virus serotypes, indicating a high rate of previous viral infection: however, hemorrhagic disease in these regions is rare. Abundant midge populations probably cause annual virus activity resulting in constant herd immunity and protection from disease. Deer in more northerly state experience hemorrhagic disease sporadically, but outbreaks are more severe with higher mortality. EHD and BT viruses may not be able to continually persist in these regions because the midge species present are inefficient vectors or generally are not very abundant. Following the outbreak, herd immunity declines over time if there are no further virus activity, and subsequent outbreaks occur as virus spreads into non-immune deer populations. A general rule for the eastern half of the United States and the Midwest, is that the frequency of hemorrhagic disease outbreaks and infection disease as latitude increases (move northward). However, the severity of clinical signs and mortality also increase with increasing latitude. Possible explanations for this regional pattern of disease include maternal protective immunity, acquired immunity from previous infections with similar serotypes of EHD or BT virus, and innate resistance of some subspecies (or regional populations) of deer to clinical disease.

Impact of Hemorrhagic Disease On Deer Populations

The severity and distribution of hemorrhagic disease are highly variable. Past occurrences have ranged from a few scattered mild cases to dramatic outbreaks. Death losses during outbreaks usually are well below 25 percent of the population but in a few instances have been 50 present or more. To date, repeated HD outbreaks have not represented a limiting factor to deer population growth. Although it is logical to assume that host population density could affect the severity of HD, there is very limited evidence that severity of disease is related to population density.

Diagnosis of Hemorrhagic Disease

A strong tentative diagnosis can be made on the basis of history and presentation, combines with field necropsy and observation of lesions. A confirmed diagnosis of EHD or BT virus infection requires virus isolation or polymerase chain reaction (PCR) detection of viral nucleic acids. The preferred specimens for the virus isolation or PCR are refrigerated whole blood in anticoagulant and refrigerated spleen, lymph node, and lung from a fresh carcass. Contact the diagnostic laboratory prior to shipping the sample in order to obtain advice on collection and shipment of specimens.

Human Health Implications

These viruses do not infect humans, and humans are not at risk by handling infected deer, eating venison from infected deer, or being bitten by infected *Culicoides* vectors. Deer that develop bacterial infections or abscesses secondary to hemorrhagic disease may not be suitable for consumption.

Livestock Implications

Past observations have revealed that simultaneous infections sometimes occur in deer, cattle, and sheep. If the vector and virus are present in the vicinity, both deer and livestock are at risk of infection. While the significance of EHD and BT viruses to white-tailed deer is established, the importance of these agents to domestic livestock is more difficult to assess. Most BT virus infections in cattle are subclinical; however, a small percentage of animals can develop fever, lameness, sore mouths, and reproductive problems. Less is known about EHD virus on cattle. EHD virus has been isolated from sick cattle, and surveys have shown that cattle often have antibodies to this virus, indicating frequent exposure. Domestic Sheep are generally unaffected by EHD, but BT can cause a serious disease similar to that in deer. Hemorrhagic disease can have severe impacts in captive white-tailed deer, especially in animals translocated from northern to endemic area in the southern United States. Vaccines are not currently available and have not been tested in white-tailed deer.

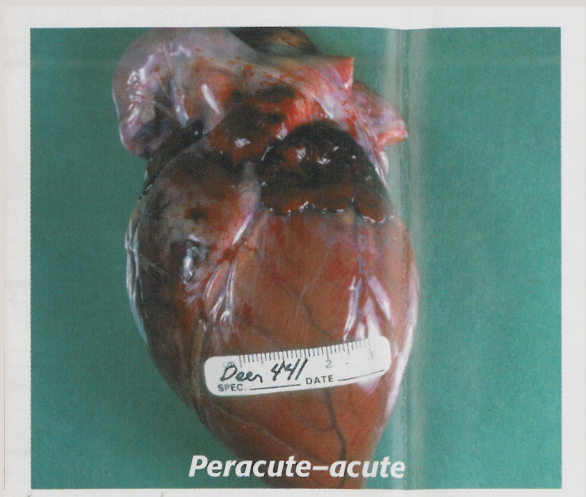
Control and Prevention of Hemorrhagic Disease

At present, there are no wildlife management tools or strategies available for prevent of control hemorrhagic disease. Although die-offs of whitetails du to hemorrhagic disease often cause alarm, past experiences have shown that mortality will not decimate local deer populations and that the outbreak will be curtailed by the onset of cold weather. Livestock owners who suspect EHD or BT virus infections should seek veterinary assistance to get diagnostic support and supportive care for their animals.

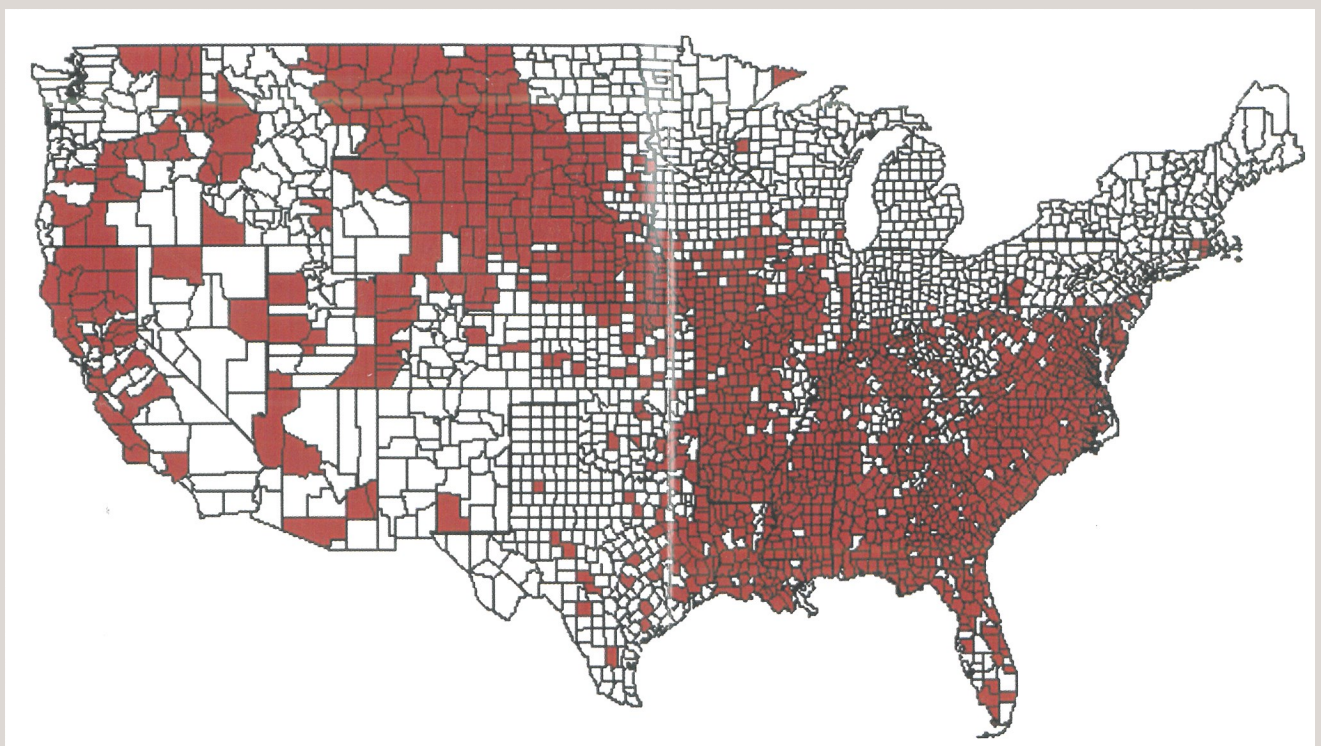
Important Questions Concerning Hemorrhagic Disease in Deer

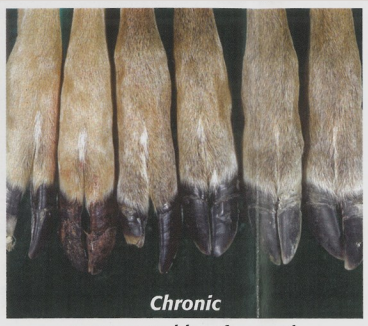
What are the Clinical Signs of Hemorrhagic Disease

Clinical signs of hemorrhagic disease are highly variable and many infected deer appear normal or show only mild signs of the illness. When illness occurs, the signs change as the disease progresses. Initially animals may be depressed and feverish, with a swollen head, neck, tongue, or eyelids and breathing difficulty. Deer may dies within 1 to 3 days. More often, deer survive longer and may become lame, lose their appetite, or reduce their activity. A smaller proportion of infected animals may be disabled for weeks and months by lameness and emaciation. Lesions, as with outward signs, can be quite variable in deer depending on the immune status of the host and duration off infection. The development of different lesions as the disease progresses has led to categorization of 3 “forms” of hemorrhagic disease: peracute, acute, and chronic. The peracute, or very rapid form, shows only severe fluid swelling (edema) of the head, neck, tongue, eyelids, and lungs. In animals living somewhat longer, the acute or “classic hemorrhagic” form occurs. These animals may have edema in the same locations but also have hemorrhagic or congestion in the heart, pulmonary artery, oral mucosa, rumen, abomasum, or intestines. There may be erosions or ulcerations on the dental pad, tongue, palate, rumen, omasum, and abomasum. The chronic form is typified by growth interruptions on the hooves and sometimes peeling of hoof walls. Other chronic lesions include ulceration, scarring, and loss of papillae in the rumen, emaciation during the winter months, and rarely antler malformations. The chronic form is a sequel or delayed manifestation of hemorrhagic disease. The virus is no longer present within the animal and therefore does not represent a truly chronic infection. It should be emphasized that all of the lesions with not be found in an individual deer and other disease also produce similar edematous, hemorrhagic, or ulcerative lesions.

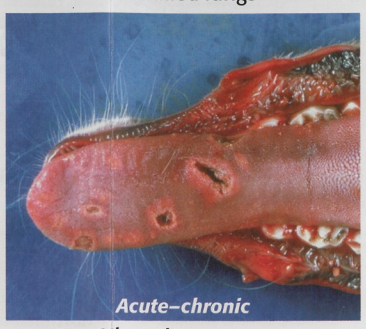


Cardiac hemorrhage

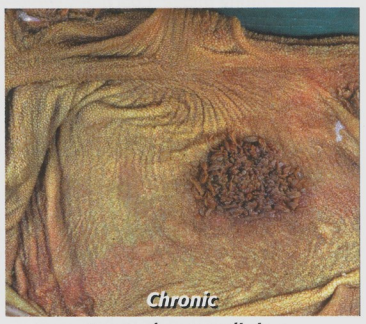

Nationwide distribution of acute or chronic hemorrhagic disease in wild deer between 1980 and 2003.



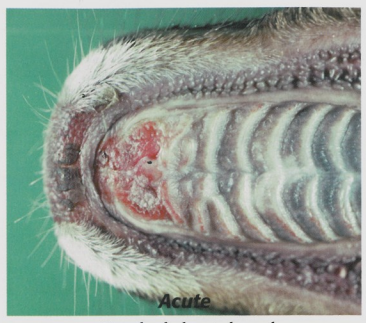
Interrupted hoof growth



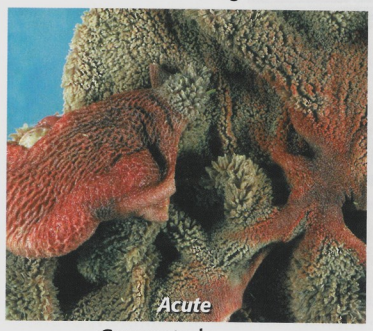
Ulcers in tongue



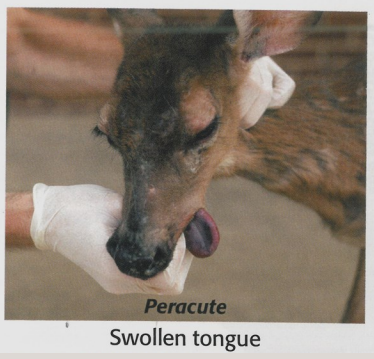
Scarred rumen lining



Eroded dental pad



Congested rumen



Swollen tongue

Hemorrhagic Disease of White-tailed Deer

Hemorrhagic disease is the most important viral disease of white-tailed deer in the United States, and outbreaks occur every year in the Southeast. The disease is caused by related orbiviruses (*Reoviridae*) in the epizootic hemorrhagic disease (EHD) or bluetongue (BT) virus serogroups. Because the disease is produced by both the EHD and BT viruses is indistinguishable, the general term, hemorrhagic disease, often is used when the specific virus responsible is unknown. The EHD and BT viruses are transmitted by biting flies, and as a consequence, hemorrhagic disease is seasonal and occurs in late summer and fall (approximately late July through November).

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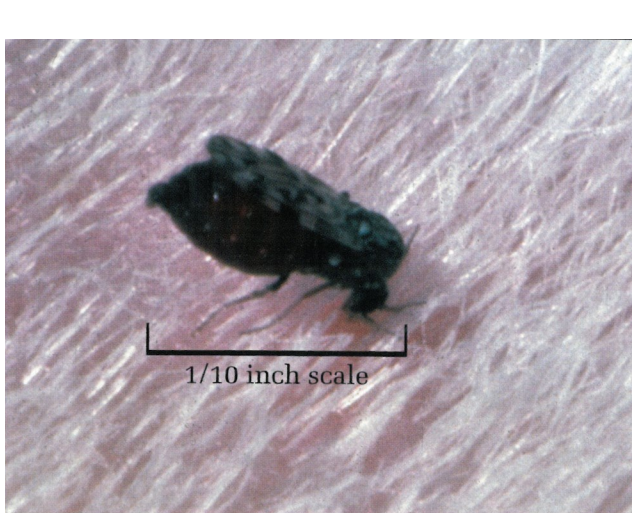
Causative Agents

In North America there are 2 subtypes of EHD virus (EHDV 1 and 2) and 5 subtypes of BT virus (BTV 2, 10, 11, 13, and 17). Isolations of EHD and BT virus from infected deer were first reported in 1955 and 1968, respectively, but white-tailed deer die-offs consistent with hemorrhagic disease were noted as early was 1886. In 2004, a sixth BT virus subtype (BTV 1) was isolated from a single white-tailed deer in Louisiana. The significance of this finding is currently under investigation.

The Vectors

In free-ranging populations of deer, EHD and BT viruses are transmitted by biting files in the genus *Culicoides*. The best documented vector in North America is *Culicoides sonorensis*, although other species of *Culicoides* may play a role in the local transmission of these viruses within certain regions, such as *C. insignis* along the Gulf Coast. These flies are known as biting midges but also are called sand gnats, sand flies, no-see-ums, and punkies. The seasonal occurrence of hemorrhagic disease coincides with periods when biting midges are abundant. The onset of freezing temperatures in late fall affects vector populations and usually brings a sudden end to hemorrhagic disease outbreaks. How it is believed that in areas with mild climate , vector populations may remain active and locally support year round virus transmission.



Adult female biting midge, *Culicoides variipennis*, feeding upon a laboratory rabbit. Hemorrhagic disease viruses are spread by these small files. (photo by Charles McKinnon, ARS, USDA)

Susceptible Wildlife Hosts

Although EHD and BT viruses are infectious to a wide range of wild ruminants, susceptibility varies among species. Clinical disease due to EHD has been reported in white-tailed deer, mule deer, bighorn sheep, elk, and pronghorn and clinical disease due to BT has been reported in these species and also black-tailed deer. Infections in these wild ruminants can range from mild or no disease to episodes of high mortality. Antibodies or virus have also been detected in bison and mountain goats; however, these infections were not associated with disease. In the Southeast, mild infections in white-tailed deer are common and are evidenced on by antibodies to the viruses in serum of normal, healthy deer.

When Should You Suspect Hemorrhagic Disease?

Hemorrhagic disease should be suspected in instances of unexplained deer mortality during late summer or early fall, especially of any of the characteristic signs of lesions are noted. An easy lesion to see in the field is the erosion on the dental pad. Because deer have a high fever, they often are found near water. Sick or dead deer should be reported promptly to state wildlife agency personnel because other native diseases and some foreign diseases resemble hemorrhagic disease. Also, prompt notification and submission of the carcass will facilitate diagnostic procedures. If hunter-harvested deer have growth interruptions in their hooves or chronic lesions of the rumen lining, previous exposure to EHD or BT virus can be suspected. However, virus is no longer present in deer with chronic lesions and therefore virus isolation is not possible. Serum tests for antibodies from hunter-harvested deer may be used to estimate previous EDH of BT virus activity in a herd.

Were Do EHD/BT Infections and Hemorrhagic Disease Occur?

Infection refers to the invasion and multiplication of virus in deer and other ruminants, while disease refers to the production of noticeable clinical signs. The location of hemorrhagic disease outbreaks in white-tailed deer from 1980-2003 are shown on the map. Monitoring natural outbreaks of hemorrhagic disease over the last thirty years has shown that infection of white-tailed deer with EHD and BT virus is much more common and geographically widespread than clinical disease. Several disease patterns exist with both EHD and BT viruses, which range from unapparent infection of various combinations of death, acute clinical signs, and/or chronic hoof lesions. Deer herds in the Southeastern coastal plains, South Florida, and Texas consistently have antibodies to multiple EHD and BT