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ABUNDANCE AND CONDITION INDICES OF COYOTES
ON HART MOUNTAIN NATIONAL ANTELOPE REFUGE, OREGON

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ABSTRACT.—U.S. Fish and Wildlife Service staff at Hart Mountain National Antelope Refuge (HMNAR), southeastern Oregon, documented high pronghorn (*Antilocapra americana*) fawn mortality, subsequent low fawn recruitment, and declining pronghorn numbers from 1996 to 1999. Coyote (*Canis latrans*) predation was the primary cause, accounting for 60–85% of fawn mortalities each year, and fawns were not physiologically predisposed to predation. Therefore, we investigated certain coyote population parameters (age structure, survival, density, physiology) to evaluate how or if these factors influence coyote predation rates on pronghorn fawns. We captured 11 coyotes (5 male and 6 female) in December 1998. Age of captured animals ranged from 1.7 to 10.7 yrs ($\bar{x}$ = 5.0 years), and all coyotes appeared healthy upon capture. There were no known mortalities through December 1999. We estimated pre-whelping (December through February 1997–1999) density from howling surveys conducted within HMNAR to be 0.40–0.53 km^{-2}. Compared to other published studies, we found significant ($P \leq 0.05$) differences in selected blood parameters (e.g., blood urea nitrogen, total protein, white blood cell counts), indicating coyote nutrition may be marginal to deficient during winter at HMNAR. A high percentage of coyotes (91%) tested positive for serum-neutralizing antibodies to canine parvovirus. We judged that parasite (*Toxascaris* spp., *Alaria* spp., *Sarcocystis* spp., and *Isospora* spp.) prevalence and intensity were not high enough to influence coyote condition. Based on our data, the coyote population at HMNAR is old aged, at a relatively high density, and stable, but their nutrition may be marginal to deficient during winter. Presently, we are unable to draw direct conclusions relating the parameters we sampled with predation rates by this unexploited coyote population.

Key words: Canis latrans, coyote, health, hematology, howling survey, Oregon, physiology.

From 1996 to 1999, the U.S. Fish and Wildlife Service conducted a study on Hart Mountain National Antelope Refuge (HMNAR) in southeastern Oregon to investigate causes of poor pronghorn (*Antilocapra americana*) fawn recruitment and the subsequent decline of the pronghorn herd. During this period it was determined that fawns were not physiologically predisposed to predation, and yet coyote (*Canis latrans*) predation accounted for 60–85% of fawn mortalities and was limiting the pronghorn population (Dunbar et al. 1999, Dunbar 1999a, 1999b, Dunbar unpublished data).

These findings led us to consider other factors that may affect predation rates. Our first objective was to examine age structure, survival, and density of the HMNAR coyote population, and to analyze blood samples to assess health and exposure to disease. Our second objective was to explore relationships between these parameters and coyote predation rates on pronghorn fawns. We suspected HMNAR coyotes would be old aged, as has been reported from other unexploited populations (Gese et al.

1989, Windberg 1995). Older animals may be more efficient predators than younger animals (Pyrah 1984, Gese et al. 1996). Based on observations over the past several years, we believe coyote density on HMNAR is high, and this may also explain, at least partially, predation rates observed over the past 4 years. Physiological data may indicate whether HMNAR coyote nutrition is adequate. Inadequate nutrition at certain times of the year may also influence predation. Disease agents, particularly canine parvovirus (CPV), can impact coyote health, population dynamics, and population size, and therefore may affect predation rates as well.

This information will be useful in evaluating impacts of possible future management actions (coyote control, habitat alterations) on the coyote population.

MATERIALS AND METHODS

During December 1998 we captured 11 (5 male and 6 female) free-ranging coyotes on

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HMNAR. Coyotes were captured either by use of a net gun (Coda Enterprises, Incorporated, Mesa, AZ) fired from a helicopter ($n = 5$) or hand-caught using a pole snare after being located from a helicopter ($n = 6$). Captured animals were restrained with a pole snare fitted loosely around the neck and a leg snare placed around both hind legs. They were also blindfolded to reduce stress from handling.

The upper first premolar was extracted from each captured coyote for age estimation. Each animal was given an injection of approximately 0.1 mL lidocaine hydrochloride around the base of the tooth to alleviate pain before extraction using a tooth elevator and dental forceps. Teeth were air-dried and age was estimated by counting cementum annuli (Matson's Laboratory, Milltown, MT).

A radio transmitter with a 2-hour mortality sensor (Advanced Telemetry Systems, Incorporated, Isanti, MN) was fitted around each animal's neck. All coyotes were located at least twice a month from January through September 1999, and then once a month from October to December 1999. If a mortality signal was detected, the transmitter was located so that the cause of the signal (e.g., death, dropped transmitter) could be determined. Survival was estimated as a simple percentage because all animals were captured during the same time period and no monitoring was conducted until after all animals were captured (Heisey and Fuller 1985).

Coyote density (coyotes · km^{-2}) was estimated from data collected during coyote howling surveys (Wenger and Cringan 1978) conducted from 1997 through 1999. These surveys were conducted along a road in the study area seldom used by vehicles. Surveys began at approximately 2100 hours on nights when wind speed was ≤16 km · hr^{-1} (Wenger and Cringan 1978). Taped coyote howls were played through a loudspeaker (Johnny Stewart Wildlife Calls, Waco, TX) at each survey station ($n = 8$) to stimulate coyote response. Taped howls were played for approximately 1 minute, followed by a 3-minute listening period. If no coyotes responded, the tape was played again for 30 seconds, followed by another 3-minute listening period before moving to the next station.

Wenger and Cringan (1978) suggested that 1.6 km was the maximum radius coyote responses could be heard from a sampling sta-

tion. Our stations were approximately 4.8 km apart, which should have decreased the likelihood that we resampled the same coyote(s) at successive stations. We adopted the 1.6-km survey radius from Wenger and Cringan (1978) as our maximum to calculate minimum coyote density. Monthly coyote density along the survey route was estimated by dividing the maximum number of coyotes responding across all surveys conducted in a month by total area surveyed (64.32 km^2).

Approximately 10 mL of blood was taken from each animal by venipuncture of the femoral vein. Blood samples were collected in EDTA tubes for a complete blood cell count (CBC) and in serum separator tubes for serology and serum chemistry analysis. Complete blood cell counts and serum chemistries were performed at Lake District Hospital, Lakeview, Oregon, on automated analyzers (Coulter T 660, Coulter Electronics, Incorporated, Hialeah, FL; opeRA Analyzer, Medium Systems, Bayer Diagnostic, Larrytown, NY).

We compared animals in our sample to animals >1 year old from other studies because we captured no animals <1 year old. We tested for differences in blood parameters due to sex in our sample using analysis of variance (Statistical Analysis System, Version 8.0, Cary, NC). We compared our results with those from Gates and Goering (1976), Rich and Gates (1979), and Smith and Rongstad (1980) using 2-tailed t tests in Corel QuattroPro 8.0 (Corel Corporation, Ottowa, ON). Analysis of variance and t-test results were considered significant at $P < 0.05$.

Because there were no between-sex differences ($P > 0.05$) in our sample for any blood parameter we examined, males and females were combined for analysis. Rich and Gates (1979) and Gates and Goering (1976) also reported means for both sexes combined. Smith and Rongstad (1980), however, reported means for both adult males and females, but there were no differences between sexes except for white blood cell count (WBC) and albumin. Therefore, we compared combined (male and female) values from our sample to male values from Smith and Rongstad (1980), except for WBC and albumin, for which we compared our combined sample to both males and females.

Serological analysis for selected infectious microorganisms was performed at Washington

Animal Disease Diagnostic Laboratory (WADDL), Washington State University, Pullman, Washington. Serum samples were tested for prevalence of disease agents using methods described by Silverstein and Greene (1998). Blood samples were tested for both canine distemper virus (CDV) and CPV using the immunofluorescent antibody/serum immunoglobulin G method. Threshold titer levels were 1:50 and 1:25 for CDV and CPV, respectively. The microaglutination test was used to test for *Leptospira interrogans* serovars: *canicola, grippotyphosa, hardjo, icterohemorrhagieae, pomona,* and *bratislava.* Threshold titer was 1:100. The rapid slide agglutination test was used to detect prevalence of *Brucella canis.* Threshold titer is qualitative (positive or negative) for this test. The virus neutralization test was used to detect canine adenovirus (CAV) at a threshold titer of 1:4.

To simplify comparisons of disease agent prevalence between HMNAR animals and those from other studies, we classified the 1.7-year-old animals from our study as yearlings, and animals >1.7 years as adults. We used this age classification only for comparisons of disease agents to other studies that classed yearlings as animals between 1 and 2 years old and adults as animals older than 2.

We collected fecal samples from 5 coyotes at time of capture. Samples were examined for parasites at WADDL, where eggs or oocytes per gram (EPG and OPG, respectively) of feces were determined by fecal flotation (Thienpont et al. 1979).

RESULTS

All coyotes captured appeared thin but otherwise in good condition. Age of coyotes ranged from 1.7 to 10.7 years ($\bar{x} = 5.0$). We captured no animals <1.7 years old, and only 36% ($n = 4$) of the sample was ≤2.7 years old. We found 1 radio-transmitter from a female coyote on 3 December 1999. No carcass or other evidence was found to suggest this animal died, but we censured her from survival estimates because it had been 2 months since she was last located. Survival of the 10 remaining HMNAR coyotes from 19 December 1998 to 19 December 1999 was 100%.

Coyote density during February 1997 was estimated at 0.50 km^{-2}. We estimated similar densities from January and December 1998 (0.42 and 0.53 km^{-2}, respectively) and from December 1999 (0.40 km^{-2}).

Tables 1 and 2 list mean hematologic and serum biochemical values, respectively, with standard deviations and *P*-values from *t* tests comparing HMNAR coyotes to other populations. Results and comparisons of tests of disease prevalence are presented in Table 3. No coyotes tested positive for *Leptospira interrogans* or any of its serovars or *Brucella canis.*

Eggs or oocytes from 4 different parasites were found in coyotes: *Toxascaris* spp., *Alaria* spp., *Isospora* spp., and *Sarcocystis* spp. All 5 coyotes tested were positive for *Toxascaris* spp., with EPG ranging from 94 to 610. The oldest coyote, a 10.7-year-old female, had the highest *Toxascaris* spp. count (610 EPG) and the highest *Alaria* spp. count (22 EPG). Three of 5 coyotes were positive for *Alaria* spp. (2–22 EPG), 2 for *Isospora* spp. (54 and 84 OPG), and 1 for *Sarcocystis* spp. (85 OPG).

DISCUSSION

We have assumed that the age structure of our sample is representative of the population at HMNAR, even though our sample size is small. Our results suggest the coyote population on HMNAR is old aged and may have experienced little recruitment in 1999, unless immigration occurred. If the older animals in our sample exhibit predation patterns as predicted in other studies (Gese et al. 1996), then age structure could be influencing predation rates on HMNAR pronghorn fawns. For example, older animals, through learned behavior, may be more efficient at locating and killing pronghorn fawns than younger, less experienced animals (Pyrah 1984, Gese et al. 1996).

Annual survival of the radio-tagged sample at HMNAR was high (100%). Gese et al. (1989) found that adult survival in a lightly exploited population in Colorado was 87%. Windberg (1995) found adult survival rates in a lightly exploited population to be 64–73%. Nellis and Keith (1976) and Andrews and Boggess (1978) found survival rates of 60% and 60.9%, respectively, in moderately to heavily exploited populations. While other factors such as habitat, season, and prey availability influence these parameters (Parker 1995), human-caused mortality can have a large impact, as comparisons

TABLE 1. Hematologic values from free-ranging adult coyotes captured on Hart Mountain National Antelope Refuge, Oregon, December 1998, and *P*-values from *t* tests comparing values from reference populations to those from this study.

			P-value		
Parameter (units)	*n*	Mean ± *s*	Gates and Goering (1976)[a]	Rich and Gates (1979)[b]	Smith and Rongstad (1980)[c]
Red blood cells (×10⁶/µL)	9	7.9 ± 0.4	0.17	0.001 (–)[d]	
Hematocrit (%)	9	57.8 ± 3.3	≤0.001 (–)	≤0.001 (–)	≤0.001 (–)
Hemoglobin (g/dL)	9	19.7 ± 1.3	≤0.001 (–)	≤0.001 (–)	≤0.001 (–)
MCV[e] (fl)	9	73.3 ± 1.2	≤0.001 (–)	≤0.001 (–)	
White blood cells (×10³/µL)	9	7.1 ± 2.4	0.06	0.04 (+)	≤0.001 (+)
Lymphocytes	11	2.2 ± 1.1	0.44	0.79	0.49, 0.002 (+)
Monocytes	11	0.1 ± 0.1	≤0.001 (+)	≤0.001 (+)	≤0.001 (+)
Segmented neutrophils	11	3.7 ± 1.0	≤0.001 (+)	≤0.001 (+)	≤0.001 (+)
Eosinophils	11	0.9 ± 0.7	0.13	0.23	0.68, –
Basophils	11	0.82[f]			
Platelets (×10³/µL)	9	299.0 ± 124.2			

[a]Data from wild-born, captive coyotes.
[b]Data from 18-month-old, wild-born, captive coyotes.
[c]Data from wild, free-ranging adult male coyotes, except WBC parameters, for which significance levels from comparisons of both males and females are reported as "males, females," unless significance level for both sexes was equal. See Methods for further explanation.
[d](+) = significantly greater than sample from this study; (–) = significantly less than sample from this study; no sign = no difference between samples.
[e]MCV = mean corpuscular volume.
[f]Only mean is reported because standard deviation included negative values.

between our study and others indicate. High survival of adult coyotes could mean that a high density of coyotes could be maintained for a relatively long period of time even with little recruitment (Knowlton and Gese 1995), possibly maintaining higher-than-average predation rates.

Coyote density is difficult to calculate accurately due largely to the elusive nature of the animal and to biases of current survey techniques (Knowlton 1984). Density estimates from howling surveys are probably biased on the low side because not all coyotes within the survey area respond on each occasion (Wenger and Cringan 1978, Okeniewski and Chambers 1984, Gese and Ruff 1998). Response rates also vary by season, and transients and non-breeders typically do not respond (Gese and Ruff 1998). In addition, howling surveys have been criticized in the literature for being too variable and not sensitive enough to detect small to moderate changes in coyote densities (Wenger and Crignan 1978, Andelt and Andelt 1984, Fuller and Sampson 1988). We believe, however, that they do allow us to follow trends from year to year, as did Harrington and Mech (1982), and can be used to estimate densities on small study areas (Pyrah 1984), including HMNAR.

Knowlton (1972) suggested coyote densities in the U.S. could reach as high as 2.3 km⁻² (6.0

mi⁻²), but that density was most likely 0.19–0.38 km⁻² (0.5–1.0 mi⁻²) over most of the coyote's range. Parker (1995) reviewed many studies from across the U.S. and reported that pre-breeding and winter density estimates ranged from 0.01 to 3.0 km⁻², with the majority of estimates falling between 0.20 and 0.57 km⁻². Our minimum density estimates were near the middle to high end of what were common pre-whelping density estimates from across the U.S., suggesting a high density for HMNAR. Also, based on 3 years of data, it appears the coyote population in our study area has remained relatively stable. Because our density estimates are a minimum, it is possible the HMNAR coyote population is at saturation density and cannot absorb any new recruits. Recent high predation levels at HMNAR, then, may be influenced by high coyote density and population stability.

We obtained blood samples from coyotes subjected to the stress of capture and handling. Some blood values, including total protein (TP), blood urea nitrogen (BUN), and hemoglobin, are relatively unaffected by the capture techniques we employed, while others, including glucose and occasionally neutrophils, are strongly influenced. Therefore, interpretation of our blood data must take this into account even though we emphasized parameters that are relatively unaffected by

Table 2. Serum biochemical values from 11 free-ranging adult coyotes from Hart Mountain National Antelope Refuge, Oregon, December 1998, and P-values from t tests comparing values from reference populations to those from this study.

Parameter (units)	Mean ± s	Rich and Gates (1979)[a]	Smith and Rongstad (1980)[b]
		P-value	
Sodium (meq/L)	143.0 ± 2.6	0.03 (+)[c]	
Potassium (meq/L)	4.9 ± 0.4	1.00	
Chloride (meq/L)	113.0 ± 2.3		
Calcium (mg/dL)	7.9 ± 0.6	≤0.001 (+)	0.003 (+)
Phosphorus (mg/dL)	3.5 ± 1.5	0.52	0.83
Total protein (g/dL)	5.7 ± 0.6	0.001 (+)	0.002 (+)
Albumin (g/dL)	3.3 ± 0.4	0.001 (−)	0.008 (−), 0.13
Uric acid (mg/dL)	0.9 ± 0.8	0.07	
Alkaline phosphatase (U/L)	10.5 ± 10.2	≤0.001 (+)	
Alanine aminotransferase (U/L)	62.0 ± 20.6		
Aspartate aminotransferase (U/L)	109.0 ± 39.0		
Lactate dehydrogenase (U/L)	468[d]		
Blood urea nitrogen (mg/dL)	39.0 ± 15.3	0.02 (−)	0.02 (−)
Glucose (mg/dL)	159.0 ± 59.4	0.09	0.92
Creatinine (mg/dL)	1.1 ± 0.3	0.03 (+)	
Total bilirubin (mg/dL)	0.02[d]		
Cholesterol (mg/dL)	148.0 ± 25.95		0.54

[a]Data from 18-month-old, wild-born, captive coyotes.
[b]Data from wild, free-ranging adult male coyotes except albumin, for which significance levels from comparisons of both males and females are reported as "males, females."
[c](+) = significantly greater than sample from this study; (−) = significantly less than sample from this study; no sign = no difference between samples.
[d]Only mean is reported because standard deviation included negative values.

Table 3. Comparison of prevalence of antibody titers to canine distemper virus (CDV), canine parvovirus (CPV), and canine adenovirus (CAV) between coyotes at Hart Mountain National Antelope Refuge (HMNAR) and coyotes from studies in California (CA; Cypher et al. 1998) and Yellowstone National Park (YNP; Gese et al. 1997).

	% positive, yearlings			% positive, adults		
	HMNAR (2)[a]	CA (37)	YNP (11)	HMNAR (9)	CA (67)	YNP (33)
CDV	100	36[b]	54	22	54	88
CPV	100	89	100	89	73	100
CAV	0	54	82	89	90	97

[a]Number tested.
[b]Only 36 coyotes were tested for antibodies to CDV.

capture and handling. Different laboratory methods used in different studies may also have some effect on values.

Red blood cell counts (RBC) were higher (Table 1) in our study compared with values of captive coyotes from Rich and Gates (1979), but they did not differ from those in a study of captive coyotes by Gates and Goering (1976). Hematocrit and hemoglobin values in our study were also higher when compared with values from 3 other studies (Gates and Goering 1976, Rich and Gates 1979, Smith and Rongstad 1980). The higher values we found could indicate that coyotes at HMNAR were in better condition than those from other studies (Gates and Goering 1976, Rich and Gates 1979, Smith and Rongstad 1980). Seal and Mech (1983), however, found higher RBC, hemoglobin, and hematocrit values in gray wolves (*Canis lupus*) during winter compared to other seasons and determined seasonal variation was responsible. Because we collected blood samples during winter, we believe this may be the cause of the difference between HMNAR studies and the others.

Because neutrophilia can result from the stress of capture and handling, it is somewhat surprising that total WBC counts of HMNAR

coyotes were lower compared with 2 other studies (Rich and Gates 1979, Smith and Rongstad 1980), and neutrophils were lower in HMNAR coyotes than in coyotes from 3 studies (Table 2). We suspect that lower WBC counts in our study may be related to poor nutrition compared to coyotes in the aforementioned studies. Any differences in the other parameters presented in Table 1 are probably due to capture stress and likely would not influence the long-term condition of the coyotes.

Blood urea nitrogen values in our study (Table 2) were higher than values from Rich and Gates (1979) and Smith and Rongstad (1980). Total protein values in our study were lower (Table 2) than those reported from the same 2 studies. Elevated BUN coupled with lowered TP levels can signify protein catabolism due to energy deprivation caused by inadequate winter nutrition, a condition we suspect is occurring in coyotes on HMNAR.

Calcium level was significantly lower in this study compared to levels from Smith and Rongstad (1980) and Rich and Gates (1979; Table 2). We believe these lower values were also due to dietary deficiency (Robbins 1983).

Among disease prevalences, the high percentage of HMNAR coyotes with titers to CPV was the most interesting and the only result that we presently consider could influence coyote condition. High CPV titers may indicate that most of the coyotes on our study area had been infected with CPV. Such high antibody prevalence rates are usually associated with a highly contagious but nonfatal infection (Thomas et al. 1984). Mech and Goyal (1995), however, predicted that the winter gray wolf population in Minnesota would decline when CPV prevalences in adults consistently exceeded 76%. They believe that CPV may be important in limiting wolf populations. The population data displayed by Windberg (1995) also illustrate the potential impact CPV can have on coyote populations. In his study, CPV contributed to high juvenile loss in 1980 and severely reduced recruitment of that age class the next year. If HMNAR coyote numbers decline over the next few years, it is possible, based on our data, that CPV may be involved.

HMNAR coyotes are old aged, at a relatively high density, and stable. Nutrition for these animals may be marginal to deficient during winter. The disease and parasite prevalences we observed did not appear to have any measurable negative influence on HMNAR coyote condition, although the high percentage of our sample with CPV may have future relevance. While we are unable to draw direct conclusions relating the parameters we sampled to predation rates on pronghorn fawns at this time, we will continue some aspects of our work so that we may do so. The data we do present, however, are relevant for an unexploited coyote population.

LITERATURE CITED

ANDELT, W.F., AND S.H. ANDELT. 1984. Diet bias in scat deposition-rate surveys of coyote density. Wildlife Society Bulletin 12:74–77.

ANDREWS, R.D., AND E.K. BOGGESS. 1978. Ecology of coyotes in Iowa. Pages 249–265 *in* M. Bekoff, editor, Coyotes: biology, behavior, and management. Academic Press, New York.

CYPHER, B.L., J.H. SCRIVNER, K.L. HAMMER, AND T.P. O'FARRELL. 1998. Viral antibodies in coyotes from California. Journal of Wildlife Diseases 34:259–264.

DUNBAR, M.R. 1999a. Health evaluation of pronghorn (*Antilocapra americana*) fawns on Hart Mountain National Antelope Refuge in southeastern Oregon, 1998. USDI Fish and Wildlife Service, Technical Report 99–01, Lakeview, OR.

_____. 1999b. Health evaluation and factors affecting survival of pronghorn (*Antilocapra americana*) fawns on Hart Mountain National Antelope Refuge, Oregon, 1999. USDI Fish and Wildlife Service, Progress Report, Sheldon-Hart Mountain Refuge Complex, Lakeview, OR.

DUNBAR, M.R., R. VELARDE, M.A. GREGG, AND M. BRAY. 1999. Health evaluation of a pronghorn antelope population in Oregon. Journal of Wildlife Diseases 35:496–510.

FULLER, T.K., AND B.A. SAMPSON. 1988. Evaluation of a simulated howling survey for wolves. Journal of Wildlife Management 52:60–63.

GATES, N.L., AND E.K. GOERING. 1976. Hematologic values of conditioned, captive wild coyotes. Journal of Wildlife Diseases 12:402–404.

GESE, E.M., O.J. RONGSTAD, AND W.R. MYTTON. 1989. Population dynamics of coyotes in southeastern Colorado. Journal of Wildlife Management 53:174–181.

GESE, E.M., AND R.L. RUFF. 1998. Howling by coyotes (*Canis latrans*): variation among social classes, season and pack size. Canadian Journal of Zoology 76: 1037–1043.

GESE, E.M., R.L. RUFF, AND R.L. CRABTREE. 1996. Intrinsic and extrinsic factors influencing coyote predation of small mammals in Yellowstone National Park. Canadian Journal of Zoology 74:784–797.

GESE, E.M., R.D. SCHULTZ, M.R. JOHNSON, E.S. WILLIAMS, R.L. CRABTREE, AND R.L. RUFF. 1997. Serological survey for diseases in free-ranging coyotes (*Canis latrans*) in Yellowstone National Park, Wyoming. Journal of Wildlife Diseases 33:47–56.

HARRINGTON, F.H., AND L.D. MECH. 1982. An analysis of howling response parameters useful for wolf pack censusing. Journal of Wildlife Management 46: 686–693.

HEISEY, D.M., AND T.K. FULLER. 1985. Evaluation of survival and cause-specific mortality rates using telemetry data. Journal of Wildlife Management 49:668–674.

KNOWLTON, F.F. 1972. Preliminary interpretations of coyote population mechanics with some management implications. Journal of Wildlife Management 36: 369–382.

_____. 1984. Feasibility of assessing coyote abundance on small areas. Final Report of Research Work Unit 909.01, National Wildlife Research Center, Fort Collins, CO. 14 pp.

KNOWLTON, F.F., AND E.M. GESE. 1995. Coyote population processes revisited. Pages 1–6 *in* C. Richardson, T. Blankenship, K. Canon, and S. Henke, editors, Coyotes in the Southwest: a compendium of our knowledge. Texas Parks and Wildlife Department, Austin.

MECH, L.D., AND S.M. GOYAL. 1995. Effects of canine parvovirus on gray wolves in Minnesota. Journal of Wildlife Management 59:565–570.

NELLIS, C.H., AND L.B. KEITH. 1976. Population dynamics of coyotes in central Alberta, 1964–68. Journal of Wildlife Management 40:389–399.

OKENIEWSKI, J.C., AND R.E. CHAMBERS. 1984. Coyote vocal response to an electronic siren and human howling. Journal of Wildlife Management 48:217–222.

PARKER, G. 1995. Eastern coyote: the story of its success. Nimbus Publishing, Halifax, Nova Scotia, Canada. 254 pp.

PYRAH, D. 1984. Social distribution and population estimates of coyotes in north-central Montana. Journal of Wildlife Management 48:679–690.

RICH, J.E., AND N.L. GATES. 1979. Hematologic and serum chemistry values of pen-raised coyotes. Journal of Wildlife Diseases 15:115–119.

ROBBINS, C.T. 1983. Wildlife feeding and nutrition. Academic Press, Inc., Orlando, FL. 343 pp.

SEAL, U.S., AND L.D. MECH. 1983. Blood indicators of seasonal metabolic patterns in captive gray wolves. Journal of Wildlife Management 47:704–715.

SILVERSTEIN, D.C., AND C.E. GREENE. 1998. Laboratory testing for infectious diseases of dogs and cats. Pages 704–715 *in* C.E. Greene, editor, Infectious diseases of the dog and cat. W.B. Saunders Company, Philadelphia, PA.

SMITH, G.J., AND O.J. RONGSTAD. 1980. Serologic and hematologic values of wild coyotes in Wisconsin. Journal of Wildlife Diseases 16:491–497.

THIENPONT, D., F. ROCHETTE, AND O.F.J. VANPARIJS. 1979. Diagnosing helminthiasis through coprological examination. Janssen Research Foundation, Beerse, Belgium. 187 pp.

THOMAS, N.J., W.J. FOREYT, J.F. EVERMANN, L.A. WINDBERG, AND F.F. KNOWLTON. 1984. Seroprevalence of canine parvovirus in wild coyotes from Texas, Utah and Idaho (1972–1983). Journal of American Veterinary Medical Association 185:1283–1287.

WENGER, C.R., AND A.T. CRINGAN. 1978. Siren-elicited coyote vocalizations: an evaluation of a census technique. Wildlife Society Bulletin 6:73–76.

WINDBERG, L.A. 1995. Demography of a high density coyote population. Canadian Journal of Zoology 73: 942–954.