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Henningson: New Diagnostic Lab Pathologist

The K-State Veterinary Diagnostic Laboratory announces the addition of Jamie Henningson, D.V.M, Ph.D., Diplomate American College of Veterinary Pathologists. Henningson is a 2004 graduate of Kansas State University's College of Veterinary Medicine. During her fourth year of veterinary school, she completed a pathology externship at the National Animal Disease Center (NADC) in Ames, Iowa.

From June 2004 until September 2008 she was a veterinary anatomic pathology resident at the Nebraska Veterinary Diagnostic Center, University of Nebraska at Lincoln. Concurrent with the pathology residency, Henningson completed a Ph.D. program at the University of Nebraska. She conducted research on the effects of viral proteins on the virulence of bovine viral diarrhea (BVD) virus in cattle. She also participated in a research project investigating the prevalence of BVD virus in alpacas in the United States and studied the tissue distribution of BVD virus in persistently infected alpacas.

After finishing her Ph.D., Henningson spent two years as a clinical instructor in veterinary pathology at the University of Wisconsin's College of Veterinary Medicine, where she was actively involved with service, teaching, and research. In 2009, while at Wisconsin, she became a Diplomate of the American College of Veterinary Pathologists. She studied the effect of vaccination with canine influenza vaccine to prevent clinical disease and lesion development in dogs infected with canine influenza virus and Streptococcus zooepidemic.

Dr. Jamie Henningson

Before coming to K-State in November, Henningson held a post-doctoral position with the Swine Virus and Prion Research Unit at the (NADC). Her research at NADC primarily involved influenza virus and porcine reproductive and respiratory syndrome virus.

Pregnancy Testing Cows Essential this Fall

Larry C. Hollis, D.V.M., M.Ag, Extension Beef Veterinarian, and Andrea Sexten, Ph.D., Animal Sciences and Industry

This year's drought has left a shortage or absence of grass and hay throughout many parts of Kansas. Producers fortunate enough to have more hay than their projected winter needs should consider the high value and increased potential selling price of hay when making culling decisions.

It is essential that cows be pregnancy checked so valuable hay or stockpiled forage is not wasted on open cows this winter. Winter feed costs are expected to be as high as $500 to $600 per cow in many areas this year, so spending a few dollars to make sure cows are pregnant makes economic sense.

Before testing, observe cows to see if they are in poor body condition or have significant feet, leg, eye, and teeth problems. Cows with existing problems are less likely to forage well during the upcoming winter and should be automatically marked for culling.

Because of high feed prices, culling cows that do not exactly fit a producer's management program may also be in order. Follow the "4 O's" culling rule: cull open cows, old cows, oddball cows, and ornery cows.

Open

Unless a producer has a specific development program that will add enough value to offset winter feeding costs, open cows should be culled. Open cows can be real profit-robbers. If cows are not carrying a calf to repay the owner for their winter feed bill, they are living on the producer's generosity.

Old, oddball, and ornery

If feed resources are limited, producers' may need to cull some pregnant cows as well. Start with old cows that are currently in poor body condition. These

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cows will likely need extra feed to make it through the winter and raise a good calf next spring. Producers can sell these cows as bred cows now. Also sell younger cows that have been determined by pregnancy exam to calve late in the calving season next spring, to be at risk for not breeding back next year because of body condition concerns the producer does not have enough feed to correct now, or those whose pregnancies are projected to result in late-born, lightweight calves that will not repay winter feed costs. If a producer has some odd-conformation or odd-colored cows that will produce calves that do not match existing calves, they should also be sold as bred cows now.

K-State research indicates that calves in larger lot sizes of similar breed, color, muscling, frame size, and weight will receive a significantly higher price when marketed than mismatched calves. When pregnancy checking, observe each cow's disposition and ask the owner or manager to identify any cows that might have a serious attitude problem. Calves tend to act like their dams, and research has shown that calves with a nervous, flighty, or aggressive disposition perform poorly in the feedlot. Ornery cows should be sold as bred cows now. They may show less of their orneriness and bring more money if sent through the sale ring with another herd mate(s). Producers should never keep replacement heifers from ornery cows.

Special considerations

Producers may need to reconsider what is done with some of their open cows. This year, because of industry economics, some producers may want to consider keeping some of the better open, young cows through the winter if they have available feed resources. This is only a consideration if they know they will need to add females next year to repopulate their cow herd to normal levels.

Open females retained should be in good body condition, have healthy reproductive tracts, and only failed to rebreed because they calved late and were short on grass or because there were fertility problems due to excessive heat this past summer (make sure no Trich or Vibrio problems exist in the herd).

The producer has to consider what it will cost to purchase a replacement female next year (all indications are that they are going to be very high priced), and balance that against winter feed costs required to retain a good, young open cow. The difference between what the open animal will sell for now and what it will cost to replace her before the start of next year's breeding season could be the difference in profit or loss. It would help to know if we will get enough moisture this winter to have adequate grass available to repopulate or try to expand the breeding herd next year, but that is impossible to predict.

A lot of important decisions need to be made after learning the pregnancy status of individual cows this year. Many of the factors that will help allow your clients to remain or become a low-cost cow/calf producer are within your arm's length at pregnancy check time this fall.

Hemorrhagic Disease of Deer

Mark G. Ruder, D.V.M., Southeastern Cooperative Wildlife Disease Study

This year's drought and high temperatures plagued animal and crop agriculture throughout a large portion of Kansas. White-tailed deer populations in eastern Kansas, however, faced another problem, hemorrhagic disease. The Kansas Department of Wildlife, Parks, and Tourism (KDWPT) documented the disease in white-tailed deer throughout the eastern third of the state (Figures 1 and 2). Hemorrhagic disease is caused by either epizootic hemorrhagic disease viruses (EHDV) or bluetongue viruses (BTV) and is one of the most significant infectious diseases of white-tailed deer in North America. Reports of large-scale epizootic mortality of white-tailed deer date back to the late 1800s and early 1900s. Documented hemorrhagic disease outbreaks have occurred regularly in the United States since the viral etiology was discovered in 1955.

Distribution and host range

The EHDV and BTV serogroups are in the genus Orbivirus, family Reoviridae. Worldwide, there are seven EHDV serotypes (1, 2, and 4 through 8) and 26 serotypes of BTV. Both EHDV and BTV are arthropod-borne, infect a variety of wild and domestic ruminants, and are distributed in a wide band around the globe from approximately 40-50 °N to 35 °S latitude in climates supportive of the *Culicoides* spp. vector. The recent emergence of multiple BTV serotypes throughout northern Europe suggests that historic climatic barriers to orbiviral spread may no longer be intact.

Hemorrhagic disease has only been reported in wildlife in North America, where it is primarily a disease of white-tailed deer. Mortality, however, is occasionally reported in mule deer, American pronghorn, elk, and bighorn sheep. In domestic ruminants, severe disease has been largely confined to sheep infected with BTV. Although infection of cattle is common, disease is normally mild with BTV and inapparent with EHDV. Historically, the only viruses endemic to the United States were EHDV- 1 and 2, and BTV-2, 10, 11, 13, and 17. Additional serotypes have been detected over the past decade (EHDV-6 and BTV-1, 3, 5, 6, 9, 12, 14, 19, 22, and 24) (Allison et al, 2010; Ostlund, 2010). These recent findings in the United States, along with the recent BTV outbreaks in northern Europe, and reports of clinical EHD in cattle, remind us that there is still much to learn about the distribution, transmission, and pathogenesis of these viruses and has renewed scientific interest in them.

Transmission and epidemiology

Both EHDV and BTV are transmitted by *Culicoides* biting midges (Diptera: Ceratopogonidae), which are small (1 to 3 mm), primarily hematophagous flies

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Figure 1. Photograph of a white-tailed deer from Lyon County with acute hemorrhagic disease. This animal was found in water unable to rise and was euthanized. EHDV-2 was isolated from spleen. Note the epistaxis. Sick and dead deer are commonly found along waterways (Photo credit: Shane Hesting of KDWPT).

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that breed in a variety of semi-aquatic habitats. Approximately 1,400 *Culicoides* spp. exist worldwide; however, only a small portion are proven vectors. In North America, known competent vectors of BTV include *Culicoides sonorensis* and *C. insignis*, whereas the only proven vector of EHDV in North America is *C. sonorensis*. Other species, however, are suspected vectors and may play a role in transmission.

The duration of the midge life cycle varies with climatic conditions. When conditions are optimal, populations can rapidly reach a very high density. Additionally, replication of the viruses within the midge is temperature-dependent. These factors, among others, likely play a significant role in spawning large outbreaks. Midge attack rates on preferred hosts are highest on warm summer nights lacking significant winds.

Hemorrhagic disease is predictably seasonal, with outbreaks peaking in late summer to early fall and ending after the first frost. In the United States, hemorrhagic disease exists in both enzootic and epizootic cycles. Which cycle predominates likely depends on the virus serotype, strain, and dosage; vector competency and abundance; and host-related factors. Examining the United States landscape via habitat type and physical geographic regions helps to elucidate whether epizootic or enzootic cycles exist, as well as how the disease will clinically manifest in white-tailed deer. The driving forces behind this variation among physical geographic regions likely results from a combination of factors such as vector activity and host-acquired and innate resistance. In endemic regions of the coastal plain in the southeastern United States where midge activity can persist much of the year, hemorrhagic disease occurs annually and most cases are subclinical or occur as the chronic form.

Moving inland into the Piedmont and Appalachian Mountains in the Southeast and in portions of the Midwest, epidemics with high mortality generally occur every 3 to 5 years. Epidemics are even more infrequent in more northern latitudes and result in significant mortality when they occur. These spatial and temporal patterns, however, are general trends, and isolated epizootics involving small numbers of animals likely occur regularly in both enzootic and epizootic regions. In addition to epizootic and enzootic cycles, it has been suggested that in certain regions of the United States, such as parts of Texas and Florida, white-tailed deer and these orbiviruses coexist in a state of enzootic stability. In these regions, seroprevalence among white-tailed deer may approach 100 percent; however, reports of clinical disease are extremely rare. This enzootic stability is likely the result of acquired immunity via frequent exposure to the viruses, high subsequent passive immunity in fawns, and mechanisms of innate immunity via coevolution of the host and pathogen.

Evidence to support these mechanisms has been demonstrated during experimental infections and discussed elsewhere (Howerth et al., 2001; Stallknecht et al., 2002). These mechanisms of immunity are potentially lost when penned deer from non-endemic regions are imported to endemic regions, possibly explaining the high mortality rates observed in some captive white-tailed deer operations.

Impacts of hemorrhagic disease to wild populations are not well documented. Mortality rates in wild populations are difficult to estimate, but may range from a few individuals to greater than 20 percent of a herd. In general, higher mortality rates are observed at more northern latitudes and losses in penned deer have approached 90 percent. Although localized population declines certainly occur after a severe hemorrhagic disease outbreak, herds likely recover within a few years. Repeated outbreaks in many regions of the United States have not limited population growth over time. Other mortality factors in the population, however, could slow the rate of recovery, or in some scenarios, have been suggested to contribute to long-term declines.

Clinical signs and pathology

Following a bite from an infected midge, primary virus replication occurs in regional lymph nodes before disseminating to secondary replication centers, including endothelium. A highly erythrocyte-associated viremia ensues, which peaks around day 6 but may persist at a very low titer for up to 60 days postinfection. The

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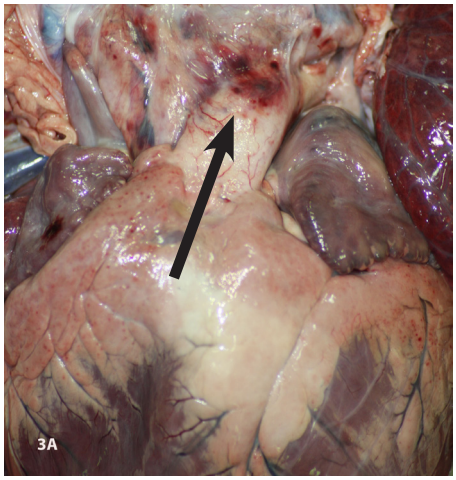
widespread viral replication in endothelium and subsequent vascular damage in susceptible species, such as white-tailed deer, results in a consumptive coagulopathy with concurrent fibrinolysis that often results in disseminated intravascular coagulation. The ensuing hemorrhagic diathesis generally culminates in death and is what gave hemorrhagic disease its name.

In white-tailed deer, disease caused by EHDV and BTV are indistinguishable, which is why both are etiologic agents of hemorrhagic disease. The disease is highly variable in white-tailed deer, ranging from subclinical to peracute death without premonitory signs. Some animals recover after mild disease, whereas some succumb to chronic sequelae months after acute disease.

The myriad of symptoms and lesions potentially observed with hemorrhagic disease is refined when the disease is categorized into one of three forms: peracute, acute, or chronic. The peracute form is primarily characterized by edema and cases generally present as peracute death with or without premonitory signs. In addition to edema, congestion, hemorrhage, and ulcerative lesions are seen in the acute or classical form. The chronic form develops following recovery and is typified by hoof abnormalities, ruminal scarring, and subsequent emaciation in the winter months. The chronic form does not represent active disease, but rather sequelae after recovery from acute hemorrhagic disease.

Common clinical signs initially include fever, loss of appetite, rough hair coat, lethargy, hyperemia of mucous membranes and non-haired regions of the skin, and ptyalism. Additional clinical signs include oral hemorrhages eventually progressing to erosions and ulcerations, hemorrhages in the skin and coronary bands with or without lameness, bleeding tendencies, respiratory difficulty, bloody diarrhea, recumbency, and terminal convulsions. Death typically occurs between day 4 to 15 postinfection.

Gross postmortem findings are highly variable and can be very subtle when animals die peracutely. Common early findings include pleural, pericardial, and peritoneal effusions, pulmonary edema, and subcutaneous edema, especially in the fascial planes of the head, neck, and ventrum. As the disease progresses and a coagulopathy ensues, widespread hemorrhages are common, especially in the gastronintestinal tract and heart. When present, two nearly pathognomonic gross findings of hemorrhagic disease in white-tailed deer are hemorrhage at the base of the pulmonary artery and hemorrhage on the serosal surface of the pylorus (Figure 3). With time, erosions and ulcers are frequently observed in the oral cavity. Survivors may present with lesions of chronic hemorrhagic disease including scaring of the tongue and rumen mucosa and cracked or sloughing hooves subsequent to interruption of growth of the hoof wall. These animals may present as over-winter mortality subsequent to malnutrition or secondary problems.



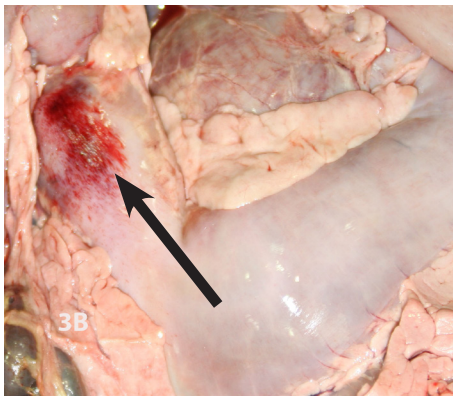


Figure 3. Hemorrhage at the base of the pulmonary artery (3A) and on the serosal surface of the pylorus (3B) are two nearly pathognomonic lesions of hemorrhagic disease in deer, when present.

Diagnosis

Epizootic mortality in a herd during transmission season should lead to the suspicion of hemorrhagic disease. Diagnosis of hemorrhagic disease should be based on virus isolation coupled with RT-PCR of blood or tissues. Preferred diagnostic samples include fresh (not frozen) spleen, lung, lymph node, or whole blood (EDTA, heparin, or citrate). Since subclinical infection of white-tailed deer is not uncommon, positive test results must be supported by clinical or postmortem findings consistent with hemorrhagic disease. This consideration is especially true when testing domestic ruminants, or other less susceptible wildlife species. The chronic form of hemorrhagic disease is typically diagnosed by consistent postmortem findings and history. Circulating antibodies against EHDV and BTV are common in white-tailed deer throughout many portions of the United States, thus serology should not be relied upon for diagnosis of recent infection unless comparison of acute and convalescent titers can be made.

Hemorrhagic disease in Kansas

This year's hemorrhagic disease outbreak occurred in the eastern third of the state, but reports of mortality extended as far west as McPherson and Butler counties. This eastern distribution is consistent with the historical distribution of hemorrhagic disease reports in the state (Figure2). Reports of hemorrhagic disease in the western half of the state are extremely rare. This apparent discrepancy of epidemiologic patterns of hemorrhagic disease in Kansas is interesting, as the state may represent a transition area between the epizootic cycle and enzootic stability. Flacke et al., (2004), suggest that the mechanisms of enzootic stability described above function in the western third of the state, whereas epizootic disease is common in eastern Kansas. This evidence was based on rare historical accounts of hemorrhagic disease from western Kansas

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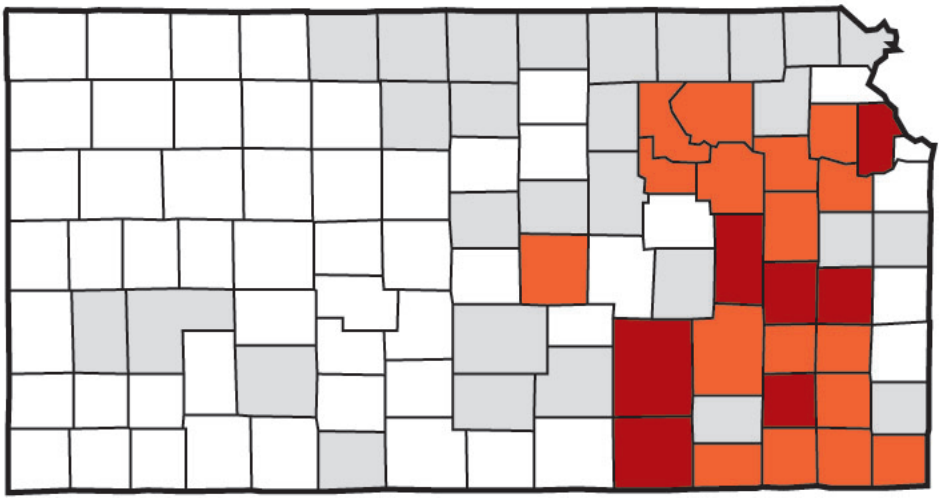

Figure 2. Map shows the distribution of hemorrhagic disease reports in Kansas. Red are counties reporting white-tailed deer mortality during 2011. Dark red are counties that EHDV-2 was isolated from dead white-tailed deer. Gray are counties that have reported hemorrhagic disease mortality from 1983 to 2011.

and a relatively high seroprevalence to EHDV and BTV among white-tailed deer and mule deer sampled for the study.

This epizootic pattern of hemorrhagic disease was apparent this past summer and fall throughout eastern Kansas. Personnel from the Kansas Department of Wildlife, Parks and Tourism investigated multiple reports of sick and dead deer this past August through October. When carcasses were recovered fresh, field necropsies were performed, and tissue samples were submitted for virus isolation to Southeastern Cooperative Wildlife Disease Study at the University of Georgia. In all, there were reports of white-tailed deer mortality in 24 counties and EHDV-2 was isolated from dead deer in seven of these counties. Although there may be localized virus transmission nearly every summer in Kansas, large outbreaks generally occur every 3 to 6 years. Before this year's outbreak, other significant outbreaks were recorded in 2007, 2003, 2002, 1996, and 1988.

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How to Live a Good Life

If a dog was the teacher, you would learn:

- When loved ones come home, always run to meet them.
- Never pass up the opportunity to go for a joyride.
- Allow the experience of fresh air and the wind in your face to be pure ecstasy.
- Take naps.
- Stretch before rising.
- Run, romp, and play daily.
- Thrive on attention and let people touch you.
- Avoid biting when a simple growl will do.
- On warm days, stop to lie on your back on the grass.
- On hot days, drink lots of water and lie under a shady tree.
- When you are happy, dance around and wag your entire body.
- Delight in the simple joy of a long walk.
- Eat with gusto and enthusiasm. Stop when you have had enough.
- Be loyal. Never pretend to be something you are not.
- If what you want lies buried, dig until you find it.
- When someone is having a bad day, be silent, sit close by, and nuzzle them gently.
- Be always grateful for each new day and for the blessing of you.
- Enjoy every moment of every day.

Continuing Education

January 13, 2012
Bull Evaluation and Management Clinic

February 3, 2012
Annual Conference on Animal Diagnostics and Field Applications

April 29, 2012
Frank W. Jordan Seminar – "The Science Behind Alternative Medicine in Animal Health"

For the most complete, up-to-date conference information visit our website at: *www.vet.ksu.edu* and click on Continuing Education, or contact: Megan Kilgore at 785-532-4528 or *meganlk@vet.ksu.edu*

Upcoming Events

January 7, 2012
Kansas Junior Beef Producer Day, Manhattan, Kan.

January 10, 2012
Winter Ranch Management Seminar, 111 Weber Hall

February 28, 2012
Swine Profitability Conference, K-State Student Union

March 1, 2012
42nd Annual LMIC Stockmen's Dinner, Clarion Hotel

March 2, 2012
99th Annual Cattlemen's Day, Weber Hall

March 2, 2012
35th Annual Legacy Bull and Heifer Sale, Purebred Unit

March 3, 2012
KSU Sheep Day and Ribbon Cutting for new Sheep and Meat Goat Center

March 31, 2012
Kansas Junior Sheep Producer Day

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