

# **A HEALTH ASSESSMENT AND CALFHOOD MORTALITY STUDY OF THE NORTHERN ALASKA PENINSULA CARIBOU HERD 2005**



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23 September 2005

## Abstract

The Northern Alaska Peninsula caribou herd (NAP) has declined by 79% since 1993, however the reasons for the decline are not well documented. The NAP caribou are an important subsistence resource for Alaska native villages and an important part of the ecosystem in the Alaska Peninsula and Becharof National Wildlife Refuge. Past live-capture/release and radio studies by the Alaska Department of Fish and Game have noted low body weights, delayed age of first calving, a decline in productivity, and lesions consistent with viral pneumonia and foot rot. High calf mortality from predation is also been documented. Retrospective serology showed no previous evidence of exposure to the bovine respiratory viral complex prior to 2000. In this study, 42 newborn calves were radio-collared to determine survival rate and causes of mortality. Additionally, four calves and three adults were euthanized for complete necropsy and diagnostic pathology, hematology, microbiology, toxicology, and parasitology. Twenty-three collared calves that died in the first 2 weeks of life were available for necropsy as well. These calves died of the following: 43% were killed by wolves, 43% by bears, 9% by unknown large predators, and 4% drowned. After 4 weeks, 74% of collared calves died, and by 8 weeks 92.5 % died. One calf had a mild bronchiolitis and intranuclear inclusion bodies consistent with Bovine Respiratory Syncytial Virus (BRSV) or Parainfluenza-3 (PI3), and serology showed exposure to PI3 but culture attempts were negative. Serology on sera collected from 2001, 2004 and 2005 demonstrate antibodies to BRSV (20%), IBR (70%), PI3 (70%), and Neospora (10%). Fecal examinations on calves detected strongyles and coccidia but were negative for *Cryptosporidium* spp. and *Giardia* sp. The adult caribou that were necropsied were heavily parasitized including: *Ostertagia* sp., *Setaria* sp., *Sarcocystis* sp., nasal bots, warbles, lungworm, *Taenia* sp., *Echinococcus granulosus*, and rumen flukes. Hematologic results from the adults were consistent with adverse health effects from parasitism. We determined that predation by wolves and bears as the primary cause of death in NAP calves. However, NAP adults are heavily parasitized and the herd has been exposed to bovine viral diseases and parasites that could significantly impact their health, reproduction, and survival.

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## Abbreviations

ADF&G	Alaska Department of Fish & Game
BRSV	Bovine Respiratory Syncytial Virus
BVD	Bovine Viral Diarrhea
EDTA	ethylenediaminetetracetic acid
Hb	haptoglobin
Hg	hemoglobin
IBR	Infectious Bovine Rhinotracheitis
NAP	Northern Alaska Peninsula
NAPCH	Northern Alaska Peninsula caribou herd
PI3	Parainfluenza-3
RBC	Red blood (erythrocyte) count
TSH	Teshekpuk caribou herd
WBC	white blood cell (leukocyte) count
WAH	Western Arctic caribou herd

## Introduction

The Northern Alaska Peninsula Caribou Herd (NAPCH) is the most important terrestrial subsistence resource for the 12 Alaska native villages on the Alaska Peninsula. The NAPCH population has declined by 79% since 1993, however the reasons for the decline have not been adequately documented (ADF&G Caribou Management Report, 2002). Evidence from past capture and monitoring work, including low body weights, delayed age of first calving, and a decline in overall productivity, suggest that poor nutritional condition may be a primary factor. Past studies have also noted significant calf mortality from predation and disease. Biologists managing the herd have documented outbreaks of mortalities due to foot rot and pneumonia among adults and calves but tissues for pathologic and etiologic diagnoses have not been examined.

In the previous two years, fall herd composition counts indicated that calf survival has not exceeded 10% over the summer period. Reliable information on factors limiting calf production and survival will be crucial in future management decisions for this herd. Recent data from a limited number of adult female caribou outfitted with satellite GPS collars indicate that two sub-herds may exist. These separate counts indicate that the northern mountain sub-herd population has remained stable, while trend counts in the southern sub-herd have declined by approximately 83%. A previous calf mortality study conducted on the NAPCH estimated that calf survival in the northern portion of the

Alaska Peninsula was 37% higher than calf survival in the southern portion of the peninsula over the first month of life, which is the critical period for calf recruitment. Understanding whether disease is a factor in this disparity of calf survival is necessary for the development of effective management for the recovery of the NAPCH.

Previous serologic analysis of this population, based on 286 blood samples collected at capture for collaring from 1982 through 1998, failed to detect antibodies to any infectious disease investigated, which includes *Brucella suis* serovar 4, Infectious Bovine Rhinotracheitis (IBR), Malignant Catarrhal Fever (MCF), Bluetongue, Bovine Respiratory Syncytial Virus (BRSV), Parainfluenza-3 virus (PI3), Bovine Viral Diarrhea (BVD), and *Toxoplasma* (Zarnke 2000; Zarnke *et al.* 2000). However, previous superficial post mortem examinations have reported lesions consistent with pneumonia (possibly due to BRSV), lungworm of unknown species, and contagious foot rot.

The objectives of the calthood mortality part of the study were to determine whether predation is a significant source of direct mortality in neonates and during early calthood in the NAPCH, and whether neonates that are preyed upon have lesions indicative of infectious or parasitic diseases. To answer these questions, the ADF&G and U.S. Fish and Wildlife Service collected calves and deceased radio collared calves for detailed post mortem examination and diagnostic pathology during June 2005 to evaluate early calthood mortality in the NAP. In addition, 3 adult caribou were collected for detailed post mortem examination and diagnostic testing. A retrospective and prospective serosurvey was undertaken to assess the potential for the introduction of infectious diseases.

## 1 Methods

### 1.1 Field Sample Collection

Venipuncture was achieved in the live-capture release adult caribou via the jugular vein using an 18 or 20 gauge 1 to 1.5 inch needle and blood collected directly into Vacutainer® brand tubes (Becton Dickinson and Company, Franklin Lakes NJ) via a Vacutainer® adaptor. In the collected caribou, blood was obtained immediately post mortem by cardiac venipuncture with a 60 ml syringe and 18 to 20 g 1 to 1.5 inch needle then transferred to Vacutainer® tubes.

Four types of Vacutainer® tubes were filled at each blood collection: serum separator tube (SST™), EDTA anti-coagulated tube, sodium heparin trace mineral tube and plain trace mineral tube. The SST™ and trace mineral plain tubes were chilled immediately after collection, allowed to clot for at least one hour and then centrifuged for 15 minutes. The serum was aspirated, aliquoted into cyrovials (Nalgene Company, Rochester NY) and immediately frozen on dry ice and stored at -50°C. A single 1 - 5 ml EDTA tube was collected for blood smears and a complete blood cell count. A 1 ml aliquot of whole blood was transferred from the heparinized trace mineral tube and frozen in a cryovial. The tube was then centrifuged for 15

minutes and the plasma harvested and transferred in to a 5 ml Falcon® tube and frozen.

### **1.1.1 Neonates**

Complete necropsies were conducted on the carcasses of previously radio collared (as 0-48 hrs old) calves brought back to the field camp by either Dr. Beckmen or Cristina Hansen. The gross examination included morphometrics and examination of all body systems with narrative description of abnormalities, injuries, and other lesions. Whenever possible, a preliminary diagnosis as to the manner of death was given. An intensive systemic collection of ten tissues were taken by sterile technique and transferred into individual sterile Whirlpac bags® for bacterial and viral (microbiology) culture including: spleen, liver, kidney, lung, thoracic and gastrointestinal lymph nodes, and intestinal tract. Tissues for microbiology are collected only when the carcass condition is suitable (freshly dead and uncontaminated). The tissues are then snap frozen on dry ice in the field and during shipping to Fairbanks. These tissues will be stored in an ultra-cold freezer in Fairbanks until shipped to the laboratory for culture. Caudate liver lobe, kidney and bone were collected and frozen for trace minerals analysis. A representative section of every tissue or organ is collected and placed in 10% neutral buffered formalin for histopathological analysis. Samples for parasitologic examination were collected, including lung, abomasal wall, and feces (or intestinal content). Additional fecal samples were collected for larvae isolation and other diagnostic tests. Necropsies were conducted on natural mortalities as well as calves collected for diagnostic purposes.

### **1.1.2 Adults**

No adult caribou were found dead during the fieldwork period. However, three obviously ill adults were observed and killed by shotgun in the cervical region for diagnostics. All edible meat was salvaged and donated to local village charities. Dr. Beckmen conducted complete necropsies on adult carcasses in the field with the assistance of Cristina Hansen and Lem Butler. The gross examination included morphometrics and examination of all body systems, and tissues were collected using the techniques described above. Fourteen tissues were collected by sterile technique for bacterial and viral (microbiology) culture (spleen, liver, kidney, lung, thoracic and gastrointestinal lymph nodes, reproductive and intestinal tracts). Hair, caudate liver lobe, kidney and bone were collected and frozen for trace minerals analysis. Samples for parasitologic examination were collected as above with the addition of feces were reserved for future diagnostic testing. A tooth was also removed and air-dried for aging. Bone marrow was collected and frozen for fat analysis.

## **1.2 Hematology**

Hematocrit was determined by measuring packed red cells as a percent of blood volume after centrifugation in a microhematocrit tube at 10,000 x g for 5 minutes. Total protein was determined from plasma with a hand-held refractometer (AO

Scientific Instruments, Buffalo NY 14215). The total leukocyte count was determined manually using the Unopette® system (Becton-Dickinson and Company, Franklin Lakes, NJ 07417) and a hemacytometer (Bright-Line, AO Scientific Instruments, Buffalo, NY 14215). The differential cell counts were made manually under oil immersion compound microscopy at 100x on fresh blood smears stained with Wright's-Giemsa type stain (Dip Quick Stain, Jorgensen Laboratories, Loveland CO 80538) by identifying 100 leukocytes based on morphology. A single operator (CH) conducted all differential cell counts to maintain consistency.

To obtain reticulocyte counts fresh whole blood was diluted 1:1 with new methylene blue stain (Jorgensen Laboratories, Loveland CO 80538) and allowed to sit for 10 minutes before making a smear to count reticulocytes. Once completed the slides were examined under oil immersion (100x) and 100 red cells were counted to determine a reticulocyte percentage and then corrected for the normal hematocrit and reported as corrected reticulocyte percentage (CRP).

Hemoglobin: In the field, 10µl of whole blood was transferred into 2.5 ml Drabkin's reagent for preservation for hemoglobin determination. The assay was performed at the ADF&G Marine Mammal Physiology Laboratory on the University of Alaska Fairbanks campus using the standard laboratory protocol. Briefly, potassium ferrocyanide oxidizes hemoglobin and its derivatives to methemoglobin. Potassium cyanide reacts with methemoglobin producing a more stable form of methemoglobin (Pointe Scientific Inc.; technical sheet P803-H7504-01, rev.1/02). Total blood hemoglobin measured as methemoglobin absorbs maximally at λ=540nm.

A stock standard is prepared by the addition of 1ml dH<sub>2</sub>O to Point Scientific Hemoglobin lyophilized standard. The resulting solution achieves a concentration of hemoglobin of ([Hb]) 2400 g/dL. The stock standard is diluted according to table below and let stand at room temperature for 3 minutes.

Stnd#	[Hb] g/dL	Hb stnd (ul)	Drabkin's (ml)
1	24	10	4.0
2	18	10	2.5
3	12	10	4.0
4	6	10	8.0
5	0	0	1.0

Absorbance is determined on standards and samples on a Spectronic™ Helios™ Gamma UV-Vis Spectrophotometer (Thermo Electron Corporation, San Jose, CA 95134). A regression equation from the standards is used to determine sample Hb concentration.

The erythrocyte (red cell) indices were calculated from the hematocrit, red cell count, and hemoglobin concentrations as follows:

Mean corpuscular volume (MCV) femtoliters =  $PCV \times 10 / RBC (10^6)$

Mean corpuscular hemoglobin (MCH) picograms =  $Hb \times 10 / RBC (10^6)$

Mean corpuscular hemoglobin concentration (MCHC) grams/dL =  $Hb \times 100 / PCV$

### 1.3 Parasitology

An attempt was made to obtain feces per rectum from adults that were immobilized and released. In addition, random fecal samples were collected off the ground during carcass collections if they appeared to be relatively fresh. At necropsy, feces were collected from the rectum or descending colon as available. Fecal samples were frozen until analysis.

Zinc sulfate fecal floatation for the identification of parasitic ova was conducted at ADFG using a sub sample of the previously frozen feces (less than 2.0 g) utilizing Ova Float Zn 118 (Butler, Dublin, OH 43017). After 10-15 minutes of floatation, wet mounted slides were examined under a compound microscope.

Quantitative floatation of fecal samples was performed at Mississippi State College of Veterinary Medicine by Dr. Lora Ballweber, and the number and type of eggs per gram of feces was determined.

The Quantitative Beaker Baermann Technique (Forrester and Lankester 1997) was used to recover live larvae. Briefly, an envelope of nylon screen with a single layer of gauze was prepared and stapled on 2 sides. An approximate 5-gram sample of feces was positioned in the screen envelope on top of the gauze and then placed horizontally submerged in a beaker containing 200 ml of tepid water. After 18 to 24 hours the envelope was removed with forceps without disturbing the sediment. The contents of the container were allowed to settle for 5 minutes before further processing. The supernatant was aspirated until approximately 30 ml of fluid remained. The contents of the beaker were swirled to mix and divided between two 15 ml centrifuge tubes. The tubes were centrifuged for 10 minutes at 1500 rpm. The supernatant was aspirated until 2-4 ml remained. The sample was resuspended and the two tube contents combined. The sample was then centrifuged again. All but the last ~2 ml of the tube contents were aspirated from the meniscus.

The sediment was resuspended in the tube and transferred with a Pasteur pipette to glass slides. The sediment was examined under a compound microscope at 40x. Larvae were differentiated into two types (i.e. dorsal-spined versus *Protostrongylus* sp.). The total number of larvae of each type recovered were divided by number of grams of feces (usually 5) to calculate the number of larvae per gram of feces (LPG).

Adult *Ostertagia* sp. and other abomasal nematodes (*Marshallagia*, *Teladorsagia*, etc.) are recovered in the manner described by Hoberg et al (1999). Briefly, the contents and mucosa of the thawed abomasal walls are washed three times with a small amount of water in a bucket. Two aliquots of the material were fixed in 10% formalin. The remaining unfixed contents were examined under a dissecting microscope and all adult nematodes were enumerated, collected and fixed in 80% alcohol for morphologic and molecular identification. Dr. Eric Hoberg, Chief Curator of the US National Parasite Collection & Animal Parasitic Disease Laboratory USDA, Beltsville, Maryland, will conduct definitive identification.



Molecular techniques were applied by Dr. Lori Bollweber of Mississippi State University to attempt to detect *Cryptosporidium* and *Giardia*.

## **1.4 Pathology**

Tissues fixed in 10% neutral buffered formalin were processed by routine paraffin imbedding, sectioned at 6 microns and stained with hematoxylin-eosin at Histology Consulting Services in Everson WA. Microscopic sections were examined and histopathologic diagnoses reported by Dr. Kathy Burek, Alaska Veterinary Pathology Services. Viral and bacterial cultures were conducted at Washington State Animal Disease Diagnostic Laboratory and reported by Dr. Lindsay Oaks.

## **1.5 Serology**

Serologic testing for BRSV, IBR, BVD, PI3, (by serum neutralization) Leptospirosis 5 serovars, EHD (by AGID), Toxoplasmosis and Neospora were conducted at the Wyoming Veterinary Diagnostic Laboratory with a cutoff of 1:4. Additional assays on retrospective sera for Neospora and Toxoplasmosis were conducted at the University of Tennessee College of Veterinary Medicine with a cutoff of 1:100. *Brucella* serology was conducted at the University of Alaska Fairbanks. *Brucella* testing consisted of both the Brucellosis Card Test (Becton Dickinson, Cockeysville, MD 21030), and a standard plate antigen test (Animal and Plant Health Inspection Service National Veterinary Services Laboratories, Ames, IA 50010). Both are macroscopic agglutination procedures. In the card test, 0.03 ml of serum is placed in a teardrop-shaped test area on the diagnostic card, then two drops (0.03 ml each) of *Brucella* antigen are placed on the card, not touching the serum. Using a stirrer, the antigen and serum are mixed and spread over the teardrop area. The card is then rocked for 4 minutes and read. Any specimen showing the characteristic agglutination pattern are read as positive, and those with dispersed clumps or none are negative.

For the standard plate antigen test, 10, 20, 40, and 80  $\mu\text{L}$  of serum were pipetted onto separate squares of an etched glass plate. 30  $\mu\text{L}$  of standard plate antigen was then added to each square and thoroughly mixed. The plate was rotated 4 times and incubated for 4 minutes under cover to prevent drying. After 4 minutes the plate is rotated again 4 times and incubated for 4 minutes. After the second incubation the plate is observed for degree of agglutination over indirect light.

## **1.6 Ancillary Assessments**

### **1.6.1 Haptoglobins**

Serum haptoglobin levels were determined using a commercial gel electrophoresis kit and haptoglobin-hemoglobin binding assay (Titan Gel High Resolution Protein Kit #3040, Helena Laboratories, Beaumont TX 77704). A 14.5% solution of reindeer

hemoglobin was obtained by dilution of washed red blood cells that had been lysed in sterile water and by freezing. Two  $\mu\text{l}$  of the 14.5% hemoglobin solution was added to each 38  $\mu\text{l}$  of test sample serum in a microcentrifuge vial and mixed by vortex. Two  $\mu\text{l}$  of the mixture was placed on the gel previously prepared according to kit instructions. Electrophoresis was conducted for 45 minutes at 105 volts. Gels were stained with an o-dianisidine solution for ten minutes and rinsed three times according to the kit specifications. Haptoglobin was recorded as positive (having a band) or negative (no band) for each animal.

### **1.6.2 Blood Urea Nitrogen**

The assays for Blood Urea Nitrogen (BUN) were performed at the ADF&G Marine Mammal Physiology Laboratory on the University of Alaska Fairbanks campus according to the manufacturers instructions for Sigma Urea Nitrogen Kit # 640A (Sigma-Aldrich Inc. St. Louis MO 63103). Samples were diluted 1:1 with deionized (DI) water (150  $\mu\text{l}$  sample + 150  $\mu\text{l}$  water) and the kit standard was diluted from 30 mg/dL to 1:1 as well. The spectrophotometer (Spectronic™ Helios™ Gamma UV-Vis Spectrophotometer, Thermo Electron Corporation, San Jose, CA, 95134) is warmed up for 30 minutes at 570 nm. The Urease Buffer reagent is reconstituted with 30 ml DI water. Two blanks, 3 standards and the samples are prepared in duplicate. Into each tube is added the following: 125  $\mu\text{l}$  Urease solution 10  $\mu\text{l}$  DI water for blank, std (1:1) or sample (1:1). The resulting solutions are gently mixed by vortex. After standing for 15-20 minutes at room temperature, the following are added in turn and mixed after each addition: 250  $\mu\text{l}$  phenol nitroprusside solution, 250  $\mu\text{l}$  Alkaline hypochlorite solution, 1.25 ml deionized water. After standing for 20 to 30 minutes at room temperature in the hood the samples are vortex mixed, and absorbance at 570 nm read. BUN is calculated as follows:

$$\text{Serum urea N (mM)} = (\text{Sample A} - \text{Blank A}) / (\text{Standard A} - \text{Blank A}) * 10.7$$

$$30 \text{ mg/dL} = 10.7 \text{ mM therefore } 75 \text{ mg/dL} = 26.75 \text{ mM}$$

### **1.6.3 Bone Marrow**

Previously frozen bone marrow samples were weighed in a tared 12 oz plastic cup. The cups were held at 65° C in a incubator for 48 hours and then weighed. Samples were returned to the incubator for an additional 24 hours and a final weight was obtained. The final dry weight was used to determine the water loss and calculate the percent fat from the remaining mass less the connective tissue. This is established as an indicator of nutritional state in caribou and moose (Nieland 1970).

### **1.6.4 Trace Minerals**

Trace mineral analysis will be conducted in the Wildlife Toxicology Laboratory at the University of Alaska Fairbanks under the supervision of Dr. Todd O'Hara. Blood, serum and plasma will be analyzed from the live captured animals and liver, kidney and bone from carcasses. Analytes will include selenium, cadmium, iron, copper, molybdenum and lead.

## **1.7 Statistical Analyses**

Statistical analyses were carried out using Sigma Stat 2.03 software (SPSS Inc., Chicago IL). Comparisons among groups were made using One-Way ANOVA with Tukey's tests. A level of alpha = 0.05 was chosen to detect differences.

The "normal" reference ranges were calculated for reindeer and free-ranging caribou as follows: A mean was calculated and the range set from 2 standard deviations above and below the mean.

## **2 Results**

### **2.1 Neonates**

#### **2.1.1 Neonate Pathology**

Forty-two newborn calves were collared between May 27<sup>th</sup> and June 3<sup>rd</sup> to determine survival rates of calves and to investigate the cause of death. Aircraft were used to check the radio signals for mortality mode and location every 1 to 3 days. Calves that were dead were collected as soon as possible and after a site investigation, were returned to camp for complete necropsy. Visual identification of a predator was the best indicator of the species of predator. However, when no predator was observed, several criteria used to distinguish wolf from bear predation.

Criterion 1. Caribou carcass remains

- Bears tended to eat all internal organs and the majority of bones. Carcasses were commonly limited to hooves, skull cap fragments, and lower mandibles
- Wolves tended to eat abdominal organs and muscle tissue first. Carcasses commonly included brain (brain case still intact), long bones, muscle tissue, and occasionally thoracic organs.

Criterion 2. Caching of carcass remains

- Bears didn't have anything left to cache
- Wolves buried parts of the carcass in neat caches that were hard to find. Often found half of a calf in the cache. The vegetation was pulled over the carcass, leaving the area looking untouched. Caches could only be found by radio-telemetry.

Criterion 3. Carcass wound examination – distance between canine punctures.

- Bears - 6cm or greater
- Wolves - 5cm or less

Criterion 4. Kill site characteristics -

- Bears tend to eat the entire carcass without moving/scattering remains. The few remains were found, were commonly in a small, discrete area.
- Wolves often scattered the carcass. This probably occurs when several wolves are present at the kill site. Wolves were seen carrying the carcass or carcass parts several hundred yards from the kill site.
- Wolf and bear foot prints, hair and scats can be distinguished easily in the field and were reported when present.

During the 4 weeks in which calf survival has been monitored, 74% of the marked calves died. Gross necropsies were performed on 23 calves carcasses (Table 1). The primary cause of death for the 23 calves were: 43% killed by wolves, 43% by bears, 9% by unknown large predators, and 4% drowned. Thus, overall predation accounted for 96% (all but one) of mortalities in calves during the first two weeks of life. Mortality was highest in the Bear/Sandy River calving area where 14 (92%) of the 15 collared calves died during the first 4 weeks of the study. By early August, two collared calves could not be located and only 3 of 40 remained alive (two collars stopped transmitting and the calves were not located) with a mortality rate of 92.5% by 2 months of age.

Table 1. Gross necropsy findings for calf mortalities

Animal ID	Date	Location	Primary cause of death
2005-100	1-Jun-05	Cinder R. N.	Drowning
2005-101	1-Jun-05	Bear River	Euthanasia by gunshot
2005-102	1-Jun-05	Bear River	Euthanasia by gunshot
Mort05-1	1-Jun-05	S. of Cinder River	Predation, wolf
Mort05-3	1-Jun-05	Old Creek	Predation? Insufficient
2005-103	2-Jun-05	Cinder R. Flats	Euthanasia by gunshot
Mort05-4	2-Jun-05	N. Cinder River	Predation, wolf
Mort05-5	2-Jun-05	Cinder flats	Predation, wolf
2005-104	3-Jun-05	Bear River	Predation, wolf
2005-105	4-Jun-05	Cinder R. Flats	Predation, wolf
Mort05-4	2-Jun-05	Cinder R. Flats	Predation, wolf
2005-106	5-Jun-05	Cinder R. Flats	Euthanasia by gunshot
2005-107	6-Jun-05	Pumice Creek	Predation, type unknown
2005-108	7-Jun-05	Cinder R. Flats	Predation, wolf
2005-109	8-Jun-05	N. Of Muddy R.	Predation, wolf
Mort05-14	8-Jun-05	Ilnik	Predation
Mort05-17	8-Jun-05	Bear River	Predation, bear?
Mort05-18	8-Jun-05	Bear River	Predation
Mort05-21	9-Jun-05	Cinder R. Flats	Predation, bear
Mort05-14	8-Jun-05	Ilnik	Predation
Mort05-17	8-Jun-05	Bear River	Predation, bear?
Mort05-18	8-Jun-05	Bear River	Predation
2005-110	9-Jun-05	Cinder R. Flats	Predation, bear

Of the four calves that were predated or found dead and were suitable for histopathologic examination, one had no unexpected histologic lesions. One calf, which died of massive trauma to the chest and inguinal areas, had a chronic mild multifocal degenerative cardiomyopathy (histiocytic myocarditis) with areas of myofiber loss. Since the calf was only 4 days old and the lesion is chronic, it may be due to a toxic or nutritional cause. The Wildlife Toxicology Laboratory at the University of Alaska Fairbanks will conduct trace mineral analysis of blood and tissues from the calves. One was found at the bottom of a pond, and had evidence of trauma. Histopathology confirmed that the calf had terminally inhaled pond water, but there was no evidence of inflammatory disease. The final calf possibly has an unusual amount of embryonic rest cells in the ventricles and white matter of the brain, a second opinion is pending, but this may be within normal limits for a young animal. The same calf also had mild lymphocytic inflammation around the bile ducts, which may be due to blockage, parasite migration, an autoimmune disorder, or a drainage reaction.

Of four calves euthanized by gunshot for the study, two had no significant histologic lesions in any of the survey tissues examined (left ventricle, abomasum, liver, kidney, lung), and one had a mild eosinophilic enteritis/colitis and very mild interstitial nephritis. However one collected calf had a mild bronchitis and bronchiolitis with possible intracytoplasmic and intranuclear inclusion bodies. The lung was sent for viral culture for PI3 or BRSV at Washington State Animal Disease Diagnostic Laboratory but results were negative. It is unusual to see these lesions in a neonate and further diagnostic testing is being pursued including PCR and immunohistochemistry.

Five of eight calves overall had a mild vacuolar hepatopathy, which is not unexpected in neonates, giving the cytoplasm a moth-eaten appearance.

### **2.1.2 Neonate Hematology**

Table 2 describes the mean, median, standard deviations, and ranges for the leukogram subpopulations obtained from collected NAP caribou calves and normal, newborn captive reindeer, age 0 to 7 days. Table 3 describes the mean, median, standard deviations, and ranges for the erythrocyte indices obtained from the same groups.

Table 2. Mean  $\pm$  SD, median, and minimum to maximum ranges of leukocyte parameters in newborn North Alaska Peninsula caribou compared with reference ranges (Mean $\pm$ 2SD) for normal, newborn captive reindeer calves.

	NAP Calves 1-3 mo., <i>n</i> = 83		Reindeer Calves <i>n</i> = 20	
	Mean $\pm$ SD	Median (Min-Max)	mean $\pm$ SD	Reference range
Total Leukocytes/ $\mu$ l	<b>2,284*</b> $\pm$ 1,185	2,206 (1,225-3,500)	5,085 $\pm$ 1957	1,171-9,000
Neutrophils:Lymphocytes	1.9 $\pm$ 1.2	2.1 (0.4-3.2)	2.6 $\pm$ 1.4	0.9-7.0
Neutrophils/ $\mu$ l	1,026 $\pm$ 1,373	1,408 (294-2,380)	2,957 $\pm$ 2,019	0-6,994
Band neutrophils/ $\mu$ l	<b>64†</b> $\pm$ 60	66 (0-124)	2 $\pm$ 10	0-22.4
Lymphocytes/ $\mu$ l	644* $\pm$ 169	649 (472-809)	1,579 $\pm$ 772	35-3,124
Monocytes/ $\mu$ l	158 $\pm$ 91	137 (79-279)	161 $\pm$ 219	0-656
Eosinophils/ $\mu$ l	61 $\pm$ 47	48 (25-124)	31 $\pm$ 54	0-139
Basophils/ $\mu$ l		0	3 $\pm$ 8	0-19

\* indicates significantly decreased compared to reindeer calves,  $p < 0.5$

† indicates significantly increased compared to reindeer calves,  $p < 0.5$

Table 3. Mean  $\pm$  SD, median, and minimum to maximum ranges of erythrocyte indices in newborn North Alaska Peninsula caribou compared with reference ranges (Mean $\pm$ 2SD) from normal, newborn captive reindeer calves.

	NAP Calves <i>n</i> = 4		Reindeer calves <i>n</i> = 25	
	Mean $\pm$ SD	Median (Min-Max)	Mean $\pm$ SD	Reference Range
Total protein g/dl	5.8 $\pm$ 0.5	5.8 (5.2-6.2)	5.3 $\pm$ 0.62	4.1-6.6
Hematocrit (Hct) %	35.4 $\pm$ 16.4	27.4 (26.7-60.0)	35 $\pm$ 4.3	30.8-39.3
Hemaglobin (g/dl)	13.1 $\pm$ 4.8	14.5 (7.8-17.2)	12.76 $\pm$ 1.42	9.92-15.6
RBC (x10 <sup>6</sup> /ul)	8.3*		7.6 $\pm$ 0.9	5.7-9.5
CRP	2.7 $\pm$ 1.4	2.5 (1.4-4.6)	na	
MCV (fl)	72.7*		45 $\pm$ 1.7	41.6-48.4
MCH (pg)	20.8*		16.9 $\pm$ 0.60	15.7-18.1
MCHC (g/dl)	28.4*		37.8 $\pm$ 1.44	34.9-40.6

\* Only on individual had a red cell count available for calculation of indices

CRP = Corrected Reticulocyte Percentage

MCV = Mean Corpuscular volume

MCH = Mean Corpuscular Hemoglobin

MCHC = Mean Corpuscular Hemoglobin content

### **2.1.3 Neonate Parasitology**

No parasites were seen grossly in calves at necropsy however a number of parasite eggs and larvae were detected on fecal exams. Positive samples by individual identification number are given in Table 4. Confirmation of identification of larvae will be by molecular analysis by Dr. Eric Hoberg, Chief Curator of the US National Parasite Collection & Animal Parasitic Disease Laboratory USDA, Beltsville, Maryland. Fecals from nine calves were analyzed by molecular methods for *Cryptosporidium* and *Giardia* and all samples were negative.



Table 4. Fecal examination results from Northern Alaska Peninsula caribou calves during late May-early June 2005.

Animal #	Beaker Baermann			Flotation (mimimum)			
	<i>Parelaphostrongylus</i> sp. Larvae	<i>Ostertagia</i> sp. larvae	Protostrongyloid larvae	<i>Marshallagia</i>	Trichostrongyle	Coccidia	Cestode <i>Monezia</i>
2005-100	-	-	+	-	-	-	-
2005-102	-	-	-	-	-	-	-
2005-103	-	-	-	-	-	-	-
2005-104	-	-	+	-	-	-	-
2005-105	-	-	-	-	-	-	-
2005-106	-	-	+	-	+	-	-
2005-107	-	-	-	-	-	-	-
2005-108	-	-	+	+	+	+	-

## 2.2 Results- Adults

### 2.2.1 Adult Pathology

Three unthrifty adult caribou from the Northern Alaska Peninsula Herd were selected for euthanasia for detailed disease assessment. The first adult (#106878, approximately 2 year old male) was in poor body condition and heavily parasitized. Grossly visible parasites included *Setaria sp*, *Sarcocystis sp.*, nasal bots (*Cephenemyia trompe*), *Taenia hydatigena*, *Echinococcus granulosus*, rumen flukes, and abomasal lesions consistent with *Ostertagia sp*. On histopathologic examination there were large numbers of eosinophils throughout the tissues examined, indicative of a heavy parasite infestation. There were eosinophils in the lymph nodes, spleen, lung (with macrophages, plasma cells, and lymphocytes), an eosinophilic and lymphocytic reaction of the lamina propria of the rumen (with a fluke on the surface), an eosinophilic reaction in the pericardium (along with intramyofibrillar protozoal cysts), and there is an eosinophilic reaction in the abomasal wall, along with encysted nematode larvae (most likely *Ostertagia*). There was also a mild meningoencephalitis in the brainstem, which may be due to parasite migration, protozoal, rickettsial or viral infection. The kidneys showed a mild interstitial nephritis, and special stains are pending to rule out Leptospirosis. Finally, there was an area in the mesentery that contained what was consistent with a hemangioma, a benign tumor that is an incidental finding.

The second animal (#106879, approximately 10 year old female) was emaciated and limping. On gross examination she was similarly heavily parasitized (warbles (*Oedemagena tarandi*), nasal bots, *Sarcocystis*, *T. krabbei*, *T. hydatigena*, rumen flukes, *Ostertagia sp*. and also lungworm (likely to be *Dictyocaulus sp.*), and had areas of alopecia (hair loss with skin and hair discoloration) on the caudolateral thighs, feet, legs, and perineum. There was also evidence of diarrhea (perineal staining), a swollen hock, evidence of gastrointestinal inflammation and possibly metritis. Much of the histologic findings were similar to the first animal, showing evidence of heavy parasitism, especially in the lungs. This animal also had lesions consistent with enteritis, and had a rumen fluke embedded in the duodenal wall (abnormal location and can cause pathology). The areas of alopecia were associated with hyperplastic dermatitis, and most likely were due to biting insects and the resultant scratching. Interestingly, this animal had several areas of inflammation in the brain, along with basophilic inclusion bodies, which could be due to the causes listed above. This case is more severe than the previous. There was mild interstitial nephritis in this animal as well as chronic active metritis. A culture of the uterus at WADDL yielded many *E coli* and moderate growth of *Enterococcus sp*. Culture of the uterus and serology were negative for *Brucella sp*. Examination of the hock remains to be undertaken.

The final animal (#106883, adult male) was in poor body condition, had some alopecia (hairloss), and was seen lagging behind the other animals and stopped to cough while being pursued by the helicopter. Gross necropsy revealed chronic pleuritis, enlarged lymph nodes, a pancreatic mass, and heavy parasitism (*Ostertagia*, *Sarcocystis*, *Echinococcus cysts*, and rumen flukes). Histologically there was evidence of heavy parasite load, as in the other adults. In the gastrointestinal tract, there is evidence of parasitic infection (nematode eggs and *Balantidium*-type organisms), along with damage to the mucosal surfaces and an eosinophilic infiltrate. A granuloma was also present on the liver. This animal also had frequent areas of inflammation in the brain (perivascular cuffs of lymphocytes, plasma cells, and macrophages), and some free red blood cells in the meninges. This is a somewhat nonspecific reaction and viral and bacterial cultures, including *Listeria*, were negative. Curiously, the thyroid was inactive, which is an incidental finding.

To summarize, these animals were heavily parasitized, all three had *Ostertagia* sp., and spectacular numbers of *Sarcocystis* in the skeletal muscles including the abdominal muscles and diaphragm, and rumen flukes from moderate to severe, while two had severe nasal bots, *Taenia hydatigena* cysts in the liver, and *Echinococcus granulosus* cysts in the lungs. Two of three had a mild interstitial nephritis, which may not be uncommon in caribou, however it may also be Leptospirosis. They also each had varying degrees of meningoencephalitis, which could be caused by several etiologic agents but culture attempts on one animal were negative so immunohistochemistry staining for protozoal parasites is pending.

A urinalysis was conducted on a sample obtained from the bladder of #106878 (adult bull). The urine was normal with a specific gravity of 1.021.

### **2.2.2 Adult Hematology**

Table 5 describes the mean, median, standard deviations, and ranges for the leukogram subpopulations obtained from NAP caribou, captive reindeer and live capture-released Teshekpuk caribou. The NAP caribou had significantly lower total white blood cell counts and among the subtypes, neutrophils, lymphocytes and eosinophils were all significantly decreased compared to TSH caribou. In light of the heavy parasitism and chronic disease state of these individuals, these leukograms indicate a poor immune response to the ongoing infections. Thus, the animals are either immunosuppressed or suffering from immune system exhaustion from the chronic stress.

Table 6 describes the mean, median, standard deviations, and ranges for the erythrocyte indices subpopulations obtained from NAP caribou, captive reindeer and live capture-released Teshekpuk caribou. The NAP caribou had significantly increased hematocrits but not total protein while the red blood cell counts were significantly decreased. This is a complicated picture and the red cell indices of

MCV and MCH, which were increased aid in the interpretation. The low RBC counts with increased corpuscle volume and hemoglobin concentration indicate macrocytic hypochromic anemia which usually results from chronic blood loss. The increase in hematocrit with a low RBC is indicative of dehydration. In a state of dehydration, the total protein is usually increased because of the loss of plasma water however, in these cases, the lack of an increase in the total protein is either from loss of plasma protein or low production of plasma protein. The loss could be again, from the same chronic blood loss that is causing the anemia. A low production would be from a poor nutritional plane or other disease state. These animals were all in very poor body condition with no fat reserves. The lack of increased total protein in the face of dehydration is likely to be a result of both loss due to the blood-sucking parasites and low production due to low protein intake or increase catabolism in these animals.

### **2.2.3 Adult Parasitology**

There were large numbers parasites were detected both on gross and fecal examinations which are summarized in Tables 7 and 8, respectively. Confirmation of the species of adult and larval parasites will be conducted by morphologic and molecular diagnosis by Dr. Eric Hoberg, Chief Curator of the US National Parasite Collection & Animal Parasitic Disease Laboratory USDA, Beltsville, Maryland and results are expect in October 2005. Feces from the seven random adult fecals and the three adult collected animals were all negative for *Cryptosporidium* and *Giardia*.

Table 5. Mean  $\pm$  SD, median, and minimum to maximum ranges of leukocyte parameters in in adult North Alaska Peninsula (NAP) caribou compared with free-ranging arctic Teshekpuk (TSH) caribou, June-July 2005.

	NAP Adults <i>n</i> = 6		TSH Adults <i>n</i> = 17	
	Mean $\pm$ SD	Median (Min-Max)	Mean $\pm$ SD	Median (Min-Max)
Total Leukocytes/ $\mu$ l	<b>5,679*</b> $\pm$ 2,963	4,675 (2,825-11,625)	<b>9,442</b> $\pm$ 2,471	8,875 (5,375-14,500)
Neutrophils:Lymphocytes	0.9 $\pm$ 0.6	0.8 (0.4-1.7)	0.8 $\pm$ 0.5	0.7 (0.1-1.9)
Neutrophils/ $\mu$ l	<b>1,296*</b> $\pm$ 257	1,219 (1,007-1,737)	<b>2,042</b> $\pm$ 694	2,048 (438-2,960)
Band neutrophils/ $\mu$ l	51 $\pm$ 44	59 (0-97)	152 $\pm$ 139	145 (0-545)
Lymphocytes/ $\mu$ l	<b>1,706*</b> $\pm$ 852	1,518 (841-2,790)	<b>3,011</b> $\pm$ 1,243	3,188 (753-5,220)
Monocytes/ $\mu$ l	369 $\pm$ 191	438 (77-581)	701 $\pm$ 480	613 (160-2,030)
Eosinophils/ $\mu$ l	<b>2,238*</b> $\pm$ 2,336	1,406 (994-6,975)	<b>3,624</b> $\pm$ 1,612	3,040 (1,350-6,630)
Basophils/ $\mu$ l	0	33 (32-34)	0	

\* indicates significantly decreased compared to TSH caribou,  $p < 0.5$

Table 6. Mean  $\pm$  SD, median, and minimum to maximum ranges of erythrocyte indices in adult North Alaska Peninsula (NAP) caribou compared with free-ranging arctic Teshekpuk (TSH) caribou, June-July 2005.

	NAP Adults <i>n</i> = 6		TSH Adults <i>n</i> = 17	
	Mean $\pm$ SD	Median (Min-Max)	Mean $\pm$ SD	Median (Min-Max)
Total protein g/dl	6.4 $\pm$ 0.4	6.6 (5.7-6.7)	6.5 $\pm$ 0.6	6.5 (5.5-8.1)
Hematocrit (Hct) %	<b>54.6*</b> $\pm$ 6.4	55.1 (44.8-62.0)	<b>46.1</b> $\pm$ 4.0	47.0 (39.0-51.0)
Hemoglobin (g/dl)	17.8 $\pm$ 2.2	18.0 (14.6-20.30)	16.8 $\pm$ 2.9	16.9 (12.3-22.0)
RBC ( $\times 10^6$ /ul)	<b>8.5†</b> $\pm$ 2.02	9.2 (5.8-10.4)	<b>10.3</b> $\pm$ 1.0	10.3 (8.8-12.1)
CRP (%)	1.5 $\pm$ 1.1	1.4 (0-3.1)	na	
MCV (fl)	<b>66.7*</b> $\pm$ 13.0	61.8 (55.4-89.9)	<b>45.0</b> $\pm$ 2.9	44.7 (36.7-56.6)
MCH (pg)	<b>21.7*</b> $\pm$ 3.8	20.1 (18.7-28.3)	<b>16.0</b> $\pm$ 5.6	15.6 (12.3-22.7)
MCHC (g/dl)	32.7 $\pm$ 0.7	32.7 (31.5-33.7)	36.3 $\pm$ 4.4	36.1 (29.7-43.1)

CRP = Corrected Reticulocyte Percentage

MCV = Mean Corpuscular volume

MCH = Mean Corpuscular Hemoglobin

MCHC = Mean Corpuscular Hemoglobin content

\* indicates significantly elevated compared to TSH caribou,  $p < 0.01$

† indicates significantly decreased compared to TSH caribou,  $p < 0.5$

Table 7. Parasites detected and identified on gross necropsy examination from adult North Alaska Peninsula caribou during late May-early June 2005. The number of + indicate the severity of infection.

Animal #	Abomasal Lesions	<i>Ostertagia</i> sp.	<i>Setaria cervi</i>	<i>Sarcocystis</i> sp.	Nasal Bots <i>Cephenemyia trompe</i>	Warbles <i>Oedemagena tarandi</i>	Lungworm <i>Dictyocaulus</i> sp.	<i>Taenia hydatigena</i> cyst	<i>Echinococcus granulosus</i> cyst	Rumen Flukes Paramphistomes	<i>Taenia krabbei</i> cyst
106878	+	+	+	+	++	-	-	+	++	++	-
106879	+	++	-	++	+	+	+	+	-	+	+
106883	+	+	-	+	-	-	-	-	+	+	-

Table 8. Fecal examination results from North Alaska Peninsula adult caribou during late May-early June 2005. \*The number of + indicate the severity of infection, (EPG) = eggs per gram of feces.

Animal #	Beaker Baermann			<i>Marshallagia</i>	Flotation (EPG)		
	<i>Parelaphostrongylus</i> sp. larvae	<i>Ostertagia</i> sp. larvae	Protostrongyloid larvae		Trichostrongyle	Coccidia	Cestode <i>Monezia</i>
106878	+	-	+	-	- (2.7)	+	+
106879	-	-	+	-	- (37.6)	-	+
106882	+	-	+	-	- (0)	+	+
106883	-	-	+	-	- (17.7)	-	+
Random A	-	+	-	-	- (0)	+	-
Random B	+	+	-	-	+ (1)	-	-
Random C	++	++	-	-	- (0)	-	-
Random D	++	++	+	-	+ (2.7)	-	-
Random E	+	+	-	-	+ (40)	-	+
Random F	-	+	+	-	- (7.5)	-	-
Random G	+	++	-	-	- (3)	-	-

Table 9. Results of serosurveillance as positive/total (% prevalence) serum samples, for selected viral, bacterial and parasitic diseases in North Alaska Peninsula (NAP) and Teshekpuk (TSH) caribou herds by years of collection.

Agent	NAP			TSH
	1982-1999	2001-2004	2005	1986-2001
Respiratory Synchronal Virus	0/189 (0%)	<b>20/34 (59% )</b>	<b>2 adults/10 (20%)</b>	1/58 (2%)
<i>Brucella suis</i>	0/177 (0%)	0/39 (0%)	0/10 (0%)	2/77 (3%)
Bovine Viral Diarrhea	0/89 (0%)	0/35 (0%)	0/10 (0%)	9/52 (17%)
Enzootic Hemorrhagic Disease	0/194 (0%)	0/34 (0%)	0/10 (0%)	0/63 (0%)
Infectious Bovine Rhinotracheitis	0/188 (0%)	<b>13/35 (37%)</b>	<b>7/10 (70%)</b>	0/60 (0%)
Parainfluenza-3	0/145 (0%)	<b>9/39 (23% )</b>	<b>7/10 (70%)</b>	0/60 (0%)
<i>Leptospirosis</i>	0/185 (0%)	0/34 (0%)	0/10 (0%)	0/45 (0%)
<i>Neospora</i>	na	<b>4/31 (13%)</b>	0/10 (0%)	1/4 (25%)
<i>Toxoplasma</i>	0%*	0/31 (0% )	1/10 (10%)	0/5 (0% )

\*state-wide 14/241 (6%) lowest prevalence on AK peninsula with 0% in NAP calves and yearlings (Zarnke et al. 2000)  
na = not available, no tests conducted



## 2.3 Results- Serology

Results of the retrospective and prospective serology are given in Table 9. Results of the same assays are given for the arctic Teshekpuk herd for comparison purposes. The emergence of relatively high antibodies prevalence to BRSV, IBR, PI-3, and *Neospora* between 1999 and 2001 is remarkable. The first three are viral diseases that comprise the bovine respiratory complex. In cattle, these agents are rarely fatal by themselves. They can establish relatively mild viral infection of the lungs (pneumonia or pneumonitis). More importantly, they damage the lungs and provide an opportunity for bacterial infections to become established especially in the presence of lungworms. The bacterial infections can then progress into more serious and potentially fatal pneumonia. The positive serology for the parasites *Neospora* and *Toxoplasma* are of interest because they may indicate exposure to domestic animals. However, seropositive caribou have been detected elsewhere in the state and at this time we have not established pathology associated with these parasites in caribou.

## 2.4 Results- Ancillary Assessments

### 2.4.1 Haptoglobins

Levels of serum haptoglobin were examined as another potential measure of sub clinical (grossly undetected) inflammation. Haptoglobin levels were compared with hemogram values to confirm an expected correlation with indicators of an inflammatory leukogram. Haptoglobin analysis was conducted on 39 NAP serum samples collected from 2001-2004, 10 NAP caribou collected June 2005 and compared to results from 80 Western Arctic herd caribou live captured from 2003-2004. All NAP caribou samples were negative whereas 5% of WAH are positive for haptoglobins.

### 2.4.2 Blood Urea Nitrogen

Concentrations of urea nitrogen were determined in the 4 calf and 6 adult NAP sera samples available from 2005 and compared with 17 neonatal captive reindeer and 61 4-16 month old Interior herd caribou from 2004. The NAP calves had significantly lower ( $p=0.016$ ) mean BUN than reindeer calves,  $6.6\pm 1.3$  (range 5.2-8.0) vs.  $20.8\pm 10.5$  (range 8 – 50) mg/dL. The NAP adults had mean BUN concentrations significantly higher than the 4 to 16 month old Interior reindeer,  $7.7\pm 2.03$  (range 4.5-10.0) vs.  $2.2\pm 0.9$  (range 1.2 – 4.8) mg/dL. This is probably an additional indicator of dehydration rather than decreased renal function.

### 2.4.3 Bone Marrow

Bone marrow was analyzed for fat content as an indicator of nutritional status from the 3 adult NAP caribou that were necropsied. The mean % fat marrow content in NAP caribou was  $56.9 \pm 22.6$  (range 31.6-75.1) vs.  $60.3 \pm 18.6$  (range 32.7-93.3) from 10 caribou marrow samples available from elsewhere. The mean %fat content tended to be lower in NAP caribou than the comparison group, however the difference was not statistically significant.

### 2.4.4 Trace Minerals

Blood and tissues were collected from NAP caribou as well as caribou from other herds in Alaska, for comparison of blood and tissue trace mineral levels. Elements that will be determined are copper, selenium, zinc, cadmium, lead, iron, molybdenum, and arsenic. These analyses are ongoing and results should be available by the end of 2005.

## 3 Discussion

Predators killed all but one of the calves examined in the first two weeks of life even though many were not consumed (especially when wolves were involved). Thus, predation by wolves and bears was the primary cause of direct mortality in young calves. This predation rate is remarkably high. In the Porcupine caribou herd predation accounted for 13% of mortality in the first two weeks of life and similarly to the present study, there 1 case of drowning (2%) (Roffe 1993). However, even as neonates NAP caribou show evidence of exposure to viral diseases and parasites that could significantly impair their health, growth and survival.

Prior to 2000, none of the bovine respiratory diseases had been detected in this herd (Zarnke 2000). However, NAP caribou serum samples collected from 2001, 2004 and 2005 indicate the herd has been exposed to bovine respiratory viruses, as well as *Neospora*. The previous single lung sample submitted for pathology suggested BRSV and one of the 2005 calves had pulmonary inclusion bodies and seroconversion consistent with PI-3. Calves were shedding larvae consistent with *Protostrongylus* sp. lungworm and have exposure to the bovine respiratory viral complex putting them at risk for morbidity and mortality from pulmonary infections. The hematologic data support the conclusion that viral disease and parasites are adversely affecting the health of NAP caribou calves. Their total leukocyte and lymphocytes counts were depressed while eosinophils and immature neutrophils were increased.

The findings of *Ostertagia* sp. abomasal lesions and pathology supporting significant adverse effects massive parasitic infections in the adults are particularly important. *Ostertagia* is a parasite that can cause poor calf survival and poor growth and body condition in *Rangifer* (Stein *et al.* 2002a, b) and food intake (Arneberg *et al.* 1996). During the perinatal period, female caribou increase the shedding of eggs and the calves are infected early. Those calves that do survive predation are subject to intense, early exposure to infective stages of parasites. In addition, the presence of *Toxoplasma*

*nativa* and *Neospora* sp. could contribute to increased fetal loss and the birth of weak calves with poor growth and low survival. Additionally, severe infections of a number of other parasites were also detected. There were large numbers of nasal bots in two of three adult caribou, severe enough to cause upper respiratory obstruction and affect the animal's ability to escape predation. Fear of fly strike in the nose has been known to drive caribou and reindeer nearly 'mad' so that they remain in unsuitable grazing habit to avoid flies or run wildly about trying to escape flies and waste energy. NAP caribou have been observed demonstrating behaviors indicative of fly avoidance. *Sarcocystis* was also present in massive numbers in the muscle of all three adult caribou. Again, this could affect the animal's stamina, growth and ability to migrate as well as escape predation.

Hematologic studies of the 2005 live-captured and collected caribou support the conclusion that disease, or other stressors, and parasites are adversely affecting the health of NAP caribou. The total circulating leukocytes as well as the subpopulations of neutrophils, lymphocytes, and eosinophils are depressed compared to Teshekpuk caribou. The hemogram demonstrates that NAP adult caribou have decreased numbers of erythrocytes compared to Teshekpuk caribou. The erythrocytes are larger and have lower concentrations of hemoglobin in them indicating a macrocytic hypochromic anemia. However, the hematocrit is increased while the total protein is slightly lower. This may indicate that the caribou are somewhat dehydrated while being hypoproteinemic. These are additional indicators of the poor nutritional plane of these animals.

## 4 Conclusion

It is crucial for this health assessment to know which pathogens this herd has been exposed to and is affected by as well as their overall health to be able to develop mitigation strategies. The health assessment can be accomplished by analysis of the samples collected during this field investigation (pathologic, serologic, and parasitic surveys) along with the retrospective serology. In addition, I suggest that a sample of 5 to 10 calves be collected again in the fall (along with fresh fecal samples off the ground) to investigate the significance of *Ostertagia* sp. and the respiratory viruses that calves may have become infected with over the summer. In order to complete the health assessment of this herd, we need the additional information of the health status and disease prevalence in calves going into winter. Blood from healthy caribou herds should also be obtained for comparison purposes and will require additional funding to complete the analyses.

## 5 Acknowledgements

This project would not have been possible without the expertise and generous assistance of Bruce Dale, Lem Butler, Justin Gude, Ron Squibb, Chris Maurer, Kristin Sesser, Dave Cox, Christine Terzi, and Patricia Rivera. Project funding in part came from the Summer Program in Conservation, Wildlife Population Medicine, and Ecosystem Health UI, ADF&G, and the Alaska Peninsula and Becharof National Wildlife Refuge.

## 6 Literature Cited

ADF&G Caribou Management Report, 2002. . Alaska Department of Fish and Game. Federal Aid in Wildlife Restoration. Research Report. Grants W-27-4 and 5 Project 3.0. Juneau, Alaska. P53-63.

Forrester, S. G., and M. W. Lankester. 1997. Extracting protostrongylid nematode larvae from ungulate feces. *Journal of Wildlife Diseases* 33: 511-516.

Mckenna, P. B. 1999. Comparative evaluation of two emigration/sedimentation techniques for the recovery of dictyocaulid and protostrongylid larvae from faeces. *Veterinary Parasitology* 80: 345-351.

Arneberg, P., Folstad, I., and Karter, A.J. 1996. Gastrointestinal Nematodes Depress Food Intake in Naturally Infected Reindeer. *Parasitology*. 112(2):213-219.

Neiland, K.A. 1970. Weight of Dried Marrow as Indicator of Fat in Caribou Femurs. *The Journal of Wildlife Management*. 34(4):904-907.

Roffe, T.J. 1993. Perinatal Mortality in Caribou from the Porcupine Herd, Alaska. *Journal of Wildlife Diseases*. 29(2):295-303.

Stien, A, Irvine, R.J., Langvatn, R., Albon, S.D., and Halvorson, O. 2002a. The Population Dynamics of *Ostertagia gruehneri* in Reindeer: a Model for the Seasonal and Intensity Dependant Variation in Nematode Fecundity. *International Journal for Parasitology*. 32:991-996.

Stein, A., Irvine, R.J., Ropstad, E., Halvorson, O., Langvatn, R., and Albon, S.D. 2002b. The Impact of Gastrointestinal Nematodes on Wild Reindeer: Experimental and Cross-Sectional Studies. *Journal of Animal Ecology*. 71:937-945.

Zarnke, R.L., Dubey, J.P., Kwok, O.C.H., and Ver Hoef, J.M. 2000. Serologic Survey for *Toxoplasma Gondii* in Selected Wildlife Species From Alaska. *Journal of Wildlife Diseases*. 36(2):219-224.

Zarnke, R.L. 2000. Alaska wildlife serologic survey, 1975-2000. Alaska Department of Fish and Game. Federal Aid in Wildlife Restoration. Research Final Report. Grants W-24-5 and W27-1 through W-27-4. Study 18.71. Juneau, Alaska. P 3-4.