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An Epizootic of Hemorrhagic Disease in White-tailed Deer in Missouri

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ABSTRACT: As part of a white-tailed deer (*Odocoileus virginianus*) survival study in Missouri (USA) we were actively monitoring 97 radio-collared deer when 8 (8%) died. This mortality, which occurred from 20 August to 23 September 1996, consisted of five adult females, two yearling females and one yearling male. Based on the seasonality of this mortality and the isolation of epizootic hemorrhagic disease virus (EHDV) serotype 2 from one of these animals, we believe that these losses resulted from an epizootic of hemorrhagic disease. The remains of five unmarked deer that may have died from HD also were found on the study area during this same period. During the fall following this mortality, we tested serum from 96 deer taken by hunters in the immediate area. Fifteen (16%) were positive for EHDV or bluetongue virus (BTV) antibodies as determined by agar gel immunodiffusion tests. Serum neutralization test results indicated that previous infections were caused by EHDV virus serotype 2. Based on these data, and assuming that there was no prior exposure to EHDV serotype 2 in this population, the exposure rate for this epizootic was 24% of which 8% died. We noted hoof interruptions in only two of the 96 deer sampled. During this mortality event, the Missouri Department of Conservation received no reports of dead deer, and without the radio-monitored animals the event would have been undetected.

Key words: Bluetongue, epizootic hemorrhagic disease, hemorrhagic disease, mortality, *Odocoileus virginianus,* white-tailed deer.

Hemorrhagic disease (HD) caused by viruses in either the bluetongue virus (BTV) or epizootic hemorrhagic disease virus (EHDV) serotypes is an important disease affecting white-tailed deer (*Odocoileus virginianus*) populations throughout much of their range (Nettles and Stallknecht, 1992). Because mortality rates as high as 84% (Fox and Pelton, 1973) have been reported, and infection rates, as determined by the presence of antibodies to these viruses, can approach 100% in some populations (Stallknecht et al., 1991), understanding potential impacts of HD on white-tailed deer populations is important for deer managers. HD-related mortality, especially when coupled with annual deer harvests, may result in harvest rates that are higher than desirable (Fischer et al., 1995). In addition, estimates of non-hunting mortality often are necessary components to many of the deer management models currently used to evaluate deer population size and structure.

Determining distribution and population impacts of HD is difficult, and in most cases these are based on mortality reports from public and resource professionals that likely underestimate mortality rates. Post-epizootic serologic testing of affected herds can help biologists identify the distribution, exposure rate, and specific BTV, or EHDV serotypes that were present during epizootics, but does not provide the mortality estimates needed by wildlife managers. Sloughed hooves or interrupted hoof growth from hunter-killed deer have been used as an indicator of disease occurrence (Couvillion et al., 1981), but like serology these observations provide limited information on exposure and no information on population impact. Population trend indices and population simulations have been used to estimate mortality caused by HD (Fischer et al., 1995), but such analysis would be difficult in cases of localized HD-related mortality or in cases where mortality rates were not excessive.

While conducting a deer survival study in Crawford and Phelps Counties in south central Missouri USA; (37°58′N, 91°21′W), utilizing white-tailed deer collared with radiotransmitters and mortality sensors, un-

expected mortality believed to be associated with HD occurred. This mortality event and subsequent serologic testing of deer in the study area allowed us to assess the possible impact of HD on this population of free-ranging deer. To our knowledge, this was the first direct measure of white-tailed deer mortality during an HD epizootic in a free-ranging white-tailed deer population.

All deer monitored during this study were captured at least 10 mo prior to the detected mortality. Deer were captured with rocket nets and modified Clover traps (Beringer et al., 2000) and all animals were fitted with radiotransmitters (Telonics, Mesa, Arizona, USA; Advanced Telemetry Systems, Isanti, Minnesota, USA) equipped with mortality sensors on a 4 hr switch. Following capture, deer were located every 40 hr. Monitoring frequency was increased to every 24 hr after the first two mortalities were detected, and responses to mortality signals were immediate. Because these mortalities occurred in late summer, we suspected HD as a probable cause of death and, where possible, samples of spleen and heparinized blood were submitted to the Southeastern Cooperative Wildlife Disease Study (SCWDS; Athens, Georgia, USA) for virus isolation.

Virus isolation was attempted using cattle pulmonary artery endothelial cells (CPAE; American Type Culture Collection, Rockville, Maryland, USA) as described by Quist et al. (1997). Isolates were identified by virus neutralization using antisera against all the North American EHDV and BTV serotypes (National Veterinary Services Laboratories, Ames, Iowa, USA).

Serum samples from 96 hunter-killed deer, harvested in the immediate area of observed mortality also were tested for antibodies to EHDV and BTV. For serologic testing, serum samples were screened using agar gel immunodiffusion (AGID) tests to both EHDV and BTV as described by the test manufacturer (Veterinary Diagnostic Technology, Inc., Wheatridge, Colorado, USA). All AGID-positive samples were tested by serum neutralization against all of the North American EHDV and BTV serotypes (supplied by National Veterinary Services) as described (Stallknecht et al., 1995). During these serum collections, deer also were examined for interrupted hoof growth, emaciation, or other signs of chronic HD.

At the time of the initial mortality ninety-seven deer were being radio-monitored, including 17 yearling females (<18 mo), 67 adult females, eight yearling males and five adult males. Of these, two died between 28 and 29 August 1996 and others were recovered on 1, 2, 9, 9, 12, and 23 September 1996. The percentage of radio-monitored deer that died during this period was 12% for yearling females, 8% for adult females, 13% for yearling males, and 0% for adult males. We isolated EHDV serotype 2 from the only deer from which samples could be obtained for virus isolation. A field necropsy of another animal showed tongue ulcers and internal hemorrhage around the heart and lungs typical of the acute form of HD (Thomas, 1981), but samples were unsuitable for VI. We found five additional unmarked deer on 20 August and 3, 6, 7, and 9 September but they were severely scavenged and decomposed and thus were unsuitable for virus isolation.

Three radio-monitored deer had long hours of inactivity i.e., the mortality sensor on the radiotransmitter would switch to mortality mode indicating the deer had not moved for ≥4 hr. These deer would flee when researchers approached but the mortality mode would be on when checked the next day. Two of these animals died within 3 days of the first episode in which the mortality mode activated, however, one radio-monitored animal whose signal switched between mortality and normal modes survived.

Of the 96 serum samples tested from the local deer population, 15 (16%) were positive on EHDV or BTV tests. Seven of these AGID positive serum samples were

suitable for additional testing by SN and all tested positive for EHDV serotype 2 with no positive results to any of the other EHDV or BTV serotypes.

Based on the assumptions that (1) all of the observed mortality from 20 August to 23 September was caused by HD; (2) the mortality rate (8%) observed in radio-monitored deer reflected the actual mortality rate in the population; and (3) detected antibodies were associated with infection during 1996 and not before, an overall infection rate of 24% was estimated, with 33% of these infected animals dying. The assumption that all of the mortality was related to HD is supported (1) other causes of deer mortality were rare, deer mortality outside of hunting season was <3% annually during our study (J. Beringer, unpubl. data); (2) deaths fit the pattern for HD cases e.g., sudden onset of mortality among generally healthy deer, death site near water, occurrence in late summer, and no sign of struggling; and (3) EHDV serotype 2 was isolated from the spleen of the only deer from which samples were submitted for virus isolation. Our assumption relating to our serologic data is supported by the fact that in the 4 yr prior to this epizootic antibody prevalence estimates for Missouri deer (21 to 47 animals sampled per year) ranged from 0% to 7% (D. E. Stallknecht, unpubl. data). Experimental infections of white-tailed deer with EHDV serotype 2 have shown a mortality rate (Quist et al., 1997) that was similar to our estimate.

Without the radio-monitored deer, we would not have known this HD epizootic had occurred. Despite an estimated 8% mortality, we received no reports of dead deer. Attendants at deer check stations noticed interrupted hoof growth on only two of the 96 deer from which they collected blood, suggesting that the detection of hoof lesions, while providing indirect evidence that HD had occurred, would underestimate exposure rates and could easily be missed under normal check station operations. During a 1988 HD epizootic in Missouri the Missouri Department of Conservation (Columbia, Missouri) received over 1,400 reports of deer mortality; and the mortality rate for this epizootic was estimated to range from 6 to 16% (Fischer et al., 1995). Unlike the localized 1996 mortality described here, the outbreak in 1988 was statewide, which suggests that there may be some threshold mortality below which dead deer are not reported. This threshold level could be affected by the total geographic affected by the outbreak and total mortality as well as mortality rate.

We were fortunate to have been monitoring deer via radio telemetry when this HD epizootic occurred. Even with this research in place, confirmation of EHDV-2 as the cause of this outbreak was difficult. Although radiotransmitters had 4 hr mortality switches, disturbance of carcasses by coyotes (*Canis latrans*) and other scavengers sometimes kept the transmitters in active mode thus delaying recovery of the deer. In these cases, the carcass was not suitable for virus isolation. In addition, some of the animals appeared inactive for 2 to 3 days prior to death resulting in premature mortality signals. This pattern is compatible with depression observed in deer with acute or peracute HD (Thomas, 1981), and although the animal was inactive it was not suitable for capture and subsequent sampling.

The fact that we received no reports of deer mortality from the public or field personnel, despite an estimated 8% mortality in the local deer herd, suggests that disease-related mortality may be significantly under reported. Even if the mortality observed in this study was not all related to HD, this observation has important implication to wildlife managers, and as this study demonstrates, questions relating to disease impacts are extremely difficult to answer even in well monitored populations.

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