Alaska Department of Fish and Game
Division of Wildlife Conservation

Alaska Wildlife Serologic Survey, 1975–1992

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INTRODUCTION AND BACKGROUND

I have been coordinating the collection and testing of wildlife sera in Alaska for over a decade. Since the survey began, I have sent memos to participating individuals and filed annual reports covering new data. Recently, a supervisor suggested that I prepare a comprehensive summary of this project (written in layman's terms) with emphasis on geographic patterns of disease. This summary could then be distributed to individuals who had contributed sera or were otherwise interested in patterns of exposure in Alaska.

The primary purpose of this document is the presentation of data derived from the serologic survey of terrestrial animals during the past 10-15 years. [We have data on marine mammals and birds, but these will not be addressed. Feel free to contact me if you would like to learn more about disease patterns in these other species.] Background information is provided for each disease agent in an effort to make this document more useful. Hopefully, it will be the kind of summary that you will want to keep for future reference. Additional copies are available upon request.

We have over 10,000 samples representing more than 30 species in our Alaska Wildlife Serum Bank. More than 50,000 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 expand.

Sera are stored in freezers at minus 40-50 C. At these temperatures, specimens should retain biochemical properties almost forever. This long-term storage aspect becomes increasingly valuable as new analytical techniques are developed and new diseases are discovered. For example, new 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 of when they are available to me.

2. Individuals who submit sera will continue to have access to those samples. In other words, they do not become "my" property once they are placed in the freezer. Sera can be shipped to the original collector or to a third party upon request.

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

There are several instances in this document where I have reported an apparent anomaly in the data without presenting an accompanying explanation. The reason that I have not provided explanations is that there are none 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, style, etc., 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. The data are 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.

An important related issue is the question of how many samples are necessary to adequately assess the frequency with which a disease occurs 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.

Presence of antibody to a particular disease agent in an animal's serum 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. 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.

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 we currently enjoy. 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 field work. Please encourage other wildlife investigators to contribute, as well. Increased participation results in increased knowledge of the health status of our wildlife populations statewide. We are entering a new era with potential game ranching and grazing of domestic livestock in areas that had previously been strictly wildlife habitat. These activities hold the potential for the spread of diseases to wildlife. Serologic surveys can provide invaluable information for protecting wildlife and minimizing negative impacts.

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 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.

VII. CONFIRMATIVE DIAGNOSIS - Serology 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 accompanying table presents data for several major caribou herds and also for bears and wolves from areas where we have enough samples to make the data meaningful. Obviously, antibody prevalence in the predators is dependent on prevalence in the prey. Considering that this disease can be transmitted orally, this observation should come as no surprise. Equally obvious is the fact that prevalence is much higher in the Arctic than elsewhere in the state. At present, we have no ready explanation for this discrepancy. Data such as these should be considered (1) when advising hunters about the possibility of human exposure, and (2) when contemplating relocation of animals from one part of the state to another.

Brucellosis (continued)

Caribou		Grizzly		Wolf	
Porcupine	9/240 (4%)	Eastern Arctic	45/296 (15%)	Eastern Arctic	1/38 (3%)
Western Arctic	16/66 (24%)	Western Arctic	56/173 (32%)		
		Noatak	21/59 (36%)		
		Seward Peninsula	10/46 (22%)		
Central Arctic	2/83 (2%)			Central Arctic	3/19 (16%)
Teshekpuk	2/21 (10%)				
Delta	0/77 (0%)	GMU 20A	6/144 (4%)	GMU 20	1/95 (1%)
Nelchina	1/84 (1%)	GMU 13	10/109 (9%)	GMU 13	1/67 (1%)
All Others	1/752				
		Kodiak	13/189 (7%)		
				Kenai	0/12

VIII. COMMENTS - Humans are susceptible to brucellosis. When humans are infected with the cattle strain (*B. abortus*), the resulting disease is sometimes referred to as "undulant fever." Signs of disease in humans include: alternate fever and chills, headaches, muscle aches and fatigue. Symptoms may recur periodically after lapses of several months. Prior to the easy availability of antibiotics, rural residents in Alaska presumably suffered extensive illness as a result of exposure to infected caribou. Current butchering and eating practices, in conjunction with medical treatment, have drastically reduced the impact of the disease on humans.

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/or 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 any confidence regarding the exposure status of any 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 - Hundreds of sera from Alaskan wildlife species have been tested for evidence of exposure to these four viruses (see table). There is some evidence of exposure in sheep, moose, and muskox, but it is rare. Dramatic divergence from the pattern of low prevalence of these agents is the case of PI3 in the Delta bison herd. Bison sera from the early to mid-1970s showed no evidence of PI3 exposure. The first serologic evidence of exposure was seen in 1977. By 1983, prevalence had reached 100% and has remained at or near that level since. Such a pattern is typical following the introduction of a new agent into a susceptible population. Domestic livestock are believed to be responsible for introduction of PI3 into the herd. There have been no outward signs of disease.

Bovine Respiratory Group Viruses (continued)

	IBR	BVD	PI3	RSV
Sheep	0/326	2/315	2/321	0/58
Moose	0/830	4/806	11/878	0/100
Muskox	0/131	4/129	4/130	0/59
Bison	1/327	5/275	240/374	0/137

For caribou, an interesting pattern has been revealed. Serum antibody prevalence of all four agents is much higher in northern herds (Western Arctic, Teshekpuk, Central Arctic, Porcupine, and to some extent Fortymile) than for herds from the remainder of the state (see below). At present, we have no ready explanation for this discrepancy. We have seen a few cases of severe pneumonia in caribou from the North Slope. However, the number of such cases is not large enough to draw any solid conclusions regarding a causal relationship between antibody prevalence and actual cases of disease. There are other diseases (brucellosis, for example) which seem to fit this pattern of higher serum antibody prevalence in the Arctic as compared with elsewhere in the state. Again, at this time there is no widely accepted explanation for these apparent patterns.

	IBR	BVD	PI3	RSV
Caribou(North)	68/640 (11%)	54/638 (8%)	60/627 (10%)	4/408 (1%)
Caribou(South)	4/532 (<1%)	2/535 (<1%)	1/499 (<1%)	0/475 (0%)

VIII. COMMENTS -

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.

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 it 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 as compared with adults.

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 - Episodes of readily identifiable disease occur sporadically in mountain goats in southeastern Alaska. Dramatic outbreaks of severe cases have been observed during 1989 and 1990. Serologic test results confirm that goats in the region have been exposed to the virus. Sample sizes have not been large enough or frequent enough to provide a complete understanding of patterns of occurrence.

Based on serologic tests, exposure of Dall sheep in the Alaska Range is common. Prevalences range from 15% to 30% (see table). There have been no significant changes in prevalence in these populations during the past 15 years. Small collections from other parts of the state indicate 10% prevalence in sheep on the Kenai Peninsula and no evidence of exposure from Atigun Pass in the Brooks Range.

Alaska Range:		
Dry Creek	65/216	(30%)
Sheep Creek	19/92	(21%)
Granite Creek	5/32	(16%)
Kenai Peninsula	2/20	(10%)
Atigun Pass	0/15	(0%)

Contagious Ecthyma (continued)

VIII. COMMENTS - Humans are susceptible to infection. Lesions are usually small and few in number. Minor scars may remain after lesions heal. Hunters would be well-advised to wear rubber gloves when skinning a sheep or goat with obvious lesions.

BLUETONGUE AND EPIZOOTIC HEMORRHAGIC DISEASE

I. AGENT(S) - viruses; closely related 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 even more dramatic. Massive hemorrhaging may occur in any of 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 a 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 suggest 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 suggestive of these hemorrhagic diseases. Additional 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.

Bluetongue and Epizootic Hemorrhagic Disease (continued)

VII. PREVALENCE - Serologic evidence of exposure to either of these diseases is rare in Alaska (see table). In fact, evidence is so sporadic that it might seem unworthy of inclusion in this compilation. However, discussions with personnel at the laboratory where the serologic tests were performed revealed a couple of interesting points: (1) sera were often tested more than once to confirm initial results and the lab personnel now feel confident that most of the results do indeed represent evidence of exposure, (2) lab personnel suspect that what we are detecting is not actually either conventional bluetongue or EHD, but rather an unknown related virus, and (3) they would be very interested in investigating if we ever find an actual case of disease.

Species	Prevalence	Percent
Caribou	2/518	0.4%
Moose	20/987	2.0%
Bison	2/362	0.6%
Deer	0/74	0.0%
Dall sheep	0/273	0.0%
Muskox	0/101	0.0%

VIII. COMMENTS -

TULAREMIA

I. AGENT - bacterium; *Francisella tularensis*

II. HOST(S) - There are two ecologically distinct cycles of this disease: (1) terrestrial and (2) aquatic. Snowshoe hares are the primary host for the terrestrial cycle. There have been numerous terrestrial outbreaks in the Interior during the past 12 years. Predators such as foxes, lynx, and domestic pets become involved in the cycle when they kill and consume an infected hare. Beaver and muskrat are the two species most commonly associated with the aquatic cycle. Any animal that consumes water from a water system (pond, river, slough) which has been contaminated by an infected animal may subsequently become infected. The remainder of this discussion will focus on the terrestrial cycle.

III. SIGNS AND SYMPTOMS - In the latter stages of disease, hares become sluggish and are easily captured by either domestic or wild predators. The most reliable internal sign of disease is an enlarged spleen. The next most reliable sign is an enlarged liver. White spots may be present on either of these organs, but not in all cases. Infected pets become sluggish and may have an elevated body temperature and loss of appetite. Signs of the disease in humans resemble a severe case of the flu--nausea, body aches, alternate chills and fever, headache, etc.

IV. TRANSMISSION - Transmission between hares occurs primarily by means of ticks (*Hemaphysalis leporis palustris*). These ticks are extremely species-specific and therefore are rarely found on any species other than hares. Ticks are only found on hares during the warmer months (late April through mid-September). Therefore, hare-to-hare transmission is largely restricted to this time period as well. Transmission from hares to predators occurs when the predator kills and consumes an infected hare. Transmission from hares to humans occurs during the gutting process. Signs such as an enlarged spleen and liver indicate that infection is localized in these organs. When a human reaches inside the gut cavity to remove the internal organs, he is essentially inserting his hand into a bacterial culture. The bacterium gains access to the human through any cuts or scratches that may be present on the hand. Muscle tissue in hares is not infected and is therefore safe to eat. Thorough cooking kills the bacteria.

V. EFFECT(S) - The disease is often fatal in hares. Large numbers of dead hares can be found in areas ranging in size from 5 to 100 acres each summer. Serologic surveys in the Interior indicate low levels of previous exposure, although we know that the disease is common. Thus, those hares that are exposed either (1) do not maintain a detectable level of antibody, or (2) die as a result of the disease and are not around to "donate" blood when live hares are captured. Dogs and cats occasionally succumb to the disease. Most pet owners seek veterinary care for pets exhibiting signs such as those described above. Treatment is relatively straight-forward once the disease is diagnosed. It is rarely fatal in humans and is easily treated with antibiotics.

Tularemia (continued)

VI. CONFIRMATORY DIAGNOSIS - Demonstration of antibody in serum (i.e., serologic testing) is one means of diagnosing this disease in survivors. Isolation, purification, and identification of the bacterium is more reliable.

VII. PREVALENCE - As indicated above, serologic surveys of hares suggest that tularemia is rare in Interior hares. However, we know that the disease is present every year. Thus, serologic surveys do not provide an accurate picture of the occurrence of this disease. To complicate matters further, the disease does not normally occur in the same location every year. So-called "hot spots" crop up in different locations each year. The proportion of hares killed at each such site can be high.

VIII. COMMENTS - Pets are susceptible to tularemia and should not be allowed to run free without human supervision. Humans should wear rubber gloves when butchering hares taken during warm weather months.

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/or 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 - Based on serologic test results, prevalence of infectious canine hepatitis (ICH) is in the 80-100% range for wolves from the Nelchina Basin, Tanana Flats, and northwest Arctic. Serum antibody prevalence in a small collection of arctic foxes from the vicinity of Prudhoe Bay was also very high. Prevalence was 2% in black bears from the Nelchina Basin and the Tanana Flats. Prevalence in grizzly/brown bears was 0% on Admiralty Island, 5-10% from the Interior and North Slope, and 26% from Kodiak Island and the Alaska Peninsula.

The absence of exposure of brown bears on Admiralty Island is attributed to the absence of wild canids that could serve as reservoirs of infection for bears. There are no readily available explanations for the other discrepancies in prevalence.

Examination of data for a period of 10-15 years indicates that prevalence in grizzly/brown bears has not increased. This suggests that exposure of bears to ICH is not a new phenomenon, but rather has occurred for a long period of time.

VIII. COMMENTS - Preventive vaccination has virtually eliminated this disease from domestic dogs. Many veterinarians indicate that they have never seen a case of the disease. However, the disease still poses a threat to free-ranging canids.

Prior to the mid-1970s, the susceptibility of bears to ICH was virtually unknown. Since that time there have been two fatal episodes of disease in captive black bears in the Lower 48.

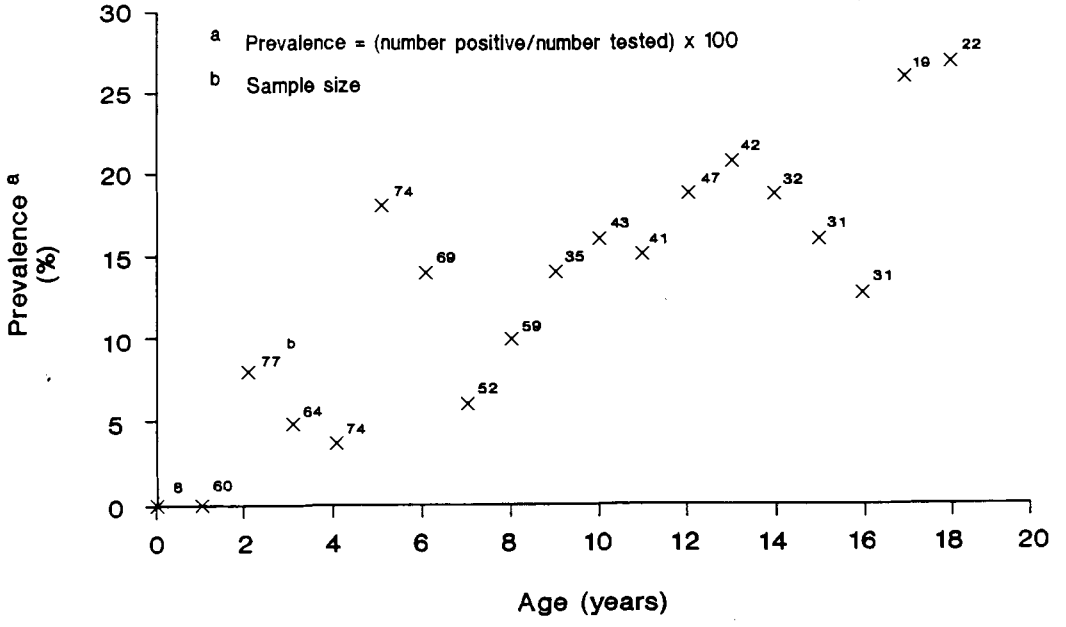
In our survey of grizzly/brown bears from Alaska, there was no serologic evidence of exposure to ICH in bears <2 years old (see figure below). Possible explanations include:

1. young bears are not exposed to the virus,
2. maternal antibody is transferred either *in utero* or in colostrum and subsequently interferes with antibody production by the young bears,
3. young bears are incapable of producing antibody,
4. antibody titers only reach detectable levels following repeated exposures over a period of at least 24 months, or
5. young bears that are exposed to ICH develop clinical disease and die as a result of the infection.

There is no evidence to support hypotheses 1-4. In fact, there are reasons to reject each of these hypotheses. There is indirect support for the fifth hypothesis:

1. clinical ICH infection is more severe in young canids, as compared with adults,
2. in captive situations, the virus is capable of killing black bear cubs, and
3. brown bear cubs at the Budapest Zoo died as a result of ICH whereas all adult bears at the Zoo survived.

This hypothesis that grizzly/brown bear cubs die as a result of exposure to ICH has not been adequately tested. We would like to conduct experiments to confirm or refute this hypothesis. In the past, it has been difficult to obtain cubs for these experiments. Please contact me if you have suggestions on obtaining animals for this purpose.



Age-specific serum antibody prevalence for infectious canine hepatitis virus in grizzly bears (*Ursus arctos*) from 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 - There have been few cases of distemper in free-ranging wildlife species in Alaska that were confirmed by means of virus isolation. Serologic tests indicate that approximately 10% of the wolves from the Nelchina Basin, Tanana Flats, and northwest Arctic have been exposed. There was no serologic evidence of exposure in a small collection of arctic foxes from the vicinity of Prudhoe Bay.

VIII. COMMENTS -

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; 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 - Serology 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 - Almost every species that has been tested has revealed some serologic evidence of exposure to one or more serovarieties of *L. interrogans* (see table). Prevalences for mountain goat, muskoxen, and deer may be artifically inflated as a result of relatively small sample sizes. Most of the grizzly/brown sera with evidence of exposure were from bears collected on Kodiak Island and near Becharof Lake. Similarly, moose sera that gave test results indicative of prior exposure were collected from the Kenai Peninsula and the Nelchina Basin. Caribou sera with similar evidence came from the Fortymile, Macomb, Delta, Denali, and northern Alaska Peninsula herds. Thus, unlike several other diseases, exposure to *L. interrogans* appears to be more common in the southern part of the state.

Mountain goat	15/38	(39%)
Muskox	5/43	(12%)
Grizzly bear	42/494	(8%)
Bison	14/229	(6%)
Deer	2/33	(6%)
Moose	48/1,020	(5%)
Caribou	15/902	(2%)
Wolf	1/210	(<1%)
Sheep	0/194	(0%)

VIII. COMMENTS - Leptospires have been isolated from a few wildlife species in Alaska. However, there have been few cases of overt disease. Leptospirosis is not thought to be a serious threat to the health of wildlife in the state.

Q FEVER

I. AGENT - rickettsium; *Coxiella burnetti*. In very simple terms, a rickettsium is an intermediate organism between a virus and a bacterium.

II. HOST(S) - Essentially any mammal can be considered to be susceptible to this disease. The primary domestic animal hosts are cattle and sheep. The wildlife species that has received the greatest attention in Alaska is the caribou.

III. SIGNS AND SYMPTOMS - In domestic species, infection is often undetectable based upon external signs of disease. When infection of pregnant females becomes severe, abortion may result. Presumably, most infections of wildlife are asymptomatic.

IV. TRANSMISSION - The organism is shed in aborted tissues and fluids. Susceptible animals are presumably exposed by means of direct contact and/or inhalation of aerosolized droplets. Ticks and other arthropods are known to serve as vectors in other parts of the world. Predation also provides a likely means of transmission.

V. EFFECT(S) - As mentioned above, detectable signs of disease are uncommon. If abortion was a widespread phenomenon, obviously productivity of a population would be affected.

VI. CONFIRMATORY DIAGNOSIS - Serologic tests provide an indication of the exposure status of a population, but are less helpful for individuals. Isolation of the organism from blood or sputum is preferred for confirmation.

VII. PREVALENCE - The only previous serologic survey data indicated that prevalence was 10% for the Delta caribou herd during the late 1960s. More current information for a variety of species is presented in the accompanying table. The high antibody prevalence in mountain goats was a surprise. In the absence of other evidence of decreased productivity of goat populations, these serologic data are not considered to be cause for alarm. Serologic evidence of exposure for other species was not more common in any specific area of the state as compared with other areas.

Mountain goat	13/60	(22%)
Dall sheep	21/345	(6%)
Wolf	15/258	(6%)
Caribou	15/501	(3%)
Muskox	2/69	(3%)
Grizzly bear	7/683	(1%)
Bison	1/300	(<1%)
Moose	0/186	(0%)

VIII. COMMENTS - Q fever is not considered to be a major health problem for wildlife in Alaska.