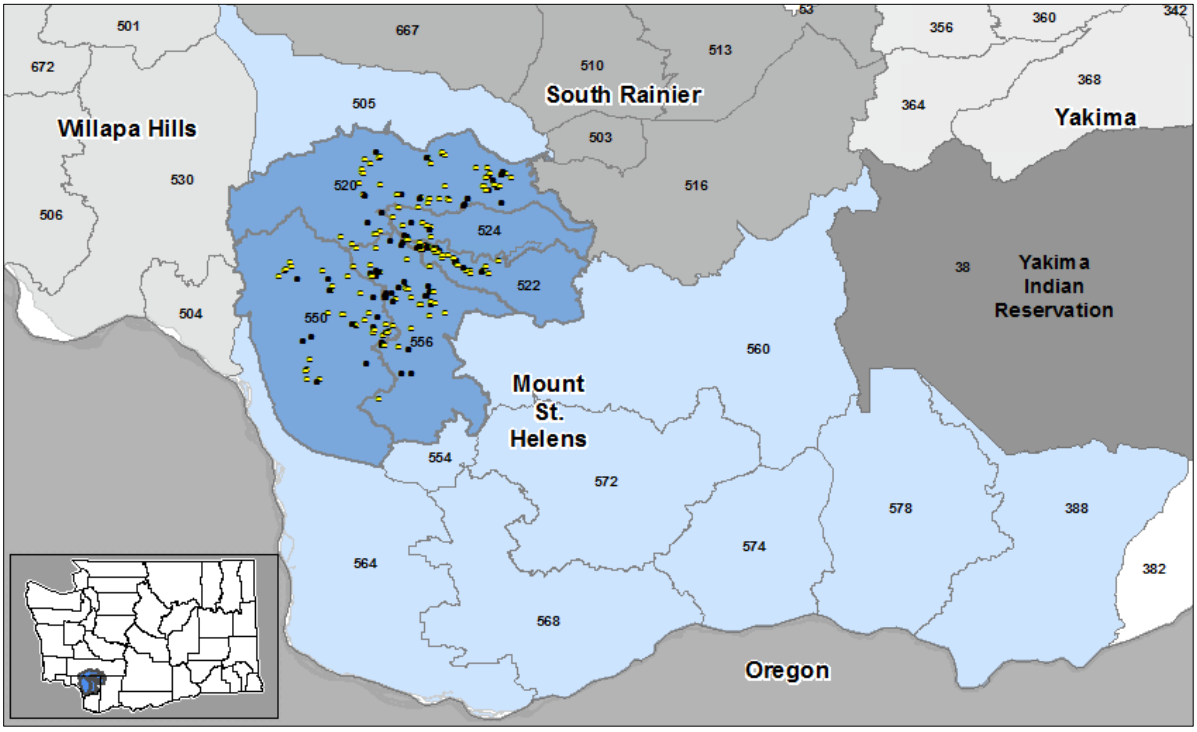

Figure 1. Map depicting the Game Management Units (GMUs) that comprise the Mount St. Helens elk herd area (light blue), the 5 GMUs that represent the core range of the herd and our study area (dark blue), and the locations where we have captured elk affected (yellow) or seemingly unaffected (black) by treponeme-associated hoof disease, February 2015–December 2017. Also included for spatial reference are GMUs associated with the Willapa Hills, South Rainier, and Yakima elk herds.

METHODS AND RESULTS

Capture and Marking

We initiated captures February 17–27, 2015 with the goal of capturing and marking 80 adult female elk at a ratio of 3 elk affected by TAHD (hereafter, diseased group) to every 1 elk that was unaffected (hereafter, control group). We conducted subsequent captures December 2015–2017, with the primary goal of maintaining our desired sample size and 3:1 ratio within each GMU. We conducted captures December 16–22 in all 3 years. When attempting to mark elk for inclusion in our diseased group, we only targeted individuals that were visibly limping, which, in most instances, was indicative of an elk having advanced stages of TAHD–of the elk we captured that were limping, only 3 were unaffected by TAHD. However, subsequent to us capturing them, we determined some elk we had captured for inclusion in our control group (i.e., not limping) had early stages of the disease. Although we were primarily interested in marking elk most severely affected by TAHD, we made the decision to include these elk in the diseased group because it afforded us the opportunity to increase our understanding of disease progression. Lastly, in order to increase the likelihood that our sample of diseased elk was an unbiased sample, we attempted to capture the first limping elk we detected within a group, regardless of their apparent condition (i.e., some elk were visibly emaciated at time of capture).

We captured female elk via aerial darting from a Bell 206B Jet Ranger helicopter using recommended immobilizing and reversal agents (Kreeger and Armeno, 2007). We blindfold elk to minimize stress during handling, administered clostridium vaccine (the first time the animal was captured), vitamin E and analgesic (flunixin meglumine) injections, and treated the dart wound. We marked each elk using a colored and numbered ear-tag and a mortality-sensitive, GPS (Global Positioning System)-equipped radio-collar. We determined disease status by having a veterinarian, knowledgeable of hoof deformities commonly associated with TAHD and other hoof diseases, examine each hoof after we had used a saline solution to remove mud and debris from the hoof. We also removed an upper canine tooth to determine age using microhistological analysis of cementum annuli (Hamlin et al. 2000; Matson's Laboratory, Milltown, MT).

We captured 80, 46, 43, and 42 female elk February 2015, December 2015, December 2016, and December 2017, respectively (Table 1). A subset of the elk we captured in December 2015 (n = 20 diseased, 10 control), December 2016 (n = 15 diseased, 8 control), and December 2017 (n = 6 diseased, 4 control) represented elk we had originally marked during previous capture events.

We recaptured these elk to accomplish three objectives: 1) to confirm disease status of elk in our control group; 2) to increase our understanding of disease progression; and 3) to index the proportion of elk known to be pregnant within each group that successfully raised a calf through late-autumn. Collectively, we captured 148 individuals during 211 capture events.

Table 1. The number of female elk we captured in each Game Management Unit (GMU) by capture event and the number of those elk that had visible signs of being affected by treponeme-associated hoof disease (Diseased Group), or appeared to be unaffected by the disease (Control Group).

	Diseased Group					Control Group				
GMU	**Feb 2015**	**Dec 2015**	**Dec 2016**	**Dec 2017**	**Total**	**Feb 2015**	**Dec 2015**	**Dec 2016**	**Dec 2017**	**Total**
520	24	10	10	3	47	6	5	4	2	17
522	11	6	5	9	31	1	2	3	5	11
524	1	4	2	0	7	3	0	0	1	4
550	15	6	4	5	30	5	0	2	5	12
556	9	5	9	6	29	5	8	4	6	23
Total	**60**	**31**	**30**	**23**	**144**	**20**	**15**	**13**	**19**	**67**

We did not mark two of the elk we captured in February 2015 because they died during the capture process (1 yearling and 1 adult; both had TAHD). In addition, we had 1 diseased elk we captured in December 2016 and 1 control elk in December 2017 that died within 1 day of being captured. In both instances, we immediately retrieved the radio-collar and redeployed it on a different elk. We included data from these elk in all analyses, except for survival.

Ages of female elk at time of initial capture that we assigned to our diseased group (n = 101) ranged 1-16 years and averaged 6 years old (95% CI = 5-7), while ages of female elk we assigned to our control group (n = 45) ranged 1-13 years and averaged 7 years old (95% CI = 6-8) (Figure 2). We were not able to collect a tooth for age determination from 2 elk in our diseased group.

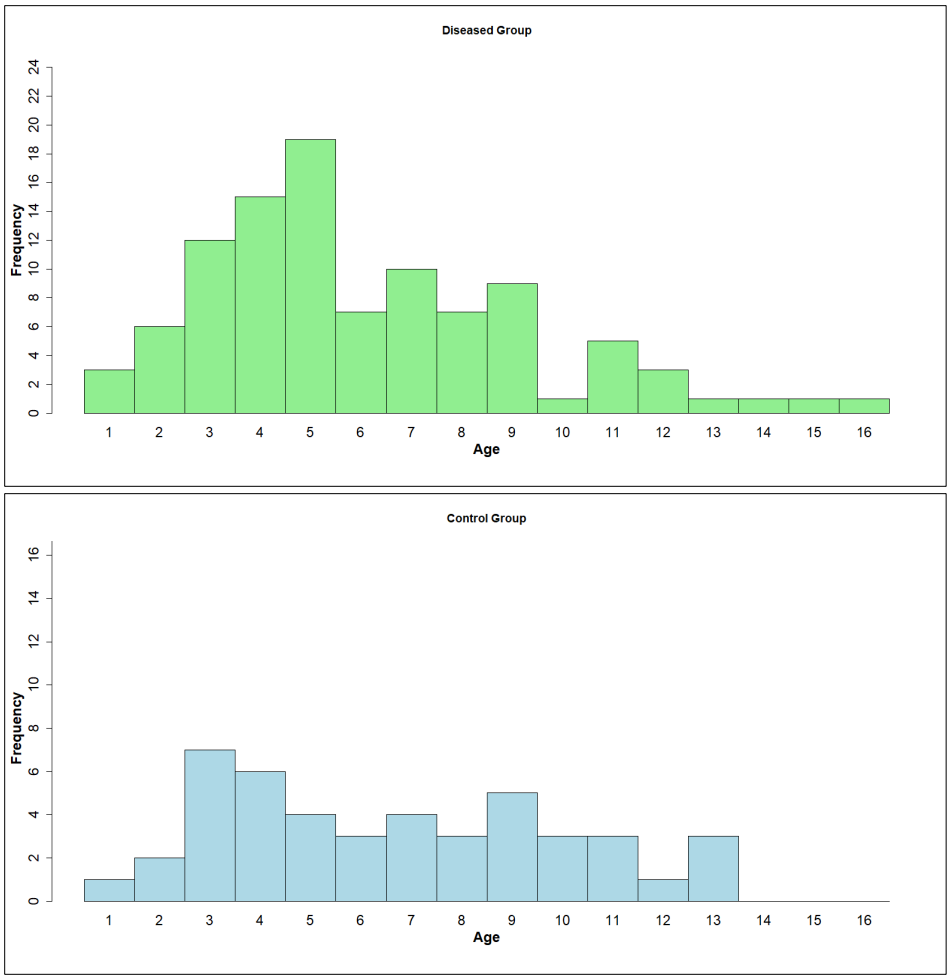

Figure 2. Distribution of ages at time of initial capture for female elk we captured, 2015–2017, that were affected by treponeme-associated hoof disease (Diseased Group) or had no visible signs of being affected by the disease (Control Group).

Disease Occurrence within Control Group

To date, we have marked and assigned 44 elk to our control group, of which, 14 are new study animals we captured for the first time in December 2017 (does not include the control elk that died during capture in December 2017). We have confirmed disease status for 25 of 30 elk we captured prior to December 2017, of which 0.48 (12/25) have contracted TAHD after we initially marked them. For elk within our control group that we captured during subsequent capture events, 0.25 (3/12), 0.22 (2/9), and 0.50 (3/6) in December 2015, 2016, and 2017, respectively, had contracted TAHD between capture events.

Disease Severity, Progression, and Recovery

We have continued to observe wide variation in hoof disease severity subsequent to our initial capture in February 2015. We initially developed grades of the disease that were related to a visual characterization of hoof deformities (Figure 3), but recognize our scoring system is subjective and may not exactly correlate with the effects of TAHD on the energy dynamics of elk. For example, we have preliminarily defined Grade IV of the disease to include any elk that is missing 1 or more hoof capsules, which would include an elk that recently sloughed its hoof capsule and is dealing with a painful, badly infected foot, and likely using a lot of energy fighting that infection. However, elk classified as having Grade IV may also include an animal that sloughed its hoof capsule several years prior and has, relatively speaking, healed and is no longer expending the same amount of energy it was when the hoof initially sloughed. Although we anticipate incorporating some measure of disease severity will strengthen the inferences we can make, our grading system is still evolving as we continue to increase our understanding of the disease during subsequent examinations of recaptured elk, from histology and microbiology examinations of hooves from study animals and hunter-harvested elk, and from evaluations of individual elk health status via clinical pathology of blood samples.

Severity.—We captured 103 elk that were affected by TAHD at the time of initial capture and we completed a full examination of all 4 hooves for 98 of them. The back hooves were involved in all 98 cases, only 1 back hoof was involved in 0.66 (65/98) of the cases, and both back hooves were involved in 0.26 (25/98) of the cases. It does not appear the rate at which TAHD involves the back right (57/98 = 0.58) or back left (66/98 = 0.67) hooves is disproportionate. The front hooves were involved in only 0.10 (10/98) of the elk we examined. The majority of elk within our diseased group either had TAHD on a single hoof with characteristics we have preliminarily associated with advanced stages of the disease (i.e., Grade 3 or Grade 4; 53/98 = 0.54) or had the disease on multiple hooves (33/98 = 0.34) (Figure 4).

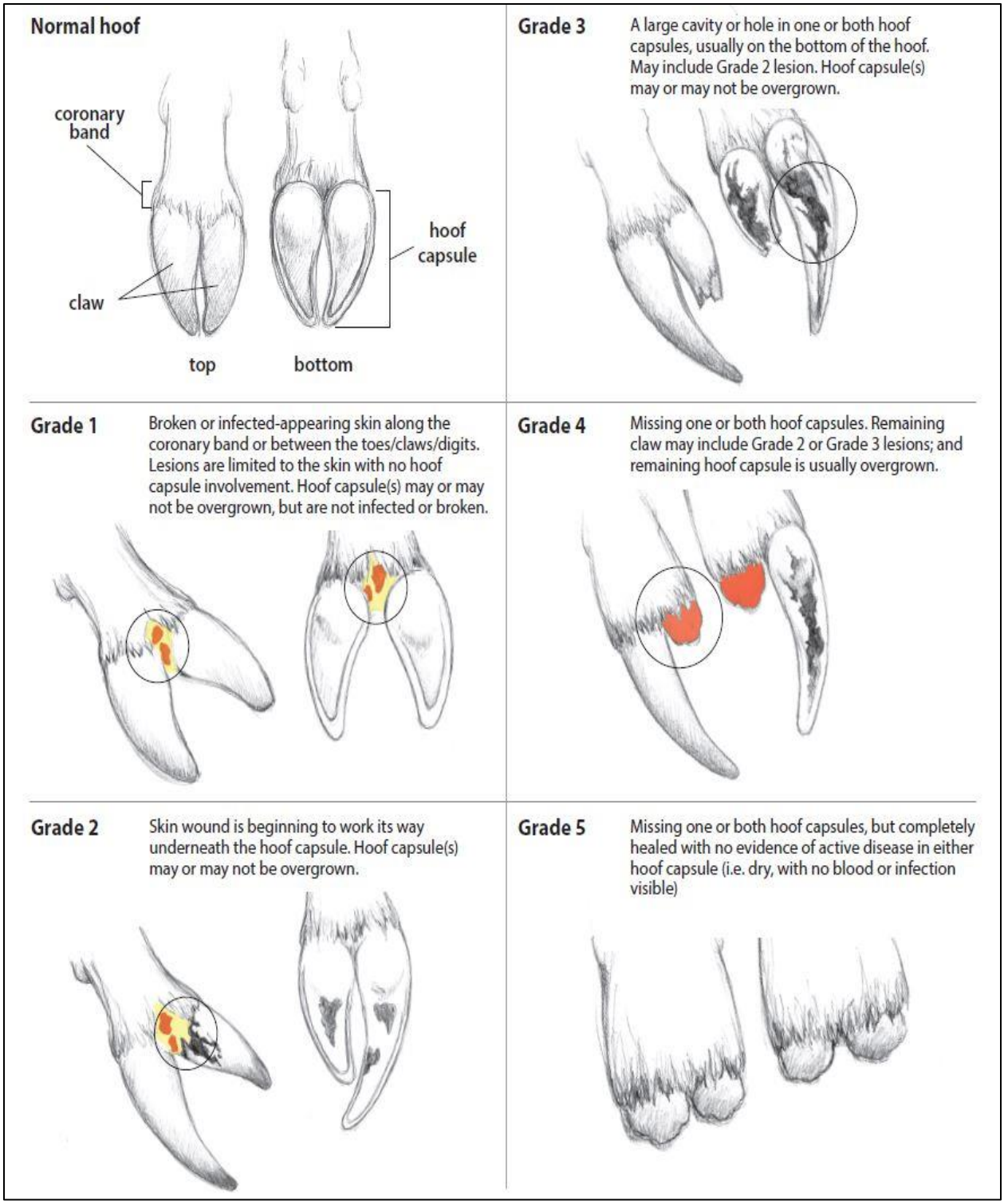

Figure 3. Diagram depicting characteristics we preliminarily associated with the 5 grades of treponeme-associated hoof disease we defined after capturing 60 female elk in February 2015, showing widely variable manifestation of the disease.

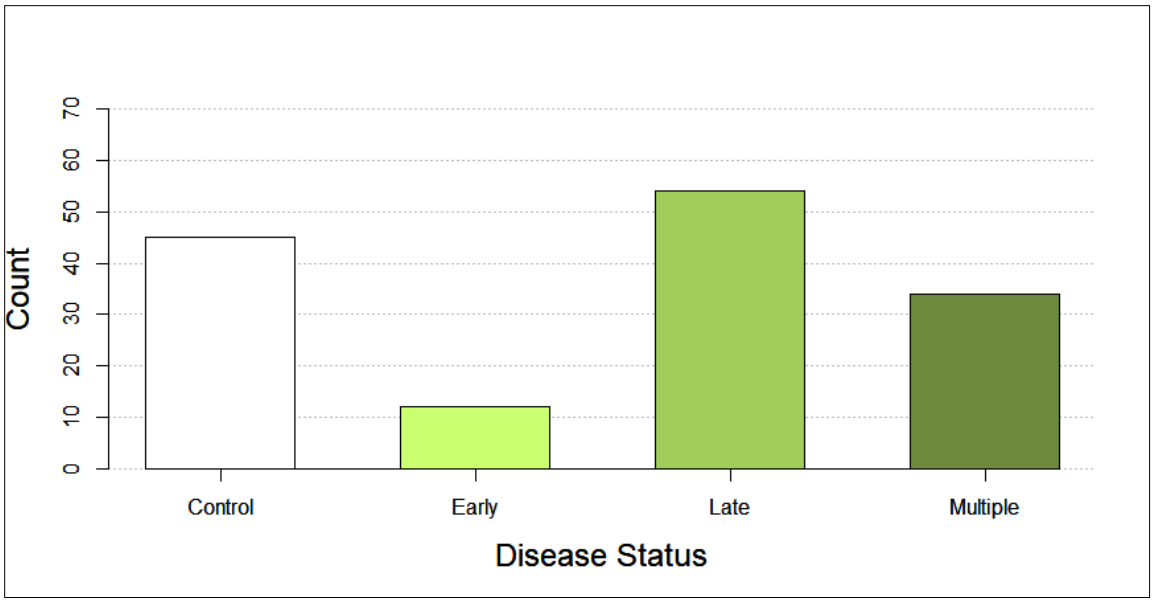

Figure 4. Distribution of hoof condition scores [Control, Early (Grade I or II), Late (Grade III or IV on a single hoof), and Multiple (present on multiple hooves)] at time of initial capture for female elk we captured February 2015–December 2017.

Progression.—We have recaptured 28 elk from our diseased group during subsequent capture events, which represented 36 hooves that were affected by TAHD during the previous capture. Of those 36 hooves, the disease progressed in 14, stayed the same in 16 (14 were Grade IV), had resolved in 6 (all were Grade I or Grade II), and 6 additional hooves had become involved. Five elk had progressed from having TAHD on a single hoof to multiple hooves, 13 had a single hoof involved during both captures, 4 transitioned from having multiple hooves involved to a single hoof, 4 had multiple hooves involved during both captures, and the disease had potentially resolved in 2 elk (Elk 161 and 162 both had Grade I on a single hoof the previous year; see below). In addition, 8 of the 27 elk from our control group had developed TAHD, with one of them having developed Grade IV on a single rear hoof between February 2015 and December 2015. Collectively, this information indicates that in many cases TAHD progresses quite rapidly and most individuals likely develop advanced stages of the disease within the first year of becoming infected.

Recovery.—We have only observed 1 case where an elk affected by TAHD had definitively recovered from the disease. We originally captured Elk 315 in December 2016, at which time we determined she had Grade II on her right hind hoof (Figure 5). She was subsequently legally harvested in November 2017 and formal examinations indicated all four hooves were grossly and

histologically normal, in addition to silver stains being negative for any spiral bacteria with typical *Treponema* morphology. We are not able to definitively claim the disease resolved in Elk 161 and Elk 162 because we only made that assessment during a gross examination of the hooves in a field setting.

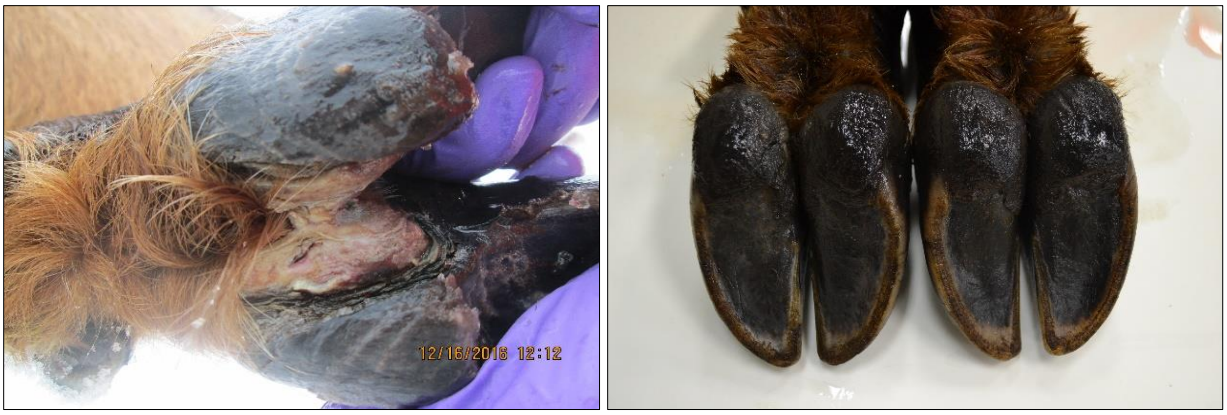

Figure 5. Photos of the right hind hoof from Elk 315 at time of initial capture on December 16, 2016 (left image) and photos of both rear hooves at time of histological examination at the Colorado State University Veterinary Diagnostic Laboratory, Fort Collins, Colorado, USA in 2017. The elk was legally harvested on November 5, 2017.

Body Condition

We determined body condition [i.e., percent ingesta-free body fat (IFBF)] at time of capture by having an experienced observer use a portable ultrasound to measure maximum subcutaneous rump fat thickness (MAXFAT) and determine a rump body condition score (rBCS) following the procedures of Cook et al. (2001*a*). We then used estimates of MAXFAT and rBCS to estimate IFBF at time of capture following the procedures of Cook et al. (2010). We also measured each elk's chest girth to estimate body mass following the procedures of Cook et al. (2003). Lastly, because lactation status has consistently been shown to be a primary determinant of the level of condition female elk are able to achieve in autumn (Cook et al. 2004, Cook et al. 2013), we classified elk as lactating (milk could be extracted from the udder) or non-lactating (milk was not present). The presence of milk indicated the female had been nursing a calf sometime within the previous 11 days (Flook 1970). Our non-lactating group undoubtedly included a combination of females that were not bred the previous autumn (true non-lactators), females that lost their calf at or near parturition, females that lost their calf at various times between parturition and capture,

and females that successfully produced a calf, but ceased lactating prior to capture. We pooled data December 2015–2017 to increase sample sizes.

Mean estimates of IFBF were consistently lowest for elk that were affected by TAHD, albeit those differences were minimal and have a low probability of being statistically significant, except for non-lactating elk in December (Table 2 and Figure 6). However, our current estimates include all elk affected by TAHD, irrespective of disease severity, which as discussed we cannot confidently quantify at this time. For example, 12 (6 lactating, 6 non-lactating) of the elk in our diseased group that we captured in December represented elk that had early stages of the disease, and given that we have learned the disease progresses quickly, there is a reasonable likelihood these elk spent a majority of the summer-autumn period unaffected by TAHD. Although sample sizes are small, our preliminary observations indicate the condition of adult female elk with early stages of the disease may be more similar to the condition of adult female elk within our control group.

Table 2. Mean estimates and associated 95% confidence intervals (CI) of percent ingesta-free body fat (IFBF) by disease and lactation status for adult female elk we captured in February and December in the Mount St. Helens elk herd area, 2015–2017.

	Non-Lactating						**Lactating**					
	Diseased Group			**Control Group**			**Diseased Group**			**Control Group**		
Season	***n***	$\bar{x}$	**CI**	***n***	$\bar{x}$	**CI**	***n***	$\bar{x}$	**CI**	***n***	$\bar{x}$	**CI**
February	56	4.2	3.6-4.7	19	5.1	3.9-6.2	---	---	---	---	---	---
December	46	5.8	5.2-6.5	16	8.5	7.7-9.2	36	5.3	4.7-6.0	31	6.3	5.7-6.94

Pregnancy

We determined pregnancy status at time of capture via ultrasonography and analysis of Pregnancy–Specific Protein B (PSPB) in serum samples collected during capture (Noyes et al. 1997). None of the elk we classified as yearlings (n = 4) were pregnant. For adult female elk, pregnancy rates have consistently been higher for our control group (range = 0.69–0.84) than for our diseased group (range = 0.32–0.59) (Figure 7). Overall, 50% (95% CI = 41–58%) of elk within our diseased group (n = 139) and 79% (95% CI = 67–87%) of elk within our control group (n = 66) have been pregnant. For comparison, McCorquodale et al. (2014) reported an overall pregnancy rate of 67% for the 109 adult female elk they captured 2009–2012.

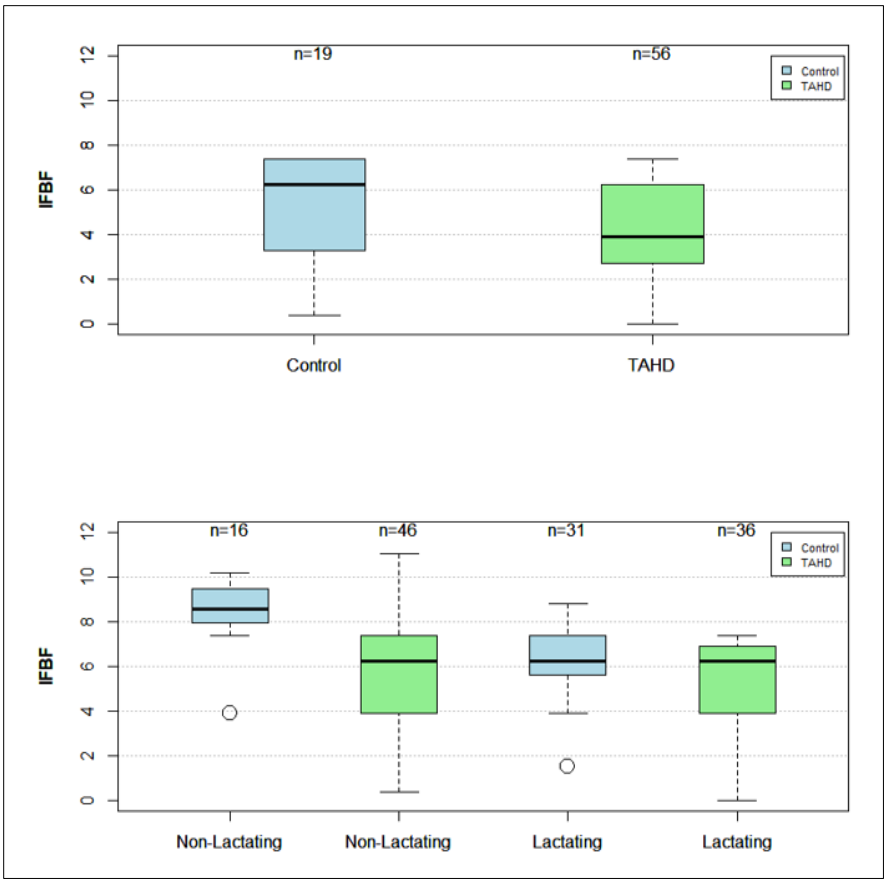

Figure 6. Boxplots of percent ingesta-free body fat (IFBF) by disease status for adult female elk we captured in the Mount St. Helens elk herd area February 2015 (top) and by disease and lactation status for adult female elk we captured December, 2015–2017 (bottom).

Productivity

In our original proposal, we defined productivity as the early survivorship of calves (e.g., to 6 months of age) and proposed we would estimate productivity using calf-at-heel ratios or lactation rates from hunter harvested elk. We have since abandoned those efforts and are only indexing calf survival using lactation rates observed in December and directly estimating calf survival from elk that we captured during subsequent capture events (i.e., we know what their pregnancy status was the previous year and assume a calf died if they were pregnant in $Year_t$, but not lactating in $Year_{t+1}$).

The proportion of adult female elk that were lactating at time of capture in December has ranged 0.63–0.69 for elk in our control group and 0.42–0.45 for elk within our diseased group (Figure 8). Overall, 0.66 (95% CI = 0.52–0.78) of elk within our control group (n = 47) and 0.44 (95% CI = 0.34–0.55) of elk within our diseased group (n = 82) have been lactating in December.

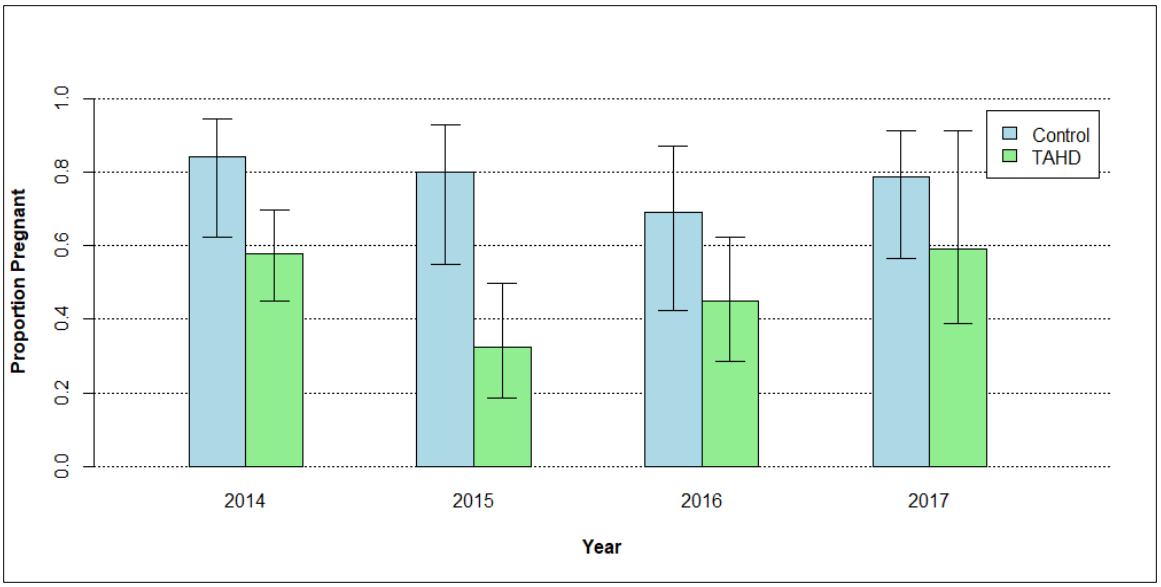

Figure 7. The proportion (and associated 95% confidence intervals) of adult female elk that were pregnant and affected by treponeme-associated hoof disease (TAHD) or had no visible signs of being affected by the disease (Control) at time of capture in the Mount St. Helens elk herd area, 2014–2017.

Although lactation rates were consistently lower for elk in our diseased group, they also had lower pregnancy rates, which indicates calf survival may not be substantially disparate between groups. Although inferences are limited by our small sample size, estimates of calf survival using pregnancy and lactation status of elk captured during subsequent capture events, also indicate calf survival to 6 months of age may be similar between groups. We estimated calf survival for our control group to be 0.60 (n = 10) in 2015, 0.75 (n = 8) in 2016, and 0.50 (n = 6) in 2017. Estimates of calf survival for our diseased group were 0.62 (n = 13) in 2015, 0.50 (n = 6) in 2016, and 0.67 (n = 3) in 2017. Overall, 0.63 of adult female elk within our control group where pregnancy status was known and 0.60 within our diseased group have successfully raised a calf through late-autumn.

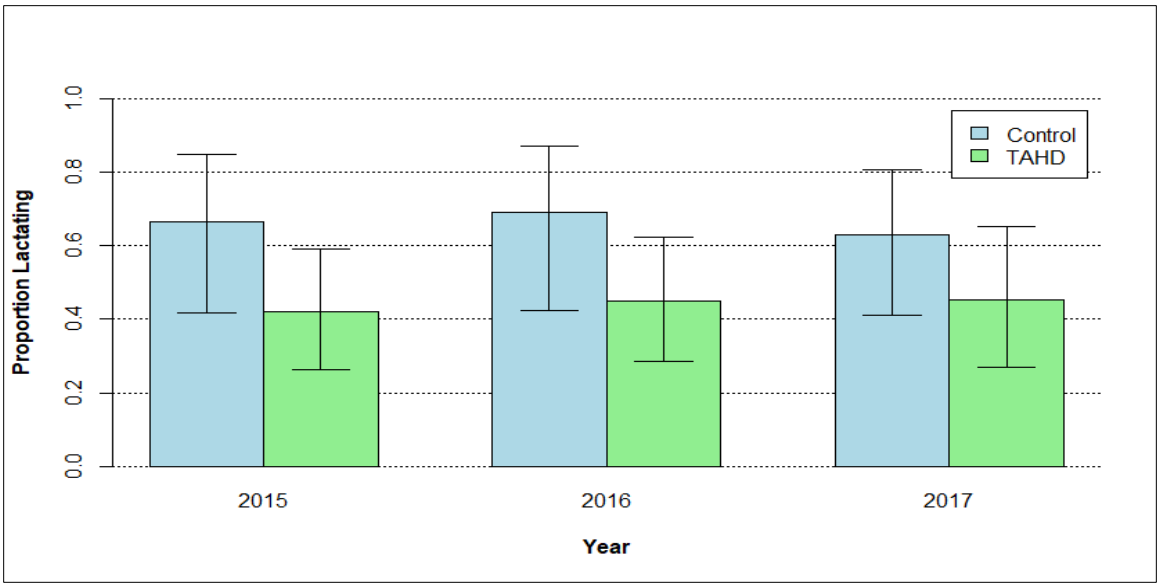

Figure 8. The proportion (and associated 95% confidence intervals) of adult female elk that were lactating in December and affected by treponeme-associated hoof disease (TAHD) or had no visible signs of being affected (Control), in the Mount St. Helens elk herd area, 2015–2017.

Survival

For our preliminary analysis, we estimated survival using the Kaplan-Meier estimator, modified for staggered-entry of individuals (Pollock et al. 1989). In addition to estimating survival since project initiation (i.e., March 2015–August 2018), we also estimated annual survival rates (i.e., May $1_{\text{Year t}}$–April $30_{\text{Year t+1}}$) and survival rates during 3 seasons that were biologically relevant to elk. These seasons included: 1) summer (May–August), the period of greatest nutritional demand for female elk supporting calves, 2) autumn (September–December), when the nutritional demands associated with lactation diminish and hunting seasons occur, and 3) winter (January–April), when elk primarily rely on fat reserves they accrued the previous summer-autumn period to meet their basic metabolic requirements.

In addition to censoring elk that died during or immediately following the capture process, we censored two mortalities from our survival analyses because, in both instances, the elk died within a couple weeks of their capture and we could not rule out capture-related stress as a contributing factor (e.g., Beringer et al. 1996). We also censored 1 elk from all analyses because she was originally captured in February 2015 as a control, missed in December 2015, and then her radio-collar quit transmitting in November 2016—thus, we have no way of knowing whether or not she had maintained her control status. In addition, we have had 5 radio-collars fail and subsequently

censored these elk from our analyses at the last point in time we received a GPS location transmission or determined the elk's status via VHF monitoring. Lastly, any elk within our control group that developed TAHD and had advanced stages of the disease was censored during the time period when disease status was unknown. For example, we censored the 3 elk confirmed to have lost their control status between February 2015 and December 2015 from our analysis during the period of February 2015–November 2015 and then brought them back into the analysis as a diseased elk in December 2015. We took this approach because we have no way of knowing when exactly they developed the disease. Lastly, we have had 2 control elk die within a few months of us capturing them (February and May, both captured the previous December) that had developed early stages of the disease by the time they died. In both instances, we kept them in the control group for this preliminary analysis. We believed this decision was justified given that disease progression appears to be quite rapid (i.e., they likely contracted the disease shortly before death) and they had spent the majority of the year as an elk unaffected by TAHD, which may have influenced their probability of survival during winter months. This decision will be considered more thoroughly as the project progresses.

Estimated survival since project initiation (i.e., March 2015–August 2018) has been 0.23 (95% CI = 0.16–0.29) for our diseased group and 0.37 (95% CI = 0.24–0.51) for our control group. Annual survival rates were similar between groups in 2017, but greater for elk in our control group in 2015 and 2016 (Table 3). Survival during summer has been similar between groups and among years within groups (Table 3). Substantial differences in estimates of survival between groups have primarily occurred during the winter season and survival of elk in both groups was lowest in winter 2016 when abnormally severe winter conditions persisted (Table 3). Although survival during autumn has not been markedly dissimilar between groups, and lower for elk in our control group 2 of 3 years, all 6 mortalities we have documented for elk in our control group during autumn have been human-caused (i.e., natural survival has been 1.00), compared to only 5 of 15 mortalities in our diseased group.

Table 3. Estimated survival rates (Ŝ) and associated 95% confidence intervals (CI) for elk affected by treponeme-associated hoof disease (Diseased Group) and for elk that were seemingly unaffected by the disease (Control Group) during 3 seasons of biological relevance to elk in the Mount St. Helens elk herd area, 2015–2017.

Diseased Group								
	Summer		**Autumn**		**Winter**		**Annual**	
Year	**Ŝ**	**CI**	**Ŝ**	**CI**	**Ŝ**	**CI**	**Ŝ**	**CI**
2015	0.93	0.86-0.99	0.92	0.85-0.99	0.80	0.70-0.90	0.68	0.57–0.79
2016	0.94	0.87-0.99	0.91	0.84-0.99	0.68	0.56-0.79	0.58	0.47–0.69
2017	1.00	–	0.86	0.76-0.96	0.75	0.65-0.86	0.65	0.54–0.76
Control Group								
	Summer		**Autumn**		**Winter**		**Annual**	
Year	**Ŝ**	**CI**	**Ŝ**	**CI**	**Ŝ**	**CI**	**Ŝ**	**CI**
2015	0.93	0.81-0.99	0.85	0.65-0.99	1.00	–	0.79	0.61–0.97
2016	0.94	0.81-0.99	1.00	–	0.83	0.66-0.99	0.78	0.60–0.97
2017	1.00	–	0.67	0.43-0.91	1.00	–	0.67	0.51–0.84

[1]Summer = May–August; Autumn = September–December; and Winter = January–April

Cause-specific Mortality

We have documented 86 mortalities (73 diseased group, 13 control group) since project initiation and attempted to investigate all deaths within 24 hours of receiving a message that a mortality event had occurred. In instances where the carcass was fully, or mostly, intact, we performed a field necropsy to determine proximate cause of death and to collect tissue samples that we submitted to the Colorado State University Veterinary Diagnostic Laboratory (CSU) for histological examination. Samples we collected and submitted to CSU included tissue samples from the heart, lungs, liver, kidney, spleen, pancreas, mammary gland, brain, popliteal and pre-scapular lymph nodes, any other tissues that seemed abnormal in appearance, and all 4 hooves. We also collected a femur and measured bone marrow fat content to estimate percent body fat at time of death (Neiland 1970). We were not able to collect all samples from every mortality event. We have received final histology reports from CSU for all but 3 mortalities to date, but have not completed bone marrow analysis for 8 elk that died April 2018–present.

To date, we have classified proximate causes of mortality as malnutrition (only applies to our control group), general debilitation (only applies to our diseased group), disease (non-TAHD),

human-caused (legal and illegal harvest), unknown, accident, and predation. Mortalities we classified as general debilitation were typically characterized by severe emaciation, the presence of advanced hoof disease, and no evidence of another primary disease based on histology of all major organs sampled. The emaciation observed in these animals indicates that they are in an extreme negative energy balance. However, we have no way of determining the relative contribution of the catabolic effects of a chronic severe disease such as TAHD (Demling 2009), compared to the catabolic effects resulting from nutritional limitations, such as those already known to occur in this herd (Cook et al. 2013, McCorquodale 2014), and how they may interact to affect the survival of elk. Mortalities we classified as disease (non-TAHD) have included cases where histological findings indicated the elk was afflicted by a severe case of pneumonia, severe renal disease, or septicemia. Lastly, mortalities we have classified as accidents have included 4 elk that have gotten stuck in bogs/mud, 1 elk that apparently drowned, and 1 elk that fell down an extremely steep and rocky slope—in all 6 cases the elk were in extremely poor condition, which we believe contributed to their plight.

Of the 13 mortalities we have documented for our control group, we have preliminarily classified 1 as unknown. Of the remaining 12, we have classified 6 (0.50) as human-caused (3 legal, 2 wounding loss, 1 illegal), which has been the leading cause of mortality (Figures 9 and 10). Of the 73 mortalities we have documented for our diseased group, we censored 3, 2 are pending histological findings, and have preliminarily classified 14 as unknown. Of the remaining 54, the leading causes of mortality have been general debilitation (0.44, n = 24) and predation (0.28, n =15). Most mortality events for our diseased group have occurred January–April (Figure 10). In instances where we have classified mortalities in our diseased group as general debilitation, predation, and unknown, 1.00, 0.83, and 0.89, respectively, have had bone marrow content levels indicative of severe negative energy balance.

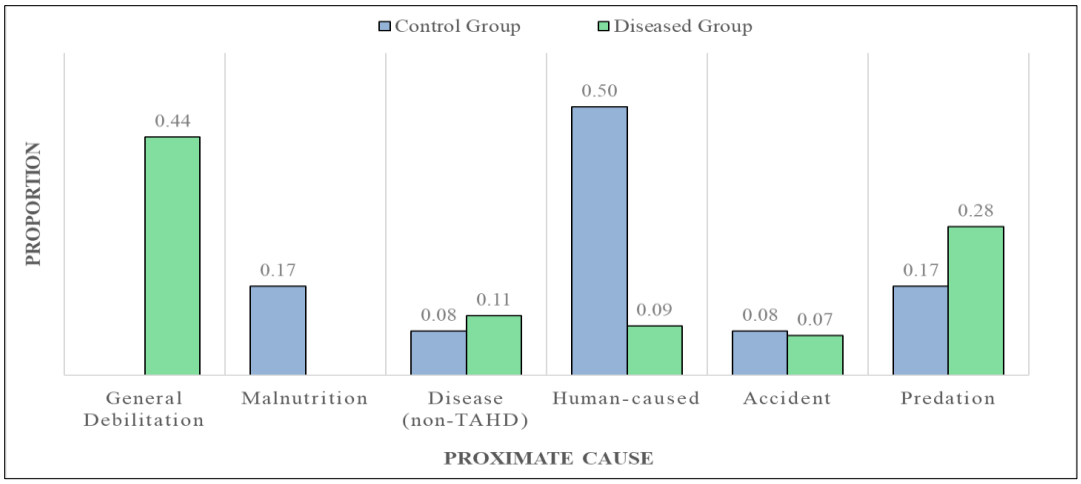

Figure 9. Proportion of deaths by proximate cause for adult female elk that were affected by treponeme-associated hoof disease (Diseased Group) or had no visible signs of being affected by TAHD (Control Group) in the Mount St. Helens elk herd area, February 2015–August 2018.

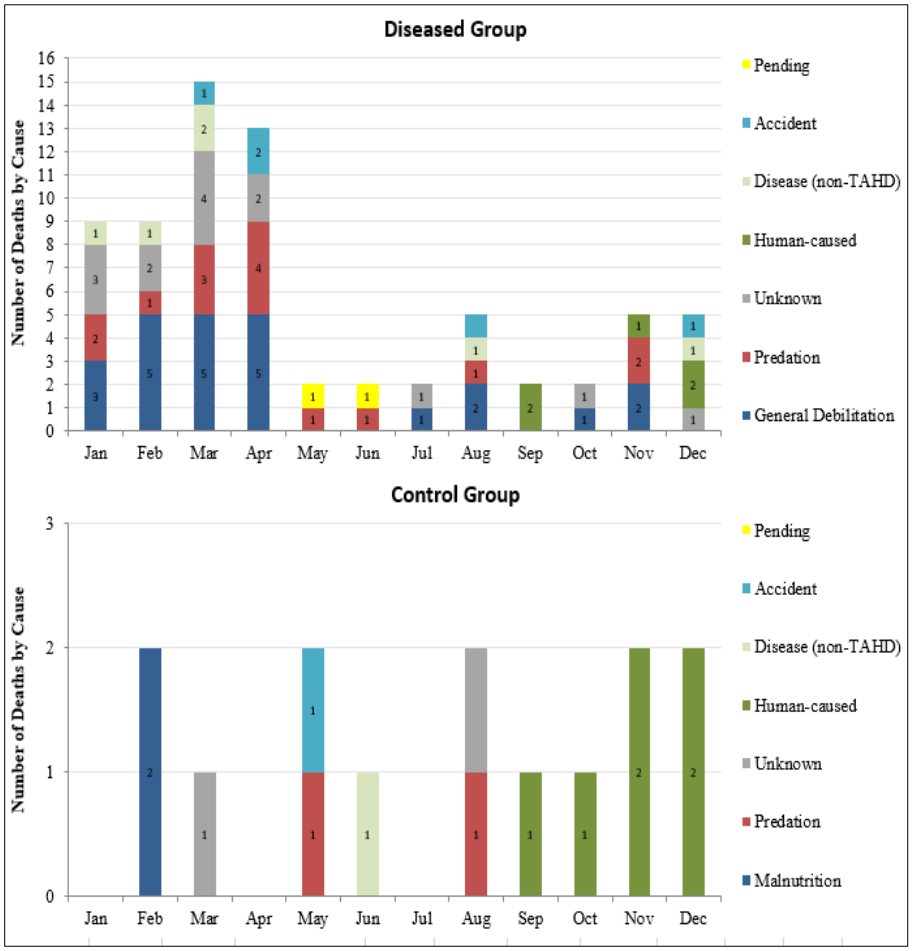

Figure 10. Number of deaths by cause and month for elk that were affected by treponeme-associated hoof disease (Diseased Group) or had no visible signs of being affected by the disease (Control Group) in the Mount St. Helens elk herd area, February 2015–August 2018.

DISCUSSION

It is far too soon for us to make any definitive statements that relate to our research objectives or to discuss our results in any detail. Preliminarily, elk affected by TAHD have had lower levels of condition in December, lower pregnancy rates, lower lactation rates, and lower annual survival rates. Our estimates of IFBF in December indicate elk in the Mount St. Helens elk herd area continue to experience strong nutritional limitations during late-summer and autumn, regardless of disease status. Irrespective of proximate cause, 0.88 of the mortalities we have documented for elk affected by TAHD, have included animals that had bone marrow content levels indicative of a severe negative energy balance. However, at this time we are not able to quantify the degree to which the catabolic effects of TAHD are contributing to those observations.

Our preliminary observations indicate that it will be important for us to consider disease severity when we complete our final analysis and we will continue to evaluate how we define disease status and severity as the study progresses. Similarly, we will continue to examine when we censor elk in our survival analysis that transition from our control group to our diseased group. At this point in time, we do not anticipate any changes to our study design and plan to conduct captures in December 2018.

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