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Source: Journal of Wildlife Diseases, 35(3) : 496-510

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-35.3.496

*Journal of Wildlife Diseases*, 35(3), 1999, p. 496–510


# HEALTH EVALUATION OF A PRONGHORN ANTELOPE POPULATION IN OREGON

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ABSTRACT: During 1996 and 1997, the U.S. Fish and Wildlife Service conducted a study to determine the cause(s) of population decline and low survival of pronghorn antelope (*Antilocapra americana*) fawns on Hart Mountain National Antelope Refuge (HMNAR) located in southeastern Oregon (USA). As part of that study, blood, fecal, and tissue samples from 104 neonatal fawns, 40 adult does, and nine adult male pronghorns were collected to conduct a health evaluation of the population. Physiological parameters related to nutrition and/or disease were studied. No abnormalities were found in the complete blood cell counts of adults ($n = 40$) or fawns ($n = 44$ to 67). Serum total protein and blood urea nitrogen (BUN) levels were lower compared to other pronghorn populations. Does had mean BUN values significantly lower ($P < 0.001$) in December 1996 than March 1997. Serum copper (Cu) levels in does (range 0.39 to 0.74 ppm) were considered marginal when compared to domestic animals and other wild ungulates. Fawns had low (0.28 ppm) Cu levels at birth and reached the does' marginal values in about 3 days. Whole blood, serum and liver selenium (Se) levels were considered marginal to low in most segments of the pronghorn population. However, serum levels of vitamin E (range 1.98 to 3.27 μg/ml), as determined from the does captured in March, were apparently sufficient to offset any signs of Se deficiency. No clinical signs of Cu or Se deficiency were observed. Fifty-five of 87 dead fawns were necropsied. Trauma, due to predation by coyotes (*Canis latrans*), accounted for 62% of the mortality during mid-May to mid-July of each year. Other causes included predation by golden eagles (*Aquila chrysaetos*) (4%), dystocia (2%), septicemic pasteurellosis (4%), starvation (5%), and unknown (23%). Adult females were tested for serum neutralizing antibodies to *Brucella* spp. ($n = 20$, negative), *Leptospira interrogans* ($n = 20$, negative), bluetongue virus ($n = 20$, 35% positive), epizootic hemorrhagic disease virus ($n = 20$, 30% positive), respiratory syncytial virus ($n = 18$, negative), parainfluenza virus type 3 ($n = 18$, 67% positive), infectious bovine rhinotracheitis ($n = 18$, negative), and bovine viral diarrhea ($n = 18$, negative). Considering the parameters examined, we found no apparent predisposing factors to mortality including those killed by coyotes, but some nutritional parameters suggest that pronghorns on HMNAR exist on a diet low in protein and Se and marginal in Cu. The effect these factors have on the population is not known.

*Key words:* Pronghorn antelope, *Antilocapra americana*, hematology, serum chemistry, trace minerals.

## INTRODUCTION

From 1990 to 1995, pronghorn antelope or pronghorns (*Antilocapra americana*) numbers on the U.S. Fish and Wildlife Service, Hart Mountain National Antelope Refuge (HMNAR) located in southeastern Oregon (USA) (42°30′N, 119°40′W) declined from 1,933 to 1,376 (29%). In mid-July of 1995, the fawn : doe ratio was <1:100, the lowest recorded in nearly 40 yr of observation (U.S. Fish and Wildlife Service, unpubl. data). Poor nutrition, predation, disease, and adverse weather during fawning have all been identified as potential factors influencing pronghorn fawn survival in different locations (Ellis, 1970; Beale and Smith, 1973; Barrett, 1978; McNay, 1980; Trainer et al., 1983).

Personnel of the HMNAR in cooperation with the Oregon Department of Fish and Wildlife, and U.S. Geological Survey's National Wildlife Health Center (NWHC), Madison, Wisconsin (USA) conducted a study in 1996 and 1997 to determine cause(s) of population decline and low sur-

vival of pronghorn fawns. As part of that study, we conducted a health evaluation of pronghorns on the HMNAR to aid managers in identifying if poor nutrition or disease were factors related to the poor fawn survival and recent population decline, to establish baseline biomedical parameters to be used in future monitoring, and provide recommendations for resource managers to enhance population growth.

## MATERIALS AND METHODS

Information was collected from 144 pronghorns, 104 neonatal fawns and 40 adult does, captured at HMNAR during 1996 and 1997. Additionally, liver samples were collected from nine adult males killed by hunters on the HMNAR in October 1996. Fifty-two fawns were captured each year during the early fawning period of mid to late May by search crews with hand held nets. When parturition was observed, no capture attempt of fawns was made for at least 4 hr to allow imprinting (Autenrieth and Fichter, 1975). Does were captured by a net gun (Coda Enterprises, Inc., Mesa, Arizona, USA) fired from a helicopter during December 1996 ($n = 20$) and late March 1997 ($n = 20$). Age was determined from known birth dates for some fawns or estimated by using behavioral criteria and condition of pelage, umbilical cord, and hooves (Von Gunten, 1978; Trainer et al., 1983) for others. Age of does was determined from tooth eruption and wear (Dimmick and Pelton, 1996) or by examination of cementum annuli (McCuthen, 1969) of incisors extracted during necropsy. Pregnancy of each doe captured in March ($n = 20$) was determined by ultrasound and verified at necropsy in 14 does that died subsequent to capture. Fawns were weighed to the nearest 0.1 kg, and examined grossly for abnormalities including signs of disease or poor condition.

Radio transmitters (Advanced Telemetry System, Isanti, Minnesota, USA) affixed to plastic ear-tags were attached to fawns captured in 1996 and 1997. Radio transmitters (Telonics, Inc., Mesa, Arizona, USA) attached to neck collars were used on does captured in March 1997. Does captured in December 1996 were marked with ear tags without radio transmitters. Marked fawns were observed twice daily until they died or until mid-July, when radio transmitters lost power. Transmitters were equipped with a mortality sensor that activated after the transmitter was stationary for 2 hr. When monitoring indicated death, fawn carcasses were found and removed within 1 to 4 days. The delay in recovering some dead fawns was due to inaccessible terrain and low transmitter output.

Blood samples (4 to 6 ml) were drawn by jugular venipuncture from 82 (32 in 1996 and 50 in 1997) fawns and 40 does. Blood samples were collected in potassium salt of ethylenediaminetetraacetic acid (EDTA) for complete blood cell (CBC) counts and analysis of selenium (Se) concentrations and in serum separator tubes for serum chemistry analysis, other trace mineral concentrations including copper (Cu), molybdenum (Mo), and iron (Fe), and serology. Vitamin E levels were determined on serum collected from 10 of the does captured in March 1997. Samples were chilled and either hand carried to a local laboratory (for CBC and serum chemistries) or frozen at −20 C and later sent by overnight delivery (for trace minerals, vitamin E, and serological analysis) to other laboratories.

Complete blood cell counts and serum chemistries were performed at Lake District Hospital, Lakeview, Oregon, on automated analyzers (Coulter T 660, Coulter Electronics, Inc. Hialeah, Florida, USA; opeRA analyzer, Medium system, Bayer Diagnostic, Tarrytown, New York, USA). Whole blood, serum, or liver was analyzed at the Animal Diagnostic Laboratory, Michigan State University (MSU), (East Lansing, Michigan, USA) for trace minerals including Cu, Mo, Fe, and Se, and vitamin E. Trace mineral concentrations in liver were determined using inductively coupled plasma atomic emission spectrometry (ICP-AES) using the method of Stowe et al. (1985) and in serum using the method of Melton et al. (1990). Selenium levels in whole blood were determined using a fluorometric method (Phosphoric Acid Method, Reamer and Veillon, 1983). Vitamin E in serum was determined by high pressure liquid chromatography (HPLC) (Widicus and Kirk, 1979). Liver Se values from the males were originally obtained on dry weight basis while liver Se values from the does were determined on wet weight basis. In order to compare both values, ppm dry weight values were converted to ppm wet weight values. For most tissues, the approximate wet weight value is equal to dry weight value/3.5 (Puls, 1994).

Serological analysis for selected disease agents was performed by National Veterinary Services Laboratory (NVSL; Ames, Iowa, USA) on serum samples collected from 20 does captured in December 1996. Serum samples were tested for antibodies to *Brucella* spp., *Leptospira interrogans,* serovars *canicola, grippotyphosa, hardjo, icterohemorrhagiae,* and *pomona,* bluetongue virus (BTV), epizootic hemorrhagic disease virus (EHDV), respiratory syn-

cytial virus (RSV), parainfluenza virus type 3 (PI3), infectious bovine rhinotracheitis virus (IBR), and bovine viral diarrhea virus (BVD). The card test (U.S. Department of Agriculture, no date) was used for *Brucella* spp. The microagglutination test (NVSL, 1997) was used for *Leptospira* serovars with ≥1:100 considered positive. The agar gel immunodiffusion (AGID) test (Pearson and Jochim, 1979) was used for BTV and EHDV. The serum neutralization (SN) test (Leannette and Schmidt, 1979) was used for RSV, PI3, IBR, and BVD with ≥1:8 considered positive. Serotyping of BTV and EHDV was conducted at NVSL using SN (NVSL, 1998).

Fecal samples were collected from does at time of capture in December 1996 ($n = 10$) and March 1997 ($n = 10$). Feces were sent to the Washington Animal Disease Diagnostic Laboratory, Washington State University, Pullman, for analysis by fecal flotation. Eggs (EPG) or oocysts (OPG) per gram of feces were determined. Fecal samples were also sent to the Wildlife Habitat Laboratory, Washington State University (Pullman, Washington, USA) for analysis of fecal nitrogen (FN) which is an index to protein intake (Leslie and Starkey, 1987).

Data were analyzed using SPSS (SPSS Inc., Chicago, Illinois, USA, 1989–92). Means were compared using one-way analysis of the variance or a Student's *t*-test. Data are presented as means and standard deviations (±SD). A three-way analysis of variance considering age, year, and sex was applied to fawn data. Statistical differences were considered significant when $P < 0.05$. A Weibull survival model (Allison, 1995) was used to determine the relationship between mortality and body weights of fawns each year. Weight was entered into the model as a continuous variable.

Partial to complete carcasses were necropsied ($n = 28$ in 1996, $n = 27$ in 1997) to determine probable cause(s) of death. Gross external and internal examinations were conducted on each carcass to identify traumatic injuries and lesions suggestive of diseases and to assess body condition based on fat reserves. Cause of death due to predation, and which predator was involved, was determined by gross examination of the carcass using criteria established by O'Gara (1978). Tissues and swabs collected from carcasses at necropsy were cultured on 5% sheep blood agar (BA) and eosin methylene blue (EMB) agar (Remel, Lenexa, Kansas, USA) and incubated in ambient air at 35 to 37 C for 18 to 24 hr. All bacterial colonies were screened based on typical colony morphology. Isolates were biochemically characterized and identified using the appropriate API system (bioMerieux, St. Louis, Missouri, USA). Isolates of *Pasteurella multocida* were serotyped using antisera from NVSL (Heddleston et al., 1972). All samples being screened for *Salmonella* were initially enriched for using Dulcitol-Selenite Broth (Raj, 1966) incubated at 42 ± 0.5 C for 16 to 18 hr. A portion of the enrichment was transferred to both Hektoen-Enteric Agar (Remel, Lenexa, Kansas) and XLT4 Agar (Difco Laboratories, Detroit, Michigan, USA) and incubated at 35–37 C for 18 to 24 hr. All bacterial colonies were screened to identify *Salmonella* spp. based on typical colony reactions and morphology. Suspected *Salmonella* isolates were biochemically characterized by the API-20E bioMerieux system. Isolates were serotyped at NVSL. In attempts to determine cause of death of fawns where cause of death could not be determined grossly, or in some cases to verify cause of death, various organ tissues were submitted to NWHC for virus isolation, histopathology, and bacterial cultures. For virus isolation, cell cultures and embryonated eggs were used. For histopathology, tissues were fixed in 10% neutral buffered formalin, embedded in paraffin, sectioned at 5 um for light micropsy, and stained with hematoxylin and eosin. Selected tissues, including kidney, liver, heart, and fetuses, from eight of 14 does that died in April 1997, following their capture in March, were analyzed grossly and histologically at the Idaho Department of Fish and Game Wildlife Health Laboratory (Caldwell, Idaho, USA), to determine cause of death.

## RESULTS

Mean age of fawns captured in 1996 was 2.4 days compared to 1.6 days in 1997. Ages of does captured in December were ≥4 yr, $n = 18$; 3 yr, $n = 1$; 2 yr, $n = 1$. Ages of does captured in March were ≥4 yr, $n = 20$. Analysis of cementum annuli from 10 of the 14 does that died after their capture in March indicated a mean age of nearly 8 yr. The males killed by hunters were all adults.

Mean weight of 1- to 3-day-old fawns in 1996 ($n = 24$) was 4.6 ± 0.6 kg (range 3.2 to 5.5) and 4.3 ± 0.7 kg (range 3.0 to 6.5 kg) in 1997 ($n = 52$). We found no association between fawn weight and mortality. No clinical signs were observed in fawns that could be attributed to unhealthy fawns or other disease.

TABLE 1. Hematological parameters and reference values of adult pronghorn does from Hart Mountain National Antelope Refuge, Oregon, 1996–97.

| Parameter (units)[a] | December 1996 mean (SD) ($n$ = 18) | March 1997 mean (SD) ($n$ = 19) | Barrett and Chalmers, 1977[a] mean ($n$ = 80) |
|---|---|---|---|
| Red blood cells ($\times 10^6/\mu l$) | 11.67 (0.90) | 11.33 (0.68) | 11.51 |
| Hematocrit (%) | 54.45 (4.55) | 53.85 (3.59) | 52.44 |
| Hemoglobin (g/dl) | 19.72 (1.32) | 19.07 (1.28) | 18.65 |
| MCH (pg) | 16.91 (0.57) | 16.82 (0.27) | 16.47 |
| MCHC (g/dl) | 36.29 (1.65)[b] | 35.41 (0.70)[b] | 36.02 |
| MCV (fl) | 46.63 (0.98)[b] | 47.52 (1.09)[b] | 46.43 |
| White blood cells ($\times 10^3/\mu l$) | 5.17 (1.70) | 5.41 (2.16) | 5.02 |
| Lymphocytes (%) | 54.0 (20.4) | 38.0 (17.9) | 31.5 |
| Lymphocytes ($\times 10^3/\mu l$) | 2.70 (0.86) | 1.90 (0.93) | |
| Monocytes (%) | 2.0 (3.3) | 1.5 (2.4) | 0.3 |
| Monocytes ($\times 10^3/\mu l$) | 0.10 (0.16) | 0.09 (0.13) | |
| Band neutrophils (%) | 0.3 (0.5) | 0.8 (1.4) | 2.1 |
| Band neutrophils ($\times 10^3/\mu l$) | 0.02 (0.03) | 0.04 (0.08) | |
| Segmented neutrophils (%) | 45.4 (20.5) | 56.2 (17.1) | 63.6 |
| Segmented neutrophils ($\times 10^3/\mu l$) | 2.49 (1.70) | 3.04 (1.41) | |
| Eosinophils (%) | 0.2 (0.4)[b] | 3.4 (5.9)[b] | 1.8 |
| Eosinophils ($\times 10^3/\mu l$) | 0.01 (0.25)[b] | 0.20 (0.37)[b] | |
| Basophils (%) | 0.7 (0.3) | 0.4 (1.5) | 0.4 |
| Basophils ($\times 10^3/\mu l$) | 0.0 | 0.02 (0.08) | |
| N/L ratio | 1.15 (1.08) | 2.26 (1.90) | |

[a] MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; N/L ratio, neutrophil/lymphocyte ratio.
[b] Significant difference ($P < 0.05$) between values in December and March.

Hematologic parameters for does and fawns are presented in Tables 1 and 2, respectively. Although there were significant statistical differences in the mean corpuscular volume (MCV) ($P = 0.01$), mean corpuscular hemoglobin concentration (MCHC) ($P = 0.04$) and number of eosinophils ($P = 0.04$) between samples from December and March in the does, they were not considered clinically relevant. Also, a lower number of lymphocytes was observed in the samples from March ($P = 0.05$).

No statistical differences in the hematologic parameters were observed between the fawns from 1996 and 1997 once the age difference between years ($\bar{x}$ = 2.2 days, $n$ = 17 compared to 1.7, $n$ = 44) was considered, therefore data were combined (Table 2). No differences attributed to sex (34 M, 27 F) or between fawns that lived or died were found. In the fawns, MCV and hematocrit fell during the first 3 days. One-day-old fawns had a higher ($P < 0.001$) MCV (46.5 fl) and hematocrit ($P = 0.01$) (45.6%) values than those measured in 3-day-old fawns, 43.9 fl and 42%, respectively. Hemoglobin concentrations and red blood cells counts remained the same during the first 3 days.

Serum biochemical parameters for does are presented in Table 3. Mean blood urea nitrogen (BUN) values were lower ($P < 0.001$) in December (15.1 mg/dl; range 11.0 to 21.0 mg/dl) compared with March (31.2 mg/dl; range 25.0 to 43.0 mg/dl). Mean phosphorus and alkaline phosphatase values also were higher ($P < 0.02$) in March. In contrast, mean albumin, creatinine, cholesterol, sodium, and total bilirubin concentrations were lower ($P \leq 0.04$) in March. Other serum chemistry values were not considered to be significantly different between the two months.

In fawns, no differences in serum biochemical values were observed between 1996 and 1997 once the age difference was considered, therefore data were combined

TABLE 2. Hematological parameters and reference values for neonatal pronghorn fawns from Hart Mountain Antelope Refuge, Oregon, 1996–97.

| Parameter (units)[a] | *n* | Mean (SD) | Min. | Max. | Barrett and Chalmers, 1979. mean (SE) |
|---|---|---|---|---|---|
| Red blood cells ($\times 10^6/\mu l$) | 66 | 9.82 (1.02) | 7.68 | 11.78 | 9.68 (0.09) |
| Hematocrit (%) | 67 | 44.0 (4.5) | 33.7 | 54.5 | 39.7 (0.38) |
| Hemoglobin (g/dl) | 67 | 14.9 (1.3) | 11.9 | 17.3 | 14.56 (0.16) |
| MCH (pg) | 66 | 15.20 (1.13) | 12.63 | 17.23 | 15.12 (0.14) |
| MCHC (g/dl) | 66 | 33.90 (1.77) | 29.36 | 38.39 | 37.12 (0.37) |
| MCV (fl) | 66 | 44.90 (3.11) | 34.22 | 49.30 | 41.25 (0.39) |
| White blood cells ($\times 10^3/\mu l$) | 64 | 4.9 (3.1) | 0.3 | 14.7 | 3.97 (0.17) |
| Lymphocytes (%) | 66 | 31 (18) | 2 | 88 | 33.9 (1.30) |
| Lymphocytes ($\times 10^3/\mu l$) | 62 | 1.33 (1.01) | 0.13 | 5.07 | |
| Monocytes (%) | 66 | 3 (2) | 0 | 10 | 0.8 (0.12) |
| Monocytes ($\times 10^3/\mu l$) | 62 | 0.10 (0.10) | 0 | 0.42 | |
| Band neutrophils (%) | 66 | 1 (1) | 0 | 6 | 3.0 (0.2) |
| Band neutrophils ($\times 10^3/\mu l$) | 62 | | 0 | 0.26 | |
| Segmented neutrophils (%) | 66 | 66 (17) | 12 | 93 | 59.9 (1.3) |
| Segmented neutrophils ($\times 10^3/\mu l$) | 62 | 3.36 (2.23) | 0.34 | 10.84 | |
| Eosinophils (%) | 66 | 0.6 (1.3) | 0 | 6.0 | 1.8 (0.19) |
| Eosinophils ($\times 10^3/\mu l$) | 62 | | 0 | 0.13 | |
| N/L ratio | 66 | 4.4 (6.7) | 0.1 | 46.5 | |
| Platelets ($\times 10^3/\mu l$) | 44 | 722 (27) | 258 | 1,518 | |

[a] MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; N/L ratio, neutrophil/lymphocyte ratio.

TABLE 3. Biochemical parameters and reference values of adult pronghorn does from Hart Mountain National Antelope Refuge, Oregon, 1996–97.

| Parameter (units) | December 1996 mean (SD) ($n = 20$) | March 1997 mean (SD) ($n = 20$) | Barrett and Chalmers, 1977b mean ($n = 106$) |
|---|---|---|---|
| Sodium (meq/L) | 159.10 (3.61)[a] | 156.30 (4.44)[a] | 167.01 |
| Potassium (meq/L) | 4.55 (0.58) | 4.55 (0.46) | 6.99 |
| Chloride (meq/L) | 113.15 (2.18) | 112.15 (2.68) | |
| Calcium (mg/dl) | 9.14 (1.29) | 8.91 (0.60) | 11.62 |
| Phosphorus (mg/dl) | 6.62 (1.55)[a] | 7.71 (0.94)[a] | 6.42 |
| Magnesium (mg/dl) | 2.89 (0.23) | 3.00 (0.78) | 2.46 |
| Total protein (g/dl) | 6.15 (0.55) | 5.92 (0.49) | 7.32 |
| Albumin (g/dl) | 4.26 (0.40)[a] | 3.91 (0.25)[a] | 4.75 |
| Globulin (g/dl) | 1.88 (0.28) | 2.01 (0.46) | |
| Albumin:globulin ratio | 2.30 (0.32) | 2.05 (0.52) | 1.97 |
| Gamma-glutamyl transferase (U/L) | 20.05 (11.51) | 16.35 (8.86) | |
| Alkaline phosphatase (U/L) | 49.25 (25.04)[a] | 75.75 (39.12)[a] | |
| Alanine aminotransferase (U/L) | 25.20 (9.64) | 23.95 (5.24) | 18.20 |
| Aspartate aminotransferase (U/L) | 226.55 (116.12) | 234.20 (132.70) | 251.50 |
| Lactate dehydrogenase (U/L) | 491.80 (161.89) | 441.45 (80.77) | |
| Glucose (mg/dl) | 202.95 (40.73) | 187.00 (30.54) | 249.95 |
| Creatinine (mg/dl) | 1.84 (0.31)[a] | 1.37 (0.20)[a] | 11.60 |
| Blood urea nitrogen (mg/dl) | 15.10 (2.77)[a] | 31.20 (5.36)[a] | 42.82 |
| Total bilirubin (mg/dl) | 0.31 (0.23)[a] | 0.18 (0.08)[a] | |
| Cholesterol (mg/dl) | 42.00 (6.31)[a] | 37.40 (6.79)[a] | 40.78 |

[a] Significant difference ($P < 0.05$) between values in December and March.

TABLE 4. Biochemical parameters and reference values of pronghorn fawns from Hart Mountain Antelope Refuge, Oregon, 1996–97.

| Parameter (units) | *n* | Mean (SD) | Min. | Max. | Barrett and Chalmers, 1979. mean (SE) |
|---|---|---|---|---|---|
| Sodium (meq/L) | 67 | 147.2 (2.9) | 139.0 | 154.0 | 145.21 (1.53) |
| Potassium (meq/L) | 67 | 4.6 (0.6) | 3.5 | 6.2 | 6.23 (0.09) |
| Chloride (meq/L) | 67 | 109.0 (2.6) | 102.0 | 114.0 | |
| Calcium (mg/dl) | 74 | 10.5 (0.7) | 9.1 | 12.8 | 12.39 (0.15) |
| Phosphorus (mg/dl) | 73 | 10.4 (1.2) | 7.8 | 13.4 | |
| Magnesium (mg/dl) | 73 | 2.04 (0.20) | 1.52 | 2.61 | 2.20 (0.03) |
| Total protein (g/dl) | 74 | 4.6 (0.6) | 3.3 | 5.9 | 4.78 (0.08) |
| Albumin (g/dl) | 74 | 1.9 (0.4) | 1.2 | 3.8 | 2.36 (0.05) |
| Globulin (g/dl) | 74 | 2.8 (0.6) | 1.7 | 4.4 | |
| Albumin:globulin ratio | 74 | 0.72 (0.28) | 0.32 | 2.23 | |
| Gamma-glutamyl transferase (U/L) | 73 | 231 (352) | 11 | 1,945 | |
| Alkaline phosphatase (U/L) | 73 | 744 (428) | 323 | 2,688 | |
| Alanine aminotransferase (U/L) | 74 | 15.8 (8.6) | 6.0 | 75.0 | |
| Aspartate aminotransferase (U/L) | 74 | 115.5 (32.2) | 56.0 | 249.0 | 106.5 |
| Lactate dehydrogenase (U/L) | 71 | 914 (231) | 531 | 1,809 | |
| Glucose (mg/dl) | 73 | 161 (40) | 60 | 292 | 203.5 (11.27) |
| Creatinine (mg/dl) | 74 | 0.9 (0.5) | 0.3 | 3.4 | 2.37 (0.85) |
| Blood urea nitrogen (mg/dl) | 74 | 15.8 (6.7) | 5.0 | 29.0 | 21.32 (1.33) |
| Total bilirubin (mg/dl) | 74 | 2.5 (1.2) | 0.5 | 5.6 | |
| Cholesterol (mg/dl) | 65 | 55.9 (23.3) | 19.0 | 107.0 | 67.40 (3.58) |

(Table 4). No differences attributed to sex (35 M, 31 F) or between fawns that lived or died were found. Blood urea nitrogen levels in fawns were negatively correlated with age with values of 20.5 ± 4.4 mg/dl, 15.1 ± 6.9 mg/dl and 11.3 ± 5.9 mg/dl for animals that were 1-, 2-, and 3-day-old, respectively. Creatinine levels also decreased between the first and the second day (1.25 ± 0.62 mg/dl compared to 0.69 ± 0.18 mg/dl). In fawns, total protein (TP) concentration was positively correlated with age (1 to 3-day-old). Mean TP increased the second day after birth, essentially due to an increase in the globulin concentration (2.61 ± 0.65 g/dl to 3.12 ± 0.67 g/dl). The mean albumin concentration also increased steadily during the first 3 days (1.66 ± 0.20 g/dl; 1.79 ± 0.30 g/dl; 2.04 ± 0.30 g/dl, respectively). Gamma-glutamyl transferase levels were higher in 1-day-old fawns (487 ± 473 U/L) than 3-day-old fawns (57 ± 33 U/L).

TABLE 5. Mean concentration of selected trace minerals in whole blood or serum in adult pronghorn does from Hart Mountain National Antelope Refuge, Oregon, 1996–97.

| Trace mineral (units)[a] | December 1996 mean (SD) ($n$ = 20) | March 1997 mean (SD) ($n$ = 20) |
|---|---|---|
| Cu (ppm) (serum) | 0.55 (0.08) | 0.56 (0.08) |
| Fe (ppm) (serum) | 2.47 (0.46)[b] | 2.94 (0.62)[b] |
| Se (ng/ml) (whole blood) | 98.55 (36.00) | |
| Se (ng/ml) (serum) | | 49.40 (9.50) |

[a] Cu, copper; Fe, iron; Se, selenium.
[b] Significant difference ($P < 0.05$) between values in December and March.

Serum Cu levels did not differ between the does captured in December and March (Table 5). There was no difference between the serum Cu values of the fawns from 1996 and 1997 once the age difference was considered, therefore data were combined (Table 6). Fawns were born with low serum Cu levels (0.28 ppm) but reached the lowest limit of the adult range (0.40 ppm) in about 3 days. No differences attributed to sex or between fawns that lived or died were found in serum Cu concentrations.

TABLE 6. Mean concentration of selected trace minerals in whole blood or serum in neonatal pronghorn fawns from Hart Mountain National Antelope Refuge, Oregon, 1996–97.

| Trace mineral (units)[a] | *n* | Mean (SD) | Min. | Max. |
|---|---|---|---|---|
| Cu (ppm) (serum) | 65 | 0.41 (0.16) | 0.2 | 1.23 |
| Fe (ppm (serum) | 65 | 3.52 (1.65) | 0.71 | 8.69 |
| Se (ng/ml) (whole blood)—1996 | 17 | 84.6 (20.6) | 51 | 120 |
| Se (ng/ml) (whole blood)—1997 | 44 | 50.6 (15.1) | 25 | 104 |

[a] Cu, copper; Fe, iron; Se, selenium.

Although mean ± SD liver Cu value of the samples collected from the does in April 1997 was lower (8.16 ± 6.86 ppm) than in the samples from the bucks in October 1996 (12.02 ± 6.86 ppm), the difference was not statistically significant (Table 7). Molybdenum liver levels were slightly higher ($P = 0.003$) in the does (1.54 ± 0.35 ppm) in March than in the bucks (1.08 ± 0.17 ppm) in October. Due to lower Cu levels and higher Mo levels in March, Cu/Mo ratio was lower ($P < 0.001$) in March (5.46 ± 1.83) than in October (10.66 ± 5.54).

Selenium values are shown in Table 5 and 6. Whole blood Se levels in fawns did not differ ($P = 0.50$) among age groups (1-, 2-, and 3-day-old) but there was a significant difference ($P < 0.001$) between years, therefore data were divided in Table 6. In 1996, 17 of 23 (74%) fawns had whole blood Se values below 100 ng/ml compared to 43 of 44 (98%) in 1997. No differences attributed to sex or between fawns that lived or died were found. After conversion to ppm wet weight, mean liver Se concentration from bucks in October was 0.11 ppm (range 0.06 to 0.22 ppm), similar to the mean of 0.13 ppm observed in does in March (range 0.10 to 0.15 ppm) (Table 7).

TABLE 7. Mean (SD) concentration (ppm, wet weight) for selected trace minerals in livers of adult pronghorns from Hart Mountain National Antelope Refuge, Oregon, 1996–97.

| Trace mineral (units)[a] | October 1996 mean (SD) ($n$ = 9, males) | March 1997 mean (SD) (n = 8, females) |
|---|---|---|
| Cu (ppm) | 12.02 (6.86) | 8.16 (2.66) |
| Mo (ppm) | 1.08 (0.17)[b] | 1.54 (0.35)[b] |
| Cu/Mo ratio | 10.66 (5.54) | 5.46 (1.83) |
| Fe (ppm) | 111.23 (27.84) | 143.87 (39.26) |
| Se (ppm) | 0.11 (0.05)[c] | 0.13 (0.02) |

[a] Cu, copper; Mo, molybdenum; Fe, iron; Se, selenium.
[b] Significant difference ($P < 0.05$) between values in October and March.
[c] Original value = 0.40 ± 0.18 ppm dry weight; approximate wet weight = dry weight/3.5 (Puls, 1994).

Iron levels in does were slightly higher ($P = 0.03$) in March than in December (Table 5). There was no difference between serum Fe values of fawns from 1996 and 1997 once the age difference was considered, therefore data were combined (Table 6). Serum Fe levels increased with age (1- to 3-day-old) in fawns. No differences attributed to sex or between fawns that lived or died were found. Mean serum vitamin E levels in adult does ($n = 10$) captured in March 1997 was 2.57 ± 0.48 μg/ml and ranged from 1.98 to 3.27 μg/ml.

All serologic tests were negative except for antibodies against BTV ($n = 20$, 35% positive), EHDV ($n = 20$, 30% positive), and P13 virus ($n = 18$, 67% positive). Of the seven samples tested using AGID, that were positive for BTV antibodies, four were positive (>1:10 dilution) for serotype 17, two for serotype 10, and one was negative using SN. Of the five samples tested using AGID, that were positive for EHDV antibodies, two were positive for serotype 2 and three were negative using SN.

Eggs of *Nematodirus* spp. were observed in seven of 14 does ($\bar{x}$ = 9 EPG; range 2 to 16 EPG). *Eimeria* spp. were found in three of 14 (range 32 to 95 OPG), and eggs of *Moniezia* spp. were found in

TABLE 8. Diagnosis of pronghorn fawn mortalities from necropsies of 55 carcasses from Hart Mountain National Antelope Refuge, Oregon, 1996–97.

| Cause of mortality | 1996 *n* (%) | 1997 *n* (%) |
|---|---|---|
| Predation by coyote | 17 (61) | 17 (63) |
| Predation by eagle | 1 (3) | 1 (4) |
| Dystocia | 0 | 1 (4) |
| Pasteurellosis | 0 | 2 (7) |
| Starvation | 3 (11) | 0 |
| Unknown | 7 (25) | 6 (22) |
| TOTAL | 28 | 27 |

two of 14 (36 and 40 EPG). No eggs or oocysts of parasites were found in the remaining eight does examined. Mean % FN for does captured in December (1.43%) was lower ($P < 0.001$) compared with those captured in March (1.87%).

Necropsies were performed on 55 of 87 dead fawns, 28 in 1996 (12 M, 15 F, 1 unknown) and 27 in 1997 (13 M, 12 F, 2 unknown) (Table 8). Predation by coyote (*Canis latrans*) accounted for 62% of the mortality. Starvation ($n = 3$) and pasteurellosis ($n = 2$), were the next leading causes of mortality.

Mean age of death for fawns ($n = 42$) in 1996 was 9.9 days (range 4 to 28) for all causes of mortality and 8.1 days (range 4 to 15) for those killed by coyotes ($n = 16$, 1 killed by coyote had unknown age). In 1997, mean age of death for fawns ($n = 28$) was 7.3 days (range <1 to 17) for all causes of mortality and 6.8 days (range 1.5 to 15) for those killed by coyotes ($n = 17$). Ten male and six female fawns in 1996 and eight male, eight females, and one unknown fawn in 1997 were killed by coyotes. Of the 104 neonates that were monitored, 17 (9 in 1996 and 8 in 1997) survived until at least mid-July of each year.

Culture of tissues from dead fawns in 1996 ($n = 32$) revealed no significant findings except isolation of *Salmonella* spp. from the small intestine of one female that died of starvation at 6 days of age. No gross lesions were observed nor were number of isolates significant enough to indicate that salmonellosis was the cause of death. *Pasteurella* spp. was not found from any tissues including tonsils ($n = 17$), in 1996. In 1997, *Salmonella* spp. was not isolated from any fawns. *Pasteurella multocida* was isolated from two bacteremic fawns, which died at 13 and 17 days of age. Serotyping of *P. multocida* resulted in recognition of two serotypes, A:3,4 and B:1. In one of these fawns, *P. haemolytica* was also isolated from the tonsillar sinus. Viral isolation attempts from tissue examined in 1996 ($n = 3$) and in 1997 ($n = 2$) were negative.

Gross and histologic findings in does that died in April after their capture in March 1997 were inconclusive but the lesions of petechia and ecchymosis noted grossly and microscopically were consistent with acute stress and probable overheating. This disease is similar to capture myopathy but affects organs other than just skeletal and the cardiac muscle (Idaho Department of Fish and Game, unpubl. data).

## DISCUSSION

Based on values from clinically normal pronghorns (Barrett and Chalmers, 1977a, 1979) and domestic animals (Smith, 1996), no abnormalities were observed in the CBC of the does and fawns. The mean RBC count, hemoglobin, and hematocrit values in does from December and March were similar to those reported by Barrett and Chalmers (1977a) for free-ranging pronghorns captured in December in Alberta (Canada). These parameters can be affected by capture technique. Higher hemoglobin, hematocrit, and RBC counts have been documented in captured animals not anesthetized or sedated (Kocan et al., 1981). In free-ranging white-tailed deer (*Odocoileus virginianus*) in Minnesota (USA) hemoglobin concentration, hematocrit and RBC counts were most elevated in March and were attributed to hemoconcentration due to dehydration that accompanies nutritional deprivation and weight loss during winter (DelGiudice et al., 1992). They also observed a 15.1% in-

crease in the MCV in March (DelGiudice et al., 1992). The increase in MCV from December to March in this study was only 2.0%. Although it was statistically significant, we do not consider it clinically relevant. The lower number of lymphocytes observed in the samples from March may indicate higher levels of stress during capture in this month (Smith, 1996).

Mean corpuscular volume and hematocrit values were higher in the fawns in this study than those described by Barrett and Chalmers (1979) for pronghorn fawns in southeastern Alberta. Barrett and Chalmers (1979) studied older fawns (0.2- to 10-day-old) which had smaller RBC and consequently lower hematocrit values than fawns in this study (<3-day-old). This decrease in the RBC size in young animals is apparently normal and has been documented in calves (Jain, 1993) and white-tailed deer fawns (Rawson et al., 1992) during the early months of life.

Some of the differences between the serum chemistry values in December and March of the does can be explained by gestation. All does captured in March were pregnant (U.S. Fish and Wildlife Service, unpubl. data) based on ultrasound. Based on later necropsy, we found 1.90 fetuses per doe and a twinning rate of 0.9 was calculated. Higher levels of alkaline phosphatase ($P < 0.02$) in March were probably due to increased osteoblastic activity and enzyme production in the placenta (LeResche et al., 1974). Total protein and albumin levels were slightly lower in March than in December. In pregnant domestic animals, total plasma protein decreases due to decreased albumin even though there is a slight increase in globulins (Kaneko, 1989), which we also observed.

In comparison with other pronghorn populations, mean TP concentrations in does in December (6.15 ± 0.55 g/dl) and March (5.92 ± 0.49 g/dl) were lower than values observed in adult pronghorns captured during the fall months in Alberta (7.32 g/dl; Barrett and Chalmers, 1977b). Fawns from HMNAR had slightly lower mean TP (4.61 g/dl compared to 4.78 g/dl) and albumin (1.86 g/dl compared to 2.36 g/dl) concentrations than fawns from Alberta. Fifty-five percent (41 of 74) of the fawns had an albumin concentration of ≤1.81 g/dl, which was the minimum value observed in fawns from Alberta.

Blood urea nitrogen has been correlated with dietary protein and protein utilization balance in cervids and appears to be relatively stable and unaffected by reproductive status and handling method (LeResche et al., 1974). Mean BUN values in HMNAR does captured in December (15.1 ± 2.77) and March (31.20 ± 5.36) and fawns (15.82 ± 6.7 mg/dl) were lower than those for Alberta does and fawns (42.82 mg/dl and 21.32 mg/dl, respectively) (Barrett and Chalmers, 1977b, 1979). In our study, 19% of the fawns had lower BUN concentrations than the lowest value (8.20 mg/dl) observed by Barrett and Chalmers (1979). This may be explained by a difference in protein quantity and/or quality in the diet of pronghorns in Alberta and HMNAR and this is reflected in the TP and BUN values in both adults and fawns.

Apparently does at the HMNAR had a low protein diet during the winter of 1996–97 which is reflected by BUN values (15.1 mg/dl). The increase in the BUN levels in March 1997 (31.2 mg/dl) indicated an increase in the protein of the diet and was not due to changes in hemoconcentration (Table 1). Similarly, FN levels were higher ($P < 0.001$) in March (1.87%) compared with December (1.43%) which also indicated an increase in the protein content of the diet during this time. Seal and Hoskinson (1978) found BUN levels of 11.3 mg/dl in a pronghorn population that wintered entirely on native range in Idaho compared with levels of 44.0 mg/dl for a population that wintered in an agricultural area with alfalfa and barley (food with a much higher average protein level) and 18.6 mg/dl for a population wintering in an area with minimal agricultural devel-

opment but higher protein content of the native food plant species.

Elevated BUN levels must be interpreted in relation to body condition and diet because hemoconcentrations, starvation, renal disease, or other processes that result in rapid tissue catabolism may increase BUN levels (Smith, 1996). Because starvation, dehydration, or renal disease were not reported in these does, we assume that the increase in BUN was due to an increase in available protein in the diet.

The duration and timing of low protein intake may affect the weight of the newborn, and, consequently, the survival potential of the fawns if it continues into a significant portion of the last trimester of gestation (April to May) which it did not in our study. This shift likely occurred in 1997, based on BUN values in late March. Blood urea nitrogen values were not related to fawn survival unlike what has been described in white-tailed deer fawns by Kunkel and Mech (1994).

Several studies have reported an inverse correlation between birth mass and mortality rate or vulnerability to predators (Verme, 1962; Guinness et al., 1978; Verme and Ullrey, 1984; Nelson and Woolf, 1987). Heavier fawns are more healthy and vigorous (Verme, 1962) and may be able to escape predators more readily. In our study, weights of fawns were not correlated with mortality.

Interpretations of trace mineral levels in pronghorns are difficult because few data are available concerning normal blood and serum levels in wild ungulates. Deficiencies and imbalances of minerals are well recognized as important determinants of animal condition, fertility, productivity, and mortality (Underwood, 1977). Chronic marginal deficiencies also cause reduced growth, lower reproductive rate or success, and poor fitness (Underwood, 1977).

Normal serum Cu concentration in most ruminants is 0.7–1.2 ppm. In sheep, cattle, and red deer (*Cervus elaphus elaphus*), serum Cu concentrations below 0.5 ppm are considered diagnostic for Cu deficiency (Mackintosh et al., 1986; Mertz, 1987; Puls, 1994). All does in this study had serum values (0.39–0.74 ppm) within the range considered marginal and difficult to interpret in domestic animals (Smith, 1996). Typical adequate liver Cu concentrations in sheep, cattle, and deer range from 25 to 100 ppm and levels below 5 ppm are considered deficient (Puls, 1994). Liver Cu levels in HMNAR adult pronghorns (range 1.05 to 26.6 ppm, wet weight) could also be considered marginal. Our results were similar to values in pronghorns from Arizona with mean serum Cu levels of 0.59 ppm (range 0.22–1.28 ppm) and liver levels of 8.3 ppm (range 1.1 to 36 ppm) (Heffelfinger and Olding, 1996). The implications of marginal serum Cu levels for HMNAR pronghorn population remains unknown, but animals with subclinical Cu deficiencies may have lower milk production, growth and reproductive efficiency without readily recognizable signs (McDowell, 1992). Other blood or serum manifestations of Cu deficiency are microcytic or normocytic, hypochromic anemia, low serum Fe levels, and hypercholesterolemia (Mertz, 1986; Smith, 1996). None of these changes were observed in pronghorns in this study. High Fe levels may have prevented anemia that would result from low Cu levels (Kaneko, 1989).

Serum Cu levels in fawns were significantly lower than does captured in March but reached the marginal levels of the does in about 3 days. This phenomenon occurs in other ungulate species. Plasma Cu levels in the bovine neonate are lower than in mature cattle (Smith, 1996) and in lambs the levels are low at birth but rise to adult values by 1 to 7 days of age (Howell et al., 1968). Adult pronghorn from HMNAR have slightly higher liver Mo levels (1.54 ppm) than most mammals (0.64 ppm) (Kaneko, 1989), but did not reach toxic levels (20–100 ppm) (Puls, 1994). Therefore, secondary Cu deficiency was not considered.

The importance of Se to pronghorns

and other wild ungulates populations has been recognized previously (Bodie and O'Gara, 1980; Stoszeck et al., 1980; Fielder, 1986; Flueck, 1994; McDowell et al., 1995). Stoszeck et al. (1980) indicated that low birthrate and increased fawn mortality for pronghorns in Idaho may result from Se deficiency, combined with reduced levels of several other trace minerals. Fifty percent of the whole blood Se values from does captured in December were considered marginal (<100 ng/ml) when compared with values from domestic sheep (Wheatley and Beck, 1988) or deer (Puls, 1994). Serum Se values in March were considered deficient when compared with values from deer (deficient, 7 to 60 ng/ml) or marginal, if cattle reference values are used (marginal, 30 to 60 ng/ml) (Puls, 1994). Liver Se values of our does were considered marginal (<0.25 ppm) or deficient (<0.18 ppm) when compared with cattle or deer values (Puls, 1994). In 1996, 17 of 23 (74%) fawns had whole blood Se values below 100 ng/ml, as did 43 of 44 (98%) in 1997. Therefore, Se levels in this population apparently were low, but the significance of this finding is not known due to absence of clinical evidence of deficiency.

Pronghorns from HMNAR had similar liver Se values to pronghorn from Idaho where Bodie and O'Gara (1980) reported clinical signs of Se deficiency in six pronghorns. Mean liver Se found in that pronghorn population in Idaho was 0.52 ± 0.16 ppm dry weight (approximately 0.14 ppm wet weight) and it was compared to a mean (±SD) value of 1.21 ± 0.20 ppm dry weight (approximately 0.35 ppm wet weight) in Montana (Stoszeck et al., 1980).

The influence and relationship between Se and vitamin E on nutritional myopathies and the immune responses of domestic animals has been reported (Smith, 1996; Finch and Turner, 1996). It is possible that we may not have observed any signs of Se deficiency because vitamin E levels were adequate (2.57 ± 0.48 μg/ml) at least in March. High dietary levels of vitamin E can reduce quantitative requirements for Se (Ullrey, 1981). Serum vitamin E levels of approximately 1.0 μg/ml were reported for white-tailed deer fed vitamin E-deficient diets for 12 mo, whereas serum levels in deer fed adequate diets averaged 2.15 μg/ml (Brady et al., 1978). However, seasonal variation in serum vitamin E levels is expected because grass and forb vitamin E levels generally increase during early growth and are higher in leaves than in stems, while they normally decrease dramatically as development approaches seed maturity (Robbins, 1993). If vitamin E stores are limited in regions of low plant Se concentration, pronghorn could experience lower reproductive efficiency and higher postnatal mortality.

Iron concentrations reflect liver Fe stores and blood hemoglobin levels. Adult and fawn pronghorns in this study have higher serum Fe values than adult sheep and cattle. This difference may be explained because pronghorns also had a higher mean hemoglobin concentration than cattle (8–15 g/dl), sheep (9–15 g/dl), or goats (8–12 g/dl) (Smith, 1996).

Trainer and Jochim (1969) found titers against BTV in eight of 96 (8%) serum samples from pronghorns from Wyoming (USA) and 35 of 97 (36%) serum samples in Colorado (USA). Epizootics of BT in free-ranging pronghorn occurred during the fall 1976 in eastern Wyoming with an estimated 3,200 animals dying and in 1984 in northeastern Wyoming resulting in 300 known pronghorn deaths (Thorne et al., 1988). Bluetongue virus serotype 17 was isolated from pronghorn in both epizootics. The reproductive rate in pronghorns was depressed following both epizootics, but the cause was not determined (Thorne et al., 1988). An epizootic of EHD occurred in western North Dakota (USA) which affected an unknown number of pronghorns (Richards, 1964). No known epizootics of BT or EHD have been documented in pronghorns in southern Oregon, but because epizootics have been

documented in other wild ungulates and in domestic livestock in this area, and because pronghorns are susceptible to BTV/EHDV, epizootics in pronghorns probably have occurred. The 35% seroprevalence for BTV/EHDV found in pronghorns in this study may indicate that the virus is circulating sporadically on HMNAR. Its effect on pronghorn production is unknown.

Sixty-seven percent of pronghorn tested had antibody titers against PI3 that ranged from 8 to 256. This was higher than that reported in Arizona (USA) in 1995 (55% positive; titer range = 32 to 1,024) (Arizona Department of Fish and Game, unpubl. data), in Alberta in 1983 (49% positive; titer range = 10 to ≥640) (Kingscote and Bohac, 1986), but lower than that reported in Idaho in 1975–77 (76% positive; titer range = 5 to 640) (Stauber et al., 1980). Although PI3 virus may predispose pronghorns to other infections, no evidence of disease associated with nor isolations of PI3 were found in our study.

Mortality of pronghorn fawns on the HMNAR during 1996 and 1997 was primarily due to predation by coyotes. Coyotes killed fawns apparently regardless of fawn health. Some researchers have concluded that the healthiest fawns may be more vulnerable to predation due to their increased activity, making them more likely to be observed by predators (Beale and Smith, 1973; Bodie, 1979; McNay, 1980). Fawns in this study were killed when they were very young (≤8.1 days) and probably were not very active.

Although starvation of fawns was observed in 1996, two could have been attributed to maternal abandonment. No data for Se or BUN levels were available for these two fawns. Poor nutrition resulting in starvation was identified as a direct cause of death in only one fawn in this study.

In this study, bacteremia, caused by *P. multocida*, killed 2 (7%) fawns in 1997. No other reports of pasteurellosis causing significant mortality in pronghorns could be found in published literature. Pasteurellosis was frequently diagnosed and appears to be sporadic in pronghorns in Wyoming (Thorne et al., 1988). Beale and Smith (1973) reported 3 of 117 (3%) pronghorn fawns died of pneumonia in their study in Utah, but no pathogen was isolated. Yoakum (1957) reported one adult male pronghorn died of pneumonia on HMNAR, but he believed it was secondary to another disease. Bacterial isolation was not attempted.

*Salmonella* spp. was isolated from the intestinal tract of one fawn that died of starvation in 1996. Salmonellosis has been documented in pronghorns only rarely. Beale and Smith (1973) diagnosed *Salmonella* in 2 of 117 (2%) pronghorn fawns examined in Utah over a 5-year period (1967–71). Mortality due to salmonellosis does not appear to be a significant factor in pronghorn populations on HMNAR. Parasites were apparently not a significant factor causing morbidity or mortality in pronghorns in this study.

The death of 14 of 20 does a few weeks following their capture in 1997 is unfortunate. Information provided by Idaho Department of Fish and Game pathologist (unpubl. data) suggested that the event was due to the nature of the animals which was closely related to nutrition, disease exposure, and trace mineral levels, and not just the handling event. However, reducing stress and being expedient in the handling process may decrease these types of mortalities. The use of capture drugs versus net-gunning may also reduce stress.

In summary, the pronghorn population on HMNAR appears to exist on a diet low in protein and Se and marginal in Cu. The low protein diet is most apparent in winter. Adequate serum vitamin E concentrations are apparently compensating for low Se levels, at least in certain seasonal periods. Although some nutritional problems, including low serum protein concentration and Se values, and pasteurellosis existed in the population, no predisposing factors to predation could be found. Presently, coy-

ote predation may be restricting population growth. No apparent cause of the low (<1:100) fawn : doe ratio that occurred in 1995 could be found. Long-term monitoring of the health and population dynamics of the pronghorn on HMNAR to determine the role nutrition, disease, and predation play is of paramount importance to successful management. We believe that limited and selective coyote control is warranted and the nutritional quality of the pronghorn diet, especially during winter, should be increased through proper habitat management.

## ACKNOWLEDGMENTS

We appreciate the work and assistance of the staff of the Hart Mountain National Antelope Refuge and the Oregon Department of Fish and Wildlife. We also express our thanks to W. Nunn, C. Scott, and L. Liedblad for their veterinary field assistance and to C. Scott, D. Van Vuren, and K. Kilbride for their critical review of the manuscript. We also thank D. Hunter for his assistance on pathology and B. Foreyt for his work on parasites.

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*Received for publication 29 June 1998.*