Alaska Wildlife Serologic Survey, 1975-2000

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Research Final Report
Grants W-24-5 and W-27-1 through W-27-4
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CONTENTS

INTRODUCTION AND BACKGROUND

I have been coordinating the collection and testing of wildlife sera in Alaska since the late 1970s. Since our serologic survey began, I have sent memos to participating individuals and filed annual reports covering new data. In the early 1990s a supervisor suggested that I prepare a comprehensive summary of this project (written for the nonspecialist) with emphasis on geographic patterns of disease. That summary was then distributed to individuals who had contributed sera or were otherwise interested in patterns of exposure in Alaska. That report had a "hot pink" cover and became known by that color.

This report is intended as an update to the original "hot pink" report. Background information is provided for each disease agent in an effort to make this document more useful. Hopefully, this report will be the kind of summary that you will want to keep for future reference. Additional copies are available upon request.

The text and tables in this report are based on the results of more than 100,000 serologic tests. The raw data was condensed into 160 "summary tables" prior to writing this report. Each summary table represents a particular host species and a particular disease agent. For example, there is a single table that summarizes all *Brucella* sp. test results for caribou. Within the body of this table, results are presented for each year and each caribou herd. A CD version of these summary tables (and the text of this report) is available to interested individuals upon request (see contacts/addresses inside front cover).

We have collected over 18,000 serum samples representing more than 30 species in our Alaska Wildlife Serum Bank. Sera are stored in freezers at –50 °C. At these temperatures, specimens should retain biochemical properties almost forever. Long-term storage becomes increasingly valuable as new analytical techniques are developed and new disease agents are discovered. For example, recent genetic analyses have provided information on the relationship between populations of several species.

I make three guarantees to individuals who submit sera:

1 Test results (and a brief analysis) will be forwarded within 1 week after they are available to me.

2 Individuals who submit sera will continue to have access to those samples. Sera can be shipped to the original collector or to a third party upon request.

3 I periodically receive requests from individuals in other agencies for sera from our collection. I always request permission from the original collector prior to shipping sera.

More than 100,000 tests have been performed on these sera. The large scale of this collection allows more accurate determination of disease patterns. A large,

uniform survey is more valuable than several small, disjunct surveys. As the collection continues to grow, our knowledge will also increase.

There are several instances in this document in which I have reported an apparent anomaly in the data without presenting an accompanying explanation. The reason that I have not provided explanations is that none is available. Some of this information is so "new" that we are not sure how to interpret the results. Please contact me if you have insight into any of these cloudy areas. Comments on format and style are also welcome.

I have attempted to avoid the use of technical jargon. Perhaps the only term that needs definition is "prevalence." Prevalence refers to the frequency with which different disease agents occur in a population. Prevalence is presented in the form of a fraction. The numerator is the number of samples with evidence of exposure to the disease agent. The denominator is the total number of samples tested.

Presence of antibody to a particular disease agent in a serum sample does not necessarily mean that the animal experienced any symptoms of that disease. When an animal is exposed to a disease organism, the animal's immune system recognizes that invading organism as being foreign or "non-self." The immune system produces antibodies in an attempt to combat this invading organism. We can detect that antibody by means of various test procedures. Thus, the presence of antibody in an animal's serum indicates ONLY that it has been exposed to the organism in question. Presence of antibody does not necessarily indicate that the animal suffered any signs of the disease.

An important issue related to disease surveys is the question of how many samples are necessary to adequately assess the frequency with which a disease is detected in a population. Sample size is critical to the interpretation of serologic survey data. A single collection of 10 samples from a population numbering in the thousands will probably not provide a meaningful estimate. On the other hand, a large one-time collection may not be the best answer, either. Several small collections spaced over several years will often be more informative than a single large collection.

I sincerely thank everyone who has collected and submitted samples for this serologic survey. Without your diligence, this project would not have achieved the level of information that is currently available. This report was funded by Federal Aid in Wildlife Restoration and the Alaska Department of Fish and Game.

I hope that all contributors will continue to collect and submit specimens obtained during fieldwork. Please encourage other wildlife investigators to contribute as well. Supplies and instructions are available upon request. Increased participation results in increased knowledge of the health status of our wildlife populations statewide. Interaction between domestic livestock and free-ranging wildlife species holds the potential for the spread of diseases to wildlife. Serologic surveys can provide invaluable information for protecting wildlife from these negative impacts.

BLUETONGUE AND EPIZOOTIC HEMORRHAGIC DISEASE

I. AGENT(S) – viruses; closely related to each other and presumably arose from a common ancestor

II. HOST(S) – Bluetongue virus is most commonly associated with domestic sheep and cattle. Epizootic hemorrhagic disease virus (EHD) is most commonly associated with wild ungulates, such as pronghorn and members of the deer family. Neither of these simplistic categorizations is 100% valid. Both viruses are capable of crossing these arbitrary boundaries. In Alaska, we consider deer, elk, caribou, moose, bison, muskoxen, and Dall sheep as potential hosts.

III. SIGNS AND SYMPTOMS – These diseases are not easily diagnosed in live, free-ranging wildlife. External manifestations are often not specific enough to provide a clear-cut picture to an investigator who is unfamiliar with the diseases. Signs may include swelling of the head and neck, increased respiration and heart rate, excess salivation, blood in urine and feces, bleeding at the hoof line, and sloughing of hooves. Internal signs are often more dramatic. Massive hemorrhaging may occur in any of the several organs including liver, heart, spleen, kidney, lung, and intestines.

IV. TRANSMISSION – Large-scale epizootics of both diseases may occur in the Lower 48 during the fall of the year. Such outbreaks are usually associated with wet weather and low-lying areas. *Culicoides* spp. midges are known to serve as biologic vectors under these conditions. The midge species responsible for the vast majority of transmission in the Lower 48 is *C. variipennis*. This species is not known to occur in Alaska. Other members of the genus are quite common here. There has been speculation that the apparent absence of *C. variipennis* indicates that an epizootic of either agent could not occur in Alaska. General ecological principles and evidence of these two diseases from other parts of the world indicate that when a niche is unoccupied, some other member of the genus will step in to fill the void.

Epizootics do not occur every year in the Lower 48. Other methods have been hypothesized for transmission during these periods between epizootics. The method receiving the most attention has been some form of oral transmission. This aspect of the epizootiology of these two diseases remains unresolved.

V. EFFECT(S) – Most of the symptoms listed above occur due to effects on blood circulation. Two related phenomena are responsible: 1) disruption of normal clotting mechanisms and 2) increased permeability of blood vessels. In simple terms, blood clots occur inside the vessels and unclotted blood then leaks out into surrounding tissues.

VI. CONFIRMATORY DIAGNOSIS – In severe cases, internal signs of disease are strongly indicative of these hemorrhagic diseases. Additional external support is gained when observed signs of disease are considered in combination with time

of year, locale, and records of previous outbreaks in the vicinity. Final confirmation depends on isolation, purification, and identification of the virus.

VII. PREVALENCE – As described in the 1992 report, antibody prevalences for both BLU and EHD are extremely low in Alaska. In the absence of independent confirmation, I would be tempted to dismiss the few positive results as incorrect (aka "false positive" results). However, the serologic testing lab has reconfirmed the validity of the results.

Positive samples for both BLU and EHD occur sporadically. For example, cumulative prevalence for EHD was 8/149 for Dall sheep from the central Alaska Range during the period 1972–1992. All 8 of those positive samples were from animals captured during 1981. Similarly, cumulative prevalence for EHD was 3/117 for sheep from the eastern Alaska Range during 1984–1992. All 3 of the positive samples were collected during 1991. Similar results are found for many of the other species included in the survey. Apparently, transmission of these viruses only occurs sporadically.

Serum antibody prevalence of bluetongue and epizootic hemorrhagic disease viruses in selected species of Alaska wildlife.

Species	Bluetongue (%)		EHD (%)	
Bison	1/813	(<1)	3/830	(<1)
Deer	0/98	(0)	2/98	(2)
Caribou	13/2908	(<1)	16/3240	(<1)
Dall sheep	2/506	(<1)	11/501	(2)
Elk	0/27	(0)	1/27	(4)
Moose	22/1903	(1)	40/1942	(2)
Mountain goat	2/88	(2)	0/91	(0)
Muskox	0/204	(0)	1/200	(<1)

BOVINE RESPIRATORY GROUP VIRUSES

I. AGENTS – viruses; infectious bovine rhinotracheitis (IBR)
bovine viral diarrhea (BVD)
parainfluenza 3 (PI3)
respiratory syncytial virus (RSV)

II. HOST(S) – As the generic name for this group of viruses implies, they were initially recognized because of their ability to cause disease in domestic cattle. When wildlife disease investigators began to monitor the health status of wildlife species, they found evidence of these agents in a wide variety of ungulates. Serologic evidence of exposure to these viruses is relatively common in some wildlife species. However, cases of actual disease have been rare. In Alaska, we can assume that the following species are at least susceptible to infection: bison, deer, caribou, moose, mountain goat, muskox, and sheep.

III. SIGNS AND SYMPTOMS – Loss of appetite, excess salivation, coughing, labored breathing, and nasal discharge.

IV. TRANSMISSION – Infected animals expel infectious virus in respiratory aerosol droplets. Susceptible animals become infected when they inhale these droplets. There is also evidence of venereal transmission.

V. EFFECT(S) – In cattle, these agents are rarely fatal by themselves. They can establish relatively mild viral infection of the lungs (otherwise known as "pneumonia"). More importantly, they can provide an opportunity for bacterial infections to become established. These infections can then progress into more serious bacterial pneumonia.

Infection can also localize in the gastrointestinal tract where it causes diarrhea. If the female reproductive tract becomes involved, abortion may result.

VI. CONFIRMATORY DIAGNOSIS – Serologic tests can provide an indication of the status of a group of animals. To have confidence regarding the exposure status of an individual animal, two or more blood samples should be collected over a period of several weeks. The ultimate confirmation is provided by isolation of the agent from the animal followed by purification and identification of the virus.

VII. PREVALENCE – Two major patterns were described in the 1992 report:

(a) Dramatic increase in antibody prevalence of PI3 in Delta bison from 0% prior to 1977 up to 100% by 1984. This pattern has remained stable since 1992 (611/639 = 96%). Presumably, the virus was introduced into the bison herd from domestic cattle (Appendix A).

(b) Higher prevalence for all four viruses in the Arctic caribou herds (Western Arctic, Teshekpuk, Central Arctic and Porcupine) as

contrasted to all other herds in the state. Apparently, some environmental factor favors transmission of these viruses in the Arctic region.

Serum antibody prevalence of respiratory viruses in caribou herds from geographic regions of Alaska.

	IBR (%)	BVD (%)	PI3 (%)	RSV (%)
Northern	53/1436 (4.0)	149/1385 (11.0)	87/1476 (6.0)	6/1090 (0.5)
Southern	17/1199 (1.5)	0/1267 (0.0)	11/1101 (1.0)	0/1150 (0.0)

There have been some minor changes to the caribou pattern:

(a) During the latter half of the 1990s, we have seen limited evidence of exposure to IBR and PI3 in herds from the southwestern portion of the state (Mulchatna, Nushagak, Northern Alaska Peninsula and Southern Alaska Peninsula). The Northern Alaska Peninsula (NAP) Herd declined significantly during the late 1980s and early 1990s. During the early 1990s, we had found clinical evidence of pneumonia in NAP animals. At that time, nematode lungworms appeared to be the cause of the pneumonia. However, respiratory viruses may also have played a role.

(b) Evidence of IBR and PI3 exposure has been found in the Galena Mountains Herd and Fortymile Herd. I conclude that this reflects interaction with the Western Arctic and Porcupine herds, respectively.

(c) Evidence of RSV exposure has appeared in the Central Arctic, Teshekpuk, and Porcupine herds. This data fits the pattern for the other three viruses of higher prevalence in the Arctic herds.

Antibody prevalence of IBR in moose from the Nelchina Basin averaged approximately 10% during the 1970s. Few test results are available since that time. Therefore, we cannot determine if this pattern has continued. Prevalence for PI3 is low but consistent for moose from the southern half of the state. Prevalence for both PI3 and BVD are higher (5–15%) for moose in the Arctic. Apparently, the factors that result in high prevalence of these viruses in the Arctic caribou herds affect the Arctic moose populations in the same manner. There was no evidence of RSV in moose.

Antibody prevalence for these viruses is very low (essentially 0%) in deer, elk, and mountain goat. With one exception, the same can be said for Dall sheep and muskox. That exception involves PI3 in the eastern Arctic. Serologic evidence of exposure appeared in both sheep and muskox in that region during the early 1990s. Presumably, those new data reflect the same factors that cause prevalence to be higher in the Arctic caribou herds.

BRUCELLOSIS

I. AGENT – bacterium; *Brucella suis* IV

II. HOST(S) – There are several species of *Brucella*, each of which is commonly associated with a particular host species. For example, *B. abortus* is usually associated with domestic cattle. *B. canis* with domestic dogs. *B. suis* IV is commonly found in reindeer, caribou, and their associated predators and scavengers such as bears, wolves, foxes, and humans.

III. SIGNS AND SYMPTOMS – Infection in caribou usually localizes in 1) the reproductive tract, 2) skeletal joints, or 3) lymph nodes. Infection of the female reproductive tract can cause abortion and retained placentas. Infection of the male reproductive organs can result in grossly enlarged testicles and sterility. Infection of joints in either sex can cause large abscesses, which may result in arthritis and lameness. Infected lymph nodes may be enlarged and pus-filled. Our knowledge of signs in other species, such as wolves and bears, is limited.

IV. TRANSMISSION – An aborted fetus and any accompanying fluids from an infected female contain extremely high levels of infectious bacteria. Caribou are curious animals and will often investigate an aborted fetus by sniffing and licking. Under this scenario, transmission occurs by means of 1) aerosol droplets containing bacteria coming into contact with mucous membranes in the corner of the eye or 2) ingestion.

Abscesses also contain large amounts of infectious bacteria. If these are cut open or rupture, the contents may be transmitted through open wounds in a susceptible animal. Venereal transmission has been hypothesized but not proven.

V. EFFECT(S) – Abortion and sterility result in decreased herd productivity. Arthritis and lameness render an animal more susceptible to predation.

VI. CONFIRMATORY DIAGNOSIS – Serologic testing is useful in determining the prevalence of exposure in a herd. Serology is less reliable for evaluating the status of any single animal. Most experts agree that if serology is going to be used for diagnosis, then each sample should be tested by more than one method. The ideal diagnostic method involves isolating, purifying, and identifying the organism from lymph nodes, aborted materials, or abscesses.

VII. PREVALENCE – The standard interpretation is that the disease occurs in all caribou herds in Alaska. That interpretation may or may not be accurate. However, our long-term large-scale serologic survey (2757 samples tested since 1975) clearly demonstrates that the disease is more common in the northern caribou herds, especially the Western Arctic and Porcupine herds. Lower prevalence was found in two herds from the western Interior (Galena Mountains and Ray Mountains). I believe these latter data reflect interaction (either direct

or indirect) between these two herds and the Western Arctic Herd. Prevalence is essentially 0% in the other herds in Alaska.

Caribou (%)			Grizzly bear (%)			Wolf (%)		
Western Arctic	50/629	(8)	Unit 26A	80/366	(22)	Unit 26A	15/60	(25)
			Unit 22	10/76	(13)			
			Unit 23	39/203	(19)			
				129/645	(20)			
Teshekpuk	1/64	(2)	Unit 26B	6/50	(12)			
Central Arctic	8/358	(2)						
Porcupine	9/306	(3)	Unit 26C	50/374	(13)	Unit 26C	1/45	(2)
Galena Mtns	5/42	(12)				Units 21C & 21D	1/26	(4)
Ray Mtn	1/21	(5)						
Delta	0/165	(0)	Unit 20A	6/270	(2)	Unit 20A	3/248	(1)
Nelchina	1/214	(0.5)	Unit 13	12/156	(8)	Unit 13	1/76	(1)
All others	0/958			38/480	(8)		3/497	(0.6)

Predators can be exposed to *Brucella* spp. while consuming infected prey species (Appendix B). Wolves and grizzly bears are the primary predators of caribou. Antibody prevalence in these two species reflects the same geographic pattern as described above for caribou. Prevalence is higher in Arctic locations, especially the western Arctic. Note the presence of positive bears in more southerly areas of Alaska in the above table, where prevalence for caribou is essentially 0%. Apparently, bears and wolves serve as a sensitive system for detecting the presence of brucellosis in the local caribou herd.

Prevalence in wolves is similar to (or perhaps slightly higher than) prevalence in caribou. However, prevalence in grizzly bears can be several-fold higher than in caribou. Bears have a longer average lifespan than caribou and wolves. Perhaps, the higher antibody prevalence in bears reflects a greater opportunity for exposure during this longer lifespan.

Brucellosis in bison and elk is a major problem in and around Yellowstone Park. Those animals are infected with *B. abortus*, which is commonly considered to be the cattle strain of *Brucella*. We do not believe that *B. abortus* or *B. suis* IV occur in any of the bison herds in Alaska. One bison sample (from the Delta Junction herd in 1988) gave a positive test result by one test method. Unfortunately, that test exhausted the supply of serum from that animal. Thus, we were unable to conduct confirmatory tests. There have been no suspicious test results since that time. I conclude that the result for the 1988 sample was a "false positive." I do not believe that *Brucella* spp. are present in any of the Alaska bison or elk herds.

Verified reports of brucellosis in moose are extremely rare. For many years, people speculated that moose were largely resistant to infection. However, serologic evidence of exposure was found in a high percentage of moose from the central North Slope following a major decline in the moose population during the late 1980s and early 1990s. Subsequently, higher than expected antibody prevalence was also found in moose from the western Arctic. Presumably, this

high prevalence in moose reflects interaction with caribou herds that also have high prevalence. Productivity of the moose population on the central North Slope was very low during the decline. We can only speculate whether brucellosis was a factor in that low productivity.

Brucella sp. have been isolated from a muskox on Nunivak Island. All of the free-ranging herds of muskox on the mainland originated from Nunivak stock. However, no serologic evidence of *Brucella* sp. has been found in any of these mainland herds.

There was no serologic evidence of exposure in deer, Dall sheep, mountain goat, or fox sera.

CANINE CORONAVIRUS

I. AGENT – virus

II. HOST(S) – canids – wolf, fox, coyote

III. SIGNS AND SYMPTOMS – Diarrhea and dehydration

IV. TRANSMISSION – Transmission occurs via the fecal-oral route. Infected animals shed virus in feces. Susceptible animals are exposed when they ingest food or water contaminated by virus.

V. EFFECT(S) – Effects of strictly coronavirus infection are usually minor to moderate. However, dual infection by both corona and parvo are common. Severity of symptoms are magnified by this dual infection.

VI. CONFIRMATORY DIAGNOSIS – Microscopic examination of feces and/or intestinal lining may reveal virus. Virus can also be isolated from these same specimens by inoculating tissue culture or live animals.

VII. PREVALENCE – Antibody prevalence exhibited a STRONG seasonal pattern for three areas in the Interior. Prevalence was 0% (0/42) for 4-month-old pups in the autumn. Prevalence in the 10-month-old pup cohort rose to nearly 60% (58/97). Prevalence in the remainder of the population averaged 25% in the autumn and 75% in the spring. Obviously, the primary period for transmission occurred during the winter. In addition, antibody decay rates for individual animals must be fairly rapid for the prevalence to decline so dramatically over the summer. See Appendix C (wolf/CCV abstract) for more information.

VIII. COMMENTS – Canine coronavirus is probably not a major source of mortality for wolves in Alaska.

CANINE DISTEMPER

I. AGENT – virus

II. HOST(S) – Canids—wolf, fox, coyote
Mustelids—weasel, mink, marten, otter, wolverine

III. SIGNS AND SYMPTOMS – Red eyes, crusty exudate around eyes and nose, loss of appetite, increased thirst, diarrhea, labored breathing, thickened foot pads, skin of head swollen, poor quality fur.

IV. TRANSMISSION – Infected animals shed virus in urine, feces, or nasal exudate. Susceptible animals may be exposed when they come into direct contact with virus in excretions or secretions, or if they inhale aerosolized virus.

V. EFFECT(S) – Infection of eye tissue can lead to blindness. Behavior may change. Infected animals may lose their fear of humans or even become aggressive toward humans. In the latter stages of disease, convulsions and paralysis may occur. Fatality rates are highly variable in captive populations, ranging from 20% to 90% of those animals that are exposed.

VI. CONFIRMATORY DIAGNOSIS – Microscopic examination of preserved lung, spleen, or bladder tissue often provides strong evidence of distemper. Ideally, such evidence should be confirmed by means of isolation, purification, and identification of virus from these same tissues.

VII. PREVALENCE – Previous studies in Alaska and the Yukon had reported fairly low and stable antibody prevalences in the neighborhood of 10% (Appendix D) for canine distemper virus. At the time of the 1992 report, data were quite limited. A substantial amount of data have been accumulated since that time.

Current data indicates that antibody prevalences for distemper in wolves ranged from 0% to 35% in Alaska. Prevalences were higher in the Yukon Territory, ranging from 33% to 64%. I cannot offer an explanation for this obvious discrepancy in prevalence between Alaska and the Yukon. There is no apparent geographic pattern in the current data for Alaska, i.e., one location may have high prevalence whereas an adjacent location has low prevalence. There was no evidence of exposure in sera of a limited number of red fox and arctic fox.

Serum antibody prevalence of canine distemper virus in wolves from selected geographic areas of Alaska.

Area	Prevalence (%)
Cordova	0/20 (0%)
Nelchina Basin	10/95 (11%)
Tanana Flats	0/240 (0%)
McKinley Park	70/237 (30%)
Fortymile	4/202 (2%)
Kanuti	6/25 (24%)
Galena	18/55 (33%)
Western arctic	4/77 (5%)
Eastern arctic	16/46 (35%)
Yukon-Aishihik	10/30 (33%)
Yukon-Finlayson	30/81 (37%)
Yukon-North Slope	14/22 (64%)

There may be a chronologic or temporal pattern of antibody prevalence for CDV in wolves. Pertinent data were available for two areas (see tables below). It appears as if distemper may occur in extended epizootic periods where a large proportion of the wolves are exposed. These epizootics are followed by extended periods when transmission is limited.

Antibody prevalence was high in Mount McKinley National Park from the mid-1980s through the mid-1990s. However prevalence then declined significantly.

Period	Prevalence (%)
1986–1995	63/173 (36%)
1996–1999	7/64 (11%)
Highest 1992	16/20 (80%)

Antibody prevalence showed a similar chronologic pattern for the southern Yukon.

Period	Prevalence (%)
1986–1994	40/100 (40%)
1997–1998	0/11 (0%)
Highest 1991–1992	30/35 (86%)

VIII. COMMENTS – Distemper has been documented as a direct source of mortality for wolves. However, it does not appear to be a major source of mortality on a widespread geographic scale.

CANINE PARVOVIRUS

I. AGENT – virus; related viruses in felids and mustelids

II. HOST(S) – Canids—wolf, fox, coyote

III. SIGNS AND SYMPTOMS – Diarrhea (sometimes bloody), dehydration, loss of appetite, lethargy.

IV. TRANSMISSION – Transmission occurs via the fecal-oral route. Infected animals shed virus in feces. Susceptible animals may be exposed when they ingest food or water contaminated by the virus.

V. EFFECT(S) – Infection of heart muscle may lead to stunted growth. In severe cases, can be fatal. Infection of gastrointestinal tract may range in intensity. Mild cases in adult canids may be almost unnoticed. Severe cases in pups may be fatal.

VI. CONFIRMATORY DIAGNOSIS – Microscopic examination of preserved feces, GI tract, or heart muscle may reveal virus. Virus can also be isolated (from fresh specimens of these same tissues) in tissue culture or live animals.

VII. PREVALENCE – The only host species that we have much data for is the wolf. Antibody prevalence in wolves ranged from a low of 10% in a small sample from the Cordova area to a high of 76% in a moderately sized sample near Galena. There was no apparent geographic pattern. Antibody prevalence rose dramatically from 0% in the late 1970s to 50% by the mid 1980s in the Nelchina Basin (Appendix D). However there have been no apparent time-specific patterns since that time.

Serum antibody prevalence of canine parvovirus in wolves from selected geographic areas of Alaska.

Area	Prevalence (%)
Cordova	2/21 (10)
Kenai Peninsula	6/11 (55)
Mat-Su Valley	18/27 (67)
Nelchina Basin	17/98 (17)
Fortymile	57/21 (27)
Tanana Flats	116/240 (48)
Denali	60/223 (26)
Kanuti	17/52 (33)
Galena	32/42 (76)
Western Arctic	42/77 (55)
Eastern Arctic	10/40 (25)

VIII. COMMENTS – Canine parvovirus appeared *de novo* in 1977. It caused a worldwide outbreak. Many domestic dogs died during the late 1970s from this

disease. The virus was spread to free-ranging canids when dogs ventured into wildlife habitat and when wild canids ventured near human habitations.

Many people have wondered about the effect of this disease on free-ranging wolves. To my knowledge, there have been two experiments that could help address this question:

(a) a traditional, controlled experiment, and

(b) a "real life" experiment.

In the controlled experiment, eight pups were intentionally exposed. Seven of the pups became ill. One of the seven died. In the "real life" experiment, 12 pups were housed in a large enclosure. A natural outbreak developed. Eleven of the 12 pups died from the disease. The results of these two outbreaks contradict each other. One suggests that the disease could be a major source of mortality for free-ranging wolves. The other suggests that parvo would be at best a minor factor. Long-term studies of wolf pack dynamics in Alaska indicate that parvo has not been a major mortality factor. See Appendix D for additional information.

INFECTIOUS CANINE HEPATITIS

I. AGENT – virus

II. HOST(S) – wolves, foxes, coyotes, bears

III. SIGNS AND SYMPTOMS – Loss of appetite, vomiting, diarrhea, mucus or blood in feces, runny nose with crusted exudate around nose and eyes, lethargy, seizures, paralysis.

IV. TRANSMISSION – Infected animals shed virus in respiratory droplets, saliva, urine, and feces. Susceptible animals become infected when they come into direct contact with these secretions or excretions.

V. EFFECT(S) – Signs and symptoms listed above indicate that infected animals outwardly appear to be very sick. Signs may last for several days. Death is not uncommon in captive wild canids. For example, mortality rates in ranch-raised foxes may reach 20% for otherwise healthy adults and 80% for juveniles.

VI. CONFIRMATORY DIAGNOSIS – Microscopic examination of preserved tissue can provide strong evidence. Confirmation by isolation of the virus from liver, kidney, or lung is preferred.

VII. PREVALENCE – Previous studies have documented high antibody prevalence of ICH in wolves from Alaska and Canada (Appendix D). Antibody prevalence of ICH in wolves remains high in all areas and all years. From 1975–1999, prevalence was 89% (932/1051) for 13 areas in Alaska. Prevalence was only slightly lower (103/128 = 80%) for four areas of the Yukon Territory from 1984–1998.

Serum antibody prevalence for infectious canine hepatitis virus in wolves from selected geographic areas of Alaska.

Area	Prevalence (%)
Cordova	20/24 (83)
Kenai	7/11 (64)
Nelchina	81/98 (83)
Fortymile	186/194 (96)
Tanana Flats	222/239 (93)
McKinley Park	200/233 (86)
Kanuti	37/43 (86)
Galena	44/51 (86)
Western arctic	73/77 (95)
Eastern arctic	38/40 (95)
Canada-Aishihik	21/28 (75)
Canada-Finlayson	59/74 (80)
Canada-North Slope	22/23 (96)
Canada-Southern Lakes	1/3 (33)

VIII. COMMENTS – There have been few confirmed cases where wolves in Alaska have died due to clinical ICH infection. Apparently, the strain of ICH that is circulating in Alaska is not highly pathogenic for wolves. Data presented here proves that most wolves are exposed to the virus. If the virus was highly pathogenic, clinical disease and death would be common.

Conversely, ICH does appear to be pathogenic for juvenile grizzly bears (Zarnke et al. 1987; Appendix E). Antibody prevalence in bears <2 years is 0%. Statistical analysis indicates that prevalence in this age cohort is lower than expected. There are several potential explanations for this pattern. The most likely explanation is that there is high mortality in the juvenile cohort following exposure. Anecdotal evidence from a European zoo supports this hypothesis.

CONTAGIOUS ECTHYMA (CE)

I. AGENT – virus

II. HOST(S) – Historically, ecthyma was known as a disease of domestic sheep and goats. During this century, the disease has been reported from numerous wildlife species worldwide. In Alaska, the two most common wildlife hosts are Dall sheep and mountain goats.

III. SIGNS AND SYMPTOMS – The virus prefers unhaired portions of the skin. Dark-colored, crusty scabs occur around the nose, eyes, ears, anus, genitalia, and the coronary band of the hoof.

IV. TRANSMISSION – Scabs contain large amounts of infectious virus. As scabs heal, they drop to the ground. Virus can remain infectious for decades in this condition. Transmission occurs when susceptible animals come into contact with these virus-laden scabs. Transmission can also occur between an infected ewe and her susceptible lamb during nursing.

Viral latency may play a role in the occurrence of clinical CE lesions in sheep (Appendix F). Viruses that employ this strategy are able to "hide" in the body. Under stressful conditions, the virus emerges from hiding and causes clinical signs of disease. After the disease has run its course, the virus goes back into hiding. This process can repeat throughout the life of an animal. One attempt to reactivate a possible latent CE infection in Dall sheep was unsuccessful (Appendix G).

V. EFFECT(S) – Scabs adjacent to the eyes can obstruct vision. In severe cases, animals have become blind. Scabs around the ear can interfere with hearing. In severe cases, the external ear has become so extensively involved that is has fallen off. Scabs surrounding the mouth can interfere with feeding. Scabs on the coronary band of the hoof can make walking so painful that an animal becomes reluctant to move. These conditions are usually more common and severe in young animals.

VI. CONFIRMATORY DIAGNOSIS – Serologic tests are a fairly reliable method of determining previous exposure to the virus. Conclusive evidence is provided by isolation, purification, and identification of the virus from scab material.

VII. PREVALENCE – There have been several easily identifiable cases in mountain goats from Southeast Alaska. Signs of disease were severe. Some animals were rendered blind and/or deaf by the infection. Serologic test results reveal no evidence of CE exposure in goats from any region, including Southeast Alaska. Obviously, these results do not reflect the known occurrence of clinical disease. Perhaps the samples were collected during periods when herd immunity had declined between outbreaks. Alternatively, perhaps the virus was so highly pathogenic that all goats exposed subsequently died from the

infection. Under this scenario, only unexposed goats would remain in the population to be captured and sampled.

Antibody prevalence in sheep ranged from 15–30% for those areas where sample sizes were adequate. These values have remained stable since the 1992 report. We still believe that CE can represent a significant source of mortality for lambs in localized areas. However, these areas are usually quite limited in size. Adult sheep that are otherwise healthy may experience debilitating illness for a few days, but will typically recover.

Clinical CE has also been observed in captive muskox. Presumably, the disease also occurs in free-ranging muskox. The serologic results shown here support this contention. The low antibody prevalence indicates that exposure is uncommon.

There has been speculation regarding the susceptibility of both moose and caribou to CE. Experimental infections have proven that both host species are indeed susceptible (Appendix H). The low prevalence reported here indicates that natural exposure is limited. There have never been any documented cases of ecthyma in free-ranging moose or caribou in Alaska.

Serum antibody prevalence of contagious ecthyma virus in selected species of Alaska wildlife.

Species	Prevalence (%)
Deer	0/30 (0%)
Caribou	16/977 (2%)
Dall sheep	97/414 (23%)
Moose	8/584 (1%)
Mountain goat	0/66 (0%)
Muskox	4/120 (3%)

LEPTOSPIROSIS

I. AGENT – spirochete bacterium, several so-called "serovarieties" of *Leptospira interrogans*; each serovariety has its own name.

II. HOST(S) – All mammals are considered to be susceptible to infection with one or more serovarieties of *L. interrogans*.

III. SIGNS AND SYMPTOMS – There are few if any external signs of leptospirosis, and certainly none that could be considered peculiar to this disease. Infected animals may be lethargic, weak, and reluctant to move. In an advanced stage of disease, an animal may appear skinny and in generally poor body condition.

IV. TRANSMISSION – Infection commonly localizes in the kidney. Infected animals shed the leptospires in their urine. The shed organisms contaminate ground water sources and subsequently gain access into a susceptible animal through mucous membranes or broken skin. Carnivores may be exposed via ingestion of infected tissue.

V. EFFECT(S) – Infection of the kidney results in dysfunction. Other less common effects include hepatitis and abortion.

VI. CONFIRMATORY DIAGNOSIS – Serologic testing can provide an indication of the status of a population relative to leptospirosis exposure but is not very helpful when dealing with a single individual. The preferred method involves isolation, purification, and identification of the agent.

VII. PREVALENCE – Serologic evidence of leptospirosis has shown up in almost every species of wildlife included in our survey. However, antibody prevalences have been low for most potential host species. The most noteworthy exception is the mountain goat. The high prevalence for goats (15/58 = 26%) was heavily influenced by one year (1983) when 15/20 animals from Southeast had significant levels of antibody. Apparently, conditions favored transmission during that year and a large proportion of the goats were exposed. Antibody prevalence for both muskox and grizzly bears was in the range of 10%. For both species, prevalence was especially high in coastal areas. Bears from Kodiak and the northern Alaska Peninsula had a combined prevalence of 20% (32/164). Muskox from the Seward Peninsula and the Yukon-Kuskokwim Delta had a combined prevalence of 13% (8/61). Perhaps the coastal environment allows longer survival of shed leptospires, thus facilitating transmission. Highest prevalence in moose was on the Kenai Peninsula at 6% (38/597) and the Nelchina basin at 5% (9/175). The higher prevalence on the Kenai is further evidence of a coastal influence. Highest prevalence in caribou was in the Western Arctic Herd at 16% (18/110). Most of these results were from the 1970s. We have few samples from other species for this period to use as a comparison.

TOXOPLASMOSIS

I. AGENT – protozoan, *Toxoplasma gondii*

II. HOST(S) – Felids are considered the only FINAL hosts in which the organism can multiply and be spread. All mammals can serve as INTERMEDIATE hosts.

III. SIGNS AND SYMPTOMS – Infected animals may have an elevated body temperature and may exhibit signs of central nervous system dysfunction.

IV. TRANSMISSION – The parasite multiplies in the gastrointestinal tract of cats. An infectious stage (the "oocyst") is shed in feces. Other animals can become infected by ingesting food or water contaminated with oocysts. The parasite multiplies in the GI tract of these secondary hosts. The resulting developmental stages circulate via the blood and lymphatic systems. Tissue cysts form in various organs. Ingestion of these tissue cysts provides another means of transmission.

V. EFFECT(S) – Effects of toxoplasmosis on free-ranging animals are difficult to assess. The disease can cause abortion in domestic sheep and goats.

VI. CONFIRMATORY DIAGNOSIS – Several serologic procedures have been developed. The modified agglutination test (MAT) is currently considered to be the most reliable. Cysts can also be isolated and identified microscopically in preserved tissue.

VII. PREVALENCE – Lynx are the only free-ranging felid in most of Alaska. Therefore, they can be considered the sole definitive host for *T. gondii*. A serologic survey of 255 lynx from four areas of the Interior revealed that prevalence ranged from 6% (5/80) to 21% (13/61). Prevalence was higher in areas with an abundance of wetlands. Prevalence was directly related to age, ranging from nearly 0% in kittens to nearly 100% in animals older than 10 years. See Appendix I for more information.

Antibody prevalence for grizzly bears ranged from 9% (18/196) in southern areas of Alaska to 16% (40/258) in the Interior to 37% (162/433) in northern regions. I cannot offer an explanation for this geographic pattern. Prevalence was directly related to age. See Appendix J for more information.

Antibody prevalence was 43% (62/143) for black bears, 9% (11/125) for wolves, 7% (22/319) for Dall sheep, 6% (14/241) for caribou, 1% (3/240) for moose and 1% (2/241) for bison. See Appendix K for more information.

VIII. COMMENTS – Humans are susceptible to toxoplasmosis. Therefore, humans should cook game meat thoroughly.

TRICHINOSIS

I. AGENT – Nematode, *Trichinella nativa*

II. HOSTS – All mammals are susceptible; most common in carnivores

III. SIGNS AND SYMPTOMS – Infected animals often exhibit diarrhea during the intestinal phase of the infection.

IV. TRANSMISSION – Transmission occurs by means of ingestion of infected meat. Adult nematodes live and reproduce in the gastrointestinal tract. Larvae use the circulatory system to spread throughout the body, where they encyst in muscles. When muscle and cyst are ingested by another animal, the larvae are released in the GI tract where they mature and mate.

V. EFFECTS – Effects on free-ranging animals are difficult to determine. Presumably, animals experience periodic muscle aches during and after the larvae encyst in the muscle. Intensity of infection (number of cysts per gram of tissue) is highest in the most active muscles such as diaphragm, tongue and masseter. There is no information to suggest that trichinosis has any effect on population dynamics of free-ranging animals.

VI. CONFIRMATORY DIAGNOSIS – Common diagnostic methods include the following two:

(a) Compression method – a small portion of muscle tissue is compressed between two glass slides and examined under a microscope, and

(b) Digestion method – a portion of muscle tissue is digested by acid and enzymes. Larvae accumulate in the bottom of the container and can be counted.

In recent years, a serologic technique known as the enzyme-linked immunosorbent assay (ELISA) has been developed. It is highly reliable.

VII. PREVALENCE – Based on a serologic survey of grizzly/brown bears, prevalence ranged from 5% (10/196) in southern Alaska to 25% (62/252) in the Interior to 83% (355/430) in the northern portion of the state. Presumably, these major discrepancies were based on differing food habits of bears in the various regions. Prevalence was higher in older age cohorts. Prevalence was not affected by year of collection or sex of the bears. See Appendix L for more information.

Tongue samples from 1065 lynx were examined for presence of larvae. Prevalence was 21%. Prevalence ranged from 4% for kittens up to 59% for lynx 5 years or older. There were no patterns based on location or year-of-collection. See Appendix M for more information.

The wolf population in Unit 20A (south of Fairbanks) was surveyed by tissue digestion of tongue samples. Prevalence was 36% (54/148). Prevalence was directly related to age. See Appendix N for more information.

VIII. COMMENTS – Humans can be exposed via consumption of undercooked meat. Meat from any carnivore should be cooked thoroughly before consumption.

APPENDIX A SERUM ANTIBODY PREVALENCE OF PARAINFLUENZA 3 VIRUS IN A FREE-RANGING BISON (*BISON BISON*) HERD FROM ALASKA

RANDALL L ZARNKE AND GA ERICKSON

ABSTRACT: Serum antibody prevalence of parainfluenza 3 virus in the free-ranging Delta bison (*Bison bison*) herd which if found near Delta Junction, Alaska (USA), increased from 0% to 100% during the period 1977 to 1984. Domestic cattle are hypothesized as the source for the infection. There has been no clinical disease or decrease in productivity in this bison herd since establishment of the infection.

Journal of Wildlife Diseases, 1990, 26:416–419

APPENDIX B FURTHER OBSERVATIONS ON RANGIFERINE BRUCELLOSIS IN ALASKAN CARNIVORES

KENNETH A NEILAND

ABSTRACT: Antibodies against rangiferine brucellosis, *Brucella suis* type 4, are commonly found in the serum of various domestic and wild Alaskan carnivores which feed on caribou, *Rangifer tarandus granti*, in arctic Alaska. Sled dogs from five native villages on the range of the Arctic caribou herd, but not from two villages on the range of the Porcupine caribou herd, are commonly infected. Wolves (*Canis lupus*) and red foxes (*Vulpes fulva*) are less commonly infected.

About 90% of the grizzly bears (*Ursus arctos horribilis*) associated with the Arctic caribou herd and 30% of those associated with the Porcupine caribou herd show serologic signs of exposure to *Brucella*, presumably the enzootic strain present in Alaskan caribou. This is the first evidence of natural *Brucella* infection in bears.

It is concluded that infection of predators by enzootic strains of *Brucella* present in prey species (e.g., ruminants) is common to many areas of the world. Evidence from the literature and unpublished experimental data suggest that such infections may interfere with reproduction in wild species, but additional study is needed to clearly resolve this question.

Journal of Wildlife Diseases, 1975, 11:45–53

APPENDIX C SEROLOGIC SURVEY FOR CANINE CORONAVIRUS IN WOLVES FROM INTERIOR ALASKA, 1994–1999

RANDALL L ZARNKE, JIM EVERMANN, JAY M VER HOEF, MARK E MCNAY, RODNEY D BOERTJE, CRAIG L GARDNER, LAYNE G ADAMS, BRUCE W DALE, AND JOHN BURCH

ABSTRACT: Wolves (*Canis lupus*) were captured in three areas of Interior Alaska, USA. Four hundred twenty-five sera were tested for evidence of exposure to canine coronavirus by means of an indirect fluorescent antibody procedure. Serum antibody prevalence averaged 70% (167/240) during the spring collection period and 25% (46/185) during the autumn collection period. Prevalence was 0% (0/42) in the autumn pup cohort (age 4–5 months), and 60% (58/97) in the spring pup cohort (age 9–10 months). These results indicate that: (a) transmission occurs primarily during the winter months, (b) antibody decay is quite rapid and (c) re-exposure during the summer is rare.

Journal of Wildlife Diseases, submitted August 2000:In press

APPENDIX D SEROLOGIC SURVEY FOR SELECTED MICROBIAL PATHOGENS OF WOLVES IN ALASKA, 1975–1982

RANDALL L ZARNKE AND WARREN B BALLARD

ABSTRACT: Serum samples were collected from 116 wolves which were captured in southcentral Alaska during 1975 through 1982. Antibodies to the following infectious disease agents were found: infectious canine hepatitis virus—72 of 87 (81%), canine parvovirus type 2—0 of 55 (0%) through 1979 and 10 of 32 (31%) after 1979, *Francisella tularensis*—16 of 67 (25%), canine distemper virus—10 of 83 (12%), *Coxiella burnetti*—5 of 95 (5%), rabies virus—1 of 88 (1%), *Brucella* spp.—1 of 67 (1%), *Leptospira interrogans*—1 of 82 (1%). Apparently rabies, brucellosis, and leptospirosis were rare and had little effect on the wolf population. Conversely, the other five infections were comparatively common and may have had a negative impact on the health of specific individual wolves, but did not appear to influence the health of the population.

Journal of Wildlife Diseases, 1987, 23:77–85

APPENDIX E SEROLOGIC SURVEY FOR INFECTIOUS CANINE HEPATITIS VIRUS IN GRIZZLY BEARS (*URSUS ARCTOS*) FROM ALASKA, 1973 TO 1987

RANDALL L ZARNKE AND MARY BETH EVANS

ABSTRACT: Serum antibody prevalence of infectious canine hepatitis virus was 12% (90 of 725) for grizzly bears (*Ursus arctos*) from Alaska (USA) during the period 1973 to 1987. Prevalence was highest on Kodiak Island at 29% (37 of 127). Prevalence of exposure at individual collection areas did not change significantly over time. There were no significant sex-specific differences in prevalence. Prevalence was directly related to age, but it was 0% for bears <2-yr-old. Young bears which are exposed to the virus may develop clinical disease and die as a result of the infection. This disease may be a factor affecting grizzly bear population dynamics.

Journal of Wildlife Diseases, 1989, 25:568–573

APPENDIX F IMMUNITY AND LATENCY IN THE EPIZOOTIOLOGY OF CONTAGIOUS ECTHYMA

RANDALL L ZARNKE

ABSTRACT: In Alaska, there have been several recent outbreaks of contagious ecthyma (CE) involving both captive and free-ranging muskoxen (*Ovibos moschatus*) and Dall sheep (Dieterich et al. 1981. J. Am. Vet. Med. Assoc. 179:1140–1143; Zarnke et al. 1983. J. Wildl. Dis. 19:170–174). Serologic surveys indicate that both CE antibody titers in specific individuals and antibody prevalence in the population rapidly decline to undetectable levels. This is interpreted as indicating periodic epizootics with disease-free, inter-epizootic periods, rather than the more traditional epizootic and enzootic situation. During the inter-epizootic periods, low antibody titers in individuals would suggest that these animals are susceptible to re-infection. However, in spite of the supposed widespread presence of the virus in the environment, these individuals do not become re-infected until a large-scale epizootic occurs. Thus, they must be protected from re-infection by some means other than antibody-mediated immunity. The most logical candidate for this role is, of course, cell-mediated immunity. Jørgenson et al. (1984. *In* Klein et al., eds. Proc. First Int. Muskox Symp., Biol. Pap. Univ. Alaska Spec. Rep. No. 4 Abstr.) present the results of a laboratory-based investigation of this matter.

Another matter that has generated considerable discussion in recent years is the *source* of virus which precipitated epizootics of CE in free-ranging animals in areas of North America where no previous episodes of disease had occurred. Two commonly accepted explanations for the origin of such outbreaks are: (1) direct contact with infected domestic sheep or goats, or (2) grazing in areas where infected animals had shed virus in past years and the virus had remained infectious. Neither of these hypotheses seem appropriate to explain several of the more recent outbreaks, i.e., there was no evidence of contact with domestic animals nor any evidence of the disease ever having been present in the area. Several researchers have considered the possibility that latent CE infections may be responsible. This possibility has been addressed in at least 2 studies (Buddle 1981. Contagious ecthyma infection in sheep; virologic and immunologic investigations. Ph.D. Thesis, Virginia Polytechnic Inst. and State Univ., Blacksburg; Zarnke and Dieterich, unpubl data), with inconclusive results. Further investigation into the role of viral latency in the epizootiology of CE is necessary.

Biological Paper University of Alaska, 1984, Special Report 4:181

APPENDIX G ATTEMPTED REACTIVATION OF CONTAGIOUS ECTHYMA IN DALL SHEEP

RANDALL L ZARNKE, PHD, AND ROBERT A DIETERICH, DVM

SUMMARY: Dexamethasone was administered to 2 Dall ewes that had clinically recovered from contagious ecthyma in an attempt to reactivate contagious ecthyma in the sheep. Clinical signs of disease were not detected within 24 days after corticosteroid injection, and virus was not detected in tissues collected at necropsy.

American Journal of Veterinary Research, 1985, 46:1775–1776

APPENDIX H SEROLOGIC AND EXPERIMENTAL INVESTIGATIONS OF CONTAGIOUS ECTHYMA IN ALASKA

RANDALL L ZARNKE, ROBERT A DIETERICH, KENNETH A NEILAND, AND GEORGEANNE RANGLACK

ABSTRACT: Serologic evidence of contagious ecthyma (CE) was found in domestic sheep (*Ovis aries*), domestic goats (*Capra hircus*), Dall sheep (*Ovis dalli*), and muskox (*Ovibos moschatus*) in Alaska. A moose (*Alces alces*) calf and a caribou (*Rangifer tarandus*) fawn were susceptible to experimental infection and both developed antibody titers as a result. CE virus was isolated from lesions of Dall sheep which were involved in a natural outbreak of the disease.

Journal of Wildlife Diseases, 1983, 19:170–174

APPENDIX I SEROLOGIC SURVEY FOR *TOXOPLASMA GONDII* IN LYNX FROM INTERIOR ALASKA

RANDALL L ZARNKE, JP DUBEY, JM VER HOEF, ME MCNAY, AND OCH KWOK

ABSTRACT: Two hundred fifty-five lynx (*Felis lynx*) carcasses were collected from trappers in Interior Alaska (USA). Serosanguinous fluids were collected from the chest cavity of each carcass. These fluids were tested for evidence of exposure to *Toxoplasma gondii* by means of a modified agglutination test using formalin fixed tachyzoites and mercaptoethanol. Thirty-nine of the samples had titers greater than or equal to the threshold (≥25). Antibody prevalence differed between areas, and was directly related to age of the host.

Journal of Wildlife Diseases, 2001, 37:In press

APPENDIX J SEROLOGIC SURVEY FOR *TOXOPLASMA GONDII* IN GRIZZLY BEARS FROM ALASKA

RANDALL L ZARNKE, JP DUBEY, OCH KWOK, AND JAY M VER HOEF

ABSTRACT: Blood samples were collected from 892 grizzly bears (*Ursus arctos*) in Alaska (USA) from 1973 to 1987. Sera were tested for evidence of exposure to *Toxoplasma gondii* by means of the modified agglutination test. Two hundred twenty sera (25%) had titers ≥25, the minimum threshold titer. Six hundred seventy-two sera (75%) had titers <25. Antibody prevalence ranged from 9% (18 positive of 196 tested) in southern areas to 37% (162 of 433 tested) in northern areas. There was no readily apparent explanation for these discrepancies in location-specific prevalence.

Journal of Wildlife Diseases, 1997, 33:267–270

APPENDIX K SEROLOGIC SURVEY FOR *TOXOPLASMA GONDII* IN SELECTED WILDLIFE SPECIES FROM ALASKA

RANDALL L ZARNKE, JP DUBEY, OCH KWOK, AND JAY M VER HOEF

ABSTRACT: Blood was collected from selected wildlife species in specific areas of Alaska (USA) during 1976–96. A modified agglutination test was used to test sera for evidence of exposure to *Toxoplasma gondii*. Serum antibody prevalence was 43% (62 positive of 143 tested) for black bears (*Ursus americanus*), 9% (11/125) for wolves (*Canis lupus*), 7% (22/319) for Dall sheep (*Ovis dalli*), 6% (14/241) for caribou (*Rangifer tarandus*), 1% (3/240) for moose (*Alces alces*), and 1% (2/241) for bison (*Bison bison*). A predictive model was developed to determine the effect of sex, age, location, and year of collection on antibody prevalence for each species. Prevalence was higher in older black bears, caribou, and wolves. For black bears, prevalence was highest in the southeast region of the state. For caribou, prevalence was lowest on the Alaska Peninsula.

Journal of Wildlife Diseases, 2000, 36:219–224

APPENDIX L SEROLOGIC SURVEY FOR *TRICHINELLA* SPP. IN GRIZZLY BEARS (*URSUS ARCTOS*) FROM ALASKA, 1973 TO 1987

RANDALL L ZARNKE, RAY GAMBLE, ROBERT A HECKERT, AND JAY VER HOEF

ABSTRACT: Blood was collected from 878 grizzly bears (*Ursus arctos*) in seven geographic areas of Alaska from 1973 to 1987. An enzyme-linked immunosorbent assay procedure was used to test sera for evidence of exposure to *Trichinella* spp. Serum antibody prevalence ranged from 5% (10 positive of 196 tested) in the Southern Region of the state to 83% (355/430) in the Northern Region. These major discrepancies may be a result of differing food habits of bears in the major geographic areas. Prevalence was higher in older age cohorts. Neither year-of-collection nor sex had a significant effect on prevalence.

Journal of Wildlife Diseases, 1997, 33:474–479

APPENDIX M PREVALENCE OF *TRICHINELLA NATIVA* IN LYNX (*FELIS LYNX*) FROM ALASKA, 1988–1993

RANDALL L ZARNKE, ALVIN A GAJADHAR, GREGORY B TIFFIN, AND JAY M VER HOEF

ABSTRACT: Lynx (*Felis lynx*) carcasses were collected during the 1989 to 1990 through 1992 to 1993 trapping seasons in Alaska (USA). Seven areas were represented. Tongue samples were removed from 1,065 carcasses. Specimens were examined for the presence of *Trichinella nativa* larvae by means of enzymatic digestion. Overall prevalence was 21%. Both prevalence and number of larvae per gram of host tissue were directly related to age of the host. Age-specific prevalence ranged from 4% for kittens up to 59% for lynx 5 yr of age and older. For infected lynx, intensity ranged from 0.27 larvae per gram of host tissue for kittens up to 2.35 larvae per gram for lynx 3 yr of age and older. Location-specific prevalence ranged from 19% to 27%. Year-specific prevalence ranged from 13% to 26%. Prevalence in both males and females was 21%.

Journal of Wildlife Diseases, 1995, 31:314–318

APPENDIX N *TRICHINELLA NATIVA* IN WOLVES FROM INTERIOR ALASKA

RANDALL L ZARNKE, DAVID E WORLEY, JAY M VER HOEF, AND MARK E MCNAY

ABSTRACT: Tongue samples were collected from 148 wolf (*Canis lupus*) carcasses during 1993 and 1994 near Fairbanks (Alaska, USA). A standard peptic digestion procedure was used to detect *Trichinella* spp. larvae. Larvae were found in 54 of 148 (36%) samples. There was no significant difference in sex-specific prevalence. Prevalence was significantly related to age. There was no relationship between the number of larvae/gm of host tissue and the age or sex of the host. *Trichinella* spp. infection may cause illness in individual wolves. However, there was no indication the parasite had any negative impact on the population.

Journal of Wildlife Diseases, 1999, 35:94–97

The Federal Aid in Wildlife Restoration Program consists of funds from a 10% to 11% manufacturer's excise tax collected from the sales of handguns, sporting rifles, shotguns, ammunition, and archery equipment. The Federal Aid program allots funds back to states through a formula based on each state's geographic area and number of paid hunting license holders. Alaska receives a maximum 5% of revenues collected each year. The Alaska Department of Fish and Game uses federal aid funds to help restore, conserve, and manage wild birds and mammals to benefit the public. These funds are also used to educate hunters to develop the skills, knowledge, and attitudes for responsible hunting. Seventy-five percent of the funds for this report are from Federal Aid.