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CARIBOU DISEASE STUDIES

by
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Final Report
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FINAL REPORT (RESEARCH)

State: Alaska

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SUMMARY

Alaskan caribou are host to a variety of parasites many of which are known to cause serious problems in *Rangifer* or other host species elsewhere. In addition there no doubt are additional parasitic species, particularly roundworms, yet to be discovered. While the currently available data when fully analyzed will probably give accurate values for the prevalence and intensities of infections in some age classes in some herds at some times, many essentially blank spots remain. Since it is clear that such infections may vary from year to year and are seldom, if ever, uniformly distributed geographically, there are no quick, easy answers, good for all times, to the many questions involving the chronic and/or acute effects of parasitism on caribou populations, whether in Alaska or elsewhere. Unfortunately many, if not most, studies on big game populations have given only very cursory or uneven attention to matters involving diseases and parasites and their chronic affects on the dynamics of wild populations. All too often the relative involvement of pathogens in population crashes or declines are examined retrospectively, a procedure which really can only reveal the circumstances under which an animal survives. More useful information on diseases, etc. of *Rangifer* may be available than for other big game ruminants because of the intensive husbandry of this species in Eurasia. Unfortunately, many apparently noteworthy reports are only available in obscure periodicals published in difficult languages (e.g. Russian, Finnish, etc.). We have hardly "scratched the surface" in discovering the epizootiological facts of life of North American species of *Rangifer*.

Rangiferine brucellosis is enzootic in at least two of our major caribou herds. At the present time it is estimated that about 10 per cent of a random sample of animals from the Arctic herd are or have been recently infected and would therefore be serologic reactors. We similarly estimate that only 1 per cent of the Nelchina herd are test reactors. Placental retention, which is associated with early *post partum* loss of fawns by affected does, remains more or less common on the Arctic calving grounds. Fifty or more per cent of the fawns born to does which experience this problem do not survive more than a day or so after birth even under ideal weather conditions. A retained placenta has only been seen once

in another Alaskan caribou herd but adequate surveys seldom have been made. The data presently available do not support the hypothesis that rangiferine brucellosis infections are solely responsible for placental retention. It appears that the condition may have several causes.

Wild or domestic carnivores that prey on caribou are readily susceptible to rangiferine brucellosis and we have serologically demonstrated infections in sled dogs, wolves, red foxes and grizzly bears. Relatively little is known about the consequences of infection by this strain of *Brucella* in carnivores. However, closely related strains commonly produce abortion and sterility. Experimental studies are sorely needed in order to evaluate the overall effects of enzootic rangiferine brucellosis on the wildlife in areas of infection. Whether or not rodents are involved as alternate (reservoir?) hosts has yet to be determined.

Necrobacillosis, particularly in the form of foot rot, is a chronic disease problem of populations of *Rangifer* spp. everywhere. Various factors, including severity of harassment by warble and bot flies and other dipteran pests, affect the yearly prevalence of the condition. Evidently even animals in otherwise good condition can become infected with serious (death?) results.

We have recently obtained the first evidence that leptospirosis may be enzootic on one Alaskan caribou range. Indigenous rodents, most likely microtids, are probably the reservoir of infection. Leptospiral infections can lead to abortion in cattle and swine, but experimental infections in sheep were followed by normal lambing. Further study is required to evaluate the importance of this disease in caribou.

Serologic evidence of one or more strains or arboviruses (particularly California encephalitis virus) in caribou and other Alaskan wildlife has recently come to hand.

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BACKGROUND

Caribou are highly important for sporting and subsistence purposes in Alaska. Individual segments of major populations may at times occupy the same range as that used by commercial reindeer herds. Thus, disease conditions (including pathogenic infestations of parasites) in caribou which can be transmitted to man or domestic animals or which are significantly harmful to the caribou are of obvious importance. The caribou in some areas is plagued by more potentially serious parasites or disease conditions than most of the other Alaskan wildlife species. Brucellosis, foot rot, warble and bot flies and gastro-intestinal roundworms, are all more or less common in all North American caribou herds and either directly cause or contribute to serious disease conditions in caribou, reindeer, man and/or his animals. Brucellosis is a particularly significant zoonotic disease of Alaskan caribou whose prevalence is documented (Neiland et al. 1968) though not well understood.

The present study, a continuation of one in progress since 1962, was primarily concerned with fully documenting the natural history (i.e. epidemiology) and pathology of rangiferine brucellosis. It seems likely that it may be cyclic with an as yet unknown periodicity. We cannot yet be sure that all of the pathological conditions (e.g. placental retention) which we suspect to be caused by the disease only involve this pathogen. We do not know whether the disease will essentially "die-out" in caribou herds, only to be reintroduced from some non-rangiferine reservoir host species in which it may occur, perhaps in "quiet" form or whether reindeer and/or caribou serve as both reservoir and secondary hosts. While these and other questions are of great scientific interest, they also point the way toward "practical" management goals. If we find the

"rangiferine" brucellosis is indeed a disease of *Rangifer* spp. and does not necessarily involve a regular reservoir host system, then we will also likely find that whereas the disease continues at a low endemic level in "close herded" reindeer, it will likely disappear in wide-ranging caribou. A recurrence of epidemic levels of the disease in caribou could be expected to come about again whenever substantial contact between caribou and infected reindeer occurred, particularly after prolonged absence of the disease from caribou. If reindeer do play the role of reservoir for the disease, the management solution is to get them off known caribou range. If a non-rangiferine reservoir may also be involved, as is the case with porcine brucellosis (wild rabbits, Europe) or bovine brucellosis (wild foxes, Argentina), then effective control is more difficult. Because of the known involvement of foxes in Argentina, the proven involvement of dogs on occasion in human brucellosis and our scant data on Eskimo sled dogs, one cannot help but wonder whether wild or semi-domestic canines are possible reservoirs of Alaskan rangiferine brucellosis.

OBJECTIVES

To determine the incidence and distribution of potential pathogens in Alaskan caribou (*Rangifer tarandus*) and alternate or reservoir hosts.

To determine whenever possible or practical the extent that such organisms may contribute to mortality, lowered productivity or economic value of affected caribou populations.

To determine the extent that wildlife pathogens depreciate the value of caribou for use as food by humans or may be a threat to domestic animal industry.

PROCEDURES

Our primary effort in rangiferine disease studies is focused on the long-term study of brucellosis in caribou. In this respect we are continuing our close cooperation with the Animal Disease Eradication Division, U. S. Department of Agriculture, who is monitoring the disease in reindeer. In these studies the following specific procedures are emphasized.

1. Serological surveillance of brucellosis prevalence in major caribou herds, particularly those in the Nelchina and Arctic areas.

2. Confirmation by isolation of suspected brucellar infections.

3. Serological studies on potential reservoir host species.

4. Aerial surveillance of the occurrence of animals displaying gross symptoms (i.e. limping, retention of afterbirth) of brucellosis during calving.

5. Surveillance from the ground of concentrations of animals during the spring and fall migrations through Anaktuvuk Pass in the Arctic to detect and collect specific animals for bacteriological and/or other studies.

6. Routine autopsies of animals taken for subsistence purposes by native or sport hunters or specifically for the purposes of various scientific studies (e.g. radiation studies, disease and parasite studies, etc.).

7. Examination of specimens submitted to our laboratory by the public.

8. Preparation of a definitive bibliography on the "Diseases, Parasites, and Disorders of Caribou and Reindeer."

9. Publication of data at suitable intervals.

FINDINGS

In addition to specifically reporting observations made during the period of this report, January 1, 1970 to June 30, 1971, a general analysis of the information obtained since caribou disease and parasites studies were first initiated in April, 1961, is also presented.

From April, 1961 to date, approximately 800 caribou have been available to employees of the Game Division for necropsy. The majority of these have been necropsied by myself, but animals collected and necropsied by others primarily for purposes other than disease and parasite studies may number several hundred. All of these animals have yielded information utilized by caribou biologists for studies on the natural history and population dynamics of this species. Many caribou were collected by various personnel for radionuclide studies by federal agencies. Those examined through early 1964, about one-half of the total, provided the basis for a Ph.D. Thesis (Univ. Calif. Berkeley, 1968) by Dr. R. O. Skoog and are reported in all respects in more of less detail in that place.

Most of the approximately 500 animals that came to hand from the Arctic herd were taken in conjunction with subsistence hunting by residents of Anaktuvuk Pass from 1961 to 1971. Nearly all of the approximately 200 animals from the Nelchina herd were strictly scientific collections, as were the 100 or so taken from the herd on the Alaska Peninsula. Although the number of animals which have been examined may seem large, it is well to bear in mind that the more heavily staffed and financed study of the Manitoba-Keewatin caribou herd by personnel of the Canadian Wildlife Service involved a full 1,000 animals. These were all scientific collections from a herd of about 350,000 animals (not a great deal larger than our Arctic herd, i.e. 250,000), and the number taken was considered to be the minimum required to adequately document the biological characteristics of this herd during the period of study.

Regardless of the inadequacy of our own data on these three important Alaskan herds, we have a very large number of individual items of data to analyze (about 30 per animal or 20,000 or so in total). An analysis of this magnitude requires a computerized procedure. The design of a suitable data sheet is under way. Even though we cannot present a full analysis of the data at this time, it seems worthwhile to summarize our findings.

I. Parasites

A. Protozoa

1. *Besnoitia tarandi* (Hadwen, 1922)

The causative agent of so-called "cornmeal" disease in Alaskan reindeer was described as a new organism by Hadwen (1922) who gave it the name *Fibrocystis tarandi*. More recently Levine (1961) removed the species to the genus *Besnoitia* Henry, 1913, which includes several other species of proven pathogenicity in domestic animals. Gibbs (1960a) reported *B. tarandi* (as *Fibrocystis* spp.) in Canadian barren-ground caribou. Choquette et al. (1967) also reported the occurrence of the organism in Canadian caribou in which in two instances it evidently caused a dermatitis. We have seen more or less commonly the periosteal and fascial cysts of *B. tarandi* in animals from each of the Alaskan herds under consideration. However, we have not seen any cases of dermatitis ascribable to this parasite. There appears to be no clear-cut evidence that this species of *Besnoitia* is a significant pathogen, although in severe cases of dermatitis it might achieve this status. Klimontov (1966) describes "fibrocystosis" of the nervous system in reindeer. I assume that this author is using Hadwen's outdated name for *B. tarandi*. Infection of the nervous system would likely be a more serious matter.

Jellison (personal communication) failed to infect rabbits with fresh material I sent to him, even though *B. besnoiti* (Marotel, 1912), a serious pathogen in cattle, will infect lagomorphs. The importance of besnoitiosis in *Rangifer* remains to be determined.

2. *Sarcocystis* sp.

This parasite has been known in Eurasian *Rangifer* spp. since it was first reported by Gruiner (1927) and subsequently named *Sarcocystis gruneri* by Yakimov and Sokolov (1934). Several other Russian publications in more recent years also refer to this parasite, but none of these reports are currently available to me. The parasite was evidently first reported in North American *Rangifer* spp. by Gibbs (1960a).

I have seen this minute parasite on several occasions in Alaskan caribou, but because of the small size of the cysts, i.e. about 2mm x 0.5mm, I assume that it has been overlooked more often than not.

The various species of *Sarcocystis* generally are not considered to be significantly pathogenic in the usually light to moderate infections

in which they are seen (Levine, 1961). However, since the parasite does destroy the muscle cell in which it occurs, heavy infections could be debilitating.

3. Other Species

No other protozoan parasites are known from *Rangifer* spp. in North America. However, piroplasms and coccidia are reported from Eurasian reindeer in the Russian literature (see Neiland and Dukeminier, 1972).

We have not done any hematologic or coprologic studies on Alaskan *Rangifer* spp., but expect to do so in the future.

B. Trematodes

1. *Paramphistomum cervi* Zeder, 1790.

It appears that only two species of trematodes have been reported from North American *Rangifer* spp. Of these, one apparently may be *P. cervi*. Bergerud (1971) reported *Paramphistomum* sp. (?) in Newfoundland caribou where about 70 per cent of a small sample of animals was infected. Bergerud neither fully identified the parasite nor saw evidence of pathogenicity.

We have commonly seen what I tentatively identify as *P. cervi* only in caribou from the Alaska Peninsula herd (one single specimen was found in a Nelchina animal). The apparent low prevalence (Nelchina herd) or absence (Arctic herd) of this fluke in caribou elsewhere in Alaska appears to be a matter of snail ecology. The known intermediate hosts for *Paramphistomum* are aquatic snails. The common final hosts in North American wildlife are *Alces* spp. It appears that *Paramphistomum* only commonly occurs in caribou in areas where the proper snail habitat is prevalent and where caribou commonly forage on such swampy range. Whether moose are involved as a primary reservoir of infection for caribou is unknown. However, the range of caribou on the Alaska Peninsula overlaps that of substantial herds of moose and is comprised of many swampy areas where caribou rest and graze throughout the summer.

I have not seen evidence of pathogenicity of *P. cervi* in caribou. However, it should be noted that this and related species of *Paramphistomum* are well known, severe pathogens in domestic ruminants and in a few instances in wild ruminants. Our failure to see signs of pathogenicity in caribou (or moose in which they also are common in certain areas in Alaska) may be due to the fact that it is the immature worms that cause the problem. We have not had the chance to look at many animals in mid or late summer when larval flukes are being ingested from infected forage.

In Eurasian *Rangifer* spp. two species of amphistome flukes have been reported: *P. cervi* by Nikolaevski (1953) and *Cotylophoron skrjabini* described as a new species by Mitskevich (1958). Neither of these reports is available to us in original form at this time so we do not know whether any pathological conditions were noted.

2. Other Species

No other species of trematode nor any pathological evidence of such have been seen in Alaskan caribou.

Pathological signs of *Fascioloides magna* (Bassi, 1875) was first reported in Canadian caribou by Erickson and Higby (1942) and recently Choquette et al. (1971) have reported finding adult worms in woodland caribou in Quebec. This liver parasite causes unmistakable lesions in ruminants which we have never seen in Alaskan species. It appears that this potentially severe parasite does not now occur in Alaska.

C. Cestodes

We know of at least five species of cestodes that infect caribou either as adults or larval worms.

1. *Avitellina arctica* Kolmarkov, 1938.

This tapeworm, which occurs in the mature form in the small intestine of *Rangifer*, was first noted in North America by Gibbs (1960b). We have found it only once in a light infection in an adult caribou from the Alaska Peninsula herd. Whether it is truly restricted to that herd is not known. It may be that the obligatory intermediate host (probably a free-living oribatid mite) is restricted to that part of the state. However, since the worm has only been found in reindeer or in caribou which have associated with reindeer, it may be that it is not naturally enzootic in North America, and was introduced with reindeer at the turn of the century.

We know of no bona fide report implicating *Avitellina* spp. as serious pathogens of ruminants. However, in chronic parasitism, especially in heavy infections, they no doubt claim their share of the host's welfare.

2. *Moniezia* sp.

Tapeworms of the genus *Moniezia* were first reported in North American *Rangifer* spp. (i.e. Alaskan reindeer) by Hadwen and Palmer (1922). More recently Gibbs (1960a) found *Moniezia* sp. (?) in Canadian barren-ground caribou.

We have seen occasional infections in late fall in the Arctic herd in calves of the year. However, we have not examined many of this age class and we have not seen infections in adults.

Polyanskaya (1961) reported that three species of *Moniezia* were responsible for chronic unthriftiness in Siberian reindeer calves which in heavier infections usually succumbed either during the first winter or the following, early spring. He further observed that the peak of infection was in July and August when 27-38 per cent of the year's calves carried up to 62 tapeworm strobilae per animal. Only 13-15 per cent of the adults were infected and no adverse effects were reported.

Whether epidemics of monieziasis occur in caribou calves in Alaska is presently not known. The fact that caribou are free ranging mediates against this commonly occurring. According to Hadwen and Palmer (1922) *Moniezia* sp.(?) occurred more frequently in closely herded reindeer and were found in young animals almost exclusively. They stated, "when the worms are numerous the fawns must necessarily be adversely affected."

3. *Taenia krabbei* Moniez, 1879.

This species of tapeworm matures in canids and uses *Rangifer* spp. and other wild ruminant prey as hosts for its larval stage. Wherever wolves are present the cysticerci of this tapeworm will be found in the muscle tissue of *Rangifer*. This is one of the most common parasites of caribou and other Alaskan cervids. No doubt many light infections have been overlooked since it is seldom practical out in the field to minutely examine every muscle of a caribou for these wheat grain-sized cysts. It may well be that in many areas any caribou that reaches 10 years of age will be infected with at least a few cysts. The cysticerci of *T. krabbei* have been widely reported in various wild ruminants in the circumboreal region (see Neiland and Dukeminier, 1972).

On occasion we have seen what appeared to be relatively severe infections. An emaciated female caribou taken from the Nelchina herd had at least 2700 cysts distributed thoughout most of the major muscles. Whether the poor condition of this animal was a result of the infection is impossible to say with certainty. Cattle have been killed by experimental infections of 10,000 or more cysts and presumably the host reaction to this parasite is a more or less variably graded response.

4. *Taenia hydatigena* Pallas, 1766.

This tapeworm also matures in canids, like its congener discussed above, and uses wild ruminants as the host for the larval stage. In this instance the cysticerci are found primarily in the liver, although also less commonly in or on other abdominal organs.

The distribution and abundance of the larvae of this species in Alaskan caribou essentially parallel that stated for *T. krabbei*, and the two most often occur together in the same animal. The cysticerci of *T. hydatigena* are much larger, about the size of an average olive, and one seldom sees more than 5-10 in a caribou liver. Why individual infections of this species usually involve fewer cysts than normally appears to be the case for *T. krabbei* is not known. Physically, the worms are almost identical.

Experimental infections of *T. hydatigena* in domestic animals involving "thousands" of larvae have proven extremely debilitating or even fatal. It seems unlikely that caribou are more than rarely exposed to enough viable tapeworm eggs on forage to produce a serious infection. Occasional small doses of eggs very likely promote an immunizing reaction.

5. *Echinococcus granulosus* (Goeze, 1782).

This tapeworm is another taeniid tapeworm that matures in canids and utilizes wild ruminants as hosts for the larval stage. In this instance the larvae occur as thousands of immature "heads" (protoscoleces) in a fluid-filled hydatid cyst which may grow as large as a grapefruit.

This parasite is world-wide in distribution, although biological strains which are more or less restricted to certain host complexes (e.g. circumboreal cervid-canid strain) do occur. Hydatid cysts are found less commonly in caribou of a given area than are the cysticerci of its taeniid relatives discussed above.

The cysts almost always locate in the lungs of the cervid intermediate host and appear not to be a serious pathogen. The usual infection will involve up to four or five walnut-sized cysts. I have never seen massive infections (e.g. 50-100 cysts) in caribou as I have on a number of occasions in moose. However, the largest hydatid I have ever seen, about the size of a large grapefruit, was found in a caribou from the Arctic herd. Judging from the massive infections seen in moose in good condition, it appears unlikely that the much lighter infections seen in caribou (frequently as not also in good condition) are seriously debilitating. In this regard I strongly question the validity of Crisler's (1958) use of observations on hydatid-bearing, wolf-killed caribou as part of a proof that wolves usually only kill debilitated caribou.

On occasion I have observed hydatids that have collapsed and were obviously degenerating in otherwise healthy animals.

The hydatid parasite can also grow in human lungs and is considered to be more or less of a human health problem around the world. In most (all?) instances non-cervid strains are involved which more frequently locate in the brain where they act like tumors and cause pressure damage. Pulmonary hydatid cysts are not uncommon in Alaskan natives in villages where sled dogs commonly harbor the adult stage which produces the infective eggs. While it used to be common practice to surgically remove such pulmonary cysts, it evidently is now considered that the "cure" may be more dangerous than the typical infection and health authorities no longer routinely resort to surgery. In any event pulmonary hydatidosis usually is symptomless and infections normally only come to light on TB X-rays.

It seems we cannot avoid concluding that the cervid strain of *E. granulosus* enzootic in Alaska is normally not a serious pathogen either to man or beast.

D. Nematodes

A variety of roundworms have been reported from Eurasian *Rangifer* spp. (Neiland and Dukeminier, 1972). At present only a couple of species have been reported from Canadian *Rangifer* spp., (Gibbs, 1960a and Erickson and Higby, 1942), and we know of only one lungworm, a body cavity worm and three or so gastrointestinal worms in Alaskan reindeer and caribou. These are separately discussed below.

1. *Dictyocaulus viviparous* (Bloch, 1782).

This lungworm is a world-wide parasite of *Rangifer* spp. and other ruminants and has been variously named *D. hadweni* and *D. eckerti* by other workers. It has been observed in Alaskan caribou in 10 per cent or less of the animals examined in relatively light, uncomplicated infections. This is no doubt ascribable to the fact that wide-ranging caribou seldom remain on a piece of summer range long enough to build up the populations of infective lungworm larvae on their forage. Furthermore, *Dictyocaulus* spp. larvae are less resistant than other nematode larvae and may only survive for up to 13 weeks and rarely through the winter in cold climate (Levine, 1968). Accordingly, only under relatively crowded conditions on restricted ranges and under milder climatic conditions do *Dictyocaulus* infections build up in wild or domestic ruminants to higher levels. The effects on helminth burdens of crowding versus moving *Rangifer* periodically to new range has been considered for Alaskan reindeer by Hadwen and Palmer (1922).

More recently Klein (1968) had the opportunity to study the growth and near extinction of a population of reindeer introduced to St. Matthew Island in the Bering Sea. He visited the 128 square mile island for short periods of time in the summers of 1957, 1963 and 1966. On these occasions he collected and necropsied for *Dictyocaulus* and other parasitic or infectious conditions the following animals (estimated total population sizes and approximate dates of collection shown in parentheses):

1957 (July-Aug.) - 12 animals (1350) - no lungworms

1963 (July) - 15 animals (6000) - 3 animals lightly infected

1966 (July) - 10 animals (42) - no lungworms

His conclusions regarding these few parasitological data are as follows: "While sample sizes are too small to enable statistical comparisons, lungworms (*Dictyocaulus* sp.) were found in three of the 1963 animals and in none of the 1957 or 1966 reindeer. None of these infestations were acute." Klein continued, "Lungworm, which has been implicated in mass mortality among other cervids (Cowan, 1951) may have contributed to reduction of the St. Matthew herd, but it certainly was not present in epizootic proportions during the summer preceding the die-off." In summing up, he draws the following conclusions regarding the cause of the "crash" die-off. "Food supply then, through interaction with climatic factors, was the dominant population regulating mechanism for reindeer on St. Matthew Island. Other factors of population control, such as disease or parasites and predation, can be ruled out and there is insufficient evidence to suggest that self-regulatory mechanisms of a behavioral (Wynne-Edwards, 1965), a genetic (Chitty, 1960), or a behavioral-physiological nature (Christian and Davis, 1964) were involved in the die-off."

While there can be little argument with the idea that under-nutrition very likely played a significant role in the catastrophic decline of the St. Matthew herd, the factual support for "ruling out" the significant

involvement of "disease or parasites" is weak at best. Observations made well before or after the fact of a die-off, even those made by personnel with considerable training or experience in animal pathology, seldom can be expected to effectively document the probable health status of the animals that died. Even if we assume that the only parasite (not to mention microbial pathogens) present in the herd was the lungworm, *Dictyocaulus*, and my 11 years of parasitiological experience with *Rangifer* elsewhere in Alaska and other considerations lead me to question this assumption, there still is a distinct possibility that parasitic bronchitis (dictyocauliasis) and pneumonia could have occurred in epizootic proportions during the actual die-off many months after Klein left the island. Indeed it is difficult to understand the factual or logical connection between Klein's initial statement (p. 359) that, "while sample sizes are too small to enable statistical comparisons lungworms (*Dictyocaulus* sp.) were found"... (only in animals taken in 1963 just before the die-off) and his claim in the following paragraph that, "lungworm... certainly was not present in epizootic proportions during the summer preceding the die-off." Regardless of these conflicting statements, the epidemiological and meteorological facts in the literature do provide for the possibility of a lungworm epizootic occurring later in the summer and fall of 1963.

The weather records for St. Lawrence Island to the north of St. Matthew Island and for the Pribilof Islands to the south (Anon. 1963) reveal that is is most likely that St. Matthew was free of frost until sometime in October. Indeed the first 32°F temperature on St. Lawrence Island to the north did not occur until September 30 in 1963 and the average temperature for the entire month was 41.7°F. Average September temperatures were over 2°F. higher on the Pribilof's in 1963, although a temperature of 32°F. was recorded on August 24. Accordingly, it appears reasonable to assume that climatological conditions were favorable for the transmission of *Dictyocaulus* on St. Matthew Island into October, which at most is a few months prior to the die-off.

According to Levine, (1968) three to four weeks after infection adults of *Dictyocaulus viviparous* begin to produce eggs. These are coughed up, swallowed and pass out in the feces. Within four days the third stage infective larvae develop and transmission occurs when these are swallowed with forage. Under cool, maritime conditions, such as occur on St. Matthew in the summer, the infective larvae may survive for up to 13 weeks. Accordingly the generation time for this parasite is about 25-30 days, and for as long as 30-72 days adult worms will produce eggs which yield larvae that are infective as long as 13 weeks later under cool, humid conditions. It appears likely that two full generations of lungworms could have occurred after Klein left the island in 1963. Indeed the first generation for the summer was likely already underway when he was there. At least a week after infection the worms (i.e. 4th stage larvae) are too small to see with normal vision. Thus early infections can be easily overlooked.

According to Dunn (1969), "...the appearance of dictyocauliasis depends upon the accumulation of infection on the pasture over the

grazing season. The high seasonality of the disease, in late summer and early autumn, is attributable to this cumulative infection." Thus factors which would have tended to concentrate the summer feeding and movements of reindeer on St. Matthew Island in 1963 would have magnified the accumulation of lungworm larvae on the forage. Klein (op. cit.) states that sedges were the predominant summer forage and "...received the brunt of the summer use." He continues that, "closely cropped sedges and grasses were present in all vegetation types supporting these plants but were most extensive in moist but well-drained meadows, on lake shores and lake flood plains, and on the drier slopes of hills." It would appear that much of the summer grazing in 1963 was restricted to moist, but well-drained foci particularly favorable to the survival and concentration of lungworm larvae.

The preceding, somewhat lengthly discussion is not intended to serve as support for the hypothesis that epizootic verminous bronchitis and pneumonia actually did play an important role in the precipitous decline of the St. Matthew herd. My intention is to establish that this could have happened and that Klein (1968) had no valid scientific grounds for arbitrarily ruling out disease as a factor in the exceptionally abrupt decline of the herd. His statement that *Dictyocaulis* "...certainly was not present in epizootic proportions during the summer preceding the die-off" is without any significant scientific merit since neither did he adequately sample the herd of 6000 animals as he admitted nor was he on the island during the time when an epizootic could have been expected to occur.

2. *Setaria yehi* Desset, 1966.

This filarial, body-cavity worm was first reported from Alaska in reindeer which were held for nutritional experiments by Dieterich and Luick (1971). All 15 animals held at their college installation and 10 of 13 held at Cantwell showed microfilariae in their blood and all five animals sacrificed in their studies yielded from five to 20 adult worms free in the body cavity. A "low-grade peritonitis" was observed in each of these five animals while three animals without microfilariae or adult worms were free of any comparable pathologic signs. No evidence was observed of cerebral-spinal nematodiasis as reported for infections by *Setaria* in other species. This parasite and a closely related form *S. labiatopapillosa* (Perroncito, 1882) were reported earlier in Canadian caribou by Becklund and Walker (1969). Another species *S. cervina* Dujardin, 1845, (see Becklund and Walker, 1969, for synonymy) was reported even earlier in Eurasian reindeer by Raevskaya (1928). The latter publication is not presently available to me, but its title, "Setaria and their pathogenic significance", suggests that Raevskaya may have seen at least some pathological signs in infections of *S. cervina* which he reported in that paper (see Becklund and Walker, 1969).

Neither in the hundreds of free-ranging caribou I have personally examined nor in the large numbers examined in the field by divisional game biologists has even one specimen of *Setaria* ever been seen. Considering the large size of the worm and the "tidy circumstances" of the

ruminant body cavity except in "gut-shot" animals, it seems likely that, if indeed the worm does occur in free-living Alaskan caribou, it must do so only very rarely. Why then were the penned reindeer commonly infected, but not other animals in the herd at Nome from which the penned animals came (Dieterich and Luick, 1971)? The answer seems to involve the specific, ecological characteristics of *Setaria* in Alaska.

We have found *S. yehi* on numerous occasions in moose in Alaska (see Becklund and Walker, 1969) but only in animals taken in the Interior, particularly more or less commonly in the general vicinity of Fairbanks. Apparently the worm is geographically restricted in its common or reservoir host by the biological requirements of the blood sucking flies by which it is transmitted. It appears that infected moose and the parasite's vector(s) (other species are transmitted by blackflies or mosquitos) do not commonly occur together, if at all, in close contact with the caribou herds we have investigated. On the other hand, the reindeer in question were brought from an area where moose are relatively scarce (and not known to be infected) into an area in which most of the infections in moose have been seen. The parasite is apparently well adapted to cervids and thus was readily transmitted from moose to reindeer when the opportunity arose. It may well have been that only one moose to reindeer transmission actually occurred and the high prevalence in the penned animals resulted from reindeer-to-reindeer transmissions among the more or less crowded animals after the parasite was introduced into the penned herd.

The failure of Dieterich and Luick (1971) to see cerebrospinal complications in their infected reindeer is not surprising. As they failed to note, the cases of such complications which are reported in the literature involve a species of worm, *Setaria digitata* (Linstow, 1906), which normally parasitizes the bovine, *Bos indicus*. It is when this bovine-adapted parasite is transmitted to aberrant hosts of other taxonomic groups, e.g. horses, sheep and goats, that the parasite wanders into abnormal anatomical sites in the aberrant host (e.g. central nervous system) and causes nervous disorders. This same situation occurs when the apparently more highly host-adapted meningeal worm of white-tailed deer, *Pneumostrongylus tenuis* (Dougherty, 1945), invades the central nervous system of moose, caribou, and other species to which it is (by definition) less well adapted. In these apparently aberrant hosts we again see serious cerebrospinal complications.

Accordingly, if *Setaria yehi* does sometimes cause nervous disorders in Alaskan wild ruminants, it would most likely do so in a non-cervid. Dall sheep may be a good candidate for such complications. There are many areas in Alaska where moose and sheep live more or less closely together during the fly season.

3. Gastro-intestinal worms.

We have found several species of trichostrongylid roundworms in the abomasa and small intestines of caribou in Alaska. These include *Teladorsagia davtiani* Andreeva and Satubaldin, 1954, first reported from Alaska in reindeer (Becklund, 1962), and *Nematodirus skrjabini* Mitskevich,

1929, previously known only from Eurasian reindeer. We have found trichostrongylids in each of the three caribou herds under consideration under field conditions, but have only had a chance to examine suitable organ samples from the Arctic herd under lab conditions. This latter collection from 50 or so animals while no doubt qualitatively representative may not be quantitatively representative of either the Arctic herd at different times or of other herds at any time. Further, we have not yet identified all of the several thousand specimens which were collected in the lab. Accordingly, we can only speculate that trichostrongylids are common parasites in all Alaskan caribou, but seldom in heavy infections. Nevertheless, because they are well known to be serious parasites of domestic ruminants when they occur in sufficient numbers, it seems likely that when conditions are favorable to their reproductive dynamics, they may also occur in significantly harmful numbers in caribou and other wild ruminants.

Another nematode parasite is also known to occur in Alaskan caribou. *Skrjabinema oreamni* Swales, 1934, a caecal pinworm first described from Canadian mountain goats *(Oreamnos americanus)*, was commonly observed in caribou of the Arctic herd during the early 1950's (personal communication, R. L. Rausch, Arctic Health Research Center). During the period 1962-1971, I examined in the field hundreds of caeca from caribou from the Arctic herd without ever seeing one worm. These parasites are large enough that it seems unlikely that an experienced person would overlook all infections that came to hand. Accordingly, one can only conclude that in recent years this pinworm has been a rare parasite of Arctic caribou, perhaps occurring in occasional very light infections, if hardly at all, that were missed under field conditions. Other species of *Skrjabinema* are not known to be pathogenic in the domestic species in which they occur (Levine, 1968).

E. Arthropods.

The parasitic and free-living blood-sucking insects that commonly associate with caribou and reindeer in Alaska in great numbers are among their most serious afflictions. These are all flies with the exception of a species of louse which recently has been found.

The blood-sucking flies, including mosquitos, blackflies, etc., are a never-ending source of harassment during the summer, which, because of their feeding habits, are a significant drain on the vitality of *Rangifer* wherever it occurs. We have not made any attempt to rigorously investigate this matter and therefore will not attempt to summarize the problem. However, it seems worthwhile before passing on to the truly parasitic insect pests of caribou to take note of some observations on insect harassment published by Kelsall (1968). It is reported that the death of "several hundred" caribou near Bathhurst Inlet in the summer of 1949 most likely resulted from extreme insect harassment. Eskimos observed the extreme agitation of a local herd and salvaged many carcasses with broken limbs from the small area in which the insect problem had apparently occurred. Kelsall also notes that reports from the "bush" of deaths of substantial numbers of caribou in other areas in Canada have involved similar circumstances.

Such acute, fatal interactions with insects involving a few hundred animals now and then are probably not markedly significant to large caribou populations. However, the chronic, non-fatal affects of continued harassment throughout the fly season may cost a caribou population a large bill for energy consumed in evasive activities and may lead to general unthriftiness of animals going into the winter regardless of the quality of the range.

The two species of flies whose larval stages parasitize caribou also are a source of great harassment. These two parasitic flies and the previously mentioned louse are separately considered below.

1. *Oedemagena tarandi* (Linnaeus).

The caribou warble fly is with few exceptions a universal parasite of *Rangifer* throughout the latter's circumpolar range. This fly is not known to parasitize any other species of ruminant and it appears that prevalence rates of 90-100 per cent are the usual thing in most areas under normal conditions. Individual infestations may involve as many as 1000 grubs (Hadwen and Palmer, 1922) and normally are heaviest in young animals suggesting that some kind of physiological resistance may develop as the host matures and is repeatedly exposed to the larvae each summer. In this regard Breev and Karazeeva (1953) report that in Eurasian reindeer healthy animals are least infested.

Other factors also influence the prevalence and intensity of infestations. It is well recognized that reduced light intensity (e.g. shade, cloudy weather, etc.) generally reduces the biting and other harassing activities of dipterous pests. Cooler temperatures also have a similar influence. This is recognized by reindeer husbandmen as an important factor in selecting summer range (Porsild, 1942). The larval and adult stages of the warble fly are reduced in numbers following a cool, cloudy summer. Late frosts after the fully developed larvae drop from the host to pupate in the ground result in much reduced numbers of adults, and therefore larvae, in the following generation (Nakhlupin and Pavlovskii, 1932). The health of the individual reindeer also affects the survival and maturation of warble larvae. Breev and Karazeeva (1953) report that fully developed larvae rarely die *in situ* in the host unless the host is noticeably emaciated.

We have noticed and reported (Neiland, 1963) that male caribou are more heavily infested with warbles on the average than females. We know of no published explanation for this sexual difference which also has been more recently reported by Kelsall (1968). However, the following explanation seems tenable. The warble fly is more inclined to strike light-colored individuals than darker ones (Espmark, 1961). During the latter part of the summer when the adult warble fly is active, male caribou, particularly older age classes, have a very light colored, extensive "cape". At the same time the females are generally quite dark colored overall. Thus the female warble fly attacks the more lightly colored male caribou more frequently than darker female caribou because of its chromotropic behavioral responses.

One might expect that caribou herds inhabitating areas which are normally windy would be less heavily parasitized than those using windy areas. This appears to be the case in Alaska. Animals from the herd on the windier Alaska Peninsula generally have fewer warble and bot fly larvae than caribou from the Arctic or Nelchina herds where prolonged, heavy winds are less common during the fly season.

No one seems as yet to have clearly demonstrated that warble infestations have a direct effect on the condition of caribou or reindeer although they are said to be an economic drain on the reindeer husbandry in Scandinavia (Nordkvist, 1971). However, that they indirectly affect the welfare of afflicted populations is beyond dispute. We have already briefly noted that the adult warble fly along with various other biting insects are sources of harassment of caribou. It actually appears that on a "per fly" basis the warble (and bot) fly is by far the greatest source of annoyance of reindeer and caribou. A single adult warble fly will provoke vigorous, and at times almost uncontrolled, evasive actions on the part of offended animals. These violent, evasive reactions frequently lead to increased accidental hoof injuries and consequent hoof infections (i.e. hoof rot). Thus it is reported by Russian reindeer husbandmen who have made studies on the relationship between prevalence of warble and bot flies and the occurrence of foot rot (i.e. necrobacillosis) that warble fly control reduces or prevents the latter disease. For example, Nikolaev et al. (1957) claimed that not a single case of necrobacillosis occurred in a reindeer herd in which warble flies had been controlled and Mezonev (1957) reported that control measures against female warble flies resulted in 2.8 times fewer deaths from the bacterial disease. Working with several reindeer herds, Terent'ev (1961) found that control of adult nose bot and warble flies greatly depressed the rate of deaths from necrobacillosis (i.e. 1.4 per cent in treated herds vs. 11 per cent untreated herds). Tarasenkov (1965) also claimed that spraying reindeer herds with insecticides reduced the incidence of the disease. Thus it might be supposed that the poorly documented "epidemics" of foot rot that apparently occur from time to time in some Alaskan caribou herds (e.g. Arctic and Alaska Peninsula) are a result, at least in part, of increased harassment by warble and bot flies during a summer when the flies are particularly abundant under conditions favorable to their attack. We have already noted the relationships of light intensity, average windiness and sex to severity of fly strike. Thus we might speculate that we would see increased prevalence of foot rot, particularly in male caribou, following a summer which had clearer, warmer, quieter weather. Late frosts in early summer during pupation would lower the numbers of adult flies and offset the disease-favoring influences of later weather indicated above. The epidemiology of diseases or parasites of wild animals may indeed be dynamic!

2. *Cephenemyia trompe* (Linnaeus).

The nose bot fly of reindeer and caribou is also circumpolar in distribution and generally occurs wherever the warble fly does. However, nose bots are seldom, if ever, as prevalent as warbles and normally only a quarter or third of a herd will carry bots (Bergerud, 1971 reported

83 per cent prevalence in Newfoundland) when nearly all will have warbles. This might be explained by the greater difficulties faced by the female bot fly in her attempts to appropriately deposit her larvae. While the warble fly simply lays her eggs anywhere on the underparts of the deer, the bot fly must maneuver, flying backwards, around the face of the deer and squirt a load of larvae into the deer's nostrils (Hadwen and Palmer, 1922). Apparently the immediate response of the deer is to snort violently, no doubt frequently expelling many if not all of the unwelcome visitors.

Otherwise the nose bot interacts with its host and the surrounding environment in much the same way as the warble fly, although it is claimed (Hadwen and Palmer, 1922) that the bot fly is much more annoying to *Rangifer*. Accordingly, the principal effect of bots on deer may involve the direct and indirect affects of harassment discussed in some detail in the section on the warble fly.

The intensities of infestations seen in Alaskan caribou range upward to about 150 individuals with an average of about 25-50. They are less common in areas where summer winds blow harder and more frequently (e.g. Alaska Peninsula). There seems to be no significant differences between the intensities of infections seen in male and female caribou. This contrasts with similar observations on warble infestations. Perhaps the nose bot fly's necessary preoccupation with the nose of its intended host, which is similarly constructed and adorned (e.g. color of pelage) in both sexes, explains the similarity of the intensities of infestations seen in both sexes, one sex being as attractive as another.

Little exact information on the direct affects of the larvae on deer seems to be available. Bergman (1917) according to Hadwen and Palmer (1922) claimed that in Lapland bot larvae sometimes produced fatal disease in reindeer, and Cowan (1951) states without obvious qualification that, "It is reported to kill caribou infrequently..." Hadwen and Palmer (1922) report that in contrast to nose bot infestations in domestic sheep, those in reindeer cause only very slight nasal discharges, but that reindeer do display symptoms similar to the "staggers or false gid" seen in bot-infested domestic sheep. They further note that deaths of reindeer from nose bot infestations have not been seen in Alaska. Bergerud (1971) reported a presumed fatal case of invasion of the cranial cavity of a stag deer by nose bot larvae. The animal was observed "walking in circles" prior to its death from an unclearly stated cause, (i.e. did the animal actually die from the bot infestation or was it collected for study after its unusual behavior was noticed?).

No doubt many interesting observations on nose bots are available in the voluminous Russian reindeer literature which is largely unavailable to me at this time (see Dukeminier and Neiland, 1972).

3. *Solenopotes tarandi* (Mjoeberg, 1915).

Specimens of this louse were recently recovered from an Arctic caribou hide sent to Dr. Christian Weisser, University of Pennsylvania,

for his use in an ectoparasite survey. This and other small species of lice can only be reliably recovered from infested hides by laboriously macerating pieces of hide with strong alkali. The chitinous exo-skeleta of lice, fleas, mites, etc. do not dissolve as does the hide and hair of the sample. Because of lack of manpower we have never felt it was worthwhile to attempt to do a reliable ectoparasite survey of caribou or other cervids. We assume that lice (and mites) may well be commonly present on caribou in Alaska.

There seems to be little reason at present to be concerned over the effects of lice infestations on caribou and reindeer in Alaska. However, various species of lice do act as vectors of disease organisms and it is clearly within the realm of possibility that a vector-parasite (e.g. virus) relationship with *Rangifer* as host could develop or may indeed already occur.

Hadwen and Palmer (1922) suggested that cases of unexpected loss of hair sometimes seen in reindeer may have been due to lice infestations.

II. Microbial Diseases.

A variety of microbial diseases are known to occur in caribou and reindeer throughout their circumpolar range (see Neiland and Dukeminier, 1972). Most of this information deals with diseases of reindeer in Eurasia where reindeer husbandry is an important agricultural practice.

In North America relatively little effort has been expended on studies on rangiferine pathogens. In one of the classic publications, Hadwen and Palmer (1922) spend only 3 1/2 of 74 pages discussing diseases of reindeer in Alaska and less than one of 339 pages of Kelsall's (1968) extensive monograph on Canadian caribou deals with this subject. Our own studies, which were initiated in cooperation with Dr. Ronald Skoog in April, 1961, and later involved cooperation from the U. S. Department of Agriculture and the Arctic Health Research Center, were briefly summarized through 1964 in Skoog's monumental thesis (University of California, Berkeley) on Alaskan caribou and by Neiland et al. (1968). Altogether, relatively little time and money has been spent on the study of diseases of *Rangifer* in North America compared to the hundreds of thousands of hours and dollars expended on other facets of this species' biology. Nevertheless, it has been clearly demonstrated that Alaskan caribou populations are infected with a number of enzootic diseases which, in at least one instance, spread to other wildlife species and also man. These include brucellosis, leptospirosis, necrobacillosis, California encephalitis virus, infectious warts (papillomas) and several other disease conditions of unknown etiology. Each is separately discussed in more or less detail in the following sections.

A. Brucellosis.

1. Description of the disease.

Brucellosis in caribou and reindeer is caused by a bacterium, *Brucella suis* type 4, for which *Rangifer* spp. apparently are the natural

reservoir hosts in nature. At present it appears that when the disease is found, as it is, in other wild species and man, it has spread from *Rangifer* to these other susceptible hosts. Thus it seems entirely proper to recognize this natural distribution and therefore to call the disease rangiferine brucellosis regardless of the host actually infected in each instance. This line of reasoning has been carried "a step further" by Russian investigators who even propose to call the causative organism *Brucella rangiferi* rather than the accepted name, based upon the recommendations of the World Health Organization's Expert Committee on the Taxonomy of Brucella, which in turn are based upon biochemical and antigenic considerations. For a more detailed analysis of this taxonomic question see Neiland et al. (1968).

A very closely related organism *Brucella suis* type 5 has recently been demonstrated as the cause of abortion and sterility in beagle dogs. This strain will be discussed in greater detail in the section dealing with brucellar infections in other Alaskan wildlife.

Rangiferine brucellosis in caribou causes many of the classic symptoms seen in other ruminants infected by other strains or species of *Brucella*. Abortion is a common and notable feature of the infection in females in their first pregnancy and may also be seen in later pregnancies. Chronic infection of the male reproductive organs occurs and transmission of the disease probably occurs most commonly during the rut. Infection of tarsal and carpal joints is another more or less common occurrence. Placental retention is frequently seen in animals from our Arctic herd in which rangiferine brucellosis has been common for at least the past ten years or so. Whether this condition is strictly a result of infection by *Brucella* is still doubtful. A more detailed discussion of this condition will be presented in the later section dealing with calving ground studies. For further discussions of the pathology of rangiferine brucellosis in caribou see Neiland et al. (1968) or the Russian references in Neiland and Dukeminier (1972).

2. Prevalence in Alaskan Caribou.

Thus far clear cut evidence of brucellosis infections has been obtained from only two of the 11 Alaskan caribou herds recognized by Skoog (see Fig. 1, Neiland et al., 1968). When studies on rangiferine brucellosis were first initiated in 1962 the disease appeared to be present in epizootic proportions in the Arctic herd (30 per cent serological reactor rate) and less common (6.5 per cent serological reactor rate) in the Nelchina herd. A few minimal, 1:20 titres obtained from a small sample of serum from the Fortymile herd are not considered indisputable evidence of infection in that herd. The absence of titres in approximately 50 sera from the Alaska Peninsula herd is not considered to be adequate evidence that the disease does not exist in that herd. In more recent years the reactor rates of the Arctic and Nelchina herds have declined to chronic levels of about 10 and 1 per cent, respectively, even though fairly high titres indicative of active infections (e.g. = 1:160) still commonly occur. Unreported serological data accumulated during the time period January, 1970 to June, 1971, are summarized in Tables 1 and 2.

Table 1. Comparison of brucellosis reactors in Alaskan caribou as revealed by complement fixing and agglutinating serologic tests.

Specimen Number	Serologic Results	
	Complement Fixation	Agglutination
A54369	4+, 1:160	4+, 1:40
A50596	2+, 1:320	4+, 1:40
A50604	4+, 1:80	Neg.
A50607	4+, 1:20	Neg.
A50609	4+, 1:20	Neg.
A50611	4+, 1:640	2+, 1:40
A50620	2+, 1:320	4+, 1:160
A50626	2+, 1:40	2+, 1:40
A50637	4+, 1:320	2+, 1:80
A50652	Neg.	2+, 1:40
A50648	2+, 1:40	Neg.
A45023	4+, 1:320	4+, 1:40
A45027	4+, 1:320	3+, 1:640
A45027 (calf)	4+, 1:320	4+, 1:640
A45002	4+, 1:160	2+, 1:320
A45005	1+, 1:320	3+, 1:40
A45008	4+, 1:40	Neg.

Table 2. Brucellosis reactors in Alaskan caribou, January, 1970 to June, 1971.

Herd	Date	Sample Size	Reactors[1] Titre	Rate
Arctic	1970	18	4+, 1:40 4+, 1:320 4+, 1:320	16%
	1971	42	2+, 1:20 4+, 1:160 4+, 1:40 1+, 1:40 4+, 1:320 2+, 1:640 2+, 1:320 1+, 1:640 4+, 1:640	21%
Nelchina	1970	36	2+, 1:40 Inc., 1:320 Inc., 1:80	8%
	1971	66	None	
Steese	1971	4	None	

[1]All titres shown except those for the Nelchina herd are for the complement fixation procedures with *Brucella abortus* smooth antigen. The Nelchina titres were obtained in the standard tube aggultination test. The Arctic reactors were primarily observed in animals showing retained placentas collected on the calving ground and reported in Table 3.

A comparison of complement fixing and agglutinating procedures was initiated several years ago when it was suspected that the latter method, which is the one commonly used by the U.S.D.A., might not detect as many reactors as the former procedure. Even a cursory inspection of the limited data shown in Table 1 reveals that the complement fixation procedure is significantly more sensitive in detecting carriers of *Brucella* antibodies. See Neiland et al. (1968) for further discussions of this point. Further comparisons of the two procedures are also made in section 4 which follows. Table 2 shows the small amount of data collected on brucella reactors in caribou herds throughout the state in 1970 and 1971. Neither Eskimos nor cooperating biologists (in 1970) were very successful in obtaining serum for testing. Most of the Arctic reactors were observed among animals I collected on the calving grounds. Since these animals were specially selected, apparently diseased animals, the reactor rates should not be taken as representative. I assume that rangiferine brucellosis is still enzootic in the Nelchina herd, though no doubt at a very low level.

Rangiferine brucellosis also occurs at chronic levels in some Alaskan reindeer herds which may be the ultimate reservoir of the disease here and elsewhere. Evidence of the disease in Canada has been recently reported. Broughton et al. (1970) observed titres of 1:25 or higher in 8.74 per cent of 1692 Mackenzie River Delta reindeer and in 4.3 per cent of 320 barren-ground caribou from the Kaminuriak herd. However, they reported that they did not see pathological signs in the reindeer tested at slaughter nor in 500 caribou necropsied during life history studies.

There is an abundant literature on rangiferine brucellosis in reindeer herds, in which the disease was first recognized about 1949 (see Neiland and Dukeminier, 1972, for detailed bibliographic citations). Various pathological conditions similar to those seen in Alaskan caribou are normally associated with brucellosis infections in Eurasian reindeer and these are noted at length in the Russian literature. It may well be that adequate testing of all distinct herds of *Rangifer* spp. throughout their circumpolar distribution will reveal that chronic brucellosis is ubiquitous, normally affecting only a relatively small proportion of a herd at any particular time.

3. Calving Ground Studies.

When it became evident that placental retention following the birth of fawns commonly occurred in Arctic caribou in which brucellosis was common, while not apparently in other herds where the disease didn't occur, it was supposed that the condition was the result of a brucellosis infection. However, it was realized that this might be a spurious correlation. Later it was learned that a substantial fraction of the does that retained placental material lost their fawns a few days post partum. At that time it was decided that an attempt to collect affected animals on the calving ground should be made in order that the causation of the condition might be determined. A three-season study was outlined for the calving periods in 1969, 1970 and 1971 during which helicopter support would be available for collecting affected animals. We also arranged for

Dr. Daniel O. Trainer, Department of Veterinary Science, University of Wisconsin, to accompany us in the field and to oversee specialized bacteriological and virological studies at the University of Wisconsin on specimens we collected on the Arctic calving ground. The data on prevalence of placental retention and serological tests for brucellosis, leptospirosis and arboviruses are given in Table 3.

The principal conclusion to be drawn from the data in Table 3 is that since only eight of the 42 animals collected showed brucella titres, it is unlikely that the condition is always caused by brucellar infections. Similarly, since the prevalence for the condition was twice as high in 1971 as in 1970, while at the same time there was an apparent decrease in leptospirosis reactors in 1971 from the 1970 level, it also appears that leptospires cannot be considered a sole cause of placental retention. It is also worth noting here that attempts to isolate leptospires from the 1970 sample failed. The data on attempts to isolate brucellae, leptospires, etc. and the arboviral serology from the 1971 collection are still not available because of circumstances beyond the control of those directly involved in the study. In any event we may likely be left with two alternative conclusions:

1. Placental retention is always caused by some single still-unknown factor, infectious or otherwise.

2. Placental retention may be caused by any one of several factors including infectious processes which may involve brucellae or leptospires.

When conditions were favorable for determining from survey aircraft if animals with retained placental materials produced fawns which survived at least a day or two post partum, it was observed that substantial fractions, i.e. 56 per cent (1968) and 43 per cent (1971), do not. In 1971 weather and snow conditions were ideal on the calving grounds but still the early loss of fawns was substantial. This suggests that the "fawn mortality factor" is not weather dependent. In any event, the absolute loss of fawns associated with placental retention has thus far been low because only a small percentage (1-5 per cent) of the does are affected by the placental retention syndrome. The information obtained from the tissues we collected on the calving ground this past year, and which are finally being processed, will be of particular interest, but may not substantially change our present conclusions.

4. Prevalence in non-rangiferine species.

a. Carnivores.

It has been known for some time that various carnivores, particularly dogs, were susceptible to various strains of *Brucella abortus*, *B. melitensis* and *B. suis*. Therefore it seemed entirely likely early in our studies on rangiferine brucellosis that the disease was probably being transmitted to sled dogs and other carnivores which ate infected caribou meat and offal (see Neiland et al., 1968). When we finally looked into this matter we found abundant evidence that our speculations were correct.

Table 3. Prevalence of placental retention and serologic reactors in Arctic caribou collected during calving activities.

Date	Retained Placenta in herd[1]			Prevalence Reactors in Affected Animals Collected		
				Brucellosis[2]	Leptospirosis[3]	Arboviruses[3]
	Sample Size	Percent Affected	Percent without calves			
1969	4357	0.94	14.6	0/15	---	---
1970	2217	1.04	---	2/7	5/6	3/9
1971	3331	2.01	43.3	6/20	14/19	---

[1]Percent affected is for total sample surveyed. Percent without calves is for those showing placental retention.

[2]Fraction of sample showing titres of 1:40 or higher.

[3]Fraction of sample showing titres of 1:100 or higher to one or more serotypes or viral agents, respectively.

Preliminary data on sled dogs and wolves were reported by Neiland (1970). We now have more extensive data on hand on sled dogs and wild carnivores and these are separately discussed below.

Sled Dogs

The results of a survey for brucellosis reactors among sled dog teams in Arctic Alaska are shown in Tables 4 and 5. The data in Table 5 represent a separate Kobuk team from which *Brucella suis* type was isolated and which was not included in Table 4. Altogether it may be concluded that wherever Arctic caribou are fed to dogs in significant amounts, canid rangiferine brucellosis reactors will be commonly found. Furthermore, meaningful titre levels are maintained in infected animals for nearly a year, if not more (see "moose," Table 5). No other data on *Brucella suis* type 4 infections in dogs are available.

However, observations on the recently discovered and closely-related "beagle dog strain" (i.e. *Brucella suis* type 5 also known as *Brucella canis*) indicate that significant titres and viable brucellae can be observed in infected dogs (i.e. beagles, greyhounds, pointers, cross of foregoing breeds) for many months (Van Hoosier et al., 1970, Moore and Kakuk, 1969). Morse (1951) reported similar observations on dogs infected by strains of *Brucella abortus* or *B. melitensis*.

A variety of pathological conditions have been noted in animals infected with canine brucellosis, i.e. *Brucella suis* type 5. These include generalized lymphadenopathy and also invasion of the spleen, liver, mammary glands, ovary, uterus, testes, kidney, prostrate glands, lung, placenta, fetus, epididymis and blood (Hill et al., 1970; McCormick et al., 1970; Moore and Kakuk, 1969; and Morse, 1951). Of special interest to understanding the possible non-sexual mechanisms of transmission are the observations on urinary excretion of viable brucellae (Moore and Kakuk, 1969) and excretion in both urine and feces (Morse, 1951). Experimental *Brucella suis* type 1 (American strain causing porcine brucellosis) infections in dogs may result in severe, general, systemic manifestations including loss of weight, lassitude, weakness and anerexia (Morse, 1951).

We have not had the opportunity to study the pathogenicity and affect on general well-being of rangiferine brucellosis infections in sled dogs. The dog from which the organism was isolated was only on hand for a short while and exhibited no unusual signs of disease just prior to sacrifice (Neiland, 1970).

Transmission of brucellosis from dogs to humans has been reported on a number of occasions (Nicoletti et al. 1967 and others). One can only wonder whether infected sled dogs are a hazard to their owners through excreting brucellae in their urine and/or feces. If abortion occurs, in cases of rangiferine brucellosis, aborted fetuses would almost certainly lead to infections in anyone who handled them.

Because rangiferine brucellosis infections would likely follow the same course in wild canines discussed in the next section, as in domestic

Table 4. Occurrence of brucellosis reactors in Alaskan sled dogs.

Number	Name	Locality	Date	Aggl. *B. abortus* Smooth	CF *B. abortus* Smooth	Aggl. *B. canis* Rough	CF *B. canis* Rough
17	German	Anaktuvuk Pass	6/67	2+, 1:640	---	---	---
18	Netah	Anaktuvuk Pass	6/67	2+, 1:160	---	---	---
21 other dogs		Anaktuvuk Pass	6/67	neg.	---	---	---
10 dogs		Ambler	5/70	neg.	neg.	1:20 to 1:160	
	Smokie	Kobuk	5/70	4+, 1:640	4+, 1:160	2+, 1:320	
7 team members		Kobuk	5/70	neg.	neg.	1:40 to 1:160	
19 dogs		Ft. Yukon	6/70	neg.	neg.	1:20 to 1:320	
2879	Darkie	Ft. Yukon	8/70	neg.	neg.	Inc., 1:100	
2884		Ft. Yukon	8/70	neg.	neg.	1:100	1:100
1970-1		Gambell	9/70	neg.	neg.	1:100	3+, 1:100
27 other dogs		Gambell	9/70	neg.	neg.	neg.	1+, 1:100 to 1+, 1:200
1970-54		Pt. Hope	9/70	2+, 1:160	2+, 1:40		2+, 1:200
26 other dogs		Pt. Hope	9/70	neg.	neg.	neg.	1+, 1:100 to 3+, 1:200
1970-67		Wainwright	9/70	neg.	1+, 1:40		2+, 1:100
1970-70		Wainwright	9/70	neg.	4+, 1:40		2+, 1:400
1970-75		Wainwright	9/70	2+, 1:640	4+, 1:320	1+, 1:100	2+, 1:200
1970-78		Wainwright	9/70	2+, 1:40	4+, 1:40		2+, 1:100
23 other dogs		Wainwright	9/70	neg.	neg.		1+, 1:100 to 2+, 1:200

Table 4 continued.

Number	Name	Locality	Date	Aggl. *B. abortus* Smooth	CF *B. abortus* Smooth	Aggl. *B. canis* Rough	CF *B. canis* Rough
3004	Captain	Barrow	10/70	4+, 1:320	neg.		
4 other dogs			Barrow	10/70	neg.		1+, 1:200 to 2+, 1:200
19 dogs		Anaktuvuk Pass	5/71	neg.	neg.		
	Red	Anaktuvuk Pass	5/71	2+, 1:320	4+, 1:160		
	Trika	Anaktuvuk Pass	5/71	3+, 1:40	neg.		

Table 5. Serologic observations[1] on a dog team infected with rangiferine brucellosis in Kobuk, Alaska.

	May 1969		July 1969			Sept. 1969		Dec. 1969		May 1970		
Name of Dog	Aggl. Test	CF Test	Aggl. Test	CF Test	USDA Card Test	Aggl. Test	CF Test	Aggl. Test	CF Test	Aggl. Test	CF Test	Aggl. *B. canis* Rough
Beaver			-	-	-	-	1:20	-	-			
Fannie	-	-	-	-	-	-	-	-	-	-	-	2+,1:40
Harry			-	1:20	-	1:320	1:640	-	?	-	2+,1:10	-
Jumbo			1:640	1:640	+	L;20	1:20	1:160	1:160			
King	-	-	1:160	1:10	+	-	-			-	-	2+,1:40
Lucy[2]	1:2560	1:640	1:640	1:1280	+							
Mila	-	-	-	1:10	-	-	-					
Moose			1:640	1:1280	+	1:80	1:10	1:160	1:40	1:80	1:10	2+1:40
Nellie			-	-	-	-	-	-	-	-	-	3+,1:80
Wendy	-	-	-	-	-	1:160	1:80	-	-	-	-	-

[1]All procedures employed *B. abortus* smooth antigen unless otherwise noted.

[2]Brucella suis type 4 isolated at sacrifice.

canines, experimental studies with domestic dogs would be of considerable value in understanding the importance of the disease in the wild canines in which it occurs.

Wolves

The first naturally occurring cases of rangiferine brucellosis in wolves appear to have been simultaneously reported in North America (Neiland, 1970) and Eurasia (Pinigin and Zabrodin, 1970). Further Alaskan serological data on wolves are shown in Table 6. The reactor rate of 7/15 (i.e. 46.6 per cent) is lower than it would have been if only sera from adult animals had been examined. On the other hand a far larger number of sera is probably needed for an accurate measure of prevalence. All of the sera came from the Brooks Range where caribou, the wolf's major prey, are commonly infected.

Pinigin and Zabrodin (1970), using bacteriological culture procedures, found rangiferine brucellosis in 12 of 110 Siberian wolves (10.9 per cent). If they had reported serological results the number of naturally infected animals would no doubt have been much higher. They made no comments on whether any pathological conditions or signs were noted and we have no information on this matter either. Careful experimentation is in order.

Rementsova (1962) reported negative bacteriologic and serologic results for 56 wolves (*Canis lupus*) taken in the SE Betpak-Dala desert. While reindeer do not occur in that area, the saiga (*Saiga tatarica* L.), a common inhabitant of that desert and a prey species for wolves, was known to be occasionally infected (1.2 per cent).

Whether or not rangiferine brucellosis is sometimes an important disease in wolves is an entirely speculative matter at this time. Data on related strains of brucellosis in domestic canines suggest that it may be.

Foxes

Because of reports in the literature on naturally occurring cases of *Brucella abortus* type (?) in wild foxes, I suggested that it was entirely likely that we would eventually find evidence of rangiferine brucellosis in Alaskan foxes in contact with caribou or reindeer (Neiland, 1970). Simultaneously, Pinigin et al. (1970) and Pinigin and Zabrodin (1970) reported naturally occurring rangiferine brucellosis infections in "blue" (?), "silver-black" (?) and arctic (*Alopex lagopus* L.) foxes. The "blue" and "silver-black" foxes were taken on an infected reindeer collective-farm while the arctic foxes were trapped in the wild. Three of 23 of the former and nine of 530 arctic foxes were found infected (Pinigin et al., 1970). Pinigin and Zabrodin (1970) found 12 of 370 arctic foxes infected. Over the past several years we have obtained sera from only 10 red foxes (*Vulpes fulva* L.) and, as seen in Table 6, two of these were serological reactors.

Table 6. The occurrence of brucellosis reactors in wild carnivore populations in Alaska.

Species	Specimen Number	Locality	Titre (*B. abortus* smooth antigen) Complement Fixation		Agglutination	
Wolf	7354	Anaktuvuk Pass	neg.		neg.	
	7355	Anaktuvuk Pass	neg.		neg.	
	7388	Anaktuvuk Pass	1:40		1:20	
	7389	Anaktuvuk Pass	neg.		1:20	
	7390	Anaktuvuk Pass	neg.	Reactor	neg.	Reactor
	7391	Anaktuvuk Pass	neg.	Rate	neg.	Rate
	7392	Anaktuvuk Pass	1:160	(7/15)	1:160	(5/15)
	3160	Anaktuvuk Pass	4+,1:20		neg.	
	3161	Anaktuvuk Pass	4+,1:320		4+,1:160	
	A50,658	Anaktuvuk Pass	2+,1:80		neg.	
	A50,660	Anaktuvuk Pass	3+,1:160		inc., 1:320	
	A50,663	Anaktuvuk Pass	neg.		neg.	
	A50,665	Anaktuvuk Pass	4+,1:640		3+,1:640	
	A50,666	Anaktuvuk Pass	3+,1:10		neg.	
	A50,667	Anaktuvuk Pass	neg.		neg.	
Red Fox	3108	Anaktuvuk Pass	2+,1:20		neg.	
	3128	Anaktuvuk Pass	neg.		neg.	
	3164	Seward Peninsula	neg.		neg.	
	3165	Seward Peninsula	neg.		neg.	
	3166	Seward Peninsula	neg.	Reactor	neg.	Reactor
	3167	Seward Peninsula	neg.	Rate	neg.	Rate
	A50,659	Anaktuvuk Pass	neg.	(2/10)	neg.	(1/10)
	A50,664	Anaktuvuk Pass	neg.		neg.	
	A50,668	Anaktuvuk Pass	neg.		neg.	
	A50,669	Anaktuvuk Pass	4+,1:640		inc., 1:320	
Grizzly Bear	3004	Brooks Range	4+,1:20		2+,1:20	
	3005	Brooks Range	4+,1:20		neg.	
	3006	Brooks Range	---		4+,1:160	
	3007	Brooks Range	4+,1:160		3+,1:80	
	3008	Brooks Range	4+,1:20	Reactor	2+,1:80	Reactor
	3009	Brooks Range	4+,1:320	Rate	3+,1:80	Rate
	3010	Brooks Range	3+,1:40	(15/17)	3+,1:20	(10/17)
	3011	Brooks Range	3+,1:40		3+,1:40	
	3012	Brooks Range	2+,1:160		3+,1:80	
	3013	Brooks Range	4+,1:80		3+,1:20	
	3014	Brooks Range	4+,1:40		neg.	
	3015	Brooks Range	4+,1:80		4+,1:80	
	3016	Brooks Range	neg.		neg.	
	3017	Brooks Range	4+,1:40		2+,1:40	
	3000	Brooks Range	4+,1:80		4+,1:40	
	3002	Brooks Range	4+,1:20		4+,1:20	
	3001	Brooks Range	4+,1:40		2+,1:40	

Pinigin and Zabrodin (1970) isolated the rangiferine organism from the spleen and postpharyngeal, submaxillary and suprascapular lymph nodes. They noted two cases of splenomegaly and two instances of submaxillary lymphadenitis. Otherwise, the infections they studied were apparently unremarkable. However, the studies were originally prompted by "frequent abortions among females on the farm and blood serum changes." It is not clear from their paper whether or not they had concluded that rangiferine brucellosis infections were actually the abortive agent. The closely related "beagle dog" strain, *Brucella suis* type 5, does readily cause abortion in canines.

The effect of rangiferine brucellosis on foxes, wild or domestic, is an open question which ultimately will require experimental studies. However, I think one can safely conclude that rangiferine brucellosis occurs in foxes in Alaska wherever they have access (e.g. wolf kills, aborted fetuses) to infected caribou or reindeer.

Bear

Neiland (1970) suggested the likelihood of finding rangiferine brucellosis in grizzly bears (*Ursus arctos*) in areas where they have the opportunity to feed on infected caribou or reindeer. Pinigin and Zabrodin (1970) make only one statement about bears in their paper, "On the natural nidality of brucellosis." They say, "Bears, wolverines and arctic foxes all feed on the meat of reindeer." I know of no other reference to bears in connection with any of the strains of brucellosis. It may be that cases have been seen in bears in zoos where they no doubt sometimes have been fed infected meat. None of the members of the Brucellosis Research Conference, Conference of Research Workers in Animal Disease know of cases of brucellosis in ursids (personal communications).

Accordingly it seems likely that the serological data on Brooks Range grizzlies in Table 6 are the first evidence that ursids are susceptible under natural conditions to infection by brucellae, in high prevalence of reactors (i.e. 15/17 (*88.3 per cent) by complement fixation test alone or 16/17 (94.1 per cent) by combined CF and agglutination tests). The fact that one bear was clearly a non reactor to both tests suggests that the serologic reactions observed were not simply due to some common peculiarity of bear blood not directly involving brucellar antibodies.

There is no basis for drawing any conclusions about brucellosis in arctic grizzlies other than that it commonly occurred during the summer of 1971. Whether or not the rangiferine or other strains of *Brucella* would be pathogenic in grizzlies or other bears is an entirely speculative matter at this time. We do now know, however, that bears are susceptible to at least one strain. Again, experimental work is needed.

Other Carnivores

Before turning our attentions to non-carnivorous, potential hosts for brucellosis it seems justifiable to consider briefly information on brucellar infections in non-canid and non-ursid carnivores elsewhere.

Rementsova (1962) summarized the data available to that time and noted that positive serologic titres for *Brucella* (sp.?) had been observed in the steppe polecat, *Mustela eversmanni* Less., in Turkemenia by Kharlampovich in 1954. More recently Pinigin and Zabrodin (1970) reported isolating a strain (?) of *Brucella* (probably rangiferine) from one of nine wolverines which they noted "readily frequents domestic deer slaughter areas..." Pritchard et al. (1971) reported on an epizootic of *Brucella abortus* type 1 in ranch mink which had been fed an aborted bovine foetus. The epizootic came to light when mink aborted their litters. Females were reported to remain infected for more than one year after the outbreak.

We have not had a chance to test sera from mustelids in Alaska, but it appears that they are also susceptible to brucellosis, sometimes with serious consequences.

It appears that we now have ample evidence that three of the seven families of carnivores (i.e. canids, ursids and mustelids) are readily susceptible to infection by one of more of the various species and strains of *Brucella*. Natural infections in species of these families can be expected to occur wherever they are in contact with wild or domestic reservoirs of the disease. Whether or not such natural infections seriously affect any susceptible carnivore population remains largely conjectural.

Rodents

A number of rodents have been found naturally infected with various species and strains of *Brucella* (Rementsova, 1962). These include several species of *Citellus*, *Spermophilopsis*, *Rattus*, *Neotoma*, *Microtus*, *Rhombomys*, *Mus* and *Apodemus*. Naturally infected *Citellus* (i.e. susliks) and *Microtus* (i.e. voles) have also been reported by Pinigin and Zabrodin (1970). Korol (1969) claimed to have discovered "self-perpetuating" brucellosis cycles in *Mus* which continued for 4-6 years after infected farm animals were not available as sources of infection for the mice.

Pinigin and Zabrodin (1970) failed to find evidence of infection in 50 Siberian lemmings (*Lemmus obensis*).

Ground squirrels (i.e. *Citellus*, called "susliks" in Siberia) were shown by Rementsova and others (see Rementsova, 1962) to commonly harbor natural infections of *Brucella* which sometimes remained active for up to 740 days. Since this rodent is more or less abundant on the Alaskan ranges of caribou and reindeer herds known to be infected with brucellosis, we have wondered whether the squirrel could serve as a reservoir of infection (Neiland, 1970). Therefore, we arranged to collect sera from squirrels in the Brooks Range where infected caribou roam and also from an infected reindeer range on the Seward Peninsula. Ten sera (Brooks Range) and 15 sera (Seward Peninsula) were negative. More testing needs to be done before the results can be considered representative. It would be particularly interesting to collect squirrel sera on the calving grounds of the Arctic caribou herd. There the squirrels are out of hibernation during calving and must now and then have access to aborted caribou fetuses.

Lagomorphs

The European hare (*Lepus europaeus*) is an important reservoir of *Brucella suis* type 2 which causes serious disease in pigs in Denmark and other parts of Europe (Stableforth and Galloway, 1959). Rementsova (1962) reported finding 22 of 174 Tolai hares (*Lepus tolai*) infected in Russia and noted that hares (and ground squirrels) are particularly susceptible to brucellosis. Kolomakin (1959) claimed that brucellosis (*B. melitensis* type ?) continued to occur in farm dogs on some premises where the disease had been eradicated in sheep, but was enzootic in hares. Dogs from premises without hares were free of the disease. It appears that hares are an effective reservoir of brucellosis and should not be overlooked in this regard.

We have tested sera from 18 hares taken in interior Alaska in areas away from established farms. These have all been negative. A much larger series of samples is needed and it would be of particular interest to examine sera from hares taken in and around the two major pig raising establishments near Fairbanks and Big Delta, respectively. As domestic animal industry continues to expand in Alaska, it seems inevitable that hares will ultimately be exposed to brucellosis. In Alaska they are a well-known reservoir of tularemia which is transmitted in part by the common hare tick, *Haemaphysalis leporis palustris*. There is conclusive evidence that at least certain strains of *Brucella* can be transmitted by ticks, and also mosquitos, (Stableforth and Galloway, 1959). It would appear that "the stage is set" for the disease eventually to become established in Alaskan snowshoe hares.

The arctic hare (*Lepus arcticus*) while seldom abundant does occur in areas where infected caribou and/or reindeer roam. One can only wonder whether or not rangiferine brucellosis might sometimes contribute to the decline following a population peak of this species. Currently there apparently is a peak population of arctic hares on the Seward Peninsula. We examined 35 specimens collected near Shishmaref in the spring, 1971, but failed to note any signs of disease. Unfortunately, serum samples were not taken by the personnel who collected the hares.

2. Leptospirosis.

Over the years we have had a substantial number of sera from caribou and moose tested for leptospiral antibodies. These earlier samples all yielded negative results. More recently, during our Arctic caribou calving ground studies in 1970 and 1971, we have again looked for serologic evidence of leptospiral infections. This time we found a number of reactor animals among those which we collected for our studies on placental retention. These data have already been briefly summarized in Table 3 and are shown in detail in Table 7. Attempts to isolate leptospirae from kidney samples taken from the animals reported in Table 7 were unsuccessful.

There are no reports in the literature of bona fide isolations of leptospirae from Alaskan wildlife. However, an unreported isolation

from rodents has recently been made by personnel of the Arctic Health Research Center (R. L. Rausch, personal communication). Several years ago a captive reindeer in Anchorage, Alaska died from an infection of *Leptospira canicola* apparently contracted through contamination from a stray dog.

I am not aware of any reports on natural infections of *Leptospira* in wild caribou or reindeer. However, antibodies against *Leptospira pomona* and *L. grippotyphosa* have been commonly observed in white-tailed deer (*Odocoileus virginianus*) in the eastern United States (Trainer and Hanson, 1960; Ferris et al., 1961, and others). According to Vander Hoeden (1964) *Leptospira grippotyphosa, L. pomona, L. australis* and *L. bataviae* have been observed in red deer (*Cervus elaphus*), roe deer (*capreolus capreolus*), and fallow deer (*Dama dama*) in Czechoslovakia, and *L. pomona* in sika deer (*Cervus nippon*) in Russia. It appears that cervids are susceptible to various serotypes of leptospirosis but that relatively few attempts have been made to find evidence of the disease in this host group.

Trainer et al. (1961) experimentally produced abortion in four of five pregnant white-tailed deer but saw no symptoms in experimental barren deer. I am not aware of other reports on the pathology of leptospiral infections in cervids.

One can only conclude that cervids are susceptible to infection by several serotypes and such infections sometimes have serious consequences. For the most part, the disease in the wild appears to be one primarily afflicting rodents and a number of serotypes have been described around the world from various species of mice, voles and rats. Various microtines have been found naturally infected, including the tundra vole (*Microtus oeconomus*), but apparently not as yet any lemmings. In any case, in many areas including Alaska tundra voles occupy caribou and reindeer ranges where transmission from infected voles to caribou via contamination of forage could take place. The swampy character of much caribou range during the summer season would certainly favor transmission. Twigg et al. (1968) noted that infection rates in various mice and voles increased markedly with increasing habitat wetness and that infections were restricted to naturally acid soils and waters. These conditions obtain in many areas in Alaska frequented by caribou, particularly the north slope of the Brooks Range and the arctic coastal plains where we collected the animals which registered the leptospiral titres reported in Table 7.

3. Necrobacillosus.

Necrobacillosus is a complex of different disease conditions caused by infection with the bacterium *Sphaerophorus necrophorus*; or *Fusobacterium necrophorum* as it is now called. One of the common forms of necrobacillosus is so-called foot rot, a widespread disease of ruminants. The causative organism is thought to commonly occur as a harmless commensal in the digestive tracts of herbivores and in soil, only causing difficulties in otherwise weakened animals. Damp, swampy terrain is the focus of most foot

Table 7. Occurrence of various leptospiral serotypes in diseased animals collected on the Arctic calving grounds, 1970-71.

Serotype	Results	
	Negative	Reactor[1]
Leptospira pomona	30	--
L. icterohaemorrhagiae	16	14[2]
L. hardjo	30	--
L. autumnalis	16	14[2]
L. hebdomadis	30	--
L. grippotyphosa	30	--
L. pyrogenes	30	--

[1]Screen titres of 1:100 or higher.

[2]Nine of 21 sera collected in 1971 cross-reacted with the two antigens.

rot problems in domestic or wild ruminants.

Foot rot is a more or less common affliction of caribou and/or reindeer throughout their range in the northern hemisphere. There is an abundant literature on this serious disease of reindeer in Eurasia (see Neiland and Dukeminier, 1972) which we briefly considered in our discussion of the warble fly (*Oedemagena tarandi* L.) and its effects on caribou.

Hadwen and Palmer (1922) in their report on "Reindeer in Alaska" claim that, "Foot rot and dermatitis are among the most troublesome of reindeer diseases", and it is also mentioned by Rausch (1953) in his paper on arctic Alaskan mammals. However, Kelsall (1968) in his monograph on Canadian barren-ground caribou does not make any mention of foot rot in caribou or reindeer in Canada. It seems unlikely that the disease is not as common in *Rangifer* spp. in Canada as elsewhere.

Over the years there have been a number of vague reports by guides, bush pilots, geologists, etc. from different areas of Alaska of lame caribou in more or less abundance. During the course of our studies we have had the opportunity to observe the condition in caribou on several occasions. In late August, 1961, we received reports from oil geologists working on the North Slope out of Umiat that they were seeing lame or dead caribou fairly commonly. We investigated the situation and in one day's flying out of Umiat in a helicopter we saw several limping or dead animals. We collected a doe which had a well advanced case. The animal was in very poor condition with a badly swollen hoof. There was a nasal discharge and lung tissue samples showed invasion by an organism morphologically similar to the necrobacillus (Rausch, Arctic Health Research Center, personal communication). There was also a well-developed lesion at the base of the tongue. It seems clear enough that the animal had probably sustained an injury to its hoof which became infected. Ultimately the infection became generalized and it is unlikely the animal would have survived much longer.

Since our first meeting with a lame caribou we have commonly seen "limpers" from the air while surveying calving animals in the Arctic herd. There are two common causes of lameness (i.e. limping) in caribou in Alaska (i.e. necrobacillary foot rot and brucellar bursitis). Together these are responsible for the lame animals we have seen in low abundance (i.e. about 1 per cent prevalence).

More recently we had a report of lame and dying animals near Port Heiden on the Alaska Peninsula. Investigations by several of our game biologists working in that area on moose and bear revealed that the reports were valid. Again only a small percentage (1 per cent or so) of limping caribou were seen in a few hours of flying. One specimen was collected. A large bull in otherwise prime, fall condition was found with a severely swollen hoof. The animal was laying down in a normal position and was so lethargic, presumably from the toxic effects of the infection, that it made no attempt to escape when approached. *Sphaerophorus necrophorus* was isolated from the foot lesion (Miller, Arctic Health Research Center, personal communication).

It appears that we may expect to hear of or see small epizootics of foot rot in caribou from time to time whether or not harassment by warble and bot flies or other dipteran pests plays an important role in the epizootiology of foot rot in Alaska, as it evidently does in Eurasia. In any case, one must assume that this disease condition is another of the normally small costs which Alaskan caribou populations are assessed each year.

4. Virus Diseases.

Relatively little information on virus diseases of *Rangifer* spp. is present in the currently available literature. Rabid caribou or reindeer are seen now and then during epizootics of the disease in arctic foxes and wolves. Foot and mouth disease has been observed in Eurasian reindeer (see Neiland and Dukeminier, 1972). Papillomas, presumably typical infectious viral warts as seen in other cervids, are observed occasionally in Alaska (Neiland, unpublished data) and elsewhere by others. Recently Hoff et al. (1970) reported serological evidence of two arbovirus groups, i.e. Bunyamwera and California encephalitis, in six of 52 Alaskan caribou sera which I had sent to them. They also reported on the isolation of a virus of the Bunyamwera group from a woodland caribou (*Rangifer tarandus terraenovae*) which may have died from a miningeal worm infection on a Wisconsin game preserve.

More is known concerning the occurrence of various arboviruses (particularly California encephalitis virus) in other cervids, principally North American white-tailed deer (Trainer and Hanson, 1969 and others) and moose (Trainer and Hoff, 1971), but also in moose in Sweden (Svedmyr et al., 1965).

We have submitted a substantial number of additional caribou sera for serologic testing and have observed additional California encephalitis titres. A strain of this virus, perhaps the one also infecting caribou, was recently isolated from Alaskan snowshoe hares and rodents (Feltz, Arctic Health Research Center, personal communication).

It seems clear enough that caribou and reindeer are susceptible to and more or less commonly infected with one or more different viruses in Alaska and elsewhere. Viral studies on *Rangifer* spp. are still in their infancy and it is expected that eventually it will be shown that these infectious agents, particularly arthropod borne forms (i.e. arboviruses), are common parasites in reindeer and caribou. Whether or not these will prove to be important pathogens in *Rangifer* spp. remains to be seen.

DISCUSSION

It seems clearly evident that caribou are susceptible to a variety of infections (i.e. microbial) diseases. The comparative lack of data on this facet of rangiferine biology is best explained by the apparent fact that most of the studies on caribou have been carried out by personnel with little or no training and/or interest in wildlife disease. The

biggest challenge, still unmet, is the evaluation of the population effects of chronic parasitic and infectious disease. Kelsall (1968) concludes,

> "With occasional exception, diseases having a virus or bacterial origin are uncommon in wild animal populations. Parasitism, on the other hand, is almost universal (Cowan, 1951). Thorough examination of any wild animal rarely, if ever, fails to disclose at least a few parasites of one or more species, and barren ground caribou are no exception. A small number of parasites dispersed through a population can be considered normal. The same might be found true of disease conditions if more were known of them. It is only when parasites or disease greatly exceed the normal, and cause direct or indirect mortality, that they limit the population in which they are found."

It seems unfortunate that the word "normal" was (and is) used to describe a condition which could have been better described as common. In using the word normal in this way, as is often done by wildlife writers, one is unconsciously led to the conclusion that animals which are commonly, even though lightly, infected by parasitic or infectious disease organisms are in spite of such infections in normal (i.e. healthy) conditions. Only acute infections, whether of parasites or so-called disease organisms, which "cause direct or indirect mortality" are said to "limit the population in which they are found." While this may seem to be a legitimately convenient way to handle the "parasites and diseases problem," and it may well be, strictly from the point of view of contemporary wildlife management, failure to consider the chronic effects of parasitic and infectious disease agents in population studies has no basis in fact from a scientific point of view. Indeed, I am not aware that any North American big game population ever has been studied thoroughly enough to establish what the "normal" burden of parasitic and infectious disease agents might be during a "typical year." After all, disease agents are also dynamic, living things.

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