Alaska Department of Fish and Game
Division of Game
Federal Aid in Wildlife Restoration
Research Progress Report

SEROLOGIC SURVEY FOR MICROBIAL PATHOGENS

by
Randall L. Zarnke
Project W-23-1
Study 18.6
October 1988

STATE OF ALASKA
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PROGRESS REPORT (RESEARCH)

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SUMMARY

Under the study title Serologic Surveys for Microbial Pathogens, 2 distinct projects were conducted. The 2 attached manuscripts constituting the progress report for this study resulted from these projects and will later be submitted for publication in the Journal of Wildlife Diseases.

Serologic tests revealed that exposure to infectious canine hepatitis virus (ICH) is widespread in Alaskan brown bears. In captive situations, ICH is capable of killing black bear cubs. The overall mortality rate for brown bear cubs from birth to 1 year of age is 50%. Some proportion of this mortality may be due to ICH. Thus this virus may play a role in bear population dynamics.

Disease has long been considered one of many factors affecting the regular fluctuations in snowshoe hare population density. Actinobacillus capsulatus is capable of causing direct mortality in hares. Serologic tests reveal that over 40% of the hares in Interior Alaska and northern Alberta have been exposed to this bacterium. A. capsulatus may or may not play a significant role in the population dynamics of hares.

CONTENTS

SEROLOGIC SURVEY FOR INFECTIOUS CANINE HEPATITIS VIRUS IN GRIZZLY BEARS (URSUS ARCTOS) FROM ALASKA, 1973-1986

by

Randall L. Zarnke
Alaska Department of Fish and Game
1300 College Rd., Fairbanks, Alaska 99701 USA

and

Mary Beth Evans
National Veterinary Services Laboratories
U.S. Department of Agriculture
P.O. Box 844, Ames, Iowa 50010 USA

ABSTRACT

Serum antibody prevalence for infectious canine hepatitis in grizzly bears (Ursus arctos) from Alaska during the period 1973 to 86 was 14% (72/526). Prevalence was highest on Kodiak Island at 27% (32/118). Prevalence at individual collection areas did not change significantly over time. No significant sex-specific difference in prevalence was observed. Prevalence was directly related to age. No samples from bears under the age of 2 years contained evidence of previous exposure to ICH.

Key Words: Infectious canine hepatitis, canine adenovirus, grizzly bear, Alaska, serology.

INTRODUCTION

Domestic dogs (Canis familiaris), wild canids and skunks are susceptible to infection by infectious canine hepatitis (ICH), also known as canine adenovirus type 1 (Cabasso, 1981). Equivocal evidence also implicates raccoons (Procyon lotor), ferret (Mustela nigripes) and mink (Mustela vison) (Cabasso, 1981). Clinical signs may range from anorexia and lethargy to ataxia, seizures, paralysis and death (Cabasso, 1981). The first evidence that members of the genus Ursus were susceptible to infection by ICH was provided by the recovery of a captive polar bear (Ursus maritimus) from a prostrate condition following administration of anti-ICH serum (Chaddock and Carlson, 1950). Two subsequent reports of clinical disease in captive black bears (Ursus americanus) were confirmed by virus isolation and histopathology (Pursell et al., 1983 and Collins et al., 1984). A serologic survey in the state of Washington revealed evidence of exposure in one of 33 free-ranging black bears and no such evidence in a single grizzly bear (Ursus arctos) (Foreyt et al., 1986). Serum antibody prevalence to ICH is high in free-ranging wolves in Alaska (Stephenson et al., 1982 and Zarnke and Ballard, 1987). The objective of the current study was to determine if there was any serologic evidence of the exposure of free-ranging black and grizzly bears in Alaska to ICH.

METHODS AND MEANS

Sera for this study were collected by personnel of the Alaska Department of Fish and Game and the U.S. Fish and Wildlife Service during the course of projects dealing with various facets of bear population ecology. Sera were stored frozen until the time of testing. Samples were tested by the serum neutralization method (Appel and Robson, 1973), utilizing the Mirandola strain of ICH. Sera with titers $\geq$ 20 were considered evidence of previous exposure to ICH and will be

hereafter referred to as "positive." All others will be referred to as "negative."

Ages of bears were determined by examining cementum annuli of premolar teeth (Craighead, et al., 1970)

RESULTS

Serum antibody prevalence for ICH in grizzly bears was 14% (72/526) statewide (Table 1). Prevalence exhibited significant differences at various collection sites (Table 1 and Figure 1). Tables 2-4 provide examples that annualized prevalence (based upon year of capture) did not change significantly over time. In areas where overall prevalence was moderate, annualized prevalences were also moderate (Table 3); this was also true for areas where overall prevalence was high (Table 2) or low (Table 4). Totals for Tables 2-4 do not correspond to totals in Table 1 because results for bears that were sampled more than one time were only included once in Table 1 and listed for each appropriate year in Tables 2-4.

No significant sex-specific difference in prevalence was observed. Prevalences for males and females were 31 of 246 (13%) and 43 of 302 (14%), respectively. Prevalence was directly related to age (Table 5 and Figure 2). Of special note was the fact that of the 55 bears under the age of 2 years none had evidence of previous exposure to ICH (Table 5).

Serial samples separated in time by at least a 1-year interval were available for 129 bears; of these, 105 bears were negative at the time of initial and subsequent captures. Fifteen bears were positive at both the time of initial and subsequent captures. Eight bears converted from negative to positive during the interval between initial and subsequent captures. One bear converted from a titer of 280 to negative over a 2-year period. One of 48 (2%) black bears from the Nelchina area were positive.

DISCUSSION

Annualized prevalence rates (Tables 2-4) do not necessarily reflect the precise rate of exposure for any specific year. Rather, they reflect relative trends of exposure over extended periods of time. Thus, taken as a whole, these annualized prevalences should reveal any significant changes in rates of exposure over time. The fact that no such significant changes were revealed (Tables 2-4) leads to the conclusion that the association between ICH and grizzly bears in Alaska is a long-standing one.

The source of ICH for bears is uncertain. Certainly, domestic dogs cannot be ignored in this discussion; however, many, if not most, dogs in Alaska are vaccinated against ICH. Thus the likelihood of dogs shedding virulent ICH in locations where bears may come into contact with the virus seem remote. Previous studies (Stephenson et al., 1982; Zarnke and Ballard, 1987) have concluded that ICH is enzootic in free-ranging wolves in Alaska. If this hypothesis is correct, ICH virus in wolf urine and feces could certainly serve as a source of infection for bears. However, there are no wolves on Kodiak Island, where serum antibody prevalence in bears was highest (Table 1). On the other hand, the red fox (Vulpes vulpes) population density is high on Kodiak. Red foxes are also susceptible to ICH (Cabasso, 1981) and thus could theoretically serve as a source of infection. Alternatively, we hypothesize that ICH has been enzootic in bear populations without the need for continued introduction of the virus from other species.

Considering the epizootiology of ICH in canids, the absence of any significant difference in sex-specific antibody prevalence rates in bears is no surprise. The 2% prevalence in black bears corresponds well with the 3% prevalence reported for Washington state (Foreyt, et al., 1986).

The results of tests on bears for which serial samples were available warrants several comments. The 81% (106/130) that remained negative on retesting corresponds well with the overall rate of 86% negative samples (Table 1) for the entire study. The 15 animals that remained positive for 2 or more tests over periods as long as 4 years suggests that (1) antibody is long-lived and/or (2) reexposure is common. The single bear that changed from positive to negative indicates that antibody decay occurs and perhaps provides indirect evidence for the reexposure hypothesis mentioned above. The 8 bears that converted from negative to positive indicate that active transmission was occurring during the course of this study.

The direct relationship between age and prevalence (Table 5 and Figure 2) suggests that the opportunity for exposure is constant throughout a bear's lifetime. The longer a bear lives, its likelihood for coming into contact with the virus becomes greater. The absence of any evidence of exposure in bears <2 years of age warrants discussion. There are 3 possible explanations: (1) young bears are not exposed to the virus, (2) young bears are not immunocompetent, and (3) if exposed to ICH, young bears develop clinical disease and die as a result of the infection. Many bears spend the first 2 years of life with their sow and thus receive protection from various hazards, including predation by adult boars. However,

it is difficult to imagine some behavioral mechanism by which sows could prevent ICH exposure to their offspring. Thus we reject the first hypothesis. Young bears are fully competent to produce antibody against other disease agents, such as Brucella suis IV (Neiland and Miller, 1981). Thus we also reject the second hypothesis. Clinical ICH is more severe in young canids, compared with adults (Cabasso, 1981). In captive situations, the virus is capable of killing black bear cubs (Pursell et al., 1983). Thus we are unable to reject the third hypothesis. The overall mortality rate for grizzly cubs from birth to 1 year of age is 50% (H. Reynolds, pers. commun.). If the third hypothesis is correct, some proportion of this mortality may be due to ICH. Thus this virus may be a significant limiting factor on bear population growth. A laboratory experiment addressing virulence of ICH in cubs is planned.

ACKNOWLEDGMENTS

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Table 1. Serum antibody prevalence for infectious canine hepatitis virus in grizzly bears (Ursus arctos) from 7 areas of Alaska, 1973-1986.

Area	Prevalence	
Kodiak Island	32/118[a]	(27%)
Alaska Peninsula	3/19	(16%)
Nelchina River Drainage	4/92	(4%)
Alaska Range	11/67	(16%)
Northwest Arctic	3/96	(3%)
Central Arctic	4/39	(10%)
Northeast Arctic	15/98	(15%)
Total	72/526	(14%)

[a] Number positive/number tested.

Table 2. Serum antibody prevalence for infectious canine hepatitis virus in grizzly bears (Ursus arctos) from Kodiak Island, Alaska.

Year	Prevalence
1981	0/1[a] (0%)
1982	16/65 (25%)
1983	0/3 (0%)
1984	8/37 (22%)
1985	9/24 (38%)
1986	5/15 (33%)
TOTAL	38/145 (26%)

[a] Number positive/number tested

Table 3. Serum antibody prevalence for infectious canine hepatitis virus in grizzly bears (Ursus arctos) from the Alaska Mountain Range, Alaska.

Year	Prevalence
1981	0/5[a] (0%)
1982	2/25 (8%)
1983	4/22 (18%)
1984	1/18 (6%)
1985	2/12 (17%)
1986	3/16 (19%)
TOTAL	12/98 (12%)

[a] Number positive/number tested

Table 4. Serum antibody prevalence for infectious canine hepatitis virus in grizzly bears (Ursus arctos) from the Nelchina River Drainage, Alaska.

Year	Prevalence
1978	0/4[a] (0%)
1979	1/41 (2%)
1980	2/25 (8%)
1983	2/28 (7%)
TOTAL	5/98 (5%)

[a] Number positive/number tested

Table 5. Age-specific serum antibody prevalence for infectious canine hepatitis virus in grizzly bears (Ursus arctos) from Alaska.

Age	Prevalence
0	0/8[a] (0%)
1	0/47 (0%)
2	5/46 (11%)
3	5/57 (9%)
4	6/59 (10%)
5	6/58 (10%)
6	6/43 (14%)
7	8/49 (16%)
8	4/51 (8%)
9	5/36 (14%)
10	6/28 (21%)
11	5/24 (21%)
12	10/35 (29%)
13	4/26 (15%)
14	6/29 (21%)
15	4/22 (18%)
16	5/28 (18%)
17	5/16 (31%)
18	3/17 (18%)
19	4/10 (40%)
20+	2/26 (8%)

[a] Number positive/number tested

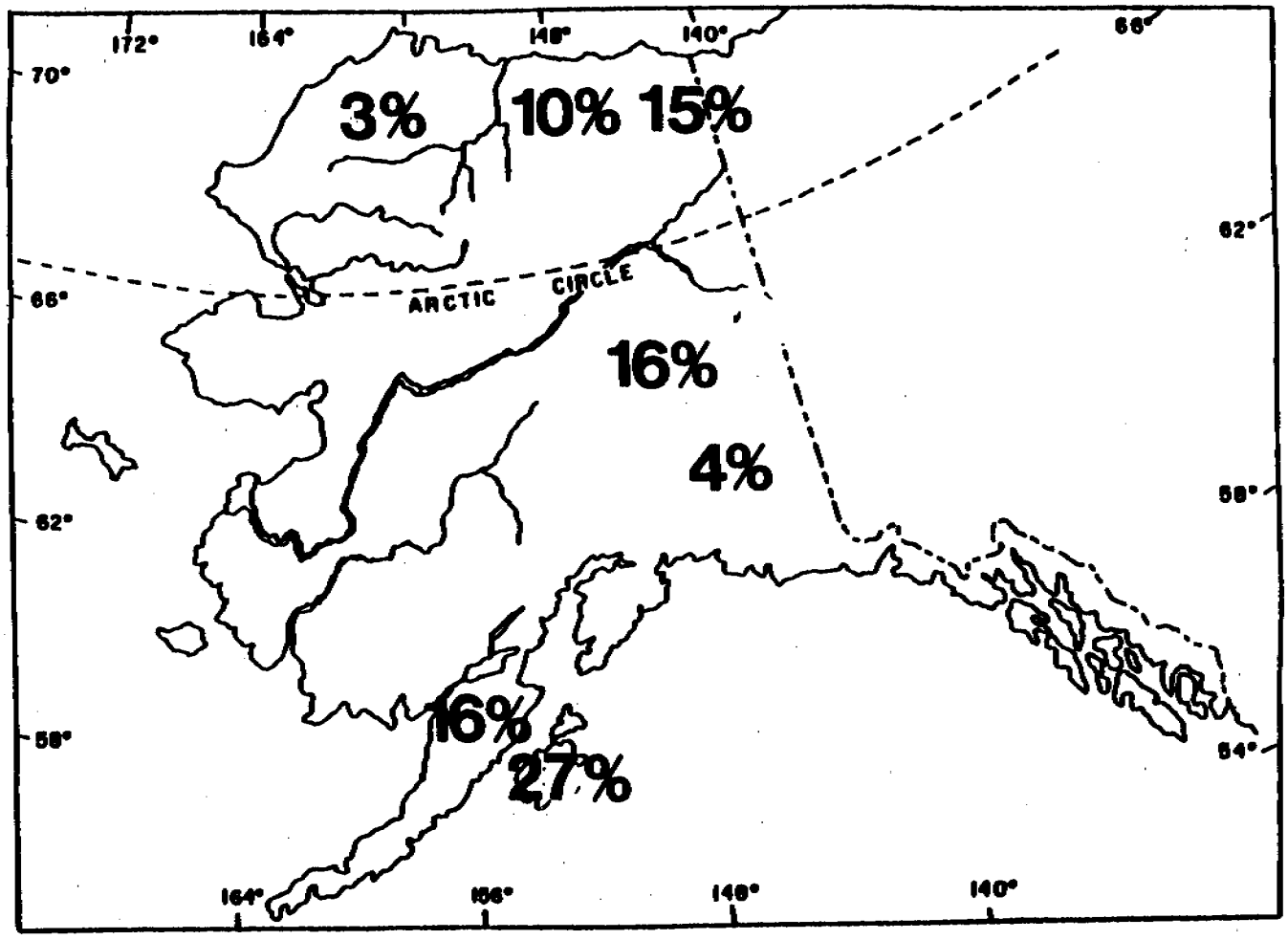

Figure 1. Serum antibody prevalence for infectious canine hepatitis virus in grizzly bears (Ursus arctos) from 7 areas of Alaska, 1973-1986.

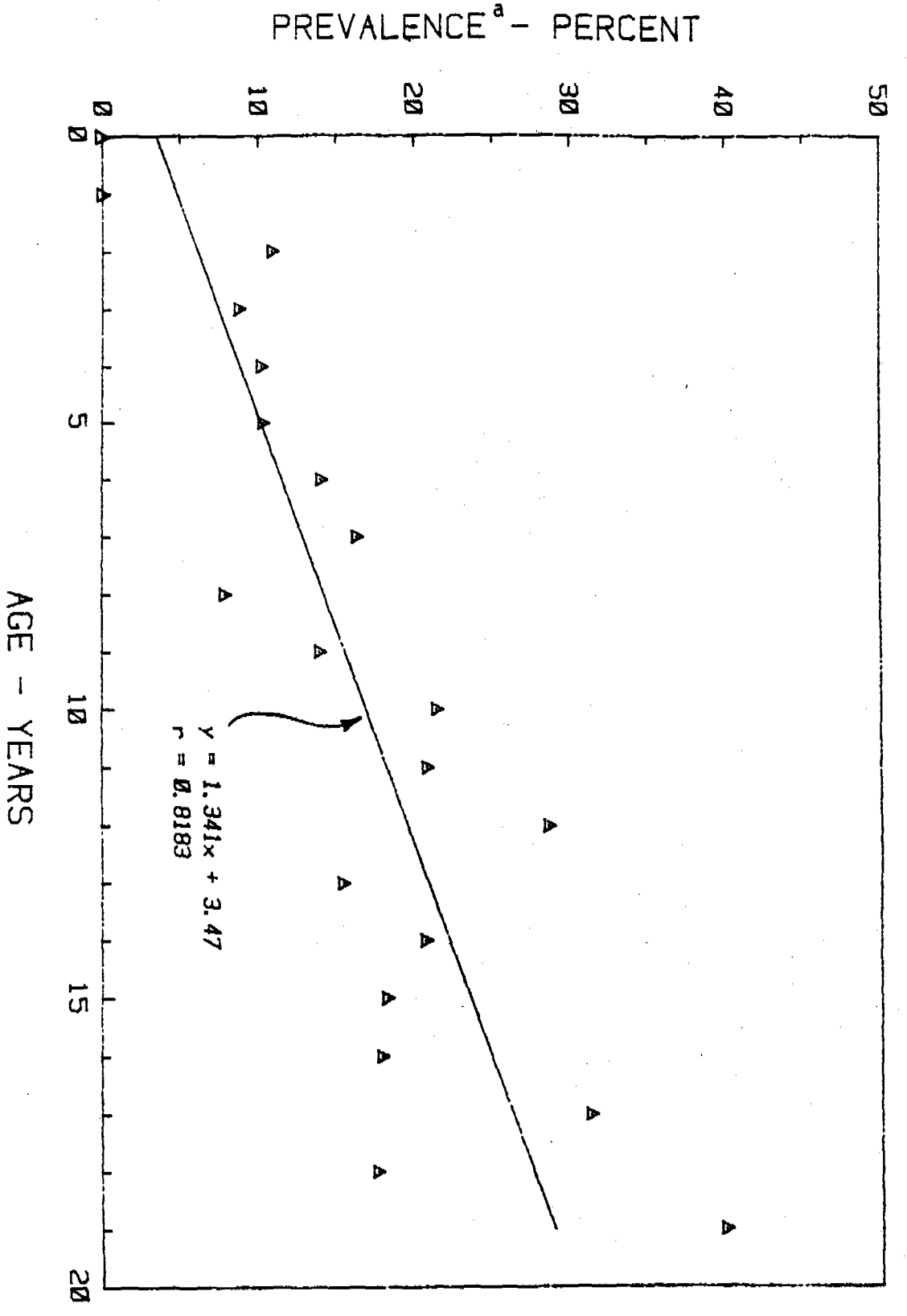

Figure 2. Age-specific serum antibody prevalence for infectious canine hepatitis in grizzly bears (Ursus arctos) from Alaska.

[a] Prevalence = (number positive/number tested) x 100

SEROLOGIC SURVEY FOR *ACTINOBACILLUS CAPSULATUS* IN FREE-RANGING SNOWSHOE HARES (*LEPUS AMERICANUS*) FROM ALASKA AND ALBERTA

by

Randall L. Zarnke
Alaska Department of Fish and Game
1300 College Road
Fairbanks, Alaska 99701, USA

and

Jamie K. Morton
Agricultural and Forestry Equipment Station
University of Alaska
Fairbanks, Alaska 99775, USA

ABSTRACT

Antiserum against Actinobacillus capsulatus was prepared in a domestic rabbit (Oryctolagus cuniculus). A concentrated suspension of formalin-killed A. capsulatus was prepared for use as an antigen. Utilizing these reagents, a plate agglutination method was developed to test sera from free-ranging snowshoe hares (Lepus americanus) captured in Alaska or Alberta. Serum antibody prevalence was 98/239 (41%) in Alaska and 51/111 (46%) in Alberta. Annual prevalence in Alaska peaked in 1981, corresponding to a minor peak in hare population density. Seasonal prevalence peaked in May in Alaska. Prevalence at 1 capture site in Alaska was significantly higher than at 4 other sites. There was no difference in sex-specific prevalence for either Alaska or Alberta.

Key words: Actinobacillus, serology, snowshoe hare, Lepus americanus, Alaska.

INTRODUCTION

Various Actinobacillus spp. are pathogens of domestic animal species (Anonymous, 1974). Members of this genus are capable of causing suppurative or granulomatous lesions in cattle, sheep, horses and swine (Anonymous, 1974). Actinobacillus spp. have been implicated in diseases of free-ranging wildlife species, including cases of pneumonia in both bighorn sheep (Ovis canadensis) and pronghorn (Antilocapra americana), splenic abscesses in pronghorn, and jaw abscesses in elk (Cervus canadensis) from Wyoming (Thorne, 1982).

A. capsulatus was originally isolated from caged Angora and mixed-breed rabbits in Ceylon (now Sri Lanka) (Arseculeratne, 1961, 1962). More recently, a closely related variant of A. capsulatus was isolated from internal organs of free-ranging snowshoe hares (Lepus americanus) in Alaska (Zarnke and Schlater, 1988). When A. capsulatus was injected intravenously into domestic rabbits (Arseculeratne, 1962), it exhibited tissue tropism similar to that observed in naturally infected hares (Zarnke and Schlater, 1988). However, little is known about natural host range, natural means of exposure or other aspects of the epizootiology of A. capsulatus infection in free-ranging wildlife species.

The purposes of the present study were to (1) develop a serologic test to aid in the diagnosis of A. capsulatus exposure and (2) utilize the test to conduct a serologic survey.

MATERIALS AND METHODS

Antiserum was prepared by injecting 2 ml of A. capsulatus suspension containing 2 x 10^9 colony forming units per ml intramuscularly into the thigh of a domestic lab rabbit (Oryctolagus cuniculus) on 2 occasions 3 days apart. Serum samples were collected on days 4, 7, 14, and 20 following the initial injection. (This work was performed at the University of Guelph, Ontario, Canada under the direction of Dr. Soren Rosendal).

For the purpose of antigen production, A. capsulatus was grown on slant tubes of sheep blood agar for 24-36 hours before being harvested. Bacterial growth from the surface of the agar slant was flushed with approximately 2 ml of 1% formalin in phosphate buffered saline (PBS). Harvested suspensions from numerous tubes were pooled and centrifuged at 5000G in a refrigerated centrifuge (Sorval model RC-2B) for 10 minutes. The supernatant fluid was discarded, and the pellet was resuspended in approximately ¼ of the volume of formalin PBS used for the original suspension. The resultant suspension was again centrifuged at 5000G for 10 minutes. The supernatant fluid was discarded, and the pellet was resuspended in a quantity of formalin PBS sufficient to create suspension with an optical density reading of approximately 580 units, as determined by the Klett spectrophotometer (model 800-3). Based upon comparison with standard McFarland nephelometer turbidity solutions, this antigen suspension contained 9 X 10^9 bacteria per ml. Antigen suspensions were tested for sterility before use.

Agglutination reactions were performed on glass slides with 30 individual 3/4-inch inside diameter raised ceramic rings (American Scientific Products, McGaw Park, IL 60085). A standard volume (0.04 ml) of antigen suspension was placed on the slide and mixed with the serum to be tested. A combination of equal volumes (0.04 ml) of both antigen and serum was assigned a dilution value of 1:40, based on comparison of reagent volumes and concentrations in other agglutination tests. This was selected as our screening dilution. After 4 minutes of gentle rocking, the mixture was evaluated for degree of agglutination and assigned a value from negative to 4(+). Sera that exhibited agglutination at the 1:40 dilution or above were considered to provide evidence of previous exposure to A. capsulatus and were referred to as "positive." All others were referred to as "negative."

Sera from free-ranging Alaska hares were collected from 1979-1986 by Alaska Department of Fish and Game personnel or wildlife professionals from other agencies. Alberta hare sera were collected during 1976 by staff and graduate students from the University of Wisconsin-Madison.

A. capsulatus antigen was tested against Francisella tularensis antiserum (Difco Laboratories, P.O. Box 1058, Detroit, Michigan 48232, USA), and A. capsulatus antiserum was tested against F. tularensis antigen (Difco, ibid). All sera from free-ranging hares were also tested against F. tularensis antigen (Difco, ibid). Differences in prevalence related to various parameters were tested for significance by means of the chi-square test (Johnson, 1980).

RESULTS

Results of agglutination tests are presented in Figure 1 and Tables 1-3. The antibody response curve for the known-infected domestic rabbit (Figure 1) fits the standard pattern for animals that are immunologically naive to the disease agent in question. Titers in "positive" snowshoe hares ranged from 40 to 3200. There was an inverse relationship between titer and number of specimens exhibiting that titer. Overall serum antibody prevalence for A. capsulatus in Alaska hares was 98/239 (41%). Corresponding values for Alberta hares were 51/111 (46%). These values were not significantly different ($p<0.25$).

Antibody prevalence for A. capsulatus in snowshoe hares from Alaska exhibited significant variation between years ($p<0.005$) (Table 1). Hare population density in Interior Alaska reached a minor peak in approximately 1981 (R. L. Zarnke, pers. observ.). Antibody prevalence and population density appeared somewhat synchronous during the 1979 to 1984 period (Figure 2). The single year of collection in Alberta did not allow for calculation of corresponding values. Antibody prevalence in hares from both Alberta and Alaska showed a similar seasonal pattern (Figure 3) with significant differences between months ($p<0.005$).

Sample sizes for hares from 5 areas of Alaska were adequate to allow comparison based upon capture location (Table 2). All sites were within 100 miles of Fairbanks. Collection areas were at least 25 miles from each other, except Fort Wainwright and Creamer's Field Refuge, which are separated by approximately 3 miles. There was no direct contact between hares from any of these areas. Prevalences were similar at all locations; however, at Fort Wainwright, the prevalence was significantly higher than those at the other locations ($p<.005$).

There was no significant difference in sex-specific prevalences for Alaska (males 22/60, 37%; females 25/64, 39%) ($p>0.9$), or Alberta (males 26/53, 49%; females 24/57, 42%) ($p>0.25$). No agglutination occurred when A. capsulatus reagents were tested against F. tularensis reagents, nor was

there any evidence of exposure to F. tularensis in any of the free-ranging hare sera.

DISCUSSION

Members of the genus Actinobacillus are serologically cross-reactive (Anonymous, 1973). We are unable to reject the possibility that the antibody that we detected in sera from free-ranging hares was actually produced in response to exposure to an Actinobacillus spp. other than A. capsulatus. However, isolation of A. capsulatus from 3 hares (Zarnke and Schlater, 1988) and the absence of isolation of any other members of the genus support our contention that the serologic reactions reported here indicated exposure to A. capsulatus. Limited testing of sera from selected herbivorous, carnivorous and omnivorous species suggested exposure in a large proportion of the species. Because of (a) the absence of isolation of A. capsulatus from any of these species and (b) the specter of cross-reactivity among the members of the genus Actinobacillus, we choose to not report these results or speculate on their significance.

A comparison of overall prevalences between Alaska (41%) and Alberta (46%) suggested the epizootiology of this disease in hares may be uniform in much of North America. The apparent correlation of annualized antibody prevalence rates and the relative population density of snowshoe hares (Table 1 and Figure 2) may or may not be reflective of the epizootiology of this disease in free-ranging hares. No conclusions were drawn regarding (a) the pattern of seasonal antibody prevalence, (b) differences in prevalence between locations (Table 2), (c) routes of exposure, or (d) modes of transmissions.

Disease has long been considered one of many possible factors in the regular decline in hare density. Effects of A. capsulatus infection on hare populations are difficult to evaluate. Obviously, the bacterium is capable of causing direct mortality (Zarnke and Schlater, 1988). However, the high antibody prevalence reported here indicates that large numbers of hares either (a) suffer no overt disease as a result of exposure, or (b) recover from actual infection. Based upon current data, we are not prepared to speculate whether A. capsulatus infections are (a) a causative factor (of undetermined magnitude) in the population decline of hares, (b) a result of decreased resistance of hares because of other factors that drive the cyclic fluctuations, or (c) unrelated to the population dynamics of hares. Experimental exposure of hares to A. capsulatus under controlled laboratory conditions would provide information of value in assessing the impact of this disease on individual hares. In addition, long-term serologic surveys spanning more than one

complete hare population cycle would provide data that would be helpful in evaluating the relationship (if any) between this disease and the population fluctuations of hares.

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PREPARED BY:

Randall L. Zarnke
Game Biologist III

APPROVED BY:

W. Lewis Pamplin, Jr. by [illegible]
W. Lewis Pamplin, Jr., Director
Division of Game

SUBMITTED BY:

Wayne L. Regelin
Research Coordinator

Donald E. McKnight
Chief of Planning

Table 1. Serum antibody prevalence for *Actinobacillus capsulatus* in snowshoe hares (*Lepus americanus*) captured in Interior Alaska, 1979-1986.

Year	Prevalence
1979	22/58[a] (38%)
1980	24/100 (24%)
1981	9/11 (81%)
1982	22/33 (67%)
1983	12/19 (63%)
1984	7/16 (44%)
Total	96/237 (41%)

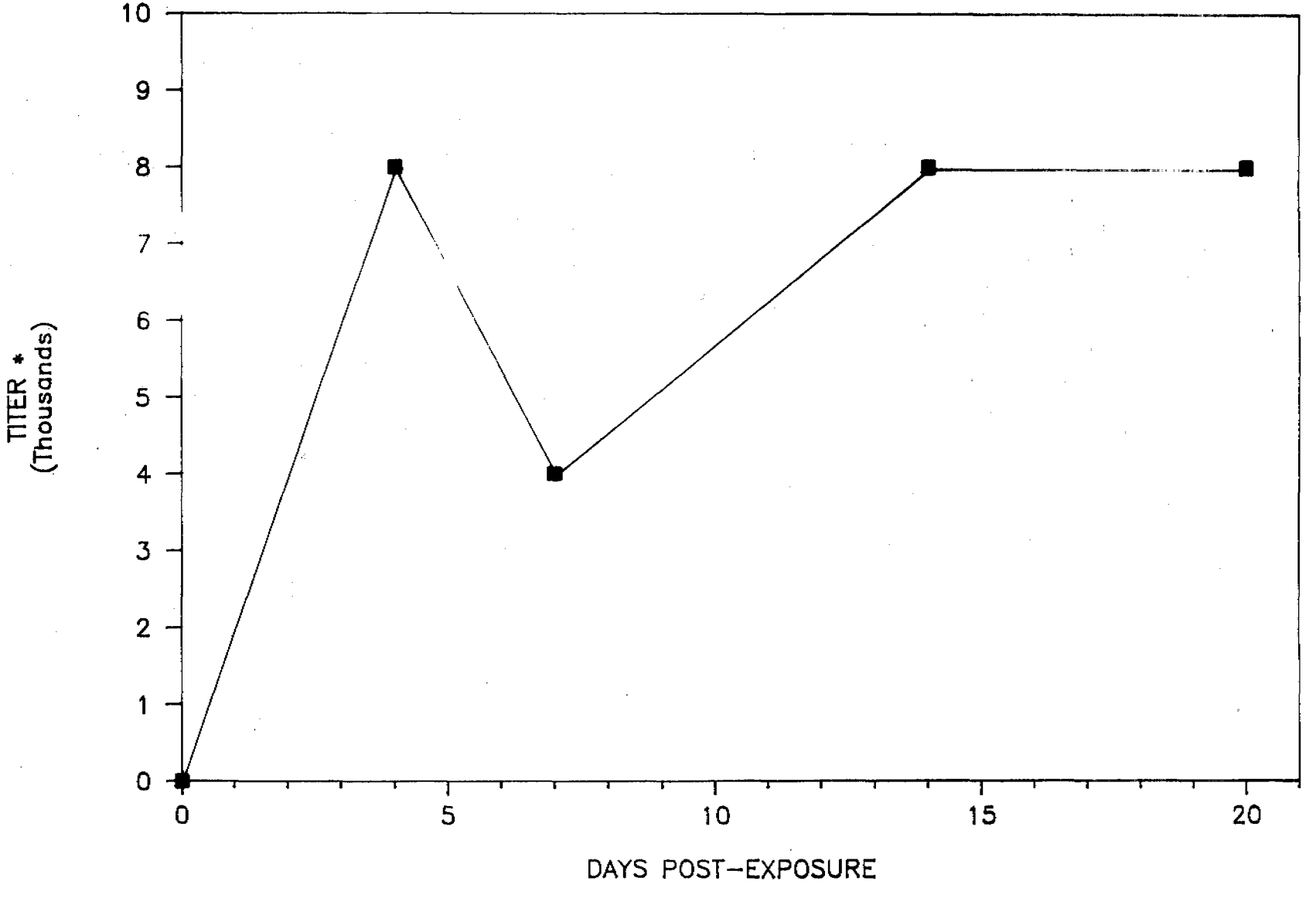
[a] Number positive/Number tested

Table 2. Serum antibody prevalence for *Actinobacillus capsulatus* in snowshoe hares (*Lepus americanus*) captured at 5 locations in Interior Alaska.

Area	Prevalence
Fort Wainwright	11/13a (85%)
Creamer's Field Migratory Waterfowl Refuge	9/18 (50%)
Eielson Air Force Base	20/39 (50%)
Washington Creek	33/84 (39%)
Delta Junction	5/19 (26%)
Total	78/173 (45%)

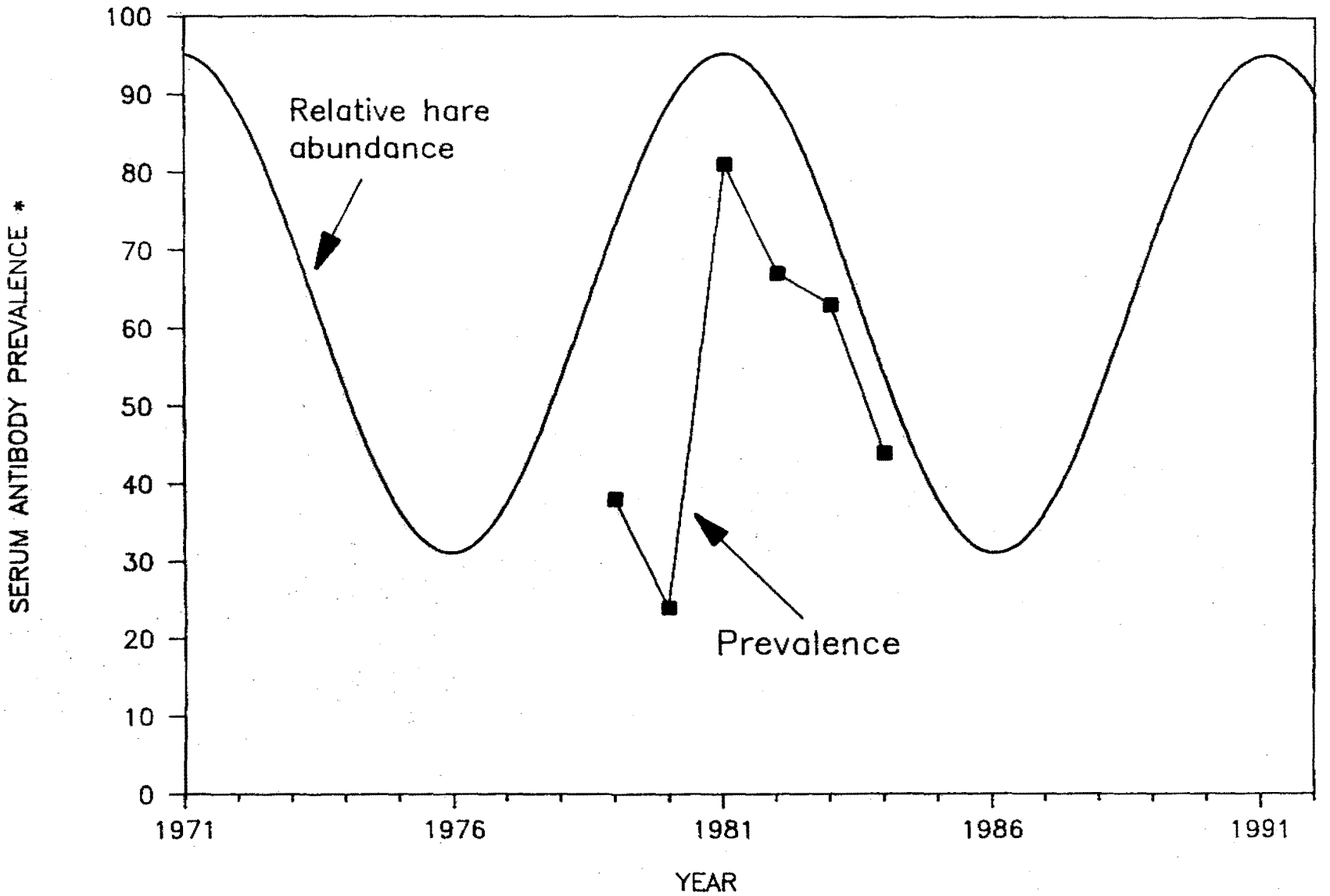
[a] Number positive/number tested

Figure 1. Serum antibody titer in a domestic rabbit (Oryctolagus cuniculi) injected intramuscularly with 2 ml of 2 X 10^9 colony forming units of Actinobacillus capsulatus.



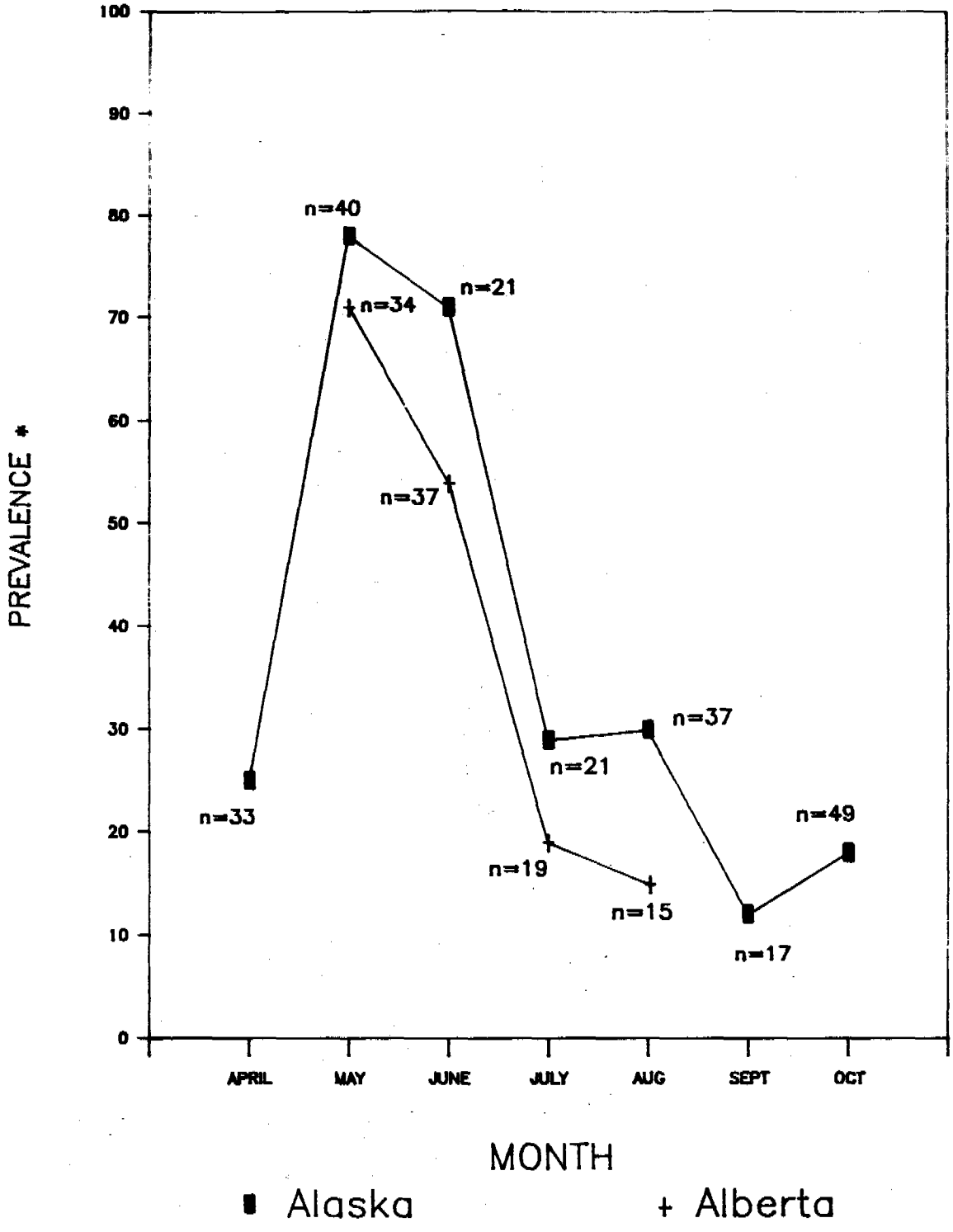
* Inverse of highest serum dilution which exhibited agglutination.

Figure 2. Comparison of serum antibody prevalence for Actinobacillus capsulatus in snowshoe hare (Lepus americanus) captured in Interior Alaska with relative hare abundance.



* (Number positive/number tested) X 100

Figure 3. Serum antibody prevalence for Actinobacillus capsulatus in snowshoe hares (Lepus americanus) captured in Alaska or Alberta.



* (Number positive/number tested) X 100

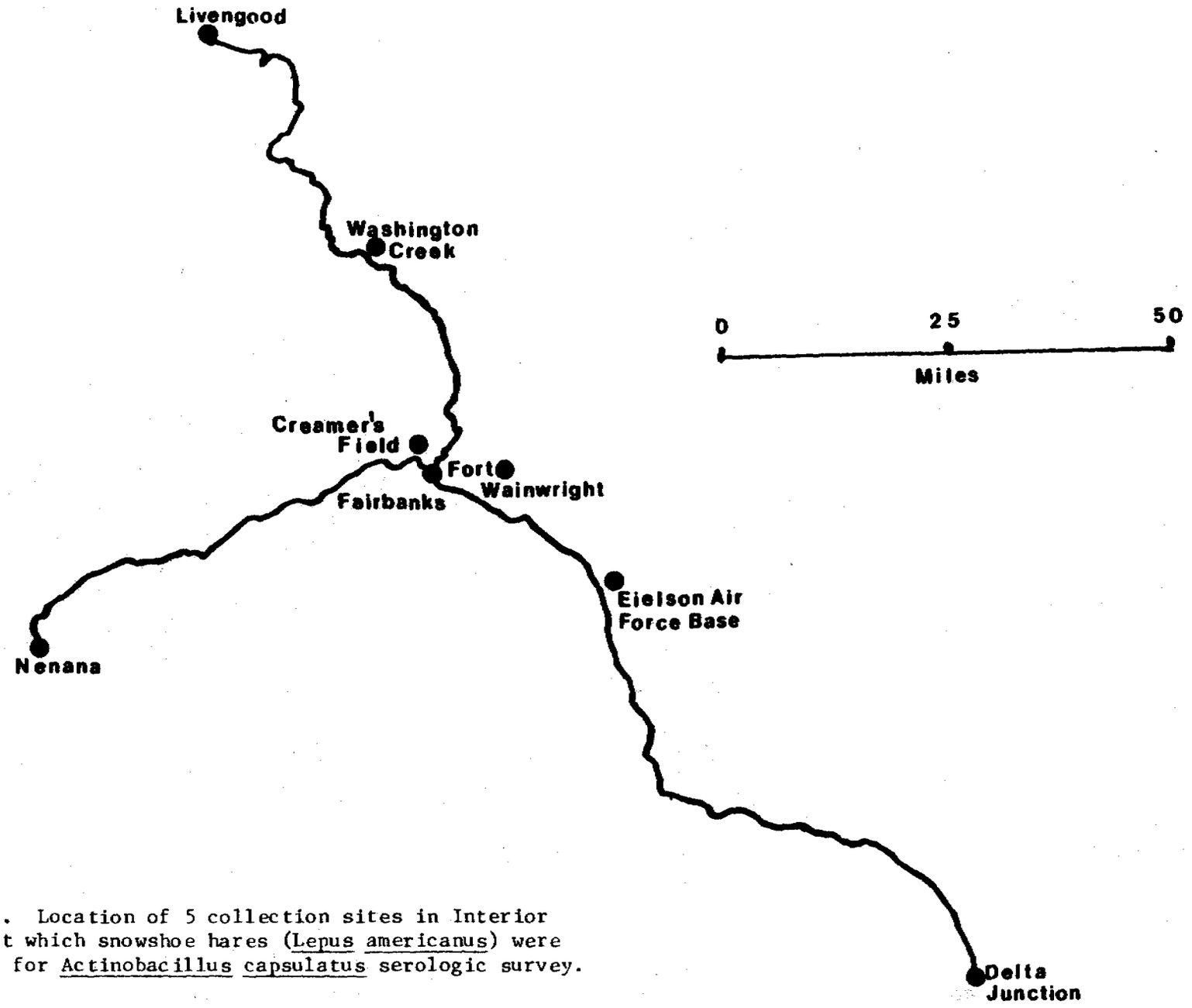


Figure 4. Location of 5 collection sites in Interior Alaska at which snowshoe hares (Lepus americanus) were captured for Actinobacillus capsulatus serologic survey.