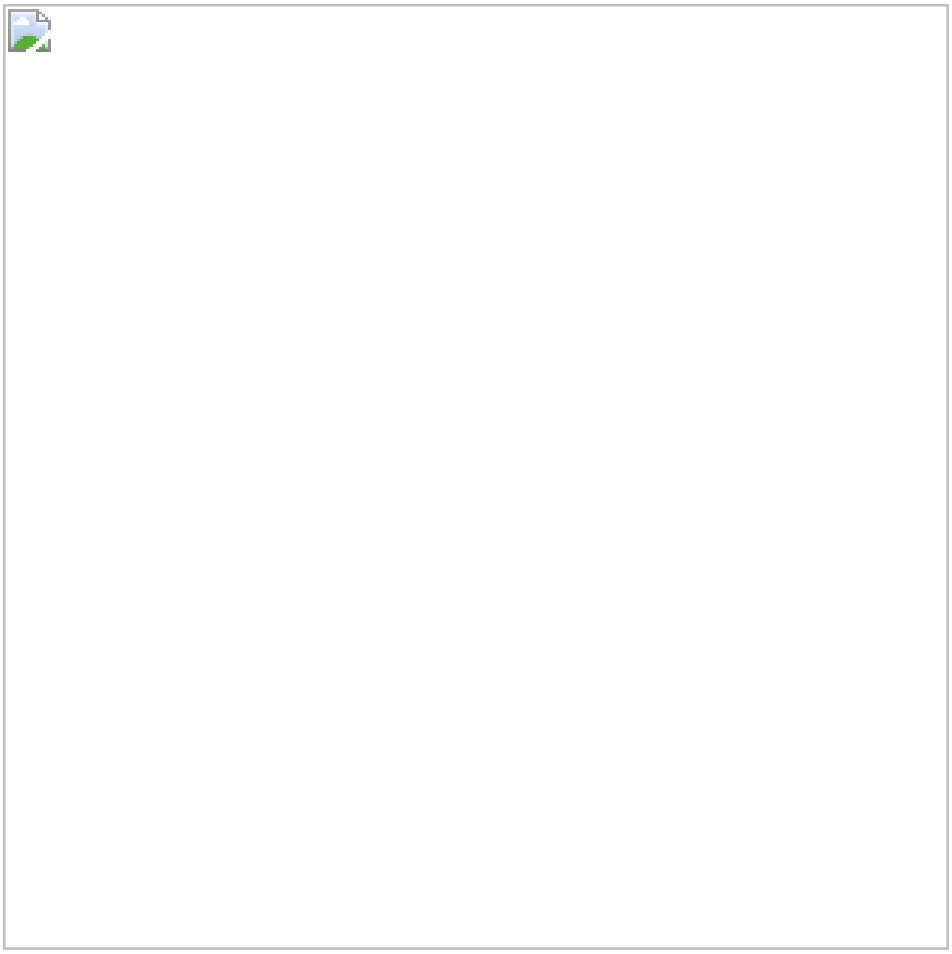

DYNAMICS OF MATERNAL ANTIBODIES TO HEMORRHAGIC DISEASE VIRUSES (REOVIRIDAE: ORBIVIRUS) IN WHITE-TAILED DEER

Authors: Gaydos, Joseph K., Stallknecht, David E., Kavanaugh, Darrell, Olson, Robert J., and Fuchs, Eugene R.

Source: Journal of Wildlife Diseases, 38(2) : 253-257

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-38.2.253

Journal of Wildlife Diseases, 38(2), 2002, pp. 253–257

DYNAMICS OF MATERNAL ANTIBODIES TO HEMORRHAGIC DISEASE VIRUSES (REOVIRIDAE: ORBIVIRUS) IN WHITE-TAILED DEER

Joseph K. Gaydos,[1,3,5] David E. Stallknecht,[1,2] Darrell Kavanaugh,[1] Robert J. Olson,[1] and Eugene R. Fuchs[4]

[1] Southeastern Cooperative Wildlife Disease Study and
[2] Department of Medical Microbiology, College of Veterinary Medicine, University of Georgia, Athens, Georgia 30602, USA
[3] Current address: Marine Ecosystem Health Program, University of California Davis Wildlife Health Center, 982 Deer Harbor Rd., Eastsound, Washington 98245, USA
[4] Texas Parks and Wildlife Department, Kerr Wildlife Management Area, Donnie E. Harmel White-tailed Deer Research Facility, Hunt, Texas, USA
[5] Corresponding author (e-mail: jkgaydos@ucdavis.edu)

ABSTRACT: Enzootic stability, potentially associated with acquired resistance and subsequent transfer of maternal antibodies, innate resistance, or both, has been hypothesized to explain the lack of reports of hemorrhagic disease (HD) in white-tailed deer (*Odocoileus virginianus*) from Texas. The objectives of this research were to determine the following: how long maternal antibodies to epizootic hemorrhagic disease (EHD) and bluetongue (BT) viruses persist; whether fawns from an enzootic site are naturally exposed to EHD and BT viruses while maternal antibodies are present; and whether field-challenged fawns develop clinical disease. Twelve of 52 fawns from Texas were moved to an indoor facility. All 12 (100%) were positive for maternal antibodies to EHD or BT viruses by agar gel immunodiffusion (AGID) and serum neutralization (SN) tests. Weekly monitoring demonstrated that precipitating antibodies disappeared by 23 wk of age and serum neutralizing antibodies disappeared by 17–18 wk of age. Fawns that remained outdoors in Texas were not observed with signs of HD. At 14–21 wk of age (October), 39 of 40 (98%) fawns that had remained outdoors were positive for EHD and/or BT virus antibodies by AGID and 32 (80%) had SN antibody titers to one or more of five viruses (EHDV-1, EHDV-2, BTV-10, BTV-11, BTV-17). Antibody titers to EHDV-1, EHDV-2, and BTV-11 all exceeded titers of same-age indoor fawns, suggesting recent exposure. Epizootic hemorrhagic disease viruses were isolated from seven (18%) of the outdoor fawns and all 40 remained clinically normal. Natural exposure of deer to EHD and BT viruses occurred at this site in the presence of maternal antibodies without causing disease. This may be due to acquired immunity and the subsequent transfer of maternal antibodies, but it does not exclude innate resistance as a possible factor in the enzootic stability of EHD and BT viruses at this location.

Key words: Bluetongue virus, enzootic stability, epizootic hemorrhagic disease virus, hemorrhagic disease, HD, maternal antibodies, *Odocoileus virginianus*, white-tailed deer.

INTRODUCTION

Hemorrhagic disease (HD) in white-tailed deer (*Odocoileus virginianus*) is caused by Orbiviruses in the epizootic hemorrhagic disease (EHD) virus and bluetongue (BT) virus serogroups (Nettles and Stallknecht, 1992). The spatial distribution of HD in the southeastern United States is not uniform. Epizootics in northern latitudes occur infrequently and are characterized by severe clinical disease and mortality, whereas epizootics in southern latitudes are more frequent and often result in chronic or inapparent disease (Davidson and Doster, 1997). Virus isolations and antibodies to viruses in both the EHD virus and BT virus serogroups have been reported from white-tailed deer in Texas, but there are few reports of HD in these populations (Nettles et al., 1992). It has been hypothesized that this represents a case of enzootic stability, whereby deer are protected from clinical HD by acquired immunity and subsequent transfer of maternal antibodies, innate resistance, or both (Stallknecht et al., 1996). A serologic survey of white-tailed deer in Texas demonstrated that exposure to EHD and BT viruses increased in a westerly direction (Stallknecht et al., 1996) in contrast to the distribution of HD in Texas, which oc-

curs primarily in the eastern part of the state where antibody prevalence is lowest (Nettles et al., 1992). Thus, high herd immunity can explain the lack of HD reported in adult deer but does not explain the lack of reported disease associated with initial exposure of fawns to these viruses. The objectives of this research were to determine how long maternal antibodies to EHD and BT viruses persist, whether fawns from an enzootic site are naturally exposed to EHD and BT viruses while maternal antibodies are present, and whether field-challenged fawns develop clinical disease.

MATERIALS AND METHODS

Twelve known-age native Texas white-tailed deer fawns were moved by 2 wk of age from an outdoor facility in Texas (Donnie E. Harmel White-tailed Deer Research Facility, Texas, USA; 30°03′47″N, 99°30′19″W) to an indoor facility at the University of Georgia (33°56′21″N, 83°22′29″W) in June and July 2000. Starting on July 17, fawns were manually restrained and bled by jugular venipuncture weekly. Serum was tested for EHD and BT virus antibodies by agar gel immunodiffusion (AGID) according to the manufacturers instructions (Veterinary Diagnostic Technology, Inc., Wheatridge, Colorado, USA) and by serum neutralization (SN) tests against all known North American EHD and BT virus serotypes as previously described (Stallknecht et al., 1995). Serum samples without evidence of SN antibodies at a dilution of 1:10 were considered negative. The percentage of fawns testing positive by AGID for EHD or BT virus antibodies, or both, was graphed by fawn age in weeks, and a logarithmic regression line was fitted to the data. A subset of fawns with SN antibodies to EHD virus serotype-1 (EHDV-1) ($n = 4$), EHD virus serotype-2 (EHDV-2) ($n = 4$), and BT virus serotype 11 (BTV-11) ($n = 3$) were tested weekly for SN antibodies. Using fawn age in weeks, geometric mean serum neutralizing antibody titers to EHDV-1, EHDV-2, and BTV-11 were plotted and again, logarithmic regression lines were fitted to the data.

Fawns remaining at the outdoor facility in Texas were observed daily by Texas Parks and Wildlife employees for visual signs of clinical disease. On October 10, 40 fawns that had remained outdoors at the facility in Texas were manually restrained and bled by jugular venipuncture. Using the same techniques as were used for the indoor fawns, serum samples from these animals were tested for EHD and BT virus antibodies by AGID. All samples that were positive for antibodies to EHD or BT virus serogroup were tested by SN test. Using known fawn age in weeks, SN antibody titers to EHDV-1, EHDV-2, and BTV-11 from outdoor fawns were plotted against the antibody regression curves developed from indoor fawns for each serotype. Virus isolation also was performed on blood collected in EDTA anticoagulant from all 40 outdoor fawns using cattle pulmonary artery endothelial (CPAE) cells (American Type Culture Collection, Rockville, Maryland, USA) as previously described (Quist et al., 1997). Viruses isolated were identified and viral titers of positive blood samples were determined by endpoint titration using techniques previously described (Quist et al., 1997).

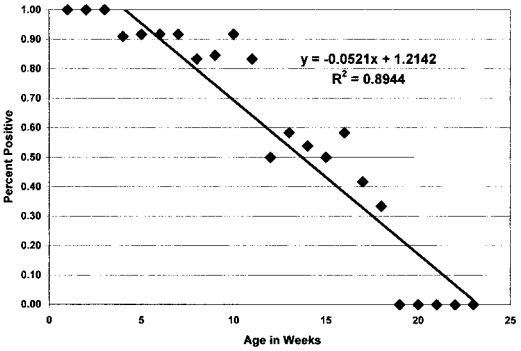

FIGURE 1. Percent of fawns ($n = 12$) positive for maternal antibodies to EHD and BT viruses as detected by AGID by fawn age in weeks.

RESULTS

On July 17, all 12 fawns moved to Georgia were positive for antibodies to EHD or BT viruses by AGID. Additionally, all were positive for SN antibodies to one or more of five EHD and BT viruses (EHDV-1, EHDV-2, BTV-10, BTV-11, and BTV-17). Precipitating antibodies to EHD or BT viruses, as detected by AGID, were not detectable in any of the 12 fawns after 18 wk of age. Extrapolation from a regression line fit to the graph of the percentage of fawns positive for EHD and BT virus antibodies suggested that fawns from this cohort would be negative for precipitating antibodies by 23 wk of age (Fig. 1). Serum neutralizing antibodies to EHDV-1, EHDV-2, or BTV-11 were not detectable

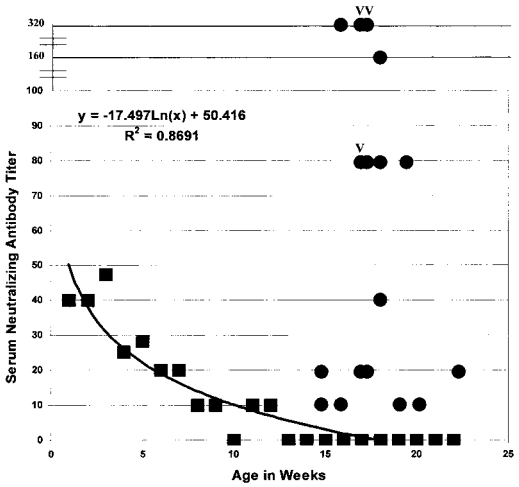

FIGURE 2. Serum neutralizing antibodies to EHDV-1 by fawn age in weeks (■ = Geometric mean serum neutralizing antibody titer of indoor fawns [n = 4]; ● = individual serum neutralizing antibody titers in outdoor fawns [n = 17]; V = serum neutralizing antibody titers from fawns from which EHDV-1 was isolated).

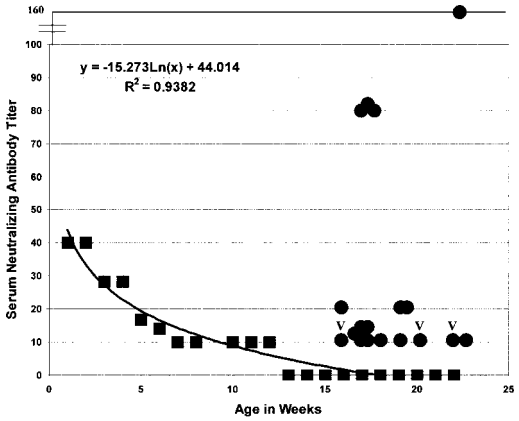

FIGURE 3. Serum neutralizing antibodies to EHDV-2 by fawn age in weeks (■ = Geometric mean serum neutralizing antibody titer of indoor fawns [n = 4]; ● = individual serum neutralizing antibody titers in outdoor fawns [n = 18]; V = serum neutralizing antibody titers from fawns from which EHDV-2 was isolated).

after 12, 13, and 10 wk, respectively, and graphs plotting the decline of antibodies to these viruses suggested SN antibodies to these viruses disappear between 17–18 wk of age (Figs. 2–4).

On October 10, the 40 fawns bled at the outdoor facility in Texas ranged from 14 to 21 wk of age. Thirty-nine of 40 (98%) fawns were positive for EHD and/or BT virus antibodies by AGID and 32 (80%) had SN antibody titers of 1:10 or greater to one or more of five viruses (EHDV-1, EHDV-2, BTV-10, BTV-11, and BTV-17). All fawns that remained outdoors in Texas had SN antibody titers to EHDV-1, EHDV-2, or BTV-11 that were higher than the titers in fawns that were moved indoors (Figs. 2–4).

Epizootic hemorrhagic disease viruses were isolated from seven of the 40 (18%) outdoor fawns. Epizootic hemorrhagic disease virus serotype 1 was isolated from four (10%) and EHDV-2 was isolated from three (8%) fawns. Virus titers for all positive blood samples were $\leq 10^{2.3}$ tissue culture infective dose 50 ($TCID_{50}$) per ml. Except for one fawn that was infected with EHDV-1, all fawns from which virus was isolated had SN antibodies to the homologous virus (Figs. 2, 3). Clinical signs of HD were not observed in fawns that remained in the outdoor Texas facility.

DISCUSSION

There is strong evidence that antibodies detected in the 12 fawns moved indoors were passively derived (maternal). The facts that fawns were moved to an indoor

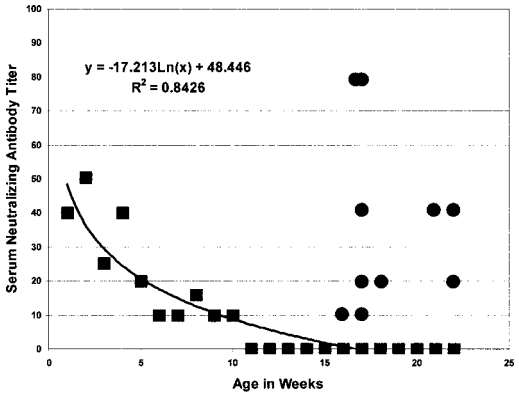

FIGURE 4. Serum neutralizing antibodies to BTV-11 by fawn age in weeks (■ = Geometric mean serum neutralizing antibody titer of indoor fawns [n = 3]; ● = individual serum neutralizing antibody titers in outdoor fawns [n = 10]).

facility by 2 wk of age, that they were seropositive at 2–4 wk of age, and that EHD and BT virus antibodies had disappeared by 17–18 wk of age support this. In the one published article on maternal antibodies to BT viruses in white-tailed deer fawns, maternal antibodies persisted for at least 8 wk in the four fawns tested (Hoff et al., 1974). In additional research evaluating white-tailed deer maternal antibody decay, all 5 wk old fawns positive for maternal antibodies to Jamestown Canyon virus had lost these antibodies when tested again at 20–24 wk of age (Grimstad et al., 1987). Although there is probably considerable variation in the duration of maternal antibodies against specific viruses in white-tailed deer, our data on EHD and BT virus maternal antibody decay are consistent with these earlier reports.

Because all fawns randomly selected and moved indoors had maternal antibodies to one or more of the EHD or BT viruses, we assumed that the majority of fawns that remained outdoors at the Texas site also had maternal antibodies to one or more of these viruses. When compared with maternal antibody decay curves developed for EHDV-1, EHDV-2, and BTV-11, which suggest that SN maternal antibodies to these viruses disappear by 17–18 wk of age, the high SN antibody titers in the 14–21 wk old outdoor fawns imply that natural exposure to these three viruses occurred. Isolation of EHDV-1 from 10% and EHDV-2 from 8% of fawns confirmed active circulation of these two viruses at the outdoor Texas site. Based on experimental data (Quist et al., 1997), virus titers $\leq 10^{2.3}$ $TCID_{50}$ suggested late infection (post-infection day 25 or greater). That all but one fawn from which EHDV-1 or 2 was isolated had SN antibodies to the homologous virus supports the idea that a late-infection viremia was detected in these animals. If viremia was detected in these animals at post-infection day 25 or greater as inferred from experimental data (Quist et al., 1997), then these fawns were no more than 14–18 wk old when infected and probably still had maternal antibodies.

Although Texas Parks and Wildlife Department personnel observed the fawns that remained at the outdoor facility daily and did not notice any marked clinical signs of HD, this does not rule out the possibility that fawns could have exhibited mild clinical signs of HD. Visual observation would have detected mortality or severe depression, but could not have detected low fever and probably was not sensitive enough to detect less apparent signs of disease like mucosal congestion, mild cutaneous erythema, or minor subcutaneous edema. Fawns received a thorough visual examination in October when they were manually restrained for venipuncture. Signs of clinical disease were not detected at this time either, despite evidence that at least 18% of the fawns were infected with EHD viruses. This confirms that clinical HD, if present in these fawns, was mild and not debilitating.

This research provides insight into the role of maternal antibodies in the enzootic stability of EHD and BT viruses in Texas. In light of the antibody regression curves developed for precipitating and neutralizing antibodies, these serologic and virus isolation data suggest that at this site, natural exposure of fawns to one or more EHD or BT viruses probably occurs while maternal antibodies are still present and does not prevent infection nor viremia. The presence of maternal antibodies may explain why severe clinical disease was not detected in any of these fawns, although the possibility of innate immunity cannot be excluded.

ACKNOWLEDGMENTS

The authors thank D. Frels and the Texas Parks and Wildlife Department for assistance in procuring deer; W. R. Davidson and E. W. Howerth for reviewing this manuscript; and K. Osborne, D. Corn, and D. Berndt for assistance rearing fawns. This work was supported through the sponsorship by fish and wildlife agencies of Alabama, Arkansas, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississip-

pi, North Carolina, Puerto Rico, South Carolina, Tennessee, Virginia, and West Virginia. Funds were provided by the Federal Aid to Wildlife Restoration Act (50-Stat. 917) and through Grant Agreement 14-45-0009-94-906, National Biological Service, U.S. Department of the Interior.

LITERATURE CITED

DAVIDSON, W. R., AND G. L. DOSTER. 1997. Health characteristics and white-tailed deer population density in the southeastern United States. *In* The science of overabundance: Deer ecology and population management, W. J. McShea, H. B. Underwood and J. H. Rappole (eds.). Smithsonian Institution Press, Washington, D.C., pp. 164–184.

GRIMSTAD, P. R., D. G. WILLIAMS, AND S. M. SCHMITT. 1987. Infection of white-tailed deer (Odocoileus virginianus) in Michigan with Jamestown Canyon virus (California serogroup) and the importance of maternal antibody in viral maintenance. Journal of Wildlife Diseases 23: 12–22.

HOFF, G. L., D. O. TRAINER, AND M. M. JOCHIM. 1974. Bluetongue virus and white-tailed deer in an enzootic area of Texas. Journal of Wildlife Diseases 10: 158–163.

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.

QUIST, C. F., E. W. HOWERTH, D. E. STALLKNECHT, J. BROWN, T. PISELL, AND V. F. NETTLES. 1997. Host defense responses associated with experimental hemorrhagic disease in white-tailed deer. Journal of Wildlife Diseases 33: 584–599.

STALLKNECHT, D. E., V. F. NETTLES, E. A. ROLLOR, 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.

———, M. P. LUTTRELL, K. E. SMITH, AND V. F. NETTLES. 1996. Hemorrhagic disease in white-tailed deer in Texas: A case for enzootic stability. Journal of Wildlife Diseases 32: 695–700.

Received for publication 2 May 2001.