Prevalence of Antibody Titers to Leptospira Spp. in Minnesota White-tailed Deer

Authors: Goyal, Sagar M., Mech, L. David, and Nelson, Michael E.

Source: Journal of Wildlife Diseases, 28(3) : 445-448

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-28.3.445

Journal of Wildlife Diseases, 28(3), 1992, pp. 445–448

Prevalence of Antibody Titers to *Leptospira* Spp. in Minnesota White-tailed Deer

Sagar M. Goyal,[1,5] **L. David Mech,**[2,3] **and Michael E. Nelson,**[2,4] [1] Department of Veterinary Diagnostic Medicine, College of Veterinary Medicine, University of Minnesota, St. Paul, Minnesota 55108, USA; [2] U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, Maryland 20708, USA; [3] Present address: North Central Forest Experiment Station, 1992 Folwell Avenue, St. Paul, Minnesota 55108, USA. [4] Present address: 305 W. Harvey Street, Ely, Minnesota 55731, USA. [5] To whom reprint requests should be sent

ABSTRACT: Serum samples (n = 204) from 124 white-tailed deer (*Odocoileus virginianus*) in northeastern Minnesota (USA) were collected from 1984 through 1989 and tested for antibodies to six serovars of *Leptospira interrogans (bratislava, canicola, grippotyphosa, hardjo, icterohemorrhagiae,* and *pomona*) using a microtiter agglutination test. Eighty-eight (43%) sera were positive at ≥1:100 for antibodies against serovars *pomona* and/or *bratislava*; none was positive for any of the other four serovars. None of the 31 sera collected in 1984–85 was positive, whereas all 54 sera collected from 1986 through 1988 had titers of ≥1:100. During 1989, only 34 (29%) of 119 sera had titers of ≥1:100. Based on these results, we believe there to be wide variability in exposure of Minnesota deer to *Leptospira interrogans*.

Key words: Leptospirosis, white-tailed deer, epizootiology, *Leptospira interrogans, Odocoileus virginianus,* regional serosurvey, microtiter agglutination.

Leptospirosis is an important disease of domestic animals and humans that occurs in wild animals throughout North America (Cirone et al., 1978). Serologic evidence of *Leptospira interrogans* infection has been found in red foxes (*Vulpes vulpes*) (Clark et al., 1960), gray foxes (*Urocyon cinereoargenteus*) (Clark et al., 1961), coyotes (*Canis latrans*) (Marler et al., 1979; Drewek et al., 1981), black-tailed deer (*Odocoileus hemionus columbianus*) (Cirone et al., 1978), white-tailed deer (*Odocoileus virginianus*) (Anthony et al., 1968; Ingebrigtsen et al., 1986; Fournier et al., 1986), moose (*Alces alces*) (Bourke and Higgins, 1984), wolves (*Canis lupus*) (Zarnke and Ballard, 1987; Khan et al., 1991), and black bears (*Ursus americanus*) (Zarnke and Ballard, 1987). Wildlife may play a role in the maintenance and spread of leptospirosis to domestic animals and humans by acting as reservoirs of *Leptospira* infection (Cirone et al., 1978; Bourke and Higgins, 1984).

The prevalence of antibody titers to *Leptospira* in deer populations of North America varies between 7 and 27% (Wedman and Driver, 1957; Ferris et al., 1961; Abdulla and Fish, 1962; Shotts and Hayes, 1970; Fournier et al., 1986). Although the role of deer in the epizootiology of leptospirosis is not clear, deer probably do not serve as reservoirs of *Leptospira* (Ferris et al., 1961; Trainer et al., 1961; Nixon, 1970). The significance of *Leptospira* infection in deer health is not known. However, *Leptospira* antibody titers in deer drop rapidly and may disappear completely within 12 to 15 weeks (Trainer et al., 1961); in contrast, bovine leptospirosis titers can persist for months (Higgins et al., 1975; Bourke and Higgins, 1984), suggesting that titers found in deer are of relatively recent origin. Our objective was to determine the prevalence of anti-*Leptospira* antibodies in a deer population of northeastern Minnesota and to compare the results with those from other studies.

One hundred twenty-four deer were captured in Central Lake County of northwestern Minnesota (USA; 47°30′ to 48°00′N and 90°45′ to 92°00′W) by Clover trap (Clover, 1954) or by rocket net from 1984 to 1989. The deer population of this area ranged between 500 and 2,000 (Mech, unpubl.). Twenty-seven of these 124 deer each were later recaptured one to eight times with capture collars (Mech et al., 1990). On each capture, deer were anesthetized with a xylazine hydrochloride-ketamine hydrochloride mixture (Mech et

TABLE 1. Prevalence of *Leptospira interrogans* antibodies in white-tailed deer of Minnesota (USA), 1984 to 1989.

Sampling frequency	Number animals sampled	Number sera tested	Number sera positive	% Sera positive
Single samples	97	97	56	58
Multiple samples	27	107	32*	30
Total	124	204	88	43

* From 19 animals.

TABLE 2. The prevalence of *Leptospira interrogans* antibodies by year.

Year	Number of sera tested	Number (%) of sera positive	Number of animals positive at ≥1:100
1984	1	0 (0)	0
1985	30	0 (0)	0
1986	16	16 (100)	15
1987	9	9 (100)	9
1988	29	29 (100)	29
1989	119	34 (29)	22
Total	204	88* (43)	75

* Since there were multiple samples from some animals, the number of positive sera is more than the number of positive animals at ≥1:100 titer.

al., 1985). Blood was drawn from their jugular veins and centrifuged; serum was removed and stored at −20 C. An incisor was pulled from each adult and the deer's age was estimated (Gilbert, 1966).

A microscopic microagglutination test (Cole et al., 1979) was used to determine antibody titers against six serovars of *Leptospira interrogans: bratislava, canicola, grippotyphosa, hardjo, icterohemorrhagiae*, and *pomona* (Tables 1, 2, and 3). A titer of ≥1:100 was considered positive (Fournier et al., 1986).

A total of 204 sera was collected; 97 single samples and 107 multiple samples. Most samples (n = 163) were taken from January through April of each year; the remainder were drawn from May through September. Fifty-two deer were <1-yr-old, including three animals subsequently recaptured as adults. Three animals could not be aged.

None of the sera had titers to serovars *canicola, grippotyphosa, hardjo*, or *icterohemorrhagiae*. Eighty-eight (43%) of the 204 sera had antibodies against serovars *pomona* and/or *bratislava* (Table 1). Seventy-five (61%) of the 124 animals had titers of ≥1:100 at least once. This is in sharp contrast to Ingebrigtsen et al.'s (1986) finding that only 2.6% of the deer in Minnesota farmlands were seropositive to *Leptospira* spp. Our results also contrast with Fournier et al.'s (1986) findings of seroprevalences of only 7 to 27%. The reasons for these differences are not known. However, the antibody titers in the present study were low and never exceeded 1:200; in the other studies some titers reached 1:1,000 (Fournier et al., 1986).

Twenty-five (66%) of 38 males and 50 (58%) of 86 females were seropositive; 31 (60%) of 52 fawns and 44 (64%) of 69 adults were seropositive. These results agree with Fournier et al.'s (1986) finding that age and sex did not affect prevalence of anti-*Leptospira* antibody titers in deer. None of the sera collected in 1984–85 was seropositive, but all sera collected in 1986–88 had antibodies (Table 2). Antibody prevalence declined to 29% in 1989. Deer mortality among radio-tagged animals, however, did not differ significantly among these three periods (15% vs. 12% vs. 8%, respectively).

Fifty-six (58%) of the 97 animals sampled only once during the study had antibody titers of ≥1:100; 43 of these animals had antibodies against both *L. bratislava* and *L. pomona*, 12 against *L. bratislava* only, and one against *L. pomona* only. One hundred seven serum samples were obtained from 27 animals that were bled more than once; 32 samples from 19 animals were positive (Table 3). Sixteen of the 19 animals had antibodies against both *L. bratislava* and *L. pomona*, two against *L. bratislava* only, and one against *L. pomona* only. On examination of multiple samples, no four-fold rise in titer was seen in any animal. In fact, the titers were never higher than 1:200. Deer developed titers as ear-

TABLE 3. Distribution of serum antibody titers to *Leptospira* in 19 of 27 deer sampled more than once.[a]

Animal number	Antibody detection[b]	Samples collected
6862	++	6 to 29 March 1986
6878	++	4 February 1986 to 9 February 1987
7278	−+	16 March to 26 April 1989
7282	−+	21 March to 2 May 1989
7058	+−	4 January 1988 to 21 March 1989
106	−−+	18 January 1985 to 18 March 1987
6381	−−+	28 January 1985 to 2 February 1988
6584	−+−	28 February 1985 to 18 March 1989
7072	++−	4 to 20 February 1989
7034	+−−	11 March 1988 to 6 April 1989
7096	−−−+	10 March to 24 April 1989
7288	−−+−−−	30 March to 17 October 1989
7300	−−+++−	8 April to 1 November 1989
7068	−−−−−++	2 January to 16 July 1989
7256	−−−++++	6 March to 5 August 1989
7270	−−−+−−−	14 March 2 to November 1989
7302	−−+++−−−	9 April to 1 to November 1989
6462	−−−−−−−−+	7 February 1985 to 17 May 1989
7092	−−−+−−−−	6 February to 26 November 1989

[a] Six animals bled twice each and two animals bled three times each were negative on all bleedings.

[b] For each animal, the number of seropositive (+) and seronegative (−) samples is shown in the order of occurrence during the range of dates sampled.

ly as 1 wk and as late as 3 yr after a negative serum sample was obtained; the median was 5 wk. Similarly, animals that became negative did so 2 days to 15 mo after a positive response, with a median of 5 wk. Animals seropositive for more than one bleeding remained positive for 2 wk to 1 yr, with a median of 7 wk. Deer 7072 and 7302 became negative within 2 and 4 days, respectively, of testing positive. These do not appear to be aberrent results because animal 7302 was positive on three consecutive bleedings before becoming negative and was negative for two additional bleedings. Trainer et al. (1961) have shown that *Leptospira* antibodies in deer usually persist <15 wk.

Fourteen of the 16 positive samples in 1986 were positive for serovar *pomona* only. Antibodies against serovar *bratislava* appeared for the first time in April 1986 when two deer had antibodies. In subsequent years, antibodies against both serovars were detected. Based on these results, we speculate that there may have been low prevalence of *Leptospira* infection in deer populations in 1984–85 followed by an increase in 1986–88 before the prevalence rate came down again in 1989. What provided these stimuli is not known, nor is the significance of these episodes. Our results confirm previous observations that *Leptospira* antibody titers in deer do not persist long, compared to titers in cattle. The widespread occurrence of low levels of anti-*Leptospira* antibody titers in deer indicate that these animals may not act as significant reservoirs of *Leptospira* for either humans or animals in Minnesota.

We thank W. K. Goml for assisting with blood sampling, and U.S. Seal for serum storage. This study was funded by the U.S. Fish and Wildlife Service, the U.S.D.A. North Central Forest Experiment Station, and the Department of Veterinary Diagnostic Medicine of the University of Minnesota.

LITERATURE CITED

ABDULLA, P. K., AND N. A. FISH. 1962. Cultural and serological evidence of leptospirosis in deer in Ontario. Canadian Veterinary Journal 3: 71–78.

ANTHONY, H. D., S. W. CAPEL, AND W. C. PEABODY. 1968. Survey of leptospirosis agglutination titers

in serums collected from deer in Kansas. Proceedings of the United States Animal Health Association 72: 192–193.

BOURKE, M., AND R. HIGGINS. 1984. Serological studies on brucellosis, leptospirosis, and tularemia in moose (*Alces alces*) in Quebec. Journal of Wildlife Diseases 20: 95–99.

CIRONE, S. M., H. P. RIEMANN, R. RUPPANNER, D. E. BEHYMER, AND C. E. FRANTI. 1978. Evaluation of the hemagglutination test for epidemiological studies of leptospiral antibodies in wild mammals. Journal of Wildlife Diseases 14: 193–202.

CLARK, L. G., J. L. KRESSE, R. R. MARSHAK, AND C. J. HOLLISTER. 1960. *Leptospira pomona* infection in an eastern red fox (*Vulpes fulva fulva*). Nature (London) 188: 1040–1041.

———, ———, ———, AND ———. 1961. Natural occurrence of *Leptospira icterohemorrhagiae* in the eastern gray fox and the eastern raccoon. Nature (London) 192: 1312–1313.

CLOVER, M. R. 1954. A portable deer trap and catch-net. California Fish and Game 40: 367–373.

COLE, J. R., H. C. ELLINGHAUSEN, AND H. L. RUBIN. 1979. Laboratory diagnosis of leptospirosis of domestic animals. Proceedings of the United States Animal Health Association 83: 189–195.

DREWEK, J., JR., T. H. NOON, R. J. TRAUTMAN, AND E. J. BICKNELL. 1981. Serological evidence of leptospirosis in a southern Arizona coyote population. Journal of Wildlife Diseases 17: 33–37.

FERRIS, D. H., L. E. HANSON, H. E. RHOADES, AND J. O. ALBERTS. 1961. Bacteriological and serological investigations of brucellosis and leptospirosis in Illinois deer. Journal of the American Veterinary Medical Association 139: 892–896.

FOURNIER, J. S., J. C. GORDON, AND C. R. DORN. 1986. Comparison of antibodies to *Leptospira* in white-tailed deer (*Odocoileus virginianus*) and cattle in Ohio. Journal of Wildlife Diseases 22: 335–339.

GILBERT, F. F. 1966. Aging white-tailed deer by annuli in the cementum of the first incisor. Journal of Wildlife Management 30: 200–202.

HIGGINS, R., P. CAYONETTE, AND J. G. COUSINEAU. 1975. A serological study of bovine leptospirosis in Quebec Province. Canadian Veterinary Journal 16: 304–307.

INGEBRIGTSEN, D. K., J. R. LUDWIG, AND A. W. MCCLURKIN. 1986. Occurrence of antibodies to the etiological agents of infectious bovine rhinotracheitis, parainfluenza 3, leptospirosis, and brucellosis in white-tailed deer in Minnesota. Journal of Wildlife Diseases 22: 83–86.

KHAN, M. A., S. M. GOYAL, S. L. DIESCH, L. D. MECH, AND S. H. FRITTS. 1991. Seroepidemiology of leptospirosis in Minnesota wolves. Journal of Wildlife Diseases 27: 248–253.

MARLER, R. J., J. E. COOK, A. I. KERR, AND S. M. KRUCKENBERG. 1979. Serological survey for leptospirosis in coyotes in northcentral Kansas. Journal of the American Veterinary Medical Association 175: 906–908.

MECH, L. D., G. D. DEL GIUDICE, P. D. KARNS, AND U. S. SEAL. 1985. Yohimbine hydrochloride as an antagonist to xylazine hydrochloride-ketamine hydrochloride immobilization of white-tailed deer. Journal of Wildlife Diseases 21: 405–410.

MECH, L. D., K. E. KUNKEL, R. C. CHAPMAN, AND T. J. KREEGER. 1990. Field testing of commercially manufactured captive collars on white-tailed deer. The Journal of Wildlife Management 54: 297–299.

NIXON, C. M. 1970. Leptospirosis—Are deer to blame? *In* Animal disease trends. Ohio Department of Agriculture/Ohio Department of Health, Cleveland, Ohio, pp. 1–3.

SHOTTS, E. B., AND F. A. HAYES. 1970. Leptospiral antibodies in white-tailed deer of the southeastern United States. Journal of Wildlife Diseases 6: 295–298.

TRAINER, D. O., L. KARSTAD, AND R. P. HANSON. 1961. Experimental leptospirosis in white-tailed deer. Journal of Infectious Diseases 108: 278–286.

WEDMAN, E. E., AND F. C. DRIVER. 1957. Leptospirosis and brucellosis titers in deer blood. Journal of the American Veterinary Medical Association 130: 513–514.

ZARNKE, R. L., AND W. B. BALLARD. 1987. Serological survey for selected microbial pathogens of wolves in Alaska, 1975–1982. Journal of Wildlife Diseases 23: 77–85.

Received for publication 3 June 1991.