A SEROLOGIC SURVEY FOR SELECTED INFECTIOUS DISEASES OF BLACK BEARS IN IDAHO

Authors: BINNINGER, C. E., BEECHAM, J. J., THOMAS, LEO A., and WINWARD, LYNN D.

Source: Journal of Wildlife Diseases, 16(3) : 423-430

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-16.3.423

A SEROLOGIC SURVEY FOR SELECTED INFECTIOUS DISEASES OF BLACK BEARS IN IDAHO

C. E. BINNINGER, Southway Animal Clinic and Hospital, 705 16th Avenue, Lewiston, Idaho 83501, USA.

J. J. BEECHAM, Idaho Fish and Game Department, 109 West 44th Street, Boise, Idaho 83604, USA.

LEO A. THOMAS, U.S. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, National Institute of Allergy and Infectious Diseases, Epidemiology Branch, Rocky Mountain Laboratories, Hamilton, Montana 59840, USA.

LYNN D. WINWARD, Animal Disease Research Unit, U.S. Department of Agriculture, SEA-Western Region, Washington State University, Pullman, Washington 99164, USA.

Abstract: Two hundred sixty-five black bears *(Ursus americanus)* from northcentral Idaho were examined serologically over a five-year period for antibodies against selected infectious disease agents. The number of positive serum samples per number of sera tested and percent positive for each infectious agent is: tularemia, 65/340 (19); brucellosis, 18/332 (5); toxoplasmosis, 23/303 (8); leptospirosis, 2/196 (1); trichinosis, 16/122 (13); Q-fever, 13/210 (6); St. Louis encephalitis, 3/340 (1); western equine encephalitis, 4/334 (1); Rocky Mountain Spotted Fever, 6/282 (2). Black bears may serve as an indicator for infection in other wildlife, domestic animals and humans in the area.

INTRODUCTION

The causes of natural mortality in free-ranging American black bears *(Ursus americanus)* remain largely unknown. Infectious disease has received little attention, and few published data exist. Any epidemiological role of black bears to other wildlife, domestic animals and humans is unknown.

In Idaho, black bear populations occur primarily in the counties occupying the northern two-thirds of the state with isolated populations along the Montana and Wyoming borders in the southeastern[2] part of the state. Domestic cattle graze all areas in which bears were sampled. Brucellosis *(Brucella abortus)* and leptospirosis *(Leptospira* spp.) have been present in some of these counties in domestic livestock during the past decade. Because the black bear is a predator of large animals as well as a scavenger of dead animals, there is a potential source of infection that other predators do not share. This paper presents serological evidence of exposure to selected diseases of American black bears in Idaho.

MATERIALS AND METHODS

A total of 265 black bears was captured in north-central Idaho (N=12) near Council or Lowell (N=253) as part of a long-term study of black bear ecology. From these 265 bears, 352 blood samples were taken, but not all samples were tested for all diseases.

Most bears were live-trapped with Aldrich spring-activated foot snares or culvert traps, while five were shot by hunters. Live-trapped bears were sedated with intramuscular injections of phencyclidine hydrochloride (Sernylan) [1] [2]

[1] Mention of a trade name, proprietary product or specific equipment does not constitute a guarantee or warranty by the U.S. Department of Agriculture and does not imply its approval to the exclusion of other products that may be suitable.

[2] Bio-Ceutic Laboratories, Inc., St. Joseph, Missouri 64502, USA.

at a dosage rate of approximately 1.3 mg/kg of body weight alone or in combination with promazine hydrochloride (Sparine)[3] at the rate of 0.6 mg/kg. Most bears (N=248) were sampled during May through August each year; 17 were sampled in dens during October-December and in March.

Blood samples were taken from either the femoral, cephalic, or jugular veins and collected in stoppered glass tubes. Each sample was allowed to clot at ambient temperature, refrigerated 1-2 hrs and then centrifuged. Serum was stored at approximately -10 C.

Sera collected in 1971-1975 were tested for antibodies at the Idaho Department of Public Health and the Bureau of Animal Industry in Boise, Idaho. Samples collected in 1976-1977 were tested at the Rocky Mountain Laboratory, U.S. Public Health Service, Hamilton, Montana, except tests for toxoplasmosis which were carried out at the Pioneering Research Laboratory, USDA-SEA Western Region, Pullman, Washington. Duplicate testing at different laboratories was not done.

The tube agglutination test[6] was used to test for antibodies to *Francisella tularensis* (Jap. Downs strain) and *Brucella abortus* antigens; the rapid plate agglutination test[5] was used in testing 10 serotypes of *Leptospira (L. canicola, L. icterohemorrhagiae, L. grippotyphosa, L. autumnalis, L. hyos, L. bataviae, L. pomona, L. pyrogenes, L. ballum* and *L. hardjo);* the complement fixation (CF) microtiter test[21] for Q-fever (QF) (nine-mile strain), Rocky Mountain spotted fever (RMSF), LaCrosse strain of California encephalities (CE), McMillan strain of western equine encephalitis (WEE), strain 85 of eastern equine encephalitis (EEE), Parton strain of St. Louis encephalitis (SLE), and Armstrong strain of lymphocytic choriomeningitis (LCM); the indirect hemagglutination (IHA) test for toxoplasmosis using the RH strain; and the IHA or bentonite floculation test (BFT) for trichinosis.

Antibody titer is designated as the reciprocal of the highest serum dilution showing a positive response to that antigen. Minimum screening titers were arbitrarily selected for each disease.

A premolar tooth was extracted for aging purposes, and sex was recorded for each bear.[16]

RESULTS

The frequency of antibody titers in 265 black bears in Idaho against nine infectious agents is presented in Table 1. Ninety-one of the 352 blood samples tested were anticomplementary. As a result, these were tested only against those antigens not utilizing the complement fixation test.

The mean age of positive and negative reactors to tularemia, brucellosis, and toxoplasmosis is presented in Table 2. Males positive to toxoplasmosis had a significantly ($P<.005$) higher mean age than the negative males. There were no significant differences ($P>.05$) in mean ages for brucellosis and tularemia. The prevalence of brucellosis and toxoplasmosis was significantly ($P<.05$) higher in males, but there was no difference in the prevalence of tularemia between sexes. Low titers were recorded for leptospirosis, Q-fever, SLE, WEE, RMSF, and trichinosis as seen in Table 1.

Fluctuations in tularemia (N=13) and brucellosis (N=3) titers are presented in Table 3.

Ten bears were serologically positive to more than one agent at a single sampling; eight had titers to tularemia and brucellosis (range 40-640 for tularemia and 20-320 for brucellosis). One bear had titers of 160 (tularemia), 40 (brucellosis), and 32 (toxoplasmosis). Another bear

[3] Wyeth Laboratories, Inc., Philadelphia, Pennsylvania 19101, USA.

TABLE 1. Frequency of antibody titers to nine infectious disease agents of black bears in Idaho. 1971-77.

Year	Tularemia 40a	Brucellosis 20	Toxoplasmosis 32	Leptospirosis 40	Trichinosis 16	Q-fever 8	SLE 8	WEE 8	RMSF 8
1971	40: 3/12b 80: 2/12 160: 2/12	20: 3/12	— —	— —	— —	8: 3/9	— —	— —	— —
1974	40: 5/27 80: 1/17	0/26	64: 1/29 128: 3/29	40: 1/29	0/26	8: 4/26	8:2/28	8:1/32	— —
1975	40: 2/66 80: 1/66 160: 4/66 320: 1/66 640: 1/66	0/64	32: 4/49 64: 3/49 128: 1/49	40: 1/52	— —	8: 4/53	32:1/53	8:3/53	8:2/53
1976	40: 8/125 80: 5/125 160: 6/125 320: 1/125	20: 2/123 40: 4/123 80: 2/123	32: 9/121	40: 0/115	16:15/122 32: 1/122	8: 2/122	8:0/249	8:0/249	8:1/122
1977	40: 6/110 80:10/110 160: 3/110 320: 1/110 640: 3/110	20: 3/107 40: 1/107 80: 2/107 320: 1/107	32: 1/104 128: 1/104	— —	— —	— —	— —	— —	8:2/108 12:1/108
TOTAL ( ) = %	65/340 (19)	18/332 (5)	23/303 (8)	2/196 (1)	16/122 (13)	13/210 (6)	3/340 (1)	4/334 (1)	6/282 (2)

a Minimum screening titer
b Titer: number positive/number tested

TABLE 2. Mean age of positive and negative reactors to tularemia, brucellosis, and toxoplasmosis in black bears in Idaho. 1971-77.

	Tularemia				Brucellosis				Toxoplasmosis			
	Positive		Negative		Positive		Negative		Positive		Negative	
Sex	N	$\bar{X}$	N	$\bar{X}$	N	$\bar{X}$	N	$\bar{X}$	N	$\bar{X}$	N	$\bar{X}$
Male	38	6.4	155	5.1	14	6.7	177	5.2	18	7.7	153	4.9
Female	27	7.2	120	6.7	3*	7.0	137	6.7	5	9.2	127	6.5
Total	65	6.7	275	5.8	17	6.7	314	5.8	23	8.0	280	5.6

*A 24-year-old female was excluded because of her extreme age.

had titers of 128 to toxoplasmosis and 40 to *L. grippotyphosa.*

DISCUSSION

Tularemia

Tularemia antibodies have not been reported previously in black bears in Idaho, although two *Francisella* species have been isolated from mule deer *(Odocoileus hemionus),* one a strain of F. tularensis.[15]

Thorpe *et al.*[17] found endemic levels of *F. tularensis* in several rodent and lagomorph species in Utah. They also reported that ticks and possibly lice and fleas are potential sources of tularemia infection for wildlife species. Even though ingestion of infected tissues through scavenging or predation is a potential source of infection for black bears, the most probable source of exposure to *F. tularensis,* however, is from ticks or other ectoparasites. The black bear in Idaho serves as a host for ticks *(Dermacentor andersoni* and *D. variabilis),* lice *(Tichodectes pinguis euarctidos),* fleas *(Chaetopsylla setosa),* and mites *(Ursicoptes americanus).*[2,4]

The magnitude in antibody titer fluctuations for *F. tularensis* (Table 3) and their subsequent recapture indicates that some black bears are capable of surviving significant exposure. Table 3 is presented to show that serial testing can be more beneficial in determining the prevalence of this disease than a single test at any given time. This table also shows that some titers (U-74, U-103) can increase significantly in one season, decrease to negative (U-103, U-34, U-20), then back to positive (U-103).

Brucellosis

Brucellosis has not been reported previously in black bears, although Neiland[11] found grizzly bears *(Ursus arctos)* were readily suscept ble to *B. suis* type 4 in Alaska.

Possible sources of *Brucella* infection in black bears include the ingestion of contaminated food [11,12] and transmission to females by infected males during copulation as in domestic carnivores.[4] The greater prevalence of *Brucella* in males than in females suggests that oral ingestion is the more likely source of infection in black bears. Male black bears have larger home ranges than females and, therefore, are more likely to encounter infected food sources.[1,14] *Brucella* has been reported in ground squirrels *(Citellus* sp.), deer mice *(Peromyscus maniculatus),* and jackrabbits *(Lepus* sp.) in Utah[20] but was not found in elk *(Cervus canadensis)* in north-central Idaho.[19]

The species of *Brucella* causing agglutinins to *B. abortus* in black bears in Idaho is unknown. However, because bears are largely herbivorous on Idaho summer ranges feeding on meadows later grazed by cattle, any potential epizootiologic role in cattle infections should be determined by further investigation.

TABLE 3. Fluctuations in tularemia and brucellosis titers in 16 black bears sampled in Idaho. 1974-77.

Disease	Bear No.	Sex	1974 Age Yr.	1974		1975		1976		1977	
				Date	Titer	Date	Titer	Date	Titer	Date	Titer
Tularemia	U-77	F	4	5/30	40	—*	—	—	—	8/8	20**
										11/22	80
	U-79	F	7	6/2	N***	—	—	6/22	20	3/17	160
								8/6	320	7/27	80
	U-103	M	1	—	—	—	—	6/11	20	7/1	20
								6/17	160	7/18	N
										8/14	80
	U-34	F	5	6/10	20	—	—	7/11	80	3/13	N
										11/17	80
	IMF-472	M	5	—	—	6/8	N	6/9	40	—	—
	S-10	M	4	—	—	—	—	6/8	N	6/12	640
	IMF-484	F	11	—	—	1/16	160	—	—	6/5	20
						6/27	80				
	U-40	M	8	—	—	8/7	160	7/25	20	—	—
	U-104	M	2	—	—	8/1	320	6/9	20	6/8	20
								7/27	20	8/10	20
	U-58	M	8	—	—	7/16	640	—	—	6/15	20
	U-88	M	3	7/16	80	5/30	N	—	—	—	—
						7/10	N				
	U-20	M	4	5/25	40	—	—	—	—	7/7	N
	U-92	F	13	—	—	—	—	8/5	80	7/16	20
Brucellosis	IMF-472	M	5	—	—	6/8	N	6/9	40	—	—
	S-10	M	4	—	—	—	—	6/8	N	6/12	320
	U-139	F	1/2	—	—	—	—	7/1	N	6/25	20

*— = not sampled
**20 = positive reaction at 1/20 dilution
***N = no titer

Toxoplasmosis

Toxoplasma gondii has been reported in black bears in Ontario, Canada, but not in the U.S.[13,18]

Quinn *et al.*[13] recognized three primary methods of transmission of *T. gondii:* congenital, fecal contamination of food from an infected feline, and from carnivorous food habits. Tizard *et al.*[18] reported that carnivores had the highest antibody titers against *T. gondii,* herbivores the lowest, and omnivores shared intermediate values. The greater prevalence of *T. gondii* antibody titers in male black bears than in females and the significantly higher mean age of positive than negative reactors in males suggest that the source of infection in Idaho black bears is from contamination of their food, possibly by an infected felid. Felid densities are not exceptionally high in those areas studied intensively. Therefore, older bears having a greater prevalence of *T. gondii* might be expected due to a longer potential exposure and males, in particular, because of their extensive home ranges. Our data support this assumption.

Quinn *et al.*[13] reported that *T. gondii* frequently may infect animals and stimulate antibody production without causing clinical disease. It is unknown whether bears in our study develop clinical toxoplasmosis.

Trichinosis

The 13% prevalence of *Trichinella spiralis* in black bears in Idaho examined during this serologic study is in agreement with the 11.9% prevalence of black bears collected in Glacier and Yellowstone National Parks as reported by Worley *et al.*[22] who used an artificial digestion technique. However, it is high compared to the 2.3% prevalence in a study conducted by Zimmerman[24] of black bears in Idaho and in black bears in other parts of the United States.[22]

On the other hand, the prevalence rates in the black bear reported here and by Worley *et al.*[22] are lower than the striped skunk *(Mephitis mephitis),* bobcat *(Lynx rufus),* and much lower than in the coyote *(Canis latrans),* fisher *(Martes pennanti),* wolverine *(Gulo gulo),* and mountain lion *(Felis concolor),* and lower still than in the grizzly bear *(Ursus arctos).* The prevalence among grizzly bears was 45.1% of those captured in Glacier or Yellowstone National Parks or environs and 58.4% among those collected in wilderness areas.

Other Diseases

Low titers recorded for QF, WEE, SLE, RMSF, and *Leptospira* spp. are believed to indicate previous exposure to the organism. The results are included here to demonstrate that the antigenic agents are present in the area at the prevalence rate recorded in Table 1. As such, the zoonotic and epizootic disease potential is probably insignificant.

In a CF control test, WEE, SLE, CE, MODOC strain M544, LCM antigens along with saline solution were tested against hyperimmune mouse sera or ascitic fluids of Powassan, yellow fever strain 17D, Yaquina, Tuleniy, EEE, Ilheus, Sindbis, CE, WEE, Japanese B encephalitis, Modoc, SLE, and LCM along with saline solution and normal mouse serum. The only cross reactions noted were between EEE and WEE and between Japanese B encephalitis and SLE. These reactions were one quarter as strong as between homologous antigen-antiserum reactions. Such hyperimmune sera would not be expected to occur in naturally exposed animals.

The public health hazards of these zoonotic diseases is beyond the scope of this paper. However, the wide distribution of black bears in the U.S., their potential as hosts for ectoparasitic disease vectors, and their omnivorous food habits (eating both the forage and the foraging animal) suggest that they may be an indicator species for epizootic outbreaks of infectious disease in other wildlife, domestic animals, and humans. Additional research in this area is needed to accurately assess this potential.

Acknowledgements

Our appreciation is extended to the Idaho Department of Public Health and Bureau of Animal Industry laboratories for testing the 1971-1975 sera. We also extend a special thanks to Susan Obenberger for her help in analyzing the data. This paper is a contribution of Federal Aid in Wildlife Restoration, Project W-160-R.

LITERATURE CITED

1. AMSTRUP, S.C. and J. BEECHAM. 1976. Activity patterns of radiocollared black bears in Idaho. J. Wildl. Manage. 40: 340-348.
2. BEECHAM, J. 1977. Some population characteristics of two black bear populations in Idaho. Int. Conf. Bear Res. and Manage. 4. (In press).
3. CHORDI, A., K.W. WALLS and G.R. HEALY. 1964. Studies on specificity of the indirect hemagglutination test for toxoplasmosis. J. Immunol. 93: 1024-1033.
4. ETTINGER, S.J. 1975. *Textbook of Veterinary Internal Medicine.* Vol. 1. W.B. Sanders Co., Philadelphia, PA. Pp. 222-233.
5. GALTON, M.M., C.R. SULZER, C.A. SANTA ROSA and M.I. FIELDS. 1965. Application of a microtechnique to the agglutination test for leptospiral antibodies. Appl. Microbiol. 13: 81-85.
6. FRANCIS, E. and A.C. EVANS. 1926. Agglutination, cross-agglutination and agglutinin absorption in tularemia. Public Health Rpt. 41: 1273-1295.
7. JACOBS, L. and M.N. LUNDE. 1957. A hemagglutination test for toxoplasmosis. J. Parasit. 43: 303-314.
8. KAGAN, I.G., H.A. FOX, K.W. WALLS and G.R. HEALY. 1967. Parasitic diseases of childhood with emphasis on the newer diagnostic methods. Clin. Pediatr. 6: 641-654.
9. LEWIS, W.P. and J. KESSEL. 1961. Hemagglutination in the diagnosis of toxoplasmosis and amebiasis. Arch. Ophthalmol. 66: 471-476.
10. MATULA, G.J., J.S. LINDZEY and H. ROTHENBACHER. 1977. Sex, age and seasonal differences in the blood profile of black bears captured in northeastern Pennsylvania. Int. Conf. Bear Res. and Manage. 4. (In press).
11. NEILAND, K.A. 1975. Further observation on rangiferine brucellosis in Alaskan carnivores. J. Wildl. Dis. 11: 45-53.
12. PRICHARD, W.D., K.W. HAGEN, J.R. GORHAM and F.C. STILES, Jr. 1971. An epizootic of brucellosis in mink. J. Am. vet. med. Ass. 159: 635-637.
13. QUINN, P.J., R.O. RAMSDEN and D.H. JOHNSTON. 1976. Toxoplasmosis: a serological survey in Ontario wildlife. J. Wildl. Dis. 12: 504-510.
14. REYNOLDS, D.G. and J. BEECHAM. 1979. Home range activities and reproduction of black bears in west-central Idaho. Int. Conf. Bear Res. and Manage. 4. (In press).
15. SHAW, W.M. 1964. Idaho big game harvest, census and range study. Job Comp. Rep. No. 15. Disease and parasitism tests and reports. Idaho Dept. Fish and Game, Boise.
16. STONEBERG, R.P. and C.J. JONKEL. 1966. Age determination of black bears by cementum layers. J. Wildl. Manage. 30: 411-414.
17. THORPE, B.D., R.W. SIDWELL, D.E. JOHNSON, K.L. SMART and D.D. PARKER. 1965. Tularemia in the wildlife and livestock of the Great Salt Lake desert region, 1951 through 1964. Am. J. Trop. Med. Hyg. 14: 622-637.

18. TIZARD, I.R., J.B. BILLETT and R.O. RAMSDEN. 1976. The prevalence of antibodies against *Toxoplasma gondii* in some Ontario mammals. J. Wildl. Dis. 12: 322-325.

19. VAUGHN, H.W., R.R. KNIGHT and F.W. FRANK. 1973. A study of reprod-disease, and physiological blood and serum values in Idaho elk. J. Wildl. Dis. 9: 296-301.

20. VEST, E.D., D.L. LUNGREN, D.D. PARKER, D.E. JOHNSON, E.L. MORSE, J.B. BUSHMAN, R.W. SIDWELL and B.D. THORPE. 1965. Results of a five-year survey for certain enzootic diseases in the fauna of western Utah. Am. J. Trop. Med. Hyg. 14: 124-135.

21. WELSH, H.H., F.W. JENSEN and E.H. LENETTE. 1959. Q-fever studies. XX. Comparison of four serological techniques for the detection and measurement of antibody to *Coxiella burnetii* in naturally exposed sheep. Am. J. Hyg. 70: 1-13.

22. WORLEY, D.E., J.C. FOX, J.B. WINTERS and K.R. GREER. 1974. Prevalence and distribution of *Trichinella spiralis* in carnivorous mammals in the United States northern Rocky Mountain region. In: *Trichinellosis.* Proc. Third Int. Conf. on Trichinellosis. C. W. Kim, ed. Intext Educational Publishers, New York, NY.

23. YUNKER, C.E., C.E. BINNINGER, J.E. KEIRANS, J. BEECHAM and M. SCHLEGEL. Clinical mange in the black bear, *Ursus americanus,* associated with *Ursicoptes americanus* (Acari: Audycoptidae). J. Wildl. Dis. (In press).

24. ZIMMERMAN, W.J. 1977. Trichinosis in bears of western and north-central United States. Am. J. Epidemiol. 106: 167-171.

Received for publication 16 May 1979