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PERINATAL MORTALITY IN CARIBOU FROM THE PORCUPINE HERD, ALASKA

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ABSTRACT: During the 1989 caribou (*Rangifer tarandus*) calving season on the Arctic National Wildlife Refuge, Alaska (USA), 61 calf carcasses were examined for cause of death and associated pathology. Dead calves were located by low-level aerial searches with two fixed-wing aircraft and a helicopter over high density calving areas between the Hulahula and Aichilik rivers. Primary diagnoses included emaciation (39%), malnutrition (8%), stillbirth (21%), trauma (16%), other primary causes (7%), and undetermined causes (8%). Twenty calves had contributory renal tubular degeneration. The findings indicate that factors contributing to nutritional deprivation in calves were the major cause of neonatal mortality; however, factors affecting stillbirth, abortion, or the urogenital system may have major effects on neonatal caribou and warrant further investigation.

Key words: Caribou, *Rangifer tarandus*, mortality, neonates, Arctic National Wildlife Refuge, north slope, Alaska.

INTRODUCTION

The Porcupine caribou (*Rangifer tarandus granti*) herd occupies the northeast corner of Alaska and northern half of the Yukon Territory in Canada. During spring, caribou migrate in a northwest direction to the calving grounds on the north slope and coastal plain of the Arctic National Wildlife Refuge (NWR) where areas are being considered for oil and gas exploration. The herd consists of about 178,000 adult caribou and has increased at an average annual rate of 5% since 1979 (Fancy, 1990). Calving commences in late May, peaks around the first few days of June, and ends by the second week of June (Fancy et al., 1990). The herd is the focus of intense research because of the potential harm from oil and gas development on Arctic refuge biota. The primary concern is a potential decrease in caribou herd size or productivity from loss of habitat to development (Cameron, 1983).

The purpose of this study was to establish baseline information on the causes of perinatal mortality in the Porcupine caribou herd. Perinatal mortality includes mortality in the last trimester of pregnancy and from birth onward for the first 2 to 3 wk. Because of the potential for oil exploration on and around the calving grounds, baseline information is needed to better identify and assess the consequences of development by comparing perinatal mortality before and after development. Baseline mortality studies were initially intended for several years. However, funding was curtailed and reliable data were collected only in 1989.

Previous information on neonatal mortality (mortality shortly after birth) in the Porcupine caribou herd (Mauer et al., 1983; Whitten et al., 1984; Whitten et al., 1987b) was based on sample sizes of only 10 to 15 animals, incomplete postmortem examinations, or techniques that precluded evaluation of death at or around the time of birth. Seventy-nine to 87% of adult females give birth each year but 16 to 35% of this herd's calves die by late June; most (59 to 74%) die ≤48 hr after birth (Whitten et al., 1992).

METHODS

The calving grounds of the Porcupine caribou herd include a large portion of the coastal plain of the Arctic NWR and some areas in western Canada bounded approximately by 69° to 70°N and 141°30′ to 144°30′W (Fancy et al., 1990). Whitten et al. (1992) provides a general description of the main calving area which occupies over 5,000 km² of the Arctic NWR. Over 178,000 caribou occupy the grounds with densities usually exceeding 20 caribou per km² (Whitten et al., 1992). Dead calves were located from 30 May to 7 June 1989 by low-level aerial searches

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between the Hulahula and Aichilik rivers. Daily, non-systematic searches over several areas exceeding 900 km² were made with two fixed-wing aircraft to locate dead calves. A helicopter was used to recover carcasses. The location was identified, and each carcass was assigned a field number, placed in a plastic bag, and transported to a field facility at Beaufort Lagoon for necropsy. Several heavily scavenged carcasses were not retrieved. One debilitated calf was euthanatized in the field by cutting the right jugular vein and carotid artery after prolonged observation confirmed the calf was abandoned.

Necropsies consisted of a systematic external and internal examination of the body and all organs and structures except the spinal cord. Portable dictation equipment powered by a portable gasoline generator was used to record history, body weight, and necropsy findings. Body weight (±0.1 kg) was determined by hanging the carcass on a precalibrated spring scale. Tissues fixed in 10% buffered neutral formalin for histopathology included brain, trachea, lung, liver, spleen, kidney, duodenum, jejunum, ileum, ileocolic juncture, heart, and skeletal muscle. Tissues were embedded in paraffin, sectioned at 5 μm, and stained with hematoxylin and eosin (H&E). Oil-red-O and periodic acid Schiff (PAS) stain (Thompson, 1966) were applied to sections when cytoplasmic vacuolation and accumulation of cytoplasmic substances were seen on H&E-stained preparations. Oil-red-O stains were applied to newly cut sections of formalin-fixed tissue prepared in a manner to minimize lipid loss and compared to control tissue known to be positive for lipid and prepared in the same manner.

Primary morphologic diagnoses were based on finding lesions judged severe enough to be significant contributors to the death of the animal. Secondary morphologic diagnoses were based on lesions judged to have been the result of primary lesions, or judged to have been of a magnitude that was unlikely to cause death by themselves.

A diagnosis of emaciation was based on severe atrophy of fat stores as indicated by severely reduced, firm, dark red to purple gross appearance of fat and lack of significant intracellular lipid histologically in adipose tissue. The criteria for malnourished calves included a less severe degree of fat atrophy. Other criteria correlated with diagnoses of emaciation and malnutrition included varying amounts of vegetation without milk in the gastrointestinal tract and lack of any other recognizable primary lesions. Starvation in newborn mammals is a result of lack of adequate milk obtained from the mother and is not necessarily the consequence of lack of food supply to the caribou population. As used in this paper, calf emaciation is not a reflection of forage quantity or quality in caribou habitat. Stillbirth diagnoses were based on presence of non-aerated, fetal lungs with clear-to-yellow fluid filling airways, lack of hoof wear, and empty stomachs. Trauma from all causes, including predation, was grouped together. Trauma, as compared to scavenging, was evaluated based on the presence of hemorrhage in the tissues surrounding the wound, and the type and location of wounds (Miller et al., 1985). Criteria such as puncture marks across the calvarium, tooth marks, and predator tracks were used to diagnose predator type (Whitten et al., 1987a).

During some preliminary work in 1988, I noticed a high frequency of renal degenerative lesions. Postmortem and acute degenerative lesions can be difficult to differentiate (Jones and Hunt, 1983). Four calves were examined for the effect of postmortem decomposition on the histologic appearance of kidneys in 1989. Calves in excellent postmortem condition were examined at necropsy in the manner described above. The kidneys and adnexa then were returned to the carcass cavity and the carcass set aside. No other organs were placed in the carcass. Carcasses were kept in the shade at ambient temperature, which varied from 2 to 13 C. Sections of kidney were sampled at 24-hr intervals through 96-hr post-necropsy. Sections were prepared for histologic examination as described above. Histologic criteria described by Jones and Hunt (1983) assisted in distinguishing degenerative and postmortem changes.

Samples for bacteriology consisted of duplicate samples of lung, liver, kidney, spleen, brain, and intestine. Placentas were evaluated if they were in suitable condition. One set of samples was refrigerated (2 to 4 C); a duplicate set was frozen in liquid nitrogen (−196 C). Refrigerated samples were maintained on chemical ice during shipment to the National Wildlife Health Research Center in Madison, Wisconsin (USA). Samples were received, chilled or frozen, depending on the appropriate sample group, on 11 June 1989. All samples were cultured on 5% sheep blood agar and eosin methylene blue. All intestinal samples were cultured for *Salmonella* spp. in selenite broth and brilliant green agar. Brucella agar and Ferrell's selective media were used on all stillbirths. These bacteriologic media are described in Balows et al. (1991).

Virology samples were frozen in liquid nitrogen and included tonsil, lung, trachea, bronchial lymph node, kidney, liver, spleen, small intestine, mesenteric lymph node, and colon contents. Samples were rapidly thawed in a 37 C water bath just before processing. Screening procedures, in absence of pathological findings, included the following: colon contents were ex-

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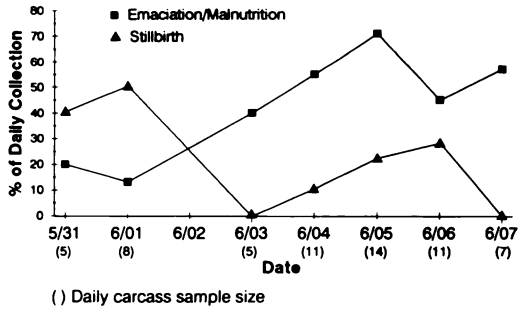


FIGURE 1. Daily proportion of caribou calves with cause of death from emaciation and stillbirth.

amined by electron microscopy for reoviruses, rotaviruses, and coronaviruses using the methods of Docherty et al. (1991) for infected chorioallantoic membrane. Tonsil and lung tissues were examined for the presence of parainfluenza 3 and infectious bovine rhinotracheitis viruses using 4- to 5-day-old 2nd passage bovine embryonic kidney (BEK) cell cultures in 24-well plates. Prior to inoculation, cells were rinsed with Hanks' balanced salt solution and fed with Eagle MEM with Earle salts (Balows et al., 1991) supplemented with pyruvate, L-glutamine, 2% chicken serum, gentamicin, and amphotericin B. Wells were inoculated with 0.1 ml supernatant of homogenized 10% tissue suspension. Uninoculated controls were included with each test. Cultures were incubated at 37 C and examined daily for 9 to 10 days for virus cytopathic effect.

Tonsil and lung also were examined for respiratory syncytial virus with a monoclonal antibody enzyme immunoassay (RSV Direct Antigen Detection System, Kallestad Diagnostics, Chaska, Minnesota, USA; trade name is not endorsed by the Federal Government).

Samples of small intestine were examined for the presence of bovine viral diarrhea virus (BVDV) using 3-day-old BEK cultures as described above in a slide, 8-well culture chamber (Lab-Tek; NUNC, Inc.; Naperville, Illinois, USA). After 3 days of incubation, the slide was prepared for direct immunofluorescent examination by rinsing with phosphate buffered saline (PBS, pH 7.2) and fixed in acetone for 10 minutes. Fixed cells were overlaid with fluorescein isothiocyanate-conjugated BVDV hyperimmune serum (National Veterinary Services Laboratory, Ames, Iowa, USA) for 1 hr, rinsed in 2 changes PBS (pH 7.8), and air-dried; I used PBS-glycerol (pH 7.8) to mount a coverslip on each slide. Slides were examined with a Model 2071 epi-fluorescent microscope (American Optical Instruments, Buffalo, New York, USA) equipped with a xenon light source. Positive and negative control slides were included with each test.

Statistical analysis included Analysis of Variance (ANOVA) and Bonferroni (Dunn) t-tests for differences in body weight among primary diagnoses (SAS Institute Incorporated, 1987).

TABLE 1. Causes of death in 61 neonatal caribou from the Porcupine caribou herd, 1989.

Diagnosis	Number
Emaciation	24
No other primary lesion (22)[a]	
Marked nephrosis (2)	
Malnutrition	5
Stillbirth	13
No other primary lesion (10)	
Congenital defects (2)	
Lymphocytosis/encephalomalacia (1)	
Trauma	10
Predation (8)	
Probable adult caribou (2)	
Other	4
Severe enteropathy (1)	
Drowning (suspect) (1)	
Low birth weight/underdevelopment (1)	
Euthanasia (1)[b]	
Undetermined	5
	61

[a] Number of calves with this lesion.
[b] Calf was found abandoned and monitored for presence of cow before being euthanatized.

RESULTS

Sixty-one calves were collected between 31 May and 7 June 1989, and evaluated for cause of death and associated pathology (Fig. 1). Maximum time elapsed between any carcass collection and necropsy was <15 hr.

Emaciation, malnutrition, stillbirth, and trauma caused 85% of the total mortality (Table 1). Almost half (47%) of the 1989 perinatal mortality was associated with poor nutritional condition (emaciation and malnutrition). Mean (±SE) body weight for 29 emaciated and malnourished calves was 5.4 (±0.11) kg and was significantly lower ($P = 0.0026$) than the weights of 27 calves that died from other causes ($\bar{x} = 6.3 \pm 0.25$ kg). I excluded four calves missing most internal organs and one euthanatized calf. Only two calves had detectable predisposing causes (significant nephroses) as-

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sociated with emaciation. The proportion of deaths from emaciation and malnutrition increased during the sampling period (Fig. 1).

Stillbirths occurred in 13 (21%) of 61 dead calves. Three of these 13 calves had recognizable predisposing factors. One calf had disseminated lymphocytic inflammation and focal encephalomalacia; however, no virus was isolated. The other two calves had severe congenital defects. One of these calves had focal subcutaneous bruising in the limbs and neck. A high ventricular septal defect, an overriding, dextraposed aorta, and renal dysplasia were observed in this calf. Hypoplasia of the intestine resulted in a markedly shortened intestine in the other calf. Signs of dystocia were observed in one other stillborn calf, but did not appear sufficient to cause death. Stillborn calves composed a high portion of total mortality early in the sampling period (Fig. 1).

Mean (±SE) body weight of 12 stillborn calves was 6.3 ± 0.39 kg; one calf was excluded because its intestine and pelvic organs were missing. Mean body weight of 44 calves that died of other causes was 5.7 (±0.15) kg. I excluded three calves missing all internal organs and one euthanatized calf. This difference was not statistically significant. The mean body weight of the stillborn calves was similar to the mean for the 15 other non-emaciated calves ($\bar{x}$ = 6.2 ± 0.34 kg), excluding four calves missing most internal organs and one euthanatized calf.

Four calves had marked (25 to 50%) atelectasis, lacked milk in their stomachs, exhibited no other significant findings, and had a mean (±SE) body weight of 6.7 (±0.34) kg. Their cause of death was undetermined, but it was noted that they had failed to suckle. A fifth calf was markedly underweight (2.7 kg), and had about 25 to 30% pulmonary atelectasis. This calf was diagnosed as having died from being underdeveloped and having a low birth weight. A sixth calf was malnourished, the gastrointestinal tract lacked food with the exception of the spiral colon, and a few minor patches of atelectatic lung were observed. The calf was diagnosed as having died from malnutrition.

Ten calves (16%) died of trauma (Table 1). Eight of these deaths were attributed to predators. The remaining two calves had internal injuries consistent with blunt trauma presumably inflicted by adult caribou. The body condition of calves killed by predators was generally good. Seven calves were in good-to-excellent condition and one in fair condition; this latter animal was considered malnourished and microscopically had mild fatty degeneration of the liver. In a few instances, predated calves in good condition were found close (<40 m) to emaciated carcasses. These emaciated carcasses had experienced greater postmortem decomposition, indicating that they were at least available at the time predators were in the area. Whether the emaciated calves were dead before predators arrived could not be determined.

A variety of secondary lesions were observed (Table 2); the most common finding was renal tubular degeneration (20/61 calves). Earliest lesions had a multifocal distribution within corticomedullary tubular epithelial cells and were characterized by variably sized, clear, round to ovoid, well-demarcated vacuoles, occasionally obliterating the cell (Fig. 2). Although the histologic appearance could have been due to a fatty change or perhaps carbohydrate accumulation, fresh sections from formalin-fixed tissues stained with oil-red-O and PAS were negative. These findings were consistent with hydropic degeneration in which the vacuoles contained primarily water (Jones and Hunt, 1983; Jubb et al., 1985). Evidence of tubular regeneration, generally characterized by areas with variably sized epithelial cell nuclei and apparent hypercellularity, was rare but observed in some necrotic areas. Severity of degenerative changes could not be assessed; based on the sequential histologic sampling of kidneys, it was evident that postmortem degeneration could mark-

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TABLE 2. Secondary morphologic diagnoses in 61 neonatal caribou from the Porcupine caribou herd, 1989.

Secondary diagnoses	Number
Renal tubular degeneration	20
Pulmonary atelectasis	6
Lymphoid depletion, spleen	5
Dystocia	3
Fatty (vacuolar) degeneration, liver	3
Focal nodular hyperplasia, adrenal	2
Peritonitis	1
Colloid depletion, thyroid	1
Granulomatous pneumonia	1
Multifocal cardiac hemorrhage	1
Adrenal hemorrhage	1
Multifocal pulmonary hemorrhage	1
Malnutrition	1

edly affect the apparent severity of the lesions. In those calves without degenerative lesions on initial sampling, postmortem change did not produce distinctive vacuolation in otherwise well-preserved tubular epithelial cells of the kidney. Diagnoses of emaciation seemed unrelated to the presence of renal lesions. Thirteen of 30 emaciated or severely malnourished calves were affected with renal degenerative changes. Seven other calves in fair to excellent body condition also had renal degenerative changes.

No significant isolates were found on virological and bacteriological analyses.

DISCUSSION

Results from this study were most comparable to studies of the Beverly (Miller et al., 1988a) and Kaminuriak herds (Miller and Broughton, 1974). Two biases in this study warrant discussion. Several heavily scavenged carcasses were excluded because, based on previous experience, the cause of death cannot be determined from heavily scavenged carcasses. Elimination of scavenged carcasses does not necessarily reduce the prevalence of predation in the results. Other causes of mortality also may be prone to being scavenged. Also, inclusion of heavily scavenged carcasses may bias samples against causes of death associated with internal organs.

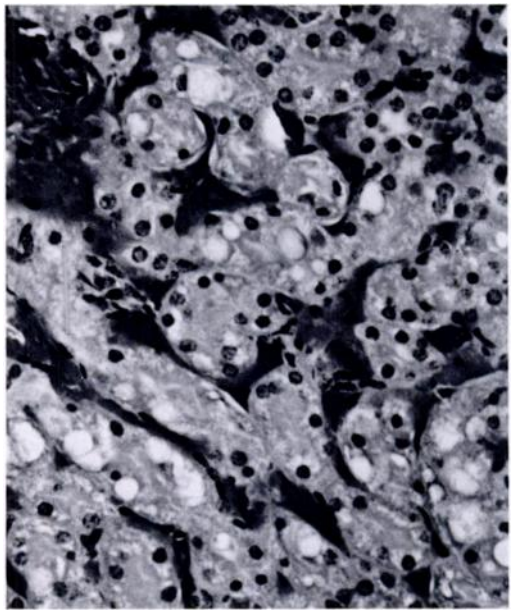


FIGURE 2. Renal tubular degeneration. Note the presence of clear, rounded vacuoles effacing cellular architecture. H&E. Bar = 40 μm.

It is highly unlikely the sampling method biased the types of mortality observed through either increased calf abandonment by females, or by decreased predation of calves. Frequent flights are routinely made over calving Porcupine caribou without producing recognizable significant disturbance or mother-calf separation (Whitten et al., 1992). My observations on flights during this study support this conclusion. In addition, the non-systematic search method used in this study rarely covered the same ground repeatedly. Study-induced abandonment is considered a problem when calves are handled and separated from their mothers for radiocollaring. Even at this level of interference only 13% of captured calves were permanently abandoned during or shortly after collaring (Whitten et al., 1992).

Predator densities are low on the arctic plain and increase significantly in higher elevations south of the core calving area (Young et al., 1990). Predation rate is related to calf distribution relative to predator distribution (Whitten et al., 1992). In

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this study, I focused on perinatal mortality in the core calving area, which has a low predator abundance. Golden eagles (*Aquila chrysaetos*) and two grizzly bears (*Ursus arctos*) were the only predators observed during the collection flights and these were not in close proximity to calving caribou.

Based on the high level of emaciation without detectable predisposing causes, failure of a calf to obtain adequate nutrition from the cow is the major cause of perinatal mortality in the Porcupine caribou herd. This is in contrast to earlier studies in which only 7% (1/14) to 27% (3/11) of radiocollared calves died from emaciation (Mauer et al., 1983; Whitten et al., 1984; Whitten et al., 1987a). However, comparisons of my findings to these studies are questionable because of the different methodologies. In populations with a high density of predators, such as the Beverly (Miller et al., 1988a) and Kaminuriak (Miller and Broughton, 1974) herds in Canada, malnutrition or starvation in calves were much less common. The proportion of unborn calves decreased during the sampling period, with most (83 to 92%) births occurring by 10 June (Whitten et al., 1992). Consequently, the proportion of mortality due to emaciation may be expected to increase during the neonatal sampling period if new mortality factors are not introduced and early mother-calf separation is the primary mechanism to produce emaciated calves. This pattern of increased emaciation deaths was observed (Fig. 1) and reflects the time required for emaciation to occur.

In the past, starvation was linked with pneumonia in unmarked calves opportunistically found (Whitten et al., 1987a); however, diagnoses were apparently made on vague gross findings which can be misleading or inaccurate. Pneumonia or microbiological evidence of pulmonary pathogens associated with starvation in calves were not documented in this study.

The causes of cow and calf separation and eventual calf starvation were not clear. In the past, separation has been interpreted as physical separation and has been accepted as maternal abandonment induced by humans or predators (Whitten et al., 1987a; Miller et al., 1988a). However, behavioral and non-morphologic problems of the calf that result in failure to suckle cannot be discounted. Maternal behavioral and physical problems unrelated to abandonment also may cause failure of the calf to receive milk. If, however, maternal abandonment behavior is the major cause of eventual emaciation in neonates, major effects could be expected if cows are disturbed on the calving grounds.

Stillbirths were relatively common on calving grounds of the Porcupine caribou herd in 1989. Using radiocollared female adults, Whitten et al. (1987a) reported that about 8% of calf mortality was attributed to stillbirth, but the criteria for determining stillbirth was not reported. Stillbirths (confirmed fetal atelectasis) in the Beverly herd were high in 1981 (10.4%) and dropped to 5.6% and 2.3%, respectively, during the following 2 yr (Miller et al., 1988a). If suspect cases are added to the confirmed cases, $\leq$18.5% of the Beverly herd calf mortality may have been due to stillbirths, a figure comparable to that for the Porcupine caribou herd.

Many factors, both infectious and noninfectious, can induce abortions and stillbirths. Brucellosis (*Brucella suis*, biovar 4) has been present in Alaskan caribou populations for many years (Huntley et al., 1963; Neiland et al., 1968) and reached a prevalence as high as 30% in the Arctic herd (Dieterich, 1981). We did not isolate *Brucella* spp. or any other pathogenic bacteria from stillborn calves.

Contrary to observations in Canadian herds, stillborn calves in the Porcupine caribou herd did not have below normal body weight. Miller et al. (1988a) suggested that low body weights of stillborn calves from the Beverly herd indicated premature births or complications in fetal development. Based on the high body weights of stillborn calves from the Porcupine cari-

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bou herd, it seems that the causes of stillbirths may not occur until very late in gestation, perhaps peripartum. Based on the relative lack of appropriate pathological findings in stillborn calves, the primary mortality factors probably are not infectious; rather, they may be environmentally related and thus have significant implications for caribou management and petroleum exploration.

The significance of the renal degenerative lesions is unknown, particularly because postmortem change affected the apparent severity of the lesions. Based on the minor regenerative changes and lack of evidence for an acute toxic insult to the kidney, I speculate that the lesions probably are metabolic in origin, secondary to other homeostatic disturbances or anoxia. The principle exogenous factors that cause renal tubular degenerative lesions and eventual necrosis, if initiating factors remain unchanged, are nephrotoxins and ischemia (Jubb et al., 1985).

Little is known about the frequency of congenital anomalies in wild animal populations. The low-level of congenital anomalies in neonatal caribou is similar to that observed in northern fur seal neonates (Roffe, unpubl.). A variety of defects have been described in animal species and those in caribou are not unique. One calf had congenital heart defects including transposition of major vessels and a ventricular septal defect. In a previous study, Whitten et al. (1987a) reported that the only neonate in which death was attributed to natural disease had unspecified congenital heart abnormalities. By comparison, atrial and ventricular septal defects and transpositions of the major vessels are the most frequently diagnosed congenital cardiac diseases in cattle (Jubb et al., 1985).

Predator abundance was low on the coastal plain where most calving occurred in 1989 (Young et al., 1990) and a low-level of predation on calves was observed in the Porcupine caribou herd. Whitten et al. (1987a) implicated predation as the major cause (≤70%) of neonatal deaths in the Porcupine caribou herd. However, his techniques of radio tracking 1- to 3-day-old calves reduced the likelihood of finding many factors associated with perinatal mortality and also biased the sampled population toward vigorous, healthy calves. Furthermore, mortality during the first 48 hr following radio collar application was considered study-induced abandonment and not included in the assessment of causes of mortality.

Preyed-upon calves were in good body condition and lacked predisposing abnormalities, suggesting predators did not select ill calves. This is similar to observations in the Beverly herd (Miller et al., 1988a) where 87.2% of calves killed by predators were apparently normal healthy animals. Considering the level of predation by wolves (*Canis lupus*) on Beverly herd neonates, Miller et al. (1988a) suggested that compensatory mortality was of minor inportance. In my study, three of the eight calves that predators killed were not consumed and an additional two were only partially consumed. Underutilization of killed prey by wolves has been documented by Miller et al. (1988b). None of the carcasses were cached as would be expected if predators were planning to return to the carcass. The lack of consumption of preyed-upon calves supports the idea that predator attention may be drawn to healthy, vigorous calves. This may be similar to the surplus killing by predators as reported by Miller and Broughton (1974) and Miller et al. (1985); however, groups of preyed-upon carcasses were not observed is this study. Surplus killing probably only occurs during periods of relative prey abundance and ease of access by predators.

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