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Disease Ecology and Adaptive Management of Brucellosis in Greater Yellowstone Elk

Gavin G. Cotterill
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DISEASE ECOLOGY AND ADAPTIVE MANAGEMENT OF BRUCELLOSIS IN
GREATER YELLOWSTONE ELK

by

Gavin G. Cotterill

A dissertation submitted in partial fulfillment
of the requirements for the degree

of

DOCTOR OF PHILOSOPHY

in

Ecology

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Disease Ecology and Adaptive Management of Brucellosis in Greater Yellowstone Elk

by

Gavin G. Cotterill, Doctor of Philosophy

Utah State University, 2019

Major Professor: Dr. Johan T. du Toit
Department: Wildland Resources

Elk (*Cervus canadensis*) and bison (*Bison bison*) of the Greater Yellowstone Ecosystem are hosts for the pathogen *Brucella abortus*, a bacterium that causes brucellosis, which can also be transmitted to cattle and humans. Test, cull, and quarantine protocols have eliminated the disease in cattle herds across the United States but these are impractical control measures for wide-ranging wildlife such as elk. Bison movements are strictly monitored to prevent comingling with cattle, but elk periodically transmit *Brucella* to cattle on rangeland where disease-induced abortions have recently occurred among infected elk. Supplemental winter-feeding offers one way to influence elk movements during part of the transmission season but simultaneously contributes to the persistence of brucellosis in the region. Using over 20 years of elk capture and testing data from fed elk on winter-feeding grounds in southwestern Wyoming, this dissertation investigates the role of winter feedgrounds in the ecology of this host-pathogen relationship, develops a fuller image of the ways in which the pathogen affects elk demography, and conversely how elk demography influences pathogen transmission. In Chapter 2, I performed a literature review of the effects winter feeding has on brucellosis...
in elk. In Chapter 3, I demonstrated a previously undocumented fertility cost associated with brucellosis exposure in elk which is not due to abortions, but which nearly doubles the estimated fertility cost to affected individuals. In Chapter 4, I used time-series serological and count data to build mechanistic SIR models for brucellosis in fed elk herds. Within that framework, I clarified the underlying transmission dynamics of the system, which allowed me to assess various management actions including test-and-slaughter of seropositive elk. In Chapter 5, I used elk in the western United States as a case study in wildlife management principles with particular attention to emerging issues in disease control. The overall picture that emerges of winter feedgrounds is one of tenuous practical compromise driven by social and political consideration, not pathogen control. The results from chapters 3 and 4 illustrate the underappreciated importance that recruitment and population turnover have on the dynamics of the system, wherein the pathogen flourishes in the reproductive tracts of individual animals and thus directly impacts vital rates at the population level. Together, these results contribute to the field of disease ecology using a unique long term disease data set of free-ranging wild ungulates by providing empirical results and mechanistic models which explain phenomena previously supported in theory, but rarely demonstrated in practice.
PUBLIC ABSTRACT

Disease Ecology and Adaptive Management of Brucellosis in Greater Yellowstone Elk

Gavin G. Cotterill

Brucellosis is a bacterial infection that primarily affects livestock and can also be transmitted to humans. In the Greater Yellowstone Ecosystem (GYE), elk (*Cervus canadensis*) and bison (*Bison bison*) are habitual carriers of *Brucella abortus*, which arrived to the region with cattle over a century ago. The disease was eliminated from cattle in the United States through widespread control efforts, but is now periodically transmitted back to cattle on open rangelands where they can come into contact with fetal tissues and fluids from disease-induced abortions that occur among elk during the late winter and spring. In Wyoming, south of Yellowstone National Park, there are 23 supplemental feedgrounds that operate annually and feed the majority of the region’s elk during a portion of the winter. The feedgrounds are controversial because of their association with brucellosis and may be shuttered in the future in part due to the arrival of chronic wasting disease. Using data collected at these feedgrounds, this study investigates the role of winter feedgrounds in the ecology of this host-pathogen relationship: it evaluates the full reproductive costs of the disease to affected elk, how herd demography influences pathogen transmission, and assesses management strategies aimed at reducing pathogen spread among elk. Using blood tests for pregnancy status and brucellosis exposure in female elk, I demonstrated a previously undocumented fertility cost associated with the pathogen which is not due to abortions, but which nearly doubles the estimated fertility cost to affected individuals. I also built mechanistic transmission
models using time-series disease and count data from feedgrounds. Within that framework, I assessed various management actions including test-and-slaughter of test-positive elk, which I found to be counterproductive due to rapid recovery times and the protective effects of herd immunity. The overall picture that emerges of winter feedgrounds is one of imperfect practicality driven by social and political consideration, not pathogen control. These results illustrate the underappreciated importance that recruitment and population turnover have on the transmission dynamics of brucellosis in elk, a pathogen which itself flourishes in the reproductive tracts of individual animals and thus impacts vital rates at the population level. Together, this study contributes to the field of disease ecology using a unique long term disease data set of free-ranging wild ungulates.
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I give special thanks to my family and friends, and especially the Raithels, who adopted me when I first arrived in Logan.

Gavin G. Cotterill
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CHAPTER 1
INTRODUCTION

Disease ecology research is motivated by both applied and basic considerations. Zoonoses directly impact human health, and only through a better understanding of how they operate in non-human host populations will we be able to predict their emergence and spread in human populations. Indirectly, the same is true for diseases that ‘spill over’ from wildlife to livestock. In both cases, conservation efforts will be harmed if humans come to perceive the wildlife hosts as pests. From a basic research perspective, there is increasing recognition of the outsized role pathogens play in evolution (Anderson & May, 1982), in classic ecological questions like population cycles (Anderson & May, 1980), and in population and community dynamics (Dobson & Crawley, 1994). The subject of this dissertation, brucellosis, touches on all of these facets.

Brucellosis refers to infection caused by any of the gram-negative, intracellular bacteria in the genus Brucella, of which there are several species, each with their own preferred hosts. These include: B. melitensis (goats), B. canis (dogs), B. suis (swine), and the species discussed herein: B. abortus (cattle). Of these, all are zoonotic. For these pathogens, humans are a dead-end, incidental host, but the symptoms of disease (sometimes referred to as ‘undulant fever’) can be awful, periodic, and clearing the pathogens can be difficult even with early detection (Young, 1995). As a case in point, human brucellosis has been known to reactivate after decades without sign or symptoms of illness (Meneses et al. 2009). Globally, it is the most common zoonosis and has enormous implications for human health and well-being, particularly in poor regions.
In the United States, food safety measures (pasteurisation of milk) and control measures in livestock have made the disease rare. Annual human cases in the United States measure in the tens, not hundreds or thousands, and are primarily an occupational hazard for veterinarians and workers in abattoirs (Heavey, 2019; Young, 1995).

*Brucella spp.* are pathogens whose niche is the mammalian reproductive tract. Erythritol (a sugar) is a preferred food source of the bacterium that is available in great quantity during fetal growth (Smith et al., 1962). The bacteria are notoriously adept at evading host immune responses, and are able to ‘hide out’ in extremely low numbers throughout the host organism including lymph nodes (Ahmed, Zheng, & Liu, 2016). Then, when the time is right (late in pregnancy), conditions allow them to proliferate, causing abortion. Among domestic livestock, this is an efficient means of transmission because of a tendency to be curious herd animals, eager to groom and inspect highly infective fetal tissues and fluids in their environment.

Apart from cattle, *B. abortus* also has the unfortunate distinction of infecting bison (*Bison bison*) and elk (*Cervus canadensis*) in the Greater Yellowstone Ecosystem (GYE). It likely arrived in North America in the late 1800s with cattle imported from Europe and quickly spread throughout the continental United States. In 1917, it is thought to have infected bison in Yellowstone National Park when infected cattle (which provided milk for park employees) were penned alongside bison (Meagher & Meyer, 1994). Although males of all species routinely contract the infection, studies have failed to demonstrate male to female transmission of *B. abortus* in elk (Thorne, Morton, Blunt, & Dawson, 1978) or bison (Robison, 1994). For these reasons, the general consensus is
that males are unimportant to brucellosis transmission.

By the 1930s, *B. abortus* infections in cattle across the U.S. were common, prompting the USDA to launch a nationwide eradication campaign. Through systematic, mandatory testing, vaccination, and cull-and-quarantine of affected herds, the disease was officially eliminated from U.S. cattle in 2000 (Ragan, 2002). This marked a significant accomplishment, but one that was short-lived. Although ‘spill back’ of brucellosis from wildlife to cattle had previously been suspected, since 2002, there have been 1-4 confirmed spillback events to livestock annually in the GYE (National Academies of Sciences, Engineering, and Medicine, 2017). Although bison were initially blamed for the spillback events, over time it became clear that elk were the source of contagion (Kamath et al., 2016). Spillback to cattle has financial repercussions for ranchers, but in the bigger picture, the existence of two wildlife reservoir hosts seriously undermines eradication efforts. Bison are not free-roaming in that their movement is carefully monitored and restricted. Nor does an interconnected population structure exist for bison across the region. By contrast, elk populations range freely across the western U.S., thus increasing the spatial extent of brucellosis. *B. abortus* is now present in elk in Montana, Wyoming, Idaho, and will probably soon be detected in Utah (National Academies of Sciences, Engineering, and Medicine, 2017). Genetic work confirmed that spillback events all came from elk, but also that elk and bison pass the pathogen between one another periodically (Kamath et al., 2016). This suggests that eradication will be impossible unless it is simultaneously accomplished in bison and elk. Additionally, disease-free bison herds in the region are at risk of exposure from elk, which has major conservation implications for the former. It seems doubtful that bison will ever be
functionally restored to landscapes outside of a few parks. Nevertheless, both private and public entities maintain bison herds, very few of which are free from cattle introgression. These disparate populations are important for the species’ continued existence. Should brucellosis be introduced to these herds (most likely via elk), then there will be enormous pressure from local cattle ranchers to depopulate bison herds, or at the very least maintain separation which may not be feasible in all cases.

The difficulty in maintaining wildlife-cattle separation is most apparent at Wyoming’s winter feedgrounds for elk. In Wyoming, south of Yellowstone National Park, there are 23 supplemental winter feedgrounds for elk. The National Elk Refuge in Jackson, WY, feeds the largest number of elk (by an order of magnitude) and is also the oldest, having been founded in 1912. The refuge annually feeds about 8000 elk (in recent years) and is operated by the U.S. Fish and Wildlife Service. The remainder are operated by the Wyoming Game and Fish Department with most having been in their current location since the 1960s (Dean et al., 2004). The original stated goals of winter feedgrounds were two-fold: (1) increase over-winter survival and elk abundance by providing food during the lean part of the year in areas where traditional winter range had been converted to residential and agricultural land, and (2) divert elk from private hay crops intended for livestock. Since their inception, disease concerns have existed at feedgrounds. Although various ailments occur (hoof rot, pasteurellosis, scabies), brucellosis has been the dominant concern since the spillback events of the early 2000s.

It has been known since well before the 1970s that feedground elk maintain the disease. Until recently it was thought that unfed herds could not maintain the disease absent constant reintroductions (National Research Council, 1998). That no longer
appears to be the case, with the disease spreading through the elk populations across WY. Nevertheless, fed elk herds likely play an important role in maintaining and spreading the disease. However, aside from some infected female elk aborting, the readily-observable effects of the disease on individual elk appear benign. This, despite seroprevalence at certain feedgrounds exceeding 60% in some years.

From a research perspective, the feedgrounds are an unusual opportunity to amass data because feedground elk are habituated to human presence, and corral traps can be used on-site to capture, mark, and test large numbers of elk relatively efficiently. However, some of the best data about brucellosis in elk comes from one captive study of elk that took place in the late 1970s (Thorne et al., 1978) in which individual elk were experimentally infected. It found that the most important means of transmission was through direct contact with fetal tissues and fluids resulting from abortions, that only about half of exposed female elk were infected and aborted, and that abortions occurred in the first year following exposure. Although the sample sizes in this study were low, it forms the basis for much of our knowledge regarding the disease in elk. Similar work is currently infeasible to conduct because the USDA classified all live *Brucella* organisms as ‘Select Agents’ in 1997 (National Academies of Sciences, Engineering, and Medicine, 2017), at a time when the importance of brucellosis research in elk and bison was not seen as vital to eradication efforts. At this point, the barrier to research imposed by Select Agent status contributes to the situation it seeks to avoid: the spread of brucellosis across the country.

By the standards of wildlife disease systems, brucellosis in feedground elk is fairly well-studied and can serve as a model for other disease ecology research, but in
part because of the impositions on captive research, much remains to be learned. The overarching goal of this study is to advance our understanding of brucellosis in elk and inform management. To do this, I used a long-term dataset collected by the Wyoming Game and Fish Department at their winter feedgrounds in the southern GYE.

REFERENCES


abortus in bovine contagious abortion. Nature, 193, 47–49. doi:
10.1038/193047a0


CHAPTER 2

WINTER FEEDING OF ELK IN THE GREATER YELLOWSTONE ECOSYSTEM AND ITS EFFECTS ON DISEASE DYNAMICS

ABSTRACT

Providing food to wildlife during periods when natural food is limited results in aggregations that facilitate disease transmission. This is exemplified in western Wyoming where institutional feeding over the past century has aimed to mitigate wildlife-livestock conflict and minimise winter mortality of elk (*Cervus canadensis*). Here we review research across 23 winter feedgrounds where the most-studied disease is brucellosis, caused by the bacterium *Brucella abortus*. Traditional veterinary practices (vaccination, test-and-slaughter) have thus far been unable to control this disease in elk, which can spill over to cattle. Current disease-reduction efforts are being guided by ecological research on elk movement and density, reproduction, stress, co-infections, and scavengers. Given the right tools, feedgrounds could provide opportunities for adaptive management of brucellosis through regular animal testing and population-level manipulations. Our analyses of several such manipulations highlight the value of a research-management partnership guided by hypothesis-testing, despite the constraints of the sociopolitical environment. However, brucellosis is now spreading in unfed elk herds while other diseases (e.g. chronic wasting disease) are of increasing concern at feedgrounds.

Therefore experimental closures of feedgrounds, reduced feeding and lower elk populations merit consideration.

1. INTRODUCTION

Central to many host-pathogen systems is the relationship by which infectious contacts increase with increasing host density. In wildlife, local aggregations often occur at sites of food provision, exemplified by winter feeding of elk (*Cervus canadensis*) at 23 locations across western Wyoming, USA. Unlike most anthropogenic food subsidies for wildlife, which exist incidentally (agricultural crops, garbage dumps) or intentionally in many small, widely dispersed loci (bird feeders), these feedgrounds are operated by government agencies and are utilised by an estimated 80% of the regional elk population [1]. Altogether, approximately 25,000 elk are fed on an annual basis [2]. Grass or alfalfa hay is generally provided *ad libitum* using horse-drawn sleighs except at the federally-managed National Elk Refuge (NER) where pelleted alfalfa is dispensed from trucks due to the large number of elk that winter there. To our knowledge, this feedground complex (figure 2-1) represents the world’s most concentrated institutional feeding programme for wildlife.

Institutional feeding began as early as 1907 and was formalised with the creation of the NER in 1912. The state of Wyoming assumed management of its first feedground in 1929 and the 22 they currently manage were mostly in place by the 1960s. They were initially established to support dwindling elk herds through the winter and provide a nutritional diversion from private haystacks, and remain popular with some sectors of the general public. They facilitate wildlife viewing and enhance sport-hunting opportunities
(important sources of revenue), limit competition on winter ranges with other ungulates, mitigate some aspects of livestock conflict, and locally offset winter starvation by elk. They are, however, implicated in disease concerns. Each feedground draws a herd of elk that congregates for weeks or months when individuals are perhaps most vulnerable to acquiring new infections. That feedgrounds facilitate disease transmission [3] has in itself created an additional reason for feeding elk — to separate them from cattle. Thus a cycle is perpetuated whereby feeding creates and mitigates the same problem: it enhances transmission among elk [3–6] while also limiting contact between elk and livestock in winter [7]. The way forward is murky and stakeholders should weigh the problems of feedgrounds maintaining disease against the opportunities of using them to adaptively manage disease. In this paper we review the effects of winter feedgrounds on disease ecology with a focus on brucellosis in elk in western Wyoming. We also offer suggestions for future research and management.

2. BRUCELLOSIS IN THE GREATER YELLOWSTONE ECOSYSTEM

For the last fifty years, much of the controversy surrounding the feedgrounds has focused on brucellosis. In the Greater Yellowstone Ecosystem (GYE), brucellosis is caused by the bacterium Brucella abortus and affects cattle, elk, and bison. Globally it is an important zoonotic disease, but human cases in the USA are generally occupation-related and rare [8]. The greatest burden now imposed by brucellosis in the USA is economic. Brucellosis causes abortions and sterility in cattle, thus state and federal livestock regulatory agencies impose restrictions on sale and movement of infected cattle herds which can reduce the profitability of affected and neighboring herds [9].
**a) Interspecific transmission among hosts**

Bison (*Bison bison*) and elk have contracted the disease from domestic cattle multiple times in the GYE since 1917 [10–12]. Although eradicated from cattle herds in the rest of the USA, brucellosis periodically spills back from elk to GYE cattle. Transmission occurs when susceptible animals have direct contact with aborted fetuses and other infective tissues and fluids [13,14]. Environmental persistence is relatively short-lived, as scavengers quickly remove infectious materials [5], although in cool, wet, shaded conditions the bacteria may remain viable for several months [15]. Thus, transmission requires either comingling or successive occupation of the same site within a limited time frame.

The respective roles of elk and cattle as reservoirs for brucellosis have changed over time, whereas the role of bison appears to have remained constant (figure 2-2). Our understanding, though, has shifted. Prior to the 2000s, bison were considered the greatest risk to cattle because they exhibit higher disease prevalence (~60%) than unfed elk (<5%), and while fed elk had higher seroprevalence (~20%) they were separated from cattle by the feedgrounds [3]. Intensive management operations preclude bison and cattle comingling [16], and spillback events to cattle in the GYE have all been attributed to elk [10,17]. Nevertheless, bison remain an important maintenance reservoir. Interspecific transmission between bison and elk has been documented via whole genome sequencing at the NER as well as in the free-ranging populations in Yellowstone National Park (YNP) [10], although there are currently insufficient data with which to estimate these rates. As a result, disease eradication is unlikely in one host without concurrent efforts across all hosts [18].
Similarly, prior to the 2000s there was broad consensus that free-ranging elk outside of the feedground complex were a non-maintenance population for brucellosis [14]. Although the spread of brucellosis in elk in most regions of the GYE traces back to the feedgrounds [10], more recently, it appears that higher levels of brucellosis seroprevalence in unfed elk herds unassociated with feedgrounds are self-sustaining, and in recent years there have been more cases of brucellosis in cattle away from, rather than in close proximity to, feedgrounds [6,7,16]. Because brucellosis prevalence is generally still higher among feedground than free-ranging elk, feedgrounds may reduce local transmission risk to cattle by facilitating elk-cattle separation.

(b) Intraspecific transmission in elk

Captive studies have failed to demonstrate male-to-female sexual transmission in elk, cattle, or bison [13,19,20], so that among and within species transmission have the same requirements with the possible exception of vertical transmission. Although calves born to infected elk exhibit a variety of outcomes, most are seronegative by 6 months of age [13]. Elk conceive in autumn and *Brucella*-induced abortions occur mainly during the third trimester due to mechanisms that are not perfectly understood (see [21]). About 50% of elk abort their first pregnancy following infection, after which the majority are thought to be recovered with immunity [13,22]. Winter feedgrounds operate between December and April, and *Brucella*-induced abortions peak between March and May [23]. Studies measuring contact and seroprevalence at different scales suggest that the probability of intraspecific transmission is correlated with elk density and aggregations along the feedlines [5,18,24].
Increased prevalence in unfed elk populations (figure 2-3) is similarly correlated with elk density and group size [6,25]. Elk group sizes in the GYE have a right-skewed distribution whereby most groups are small (e.g. <10), but most of the individuals in the population occur in groups of several hundred to several thousand [26]. These large groups likely play a disproportionate role in brucellosis maintenance and spread [6,18]. As unfed elk herds in the GYE have grown, so too have regional density and large winter aggregations associated with increasing brucellosis prevalence [25,27]. These large, unfed groups occur most frequently on private land or public land with late-season management closures where elk can escape hunting pressure, are larger on grasslands, and even larger still on irrigated land [25,26]. Irrigated land may represent another form of anthropogenic food subsidy which is largely outside of management control and poorly studied in the context of disease ecology.

A potential hidden cost associated with feedgrounds is increased stress, which can increase disease susceptibility [30,31] and enhance intraspecific transmission. Forristal [32] compared levels of fecal glucocorticoids (fGCs), a stress hormone, between fed and unfed elk during winter and found higher levels in fed populations. Feedgrounds differ in their localised elk density, predator densities, and human activity, all of which could lead to elevated fGCs. Agonistic behavior may also increase at feedgrounds as the result of elk sex- and age-class mixing that normally does not occur in winter.

Likewise, co-infections play an important role in the susceptibility, duration, transmission, and expression of diseases [33–36]. Cytokines, which are cell-signalling proteins that mediate a host’s anti-parasitic response, can be modulated by parasites themselves, and thus have been proposed as a useful way to gauge interactions like
competition or synergism between co-infecting parasites [37]. Some evidence for synergism between *B. abortus* and the weakly pathogenic *Trypanosoma cervi*, with which Wyoming elk are chronically infected [38], has recently emerged [39]. Both appear to share a strategy in which they upregulate host production of the cytokine interleukin-10 (IL-10), which can impair immune response and facilitate chronic infections [40–43]. This effect may also hinder vaccine efficacy [39,43]. Any number of diseases carried by elk have the potential to interact in ways that are relevant to feeding. *T. cervi* provides a useful illustration for this line of questioning which is relatively new to wildlife disease. Methods exist for quantifying elk cytokines using reverse transcription real-time polymerase chain reaction [44], and present a new approach to assess the effects of winter feeding on elk health.

(c) Scavengers and predators

*Brucella*-induced abortions are infrequently detected on feedgrounds, in part because scavengers quickly consume or remove the fetuses [5,15,24]. Although transmission has occurred under experimental conditions, scavenger species are not thought to be important vectors for the spread or maintenance of brucellosis [45–48], and have the potential to mitigate transmission [18,49]. Coyotes (*Canis latrans*) are important fetal scavengers and feedgrounds have higher scavenging rates than unfed locations [5,15,24]. This has important implications for increasing prevalence of brucellosis in large, unfed aggregations of elk, as coyotes can be hunted year-round in most of the western USA, but benefit from relative protection at established feedgrounds. The effects of bears (*Ursus arctos* and *Ursus americanus*) on disease dynamics of the feeding
grounds is unknown, but is likely minor because they are still hibernating for some of the transmission season.

Wolves (Canis lupus) have expanded their range since their reintroduction to Yellowstone National Park in 1995 and routinely kill elk at some feedgrounds. On several occasions wolves have chased elk off of feedgrounds, effectively halting feeding operations for most of the winter. Their impacts on feeding operations and winter elk survival at the NER have been minimal, but wolf presence is associated with an increase in the proportion of the Jackson herd that attend feedgrounds. Hunting by humans could have a similar effect, with most hunting occurring in the fall and winter, and hunter avoidance by elk (movement to private land) being well documented in the western USA [50–52]. In areas where elk are sensitized to the risk of predation by both humans and wolves, elk may seek refuge at feedgrounds or on private land where hunting is restricted, leading to dense aggregations with increased brucellosis transmission risk [48].

The effect of wolves on winter aggregations of elk has yet to be fully explored. Wolf presence is associated with larger elk groups [26], but it is unknown whether wolves are simply following large numbers of elk, if the aggregation pattern represents a defensive behavior in response to predators, or both. Finer spatial resolution is needed to assess the effect of wolves on brucellosis transmission in the context of winter feeding, since stagnant elk herds or elk returning to feedlines would be more likely to encounter fetuses compared with groups that spend more time on ‘fresh ground’ as a consequence of evading predators. The potential attraction of wolves to feedgrounds represents an additional concern to neighboring ranchers where displacement of elk to private property may increase spillback risk or result in incidental livestock depredation.
3. ADAPTIVE MANAGEMENT

Wildlife feeding programmes provide valuable opportunities for learning about disease dynamics in free-ranging populations. The GYE elk feedground system is currently operated – to the extent allowed by logistics, funding, and politics – within an adaptive management framework to allow outcomes-based comparisons of alternative interventions [53]. Our experiences with this system underscore the need for a priori considerations of statistical design: treatments and controls applied with randomisation, replication, stratification, and calibration. Finally, alternative formulations of plausible models that account for a broad range of ecological and disease dynamic processes will speed up the learning process.

(a) Vaccination

Feedgrounds offer enormous potential for vaccine delivery. The Wyoming Game and Fish Department (WGFD) began vaccinating feedground elk by airgun in 1985 [54] using a vaccine developed for use in cattle [55] and later used in elk [56]. By the time the program ended in 2015, coverage exceeded 97% of elk calves, but there was no significant reduction in seroprevalence or abortion events [57]. Were an effective vaccine available, feedgrounds could facilitate an annual ‘doctor’s visit’ to reduce contagion and risk. Unfortunately, the tools developed for use in cattle have yet to overcome either the immunological differences of elk, or some other unknown element such as co-infection [39]. Vaccine-development efforts are further constrained by the Select Agent status designated to *B. abortus* in the USA, which increases the regulations associated with handling live *Brucella* cultures. Should a new vaccine be developed, at least half of the
female elk population would require vaccination [22], which may only be feasible where elk are fed. It is also vital that any new vaccine not impair surveillance efforts, i.e. that the vaccine strain be discernible from the pathogenic strain in serologic tests.

While empirical results ultimately contributed to the cessation of elk vaccination, the effects (or lack thereof) may have been more readily apparent with better implementation of experimental design. Initially there was only one control site and until recently there were no competing models to explain potential changes in seroprevalence (e.g. altered feeding seasons). In addition, treatments could have been phased in or out over different years across sites in order to control for annual variation and allow for cleaner pre-post comparisons.

(b) Test-and-slaughter

Experimental removal of seropositive elk took place on the Muddy Creek, Fall Creek, and Scab Creek feedgrounds between 2006 and 2010. Seroprevalence decreased among yearling or older female elk from 37% to 5% over the 5 years of treatment at Muddy Creek, with approximately 50% of yearling and older females being tested. Elk were removed at Scab Creek and Fall Creek for only two years. While prevalence dropped at all three feedgrounds during treatment, prevalence among elk at Scab Creek was higher post-treatment compared to pre-treatment, and there was minimal change at Fall Creek (figure 2-4). Some of this variation can likely be explained by pre-treatment seroprevalence and feeding season lengths. Both Scab Creek and Fall Creek had lower pre-treatment disease prevalence than Muddy Creek and Fall Creek feedground has the shortest-duration feeding season among feedgrounds; see section d). This experiment
suggests that multiple years of test-and-slaughter are required to reduce seroprevalence but the varying treatment periods, limited pre-treatment data, and different outcomes across sites, all limit the strength of that conclusion. Culling based on serology results might be useful where a feedground closure is anticipated, but absent significant changes to aggregation patterns during the transmission season, depleting the pool of recovered (and still seropositive) animals could lead to more infectious contacts in subsequent years and a return to pre-treatment prevalence [58,59].

(c) Density manipulation

A relatively low-cost method to reduce elk densities during feeding is by distributing feed over a larger area. The probability of a susceptible elk becoming infected with brucellosis is correlated with contact rate and duration of contact with infected-aborted fetuses, which both increase with local elk density and are thus elevated on feedgrounds [18]. Therefore, management strategies which reduce adult-fetus contacts [4] and adult-adult contacts [60] should lead to reduced disease transmission. In one experiment, *Brucella*-free elk fetuses were randomly placed along feedlines under high and low-density treatments [4]. Under low-density conditions, the number of fetal contacts fell dramatically, and elk density itself dropped by 80%.

Low-density feeding has been adopted on 9 state feedgrounds, although uniform implementation at these sites has been logistically constrained. Available space for feeding differs among locations, leading to non-random selection of treatment sites, and the experience level of elk feeders, who must work a team of horses through deep snow, is variable. Both of these concerns have the potential to obscure treatment effects.
(d) Shortened feeding season

In a comprehensive study, over 55% of the spatial variation in brucellosis seroprevalence among feedground elk was explained by the length and ending date of the supplemental feeding season using the average season end-date for a feedground over the previous 8 years [61]. This also appears to be the case over time at the two sites for which there is strong longitudinal sampling (figure 2-5). This relationship is likely a function of abortion events driving the spread of the disease, and the timing of abortion events, which peak between March and May [23]. As with low-density feeding, WGFD has adopted earlier end dates at several feedgrounds. In order to mitigate the potential risk of displacing elk onto private property, targets for ‘early’ ending are based on relative snow conditions and when elk would normally depart voluntarily, as opposed to calendar date. This approach reduces risk to cattle, but limits our ability to evaluate treatment effect.

(e) Temporary sterilisation

One recently proposed intervention is the use of a gonadotropin-releasing hormone (GnRH) vaccine to temporarily sterilise female elk and bison [58,62]. In both captive and free-ranging elk, it reduces pregnancy for 1-3 years following a single dose [62,63]. It neither disrupts pregnancy upon initial delivery [66] nor affects the reproductive development of offspring [64]. In theory, it could be selectively administered to infected animals to reduce the risk of abortions in years 2 and 3, however most abortions are thought to occur in year 1. This is further complicated by imperfect detection of brucellosis and the potential for reductions in population growth (see [58] for an in depth discussion in the context of bison). Importantly, if transmission is driven by
animals aborting in the season in which they become infected, then sterilisation will not be effective. If, however, successive abortions in the years following initial infection are more important than limited captive studies have suggested, then targeted GnRH vaccination should circumvent transmission events and increase herd immunity. Many of the feedground herds are considered to be ‘over management objective’ so that some loss of reproduction could help accomplish desirable disease and population goals, but careful consideration of which animals to target for vaccination is required.

4) CONCLUSION AND FUTURE DIRECTIONS

Prevalence of brucellosis remains high in fed elk and has become self-sustaining in unfed herds too. Past control efforts at feedgrounds, including vaccination and test-and-slaughter, have not changed the dynamics of this host-pathogen system. Current efforts, including density and feeding-duration manipulations, have yet to be thoroughly assessed but from current serology they seem unlikely to resolve brucellosis by themselves. Temporary sterilisation is an option, but it remains unclear how this could or should be implemented. This leads to the inevitable discussion of closing feedgrounds and/or reducing feeding operations. Neighboring states have closed feedgrounds in the past, although none have operated on the same scale as Wyoming and so serious questions remain. Could the winter range support the current elk population without supplemental feeding? If not, should feedground closure be combined with culling and how would that be received by the public? If feedgrounds were closed, where would elk spend the winter? How much additional hazing would be necessary to achieve the same spatiotemporal separation between elk and cattle? If some feedgrounds were phased out
before others, would that cause larger elk aggregations at the remaining ones? Are private refugia and irrigated fields essentially functioning as feedgrounds outside of management control? Answers to such questions have real implications for not only disease management but also ranching, hunting and guiding, non-consumptive tourism, highway safety, and animal welfare ethics.

So far, continued feeding has mollified most stakeholders within Wyoming. However, changing disease patterns, including the arrival of chronic wasting disease (CWD) (see [65]), might shift the balance of opinion. CWD has now been detected in mule deer (Odocoileus hemionus) or moose (Alces alces) in two of the seven elk herd units containing feedgrounds, although no cases have yet been reported in elk in those areas. Unlike brucellosis, CWD is fatal in cervids. It is transmitted both directly and indirectly [66], persists in the environment [67], and the known transmission routes continue to expand [68–72]. Feedgrounds are poised to concentrate infectious material and spread the disease among elk and other susceptible ungulates that utilise or pass through those areas. CWD likely represents a much bigger threat to cervid populations in the GYE than brucellosis, but has not yet been shown to infect cattle [73] or humans [74]. Unless an efficacious vaccine against CWD becomes available, the only useful applications of feedgrounds to CWD management could be surveillance and removal of infected animals, the benefits of which would likely be outweighed by the risks of concentrating and spreading CWD.

Despite the risks, the continued adaptive management of GYE feedgrounds, together with experimental closures, will enhance knowledge on disease dynamics and the consequences of terminating feeding should that be deemed necessary. Winter
feeding in the southern GYE draws in most of the local elk population to fixed locations for at least three months each year. By using baited corral traps and restraint chutes at these feedgrounds it is possible to handle, mark, and sample hundreds of elk each winter. A before-after control-impact study could assess the interactive effects of supplemental feeding on host stress, the microbiome, reproduction, immune function, and disease susceptibility, with marked elk sampled for several years before and after experimental closures. Fecal pellets and blood from restrained elk can be used to determine relative stress levels, pregnancy, bacterial killing ability, cytokine levels, genetics and brucellosis serostatus. Fine-resolution spatial data on wolf and elk movement could elucidate predator-host-disease interactions during winter that catalyze or antagonize brucellosis transmission. Together with the results of previous and ongoing telemetry studies, such information would provide early insight into the effects of feedground closures on elk population, movement, and disease ecology.

**DATA ACCESSIBILITY**

The datasets and code supporting this article are available online:


**REFERENCES**


19. Thomsen A. 1943 Does The Bull Spread Infectious Abort-Ion in Cattle?


32. Forristal VE, Creel S, Taper ML, Scurlock BM, Cross PC. 2012 Effects of supplemental feeding and aggregation on fecal glucocorticoid metabolite


There are 23 supplemental winter feedgrounds for elk in Wyoming. The National Elk Refuge, north of Jackson, is operated by the US Fish and Wildlife Service while the remainder are operated by the Wyoming Game and Fish Department.
Figure 2-2. Maintenance and reservoir hosts for *B. abortus* in the GYE during the three stages of the disease to-date. Initially (A), cattle (bottom left) were a source population that infected bison (top left), fed elk (top right), and unfed elk (bottom right). After effective control measures were implemented in cattle (B) they were no longer a maintenance host but could be re-infected from fed elk. After 2000 (C), unfed elk became part of the reservoir community, able to maintain the infection in the absence of other host populations and are now a source of infection to cattle. Arrows depict established transmission paths. Arrow thickness denotes relative importance.
Figure 2-3. Seroprevalence trends from three unfed herd units in western Wyoming. Historically unfed herds rarely exceeded 5% prevalence, however, seroprevalence of some unfed herds has crept upwards, now reaching the 10-40% seroprevalence observed on feedgrounds. Circle radius represents sample size, smoother lines were fit using generalised additive models with the mgcv package [28] in R [29].
Figure 2-4. Test-and-slaughter of seropositive female elk between 2006 and 2010 reduced seroprevalence for brucellosis at the Muddy Creek feedground from 37% to 5%. In comparison, Scab Creek and Fall Creek received two years of treatment, the impacts of which are less clear. Points represent the proportion of seropositive animals tested in a given year, with 95% confidence intervals (bars). Vertical dotted lines represent years in which test-and-slaughter occurred at each site. Smoother lines were fit using generalised additive models with the mgcv package [28] in R [29].
Figure 2-5. Seroprevalence estimates (empty circles, scaled to sample size) and rolling average feedground end date in the previous 8 years (solid circles) at Dell Creek and Greys River feedgrounds. Smoothed seroprevalence estimates (lines) were fit using generalised additive models in the mgcv package [28] in R [29].
CHAPTER 3

HIDDEN COST OF DISEASE IN A FREE-RANGING UNGULATE: BRUCELLOSIS REDUCES MID-WINTER PREGNANCY IN ELK.

ABSTRACT

1. Demonstrating disease impacts on the vital rates of free-ranging mammalian hosts typically requires intensive, long-term study. Evidence for chronic pathogens affecting reproduction but not survival are rare, but have the potential for wide-ranging effects. Accurately quantifying disease-associated reductions in fecundity is important for advancing theory, generating accurate predictive models, and achieving effective management.

2. We investigated the impacts of brucellosis (*Brucella abortus*) on elk (*Cervus canadensis*) productivity using serological data from over 6000 captures since 1990 in the Greater Yellowstone Ecosystem, USA. Over 1000 of these records included known age and pregnancy status.

3. Using Bayesian multilevel models, we estimated the age-specific pregnancy probabilities of exposed and naïve elk. We then used repeat-capture data to investigate the full effects of the disease on life history.

4. Brucellosis exposure reduced pregnancy rates of elk captured in mid- and late-winter. In an average year, we found 60% of exposed 2-year-old elk were pregnant

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compared to 91% of their naïve counterparts (a 31 percentage point reduction, 89% HPDI = 20-42%), whereas exposed 3- to 9-year-olds were 7 percentage points less likely to be pregnant than naïve elk of their same age (89% HPDI = 2-11%). We found these reduced rates of pregnancy to be independent from disease-induced abortions, which afflict a portion of exposed elk.

5. We estimate that the combination of reduced pregnancy by mid-winter and the abortions following mid-winter reduces the reproductive output of exposed female elk by 24%, which affects population dynamics to a similar extent as severe winters or droughts. Exposing hidden reproductive costs of disease is essential to avoid conflating them with the effects of climate and predation. Such reproductive costs cause complex population dynamics, and the magnitude of the effect we found should drive a strong selection gradient if there is heritable resistance.

1 | INTRODUCTION

Acute infections that increase host mortality garner broad interest. By virtue of their pathogenicity they can locally threaten species persistence (Frick et al., 2015; McCallum, 2012) and trigger disturbances that reverberate through the ecosystem (Holdo et al., 2009; Hollings, Jones, Mooney, & McCallum, 2013). By comparison, the effects of chronic infections are understudied. Parasites of low pathogenicity certainly have the potential for population-level effects (McCallum, 1994), but isolating the effects of chronic disease from other environmental stressors requires intensive long-term study (P. C. Cross et al., 2009; Gorsich, Ezenwa, Cross, Bengis, & Jolles, 2015; Jolles, Cooper, & Levin, 2005). Chronic diseases may impact host reproduction either directly by reducing
pregnancy or causing abortions, or indirectly by reducing energy reserves and body condition. In both cases, the importance of disease may be underappreciated. As a case-in-point, some elk \((Cervus canadensis)\) herds in the Greater Yellowstone Ecosystem (GYE) of the western USA have a high prevalence of brucellosis - a chronic reproductive disease - yet the costs to elk have long been considered “unimportant as a practical matter” (National Research Council, 1998). This is despite the hallmark symptom of the pathogen, and its primary means of transmission, being abortion (Thorne, Morton, Blunt, & Dawson, 1978). In the GYE, brucellosis is caused by the bacterium \(Brucella abortus\) and is routinely detected in elk, bison \((Bison bison)\), and occasionally domestic cattle. Significant reductions in bison reproduction and recruitment have been attributed to this disease (Fuller et al., 2007; Geremia et al., 2008; Thompson Hobbs et al., 2015), but attempts to formally characterize the reproductive consequences of this disease in elk have lagged. There is no evidence that brucellosis affects survival in bison (Fuller et al., 2007) or elk (Benavides et al., 2017), and limited captive trials with elk in the 1970s suggested that major reproductive costs were limited to the first year post-infection (Thorne et al., 1978). More recently, Oldemeyer, Robbins, & Smith (1990) observed non-significant declines in calf production by seropositive female elk, while Foley et al. (2015) were unable to detect changes in calf:cow ratios in herds where prevalence for the disease was comparatively high.

Elk vital rates in the GYE have been the subject of intense scrutiny since the reintroduction of gray wolves \((Canis lupus)\) in the 1990s, which has also coincided with increasing brown bear \((Ursus arctos)\) (Schwartz, Haroldson, Gunther, & Moody, 2006) and cougar \((Puma concolor)\) numbers (Clark, Rutherford, & Casey, 2013). As with many
ungulate species, adult elk survival is generally high, so that population dynamics are largely driven by variable recruitment (Cole et al., 2015; Middleton, Kauffman, McWhirter, Cook, et al., 2013; Raithel, Kauffman, & Pletscher, 2007) and numerous studies have evaluated the relative influences of top-down and bottom-up mechanisms on elk productivity and abundance (Creel, Christianson, Liley, & Winnie, 2007; Mech, Smith, Murphy, & MacNulty, 2001; Middleton, Kauffman, McWhirter, Jimenez, et al., 2013; Proffitt, Cunningham, Hamlin, & Garrott, 2014; White et al., 2011). Reduced elk pregnancy due to nutritional limitation is well established (Cook et al., 2004) as a demographic response to adverse climatic conditions. For example, a 7% difference in yearling pregnancy and 15% difference in adult pregnancy have been attributed to winter severity and reduced summer precipitation, respectively (Proffitt et al., 2014). Similarly, a 4-year, 19% decline in pregnancy among migrant elk was found to be largely driven by poor reproduction of young and lactating females (Middleton, Kauffman, McWhirter, Cook, et al., 2013). More controversially, studies have attributed herd-level variation in elk pregnancy to the indirect costs of predation acting through increased vigilance and predator avoidance. One study attributed as much as a 43% relative decline in elk pregnancy to the stress-induced non-consumptive effect of wolf presence (Christianson & Creel, 2014), while others found no evidence for such an effect (Proffitt et al., 2014; White et al., 2011). Brucellosis may have been at low levels or even absent when and where many of these studies were conducted and, as such, the effects of this disease were not investigated. In recent years, however, seroprevalence for brucellosis among elk has been increasing and expanding to new areas around the GYE (National Academies of Sciences, Engineering, and Medicine, 2017) so that ignoring its effects may no longer be
a tenable assumption. Furthermore, calibration for the full effect of brucellosis will be essential if the well-studied GYE elk population is to be used as a model for understanding the effects of climate and predation on other elk populations or ungulate species.

Historically, supplemental winter feedgrounds for elk in the GYE have been focal points for brucellosis contagion. In the Wyoming portion of the southern GYE there are 23 feedgrounds operating annually and at times local seroprevalence can reach as high as 60% (Cotterill et al., 2018). With such high exposure, one might expect that abortions should be frequent and readily observed, but on average fewer than two fetuses were detected annually over a 50-year period (Cross et al., 2015). Because of the difficulty of observation, a more rigorous approach for detecting abortions was undertaken using vaginal-implant transmitters (VITs) which were cultured for \textit{B. abortus} within days after being expelled due to abortion or parturition. A comprehensive study found that, on average, 16% of seropositive and pregnant elk abort per year (Cross et al., 2015) which is consistent with previous findings (Thorne et al., 1978). Use of VITs also confirmed that most abortions occur late in pregnancy (March-May), which is consistent with \textit{Brucella} spp. across host species (Jamil et al., 2017). The shortcoming of this methodology lies in its potential to underestimate the total reproductive cost of the disease. VITs are designed to be expelled by the passage of a fetus through the birth canal, and so are only inserted into those animals determined to be pregnant through ultrasound. Because VITs are inserted in winter (January and February), this method excludes any elk that either failed to conceive in the previous autumn or else lost the pregnancy early in gestation.

Here we report on a study of mid- and late-winter age-specific pregnancy rates
and brucellosis serology for elk in free-ranging herds that attended supplementary feedgrounds in the GYE spanning two decades. Contrary to the conventional assumption that the only effect of brucellosis on elk reproduction is spontaneous induction of late-term abortion (National Research Council, 1998), we found exposed females to also incur a substantial risk of lost reproductive opportunity prior to when abortions occur. Since an individual must first be pregnant in order to have an abortion, we assumed that the difference in pregnancy and abortion losses are additive when calculating the total reproductive costs of disease. Taken together, these double what was previously thought to be the total reproductive cost of brucellosis to elk.

2 | MATERIALS AND METHODS

2.1 | Study area and data collection

Data were collected in western Wyoming, south of Yellowstone National Park, USA, where supplementary winter feedgrounds are used by approximately 80% of the region’s elk (Dean et al., 2004). The National Elk Refuge in Jackson, WY, is operated by the US Fish and Wildlife Service (USFWS) and 22 additional feedgrounds are operated by the Wyoming Game and Fish Department (WGFD; see Cotterill et al. 2018). Our data were collected by the WGFD for research and management purposes across all 23 feedgrounds and 2 nearby unfed wintering locations between 1995 and 2017. All captured elk receive permanent ear tags, which enable identification at subsequent recaptures and this allowed us to track a subset of individual elk over time. Age, pregnancy, and brucellosis serostatus were known for 1236 records of female elk. All serology and pregnancy data were collected between January 3 and April 15, with 90% of
data collection occurring prior to March 1, of any year.

2.2 | Covariates

Age ranged from 1½ to 19½ years at the time of capture and was determined either through recapture of animals marked as calves or yearlings (when elk are morphologically distinct, n=824) or through cementum annuli analysis (Matson’s Lab, Milltown, MT, USA) of a vestigial canine taken from new captures (n=412). Yearlings were excluded from most pregnancy testing for logistical reasons: testing is expensive and elk generally do not reach reproductive maturity until age two, meaning that only one in about five female yearlings might be expected to be pregnant. As such, the yearling age class is poorly represented (n=18). Two-year-olds were the best-sampled age and accounted for nearly 25% of the data. Sample size decreased with age and animals of 10 years or older accounted for only 10% of the data.

The collection of serological data from GYE elk has been described elsewhere (Cross, Edwards, Scurlock, Maichak, & Rogerson, 2007; Maichak et al., 2017; Scurlock & Edwards, 2010). Briefly, to obtain blood samples and teeth for cementum analysis, baited corral traps and/or chemical immobilization were used. Serological assays were conducted and interpreted using current National Veterinary Services Laboratories protocols. Serological profiles were categorized using the United States Department of Agriculture’s brucellosis eradication uniform methods and rules for cervids (APHIS 91-45-013), resulting in a binary (seronegative or seropositive) determination. In a secondary analysis, fluorescent polarization (FP) assay results (in millipolarization units) were included as a continuous variable (Gall et al., 2001).
Pregnancy status was determined either by transrectal ultrasound at the time of capture (n=260), or a blood test for pregnancy-specific protein-B (PSPB, n=976) performed by BioTracking, Inc., Moscow, ID, USA, and previously validated in elk with 97% accuracy (Noyes, Sasser, Johnson, Bryant, & Alexander, 1997). If both ultrasound and PSPB results were available for the same animal and capture, we took PSPB as the more accurate indicator.

WGFD performs ground counts at each of their feedgrounds in peak winter. From these we were able to construct calf:cow ratios for each feedground. In the case of four locations, we also had adequate time-series data for seroprevalence with which to test for a relationship between disease prevalence or changes in prevalence and calf:cow ratios. Due to the timing of transmission (abortion) events and the length of time required to develop a titer (test seropositive following exposure) it is not perfectly clear whether the change in seroprevalence at a site from the previous year \((t-1)\) to \(t\) or the change from two years previous \((t-2)\) to \(t-1\) should be a stronger predictor of current year \((t)\) calf counts. Therefore we tested both, along with seroprevalence in the previous year \((t-1)\) and two years prior \((t-2)\).

2.3 | Statistical modeling and evaluation

Using a Bayesian hierarchical approach, we evaluated the effect of serostatus on pregnancy while accounting for variation between age groups, year effects, and possible effects of pregnancy test method. A preliminary analysis determined that binning ages 3-9, as well as 10 or older, while keeping yearlings and two-year-olds separate, yielded the best balance of model fit and simplicity based on information criteria. Location was
initially included as a model term but dropped due to insignificant variance in site effect.

Our response variable was the pregnancy status $Y$ for individual elk $i$ at age $j$ in year $k$. We assumed that $Y_{ijk}$ was a Bernoulli trial with a probability of being pregnant, $p_{ijk}$. We then used a logit link function to relate the probability of pregnancy to covariates. Let $\alpha_0$ represent an overall intercept term, where $\alpha_i$ and $\alpha_k$ represent age- and year-specific offsets, respectively. Let $\beta_0$ represent the regression coefficient associated with a dummy variable for serostatus, $s_i$, where additional age-varying effects of serostatus are represented as $\beta_i$. In a post-hoc analysis we also tested for year-varying effects of serostatus, represented as $\beta_k$. Let $\gamma$ represent the regression coefficient associated with the dummy variable for pregnancy test method, $m_i$. Thus, the model including all terms takes the form:

$$logit(p_{ijk}) = \alpha_0 + \alpha_{jk} + (\beta_0 + \beta_{jk})s_i + \gamma m_i.$$ 

Six alternative models were compared against this one to determine the effect of serostatus on pregnancy probability, while accounting for the relative importance of age-and year-specific variation, as well as the effect of pregnancy test method (Table 3-1).

Nearly all of our data were collected before March, which we assumed would be prior to when elk typically abort due to brucellosis (Cross et al., 2015). We tested that assumption using seropositive records by modeling pregnancy status as a function of day of the calendar year, collapsing all years. If our results were influenced by early abortions occurring prior to March, then the probability of pregnancy should have decreased over our sampling period. Otherwise, animals in our sample that were not pregnant either failed to conceive during the preceding rut, or suffered intrauterine mortality prior to
January. A logistic regression was performed using day of calendar year to predict the probability of pregnancy for seropositive elk with and without age effects.

Within 1236 records there were 869 unique individuals. Individual was not included as a variable in our pregnancy models because relatively few animals were recaptured more than twice. However, these longitudinal data provided an additional opportunity to test the hypothesis that infected animals recover from the fertility consequences of brucellosis and that any reductions in pregnancy probability attributed to serostatus would disappear over time. For the subset of recaptured individuals that ever tested seropositive, we created a coarse metric of ‘time-since-infection’ by tracking the year in which an individual was first observed to be seropositive (see Appendix B). We then tested whether time-since-infection or fluorescent polarization (FP) assay values (when available) were significant predictors of pregnancy status. We also regressed time-since-infection against FP values to test whether our data support the belief that FP values decline in individuals as elk lose detectable Brucella antibodies over time (Benavides et al., 2017). 

For four feedgrounds with the most consistent serologic testing effort over time we generated smoothed seroprevalence estimates using generalized additive models. We then calculated a calf:cow ratio for each site and year resulting in at least a decade of serological data per site. We modeled the number of calves per 100 adult female elk, \( y \), as being normally distributed with a mean, \( \mu \) and standard deviation, \( \sigma \). The linear model for \( \mu \) then consisted of an overall intercept, \( \alpha_0 \), with and without site-varying intercept offsets, \( \alpha_j \), and a fixed effect, \( \beta \), for a particular serologic parameter, \( \rho \). Thus: \( \mu = \alpha_0 + \)
\(\alpha_i + \beta \rho\). We tested 4 different serological parameters, for a total of 8 models. Accounting for the timing of transmission (abortion) events as well as the incubation period of brucellosis, we hypothesized that the change in seroprevalence from year \(t-2\) to year \(t-1\) as well as the change in seroprevalence from year \(t-1\) to year \(t\) should both strongly correspond to the rate of new infections immediately preceding periods when conception or abortion occur which would be relevant for calf attendance in year \(t\). We also hypothesized that herd level exposure would be sufficient to detect an effect, and so used seroprevalence at year \(t-2\) and year \(t-1\) as predictors. We standardized all four of our serological predictors and used vague priors for all terms. The prior for the overall intercept, \(\alpha_0\), was normally distributed with a mean of 20 and standard deviation of 20. The slope coefficient, \(\beta\), was distributed normal with a mean of zero and standard deviation of 10. The standard deviations for the intercept terms were distributed half-Cauchy with a location parameter of zero and scale parameter of 2.

In our pregnancy models, the prior for the overall intercept for the probability of pregnancy, \(\alpha_0\), was drawn from a normal distribution with a mean of 2 and standard deviation of 1. Age- and year-specific offsets, \(\alpha_j\) and \(\alpha_k\), were given weakly-informative normal priors with a mean of zero, and standard deviations which were drawn from a half-Cauchy distribution with a location parameter of zero and scale parameter of 2. All other effects, including serostatus, pregnancy test method, time-since-infection, and FP, were given weakly-informative normal priors with a mean of zero and standard deviation of 10. For models which featured varying-intercepts and slopes for the age- and year-varying effects of serostatus, a joint multivariate normal prior was used, \([\alpha_{jk}] \sim [\beta_{jk}] \sim\)
The mean vector consisted of zeros, while the covariance matrix, $\Sigma$, received hyperpriors and was further decomposed into a standard deviation matrix and correlation matrix, $\Omega$, $\Sigma = \begin{pmatrix} \sigma_{\alpha_{jk}} & 0 \\ 0 & \sigma_{\beta_{jk}} \end{pmatrix} \Omega \begin{pmatrix} \sigma_{\alpha_{jk}} & 0 \\ 0 & \sigma_{\beta_{jk}} \end{pmatrix}$. Standard deviations were drawn from the same half-Cauchy distribution previously used, $(\sigma_{\alpha_{jk}}, \sigma_{\beta_{jk}}) \sim \text{HalfCauchy}(0,2)$, while the correlation matrix received a relatively diffuse, unimodal Lewandowski, Kurowicka, and Joe (LKJ) prior, $\Omega \sim \text{LKJcorr}(2)$.

All models were fit in R version 3.3.3 (R Core Team, 2016) using the package rethinking (McElreath, 2016) and Stan (Stan Development Team, 2017). To quantify support for our models estimating the effect of serostatus on pregnancy probability we used Widely Applicable Information Criterion (WAIC) (Vehtari, Gelman, & Gabry, 2017; Watanabe, 2010) and Akaike model weight. All models were run for 5,000 iterations after warm-up with three chains. We assessed convergence by monitoring trace plots and using the Gelman-Rubin statistic ($\hat{R}$). $\hat{R}$ values were all less than or equal to 1.1.

3 | RESULTS

3.1 | Effects of serostatus, test method, age and calendar day

Disease exposure status was the single best predictor of pregnancy in all of our models. Age was not by itself a strong predictor, but was important after accounting for disease status. Pregnancy test method also emerged as an important predictor in our models, with false-positive ultrasound tests being the greatest source of error. Our models
predicted a lower probability of pregnancy based on PSPB results than ultrasound ($\gamma = -0.95$, 89% HPDI -1.39, -0.52). This testing error did not vary by serostatus or age. Including age- and year-varying intercepts further reduced WAIC of our models (Table 3-2).

Being seropositive reduced the probability of pregnancy across all ages, with greater effects among younger elk. The largest percentage point difference was in the 2-year-old age class (Fig. 3-2). Being seropositive reduced the probability of pregnancy by 31 percentage points for 2-year-olds (89% HPDI = 20-42 percentage point difference from seronegative mean) but only 7% for 3- to 9-year-olds (89% HPDI = 2-11%). Relatively few pregnancy test results were available for yearlings, resulting in low precision of the estimates for this age class. The mean percentage point difference for yearlings was 16% (89% HPDI = -3-39%).

We found no evidence that the day of calendar year in which an individual pregnancy test was administered had any effect on the probability of pregnancy for seropositive elk (see Appendix A-1). The modeled effect for each standard deviation of the time covariate was 0.02, with a wide 89% credible interval (-0.30, 0.38).

### 3.2 Effect of year

Most years in our data exhibited similar levels of pregnancy but there was considerable variation in pregnancy probability in a handful of years (Fig. 3-3). In a post-hoc analysis, we added year-varying disease effects but found no evidence to suggest that the effect of disease changed over time or in years of relatively high or low overall pregnancy. Although the percentage point difference between mean estimates for the
probability of pregnancy for seropositive and seronegative 2-year-old and 3- to 9-year-old elk increased in years of low overall pregnancy, these fell within the 89% HPDIs for age-specific differences based on our top model (Fig. 3-2).

3.3 | Longitudinal data

For more than 500 seropositive records with FP assay values, there was weak evidence supporting the hypothesis that probability of pregnancy increases as Brucella antibodies decrease (see Appendix A-2). A decrease in one standard deviation of FP value increased pregnancy probability by <2% and the 89% credible interval for effect of FP value overlapped zero. Similarly, ‘time-since-infection’ was a poor predictor of pregnancy. The mean estimates for the effect of time-since-infection were near zero, and these estimates remained unchanged whether or not age-varying intercepts were included. In addition, for repeatedly sampled individuals, FP values declined at a rate of 15 mpu/year (89% HPDI = -19, -11). This appears to suggest that our metric for time-since-infection was adequate for testing recovery, despite not knowing with certainty when individuals were first infected. This finding also corroborates the idea that low FP results may be indicative of older exposures.

3.4 | Calf:cow ratio

The change in seroprevalence from years $t-2$ to $t-1$ and the change in seroprevalence from years $t-1$ to $t$ had a negative association with the number of calves counted per 100 female elk in year $t$, and credible intervals did not overlap zero. Neither the seroprevalence at year $t-2$ nor at $t-1$ exhibited a statistically significant association with calves per 100 females. Allowing site-varying intercepts reduced WAIC, but Akaike
model weight was distributed across 6 of the 8 models and none appeared to be a particularly good fit to the data (Appendix A-4). All four models including the ‘change in prevalence’ predictors provided similar estimates. The top model predicts that for one standard deviation increase in prevalence from year \(t-2\) to \(t-1\) (approximately 6%) 1.6 fewer calves per 100 adult female elk would be counted in year \(t\) (89% HPDI = -0.31, -2.79).

4 | DISCUSSION

Chronic disease impacts can be difficult to detect or go misdiagnosed because they typically require large, long-term data sets. Our results show that, despite decades of research, there is a significant and previously undetected reduction in the probability of pregnancy in elk cows exposed to brucellosis. This effect appears to be additive to the abortions that the disease is notorious for, which effectively doubles the previously estimated reproductive cost of brucellosis to elk. We also found evidence that this hidden reproductive cost is long-lived. It is uncertain, however, how this might influence population growth, in part because the mechanisms by which the disease might affect conception, implantation, and/or early pregnancy remain unidentified.

The estimated overall effect of reduced pregnancy on reproductive output, contingent on age-specific prevalence and herd age structure, was 12 percentage points less than the seronegative mean (see Appendix A-3). We found substantial variation in effect size between age classes, which appeared to diminish in older age. This difference attributable to serostatus could result from failure to conceive during the previous rut or fetal loss, although we found compelling evidence that this reduction in pregnancy is not
the result of abortions occurring during our sampling period. Because pregnancy is a prerequisite for abortion, the total reduction in reproductive output from this disease must be the sum of the two. By our estimation this would suggest that seropositive elk produce approximately 24% fewer calves than seronegative elk (see Appendix A-3). Similar reductions in pregnancy and recruitment attributable to brucellosis have been found for bison in Yellowstone, which lend additional support to our findings (Fuller et al., 2007; Geremia et al., 2008). This indicates the potential for the disease to have as much of an effect on reducing pregnancy as severe winters or droughts.

A 24% reduction in reproductive output is substantial, but a few caveats must be considered. That value applies to seropositive females and, with periodic exceptions, it is the minority of elk within any given herd in our study area that test seropositive for brucellosis (Cotterill et al., 2018). Except in herds with particularly high seroprevalence, the expected decline in pregnancy probability across the herds we studied should generally be below 7%. In a typical GYE herd with 25% seroprevalence, in which the disease causes 12% of seropositive female elk to lose their reproductive opportunity by mid-winter, and an additional 16% of seropositive elk go on to abort conditional upon being pregnant, then under average feedground conditions (76% of our seropositive elk were pregnant), the combined effect is a 6% reduction in healthy births as follows:

\[
(0.25 \times [0.12 + (0.16 \times 0.76)]) = 0.06
\]

It must be noted, however, that local seroprevalence can at times exceed 60%, in which case the expected overall reduction in calf production for the herd would be 14 percentage points lower. As we saw when we modeled the effect of year on pregnancy probability, in years when overall pregnancy
rates are depressed for other reasons, the reproductive costs of brucellosis further compound low productivity. While we did detect a decrease in calf:cow ratio attributable to increases in seroprevalence, the observed effect was weaker than expected. This could be because the calf data included sampling error arising from the use of ground counts on highly aggregated herds and also because changes in smoothed seroprevalence estimates are imprecise. A thorough accounting of other variables that influence recruitment to calfhood was simply beyond the scope of this study.

The mechanism behind the observed reductions in pregnancy remains unidentified. Robust longitudinal data would add to our understanding of the cause, but are costly and difficult to obtain. A captive study would be ideal, but is ruled out in the USA by the Select Agent Status imposed on live Brucella cultures (National Academies of Sciences, Engineering, and Medicine, 2017). We were unable to resolve this issue, but our analysis of the available repeat-capture data suggest that female elk incur longer-term reproductive consequences of this disease than previously thought. Both ‘time-since-infection’ and FP assay were poor predictors of pregnancy probability for seropositive elk, which does not support the conventional belief that elk recover fully from this disease after the initial abortion risk has passed. Chronic inflammation of the endometrium (endometritis) has been described across the Brucella genus in other hosts, which could interfere with implantation (Enright, 1990; Meador, Hagemoser, & Deyoe, 1988; Rhyan et al., 2009; Verma, Katoch, Sharma, & Nigam, 2000). Although this seems a plausible biological cause, it does not fit well with the large effect in 2-year-olds, most of which are expected to be pregnant for the first time.

These findings complicate studies of GYE elk which have shown reduced
recruitment over time but failed to take brucellosis into account. In some instances there
may exist data to show the disease was absent or at very low levels for the herds in the
question, while in others there may simply be no relevant disease data. Reduced
fecundity because of disease may also add complexity to disease dynamics. When time-
series prevalence data are used to evaluate disease-management strategies, then
differential vital rates must be taken into account along with other potentially relevant
factors like seasonality of transmission (Altizer et al., 2006), climate (Pascual, Cazelles,
Bouma, Chaves, & Koelle, 2008), and cohort entry effects (He & Earn, 2016). A chronic
disease like brucellosis, which affects fecundity, might provide an excellent model
system in which to study the fitness consequences of disease and the evolution of disease
resistance or tolerance. This line of inquiry is particularly relevant now with the spread of
chronic wasting disease (CWD), a spongiform encephalopathy that affects cervids,
causing global concern (Galloway, Monello, Brimeyer, Cole, & Hobbs, 2017; Mysterud
& Rolandsen, 2018). It has been suggested that selection for prion disease resistance may
play an important role in the long term dynamics of infected populations as CWD
continues to spread (Monello et al., 2017; Robinson, Samuel, Johnson, Adams, &
McKenzie, 2012; A. L. Williams, Kreeger, & Schumaker, 2014). Similarly, the
magnitude of the effect of brucellosis on elk fitness should drive selection for increased
resistance or tolerance. Whereas CWD is eventually fatal for elk (E. S. Williams,
Kirkwood, & Miller, 2008), the fitness consequences of brucellosis take effect much
sooner (though they may also be prolonged). If coinfection becomes common in GYE elk
then an intriguing question will arise: could selection for resistance to one disease disrupt
selection for resistance to the other?
Lastly, the operation of supplementary feedgrounds for wildlife is a subject of perennial debate and, for the GYE elk in particular, the brucellosis problem places feedground managers on the horns of a dilemma. First, elk-cattle transmission risk is reduced by the spatial separation achieved by feedgrounds (Brennan, Cross, Portacci, Scurlock, & Edwards, 2017), but feedgrounds increase elk-elk transmission while maintaining high local prevalence (Cross et al., 2007) and serving as a source to elk that are remote from feedgrounds (Kamath et al., 2016). Second, while feedgrounds increase the reproductive cost of brucellosis they may also offset them by nutritional supplementation of the elk cows that proceed to healthy parturition. The proportion of seronegative feedground elk that are pregnant rival some of the higher reported pregnancy rates for Rocky Mountain populations (Raithel et al., 2007). Consequently, at the scale of the GYE, the overall effect of brucellosis on population growth appears benign under normal conditions for nutritionally supplemented elk. Closing feedgrounds while brucellosis prevalence is high might result in poor recruitment in the near term, particularly if nutritional supplementation has been offsetting disease costs. Because brucellosis in elk has historically been considered a ‘feedground problem’, and feedgrounds facilitate handling and sampling opportunities, the bulk of the ecological research surrounding it has taken place within these fed herds. The full effects of brucellosis remain to be tested in unsupplemented elk herds where food resources, herd demographics, local force-of-infection, and other factors differ. With brucellosis prevalence increasing among many elk herds in the GYE and beyond (P. C. Cross et al., 2010; National Academies of Sciences, Engineering, and Medicine, 2017), brucellosis and other disease impacts should be an important consideration in future demographic
Failure to account in full for disease-induced reproductive costs could cause over-estimates of the effects of other demographic stressors, all of which require different management and policy strategies.

**DATA ACCESSIBILITY**

Data and supporting code available through the Utah State University Digital Commons: [https://doi.org/10.15142/T39M0B](https://doi.org/10.15142/T39M0B)

**LITERATURE CITED**


Ecosystem. *Ecological Applications*, 17(4), 957–964. doi:10.1890/06-1603


Serengeti and its Implications for Ecosystem C. *PLOS Biology*, 7(9), e1000210. doi:10.1371/journal.pbio.1000210


doi:10.1002/jwmg.792


(Cervus elaphus). *Ecosphere*, 5(5), art60. doi:10.1890/ES14-00013.1

TABLES AND FIGURES

TABLE 3-1. Seven models to estimate the effect of serostatus on the probability of pregnancy. *Model 7 was added post-hoc to test for a significant disease by year interaction.

<table>
<thead>
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<th>model</th>
<th>formula</th>
<th>intercepts</th>
<th>slopes</th>
</tr>
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<td>logit($p_i$) = $\alpha_0 + \beta_0 s_i + \gamma m_i$</td>
<td>fixed</td>
<td>serostatus; method</td>
</tr>
<tr>
<td>2</td>
<td>logit($p_{ik}$) = $\alpha_0 + \alpha_k + \beta_0 s_i + \gamma m_i$</td>
<td>year-varying</td>
<td>serostatus; method</td>
</tr>
<tr>
<td>3</td>
<td>logit($p_{ij}$) = $\alpha_0 + \alpha_j + \beta_0 s_i + \gamma m_i$</td>
<td>age-varying</td>
<td>serostatus; method</td>
</tr>
<tr>
<td>4</td>
<td>logit($p_{ij}$) = $\alpha_0 + \alpha_j + (\beta_0 + \beta_j) s_i + \gamma m_i$</td>
<td>age-varying</td>
<td>age-varying serostatus; method</td>
</tr>
<tr>
<td>5</td>
<td>logit($p_{ijk}$) = $\alpha_0 + \alpha_{j,k} + \beta_0 s_i + \gamma m_i$</td>
<td>age- and year-varying</td>
<td>serostatus; method</td>
</tr>
<tr>
<td>6</td>
<td>logit($p_{ijk}$) = $\alpha_0 + \alpha_{jk} + (\beta_0 + \beta_{jk}) s_i + \gamma m_i$</td>
<td>age- and year-varying</td>
<td>age-varying serostatus; method</td>
</tr>
<tr>
<td>7*</td>
<td>logit($p_{ijk}$) = $\alpha_0 + \alpha_{jk} + (\beta_0 + \beta_{ijk}) s_i + \gamma m_i$</td>
<td>age- and year-varying</td>
<td>age- and year-varying serostatus; method</td>
</tr>
</tbody>
</table>
**TABLE 3-2.** Model comparison for the effect of serostatus on pregnancy probability. Model 6, including age and year-varying intercepts, age-varying effect of serostatus, and effect of pregnancy test method had the lowest WAIC, and received more than half of the Akaike model weight. Model 7, which differed from model 6 only in that it included year-varying effects of serostatus, failed to improve model fit, and received the remainder of the Akaike model weight.

<table>
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<th>dWAIC</th>
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<th>SE</th>
<th>dSE</th>
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<td>0</td>
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<tr>
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<td>43.42</td>
<td>19.09</td>
</tr>
</tbody>
</table>
FIGURE 3-1. Elk at a winter feedground in Wyoming. Photo credit: Mark Gocke, WY Game & Fish Dept.
FIGURE 3-2. Left: Mean posterior predictive estimates of pregnancy probability by age group and serostatus from model 6, for the average year, with 89% highest posterior density interval (HPDI) estimates indicated. Right: The percentage point difference in mean estimates by age, for the average year, with 89% HPDI indicated. Mean estimates were 16% for yearlings, 31% for 2-year-olds, 7% for 3- to 9-year-olds, and 2% for animals 10 years of age and older.
FIGURE 3-3. Mean posterior predictive estimates by age class of the probability of pregnancy for seronegative (left) and seropositive (right) elk in years of highest (2011, top line), average (2012, middle line), and lowest (2017, bottom line) overall pregnancy.
CHAPTER 4
PARSING THE EFFECTS OF DEMOGRAPHY, CLIMATE, AND MANAGEMENT ON RECURRENT BRUCELLOSIS OUTBREAKS IN ELK

ABSTRACT

1. Zoonotic pathogens can harm human health and well-being directly or by impacting livestock. Pathogens that spillover from wildlife can also hurt conservation efforts if humans come to perceive wildlife as pests. Brucellosis caused by the bacterium *Brucella abortus* circulates in elk and bison herds of the Greater Yellowstone Ecosystem which in turn pose a risk to cattle and humans. Our goal was to develop a better understanding of brucellosis transmission dynamics in elk—the host driving expansion of the pathogen across the region—and how management actions might reduce infection.

2. Using >20 years of serologic, demographic, and environmental data on brucellosis in elk, we built stochastic compartmental models to assess the influences of climate forcing, herd immunity, population turnover, and management interventions on pathogen transmission. Data were collected at feedgrounds visited in winter by free-ranging elk in Wyoming, USA.

3. Snowpack, hypothesized as a driver of elk aggregation and thus brucellosis transmission, was strongly correlated across feedgrounds. We expected this

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variable to drive synchronized disease dynamics across herds. Instead, we demonstrate asynchronous epizootics driven by variation in demographic rates.

4. We evaluated the effectiveness of test-and-slaughter of seropositive female elk at two feedgrounds. Test-and-slaughter temporarily reduced herd-level seroprevalence but likely reduced herd immunity while removing few infectious individuals, resulting in subsequent outbreaks once the intervention ceased. We simulated an alternative strategy of removing seronegative female elk and found it would increase herd immunity, yielding fewer infections. We evaluated a second experimental treatment wherein feeding density was reduced at one feedground, but we found no evidence for an effect despite a decade of implementation.

5. Synthesis and applications: Positive serostatus for a wildlife disease is often imperfectly correlated with infective status but is nevertheless used to make management decisions including lethal removal. This can have adverse consequences whereas efforts that maintain herd immunity can have longer-term protective effects. In a metapopulation of a long-lived species it is unlikely that serological trends will be synchronous across subpopulations unless vital rates are constant because demographic factors have a large influence on disease patterns even where strong climatic forcing occurs.

1 | INTRODUCTION

Controlling disease spread is especially problematic in the case of wide-ranging wildlife populations. Part of the problem is that disease surveillance typically relies on serological testing, the results of which can only be properly interpreted within the
context of a specific combination of test, host and pathogen (Gilbert et al., 2013).

Assuming test accuracy, a common misconception is that positive serostatus denotes 
current infection. Instead, seropositivity indicates detectable antibodies due to previous 
exposure. Conversely, seronegativity could mean no exposure, recent exposure within the 
period required to seroconvert, or historical exposure followed by the loss of detectable 
antibodies. Evidence from human systems shows that antibodies to many pathogens 
decline in the months or years following infection (Edwards, 2005; Gijsen, Land, 
Goossens, Slobbe, & Bruggeman, 2002) and in cases like brucellosis long-term immunity 
can be retained in test-negative individuals through adaptive cell-mediated responses 
(Yingst & Hoover, 2003). Consequently serostatus alone is not a reliable indicator of 
infection status, especially when antibodies are short-lived relative to the lifespan of the 
host. Such is the case with brucellosis in elk (Cervus canadensis), caused by the 
bacterium Brucella abortus. Detectable antibodies are lost over time, and modeling 
results suggest that immunity to reinfection may be retained (Benavides et al., 2017).

‘Brucellosis’ refers to infection by members of the genus Brucella and is one of 
the most common zoonotic diseases worldwide (Boschirolni, Foulongne, & O’Callaghan, 
2001). In the Greater Yellowstone Ecosystem of the western USA, elk and bison (Bison 
bison) are reservoirs of B. abortus which arrived with cattle a century ago (Meagher & 
Meyer, 1994). There is low spillover risk from bison to cattle (National Academies of 
Sciences, Engineering, and Medicine, 2017) despite seasonal movements of bison out of 
the park as influenced by climate and population size (Kilpatrick, Gillin, & Daszak, 
2009). Elk, however, are widely distributed inside and outside the park, and brucellosis 
periodically spills back from elk to cattle (Kamath et al., 2016) at significant cost to the
affected cattle industry. The pathogen is transmitted by direct contact with fetal tissues and fluids resulting from disease-induced abortions (National Research Council, 1998). Live births from infected mothers can also cause horizontal transmission if other herd members inspect the newborn calf or birth tissues, although parturient elk sequester themselves and their newborn calves (Van Campen & Rhyan, 2010). Vertical transmission does not occur. Bison and elk born to seropositive mothers can have detectable antibodies, but these disappear after several months and do not provide lifelong immunity (Rhyan et al., 2009; Thorne, Morton, Blunt, & Dawson, 1978). Thus elk-to-elk and elk-to-cattle transmissions are most likely during and after abortion events, which primarily occur between March and May (Cross et al., 2015) and in the first year following infection (Thorne et al., 1978).

Due to the seasonality of transmission, winter feedgrounds for elk feature prominently in ongoing efforts to understand and control brucellosis. Elk-to-elk transmission is facilitated by 23 feedgrounds in Wyoming that aggregate large herds during part of the transmission season. Hay is provided daily at these diversionary locations to reduce depredation of private haystacks and minimize comingling with cattle in winter as part of a disease-risk mitigation strategy. Feedgrounds reduce local spillover risk to cattle in the short term (Brennan, Cross, Portacci, Scurlock, & Edwards, 2017), yet simultaneously contribute to disease persistence (Scurlock & Edwards, 2010). The seasonality of transmission, coinciding with winter feeding and high site fidelity of elk to particular feedgrounds (supporting reference?), creates a metapopulation structure where feedground herds are subpopulations within which brucellosis circulates. This presents a fortuitous study system for investigating the drivers of pathogen transmission. Drivers
can be broadly categorized as those that are exogenous, or ‘external’ to the host and pathogen, like climatic variables, and those that are endogenous, or ‘internal’, like vital rates or epidemiological processes. Interactions between the two can make it difficult to infer the underlying processes from the observed serological patterns (Koelle & Pascual, 2004; Paull et al., 2017). In the context of a metapopulation, strong environmental forcing might generate a Moran effect, with strong synchrony in outbreak size or timing across subpopulations (Moran, 1953). Yet, if disease trends are sensitive to stochasticity, vital rates, or epidemiological rates, we might instead expect asynchrony despite common external forcing (Rohani et al. 1999). Exogenous effects also depend on conditions within the host population. Strong forcing could facilitate pathogen transmission, but not if herd immunity is already high.

Previous work suggested that heavy snowpack increases elk-to-elk brucellosis transmission (Cross, Edwards, Scurlock, Maichak, & Rogerson, 2007) and that dispersing haypiles across greater area on the feedground could reduce elk density by 83% and contagious contacts by 91% (Creech et al., 2012). The Wyoming Game and Fish Department (WGFD) has thus experimented with ‘low-density feeding’ in addition to a test-and-slaughter program during late winter from 2006 to 2010 at three feedgrounds. The effectiveness of these actions has not been fully evaluated, which motivated our current work. We modeled the underlying infection dynamics of brucellosis in free-ranging, winter-fed elk using compartmental SIR models and explored the relative influences of demographic and environmental drivers on transmission. This provided a baseline understanding of seroprevalence trends within a metapopulation context while accounting for the ambiguity of serological status. Against this backdrop we assessed two
management interventions in relation to their intended effectiveness in reducing disease prevalence: ‘low-density’ feeding and test-and-slaughter.

2 | MATERIALS AND METHODS

2.1 | Study area and data collection

Our study area is western Wyoming, south of Yellowstone National Park, USA, at winter feedgrounds that are used by approximately 80% of the region’s elk annually (Figure 4-1; Dean et al., 2004). Elk captures occurred principally in February of each year for the purpose of disease surveillance. Blood was drawn only from female elk because males are insignificant as vectors of infection (National Research Council, 1998). Serological testing was performed in accordance with National Veterinary Services Laboratory protocols as described by Maichak et al. (2017). Elk calves were excluded from serological testing. Serologic test results were aggregated by site and year. Greys River, Dell Creek, and Muddy Creek feedgrounds each had 15-25 years of serology data with robust sample sizes, despite periodic gaps (Table B-1). Demographic data included adult counts by sex and the number of calves present. Attendance at these feedgrounds ranged from 100-700 adult female elk per year. Counts and age/sex classifications were recorded during peak-winter along feedlines, when feedground attendance by elk is presumed highest. One additional feedground, Scab Creek, provided sufficient data with which to test our top model and parameter estimates.

Beginning in 2009 ‘low-density’ feeding practices were adopted at Greys River feedground. Reliable feed distribution and elk density data were unavailable and so we characterized the experimental treatment as a categorical (before-and-after) variable.
Test-and-slaughter of seropositive female elk took place at Muddy Creek and Scab Creek, where capture and testing rates ranged from 29-62% of attending female elk per year (Table B-2; Scurlock, Edwards, Cornish, & Meadows, 2010). At Muddy Creek 107 seropositive female elk were removed over five years (2006-2010) when the female count averaged 260. At Scab Creek 58 seropositive female elk were removed over two years (2009-2010) when the female count averaged 486.

2.2 | Partially-observed Markov process models

Deterministic compartmental models are central to the study of disease dynamics (reference). Although statistical inference is simpler with deterministic models, “many infectious systems are fundamentally individual-based stochastic processes, and are more naturally described by stochastic models” (Roberts, Andreasen, Lloyd, & Pellis, 2015). Partially-observed Markov process (POMP) models combine the mechanistic processes in compartmental SIR models with probabilistic models linking the observed data to the latent process (King, Nguyen, & Ionides, 2016). Our latent process was a four-compartment model (Figure 4-2), alternatively described as a series of discretized equations (Appendix B). We modeled a discrete-time process at annual intervals because disease transmission and birth pulses are seasonal and infected females are likely to abort, and thus transmit infection, in the following year. Additionally, 90% of our data were collected in February, a length of time less than the average time to seroconvert following exposure (Thorne et al., 1978) and prior to peak transmission season (Cross et al., 2015).

The compartments of our models include susceptible and seronegative (S), infected, infectious, and seropositive (I), seropositive but no longer abortive (R₁), and
sero reverted—seronegative and recovered with immunity (R2). Entry occurs via calf recruitment. Because we modeled female elk only, the number of calves (C) in year \( t \) was the number observed divided by two under the assumption of equal sex ratio in calves (Johnson, 1951). Hunting is the dominant source of mortality in this population and so individuals across all compartments experienced an equal probability of mortality \( \mu_j \) within each defined period of the timeseries (\( j = 1 \text{-} 3 \) periods depending on feedground) when there was a new management objective for that hunt unit. Susceptibles are exposed at a rate corresponding to the force of infection (\( \lambda \)), but not all that are exposed and seroconvert become infectious (abort), which allows a proportion to transition straight from S to R1 (\( \rho \)). Elk that do become infectious recover with probability \( \sigma \) and detectable antibodies are lost with probability \( \gamma \). Because serology does not distinguish between compartments I and R1, the test-and-slaughter models (Muddy Creek and Scab Creek) included \( v \), the probability of seropositives exiting I and R1 in years with removals. Conditional on being in compartments I and R1, the probability of removal was equal to the proportion of females captured for testing at a feedground in a given year. This approach is integer-based, therefore probabilities and rates were incorporated into the process model using random draws from an eulermultinomial distribution within the software package ‘pomp’ (King et al., 2018) in R (R Core Team, 2018).

The force of infection, \( \lambda \), took one of three basic forms each corresponding to a model where transmission was internally-driven (endogenous), driven by climate (exogenous), or driven by both factors (combination). In the endogenous model, we assumed \( \lambda_1 \) is equal to the product of a constant transmission parameter \( \beta \), and the sum of the annual number of infecteds I and imported infections from outside the herd \( i \), divided
by the population size $N$ raised to a scaling parameter $\Theta$. The scaling parameter describes the degree to which the transmission process is density-dependent ($\Theta = 0$) or frequency-dependent ($\Theta = 1$) (Cross et al., 2013). In the exogenous forcing model, we assumed $\lambda_2$ is related to a winter severity covariate $\psi$ that varied annually and by feedground. The final form was a combination of the previous two.

\[
\lambda_1(t) = \frac{\beta[I(t) + \iota]}{N(t)^\Theta} \quad \text{endogenous}
\]

\[
\lambda_2(t) = \frac{[\beta^* \psi(t)][I(t) + \iota]}{N(t)^\Theta} \quad \text{exogenous}
\]

\[
\lambda_3(t) = \frac{[\beta + [\beta^* \psi(t)]][I(t) + \iota]}{N(t)^\Theta} \quad \text{combined} \quad \text{(Equations)}
\]

The observed process was the number of seropositive test results divided by the total number tested for a given feedground in a given year (‘apparent seroprevalence’). The probability of the data (number of seropositive test results) in year $t$ was binomially distributed and conditional on the probability $p(t)$, which was the ‘true seroprevalence’ from the latent process and $n(t)$, the total number of tests. We also modeled female elk counts to ensure that our model predictions conformed both to the observed disease and population trends. Observed counts were modeled as a draw from a normal distribution with a mean at the ‘true population size’ $N$ (the sum of the four compartments), and a standard deviation of 20, which represents approximately 5% of an intermediate-sized feedground herd.
2.3 | Incorporating winter severity

Based on previous studies we expected that environmental conditions causing larger elk aggregations for longer periods during late winter should result in more transmission (Creech et al., 2012; Cross et al., 2007). We thus tested models in which ‘heavy snow’ and ‘late green-up’ contributed to environmentally-driven transmission ($\psi$) via the force of infection. Snow-depth data were unavailable across the temporal and spatial extent of our study area so we used snowmelt water equivalent (SWE) values from nearby SNOTEL sites for each feedground between March and June. These values were strongly correlated with one another across sites within each year (Figure B-7). We summed the SWE values of the first day in each of these months to arrive at a single value per site and year.

Often the nearest SNOTEL stations are several kilometers from the feedground and hundreds of meters higher in elevation, so we also calculated green-up metrics using normalized difference vegetation index (NDVI) MODIS data and the 3x3 square of pixels around each feedground where each pixel was 250x250m. We excluded pixels that included roads or buildings. We fit double logistic curves to NDVI time series to calculate these metrics following the methods of Bischof et al. (2012) and Merkle et al. (2016). Metrics were strongly correlated so we only investigated the date of ‘spring start’, which we defined as the peak values of the 2nd derivative of the spring side of the NDVI curve (Johnston, Beever, Merkle, & Chong, 2018). Annual SWE values were standardized across the years 1991-2018 for each SNOTEL station corresponding to an individual feedground, whereas NDVI values were standardized across 2000-2017 (beginning when these data were first available). Standardized values were then
exponentiated because λ is a rate which cannot be negative.

### 2.4 | Candidate models

All of our models follow the four-compartment plan (Figure 4-2). Alternative models featuring no seroreversion or seroreversion without retained immunity did a poor job of describing the data and received less support using AIC in a preliminary analysis (Figures B-1, B-2). The possibility of a ‘low-density feeding’ treatment effect was tested at Greys River feedground with models where λ was allowed to vary ‘before’ and ‘after’ treatment initiation in 2009. Testing all possible combinations for a time-dependent low-density feeding treatment effect yielded 5 additional models (Table B-3).

### 2.5 | Inference, comparison, and constraints

We used sequential Monte Carlo to obtain the log likelihood following the maximum-likelihood approach of Ionides et al. (2015), and iterated filtering in the software package ‘pomp’ (King et al., 2018). A broad exploration of parameter space was initiated using 100 sets of parameter values, with each value drawn from a uniform distribution. Likewise, initial starting conditions were generated for the 4 compartments (full details in Appendix B). During iterative filtering (‘mif2’ in ‘pomp’) all parameters were perturbed except ρ and γ. We used 20,000 particles, 800 iterations, and 20 replicates. For each replicate we ran 20 particle filters with 20,000 particles to calculate the log likelihood and standard error of the Monte Carlo approximation. The standard error for the log likelihoods of all models was less than 0.1.

With limited data at Scab Creek, we used it to test our top model and a reduced range of parameter estimates based on the results of other feedgrounds. Diagnostic
checks included monitoring the effective sample size of our filtering procedure, the conditional log likelihood at the last iteration of the MLE search and traceplots of the parameter estimates. We calculated the Akaike Information Criterion (AIC) and Akaike model weights (Burnham & Anderson, 2002) using the MLEs of each model for comparison. We further assessed parameter uncertainty following the methods of King et al. (2015; see Appendix B).

2.6 | Testing expectations of synchrony

Assuming heavy snowfall causes elk to aggregate, thereby increasing transmission, we expected synchronous seroprevalence trends across feedgrounds because all feedgrounds experienced synchronous snowfall (Figure B-7). Following model comparison we simulated three hypothetical subpopulations with varying demographic rates and projected count and seroprevalence trends out 100 years to explore if we should expect synchronous trajectories across subpopulations. Hypothetical subpopulations received the same starting size ($N = 400$, female only) and initial conditions of the four disease compartments. We used the MLE values of parameters from the endogenous, exogenous, and combined models for Greys River and held them constant across space and time. We used identical climate values for each subpopulation, which was a vector sampled with replacement from our Greys River SWE data. Calf recruitment in year $t$ was a random draw that broadly encompassed a plausible range of calf recruitment values from Rocky Mountain elk populations across the Western U.S. ($\sim \text{Uniform}(0.1, 0.4)$; Raithel, Kauffman, & Pletscher, 2007) multiplied by the subpopulation size in year $t-1$. Mortality ($\mu$) varied by subpopulation but was time-
constant and took values \((1/8, 1/9, \text{and } 1/10)\) that were found to yield decreasing, stable, or increasing count trends across subpopulations. Additional sources of stochasticity were (1) a multinomial draw on the number of new infectives at each time step, and (2) infectious individuals were imported as a Bernoulli draw with an annual probability corresponding to rate \(i\). We compared the projections of our three transmission models.

### 2.7 Comparing test-and-slaughter with other possible regimes at Muddy Creek

Using the MLE parameter values for our endogenous model at Muddy Creek, we estimated the number of abortive elk \((I)\) over the modeled 15 year period and constructed 90% prediction intervals using the 5% and 95% quantiles of 2000 simulations of the model. We repeated this process for three hypothetical management regimes over the same time period: (1) no slaughtering; (2) slaughtering seronegative elk only; (3) indiscriminate culling. Removals (options 2 and 3) were set to maintain population sizes comparable to those observed when only seropositive female elk were slaughtered. Finally, we calculated the difference of median estimates over time between the 4 regimes and estimated the number of infectives removed during test-and-slaughter.

### 3 RESULTS

We found substantial temporal variability in seroprevalence within individual feedground herds suggesting recurrent outbreaks (Figure 4-3) that were asynchronous across subpopulations. At Greys River feedground, the site for which we had the most years of data, the endogenous model received the highest support by AIC, while at Dell Creek and Muddy Creek, exogenous models received similar model weight to the alternatives (Table B-5). Substituting vegetation green-up for snow data did not
substantially alter these results (Table B-6), except that the exogenous model received stronger support at Dell Creek. This might suggest that NDVI improved on SWE for modeling. The incorporation of a treatment effect from low-density feeding practices at Greys River failed to improve model fit. There were not sufficient data at Scab Creek to perform a formal model comparison, but the endogenous model and parameter estimates from other feedgrounds were in rough agreement (Figure 4-3).

Because $\lambda$ depends on the relative fraction of susceptible and infectious individuals, it can vary substantially over time despite a constant transmission term (Figure 4-4). By simulating with the parameter values at the MLE, it becomes apparent that relatively few infective elk are needed to achieve high levels of seroprevalence. This is consistent with the difficulty in detecting abortions at the feedgrounds and previous work which estimated that 16% of seropositive elk abort in any given year (95% CI: 0.10, 0.23; Cross et al., 2015). $I$, which we defined as abortive in our models, provided this additional point of comparison. Indeed, the sum of $I/(I + R_1)$ for the full time series of the endogenous models from 2000 stochastic simulations had a median of 16.6% at Greys River (90% prediction interval: 0.14, 0.19), 13.6% at Dell Creek (90% PI: 0.08, 0.18), and 17.0% at Muddy Creek (90% PI: 0.12, 0.20). Our models consistently estimated quick transitions from $I$ to $R_1$, indicating that the majority of elk that do abort only do so in the first year following infection, consistent with work on captive elk (Thorne et al., 1978; see Appendix B).

Our simulation revealed that if demographic rates vary between feedgrounds then asynchronous disease trends can emerge even when synchronous exogenous forces drive
\[ \lambda \]. As expected, the endogenous model predicted the greatest asynchrony across sites, but all models generated increasing asynchrony as time progressed and population growth trajectories diverged (Figure 4-5).

At Muddy Creek we estimate that of 107 seropositive elk that were removed over 5 years, only 6 were infective, but this prevented an additional 20 infections in the following 8 years. These effects translate to two fewer infectives in the remaining population per year compared to models without test-and-slaughter (Figure 4-6).

Annually removing 10\% of female seronegative elk during the same 5-year period was predicted to generate a similar reduction to the number of infectives present during control efforts, but yield additional benefits over the remaining time period (37 fewer infectives compared to seropositive removal). The simulation of culling without regard to serostatus of 7.5\% of female elk per year fell between the other two predictions: it achieved fewer infectives compared to slaughter of seropositive elk, but underperformed compared to seronegative slaughter.

4 | DISCUSSION

We found brucellosis seroprevalence trends that were asynchronous across elk subpopulations despite their exposure to similar environmental forcing and we used mechanistic modeling to tackle the inverse problem of inferring the latent processes from the observed serological dynamics. Although brucellosis is widely considered a chronic ailment (Ahmed, Zheng, & Liu, 2016), our results, in conjunction with previous research, suggest the dynamics of brucellosis in elk may actually fall more in line with acute and highly-immunizing diseases. This has repercussions for management. The time to
seroconversion following exposure (approximately 1 month), the timing of testing (prior to disease-induced abortions), and the chances that a newly-infected elk will actually abort (approximately 50%), along with quick recovery time and lifelong immunity, all contribute to a situation in which targeting seropositives rarely removes infectious individuals. Though at first counterintuitive, it follows that removing seronegative elk would have longer-term protective effects. This is consistent with brucellosis work in bison suggesting that the loss of herd immunity created by removal of seropositive individuals can result in ricochet effects (Ebinger, Cross, Wallen, White, & Treanor, 2011). Timing of interventions is also paramount. It appears that test-and-slaughter at Muddy Creek coincided with a ‘fadeout period’ when seroprevalence was high, but declining. Therefore, the number of infectious elk was low (Figure 4-4). If removals instead targeted seronegative elk when seroprevalence is high, this would drive a spike in seroprevalence but a decline in newly infected elk, a longer period with reduced spillover risk, and thereafter a decline in seroprevalence. Achieving public support for such action might require substantial outreach. Alternatively, sustained culling without regard to serostatus (through increased hunter harvest) might garner wider support. This result stands in contrast to a finding that hunting can increase disease prevalence (Choisy & Rohani, 2006), but which involved a region of the parameter space that is unlikely in our system (large annual fluctuations in host population size and rapid, explosive spikes in prevalence).

Based on the simulation results of our hypothetical herds we should not necessarily expect synchronous seroprevalence trends even in scenarios with strong climate forcing. Outbreaks might periodically align following severe winters, but
intervening years exhibit asynchrony like that detected in the actual seroprevalence data. Severe winters can only trigger outbreaks if a large pool of susceptible female elk already exists. This underscores the importance of birth rate and population turnover to the disease dynamics of this system. These findings are consistent with existing literature (Lloyd & Sattenspiel, 2010), yet this may be among the first applications for long-lived, free-ranging wildlife. Lastly, we found no evidence that ‘low-density feeding’ has reduced the force of infection at Greys River feedground. Additional data on elk density and feed distribution would permit more explicit modeling. The course of an outbreak for any one of our subpopulations appears to exceed a decade, and our longest time-series was 25 years. In that context, small treatment effects may be difficult to detect.

These findings prompt a review of the options for reducing brucellosis transmission among feedground elk. A vaccination program persisted at feedgrounds for decades, in part because its implementation coincided with a brief dip in seroprevalence, although it was later deemed ineffective (Maichak et al., 2017). Further vaccine development is hampered by the USDA’s Select Agent Status for Brucella spp. (National Academies of Sciences, Engineering, and Medicine, 2017). Quarantine is infeasible for free-ranging elk and wide-scale fencing is problematic (Mysterud & Rolandsen, 2018). In general, removing infectives should reduce contagion in situations where ‘infectiousness’ can be accurately identified, rates of capture and monitoring are high, and mixing with other populations is minimal. Although all these conditions can exist in some wildlife systems (Garwood, 2018), it is more common that the inability to meet one or all of them diminishes the efficacy of this practice (Wolfe, Watry, Sirochman, Sirochman, & Miller, 2018).
Managing brucellosis in elk is ultimately about limiting risk to cattle because the disease does not pose a major threat to elk abundance. This means minimizing the risk of cattle encountering elk fetuses from brucellosis-induced abortions. Although feedgrounds contribute to the persistence of this dilemma, suggestions of closing them have met with opposition. After all, the feedgrounds divert elk from areas of their winter range where they would comingle with cattle. Our models suggest that this seasonal sequestration has created subpopulations (different feedground herds) within which recurrent brucellosis outbreaks occur, and when local seroprevalence is high the period of greatest spillover risk has likely passed. This reframing of risk, combined with spatial modelling of resource selection, should help identify risky times and places for cattle (Merkle et al., 2018). Finally, scavengers are effective in removing infective tissues (Maichak et al., 2009) and so conserving the scavenger guild is likely beneficial for reducing brucellosis contagion on open rangeland. Nevertheless, these options remain limited while brucellosis is spreading through the growing elk populations in the GYE. Also, chronic wasting disease (CWD) has recently arrived to the GYE and so management actions aimed at controlling one will necessitate consideration of the impacts on both. For example, CWD could create a younger age structure and reduce population growth, but any management efforts to maintain the total abundance of (younger) elk could increase the frequency and intensity of brucellosis outbreaks. Our findings emphasize a need to move beyond traditional control measures and should serve as a warning to agencies that face the possibility of increasing brucellosis infections in elk elsewhere in North America or red deer (Cervus elaphus) in other countries. If infected subpopulations become interconnected then eradicating this troublesome disease could quickly become
impossible without extremely costly and controversial culling campaigns.

**DATA ACCESSIBILITY**

Data and supporting code will be available through the Utah State University Digital Commons.

**REFERENCES**

doi: 10.3389/fcimb.2016.00030


dynamics of a bacterial disease among wildlife and livestock. *Nature Communications*, 7, 11448. doi: 10.1038/ncomms11448


Maichak, E. J., Scurlock, B. M., Cross, P. C., Rogerson, J. D., Edwards, W. H., Wise, B.,


slaughter to reduce prevalence of brucellosis in elk attending feedgrounds in the Pinedale Elk Herd Unit of Wyoming; Results of a 5 year pilot project (p. 20).

Retrieved from Wyoming Game and Fish Department website:
https://wgfd.wyo.gov/WGFD/media/content/PDF/Wildlife/TR_REPORT_2010_FINAL.pdf


TABLES AND FIGURES

TABLE 4-1. Model results using full time series and snowmelt water equivalent data as environmental covariate. At Greys River the endogenous model with a constant transmission term, $\beta$, received the greatest support by AIC and Akaike model weight. At Dell Creek and Muddy Creek, all three models were <2 dAIC different. The effect of a ‘low-density’ feeding treatment was tested with 5 models at Greys River, the only one of the three feedgrounds to adopt the practice.

<table>
<thead>
<tr>
<th>Feedground</th>
<th>Model</th>
<th>loglik</th>
<th>AIC</th>
<th>dAIC</th>
<th>Weight</th>
</tr>
</thead>
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<tr>
<td>Greys River</td>
<td>endogenous</td>
<td>-64.22</td>
<td>152.44</td>
<td>•</td>
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</tr>
<tr>
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<td>9.21</td>
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</tr>
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<td>11</td>
<td>&lt;0.01</td>
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<tr>
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</tr>
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</tr>
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<td>115.97</td>
<td>•</td>
<td>0.44</td>
</tr>
<tr>
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<tr>
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<td>1.97</td>
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<tr>
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</tr>
<tr>
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<td>-33.12</td>
<td>88.24</td>
<td>0.99</td>
<td>0.27</td>
</tr>
</tbody>
</table>
FIGURE 4-1. Wyoming has 23 winter feedgrounds for elk located south of Yellowstone National Park (YNP). The National Elk Refuge (NER) is operated by the U.S. Fish and Wildlife Service, while the remainder are operated by the Wyoming Game and Fish Department. Greys River, Dell Creek, Muddy Creek, and Scab Creek feedgrounds contributed to our analyses.
FIGURE 4-2. Flow diagram for the disease process model with four compartments: S, susceptible; I, infectious; R₁, recovered but seropositive; and R₂, having lost detectable antibodies and immune. Seropositive states outlined in red; seronegative in blue. Female elk are born naïve and enter via calf recruitment (C). All compartments experience an equal period-dependent probability of mortality (μⱼ). Susceptible elk are exposed at a rate corresponding to the force of infection (λ), but not all elk that seroconvert will abort, which allows a proportion (ρ) to transition straight from S to R₁. Recovery occurs with probability σ and detectable antibodies are lost with probability γ. Conditional on being seropositive, the probability of removal ν(t) for test-and-slaughter (TAS) models equaled the proportion of the herd captured for testing in a given year. The measurement model assumed that the number of positive tests (+) was a binomial draw and the probability of the observed female elk count was drawn from a normal distribution.
FIGURE 4-3. Time series simulations for endogenous models at four feedgrounds showing disease trends (left) and female elk count trends (right). Red lines denote observed data; black error bars represent 90% binomial confidence intervals; blue shading indicates 90% prediction intervals from 2000 stochastic simulations with parameter values set at the maximum likelihood estimate.
FIGURE 4-4. Predictions from the endogenous models for Greys River (top), Dell Creek (middle), and Muddy Creek (bottom). Left, estimated force of infection over time with parameter values set at the maximum likelihood estimate. Right, corresponding estimates of the number of individuals in each of the four compartments over time for S (susceptibles), I (infecteds), R1 (recovereds), and R2 (lost antibodies, immune). Dashed lines represent median estimates and 90% prediction intervals from 2000 simulations are shaded.
FIGURE 4-5. Simulations for three hypothetical subpopulations over 100 years under the same starting conditions including initial population size and disease parameters, but with varying death and replacement. The model drivers are endogenous (top), exogenous (bottom), and both combined (middle).
FIGURE 4-6. Left: the estimated number of infective elk present at Muddy Creek feedground during 2005-2018 including 5 years of management intervention (2006-2010, denoted by vertical dashed lines) with four management options: test-and-slaughter of all seropositive female elk (red); no action (grey); test-and-slaughter of 10% of seronegative female elk (blue); indiscriminate culling of 7.5% of female elk (orange). Center: corresponding estimates for seroprevalence under the four regimes. Right: corresponding female count estimates. Colored dashed/dotted lines represent median estimates; shaded areas are 50% prediction intervals from 2000 simulations.
CHAPTER 5
UNINTENDED CONSEQUENCES OF A SUCCESSFUL WILDLIFE RECOVERY:
DEPREDATION AND DISEASE CHALLENGE ELK MANAGEMENT IN THE
AMERICAN WEST

Abstract:
The resurgence of once-dwindling elk populations across the American West constitutes a success story resulting from concerted policy and reintroduction efforts. Over time, new management framings have emerged, from restoration, to reducing depredation, and now to controlling disease, with each adding another layer of challenges above the other. Whereas extirpation and depredation can both be characterized as cost-benefit mismatches across the public-private divide, disease conflicts are variable: their impacts can sprawl across sectors, agencies and industries. As such, disease creates different policy challenges that, unchecked, may erode hard-won conservation successes. Aligning the costs and benefits of disease management will require novel approaches tailored to specific disease systems that in some cases may risk violating tenets of the North American model of wildlife management, but as exemplified by elk, inadequate disease control can have ripple effects across multiple species resulting in worse conservation outcomes.

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In a nutshell:

- Elk management in the Rocky Mountains has become framed in different ways when new challenges have presented themselves as negative consequences of earlier successes.
- The first framing of formal management efforts for elk is population recovery to restore an iconic big game species and provide recreational hunting opportunities.
- The second framing is to reduce depredation of winter food resources for livestock and to reduce negative impacts on habitat.
- The third framing is to control the spread of diseases and spillover to livestock.
- The three framings have not replaced one another and have become superimposed, adding complexity and conflict to the challenges faced by wildlife managers.
- The lesson is that the response of wildlife populations to conservation efforts can outpace the evolution of the policy framework in which managers operate.

In the late 1800s, the Greater Yellowstone Ecosystem (GYE) was a refuge for remnant elk (*Cervus canadensis*) populations by accident, then design: rugged terrain precluded easy access and land conversion, providing protection from unregulated killing which was later codified with the establishment of Yellowstone National Park (YNP) in 1872 and the establishment of state game agencies which regulated ‘take’. These populations served as the source for reintroduction efforts across the West and later into eastern states (Bryant and Maser 1982). The successful recovery of elk populations created new ecological and social conflicts along the way for which existing policy was
unprepared. We outline 3 conservation framings, which, rather than replacing one another reflect the radiation of abundant elk outwards from the GYE and the evolution of policies meant to address changing objectives. As one of the most popular hunted species in North America, elk offer a useful illustration of changing management frameworks because they are both a posterchild for conservation success and challenges.

**Framing 1: Restoration and reestablishment**

In the early 1900s, Wyoming had the largest remnant elk population and became the source for most reintroduction efforts. Following a grassroots effort to mitigate winter starvation and hay crop losses, the National Elk Refuge was established in 1912 and its success in these respects motivated the establishment of additional diversionary and supplemental feeding sites for ungulates by the state (Figure 5-2). The two-fold goal was to increase elk abundance and limit depredation.

In principle, the dilemma of ‘too few animals’ is simple when the cause is unregulated killing. In practice, rectifying the situation meant creating new laws and institutions with enforcement powers. Formalizing and enforcing hunting regulations (seasonal closures, bag limits, restricted methods of take) was a first step. State game agencies were established for this purpose, and their revenue derived primarily from the sale of hunting licenses and later also from federal taxes on firearms and ammunition (Organ *et al.* 2012). The reliance of agency budgets on hunting incentivized abundant game, which was perfectly aligned with the original goal of restoration. In at least this one respect there has been success, with elk populations approximately doubling since the 1970s (Popp *et al.* 2014). But over time this led to conflicts among stakeholders for
which existing rules and norms were unprepared. The centrality of hunting to wildlife management thus has an organic historical basis and remains an important driver and funding source for conservation efforts (Jacobson et al. 2010), but it cannot solve issues which its governing institutions are incentivized to create. Further, arguments that hunting is, and must remain, the basis of wildlife conservation in North America are facile: they ignore the contributions of non-hunting partners in conservation (Nelson et al. 2011), they implicitly but arbitrarily (from an ecological perspective) value certain species above others, and they rest on the logical fallacy that the future will be like the past (appeal to tradition). As land use, land ownership and human demographics change, hunting interest and opportunity can wane, shrinking license sales and thus budgets. For proper conservation and management to continue, alternative revenue streams that extend beyond hunting activities are desperately needed (Jacobson et al. 2010).

**Framing 2: Managing socioecological conflict**

‘Depredation’ describes property damage caused by wildlife. The conflict arises when the property is privately-owned, and the wildlife are publicly-owned. In the case of elk, this typically means consuming hay crops meant for domestic livestock and damage to fences. Historically this was a seasonal phenomenon but increasingly occurs year-round as elk behavior responds to irrigated landscapes (Barker et al. 2019). Framed as a misalignment of costs and benefits, the publicly-owned elk exist at a cost to private citizens. State agencies have responded by creating additional diversionary feedgrounds (Rimbey et al. 1991), subsidizing fencing costs of private hay crops, or by offering direct reimbursements to ranchers (Wagner et al. 1997). Increased hazing operations and lethal
removal are also in widespread use. Depredation issues are more apt to be resolved through hunting at low abundance because it pressures elk to relocate, but as elk abundance increases, or norms, attitudes and land use changes, this becomes less effective (Haggerty and Travis 2006).

Recent trends in the GYE toward restricting hunting access on private lands have further exacerbated conflict. Hunting interests aside, the issue of public wildlife on private land presents a major dilemma. Across a patchworked ownership landscape, elk adapt to variable hunting pressure (or human presence) and take refuge on private lands (Burcham et al. 1999). The reduced access and opportunity is a problem for state agencies. Some states have adopted incentive programs wherein private property owners are given hunting tags which they can sell on the open market in return for allowing hunting access or making habitat improvements (see Messmer et al. 1998). These programs ‘indirectly’ align costs and benefits: private landowners are provided a mechanism through which to profit from wildlife without transferrance of legal ownership in return for allowing public access (Watson 2012). Programs like this highlight a central policy debate. Regulated commercial harvest is occasionally proposed as a solution to overabundant game (VerCauteren et al. 2011), but any weakening of the public trust doctrine sets a dangerous precedent that could erode protections for wildlife. At the same time, conflict itself is perceived as a failure in management contributing to negative conservation outcomes (Jacobson et al. 2010).

Habitat degradation is another unforeseen consequence of the emphasis on abundance amplified by feeding operations. Across the West, aspen (Populus tremuloides) are disappearing due to poor regeneration, which is partly due to
overbrowsing by elk (Bartos and Campbell 1998). Overbrowsing by ungulates directly reduces aspen cover which provides critical habitat for a range of species and has negative hydrologic consequences (LaMalfa and Ryle 2008). Notably, this intersects with predator restoration and conservation. Wolf (*Canis lupus*) reintroductions and predator protections were proposed as a way of limiting elk numbers, promoting aspen regenerations, and thereby improving hydrologic function although whether this ‘cascade’ of effects has or is occurring is hotly debated (Ripple and Beschta 2007; Kauffman *et al.* 2010; Kohl *et al.* 2018). In like fashion, abundant elk and feedgrounds have contributed to conflicts surrounding disease.

**Framing 3: The era of disease**

Major disease conflict with GYE elk emerged in the early 2000s when elk were found to be the source of brucellosis (*Brucella abortus*) spillover to cattle (Rhyan *et al.* 2013). Since then, that bacterium has been pivotal in shaping the management of GYE elk and bison. The disease originated with cattle and was transmitted to bison and elk which are now reservoir hosts (Meagher and Meyer 1994). Elk periodically transmit the disease back to cattle, which is an expanding problem (Figure 5-3). Changes in land use and land ownership complicate management in the GYE, with a recent influx of wealthy absentee or amenity landowners comprising a prominent shift. When private land issues predominantly involve working landscapes, there is mutual interest in disease management and working with state agencies to reduce risk, but the motivations of this newly arrived group for owning property are more apt to center on privacy and wildlife viewing. While this precludes their own potential for depredation conflict, it can
exacerbate depredation and disease conflict with neighbors and have other negative conservation outcomes like lost migratory behavior (Figure 5-4). Amenity landowners are less likely to allow hunting or other management actions, creating large areas (and wildlife populations) ‘outside of administrative control’ (Haggerty and Travis 2006). This is a particularly pernicious problem with brucellosis because the effects to elk are not obvious to casual observers. These herds on private land nevertheless expose neighboring elk, bison, and cattle to increased risk. Disease-free bison herds have been reestablished in a number of regions, but these are susceptible to brucellosis exposure from free-ranging elk, after which disease eradication in the bison herd may be impossible without culling. This could put bison owners/managers in the same position as YNP officials in managing spillover potential to cattle, which is restricting movement and conducting test-and-slaughter of seropositive dams (see Box 1).

Managing for fewer ungulates might seem anathema to much of the public, although the encroachment of chronic wasting disease (whose effects are more visible) could eventually change attitudes. Framed as an issue of overabundance, ecologically sound remedies have been proposed which do not have broad social or political support across the GYE: (1) ban feeding, (2) increase hunting until populations are smaller (manage for quality not quantity), and (3) restore habitat, and in this case specifically, native winter range (Gortázar et al. 2006). An ecological lesson that merits consideration and could be used to advantage in disease control is that of population cycling. Although the mechanisms driving the ‘10-year population cyle’ remain a mystery (Myers 2018), there is at least one clear evolutionary benefit to the phenomenon, which is the disruption of specialist predator population growth that allows prey populations to rebound. In elk,
this would mean rotationally focusing hunting pressure to drastically reduce elk numbers for a time and would have to be coordinated in such a way that herds are not simply displaced. Across an entire state, the objective might be for stable elk numbers, but within hunt areas, numbers might fluctuate wildly over time. The ultimate goal would be healthier herds achieved by mimicking predation to disrupt transmission (Roy and Holt 2008).

**Box 1: Bison restoration despite brucellosis: Utah and Montana**

Bison are inextricably linked to elk conservation in the West by virtue of their association with Yellowstone National Park and brucellosis. At the northern park boundary with Montana, seasonal bison movements outside the park have been a major source of conflict: first with ranchers due to depredation and disease risk, then due to agency cullings of bison. The disease was brought under control in cattle by the late 1990s, at which point all states were classified ‘brucellosis free’. It is somewhat of a historical accident that the first ‘spillback events’ of the disease from wildlife to cattle occurred in this region, and initially the blame was placed on bison. Later it became clear that spillback originated from elk, but it was already too late; the public perception was set. Over the ensuing decades and court battles, Montana designated bison as ‘livestock’ and therefore private property rules apply. This allows private entities to manage bison herds, profit from them, and also ‘restore’ bison herds to new areas (eg. American Prairie Reserve) without state agency involvement. By placing bison outside of state agency control bison hunting does not exist in Montana in the traditional sense.

By contrast, Utah is home to several bison herds which are managed by the state
and are highly valued by hunters. Bison from YNP were relocated to the Henry Mountains in Southern Utah in the 1940s and fortuitously have remained both disease-free and free from genetic introgression with cattle (Ranglack et al. 2015). The Utah state agency has continued to manage them, and used them as source populations for new herds within Utah. Bison in the Henry Mountains have been a source of classic private/public conflict: local ranchers see them as competing with cattle, and are unable to profit from their presence, while the state is incentivized to keep as many bison as possible for trophy hunting opportunity.

Despite the contrasting approaches of the two states to bison management, neither has been able to avoid conflict and legal battles. Meanwhile, brucellosis in free-ranging elk is expanding, and therefore poses a threat to bison restoration efforts that are no longer connected to YNP bison.

**Conclusions**

A single ‘right’ number of individuals of a given species does not exist. Nevertheless ‘abundance’ forms the cornerstone statistic that informs much of wildlife management and the agencies’ very own ability to operate. Whereas the long term viability of populations, or the resilience of systems, may be more laudable goals, they involve so many other factors as to be abstract. (E.g., no funding mechanisms exist which align management principles with resilience.)

With unique respect to disease management, the emphasis on continuously growing or stable host numbers may be at direct odds with pathogen control. Managing for maximum sustainable yield ensures a constant supply of young, susceptible
individuals to perpetuate disease outbreak, and winter feeding operations maintain high
over winter survival which supports higher densities of elk than native winter range can
support (Chapter 4). Fluctuating host population size might be a good thing for pathogen
control to the extent that transmission is density-dependent. Periods of declining growth
might disrupt pathogen spread and increase the probability of local pathogen extinction.
In theory predators could fill this role (Roy and Holt 2008), on the other hand they
prepresent their own source of conflict. A surrogate for intensive predation could be high
hunter harvest which is focused in space and time on specific herds.

Currently demographic responses of managed populations are mismatched with
the rate of change of the policy framework that governs management. Because of this,
‘too much of good thing’ can rapidly become a problem. Conflict arising from disease is
is projected to increase. Because managing public resources on private lands requires
voluntary action of landowners, as private parcels become smaller, achieving common
purpose and coordinating management efforts at sufficient scale becomes intractable. The
financial incentives of stakeholders are diverse, and can be at odds with conservation
goals. Social attitudes also vary geographically, and needs will vary depending on
specific disease systems. New paradigms and possibly new institutions are required to
improve the integration of policies governing diseases in livestock and wildlife, as
complicated by emerging land tenure patterns and evolving public attitudes towards
hunting and wilderness.
References


FIGURES

Figure 5-1. A group of bull elk in Wyoming in winter. Photo credit: Mark Gocke, Wyo. Game & Fish Dept.

Figure 5-2. A bull elk feeding alongside calves during winter at one of Wyoming’s state run feedgrounds. Photo credit: Mark Gocke, Wyo. Game & Fish Dept.
Figure 5-3. Nationwide, elk abundance has approximately doubled in the last half century. Solid blue dots indicate states with remnant elk populations in the early 1900s. Thin inner circles indicate relative abundance of free-ranging elk in the 1970s. Thick outer circles indicate state-specific elk abundance. The current Designated Surveillance Area boundary (DSA) for brucellosis is shaded in red, with the projected DSA expansion by the year 2038 in yellow.
Figure 5-4. Misalignment of costs and benefits within the public trust framework. Arrows indicate directionality of economic burden. Historically private landowners suffered costs associated with damages from abundant elk, for which states have developed partial solutions. Although depredation is still a concern, disease burdens at the wildlife-livestock interface are an expanding problem. Amenity landownership creates ‘private herds’ which exacerbate conflict by putting wildlife outside of management oversight.
CHAPTER 6
DISCUSSION

Although nearly eradicated in the United States, bovine brucellosis (*Brucella abortus*) is once more rising to prominence due to growing and interconnected elk (*Cervus canadensis*) populations in the West. *Brucella* organisms fall under USDA Select Agent status and this barrier not only hinders vaccine development, but captive trials which could illuminate other factors important to disease control in wildlife like host-specific epidemiological transition rates. Such information would likely expand our ability to accurately model transmission dynamics of brucellosis within and across species, and lend greater confidence to spatial and predictive models. In the absence of this ability, and as the preceding chapters demonstrate, increasingly sophisticated methods can be applied to ever-growing datasets of free-ranging animals to reveal new information vital to disease control in wildlife.

In Chapter 2, I reviewed the current state of our knowledge surrounding the disease ecology of brucellosis in elk and the role that supplemental winter feedgrounds have played. In Chapter 3, I demonstrated that, through still unknown mechanisms, female elk experience reduced reproductive output as a consequence of brucellosis exposure apart from the disease-induced abortions which afflict some, but not all, female elk that become infected. The size of this effect nearly doubles what we believed was the total cost to exposed individuals. In Chapter 4, I evaluated test-and-slaughter of seropositive female elk as a management strategy to control the spread of brucellosis and in the process developed mechanistic models for the underlying transmission process in
feedground elk herds. Finally, in Chapter 5, I used brucellosis in elk to highlight changes in management and policy that have occurred or need to occur in order to address the unforeseen dilemmas that arise from species abundance, including the proliferation of zoonotic and spillover diseases.

The understanding of brucellosis in elk and the role of supplemental winter feeding that I develop in Chapter 2 sets the scene for the remainder of the dissertation. Critically, the dynamics of this multihost pathogen have changed over time as other circumstances have changed. *B. abortus* in the Greater Yellowstone Ecosystem (GYE) originated with domestic cattle, but the balance—in terms of culpability for transmission and expanding the spatial extent of the pathogen—has shifted away from bovids towards fed and unfed elk. This motivates our focus on this one host in terms of the disease’s effects and intraspecific transmission dynamics. It also paints feedgrounds in a slightly more complicated light. Whereas in the past some have viewed elimination of winter feedgrounds as a silver bullet solution (Bienen & Tabor, 2006), in light of the increasing seroprevalence observed in growing elk herds far from feedgrounds (Cross et al., 2010), it is apparent that the underlying conditions that facilitate intraspecific transmission exist elsewhere in the GYE. This does not mean that feeding should continue indefinitely, rather that winter feeding is an imperfect compromise to meet certain management objectives and that once a feedground exists, it is difficult to close.

In Chapter 3 I demonstrated that seropositive female elk are less likely to be pregnant in mid-winter than seronegative female elk. The mechanism driving this phenomenon is unknown, but we found compelling evidence suggesting this difference is not due to disease-induced abortions. Were this reduced pregnancy the result of
additional abortions, we should have seen the magnitude of the effect change over the
course of the sampling season, since abortions peak between March and May (Cross et
al., 2015). There is additional evidence corroborating these findings from microbiological
research (Ahmed, Zheng, & Liu, 2016; Jamil et al., 2017), and feedground observations:
abortions (and fetuses) are rarely detected on feedgrounds. As a byproduct of how these
data were collected I structured this question by age class and found the effect was most
pronounced among two year-olds. However, because the probability of exposure
increases linearly with age, it is equally plausible that probability of pregnancy is
impacted as a function of recency-of-exposure, not age itself. This is a good example of a
question that might be definitively answered through captive trials but that we cannot
adequately address with our current longitudinal data. There are other intriguing
implications from this work as well. The magnitude of the cost to individual reproductive
output as a consequence of infection is large enough that if there is heritable resistance to
brucellosis infection, this should drive a selection gradient. Additionally, if recency-of-
infection, rather than age, is associated with reduced reproductive output like it is for the
probability of abortion, then there should be a strong temporal signal. In other words, in
the years during an outbreak, there should be drastically lower calf production. If
outbreaks have a strong impact on demographic rates, the pathogen itself could be an
additional driver of asynchronous or unstable disease dynamics across subpopulations.
This should be of interest to theoretical ecologists and is relevant to Chapter 4. Another
important finding from this work (presented in Appendix B) is the statistically significant
decline in fluorescent polarization assay score (a serological test) that occurs over time,
corroborating the findings in Benavides et al. (2017).
In Chapter 4 I found a pattern of recurrent brucellosis outbreaks at individual feedgrounds over the preceding 25 years. These outbreaks were not perfectly synchronized with one another across the region-wide metapopulation of fed elk. I used partially-observed Markov process models to develop a mechanistic understanding of the transmission dynamics of the system while accounting for the opacity of serological data and tried to disentangle the effects of various drivers of transmission. Lacking explicit elk density data, I instead sought to use environmental data to inform transmission. This proved, perhaps, too imprecise for our modeling approach. However, it turns out that demography, and specifically calf recruitment, explained much of the elk-to-elk transmission dynamics within individual feedground herds. This is in line with previous theoretical work on metapopulation disease dynamics (Lloyd & Sattenspiel, 2010), and well-studied human systems like measles (Rohani, Earn, & Grenfell, 1999). Nevertheless it shines the light on a salient and underappreciated point, which is that the predisposition of populations to a disease outbreak is particularly sensitive to the inflow of new susceptibles for immunizing pathogens in a long-lived host. This variability of calf recruitment, which in the previous chapter we demonstrated can be influenced by the pathogen itself, in turn can lead to divergent population growth trajectories which we should expect to produce asynchronous disease trends across subpopulations even if they experience (and are in part driven by) identical climatic forcing.

These mechanistic models also give us a glimpse behind the veil of serostatus and reveal important insights, chief among them being that there is a strong temporal component in this system. The effects to an individual (and their ability to transmit the pathogen to others) is largely restricted to a single year, and nearly half of all exposed
female elk likely never transmit the pathogen. On the other hand, by the time seroconversion occurs and winter elk handling occurs at feedgrounds, the vast majority of seropositive individuals are likely recovered. Management interventions to elk that use brucellosis seroprevalence or serostatus as a metric must be aware of this fact, and the counterintuitive conclusion that seronegative individuals may pose a greater transmission risk than their seropositive counterparts. This explains why test-and-slaughter of seropositive elk is a counterproductive strategy for reducing contagion and spillback risk to cattle in this system.

Chapter 5 puts the dilemma of brucellosis in the GYE in the wider historical, conservation, and management context. Elk are highly valued by the public at large and likely have been since humans first arrived on the continent. This has helped to drive a rebound in elk populations across the West and into eastern states from which they were extirpated following European colonization. Despite a broad appreciation of the species, there are those who experience the brunt of the costs associated with living near this large ungulate. Originally depredations to private hay crops was the main (perhaps only) conflict which garnered attention. These were also focused in winter and motivated the creation of diversionary feedgrounds. Spillback risk of brucellosis from elk to cattle has amplified conflict, and there has also been a ‘spillover’ of negative conservation outcomes for bison due to continued perception among ranchers that bison pose a major risk. This leads to the idea that current levels of elk abundance may, in fact, be ‘too much of a good thing’. From this, in conjunction with previous chapters and preexisting theoretical work, we arrive at the idea that managing game species in a way that accommodates fluctuating abundance might facilitate disruptive cycling of pathogen
transmission.

The overall goal of this dissertation was to increase our understanding of the disease ecology of brucellosis in elk and provide new insights that inform future management solutions. To do this I used a unique longterm disease dataset from Wyoming’s state run winter feedgrounds. This information allowed me to identify unrecognized effects that the pathogen has on host demography, and in complementary fashion, the important role that host demography plays on the transmission dynamics of the pathogen. An inescapable applied result for managers is to exercise caution when using seroprevalence as a metric for evaluating management actions, and that serostatus alone says little of an individual elk’s ability to transmit Brucella. Serological testing forms the basis of this powerful dataset, but mechanistic modeling may be required in order to make sense of these data in time series.

REFERENCES


APPENDICES
Section A-1: Does the probability of pregnancy among seropositive female elk decrease over the sampling period?

From previous work we expected that February was the earliest possible time that abortions could occur (Cross et al., 2015), but that they should not happen with any great frequency until March, April, and May. Blood samples were collected between January and April at feedgrounds for any given year. 90% were collected prior to March 1. If the difference in pregnancy between seropositive and seronegative elk was due to disease-induced abortions, then, using seropositive pregnancy test data, we would expect a decreased probability of pregnancy later in the sampling period. The raw pregnancy data for seropositive elk provides a sense of data distribution (Fig. A-1).
Figure A-1. Proportion of the sample pregnant by 10-day period of the calendar year (n=587). The width of the bars reflects sample size per 10-day window. Almost all of the data was collected in the 40-day period between January 20th and March 1st. Approximately 80% of individuals represented in the sample were pregnant (lighter shading where ‘pregnant = 1’ corresponds to pregnant).

We tested for a significant change in pregnancy over the sampling period with 3 models. First, a generalized linear model where, $\alpha_0$ was the intercept for the probability that a seropositive elk is pregnant and $\beta_0$ was the slope coefficient for the time covariate, $\zeta_i$:

$$logit(p_i) = \alpha_0 + \beta_0 \zeta_i$$

The second model incorporated an age-varying intercept, while the third incorporated an age-varying intercept, $\alpha_j$, and age-varying slope, $\beta_j$:

$$logit(p_{ij}) = \alpha_0 + \alpha_j + (\beta_0 + \beta_j) \zeta_i$$

We standardized Julian day by subtracting the mean and dividing by the standard
deviation. Model 3 received the lowest WAIC and the majority of the model weight (table A-1).

Table A-1. Model results for the effect of Julian day on the probability of a seropositive elk being pregnant. Model 3, which included an age-varying intercept and slope, was the top model.

<table>
<thead>
<tr>
<th>model</th>
<th>intercept</th>
<th>slope</th>
<th>WAIC</th>
<th>weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>age-varying</td>
<td>age-varying</td>
<td>626.7</td>
<td>0.65</td>
</tr>
<tr>
<td>2</td>
<td>age-varying</td>
<td>fixed</td>
<td>627.9</td>
<td>0.35</td>
</tr>
<tr>
<td>1</td>
<td>fixed</td>
<td>fixed</td>
<td>648.8</td>
<td>0.00</td>
</tr>
</tbody>
</table>

The overall beta estimate from this model was 0.02 (89% HPDI = -0.30, 0.38). The point prediction of the model corresponded to a 2 percentage point increase in the probability of pregnancy for seropositive elk between early January and mid-April, however the credible intervals are very wide and overlap zero. Thus we found no evidence to suggest the probability of pregnancy changes over the sampling period.

These model results suggest that the difference in pregnancy probability between seropositive and seronegative elk was either due to reduced rates of conception among seropositive elk, or intrauterine mortality prior to January. In either case, this appears to be a separate phenomenon from the disease-induced abortions which occur annually among ~16% of seropositive and pregnant elk.
Section A-2: Do lower fluorescent polarisation assay (FP) test results correspond to higher pregnancy probability in seropositive elk?

We found a larger apparent effect of serostatus on pregnancy probability among young animals. Antibodies for brucellosis in elk are retained for many years but can eventually be lost (Benavides et al., 2017). If that were not the case, then seroprevalence should increase linearly with age following Muench's catalytic model (Muench, 1959). Instead, age is only generally correlated with disease exposure and time-since-infection. Elk could recover from the reproductive consequences of the disease, but remain seropositive, which is possibly why the effect of serostatus on pregnancy is attenuated at older age. In other words, the recency with which the infection was acquired may be more relevant than age. FP scores give us a continuous measure of serology to work with, so a reasonable question to ask is whether seropositive elk are more likely to be pregnant when FP scores are lower.

Our sample size for this analysis was 510 records. The GLM, \( \logit(p_i) = \alpha_0 + \beta_0 \eta_i \), where \( \eta_i \) corresponded to the standardized FP value of individual \( i \), generated a beta estimate of -0.07 and CIs overlapping zero (89% HPDI = -0.25, 0.09). These results do not appear significant. The 89% HPDI estimates correspond to a span from a 5% increase to 2% decrease in pregnancy probability per one standard deviation of FP score decrease and the standardized values of FP score in our data ranged from -2.8 to 2.1.

Does the probability of pregnancy increase as time-since-infection increases?

Based on the repeat capture of marked individuals with repeat serology and pregnancy test results we were able to generate 242 records where we knew the year in
which the individual elk first tested seropositive. Sample sizes are indicated in table A-2.

**Table A-2.** The number of pregnancy-specific protein B results for individuals with a known seroconversion date based on longitudinal records. These observations represent 134 unique individuals.

<table>
<thead>
<tr>
<th>Time since first seropositive test</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample size</td>
<td>134</td>
<td>46</td>
<td>35</td>
<td>6</td>
<td>7</td>
<td>7</td>
<td>3</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

Thorne et al. (1978) reported an average incubation period of 39 days following artificial infection to become seropositive, and due to the timing of abortions (transmission) we strongly suspect that in almost all cases individuals can only first be detected as seropositive in the winter following the winter/spring in which they contracted the infection. As such, 0 'time since first seropositive test' should equate to 1 year since becoming infected. And indeed the proportion of those records that were pregnant is 77%, which is very similar to what we expect from the data for all seropositives, ignoring age. By contrast, if these reflected individuals which had become infected in the previous month or two, the proportion that are pregnant should be closer to 90%.

With this new dataset, we can also test whether FP scores actually decrease with time the way we anticipated. We end up with 212 observations to test this notion. This is a simple linear model $FP \sim Normal(\mu, \sigma)$, and $\mu = \alpha + \beta\delta$, where $\delta$ is the 'time since first seropositive test' covariate. Our intercept ends up being 218, with a beta estimate of -15.09 (89% HPDI = -18.95, -11.02). This looks significant and supports the hypothesis that lower FP scores may be indicative of longer time since infection. Still, caution is
warranted lest we read too much into FP values.

A generalized linear model gives us a beta estimate for the effect of time-since-infection of -0.01 with wide credible intervals (89% HPDI = -0.15, 0.13). This intercept translates into a 77% probability of pregnancy and an estimated 2% increase in the probability of pregnancy for each year afterwards, but the effect is not significant. This does not lend support for the idea of recovery. Although these data are imperfect, we do have >30 samples for the 1st, 2nd, and 3rd years following first seropositive test.

The conventional belief among brucellosis/elk/feedground researchers since Thorne et al.’s captive study in the 1970s was that some elk experience reproductive failures in the year or two following infection, and afterwards emerge recovered and relatively unscathed. We expected that the same would hold true for what appears to be ‘failure to conceive’, but so far we have not found evidence to support that belief.

Section A-3: Estimating a ‘total apparent effect’ of serostatus.

Method 1: “Accounting for herd age structure and age-specific prevalence”

One way to do this is to calculate an average based on the age-specific prevalence curve (Fig. A-2), some population structure (Fig. A-3), and the point estimates from the age-specific percentage point differences in pregnancy probability by serostatus (from our top model). Age-specific prevalence follows that reported in Benavides et al. (2017). Here we used all known-age data from the Wyoming feedgrounds and fit a smoother.
Figure A-2. Age-specific seroprevalence for brucellosis of Wyoming feedground elk. Open blue circles represent raw data, while the black line represents model estimates from a generalized additive model, and the shaded area represents 95% confidence intervals.
Figure A-3. A simulated population structure for elk spanning from 6 months old to 18 ½ years old. Dots represent the proportion of the total population belonging to each age.

An approximate population structure was simulated using the density of the exponential function with a rate of 0.25, which fit the distribution of our known-age data well. The point estimates for the difference in pregnancy probability (the percentage point difference from the seronegative mean) were 0.16 (yearling), 0.31 (2-year-olds), 0.7 (3- to 9-year-olds), and 0.2 (10+).

If, for each age from 6 months to 18 ½ years, we multiply the age-specific prevalence by the proportion of the population by the expected difference in pregnancy probability and take the average, we arrive at 0.12, or 12 percentage points fewer than than the seronegative expectations.

Method 2: accepting an average effect of serostatus on pregnancy that includes some age-bias
Ignoring the age-varying effects of serostatus but keeping age-varying intercepts (as in model 3), we can calculate the average expected effect across all ages, again using point estimates. The percentage point differences from model 3 end up being 13.4% (yearling), 12.9% (2-year-olds), 11% (3- to 9-year-olds), and 16% (10+). The weighted average for all ages up to 18 ½ years old is then 13.7%.

Section A-4

Table A-3. Model rankings for 8 models to predict the number of calves counted per 100 adult female elk in year \( t \) by serological parameters. Increases in seroprevalence at a site in the last 1 or 2 years were associated with fewer expected calves, shown here with 89% HPDI.

<table>
<thead>
<tr>
<th>rank</th>
<th>intercepts</th>
<th>serological parameter</th>
<th>WAIC</th>
<th>weight</th>
<th>estimated effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>site-varying</td>
<td>( \Delta \ t-2 ) to ( \Delta \ t-1 )</td>
<td>450.7</td>
<td>0.41</td>
<td>-1.60 (-2.79, -0.31)</td>
</tr>
<tr>
<td>2</td>
<td>site-varying</td>
<td>( \Delta \ t-1 ) to ( \Delta t )</td>
<td>451.9</td>
<td>0.23</td>
<td>-1.39 (-2.64, -0.10)</td>
</tr>
<tr>
<td>3</td>
<td>site-varying</td>
<td>prevalence ( t-1 )</td>
<td>452.9</td>
<td>0.14</td>
<td>-0.90 (-2.16, 0.38)</td>
</tr>
<tr>
<td>4</td>
<td>site-varying</td>
<td>prevalence ( t-2 )</td>
<td>453.7</td>
<td>0.09</td>
<td>-0.30 (-1.51, 1.03)</td>
</tr>
<tr>
<td>5</td>
<td>fixed</td>
<td>( \Delta \ t-2 ) to ( \Delta \ t-1 )</td>
<td>454.1</td>
<td>0.08</td>
<td>-1.98 (-3.24, -0.74)</td>
</tr>
<tr>
<td>6</td>
<td>fixed</td>
<td>( \Delta \ t-1 ) to ( \Delta t )</td>
<td>455.3</td>
<td>0.04</td>
<td>-1.83 (-3.04, -0.59)</td>
</tr>
<tr>
<td>7</td>
<td>fixed</td>
<td>prevalence ( t-1 )</td>
<td>459.5</td>
<td>0.00</td>
<td>-0.89 (-2.17, 0.39)</td>
</tr>
<tr>
<td>8</td>
<td>fixed</td>
<td>prevalence ( t-2 )</td>
<td>461.1</td>
<td>0.00</td>
<td>-0.07 (-1.38, 1.22)</td>
</tr>
</tbody>
</table>
Figure A-4. Results from the top model for the effect of serological parameters on the number of calves present during peak winter per 100 adult female elk. The solid black lines represent the mean estimate for the effect of a change in seroprevalence from year $t-2$ to $t-1$ (standardized), while the shaded portion represents the 89% highest posterior density interval. Allowing the intercept to vary by site modestly improved model fit. The model predicts 1.6 fewer calves per 100 adult female elk in year $t$ for an increase in one standard deviation of seroprevalence (5.8%) from year $t-2$ to $t-1$. 
Figure A-5. Estimates for the year-varying intercepts from the top model for pregnancy. In only 4 of 23 instances does it appear that a year has been significantly different from the mean in terms of overall levels of elk pregnancy at the feedgrounds. Alpha_k[23], which corresponds to 2017, is the outlier.
APPENDIX B

CHAPTER 4 SUPPLEMENTAL INFORMATION

Section A. Serologic sample size and capture rates

Table B-1. Serologic sample sizes for Greys River, Dell Creek, Muddy Creek, and Scab Creek feedgrounds.

<table>
<thead>
<tr>
<th>Year</th>
<th>Greys River</th>
<th>Dell Creek</th>
<th>Muddy Creek</th>
<th>Scab Creek</th>
</tr>
</thead>
<tbody>
<tr>
<td>1993</td>
<td>56</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1994</td>
<td>22</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1995</td>
<td>38</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1996</td>
<td>33</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1997</td>
<td>38</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1998</td>
<td>43</td>
<td>34</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1999</td>
<td>35</td>
<td>37</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2000</td>
<td>36</td>
<td>22</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2001</td>
<td>37</td>
<td>35</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2002</td>
<td>42</td>
<td>33</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2003</td>
<td>55</td>
<td>30</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2004</td>
<td>38</td>
<td>36</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>2005</td>
<td>66</td>
<td>34</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>2006</td>
<td>24</td>
<td>25</td>
<td>171</td>
<td>16</td>
</tr>
<tr>
<td>2007</td>
<td>36</td>
<td>37</td>
<td>81</td>
<td>39</td>
</tr>
<tr>
<td>2008</td>
<td>38</td>
<td>43</td>
<td>158</td>
<td>4</td>
</tr>
<tr>
<td>2009</td>
<td>43</td>
<td>44</td>
<td>114</td>
<td>151</td>
</tr>
<tr>
<td>2010</td>
<td>48</td>
<td>37</td>
<td>141</td>
<td>141</td>
</tr>
<tr>
<td>2011</td>
<td>50</td>
<td>46</td>
<td>72</td>
<td></td>
</tr>
<tr>
<td>2012</td>
<td>42</td>
<td>24</td>
<td>70</td>
<td>65</td>
</tr>
<tr>
<td>2013</td>
<td>42</td>
<td>67</td>
<td>55</td>
<td></td>
</tr>
<tr>
<td>2014</td>
<td>31</td>
<td>71</td>
<td>86</td>
<td></td>
</tr>
<tr>
<td>2015</td>
<td>44</td>
<td>30</td>
<td>56</td>
<td>3</td>
</tr>
<tr>
<td>2016</td>
<td>59</td>
<td>49</td>
<td>68</td>
<td>73</td>
</tr>
<tr>
<td>2017</td>
<td>74</td>
<td>1</td>
<td>78</td>
<td>7</td>
</tr>
<tr>
<td>2018</td>
<td>24</td>
<td>9</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table B-2. The number of female elk captured from Muddy Creek and Scab Creek during test-and-slaughter from Scurlock et al. (2010).

<table>
<thead>
<tr>
<th>Feedground</th>
<th>Year</th>
<th>No. captured and tested</th>
<th>No. removed</th>
<th>Percent of attending females captured</th>
<th>Total female count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muddy Creek</td>
<td>2006</td>
<td>158</td>
<td>58</td>
<td>60%</td>
<td>263</td>
</tr>
<tr>
<td></td>
<td>2007</td>
<td>79</td>
<td>13</td>
<td>35%</td>
<td>228</td>
</tr>
<tr>
<td></td>
<td>2008</td>
<td>154</td>
<td>21</td>
<td>62%</td>
<td>249</td>
</tr>
<tr>
<td></td>
<td>2009</td>
<td>114</td>
<td>8</td>
<td>38%</td>
<td>301</td>
</tr>
<tr>
<td>Scab Creek</td>
<td>2010</td>
<td>141</td>
<td>7</td>
<td>56%</td>
<td>253</td>
</tr>
<tr>
<td></td>
<td>2009</td>
<td>149</td>
<td>31</td>
<td>29%</td>
<td>514</td>
</tr>
<tr>
<td></td>
<td>2010</td>
<td>141</td>
<td>27</td>
<td>31%</td>
<td>457</td>
</tr>
</tbody>
</table>

Section B. Discretized equations of the basic models.

Let $\delta$ equal one year. We modeled female elk only and so divided the number of calves counted in each year at each feedground by two under the assumption of equal sex ratio in calves. Susceptible female elk ($S$) are exposed and seroconvert according to force of infection $\lambda$. A proportion of those, $\rho$ never become infectious (abort) and transition straight to $R_1$. With no disease-induced mortality, all compartments are equally subjected to natural mortality $\mu_j$, where $j$ indicates different time periods 1 - J: at Muddy Creek J=1, At Greys River J=2, at Scab Creek J=3. At Dell Creek J=2, but $j = 1$ defines the period prior to 2008 and after 2012, $j = 2$ from 2008-2012. $\sigma$ is the probability of recovery from $I$ to $R_1$, while $\gamma$ is the probability of seroreversion (losing detectable antibodies), governing the $R_1$ to $R_2$ transition. The true population size of the latent process, $N(t)$ is the sum of the four compartments. The true seroprevalence of the latent process is the sum of the seropositive compartments over $N$.

$$S(t + \delta) = S(t) + \left[\frac{\text{calves}(t)}{2}\right] - [S(t)\lambda] - [S(t)\mu_j]$$

$$I(t + \delta) = I(t) + [S(t)\lambda(1 - \rho)] - [I(t)\sigma] - [I(t)\mu_j]$$
\[ R_1(t + \delta) = R_1(t) + [S(t)\lambda \rho] + [I(t)\sigma] - [R_1(t)\gamma] - [R_1(t)\mu j] \]

\[ R_2(t + \delta) = R_2(t) + [R_1(t)\gamma] - [R_2(t)\mu j] \]

\[ N(t) = S(t) + I(t) + R_1(t) + R_2(t) \]

true seroprevalence(t) = \([I(t) + R_1(t)]/N(t)\]

In test-and-slaughter models (Muddy Creek and Scab Creek) removals are a function of the estimated year and feedground-specific capture rates \(\nu(t)\). With test-and-slaughter, \(I(t + \delta)\) includes the additional term \(-[I(t)\nu(t)]\) and \(R_1(t + \delta)\) includes \(-[R_1(t)\nu(t)]\).

**Section C. Some evidence against alternative SIR and SIRS models from a preliminary analysis.**

![Figure B-1](image_url). Fitted seroprevalence and population count trends for Greys River using an endogenous, three-compartment SIR process. 90% prediction intervals of 2000 simulations with parameters fixed at the maximum likelihood estimate shaded in blue. The model does a poor job of describing the data, as evident by the left panel and AIC = 171.
Figure B-2. Fitted seroprevalence and population count trends for Greys River using an endogenous process model in which antibodies are lost over time and individuals lose immunity (SIRS). The model does a poorer job of describing the data unless parameter constraints are relaxed and allowed to take any (unrealistic) value. The best fit with parameter constraints (shown) received less support by AIC than the SIRR immunity model (157, dAIC = 5). 90% prediction intervals of 2000 simulations with parameters fixed at the maximum likelihood estimate shaded in blue.

Section D. Search settings and parameter constraints.

We used 20,000 particles and 800 iterations of the fitting procedure. Cooling type was set to geometric and the cooling fraction per 50 iterations was set to 0.25. The perturbation step size (random walk standard deviation size) was set to 0.02 for all parameters except $\gamma$ and $\beta$, which were set to zero (not perturbed in the random walk).

For models at Greys River and Dell Creek 100 starting value vectors were generated as follows:

Transmission parameter terms, $\beta$ and/or $\beta'$ $\sim$ Uniform(0,10). In Raithel et al. (2007) average annual survival for female Rocky Mountain elk across the West ranged across yearling and older age classes 0.724-0.873. Corresponding annual mortality probabilities equal 0.127-0.276, which we broadly encompassed: $\mu_j$ $\sim$ Uniform(0.11,0.33). Recovery probability, $\sigma$ $\sim$ Uniform(0,1). We constrained two
parameters for which we had strong prior information and which are physiological and thus expected to be invariant across space and time. The proportion of individuals transitioning straight from \( S \) to \( R_1 \) (never infectious, \( \rho \)) was constrained between 0.49-0.51/year following Thorne et al.’s (1978) finding that only approximately 50% of exposed female elk ever abort. The frequency/density-dependence scaling parameter encompassed the full possible range, \( \theta \sim \text{Uniform}(0,1) \). The imported infections rate was presumed to be less than 5 per year, \( \iota \sim \text{Uniform}(0,5) \).

Using fluorescent polarization assay scores compared against overall NVSL brucellosis testing protocols and time since infection, we estimated the seroreversion probability (\( \gamma \)) from \( R_1 \) to \( R_2 \) to be between 0.08-0.10/year and constrained it thus (Fig. B-3; and see Benavides et al., 2017).
Figure B-3. Left, density distribution of fluorescent polarization assay (FP) scores for seronegative (blue) and seropositive (red) serological test results for female elk based on NVSL protocols, with vertical dashed blue line indicating our chosen lower cutoff of 40 millipolarization units (mpu) and a red dashed line indicating our chosen upper cutoff of 78 mpu. Right, FP scores as a function of the number of years since infection based on 227 longitudinal records, with a solid black line representing fitted linear model predictions, with an intercept of 218 mpu and slope of -15/yr, p-value=3.5e-9. Dashed blue and red lines indicating the cutoffs used to define the parameter space for our seroreversion parameter $\gamma$.

100 unique initial starting conditions were also generated for the 4 compartments (S, I, $R_1$, and $R_2$). These were expected to vary by feedground, and since their values sum to one, achieving uniformity is a minor challenge. We generally assumed S and $R_1$ would be the most populous compartments, followed by I and $R_2$. The procedure for generating these values is available along with model code as electronic supplement.

Muddy Creek had fewer years of data, nevertheless, the parameter space explored differed only for $\sigma$, whose starting values $\sim$ Uniform(0.9,1) based on MLEs from Greys River and Dell Creek. Further, these are not ‘hard constraints’. During iterative
filtering parameters are free to explore parameter space outside of the initial values ranges if it helps to maximize the likelihood.

Section E. Models and parameter estimates.

Table B-3. Full time-series models, with baseline and environmentally-driven transmission parameters. Transformed, cumulative March to June snowmelt water equivalent (SWE) values comprise the environmental covariate used ($\psi$). Low-density feeding treatment models at Greys River had time-varying transmission parameters $\beta_k$, $\beta'_k$ and $\theta_k$ for $k = 1 - 2$ where $k = 1$ prior to 2009. The total number of model parameters is stated, as well as the years available for modeling. *Due to insufficient data, Scab Creek was not used as a site for inference, but rather, plausible parameter estimates based on the other sites were plugged in to see whether these could also explain the disease trends at this site.
Table B-4. Models run on the subset of years for which NDVI data was available for inclusion as the environmental covariate $\psi$.

<table>
<thead>
<tr>
<th>Feedground</th>
<th>Model</th>
<th>$\lambda$</th>
<th>No. Params.</th>
<th>Timespan</th>
</tr>
</thead>
<tbody>
<tr>
<td>Greys River</td>
<td>endogenous</td>
<td>$\frac{\beta[I(t)+\epsilon]}{N(t)^0}$</td>
<td>8</td>
<td>2000 - 2017</td>
</tr>
<tr>
<td></td>
<td>exogenous</td>
<td>$\frac{\beta'\psi[I(t)+\epsilon]}{N(t)^0}$</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td></td>
<td>combined</td>
<td>$\frac{[\beta+(\beta'\psi)][I(t)+\epsilon]}{N(t)^0}$</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>Dell Creek</td>
<td>endogenous</td>
<td>$\frac{\beta[I(t)+\epsilon]}{N(t)^0}$</td>
<td>8</td>
<td>2000 - 2017</td>
</tr>
<tr>
<td></td>
<td>exogenous</td>
<td>$\frac{\beta'\psi[I(t)+\epsilon]}{N(t)^0}$</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td></td>
<td>combined</td>
<td>$\frac{[\beta+(\beta'\psi)][I(t)+\epsilon]}{N(t)^0}$</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>Muddy Creek</td>
<td>endogenous</td>
<td>$\frac{\beta[I(t)+\epsilon]}{N(t)^0}$</td>
<td>8</td>
<td>2004 - 2017</td>
</tr>
<tr>
<td></td>
<td>exogenous</td>
<td>$\frac{\beta'\psi[I(t)+\epsilon]}{N(t)^0}$</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td></td>
<td>combined</td>
<td>$\frac{[\beta+(\beta'\psi)][I(t)+\epsilon]}{N(t)^0}$</td>
<td>9</td>
<td></td>
</tr>
</tbody>
</table>
Table B-5. Model results using full time series and snowmelt water equivalent data as the winter-severity covariate. At Greys River the endogenous model with a constant transmission parameter term, \( \beta \), received greater support than alternative models by AIC. At Dell Creek and Muddy Creek, all three models were \(<2 \delta \text{AIC} \text{ different}. \beta \) is a base transmission potential term, while \( \beta' \) is a climate-forced/exogenous transmission term. Low-density feeding treatment models at Greys River took all combinations of time-varying \( \beta \) and \( \beta' \), where \( \beta_1 \) is the transmission term corresponding to the pre-treatment period \(<2009\) and \( \beta_2 \) corresponds to post-treatment \( \geq 2009\). The same holds for \( \beta' \). \( \mu \) represents annual mortality, where subscripted numbers denote time-variance. \( \sigma \) represents annual recovery I to R1; \( \rho \) is the proportion which seroconvert but never abort; \( \gamma \) is annual seroreversion probability (antibody loss); \( \theta \) is a dependency- vs. frequency-dependent scaling parameter. \( \theta_1 \) and \( \theta_2 \) accommodate time-period variant transmission terms \( \beta_k \) and \( \beta'_k \) in ‘low-density’ feeding treatment models; \( \iota \) is the average number of imported infections per year.
<table>
<thead>
<tr>
<th>Feedground</th>
<th>Model</th>
<th>$\beta$</th>
<th>$\beta_1$</th>
<th>$\beta_2$</th>
<th>$\beta_3$</th>
<th>$\mu_1$</th>
<th>$\mu_2$</th>
<th>$\mu_3$</th>
<th>$\sigma$</th>
<th>$\rho$</th>
<th>$\gamma$</th>
<th>$\theta_1$</th>
<th>$\theta_2$</th>
<th>$\lambda$</th>
<th>loglik</th>
<th>AIC</th>
<th>$\delta$AIC</th>
<th>Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Greys River</td>
<td>endog.</td>
<td>0.02</td>
<td>0.21</td>
<td>0.16</td>
<td>0.95</td>
<td>0.51</td>
<td>0.10</td>
<td>0.18</td>
<td>0.11</td>
<td>0.85</td>
<td>0.73</td>
<td>0.11</td>
<td>0.85</td>
<td>0.73</td>
<td>-64.22</td>
<td>152.44</td>
<td>0</td>
<td>0.75</td>
</tr>
<tr>
<td>Greys River</td>
<td>endog., LD treat.</td>
<td>0.01</td>
<td>0.88</td>
<td>0.21</td>
<td>0.16</td>
<td>0.95</td>
<td>0.49</td>
<td>0.11</td>
<td>0.11</td>
<td>0.85</td>
<td>0.73</td>
<td>0.11</td>
<td>0.85</td>
<td>0.73</td>
<td>-63.94</td>
<td>158.88</td>
<td>3.44</td>
<td>0.13</td>
</tr>
<tr>
<td>Greys River</td>
<td>comb.</td>
<td>0.23</td>
<td>0.04</td>
<td>0.2</td>
<td>0.17</td>
<td>0.93</td>
<td>0.49</td>
<td>0.12</td>
<td>0.63</td>
<td>0.35</td>
<td>0.40</td>
<td>0.17</td>
<td>0.49</td>
<td>0.12</td>
<td>-65.40</td>
<td>156.79</td>
<td>4.35</td>
<td>0.08</td>
</tr>
<tr>
<td>Greys River</td>
<td>comb., LD treat. B</td>
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<td>0.06</td>
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<td>0.16</td>
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<td>0.50</td>
<td>0.12</td>
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<td>0.50</td>
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<td>0.51</td>
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<td>0.51</td>
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<td>0.94</td>
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<td>0.10</td>
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<td>0.33</td>
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</tbody>
</table>
Table B-6. Model results using timespans for which NDVI MODIS data were available and ‘spring start’ is the environmental covariate. At Greys River and Muddy Creek the endogenous model with a constant transmission parameter received the majority of the Akaike weight. At Dell Creek, the results were reversed: the exogenous model received the most support, followed by the combined and endogenous models. A description of the parameters can be found above in the caption for Table B-5.

<table>
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<tr>
<th>site</th>
<th>model</th>
<th>$\beta$</th>
<th>$\beta'$</th>
<th>$\mu$</th>
<th>$\mu_1$</th>
<th>$\sigma$</th>
<th>$\rho$</th>
<th>$\gamma$</th>
<th>$\theta$</th>
<th>$\iota$</th>
<th>loglik</th>
<th>AIC</th>
<th>$\delta$AIC</th>
<th>Weight</th>
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<td>0.61</td>
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<td>1.09</td>
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<td>0.08</td>
<td>0.34</td>
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<tr>
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<td>0.19</td>
<td>0.77</td>
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<td></td>
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<td>0.96</td>
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<td>0.50</td>
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<td>0.91</td>
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</table>
Section F. Characterizing parameter uncertainty

We followed the procedures of King et al. (2015) to sample parameter space in proportion to the likelihood for each of the four feedground herds using the endogenous model. Here we illustrate with Greys River. we first set up a collection of parameter vectors in a neighborhood of the maximum likelihood estimate containing the region of high likelihood by using a likelihood ratio test at the 99% confidence level.

For all parameters except $\beta$ and $\theta$ we generated a Latin hypercube design based on the Sobol low-discrepancy sequence using sobolDesign in R package pomp. $\beta$ and $\theta$ are log-linearly related. For these two variables we sampled corresponding value pairs from the predicted log-linear model fit.

We then carried out a particle filter at each parameter vector, which gave us estimates of both the likelihood and the filter distribution at that parameter value. We then simulated forward from each filter distribution to give the prediction distribution for each parameter vector. We then sampled from the prediction distributions with probability proportional to the estimated likelihood of the parameter vector.
Table B-7. Ranges of parameter values in the neighborhood of the maximum likelihood estimate for Greys River. \( \rho \) (rho) and \( \gamma \) (gamma) were essentially fixed at 0.11 and 0.50, respectively.

<table>
<thead>
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<th>Parameter</th>
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</thead>
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<tr>
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<tr>
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<tr>
<td>mu2</td>
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<tr>
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</table>

Figure B-4. \( \beta \) and \( \theta \) are log-linearly related. Points represent model estimates in the neighborhood of the MLE from the endogenous model for Greys River. The black line is the fitted linear model predictions, with 95% confidence intervals shown. We sampled evenly for values of \( \beta \) and \( \theta \) along the predicted fit.
Figure B-5. Pairs plots illustrating the values for each of the 20 parameter vectors from the sampling procedure for Greys River.
Figure B-6. Time series simulations for endogenous models at four feedgrounds. Left, disease trends, and right, female elk count trends. Red lines denote the observed data; black error bars represent the 90% binomial confidence interval for seroprevalence; blue shading indicates the 90% intervals of the prediction distributions sampled with probability proportional to the estimated likelihood of the parameter vectors. Disease trends yield a noticeably worse fit compared with predictions from the MLE for Greys River and Dell Creek. At Muddy Creek the disease trend appears to have similar coverage of the data as predictions using the MLE, but at the cost of a worse fit to the count data.
Figure B-7. The predicted number of infectives from the endogenous model for Muddy Creek with parameter and demographic uncertainty under seropositive test-and-slaughter (red), no action (grey), seronegative test-and-slaughter (blue), and culling without regard to serostatus (orange). Colored dashed lines show median estimates. Left, shading represents 90% prediction intervals using equal tail quantiles from 2000 stochastic simulations. Right, shading represents 50% prediction intervals. Removals occurred between 2006 and 2010 (inclusive), denoted by vertical dashed lines. These results likely overestimate the uncertainty, yet the key result remains compelling: test-and-slaughter of seropositive female elk is a poor strategy for reducing brucellosis transmission among elk. Using median estimates compared to seropositive test-and-slaughter: seronegative test-and-slaughter (blue) would have produced 118 fewer infectives; culling, 71 fewer; no action, 68 fewer.

Section G. Additional figures.

Figure B-8. Left, cumulative March to June snowmelt water equivalent values from 3 SNOTEL stations used for analysis. Color denotes location. Right, the rolling 8-year average for corresponding sites as per Cross et al. (2007). SWE trends were strongly correlated across the region. Spearman’s rho for cumulative March-June SWE ranged 0.8-0.9 and Pearson’s correlation coefficient ranged 0.71-0.91.
References


Pinedale Elk Herd Unit of Wyoming; Results of a 5 year pilot project (p. 20). Retrieved from Wyoming Game and Fish Department website:

https://wgfd.wyo.gov/WGFD/media/content/PDF/Wildlife/TR_REPORT_2010_FINAL.pdf

CURRICULUM VITAE

Gavin Cotterill
(November 2019)

EDUCATION

Ph.D. in Ecology, G.P.A. 3.9  
August 2015 – December 2019
Utah State University, Logan, Utah, USA; Dept. of Wildland Resources and Ecology Center.
Advisors: Dr. Johan du Toit and Dr. Paul Cross
- Awarded S.J. and Jessie E. Quinney Doctoral Research Fellowship
- 2020 Quinney College of Natural Resources Doctoral Researcher of the Year
- My dissertation research focused on the disease ecology of brucellosis in elk, the role of supplementary winter feeding in perpetuating and managing the disease, estimating the impacts to vital rates of the disease, and using stochastic mechanistic models to infer the latent disease processes from observed seroprevalence time series data within the Greater Yellowstone Ecosystem.
- Skills: programming and statistical analyses (e.g. Bayesian hierarchical models, partially-observed Markov process models, mark-recapture models, matrix population models), field research and elk handling, professional presentations, grant proposal and publication writing.
- Software: ArcGIS, BASH, Canvas, JAGS, Microsoft Access, R, RMark, STAN, SLURM.

August 2007 – May 2010
University of Vermont, Burlington, Vermont, USA; Rubenstein School of Environment and Natural Resources.

PUBLICATIONS


brucellosis reduces mid-winter pregnancy in elk. Ecology and Evolution, 8(22), 10733-10742, DOI: 10.1002/ece3.4521, IF = 2.5.


**PROFESSIONAL EXPERIENCE**

**Teaching Assistant**

Spring 2016
“Genetics in Conservation and Management”
Helped students individually and in groups with test and homework preparation, graded assignments and tests, facilitated classes.

**Avian Technician**

April 2015 - August 2015
*Center for Natural Lands Management*
Assisted on various projects alongside the Dept. of Defense on Joint Base Lewis-McChord. Duties included mist-netting and banding Western Bluebirds, nest monitoring, point counts and line transects, Oregon Vesper Sparrow and Streaked Horned Lark surveys.

**Dog Handler, Trainer, Kennel Manager**

October 2012 – April 2015
*Conservation Canines, Center for Conservation Biology at the University of Washington*
Projects included caribou, wolf, and moose scat surveys in Alberta, CA, Washington ground squirrel surveys in Oregon for The Nature Conservancy, forest carnivore scat surveys in Sarikamis, Turkey and wind farm mortality surveys in Illinois.

**Avian Technician**

Spring/Summer 2012 and 2013
*Institute for Bird Populations and the National Park Service*
Conducted backcountry breeding bird point count surveys in 7 park units in Washington and Oregon.

**Cougar Diet Study Field Technician**

August 2012 – October 2012
Utah State University
Backcountry surveys of GPS clusters for prey remains, collar recovery using radio telemetry, habitat surveys.

Land Stewardship Intern (LANDS)  
June 2009 – August 2009
University of Vermont in Partnership with the Student Conservation Association
Creation of resource inventory reports for client partners including The Nature Conservancy, Green Mountain National Forest, the City of Charlotte, and the City of South Burlington.

PRESENTATIONS

Brucellosis Research Group Meeting, Idaho Falls, ID, August 2019
“Modeling seroprevalence trends in elk at WY feedgrounds”

Brucellosis Research Group Meeting, Cody, WY, September 2018
“Partially-observed Markov process models of feedground seroprevalence”

Ecological Society of America Annual Conference, New Orleans, LA, August 2018
“Hidden cost of disease in a free-ranging ungulate: brucellosis reduces mid-winter pregnancy in elk”

Learn to Hunt Grant Proposal Outreach, Salt Lake City, UT, May 2018
“Learning to hunt as an adult”

WILD Research Symposium, Logan, UT, April 2018
“Hidden costs of brucellosis in free-ranging elk: reduced pregnancy in mid-winter, not just reproductive failures in spring”

Brucellosis Research Group Meeting, Bozeman, MT, September 2017
“Preliminary results from Wyoming Feedgrounds PSPB testing”

Brucellosis Research Group Meeting, Idaho Falls, ID, September 2016
“Relating elk population ecology to brucellosis serology using feedground data and a new field diagnostic approach”

WILD Research Symposium, Logan, UT, April 2016
“Disease ecology and adaptive management of brucellosis in Greater Yellowstone elk”

Field Demonstation and Forum, Eatonville, WA, May 2014 and October 2014
“Using Conservation Canines for wildlife scat detection”
SERVICE

University Contributions

- Graduate Student Liaison to the Wildland Resources Department Head Search Committee. Attended candidate interviews and events. Solicited and coordinated graduate student feedback and presented summary to the search committee. Fall 2019.
- Facilitator of Animal Ecology Working Group. Weekly discussion group composed of graduate students within the WILD department. Spring 2018
- Graduate Student Liaison to the Departmental Seminar. Helped select, invite, and host guest speakers. Spring 2018

Press Coverage


Reviewed Manuscripts for:

- Ecosphere

Membership in Professional Organizations:

- Ecological Society of America
- The Wildlife Society