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## Hemorrhagic Disease in White-tailed Deer in Texas: A Case for Enzootic Stability

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**ABSTRACT:** Although antibodies to viruses in both the epizootic hemorrhagic disease virus (EHDV) and bluetongue virus (BTV) serogroups have been reported from white-tailed deer (*Odocoileus virginianus*) in Texas (USA), there are few reports of hemorrhagic disease (HD) in these populations. To understand the extent and diversity of exposure to the North American EHDV and BTV serotypes in these deer populations, we serologically tested 685 white-tailed deer collected from November 1991 through March 1992 throughout their range in Texas. Overall, 574 (84%) of deer had antibodies to EHDV or BTV. Prevalence estimates varied according to ecological region, from 57% in the Gulf Prairies to 100% in the northwest Edwards Plateau. Based on serum neutralization tests, the deer had evidence of previous exposures to multiple EHDV and BTV serotypes, with evidence of exposure to two to five serotypes detected in each ecological region. The apparent lack of HD in relation to this high antibody prevalence cannot be explained, but may be related to enzootic stability in which a near perfect host-virus relationship exists.

**Key words:** Hemorrhagic disease, white-tailed deer, *Odocoileus virginianus*, epizootic hemorrhagic disease virus, bluetongue virus, epizootiology.

Hemorrhagic disease (HD) is caused by viruses in either the epizootic hemorrhagic disease virus (EHDV) or bluetongue virus (BTV) serogroups (Reoviridae: Orbivirus) and represents the most important viral disease known to affect white-tailed deer (*Odocoileus virginianus*) (Nettles and Stallknecht, 1992). Two serotypes of EHDV (serotypes 1 and 2) and five serotypes of BTV (serotypes 2, 10, 11, 13, and 17) have been isolated in the United States (Pearson et al., 1992). Clinical responses of white-tailed deer to infections with these viruses are variable, ranging from inapparent infection to death (Kocan et al., 1982; Thomas et al., 1974). Reasons for this variation currently are unclear.

Hemorrhagic disease in Texas (USA) was first reported in 1966 in a captive white-tailed deer (Stair et al., 1968) and a bighorn sheep (*Ovis canadensis*) (Robinson et al., 1967). However, only isolated reports of HD in wild ungulates were documented from Texas during a nationwide mortality and morbidity survey conducted from 1980 to 1989 (Nettles et al., 1992). During this period, only 10 of 1,608 HD reports were from Texas; all but one of these reports represented counties located in the extreme eastern part of the state. In addition, there are few reports of EHDV or BTV isolations from either domestic or wild species in Texas. Since 1976, only three virus isolations of BTV serotypes 11 and 17, exclusively from domestic sheep, have been reported (Pearson et al., 1992).

The relative scarcity of clinical and virologic evidence of EHDV or BTV infection in Texas white-tailed deer is in contrast with results from previous serologic surveys. Wilhelm and Trainer (1966) reported a high prevalence of antibodies to BTV (78%) and EHDV (91%) in white-tailed deer in San Patricio County. Antibodies to EHDV (75%) and BTV (75%) were also reported in 122 deer from the Edwards Plateau, Cross Timbers, and Post Oak Savannah ecological areas (Corn et al., 1990). Waldrup et al. (1989) reported a prevalence of 32% for antibodies to EHDV and 34% for antibodies to BTV in 274 deer of numerous species (native and exotic) in several Texas counties. Thus there is evidence for a relatively high prevalence of EHDV and BTV infection in deer in many areas of Texas without corresponding reports of HD. There is no information, however, on the specific EHDV and BTV serotypes responsible for this exposure, and such information may help in

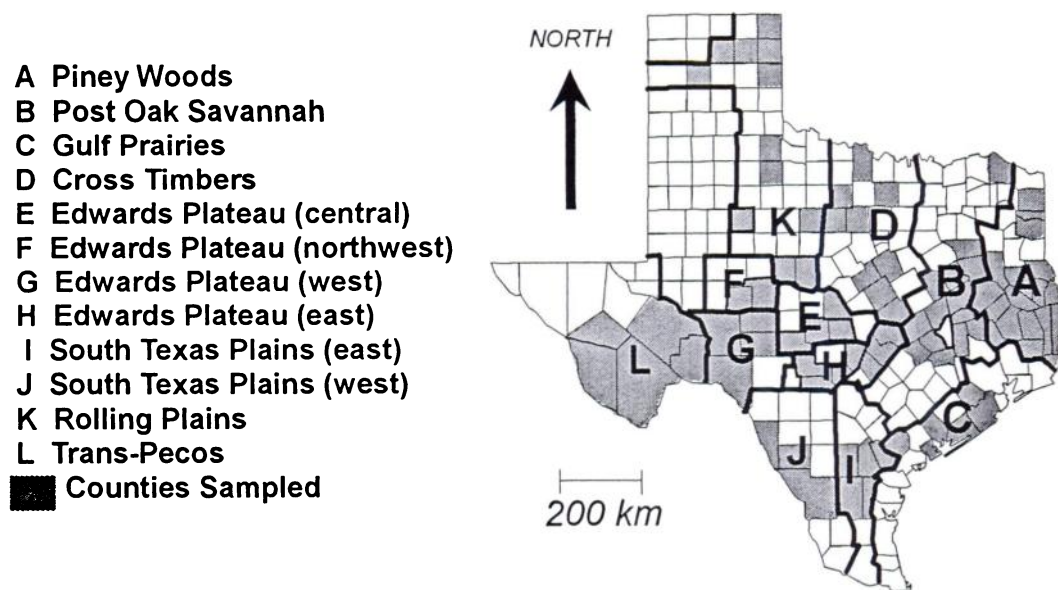


FIGURE 1. Counties and ecological regions from which white-tailed deer were sampled including: Piney Woods (32°0'N, 95°0'W); Post Oak Savannah (32°0'N, 96°0'W); Gulf Prairies (29°0'N, 96°0'W); Cross Timbers (33°0'N, 97°0'W); Edwards Plateau central (31°0'N, 100°0'W); Edwards Plateau northwest (34°0'N, 100°0'W); Edwards Plateau west (30°0'N, 100°0'W); Edwards Plateau east (30°0'N, 96°0'W); South Texas Plains east (26°0'N, 96°0'W); South Texas Plains west (26°0'N, 100°0'W); Rolling Plains (34°0'N, 100°0'W); and Trans Pecos (31°0'N, 104°0'W).

understanding the apparent lack of HD in these populations. In order to better define the extent and diversity of exposure to the EHDV and BTV serotypes in these populations we serologically tested white-tailed deer throughout their range in Texas.

Deer were collected by Texas Parks and Wildlife Department personnel by shooting from November 1991 through March 1992. Serum samples were collected from 685 animals from 89 counties representing the eight ecological regions (Gould, 1975) where deer are present (Fig. 1). Due to a large sample size, two of these regions, the Edwards Plateau and South Texas Plains, were further subdivided for analysis (Fig. 1).

Serum samples were tested for antibodies to the EHDV and BTV serotypes as described by Stallknecht et al. (1995). Briefly, serum samples were screened using EHDV and BTV agar gel immunodiffusion (AGID) tests (Pearson and Jochim, 1979). To test for serotype-specific antibodies, at least 20 randomly selected

AGID-positive samples from each ecological region were tested by serum neutralization (SN) test against all enzootic serotypes of EHDV and BTV as described (Stallknecht et al., 1995). All sampled counties in each ecological region were represented in this subsample. Evidence of previous exposure to a given serotype was determined by detection of monospecific reactions or clusters of seropositive results against a given serotype as suggested by Taylor et al. (1985) and modified by Stallknecht et al. (1995). A monospecific reaction was accepted as evidence of previous exposure to a given serotype only if positive serologic results were observed in one or more animals against a single EHDV or BTV serotype at a serum dilution of 1:20 or higher. Exposure based on clusters was accepted if more than 50% of the seropositive deer tested by SN in a given ecological region had neutralizing antibodies to a given serotype at a serum dilution of 1:10 or higher.

TABLE 1. Prevalence of precipitating antibodies to epizootic hemorrhagic disease or bluetongue viruses in white-tailed deer in Texas by ecological region, 1991 to 1992.

| Ecological region           | Number of counties | Number of deer | Number positive (%) <sup>a</sup> | 95% confidence limit |
|-----------------------------|--------------------|----------------|----------------------------------|----------------------|
| Gulf Prairies               | 5                  | 87             | 50 (57%)                         | 46%–68%              |
| Piney Woods                 | 18                 | 54             | 37 (68%)                         | 54%–80%              |
| Post Oak Savannah           | 14                 | 100            | 76 (76%)                         | 68%–85%              |
| South Texas Plains (east)   | 5                  | 46             | 41 (89%)                         | 76%–96%              |
| South Texas Plains (west)   | 3                  | 40             | 36 (90%)                         | 75%–97%              |
| Cross Timbers               | 9                  | 95             | 85 (89%)                         | 81%–94%              |
| Edwards Plateau (central)   | 4                  | 21             | 19 (90%)                         | 68%–98%              |
| Trans Pecos                 | 5                  | 58             | 53 (91%)                         | 80%–97%              |
| Edwards Plateau (east)      | 8                  | 68             | 63 (93%)                         | 84%–97%              |
| Edwards Plateau (west)      | 4                  | 26             | 25 (96%)                         | 78%–100%             |
| Rolling Plains              | 12                 | 71             | 70 (99%)                         | 92%–100%             |
| Edwards Plateau (northwest) | 2                  | 19             | 19 (100%)                        | 79%–100%             |

<sup>a</sup> Number (percent) of animals with antibodies to epizootic hemorrhagic disease or bluetongue viruses.

Differences in antibody prevalence between groups of regions and between age classes were tested by Chi-square analysis using Yate's correction (Sokal and Rohlf, 1981). Confidence limits (95%) on antibody prevalence estimates for individual ecological regions also were calculated (Fleiss, 1981).

Overall, 574 (84%) of 685 deer had antibodies to EHDV or BTV (Table 1). Antibody prevalence varied from 57% in the

Gulf Prairies to 100% in the northwest Edwards Plateau. Antibody prevalence increased in a westerly direction, and significant differences ( $P < 0.04$ ) were detected in pair-wise comparisons between regions grouped into eastern (Piney Woods, Gulf Prairies, Post Oak Savannah; 163 of 241 deer, 68%), central (Cross Timbers, South Texas Plains east and west, and Edwards Plateau east and central; 244 of 270 deer, 90%), and western (Edwards Plateau west and northwest, Rolling Plains, and Trans Pecos; 167 of 174 deer, 96%) zones. Antibody prevalence increased to greater than 80% in the 2.5-yr and older age classes (Fig. 2). Significant age differences ( $P < 0.05$ ) were detected only between the 1.5-yr age class and the 2.5-yr and older age classes.

We tested 240 AGID-positive serum samples by SN (Table 2). Statewide, evidence of previous exposure to EHDV serotypes 1 and 2 and BTV serotypes 11, 13, and 17 were detected. Presence of these serotypes varied by region, and of all serotypes detected, EHDV serotype 2 was most widely distributed, followed by BTV serotype 13, BTV serotype 17, BTV serotype 11, and EHDV serotype 1. Grouped by eastern, central, and western zones, evidence of previous exposure to at least four serotypes (BTV and EHDV) was detected in each zone.

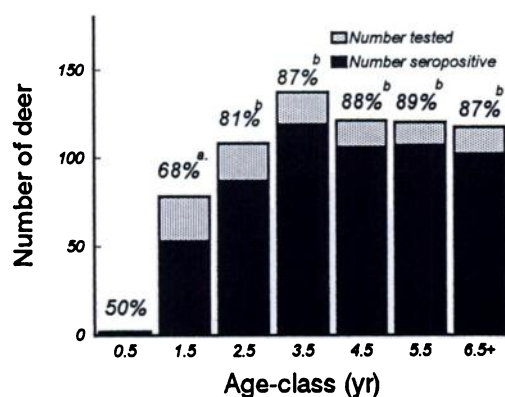


FIGURE 2. Age class distribution of white-tailed deer sampled in Texas for serologic testing for antibodies to epizootic hemorrhagic disease and bluetongue viruses. Numbers above bars represent antibody prevalence estimates for each age class. Prevalence estimates with different superscript letters are significantly different ( $P < 0.05$ ). The 0.5-yr age class is not included in the statistical analysis due to low sample size.

TABLE 2. Neutralizing antibodies to epizootic hemorrhagic disease virus (EHDV) and bluetongue virus (BTV) serotypes in white-tailed deer in Texas by ecological region.

| Ecological region           | Number of sera | Serotype <sup>a</sup> |                     |      |       |                    |                    |                    |
|-----------------------------|----------------|-----------------------|---------------------|------|-------|--------------------|--------------------|--------------------|
|                             |                | EHDV1                 | EHDV2               | BTV2 | BTV10 | BTV11              | BTV13              | BTV17              |
| Gulf Prairies               | 24             | 4% <sup>m</sup>       | 96% <sup>m,c</sup>  | 29%  | 0%    | 17%                | 62% <sup>m,c</sup> | 29% <sup>m</sup>   |
| Piney Woods                 | 22             | 27%                   | 95% <sup>m,c</sup>  | 5%   | 0%    | 0%                 | 45% <sup>m</sup>   | 9%                 |
| Post Oak Savannah           | 22             | 27% <sup>m</sup>      | 91% <sup>m,c</sup>  | 5%   | 23%   | 18%                | 36% <sup>c</sup>   | 41%                |
| South Texas Plains (east)   | 24             | 25%                   | 83% <sup>m,c</sup>  | 21%  | 37%   | 71% <sup>m,c</sup> | 79% <sup>m,c</sup> | 67% <sup>m,c</sup> |
| South Texas Plains (west)   | 25             | 12%                   | 96% <sup>m,c</sup>  | 16%  | 8%    | 52% <sup>c</sup>   | 80% <sup>m,c</sup> | 80% <sup>m,c</sup> |
| Cross Timbers               | 24             | 37%                   | 100% <sup>m,c</sup> | 33%  | 29%   | 54% <sup>c</sup>   | 92% <sup>m,c</sup> | 54% <sup>c</sup>   |
| Edwards Plateau (central)   | 12             | 8%                    | 100% <sup>m,c</sup> | 33%  | 8%    | 25%                | 75% <sup>m,c</sup> | 42%                |
| Trans Pecos                 | 21             | 14%                   | 100% <sup>m,c</sup> | 29%  | 19%   | 43%                | 57% <sup>c</sup>   | 67% <sup>m,c</sup> |
| Edwards Plateau (east)      | 15             | 40%                   | 100% <sup>m,c</sup> | 0%   | 13%   | 33%                | 73% <sup>m,c</sup> | 47%                |
| Edwards Plateau (west)      | 12             | 33%                   | 92% <sup>m,c</sup>  | 42%  | 42%   | 33%                | 83% <sup>m,c</sup> | 67% <sup>c</sup>   |
| Rolling Plains              | 22             | 45%                   | 100% <sup>m,c</sup> | 41%  | 9%    | 45%                | 82% <sup>m,c</sup> | 82% <sup>m,c</sup> |
| Edwards Plateau (northwest) | 12             | 33% <sup>m</sup>      | 92% <sup>m,c</sup>  | 25%  | 8%    | 75% <sup>c</sup>   | 83% <sup>c</sup>   | 67% <sup>c</sup>   |

<sup>a</sup> Superscript <sup>m</sup> = meets criteria for monospecific reactions, superscript <sup>c</sup> = meets criteria for clusters.

There was good agreement in serotype exposure results as determined by monospecific reactions and clusters (Table 2). Our criteria (clusters or monospecific antibodies) for previous exposure to a given serotype in an ecological region was met in 39 cases (Table 2). Of these, both clusters and monospecific antibodies were detected in 25 cases. Evidence of previous exposure was limited to detection of clusters or monospecific reactions in eight and six cases, respectively. The failure to detect serological evidence of previous exposure to BTV serotype 2 based on these criteria also was encouraging. To date, isolations of BTV serotype 2 have been restricted to Florida, USA (Gibbs et al., 1983). Although neutralizing antibodies to BTV serotype 2 were observed in this study (Table 2), it is likely that they represent cross reactions due to exposure with other BTV serotypes.

There also was very good agreement between the combined EHDV and BTV AGID and SN results. Of the 240 AGID-positive samples tested by SN, only 5 (2%) were negative for all of the EHDV and BTV serotypes. These results are comparable with serologic results from a similar study in Georgia where AGID false positive results, as determined by SN results, ranged from 2% to 6% depending on year

(Stallknecht et al., 1995). Although AGID-negative samples observed in the present study were not tested by SN, the fact that more than 89% of deer tested AGID-positive in nine of 12 ecological regions is evidence that false negative results were not a major problem.

Specific antibody prevalence estimates to the EHDV or BTV serogroups based on AGID results are not reliable due to cross reactions (Stallknecht et al., 1991). It was for this reason that we elected to express our AGID prevalence estimates (Table 1) in relation to the combined EHDV and BTV AGID results. In addition, the combined AGID results provide a very rapid, reliable, and inexpensive means for screening serum samples for subsequent SN tests. At present, the SN test provides the only means for gaining serologic evidence of specific serotype exposure in deer or any other species.

Results were consistent with past serological surveys of white-tailed deer in Texas and are evidence for a very high frequency of exposure to multiple serotypes of both EHDV and BTV. This exposure increased in a westerly direction. It is interesting that this increase in exposure towards the west was in contrast to the distribution of clinical HD in Texas that occurs primarily in the eastern part of the

state (Nettles et al., 1992) where antibody prevalence is lowest. Based on the extent of herd immunity in white-tailed deer populations in Texas, a low number of reports of HD in adult animals is understandable. However, the lack of reported disease associated with initial exposure of fawns to these viruses is puzzling; this may relate individually or in combination to innate host resistance, protection of fawns through maternal antibody transfer, or poor detection of affected animals. In addition, vector species composition and seasonality may interact with these potential variables, especially maternal antibody transfer. At present, all of these possibilities are poorly understood and at best are speculative.

Regardless of the mechanisms involved, HD currently is not recognized as a significant problem in white-tailed deer in Texas. If this apparent lack of disease is related to enzootic stability and not to poor detection, the potential impact of HD on white-tailed deer management in Texas is minimal. However, the lack of disease in indigenous deer populations does not mean that HD could not present a disease risk to other ungulate species, especially those translocated from nonenzootic areas. Robinson et al. (1967) suggested, for example, that bluetongue may have played a role in the disappearance of desert bighorn sheep in the Trans Pecos ecological region. Such considerations may be important in re-introduction efforts involving endangered or threatened ungulate species.

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#### LITERATURE CITED

- CORN, J. L., D. M. KAVANAUGH, D. A. OSBORN, S. DEMARAIS, D. M. MILLER, AND V. F. NETTLES. 1990. Survey for diseases and parasites in exotic ruminants in Texas. *Proceedings of the Annual Meeting of the United States Animal Health Association* 94: 530-540.
- FLEISS, J. L. 1981. *Statistical methods for rates and proportions*. John Wiley and Sons, New York, New York, 321 pp.
- GIBBS, E. P. J., E. C. GREINER, W. P. TAYLOR, T. L. BARBER, J. A. HOUSE, AND J. E. PEARSON. 1983. Isolation of bluetongue virus serotype 2 from cattle in Florida: Serotype of bluetongue hitherto unrecognized in the Western Hemisphere. *American Journal of Veterinary Research* 44: 2226-2228.
- GOULD, F. W. 1975. Texas plants—A check list and ecological summary. MP-585. Texas Agricultural Experiment Station, Texas A&M University, College Station, Texas, 121 pp.
- KOCAN, A. A., E. CASTRO, B. ESPE, R. T. DOYLE, AND S. K. OLSEN. 1982. Inapparent bluetongue in free-ranging white-tailed deer. *Journal of the American Veterinary Medical Association* 181: 1415-1417.
- NETTLES, V. F., AND D. E. STALLKNECHT. 1992. History and progress in the study of hemorrhagic disease of deer. *Transactions of the North American Wildlife and Natural Resources Conference* 57: 499-516.
- , W. R. DAVIDSON, AND D. E. STALLKNECHT. 1992. Surveillance for hemorrhagic disease in white-tailed deer and other wild ruminants, 1980-1989. *Proceedings of the Annual Conference of the Southeastern Association of Fish and Wildlife Agencies* 46: 138-146.
- PEARSON, J. E., AND M. M. JOCHIM. 1979. Protocol for the immunodiffusion test for bluetongue. *Proceedings of the American Association of Veterinary Diagnosticians* 22: 463-475.
- , G. A. GUSTAFSON, A. L. SHAFER, AND A. D. ALSTAD. 1992. Distribution of bluetongue in the United States. *In* *Bluetongue, African horse sickness, and related orbiviruses*, T. E. Walton and B. I. Osburn (eds.). CRC Press, Boca Raton, Florida, pp. 128-139.
- ROBINSON, R. M., T. L. HAILEY, C. W. LIVINGSTON,

- AND J. W. THOMAS. 1967. Bluetongue in desert bighorn sheep. *The Journal of Wildlife Management* 31: 165–168.
- SOKAL, R. R., AND F. J. ROHLF. 1981. *Biometry*. W. H. Freeman and Company, New York, New York, 858 pp.
- STAIR, E. L., R. M. ROBINSON, AND L. P. JONES. 1968. Spontaneous bluetongue in Texas white-tailed deer. *Pathologia Veterinaria* 5: 164–173.
- STALLKNECHT, D. E., J. L. BLUE, E. A. ROLLOR, III, V. F. NETTLES, W. R. DAVIDSON, AND J. E. PEARSON. 1991. Precipitating antibodies of the epizootic hemorrhagic disease and bluetongue viruses in white-tailed deer in the southeastern United States. *Journal of Wildlife Diseases* 27: 238–247.
- , V. F. NETTLES, E. A. ROLLOR, III, AND E. W. HOWERTH. 1995. Epizootic hemorrhagic disease virus and bluetongue virus serotype distribution in white-tailed deer in Georgia. *Journal of Wildlife Diseases* 31: 331–338.
- TAYLOR, W. P., I. D. GUMM, E. P. J. GIBBS, AND J. HOMAN. 1985. The use of serology in bluetongue epidemiology. *Progress in Clinical and Biological Research* 178: 461–468.
- THOMAS, F. C., N. WILLIS, AND G. RUCKERBAUER. 1974. Identification of viruses involved in the 1971 outbreak of hemorrhagic disease in the southeastern United States white-tailed deer. *Journal of Wildlife Diseases* 10: 187–189.
- WALDRUP, K. A., E. COLLISON, S. E. BENTSEN, C. K. WINKLER, AND G. G. WAGNER. 1989. Prevalence of erythrocytic protozoa and serologic reactivity to selected pathogens in deer in Texas. *Preventive Veterinary Medicine* 7: 49–58.
- WILHELM, A. R., AND D. O. TRAINER. 1966. A serological study of epizootic hemorrhagic disease of deer. *The Journal of Wildlife Management* 30: 777–780.

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