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AN EPIZOOTIC OF HEMORRHAGIC DISEASE IN WHITE-TAILED DEER (*ODOCOILEUS VIRGINIANUS*) IN MISSOURI: NECROPSY FINDINGS AND POPULATION IMPACT

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ABSTRACT: An epizootic occurred among white-tailed deer (*Odocoileus virginianus*) from July through October 1988 in Missouri (USA). From late July through September, nine necropsied deer had lesions of the peracute or acute forms of hemorrhagic disease (HD) or no apparent lesions, whereas two deer necropsied in October had lesions of the chronic form of HD. Epizootic hemorrhagic disease virus was isolated from two necropsied deer. Based on changes in population indices, there is evidence that deer populations declined in seven of Missouri's 57 deer management units from 1987 to 1990. Based on a deterministic model designed to simulate deer populations in management units, it appeared that summer and fall 1988 mortality ranging from 6% to 16% accounted for the population decreases in deer management units with population declines. Heavily hunted areas where high deer mortality was not reported in the summer and fall of 1988 did not have population declines. Based on these results, we believe that HD mortality was high and resulted in deer population declines in parts of Missouri when combined with hunting harvest.

Key words: White-tailed deer, *Odocoileus virginianus*, hemorrhagic disease, bluetongue, epizootic hemorrhagic disease, mortality, populations.

INTRODUCTION

Hemorrhagic disease (HD) has been recognized as a cause of mortality of white-tailed deer (*Odocoileus virginianus*) and other wild ruminants in the United States since 1955 (Nettles and Stallknecht, 1992). The disease occurs in late summer and early fall and is caused by bluetongue virus (BLU) or epizootic hemorrhagic disease virus (EHD) serotypes. Biting midges, *Culicoides variipennis*, are capable vectors (Jones et al., 1977).

Peracute, acute, and chronic forms of HD have been identified by gross lesions (Prestwood et al., 1974) which are attributable to viral replication within vascular endothelium (Tsai and Karstad, 1973). The resulting vascular damage and thrombosis cause hemorrhage and ischemic tissue necrosis. Virus usually is isolated from <25% of deer necropsied during HD epizootics (Nettles et al., 1994); therefore, diagnosis frequently is made on the basis of gross or microscopic lesions.

The impact of HD on free-ranging white-tailed deer populations is difficult to assess. In the southeastern United States, infection with BLU or EHD usually results in fairly mild disease with little mortality, whereas epizootics in the midwest and northeastern regions may be characterized by high mortality (Nettles et al., 1992). Although morbidity and mortality have been estimated during HD epizootics in captive herds and in national parks (Prestwood et al., 1974), scant information is available concerning the effect on free-ranging populations. Mortality rates appeared high in dense populations of free-ranging deer (Swenson, 1979).

Epizootics of HD were reported in Missouri (USA) in 1976 and 1980 (Brannian et al., 1983). During the summer and fall of 1988, HD was identified in white-tailed deer in 26 states (Nettles and Stallknecht, 1992). In Missouri, over 1,400 suspected cases of HD were reported in white-tailed deer. Furthermore, following the 1988 firearms deer season, there was an un-

precedented number of reports from Missouri Department of Conservation (MDC) agents and concerned citizens citing reduced deer numbers in parts of Missouri. Based on these reports, we believed that the HD epizootic may have had a significant impact on deer populations. Our objectives were to identify the causative agent, determine the pathology among deer submitted during this epizootic, and assess the impact of the disease on selected deer populations in Missouri.

MATERIALS AND METHODS

Necropsies were performed on white-tailed deer submitted to the University of Missouri Veterinary Medical Diagnostic Laboratory (VMDL) by private deer herd owners and MDC personnel. The distribution of submitted deer by county was Audrain County (39°10′N, 91°55′W), n = 1; Laclede County (37°30′N, 92°40′W), n = 1; Macon County (39°50′N, 92°15′W), n = 2; Moniteau County (38°40′N, 92°15′W), n = 1; Randolph County (39°25′N, 92°15′W), n = 6. During gross examination, tissues were collected for microscopic examination and microbiologic studies. Blood samples were obtained from any deer presented alive.

Tissues were fixed in phosphate-buffered formalin, embedded in paraffin, sectioned at 5 μm, stained with hematoxylin and eosin, and examined by light microscopy. Tissue samples were submitted for culture of bacterial pathogens on the basis of gross lesions. Aerobic and anaerobic bacterial cultures were performed according to procedures described by Carter (1984). Serum was separated from blood samples and submitted to the National Veterinary Services Laboratory (NVSL), Ames, Iowa (USA) for serologic studies. Serum samples were tested for antibodies to BLU and EHD by agar gel immunodiffusion (AGID) (Pearson and Jochim, 1979). Serum from one deer also was tested by serum neutralization (Pearson et al., 1991).

Lung, spleen, and heparinized blood were submitted for virus isolation to the NVSL. Tissue suspensions were sonicated and inoculated onto monkey kidney (Vero-M) cells (American Type Culture Collection, Rockville, Maryland, USA) and baby hamster kidney (BHK-21) cells (American Type Culture Collection), and into embryonating chicken eggs by the intravenous route. Two serial passages were made in each cell culture system. After the last passage in Vero-M and BHK cell cultures, the cells were stained with BLU and EHD fluorescent antibody conjugates (National Veterinary Services Laboratory, Ames, Iowa, USA). After one passage in embryonating chicken eggs inoculated by the intravenous route, suspensions of the dead embryos and all live embryos were passed into embryonating chicken eggs by the egg sac route and onto Vero-M cells. The Vero-M cells were stained with BLU and EHD fluorescent antibody conjugate. Viral isolates were typed by the antibody disc neutralization test (Stott et al., 1978).

Mortality due to HD and the impact on white-tailed deer populations in Missouri were evaluated from late July 1988, when the first suspected HD mortalities were reported, through December 1990. Information on the distribution and relative number of suspected HD cases was derived from MDC conservation agent annual reports of confirmed deer mortality, and responses to a request for MDC agents to submit unconfirmed, but reliable public reports of unexplained deer mortality during summer and fall of 1988. Gender was not available for most deer in these reports.

Deer population indices including hunter success rates, antlered deer harvest, and road kills were compiled from hunter-kill data and deer death reports submitted by MDC agents from 1986 to 1990. There were 57 deer management units in Missouri, and a quota of any-deer permits that allowed the taking of antlerless deer during the firearms season was issued in each of these units. Bonus anterless-only permits were issued in units where the quota of any-deer permits was undersubscribed. Permits to take antlered deer were not limited to a quota. Successful deer hunters were required to submit their deer to a check station where sex and age of the deer, kill location, and permit type data were collected. Annual hunter success equalled the number of deer taken by permit type divided by the number of permits issued for a unit. Impact of the kill on antlered deer, as reflected in age ratios in killed animals, varied little during the study period, so antlered deer kill totals served as a population index.

Road-killed deer were reported to MDC agents or Missouri Highway and Transportation Department personnel, both of whom submitted records to MDC Wildlife Research Section for compilation and analysis. Roadkills were standardized for annual vehicle miles traveled (Anonymous, 1987, 1988, 1989, 1990), and summarized by unit.

Percentage change in the population indices for each year from 1987, the base year, through 1990 was calculated on a statewide basis as well as for individual deer management units. The changes between 1987 and 1988 were believed to reflect the immediate impact of HD and the 1987 to 1990 trends were believed to reflect the

long-term effects of HD combined with liberal hunting seasons.

A deterministic model simulating deer populations (Nixon et al., 1991) was used to assess the extent of HD mortality and population decreases in seven deer management units with apparent population declines. Input for the model included reproductive rates collected from road-killed females, non-hunting mortality rates determined by monitoring deer marked with radio transmitters (Hansen and Beringer, 1990), and annual kill during hunting seasons derived from information collected at mandatory check stations (L. P. Hansen, unpubl.). Density-dependent reproduction and mortality were not incorporated into the models. Based on high reproductive rates and low mortality rates (Hansen and Beringer, 1990), we inferred that deer in most units were well below carrying capacity (McCullough, 1984). Sex and age of deer suspected of dying from HD were not available; thus, for modeling purposes, HD was assumed to affect sex and age classes equally.

The starting number of deer used in the model for each unit was varied in repeated runs until the simulation best matched independent population trend information such as hunter success, road kills, and annual harvest. The ratio of the number of antlered bucks actually taken during the hunting seasons to the preseason number of antlered bucks predicted by the simulation also served as a measure of how well the simulation was performing. Once a matching combination of population trend and percentage antlered buck harvest was achieved for a unit, the only input variable that was changed annually was harvest.

RESULTS

Eleven deer, ranging from 3 mo to 4 yr in age, were submitted for necropsy from 28 July through 31 October 1988. The deer came from mid-Missouri, in part a reflection of the short driving distance to the VMDL. Four deer were free-ranging and seven were captive. Hemorrhagic disease was suspected on the basis of clinical history or gross lesions. Clinical illness was characterized by severe depression, anorexia, blood-tinged oral and nasal discharge, and severe respiratory distress with death occurring within 48 hr of the onset of clinical signs.

Necropsy findings were variable. Hydrothorax and pulmonary edema (n = 5) were the most common gross lesions in the nine deer submitted through September. Other lesions among these animals included hemorrhagic enteritis, abomasitis or rumenitis (n = 4); submandibular edema (n = 2); multifocal hemorrhage (n = 1); and mucopurulent tracheitis (n = 1). Microscopically, in addition to changes consistent with gross lesions, there were necrotizing glossitis (n = 2), centrilobular hepatocellular necrosis (n = 1), and interstitial pneumonia and lymphocytic vasculitis (n = 1). Three deer submitted during this period lacked significant gross or microscopic lesions. The two deer submitted in October had chronic glossitis and stomatitis (n = 2), multifocal hemorrhages and abscessed hooves (n = 1). One of these deer had lymphocytic vasculitis and interstitial myositis.

Microbiologic findings included the isolation of *Actinomyces pyogenes* from the pneumonic lung of the deer with mucopurulent tracheitis, and *Clostridium perfringens* from small intestines of deer with hemorrhagic enteritis. A virus identified as EHD, serotype 2 (EHD-2), was isolated from the deer with tracheitis, and an untyped EHD was isolated from one deer with acute and chronic HD lesions; virus isolation results were negative from the remaining nine deer. The serum of five of six deer tested by AGID contained antibodies against EHD or BLU and serum neutralization results from the deer from which EHD-2 was isolated were positive for antibodies to BLU, serotype 11 at 1:20 and to EHD-2 at 1:160.

Based on public reports collected by MDC agents, 1,457 cases of unexplained deer mortality occurred in 71 of 114 counties, with 28 counties having reports of ≥20 dead deer. Counties reporting ≥20 dead deer were scattered throughout the state, but were concentrated within east-central (90°30′N to 92°15′N, 38°5′W to 39°10′W) and southwestern Missouri (36°35′N to 37°10′N, 91°55′W to 94°5′W). Statewide, there were 758 reports of unexplained deer mortality confirmed by MDC agents in 1988, compared to averages of

only 189 and 156 annually from 1985 to 1987 and from 1989 to 1992, respectively. The increase in reported unexplained deer mortality in 1988 was four- to five-fold statewide, and one- to 165-fold among individual deer management units. These results may serve only as gross measures of regional differences in mortality because reporting varied among agents.

When percentage change in deer population indices was calculated, there was a statewide decrease in four indices between 1987 and 1988 (Table 1) despite steady increases during the preceding years. Percentage decreases from 1987 to 1988 were greater in the population indices of seven deer management units when compared to statewide indices (Table 1). These seven deer management units comprised two separate blocks (Fig. 1) located within regions of the state that reported ≥20 dead deer per county for summer and fall of 1988 and from which numerous reports of fewer deer were received. When changes in population indices from these seven units were examined over the period from 1987 through 1990, a downward population trend was observed (Table 1). Moderate decreases in population trend indices occurred in some other deer management units, but those units were not included in the detailed analysis because of smaller long-term effects.

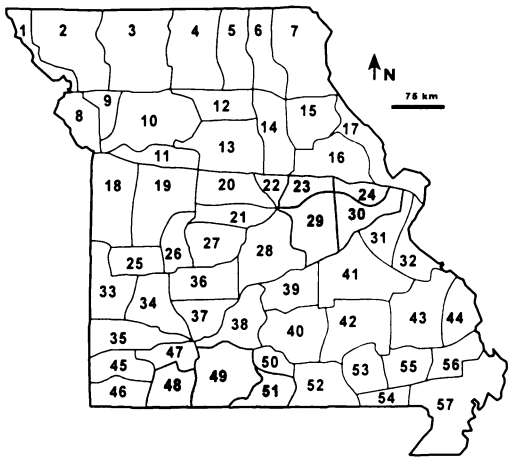

FIGURE 1. White-tailed deer population indices in the shaded deer management units had a greater decrease than statewide indices from 1987 to 1988 and had apparent population declines from 1987 to 1990. Numbers refer to deer management units.

Based on results of deterministic population modeling, HD mortality of 16% occurred among white-tailed deer in units 23, 24, 28, 29, 48; 6% in unit 49; and 10% in unit 51. From 1987 to 1988, simulated deer populations in the seven units decreased by 12% to 23%, whereas the simulated statewide population increased by 1% according to the model (Table 1). De-

TABLE 1. Changes in deer population trend indices and simulated populations in Missouri deer management units.

Deer management unit	Percentage change from 1987 to 1988					Percentage change from 1987 to 1990				
	Hunter success		Antlered harvest	Road-killed deer	Simulated population	Hunter success		Antlered harvest	Road-killed deer	Simulated population
	Any-deer	Bonus				Any-deer	Bonus			
23	−30	−5	−12	−34	−16	−43	−12	−23	−31	−23
24	−30	−24	−15	−30	−19	−38	−32	−32	−35	−23
29	−28	−11	−24	−35	−23	−23	−14	−28	−47	−31
30	−27	−15	−21	+18	−23	−30	−21	−28	+14	−33
48	−27	—	−21	−22	−12	−27	—	−5	−43	−10
49	−38	−27	−35	−39	−14	−29	−21	−15	−31	−18
51	−35	−6	−24	−17	−12	−40	−9	−26	−43	−18
Mean of units 23, 24, 29, 30, 48, 49, 51	−30	−14	−22	−23	−14	−32	−19	−22	−31	−19
Statewide	−16	−7	−7	−6	+1	−21	−9	+5	−14	+4

creases of 10% to 31% in simulated deer populations in the seven affected units (Fig. 1) were observed from 1987 through 1990, although the simulated statewide population increased by 4% during the same period (Table 1). The positive values of the simulated statewide populations during periods with decreasing population trend indices reflected the inclusion of data other than these indices in the input for the model.

DISCUSSION

Hemorrhagic disease activity in Missouri was confirmed by isolation of EHD from two white-tailed deer necropsied during the epizootic. Diagnosis of HD by necropsy findings was complicated by the variability of gross lesions in individual animals, as well as by the paucity of the typical histologic lesions of endothelial swelling, thrombosis, fibrinoid necrosis, and vasculitis (Howerth and Tyler, 1988). In general, gross lesions in deer submitted through September resembled the peracute and acute forms of HD. Deer submitted in October had lesions of the chronic or acute forms of HD. However, three deer had no apparent gross lesions and a fourth had tracheitis, but lacked gross lesions suggestive of HD. One of the two EHD isolates came from the animal with tracheitis.

Results of virus isolation attempts and serologic studies were consistent with those recorded during other epizootics. The low percentage of virus isolation typified the difficulty encountered in confirming the diagnosis of HD and was consistent with the diagnostic efforts of other investigations in which virus was isolated from only 57 (23%) of 252 deer necropsied during HD epizootics (Nettles et al., 1994). Antibodies against EHD or BLU commonly were found during and after HD epizootics, but also were found in healthy deer in areas not reporting HD (Couvillion et al., 1981; Stallknecht et al., 1991). Therefore, although the ability to identify previous exposure to the viruses may be helpful, the use of serologic tests as a diagnostic aid was limited.

Hemorrhagic disease was diagnosed in seven deer by gross lesions and in one deer by isolation of EHD-2. Although this latter animal had microscopic lesions of HD, the isolation of virus in the absence of gross HD lesions is evidence that clinical HD may occur in the absence of typical gross lesions. Virus isolation results were negative on three deer without HD lesions. Lesions in one of these deer may have been obscured by gunshot wounds. Hemorrhagic disease was suspected in these deer because of clinical signs, HD activity in the area, and their presentation during the time of year that is associated with HD and is used as a criterion for HD surveillance (Nettles et al., 1994), but could not be diagnosed by lesions or virus isolation.

Decreases in hunter success and antlered deer harvest from 1987 to 1988, as well as from 1987 to 1990, could not be attributed to poor hunting conditions or low hunting pressure as conditions were good and hunting pressure was high in 1988 through 1990. Thus, we believe that fewer deer were present in the seven deer management units at the start of the firearms season in 1988 through 1990 than at the start of the 1987 season.

The apparent declines observed in the seven deer management units from 1987 through 1990 could be explained by a heavy kill during the hunting seasons, or by the 1988 HD epizootic. Simulated deer populations in the units with apparent population declines from 1987 to 1990 increased or remained stable when no summer and fall mortality in 1988 due to HD was included in the model and therefore, did not match observed field population trend information. Hemorrhagic disease was not reported in Missouri from 1985 through 1987, 71 counties reported HD in 1988, one county reported HD in 1989, and none was reported in 1990 (Nettles and Stallknecht, 1992). Allowable deer kill in most of Missouri, especially of females, was liberalized between 1985 and 1990 in

an attempt to slow or stop growth of the deer herd. This was especially true in east-central Missouri and in the agricultural region across the northern third of the state. However, the apparent declines after 1987 in the seven units in east-central and southwestern Missouri did not occur in the heavily hunted agricultural units in northern Missouri, or in other parts of the state with heavy kills during the hunting season. Thus, we propose that HD mortality in 1988 contributed to the declines.

Models must be regarded as tools that are inherently deficient to some extent due to necessary simplification (McCullough, 1984). The performance of our model was dependent on the accuracy and precision of the input, and although data used as input were collected in Missouri, temporal and regional variability were not always measured. We believe that the results of the simulations provide insight into the population processes, but specific changes as defined by the simulations should be regarded with caution.

Although direct measures of mortality due to HD in Missouri in 1988 were not available, the evidence from trend information and population simulations was that mortality was high in parts of Missouri and may have caused deer population declines when combined with hunting kill. Measures of population impacts rarely have been reported, but similar evidence of high HD mortality has been demonstrated in free-ranging (Swenson, 1979) and in captive (Roughton, 1975) white-tailed deer. State agencies should be aware of the potential population impact and the possible need to adjust hunting mortality.

This epizootic of HD may be evidence that the effects of disease on wildlife populations can be far more severe and widespread than indicated by the reporting and investigation of mortality cases. Based on simulated figures from the management units experiencing population declines, we believe that more than 14,000 deer died in the small portion of Missouri represented by the seven deer management units. Simulated figures for statewide mortality due to HD were not available, but were assumed to be much greater than the 14,000 of the seven management units. Only 1,457 cases of unusual deer mortality were reported by the public, of which 758 were confirmed by MDC agents. Eleven deer were submitted for post mortem examination during the period of high mortality, but only four were free-ranging animals and the causative virus was isolated from only two deer.

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