

DEER DOWNTOWN: URBAN DEER DISEASE AND MANAGEMENT

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US DEER SPECIES

Two species of deer occur in the United States, mule and black-tailed deer (*Odocoileus hemionus*) and white-tailed deer (*O. virginianus*). The two species are morphologically and behaviorally distinct but hybridization in the wild has been reported. White-tailed deer are distinguished most easily by having a tail that is brown above and white laterally and below. Their antlers have one main beam with minor branches, the length of the ear is half the length of the head, the preorbital pit is shallow, and the metatarsal gland is less than 42 mm long. Mule deer have a tail that is white or black above and tipped with black. Their antlers branch into two nearly equal parts, the length of the ear is two-thirds to three-fourths the length of the head, the preorbital pit is about 22 mm deep, and the metatarsal gland is greater than 70 mm long.

MANAGEMENT

Following the arrival of European settlers in North America, hunting for the massive procurement of deer hides and meat significantly reduced most deer populations throughout the United States. Highly regulated hunting seasons, restocking efforts, and conversion of habitat to more “deer-friendly” areas has resulted in the recovery of most deer populations and in certain locations, deer populations are believed to be larger now than during pre-European settlement. Exceptions include the Key deer subspecies, *O. virginianus clavium*, and the Columbian white-tailed deer subspecies, *O. virginianus leucurus*, which are both listed as endangered under the US Endangered Species Act.

Odocoileus deer are host to numerous bacteria, viruses, fungi, internal and external parasites as well as a prion protein.¹ Some are capable of causing disease in deer, others in domestic animals, and some in humans. Understanding the epidemiology of these agents is becoming ever more important as deer populations expand into more suburban and urban environments.

ORBIVIRUS HEMORRHAGIC DISEASES

Viruses in the epizootic hemorrhagic disease (EHD) and bluetongue (BLU) virus serogroups cause a clinically and pathologically indistinguishable disease often referred to as *hemorrhagic disease*. Undoubtedly, hemorrhagic disease is one of the most important diseases of deer in North America. Within the United States, BLU serotypes 1, 2, 3, 5, 6, 10, 11, 13, 14, 17, 19 and 22 and EHD serotypes 1, 2 and 6 are reported. Accounts of mortality associated with these viruses in white-tailed deer and mule deer date back to 1886 and 1901. Both BLU and EHD viruses are vectored by

Culicoides midges and hemorrhagic disease generally occurs in the late summer and early fall, likely in association with the seasonal patterns of vector activity. Disease can be peracute, acute, or chronic. Peracute cases present with pulmonary congestion, hemorrhage and edema. In acute cases, petechia, ecchymoses, hydropericardium, oral erosion, necrotizing glossitis, and hemorrhage in multiple tissues is seen. Chronic cases exhibit healed or healing erosions in the oral cavity, forestomach and abomasum, atrophy and fibrosis of rumen papillae, partial or complete hoof sloughing, and severe emaciation or winter mortality associated with reduced absorption of nutrients from the gastrointestinal tract. Cattle often develop subclinical infection when infected with BLU and EHD viruses, but more severe disease, including mortality, can occur. Sheep are very susceptible to BLU infection, but clinical disease in sheep has not been reported with EHD. Important differential diagnosis for deer with clinical signs of Orbivirus hemorrhagic disease include adenovirus hemorrhagic disease, vesicular stomatitis, malignant catarrhal fever, and some foreign animal diseases such as heartwater (caused by *Ehrlichia ruminantium*), foot-and-mouth disease, Rinderpest, and peste des petits ruminants.

ADENOVIRUS HEMORRHAGIC DISEASE

In 1993 a novel Adenovirus was discovered after it caused a hemorrhagic disease epizootic in mule deer throughout northern and central California. Clinical signs were similar to those caused by EHD and BLU viruses with the exception that microscopically, deer had widespread systemic vasculitis with endothelial intranuclear inclusions. In 1997, the virus also has caused mortality in three captive white-tailed deer in Iowa. Likely the disease is much more widely distributed but due to the lack of a good serologic test, the current distribution is unknown. It is possible that Adenovirus hemorrhagic disease mortality often goes undiagnosed or misdiagnosed as Orbivirus hemorrhagic disease, underscoring the need to establish an etiology during hemorrhagic disease outbreaks when possible.

BOVINE TUBERCULOSIS

Prior to 1994, infections of bovine tuberculosis (TB) in free-ranging white-tailed deer were considered sporadic spill-over cases from TB-infected domestic cattle. In 1994 a hunter-harvested deer tested positive for Bovine TB and further testing of other deer revealed that the disease was being maintained in the free-ranging white-tailed deer population and not in local infected livestock. Previously, it was thought that Bovine TB, caused by the acid-fast bacilli *Mycobacterium bovis*, could not be maintained in free-ranging deer populations. High deer densities, supplemental deer feeding, and subsequent artificial crowding of deer were implicated as reasons that this deer population was able to maintain the disease. Since its discovery in free-ranging white-tailed deer, other wildlife species (spill-over infections) have been detected with Bovine TB as have numerous cattle

herds. Current control strategies include reduction of deer density through increased hunter harvest and restrictions on artificial feeding and baiting of deer.

Since 2005, Bovine TB similar to a strain found in cattle in the southwestern US and Mexico has been identified in multiple cattle herds. Bovine TB also has been diagnosed in free-ranging white-tailed deer proximal to infected cattle herds. Unlike in Michigan, Bovine TB in Minnesota white-tailed deer is believed to be the result of spill-over from infected domestic cattle. Efforts are underway to dramatically reduce deer density and the potential for the disease to become established in free-ranging deer.

BOVINE VIRAL DIARRHEA

Bovine viral diarrhea virus (BVDV) is a positive-stranded RNA pestivirus in the family *Flaviviridae* that causes early embryonic death, abortion, congenital defects and a variety of postnatal clinical manifestations in cattle. Mule deer and white-tailed deer also can be infected with BVDV and if pregnant when infected, can result in persistently infected offspring. Some experimental infections of deer (both white-tailed and mule deer) with BVDV have demonstrated subclinical infections, while others have resulted in fetal resorption, mummification and abortions. While persistently infected cattle are considered the most important source of infection in cattle, deer transiently or persistently infected with BVDV also could be a source of infection in non-infected cattle herds. The role of BVDV in causing disease in deer needs to be better investigated, as does the role that deer could play in infecting noninfected cattle herds.

CHRONIC WASTING DISEASE

Chronic wasting disease (CWD) is a prion-caused transmissible spongiform encephalopathy of wild cervids. First documented in mule deer in 1967 and white-tailed deer in 1981, CWD causes intracytoplasmic vacuolization in neuronal perikarya and neuropil in the gray matter of the brain and spinal cord. Clinical signs include ataxia, weakness, inability to stand, dehydration, rough hair coat, excessive salivation, and severe emaciation. Once thought to be limited to an endemic focus in northeastern Colorado and southeastern Wyoming, CWD has now also been identified in captive or free-ranging white-tailed or mule deer in Nebraska (1999), Wisconsin (2001), New Mexico (2002), Illinois (2002), Alberta (2002), Utah (2003), New York (2005), West Virginia (2005), Kansas (2006), Minnesota (2006), and Michigan (2008). Disease transmission is believed to be lateral and transmission via environmental contamination likely plays an important role in disease persistence and spread. The multiple independent foci of disease across the country and frequent association with captive cervid farming suggest that in at least some instances, the spread of CWD is related to the movement of infected captive cervids. Strategies for limiting the spread of CWD include the reduction of deer

density in infected areas and minimizing the movement of infected deer or deer carcasses.

HAIR LOSS SYNDROME IN BLACK-TAILED DEER

During 1995, biologists in Washington State first noticed an unusual syndrome of hair loss in Columbian black-tailed deer (*O. hemionus columbianus*), a subspecies of mule deer. The condition spread from the original nidus in west-central Washington, throughout the western portion of the state, and by 1998 it also was seen in deer west of the Cascade Mountains in Oregon. Animals with hair-loss syndrome (HLS) are usually thin or emaciated with loss of pelage over the thorax, flanks, and hindquarters. This loss of hair often is asymmetric and perceived first as a change in coat color, probably due to removal of outer guard hairs and thinning of hair coat. The disease is distinctly seasonal with highest prevalence in winter and spring and is most prominent in does and fawns, especially deer between 6 and 12 months of age. A florid infestation with chewing lice is one of the few consistent pathologic findings in HLS-affected deer, and it appears that an exotic louse *Damalinia (Cervicola)* sp is responsible for the condition. *Damalinia (Cervicola)* is an Old World louse that transferred from another species. A hypersensitivity/inflammatory response is likely an important component of the syndrome as are the indirect effects of pediculosis including decreased feeding time due to pruritus or decreased thermoregulatory ability due to hair loss. The impact of these could be especially important for deer that have minimal energy reserves due to poor quality winter browse and to animals carrying large numbers of endoparasites. Affected deer experimentally treated with ivermectin subcutaneously at doses between 0.2 and 1.3 mg/kg of body weight monthly for four consecutive months gained significantly more weight than untreated deer and regrew their hair at a faster rate than untreated deer. Lice and all nematode eggs and larval stages in feces were eliminated or greatly reduced following treatment. Strategies for controlling the disease on the population level have not been tried and it will be interesting to see if this host–novel parasite relationship develops a less pathogenic equilibrium.

TAKE-HOME MESSAGES

Although few diseases of deer permit widespread opportunities for intervention and treatment at the population level, a thorough understanding of the epidemiology of these diseases lends itself to several important take-home messages regarding the management of deer and other wildlife diseases:

1. Work with local natural resource agencies and diagnostic labs to identify the etiologic agent involved in deer epizootics. Commonly seen diseases like Orbivirus hemorrhagic disease can be clinically indistinguishable from other important diseases like Adenovirus hemorrhagic disease or numerous foreign animal diseases.

2. Modifying the natural history of wildlife through artificial feeding is a prescription for problems, as was demonstrated in the Bovine TB outbreak in Michigan.
3. Avoid moving deer and other wildlife when possible. Movement of wildlife was likely involved in the spread of CWD as well as in the introduction of the exotic chewing louse *Damalinia (Cervicola)* sp implicated in hair loss syndrome.
4. Understanding the epidemiology of deer diseases allow us to better assess risk (to wildlife and humans) as well as to manage them. Bovine TB in Minnesota is different than Bovine TB in Michigan

and our true understanding of the role of deer persistently infected with BVDV in infecting cattle will only come with a better understanding of the pathogenesis and epidemiology of the disease in deer.

REFERENCES

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