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SEROLOGIC SURVEY OF *TOXOPLASMA GONDII* IN GRIZZLY BEARS (*URSUS ARCTOS*) AND BLACK BEARS (*URSUS AMERICANUS*), FROM ALASKA, 1988 TO 1991

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ABSTRACT: We tested 644 serum samples from 480 grizzly bears and 40 black bears from Alaska (USA), collected between 1988 and 1991, for *Toxoplasma gondii* antibodies, using a commercially available latex agglutination test (LAT). A titer ≥64 was considered positive. Serum antibody prevalence for *T. gondii* in grizzly bears (*Ursus arctos*) was 18% (87 of 480). Prevalence ranged from 9% (seven of 77) on Kodiak Island to 28% (15 of 54) in northern Alaska. Prevalence was directly correlated to age. No grizzly bears <2-year-old had *T. gondii* antibody. High antibody titers were found mainly in grizzly bears captured north of the Arctic Circle. Antibody prevalence in black bears (*Ursus americanus*) from Interior Alaska was 15% (six of 40), similar to the prevalence in grizzly bears from the same area (13%; five of 40).

Key words: Toxoplasmosis, *Toxoplasma gondii*, grizzly bear, *Ursus arctos*, black bear, *Ursus americanus*, serologic survey, prevalence, Alaska.

INTRODUCTION

There are an estimated 30,000 grizzly bears (*Ursus arctos*) in Alaska (USA) (Hummel and Pettigrew, 1991) and less than 1,000 in the other 48 continental U.S. states. Populations of black bears (*Ursus americanus*) were estimated to be between 300,000 and 400,000 in the United States including 140,000 in Alaska (Hummel and Pettigrew, 1991). As predators and scavengers, bears might come in contact with zoonotic disease agents such as *Toxoplasma gondii, Mycobacterium tuberculosis, Coxiella burnetii, Yersinia pestis, Leptospira* spp., *Trichinella* spp., *Brucella* spp., and *Francisella tularensis* (Ruppaner et al., 1982). Toxoplasmosis is a protozoan zoonosis found throughout the world (Dreesen, 1990). Infection by the intracellular parasite, *T. gondii*, was reported to be prevalent in many warm blooded species, including humans (Dubey and Beattie, 1988). The role of wildlife in the epidemiology of the disease has not yet been fully investigated. Wildlife species could serve as intermediate reservoir hosts for the organism (Dubey and Beattie, 1988). Based on serologic studies, many wildlife species (Franti et al., 1975; Riemann et al., 1975) have been implicated, including many furbearing species (Dreesen, 1990) in the epidemiology of toxoplasmosis in the United States. Black bears (*Ursus americanus*) with serologic evidence of exposure were reported from Ontario, Canada (Quinn et al., 1976), as well as Florida (Burridge et al., 1979), Idaho (Binninger et al., 1980), California (Ruppaner et al., 1982), and Pennsylvania of the USA (Briscoe et al., 1993). None of 21 polar bear (*Ursus maritimus*) sera collected in an area 250 km north of Jamesonland, Greenland, had *Toxoplasma* spp. antibodies (Clausen and Hjort, 1986). Serologic evidence of exposure has been reported in one captive polar bear (Kiupel et al., 1987). The only known clinical cases of toxoplasmosis in bears were reported in young captive Kodiak bears (*Ursus arctos middendorffi*) kept at the Rostock Zoological garden in Germany (Kiupel et al., 1987). More recently, *T. gondii* was isolated from five hunter-killed black bears from Pennsylvania (Dubey et al., 1994), and during the hunting season of 1993, viable *T. gondii* were isolated from 10 of 28 black bears from Pennsylvania (Dubey et al., 1995).

Reports of *Toxoplasma* spp. exposure in animals and humans in Alaska are limited.

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Serologic evidence has been reported for indigenous Alaska natives (Peterson et al., 1974), and in moose (*Alces alces*) (Kocan et al., 1986). There also was histologic evidence in a harbor seal (*Phoca vitulina richardii*) (VanPelt and Dieterich, 1973). Our objective was to determine the antibody prevalence of *T. gondii* in free-ranging grizzly bears (*Ursus arctos horribilis*) and black bears from Alaska.

MATERIAL AND METHODS

Personnel of the Alaska Department of Fish and Game and the U.S. Fish and Wildlife Service captured 480 grizzly bears and 40 black bears in the course of performing population ecology studies. Some bears were captured more than once; 644 serum samples were available for testing. Sampling was opportunistic. Seventy-six blood samples were collected from 40 black bears between 1988 and 1991 in Interior Alaska on the Tanana Flats, south of Fairbanks (63°N to 66°N, 145°W to 150°W) (Fig. 1). The 568 grizzly bear blood samples were collected between 1988 and 1991 from eight different geographical areas (Fig. 1). In southern Alaska, 79 serum samples were collected on Kodiak Island (57°N to 58°N, 152°W to 155°W), and 86 samples came from the Alaska Peninsula (Katmai Coast and Black Lake) (56°N to 59°N, 153°W to 159°W). In Interior Alaska, 53 serum samples were collected in the Tanana Flats, Denali Park and Fairbanks areas (64°N to 66°N, 147°W to 153°W). In Western Alaska, 40 serum samples were collected from Seward Peninsula (64°30′N to 66°30′N, 162°W to 168°W) and 99 samples from Noatak river drainage (67°45′ to 68°30′N, 156°W to 162°W). In Northern Alaska, serum samples were collected from three different areas, 133 samples in northwestern Alaska (68°30′N to 71°N, 159°W to 163°W), six samples in north central Alaska (Prudhoe Bay, Dead Horse; 70°20′N, 148°W), and 72 samples from northeastern Alaska (68°30′N to 70°30′N 141°W to 147°W).

Blood samples were collected in glass tubes by femoral, saphenous or cephalic venipuncture. Serum was separated by centrifugation and stored at −20 C until tested. Blood samples were collected from 25 (63%) of the 40 black bears more than once over the four-year survey: two bears had blood samples collected four times, seven bears had blood samples collected three times and 16 bears had blood samples collected twice. Among the 480 grizzly bears, blood samples were obtained three times from nine bears and twice from 68 bears.

Serum samples were tested between April and June 1992 by latex agglutination (LAT) using the commercial kit Toxotest-MT "Eiken" (Eiken Chemical Co, Ltd., Tokyo, Japan). A titer ≥64 was considered positive, in accordance with the manufacturer's instructions. Titers of 16 and 32 were reported as weakly positive.

Ages were estimated by examining cementum annuli of premolar teeth for black bears (Stoneberg and Jonkel, 1966) and grizzly bears (Craighead et al., 1970). For this analysis, black bears were classified in four inclusive age groups: 0 to 2 yr old, 3 to 4 yr old, 5 to 7 yr old, and ≥8 yr old. Grizzly bears were classified into five inclusive age groups (MacGuire and Servheen, 1992): 0.5 to 2 yr (young bears), 2.5 to 4 yr (end of puberty), 4.5 to 8 yr (reproductively mature bears), 8.5 to 12 yr (prime reproductive capability), and ≥13 yr (old bears). Grizzly bears from the Seward Peninsula and McKinley Park all were reported as adults (≥4.5 years).

Year of collection is not necessarily indicative of year of exposure. However, in this study, test results were assigned to year of collection.

Data concerning species, sex, age, geographical location at time of blood collection, and date of blood collection were analyzed using Epi Info version 5.01 (Dean et al., 1990). Frequency distributions were obtained and chi-square tests of homogeneity for 2 × 2 contingency tables were used to evaluate the statistical significance of any associations.

RESULTS

Serum antibody prevalence for *T. gondii* in grizzly bears from 1988 to 1991 was 18% (87 of 480) (Table 1). Prevalence ranged from 9% on Kodiak Island to 28% for northern Alaska (Table 1). There was a distinct geographic pattern of increasing antibody prevalence ($P = 0.04$), as prevalence increased gradually from southern Alaska (14%) and Interior Alaska (13%) to western Alaska (17%) and northern Alaska (25%). Prevalence in males (34 of 183, 19%) was similar to the prevalence in females (53 of 297, 18%).

In black bears, *T. gondii* antibody prevalence was 15% (six of 40) (Table 1). This prevalence was similar to the prevalence (five of 40, 13%) observed in grizzly bears captured in the same geographical area (Interior Alaska). Prevalence in males (two of 18, 11%) was lower than in females (four of 22, 18%).

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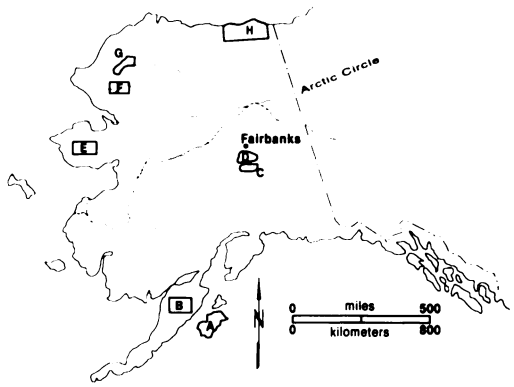


FIGURE 1. Location of study sites where grizzly bear (*Ursus arctos*) and black bear (*Ursus americanus*) sera were collected for toxoplasmosis survey. For grizzly bears, A: Kodiak Island; B: Alaska Peninsula; C and D: Interior Alaska; E: Seward Peninsula; F: Noatak River drainage; G: Western Arctic; H: Eastern Arctic. For black bears: D: Interior Alaska.

High *Toxoplasma* spp. antibody titers (≥1,024) were found mainly in grizzly bears. Eight (73%) of 11 of these bears were captured north of the Arctic Circle (Fig. 2). One black bear from Interior Alaska had an antibody titer of 4,096. Twenty-nine grizzly bears (6%) and one black bear (2.5%) had only weakly positive antibody titers. Overall, 5.4% of the grizzly bears serum samples (31 of 568) and 4% (three of 76) of the black bears serum samples had weakly positive antibody titers.

Year-specific prevalence in grizzly bears decreased from 22% (45 of 206) in 1988 to 16% in 1989, 14% in 1990 and 15% in 1991. The highest year- and location-specific prevalence (38%) was observed in 1988 in the western Arctic (Table 2).

Age of the bears at the time of bleeding was determined for 600 (93%) of the 644 blood samples collected; 44 grizzly bears were reported only as adults. Sixty-five (93%) of 70 young grizzly bears (0.5 to 2 years old) were from interior Alaska (n = 31 bears) or the Noatak River drainage (n = 34 bears).

Antibody prevalence in black bears varied from 4% (one of 26) in the 0 to 2 yr-old group to 10% (two of 20) in the 2.5 to 4 yr-old group, 18% (three of 17) in the 4.5 to 8 yr-old group, 11% (one of nine) in the 8.5 to 12 yr-old group and 25% (one of four) in the ≥13 yr-old group. There was no significant difference in antibody prevalence between adults and young black bears.

Mean (±SE) age of positive grizzly bears was 12 (±0.6) yr whereas the mean age of negative grizzly bears was 8 (±0.3) yr ($P < 0.05$). Adult grizzly bears were four times more likely to have been exposed than juveniles (relative risk (RR) = 4.06, 95% confidence interval (CI) = 2.01 to 8.22). *Toxoplasma gondii* antibody prevalence increased with age, from 0% (zero of 60) in the 0 to 2 yr-old group to 8% (eight of 97)

TABLE 1. Antibody prevalence of *Toxoplasma gondii* in grizzly bears (*Ursus arctos horribilis*) and black bears (*Ursus americanus*) from Alaska by geographical origin and by sex, 1988 to 1991.

Species	Site[a]	Prevalence		
		Male	Female	Total
U. arctos	A	0/10[b] (0)	7/67 (10)	7/77 (9)
	B	6/36 (17)	10/50 (20)	16/86 (19)
	C, D	4/17 (23)	1/23 (4)	5/40 (13)
	E	2/16 (12)	7/24 (29)	9/40 (23)
	F	3/34 (9)	9/53 (17)	12/87 (14)
	G	10/42 (24)	13/54 (24)	23/96 (24)
	H	9/28 (32)	6/26 (23)	15/54 (28)
U. americanus	C, D	2/18 (11)	4/22 (18)	6/40 (15)
Total		36/201 (18)	57/319 (18)	93/520 (18)

[a] Location of site of capture given in Figure 1.
[b] Number positive/number tested (percent positive).

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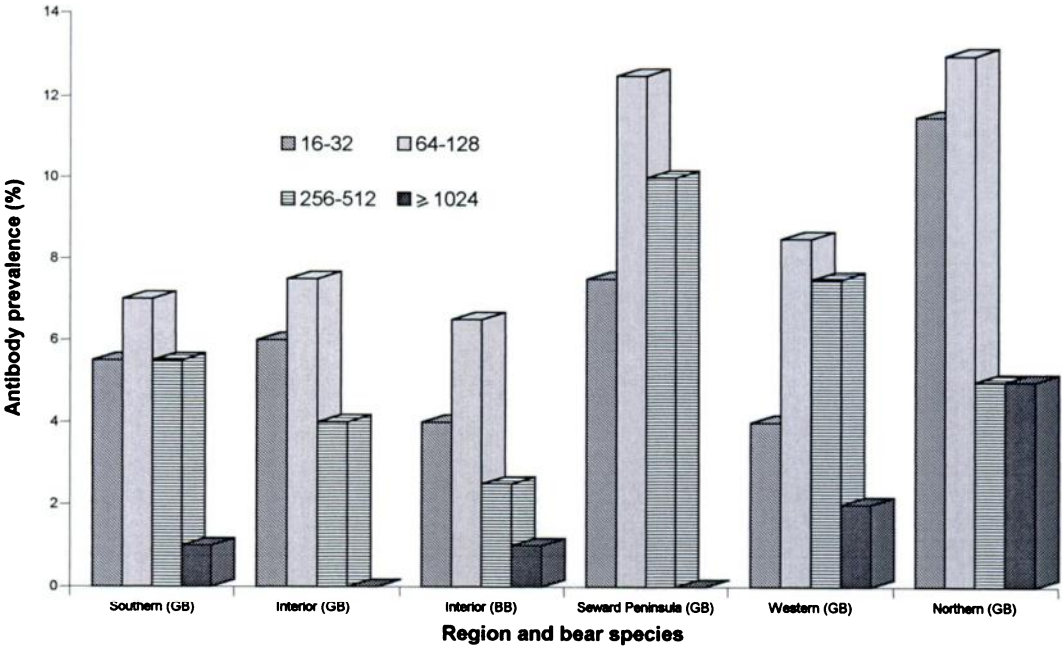


FIGURE 2. Antibody titers to *Toxoplasma gondii* in black bears (BB) and grizzly bears (GB), by regional location, Alaska, 1988 to 1991. Variation in titers is illustrated by hatching in boxes.

in the 2.5 to 4 yr-old group, 12% (11 of 89) in the 4.5 to 8 yr-old group, 20% in both the 8.5 to 12 yr-old group (20 of 101) and in the >12 yr-old group (34 of 167). Very low antibody titers (titer = 16) were observed mainly in old bears, as nine of the 11 bears with such a titer, and for which a specific age was determined, were more than 10-yr-old.

Among the 77 grizzly bears bled more than once, 17 bears (22%), ranging from 2 to 20 yrs old, had at least one positive sample for *T. gondii*. Nine grizzly bears were positive at two or three different bleedings, including two young females which had high titers (≥1,024). The seven other bears were more than 10-yr-old (range 10.5 to 20 yr) at time of the first bleeding.

Among the 25 black bears bled more than once, six (24%) bears, ranging in age from 1 to 12 yr, had *T. gondii* antibodies titer ≥1:32. Three of these six black bears converted from negative to positive or weak positive during the sampling period. Two other bears, initially positive, became weakly positive, and one black bear which was positive became negative.

DISCUSSION

Grizzly bears and black bears, like many other scavenger species, are susceptible to *T. gondii* infection. Dubey et al. (1994) isolated *T. gondii* from five bears from Pennsylvania, confirming natural infection. We observed an antibody prevalence in black bears of 15%. This prevalence was within the range of the 27% (40 of 149) which was previously reported in California (Ruppanner et al., 1982), and the 8% (23 of 303) reported from Idaho (Binninger et al., 1980), but lower than the 44% (seven of 16) reported from Ontario, Canada (Tizard et al., 1976), the 80% (535 of 665) reported from Pennsylvania by Briscoe et al. (1993) and the 80% (257 of 322) reported by Dubey et al. (1994), also from Pennsylvania. The mean prevalence in grizzly bears was 18%. This is the first report of serologic evidence of exposure in grizzly bears.

The lower prevalence of *T. gondii* an-

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TABLE 2. Year-specific serum antibody prevalence of *Toxoplasma gondii* in grizzly bears (*Ursus arctos horribilis*) and black bears (*Ursus americanus*) from Alaska.

Species	Location	Year				Total
		1988	1989	1990	1991	
U. arctos	A	2/41 (5)[a]	5/37 (14)	0/1 (0)	NS[b]	7/79 (9)
	B	12/48 (25)	NS	4/38 (11)	NS	16/86 (19)
	C, D	3/14 (21)	0/1 (0)	2/17 (12)	1/21 (5)	6/53 (11)
	E	NS	9/40 (23)	NS	NS	9/40 (23)
	F	4/30 (13)	4/35 (12)	NS	5/34 (15)	13/99 (13)
	G	13/34 (38)	NS	7/44 (16)	9/55 (17)	29/133 (22)
	H	10/35 (29)	0/1 (0)	4/21 (19)	4/21 (19)	18/78 (23)
U. americanus	C, D	0/11 (0)	4/20 (20)	3/22 (14)	1/23 (4)	8/76 (11)

[a] Number positive/number sampled (percent positive).
[b] NS = not sampled.

tibodies in black bears from Alaska than in black bears from Pennsylvania may have resulted from the lower sensitivity of the LAT used in our study when compared to other serological tests such as the modified agglutination test (MAT) (Dubey et al., 1994). Dubey et al. (1995) reported a seroprevalence of 79% in black bears from Pennsylvania by MAT (Dubey and Desmonts, 1987), but the prevalence was only 32% (nine of 28) when using the same LAT we used. Furthermore, the isolation of *T. gondii* from six bears without LAT antibodies is evidence that this test was not as sensitive as the MAT (Dubey et al., 1995), but such data were not available at the time we performed our study. We considered positive any serum sample with a titer $\geq$1:64, as recommended by the manufacturer for species other than humans. It may have been more accurate to consider positive any LAT $\geq$1:16; five of the six bears reported by Dubey et al. (1995), for which *T. gondii* was isolated, had a LAT titer of 1:16. All six bears had positive MAT.

Year-specific antibody prevalence varied by location: 0% to 20% in black bears and 0% to 38% in grizzly bears. No specific pattern was noted. There was no significant difference ($P > 0.05$) in antibody prevalence by sex for either grizzly or black bears; this differed from reports by Binninger et al. (1980) in black bears in Idaho.

Prevalence varied between geographic areas. In grizzly bears, prevalence increased from south (14%) to north (25%). Such an increase eventually could be related to higher prevalences in wildlife on which grizzly bears preyed and scavanged.

First exposure to *T. gondii* seemed to occur mainly after 2 yr of age in grizzly and black bears. Apparently, opportunity for exposure was high in grizzly bears, based on the increasing age-specific antibody prevalence. Low antibody titers were observed mainly in very old grizzly bears; this probably was related more to residual antibodies than cross-reactivity with other parasitic organisms. In false positive cases, one would expect to have weakly positive

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results evenly distributed in all age groups. Our results were in agreement with those of Binninger et al. (1980) who reported that older bears had a greater prevalence of *T. gondii* due to a longer exposure potential. Such an observation also was reported in grizzly bears for infectious canine hepatitis (Zarnke and Evans, 1989). Based on our test results on serial samples, we believe that infection occurred during the study as well as re-infection or reactivation of bradyzoites. Age-related prevalence increase also has been described in Alaska natives (Peterson et al., 1974). These villagers routinely ate moose, reindeer (*Rangifer tarandus*) and bear meat. Flesh sometimes was eaten raw, or lightly cooked, particularly while on hunting expeditions. Such eating habits could explain *T. gondii* exposure among indigenous humans throughout Alaska.

There was limited data on the serum antibody prevalence of toxoplasmosis in various wildlife in Alaska. Prevalence was 23% among 110 free-ranging moose from the Susitna River, the Alaska Peninsula, and the Kenai Peninsula (Kocan et al., 1986). Bears certainly could be exposed from consumption of infected rodents or wild herbivores. The highest *T. gondii* antibody prevalence observed in grizzly bears was in geographic areas where caribou and reindeer were abundant (Davis, 1980). Therefore the higher infection rate in grizzly bears could be associated with a higher prevalence in reindeer. Such a hypothesis was based on the fact that *Brucella* spp. antibody prevalence in reindeer and humans was also the highest in northern Alaska where the major herds ranged (Huntley et al., 1963). In grizzly bears, *Brucella* spp. infection occured when eating infected reindeer or caribou (Neiland, 1975). *Brucella* spp. antibody prevalence in grizzly bears provided support for such a hypothesis (Zarnke, 1991). Therefore, serologic surveys in reindeer and caribou populations would be useful to determine if our hypothesis was valid. Serologic surveys could provide evidence that toxoplasmosis was transmitted to bears via a natural predator-prey cycle involving larger prey species. Serologic surveys and body infestation surveys should be conducted also in other prey species of grizzly bears, such as small rodents, to determine their respective role in the transmission of the infection to bears.

Reports from countries around the polar circle provided evidence for such cycles between rodents and carnivores in Norway (Kapperud, 1978), and in Siberia, Russia (Rogatykh et al., 1977). Rogatykh et al. (1977) and Galuzo (1977) reported that in the Taimyr and Khanty-Mansiik regions, Russia, 4% of the reindeer had serologic evidence of *Toxoplasma* spp. exposure. Toxoplasmosis is maintained by lemmings (*Lemmus lemmus*) and transmission to indigenous people occurs via reindeer meat which is normally eaten raw (Rogatykh et al., 1977). Leonova and Akin'shina (1975) reported low antibody titers in humans, reindeer, and rodents from the Chukotka Peninsula and Wrangel Island, Siberia.

Indigenous Alaskans as well as visiting hunters should be educated as to the risks associated with the consumption of raw or incompletely cooked bear meat. In Alaska, toxoplasmosis should be considered along with trichinellosis as a zoonosis transmitted by consumption of undercooked bear meat. As advocated by Briscoe et al. (1993), all meat should be thoroughly cooked. The internal temperature of meat should reach 66 C for 3 min (Dubey et al., 1990).

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