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AN OUTBREAK OF A HEMORRHAGIC DISEASE IN WHITE-TAILED DEER IN KENTUCKY[1]

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Abstract: In 1971, an outbreak of a hemorrhagic disease occurred in captive and free-ranging white-tailed deer (*Odocoileus virginianus*) in Mammoth Cave National Park, Kentucky. Clinical signs and gross pathological lesions were consistent with those of epizootic hemorrhagic disease and bluetongue, as were serological and histopathological findings for samples sent to other laboratories. The infection rate among the 104 captive deer was 88-92%, and that among the free-ranging Park deer appeared to be similar. Mortality was negligible in the Park deer, but 65 (62%) of the captive deer died. The deaths were bimodally distributed over a 36-day period, and the mortality rate decreased from 97-100% for deer clinically ill during the first 17 days of the outbreak to 58% for deer first exhibiting clinical signs on day 16 or later. Mortality was equal in males and females, but less in yearlings than among fawns or adults. Winter mortality among survivors of the initial outbreak was associated with low ambient temperatures and sometimes fungal and bacterial abscesses, possibly sequelae or complications of the hemorrhagic disease. The pregnancy and birth rates among surviving does appeared to be normal.

INTRODUCTION

In mid-September 1971, an outbreak of a hemorrhagic disease occurred in captive and free-ranging white-tailed deer in Mammoth Cave National Park, Kentucky, coinciding with similar outbreaks in several other southeastern states. The clinical signs and gross lesions were consistent with those of epizootic hemorrhagic disease (EHD),[8,19] but because of similarities in the pathology of EHD and bluetongue (BT),[7,13,18,20] the controversy over whether these diseases are caused by the same or different viruses,[4,14] and the fact that both viruses were active in the southeastern states during the outbreak,[16] the disease is referred to here simply as a hemorrhagic disease (HD).

The 1971 outbreaks were the first recognized occurrence of a widespread epizootic of HD in deer in the eastern half of the continent since the 1955 outbreaks of EHD in New Jersey and Michigan.[3,10,11] The Kentucky outbreak was the first confirmed occurrence of HD in deer in that state. Since it was also the first epizootic involving EHD in captive deer, it afforded a unique opportunity for study of the disease. This paper reports observations made during and subsequent to the outbreak in Mammoth Cave National Park.

DEER HERD HISTORY

Native deer were virtually exterminated in Kentucky by 1914, but 30 white-tailed deer were imported from an unrecorded source and released on the newly created Kentucky Wodlands Game Refuge about 1922 (P. W. Sturm, personal communication). Progeny of these deer were used for restocking other suitable areas in Kentucky, including Mam-

[1] A contribution of a cooperative project between the National Park Service and the U.S. Fish and Wildlife Service, USDI.

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moth Cave National Park. Park and refuge records indicate that the Park herd was established by transplanting about 50 of these deer between 1942 and 1949.

In the fall of 1949 an epizootic of undetermined etiology decimated the refuge herd[1] and other deer populations in eastern Kentucky.[9] This was the only major deer die-off recorded in Kentucky before 1971, and there is no record of it having reached the then sparse deer population in the Park.

PRE-OUTBREAK SITUATION

Mammoth Cave National Park encompasses 210 km^2 of wooded, karst hill country in southcentral Kentucky and is surrounded by agricultural land on which crops and livestock (cattle, horses, and swine) are raised. The Park is roughly bissected by the Green River, the major surface drainage system. Because of the karst geography, there are only a few year-round surface streams and shallow ponds in the Park. There are also few roads and, because the Park's primary attraction is Mammoth Cave, visitors seldom penetrate the roadless areas.

Park Deer Herd

No reliable census data exist for the Park deer herd, but our field observations and productivity studies suggest that the pre-outbreak density of the free-ranging herd in 1971 was approximately 10 deer/km^2 and increasing after a 1969-1970 populaiton low. Necropsies of 35 free-ranging Park deer collected during the summer of 1971 disclosed no evidence that HD was present.

Experimental Deer Colony

At the time of the outbreak, an experimental colony of 104 deer was being maintained in a 2 ha pen complex at the Mammoth Cave Wildlife Research Station.[3] The colony consisted of 26 fawns, 18 yearlings, and 15 adults of both sexes that had been bottle-reared at the station, 32 adult does and 5 adult bucks that had been captured from the free-ranging herd in the Park but had been in captivity more than a year in most cases, and 8 adult does that had been captured between 2 months and 2 weeks before the outbreak began within the colony. All deer were fed *ad libitum* with a proven deer ration.[21] The last fawn had been weaned from evaporated milk to this diet 4 weeks before the outbreak. The bottle-reared deer were kept in open stalls or paddocks and could be readily observed. The wild captives were in 2,000-m^2 or larger paddocks and could not be approached without causing them to run about the enclosures and into the fence.

THE OUTBREAK AND PROCEDURES

The first colony deer, a yearling buck, died on 14 September 1971; the second, a fawn, died on 19 September. No further deaths occurred until 23 September, when the deer began dying at the rate of two to five per day. By 28 September, 20 had died. Upon contacting the Southeastern Cooperative Wildlife Disease Study (SCWDS), University of Georgia, Athens, Georgia, I learned that deer in five other southeastern states were dying with gross lesions similar to those observed in our colony. The Kentucky Division of Fish and Wildlife Resources was advised of the die-off and requested assistance from the SCWDS. I also requested diagnostic aid from the Animal Health Division, U.S. Department of Agriculture (USDA). Three fawn carcasses were sent to the Kentucky Department of Agriculture Diagnostic Laboratory in Hopkinsville, and tissues from other deer were sent to the USDA Animal Disease Research Laboratory;[4] to the Canada Department of Agriculture (CDA), Animal Diseases Research Institute, Hull, Quebec; and to the Department of Bacteriology, Utah State University, Logan, Utah.

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[3] A field station of the Denver Wildlife Research Center, USDI, U.S. Fish and Wildlife Service, Building 16, Federal Center, Denver, Colorado 80225.

[4] USDA, Agricultural Research Service, Building 45-B, Federal Center, Denver, Colorado 80225.

In an effort to monitor clinical signs of disease, our staff observed all captive deer as closely as possible using binoculars and flashlights as needed) at 4 h intervals around the clock for 9 days during the peak of the die-off, and systematically recorded the presence or absence of 40 clinical signs. This frequency of observations was not always maintained because of the need to necropsy animals that died. Moribund animals were kept under close surveillance and were generally removed from the pens soon after death, often within minutes. Necropsies were conducted in the order in which the deer died but were sometimes delayed several hours if a backlog of carcasses developed. Anatomical, morphological, and physiological data were recorded for each carcass according to a routine procedure at this station. Blood samples were collected when possible. One sample was mixed 50:50 with OPG[5] and refrigerated; another was centrifuged and the serum fraction was frozen. Tissues collected for histopathological examination were preserved in 10% buffered formalin, and those for virus isolation were snap frozen in either acetone and dry ice or liquid nitrogen.

To determine the infection rate in the free-ranging herd, blood and serum samples were also obtained from 161 of 170 deer (including 16 "recaptures") live-trapped in the Park by the Kentucky Division of Fish and Wildlife Resources between 21 October and 23 December 1971. Of these deer, 39 were retained for further observation and as replacements for captive deer lost to the disease, 23 were killed and necropsied, and 108 were tagged and released. All but 7 of the 108 released deer were examined for clinical signs of disease, and blood samples were taken from 103 of them. Fifty-eight free-ranging deer that our staff killed for other purposes within 6 months after the outbreak were also bled and examined.

Five serologically negative white-tailed deer fawns from an epizootic-free area were contributed by the SCWDS and introduced into our colony on 19 October to serve as sentinels. These were confined in locations adjacent to paddocks containing infected deer, and held for 6 months, during which they were observed for signs of illness and bled periodically.

RESULTS AND DISCUSSION

Chronology and Impact of the Outbreak Among Captive Deer

The die-off lasted 36 days (14 September through 19 October) and resulted in the deaths of 65 deer (62% of the colony). The distribution of deaths within this 36-day period was examined in two ways: the percent of the total number of deaths by day (Fig. 1), and the percent of the remaining population that died each day. Both analyses suggested a bimodal distribution of mortality with peaks on days 15-16 and 27-28.

Of the 39 deer surviving this 36-day period, 27 were clinically ill. During 14-28 September, when the first 20 deer died, few necropsies or records of clinical signs were made. However, interviews with personnel present during this period and my observations thereafter indicated that the duration of clinical signs before death averaged 24 hr or less during the first 15 days of the outbreak, that the mortality rate among deer clinically ill during the first 17 days of the outbreak was 97-100%, and that the onset of clinical signs peaked on day 17 (Fig. 2). The 27 deer that recovered showed clinical signs no earlier than day 16, and the mortality rate from that time on was 58%. In addition, the duration of clinical signs before death during this second half of the outbreak averaged 5 days (range, 1-13 days), and deer that recovered showed signs for an average of 10½ days (range, 6-18 days). Thus, there appeared to be an inverse relationship in time between the onset and duration of clinical signs and the fatality of the infection.

Chi-square analysis (using Yates' correction for continuity) indicated no sig-

[5] OPG = 5 g potassium oxalate, 5 g phenol, 500 ml glycerol, and 500 ml distilled water.

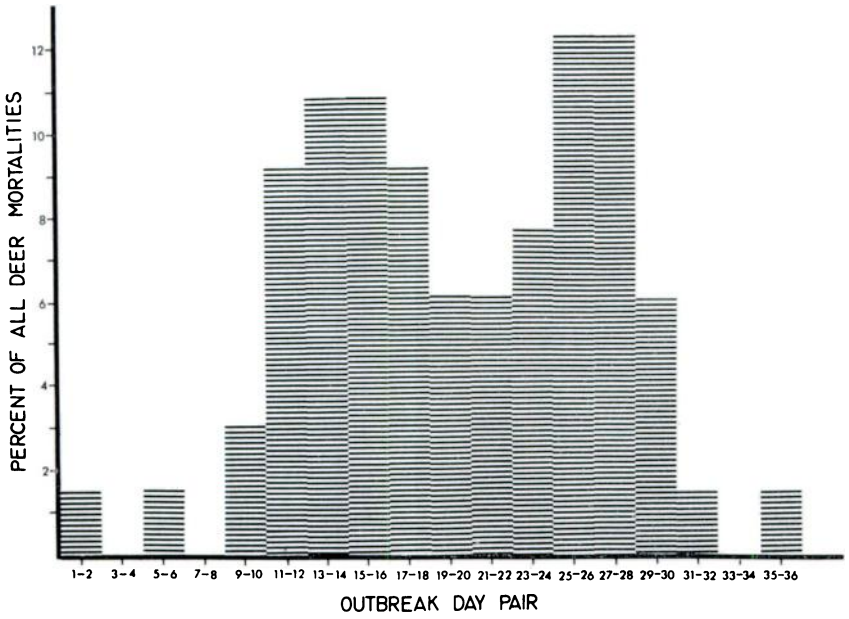

FIGURE 1. Distribution of mortality in captive deer by outbreak days. Two-day units are used to facilitate interpretation.

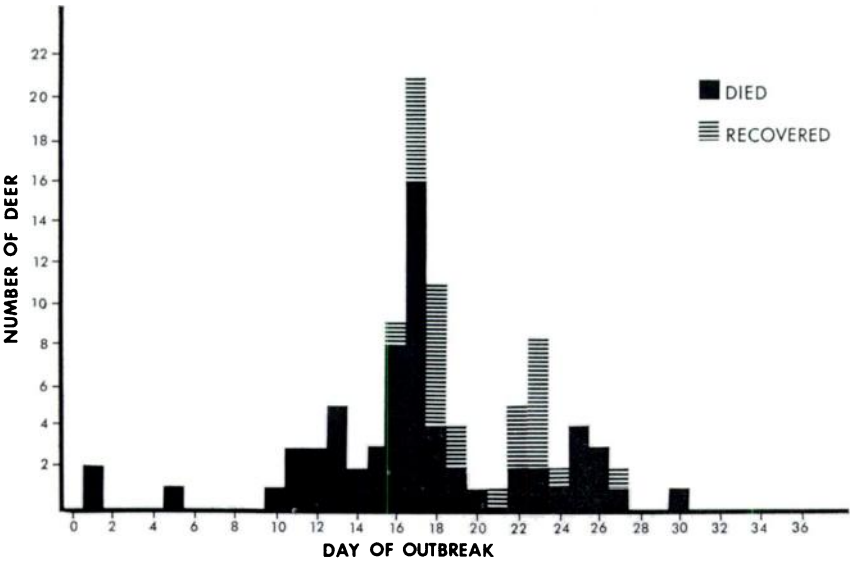

FIGURE 2. First appearance, by outbreak day, of definite clinical signs of HD in captive deer.

nificant difference in infection rates between sexes (P = 0.75-0.90), among age classes (P = 0.50-0.75), or between captured and pen-reared deer (P = 0.25-0.50). Similarly, there was no significant difference in mortality rates between sexes (P = 0.75-0.90), wild and tame deer (P $\cong$ 0.90), or fawns and adults (P = 0.75-0.90), but the 39% mortality rate for yearlings was significantly lower than the 73% for fawns (P $\cong$ 0.05) or the 65% for adults (P = 0.05-0.10). The times of death were equally distributed between day and night hours (0600-1800 and 1800-0600). Mortality differed greatly (40-83%) among the 8 paddocks and 11 stalls, but there were no obvious patterns that could be related to locations of vector breeding sites or an initial locus of infection within the facility.

Clinical Signs

Clinical signs observed, in order of decreasing frequency, included lethargy (98%), stiffness or lameness (92%), drooping ears (83%), swollen eyelids (81%), swollen cheeks (80%), hyperemia of the skin and of mucus membranes at body openings (79%), unwariness in wild captives (76%), swollen jaws (72%), excessive — sometimes blood-tinged—saliva or froth from the nostrils (64%), and dyspnea (50%). Audible "grinding" of molar teeth, rough coats, trembling, protruding eyes, lateral tilting of the head, and beads of blood on the muzzle epidermis were observed occasionally. Bloody diarrhea or blood-flecked fecal pellets were seldom seen, but fecal coatings of stringy, yellow mucus were common. Our few observations of pulse rate and body temperature before death revealed tachycardia and normal to depressed temperatures. However, 62% of the deer examined at death or shortly after were febrile, with rectal temperatures 40-44 C, and, in one case, 48 C.

There was no consistent sequence of clinical signs, but the most common pattern began with swelling of the eyelids, or of the cheeks, jaw, or muzzle, and was followed by stiffness or lameness and sluggishness. Anorexia also probably occurred early but was not readily recognized among group-fed deer. Except for obviously terminal signs such as dyspnea and coma, the sequence and variety of clinical signs in deer that died were not conspicuously different from those in deer that recovered.

Direct observation or disturbed surface soil or litter around carcasses revealed that prolonged thrashing usually preceded death, and the carcasses were almost always laterally prostrate, usually with the limbs extended and often with the neck arched caudally. Fluid or froth, clear or blood-tinged, flowed from the mouth or nostrils at death in 60% of the cases, but terminal bloody flows from the penis, vulva, or anus were rarely observed. The conspicuous swelling of the head commonly seen during the clinical course of the disease had usually subsided prior to death, but slight to severe hyperemia of the tongue, ears, mammary region, perianal zone, coronary band, eyelids, lips, or nostrils was usual, and the lips and tongue were occasionally cyanotic. Conjunctivitis and scleritis were also very common, but nasal crusts and sloughing of the muzzle epidermis were rare.

Gross Pathology

Gross internal lesions observed during necropsy of 45 captive deer were suggestive of EHD or BT.[3,4,8,10,17,19] Petechial to frank hemorrhages occurred throughout the body, as did edema (Table 1). Amounts of edematous fluid present varied from approximately 1 ml around the joints of some long bones to 1,800 ml in the pleural cavity of one deer. The distribution and frequency of other gross lesions are summarized in Table 2.

Overall, the gastrointestinal tract was the system most consistently affected. Gross hemorrhagic lesions and epithelial sloughing, although varying in degree and location along the tract, were seen in all necropsies of diseased deer during the epizootic. Since these were the most frequent and sometimes the only hemorrhagic lesions seen in necropsies of free-ranging deer killed during the winter following the outbreak, they may also be the last gross lesions to disappear in convalescing deer.

TABLE 1. Frequency of gross hemorrhages, hemorrhagic congestion, and edema in 45 captive deer that died during days 16-36 of the HD outbreak.

Tissue or area	Frequency (%)
HEMORRHAGES OR CONGESTION:	
Heart (epicardial):	
Left ventricle	80
Right ventricle	69
Left atrium	87
Right atrium	90
Heart (endocardial):	
Left ventricle	85
Right ventricle	74
Left atrium	47
Right atrium	71
Gastrointestinal tract:	
Rumen	91
Reticulum	90
Omasum	76
Abomasum	84
Intestine	58
Caecum	87
Colon and rectum	87
Omenta and mesenteries	26
Urogenital tract:	
Kidneys	33
Urinary bladder mucosa	<30
Ovaries	42
Vagina and uterus	<20
Testes	75
Epididymides and Spermatic cord	<20
Other tissues:	
Adrenals	62
Costal pleura	83
Diaphragm	82
Liver	79
Lungs	83
Lymph nodes	88
Pancreas	58
Pituitary	32
Salivary glands	29
Spleen	86
Thymus	56
Thyroid	30

TABLE 1 — Continued

Tissue or area	Frequency (%)
EDEMA:	
Peritoneal cavity	83
Pleural cavities	62
Pericardial sac	73
Cranium	52
Carpometacarpal joint	64
Metacarpophalangeal joint	64
Subcutaneous, head and neck	69
Omenta and mesenteries	41

TABLE 2. Frequency of miscellaneous gross lesions and other conditions in 45 captive deer that died during days 16-36 of the HD outbreak.

Tissue or characteristic	Condition and frequency
Tongue, base	Swollen and hard (41%)
Tongue, dorsal surface	Whitish, cheesy, or crusty lesions (23%)
Tongue, epithelium	Epithelial sloughs (13%)
Dental pad	Epithelial sloughs (13%)
Lips	Epithelial sloughs (5%)
Oral papillae	Hyperemic (46%)
Pharynx	Swollen and hyperemic (20%)
Larynx	Swollen and hyperemic (20%)
Trachea and bronchi	Severely and extensively hyperemic (71%)
Trachea and bronchi	Lined with serous or clear froth, or free blood (68%)
Lungs	Edematous, congested with blood, or both (79%)
Rumen contents	Scant amount (58%)
Rumen contents, pH	Mean 6.4, range 5.5 - 8.0
Rumen contents, color	Normal (51%), dark gray-brown to chocolate brown (47%), maroon (2%)
Rumen contents, consistency[1]	Normal (11%), liquefied (67%), dehydrated (22%)
Rumen mucosa	Sloughs readily (61%)
Reticulum and omasum mucosae	Gross lesions and sloughing (about 50%)
Abomasum mucosa, pyloric	Darkened and hemorrhagic (94%), sloughs readily (about 50%)
Small intestine contents	Bloody (65%)
Feces, form	Pelleted (88%), diarrhetic (12%)
Feces, coating	Normal (9%), bloody mucus (20%), yellowish stringy mucus (71%)

[1] Dry ingesta was invariably dark brown, felt fibrous or gritty, and was usually (90%) associated with a darkened rumen mucosa and a febrile condition shortly before death; liquefied ingesta was also associated (88%) with a darkened, sometimes black, rumen mucosa.

Histopathology

Tissues from 14 captive and 12 free-ranging deer that died or were killed during the epizootic were found to have microscopic lesions consistent with either EHD or BT.[4,7,8]

Serology

Forty-one sera from 12 captive deer and 168 sera from 155 free-ranging deer were tested for bluetongue virus (BTV) antibody by the USDA Animal Disease Research Laboratory using an agar gel precipitin (AGP) test with undiluted serum. Only six of these samples, all from captive deer, were considered positive. One was from a dead adult necropsied during the middle of the outbreak, one was from a convalescent yearling buck 65 days after the onset of clinical signs that persisted for 2 weeks, two were from two of the sentinel fawns 63 days after the onset of clinical signs that persisted for about 2 months, and two were from the same fawns 165 days after the onset of signs. Sera from these two fawns were obtained weekly for 10-11 weeks beginning 2 days before the onset of clinical signs, but all of these other samples were negative for BTV antibodies.

The CDA Animal Diseases Research Institute performed additional serological tests on 26 of the samples, 12 from free-ranging deer and 14 from clinically ill captive deer. None of the 12 serum samples from free-ranging deer yielded positive results, but seven samples from captive deer were positive and an eighth was questionable for EHDV antibodies.[10] Of these eight, five were from the five sentinel fawns: two positive samples were obtained at death 9 and 10 days after initial exposure and within 24 h of the onset of clinical signs; one positive was obtained 2 days before death, 17 days after initial exposure and 8 days after the onset of clinical signs; and one positive and the one questionable were obtained 73 days after initial exposure and 63 days after the onset of clinical signs that persisted for about 2 months. Of the three remaining positive sera, two were drawn from adult captives at death and one was drawn from a yearling buck 65 days after the onset of clinical signs that persisted 10 days. On 14 serum samples from captive deer, plaque neutralization tests for BTV antibodies indicated one positive and five questionables.[16]

Virus Isolation Efforts

The USDA Animal Disease Research Laboratory, using intravascular inoculation of 8-day embryonating chicken eggs, was unable to isolate BTV from either OPG-preserved blood samples or fresh and frozen tissues (spleen, kidney, liver, bone marrow, lymph nodes) taken from HD-infected deer (J. G. Bowne, personal communication). Among the blood samples tested were 23 from acutely ill, captive deer. All assays of blood samples from free-ranging deer were also negative.

Impact on the Free-Ranging Herd

Six two-man crews searched several Green River tributaries and hollows on 30 September 1971 but found no dead or sick deer. Ground searches on 4 October also proved futile, but periodic boat patrols along the Green River revealed several deer carcasses in or near the water. Five of these were examined and had gross lesions similar to those in dead colony deer. Several other reports of sick or dead deer in the Park were received during the fall and winter, but we were unable to recover further carcasses. Reports of sick or dying deer elsewhere in the state arrived sporadically during late summer and fall but were not investigated.

Because of our preoccupation with the outbreak in the experimental colony, we were unable to survey conditions in the free-ranging herd until mid-October. At this time the outbreak had essentially ended in the colony and apparently also in the free-ranging herd.

POST-OUTBREAK SITUATION

Winter Mortality

During the winter and spring following the outbreak in the experimental colony, 5 of the 39 survivors died, usually after a brief clinical illness, and showed hemorrhagic lesions at necropsy. Three of these also had multiple abscesses that involved the lungs and other organs, as did six free-ranging deer killed during the winter and spring. Only one deer that died during the outbreak—after a lingering illness—had such suppurative lesions. Histopathological observations associated with these late deaths included the following: hyperkeratosis of the rumen and reticulum; intestinal ulcers and peritonitis; intestinal pseudomembranes of mainly fibrin and blood; hemosiderosis of the spleen and liver; thickening of the liver capsule with organizing fibrinous exudate; fibrous adhesions on the spleen capsule; suppurative meningitis; hyaline thrombi in capillaries and venules in the subepithelial connective tissues of the tongue; massive disseminated fibrin and hyaline thrombosis of lymph nodes; abscesses of skeletal muscles; thickened pleurae; lung foci of chronic suppuration and adenomatous reaction; and chronic, severe, interlobular edema of the lungs. The fungal and bacterial abscesses were regarded as possible late sequelae or complicaitons of the HD.

Including the five "relapse" cases and the deaths of three other replacement deer of unknown case history, there were eight winter or spring deaths related to the HD or possible complications. All eight deaths were associated with marked decreases in ambient temperature during an otherwise mild winter. The last death occurred during a cold snap on April 8 after the weather began warming. The deer that died, a 10-month-old male, had survived clinical illness during the outbreak but 6 months later showed lesions typical of acute HD as though recently infected. All eight dead deer and most of the survivors that could be observed showed evidence of hoof sloughing.

Effect on Productivity

Although the well-documented, late-summer timing of most HD outbreaks probably precludes any significant effect on the pregnancy rate in species, such as deer, that normally breed in November or December, it has been shown that BT causes early absorption or uncomplicated abortion in white-tailed deer,[15,20] and congenital infection and fetal abnormalities in sheep and cattle.[5,6,12] Experimental EHD may affect the deer fetus *in utero*[4] as well as the viability of the fawn.[2] However, 11 fertile captive does that survived the outbreak and had an opportunity to breed became pregnant; two of these died during the winter and nine gave birth to a normal number of fawns. None of these does appeared clinically ill during the breeding season, but nine (including the two that died) had shown frank signs during the outbreak. The two that died during pregnancy had typical lesions of HD and/or HD sequelae at necropsy. In addition, of 21 replacement does that were captured after the outbreak and had an opportunity to breed either in captivity or in the wild, 19 became pregnant; two died during the first trimester of apparently normal pregnancies, and 17 fawned in 1972. At least 11 of these, examined between capture and release into the pens, had clinical signs suggestive of exposure to the HD. The high rate of successful pregnancies among suspected survivors of HD indicates that ovulation and conception can occur either during the course of sublethal infection or soon after and that such infection does not result in conspicuous fetal mortality.

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