Abstract

Viral, bacterial, and parasitic diseases are considered important potential causes of mortality among mule deer populations. Various aspects on the pathologic manifestations and epizootiology are presented for the hemorrhagic disease complex (bluetongue and epizootic hemorrhagic disease), necrobacillosis, gastrointestinal trichostrongylosis and lungworm disease.

The importance of diseases affecting human and livestock health long has been recognized and is evidenced in the United States today by enormous expenditures by the Departments of Health, Education, and Welfare and Agriculture as well as by states and private philanthropic agencies. In contrast, diseases of free-living wild animals largely have been ignored, and only recently have diseases of wildlife received attention. As a result, a relative dearth of knowledge exists on diseases affecting wild populations. This fact was all too evident when searching the literature for accounts of diseases affecting mule deer (Odocoileus hemionus). As a result, this presentation is largely speculative based on information derived from the literature and extrapolated from our experiences with diseases of white-tailed deer (O. virginianus).

In 1969 we presented a list of 12 fundamental causes of morbidity and mortality among wildlife populations (Table 1) and described a process of elimination whereby potential causes of death could be narrowed from 12 to 3 or 4 (Hayes and Prestwood 1969). When considering diseases potentially capable of causing a widespread decline of mule deer populations, we applied this method of elimination. As a result, a list of diseases of potential importance to mule deer populations was compiled (Table 2).

Viral Diseases - Three viral agents producing two clinical syndromes, hemorrhagic disease (HD) and malignant catarrhal fever (MCF) respectively, are considered threats to mule deer populations.

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<th>Table 1. Fundamental Causes of Morbidity and Mortality Among Wild Animal Populations</th>
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The so-called "hemorrhagic complex" consists of two viral diseases, bluetongue (BT) and epizootic hemorrhagic disease (EHD), which cannot be distinguished clinically or by gross or microscopic lesions. Hemorrhagic disease is characterized by hemorrhage and vascular thrombosis with resultant necrosis of affected tissues.

Bluetongue is enzootic throughout much of the United States. In 1974 BT virus was recovered from
herds of cattle in Colorado and Oregon and from sheep flocks in California, Idaho, New Mexico, Oregon, and Texas (Shoenfeld et al. 1974). In addition, BT-positive modified complement-fixation test reactors were detected in Arizona, Arkansas, California, Colorado, Florida, Idaho, Indiana, Kansas, Kentucky, Louisiana, Missouri, Montana, Nebraska, Nevada, New Mexico, New York, North Carolina, North Dakota, Ohio, Oklahoma, Oregon, South Dakota, Texas, Utah, Virginia, Washington, Wisconsin, and Wyoming (Shoenfeld et al. ibid). This agent appears enzootic in the south Atlantic and Gulf coastal plain of the southeastern United States (Thomas and Prestwood 1976) and Texas (Hoff et al. 1974; Marburger et al. 1970). The virus of EHD similarly is widespread throughout much of the United States and Canada. Sero-positive EHD plaque reduction neutralization test reactors were found widely distributed among white-tailed deer populations of the southeastern United States prior to the 1971 epizootic of HD (Thomas and Prestwood 1976). Hemorrhagic disease has been seen annually in this region since that time.

During the 1975 epizootic of HD in New Jersey, only one virus--EHD--was isolated (Anon. 1976). This was in contrast to the 1971 outbreak in the Southeast where both BT and EHD viruses were involved (Prestwood et al. 1974). Similarly only EHD virus was isolated from deer dying of HD in North Dakota during 1971, however, sero-positive reactors to BT were found among hunter-killed deer following that mortality (Hoff et al. 1973).

The virus of BT disease is transmitted by biting midges (Culicoides). Recent unpublished evidence suggests that EHD virus also is transmitted by this vector. Hemorrhagic disease is seasonal and usually occurs in the late summer and early fall when flies are abundant. Epizootics characteristically cease with the first frost.

Sheep are severely affected by BTv, and vaccination is practiced by some sheep herders. Only occasionally is BT manifest clinically in cattle, however, these animals may harbor the virus inapparently for extended periods. This finding led to the hypothesis that cattle are reservoirs for BT virus (Bowen 1975).

The role of cattle in the epizootiology of EHD presently is unknown, however, antibodies to EHD have been detected among sentinel cattle placed in contact with white-tailed deer during an outbreak of HD and also in feral cattle in contact with whitetails where HD was not recognized (Thomas and Prestwood 1976).

Although we were unable to locate published reports of clinical BT in mule deer, one of the authors (CPH) has recovered BT virus from captive mule deer which were dying of an acute hemorrhagic disease syndrome. Mule deer reportedly are relatively resistant to infection with EHD virus, and in outbreaks of EHD, carcasses of white-tailed deer outnumber those of mule deer 23 to 1. Sero-positive reactors to EHD virus were prevalent among mule deer following an outbreak of EHD in deer of North Dakota during 1970 and 1971, suggesting that they were similarly exposed to this viral agent (Hoff et al. 1973). The effects of BT and EHD viruses, particularly latent infections, in mule deer require further study before these diseases can be properly assessed for mule deer populations.

Acute and chronic hemorrhagic disease in white-tailed deer have been delineated (Prestwood et al. 1974). The primary pathogenic mechanism appears to be disseminated intravascular coagulation (Tsai and Karstad 1973). The acute disease is characterized by extensive hemorrhage and thrombosis. In chronic hemorrhagic disease, pathologic lesions are quite variable but reflect sequela of vascular damage. These changes are most pronounced in the digestive tract but also may involve the coronary band and sensitive laminae of the hooves. Necrotic stomatitis, rumenitis, and omasitis often occur, and ulceration of the ruminal pillars may be pronounced. Laminitis may be sufficiently severe to cause sloughing of the hooves. Fawns infected while nursing frequently exhibit severe destruction of the ruminal lining. These animals may survive initial infection only to succumb after weaning when the diet shifts to roughage. Starvation or predation are the usual fate for these animals. In adult survivors, secondary infections of the digestive tract and feet are usual sequela. These lesions may be manifest as necrotic stomatitis, ulcerative rumenitis, or pyogenic infections of the feet. Involvement of the feet often is observed in hunter-killed deer. Infection of does during the early stages of gestation may result in therapeutic abortions (Thomas and Trainer 1970) and possibly stillbirths or malformed fetuses as observed with domestic livestock. The long term effect of BT on productivity of white-tailed or mule deer populations has yet to be explored.

Presumptive diagnosis of HD can be made on the basis of gross and microscopic lesions, however isolation and characterization constitute the only means for distinguishing the viruses of BT and EHD.

Malignant catarrhal fever (MCF) is a viral disease of domestic and wild ruminants characterized by catarrhal, mucopurulent, or necrotizing inflammation of the respiratory and digestive tracts and eyes. The disease is worldwide in distribution, and most outbreaks in North America are sporadic in occurrence. In the United States, MCF has been diagnosed in axis (Axis axis) and white-tailed deer in southern Texas (Clark et al. 1970; Clark and McConnell 1972), in white-tailed deer of New Jersey and Connecticut (Wyand et al. 1971), and in greater kudu (Tragelaphus strepsiceros) in Missouri (Bolver and Kurka 1974). Additionally, MCF has been recognized in American bison (Bison bison) and...
captive mule deer in Colorado (Pierson et al. 1974). One of the authors (CPH) has experimentally transmitted MCF to mule deer. Among cattle the disease appears sporadically in sheep-raising areas of the midwest and west.

The mode of transmission of MCF is unknown, however the recent discovery of MCF virus in nasal secretions of blue wildebeest (Connochaetes taurinus) led to the hypothesis that nasal shedding of MCF virus may be a mechanism for transmission of virus among wildebeest and from wildebeest to cattle (Rweyemamu et al. 1974). In Africa, wildebeest are natural reservoirs of MCF virus. Sheep are considered natural reservoirs elsewhere.

In cattle, peracute, alimentary, head and eye, and mild forms of MCF have been described. Among deer, the disease was best described for axis deer (Clark et al. 1970). Axis deer with MCF were lethargic, emaciated, weak, ataxic, and apparently blind. Neurologic disturbance was present in some animals. Gross lesions consisted of corneal opacity, areas of alopecia encrusted with thick, dried exudate, greatly enlarged lymph nodes and enlarged arteries. Gross lesions in white-tailed deer ranged from none to hemotherax to sudden death. Diagnosis of MCF is based on histologic lesions consisting of fibrinoid necrotizing vasculitis which are considered pathognomonic for this disease (Jubb and Kennedy 1970). Eye lesions and neurologic signs, when present, serve to distinguish MCF from BT and EHD.

Bacterial Diseases - Only one bacterial disease, necrobacillosis, is considered a serious threat to mule deer populations. The disease is caused by Fusobacterium (Spherophorus) necrophorum. Considerable controversy exists on whether F. necrophorum acts as a primary or secondary invader. Jubb and Kennedy (1970) state, "In no instance has it yet been credited with the role of primarily penetrating pathogen." Rosen (1970) considered F. necrophorum a probable opportunist awaiting an abrasion or injury to serve as an avenue for invasion.

Necrobacillosis has been diagnosed in a variety of wild cervidae, primarily in the western United States. It has been considered a major cause of mortality among mule deer in California (Rosen et al. 1951; Rosen 1970) and among wapiti (Cervus canadensis) of Wyoming (Murie et al. 1944 cited in Rosen 1951). Periodic outbreaks of necrobacillosis occur in many species of domestic livestock. Among sheep and cattle, frequently necrobacillosis is encountered when the environment is dark, dirty, damp, and overcrowded. Epizootics in California deer have occurred when animals have been overly concentrated near muddy water holes due to drought (Rosen et al. 1951). When the environment is sufficiently seeded with F. necrophorum organisms the disease apparently becomes contagious (Jubb and Kennedy 1970).

Lesions produced by F. necrophorum in mule deer are variable depending on the site of infection. Foot rot syndrome often is present, which is characterized by necrosis of the interdigital tissue, inflammation of the coronary band and sensitive laminae, and extension into the proximal joint above the hoof (Rosen et al. 1951). Necrotic stomatitis, ulcerative rumenitis and abscesses in other organs also may occur.

Diagnosis of necrobacillosis is based on isolation of F. necrophorum from affected tissues. It should be mentioned that the lesions of chronic hemorrhagic disease with secondary bacterial infection in white-tailed deer are markedly similar to those described for necrobacillosis in mule deer. The role of BT virus in producing a portal of entry for F. necrophorum should be a fruitful area for future study.

Parasitic Diseases - Two major groups of nematodes, trichostrongyles and lungworms, are of potential significance in portions of the mule deer's range.

The gastrointestinal trichostrongyles infecting mule deer comprise several genera (Walker and Becklund 1970), and in areas where climatic conditions are conducive to parasitism, Haemonchus contortus s.l.; the large stomach worm, is of particular importance. This helminth may cause considerable blood loss among fawns. The occurrence of H. contortus is worldwide, however it is locally more common in some areas than others in humid environments. In the southeastern United States, for example, H. contortus is more prevalent and has a higher intensity of infection in deer of the lower coastal plain than in more upland terrain (Prestwood et al. 1972). The host range for H. contortus includes cattle, sheep, goats, and numerous wild ruminants. Although species of Haemonchus infecting cattle and sheep have been separated, the genus appears to be in a state of evolutionary flux. Das and Whitlock (1960) considered H. contortus to be actively evolving and that the species contains a number of well defined demes, each adapted to a particular host-microclimate interaction.

The principal effects of Haemonchus are due to anemia, which in heavily infected animals is accompanied by edema, emaciation, and generalized digestive disturbances. Both 4th stage larvae and adult H. contortus cause blood loss. At necropsy submandibular edema ("bottle jaw"), enlarged lymph nodes, and thin, watery blood may be obvious. The lining of the stomach may be swollen, and have petechial hemorrhages with shallow ulcerations. Young deer usually are more heavily infected than the adults (Prestwood and Kellogg 1971).

Conditions that predispose animals to H. contortus also are conducive to heavy infections with other abomasal or intestinal trichostrongyles, and pure infections with a single species of nematode are uncommon. Similarly, nutritional status of the animal is of paramount importance since poor nutrition and parasitism often occur concomitantly. Although one helminth, e.g. H. contortus, may be the actual cause of death in deer, overcrowding, food shortage, and competition by other ruminants or swine are important contributing factors leading to death from gastrointestinal parasitism.

Two types of lungworms infect mule deer, viz. 1. those whose adult stage is found in the lung (Dictyocaulus viviparus) and 2. those whose adult stage occurs in sites remote from the lungs but whose eggs and larvae pass through the lungs to the external environment (Paraclostrhongylus spp.).

The large lungworm, Dictyocaulus viviparus, is located in the bronchi and bronchioles of mule deer.
where it produces a parasitic bronchitis. Dictyocaulus is worldwide in distribution, with increased prevalence in cool, moist areas. Like other trichostrongyles, D. viviparus has a direct life cycle. Cattle and various wild ruminants serve as definitive hosts for large lungworms, however, recent work has shown that Dictyocaulus of wild ruminant origin has low infectivity in cattle suggesting that there may be host specific strains of this helminth (Presidente et al. 1972, 1973; Gupta and Gibbs 1971).

In the southeastern United States, about 30 percent of white-tailed deer harbor D. viviparus. Infection is most prevalent and intense in young animals, particularly buck fawns. Dictyocauliasis is seasonal in occurrence; the prevalence and intensity of infection are higher in late summer and early fall. The parasite is least common during the winter (Prestwood et al. 1971). Outbreaks of D. viviparus pneumonia have been recorded in captive black-tailed deer in Oregon (Presidente et al. 1973).

At necropsy, lesions caused by D. viviparus may be mild to severe. Extensive pneumonia may be present, and there may be numerous lungworms and exudate in the bronchi. Pleuritis and interlobular thickening may be obvious. Occasionally edema and enlarged lymph nodes and lymph vessels are detected. Lung damage may be particularly severe when first-stage larvae of Parelaphostrongylus sp. also are present (Prestwood et al. 1971).

Mule deer may be infected by at least two species of Parelaphostrongylus, viz. P. odocoilei and P. tenuis. Parelaphostrongylus odocoilei is located adjacent to or within small vessels in the musculature of the hindbody of black-tailed and mule deer. It also has been found within small vessels in the lungs. This helminth has been reported only from deer of California (Hohmaier and Hohmaier 1954; Brunetti 1969). Recently, however, similar protostrongylid larvae were found in the feces of mule deer from Western Canada (Samuel and Holmes 1974). One of the authors (CPH) has seen protostrongylid larvae in feces of southwestern mule deer.

The life cycle of P. odocoilei is indirect. Eggs are deposited in the circulation and arrive as emboli in the lungs where hatching occurs. First-stage larvae ascend the bronchial passageways and trachea, are swallowed and eliminated with the feces. Various terrestrial snails and slugs (Helix aspersa, Agriolimax agrestis, A. compestris, Planorbis sp.) serve as intermediate hosts. Deer become infected after ingesting snails containing infective P. odocoilei larvae. The pre-patent period is approximately 2-1/2 months.

Adult P. odocoilei produce small hemorrhages in the musculature. Extensive tissue damage caused by eggs and larvae of P. odocoilei has been seen histologically in the lungs and lymph nodes and has been considered a cause of death among California deer (Brunetti 1969).

Mule deer have been experimentally infected with the meningeal worm, Parelaphostrongylus tenuis (Anderson et al. 1966). This helminth is widely distributed throughout the range of its usual host, the white-tailed deer, and has been recorded as far west as Oklahoma and Minnesota. Adult helminths are located in the subdural space and venous sinuses of the cranial meninges of white-tailed deer. The life cycle is indirect, and similar to that of P. odocoilei. Detailed studies by Anderson (1963; 1965) and Anderson and Strelive (1967) have shown that after ingestion, infective larvae penetrate the abomasal wall and travel to the spinal cord of white-tailed deer. Development occurs in the dorsal horns of gray matter for 30-40 days, after which worms move to the subdural space. First-stage larvae appear in the feces 90 days or more post-infection.

Neurologic signs are rare in white-tailed deer, however, neurologic disturbances leading to paralysis and death have been observed in unusual hosts, e.g. domestic sheep, moose (Alces alces), wapiti, caribou (Rangifer tarandus terranovae) and reindeer (R. t. tarandus) (Anderson 1970). A mule deer fawn was experimentally infected with P. tenuis, and a fatal paralysis ensued (Anderson et al. 1966). Black-tailed deer translocated into Tennessee from Oregon also were afflicted with fatal neurologic disease caused by P. tenuis, which apparently has been a primary limiting factor for establishing black-tailed deer in Tennessee. Experimental infection of a hybrid deer (O. h. columbianus x O. virginianus) resulted in fatal paralysis 52 days post-infection (SCWDS, unpublished).

It appears that the encroachment of white-tailed deer onto the range of mule deer is a definite threat because of the likelihood of exposing mule deer populations to P. tenuis.

Although each of the aforementioned diseases has been considered a specific potential mortality factor for mule deer populations, we should emphasize that seldom does one entity alone cause significant mortality of a wild population. Rather, multiple factors usually are involved, and the specific disease is a product of complex interactions between the animal and its environment. Judging from the apparent lack of information on diseases of mule deer, we respectfully suggest that concerned game and fish agencies place more emphasis on investigating diseases of mule deer. These investigations should include specific information on causes of "die-offs" and probably more importantly, should consider what potential mortality factors are present within apparently healthy populations. Only until we have a basic understanding of the interactions between the disease agent, the animal, and the environment can the causes of the mule deer decline in the west be ascertained.

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LITERATURE CITED


