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Elaeophorosis in Bighorn Sheep in New Mexico

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ABSTRACT: Two bighorn sheep (*Ovis canadensis*) in New Mexico (USA) were found to be naturally infected with *Elaeophora schneideri*. An adult ram examined in 1997 in the Fra Cristobal Mountains had 26 nematodes in the carotid and iliac arteries, and microfilariae were present in the skin, nasal mucosa, brain, and lungs. This ram was markedly debilitated prior to euthanasia and extensive crusty, scabby lesions were observed on its head. In 1998, a yearling ewe found dead adjacent to Watson Mountain near the Gila Wilderness area was found to have 13 nematodes present in its heart. This is the first report of *E. schneideri* in bighorn sheep, and we suggest that bighorn sheep are susceptible to *E. schneideri* infection wherever they coexist with mule deer (*Odocoileus hemionus hemionus*) and appropriate tabanid vectors.

Key Words: Case report, *Elaeophora schneideri*, bighorn sheep, *Ovis canadensis*, mule deer, Tabanidae

The nematode *Elaeophora schneideri* normally parasitizes mule deer (*Odocoileus hemionus hemionus*) and black-tailed deer (*O. h. columbianus*) in the western United States (Weinmann et al., 1973; Hibler et al., 1974). It has been reported also from atypical hosts including elk (*Cervus elaphus*), moose (*Alces alces*), barbary sheep (*Ammotragus lervia*), domestic sheep (*Ovis aries*), and sika deer (*Cervus nippon*) (Adcock and Hibler, 1969; Worley, 1975; Robinson et al., 1978; Pence and Gray, 1981; Madden et al., 1991). This report describes the initial detection of two cases in bighorn sheep (*Ovis canadensis*) in New Mexico (USA) by field biologists, and the subsequent confirmation of *E. schneideri* infection following necropsy and parasitologic examination.

In October 1995, 37 bighorn sheep captured at Red Rock (New Mexico; 32°42′17″N, 108°44′21″W), were translocated to the Fra Cristobal Mountains (New Mexico; 33°22′30″N, 107°07′30″W), to reestablish a permanent population in what was presumably historic range. Physical examination at the time of capture did not reveal clinical signs or lesions suggestive of clinical disease, with the exception of one animal that had a healing ulcer on its lip and another that had an apparent chronic abscess on its dorsal rump. No ticks or mites were found on the integument or in the ear canal of any of the animals.

All translocated ewes ($n = 24$) and rams ($n = 13$) were fitted with drop-off radiocollars, and the rams also had passive integrative transponder (PIT) tags (Destron-Fearing, St. Paul, Minnesota, USA) placed in their horns. By November 1997, the herd had increased to 43 animals through the recruitment of lambs, despite the fact that several adults had been killed by mountain lions (*Puma concolor*). On 4 November 1997 a ram in the Fra Cristobal Mountains was observed to be in poor condition with skin lesions affecting much of its face and head. The animal's right eye was swollen closed and there was a fluid discharge from that eye and the external nares. Over the next few weeks, new scablike lesions appeared at the base of both horns and the animal's condition deteriorated. At the same time, three other animals in the herd with similar skin lesions were observed to scratch their heads and ears using their hooves.

The ram was euthanized by gunshot in

the late afternoon on 2 December 1997 and transported to the New Mexico Department of Agriculture Veterinary Diagnostic Services Laboratory (Albuquerque, New Mexico, USA). The next day a complete necropsy was performed and representative tissues from all major organs were fixed in 10% neutral buffered formalin. A PIT tag recovered from one horn confirmed that the ram was originally translocated to the Fra Cristobal Mountains from Red Rock in 1995 (he had lost his drop-off radiocollar). Gross examination revealed multiple, raised, crusty skin lesions on the forehead region, around the base of the horns, and on the inner surface of the pinnae of the ears. No mites or ticks were found in or around these lesions. The largest lesion was about 5 cm in diameter, and the skin in affected areas was about twice normal thickness. Lesions on the forehead extended from the horns to the external nares, and the general appearance of these lesions was remarkably similar to that described and photographed in barbary sheep affected by elaeophorosis (Pence and Gray, 1981). Twenty-four nematodes, ranging from 4 to 10 cm in length, were found in the carotid arteries and a single nematode was found in each of the two iliac arteries. These nematodes were identified as *E. schneideri* (Hibler and Adcock, 1968), and representative specimens have been deposited in the U.S. National Parasite Collection (Beltsville, Maryland, USA; USNPC No. 87636). Multiple raised yellow-white plaques were found in the nasal mucosa covering the turbinates. No dipterous larvae (e.g., *Oestrus ovis*) were found in the nasal cavity or frontal sinus. No gross lesions were found in the lungs, kidneys, spleen, gastrointestinal tract, or brain.

Formalin-fixed tissues were embedded in paraffin, sectioned at 4 μm, stained with hematoxylin and eosin, and examined for microscopic lesions. Sections of skin from the head revealed focal erosion and ulceration and a dense infiltration of macrophages, eosinophils, lymphocytes, and plasma cells into the dermis. There were scattered microfocal areas of necrosis and microfilariae present in some of these foci. Microscopic examination of the nasal plaques seen during gross necropsy revealed a dense infiltration of mixed inflammatory cells, microfocal areas of necrosis, and rare degenerating microfilariae. Examination of multiple sections of lung revealed numerous vessel-associated infiltrates of inflammatory cells, and degenerate microfilariae were present in the lumen of scattered pulmonary arterioles and capillaries. Multifocal inflammatory cell infiltrates were found in the meninges, and thick perivascular cuffs composed primarily of plasma cells and lymphocytes were found in all major areas of the brain. Degenerate microfilariae were found in small vessels scattered throughout the brain. The eyes and associated optic nerves and vessels were not examined histologically.

The second case of elaeophorosis was detected in October 1998 when a yearling ewe was found dead near Watson Mountain in western New Mexico (33°05′25″N 108°31′44″W). This ewe was a member of a small herd of resident bighorn sheep established in the Turkey Creek area by translocations from Banff National Park (Alberta, Canada) in 1964. The cause of death could not be determined, but gross examination of the heart revealed 13 nematodes, ranging in length from 4 to 11 cm, that were subsequently identified as *E. schneideri* (USNPC No. 88758). Carotid arteries, skin and other organs were not available for detailed parasitologic examination. However, the biologist who found the ewe noted that additional nematodes (not collected) were protruding from arteries in the neck when he cut off the ewe's head. *Elaeophora schneideri* adults are typically found in the carotid arteries, and the presence of nematodes in the heart may have resulted from postmortem migration. Based on examination of the two female worms found in the heart, we concluded that this may have been a prepatent infection since undeveloped eggs

rather than microfilaria were present in the uteri.

Mule deer and black-tailed deer are considered the normal definitive hosts for *E. schneideri* in the western United States and typically do not exhibit clinical signs of infection (Weinmann et al., 1973; Hibler et al., 1974). In contrast, several other wild and domestic ruminants have been found to be clinically affected including elk, moose, barbary sheep, domestic sheep, and sika deer (Adcock and Hibler, 1969; Worley, 1975; Robinson et al., 1978; Pence and Gray, 1981; Madden et al., 1991). The presence of *E. schneideri* in the adult ram and yearling ewe examined in this study clearly indicates that bighorn sheep are susceptible to elaeophorosis. However, it is surprising that this is the first time *E. schneideri* has been found in bighorn sheep. Bighorn sheep are intensively managed and it seems likely that the lesions caused by elaeophorosis would have been detected previously if clinical infection was present. In fact, in November 1997 (prior to finding *E. schneideri* in any bighorn sheep) we also captured and examined 47 additional bighorn sheep at Red Rock, and 7 bighorn sheep in the Turkey Creek herd. None of these animals were observed to have lesions consistent with elaeophorosis; however, we did not examine skin biopsies for the presence of microfilariae and thus we could not detect subclinical infections. Although speculative, it is possible that *E. schneideri* infections have not been detected previously in bighorn sheep because heavily infected animals are likely to die.

The occurrence of *E. schneideri* in atypical hosts such as bighorn sheep is likely to be restricted to areas where there are adequate densities of deer definitive hosts and tabanid vectors (Clark and Hibler, 1973; Worley, 1975). These conditions clearly exist for the Turkey Creek herd located near to the Gila Wilderness area where *E. schneideri* infections in elk, mule deer, and tabanid vectors have been well-documented (Hibler et al., 1968; Clark and Hibler, 1973). In 1997 and 1998 biologists monitoring bighorn sheep in the Fra Cristobal Mountains observed mule deer within 200 m of bighorn sheep on over 20 occasions, and >100 deer were counted during ground surveys of the range. In addition, in 1997 biting flies frequently attacked both humans and bighorn sheep in the spring and summer months. We speculate that the nearby Elephant Butte Reservoir (<10 km away on the Rio Grande River) may have created appropriate habitat for the production of large numbers of competent vectors, and that the resident mule deer population has served as a reservoir of infection for the newly established bighorn sheep population. Additionally, conditions also may be favorable for *E. schneideri* transmission to bighorn sheep in other areas such as the Red Rock facility where bighorn sheep, mule deer, and tabanid vectors all co-occur.

It remains to be demonstrated what effect elaeophorosis has on the fitness and viability of bighorn sheep populations. We encourage other investigators to examine bighorn sheep for characteristic lesions and the presence of *E. schneideri* in the heart and arteries. Although *E. schneideri* microfilariae are not likely to be found in the blood, we have successfully used the methods of Weinmann et al. (1973) to isolate microfilariae from skin punch biopsies of infected deer. In the absence of a serologic assay, the collection and analysis of skin biopsies during routine capture operations may provide the best approach for expanding our knowledge of the distribution and prevalence of *E. schneideri* infection in bighorn sheep.

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