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Incidence of Some Disease Antibodies in Iowa Deer¹

Abnold O. Haugen²

Abstract. Serum samples from white-tailed deer (Odocoileus virginianus) in Iowa were checked for various disease antibodies from 1959 through 1963. Leptospirosis antibodies were found in 9.5% of 369 sera checked. L. grippotyphosa (3.9%), L. pomona (3.6%) and L. autumnalis (2.8%) were found most frequently. L. ballum, L. hardjo, L. sejroe, and L. autralis were also noted. No evidence of brucellosis was found in 231 sera checked. Evidence for western or California strains of encephalitis was noted. Anaplasmosis was also present. Johne's disease antibodies were not found in a sample of deer from western Iowa.

All populations of game animals have annual increases and losses. Losses result from a wide variety of causes. Our discussions today, however, will deal only with information on disease, tests for disease antibodies and information from autopsies on deer in Iowa.

A search of the literature has not been made to learn if disease caused any losses of deer between the late 1800's, when white-tailed deer were reintroduced in Iowa, and the early 1950's. This paper, therefore, reflects mainly what has been learned about disease antibodies in Iowa deer since 1950. There are no records to show that the Iowa State University Diagnostic Laboratory necropsied any deer between 1946 and 1950 (Table 1). The deer population in Iowa at that time was still rather small, and little chance for detecting mortality existed under those conditions. Five deer were checked in the period 1951-53.

The Unit annual report for 1954-55 notes that four deer had been brought in for autopsy. Cause of death of one was diagnosed as enterotoxemia. One checked in August 1955 was found with encepholomalacia. Seven specimens submitted between Aug. 20 and Nov. 18, 1956, showed various symptoms, including some hemorrhagic enteritis and some hemorrhages in musculature, pericardial sac, and/or on the heart. Three of these were suspected of having died of enterotoxemia, and one of algae poisoning. Dr. Hendrickson stated in 1957 that "a disease, possibly an epizootic hemorrhagic disorder, reduced deer numbers in scattered areas during the summer of 1956." This was about the same vear in which Frank Heidelbauer, State Conservation Commis-

¹ Journal Paper No. J-5698 of the Iowa Agricultural and Home Economics Experi-ment Station, Ames, Iowa. Project No. 1395. Jointly financed by the Iowa Cooperative Wildlife Research Unit, the Bureau of Sport Fisheries and Wildlife (U.S. Dept. Inter-jor), Iowa State University of Science and Technology, Iowa State Conservation Com-mission, and the Wildlife Management Institute. Part of the work was done in connec-tion with a National Science Foundation Undergraduate Research Participation grant.

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DEER DISEASES

79

sion pilot, observed a number of dead deer during his flights at low levels in western Iowa.

No evidence is available to indicate any occurrence of disease outbreaks or unusual mortality in deer in Iowa in the period 1957 through 1963. However, there have been a number of autopsies of deer at the Diagnostic Laboratory. Two of these were brought in because they were "queer" acting. Both had brain abscesses. A third had a "softening" of the brain due to a previous skull fracture. Bacterial infection in the rib section was found in two specimens. Streptococcus infection of the foot was found in one. arthritis of the hock joint was found in one, and five had suffered traumatic injuries of some sort. Six fawns that had been held in captivity died from different causes, some had scours. One fawn had suffered mower cuts and had an infection about the navel. One game-farm yearling male, autopsied by Eldie Mustard of the State Conservation Commission, showed some suspicious hemorrhages and some enteritis. A 6-month buck, killed with a hammer because it chased a farmer's dog, evidently was only acting like any normal buck ought to behave in late November. The rut was in progress. No evidence of disease was found.

Evidence of past experience of animals with disease may be determined through testing for antibodies. Such evidence is mostly lacking and not well understood in wild animals. The presence of antibodies of certain diseases can be detected, however, even though the seriousness of the animal's past experience with the disease is unknown. Obviously, such an approach only provides information on animals that have been infected and have survived.

A good general discussion on leptospirosis in wildlife is presented by Roth et al. (1961). Fay (1961) published a similarly good summary of brucellosis in deer. The availability of these publications makes it unnecessary to here repeat general information on these diseases.

MATERIALS AND METHODS

Blood samples for the study were secured from 1959 through 1963. The samples from 1959-61 were collected mainly by dipping a vial into any pool of blood found in the body cavity when hunters brought their deer to checking stations operated at designated locations in the state. Samples at the DeSoto Bend Refuge in 1962 and 1963 were taken directly from the aorta with a hypodermic syringe by biologists who gutted the deer as part of the operational procedure at the checking station. The samples were allowed to clot; then the serum was removed by centrifuging and stored frozen at about -20°F. until tested.

Tests for brucellosis were performed by Drs. Frank C. Stiles,

IOWA ACADEMY OF SCIENCE [V

Jr., of the Brucellosis Unit of the ADE Diagnostic Laboratory and George Lambert of the National Animal Disease Laboratory, Ames, using the 1:25 brucellosis plate test.

Information on antibodies of Leptospira was secured by the microscopic agglutination test performed by personnel at the National Animal Disease Laboratory, Ames, including Drs. Edward A. Carbrey, D. E. Hughes, and O. H. V. Stalheim.

Tests for encephalitis and vesicular stomatitis were performed by Dr. Daniel O. Trainer of the University of Wisconsin by the Hela cell metabolic-inhibition test. Sera for those tests were packed in dry ice and airmailed from Ames, Iowa, to Madison, Wisconsin.

The tests for Johne's disease antibodies were performed with an experimental antigen developed and used by Dr. Aubrey B. Larsen of the National Animal Disease Laboratory, Ames. Dr. Larsen also made direct observation on sections of the gut of many deer, looking for lesions of the disease.

RESULTS

Leptospirosis. During 1959-63, 285 samples of deer sera collected during December hunting seasons were tested for Leptospira antibodies; 9.8% were positive (Table 2). L. pomona had an incidence of 3.6%. Other serotypes involved were L. grippotyphosa 3.9%, L. autumnalis 2.8%, L. ballum 1.1%, L. hardjo 1.1% and L. sejroe 0.7%. Of the 28 animals showing a positive test, there was a cross-reaction with a second serotype in 8 tests, with the cross-reaction most commonly involving pomona and autumnalis.

Table 1. Deer examined at Iowa Veterinary Diagnostic Laboratory up through 1956.*

Year and N	lum-		
date	ber	Origin	Diagnosis
1946-'50	Non	е	
1951 June 18			Malnutrition
		Marshalltown	Blackleg
Sept. 26 1953 Feb. 11	5	Fort Dodge	Rabies-negative
			Corynebacteriosis
Nov. 16		Des Moines	Toxemia
1954 Jan. 4	3	E. L. Kozicky, I.S.U.	Enterotoxemia
Nov. 12	2	"	Traumatism
Nov. 28	}	Jewell	Decomposed
1955 Aug. 15	i 1	E. L. Kozicky, I.S.U.	Encephalomalacia
1956 Aug. 20) 7	Boone	Enterotoxemia
Sept. 11		Muscatine	Bacteriological examnegative
Sept. 17	,	Des Moines	Poisoning, algae
Sept. 19)	Fort Dodge	Rabies-negative
Oct. 8		I.S.U.	Enterotoxemia
Oct. 31		Boone	Polyarthritis
Nov. 18	}	Jefferson	Enterotoxemia

* Based on a search of old records by Dr. Paul Bennett in May, 1963.

				Number					Serotype a	nd titer		
Year	Location	Age	Sex	sera samples	Number negative	Number positive	L. pomona	L. sejroe	L. grippo- typhosa	L. hardjo	L. autumnalis	L. ballum
1959	Allamakee and											
	Cherokee Cos.			35	29	6	Т	Т	Т		Т	
	Allamakee Co.	11/2	\mathbf{F}				1/400					
	"	11/2	M					1/400				
	"	$1\frac{1}{2}$	M				1 / 100		1/400		1 / 100	
		21/2	M				1/400				1/400	
	Cherokee Co.		r F				1/400	1 / 400				
1960	Allamakee Co.	21⁄2	г	30	22	8	Т	1/400 T	Т		Т	т
1900	Anamakee Co. $''$	F	\mathbf{F}	30	22	0	1	I	1/100		1	1
	"	$1^{1}/_{2}$	Ň				1/1000		1/100		1/100	
	//	$1\frac{1}{2}$	M				1/1000				1/100 $1/100$	
	"	11/2	M								1/100	
	"	21/2	M				1/100				1/1000	
1960	Allamakee Co.	31/2	Μ				1/1000				1/1000	
	"	31⁄2	\mathbf{F}						1/100		1/100	
	"	31⁄2	Μ				1/100				1/100	
1961	"			19	18	1	Т		Т	Т	Т	Т
	"	41⁄2	\mathbf{F}							1/400		

Table 2. Serotypes and titer for serotypes of Leptospirosis as indicated from blood samples routinely secured from deer shot in December hunting seasons in Iowa and the adjacent Nebraska portion of the DeSoto Bend National Wildlife Refuge (microscopic agglutination tests).

18

1967]

Table 2. (Continued.)

				Number					Serotype a	nd titer	_	~
Year	Location	Age	Sex	sera samples	Number negative	Number	L. pomona	L. sejroe	L. grippo- typhosa	L. hardjo	L. autumnalis	L. ballum
1961	Lucas Co.	1180		22	14	8	pointina T	30,100	T	T	T	T
1001	Lucas Co.	\mathbf{F}	\mathbf{F}	22	14	0	1		1/100	-	-	-
		F	Μ						1/100			
		년 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1	M M						$\frac{1}{100}$ $\frac{1}{400}$			1/100
		г F	F						1/400 1/400	1/100		1/100
		F	\mathbf{F}						1/100			
		31/2	М							1/100	1 (100	
1962	DeSoto Bend	31⁄2	М								1/100	
1002	Refuge			109	104	5	Т		т	Т	Т	Т
	0	F F 2½	Μ						1/20			
		F 014	F				1 /000		1/20			1/20
1962	DeSoto Bend	$\frac{2}{71/2}$	F F F				$\frac{1}{200}$ $\frac{1}{20}$					1/20
1001	Refuge	• / 2	_				1/20					1/20
1000	"	?	\mathbf{F}									
1963 Total	positive for single	a corotra	n	70	70	0	T	2	$\frac{1}{8}$	$\frac{1}{2}$	3	1
	positive for two						$\frac{4}{5}$	2	3	1	5	$\frac{1}{2}$
	totals	50100,000	5	285	257	28	9	2	11	3	8	$\frac{2}{3}$
Percer	nt positive					9.8	3.6	0.7	3.9	1.1	2.8	1.1
1960-6	33 and 1961			also te	sted for a	icterohem iebdomad	orrhagiae, canio	cola, aust	ralis, pyroge	nes, bata	<i>viae</i> and <i>hy</i>	08.
1962	and 1901				sted for n		ıs.					
1963							shown with a '	"Т".				

1967]

DEER DISEASES

Fifteen deer killed accidentally during 1960-61 in scattered locations of Iowa, when tested, were found positive for *L. australis* -1, *L. grippotyphosa*-1, *autumnalis*-1, and 1 showed cross-reaction between *L. pomona* and *L. autumnalis* (Table 3).

Table 3. Serotypes and titre for Leptospirosis as indicated by blood samples from deer accidentally killed in various months and locations in Iowa.

Year	Location		Posi- tive	L. pomona	L. grippo- typhosa	L. australis	L. au- tumnalis
1/10/60	Johnson	1					
2/15/60	Fayette	1					
2/19/60	Polk	1					
3/8/60	"	1					
3/17/60	11	1					
3/24/60	Allamakee	1					**
4/7/60	Greene	1					
6/20/60	Story	1					
7/27/60	Hamilton	1					
9/7/60	Boone	1					
10/21/60	"		1			1/100	
11/12/60	Iowa		1	1/1000			1/1000
11/16/60	Johnson	1					
11/21/60	- <i>//</i>	1					
11/28/60	Story	1					
12/20/60	Iowa		1				1/100
3/26/61	Johnson	1					
3/30/61	Boone		1		1/100		
8/6/61	Bremer	1					
Total de	eer	15	4				

Conservation Officer Harlan Frankel (1960), with assistance from local veterinarians, made tests for *L. pomona* on 32 blood samples from Clayton County in 1958 and on 37 from Clayton (14), Winneshiek (6), Jackson (11), Fayette (5), and Delaware (3) counties in 1959. All the 1958 samples were negative, but 3 (2 bucks and 1 doe) of the 1959 samples were positive for *L. pomona*.

An overall occurrence of leptospirosis antibodies of 9.5% is indicated when the 369 samples of Iowa deer from this study and those of Frankel are considered collectively.

Bennett (1964), who checked 2,298 samples of blood secured from deer hearts collected in the 1963 December hunting season in Iowa, found 91 reactors (3.9%) for *L. pomona*. He did not check for other serotypes of leptospirosis.

In Carroll County, Illinois, Ferris and Verts (1964) checked 319 sera and found that 10% (32) of the dear reacted with one or more antigens of Leptospira serotypes. Wisconsin studies indicate approximately 28% of the deer tested (Trainer and Hanson, 1960) were positive for *L. pomona* but the incidence varied considerably from area to area, with the higher prevalence in dense populations.

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84.

IOWA ACADEMY OF SCIENCE

7

Tests for L. pomona on 190 deer mainly from the wildland area of the upper part of Michigan's Lower Peninsula showed 26.3%positive (Youatt et al., 1959). This part of Michigan supports its densest deer population.

Suggestions were made by each of the authors from Wisconsin, Michigan and Illinois that Leptospiral infections due to *L. pomona* were related to the population density of deer. This may not be true for Iowa. As an example, only 2 *L. pomona* reactors were found in a sample of 109 tests from 188 deer harvested from a herd estimated at 600 animals on a 2500-acre "island area," the DeSoto Bend National Wildlife Refuge in 1962. A year later, there were no reactors in a sample of 70 sera from 92 deer killed on the same area.

The numbers of reactors are too few for conclusions, but there is evidence that both sexes and all age groups, including fawns, are susceptible.

Brucellosis. From 1958 through 1962, 231 blood samples taken from Iowa deer during the December hunting seasons were checked for the presence of antibodies of Brucella abortus. All 231 were negative (Table 4). An additional 13 samples taken from deer killed accidentaly in various seasons of the year were also negative (Table 5).

		Number		
		sera	Test	Results
Date	Location	samples	No. —	No. +
1958	Clayton Co.	32	32	0
1959	NE Iowa	37	37	0
1960	Various counties	12	12	0
1960	Lansing and			
,	Allamakee Cos.	25	25	0
1960	Cherokee	6	6	0
1961	Allamakee and			
	Lucas Cos.	41	41	0
1961	Bremer Co.	1	1	0
1962	DeSoto Bend	77	77	0
		231	231	0

Table 4. Results of plate tests for antibodies of Brucellosis in sera from Iowa deer.

Bennett (1964), who ran tests on blood samples from the hearts of 2,298 deer taken from throughout Iowa in the 1963 hunting season, also found all sera negative. Iowa tests are in agreement with findings of Fay (1961) who concluded that brucellosis is of no importance and almost non-existent in white-tailed deer. His conclusion was based on results from 12,706 white-tailed deer tests in 24 states, with only 20 (0.16%) deer blood samples reacting.

Encephalitis. Eighty-seven serum samples from deer taken in the December 1962 and 1963 hunting seasons on the Nebraska Published by UNI ScholarWorks, 1967

85

1967]

DEER DISEASES

	No. sera	Test Results
Date Location	samples	No. – No. +
4/7/60 Greene	1	1 0
4/7/60 Boone	1	1 0'
4/14/60 Hamilton	1	1 0
6/20/60 Story	1	1 0
9/7/60 Boone	1	1 0
10/21/60 Boone	1	1 0
11/15/60 Johnson	1	1 0
11/12/60 Iowa	1	1 0
11/21/60 Johnson	1	1 0
3/26/60 Johnson	1	1 0
3/30/60 Boone	1 .	1 0
12/20/60 Iowa	1	1 0 🗄
8/6/61 Bremer	1	1 0
	13	13

Table 5.	Results of plate tests for antibodies of Brucellosis in blood sam-
	ples secured from Iowa deer during various seasons of the year.

portion of the DeSoto Bend National Wildlife Refuge, on the east side of the Missouri River, were checked for various strains of encephalitis. Two male fawns of 67 deer from 1962 showed antibodies for western viral encephalitis, but were negative for eastern, St. Louis, and California strains. In 1963, 6 (1 female and 5 males, all 1½ years and older) of 20 deer sampled were positive for the California strain, but negative for eastern, St. Louis, and western strains.

Sera from 12 deer taken near Burlington in Des Moines County in southeastern Iowa were negative for encephalitis.

Vesicular Stomatitis. All of 20 DeSoto Bend and 12 Burlington samples of blood serum tested for vesicular stomatits were negative.

Anaplasmosis. Bennett (1964), using the capillary agglutination test on 2298 Iowa deer heart blood samples from the 1963 season, found 91 (3.9%) reactors for anaplasmosis. The positive animals came from scattered locations throughout the state.

Johne's Disease. In a check of 50 intestinal specimens and 91 serum samples from the 1962 DeSoto Bend deer season, no evidence was found of *Mycobacterium paratuberculosis*, the organism that causes Johne's disease (Larsen, 1963).

Chlorinated Hydrocarbon Insecticides in Deer. Body fat from about half a dozen white-tailed deer shot on the DeSoto Bend National Wildlife Refuge in 1962 was sent to the Denver Wildlife Research Center. Analysis at that center indicated the presence of DDE in a concentration of less than 0.5 p.p.m.

Acknowledgments

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86

IOWA ACADEMY OF SCIENCE

[Vol. 74

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