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Author: TTTCHE, A. R.

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EXPERIMENTAL INFECTION OF WHITE-TAILED DEER WITH *Elaeophora schneideri* [1]

A. R. TITCHE, [2] Wild Animal Disease Center, Colorado State University, Fort Collins, Colorado 80523, USA.
A. K. PRESTWOOD, Southeastern Cooperative Wildlife Disease Study, Department of Parasitology, College of Veterinary Medicine, University of Georgia, Athens, Georgia 30602, USA.
C. P. HIBLER, Wild Animal Disease Center, Colorado State University, Fort Collins, Colorado 80523, USA.

Abstract: An attempt was made to infect fawn and adult white-tailed deer, *Odocoileus virginianus,* with *Elaeophora schneideri.* Experimental infection of fawns caused a relative eosinophilia that persisted. Obstruction of a coronary artery caused death of one fawn, and weakness, dyspnea, and locomotor difficulties were observed in another fawn and an adult. Plaque-like lesions were observed grossly in the intimal lining of carotid arteries, and subintimal thickening and proliferation of fibrous tissue in vessel walls were observed microscopically. Nematodes were recovered from 3 of 4 fawns and 0 of 4 adults, suggesting an age-related resistance in older animals. Microfilariae were recovered via facial skin biopsy of a single fawn. This study suggests that white-tailed deer serve as usual hosts for *E. schneideri,* although the host-parasite relationship may be tenuous.

INTRODUCTION

The arterial worm, *Elaeophora schneideri,* occurs in domestic sheep *(Ovis aries)* and a variety of North American cervids.[3,8] Mule deer *(Odocoileus hemionus)* are considered usual hosts for the parasite.[5] Infections often cause "filarial dermatosis" in domestic sheep,[6] ischemic necrosis of brain and cephalic tissues in wapiti *(Cervus canadensis)*[1] and moose *(Alces alces),*[10] or unilateral tumorous masses on the head or feet of sika deer *(Cervus nippon).*[8]

Prior to 1962, arterial worm had not been reported in white-tailed deer. Since that time, low prevalences of infections with *E. schneideri* in this host were reported in Arizona,[2] Florida, Georgia, South Carolina[7] and Texas. White-tailed deer were considered the source of infection for *E. schneideri* in sika deer of Texas.[8] Impaired function of mylohyoideus muscles was associated with and possibly caused by *E. schneideri* in the only case of clinical elaeophorosis reported in white-tailed deer.[7] The investigation described herein was conducted to clarify the host-parasite relationship of *E. schneideri* and white-tailed deer.

MATERIALS AND METHODS

Experimental animals — Adult white-tailed deer used for study were two to four years old and, with exception of one deer obtained from a zoo in Norfolk, Virginia, had been reared inside a building where contact with hematophagous arthropods was minimal. To assure freedom of pre-

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[2] Present address: Department of Medicine, College of Veterinary Medicine, University of California, Davis, California 95616, USA.

existing *E. schneideri* infections, a facial skin biopsy[3] was examined from each adult prior to infection. Fawns were obtained locally by Georgia Department of Natural Resources personnel. They were used for infection when three to six weeks old. All deer were housed inside a barn on concrete for the duration of the experiment.

Inoculum — Collections of horseflies, predominately *Hybomitra laticornis*, were made between 23 June and 2 July 1975, in the Gila National Forest of New Mexico. Horseflies were transported on wet ice to facilities of the Southeastern Cooperative Wildlife Disease Study at the University of Georgia's College of Veterinary Medicine. Within three to five days after collection of horseflies, infective third-stage larvae of *E. schneideri* were dissected from the heads and mouthparts, counted, and drawn into syringes.

Experimental design — Four fawns and four adult deer were used for infection. Two fawns and one adult were maintained as uninfected controls. Commencing two weeks before inoculation and at weekly intervals thereafter, blood samples in EDTA were collected for hematocrits and leukocyte counts. A deer in each age class was given 25, 50, 100, or 200 infective larvae via jugular venipuncture. Necropsy schedules were scheduled for 18 and 30 weeks postinfection (wpi). Facial biopsies were taken from surviving deer at 26, 29, and 30 wpi.

At necropsy particular attention was given the eyes, brain, and major arteries. Representative samples of other organs and tissues were taken. Eyes were preserved in Zenker's fluid; other tissues were fixed in 10% buffered formalin. Eyes were sectioned through the equator and optic nerve. Brains were preserved intact, and coronal sections were taken at 5 mm intervals. Tissues were embedded in paraffin and cut at 6 μm for histopathologic study. Hematoxylin and eosin and Verhoeff-Van Gieson stains were used for staining tissue.

RESULTS

Clinical signs — Commencing approximately two weeks postinfection, a relative eosinophilia was observed in fawns given infective larvae of *E. schneideri* (Fig. 1). Although eosinophilia was not absolute, values were sustained above those of the helminth-free controls throughout most of the study.

Clinical signs of disease were observed in 2 of 4 fawns and 1 of 4 adults. The fawn given 50 infective larvae collapsed suddenly 28 days postinfection (dpi). Upon admission to the teaching hospital at the University of Georgia, the heart beat was erratic and a grade three systolic murmur was auscultated. Dyspnea was evident. Thoracic radiography revealed an enlarged left ventricle and suggested the presence of fluid in the lungs. Lactated ringers solution and dexamethasone were administered; however, the condition of the animal deteriorated, and it died approximately 19 hours after discovery of illness.

Simultaneously, a second fawn and an adult developed clinical signs which consisted of weakness, dyspnea, cyanosis, and locomotor difficulties. Signs were observed most frequently between 28 and 35 dpi, although they persisted intermittently in the adult deer for several months.

Post-mortem findings — At necropsy, significant lesions were found in the pulmonary and circulatory systems of the fawn that died 29 dpi. The lungs were consolidated, particularly in the dorsal aspects. Approximately 10 ml of clear fluid was found in the pericardial sac. An infarct approximately 1 cm^2 was located near the apex of the left ventricle (Fig. 2). Upon dissection, an immature *E. schneideri* was found in the adjacent coronary artery. Other coronary arteries appeared swollen, and the surrounding myocardium was blanched in appearance. Fourteen immature *E.*

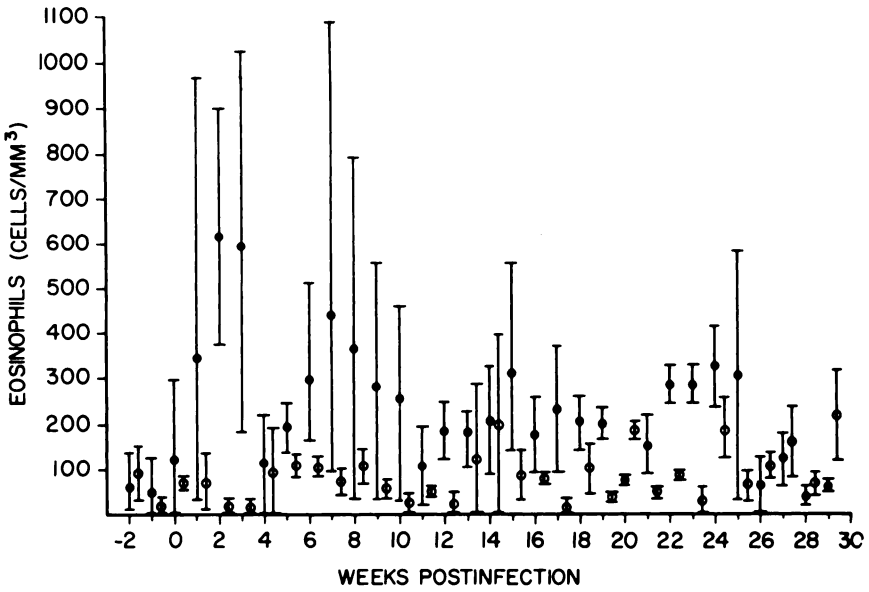

FIGURE 1. Eosinophil counts in fawns given infective larvae of *Elaeophora schneideri* compared with helminth-free control fawns. (0).

schneideri were recovered from this animal (Table 1).

In deer euthanitized 18 and 30 wpi, gross lesions were not pronounced. Although no parasites were found, plaque-like lesions were observed in the intimal lining of carotid arteries in adult deer. Nematodes were recovered from the carotid arteries and, on one occasion, the aorta of fawns given 100 or 200 larvae. Parasites were not recovered from the fawn given 25 larvae. A few microfilariae were detected in a facial skin biopsy of a fawn at 30 wpi. All other biopsies were negative.

Microscopic lesions in the vascular system were mild to moderately severe (Table 2). Seven of eight experimental animals had subintimal thickening in leptomeningeal arteries or arterioles in transverse cerebral sections of the corpus striatum and/or thalamus. Identical lesions were present in the ventral spinal artery of two deer. Only one animal had lesions in the leptomeningeal arteries supplying the cerebellum. Two deer, each of which harbored *E. schneideri* in the carotid arteries, had moderately severe intimal proliferation in cerebral leptomeningeal or carotid arteries. Similar vascular lesions were present in the carotid arteries or their terminal branches in four of the eight experimental deer. In one adult deer, vasculitis of retinal veins was present.

DISCUSSION

Elaeophora schneideri developed in three of four fawns given infective larvae, and percentage recovery (11 to 28%), albeit low, was comparable to that in experimentally-infected mule deer fawns.[5] The development and migration of *E. schneideri* in white-tailed fawns apparently was similar to that in mule deer[4] as evidenced by the presence of immature nematodes in carotid and cor-

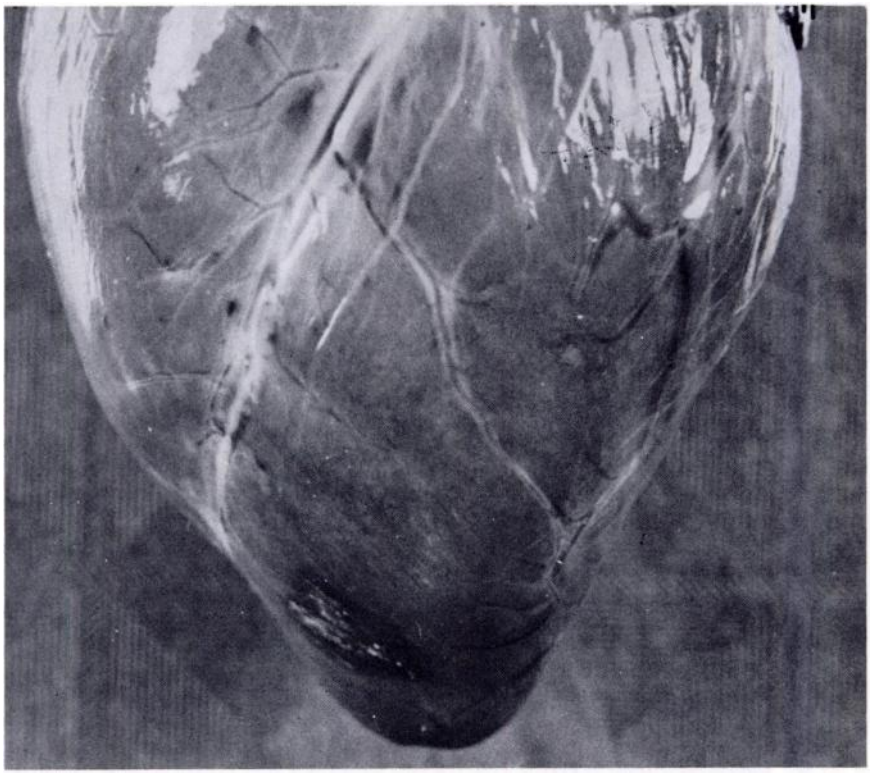

FIGURE 2. Heart of fawn given 50 infective larvae of *E. schneideri*. Note infarct on apex of ventricle.

onary vessels of a fawn that died 29 dpi. The occurrence of microfilariae in a biopsy of facial skin of one deer at 30 wpi reveals for the first time that patent infections occur in white-tailed deer. Absence of microfilariae in a biopsy taken 29 wpi suggests 30 weeks may be the minimum prepatent period in the white-tailed deer.

Failure to recover immature or adult *E. schneideri* from adult white-tailed deer given infective larvae suggests an effective age resistance to infection. The presence of vascular damage in these animals indicates that this age-related protection is not complete; however, it appears sufficient to prevent establishment of the parasites.

The distribution and nature of vascular lesions in experimental animals of this investigation were consistent with those found in wapiti by other investigators.[1] Subintimal thickening and formation of proliferative fibrous tissue within vessel walls characteristic of *Elaeophora* infections in wapiti were observed in white-tailed deer. Occurrence of vascular lesions most frequently in cerebral leptomeningeal arteries and arterioles and least frequently in cerebellar vessels parallels Adcock's proposed sites of predilection for neurologic damage in wapiti.[1] However, the lesions in the white-tailed deer were consistently and dramatically less severe than those in wapiti infected with

comparable numbers of larvae, indicating a considerably less harmful relationship between *Elaeophora* and white-tailed deer. Only in the few instances of relatively severe intimal proliferation was blood flow likely to

TABLE 1. Results of experimental infections of white-tailed deer *(Odocoileus virginianus)* with *Elaeophora schneideri.*

Animal	Number Larvae	Death Weeks Post-infection	Number Parasites Recovered
Adult 1	25	30	0
Adult 2	50	30	0
Adult 3	100	18	0
Adult 4	200	18	0
Control	0	30	0
Fawn 1	25	30	0
Fawn 2	50	4[a]	14
Fawn 3[b]	100	30	11
Fawn 4	200	18	26
Control	0	30	0
Control	0	Not Killed	—

[a]Animal died; all others euthanitized.

[b]Microfilariae recovered from facial skin biopsy of this animal.

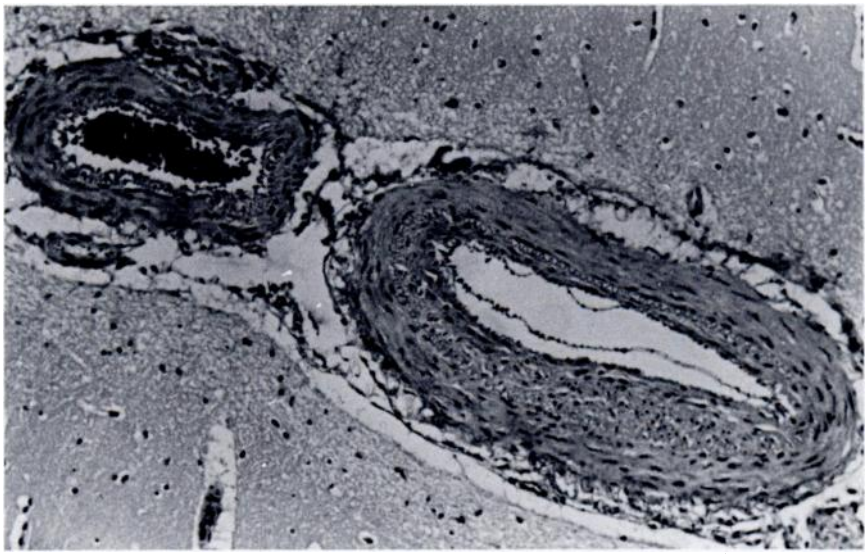

FIGURE 3. Mild subintimal thickening, leptomeningeal artery, level of corpus striatum, Fawn 4. Artifactural detachment of endothelium in affected vessel at right. H&E × 140.

TABLE 2. Summary of lesions produced by experimental infections of white-tailed deer *(Odocoileus virginianus)* with *Elaeophora schneideri*.[a]

	Level of Corpus Striatum				Level of Thalamus				Level of	Anterior		Carotids[c]		Ocular Forehead		
Animal	I[b]	II	III	IV	I	II	III	IV	Cerebellum	Cord	Retes	Left	Right	Venules	Skin	Ear
Adult 1	s	s	s	s	-	-	-	s	s	-	-	s	s	-	s	a
Adult 2	-	s	s	s	-	s	-	-	-	-	s	-	-	v	-	-
Adult 3	-	s	s	s	-	-	s	s	-	s	-	-	s	-	-	-
Adult 4	s	-	-	s	-	s	s	s	-	s	-	-	-	-	-	-
Control	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Fawn 1	-	-	s	-	-	-	-	-	-	-	-	-	-	-	-	-
Fawn 2	p	s	spv	p	-	-	s	-	-	-	-	-	-	-	-	-
Fawn 3	-	-	-	-	-	-	-	-	-	-	-	s	s	-	a	-
Fawn 4	s	s	s	s	s	s	s	s	-	-	-	psa	p	-	-	-
Control	-	-	-	-	-	-	-	-	-	-		-	-	-	-	-

[a]p = intimal proliferation; s = subintimal thickening; v = vasculitis; a = arteriole in region thrombosed.

[b]Transverse sections of cerebrum divided into Cartesian quadrants.

[c]Includes terminal branches of carotid arteries.

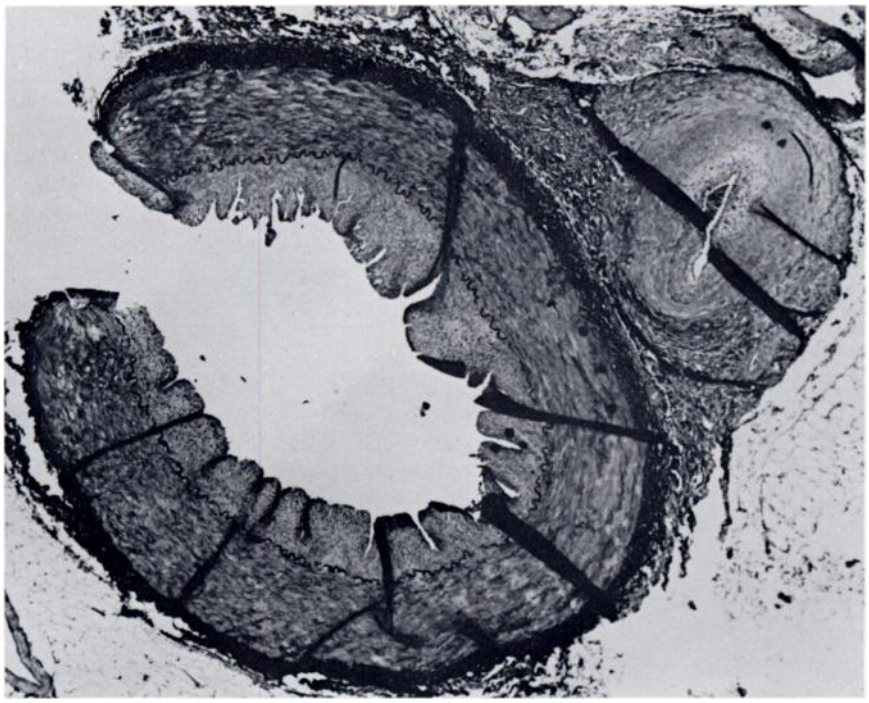

FIGURE 4. Moderately severe villous fibroblastic intimal proliferation, terminal branch of left carotid artery, Fawn 4. Involvement of vessels also includes interruption of internal elastic membrane. Verhoeff-Van Gieson × 35.

have been affected significantly. Thus, absence of classical clinical signs of elaeophorosis (neurologic disease and ischemic necrosis of extremities) can be explained by absence of vascular obstruction of sufficient severity.

Obstruction of a major coronary artery by a migrating *E. schneideri* caused death of one white-tailed fawn, and clinical signs occurred in two additional animals at the time of migration of *E. schneideri* from small cerebral arteries to the larger arteries of the neck and body. Although gross and histologic examinations did not reveal the cause of abnormal signs in these latter animals, lesions may have regressed in severity by the time of necropsy several months later. When considering the results reported herein, previously reported abnormalities associated with heavy infections of *E. schneideri*,[7] and low prevalences of infection in white-tailed deer,[2,7,9] this host-parasite relationship probably should be considered tenuous.

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