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PARASITES, DISEASES, AND HEALTH STATUS OF SYMPATRIC POPULATIONS OF FALLOW DEER AND WHITE-TAILED DEER IN KENTUCKY[1]

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ABSTRACT: In August 1983, a study on parasites, diseases, and health status was conducted on sympatric populations of fallow deer (*Dama dama*) and white-tailed deer (*Odocoileus virginianus*) from Land Between The Lakes, Lyon and Trigg counties, Kentucky. Five adult deer of each species were studied. White-tailed deer had antibodies to epizootic hemorrhagic disease (EHD) virus and *Leptospira interogans* serovariety *icterohemorrhagiae*, and fallow deer had antibodies to bluetongue and EHD viruses. Serologic tests for bovine virus diarrhea virus, infectious bovine rhinotracheitis virus, parainfluenza$_3$ virus, and *Brucella* spp. were negative. One white-tailed deer had an infectious cutaneous fibroma, and one fallow deer had pulmonary mucormycosis. White-tailed deer harbored 16 species of parasites, all of which are considered typical of the parasite fauna of this host in the southeastern United States. Fallow deer harbored nine species of parasites, including eight species known to occur in white-tailed deer on the area and one species (*Spiculopteragia assymmetrica*) that is not. All fallow deer had inflammatory lesions in the spinal cord and/or brain that were attributed to prior infection with meningeal worm (*Parelaphostrongylus tenuis*), indicating that *P. tenuis* infections are not always fatal for this species. The apparent high rate of exposure of Land Between The Lakes fallow deer to *P. tenuis* without a resultant high rate of clinical cerebrospinal parelaphostrongylosis is hypothesized to be due to (1) a low prevalence and intensity of *P. tenuis*, (2) partial innate resistance of fallow deer, and (3) acquired immunity.

INTRODUCTION

White-tailed deer (*Odocoileus virginianus*) are the most abundant and widely distributed wild cervids in North America (Halls, 1978). Because white-tailed deer coexist with either domestic livestock, other native cervids or introduced exotic ungulates throughout most their range, numerous studies have been made on the potential for cross-transmission of infectious agents between white-tailed deer and these hosts (see Davidson et al., 1981).

A population of free-ranging fallow deer (*Dama dama*) on Land Between The Lakes (LBL), Lyon and Trigg counties, Kentucky, has coexisted with white-tailed deer since the 1920's; however, the fallow deer population has exhibited slow growth and range expansion during this time. One factor considered to be a significant cause of mortality in fallow deer on LBL is cerebrospinal parelaphostrongylosis due to the meningeal worm (*Parelaphostrongylus tenuis*) of white-tailed deer (Nettles et al., 1977). Fallow deer on the area also are known to harbor at least five other species of parasites that occur in white-tailed deer (Phillips et al., 1974; Nettles et al., 1977);

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however, a detailed comparison has not been made of the parasite fauna and diseases of the two species of deer. This report presents an evaluation of the parasites, diseases, and health status of fallow and white-tailed deer on LBL.

MATERIALS AND METHODS

This study was conducted in August 1983, on the Tennessee Valley Authority's LBL, a 68,800-ha strip of land that separates two large reservoirs (Kentucky Lake and Lake Barkley). The area is managed as a national demonstration area for outdoor recreation, environmental education, and natural resource management. Descriptions of the wildlife habitat, forest resources, and hydrologic condition of the areas are available elsewhere (Burbank and Smith, 1966). The present study was conducted within a 2,000-ha area known as the Environmental Education Area (EEA) which straddles the border between Lyon and Trigg counties, Kentucky. Within the EEA were an estimated 250 white-tailed deer and 200 fallow deer. Other ungulates were not present on the study area, although four cattle and two horses had been kept in a small EEA pasture the preceding winter. Surrounding portions of LBL also are inhabited by white-tailed and fallow deer, and a confined herd of bison (*Bison bison*) was kept several miles from the study area.

Five adult animals (≥1 yr of age) of each species were collected by shooting in the cervical spine. Blood samples (with and without anticoagulant) were obtained by cardiac puncture immediately after death. The carcasses were tightly sealed in plastic and refrigerated prior to necropsy within 18 hr. Ages of white-tailed and fallow deer were determined by the methods of Severinghaus (1949) and Chapman and Chapman (1975), respectively. Physical condition was assessed by the method of Stockle et al. (1978). Detailed necropsies (Nettles, 1981) were performed on each animal including the following procedures: (1) Baermann examinations of macerated lung tissue and feces, (2) abomasal parasite counts (APC), and (3) $NaNO_3$ flotations of feces for coccidian oocysts. The brain and spinal cord of each fallow deer were removed intact, examined visually while fresh, preserved in 10% neutral buffered formalin, and later reexamined grossly and histologically for lesions and nematodes. Sections of all major organs and any lesions were examined using standard histologic procedures including special staining techniques where appropriate. Serum samples were tested for antibodies to the etiologic agents of the following infectious diseases: bovine virus diarrhea, infectious bovine rhinotracheitis, and parainfluenza group 3 by serum neutralization; bluetongue (BT) and epizootic hemorrhagic disease (EHD) by agar gel immunodiffusion; brucellosis by the card test; and leptospirosis by microscopic agglutination. Serologic tests for the etiologic agents of leptospirosis included the *pomona, hardjo, grippotyphosa, icterohemorrhagiae,* and *canicola* serovarieties.

RESULTS AND DISCUSSION

The overall physical condition of the fallow deer (two males, three females) was generally higher than that of the white-tailed deer (one male, four females). Fallow deer condition ratings were good (three animals) and excellent (two animals), whereas white-tailed deer were rated fair (two animals) and good (three animals). Previous studies (Harmel, 1979; Armstrong and Harmel, 1981) have demonstrated that under identical nutritional conditions axis deer (*Axis axis*) and sika deer (*Cervus nippon*) are able to maintain physical condition better than white-tailed deer. Studies on food habits have revealed close similarities among axis, sika, and fallow deer (Armstrong and Harmel, 1981), and these authors have implied that fallow deer would have a similar competitive advantage compared to white-tailed deer.

Serologic tests disclosed antibodies to EHD virus in two fallow deer and one white-tailed deer, antibodies to BT virus in one fallow deer, and antibodies (1:200) to *Leptospira interogans* serovariety *icterohemorrhagiae* in one white-tailed deer. Previous serologic testing of white-tailed deer in southeastern U.S.A. for antibodies to EHD and BT viruses (Couvillion et al., 1981) and to *L. interogans* (Shotts, 1981) have produced similar results. The remaining serologic tests were negative.

Two infectious diseases were found at necropsy. A white-tailed deer had a 1-cm infectious cutaneous fibroma on a front

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TABLE 1. Parasites recovered from fallow and white-tailed deer from Land Between The Lakes, Lyon and Trigg counties, Kentucky.[a]

Parasite	Fallow deer	White-tailed deer
Sarcocystis sp. (78139)[b]	—	80, M, M
Theileria cervi (78140; 78141)	60, L, L[c]	40, L, L
Trypanosoma cervi (78142)	—	20, L, L
Parelaphostrongylus tenuis (78143)	—[d]	60, 2, 5
Protostrongylid larvae (78144)	—	100, M, M
Setaria yehi (78145)	—	60, 2, 3
Gongylonema pulchrum (78146; 78147)	20, 4, 4	100, 43, 200
Apteragia odocoilei (78148; 78149)	20, 52, 52	100, 556, 1,014
Apteragia pursglovei (78150)	20, 16, 16	—
Ostertagia dikmansi (78151)	—	60, 68, 102
Ostertagia mossi (78152)	—	100, 244, 450
Spiculopteragia assymetrica (78153)	100, 684, 1,648	—
Trichostrongylus askivali (78154)	—	20, 38, 38
Trichostrongylus axei (78155)	—	40, 1, 1
Capillaria bovis (78156; 78157)	40, 22, 25	40, 7, 12
Nematodirus odocoilei (78158; 78159)	20, 1, 1	20, 1, 1
Oesophagostomum venulosum (78160; 78161)	80, 3, 6	80, 12, 24
Amblyomma americanum (1984.141)	100, M, M	100, M, M
Tricholiperus parallelus (1984.141)	—	20, L, L

[a] Figures in columns are prevalence, mean number per infected deer, and maximum intensity of infection.
[b] Numbers in parentheses are U.S. National Parasite Collection (Beltsville, Maryland) accession numbers (protozoans and nematodes) and Bishop Museum (Honolulu, Hawaii) collection numbers (arthropods).
[c] Actual counts not made; intensities estimated as low (L = ≤25), moderate (M = 25 to 100), or high (H = ≥100).
[d] Lesions compatible with *P. tenuis* infection were found in the brain and/or spinal cord of all fallow deer.

leg. A fallow deer had a 5-cm firm, gray mass that was surrounded by a 2–4-cm zone of inflammation in the left lung. The center of the mass appeared necrotic. Histologically the lesion was a granulomatous nodule with multiple areas of necrosis. Surrounding lung tissue was atelectatic, congested, and contained infiltrates of eosinophils, neutrophils, mononuclear cells, and macrophages. Within the necrotic foci were branching, non-septate, PAS-positive fungal hyphae that were morphologically compatible with the genus *Mucor*. The lesion was diagnosed as focal pulmonary mucormycosis.

Parasitologic studies disclosed a total of 18 species of parasites including three protozoans, 13 nematodes, and two arthropods (Table 1). No coccidial oocysts were found in feces. White-tailed deer harbored 16 species and fallow deer harbored nine species of parasites. The species found in both white-tailed and fallow deer were typical of the parasite fauna of white-tailed deer in southeastern U.S.A. (Davidson et al., 1981) with a single exception. This exception was the occurrence of the abomasal nematode *Spiculopteragia assymetrica* in fallow deer. Doster and Friend (1971) reported *S. assymetrica* from free-ranging fallow deer on St. Simons Island, Glynn County, Georgia, and this species also has been reported from fallow deer in Europe (Travassos, 1937). Apparently *S. assymetrica* was introduced into North America with one or more early stockings of fallow deer and has survived in their progeny. The remainder of the parasite fauna of fallow deer on LBL probably was derived via cross-transmission from white-tailed deer, and now both species of deer serve as a "host pool" for these parasites. Previous studies also have found fallow deer to be

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FIGURE 1. Subdural nodular lymphoid hyperplasia at the *cauda equina* in a fallow deer. Formalin fixed.

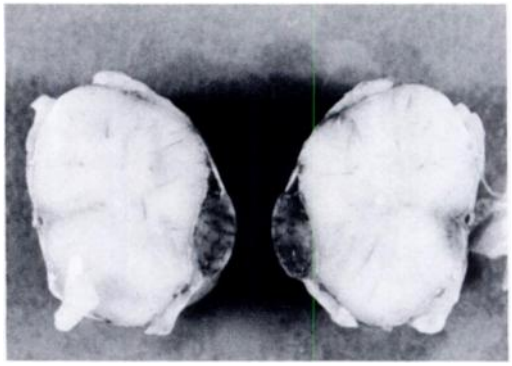

FIGURE 2. Cross-section of lesion in Figure 1 showing compression and displacement of spinal cord in a fallow deer. Formalin fixed.

parasitized by species commonly associated with white-tailed deer including: *Dictyocaulus viviparus, Ostertagia mossi* (Phillips et al., 1974), *Setaria yehi* (Brugh, 1971; Phillips et al., 1974), *Gongylonema pulchrum, Apteragia odocoilei, Cooperia punctata, Haemonchus contortus, Capillaria* spp. (Brugh, 1971), *Oesophagostomum* spp., and *Parelaphostrongylus tenuis* (Nettles et al., 1977).

Lesions attributable to parasitism were noted in both white-tailed and fallow deer. White-tailed deer had the following parasite-induced lesions: (1) mild granulomatous meningitis due to *P. tenuis* (two deer); (2) scattered microgranulomas surrounding protostrongylid eggs and larvae in alveolar capillaries (four deer); (3) mild focal to diffuse fibrinous peritonitis associated with *Setaria yehi* (two deer); and (4) mild focal cutaneous and subcutaneous inflammation associated with attachment sites of *Amblyomma americanum* (five deer).

All fallow deer had similar lesions associated with *A. americanum*. All fallow deer also had lesions in the brain and/or spinal cord that were attributed to *P. tenuis* infection, although repeated gross and histologic examinations failed to disclose meningeal worms. These central nervous system lesions were: (1) focal nodular lymphoid hyperplasia (Figs. 1–3) in the lumbar spinal meninges (two deer); (2) focal leuko- and/or myleomalacia with varying degrees of degenerative and inflammatory changes (Fig. 4) in the lumbar and thoracic cord (two deer); (3) focal non-suppurative spinal and cerebral meningitis (two deer); and (4) minor to extensive perivascular cuffing with eosinophils and mononuclear cells (five deer).

The APC value (848), physical condition ratings, body weights, and other herd health parameters suggested that the white-tailed deer population was near nutritional carrying capacity of the habitat and that mortality due to disease was at an inconsequential level (Eve, 1981). This concept is supported by a low prevalence of moribund or dead deer found on the area despite a high rate of human activity for several years prior to this study. The only recent disease-related losses were a few deer found dead of suspected hemorrhagic disease in 1981 and two fawns with extensive infestations of *A. americanum* around the eyes in the summer of 1980 (SCWDS, unpubl. data).

Fallow deer also appeared to be in a generally good health status although interpretation of parasite levels and other data is less precise due to a lack of comparative baseline information. Of particular interest, however, was the presence in all fallow deer of lesions that were

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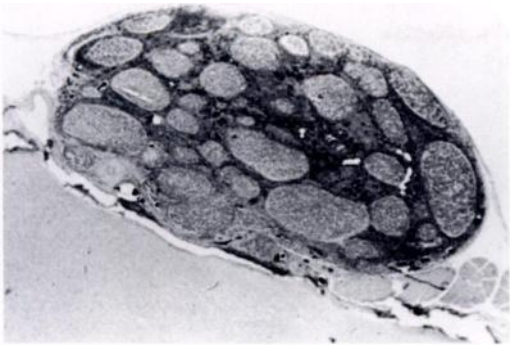

FIGURE 3. Histologic section of lesion in Figure 1 showing multiple germinal centers and location within the *pia mater* of a fallow deer. H&E, ×25.

FIGURE 4. Focal inflammation characterized by malacia, microhemorrhages, infiltration of mononuclear cells and eosinophils, and gliosis in the lateral white matter of the lumbar spinal cord in a fallow deer. H&E, ×125.

strongly suggestive of prior infection by meningeal worms. Nettles et al. (1977) previously demonstrated that cerebrospinal parelaphostrongylosis is an important mortality factor in fallow deer on LBL. These authors noted, however, that on a population basis the disease was "considerably less devastating than that reported for other susceptible ruminants." They also speculated on potential causal factors including a low prevalence and intensity of *P. tenuis* in white-tailed deer on the area and a degree of innate resistance to the parasite by fallow deer.

We believe that the diminished impact of *P. tenuis* on this fallow deer population is a function of (1) a low prevalence and intensity of *P. tenuis*, (2) innate resistance of fallow deer to *P. tenuis*, and (3) acquired immunity. White-tailed deer on LBL do, in fact, have a lower prevalence and mean intensity of *P. tenuis* than most populations of white-tailed deer in the southeastern U.S.A. When compared to the findings of a survey of populations of white-tailed deer in southeastern U.S.A. by Prestwood and Smith (1969), the prevalence was lower than more than half (58%) of the populations and the mean intensity was lower than three-fourths (78%) of the populations (mean intensity recalculated from data of Prestwood and Smith). In addition, previous studies have demonstrated consistently low numbers of larvae per naturally infected gastropod which led Anderson and Prestwood (1981) to conclude that "most deer probably acquire infections from ingesting small numbers of larvae." Exposures of LBL fallow deer to *P. tenuis*, therefore, are likely to involve relatively low numbers of third-stage larvae. Furthermore, based on lesions observed in this study, many fallow deer are able to survive natural exposure suggesting less vulnerability to the parasite than that exhibited by some other species, e.g., moose (*Alces alces*) and caribou (*Rangifer tarandus*) (Anderson and Prestwood, 1981). High initial infection levels (75 larvae) in experimental studies, however, have been routinely fatal for fallow deer (Kocan, pers. comm.) indicating that possible innate resistance is limited. Based on experimental infection studies, Tyler et al. (1980) similarly suggested that mule deer (*O. hemionus*) might be refractory to low but not high doses of infective larvae of *P. tenuis*. Once exposed to and having survived an initial infection, fallow deer likely develop an immunity against reinfection. A similar phenomenon has been demonstrated with repeated weekly low-level (five larvae) in-

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fections of white-tailed deer with the closely related species *P. andersoni* (Prestwood and Nettles, 1977). It also has been suggested to occur in white-tailed deer naturally infected with *P. tenuis* (Anderson and Prestwood, 1981).

Thus, our hypothesis is that many LBL fallow deer, having perhaps a slight degree of innate resistance, survive an initial low-level *P. tenuis* infection and subsequently develop an immunity to reinfection. Clinical disease probably is restricted to the occasional fallow deer that initially acquires higher numbers of larvae or those in which developing meningeal worms produce more severe lesions. This series of events would account for prior field observations (Nettles et al., 1977) of an absence of clinical disease in older (immunized?) fallow deer and a diminished impact of cerebrospinal parelaphostrongylosis on the herd. Similar patterns of clinical cerebrospinal parelaphostrongylosis involving primarily young animals and sporadic clinical disease also have been observed in populations of elk (*Cervus canadensis*) in Oklahoma (Carpenter et al., 1973; Kocan, pers. comm.) and Michigan (Schmitt, pers. comm.) and in red deer (*C. elaphus*) in Pennsylvania (Woolf et al., 1977; Olsen and Woolf, 1978, 1979).

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