DISSERTATION

EFFECT OF ENHANCED NUTRITION DURING WINTER ON THE UNCOMPAHGRE PLATEAU MULE DEER POPULATION

Submitted by

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WE HEREBY RECOMMEND THAT THE DISSERTATION PREPARED UNDER OUR SUPERVISION BY CHAD JEFFREY BISHOP ENTITLED EFFECT OF ENHANCED NUTRITION DURING WINTER ON THE UNCOMPAHGRE PLATEAU MULE DEER POPULATION BE ACCEPTED AS FULFILLING IN PART REQUIREMENTS FOR THE DEGREE OF DOCTOR OF PHILOSOPHY

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ABSTRACT OF DISSERTATION

EFFECT OF ENHANCED NUTRITION DURING WINTER ON THE UNCOMPAHGRE PLATEAU MULE DEER POPULATION

Mule deer (*Odocoileus hemionus*) populations declined across much of the West during the 1990s, prompting state wildlife agencies to explore mule deer limiting factors. The greatest concern of agencies and sportsmen was whether declining habitat quality, predation, or both were responsible for the observed declines. In Colorado, the Uncompahgre Plateau mule deer population received the most attention because of a steep population decline from the 1980s through the late 1990s. Biologists hypothesized that poor quality of the pinyon (*Pinus edulis*) and juniper (*Juniperus osteosperma*) winter range was the primary cause of the observed decline. In contrast, many of the Colorado Division of Wildlife's (CDOW) constituents hypothesized that high predation rates were keeping the mule deer herd below nutritional carrying capacity. These hypotheses represented very different paradigms of population limitation. Perhaps more importantly, the competing views suggested that CDOW should pursue one of two very different management strategies: 1) implement habitat improvements in the pinyon-juniper winter range, or 2) implement efforts to reduce predator populations, particularly coyote (*Canis latrans*) populations. Information was needed to guide the decision process. I therefore evaluated the effect of enhanced nutrition during winter on the Uncompahgre deer population as a way to evaluate the importance of habitat quality versus that of predation.

I conducted a field study incorporating a crossover experimental design to quantify the effect of enhanced nutrition on fetal, neonatal, overwinter fawn, and annual

adult doe survival rates. I captured and radio-collared samples of deer in 2 experimental units (EUs) on winter range. I delivered the nutrition treatment to deer occupying one EU (treatment) and did not administer the treatment to deer in the other EU (control). Established field techniques were not sufficient to allow me to quantify the effect of the treatment on fetal and neonatal survival. I therefore pursued an exploration of vaginal implant transmitters as a mechanism to capture necessary samples of newborn fawns on summer range exclusively from radio-collared does that occupied the winter range EUs (Chapter 1). This effort allowed me to estimate fetal and neonatal survival as a function of the treatment. In broad terms, I demonstrated that direct estimates of fetal and neonatal survival may be obtained from previously marked female mule deer in free-ranging populations, thus expanding opportunities for conducting field experiments.

I encountered additional challenges with estimation of fetal and neonatal survival. First, I was unable to determine the fate of all fetuses that I documented in utero. I therefore developed a likelihood function for estimating fetal survival when the fates of some fetuses are unknown (Chapter 2). Second, a majority of my fetal and neonatal samples were comprised of siblings, indicating my data were potentially overdispersed. Overdispersion causes sample variances to be underestimated and requires a variance inflation factor, c. To estimate c, I compared theoretical variance estimates with empirical variance estimates obtained from bootstrap analyses of the data (Chapter 2). I found little evidence of overdispersion in my fetal survival data, and I found modest overdispersion in my neonatal sample data ($\hat{c} = 1.25$). Although some overdispersion was detected, my results indicated that fates of sibling mule deer neonates may often be

independent even though they have the same dam and use the environment similarly. I discuss reasons for this in Chapter 2.

After resolving issues with fetal and neonatal survival estimation, I quantified the effect of the nutrition enhancement treatment on fetal, neonatal, overwinter fawn, and annual adult doe survival (Chapter 3). I then used these parameter estimates, along with estimated fecundity rates, in an age-structured, deterministic population model to estimate the effect of the treatment on the population rate of change, λ. The treatment caused $\hat{\lambda}$ to increase by an average of 0.133 (SD = 0.0168) during the 3 years of my study. I documented density dependence in the Uncompahgre deer population because survival of fawns and does increased considerably in response to enhanced nutrition. I found strong evidence that coyote predation of ≥6-month-old fawns and adult does was compensatory. Finally, I found that winter range habitat quality was a limiting factor of the Uncompahgre Plateau deer population.

I completed my principal study objectives in the first 3 chapters of the dissertation. However, my research afforded the opportunity to evaluate the utility of serum thyroid hormones in mule deer as an index to body condition (Chapter 4). Concentrations of total thyroxine (T4) and free T4 (FT4) were substantially higher in treatment deer than control deer. I also found that serum thyroid hormones were highly correlated with estimated body fat in mule deer during late winter. Concentrations of T4 and FT4 could be useful for evaluating relative condition of different deer groups or populations, and for roughly estimating body fat of individual animals during late winter.

In summary, I demonstrated that winter range habitat quality was ultimately limiting the Uncompahgre mule deer population. Observed predation was primarily

compensatory, particularly of ≥6-month-old fawns and adult does. My findings indicate that CDOW should evaluate habitat treatments in late-seral pinyon-juniper habitat as a means to increase habitat productivity for mule deer.

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Chad J. Bishop

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CHAPTER 1

USING VAGINAL IMPLANT TRANSMITTERS TO AID IN CAPTURE OF MULE DEER NEONATES

Abstract: Estimating survival of the offspring of marked female ungulates has proven difficult in free-ranging populations yet could improve the present understanding of factors that limit populations. I evaluated the feasibility and efficiency of capturing large samples (i.e., >80/year) of neonate mule deer (*Odocoileus hemionus*) exclusively from free-ranging, marked adult does using vaginal implant transmitters (VITs, $n = 154$) and repeated locations of radio-collared does without VITs. I also evaluated the effectiveness of VITs, when used in conjunction with in utero fetal counts, for obtaining direct estimates of fetal survival. During 2003 and 2004, after I placed VIT batteries on a 12-hour duty cycle to lower electronic failure rates, the proportion that shed ≤3 days prepartum or during parturition was 0.623 (SE = 0.0456), and the proportion of VITs shed only during parturition was 0.447 (SE = 0.0468). My neonate capture success rate was 0.880 (SE = 0.0359) from does with VITs shed ≤3 days prepartum or during parturition and 0.307 (SE = 0.0235) from radio-collared does without VITs or whose implants failed to function properly. Using a combination of techniques, I captured 275 neonates and found 21 stillborns during 2002–2004. I accounted for all fetuses at birth (i.e., live or stillborn) from 78 of the 147 does (0.531, SE = 0.0413) having winter fetal counts, and this rate was heavily dependent on VIT retention success. Deer that shed VITs prepartum were larger than deer that retained VITs to parturition, indicating a need

to develop variable-sized VITs that may be fitted individually to deer in the field. I demonstrated that direct estimates of fetal and neonatal survival may be obtained from previously marked female mule deer in free-ranging populations, thus expanding opportunities for conducting field experiments. Survival estimates using VITs lacked bias that is typically associated with other neonate capture techniques. However, current vaginal implant failure rates, and overall expense, limit broad applicability of the technique.

Key Words: birth site, Colorado, fawn, fetus, mule deer, neonate, *Odocoileus hemionus*, survival, vaginal implant transmitter, VIT.

1.1 INTRODUCTION

Measuring fetal and neonatal deer survival as a function of dam characteristics (e.g., body condition, disease status) is necessary to understand components of reproductive ecology, population productivity, and disease transmission. Conducting such research in enclosure facilities is typically necessary to achieve experimental rigor, although important insights may be obtained by conducting similar research in free-ranging populations. For example, experimental studies that relate nutrition of female ungulates to reproductive success have been restricted largely to enclosures (Verme 1965, 1969; Robinette et al. 1973; Thorne et al. 1976; Cook et al. 2004). Applying similar experimental treatments to adult females in free-ranging populations, while quantifying neonatal production and survival, may provide an understanding of nutritional effects on these parameters relative to other potential limiting factors (e.g., predation, disease).

Feasibility of such studies depends on whether necessary numbers of neonates can be captured exclusively from radio-marked females with known treatment status, at least whenever treatments are applied to individuals or groups rather than whole populations. Capturing neonates from previously marked females enables the relation of doe-specific data to reproductive success, which has broad applicability for field studies if sample size objectives can be met.

Huegel et al. (1985) captured white-tailed deer (*O. virginianus*) neonates from radio-collared females in south-central Iowa by repeatedly locating each doe during the fawning period and searching for fawns when successive locations indicated a reduction in daily movements. Carstensen et al. (2003) employed similar methods using a fixed-wing aircraft to obtain successive doe locations but concluded the technique was inefficient and not viable for capturing large samples of white-tailed deer fawns in north-central Minnesota. A fixed-wing location of radio-collared does combined with aerial fawn searches is a possibility in relatively open habitats but not in closed-canopy habitats (Hamlin et al. 1984). M. A. Hurley (Idaho Department of Fish and Game, personal communication) and T. M. Pojar (Colorado Division of Wildlife, personal communication) attempted to locate radio-collared mule deer does from the ground and conduct searches when doe behavior or appearance indicated fawn(s) may be present, but such attempts were inefficient and not considered useful for capturing large samples of neonates.

The most promising technique employed to capture neonates from marked does is use of vaginal implant transmitters (VITs). Initial applications of VITs relied on vulvar sutures for transmitter retention, which were largely ineffective and raised animal welfare

concerns (Garrott and Bartmann 1984, Giessman and Dalton 1984, Nelson 1984). More recently, modified VITs were used in white-tailed deer with better success (Bowman and Jacobson 1998, Carstensen et al. 2003). The modified VIT has flexible, silicone wings that induce pressure against the vaginal wall to retain the transmitter, thus eliminating sutures and facilitating a quick, non-surgical insertion process. Other studies using newly-designed VITs were recently conducted on Columbian black-tailed deer (*O. hemionus columbianus*; Pamplin 2003), mule deer (Johnstone-Yellin et al. 2006), and elk (*Cervus elaphus*; Vore and Schmidt 2001, Johnson et al. 2006). These studies did not document any detrimental effects to the adult females, fetuses, or neonates by use of VITs. In a study focused on animal welfare, Johnson et al. (2006) found that VITs in elk caused minimal tissue irritation and did not impact reproductive performance. Recent data indicate VITs are potentially a viable technique for locating and capturing neonates from radio-marked adult female deer shortly after parturition (Carstensen et al. 2003, Pamplin 2003, Johnstone-Yellin et al. 2006).

Vaginal implant transmitters could also permit measurement of fetal survival in free-ranging populations. Fetal survival estimates are needed in populations where stillborn mortality is known to occur but is poorly understood or quantified (Ricca et al. 2002, Pojar and Bowden 2004). Survival could be estimated by counting the number of fetuses in utero during winter and using VITs to document the fate of each fetus at parturition. However, each of a doe's documented in utero fetuses would need to be accounted for at birth to represent a valid data point. Precision of the survival estimate would therefore depend on the proportion of birth sites located where the number of fawns observed equals the number of known fetuses.

An additional advantage of using VITs to capture neonates may be a reduction in sample bias when compared to capture techniques that rely on opportunistic fawn capture (White et al. 1972, Ballard et al. 1998, Pojar and Bowden 2004). These techniques are susceptible to bias because of unequal capture success among vegetation types, road densities, fawn ages, and stages of fawning. When using VITs, neonate captures should be more random as long as VIT signals are monitored with equal intensity during fawning, and assuming the sample of radio-collared does was captured with minimal bias. Thus, VITs could have more broad applicability regardless of whether study objectives require that fawns be captured from previously marked does.

My principal objective was to evaluate the feasibility of capturing large samples of newborn fawns exclusively from radio-collared adult does in a free-ranging, migratory mule deer population using a combination of 2 approaches: 1) VITs placed in adult doe mule deer during winter as a mechanism for determining the timing and location of birth sites the following June, and 2) repeated ground relocations of radio-collared does without VITs during the fawning period. My secondary objectives were to evaluate the effectiveness of VITs for estimating fetal survival when used in conjunction with fetus counts, and to provide an evaluation of VITs as a neonate capture technique for migratory mule deer in the Intermountain West.

1.2 STUDY AREA

I conducted my research in southwest Colorado on the southern half of the Uncompahgre Plateau and in the adjacent San Juan Mountains (Fig. 1). I restricted the winter range study area to 2 sites, or experimental units (EUs), to meet research objectives fully detailed in Chapter 3. For clarity, I define the core of each EU as the area

containing 90% of the radio-collared deer captured in that unit. The core of the Colona EU (38°21'N, 107°49'W) covered 12 km^2 and the core of the Shavano EU (38°27'N, 108°01'W) covered 22 km^2. Each EU encompassed approximately 40 km^2 when considering all radio-collared deer, ranging in elevation from 1,830 m to 2,290 m. Winter range EUs were comprised of pinyon (*Pinus edulis*) and Utah juniper (*Juniperus osteosperma*) woodlands with interspersed big sagebrush (*Artemisia tridentata*) adjacent to irrigated agricultural fields. During my study, annual precipitation averaged 22.3 cm and the minimum temperature in January averaged –8.2° C in Montrose, Colorado (Western Regional Climate Center [WRCC] 2005), which is 60 m below the lowest winter range elevation in either EU. Deer occupied the winter range EUs from November through April each year. Estimated deer densities typically varied between 31 deer/km^2 and 59 deer/km^2 in the core of each EU during the study, with densities periodically reaching 85 deer/km^2 in portions of an EU (C. J. Bishop, Colorado Division of Wildlife, unpublished data).

I defined summer range based on migratory movements of radio-collared deer captured in the winter range EUs. Summer range for 95% of the radio-collared deer covered 2,500 km^2, whereas the total summer range encompassed approximately 4,000 km^2 between 37°49' and 38°28'N latitude and 107°26'and 108°17'W longitude (Fig. 1). Elevations ranged from 1,830 m to 3,500 m, with a majority of deer summering between 2,600 m and 3,000 m. Dominant summer range habitat types, from lower to higher elevations, were pinyon–juniper, Gambel oak (*Quercus gambelii*), ponderosa pine (*Pinus ponderosa*), big sagebrush, aspen (*Populus tremuloides*), and mixed forests of Engelmann spruce (*Picea engelmannii*) and subalpine fir (*Abies lasiocarpa*). Diverse

habitat mosaics occurred at interfaces of each of the major habitat types. Snowberry (*Symphoricarpos* spp.) was a common understory shrub in Gambel oak, ponderosa pine, and aspen habitats, and it occasionally occurred in sagebrush habitats. Annual precipitation averaged 57.4 cm and the maximum temperature in July averaged 26.7° C at a weather station in the summer range situated at 2,438 m elevation (WRCC 2005).

1.3 METHODS

1.3.1 Sample Size

I placed VITs in 154 pregnant, adult mule deer during 26 February–2 March, 2002–2004. During 2002, I placed VITs in 18 adult females in each EU as a pilot study to evaluate effectiveness of VITs relative to equipment functionality and logistical feasibility. I based sample size calculations on a success-failure analysis of VIT retention to parturition and the proportion of VITs resulting in a fawn capture. During 2003 and 2004, I attempted to insert VITs in 30 adult females in each EU each year. I based sample sizes on precision of resulting neonate survival estimates necessary to meet other research objectives (Chapter 3). I assumed 30 VITs would facilitate the capture of ≥40 fawns, when combined with opportunistic fawn captures from other radio-collared does during the fawning period.

I captured and radio-collared an additional 139 adult does that did not receive VITs during 20 November–14 December, 2000–2003 (Chapter 3). I permanently attached radio collars on all captured adult does; thus, many of the does were present in multiple years' samples. Most adult does receiving a VIT in a given year did not receive a VIT the following year, but I retained the does in the radio-collared sample if they survived to the next year. My total samples of radio-collared does with VITs were 36,

58, and 60 during 2002, 2003, and 2004, respectively. My total pre-fawning samples of radio-collared does without VITs were 85, 114, and 145 during 2002, 2003, and 2004, respectively.

1.3.2 Capture and Handling

I captured adult does during November and December primarily using baited drop nets (Ramsey 1968, Schmidt et al. 1978) and secondarily using helicopter net-gunning (Barrett et al. 1982, van Reenan 1982). I used helicopter net-gunning during late February and early March to capture all deer receiving VITs. All deer were hobbled and blind-folded prior to handling.

Adult does receiving VITs were ferried ≤3.5 km by the helicopter to a central processing location. During February–March 2002, I chemically immobilized 12 deer in each EU immediately following net-gun capture using a combination of 5:1 ketamine (5–7 mg/kg) and xylazine (1–3 mg/kg) given intravenous to facilitate ultrasonography and insertion of VITs. Immediately prior to release, I reversed xylazine with an intravenous injection of yohimbine at a rate of approximately 12 mg/45 kg animal body mass. I physically restrained all other captured deer in each experimental unit during 2002, thereby evaluating the need for chemical restraint to perform the necessary handling procedures. I found that physical restraint alone worked well and therefore did not chemically immobilize deer during 2003 and 2004.

Following capture, I fitted adult does with vinyl-belted radio collars equipped with mortality sensors (Lotek, Inc., Newmarket, Ont., Canada; Advanced Telemetry Systems, Inc., Isanti, Minn.), which activated after remaining motionless for 4 hours. I stitched neck band material (Ritchey Mfg. Co., Brighton, Colo.) to the left side of each

radio collar, which I engraved with a unique, 2-symbol marking for visually identifying deer. I measured mass, hind foot length, and chest girth of each deer and estimated deer age using tooth replacement and wear (Severinghaus 1949, Robinette et al. 1957, Hamlin et al. 2000). During captures in February–March, I performed transabdominal ultrasonography using an Aloka 210 (Aloka, Inc., Wallinford, Conn.) portable ultrasound unit with a 3-MHz linear transducer to establish pregnancy status and measure fetal rates (Stephenson et al. 1995). I shaved the left caudal abdomen from the last rib and applied lubricant to facilitate transabdominal scanning. I fitted each pregnant deer with a VIT and released non-pregnant does without a radio-collar or VIT. The ultrasound and VIT insertion procedures were performed in a 4.3 × 4.9-m wall-frame tent to minimize disturbance from helicopter rotor wash and adverse weather conditions and to create a dim environment to facilitate ultrasonography.

1.3.3 Vaginal Implant Transmitters

I used VITs (M3930, Advanced Telemetry Systems, Inc., Isanti, Minn.), which have been described in detail elsewhere (Bowman and Jacobson 1998, Carstensen et al. 2003, Johnstone-Yellin et al. 2006). However, I made several noteworthy alterations. Antennas were pre-cut by the manufacturer to 6 cm in length with antenna tips encapsulated in a resin bead to eliminate sharp edges. During 2003 and 2004, I placed VITs on a 12-hour on-off duty cycle to extend battery life. Immediately prior to deer capture, I initiated the duty cycle by removing magnets from the transmitters at 0430 hours, which caused the transmitters to become active at 0530 hours during the fawning period because of daylight savings time. Similar to others (Carstensen et al. 2003, Johnstone-Yellin et al. 2006), I used a temperature-sensitive switch that caused VITs to

increase pulse rates from 40 pulses to 80 pulses per minute when the temperature dropped below 32° C. A temperature drop below 32° C was indicative of the VIT being expelled from the deer.

After I initiated the 12-hour on-off cycle, I sterilized VITs in a chlorhexidine solution, rinsed them with sterile saline solution, and allowed them to air-dry before sealing them in 7.6 × 20.3-cm sterile pouches. I inserted VITs using a clear, plastic swine vaginoscope (Jorgensen Laboratories, Inc., Loveland, Colo.) and alligator forceps. The vaginoscope was 15.2 cm long with a 1.59 cm internal diameter and had a smoothed end to minimize vaginal trauma. I measured lengths of adult mule deer vaginal tracts from road-killed deer obtained in the study area to gauge approximate insertion distance. I placed vaginoscopes and alligator forceps in cold sterilization containers with chlorhexidine solution between each use and I used a new pair of nitrile surgical gloves to handle the vaginoscope and VIT for each deer. To insert a VIT, I folded the silicone wings together and placed the VIT into the end of the vaginoscope. I liberally applied sterile KY Jelly® to the scope and inserted it into the vaginal canal until the tip of the VIT antenna was approximately flush with the vulva. I used the alligator forceps, which extended through the vaginoscope, to firmly hold the VIT in place while the scope was pulled out from the vagina. The transmitter antenna was typically flush with the vulva, but it occasionally extended up to 1 cm outward. All capture and handling procedures, including VIT techniques, were approved by the Colorado Division of Wildlife's Animal Care and Use Committee (project protocols 11–2000 and 1–2002).

1.3.4 Radio-monitoring and Neonate Capture

I monitored live-dead status and general location of all radio-collared does daily from the ground during winter and spring. I located each of the adult does with VITs using aerial telemetry every 2–3 weeks from March through May. During each morning of June VIT signal status was checked by aerially locating each radio-collared doe having a VIT. Flights began at 0530 hours and were usually completed by 1000–1100 hours. Early flights were necessary to detect fast signals because temperature sensors of VITs expelled in open habitats and subject to sunlight often exceeded 32° C by mid-day, which caused VITs to switch back to a slow (i.e., prepartum) pulse. When a fast (i.e., postpartum) pulse rate was detected, very high frequency (VHF) receivers and directional antennae were used from the ground to simultaneously locate the VIT and radio-collared doe, which were typically in proximity to one another. My goal was to observe behavior of the collared doe, establish whether the VIT was shed at a birth site, and search for fawns in the vicinity of the doe and expelled VIT. In cases where the doe had moved away from the VIT (i.e., >200 m), the VIT was located to determine whether shedding occurred at a birth site and whether any stillborn fawn(s) were present, and the collared doe was subsequently located to search for fawns at her location. I attempted to account for each doe's fetus(es) as live or stillborn fawns in order to quantify in utero fetal survival from February to birth. I classified each fawn found dead at a birthsite as stillborn unless evidence was present to suggest the fawn was born alive. In most cases, I confirmed that the fawn had died before birth via laboratory necropsy. Surgical gloves were worn when handling fawns to help minimize transfer of human scent.

Most radio-collared does, that did not receive VITs, were located from the ground approximately every other day during June, and fawns were located by observing doe

behavior and conducting searches in the vicinity of the does. The same procedure was used for any VIT doe whose implant failed because of premature expulsion or battery failure. My technicians and I worked in pairs and partitioned the study area into segments, whereby each 2-person team was responsible for one segment. I used 3–4 teams during 2002 and 5–6 teams during 2003 and 2004.

1.3.5 Effectiveness of Vaginal Implant Transmitters

I assessed VIT effectiveness in terms of function, retention to parturition, and fawn capture success. I assigned the fate of each VIT to one of 7 categories: 1) censor, 2) migration loss, 3) battery or transmitter failure, 4) early prepartum shed (i.e., >3 days prepartum), 5) late prepartum shed (i.e., ≤3 days prepartum), 6) parturition shed within 200 m of the birth site, and 7) parturition shed at the birth site. I combined categories 6 and 7 for analysis because 92% of all parturition sheds were at the birth site or only several meters away. I censored VITs associated with prepartum doe mortalities and missing does (i.e., unable to detect radio-collar signal) because these deer failed to provide an adequate test of VIT effectiveness. In each case, the VIT was functioning correctly when the doe died or disappeared. There was no evidence to link VITs to the mortality events, which in most cases were caused by predation.

I considered migration losses, battery failures, and early prepartum sheds as VIT failures. Migration losses refer to VIT signals that disappeared during spring migration and represent either battery failures or early prepartum sheds between winter and summer range. In either event, I was unable to recover the VITs but lacked conclusive evidence to declare battery failures. I documented battery failures based on the disappearance of a doe's VIT signal after having consistently heard the signal on a daily basis. I allocated

≥20 minutes of aerial searching around each doe for 3 additional days before classifying a VIT as a battery failure. I confirmed 3 battery failures by opportunistically finding VITs when conducting ground searches for fawns in the vicinity of radio-collared does that's VITs had failed. In each case the VIT battery had failed.

I considered late prepartum sheds and parturition sheds as 2 levels of VIT success. I quantified the proportion of successful fawn captures associated with each level of VIT success, as well as the proportion of fawn captures from all non-VIT does and does with VIT failures. I describe effort associated with each type of fawn capture by calculating the number of person-hours per captured fawn. I describe costs associated with the 2 types of fawn capture by considering all operating and personnel expenses, including capture and transmitter costs for adult does. I used constant rates of $450/doe for helicopter net-gunning expenses and $215/transmitter (i.e., radio collars and VITs) in my calculations. I also determined the expected reduction in fawn capture costs if all VITs had been successful.

I evaluated the utility of VITs for quantifying direct estimates of fetal survival by determining the number of does with VITs for which the number of postpartum fawns observed (live or stillborn) equaled the number of fetuses measured in February–March. Each fetus was indirectly marked via the radio-marked doe, thereby allowing a direct survival estimate. The sample would be more robust by including all does in which ≥1 fetus was accounted for at parturition and right-censoring the missing fetuses. However, in this application, censoring would bias fetal survival estimates upward because I typically located only live fawns when ≥1 fetuses were missing. Additionally, such censoring would necessitate assuming the fetuses never existed because I could not

measure fetal survival as a function of time. This approach also assumes that no fetuses were resorbed, which appears reasonable for mule deer (Robinette et al. 1955, Medin 1976, Carpenter et al. 1984). I therefore considered the subset of does for which I recovered all fetuses at parturition as the viable sample for estimating fetal survival. I ultimately discovered that a known fates analysis was not practical and therefore developed a likelihood for estimating fetal survival when the fates of some fetuses are unknown (Chapter 2).

1.3.6 Deer Body Size and Implant Retention

I hypothesized that probabilities of VIT retention to parturition were related to deer behavior and morphology. I could not easily modify the former whereas I could address the latter by manufacturing different sizes of the silicone wings used to retain the implant in the vaginal canal. Modifications to the silicone wings would be costly and require additional research and development to accommodate on-site field application of different-sized wings (C. O. Kochanny, Advanced Telemetry Systems, personal communication). I evaluated VIT retention as a function of deer morphometric variables and age using PROC LOGISTIC in SAS (SAS Institute 2003) to determine whether retention probabilities decreased for larger adult does. I used a binary response model where the 2 levels of VIT retention were synonymous with my success-failure definitions: 1) VIT retained until ≤3 days prepartum (i.e., retained) and 2) VIT shed >3 days prepartum (i.e., not retained). I then performed a second modeling analysis to distinguish between the 2 levels of successful retention; the binary levels of the response variable were: 1) VIT retained to parturition (i.e., fully retained) and 2) VIT shed 1–3 days prepartum (i.e., partially retained). Independent variables were doe mass (kg), chest

girth (cm), hind foot length (cm), and age (yr). Ungulate mass and chest girth have been shown to be correlated (Weckerly et al. 1987, Cook et al. 2003). I used both variables in my analysis to provide a more complete evaluation of VIT retention and to avoid assumptions regarding which variable would be more informative. I only considered models with additive effects because I lacked a strong rationale for testing interactions among the variables. I evaluated model fit using the Hosmer-Lemeshow goodness-of-fit test (Hosmer and Lemeshow 2000). I performed model selection using Akaike's Information Criterion corrected for sample size (AIC_c) (Burnham and Anderson 2002).

1.3.7 Potential Bias of Differing Fawn Capture Strategies

I caused minimal disturbance during the fawning period to adult does with successful VITs because the does were only located from the ground once or twice to capture their fawns. Conversely, a majority of non-VIT does were tracked every 1–3 days during June until their fawns were captured. I compared survival rates between fawns captured with the assistance of VITs and fawns captured opportunistically by repeatedly tracking radio-collared does. I analyzed survival through 6 months of age using a common entry date and incorporating right-censoring as appropriate (Kaplan and Meier 1958, Pollock et al. 1989).

On occasion, newborn fawns were found when a radio-collared doe was located in the immediate vicinity of unmarked doe(s). I did not place radio-collars on newborn fawns when the identity of the dam was in doubt. However, some probability existed for mistakenly capturing fawns from a nearby unmarked doe. The probability was higher for fawns captured opportunistically following repeated locations of a collared doe, as compared to successful VIT sheds where the timing and location of birth was known. I

evaluated success of capturing the correct fawns by subsequently observing the radio-collared doe and fawn(s) together and by evaluating the return rate of surviving fawns to the correct winter range EU. For the latter analysis, I considered the sample of all radio-collared fawns that survived long enough to migrate to winter range and calculated the proportion that migrated to the correct winter range EUs.

1.4 RESULTS

1.4.1 Effectiveness of Vaginal Implant Transmitters

The proportion of all VITs that shed ≤3 days prepartum or during parturition was 0.565 (SE = 0.0410, n = 147), whereas the proportion of VITs shed during parturition was 0.401 (SE = 0.0406, n = 147). I censored VITs from 7 does; 5 died prepartum and 2 disappeared following spring migration. Of the remaining 147 VITs, I observed 7 migration losses, 23 battery failures, 34 early prepartum sheds, 24 late prepartum sheds, 5 parturition sheds within 200 m of the birth site, and 54 parturition sheds at the birth site. Of the battery failures, 16 of 23 occurred during 2002. Considering only data from 2003 and 2004, the proportion of VITs that shed ≤3 days prepartum or during parturition was 0.623 (SE = 0.0456, n = 114), and the proportion of VITs shed during parturition was 0.447 (SE = 0.0468, n = 114). During 2003–2004, I observed 4 censors, 7 migration losses, 7 battery failures, 29 early prepartum sheds, 20 late prepartum sheds, and 51 parturition sheds (48 at the birth site).

My neonate capture success rate was 0.915 (SE = 0.0366, n = 59) for does with VITs shed during parturition and 0.792 (SE = 0.0847, n = 24) for does with VITs shed late prepartum. Combining both levels of VIT success (i.e., late prepartum and parturition sheds), my neonate capture success rate was 0.880 (SE = 0.0359, n = 83).

Neonate capture success was 0.438 (SE = 0.0625, n = 64) for does with VIT failures and 0.282 (SE = 0.0251, n = 323) for all non-VIT, radio-collared does. I intensively located does with failed VITs from the ground because of my previous data investment (i.e., fetus counts and body condition measures) and to document the number of days VITs were prematurely shed by identifying timing of birth. Conversely, frequency of locations was more variable for non-VIT, radio-collared does during the fawning period, particularly those located on the fringes of the study area or in remote areas. My overall neonate capture success rate based on repeated ground telemetry locations and corresponding fawn searches for all radio-collared does without VITs or where VITs were ineffective was 0.307 (SE = 0.0235, n = 387).

I captured 296 neonates (including stillborns) from radio-collared does during the study: 89 from does with VITs shed during parturition, 25 from does with VITs shed late prepartum, 43 from does with failed VITs, and 139 from does not receiving VITs. Daily capture crews during June included 6 individuals in 2002, 8 individuals in 2003, 9 individuals in 2004, and part-time assistance from 3–4 additional personnel during all years. Total person-hours also included 4–5 fixed-wing pilot-hours per day for VIT monitoring and occasional non-VIT doe monitoring. Approximately 8,660 total person-hours were committed to capture the 296 neonates, or an average of 29.3 person-hours per captured fawn. Approximately 7 person-hours were required per captured fawn from does with parturition-shed VITs, 16 person-hours per captured fawn from does with VITs shed late prepartum, and 42 person-hours per captured fawn from does with failed VITs and does not receiving VITs. I attributed 75% of the pilot-hours toward fawns captured from does with successful VITs.

Considering all capture, transmitter, and personnel costs, I spent approximately $1,325 per fawn captured from does receiving VITs and $890 per fawn captured from does not receiving VITs. I used helicopter net-gunning to capture all does that received VITs whereas I used drop nets to capture most does that did not receive VITs, which partially explains the observed difference. If all does had been captured via net-gunning, the cost per fawn associated with non-VIT does would increase to approximately $1,200. My estimate of $1,325 per fawn when using VITs included the 43 fawns captured from does with VIT failures. My cost was $1,670 per fawn captured from does with successful VITs, which more appropriately reflects costs of VIT application in this study. If all VITs had been successful, my cost per captured fawn would reduce to approximately $860.

I accounted for all fetuses at birth (i.e., live or stillborn) from 78 of the 147 available does (0.531, SE = 0.0413) with in utero fetal measurements. The ability to locate each of a doe's fawns was heavily dependent on VIT success. I accounted for all fetuses from 0.780 (SE = 0.0544, $n = 59$) of does with VITs shed during parturition, 0.458 (SE = 0.104, $n = 24$) of does with VITs shed late prepartum, and 0.328 (SE = 0.0592, $n = 64$) of does with VIT failures.

1.4.2 Deer Body Size and Implant Retention

My models of VIT retention to ≤3 days prepartum ($\chi^2_8 = 4.00$, $P = 0.857$) and of VIT retention to parturition ($\chi^2_8 = 3.41$, $P = 0.906$) adequately fit the data. I considered only 11 models in each analysis. My model selection results provided strong evidence that VIT retention varied as a function of deer body mass and chest girth (Table 1). The probability of VIT retention decreased for larger deer. Mass and chest girth were highly

correlated as expected (R^2 = 0.566). Mass explained the most variation in VIT retention to within 3 days of parturition (Table 1, Figure 2). The beta estimate for mass based on the best model {VIT retention (mass)} was –0.0967 (SE = 0.0340). Mean mass of does shedding VITs early prepartum was 67.2 kg (SE = 1.24) whereas mean mass of does retaining VITs until ≤3 days prepartum was 63.0 kg (SE = 0.687). Chest girth explained the most variation in VIT retention to parturition, which distinguished between late prepartum and parturition sheds (Table 1, Fig. 3). The beta estimate for chest girth based on the best model {VIT retention (chest girth)} was –0.0923 (SE = 0.0542). The next best model {VIT retention (chest girth + age)} suggested an effect of age on VIT retention ($\hat{\beta}_{age}$ = –0.190, SE = 0.149, Table 1), indicating VIT retention to parturition decreased for older does. Mean chest girth was 96.8 cm (SE = 1.06) and mean age was 4.5 years (SE = 0.44) for does shedding VITs 1–3 days before birth, as compared to 94.7 cm (SE = 0.612) and 3.7 years (SE = 0.19) for does retaining VITs until parturition. In each analysis the intercept-only model received minimal AIC_c weight (Table 1), providing additional evidence that the morphometric variables were important effects.

1.4.3 Potential Bias of Differing Fawn Capture Strategies

I found minimal evidence that capture strategy affected fawn survival. Survival of fawns captured from does with successful VITs ($S(t)$ = 0.558, SE = 0.0618, n = 104) was relatively similar (χ^2_1 = 1.51, P = 0.220) to survival of fawns captured through repeated locations of radio-collared does ($S(t)$ = 0.471, SE = 0.0458, n = 171).

My rate of correctly capturing neonates from the targeted, radio-collared does based on fawn migrations to the appropriate winter range EUs was 0.947 (SE = 0.0196, n = 131). Seven fawns were incorrectly identified and radio-collared during the study,

which included 2 sets of twins and 3 singletons. The 5 incorrect capture incidents occurred in high-density deer areas and involved 3 non-VIT does, a doe with an early-prepartum VIT shed, and a doe with a late-prepartum VIT shed. In 2 cases, an uncollared adult doe was present with the radio-collared doe at the time of capture. I decided to radio-collar the neonate in each case, fully realizing the decision was debatable, based on behavioral cues of the radio-collared doe. These were the only 2 incidents in the study where I opted to radio-collar neonates when ≥1 doe was present. With the exception of these 2 questionable incidents, the rate of capturing the correct neonates from targeted does was 0.961 (SE = 0.0171, n = 129).

1.5 DISCUSSION

I found VITs to be an effective technique for capturing mule deer fawns from targeted, radio-collared adult does. I captured fawns from 88% (SE = 3.59) of adult does expelling VITs within 3 days of parturition. Similarly, Carstensen et al. (2003) reported 89% neonate capture success for white-tailed deer with VITs expelled at or near birth sites. Following technique improvements, Pamplin (2003) reported 61% capture success for black-tailed deer with VITs expelled at birth sites in thick vegetative cover. The main disadvantage of VITs was the inefficiency associated with a relatively high failure rate, which has been a common issue in studies of free-ranging deer (Bowman and Jacobson 1998, Carstensen et al. 2003, Johnstone-Yellin et al. 2006). The 2 main causes of VIT failures in my study were battery failures and early prepartum sheds. I greatly reduced battery failures by incorporating a 12-hour duty cycle, and I found evidence that early prepartum VIT sheds were a function of deer body size. The latter indicates a need to manufacture variable-sized silicone retention wings that may be fitted to deer much in the

same way radio collars are individually fitted. The future applicability of VITs may depend on how well subsequent VIT modifications lower the failure rate, which could reduce overall expense of the technique by as much as half. Aside from specific applications such as capturing fawns from target does, VITs could be applied broadly to facilitate random neonate capture spatially and temporally, thereby minimizing sample bias that is innate to most other capture techniques.

Neonate captures associated with VITs were spatially random in the sense that adult does captured on winter range determined neonate capture locations instead of road access or vegetation type. I maintained this spatial randomness during fawning by aerially monitoring all VIT signals with equal effort regardless of deer location. Other neonate capture techniques (White et al. 1972, Hamlin et al. 1984, Ballard et al. 1998, Pojar and Bowden 2004) have likely led to collared fawn samples that were biased towards roads, open habitats, or both. Vaginal implants facilitated temporal randomness at both the individual level (i.e., capturing fawns as newborns rather than as older, mixed-aged fawns) and population level (i.e., capturing a representative sample of fawns from the beginning to end of fawning).

Nearly all fawns captured from does with VITs shed during parturition were <2 days old and 75% were ≤1 day old. Biases arise when older fawns are captured because early mortality is missed (Pamplin 2003, Pojar and Bowden 2004). I captured several newborn fawns during the first week of June and again during the first 2 weeks of July, and I had 3 VITs remain in deer until late July indicating the respective does had not yet given birth. Similarly, Johnstone-Yellin et al. (2006) observed 2 of 19 captive does give birth in early-mid July. When other techniques are used (e.g., opportunistic fawn

searches), capture periods typically end when a target sample has been captured or a point of diminishing returns is reached, neither of which fosters a representative sample of late-born fawns nor an accurate understanding of the proportion of adult females conceiving late in estrous or during a second estrous. Finally, I demonstrated that VITs can be used to directly measure fetal survival in free-ranging populations. Vaginal implants facilitated an optimal neonate sampling approach and could therefore be considered a preferred capture strategy even in situations where adequate samples of fawns have been captured using other techniques. Additional benefits of using VITs include measurements of birth site characteristics and deer fidelity to birth sites. Current failure rates and expense remain limiting factors to widespread application.

I demonstrated that large samples of fawns can be captured from radio-collared adult does via repeated ground locations and associated searches during the fawning period. Prior to this research, communications with peers and my own past experiences suggested this approach would not be successful over large areas of forest-shrub habitats in the Intermountain West; I have shown that such efforts can be successful with adequate technology and personnel. Overall capture costs associated with this technique were less than those associated with VITs even though personnel costs were much higher. Vaginal implants were expensive because of observed failure rates and annual costs to redeploy them. Conversely, I accumulated radio-collared adult does without VITs over the course of the study at comparatively minimal expense, thereby creating a large pool of fawns available for capture. I only met my neonate sample size objectives by combining the use of both capture techniques, which has important implications for any research where there is an advantage to capturing fawns from previously-marked adult

does. The necessity of capturing fawns by repeatedly locating collared does will diminish as VIT failure rates are reduced. Even so, neonate capture via ground-telemetry of radio-collared adult does affords a viable opportunity to bolster sample sizes of neonates from target does.

I did not detect a difference in survival between fawns captured from does with successful VITs and fawns captured from does through repeated ground telemetry. Although I had large samples (i.e., >100/group), I lacked power to detect a small but biologically significant difference (e.g., <10%). The point estimate for survival of fawns captured with VITs was higher than that for fawns captured without VITs, which should lessen potential concerns regarding impacts of VITs on fawn survival. Aside from the physical intrusion of the implant itself during gestation, VITs were beneficial by requiring only 1 or 2 site visits to capture fawns during the fawning period as opposed to repeated site visits and associated doe disturbances.

Vaginal implants enhanced the ability to capture the correct fawns from target does. Overall, 96% of fawns were correctly associated with target does when excluding 2 questionable incidents that were not representative of neonate capture in my study. Although parturient does typically isolate themselves from other deer (Downing and McGinnes 1969, Robinette et al. 1977, Ozaga et al. 1982, Schwede et al. 1993), I occasionally observed ≤3-day-old fawn(s) together with ≥2 does, thereby making it difficult to determine which fawns belonged to which doe.

1.6 MANAGEMENT IMPLICATIONS

I demonstrated that VITs can be used in conjunction with repeated locations of radio-collared adult does during fawning to capture large samples of neonates exclusively

from marked deer, which expands opportunities for conducting experimental studies of free-ranging, migratory deer populations. Direct estimates of fetal survival may be obtained by combining in utero fetal counts with VITs. Current VIT failure rates and overall expense limit applicability of the technique to well-funded studies with adequate personnel. Nevertheless, VITs have broad applicability for use in capturing random samples of neonates and generating unbiased estimates of neonate survival. Additional design modifications of VITs should incorporate different-sized silicone retention wings that may be fitted to individual deer to minimize premature expulsion of VITs.

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Table 1. Model selection results, based on Akaike's Information Criterion with small sample size correction (AIC_c), for evaluating vaginal implant transmitter (VIT) retention in adult female mule deer using logistic regression as a function of deer mass (kg), chest girth (chest; cm), hind foot length (foot; cm), and age (yr) in southwest Colorado, 2002–2004.

Dependent variable[a]	Model	No. parameters	AIC_c	ΔAIC_c	Akaike weight
VIT retention to ≤3 days prepartum	Mass	2	135.9	0.00	0.30
	Mass, chest	3	137.1	1.18	0.17
	Chest	2	137.7	1.79	0.12
	Mass, age	3	137.8	1.92	0.12
	Chest, age	3	138.1	2.23	0.10
	Mass, chest, age	4	138.9	3.02	0.07
	Mass, chest, foot	4	139.0	3.07	0.06
	Age	2	140.9	5.00	0.02
	Mass, chest, foot, age	5	140.9	5.03	0.02
	Intercept only	1	143.1	7.15	0.01
	Foot	2	143.6	7.64	0.01
VIT retention to parturition	Chest	2	97.7	0.00	0.28
	Chest, age	3	98.3	0.56	0.21
	Mass, chest, age	4	99.1	1.40	0.14
	Mass, chest	3	99.5	1.76	0.12
	Age	2	100.3	2.62	0.08

Table 1. Continued.

Dependent variable[a]	Model	No. parameters	AIC_c	ΔAIC_c	Akaike weight
	Mass, chest, foot, age	5	101.3	3.61	0.05
	Mass, chest, foot	4	101.7	3.96	0.04
	Intercept only	1	101.9	4.15	0.03
	Mass, age	3	102.5	4.77	0.03
	Foot	2	103.1	5.38	0.02
	Mass	2	103.3	5.55	0.02

[a]I present 2 model selection analyses; I define levels of the binary response variable, VIT retention, differently in each analysis. In the first analysis, the 2 levels of VIT retention are: 1) retained (i.e., VIT retained to ≤3 d of parturition) and 2) not retained (i.e., VIT shed >3 d prepartum). In the second, the levels of VIT retention are: 1) fully retained (i.e., VIT retained to parturition) and 2) partially retained (i.e., VIT shed 1–3 d prepartum).

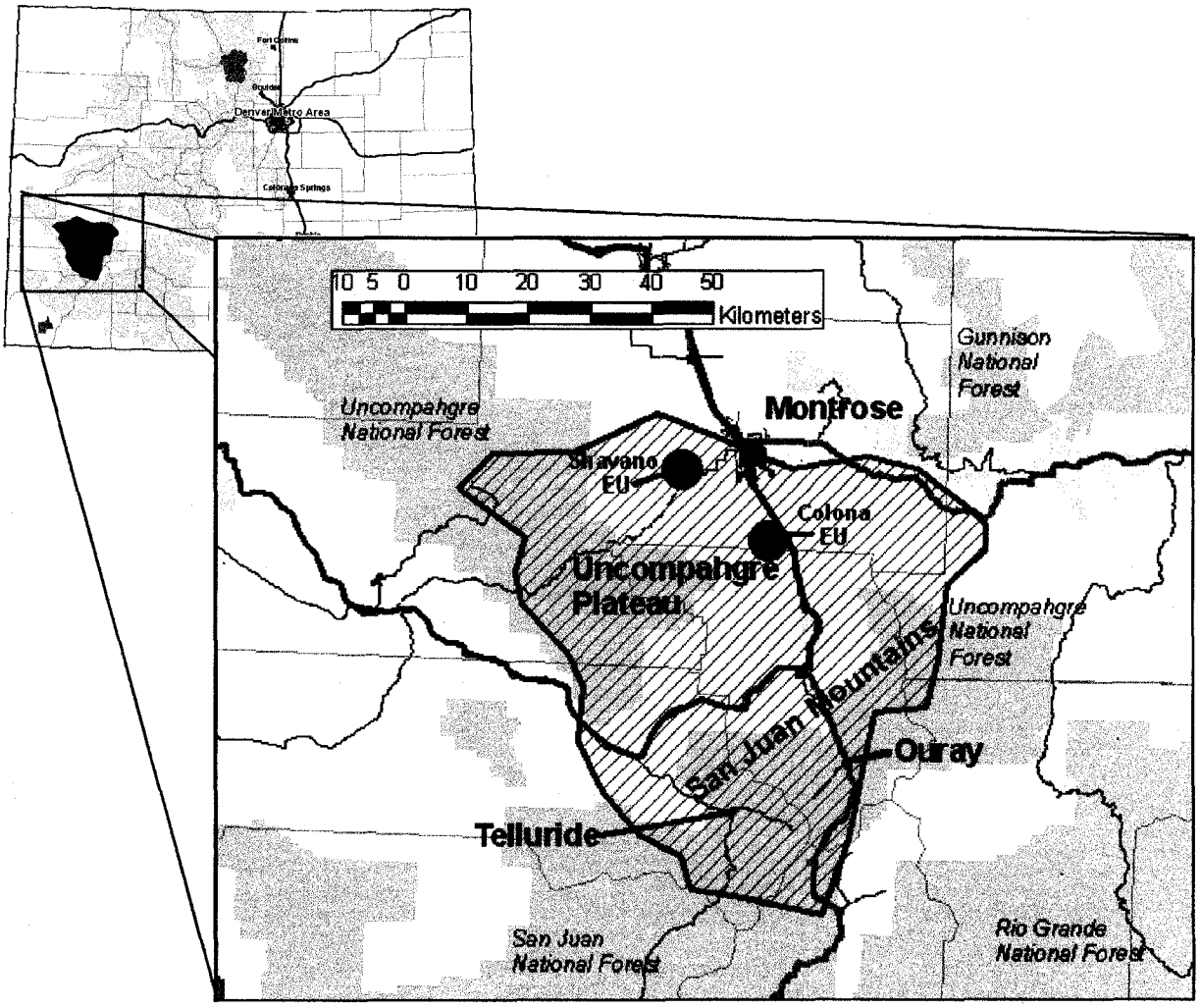

Figure 1. Location of winter range experimental units (EU; ●) and summer range study area (▨) on the Uncompahgre Plateau and adjacent San Juan Mountains in southwest Colorado, where I studied vaginal implant transmitters in mule deer, 2002–2004.

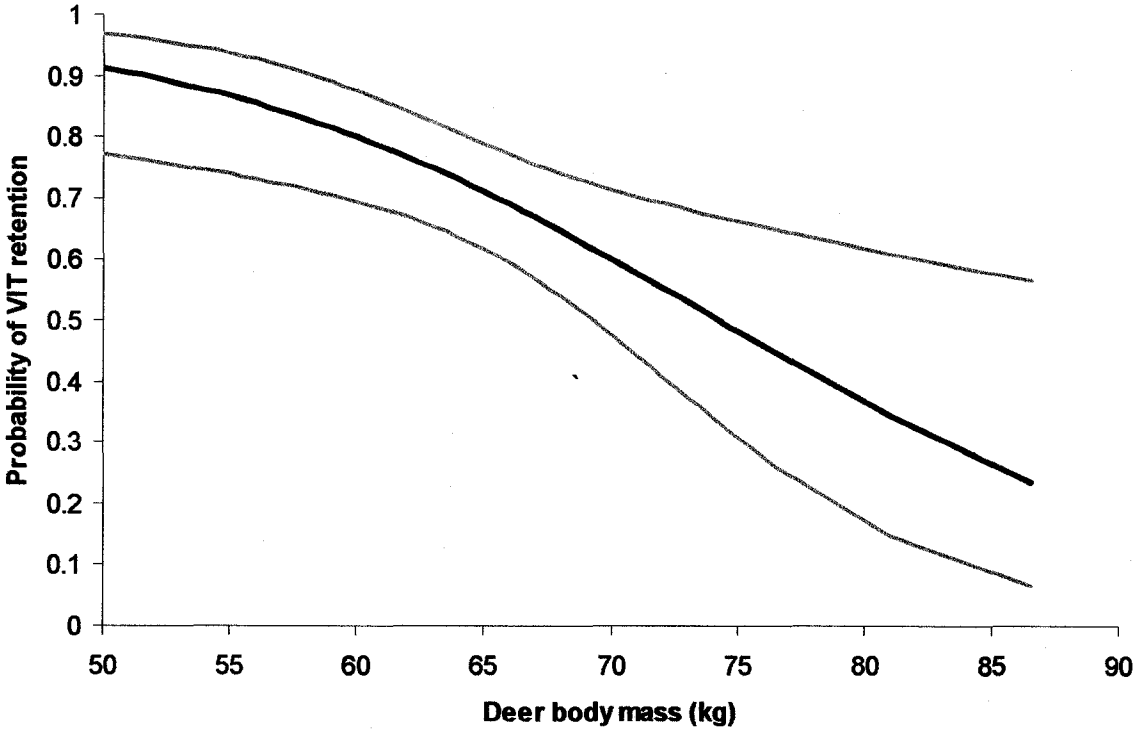

Figure 2. Estimated probability and 95% confidence interval of adult female mule deer retaining vaginal implant transmitters (VITs) until ≤3 days of parturition as a function of deer body mass, southwest Colorado, 2002–2004. The underlying dataset includes all deer receiving VITs during late February or early March, except those with VITs that were never recovered due to battery failure or transmitter loss.

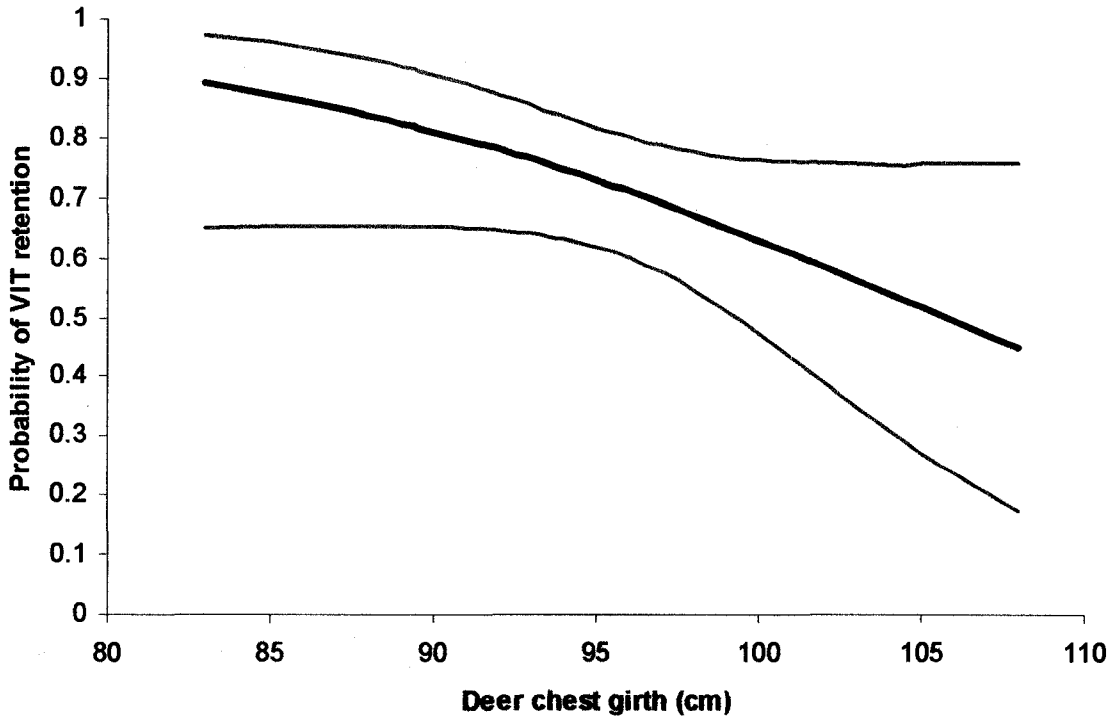

Figure 3. Estimated probability and 95% confidence interval of adult female mule deer retaining vaginal implant transmitters (VITs) to parturition as a function of deer chest girth, southwest Colorado, 2002–2004. The underlying dataset includes only those deer retaining VITs until ≤3 days of parturition (i.e., successful VITs), thereby distinguishing between deer shedding VITs 1–3 days prepartum versus deer shedding VITs at parturition.

CHAPTER 2

EVALUATING DEPENDENCE AMONG MULE DEER SIBLINGS IN FETAL AND NEONATAL SURVIVAL ANALYSES

Abstract: The assumption of independent sample units is potentially violated in deer (*Odocoileus* spp.) fetal and neonatal survival analyses where twin and triplet siblings comprise a high proportion of the sample. Violation of the independence assumption causes sample data to be overdispersed relative to a binomial model, and therefore requires a variance inflation factor, c, to obtain appropriate estimates of sampling variances. I evaluated overdispersion in fetal and neonatal mule deer (*O. hemionus*) datasets where more than half of the sample units were comprised of siblings. I developed a likelihood function for estimating fetal survival when the fates of some fetuses are unknown, and I used extensions of the binomial model to estimate neonatal survival. I compared theoretical variance estimates obtained from these analyses with empirical variance estimates obtained from data bootstrap analyses to estimate the overdisperion parameter, c. My estimates of c for fetal survival ranged from 0.678 to 1.118, which indicate little to no evidence of overdispersion. For neonatal survival, mean $\hat{c}$ was 1.25, providing evidence of limited overdispersion (i.e., limited sibling dependence). In biological terms, the fates of sibling mule deer neonates may often be independent even though they have the same dam and use the environment similarly. Predation tends to act independently on sibling neonates because of dam-neonate behavioral adaptations, although I observed evidence of a predator(s) killing siblings on

several occasions. The effect of maternal characteristics on sibling fate dependence is less straightforward and may vary by circumstance. I recommend that future neonatal survival studies incorporate additional sampling intensity to accommodate modest overdispersion (i.e., $\hat{c} = 1.25$), which would facilitate a corresponding $\hat{c}$ adjustment in a model selection analysis using quasi-likelihood without a reduction in power.

Key Words: bootstrap, c-hat, dependence, extra-binomial variation, fawn, fetal survival, goodness-of-fit, mule deer, neonatal survival, *Odocoileus hemionus*, overdispersion, sibling.

2.1 INTRODUCTION

Independence of sample units is required in survival analyses to obtain appropriate estimates of sampling variance. The independence assumption is likely violated when sample units include monogamous mates or siblings. For example, adult pairs of Canada geese (*Branta canadensis*) typically mate for life and therefore do not behave independently of one another (Anderson et al. 1994, Sheaffer et al. 2004). Littermates of numerous species access the same nutrient supply and are often subjected to the same mortality factors. Generally speaking, some degree of biological dependence exists among individuals that use the same resources in time and space.

The independence assumption is potentially violated in studies of fetal and neonatal deer (*Odocoileus* spp.) survival. Adult female deer commonly produce twins and occasionally triplets. Siblings should not be assumed to have independent fates, because they have the same dam, share at least 0.25 of their genome, and are exposed to

nearly identical environmental conditions postpartum. Maternal body condition and disease status as well as predation are mechanisms that could cause dependence among sibling fates. However, siblings are commonly radio-collared in neonatal survival studies (Hamlin et al. 1984, Whittaker and Lindzey 1999, Carstensen et al. 2003, Jarnemo and Liberg 2005). There are advantages to marking siblings, but the potential dependence of survival outcomes should be addressed. Sample unit dependence is also encountered in measurements of fetal survival, which can be made in free-ranging deer populations with the aid of ultrasonography and vaginal implant transmitters (Chapter 1). In fact, inclusion of siblings is required when estimating fetal survival because the fetus sample is obtained from in utero counts, and each of a doe's fetuses must be counted without error. Twin or triplet fetuses cannot be individually marked in utero, and therefore 1 fetus cannot be randomly chosen to include in the sample while the other(s) is disregarded.

When the independence assumption is violated, survival point estimates are typically unbiased but sample variances are underestimated (Wedderburn 1974, Cox 1983, Firth 1987, Breslow 1990, Schmutz et al. 1995). This condition is referred to as overdispersion or extra-binomial variation. An overdispersion parameter, or variance inflation factor, c, is required to correct for overdispersed data, thereby facilitating appropriate inference using quasi-likelihood theory (Wedderburn 1974, Burnham and Anderson 2002). The simplest approach for estimating c requires only the standard goodness-of-fit (GOF) test statistic for the global (i.e., most parameterized) model and its degrees of freedom (Cox and Snell 1989):

$$\hat{c} = \chi^2 / df .$$

The global model is needed to avoid mistaking model-structure variation (i.e., lack of model fit) for overdispersion (Burnham and Anderson 2002). Unfortunately, this simple approach fails to provide an appropriate estimate of c under many circumstances because of inadequate sample sizes. Instead, as in the case of deer fetal and neonatal survival, more sophisticated numerical approaches are required (Schmutz et al. 1995).

There is a need to evaluate whether theoretical variance estimates of deer fetal and neonatal survival are valid, or whether variance inflation procedures are required to account for dependence among siblings. My objective was to determine the degree of overdispersion in fetal and neonatal mule deer (*O. hemionus*) datasets where more than half of the sample units comprised siblings. In accomplishing this objective, I develop and present a likelihood function for estimating deer fetal survival when the fates of some fetuses are unknown, and I describe bootstrap procedures for evaluating overdispersion in survival data from siblings. My results have implications for (1) fetal survival estimation, (2) confidence associated with point estimates of fetal and neonatal deer survival, (3) power calculations in the design of future sibling survival studies, and (4) biological interpretation of sibling relationships in free-ranging mule deer populations.

2.2 STUDY AREA

I conducted field work in southwest Colorado on the southern half of the Uncompahgre Plateau and in the adjacent San Juan Mountains. Winter range habitat comprised pinyon pine (*Pinus edulis*) and Utah juniper (*Juniperus osteosperma*) woodlands with interspersed big sagebrush (*Artemisia tridentata*) adjacent to irrigated agricultural fields; elevations ranged from 1,830 m to 2,290 m. During my study, annual precipitation averaged 22.3 cm and the minimum temperature in January averaged –8.2°

C in Montrose, Colorado (Western Regional Climate Center [WRCC] 2005), which is 60 m below the lowest winter range elevation. Deer occupied winter range from November through April each year.

Summer range habitat types included pinyon–juniper, Gambel oak (*Quercus gambelii*), ponderosa pine (*Pinus ponderosa*), big sagebrush, aspen (*Populus tremuloides*), and mixed forests of Engelmann spruce (*Picea engelmannii*) and subalpine fir (*Abies lasiocarpa*). Elevations ranged from 1,830 m to 3,500 m, with a majority of deer summering between 2,600 m and 3,000 m. Diverse habitat mosaics occurred at interfaces of each of the major habitat types. Annual precipitation averaged 57.4 cm and the maximum temperature in July averaged 26.7° C at a weather station in the summer range situated at 2,438 m elevation (WRCC 2005). I provide a detailed study area description in Chapter 3.

2.3 METHODS

2.3.1 Fetus and Neonate Samples

All fetuses and neonates used in my study were offspring of free-ranging, radio-collared adult doe mule deer. I administered a nutrition enhancement treatment to one-half of the adult does during winter and early spring to meet research objectives detailed in Chapter 3. I did not provide any treatment to the other half of the adult does, which I will hereafter refer to as control deer.

I used transabdominal ultrasonography (Stephenson et al. 1995) to count the number of fetuses in utero in each of 154 pregnant, adult doe mule deer during 26 February–2 March, 2002–2004 (Chapter 1). I placed a vaginal implant transmitter (VIT) in each doe as a mechanism to help determine the timing and location of birth the

following spring or summer. I then attempted to find birth sites and determine the fate of each of a doe's fetuses as live or stillborn (Chapter 1). I assumed that no fetuses were resorbed, which is a reasonable assumption for mule deer (Robinette et al. 1955, Medin 1976, Carpenter et al. 1984). I was unable to obtain an accurate fetal count for 9 does, which I removed from the analysis. I also removed 5 does from the analysis that died prior to giving birth and 2 does that I could not locate following spring migration. My resulting sample for analysis comprised 255 fetuses from 138 does (29 does with 1 fetus, 101 does with 2 fetuses, 8 does with 3 fetuses). The proportion of the fetal sample comprised of twins or triplets was 0.886, indicating a high potential for dependence among sample units.

I captured neonates from radio-collared does with the assistance of VITs and by repeatedly locating does during the fawning period (Chapter 1). I captured and radio-collared 276 neonates during 4 June–8 July, 2002–2004. I removed 6 fawns from the sample because of possible capture-related abandonment or injury, resulting in a total sample of 270 radio-collared neonates from 178 radio-collared does (88 does with 1 marked fawn, 88 does with 2 marked fawns, 2 does with 3 marked fawns). The proportion of the sample comprising sibling neonates was 0.674 (182/270), indicating a high potential for dependence among sample units. I radio-monitored all neonates daily to identify mortalities during June–December each year. I assessed evidence at the mortality site and performed necropsies on intact carcasses in a laboratory to identify causes of death. I classified mortalities as coyote (*Canis latrans*) predation, bear (*Ursus americanus*) predation, felid predation, undetermined predation, disease or illness, starvation, malnutrition, trauma or injury, abandonment, and unknown. I right-censored

46 fawns during the analysis period (i.e., June–December, 2002–2004). Right censoring occurred when fawns prematurely shed radio collars, typically while crossing fences during migration from summer range to winter range, which indicated the censored individuals were likely independent of fawn fates. The right censored data had relatively little impact on survival analyses because most occurred during fall after much of the mortality had occurred.

2.3.2 Overdispersion in Relation to Model Structure

I assessed overdispersion in my fetus and neonate sample data by comparing the results of 2 analyses. I first analyzed fetal and neonatal survival using methods that assumed independence among sample units. I then conducted data-bootstrap analyses to obtain variance estimates that reflected dependence among sample units, which are described in detail below. To correctly estimate c from these analyses, variation due to model structure must be removed or isolated (Burnham and Anderson 2002). For example, I expected neonatal deer survival to vary by year because of fluctuating environmental conditions. If I disregarded year in my analysis, unexplained annual variation could be misinterpreted as overdispersion, resulting in an inflated estimate of c. Stated differently, a lack of structural fit of the model could be misinterpreted as overdispersion. Treatment status and year were the key structural variables common to all fetal and neonatal survival models. I therefore evaluated overdispersion using highly parameterized models that resulted in separate estimates of fetal and neonatal survival for each of the 6 combinations of treatment status (treatment, control) and year (2002–2004).

2.3.3 Fetal Survival

I was unable to determine the fate of 96 of the 255 (0.376) fetuses because some VITs were ineffective and newborn fawns were difficult to detect. I did not censor the missing fetuses because the censoring mechanism would not be independent of fetus fate. Specifically, censoring would tend to bias fetal survival estimates upward because I typically located only live fawns when ≥ 1 fetuses were missing. Additionally, such censoring would remove the missing fetuses from the analysis altogether because I could not measure fetal survival as a function of time (i.e., undetected fetuses would be treated as if they never existed). I therefore developed a joint likelihood to estimate fetal survival that incorporated all fetuses into the analysis. The likelihood includes several nuisance detection parameters which allow estimation of fetal survival in the absence of known fates. I constructed the likelihood based on the following scenarios: 1) fetus survived and was monitored and detected as a neonate ≤ 1 day postpartum; 2) fetus died, was stillborn at the birth site next to a successful VIT, and detected solely as a result of the VIT; 3) fetus survived and was monitored but not detected ≤ 1 day postpartum, and survived and was detected as a neonate during the first week postpartum; 4) fetus survived, was not monitored ≤ 1 day postpartum, and survived and was detected as a neonate during the first week postpartum; 5) fetus died, was not located with a successful VIT, but was detected opportunistically while searching for fawns after radio-locating a doe; and 6) fetus was never detected. The joint likelihood is:

$$L(S_1,a,p_1,b,r,S_2,p_2 \mid n,x_i,k,u_j,l,w) = \binom{n}{x_1x_2x_3x_4x_5x_6}[S_1ap_1]^{x_1}[(1-S_1)b]^{x_2}[S_1a(1-p_1)S_2p_2]^{x_3}$$
$$[S_1(1-a)S_2p_2]^{x_4}[(1-S_1)(1-b)r]^{x_5}[S_1a(1-p_1)(1-S_2)+S_1(1-a)(1-S_2)+S_1a(1-p_1)$$
$$S_2(1-p_2)+S_1(1-a)S_2(1-p_2)+(1-S_1)(1-b)(1-r)]^{x_6} \bullet \binom{k}{u_1u_2u_3u_4}[ab]^{u_1}[(1-a)b]^{u_2}$$
$$[a(1-b)]^{u_3}[(1-a)(1-b)]^{u_4} \bullet \binom{l}{w}S_2{}^{w}(1-S_2)^{l-w}.$$

Model parameter and data descriptions follow:

S_1 = fetal survival probability.

a = probability of radio-locating a doe and searching for her fawn(s) ≤ 1 day postpartum (i.e., probability of monitoring a doe's fawns ≤1 day postpartum).

p_1 = probability of detecting a neonatal fawn ≤ 1 day old given that a search was conducted ≤1 day postpartum.

b = probability a VIT was shed at a birth site.

r = probability of detecting a stillborn fetus when a VIT was not shed at a birth site.

S_2 = neonatal survival probability from birth to 5 days old.

p_2 = probability of detecting a neonatal fawn >1 day old given that a search was conducted >1 day postpartum.

n = total sample of fetuses documented in utero during winter.

x_1 = number of fetuses that survived and were detected as neonatal fawns ≤ 1 day old.

x_2 = number of fetuses that were stillborn and were found at birth sites along with a VIT.

x_3 = number of fetuses that survived, were monitored but not detected ≤ 1 day postpartum, and were later detected as neonatal fawns >1 day postpartum.

x_4 = number of fetuses that survived, were not monitored ≤ 1 day postpartum, and were detected as neonatal fawns >1 day postpartum.

x_5 = number of fetuses that were stillborn and were detected without assistance from a VIT.

x_6 = number of fetuses that were never detected.

k = total sample of adult does that received VITs and whose fetuses were counted in utero during winter.

u_1 = number of adult does that shed VITs at birth sites and were monitored for newborn fawns ≤ 1 day postpartum.

u_2 = number of adult does that shed VITs at birth sites but were not monitored for newborn fawns ≤ 1 day postpartum.

u_3 = number of adult does that did not shed VITs at birth sites but were monitored for newborn fawns ≤ 1 day postpartum.

u_4 = number of adult does that did not shed VITs at birth sites and were not monitored for newborn fawns ≤ 1 day postpartum.

l = total sample of neonatal fawns that were captured and radio-collared ≤ 1 day postpartum (includes newborn fawns that were not part of the fetus sample).

w = number of neonatal fawns captured ≤ 1 day postpartum that survived until ≥5 days postpartum.

If a VIT was shed at a birth site, I assumed stillborn detection probability was 1. This assumption seemed reasonable because these stillborns were found next to the VITs and were detected with ease. I defined S_2 as survival probability from birth to 5 days old

because neonatal fawns detected >1 day postpartum were generally found within 5 days of birth.

I numerically maximized the natural logarithm of the likelihood function using a quasi-Newton optimization algorithm in PROC NLMIXED in SAS (SAS Version 9.1, 2003) to obtain parameter estimates and the variance-covariance matrix. To test validity of the likelihood function, I generated 1000 fetal datasets in SAS using specified parameter values and then used maximum likelihood (ML) to analyze the simulated datasets. The mean of the 1000 parameter estimates were identical to the parameter values used to generate the data. I then analyzed my observed fetal data using the model, $\{S_1$(year×treatment) S_2(year×treatment) p_1(year×treatment) p_2(year×treatment) r(.) a(year×treatment) b(year×treatment)$\}$. A constraint on r was needed because I failed to detect stillborns without assistance of a VIT during several year×treatment combinations, and therefore r could not be separately estimated.

2.3.4 Neonatal Survival

I analyzed neonatal survival using extensions of the binomial model that accommodated data structures ranging from simple to complex, which facilitated a comprehensive assessment of overdispersion. First, I used a binomial estimator to estimate survival from birth through August, prior to when most right-censoring occurred. All fawns were assigned a 1 (survived) or 0 (died) with the exception of 5 fawns that were right-censored prior to August. I analyzed survival as a function of treatment status and year. I also considered neonate sex as a variable, but sex explained relatively minimal variation in survival and would have complicated litter resampling in the bootstrap analysis. I expected dependence of sibling fates to be greatest during the

first weeks and months following birth, making this approach reasonable. I chose to perform this analysis primarily because it facilitated a straight-forward assessment of overdispersion.

Second, I analyzed survival using a staggered-entry Kaplan-Meier analysis to accommodate different entry times of animals into the analysis and to accommodate right-censoring (Kaplan and Meier 1958, Pollock et al. 1989). I analyzed survival as a function of fawn age (i.e., days survived since birth), treatment status, and year. For clarification, fawn age could be thought of as a temporal variable during the first 6 months of life. I refer to this variable as fawn age throughout the text to avoid confusion. I used 182 daily survival intervals to construct encounter histories of survival from birth to 6 months of age. Fawns that were ≤1 day old when captured were included in the first survival interval, fawns that were >1 and ≤2 days old when captured entered the analysis in the second survival interval, and so forth. A majority of neonates in my sample (0.748) were ≤2 days old when captured and most (0.904) were ≤4 days old when captured. I estimated variance using 2 different estimators that have been discussed elsewhere (Cox and Oakes 1984, Pollock et al. 1989). The first estimator was developed by Greenwood (1926):

$$\hat{\text{var}}[\hat{S}(t)] = [\hat{S}(t)]^2 \sum_{1}^{j|a_j<t} \frac{d_j}{r_j(r_j - d_j)},$$

where $\hat{S}(t)$ is the estimated survivor function for any arbitrary time t, a_j is the j^{th} point in time that one or more deaths occurred, r_j is the number of animals at risk at a_j, d_j is the number of deaths at a_j, and the summation is for all death times $a_j < t$. The Greenwood (1926) estimator corresponds to the variance estimated from a binomial model with no

censoring (White and Garrott 1990). The second estimator was presented by Cox and Oakes (1984:51):

$$\hat{\text{var}}[\hat{S}(t)] = \frac{[\hat{S}(t)]^2[1-\hat{S}(t)]}{r(t)},$$

where $r(t)$ is the number of animals at risk at time t.

The latter estimator often produces larger variance estimates than the former estimator but has better properties when $\hat{S}(t)$ is located in the tails of the distribution (i.e., when $\hat{S}$ approaches 0 or remains close to 1). In these situations, the variance estimated by a binomial model approaches 0 and is therefore an underestimate of the true sampling variance. I chose to analyze survival using the staggered-entry Kaplan-Meier model in part because variance estimators exist in closed form and have been commonly used in survival analyses. The main disadvantage with this approach is that I could not incorporate individual covariates.

Last, I used the Known Fates option in Program MARK to estimate survival (White and Burnham 1999), which is equivalent to a staggered-entry Kaplan-Meier analysis of daily survival rates (Kaplan and Meier 1958, Pollock et al. 1989). However, Program MARK allowed incorporation of individual covariates into survival models, model parameterization flexibility, and model selection (White and Burnham 1999). I analyzed survival as a function of fawn age (i.e., days survived since birth), Julian date of birth, treatment status, year, fawn sex, estimated fawn mass at birth (kg), and estimated fawn hind foot length at birth (cm). I again used 182 daily survival intervals to construct encounter histories of survival from birth to 6 months of age. Survival was estimated using a logistic transformation where:

$$S_j = \frac{1}{1+e^{-[\beta_0+\beta_1(X_{1j})+\ldots+\beta_i(X_{ij})]}}.$$

The model parameters are $\beta_0, \beta_1, \ldots, \beta_i$. The sampling variance-covariance matrix of the 182 daily survival rates was then estimated using an application of the delta method (Seber 1982). Survival for the 6-month interval was estimated by the product of the 182 daily survival rates, and the variance was estimated using the delta method. I used Akaike's information criterion adjusted for sample size (AIC_c) to evaluate which variables explained measurable variation in neonatal survival (Burnham and Anderson 2002). These results were then used to select appropriately structured models for the overdispersion analysis explained below.

I measured fawn mass and hind foot length at the time of capture rather than at birth, which meant the measurements were not directly comparable across fawns. I therefore estimated fawn mass and hind foot length at birth using a regression analysis in SAS (PROC REG, SAS Version 9.1, 2003). I modeled fawn mass and hind foot length at the time of capture as a function of fawn age at capture, Julian date of birth, sex, treatment status, and year. I considered several age-at-capture variables (age, age^2, age^3) to evaluate nonlinear relationships between fawn capture mass (or hind foot length) and capture age. I performed model selection using AIC_c (Burnham and Anderson 2002). Treatment status, year, sex, age, age^2, and age^3 were important variables in the analysis of capture mass based on model weights. I found similar results for the analysis of hind foot length, except year was not important. To estimate birth mass for each neonate, I separately regressed capture mass as a function of age for each treatment × year × sex combination. Similarly, I regressed capture hind foot length as a function of age for each

treatment × sex combination. I considered 3 models in each regression: 1) mass (or hind foot length) = age, 2) mass (or hind foot length) = age + age^2, and 3) mass (or hind foot length) = age + age^2 + age^3. I then used the model-averaged beta estimates from the 3 models to estimate the y-intercept (i.e., mass or hind foot length at birth) for each fawn given its age and mass (or hind foot length) at the time of capture.

2.3.5 Overdispersion: Chi-square GOF

I initially used a chi-square goodness-of-fit (GOF) test to evaluate the distribution of mortalities among neonatal siblings and to estimate overdispersion ($\hat{c}$) for neonatal survival from birth through August. I began with this approach because it was straight-forward, it allowed a coherent summary of observed versus expected sibling fate scenarios, and it has recently been used by others (Gaillard et al. 1998, Schwartz et al. 2006, Wiens et al. 2006). For each litter size (i.e., 1, 2, or 3) within each treatment × year combination, I computed the expected number of litters where 0 fawns lived, 1 fawn lived, etc., using the overall neonatal survival rate for that treatment × year combination. Survival was calculated simply as the number of surviving fawns divided by the total number of fawns in the sample. I computed expected counts using the binomial distribution:

$$P(X = k) = \binom{n}{k} p^k (1-p)^{n-k},$$

where n = litter size, k = number of survivors, and p = survival probability for a specific treatment × year combination. I then compared my observed litter fates to the expected counts. I computed the chi-square statistic as the sum over all cells of (observed – expected)^2/expected. The technique did not work well for my data because I captured

only 2 sets of triplets and I lacked power. However, the technique provided an effective means for summarizing data in the context of sibling dependence. I did not use this approach for any other analyses because of small cell counts or too much right-censoring when considering the full analysis period.

2.3.6 Overdispersion: Bootstrap Analysis

I used data bootstrap analyses as my principal means to evaluate overdispersion. I performed all bootstrap analyses in SAS (SAS Version 9.1, 2003) following methodologies presented by Efron and Tibshirani (1993). Each fetal and neonatal survival bootstrap analysis comprised 10,000 replicate datasets generated by resampling my data with replacement. I resampled the litters of radio-collared adult does rather than individual fetuses or neonates. The number of samples drawn in each replicate equaled the number of litters (i.e., number of adult does) in the original dataset. Each replicate dataset comprised the fetuses, or neonates, associated with the resampled litters. The sample size of each replicate dataset varied according to the proportion of resampled litters comprising a single offspring versus the proportion comprising twins or triplets. In the context of the neonate analysis, litter size refers to a doe's radio-collared fawns only, which was occasionally less than the true litter size when I failed to capture 1 of a doe's fawns.

To estimate overdispersion, I performed the fetal and neonatal survival analyses described earlier on each replicate dataset using a macro function in SAS (SAS Version 9.1, 2003). I programmed the MARK analyses in SAS by generating a 1 (survived) or 0 (died) for each fawn each day it was in the sample during the analysis period. The resulting dataset comprised 28,195 1's and 0's reflecting the total number of fawn

monitoring days in my study. I analyzed this dataset using PROC LOGISTIC in SAS (SAS Version 9.1, 2003) and output the beta parameter estimates and beta variance-covariance matrix in order to compute daily survival rate and variance estimates for each treatment × year combination. I used the mean value of individual covariates when estimating survival. I estimated daily survival rates using a back-transformation of the logit link function used in PROC LOGISTIC. I estimated the sampling variance-covariance matrix of the 182 daily survival rates in SAS (PROC IML, SAS Version 9.1, 2003) using the delta method (Seber 1982). Specifically, I computed the partial derivatives of the 182 survival rate functions with respect to each of the beta parameters. I multiplied the partial derivatives matrix by the beta variance-covariance matrix, which I then multiplied by the transpose of the partial derivatives matrix. I estimated survival for the 6-month interval by taking the product of the 182 daily survival rates. I estimated variance of this survival estimate within PROC IML in SAS using another application of the delta method. Specifically, I computed the partial derivatives of the product of daily survival rates with respect to each of the individual daily survival rates. I multiplied the partial derivatives vector of daily survival rates by the daily survival variance-covariance matrix, which I then multiplied by the transpose of the partial derivatives vector.

I validated my neonatal survival models programmed in SAS by comparing my results to Program MARK output. Survival and variance estimates were identical out to 5 decimal places. Once I validated a model in SAS, I conducted the bootstrap analysis by repeating the survival analysis on 10,000 replicate datasets generated by resampling the data with replacement.

Each of my bootstrap analyses resulted in 10,000 fetal or neonatal survival estimates for each combination of treatment status and year. I then calculated the mean and standard deviation (SD) of the 10,000 survival estimates. The SD of the survival estimates provided an empirical sampling variance estimate that reflected dependence among sample units. To estimate overdispersion, I compared the SDs of replicate survival estimates with the theoretical SEs obtained from the ML analyses of my fetal and neonatal survival data. Overdispersion was indicated when the SD from the bootstrap analysis exceeded the theoretical SE. I estimated c as the ratio of the empirical (i.e., bootstrap) variance ($[SD(\hat{S})]^2$) to the theoretical variance ($[SE(\hat{S})]^2$), which is approximately chi-square distributed with 1 df. Overdispersion is indicated when $\hat{c} > 1$. I separately estimated c for each treatment-year group, and used the average of those estimates as the optimal predictor of c. As an a priori guideline, I considered $1.0 < \hat{c} \leq 1.2$ as providing only weak evidence of overdispersion, thereby reflecting the uncertainty in $\hat{c}$.

2.4 RESULTS

2.4.1 Fetal Survival

My estimates of c for fetal survival ranged from 0.678 to 1.118 and averaged 0.950 (Table 1). The highest estimate provided weak evidence of overdispersion ($\hat{c} = 1.118$), which pertained to control deer during 2004, when I observed sibling stillbirths on 3 occasions. I found a set of stillborn twins on 1 occasion, and I found 2 stillborn fetuses from triplet litters on the other 2 occasions. I also observed 3 sets of twins with only 1 stillborn fetus and a set of triplets with no stillborn fetuses among control deer during 2004. I observed no more than 2 stillborns during any other treatment-year

combination, which did not include any sibling stillbirths. As expected, fetal survival point estimates from the bootstrap analysis were nearly identical to those obtained from the ML analysis, indicating there was no bias.

2.4.2 Neonatal Survival

My estimate of c for neonatal survival from birth through August using a chi-square GOF approach was 1.4701 when including the only 2 sets of triplet neonates in my dataset and 0.9486 when excluding the 2 sets of triplets. Observed versus expected mortality distributions for different litter sizes are listed in Table 2. My estimates of c for neonatal survival from birth through August using a binomial estimator and data bootstrap ranged from 0.8680 to 1.2568 and averaged 1.0807 (Table 3).

My estimates of c for neonatal survival based on the staggered-entry Kaplan-Meier model ranged from 0.8777 to 2.433 when using the variance estimator presented by Cox and Oakes (1984) and from 1.1267 to 1.4085 when using the Greenwood (1926) variance estimator (Table 4). The highest overdispersion estimates were for treatment fawns in 2002. However, these estimates were high in part because sample sizes were small during the first daily survival interval. I captured only 5 treatment fawns ≤1 day old during 2002, of which 1 died during the first interval. The first daily survival rate was therefore estimated as 0 during numerous replicates of the bootstrap analysis. Survival during the 6-month analysis period was also estimated as 0 because it was computed as the product of daily survival rates. In result, the bootstrap variance estimate was inappropriately inflated for treatment fawns in 2002. Sample sizes were larger during 2003–2004, particularly during the first daily survival interval, resulting in more

representative estimates of overdispersion (mean $\hat{c}_{\text{Cox and Oakes}} = 1.0543$, mean $\hat{c}_{\text{Greenwood}} = 1.2195$).

My estimates of c for neonatal survival using Program MARK were based on models that included fawn age, year, treatment status, Julian date of capture, and estimated birth mass. I obtained similar estimates of c for 2 different model parameterizations incorporating these variables. The model with reduced parameterization estimated survival as a function of fawn age using a non-linear trend (no. model parameters = 13): {S(age-trend age-trend^2 age-trend^3 year treatment year×treatment capture-date birth-mass year×birth-mass)}. My estimates of c for this model ranged from 1.1128 to 1.4123 and averaged 1.2493 (Table 5). The more highly parameterized model estimated 26 weekly survival rates corresponding to fawn age (no. model parameters = 35): {S(weekly-age year treatment year×treatment capture-date birth-mass year×birth-mass)}. The corresponding estimates of c ranged from 1.1110 to 1.4291 and averaged 1.2460 (Table 6). Given the similarity in $\hat{c}$ between these models, I did not pursue more highly parameterized models. As with fetal survival, average neonatal survival point estimates from the bootstrap analyses were nearly identical to survival estimates from the ML analyses of the original data, indicating there was minimal or no bias.

2.5 DISCUSSION

2.5.1 Fetal Survival

Fetal survival in free-ranging deer populations may now be directly estimated given recent technological advancements in ultrasonography and VITs. I presented a likelihood function for estimating fetal survival when the fates of some fetuses are

unknown. I found minimal evidence of overdispersion in my fetal survival data, indicating that a variance adjustment is not necessary, at least for this particular dataset.

In biological terms, my analysis indicates that fates of sibling fetuses were largely independent even though they shared the same maternal resources. Adult doe malnutrition or disease status could explain dependence among fates of sibling fetuses (Robinette et al. 1955, Verme 1962, 1963, 1977, Eve 1981). Specifically, sibling fetuses of a malnourished or diseased doe may jointly have an increased risk of being stillborn and therefore fail to represent independent sample units. During 5 of 6 treatment-year combinations, however, I observed ≤2 stillborns total, and I did not observe any sibling stillborns. The low mortality rates overall lowered the likelihood of overdispersion being a significant issue. Even when mortality was higher (control-2004), I found minimal evidence of overdispersion. The design of future fetal survival studies should not require oversampling to accommodate sibling sample units, although I urge caution because my results are based on 1 dataset.

2.5.2 Neonatal Survival

I found evidence of limited overdispersion in my neonatal survival data. My various bootstrap analyses indicated that $1.05 \leq \hat{c} \leq 1.25$ when considering the mean $\hat{c}$ across the various treatment-year combinations and when excluding 2002 data in the staggered-entry Kaplan Meier model because of small sample size during the first survival interval. Within this general range, $\hat{c}$ varied depending on the analysis approach used. I observed minimal evidence of overdispersion with the simple binomial estimator, although this approach relied on a simplified data structure. I also found minimal evidence of overdispersion when the Cox and Oakes (1984) variance estimator was used

with the staggered-entry Kaplan-Meier model. In contrast, $\hat{c}$ was closer to 1.25 when using the Greenwood (1926) variance estimator or Program MARK, each of which provided ML estimates of variance. The lower $\hat{c}$ associated with the Cox and Oakes (1984) variance estimator can likely be explained by its relative inefficiency. The inefficiency is a result of the estimator being more robust to assumption violations of the binomial model.

My results suggest that previously reported variance estimates of mule deer neonatal survival are likely adequate, particularly those based on the Cox and Oakes (1984) variance estimator. My sample likely comprised a higher proportion of siblings than neonatal datasets in past studies because I employed VITs. Thus, I expect my estimates of overdispersion to be on the high end when compared to past research. The design of future neonatal survival studies should consider a slight inflation of sample sizes to accommodate potential overdispersion (i.e., $\hat{c} \cong 1.25$). However, overdispersion should be assessed in additional mule deer neonatal datasets before any general conclusions are drawn. The potential for overdispersed data may increase in the future as VITs facilitate the routine capture of siblings.

Biological mechanisms underlying observed dependence among neonatal siblings include dam condition and predation. Similar to my discussion of fetal dependence, one might expect sibling neonates born of a malnourished or diseased doe to lack independence because each should have an increased probability of dying from starvation or disease (Verme 1962, 1977, Eve 1981, Sams et al. 1996). Sibling neonates born of a particularly healthy doe might lack independence because each should have an increased probability of survival. Gaillard et al. (1998) observed dependence of fates among

sibling roe deer (*Capreolus capreolus*) neonates in France and Norway during unfavorable years (i.e., poor summer deer survival or high population density), but not during favorable years. They discussed that maternal characteristics likely had a greater influence on neonatal survival during unfavorable years. In my study of mule deer, I observed limited evidence of overdispersion seemingly irrespective of maternal condition. Estimated body fat of treatment does was 11.8% (SE = 0.410) whereas estimated fat of control does was 7.1% (SE = 0.174) in late February (Chapter 3). My treatment simulated optimum habitat conditions for deer whereas my control was representative of prevailing conditions. Although fawns born from treatment does experienced overall higher survival than fawns born from control does, my treatment and control neonatal survival datasets provided roughly similar evidence of overdispersion (Tables 4, 5, 6).

I observed 1 set of twin neonates where both fawns died of starvation. Similarly, I observed 1 set of twins where 1 fetus was stillborn and the other died of starvation a few days after birth. Aside from these examples, and perhaps some evidence of twin sets surviving more often than expected, sibling neonate fates appeared to be mostly independent of one another from the standpoint of maternal characteristics. A possible explanation is that the death of 1 sibling might actually enhance the survival probability of the remaining sibling(s) because of reduced competition for the available milk supply (Smale et al. 1995, Drummond et al. 2000).

Predation can cause dependence of neonatal sibling fates if the same predator kills each sibling, or if sibling neonates are in a location especially vulnerable to predation. I observed evidence indicating a black bear killed triplet neonates in a single episode. I

observed evidence indicating coyotes killed 3 sets of twins over the course of the study, 2 of which were located near coyote dens. In each case, the twin neonate was killed 1–7 days after its sibling was killed. I also observed predation of 2 neonates only a few days before or after their siblings died of starvation or illness, which could represent partially dependent fates if the neonates were especially vulnerable to predation given their poor condition. However, all other predation events involved only 1 fawn and did not appear to influence the fate of the fawn's sibling(s). These predation observations are consistent with my results indicating modest evidence of sibling dependence. A logical explanation for limited dependence with respect to predation is that does commonly bed their fawns some distance apart (White et al. 1972, Lent 1974, Ozoga et al. 1982, Schwede et al. 1994). Sibling isolation during the first weeks of life is considered to be a behavioral strategy adopted specifically to prevent predators from finding more than 1 fawn (Schwede et al. 1994). This behavioral pattern therefore acts to minimize sibling dependence with respect to predation, which is significant in my study because 60.6% of fawn mortalities were caused by predation. Gaillard et al. (1998) studied roe deer populations that did not have natural predators, which may largely explain why they observed stronger evidence of overdispersion in neonatal survival than I did.

2.6 MANAGEMENT IMPLICATIONS

Technology now facilitates direct measurements of fetal survival in free-ranging deer, and I provide quantitative methods for the analysis of fetal survival data. It was reasonable to treat sibling fetuses as independent sample units in this study. However, the degree of sibling dependence among deer fetuses should be expected to vary by circumstance. For neonatal survival, I recommend a slight variance adjustment to

accommodate modest overdispersion (i.e., $\hat{c} \cong 1.25$) when computing sample size requirements, particularly if the sample will comprise a high proportion of siblings. This would then allow a corresponding $\hat{c}$ adjustment in subsequent quasi-likelihood model selection analyses using Akaike's information criterion ($QAIC_c$). Additional overdispersion analyses of neonatal survival data are needed to either support or contradict my recommendation. While assessment of overdispersion has direct quantitative relevance, it also elucidates important biological relationships. Specifically, the fates of sibling mule deer neonates may often be independent even though they have the same dam and use the environment similarly. Dam-neonate behavior facilitates independence of sibling fates with respect to predation, although I documented several cases of predators killing siblings. The effect of maternal condition on sibling fate relationships warrants further study because it is not straightforward and may partially depend on the prevalence of predation.

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Table 1. Overdispersion estimates ($\hat{c}$) of mule deer fetal survival reflecting sibling dependence in southwest Colorado, 2002–2004. Survival was estimated using a maximum likelihood (ML) analysis and a data bootstrap analysis, and overdispersion was estimated as the ratio of variance estimates of the 2 survival estimates.

			MLE		Bootstrap analysis		
Year	Group	n	$\hat{S}$	SE($\hat{S}$)	Avg $\hat{S}$	SD($\hat{S}$)	$\hat{c}$
2002	Control	33	0.8577	0.11190	0.8506	0.11446	1.0462
	Treatment	24	0.8016	0.15390	0.8060	0.12676	0.6784
2003	Control	44	0.9622	0.03821	0.9635	0.03784	0.9810
	Treatment	38	0.9639	0.03601	0.9645	0.03467	0.9271
2004	Control	59	0.7560	0.09007	0.7612	0.09524	1.1180
	Treatment	57	1.0000	0.00000	1.0000	0.00000	---

Table 2. Observed versus expected distributions of neonatal mule deer mortalities for different sizes of marked litters in southwest Colorado, 2002–2004. Expected values were computed from the binomial distribution separately for each treatment × year combination and then summed across treatment × year combinations.

Marked Litter Size[a]	No. Surviving Fawns	Observed No. Litters	Expected No. Litters
1	0	35	31.43
	1	54	57.57
2	0	12	11.69
	1	31	37.78
	2	42	35.53
3	0	1	0.13
	1	1	0.58
	2	0	0.86
	3	0	0.43

[a]Marked litter size refers to the number of newborn fawns captured and marked from an adult doe. In some cases, primarily for litters of size 1, the actual litter size was larger (2 or 3) but I was unable to mark each of the doe's fawns.

Table 3. Overdispersion estimates ($\hat{c}$) of mule deer neonatal survival reflecting sibling dependence from birth through August in southwest Colorado, 2002–2004. Survival was estimated using a binomial estimator and a data bootstrap analysis, and overdispersion was estimated as the ratio of variance estimates of the 2 survival estimates.

			Binomial estimator		Bootstrap analysis		
Year	Group	n	$\hat{S}$	SE($\hat{S}$)	Avg $\hat{S}$	SD($\hat{S}$)	$\hat{c}$
2002	Control	23	0.5652	0.10569	0.5658	0.11565	1.1974
	Treatment	30	0.7000	0.08510	0.7008	0.08192	0.9267
2003	Control	45	0.6222	0.07309	0.6201	0.07330	1.0058
	Treatment	55	0.8000	0.05443	0.7995	0.06035	1.2294
2004	Control	49	0.5714	0.07143	0.5729	0.06655	0.8680
	Treatment	63	0.5714	0.06285	0.5713	0.07046	1.2568

Table 4. Overdispersion estimates ($\hat{c}$) of mule deer neonatal survival reflecting sibling dependence from birth to 6 months old in southwest Colorado, 2002–2004. Survival was estimated using a staggered-entry Kaplan-Meier model and a data bootstrap analysis, and overdispersion was estimated as the ratio of variance estimates of the 2 survival estimates.

			Kaplan-Meier estimator			Bootstrap analysis			
Year	Group	n	$\hat{S}$	SE($\hat{S}$)[a]	SE($\hat{S}$)[b]	Avg $\hat{S}$	SD($\hat{S}$)	$\hat{c}^{a,c}$	$\hat{c}^{b,c}$
2002	Control	23	0.4535	0.10108	0.11040	0.4531	0.12413	1.5079	1.2642
	Treatment	30	0.5170	0.10372	0.13631	0.5099	0.16177	2.4326	1.4085
2003	Control	45	0.4264	0.08630	0.08595	0.4198	0.10040	1.3535	1.3647
	Treatment	55	0.5994	0.08088	0.07285	0.5968	0.07765	0.9217	1.1362
2004	Control	49	0.3963	0.08540	0.07538	0.3978	0.08001	0.8777	1.1267
	Treatment	63	0.4191	0.06810	0.06283	0.4168	0.07025	1.0642	1.2503

[a]Variance and overdispersion estimates were calculated using the variance estimator presented by Cox and Oakes (1984:51).

[b]Variance and overdispersion estimates were calculated using the variance estimator developed by Greenwood (1926) and later presented by Cox and Oakes (1984:51).

[c]Small sample size and a mortality among treatment neonates during the first daily survival interval in 2002 caused the bootstrap analysis to incorrectly estimate $\hat{S} = 0$ during numerous replications, which inflated the bootstrap variance. Therefore, overdispersion estimates for treatment deer during 2002 are not necessarily representative of neonatal sibling dependence.

Table 5. Overdispersion estimates ($\hat{c}$) of mule deer neonatal survival reflecting sibling dependence from birth to 6 months old in southwest Colorado, 2002–2004. Survival was estimated as a function of a non-linear trend in fawn age, year, a nutrition treatment, year × treatment interaction, capture date, birth mass, and year × birth mass interaction (no. parameters = 13). Survival was estimated using maximum likelihood (ML) followed by a data bootstrap analysis, and overdispersion was estimated as the ratio of variance estimates of the 2 survival estimates.

			ML		Bootstrap analysis		
Year	Group	n	$\hat{S}$	SE($\hat{S}$)	Avg $\hat{S}$	SD($\hat{S}$)	$\hat{c}$
2002	Control	23	0.5183	0.11433	0.5230	0.13103	1.3134
	Treatment	30	0.7364	0.09385	0.7287	0.11154	1.4123
2003	Control	45	0.4649	0.07631	0.4625	0.08426	1.2193
	Treatment	55	0.6121	0.06931	0.6135	0.07311	1.1128
2004	Control	49	0.4255	0.07296	0.4271	0.07722	1.1202
	Treatment	63	0.4025	0.06377	0.4035	0.07320	1.3178

Table 6. Overdispersion estimates ($\hat{c}$) of mule deer neonatal survival reflecting sibling dependence from birth to 6 months old in southwest Colorado, 2002–2004. Survival was estimated as a function of weekly fawn age, year, a nutrition treatment, year × treatment interaction, capture date, birth mass, and year × birth mass interaction (no. parameters = 35). Survival was estimated using maximum likelihood (ML) followed by a data bootstrap analysis, and overdispersion was estimated as the ratio of variance estimates of the 2 survival estimates.

			ML		Bootstrap analysis		
Year	Group	n	$\hat{S}$	SE($\hat{S}$)	Avg $\hat{S}$	SD($\hat{S}$)	$\hat{c}$
2002	Control	23	0.5201	0.11417	0.5259	0.12989	1.2943
	Treatment	30	0.7406	0.09306	0.7349	0.11125	1.4291
2003	Control	45	0.4685	0.07623	0.4672	0.08375	1.2070
	Treatment	55	0.6146	0.06902	0.6153	0.07275	1.1110
2004	Control	49	0.4285	0.07292	0.4310	0.07734	1.1249
	Treatment	63	0.4069	0.06374	0.4072	0.07295	1.3098

CHAPTER 3

EFFECT OF ENHANCED NUTRITION ON MULE DEER POPULATION RATE OF CHANGE

Abstract: Concerns over declining mule deer (*Odocoileus hemionus*) populations during the 1990s prompted research efforts to identify and understand key limiting factors of deer. Similar to past deer declines, a top priority of state wildlife agencies was to evaluate the relative importance of habitat and predation. I therefore evaluated the effect of enhanced nutrition of deer during winter and spring on fecundity and survival rates using a life table response experiment involving free-ranging mule deer on the Uncompahgre Plateau in southwest Colorado. The nutrition enhancement treatment represented an instantaneous increase in nutritional carrying capacity of a pinyon (*Pinus edulis*) and Utah juniper (*Juniperus osteosperma*) winter range, and was intended to simulate optimum habitat quality. Prior studies on the Uncompahgre Plateau indicated predation and disease were the most common proximate causes of deer mortality. By manipulating nutrition and leaving predation as it was, I determined whether habitat quality was ultimately a critical limiting factor of the deer population. I measured fetal, neonatal, and overwinter fawn survival, and annual adult doe survival, which I then used to estimate population rate of change as a function of enhanced nutrition. Pregnancy and fetal rates were high for all deer, regardless of the nutrition treatment. Fetal and neonatal survival rates were higher among deer that received the nutrition enhancement treatment than deer that served as experimental controls. Overwinter fawn survival was

considerably higher among treatment deer than control deer. Overwinter survival increased by 0.16–0.31 depending on year and fawn sex, and none of the 95% confidence intervals associated with the effect overlapped 0. The nutrition enhancement treatment increased survival of fetuses to the yearling age class by 0.14–0.20 depending on year and fawn sex, although 95% confidence intervals slightly overlapped 0. The nutrition treatment also had a positive effect on annual adult doe survival. Survival of does receiving the treatment ($\hat{S}$ = 0.879, SE = 0.0206) was higher than survival of control does ($\hat{S}$ = 0.833, SE = 0.0253). My estimate of the population rate of change, $\hat{\lambda}$, was 1.165 (SE = 0.0358) for treatment deer and 1.033 (SE = 0.0380) for control deer. The treatment caused $\hat{\lambda}$ to increase by 0.133 (SE = 0.0428). I documented density dependence in the Uncompahgre deer population because survival of fawns and does increased considerably in response to enhanced nutrition. I found strong evidence that coyote (*Canis latrans*) predation of ≥6-month-old fawns and adult does was compensatory. My results demonstrate that observed coyote predation is not useful for evaluating whether coyotes are negatively impacting a deer population. I also found evidence that mountain lion (*Puma concolor*) predation was compensatory. Disease mortality was not compensatory among adult does. I found that winter range habitat quality was a limiting factor of the Uncompahgre Plateau mule deer population. Therefore, I recommend evaluating habitat treatments for deer that are designed to set back succession and increase productivity of late-seral pinyon-juniper habitats that presently dominate the winter range.

Key Words: carrying capacity, Colorado, compensatory mortality, coyote predation, density dependence, fecundity, fetal survival, habitat quality, lambda, life table response

experiment, mountain lion predation, mule deer, nutrition, neonatal survival, *Odocoileus hemionus*, overwinter fawn survival, pinyon-juniper.

3.1 INTRODUCTION

Mule deer (*Odocoileus hemionus*) populations apparently declined during the 1990s across much of the West, and present numbers are well below the peak population levels documented during the 1940s–1960s (Unsworth et al. 1999, Gill et al. 2001, Heffelfinger and Messmer 2003). An understanding of limiting factors is necessary to understand why populations may have declined, and to guide management efforts aimed at increasing deer numbers (Gill et al. 2001, de Vos et al. 2003). Limiting factors of mule deer are difficult to understand because they are numerous, interacting, and subject to variability. Climatic variation can cause wide population fluctuations, and may often be the primary reason for observed changes, yet managers are frequently concerned with factors that can be manipulated through management actions. Predation and habitat have typically received the most attention from wildlife agency administrators, biologists, and sportsmen alike. Predation is a concern because it is routinely identified as the most common proximate cause of deer mortality. Habitat quality is believed to have declined across much of the West because of altered fire regimes and associated plant successional changes, invasion of noxious weeds, overgrazing, energy development, and habitat loss caused by urban development (Lutz et al. 2003, Watkins et al. 2007).

Identification of the principal limiting factor(s) is necessary to make informed management decisions. Some mule deer populations may be driven by extreme environmental variation that is primarily density-independent, in which case the preferred

management strategy may be to monitor populations, or perhaps climate variables, and make responsive harvest decisions (Mackie et al. 1998). However, in less-variable environments, determining whether habitat or predation is most limiting has significant management implications because the 2 factors represent very different limitation scenarios.

The relationship between habitat quality and deer population size is heavily rooted in density dependence theory (McCullough 1979). As populations approach or exceed nutritional carrying capacity (NCC) of a given environment, fecundity and survival are expected to decline. Nutritional carrying capacity refers to the number of animals that can be supported on a specified landscape given animal nutrient requirements relative to nutrient availability (McLeod 1997). Density dependent effects have been demonstrated in body condition (Gaillard et al. 1996, Stewart et al. 2005, Kjellander et al. 2006), fecundity (Clutton-Brock et al. 1987, Stewart et al. 2005), and survival (Clutton-Brock et al. 1987, Bartmann et al. 1992, Singer et al. 1997, White and Bartmann 1998) of ungulates. If a population is limited by NCC and demonstrating density dependent feedback, wildlife managers have 2 main options for improving fawn production and survival. One option is to alter adult doe harvest strategies in an attempt to increase fawn production and survival. Under this option, the management goal is to optimize age and sex ratios as a way to increase the number of bucks available for harvest (McCullough 1979, 2001). A second option is to improve habitat quality for deer with the goal of increasing total deer numbers.

Predation is a concern when deer populations are below NCC, because it will more likely be a source of additive mortality (Ballard et al. 2001). If a population is

limited by predation, wildlife managers should pursue management options different than those mentioned above. First, doe harvest should be minimized, or at least conservatively managed, to maximize production and survival of young. Second, predator control or liberalized harvest of predator species may be considered as ways to lessen mortality and increase deer numbers. Habitat treatments and predator control can be costly in terms of both economic and social capital. Neither option should be pursued without adequate justification.

To determine the importance of different limiting factors, a specific effect must be isolated, often in the context of considerable background variation (i.e., process variance). The relative importance of habitat quality versus predation can be ascertained by manipulating one factor and leaving the other alone in a field experiment. If habitat quality is ultimately limiting the deer population, such that further population growth is restricted by NCC, then I would expect observed predation to have minimal effect on population growth (Bartmann et al. 1992, Ballard et al. 2001). In contrast, if the population is below NCC, and predation is a common proximate mortality cause, I might expect some threshold of predator removal to cause an increase in the deer population. Ideally, 2 field experiments should be conducted: one that manipulates predation and one that manipulates habitat. Hurley and Zager (2006) conducted an intensive predator control study in southeast Idaho. They measured deer population parameters in response to coyote (*Canis latrans*) and mountain lion (*Puma concolor*) reductions. I complemented their research by manipulating deer nutrition while not manipulating coyote and mountain lion predation.

I studied a deer population in southwest Colorado that had declined prior to my research (Watkins et al. 2001). I hypothesized that poor habitat quality on winter range, and possibly disease, had contributed to the decline, which was largely caused by reduced December fawn recruitment (Watkins et al. 2001, Pojar and Bowden 2004). Winter range habitat predominantly comprised late-seral pinyon (*Pinus edulis*) and Utah juniper (*Juniperus osteosperma*) woodlands with minimal understory vegetation and limited species diversity. Predation by coyotes and mountain lions was presented as a competing hypothesis as to why the deer population declined. I implemented an instantaneous increase in NCC of winter range habitat and measured a series of deer population responses in the context of a life table response experiment (LTRE, Caswell 2001). I did not manipulate predator numbers or any other potential limiting factor, and I conducted the entire study with free-ranging mule deer. I increased NCC by enhancing deer nutrition using supplemental pellets, which was intended to simulate optimum habitat quality from a nutritional standpoint. I opted not to use mechanical treatments or prescribed fire because the treatments could have failed to effectively increase NCC, making it impossible to determine the relative importance of habitat quality and predation. Additionally, I did not want to study the effectiveness of habitat treatment strategies until after I determined whether habitat was indeed limiting.

My research objective was to evaluate the effect of enhanced nutrition on a mule deer population using a LTRE (Caswell 2001). Specifically, I evaluated the effect of enhanced nutrition on pregnancy rates and numbers of fetuses produced; fetal, neonatal, and overwinter fawn survival; and annual adult doe survival. I then used these estimates to quantify the effect of enhanced nutrition on population rate of change. My ultimate

goal was to determine whether habitat was limiting a deer population in which predation was the most common proximate mortality factor.

3.2 STUDY AREA

I conducted my research in southwest Colorado on the southern half of the Uncompahgre Plateau and in the adjacent San Juan Mountains (Fig. 1). My winter range study area comprised 2 sites, or experimental units (EUs). The Colona EU (38°21'N, 107°49'W) received a nutrition enhancement treatment during 2000–2002 and the Shavano EU (38°27'N, 108°01'W) served as a control. I then reversed the treatment-control designations during 2002–2004 consistent with a crossover experimental design (Fig. 2). I selected the EUs based on several criteria. First, I selected EUs that were separated by ≥15 linear km to prevent individual deer from occupying more than one EU. Second, I restricted the size of EUs to roughly 15 km^2 to lessen logistical constraints associated with daily delivery of the nutrition enhancement treatment. Third, I selected EUs with relatively high deer densities (i.e., >30 deer/km^2, B. E. Watkins, Colorado Division of Wildlife, unpublished data) so that I could achieve sample size objectives. Finally, I selected EUs that comprised similar habitats with relatively low numbers of wintering elk (i.e., <50 elk in a normal winter).

I studied free-ranging deer and therefore EU size was not static. I defined the core of each EU as the area that received the nutrition treatment and contained roughly 90% of the radio-collared deer captured in that unit. The core of the Colona EU was 7 km^2 when it received the treatment during 2000–2002. However, during 2002–2004, I expanded the core area to 12 km^2 in response to periodic shifts in deer distribution, which was necessary to achieve sample size objectives. The core of the Shavano EU was 22

km^2 throughout the study. Each EU encompassed approximately 40 km^2 when considering all radio-collared deer, ranging in elevation from 1,830 m to 2,290 m.

Winter range EUs were comprised of pinyon and Utah juniper woodlands with interspersed big sagebrush (*Artemisia tridentata*) adjacent to irrigated agricultural fields. During my study, annual precipitation averaged 22.3 cm and the minimum temperature in January averaged –8.2° C in Montrose, Colorado (Western Regional Climate Center [WRCC] 2005), which is 60 m below the lowest winter range elevation in either EU. Deer occupied the winter range EUs from November through April each year. Estimated deer densities typically varied between 31 deer/km^2 and 59 deer/km^2 in the core of each EU during the study, with densities periodically reaching 85 deer/km^2 in portions of an EU (C. J. Bishop, Colorado Division of Wildlife, unpublished data).

I defined summer range based on migratory movements of radio-collared deer captured in the winter range EUs. Summer range for 95% of the radio-collared deer covered 2,500 km^2, whereas the total summer range encompassed approximately 4,000 km^2 between 37°49' and 38°28'N latitude and 107°26'and 108°17'W longitude. Elevations ranged from 1,830 m to 3,500 m, with a majority of deer summering between 2,600 m and 3,000 m. Radio-collared deer from the 2 winter range EUs were intermixed throughout most of the summer range, lessening any potential confounding of summer range habitat use on the effect of the winter range nutrition treatment (Fig. 3). The notable exception was an area directly southwest of the Shavano EU, which was used exclusively by deer from the Shavano EU.

Dominant summer range habitat types, from lower to higher elevations, were pinyon–juniper, Gambel oak (*Quercus gambelii*), ponderosa pine (*Pinus ponderosa*), big

sagebrush, aspen (*Populus tremuloides*), and mixed forests of Engelmann spruce (*Picea engelmannii*) and subalpine fir (*Abies lasiocarpa*). Diverse habitat mosaics occurred at interfaces of each of the major habitat types. Snowberry (*Symphoricarpos* spp.) was a common understory shrub in Gambel oak, ponderosa pine, and aspen habitats, and it occasionally occurred in sagebrush habitats. Annual precipitation averaged 57.4 cm and the maximum temperature in July averaged 26.7° C at a weather station in the summer range situated at 2,438 m elevation (WRCC 2005).

3.3 METHODS

3.3.1 Treatment

I enhanced nutrition of deer in the treatment EU from early-mid December through April each year by providing a pelleted supplemental feed. The supplement was developed through testing with both captive and wild deer and has been safely used in applied research and management (Baker and Hobbs 1985, Baker et al. 1998). Pellets were distributed daily from 22.7 kg bags using pickup trucks, all-terrain vehicles (ATVs), and snowmobiles on primitive roads throughout the EU. Each bag of pellets was distributed in approximately 20–25 small piles in a linear fashion. I spread pellets throughout the entire EU to minimize animal concentrations and to prevent dominant animals from restricting fawn access to the feed. I generally supplied pellets ad libitum such that residual pellets remained throughout the EU when the next day's ration was provided. This required the distribution of 800–2000 kg of feed per day, depending on number of elk present, weather, and availability of natural forage. My approach typically allowed all ages and sexes of deer unlimited access to the supplement. I documented deer use of the feed using visual observations and daily monitoring of radio-collared deer.

I recorded 1,957 visual observations of radio-collared deer consuming the supplement during the study.

The pelleted ration was commercially produced in the form of 2×1×0.5-cm wafers (Baker and Hobbs 1985) by Ranch-Way Feed Mills (Fort Collins, CO). Feed quality (e.g., digestibility, protein) greatly exceeded that of typical winter range deer diets; exact constituent values were provided by Baker et al. (1998). When provided ad libitum, the feed should have allowed deer to meet or exceed maintenance nutritional requirements during winter (Ullrey et al. 1967, Thompson et al. 1973, Smith et al. 1975, Baker et al. 1979, Holter et al. 1979, Swift 1983). My intent was not to determine the exact level of nutritional enhancement necessary to effect a change in fecundity or survival, but rather to determine if nutrition was a significant factor limiting fawn recruitment in a declining population where predation and disease were common proximate mortality factors.

3.3.2 Response Variables

December fawn recruitment on the Uncompahgre Plateau had been declining prior to my research (Watkins et al. 2001). I hypothesized that deteriorating winter range habitat quality caused adult doe body condition to decline, which in turn had a negative effect on fawn production and survival. I therefore planned to use December fawn:doe ratios as a response variable to reflect fecundity and neonatal survival. However, I struggled to measure fawn:doe ratios with desired precision and without bias (Bishop et al. 2005*b*). High deer densities and heavy cover in combination with the small size of EUs contributed to the problem of measuring age ratios adequately. In response, I measured fecundity and survival rates directly beginning in year 2 of the study. Specifically, I measured adult doe pregnancy and fetal rates (Feb), fetal survival

(Feb–Jun), neonatal survival (Jun–Dec), and overwinter fawn survival (Dec–Jun). Pregnancy rate is defined here as the proportion of adult does having ≥1 fetus in utero during late February, and fetal rate is defined as the mean number of fetuses per pregnant adult doe during late February. Fetal survival refers to the survival rate of fetuses in utero from February to birth. I evaluated the effect of the treatment on fawn production and survival exclusively using the direct measures of fecundity and survival rates; I did not use fawn:doe ratios because of the aforementioned problems. I also measured annual adult doe survival each year of the study. I then used each of the fecundity and survival parameters in a matrix population model to quantify the population rate of change, λ (Caswell 2001).

3.3.3 Sample Size Objectives

My initial objective was to maintain ≥40 radio-collared adult does in each EU throughout the study to facilitate measurement of December fawn:doe ratios in response to the nutrition treatment (Bishop et al. 2005*b*). During 2002–2004, I captured additional adult does during late February and early March to allow measurement of fetal and neonatal survival in response to the treatment. In 2002, I based adult doe sample size on an evaluation of vaginal implant transmitters (VITs) for capturing newborn fawns exclusively from treatment and control radio-collared does (Chapter 1). During 2003 and 2004, I based adult doe sample sizes on the number of does needed to achieve target samples of fetuses and neonates. Adult doe, fetus, and neonate samples were interdependent because all fetuses and neonates used in my study were offspring of radio-collared does. I had difficulty determining fetus sample size requirements because of uncertainty in identifying fetus fates, and I was not aware of previous fetal survival

studies to inform calculations. I therefore based my sample size calculations on quantifying neonatal survival because it was my highest priority and I was able to generate reliable estimates.

I desired to detect a difference in neonatal survival of ≥0.15 between experimental groups (EGs). Experimental group refers to deer that directly (does and ≥6-month-old fawns) or indirectly (fetuses and neonates) received the treatment (treatment EG) or did not receive the treatment (control EG). For example, the treatment EG included neonatal fawns born from radio-collared does that occupied the treatment EU the previous winter. A sample size of 40 neonates per EG per year provided power of 0.81 to detect a difference of 0.15 in survival between treatment and control fawns, assuming survival of control fawns was 0.40. I assumed a control survival rate of 0.40 based on previous neonatal survival rates measured on the Uncompahgre Plateau (Pojar and Bowden 2004) and December fawn:doe ratios measured during the late 1980s and 1990s, when the Uncompahgre population declined (Watkins et al. 2001). I determined that 60 radio-collared does (30 treatment and 30 control) equipped with VITs would be necessary to capture a minimum of 80 newborn fawns (Bishop et al. 2002, Chapter 1). I also assumed that I would capture some fawns from treatment and control radio-collared does that did not receive VITs.

My target sample size for estimating overwinter fawn survival was the same as my sample size for estimating neonatal survival (n = 40 fawns/EG/yr). I expected overwinter fawn survival to increase in response to the treatment by approximately 0.15 because this was the difference measured in a density reduction experiment conducted by White and Bartmann (1998) in northwest Colorado. I assumed a control survival rate of

0.40 based on long-term data from Colorado, Idaho, and Montana (Unsworth et al. 1999). However, data from 5 deer populations across Colorado indicated that overwinter fawn survival was typically ≥0.65 during my study (Colorado Division of Wildlife, unpublished data).

3.3.4 Capture, Handling, and Radio-marked Samples

I captured 139 adult does during 20 November–14 December, 2000–2003, and 241 6-month-old fawns during 20 November–19 December, 2001–2003, using baited drop nets (Ramsey 1968, Schmidt et al. 1978) and helicopter net-gunning (Barrett et al. 1982, van Reenen 1982). I captured 154 pregnant adult does (including 19 recaptures) during 26 February–2 March, 2002–2004, using helicopter net-gunning (2002: $n = 36$, 2003: $n = 58$, 2004: $n = 60$). All deer were hobbled and blind-folded prior to handling. During drop-net captures, stretchers were used to carry deer away from nets prior to release. During net-gun captures, deer were ferried ≤3.5 km by the helicopter to a central processing location.

I fitted deer with vinyl-belted radio collars equipped with mortality sensors (Lotek, Inc., Newmarket, ON, Canada; Advanced Telemetry Systems, Inc., Isanti, MN), which activated after remaining motionless for 4 hours. I permanently attached radio collars on all adult does; thus, many of the does were present in multiple years' samples. I temporarily attached radio collars on 6-month-old fawns by cutting the collar belting in half and reattaching the two ends using rubber surgical tubing. Fawns shed the collars ≥6 months post-capture. I stitched neck band material (Ritchey Mfg. Co., Brighton, CO) to the left side of each radio collar, which I engraved with a unique marking for visually identifying deer. I measured mass (kg), hind foot length (cm), and chest girth (cm) of

each deer and estimated deer age using tooth replacement and wear (Severinghaus 1949, Robinette et al. 1957, Hamlin et al. 2000).

During captures in February–March, I measured maximum subcutaneous fat thickness on the rump (cm) and thickness of the longissimus dorsi muscle (cm) of each doe using a SonoVet 2000 portable ultrasound unit (Universal Medical Systems, Bedford Hills, NY) with a 5 MHz linear transducer (Stephenson et al. 1998, 2002; Cook et al. 2001). A small area of hair was plucked at each measurement point and lubricant was used to enhance contact between the transducer and skin. I determined a body condition score (BCS) for each deer by palpating the rump (Cook et al. 2001, 2007). I combined ultrasound measurements with the BCS score to estimate body fat of each deer (Cook et al. 2007).

During captures in February–March, I also established pregnancy status and measured fetal rates of each doe by performing transabdominal ultrasonography using an Aloka 210 portable ultrasound unit (Aloka, Inc., Wallinford, Conn.) with a 3-MHz linear transducer (Stephenson et al. 1995). The left caudal abdomen was shaved from the last rib and lubricant was applied to facilitate transabdominal scanning. I was unable to obtain accurate fetal counts for 9 does, which I excluded from the fetal sample. I also excluded fetuses from 5 does that died prior to giving birth and from 2 does that I could not locate following spring migration. My resulting fetal sample comprised 255 fetuses from 138 does (29 does with 1 fetus, 101 does with 2 fetuses, 8 does with 3 fetuses).

I fitted each pregnant deer with a VIT (Advanced Telemetry Systems, Inc., Isanti, MN) and released non-pregnant does without a radio collar or VIT. I performed the ultrasound and VIT insertion procedures in a 4.3 × 4.9-m wall-frame tent to minimize

disturbance from helicopter rotor wash and adverse weather conditions and to create a dim environment to facilitate ultrasonography. My VITs had a temperature-sensitive switch that caused them to increase pulse rates from 40 pulses to 80 pulses per minute when the temperature dropped below 32° C. A temperature drop below 32° C was indicative of the VIT being expelled from the deer. I used VITs as an aid to determine the adult does' times and locations of birth the following June. I provided a detailed description of VITs and VIT insertion procedures in Chapter 1.

I located each of the adult does with VITs using aerial telemetry every 2–3 weeks during March–May and every morning during June. When a VIT was detected with a fast (i.e., postpartum) pulse rate, very high frequency (VHF) receivers and directional antennae were used from the ground to simultaneously locate the VIT and radio-collared doe, which were typically in proximity to one another. I attempted to account for each doe's fetus(es) as live or stillborn fawns to quantify in utero fetal survival from February to birth. I assumed that no fetuses were resorbed, which is a reasonable assumption for mule deer (Robinette et al. 1955, Medin 1976, Carpenter et al. 1984). I classified each fawn found dead at a birth site as stillborn unless evidence was present to suggest the fawn was born alive. In most cases, I confirmed that the fawn had died before birth via laboratory necropsy. Most radio-collared does, that did not receive VITs, were located from the ground approximately every other day during June, and doe behavior and searches in the vicinity of the doe were used to locate neonates. The same procedure was used for any VIT doe whose implant failed because of premature expulsion or battery failure. Unsuccessful neonate searches were usually terminated 30–45 minutes following

the initial location of the radio-collared doe, although search times occasionally lasted an hour in heavy cover.

I captured and radiocollared 276 neonates born from radiocollared does during 4 June–8 July, 2002–2004 (2002: $n = 54$, 2003: $n = 103$, 2004: $n = 119$). I removed 6 fawns from the sample because of possible capture-related abandonment or injury, resulting in a total sample of 270 radio-collared neonates from 178 radio-collared does (88 does with 1 marked fawn, 88 does with 2 marked fawns, 2 does with 3 marked fawns). Surgical gloves were worn when securing and handling neonates to help minimize transfer of human scent. I captured 75% of the neonates in my sample within 2 days of birth. These neonates were secured and handled with little or no effort because they rarely attempted to run or resist handling. A short chase was occasionally required to capture older neonates, which often struggled during handling. A drop-off radio collar with a 2-hour mortality sensor (Advanced Telemetry Systems, Inc., Isanti, MN) was placed on each captured neonate. Radiocollars were constructed with elastic neck-band material to facilitate expansion. Hole-punched, vinyl-belting tabs extended from the end of the elastic and from the transmitter for attachment purposes. I made collars temporary by cutting the vinyl tab extending from the elastic and reattaching the belting with latex tubing, which generally caused the collars to shed from the animal >6 months post-capture. I right-censored 46 neonates that shed their collars prematurely in association with fences during fall migration, typically 4–5 months post-capture.

I recorded mass (kg), hind foot length (cm), age (days), and sex of each captured neonate. Neonates were placed in a bag to measure mass. Neonate age was estimated primarily based on radio-monitoring of the adult does, and secondarily based on hoof

characteristics, condition of the umbilical cord, pelage, and behavior (Haugen and Speake 1958, Robinette et al. 1973, Sams et al. 1996, Pojar and Bowden 2004). Daily monitoring of does with functioning VITs allowed us to determine specific dates of birth, and monitoring of other radio-collared does often allowed us to identify dates of birth within a 1–2 day period. Handling times were roughly 5 minutes per fawn. All deer capture and handling procedures, including VIT techniques, were approved by the Colorado Division of Wildlife's Animal Care and Use Committee (project protocols 11–2000 and 1–2002).

3.3.5 Monitoring and Cause-Specific Mortality

I radio-monitored deer daily from the ground, and approximately biweekly from the air, throughout the study to determine fates and mortality causes. I detected signals daily from all radio-collared neonates during the summer and fall and from most radio-collared deer during winter, which typically allowed mortalities to be retrieved within 24 hours of the mortality event. During summer and migration periods, approximately 15–25% of adult and yearling deer could not be ground-monitored on a routine basis. I therefore failed to detect some deer mortalities for several days, or on occasion, for one or more weeks.

When I located a deer mortality in the field, I conducted a thorough site inspection to record tracks, scat, drag trails, blood, hair, and any other signs that could help determine the cause of death. I then collected the carcass or performed a field necropsy on site. I collected and submitted all fresh, intact neonate carcasses to the Colorado Division of Wildlife's Wildlife Health Laboratory (Fort Collins, CO) or the Colorado State University Diagnostic Laboratory (Fort Collins, CO) for necropsy. I also

submitted fresh, intact adult and 6-month-old fawn carcasses to the laboratory for necropsy when logistically feasible. During laboratory necropsies, various tissue samples were extracted for bacteriology, virology, polymerase chain reaction (PCR), and virus isolation. I performed field necropsies on all other deer mortalities when at least some portion of the carcass was present. When feasible, I collected and submitted heart, lung, liver, kidney and spleen samples to the laboratory for analysis. I submitted one fresh sample and one formalin-fixed sample of each tissue. Myers (2001) provided a detailed explanation of necropsy protocols and laboratory diagnostic techniques.

I identified coyote and domestic dog (*Canis lupus familiarus*) predation based on canine puncture wounds and associated hemorrhaging, torn tissue on the hind legs, tracks, sign indicating a chase or struggle, blood on the ground or vegetation, and buried carcasses (neonates only). Carcasses of deer killed by coyotes were sometimes dismembered and spread out across the site, although I did not rely on this observation alone to confirm coyote predation. I identified mountain lion and bobcat (*Lynx rufus*) predation based on cached carcasses, canine puncture wounds and associated hemorrhaging, and tracks. I identified black bear (*Ursus americanus*) predation based on canine punctures and associated hemorrhaging, bruising, peeled hide, and bear sign. I identified malnutrition as a cause of death based on an intact carcass with minimal or no femur marrow fat, and the lack of any sign indicating disease, predation, or hemorrhaging. I evaluated femur marrow fat based on appearance and texture (Riney 1955), which was sufficient for identifying deer that had mostly or entirely depleted their fat reserves.

I classified fawn mortalities as canid predation, black bear predation, felid predation, unknown predation (i.e., unidentified predator), disease, starvation or malnutrition, injury or accident, and unknown. Canid predation was caused by coyotes, and to a much lesser extent, domestic dogs. Felid predation was caused by mountain lions and bobcats. Disease mortalities included deaths caused by or associated with hemorrhagic disease, severe diarrhea, pneumonia, infections, and congenital deformities. Injuries and accidents included fence injuries, blunt trauma, drowning, becoming trapped, and collisions with vehicles.

I classified adult doe mortalities as mountain lion predation, coyote predation, black bear predation, unknown predation, disease, suspected disease, malnutrition, injury, parturition death, and unknown. I did not include harvest as a mortality factor because no doe hunting occurred during my study. Disease mortalities included deaths caused by hemorrhagic disease, pneumonia, and malignant catarrhal fever (MCF, Schultheiss et al. 2007). Suspected disease mortalities comprised deaths that I could not specifically diagnose, yet were consistent with disease. I suspected disease because carcasses were intact, and field necropsies indicated the does did not die of predation, malnutrition, or trauma. Injuries primarily included collisions with vehicles, and parturition deaths included any death associated with giving birth.

3.3.6 Statistical Methods

I separately modeled adult doe body fat, pregnancy rates, fetal rates, fetal survival, neonatal survival, overwinter fawn survival, and annual adult doe survival as a function of the nutrition treatment and other relevant variables. I also modeled cause-specific mortality separately for neonates, wintering fawns, and adult does. For each

analysis, I developed a priori model sets based on my expectations of important variable relationships with the ultimate goal of quantifying the effect of the nutrition enhancement treatment. I used Akaike's information criterion adjusted for sample size (AIC_c) to select among candidate models and I corrected for overdispersion when appropriate using quasi-likelihood ($QAIC_c$). I used model-averaging to reflect model selection uncertainty in estimates of parameters (Burnham and Anderson 2002). In a few instances, however, I based parameter estimates on the model with the lowest AIC_c, generally because that model received all of the Akaike weight.

Body Fat and Reproductive Rates.—I modeled estimated adult doe body fat as a function of treatment and year using PROC MIXED in SAS (SAS Version 9.1, 2003). I modeled adult doe pregnancy rates as a function of treatment and year using PROC LOGISTIC in SAS, and I modeled adult doe fetal rates as a function of treatment, year, and age class (yearling or ≥2-yr-old does) using PROC MIXED (SAS Version 9.1, 2003). I did not obtain any data on yearling fetal rates during 2002, and I obtained fetal counts from only 9 yearlings during 2003 and 2004. Thus, I only had power to detect large fetal rate differences between yearlings and older does.

Fetal Survival.— I was unable to determine the fate of 96 of the 255 fetuses documented in utero because some VITs were ineffective and newborn fawns were difficult to detect. I therefore developed a joint likelihood that included several nuisance detection parameters to estimate fetal survival in the absence of known fates (Chapter 2). I numerically maximized the natural logarithm of the likelihood function using a quasi-Newton optimization algorithm in PROC NLMIXED in SAS (SAS Version 9.1, 2003) to obtain parameter estimates and the variance-covariance matrix. I modeled fetal survival

as a function of treatment and year. There was a potential for overdispersion because my fetus sample comprised a high proportion of siblings. Sibling fetuses may have lacked independent fates because they shared the same maternal resources. However, I did not find evidence of overdispersion (Chapter 2). I therefore did not inflate variance estimates and I used AIC_c to select among models.

Neonatal Survival.— I analyzed neonatal survival using the Known Fates option in Program MARK (White and Burnham 1999), which accommodated staggered entry and exit times of marked fawns during the analysis period (Kaplan and Meier 1958, Pollock et al. 1989). I modeled survival as a function of fawn age (i.e., days survived since birth), Julian date of birth, treatment, year, fawn sex, estimated fawn mass at birth (kg), and estimated fawn hind foot length at birth (cm). I used 182 daily survival intervals to construct encounter histories of survival from birth to 6 months of age. Fawns that were ≤1 day old when captured were included in the first survival interval, fawns that were >1 and ≤2 days old when captured entered the analysis in the second survival interval, and so forth. A majority of neonates in my sample (0.748) were ≤2 days old when captured and most (0.904) were ≤4 days old when captured. I measured fawn mass and hind foot length at the time of capture rather than at birth, which meant the measurements were not directly comparable across fawns. I therefore estimated fawn mass and hind foot length at birth using a regression analysis in SAS (PROC REG, SAS Version 9.1, 2003). I modeled fawn mass and hind foot length at the time of capture as a function of fawn age at capture, Julian date of birth, sex, treatment, and year. I provided a detailed explanation of this analysis in Chapter 2.

Similar to fetal survival, my neonatal survival data were potentially overdispersed because my sample included 88 sets of twins and 2 sets of triplets. Sibling neonates shared maternal resources and used the environment similarly in time and space, which could have caused dependence among neonate fates. I found evidence of modest overdispersion in these data and recommended setting the overdispersion parameter, c, equal to 1.25 in a quasi-likelihood analysis (Chapter 2). I therefore used $QAIC_c$ to select among neonatal survival models with $\hat{c} = 1.25$.

Overwinter Fawn Survival.— I analyzed overwinter fawn survival using the Nest Survival option in Program MARK (White and Burnham 1999) because it allowed data with irregular radio-monitoring of collared animals, referred to as ragged telemetry data (Rotella et al. 2004). On winter range, signals of most radiocollared fawns were monitored daily, whereas a few fawns were monitored weekly or biweekly. Once deer left winter range, monitoring of all fawns became more sporadic. I typically determined the exact dates of fawn mortalities, although in some cases, I could only determine an approximate date. The ragged telemetry analysis allowed me to incorporate all the available information from these different scenarios.

I modeled overwinter fawn survival as a function of time, treatment, year, fawn sex, early winter mass (kg), chest girth (cm), and hind foot length (cm). I estimated survival from 17 December to 16 June, which resulted in 182 daily survival intervals. I selected 17 December as the start date because 16 December was the mean 6-month birthday of fawns captured as neonates. I estimated survival over a 6-month period (i.e., through 16 June), which is when fawns reached 1 year of age. I constrained time 4 different ways in my models: weekly, monthly, seasonal (i.e., winter or spring), and as a

trend. All fawns captured and radiocollared in the treatment EU were included in survival analyses with a treatment designation regardless of whether they accessed the supplement or not.

Annual Adult Doe Survival.— I analyzed annual adult doe survival using the Nest Survival option in Program MARK (White and Burnham 1999) because my radio-monitoring was irregular among individuals and throughout the year. I modeled annual doe survival as a function of time, treatment, year, age, timing of capture, early winter mass (kg), chest girth (cm), and hind foot length (cm). I estimated annual survival from 15 December to 14 December, which resulted in 365 daily survival intervals. I constrained time 3 different ways in my models: biweekly, monthly, and seasonal (i.e., winter-spring or summer-fall). Many adult does were included in multiple years' samples, although I only measured individual covariates when the does were initially captured and radiocollared. I used these individual covariate values in multiple years' samples because they reflected overall differences in deer body size. I included timing of capture (i.e., Nov–Dec or Feb–Mar) as a variable to evaluate whether adult doe individual covariates varied depending on what time of year they were measured. Similar to 6-month-old fawns, all adult does captured and radiocollared in the treatment EU were included in survival analyses with a treatment designation regardless of whether they accessed the supplement or not.

Deer-vehicle collisions (DVCs) were a common cause of mortality of adult does captured in the Colona EU but not the Shavano EU. Deer from the Colona EU were commonly in close proximity to highways during spring and fall whereas most Shavano deer were not. I analyzed adult doe survival in the context of a balanced crossover

experimental design, which should have minimized confounding of DVCs with the nutrition treatment. However, to evaluate the potential for confounding, I performed 2 separate analyses of annual adult doe survival. I included all observed DVCs in the first analysis, whereas I right-censored DVCs in the second analysis.

Cause-specific Mortality.— I modeled cause-specific mortality of neonates, wintering fawns, and adult does using a generalized logits model (i.e., multinomial logistic regression) in SAS (PROC LOGISTIC, SAS Version 9.1, 2003). I modeled neonatal mortality causes during summer and fall as a function of fawn age (i.e., days survived since birth), Julian date of birth, treatment, year, sex, and estimated fawn mass at birth (kg). I modeled mortality causes of fawns during winter and spring as a function of year, treatment, sex, time, and early winter mass (kg). I modeled annual mortality causes of adult does as a function of year, treatment, season, age (years), and mass (kg). I evaluated 2 different season variables; the first comprised 4 levels (winter, spring, summer, and fall), whereas the second had 2 levels (winter-spring and summer-fall). I only included deer mortalities in these analyses rather than the entire sample of radio-collared deer. My objective was to evaluate variability in the relative contributions of different mortality factors to the total observed mortality.

I performed 2 separate analyses based on 2 resolutions of the mortality data for each deer age group (i.e., neonates, winter fawns, adult does). In the first analysis, the dependent variable comprised a separate level for each individual mortality category except the unknown category. I excluded unknown mortalities because they did not represent a unique mortality cause, but rather some combination of the other mortality categories. Unknown mortalities comprised 11% of all neonatal and wintering fawn

mortalities and 18% of all adult doe mortalities. In the second analysis, I reduced the number of mortality categories to 3: predation, disease-malnutrition-starvation, and injury-accident. The latter analysis considered widely differing mortality factors that were easily discernible from one another.

Continuous Survival Rates.— I estimated fawn survival from the fetal stage to 6 months old as the product of fetal and neonatal survival rates. Similarly, I estimated fawn survival from the fetal stage to 1 year old as the product of fetal, neonatal, and overwinter fawn survival rates. I estimated a treatment effect as the difference in survival between treatment and control EGs. I estimated variances using the delta method (Seber 1982).

My estimate of survival from fetus to 1 year of age was structured to represent the treatment effect rather than any specific cohort of deer. This was necessary because any given winter's treatment applied to 2 cohorts of fawns. Overwinter fawn survival was measured as a function of the treatment using the current year's cohort of 6-month-old fawns whereas fetal and neonatal survival was measured using the upcoming year's cohort of fawns. Additionally, the crossover point of the experimental design occurred in December, meaning that the fawn cohort associated with the Colona EU switched from a treatment designation to a control designation in December 2002, and vice versa for the fawn cohort associated with the Shavano EU. In this case, estimating survival from the fetal stage to the yearling age class for a specific cohort of fawns would mix treatment and control assignments. Thus, for each year of the study, I estimated survival from the fetal stage to the yearling age class, as a function of the treatment, by taking the product of fetal and neonatal survival rates measured immediately post-treatment and the

overwinter fawn survival rate measured during administration of the treatment. For example, during 2001–02, I estimated survival of fetuses to the yearling age class as the product of 2002 fetal survival, 2002 neonatal survival, and 2001–02 overwinter fawn survival.

Population Rate of Change.— I used my fecundity and survival parameter estimates to construct year-specific matrix population models (Leslie 1945, 1948, Lefkovitch 1965, Caswell 2001) that reflected treatment and control conditions in this study. My population models included estimates of adult doe pregnancy rates, yearling doe fetal rates, ≥2-yr-old doe fetal rates, fetal survival rates, male and female neonatal survival rates, male and female overwinter fawn survival rates, and adult doe survival rates. I estimated the finite rate of population change, λ, by applying the same fecundity and survival rate estimates over time to an artificial population until age ratios reached a steady state. This approach provided a theoretical estimate of λ that was representative of the set of input parameters. I used the λ estimates as a means to quantify the effect of the nutrition enhancement treatment on mule deer population performance. When estimating year-specific λ, I structured the population models to represent the treatment effect rather than any specific cohort of fawns, as explained above. I imputed an expected value of yearling fetal rate in 2002 based on my fetal rate models because I lacked data to directly estimate the rate.

I estimated the variance of my λ estimates using the delta method (Seber 1982). Specifically, I numerically computed the partial derivatives of each λ function with respect to each fecundity and survival parameter used in my matrix population models. I

multiplied the partial derivatives matrix times the variance-covariance matrix of fecundity and survival estimates, which I then multiplied by the transpose of the partial derivatives matrix to obtain a variance-covariance matrix for the set of λ estimates.

3.4 RESULTS

3.4.1 Adult Doe Body Fat and Reproductive Rates

The optimal model of estimated adult doe body fat included a treatment × year interaction (Table 1). Estimated percent body fat of treatment adult does was higher than that of control does each year, although the magnitude of the effect varied annually (Table 2). I found no evidence of variation in pregnancy rates between treatment and control adult does or among years (Table 3). Adult doe pregnancy rate for all deer during the study was 0.935 (SE = 0.0191). Adult doe fetal rates varied among years and between age classes, but did not vary as a function of the treatment (Tables 4, 5).

3.4.2 Fetal Survival

Fetal survival varied between treatment and control EGs and among years (Table 6). Fetal survival was higher overall in the treatment EG than in the control EG, although I observed considerable annual variation in the magnitude of the effect. I observed virtually no difference in fetal survival between treatment and control EGs in 2003, whereas I observed a large difference between EGs in 2004 (Table 7).

3.4.3 Neonatal Survival

The 4 most parsimonious models of neonatal survival had nearly identical $QAIC_c$ weights. Based on these models, neonatal survival varied as a function of neonatal sex, treatment, year, a 3rd order polynomial trend in fawn age, Julian date of birth, estimated birth mass, and a year × birth mass interaction (Tables 8, 9). I found only marginal

evidence of a treatment effect because addition of the treatment parameter to the most parsimonious models did not lower $QAIC_c$. The 95% confidence interval of the treatment beta estimate overlapped 0 ($\hat{\beta}_{trt}$ = 0.276, 95% CI: –0.123, 0.675). Survival of treatment neonates averaged 0.528 (SE = 0.0549), and survival of control neonates averaged 0.482 (SE = 0.0565), during the study (Table 9). The sex effect was also marginal based on the beta parameter estimate ($\hat{\beta}_{sex}$ = 0.322, 95% CI: –0.083, 0.728). Although I lacked strong evidence, I found that survival of female neonates was higher than that of male neonates (Table 9).

The polynomial trend in fawn age indicated that neonatal daily survival probability was lowest immediately post-birth, increased markedly over the first 70 days of life ($\hat{\beta}_{Age}$ = 0.101, 95% CI: 0.068, 0.134), decreased slightly from 71 to 134 days of life ($\hat{\beta}_{Age^2}$ = –0.00111, 95% CI: –0.00161, –0.00060), and increased slightly from 135 to 182 days of life ($\hat{\beta}_{Age^3}$ = 0.0000036, 95% CI: 0.0000015, 0.0000057). Neonatal survival probability decreased the later fawns were born ($\hat{\beta}_{bdate}$ = –0.223, 95% CI: –0.409, –0.037), and survival probability increased with greater birth mass ($\hat{\beta}_{bmass}$ = 0.260, 95% CI: 0.054, 0.465). Birth mass had a greater effect on survival probability during 2002 than either 2003 or 2004 ($\hat{\beta}_{year02\times bmass}$ = 0.667, 95% CI: 0.024, 1.310; $\hat{\beta}_{year03\times bmass}$ = 0.104, 95% CI: –0.345, 0.554). The effect of birth mass and birth date on survival was partially related to the treatment. Fawns in the treatment EG averaged 3.64 kg (SE = 0.0578) at birth whereas fawns in the control EG averaged 3.49 kg (SE = 0.0573). Mean birth date, expressed as the number of days following the first fawn birth (i.e., 2 June), was lower

for fawns in the treatment EG (13.95, SE = 0.509) than fawns in the control EG (15.76, SE = 0.498).

In the cause-specific mortality analysis that distinguished among the 7 mortality categories, the model including an intercept and fawn age received all of the Akaike weight (No. parameters = 12, AIC_c weight = 1.000). Causes of fawn mortality changed as fawns aged and as the summer-fall season progressed (Fig. 4). In the analysis that lumped mortality causes into 3 categories, the intercept-only model received slightly more Akaike weight than any other model (Table 10). The relative proportions of total mortality comprised of predation, starvation-disease, and injuries-accidents remained roughly the same during the study and among EGs (Fig. 5).

3.4.4 Overwinter Fawn Survival

Overwinter fawn survival varied as a function of the nutrition enhancement treatment, year, sex, time (monthly), and early winter mass and chest girth (Tables 11, 12). I found strong evidence of a treatment effect ($\hat{\beta}_{trt}$ = 1.350, 95% CI: 0.723, 1.978) (Table 12). Survival of fawns receiving the treatment averaged 0.905 (0.0259) whereas survival of control fawns averaged 0.684 (SE = 0.0438). Similar to neonates, I found some evidence that female fawns had higher survival than male fawns ($\hat{\beta}_{sex}$ = 0.362, 95% CI: –0.200, 0.925) (Table 12). Lowest monthly survival occurred between mid-January and mid-February ($\hat{\beta}_{month2}$ = –1.552, 95% CI: –2.520, –0.584), whereas highest monthly survival occurred between mid-March and mid-April ($\hat{\beta}_{month4}$ = 0.291, 95% CI: –1.139, 1.720). The probability of fawn survival increased as early winter mass and chest girth

increased, although the effect of mass was much greater than that of chest girth ($\hat{\beta}_{mass}$ = 0.145, 95% CI: 0.087, 0.202; $\hat{\beta}_{chest}$ = 0.043, 95% CI: –0.032, 0.117).

In the cause-specific mortality analysis that distinguished among each individual mortality cause of wintering fawns, the intercept-only model received virtually all of the Akaike weight (No. parameters = 5, AIC_c weight = 0.911), suggesting that relative prevalence of different mortality causes did not vary. Coyote predation was the most common proximate cause of overwinter fawn mortality (Fig. 6). In the second analysis, with only 3 mortality categories, I observed marginal evidence of annual and sex-specific variation (Table 13, Fig. 7). I lacked evidence to suggest mortality causes varied between EGs, in part because very few treatment fawns died during the study (n = 13).

3.4.5 Annual Adult Doe Survival

Annual adult doe survival varied as a function of the nutrition enhancement treatment, season, age, and hind foot length regardless of whether DVCs were included in the analysis (Tables 14, 15). Including DVCs, model-averaged annual survival estimates were 0.879 (SE = 0.0206) for treatment adult does and 0.833 (SE = 0.0253) for control adult does. Excluding DVCs, model-averaged annual survival estimates were 0.898 (SE = 0.0191) for treatment adult does and 0.867 (SE = 0.0227) for control adult does. Also, there was a treatment × season interaction regardless of whether DVCs were included in the analysis. Treatment deer experienced higher survival during winter-spring than summer-fall, whereas control deer did not (Table 16). The probability of adult doe survival increased as hind foot length increased; the effect was most pronounced in the analysis that excluded DVCs ($\hat{\beta}_{foot}$ = 0.116, 95% CI: –0.032, 0.263). Regarding age,

models that evaluated a linear relationship between doe survival probability and doe age had roughly similar weight to models that tested a quadratic relationship between survival and age. The former received the most AIC_c weight in the analysis that excluded DVCs, which indicated a declining survival probability with increasing age ($\hat{\beta}_{age} = -0.109$, 95% CI: –0.196, –0.023). The quadratic effect received most support in the analysis that included DVCs, which indicated that survival probability increased until does were 5 years old, after which survival probability declined with age ($\hat{\beta}_{age} = 0.188$, 95% CI: –0.134, 0.509; $\hat{\beta}_{age^2} = -0.018$, 95% CI: –0.039, 0.004).

In the adult doe cause-specific mortality analysis that distinguished among each individual mortality cause, the model with an intercept and doe age received most of the Akaike weight (No. parameters = 16, AIC_c weight = 0.834). Adult does ≤8 years old died principally from collisions with vehicles, disease, and mountain lion predation. Older does died principally from malnutrition and coyote predation (Fig. 8). All but 2 does killed by coyotes had minimal or no femur marrow fat remaining, indicating they were severely malnourished. In the second analysis, with only 3 mortality categories, 2 models received most of the Akaike weight: 1) intercept + season (No. parameters = 8, AIC_c weight = 0.521) and 2) intercept + season + age (No. parameters = 10, AIC_c weight = 0.412). During winter and summer, a majority of the mortality was caused by disease or malnutrition, and there were few DVCs. The opposite was true during spring and fall (Fig. 9). Predation was relatively constant throughout all seasons. I lacked sufficient evidence to suggest mortality causes varied between EGs, even though most DVCs were associated with the Colona EU, which indicates the crossover experimental design

minimized any potential confounding between the nutrition enhancement treatment and DVCs.

3.4.6 Continuous Survival Rates

I estimated fawn survival from the fetal stage to 6 months old separately for each treatment, year, and sex combination, consistent with model selection results from my fetal and neonatal survival analyses (Table 17). I likewise estimated fawn survival from the fetal stage to 1 year of age separately for each treatment, year, and sex combination (Table 18). The nutrition enhancement treatment increased survival of fetuses to the yearling age class by 0.14–0.20, although 95% confidence intervals slightly overlapped 0 (Table 19). When averaging estimates across sexes and years, survival of treatment fetuses to the yearling age class was 0.447 (SE = 0.0519), whereas survival of control fetuses to the yearling age class was 0.271 (SE = 0.0418). Thus, the treatment caused survival to increase by 0.177 (SE = 0.0818, 95% CI: 0.0163, 0.3370).

3.4.7 Population Rate of Change

In my population models, I used adult doe survival estimates that included DVCs because I did not find evidence that DVCs were confounded with the nutrition treatment. My estimates of the population rate of change, $\hat{\lambda}$, were 1.15–1.17 for treatment deer and 1.02–1.06 for control deer, with some overlap in 95% confidence intervals (Fig. 10). Average $\hat{\lambda}$ was 1.165 (SE = 0.0358) for treatment deer and 1.033 (SE = 0.0380) for control deer. The treatment caused $\hat{\lambda}$ to increase by 0.139 (95% CI: 0.0197, 0.2592) during 2001–02, 0.113 (95% CI: 0.0230, 0.2040) during 2002–03, and 0.145 (95% CI:

0.0477, 0.2422) during 2003–04. When averaged across years, the treatment caused $\hat{\lambda}$ to increase by 0.133 (95% CI: 0.0487, 0.2166).

3.5 DISCUSSION

3.5.1 Fecundity and Survival Estimates

I adequately delivered the nutrition enhancement treatment to mule deer occupying the treatment EU each winter (Table 2). Therefore, any results indicating minimal or weak treatment effects cannot be explained by a failure to deliver the treatment. I found no differences in pregnancy and fetal rates between EGs. Any treatment effects likely would have been carried over from the previous year's treatment because most adult does were bred just prior to the start of treatment delivery each year. Both pregnancy and fetal rates were high for each EG, equaling or exceeding previous estimates measured on the Uncompahgre Plateau and elsewhere across Colorado (Andelt et al. 2004). Pregnancy and fetal rates were not a limiting factor to the mule deer population during the years of my study.

I detected treatment effects in each survival parameter I measured, which encompassed survival of all age and sex classes except adult males. I observed relatively strong support for a treatment effect in fetal survival, primarily because the effect was large during 2004. I found marginal evidence of a treatment effect in neonatal survival. My sample sizes were insufficient to detect small to moderate effects (i.e., survival increase of 0.05–0.10) with desired power, especially during 2002. Small-moderate effects in neonatal survival are biologically meaningful, however, particularly when considered over time. Overdispersion in my neonatal survival data further reduced power to detect a treatment effect (Chapter 2). Principal drivers of neonatal survival included

birth mass and birth date, which were only partially explained by the treatment. Survival increased with increased birth mass and earlier birth dates, which has been observed previously in deer (Lomas and Bender 2007) and other ungulates (Singer et al. 1997, Keech et al. 2000, Cook et al. 2004).

I found strong evidence of a treatment effect on overwinter fawn survival, even when survival of control fawns was high. Overwinter survival of treatment fawns averaged 0.905 (SE = 0.0259) during the study, which is exceptionally high when compared to other recorded survival estimates of free-ranging mule deer fawns (White et al. 1987, Unsworth et al. 1999, Bishop et al. 2005*a*). Early winter mass explained additional variation in the data. Probability of survival increased as early winter mass increased, which has been documented previously (White et al. 1987, Unsworth et al. 1999, Bishop et al. 2005*a*, Hurley and Zager 2006, Taillon et al. 2006). The effects of the nutrition treatment and early winter mass on survival probability provide strong evidence that fawn body condition dictated overwinter survival.

I observed higher survival of female fawns than male fawns during both the neonatal and overwinter survival periods. Higher female neonatal survival has been documented in deer previously (Jackson et al. 1972), but most studies have found little or no evidence for sex differences in neonatal survival (Gaillard et al. 1997, Ricca et al. 2002, Pojar and Bowden 2004, Lomas and Bender 2007). Sex differences in overwinter fawn survival have been documented more commonly, with females having higher survival (Bartmann et al. 1992, White and Bartmann 1998, Unsworth et al. 1999, Bishop et al. 2005*a*). A key relevance of differential fawn survival between sexes is the effect on male and female yearling recruitment. During the 3 years of my study, survival from the

fetal stage to the yearling age class averaged 0.478 (SE = 0.0606) for treatment females and 0.417 (SE = 0.0616) for treatment males, and survival averaged 0.306 (SE = 0.0528) for control females and 0.238 (SE = 0.0458) for control males. Thus, survival to the yearling age class was roughly 0.06–0.07 higher for females than males, thereby creating a reduced buck:doe ratio prior to any harvest effects.

The nutrition treatment had a positive effect on adult doe survival during winter and spring, when deer received the treatment. During summer and fall, however, survival was similar among treatment and control does. Of note, summer-fall doe survival did not exceed winter-spring survival in my study. Survival monitoring of does across the Uncompahgre Plateau over the past 10 years has shown a similar trend in seasonal survival rates (B. E. Watkins and B. Banulis, Colorado Division of Wildlife, unpublished data). This is based entirely on natural mortalities because antlerless hunting was not allowed. Most summer doe mortalities appeared to be disease-related, and were apparently independent of nutrition. This seasonal pattern of doe survival is not typical of other populations in Colorado and across the West (Bartmann et al. 1992, Ricca et al. 2002, Bender et al. 2007, Colorado Division of Wildlife, unpublished data).

3.5.2 Dependence in Stage-Specific Fawn Survival Rates

Fetal rates and fetal, neonatal, and overwinter fawn survival varied annually, as did the magnitude of the treatment effects, but not in synchrony. The largest treatment effect in fetal survival occurred during 2004, when the treatment had the least effect on neonatal survival. The highest measured fetal rates occurred during 2004, when fetus-neonate survival rates (i.e., survival from fetus to 6 months old) were the lowest. Neonatal survival rates declined over the course of the study, whereas overwinter

survival rates increased each year of the study. I observed annual variation in each fecundity and survival parameter, yet recruitment of yearlings as a function of the treatment was relatively constant. Likewise, I observed relatively minimal temporal variability in the estimated population rate of change ($\hat{\lambda}$) for each EG, particularly treatment deer (Fig. 10). These results suggest a compensatory relationship among stage or season-specific survival rates, and therefore, emphasize the need to consider all stages when assessing populations (Caswell 2001).

A possible explanation for this compensatory relationship is the timing of death of lightweight or otherwise unthrifty fawns, which have a lower probability of surviving to the yearling age class. In some years, conditions may facilitate relatively high survival of these fawns to winter, at which point their survival probability declines significantly. In other years, these fawns may have low survival probabilities during summer and fall, which reduces December fawn recruitment, but increases overwinter fawn survival because the poorest condition fawns have already been removed from the population. The same relationship could apply to fetuses and neonates, as I saw among control deer in 2004. The stillborn fetuses in 2004 were mostly small, lightweight, and seemingly undernourished. If these fetuses had been born alive, they invariably would have suffered high mortality rates as neonates. The effect would have been to increase fetal survival and decrease neonatal survival.

3.5.3 Effect of Enhanced Nutrition on Population Rate of Change

The treatment caused λ to increase by an average of 0.133 (SE = 0.0428) during the 3 years of my study. The 95% confidence intervals on my estimates of the treatment effect on λ did not overlap 0, providing strong evidence for the effect. The mean

estimate of λ for the treatment EG was 1.165 (SE = 0.0358), which would cause a population to double in size in approximately 5 years. For perspective, the Uncompahgre Plateau deer population is currently estimated at roughly 38,000 deer (B. Banulis, Colorado Division of Wildlife, unpublished data). The treatment conditions would cause the population to increase by >7,000 deer per year. This level of response supports the hypothesis that the deer population was limited by NCC through density-dependent feedback. My results demonstrate that deer nutrition, and therefore habitat quality, is ultimately a critical limiting factor of the population. My finding is particularly noteworthy considering predation and disease were overall the most common proximate causes of deer mortality prior to and during my study (Watkins et al. 2001, Pojar and Bowden 2004, B. Banulis, Colorado Division of Wildlife, unpublished data). Furthermore, my study took place during 4 mild to average winters, when nutrition might be expected to play a lesser role.

My research provides additional insights into the role of nutrition in ungulate population regulation. My results are consistent with research linking nutrition to fecundity and survival in deer (Verme 1969, Robinette et al. 1973, Ozoga and Verme 1982, Baker and Hobbs 1985, Mech et al. 1991) and other ungulates (Thorne et al. 1976, Cameron et al. 1993, Keech et al. 2000, Cook et al. 2004). These studies directly link fecundity and survival to adult female body condition throughout the year, the rates of growth and fat accretion in young animals during late summer and fall, and the rates at which fat and protein are depleted during winter. I associated mule deer population rate of change with each of these nutritional factors in a free-ranging population exposed to diverse mortality factors. My results are also consistent with studies that documented

density dependent effects on fecundity or survival of ungulates by manipulating density (Clutton-Brock et al. 1987, Bartmann et al. 1992, White and Bartmann 1998, Stewart et al. 2005). Strong density-dependent effects were only observed when density manipulations occurred in enclosures. However, by manipulating nutrition, I documented a strong density-dependent effect in a free-ranging population.

3.5.4 Compensatory Mortality

I found minimal evidence of differences in fawn or doe mortality causes between EGs. The increased survival rates associated with the treatment effect were explained by the reductions in rates of all mortality causes rather than any specific mortality cause. That is, incidents of predation, malnutrition, starvation, disease, and injuries-accidents all declined as a result of enhanced nutrition. Naturally, the magnitude of the decline was more pronounced for ≥6-month-old fawns because that is where I observed the greatest treatment effect.

Considering neonates, it makes sense that enhanced nutrition of dams would cause lower rates of starvation and malnutrition, and possibly disease. However, the effect of nutrition on predation rates of neonates is less straightforward. There are several explanations as to why predation on neonates might decline in response to enhanced doe nutrition. First, does may be better able to detect predators and defend their fawns. Second, enhanced nutrition might reduce rates of diarrhea that increase fawn scent, making fawns less vulnerable to detection by predators. Third, as fawns become older, those in better condition may be better able to escape predators. Invariably, however, some amount of predation on newborn fawns occurs irrespective of doe or fawn

nutrition (Hamlin et al. 1984, Ballard et al. 2001, Hurley and Zager 2006). I observed only modest evidence that predation of neonates was compensatory.

In contrast, I found strong evidence that predation of ≥6-month-old fawns and adult does was compensatory. Coyote predation was the most common mortality cause of wintering fawns. Thus, the treatment effect in overwinter fawn survival resulted, in large part, from a reduction in coyote predation. Studies have found that coyotes primarily killed malnourished fawns during winter (Bartmann et al. 1992, Bishop et al. 2005*a*). I found that most fawns killed by coyotes in the control EU were malnourished, as evidenced by minimal or no femur marrow fat. Fawns in the treatment EU were in better condition, which explains why I observed a significant reduction in coyote predation as a result of the nutrition enhancement treatment. My results indicate that coyote predation of ≥6-month-old fawns was compensatory, which is consistent with findings from other field experiments where coyote numbers were manipulated instead of deer nutrition (Bartmann et al. 1992, Hurley and Zager 2006). Observed coyote predation of wintering fawns in the intermountain West, albeit common, is not evidence that coyotes are having a negative impact on deer populations. I also found that coyote predation on adult does was predominantly compensatory because coyotes selected for older does in poor condition, which is consistent with Hurley and Zager (2006).

Felid predation occurred at a relatively constant rate among neonates, ≥6-month-old fawns, and adult does, accounting for roughly 15% of the total mortality. Most felid predation of ≥6-month-old fawns and adult does was caused by mountain lions. I expected mountain lion predation to account for a greater proportion of the total mortality of treatment deer, because lions are capable of killing healthy animals and of potentially

having a negative impact on populations (Bleich and Taylor 1998, Ballard et al. 2001, Robinson et al. 2002, Hurley and Zager 2006). Instead, mountain lion predation declined in response to the treatment for ≥6-month-old fawns and adult does, suggesting that it was compensatory as well. I observed no mountain lion predation of treatment fawns ≥6 months old, whereas I observed 6 mountain lion kills and 2 bobcat kills among control fawns. Four of the 6 control fawns killed by mountain lions, and both fawns killed by bobcats, were malnourished based on emaciation and/or minimal or no femur marrow fat. The other 2 fawns were in poor condition, but had some femur marrow fat remaining. Similarly, I observed 1 treatment adult doe, versus 8 control adult does, killed by mountain lions.

It is possible that daily activity in the treatment EU (delivering pellets) deterred predation; however, roughly equal amounts of time were spent in each EU each winter monitoring radio-collared deer and collecting fawn:doe ratio data from the ground. The treatment was delivered by ≤3 individuals during morning hours, when deer were typically bedded. My largest winter field crew comprised 4 individuals, which divided tasks among treatment and control EUs. Additionally, my winter range EUs were situated in a rapidly developing area where human activity was common. The presence of my field crew likely had little influence on predator activities.

The large effect of enhanced nutrition on $\hat{\lambda}$ in my study suggests habitat was ultimately the critically limiting factor of the Uncompahgre deer population. Predation should have minimal impact on populations that are at or near NCC (Ballard et al. 2001). In contrast to my results, Hurley and Zager (2006) observed no increase in $\hat{\lambda}$ in response to coyote reductions, and only a slight increase in $\hat{\lambda}$ in response to mountain lion

reductions. My findings regarding relative effects of habitat versus predation should not be extrapolated to more complex predator-prey systems that include additional predator species such as wolves (*Canis lupus*).

3.5.5 Disease

Disease was a common mortality factor among neonatal fawns and adult does, but not ≥6 month old fawns. I found minimal evidence that disease was compensatory in adult does; in fact, I observed equal numbers of disease cases in each EG. Disease actually represented a higher proportion of total mortality among treatment adult does than control adult does in my sample data, although I lacked power to detect the difference. I found no evidence of novel diseases or chronic wasting disease (CWD), the latter of which has not been documented in southwest Colorado. I observed several cases each of hemorrhagic disease, MCF (Schultheiss et al. 2007), and pneumonia among adult does. Hemorrhagic disease and severe diarrhea were most commonly associated with neonatal disease-related deaths. I failed to identify the exact cause of death in a number of disease-related cases for both does and neonates. Additionally, greater than 50% of adult does were seropositive (i.e, titers ≥1:32) for bovine viral diarrhea virus (BVDV) during 2000–02, and I isolated the virus from a neonate (C. J. Bishop, Colorado Division of Wildlife, unpublished data). For unexplained reasons, seroprevalence dropped to <25% in 2002–03. Of note, I did not link BVDV to fecundity or mortality. Deer intermixed with sheep and cattle on summer range and were occasionally in close proximity to livestock on winter range, which likely explains the prevalence of BVDV and MCF. The degree to which disease may be negatively impacting the population

remains unclear; however, the overall large effect of enhanced nutrition suggests habitat quality is ultimately of greater importance.

3.5.6 Diet Quality and Habitat Enhancement

Deer receiving the supplemental pellet consumed a higher quality diet than deer consuming natural vegetation only. Average-sized fawns and adult does in the treatment EU should have met maintenance energy requirements during winter by consuming 0.7–0.9 kg and 1.1–1.4 kg of the supplement per day, respectively (Baker et al. 1979, Swift 1983, Baker et al. 1998). I estimated that consumption was roughly 1.4–2.0 kg/deer/day based on estimated deer and elk densities in the treatment EUs (C. J. Bishop, Colorado Division of Wildlife, unpublished data), expected elk consumption rates (i.e., 4.5 kg/elk/day), and daily quantities of the supplement provided. In contrast, deer consuming only natural forage likely failed to meet maintenance nutrient requirements from dietary intake during winter.

The principal forage species of deer on the winter range EUs were alfalfa (*Medicago* spp.), Utah juniper, big sagebrush, black sagebrush (*Artemisia nova*), cheatgrass (*Bromus tectorum*) and crested wheatgrass (*Agropyron cristatum*). In vitro dry matter digestibility (IVDMD) of alfalfa generally ranges from 50 to 70% (Swanson and Herman 1952, Weir et al. 1960, Robles et al. 1981, Lenssen et al. 1988, Belyea et al. 1989), and crude protein (CP) of alfalfa is roughly 17–25% (Weir et al. 1960, Lenssen et al. 1988). Alfalfa was clearly a valuable forage item for deer, but it was limited in quantity and only available through mid December. Sagebrush and juniper were the main forage species available from late December through early March. Winter estimates of IVDMD are 40–48% for Utah juniper (Bunderson et al. 1986, Welch 1989), 45–65% for

big sagebrush (Ward 1971, Kufeld et al. 1981, Welch and Pederson 1981, Welch 1989), and 53% for black sagebrush (Welch et al. 1983, Welch 1989). Winter estimates of CP range from 6 to 12% for these same species (Welch 1989, Wambolt 2004). Sagebrush and juniper species contain terpenoids, which likely further reduced quality of winter deer diets due to microbial inhibition (Nagy et al. 1964, Carpenter et al. 1979, Schwartz et al. 1980). Spring IVDMD and CP estimates of immature, green cheatgrass (IVDMD: 65–72%, CP: 17–21%) and crested wheatgrass (IVDMD: 71–73%, CP: 27–28%) are high (Austin et al. 1994, Bishop et al. 2001), although they offer minimal forage value during winter. On a similar pinyon-juniper-sagebrush winter range in northwest Colorado, deer diets during January–March ranged from 24 to 38% IVDMD and 5 to 7% crude protein (Bartmann 1983). In contrast, the supplemental pellet provided 63% digestibility and 22% crude protein (Baker et al. 1998).

Habitat treatments in the pinyon-juniper woodlands could improve habitat productivity by increasing the quantity and diversity of higher-quality forage. Treatments would likely cause the greatest increase in diet quality during winter, although late fall and spring diets might also improve because of increased forage availability. During the past decade, roller chop and hydro axe treatments performed in pinyon-juniper woodlands on the Uncompahgre Plateau, coupled with reseeding of mostly native species, have caused an increase in the quantity and diversity of forbs, grasses, and certain browse species (UPP 2007; Bureau of Land Management, unpublished data).

The ultimate question is whether habitat enhancement treatments improve mule deer population performance, or conversely, minimize population declines as habitat is lost. My study establishes the need to evaluate habitat enhancement strategies on the

Uncompahgre Plateau. Specifically, my findings provide a scientific basis for pursuing and evaluating vegetative manipulation techniques in late-seral pinyon-juniper winter range as a means to set back succession and increase habitat productivity for deer. However, my measured rates of population increase, in response to artificial nutrition enhancement, would not be feasible or sustainable in response to habitat improvements via vegetative manipulations. The objective of such habitat management should be to achieve smaller, yet sustainable, deer population increases over time. Availability of quality habitat is likely to become even more limiting because the most productive winter and summer range habitats on the Uncompahgre Plateau and adjacent San Juan Mountains are being lost to human development at a rapid rate. A coordinated effort to manage habitat at a landscape scale is underway on the Uncompahgre Plateau, referred to as the Uncompahgre Plateau Project (UPP 2007). To evaluate effectiveness of the Project from a deer perspective, an ongoing study is quantifying the effects of habitat treatments in pinyon-juniper on deer population parameters (Bergman et al. 2007).

I did not randomly select the Uncompahgre Plateau as a study site. It was chosen specifically because the deer population had declined, and there were competing hypotheses with respect to habitat versus predation as limiting factors. My results should not be extrapolated beyond the Uncompahgre Plateau for these reasons. However, given resource limitations that prevent similar studies from being conducted in numerous populations, it may be reasonable to make cautious inference to other pinyon-juniper winter ranges across the Colorado Plateau, which are uniquely dominated by *Pinus edulis* and *Juniperus osteosperma* (West 1999). The current status of pinyon-juniper on the Uncompahgre Plateau, which was the basis for the hypothesis of why deer declined, is

not unique. Many pinyon-juniper communities are considered degraded primarily because of excessive grazing and altered fire patterns, and therefore, warrant proactive management (Gruell 1999, West 1999). Proposed strategies to restore pinyon-juniper communities may likewise improve deer habitat productivity, and therefore, may be advisable anywhere in the Colorado Plateau ecoregion where deer populations have declined (Watkins et al. 2007). However, there is a need to evaluate the effectiveness of various habitat treatments for mule deer (Bergman et al. 2007).

3.5.7 Winter Feeding

I caution against the use of my findings to justify winter feeding management programs for deer. I did not administer the pelleted supplement in a manner consistent with a typical winter feeding program. I provided the supplement ad libitum, and I spread it out to avoid creation of feed grounds. I expended, on average, $40,000 and roughly 1000 person hours per winter to purchase and deliver the supplemental feed to <1000 deer, and up to 200–300 elk, across 7–22 km^2. As a rough extrapolation, it would require >40,000 person hours and ~$1.75 million in feed costs to provide the supplement in this manner to most of the Uncompahgre deer population for a winter. Clearly, I did not employ methods in order to evaluate winter feeding as a possible management strategy for deer. My objective was to simulate optimum habitat conditions for deer. Others have evaluated the utility of winter feeding as a management strategy to mitigate deer mortality (Baker and Hobbs 1985, Peterson and Messmer 2007).

3.6 MANAGEMENT IMPLICATIONS

I demonstrated density dependence in a free-ranging mule deer population by manipulating nutrition on winter range and measuring responses in fecundity and

survival. Fetal, neonatal, and overwinter fawn survival, and annual adult doe survival, increased as a result of enhanced nutrition. Enhanced nutrition therefore had a large, positive effect on population rate of change, indicating habitat quality was a critical limiting factor of the deer population. My study provides support for evaluating the effectiveness of habitat treatments for deer in pinyon-juniper winter range. If effective, such treatments would provide a strategy to increase the Uncompahgre Plateau deer population, and to lessen impacts of future habitat loss. My findings could be reasonably used to justify evaluation of habitat treatments in other deer populations that have declined across the Colorado Plateau given similarity of the pinyon-juniper winter ranges and associated management challenges.

Predation and disease were common proximate causes of deer mortality. Coyote predation was compensatory, at least for ≥6-month-old fawns and adult does. I also found evidence that mountain lion predation was compensatory for ≥6-month-old fawns and adult does. The magnitude of the nutrition treatment effect on population rate of increase, and evidence of compensatory mortality, suggests predation was not having a negative effect on the deer population. My findings demonstrate that observed coyote predation is not useful for evaluating whether coyotes are negatively impacting a deer population. The effect of disease was unclear, although I found virtually no evidence it was compensatory in adult does.

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Table 1. Model selection results, based on Akaike's Information Criterion with small sample size correction (AIC_c), from an analysis of estimated adult doe body fat percentage in mid-late winter as a function of year and a nutrition enhancement treatment in southwest Colorado, USA, 2002–2004.

Model	No. parameters	AIC_c	ΔAIC_c	Akaike wt
Year, treatment, year × treatment	6	716.66	0.00	1.000
Year, treatment	4	743.67	27.01	0.000
Treatment	2	753.74	37.08	0.000
Year	3	828.12	111.47	0.000
Intercept only	1	836.13	119.47	0.000

Table 2. Estimated percent body fat of adult doe mule deer occupying a pinyon-juniper winter range during late February–early March in southwest Colorado, USA, 2002–2004. Approximately half of the adult does received enhanced nutrition via supplementation (treatment) whereas the other half received no supplementation (control).

			Percent body fat	
Year	Experimental group	*n*	Estimate	SE
2002	Treatment	18	10.21	0.597
	Control	18	7.60	0.597
2003	Treatment	30	13.90	0.463
	Control	28	6.64	0.479
2004	Treatment	30	10.63	0.463
	Control	30	7.28	0.463

Table 3. Model selection results, based on Akaike's Information Criterion with small sample size correction (AIC_c), from an analysis of adult doe mule deer pregnancy rates as a function of year and a nutrition enhancement treatment in southwest Colorado, USA, 2002–2004.

Model	No. parameters	AIC_c	ΔAIC_c	Akaike wt
Intercept only	1	83.26	0.00	0.631
Treatment	2	85.24	1.97	0.235
Year	3	87.03	3.77	0.096
Year, treatment	4	89.06	5.80	0.035
Year, treatment, year × treatment	6	93.27	10.01	0.004

Table 4. Model selection results, based on Akaike's Information Criterion with small sample size correction (AIC_c), from an analysis of adult doe mule deer fetal rates as a function of year, age class (yearling or ≥2 years old), and a nutrition enhancement treatment in southwest Colorado, USA, 2002–2004.

Model	No. parameters	AIC_c	ΔAIC_c	Akaike wt
Year, age class, year × age class	6	202.00	0.00	0.503
Year, age class	4	202.47	0.47	0.398
Age class	2	205.26	3.26	0.099
Year	3	217.45	15.44	0.000
Intercept only	1	219.22	17.22	0.000
Year, treatment	4	219.74	17.74	0.000
Year, treatment, year × treatment	6	221.25	19.24	0.000
Treatment	2	221.70	19.70	0.000

Table 5. Adult doe mule deer fetal rates during late February–early March in southwest Colorado, USA, 2002–2004.

Year	Age class	*n*	Fetal rate[a]	
			Estimate	SE
2002	≥2 Years Old	36	1.79	0.075
	Yearling	0	---	---
2003	≥2 Years Old	45	1.82	0.066
	Yearling	5	1.07	0.190*
2004	≥2 Years Old	56	2.01	0.061
	Yearling	4	1.41	0.206

[a]I used model-averaging to estimate fetal rates (Burnham and Anderson 2002).

Table 6. Model selection results, based on Akaike's Information Criterion with small sample size correction (AIC_c), from an analysis of mule deer fetal survival as a function of year (yr) and a nutrition enhancement treatment (trt) in southwest Colorado, USA, 2002–2004.

Model[a]	No. Parameters	AIC_c	ΔAIC_c	Akaike wt
S_1(trt × yr) S_2(.) p_1(yr) p_2(yr) r(.) a(yr) b(yr)	20	1137.79	0.00	0.293
S_1(trt × yr) S_2(trt) p_1(yr) p_2(yr) r(.) a(yr) b(yr)	21	1138.27	0.48	0.231
S_1(trt + yr) S_2(.) p_1(yr) p_2(yr) r(.) a(yr) b(yr)	18	1139.20	1.41	0.145
S_1(trt) S_2(.) p_1(yr) p_2(yr) r(.) a(yr) b(yr)	16	1139.39	1.60	0.132
S_1(trt × yr) S_2(.) p_1(.) p_2(yr) r(.) a(yr) b(yr)	18	1140.30	2.51	0.084
S_1(trt × yr) S_2(yr) p_1(yr) p_2(yr) r(.) a(yr) b(yr)	22	1141.06	3.27	0.057
S_1(trt × yr) S_2(.) p_1(yr) p_2(yr) r(.) a(trt × yr) b(trt × yr)	26	1142.06	4.27	0.035
S_1(yr) S_2(.) p_1(yr) p_2(yr) r(.) a(yr) b(yr)	17	1143.78	5.99	0.015
S_1(trt × yr) S_2(.) p_1(yr) p_2(.) r(.) a(yr) b(yr)	18	1146.00	8.21	0.005
S_1(trt × yr) S_2(.) p_1(yr) p_2(trt × yr) r(.) a(trt × yr) b(trt × yr)	29	1146.93	9.14	0.003
S_1(trt × yr) S_2(.) p_1(trt × yr) p_2(trt × yr) r(.) a(trt × yr) b(trt × yr)	32	1153.51	15.72	0.000

Table 6. Continued.

Model	No. Parameters	AIC_c	ΔAIC_c	Akaike wt
S_1(trt × yr) S_2(trt) p_1(trt × yr) p_2(trt × yr) r(.) a(trt × yr) b(trt × yr)	33	1155.15	17.36	0.000
S_1(trt × yr) S_2(trt × yr) p_1(trt × yr) p_2(trt × yr) r(.) a(trt × yr) b(trt × yr)	37	1163.96	26.17	0.000
S_1(trt × yr) S_2(.) p_1(.) p_2(.) r(.) a(.) b(.)	12	1167.19	29.40	0.000
S_1(.) S_2(.) p_1(.) p_2(.) r(.) a(.) b(.)	7	1174.95	37.16	0.000

[a]Fetal survival probability is represented by parameter S_1; all other model parameters are nuisance parameters explained in detail in Chapter 2.

Table 7. Estimated in utero survival of mule deer fetuses from February until birth on a pinyon-juniper winter range in southwest Colorado, USA, 2002–2004. Roughly half of the adult does carrying the fetuses received enhanced nutrition via supplementation during winter (treatment) whereas the other half received no supplementation (control).

			Fetal survival[a]	
Year	Experimental group	n	$\hat{S}$	SE($\hat{S}$)
2002	Treatment	24	0.857	0.1134
	Control	33	0.779	0.1579
2003	Treatment	38	0.966	0.0327
	Control	44	0.935	0.0594
2004	Treatment	57	0.983	0.0277
	Control	59	0.747	0.0899

[a]I used model-averaging to estimate survival (Burnham and Anderson 2002).

Table 8. Model selection results, based on quasi-likelihood using Akaike's Information Criterion with small sample size correction ($QAIC_c$), from an analysis of mule deer neonatal survival as a function of sex, year (yr), a nutrition enhancement treatment (trt), fawn age trend (A), Julian date of birth (bdate), estimated birth mass (bmass, kg), and estimated birth hind foot length (bhft, cm), in southwest Colorado, USA, 2002–2004.

Model[a]	No. Parameters	$QAIC_c$[b]	$\Delta QAIC_c$	$QAIC_c$ wt
Sex, bmass, A, A^2, A^3, bdate	7	1212.49	0.00	0.129
Sex, bmass × year, A, A^2, A^3, bdate	11	1212.52	0.03	0.127
Sex, trt, bmass × year, A, A^2, A^3, bdate	12	1212.70	0.21	0.116
Bmass, A, A^2, A^3, bdate	6	1212.92	0.43	0.104
Sex, trt, bmass, A, A^2, A^3, bdate	8	1213.33	0.84	0.085
Bmass × year, A, A^2, A^3, bdate	10	1213.53	1.04	0.077
Trt, bmass, A, A^2, A^3, bdate	7	1214.07	1.58	0.059
Trt, bmass × year, A, A^2, A^3, bdate	11	1214.14	1.66	0.056
Trt, bmass, year, A, A^2, A^3, bdate	9	1214.70	2.21	0.043
Trt, bmass, year, A, A^2, A^3	8	1215.18	2.69	0.034

Table 8. Continued.

Model	No. Parameters	$QAIC_c$	$\Delta QAIC_c$	$QAIC_c$ wt
Trt × year, bmass × year, A, A^2, A^3, bdate	13	1215.60	3.11	0.027
Trt, bmass × year, A, A^2, A^3, bdate, bhft	12	1215.61	3.12	0.027
Trt × year, A, A^2, A^3, bdate × bmass	12	1216.39	3.90	0.018
Trt × year, bmass × year, A, A^2, A^3, bdate × bmass	14	1216.50	4.01	0.017
Trt × year, bmass × year, A, A^2, A^3, bdate, bhft	14	1216.82	4.33	0.015
Trt × year, bmass, A, A^2, A^3, bdate	11	1217.02	4.53	0.013
Trt × year, bmass, A, A^2, A^3	10	1217.48	4.99	0.011
Trt × year, bmass, A, A^2, A^3, bdate, bhft	12	1217.63	5.14	0.010

[a]I considered a total of 40 models. I listed in the table only those models that received ≥0.01 $QAIC_c$ weight. All models that included daily, weekly, or biweekly variation in fawn age received 0 $QAIC_c$ weight.

[b]Model selection results were based on $\hat{c} = 1.25$ (Chapter 2).

Table 9. Estimated survival of neonatal mule deer from birth to 6 months old in southwest Colorado, USA, 2002–2004. Roughly half of the neonates' dams received enhanced nutrition via supplementation during the previous winter (treatment) whereas the other half of the neonates' dams received no supplementation (control).

				Neonatal survival[a]	
Year	Experimental group	Sex	*n*	$\hat{S}$	SE($\hat{S}$)
2002	Treatment	Female	17	0.615	0.1161
	Treatment	Male	13	0.565	0.1243
	Control	Female	10	0.560	0.1201
	Control	Male	13	0.508	0.1186
2003	Treatment	Female	27	0.573	0.0761
	Treatment	Male	28	0.519	0.0881
	Control	Female	26	0.522	0.0776
	Control	Male	21	0.465	0.0813
2004	Treatment	Female	29	0.478	0.0832
	Treatment	Male	37	0.421	0.0787
	Control	Female	32	0.450	0.0840
	Control	Male	17	0.391	0.0881

[a]I used model-averaging to estimate survival (Burnham and Anderson 2002).

Table 10. Model selection results, based on Akaike's Information Criterion with small sample size correction (AIC_c), from an analysis of neonatal mule deer mortality causes as a function of fawn age (i.e., days survived since birth), Julian date of birth (bdate), a nutrition enhancement treatment, year, fawn sex, and estimated fawn mass at birth (bmass, kg) in southwest Colorado, USA, 2002–2004.

Model	No. parameters	AIC_c	ΔAIC_c	AIC_c wt
Intercept only	2	188.60	0.00	0.205
Bmass	4	189.27	0.67	0.147
Year, treatment, year × treatment	12	189.41	0.81	0.137
Age	4	189.41	0.81	0.136
Year	6	190.27	1.67	0.089
Bmass, age	6	190.78	2.18	0.069
Treatment	4	190.99	2.40	0.062
Bdate	4	191.47	2.89	0.049
Year, treatment, year × treatment, bmass	14	191.78	3.19	0.042
Sex	4	192.38	3.78	0.031
Year, treatment	8	193.28	4.68	0.020
Year, sex	8	194.42	5.82	0.011
Year, treatment, sex	10	197.46	8.86	0.002
Year, sex, year × sex	12	201.49	12.89	0.000

Table 11. Model selection results, based on Akaike's Information Criterion with small sample size correction (AIC_c), from an analysis of mule deer overwinter survival as a function of a nutrition enhancement treatment (trt), year (yr), sex, time (t), early winter mass (mass, kg), early winter chest girth (chest, cm), and hind foot length (hft, cm), in southwest Colorado, USA, 2001–2004. I constrained time 4 different ways: weekly, monthly, seasonally (i.e., winter, spring), and as a trend (T).

Model[a]	No. parameters	AIC_c	ΔAIC_c	AIC_c wt
Trt, yr, sex, t(month), mass	11	650.42	0.00	0.435
Trt, yr, sex, t(month), mass, chest	12	651.12	0.70	0.307
Trt × t(month), yr, sex, mass	16	653.03	2.61	0.118
Trt, yr × sex, t(month), mass	13	653.76	3.34	0.082
Trt, yr, sex, t(T), mass	7	655.71	5.29	0.031
Trt, yr × t(month), sex, mass	21	657.82	7.40	0.011
Trt, yr, sex, t(season), mass	7	658.52	8.11	0.008
Trt, yr, sex, t(week), mass	31	660.52	10.10	0.003
Trt, sex, mass	4	661.99	11.57	0.001
Trt, mass	3	662.04	11.62	0.001
Trt, yr, mass	5	662.08	11.66	0.001
Trt, yr, sex, mass	6	662.25	11.83	0.001

[a]I considered a total of 23 models. I listed in the table only those models that received ≥0.001 AIC_c weight.

Table 12. Estimated overwinter survival of mule deer fawns occupying a pinyon-juniper winter range in southwest Colorado, USA, 2001–2004. Half of the fawns received enhanced nutrition via supplementation (treatment) whereas the other half received no supplementation (control).

				Overwinter survival[a]	
Year	Experimental group	Sex	n	$\hat{S}$	SE($\hat{S}$)
2001–02	Treatment	Female	18	0.894	0.0383
	Treatment	Male	21	0.853	0.0471
	Control	Female	15	0.648	0.0812
	Control	Male	24	0.542	0.0867
2002–03	Treatment	Female	18	0.932	0.0267
	Treatment	Male	22	0.902	0.0350
	Control	Female	17	0.763	0.0692
	Control	Male	21	0.671	0.0821
2003–04	Treatment	Female	19	0.938	0.0252
	Treatment	Male	19	0.912	0.0336
	Control	Female	25	0.780	0.0644
	Control	Male	16	0.702	0.0823

[a]I used model-averaging to estimate survival (Burnham and Anderson 2002).

Table 13. Model selection results, based on Akaike's Information Criterion with small sample size correction (AIC_c), from an analysis of wintering mule deer fawn mortality causes as a function of time, a nutrition enhancement treatment, year, fawn sex, and fawn early winter mass (mass, kg) in southwest Colorado, USA, 2001–2004.

Model	No. parameters	AIC_c	ΔAIC_c	AIC_c wt
Year	6	83.91	0.00	0.269
Year, sex	8	84.35	0.45	0.215
Intercept only	2	85.22	1.31	0.140
Year, mass	8	86.39	2.49	0.078
Mass	4	86.43	2.53	0.076
Sex	4	86.57	2.66	0.071
Year, treatment, sex	10	87.67	3.76	0.041
Year, treatment	8	87.71	3.80	0.040
Treatment	4	88.18	4.27	0.032
Time	4	89.08	5.18	0.020
Year, time	8	89.52	5.61	0.016
Year, treatment, year × treatment	12	93.85	9.94	0.002
Year, sex, year × sex	12	94.61	10.71	0.001

Table 14. Model selection results, based on Akaike's Information Criterion with small sample size correction (AIC_c), from an analysis of adult doe mule deer annual survival as a function of a nutrition enhancement treatment (trt), year (yr), time (t), age, mass (mass, kg), chest girth (chest, cm), and hind foot length (foot, cm), in southwest Colorado, USA, 2000–2004. I constrained time 3 different ways: biweekly, monthly, and seasonally (i.e., winter-spring, summer-fall).

Model[a]	No. parameters	AIC_c	ΔAIC_c	AIC_c wt
Trt × t(season), age, age^2	6	1275.18	0.00	0.130
Trt × t(season), age	5	1275.58	0.40	0.106
Trt × t(season), age, age^2, foot	7	1275.77	0.59	0.096
Trt × t(season)	4	1276.06	0.89	0.083
Trt, age	3	1276.16	0.98	0.079
Trt	2	1276.62	1.44	0.063
Trt, age, foot	4	1276.62	1.45	0.063
Trt × t(season), age, age^2, age^3	7	1276.79	1.61	0.058
Trt × t(season), foot	5	1276.91	1.74	0.054
Trt, t(season), age	4	1277.14	1.96	0.049
Trt × t(season), age × foot	7	1277.69	2.51	0.037
Trt, t(season)	3	1277.73	2.55	0.036
Trt × t(month)	24	1277.84	2.66	0.034
Trt, t(season), foot	4	1278.60	3.43	0.023

Table 14. Continued.

Model[a]	No. parameters	AIC_c	ΔAIC_c	AIC_c wt
Trt, t(biweekly)	27	1279.40	4.22	0.016
Trt, t(season), chest	4	1279.66	4.49	0.014
Trt, t(season), mass	4	1279.69	4.51	0.014

[a]I considered a total of 32 models. I listed in the table only those models that received $\geq$0.01 AIC_c weight.

Table 15. Model selection results, based on Akaike's Information Criterion with small sample size correction (AIC_c), from an analysis of adult doe mule deer annual survival in which all mortalities resulting from deer-vehicle collisions were censored. I analyzed survival as a function of a nutrition enhancement treatment (trt), year (yr), time (t), age, mass (mass, kg), chest girth (chest, cm), and hind foot length (foot, cm), in southwest Colorado, USA, 2000–2004. I constrained time 3 different ways: biweekly, monthly, and seasonally (i.e., winter-spring, summer-fall).

Model[a]	No. parameters	AIC_c	ΔAIC_c	AIC_c wt
Trt × t(season), age, foot	6	1053.20	0.00	0.215
Trt × t(season), age, age^2, foot	7	1053.52	0.32	0.184
Trt × t(season), age	5	1053.53	0.33	0.182
Trt × t(season), age, age^2, age^3, foot	8	1053.11	1.91	0.083
Trt, age	3	1055.18	1.98	0.080
Trt × t(season), age × foot	7	1055.20	2.00	0.079
Trt × t(season)	4	1056.58	3.38	0.040
Trt, t(season), age	4	1056.89	3.69	0.034
Trt × t(season), foot	5	1056.93	3.74	0.033
Trt	2	1058.33	5.13	0.017
Trt, t(month)	13	1058.49	5.29	0.015

[a]I considered a similar list of models as those represented in Table 14. I listed in the table only those models that received ≥0.01 AIC_c weight.

Table 16. Estimated annual survival of adult doe mule deer (n = 274) in southwest Colorado, USA, 2000–2004. Half of the deer received enhanced nutrition via supplementation (treatment) during winter-spring whereas the other half received no supplementation (control). I present estimates from 2 analyses. Mortalities resulting from deer-vehicle collisions (DVCs) were included in the first analysis, whereas DVCs were right-censored in the second analysis.

			Annual survival[a]	
Analysis	Experimental group	Season	$\hat{S}$	SE($\hat{S}$)
DVCs included	Treatment	Winter-spring	0.952	0.0159
	Treatment	Summer-fall	0.924	0.0172
	Control	Winter-spring	0.911	0.0180
	Control	Summer-fall	0.915	0.0185
DVCs censored	Treatment	Winter-spring	0.964	0.0135
	Treatment	Summer-fall	0.932	0.0162
	Control	Winter-spring	0.922	0.0175
	Control	Summer-fall	0.941	0.0160

[a]I used model-averaging to estimate survival (Burnham and Anderson 2002).

Table 17. Estimated survival of mule deer fawns from the fetal stage to 6 months old as a function of a nutrition enhancement treatment, year, and fawn sex in southwest Colorado, USA, 2001–2004.

Year	Experimental group	Sex	$\hat{S}$	SE($\hat{S}$)
2001–02	Treatment	Female	0.527	0.1215
	Treatment	Male	0.485	0.1243
	Control	Female	0.436	0.1287
	Control	Male	0.395	0.1223
2002–03	Treatment	Female	0.553	0.0759
	Treatment	Male	0.501	0.0868
	Control	Female	0.488	0.0789
	Control	Male	0.435	0.0809
2003–04	Treatment	Female	0.470	0.0829
	Treatment	Male	0.413	0.0783
	Control	Female	0.336	0.0746
	Control	Male	0.292	0.0746

Table 18. Estimated survival of mule deer fawns from the fetal stage to 1 year old as a function of a nutrition enhancement treatment, year of treatment delivery, and fawn sex in southwest Colorado, USA, 2001–2004.

Treatment year	Experimental group	Sex	$\hat{S}$	SE($\hat{S}$)
2001–02	Treatment	Female	0.471	0.1105
	Treatment	Male	0.414	0.1085
	Control	Female	0.282	0.0905
	Control	Male	0.214	0.0746
2002–03	Treatment	Female	0.516	0.0723
	Treatment	Male	0.452	0.0802
	Control	Female	0.372	0.0690
	Control	Male	0.292	0.0651
2003–04	Treatment	Female	0.441	0.0786
	Treatment	Male	0.377	0.0728
	Control	Female	0.262	0.0621
	Control	Male	0.205	0.0576

Table 19. Effect of an overwinter nutrition enhancement treatment on survival of mule deer fawns from the fetal stage to 1 year old in southwest Colorado, USA, 2001–2004. The treatment effect represents the increase in survival attributable to the treatment.

		Treatment effect		
Treatment year	Sex	Estimate	95% LCL	95% UCL
2001–02	Female	0.189	–0.1078	0.4857
	Male	0.199	–0.0815	0.4780
2002–03	Female	0.144	–0.0202	0.3076
	Male	0.160	–0.0037	0.3230
2003–04	Female	0.179	–0.0091	0.3669
	Male	0.172	–0.0116	0.3562

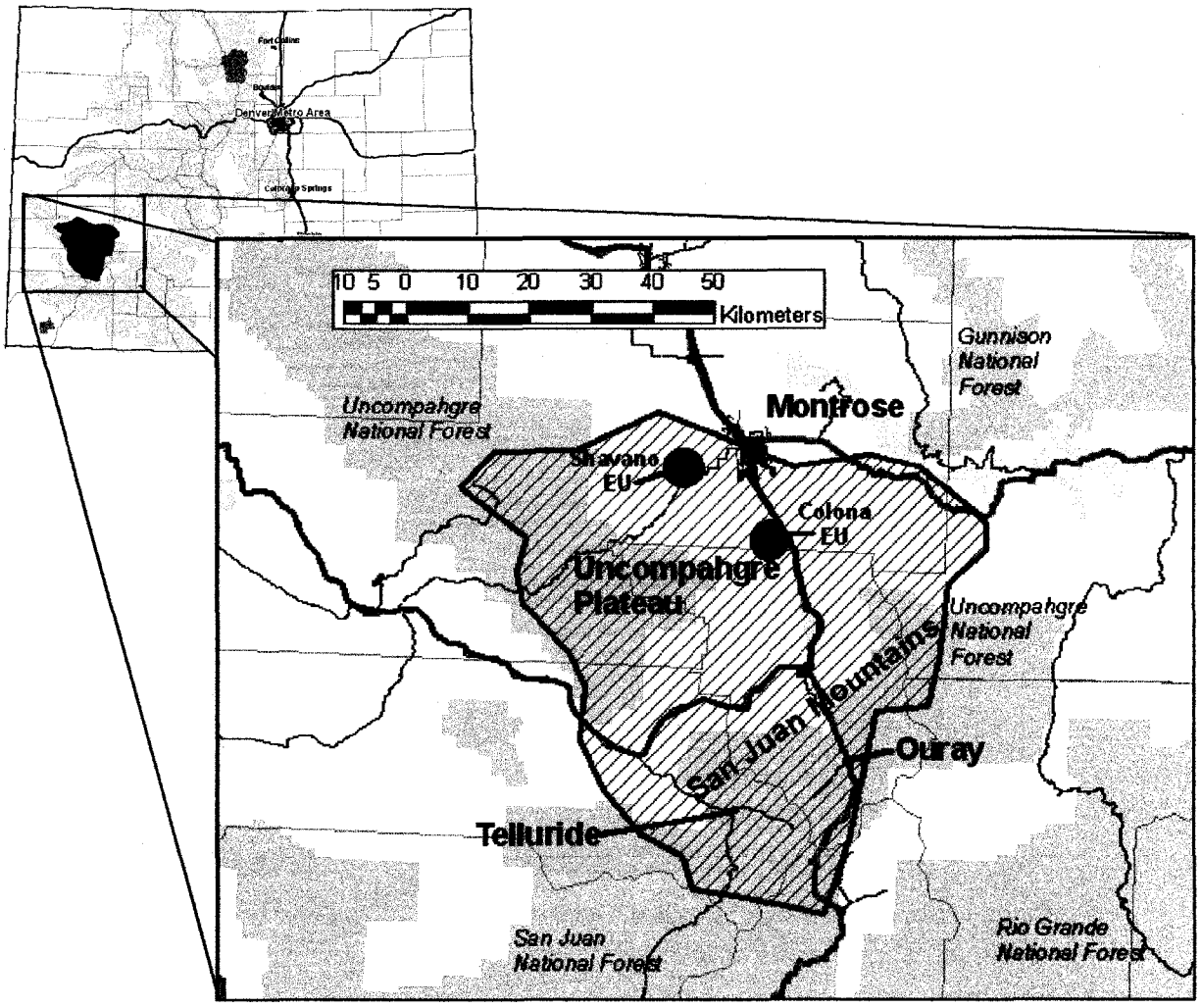

Figure 1. Location of winter range experimental units (EU; ●) and summer range study area (▨) on the Uncompahgre Plateau and adjacent San Juan Mountains in southwest Colorado, where I studied the effects of enhanced nutrition on mule deer population performance, 2000–2004.

Year	Colona EU	Shavano EU
2000-01	Treatment	Control
2001-02	Treatment	Control
2002-03	Control	Treatment
2003-04	Control	Treatment

Figure 2. Depiction of the crossover experimental design I used to evaluate the effect of enhanced nutrition on mule deer population performance in southwest Colorado, 2000–2004. Colona and Shavano experimental units (EUs) were study sites located on mule deer winter range where I administered a nutrition enhancement treatment. I measured only adult doe survival during 2000–01, whereas I measured fecundity, fawn survival, and adult doe survival rates in response to enhanced nutrition during 2001–2004.

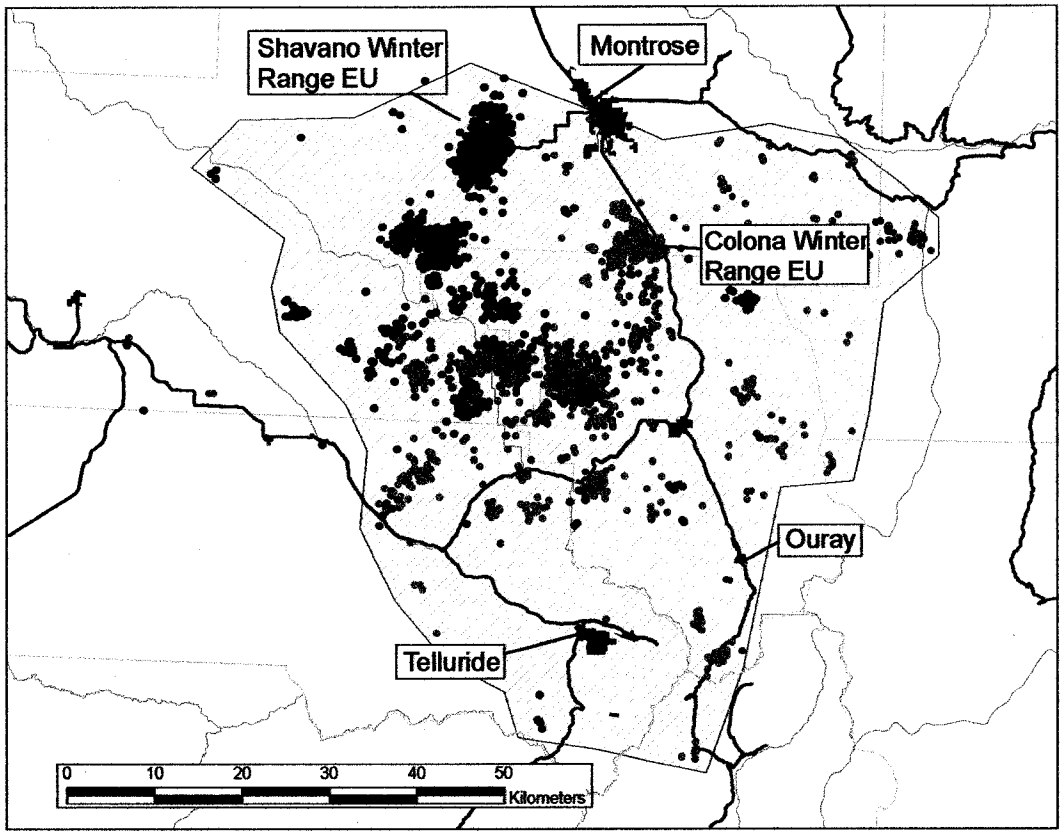

Figure 3. Annual locations of radio-collared mule deer on the Uncompahgre Plateau and adjacent San Juan Mountains in southwest Colorado, 2000–2004. Locations of deer captured in the Shavano experimental unit (EU) are shown in black; locations of deer captured in the Colona EU are shown in gray.

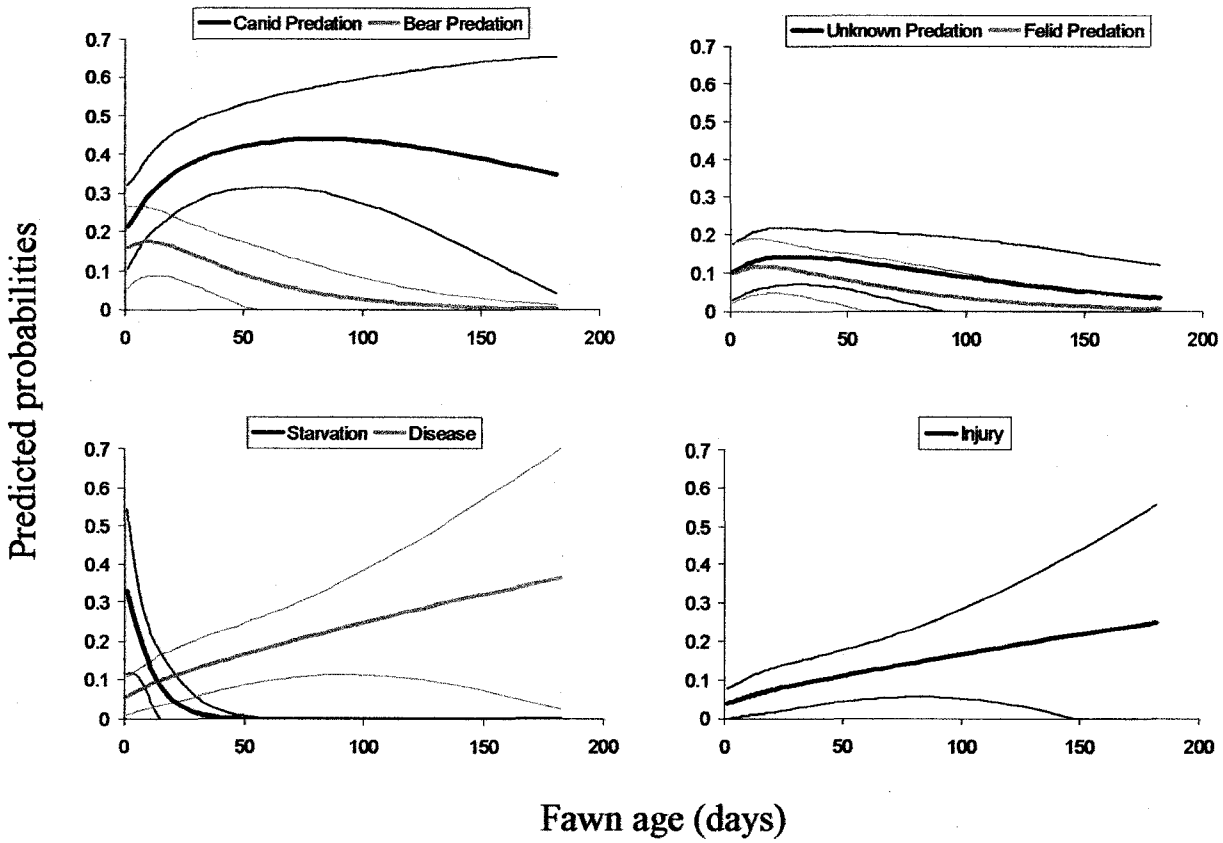

Figure 4. Predicted probabilities and 95% confidence intervals of mule deer neonatal mortality causes as a function of fawn age in southwest Colorado, 2002–2004. Cause-specific mortality probabilities are based on total mortality (i.e., Σ probabilities = 1), and therefore, represent a deer's relative likelihood of dying from a particular cause given that the deer dies.

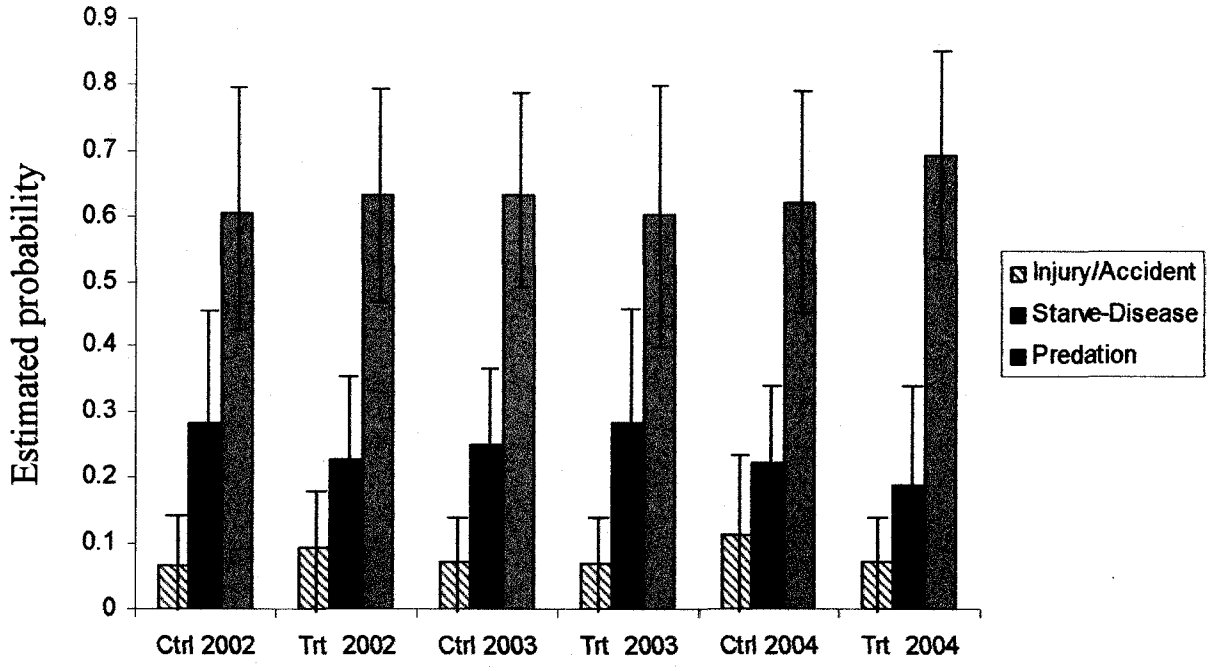

Figure 5. Estimated probabilities and 95% confidence intervals of mule deer neonatal mortality causes as a function of year and a nutrition enhancement treatment in southwest Colorado, 2002–2004. Cause-specific mortality probabilities are based on total mortality (i.e., Σ probabilities = 1), and therefore, represent a deer's relative likelihood of dying from a particular cause given that the deer dies. Individual mortality causes were lumped into 1 of the 3 categories shown.

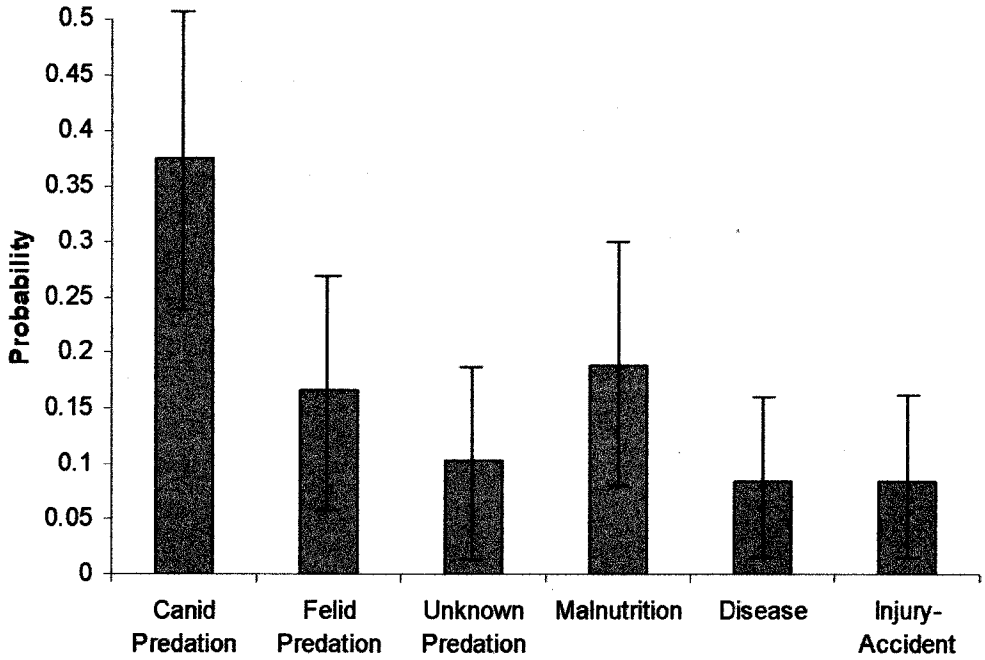

Figure 6. Estimated probabilities and 95% confidence intervals of mortality causes of ≥6-month-old mule deer fawns in southwest Colorado, 2001–2004. Cause-specific mortality probabilities are based on total mortality (i.e., Σ probabilities = 1), and therefore, represent a deer's relative likelihood of dying from a particular cause given that the deer dies.

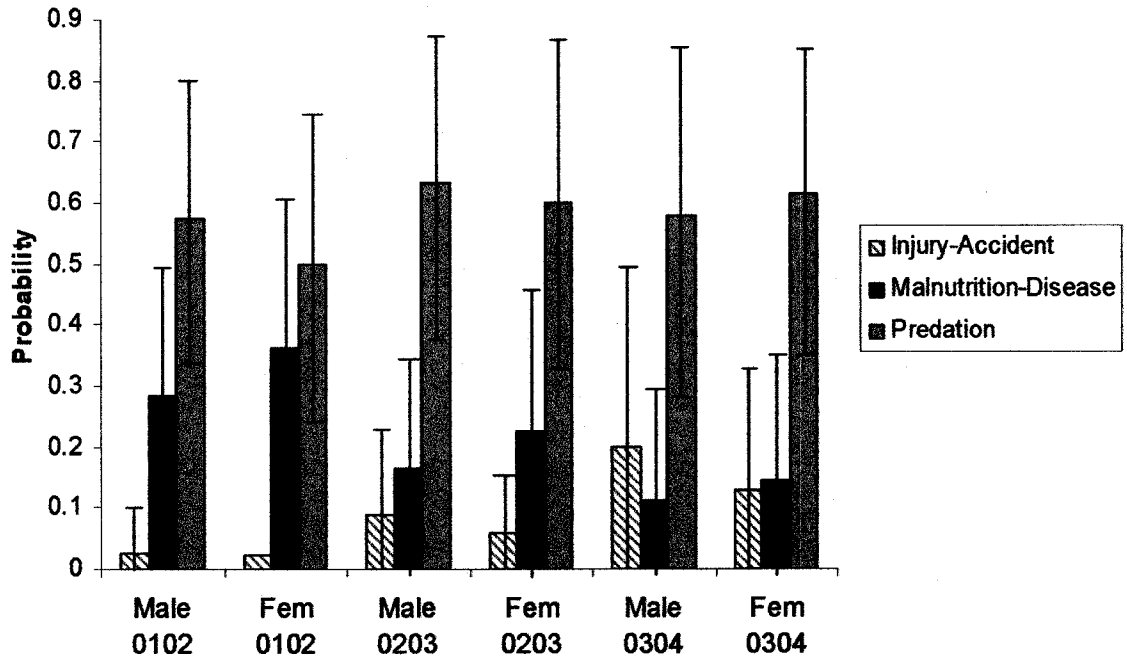

Figure 7. Estimated probabilities and 95% confidence intervals of ≥6-month-old mule deer fawn mortality causes as a function of year and sex in southwest Colorado, 2001–2004. Cause-specific mortality probabilities are based on total mortality (i.e., Σ probabilities = 1), and therefore, represent a deer's relative likelihood of dying from a particular cause given that the deer dies. Individual mortality causes were lumped into 1 of the 3 categories shown.

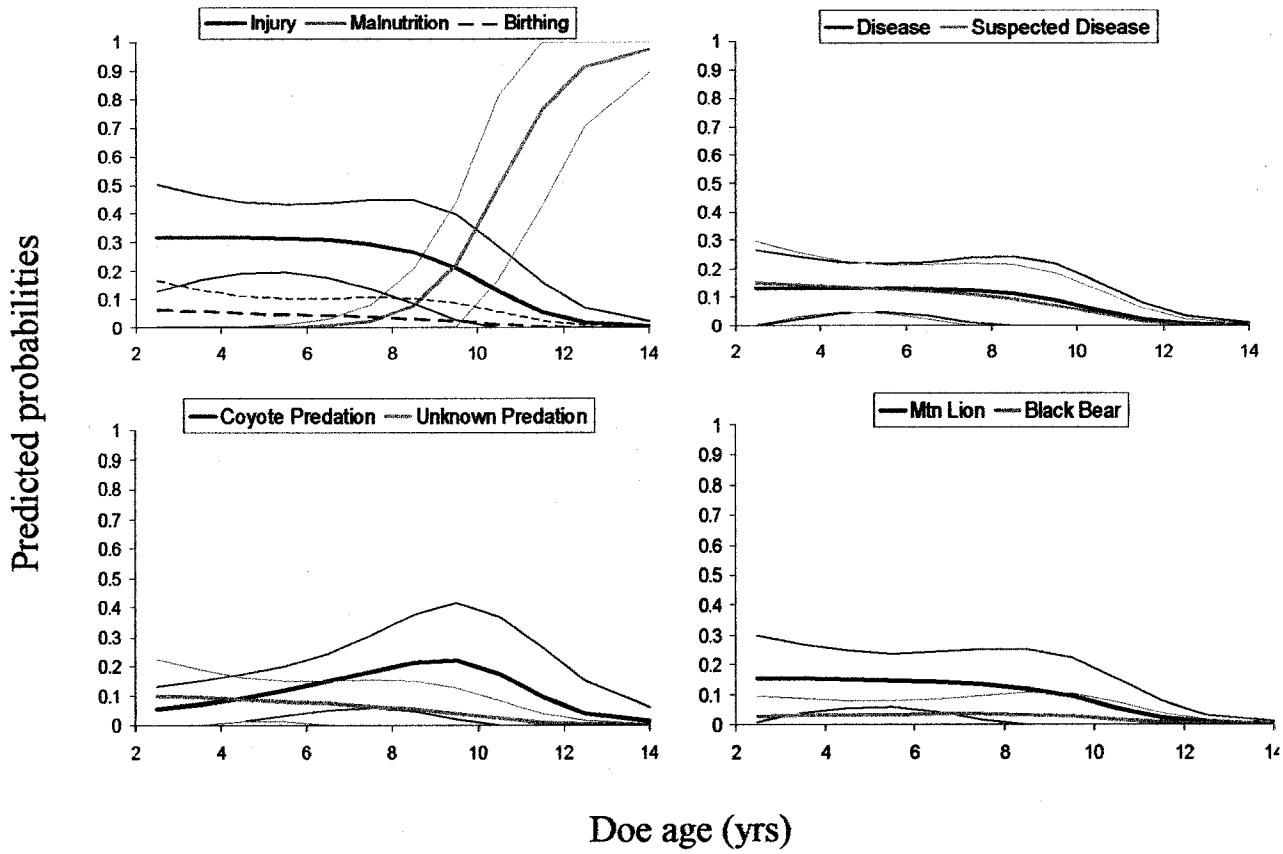

Figure 8. Predicted probabilities and 95% confidence intervals of adult doe mule deer mortality causes as a function of doe age in southwest Colorado, 2000–2004. Cause-specific mortality probabilities are based on total mortality (i.e., Σ probabilities = 1), and therefore, represent a deer's relative likelihood of dying from a particular cause given that the deer dies.

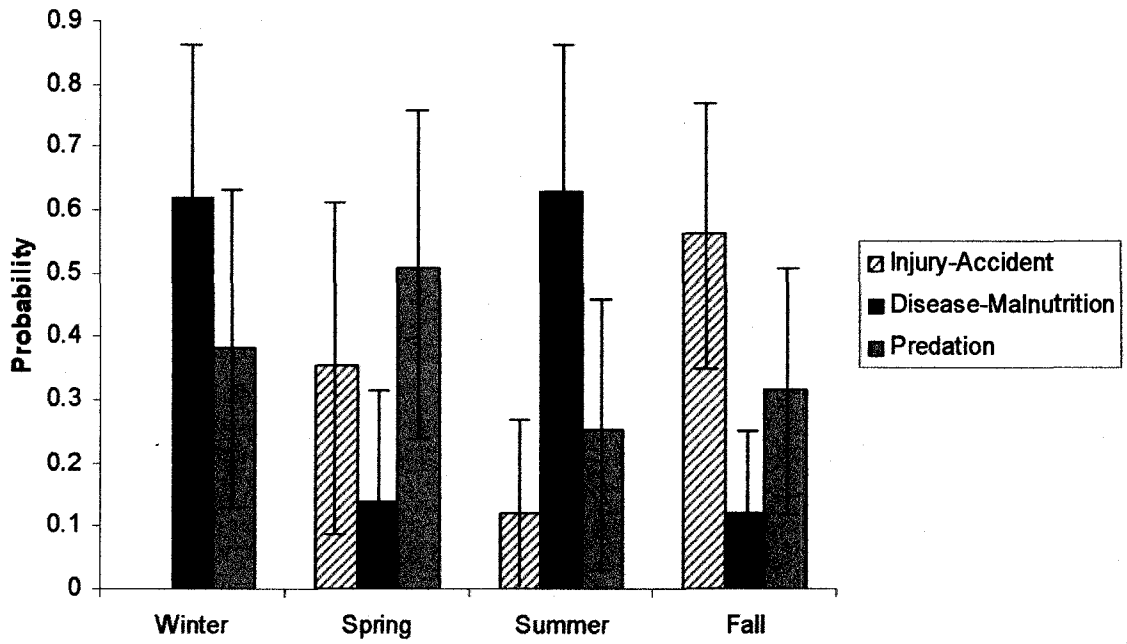

Figure 9. Estimated probabilities and 95% confidence intervals of adult doe mule deer mortality causes as a function of season in southwest Colorado, 2000–2004. Cause-specific mortality probabilities are based on total mortality (i.e., Σ probabilities = 1), and therefore, represent a deer's relative likelihood of dying from a particular cause given that the deer dies. Individual mortality causes were lumped into 1 of the 3 categories shown.

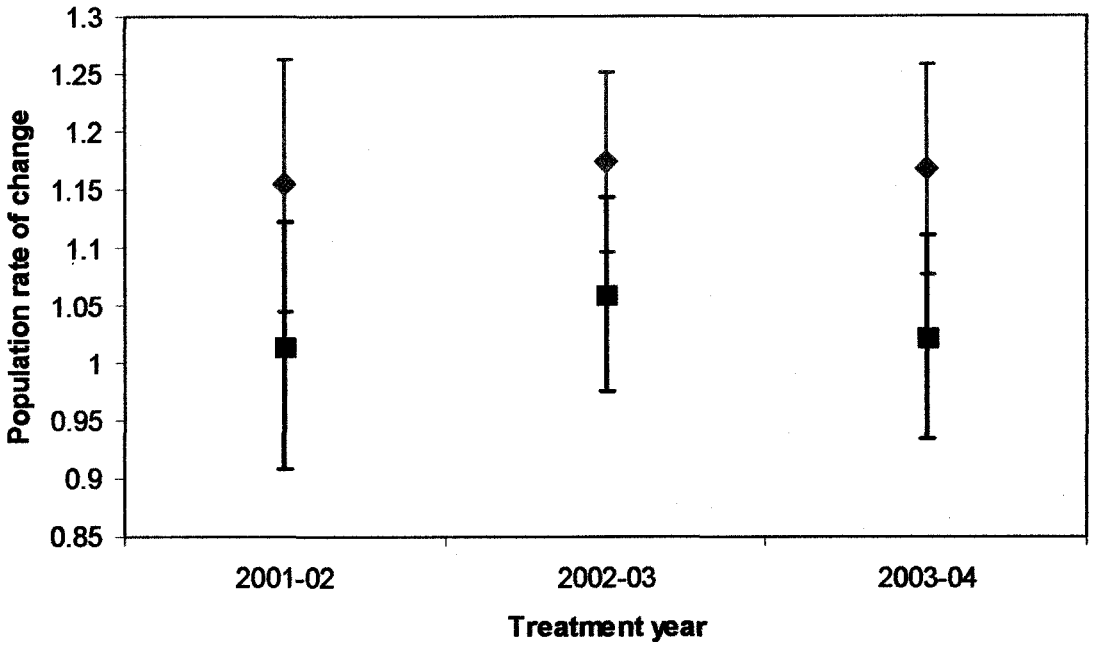

Figure 10. Estimates and 95% confidence intervals of the population rate of change, λ, as a function of enhanced nutrition during winter and early spring for mule deer in southwest Colorado, 2001–2004. Gray symbols and bars represent deer that received the nutrition treatment, whereas black symbols and bars represent deer that did not receive the treatment.

CHAPTER 4

EVALUATING MULE DEER BODY CONDITION DURING LATE WINTER USING SERUM THYROID HORMONE CONCENTRATIONS

Abstract: Body condition of ungulates is ultimately a determinant of fecundity and survival rates. Researchers have recently developed ultrasonography and body condition scoring techniques that allow reliable estimation of body fat in several ungulate species, but the approach is not feasible to employ in all circumstances, particularly in routine population monitoring programs. There remains a need for a reliable blood chemistry index that could be used to assess relative condition of different deer populations or groups. I evaluated the relationship between estimated body fat of free-ranging mule deer and serum concentrations of total thyroxine (T4), total triiodothyronine (T3), free T4 (FT4), and free T3 (FT3), during late February–early March in southwest Colorado. Deer body fat varied widely because I imposed a nutrition treatment on one-half of my sample. Concentrations of T4 and FT4 were 48.23 nmol/l (SE = 3.88) and 12.69 pmol/l (SE = 1.12) higher, respectively, in deer that received the nutrition treatment than deer that did not receive the treatment. My optimal model of estimated body fat included T4, $T4^2$, FT4 and deer chest girth ($\%Fat = -4.8015 - 0.0946 \times T4 + 0.000603 \times T4^2 + 0.1474 \times FT4 + 0.1426 \times$chest girth, $r^2 = 0.609$). Ultrasound and body condition scoring should be used to estimate body fat whenever possible. However, in cases where only a

blood sample can be obtained, I documented the potential utility of T4 and FT4 during late winter for evaluating relative body condition of mule deer.

Key Words: body condition, body fat, Colorado, mule deer, nutrition, *Odocoileus hemionus*, serum thyroid hormones, T3, T4, thyroxine, triiodothyronine, ultrasound.

4.1 INTRODUCTION

Body condition of ungulates relates directly to reproduction and survival. Body condition indices may be useful for wildlife managers when fecundity and survival rates cannot be measured directly. Various blood and urine chemistry variables have been evaluated as potential condition indices, but they are generally unreliable for one reason or another (Saltz et al. 1995, Cook et al. 2001*b*). More recently, ultrasound technology has facilitated direct measurements of fat and muscle layers in live animals, which along with a body condition score, have been calibrated to allow body fat estimation in moose (*Alces alces*), elk (*Cervus elaphus*), and mule deer (*Odocoileus hemionus*) (Stephenson et al. 1998, 2002; Cook et al. 2001*a*, *b*; Cook et al. 2007). This technique represents the optimal approach for estimating body condition in live animals. However, at least in the near term, several constraints may limit its widespread application: 1) portable ultrasound equipment is expensive (i.e., $10,000–$15,000 per unit), 2) formal training is required to prevent gross application errors, and 3) equipment and personnel logistics associated with the technique may hinder some capture operations in remote locations. In contrast, blood samples can be collected with minimal training and effort.

Serum concentrations of thyroid hormones are one of the few serum variables that have shown promise as a condition index (Bahnak et al. 1981; Watkins et al. 1982, 1983, 1991; Willard et al. 1998; Cook et al. 2001*a*). Serum thyroid hormone variables include total thyroxine (T4), total triiodothyronine (T3), free T4 (FT4), and free T3 (FT3). The hormones are released into the bloodstream in pulses that correspond to metabolic changes. Circannual changes in serum hormone concentrations have been documented in deer (Bahnak et al. 1981, Bubenik et al. 1983, Watkins et al. 1983). Watkins et al. (1991) and Cook et al. (2001*a*) documented correlations between thyroid hormones and percent body fat during winter in white-tailed deer (*O. virginianus*) and elk, respectively.

I evaluated the relationship between estimated body fat of free-ranging mule deer and T4, T3, FT4 and FT3, during late February–early March in southwest Colorado. I enhanced the nutrition of one-half of the deer in my sample during winter and spring (i.e., treatment), while the other half had access only to existing winter range habitat (i.e., control). Deer body fat varied widely because of the nutrition treatment, which made my dataset conducive to evaluating a condition index. My objectives were to determine 1) whether thyroid hormones were different between treatment and control deer, and 2) whether any of the thyroid hormone variables could be used to reasonably predict body fat in mule deer.

4.2 STUDY AREA

I conducted my research in southwest Colorado on the southern half of the Uncompahgre Plateau (Fig. 1). My winter range study area comprised 2 sites, or experimental units (EUs). The Colona EU (38°21'N, 107°49'W) was located south of Montrose, CO, and the Shavano EU (38°27'N, 108°01'W) was located west of Montrose.

I studied free-ranging deer, and therefore, defined EU boundaries from movements of radio-collared deer. The size of each EU ranged up to 22 km^2 when considering 90% of the radio-collared deer, and up to 40 km^2 when considering all deer. Winter range EUs were comprised of pinyon (*Pinus edulis*) and Utah juniper (*Juniperus osteosperma*) woodlands with interspersed big sagebrush (*Artemisia tridentata*) adjacent to irrigated agricultural fields. Elevations ranged from 1,830 m to 2,290 m. During my study, annual precipitation averaged 22.3 cm and the minimum temperature in January averaged –8.2° C in Montrose, Colorado (Western Regional Climate Center [WRCC] 2005), which is 60 m below the lowest winter range elevation in either EU. Deer occupied the winter range EUs from November through April each year.

4.3 METHODS

4.3.1 Adult Doe Capture and Handling

I captured 118 pregnant adult does during 26 February–2 March, 2003–2004 (2003: $n = 58$, 2004: $n = 60$), using helicopter net-gunning (Barrett et al. 1982, van Reenen 1982). I captured one-half of the deer in the Shavano EU and the other half in the Colona EU. During December–April, 2002–03 and 2003–04, I enhanced nutrition of deer in the Shavano EU using a supplemental pellet (Baker et al. 1998). The pellet was not supplemented with iodine; estimated iodine composition was <0.1 ppm (Ranch-Way Feeds, Fort Collins, CO). Deer in the Colona EU had access only to existing winter range habitat. The nutrition enhancement treatment was part of a larger study described in detail in Chapter 3.

All deer were hobbled and blind-folded prior to handling and ferried ≤3.5 km by helicopter to a central processing location. I measured maximum subcutaneous fat

thickness on the rump (cm) and thickness of the longissimus dorsi muscle (cm) of each doe using a SonoVet 2000 portable ultrasound unit (Universal Medical Systems, Bedford Hills, NY) with a 5 MHz linear transducer (Stephenson et al. 1998, 2002; Cook et al. 2001*a*). A small area of hair was plucked at each measurement point and lubricant was used to enhance contact between the transducer and skin. I also recorded body mass (kg) and chest girth (cm). The ultrasound procedures were performed in a 4.3 × 4.9-m wall-frame tent to minimize disturbance from helicopter rotor wash and adverse weather conditions and to create a dim environment for ultrasonography. I determined a body condition score (BCS) for each deer by palpating the rump (Cook et al. 2001*a*, 2007). I combined ultrasound measurements with the BCS score to estimate body fat of each deer (Cook et al. 2007).

I drew blood samples from the jugular vein of each captured deer. I separated and froze serum samples at –20°C, and submitted them to the Michigan State University Animal Health Diagnostic Laboratory (East Lansing, Michigan) for analysis of T4, T3, FT4, and FT3 concentrations. Serum hormone analytical procedures were described by Watkins et al. (1983, 1991). The laboratory did not know which samples were taken from treatment deer and which were taken from control deer. All deer capture and handling procedures were approved by the Colorado Division of Wildlife's Animal Care and Use Committee (project protocols 11–2000 and 1–2002).

4.3.2 Statistical Methods

I first evaluated whether mean values of T4, T3, FT4, and FT3 differed between treatment and control EUs. I knew a priori that estimated body fat of treatment deer was 7.26% (SE = 0.666) higher than control deer in 2003, and 3.35% (SE = 0.655) higher

than control deer in 2004 (Chapter 3). My initial analysis determined how effectively serum thyroid hormones could distinguish between a group of deer in good condition and a group in relatively poor condition.

I then modeled estimated percent body fat as a function of T4, FT4, T3, and FT3 using PROG REG in SAS (SAS Version 9.1, 2003) to evaluate how well serum hormones could predict estimated body fat. When developing my a priori model set, I did not consider models with greater than 3rd order polynomials. I used Akaike's information criterion adjusted for sample size (AIC_c) to select among candidate models (Burnham and Anderson 2002). I initially did not include other independent variables (e.g., body mass) because my ultimate goal was to determine how well a blood sample, by itself, could predict estimated body fat. However, even in helicopter net-gun captures in remote areas, handlers are often able to measure body mass and chest girth of adult deer with little trouble. Therefore, as a follow-up analysis, I added body mass and chest girth to my best models of thyroid hormone variables to evaluate relative improvement in predictive capability.

4.4 RESULTS

I found that T4, FT4, and T3 concentrations were higher in adult does that received the nutrition treatment than adult does that did not (Table 1). The largest differences were observed in T4 and FT4 (Table 2). Models of estimated body fat receiving any AIC_c weight each incorporated a 3rd order polynomial term. However, when I examined predicted values, these models were overfitting the data and not biologically reasonable. A few data points at the lowest fat levels and highest fat levels were causing the cubic polynomial models to receive most of the model weight. I

therefore removed these models from the model set. The optimal model for predicting estimated body fat included T4, $T4^2$, and FT4 ($\%\hat{Fat} = 7.540 - 0.0743 \times T4 + 0.000518 \times T4^2 + 0.155177 \times FT4$, $r^2 = 0.573$) (Table 3). Estimated percent body fat was linearly related to FT4 (Fig. 2), and curvilinearly related to T4 (Fig. 3). Chest girth, but not body mass, improved predictability of estimated body fat ($\%\hat{Fat} = -4.8015 - 0.0946 \times T4 + 0.000603 \times T4^2 + 0.1474 \times FT4 + 0.1426 \times$chest girth, $r^2 = 0.609$) (Table 4). Estimated percent body fat was linearly related to chest girth (Fig. 4).

4.5 DISCUSSION

I observed large differences in T4, FT4, and T3 between adult does that received enhanced nutrition and adult does that did not, during late February–early March (Table 2). Seal et al. (1972) documented differences in T4 between captive white-tailed deer maintained on high quality diets versus those on moderate quality diets, during March and April. Both deer groups had higher T4 levels than a group of wild deer fed cedar browse. Bahnak et al. (1981) observed higher levels of T3 and T4 in white-tailed deer given high-quality diets than deer given low-quality diets during winter and early spring. Similarly, Watkins et al. (1982) observed higher levels of T3 and T4 in fed white-tailed deer fawns than fasted deer fawns during April. My results, which are consistent with past research on captive white-tailed deer, suggest that blood samples could be used during late winter to assess relative condition of different deer populations, or relative condition of deer groups occupying different habitats. Body fat differences between my treatment and control deer were larger than what would be expected under natural conditions. However, in 2004, estimated body fat of treatment deer was only 3.35% (SE

= 0.655) higher than that of control deer, and differences in serum hormone concentrations were large.

I delivered the nutrition treatment in each EU during 2000–2004 as part of a crossover experimental design (Chapter 3), but I did not measure serum thyroid hormones until the final 2 years of research. I therefore lacked spatial replication of the treatment group effect, which means it is possible that inherent differences between the Shavano EU and Colona EU were partially responsible for the observed differences in serum thyroid hormones. However, the winter range EUs were separated by only 15 km, and radio-collared deer from the 2 EUs overlapped extensively on summer range. I lacked a biological explanation as to what other factor(s) would create such large differences between the 2 groups other than the imposed nutritional differences. More importantly, I observed strong relationships between estimated body fat and T4 and FT4, indicating that the observed differences between EUs were legitimate (Figs. 2, 3).

The relationship I found between body fat and serum thyroid hormones in mule deer during late winter was similar to that documented in white-tailed deer (Watkins et al. 1991) and elk (Cook et al. 2001*a*). However, Watkins et al. (1991) found the strongest relationship with T3 rather than T4. Serum thyroid hormones vary over time, and in particular, T4 is known to vary seasonally (Bahnak et al. 1981, Watkins et al. 1983). Watkins et al. (1991) used deer samples obtained from late fall through early spring, which may explain why they observed a weaker relationship between body fat and T4. Cook et al. (2001*a*) observed strong relationships between body fat and T4 during December and March, but they observed no relationship during September. Watkins (1980) found no relationship between serum thyroid hormone concentrations and plane of

nutrition (ad libitum versus 50% ad libitum) in white-tailed deer fawns during the fall. These results suggest serum thyroid hormone concentrations are most related to body fat when deer are in a catabolic state in late winter or early spring.

Chest girth was weakly related to estimated body fat (Fig. 4) and improved my optimum body fat model. Of note, body mass was not related to estimated body fat. Morphometric variables explained far less variation in body fat than hormone variables. Considering only single-variable models and using FT4 as the reference, the ΔAIC_c was 77.32 for chest girth and 82.20 for mass.

I recommend using ultrasound and BCS ratings to estimate body fat whenever possible (Stephenson et al. 1998, Cook et al. 2001*a*, *b*, Cook et al. 2007). However, there may be occasions where blood samples can be collected during winter or early spring but not ultrasound measurements. For example, the Colorado Division of Wildlife captures large samples of deer (i.e., 400–500) annually for estimating survival. In most cases, helicopter net-gun capture crews handle and release animals at the capture site. Handling generally involves placing a radio-collar on the deer, measuring mass, and collecting a blood sample. The procedure can be done quickly with minimal handling time. As such, it would not be logistically feasible or cost-effective to estimate body fat of deer using ultrasound and BCS ratings during each of these captures each year, particularly when survival is the primary objective. With adjustments in the timing of adult doe captures (i.e., late winter instead of early winter), serum thyroid hormone analyses could be used to monitor relative herd condition, and possibly to evaluate changes in herd condition relative to landscape-scale habitat treatments over time. Samples would need to be collected at the same time each year to be comparable across years and areas.

4.6 MANAGEMENT IMPLICATIONS

Ultrasound and BCS ratings provide superior estimates of body fat (Stephenson et al. 1998, Cook et al. 2001*a*, *b*, Cook et al. 2007) but may not always be feasible to obtain. I found that serum thyroid hormones are highly correlated with estimated body fat in mule deer during late winter. Concentrations of T4, FT4, and T3 could be useful for evaluating relative condition of different deer groups or populations, and for roughly estimating body fat of individual animals during late winter. Chest girth improved predictability of my optimum body fat model, and therefore should be measured anytime blood samples are collected for evaluating body condition. Serum thyroid hormones may be most useful as a body condition index in large scale population monitoring where deer are routinely captured on an ongoing basis.

4.7 ACKNOWLEDGMENTS

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Table 1. Estimated serum concentrations of total thyroxine (T4, nmol/l), total triiodothyronine (T3, nmol/l), free T4 (FT4, pmol/l), and free T3 (FT3, pmol/l) of adult doe mule deer occupying a pinyon-juniper winter range during late February–early March in southwest Colorado, USA, 2003–2004. Approximately one-half of the adult does received enhanced nutrition via supplementation (treatment) whereas the other half received no supplementation (control).

			Hormone concentration estimates			
Year	group	*n*	T4 (SE)	FT4 (SE)	T3 (SE)	FT3 (SE)
2003	Treatment	30	146.57 (3.53)	29.97 (1.27)	1.647 (0.058)	4.097 (0.130)
	Control	28	92.32 (3.56)	17.07 (0.65)	1.418 (0.080)	3.707 (0.210)
2004	Treatment	30	131.93 (4.48)	24.83 (1.39)	2.077 (0.075)	4.210 (0.154)
	Control	30	90.03 (3.54)	12.50 (0.59)	1.700 (0.104)	4.247 (0.672)

Table 2. Differences in serum concentrations of total thyroxine (T4, nmol/l), total triiodothyronine (T3, nmol/l), free T4 (FT4, pmol/l), and free T3 (FT3, pmol/l) between adult doe mule deer that received enhanced winter nutrition (treatment) and adult does that did not (control), in southwest Colorado, USA, 2003–2004. Differences reflect the effect of enhanced nutrition on serum thyroid hormones.

		Difference (Treatment – Control)		
Variable	Year	Estimate	95% LCL	95% UCL
T4	2003	54.245	44.210	64.281
	2004	41.898	30.475	53.320
FT4	2003	12.895	10.037	15.754
	2004	12.333	9.313	15.353
T3	2003	0.229	0.031	0.427
	2004	0.377	0.120	0.634
FT3	2003	0.389	–0.105	0.883
	2004	–0.037	–1.415	1.342

Table 3. Model selection results, based on Akaike's Information Criterion with small sample size correction (AIC_c), from an analysis of adult doe mule deer body fat (%) as a function of serum thyroid hormones in southwest Colorado, USA, 2003–2004. Serum thyroid hormone variables were total thyroxine (T4, nmol/l), total triiodothyronine (T3, nmol/l), free T4 (FT4, pmol/l), and free T3 (FT3, pmol/l).

Model	No. parameters	AIC_c	ΔAIC_c	Akaike wt
T4, $T4^2$, FT4	4	210.29	0.00	0.599
T4, FT4, T3, FT3	5	212.54	2.25	0.195
T4, FT4	3	213.57	3.28	0.116
T4, FT4, T3	4	214.68	4.39	0.067
T4, $T4^2$	3	217.82	7.53	0.014
FT4, FT3	3	219.59	9.30	0.006
FT4	2	221.23	10.94	0.003
FT4, $FT4^2$	3	223.19	12.89	0.001
T4	2	223.65	13.36	0.001
T3	2	303.23	92.94	0.000
FT3	2	304.44	94.15	0.000
Intercept only	1	305.46	95.17	0.000

Table 4. Model selection results, based on Akaike's Information Criterion with small sample size correction (AIC_c), from an analysis of adult doe mule deer body fat (%) as a function of serum thyroid hormones, chest girth (cm, chest), and body mass (kg, mass) in southwest Colorado, USA, 2003–2004. Serum thyroid hormone variables were total thyroxine (T4, nmol/l), total triiodothyronine (T3, nmol/l), free T4 (FT4, pmol/l), and free T3 (FT3, pmol/l).

Model	No. parameters	AIC_c	ΔAIC_c	Akaike wt
T4, $T4^2$, FT4, chest	5	202.20	0.00	0.599
T4, $T4^2$, FT4, chest, mass	6	204.24	2.04	0.216
T4, $T4^2$, FT4, mass	5	206.01	3.81	0.089
T4, FT4, T3, FT3, chest	6	206.28	4.08	0.078
T4, $T4^2$, FT4	4	210.29	8.09	0.011
T4, FT4, T3, FT3	5	212.54	10.34	0.003
FT4	2	221.23	19.03	0.000
T4	2	223.65	21.45	0.000
Chest	2	298.55	96.35	0.000
T3	2	303.23	101.03	0.000
Mass	2	303.43	101.23	0.000
FT3	2	304.44	102.24	0.000
Intercept only	1	305.46	103.26	0.000

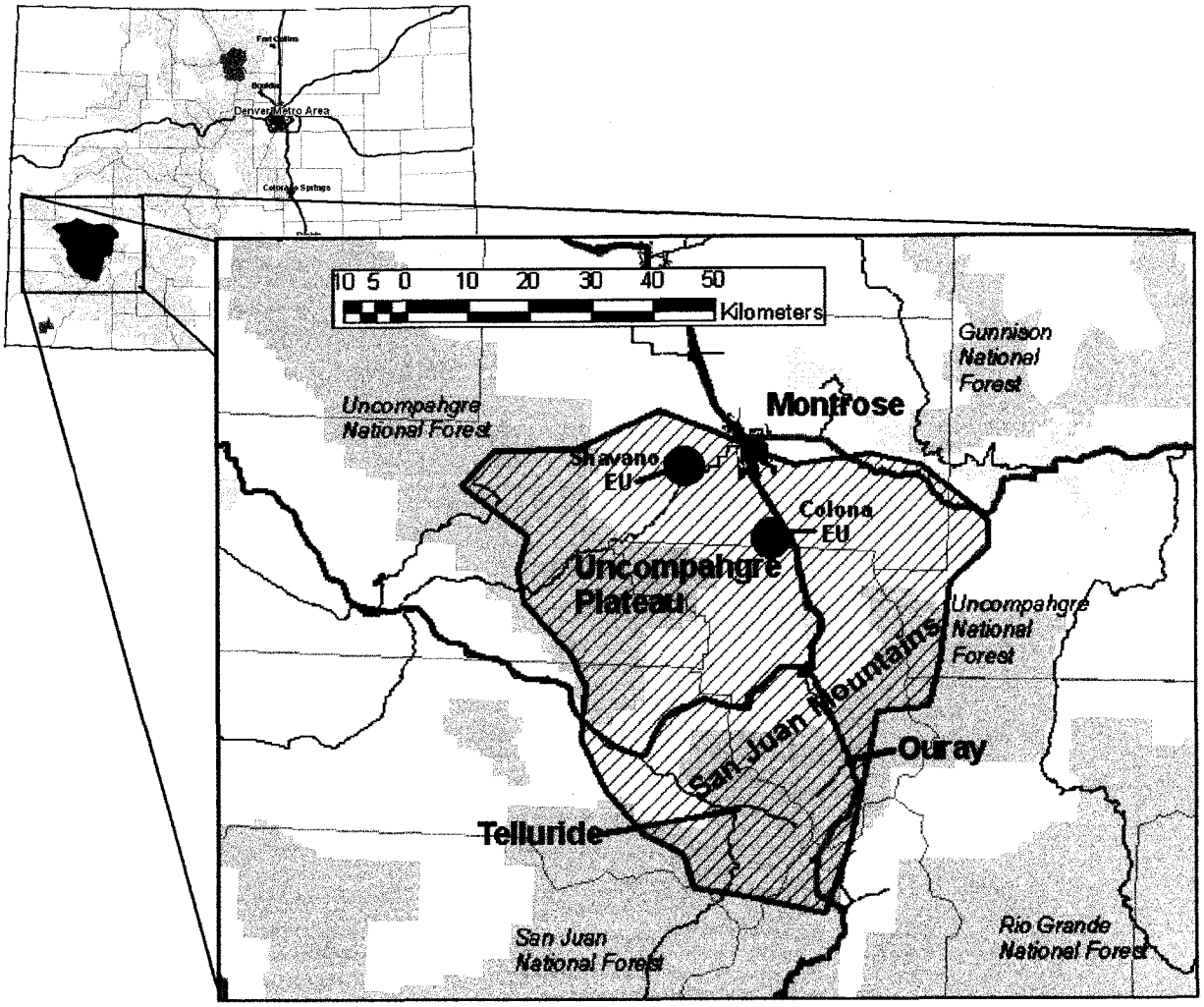

Figure 1. Location of winter range experimental units (EU; ●) on the Uncompahgre Plateau in southwest Colorado, where I studied serum thyroid hormones in mule deer, 2003–2004. Summer range for these deer is also indicated (▨).

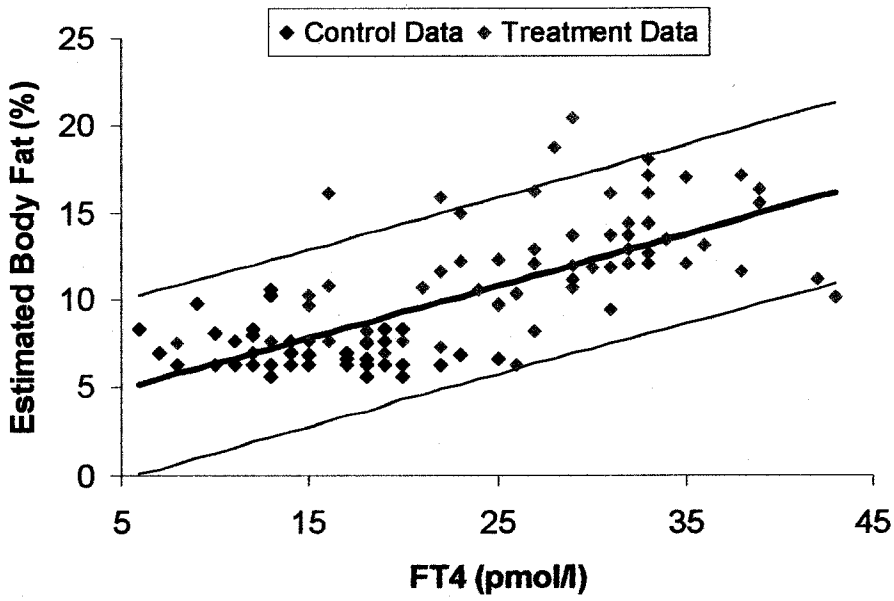

Figure 2. Predicted relationship and 95% confidence interval between estimated body fat and serum concentrations of free thyroxine (FT4) in mule deer, southwest Colorado, 2003–2004. One-half of the deer received enhanced nutrition (treatment, ♦) whereas the other half did not (control, ♦).

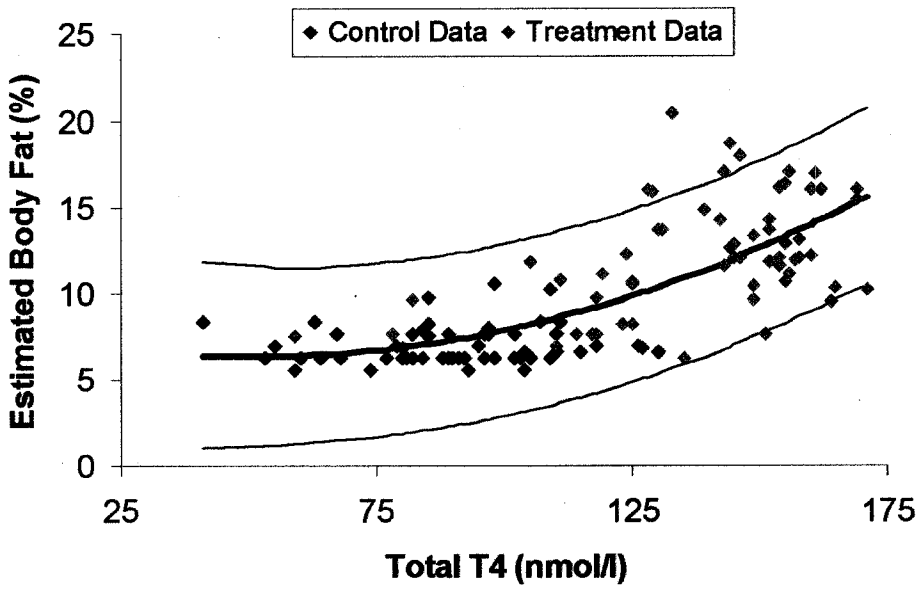

Figure 3. Predicted relationship and 95% confidence interval between estimated body fat and serum concentrations of total thyroxine (T4) in mule deer, southwest Colorado, 2003–2004. One-half of the deer received enhanced nutrition (treatment, ♦) whereas the other half did not (control, ♦).

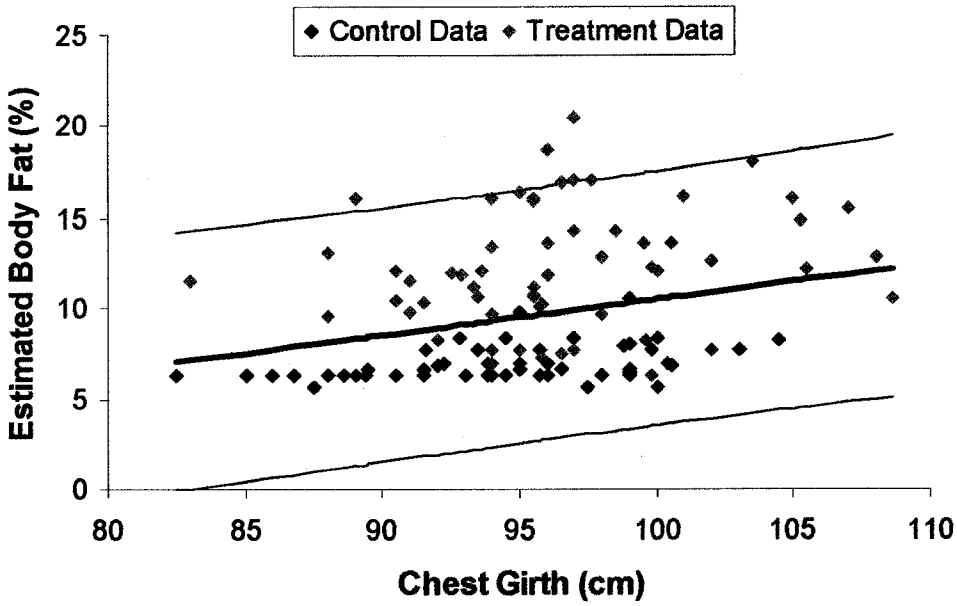

Figure 4. Predicted relationship and 95% confidence interval between estimated body fat and chest girth of mule deer, southwest Colorado, 2003–2004. One-half of the deer received enhanced nutrition (treatment, ♦) whereas the other half did not (control, ♦).