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

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

Key words: Infectious canine hepatitis virus, canine adenovirus, grizzly bear, *Ursus arctos*, serologic survey, field study, prevalence.

INTRODUCTION

Domestic dogs, coyotes (*Canis latrans*), red foxes (*Vulpes vulpes*), wolves (*Canis lupus*) and striped skunks (*Mephitis mephitis*) are susceptible to infectious canine hepatitis virus (ICH), also known as canine adenovirus type 1 (Cabasso, 1981). Clinical signs of the disease may range from anorexia and lethargy to ataxia, seizures, paralysis and death (Cabasso, 1981). Equivocal evidence also implicates raccoons (*Procyon lotor*), ferrets (*Mustela putorius*) and mink (*Mustela vison*) (Cabasso, 1981) as susceptible hosts. A captive polar bear cub (*Ursus maritimus*) recovered from an illness following intraperitoneal administration of anti-ICH serum (Chaddock and Carlson, 1950). This incident provided the first reported evidence that members of the genus *Ursus* are susceptible to ICH. Captive black bears (*Ursus americanus*) at two locations showed clinical signs similar to those listed above (Pursell et al., 1983; Collins et al., 1984). Diagnoses of ICH infection in these bears were supported by virus isolation and histopathology in both instances. Rigorous laboratory tests later confirmed the identity of the virus (Whetstone et al., 1988). A serologic survey in 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 ranges from 40% to 100% in free-ranging wolves from various parts of Alaska (Stephenson et al., 1982; Zarnke and Ballard, 1987). The objective of the present study was to determine if there was serologic evidence of exposure of free-ranging grizzly bears to ICH in Alaska.

MATERIALS AND METHODS

Sera were collected by personnel of the Alaska Department of Fish and Game (Juneau, Alaska 99802, USA) and the U.S. Fish and Wildlife Service (King Salmon, Alaska 99613, USA) during population ecology studies. Localities for capture sites are shown in Figure 1. Sera were stored temporarily at −12 C and at −40 to −46 C for up to 13 yr until the time of testing. Samples were tested by a serum microneutralization method (Appel and Robson, 1973) utilizing the Mirandola strain of ICH. A threshold titer of 1:20 was selected based upon reported antibody response in naturally exposed black bears (Collins et al., 1984). Titers ≥20 were considered evidence of previous exposure to ICH and are hereafter referred to as "positive." All others are referred to as "negative."

Ages of bears were determined by examining cementum annuli of premolar teeth (Craighead et al., 1970). Bear population densities were reported previously (Reynolds, 1976; Miller and Ballard, 1982; Reynolds and Hechtel, 1984; Reynolds et al., 1987; Ballard et al., 1988).

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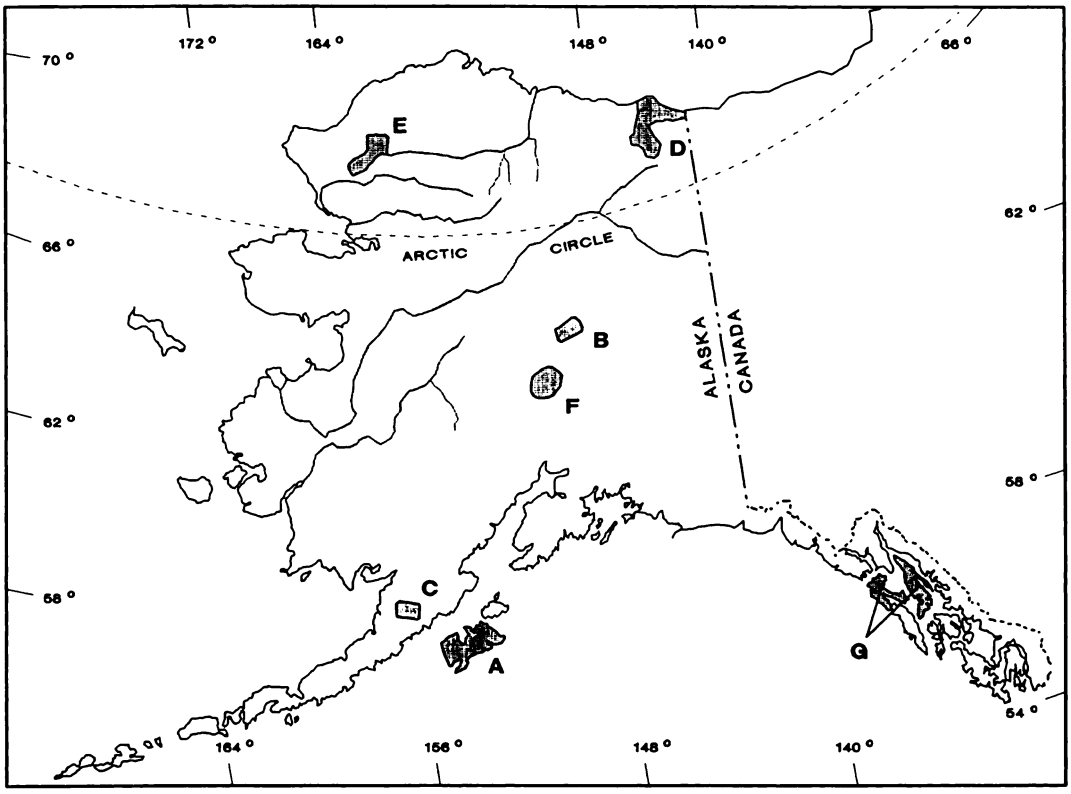


FIGURE 1. Location of study sites where grizzly bear (*Ursus arctos*) sera were collected for infectious canine hepatitis virus survey: A, Kodiak Island; B, Central Alaska Range; C, Alaska Peninsula; D, Northeast Arctic; E, Northwest Arctic; F, Lower Susitna River Drainage; G, Southeast Alaska.

Data were analyzed using a logit loglinear model (Agresti and Yang, 1987) to test for the effect of age, gender and geographic location on the prevalence of ICH. Many bears were sampled more than once. Only the first observation for each bear was used. Test results from areas with inadequate sample size or having incomplete age or gender data were not considered. For purposes of this analysis, bears were classified into four age categories: 0 to 1.5, 2.5 to 5.5, 6.5 to 11.5 and >12.5 yr. In a logit loglinear approach, a complete model including terms for each of the independent variables (age, gender and location) and their interactions was specified. A backward selection process using $a = 0.10$ was employed to remove terms from the model until the most parsimonious model that explained the data was identified.

RESULTS

Serum antibody prevalence for ICH in grizzly bears during the period 1973 to 1987 was 12% (90 of 725) statewide (Table 1). Antibody prevalence was 10% (32 of 317) for males and 14% (58 of 408) for females. Based upon loglinear analysis, neither gender nor interactions among any of the three parameters (age, gender and location) were significantly related to ICH prevalence ($P > 0.10$). The two most important parameters in the loglinear model were area and age, respectively. Prevalence ranged from a low of 0% in southeastern Alaska to a high of 27% on Kodiak Island, although there was no apparent geographic pattern of prevalence values (Table 1). Prevalence was positively correlated to age (Fig. 2). None of the 55 bears <2-yr-old had evidence of previous exposure to ICH.

Positive titers ranged from 1:20 to >1:2,759. Frequency distribution of positive titers exhibited an inverse relationship; low

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TABLE 1. Serum antibody prevalence for infectious canine hepatitis virus in grizzly bears (*Ursus arctos*) from seven areas of Alaska, 1973–1987.

Site[a]	Sample collection period	Prevalence		Density
		Number examined/ number infected	%	
Kodiak Island	1981–1986	37/127[b]	29	39[c]
Central Alaska Range	1981–1987	13/72	18	2
Alaska Peninsula	1986	3/19	16	6
Northeast Arctic	1973–1975			
	1982–1987	22/174	13	1
Northwest Arctic	1977–1987	10/179	6	2
Lower Susitna River drainage	1978–1987	5/130	4	2
Southeast Alaska	1984–1987	0/24	0	39
Total		90/725	12	

[a] See Figure 1 for localities of study sites.
[b] Number positive/number tested.
[c] Number bears/100 km².

titers were common and high titers were rare. Prevalences calculated based upon year of capture were relatively stable at individual study areas. For example, annualized prevalences for the lower Susitna River drainage ranged from 0% to 8% over a 6 yr period, whereas corresponding values for Kodiak Island ranged from 22% to 38% over a 5 yr period. Prevalence was not correlated to bear density (Table 1).

Serial samples (≥ 1 yr between samples) were available for 172 bears. Of these, 139 bears were negative at the time of initial and subsequent captures. Fourteen bears were positive at initial and subsqunt captures. Fourteen bears ranging in age for 3.5 yr to 18.5 yr converted from negative to positive during the interval between captures. Five bears ranging in age from 8.5 yr to 20.5 yr converted from positive to negative.

DISCUSSION

We have no explanation for the differences in prevalence among geographic areas (Table 1). The relatively stable annualized prevalences at individual study areas suggest that the association between ICH and grizzly bears in Alaska is not a recent phenomenon. A dramatic increase in prevalence over time might suggest the introduction of a new disease agent into a susceptible host population. Such was not the case here. Based upon accepted methods of transmission and observations in other species, the absence of any significant difference in sex-specific antibody prevalence rates in bears was not unexpected.

The source of ICH for bears is uncertain. Infected canids shed ICH in saliva, urine and feces (Cabasso, 1981). Transmission occurs when susceptible animals contact contaminated excreta (Cabasso, 1981). Serologic tests revealed 11 of 12 (88%) arctic foxes (*Alopex lagopus*) captured near Prudhoe Bay in 1982 had been exposed to ICH (R. L. Zarnke, unpubl. data). Red fox (*Vulpes vulpes*) are also susceptible to this infection (Cabasso, 1981). Previous studies (Stephenson et al., 1982; Zarnke and Ballard, 1987) have concluded that ICH is enzootic in free-ranging wolves in Alaska. Presumably, ICH-contaminated urine and feces from these wild canids could serve as a source of infection for bears in areas of mainland Alaska where bear and canid populations are sympatric.

There are no wolves on Kodiak Island where ICH serum antibody prevalence in

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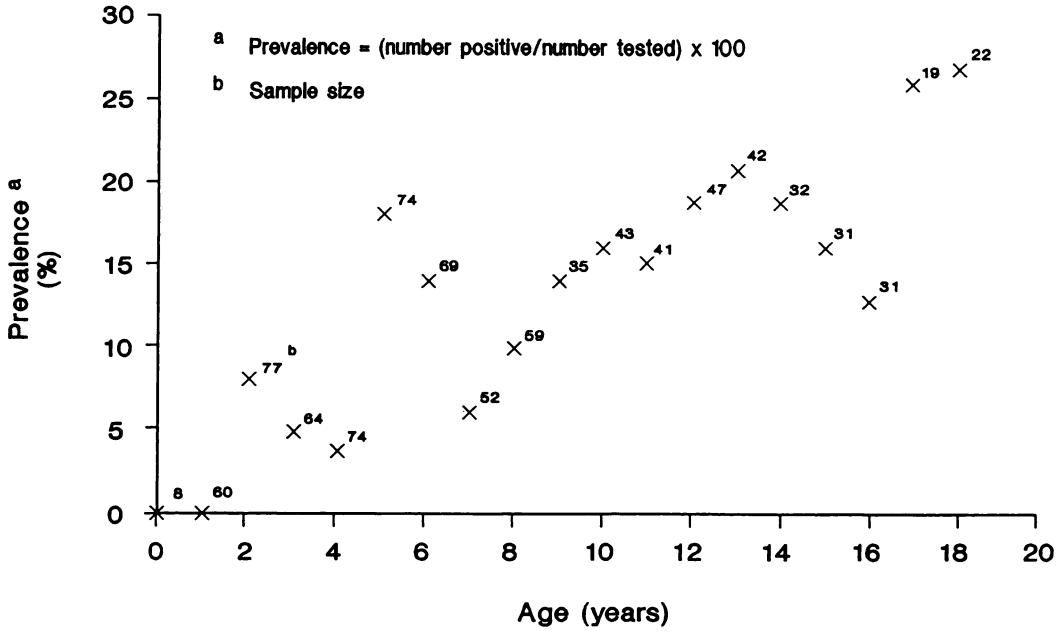


FIGURE 2. Age-specific serum antibody prevalence for infectious canine hepatitis virus in grizzly bears (*Ursus arctos*) from Alaska.

bears was highest (Table 1). However, red fox population density is high. Neither wolves nor foxes are found on Admiralty or Chichagof Islands in southeastern Alaska where prevalence was 0% (Table 1). We speculate that free-ranging canids are necessary for the introduction and perhaps periodic re-introduction of ICH in grizzly bear populations in Alaska.

Test results on serial samples revealed that 81% (139 of 172) which were negative on initial sampling remained negative on subsequent sampling. This corresponds well with the overall rate of 88% negative samples (Table 1) for the entire study. The 14 animals which remained positive for two or more tests over as long as a 6 yr period suggests that (1) antibody is long-lived and/or (2) re-exposure is common. The five bears which changed from positive to negative suggest that antibody decline does occur and perhaps provides indirect evidence for the re-exposure hypothesis mentioned above. The largest fall in titer among these five bears was a decrease from 1:61 to negative over a 2 yr period. The remainder were declines from in the 1:30's to negative. These low positive titers may have represented false positive values. The 14 bears which converted from negative to positive indicate that active transmission occurred during the study.

The direct relationship between age and prevalence (Fig. 2) suggests that the opportunity for exposure is present throughout a bear's lifetime. The longer a bear lives, the greater its likelihood of having contact with the virus.

There was no evidence of exposure in bears <2-yr-old. Possible explanations include (1) young bears are not exposed to the virus, (2) passively acquired maternal antibody interferes with active antibody production in young bears, (3) young bears are incapable of antibody production, (4) antibody titers only reach our threshold value of 20 following repeated exposures over a period of at least 24 mo or (5) young bears which are exposed to ICH develop clinical disease and die as a result of the infection. The first hypothesis is unlikely. Many bears spend the first 2 yr of life with their mother and thus receive protection from various hazards. However, it is un-

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likely that some behavioral mechanism of mother bears or their offspring could preclude young bears from exposure to ICH for 2 yr. The second hypothesis is unacceptable. In dogs, even high levels of maternal antibody are not protective against exposure to ICH (Wright et al., 1974). If maternal antibodies in young bears were at a high enough level to be protective, they would have been detected by the test system utilized in this study (T. O. Bunn, pers. comm.). The third hypothesis is even more unlikely. Six-mo-old black bears had high titers approximately 5 wk postexposure (Collins et al., 1984). There is no reason to believe that grizzly cubs would react differently. Similar arguments pertain to the fourth hypothesis. There is indirect support for the fifth hypothesis: (1) clinical ICH infection is more severe in young canids, as compared with adults (Cabasso, 1981); (2) in captive situations, the virus is capable of killing black bear cubs (Pursell et al., 1983; Collins et al., 1984); and (3) brown bear (*Ursus arctos*) cubs at the Budapest Zoo died as a result of ICH whereas no signs of disease were observed in adult bears (Kapp and Lehoczki, 1966).

The mortality rate for grizzly bear cubs during their first year of life can reach 45% in some areas of Alaska (Reynolds and Hechtel, 1984). If the fifth hypothesis is correct, some proportion of this mortality may be due to ICH. Thus, this virus may be a factor affecting bear population dynamics.

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