ELAEOPHOROSIS IN DEER AND ELK IN THE GILA FOREST, NEW MEXICO *

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The Gila National Forest in southwestern New Mexico contains about 4,000 square miles of extremely rugged, mountainous terrain. Altitudes range from 6,000 feet to over 10,000 feet, and the climax vegetation is primarily Ponderosa pine, with some spruce and fir at the higher elevations. Streams and ponds are abundant. The center of the forest has been designated as the Gila Wilderness, approximately 600 square miles of inaccessible land even more rugged than the surrounding Gila Forest.

Historically, the forest is a significant site for the study of elaeophorosis because it was here that Kemper (1938, N. Am. Vet., 19:36-41) discovered*Elaeophora schneideri* Wehr and Dikmans, 1935, and incriminated it as the causative agent of the disease called "sorehead," a filarial dermatitis affecting the forehead and face of domestic sheep sent to summer range in these mountains. For many years, Kemper worked with elaeophorosis among sheep in the forest, laying a firm foundation for many of the studies being undertaken now that elaeophorosis has been established as a disease of North American elk, *Cervus canadensis* (Adcock, Hibler, Abdelbaki and Davis, 1965, Bull. Wildl. Dis. Assoc., 1:48; Adcock, 1967, Dissert., Colo. State Univ.).

The first recorded case of elaeophorosis in elk in New Mexico was in a young bull calf found at the southern edge of Gila Forest in the late summer of 1964. This animal survived the disease and has been maintained in captivity for long-range studies. It has all the characteristic signs of elaeophorosis described by Adcock *(op cit.):* total blindness, nystagmus, circling, abnormal antler growth, cropped ears, and necrosis of the muzzle. In September, 1965, another blind elk calf was found in the same area. At the necropsy of this animal, 123 fifth stage, but sexually immature *E. schneideri* were recovered from arteries throughout the body (Adcock, Hibler, Abdelbaki and Davis, *op cit.*). This case showed that *E. schneideri* is responsible for the disease in elk referred to as "clear-eyed blindness." On October 20, 1967, a blind elk calf was found in the Gila Wilderness, and 50 *E. schneideri* were recovered at necropsy. Five days later, another blind elk calf was found in the northern edge of the Forest. This animal had 110 *E. schneideri* in its arteries. These findings, especially with respect to the number of worms per animal, strongly suggested that elaeophorosis might be extremely prevalent in the forest. Thus, in May, 1968, 10 mature elk were killed and examined for signs of infection with *E. schneideri.* The results confirmed earlier expectations; nine of the 10 elk examined had moderate to severe arterial lesions and dead, calcified parasites in the common carotid, internal maxillary, and other arteries supplying the cephalic region of the body. Damage to the arteries was often so severe it was

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surprising that the animal could live. One elk had a cropped ear, an excellent indication of parasitism by *E. schneideri*. Observation of additional elk in the forest revealed that five of eight had at least one ear cropped.

On September 12, 1968, another blind elk calf was found in the Gila Wilderness. It had the most severe case of parasitism seen thus far; 145 living *E. schneideri* were found in arteries throughout the body. In addition, many dead parasites were present in the right common carotid and internal maxillary arteries.

Game and fish biologists in New Mexico have been concerned about the elk herd in Gila Forest since they were transplanted from Yellowstone National Park in 1954-56 because the population was not increasing at the rate anticipated. Consequently, the herd has been carefully surveyed for a number of years. Present information indicates that at least 90% of the cows conceive and have a normal calf. However, in late summer and early fall, the surveys reveal only 15-20% survival of elk calves. The extent to which elaeophorosis is responsible for this severe mortality is presently unknown, but our present information indicates that *E. schneideri* does much of its damage during late summer and early fall.

E. schneideri was found in a mule deer, *Odocoileus hemionus hemionus,* in Utah shortly after Kemper discovered the nematode in domestic sheep (Wehr and Dikmans, 1935, Zool. Anz., 110:202-208). Subsequently it has been found in deer in a number of other states and Canada. Strangely enough, however, *E. schneideri* has never been reported from deer in New Mexico. Since the authors believe deer play an important role in the biological cycle of this disease, 10 mature mule deer were killed in June, 1968, and examined for *E. schneideri.* Four of six animals killed in the northern edge of the Forest were infected with *E. schneideri;* two killed near the center of the Forest (at the Gila Cliff Dwellings) were infected; and two killed in the southeastern edge of the Forest (in the Black Range Primitive Area) were uninfected.

Data obtained thus far indicate that the majority of elk calves become infected with *E. schneideri* about two to three weeks following birth, which in the enzootic areas of Arizona and New Mexico places the time of infection near mid-June (Adcock, *op. cit.*). Therefore, in June bloodsucking arthropods feeding on deer and elk were collected and examined for filarioid larvae. Simuliidae, Tabanidae, and Rhagionidae were abundant. The simuliids were observed feeding on the skin deep inside the ears of deer, a site where microfilariae of *E. schneideri* have not been found. The tabanids and rhagionids, however, fed almost exclusively on the forehead and face of deer, sites where microfilariae are abundant. They fed in such numbers that the faces of deer were covered with thick crusts of dried blood. The skin of the nose was swollen three to four times its normal thickness, and irritation had apparently stimulated the animals to rub and scratch because the face was almost devoid of hair.

Three hundred and eighteen horseflies were collected from an area where deer had been bedding for an extended period of time. They were infected with a total of 140 first, second and third stage filarioid larvae. An additional 1,298 flies were collected at random from sites all over the northern edge of the forest. A total of 60 larvae were found in these. Most of the flies were macerated and soaked in physiological saline to collect the larvae, so accurate information on the incidence of infection is not available.

The third stage, or infective, larvae are 3.8 to 4.2 mm long by 0.048 to 0.052 mm thick. These extremely large larvae have the number and arrangement of cephalic papillae identical to the pattern found on adult *E. schneideri* (Anderson, 1968, Can. J. Zool., 46:181-199; Hibler and Adcock, 1968, J. of Parasitol., In Press). Moreover, they have three protuberances on the tail, a feature also possessed by the adults (Hibler and Adcock, *op. cit.*). This finding strongly indicates that the larvae found in the tabanids are larval stages of *E. schneideri* and efforts to prove this are currently underway.

Two species of Tabanidae were found in the forest, and both of these were infected. *Hybomitra sonomensis* var. *phaenops* Osten Sacken, commonly called the "Green-headed Horsefly," or the "Green-headed Fly" was the more abundant, making up approximately 85% of the tabanid population. *Tabanus marginalis* Fabricius constituted the remaining 15%. The "Green-headed Horsefly" is also prevalent in enzootic areas of elaeophorosis in Arizona and Colorado. If the tabanids are intermediate hosts, this fly is so common that it may, by virtue of numbers alone, be the most important intermediary.

Observation of tabanids feeding showed that complete engorgement required about one minute, and within one hour 20 to 25 would feed on the face of one deer. The flies were active from about 9:00 A.M. until 4:00 P.M., with the greatest amount of activity occuring between 11:00 A.M. and 2:00 P.M.

Examination of species of *Symphoromyia* (Diptera: Rhagionidae) revealed that they, too, were infected with the same filarioid larvae found in tabanids, but only a few of these insects were infected and the only larvae recovered were in the first stage of development. Thus, the role of the rhagionids in transmitting the parasite is unclear. While present information suggests their role is of secondary importance, they must be considered in all future work.

In the Gila Forest, tabanids and rhagionids emerge about the last week in May and reach a peak in number during the hot, dry month of June. They disappear almost entirely with the advent of summer rains, which begin during the first week in July. This, if true of all enzootic areas of elaeophorosis, may explain why infection in elk calves generally occurs during the second and third week of life.

As stated above, deer have an abundance of microfilariae of *E. schneideri* in capillaries of the skin on the forehead and face, but they are seldom found in samples of skin from sheep and elk. Thus, it can be postulated that tabanids feeding on deer in early June become infected, and after a period of development in the fly (present information indicates about two weeks), the infective larvae are transmitted to other deer, elk, or domestic sheep. The month-long life span of the flies would also explain the absence of infection in fawns, which in these areas usually are not born until after mid-July.

If, as the above data indicate, species of *Hybomitra* and *Tabanus* are intermediate hosts for *E. schneideri*, the observations on the number of flies present during June, number feeding per hour, and the incidence of infection, makes it extremely unlikely that any deer or elk in the Gila Forest could escape exposure to infection. While the high rate of mortality among elk calves in the Gila is probably the result of many factors, when all aspects of this situation are considered, elaeophorosis may prove to be the most serious.

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DISCUSSION CONFERENCE ON DISEASES OF WATERFOWL

The Wildlife Disease Association and the Patuxent Wildlife Research Center are cosponsoring a two day discussion on diseases of waterfowl, March 6-7, 1969 at the Research Center in Laurel, Maryland.

Program will be centered on the topic of diagnosis of major waterfowl disease problems. March 6 will be devoted to a discussion of various parasitic diseases of waterfowl including blood protozoa, coccidia, amoeba-like organisms, and pathogenic helminths.

The second day, March 7, will be devoted to a discussion of bacterial, fungal, and viral diseases of waterfowl, including fowl cholera, duck viral enteritis (= duck plague), aspergillosis and salmonellosis.