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# EXPERIMENTAL INFECTION OF IMMATURE MULE DEER WITH *Elaeophora schneideri**

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*Abstract:* Five neonatal mule deer *(Odocoileus hemionus)* and five yearling mule deer were experimentally infected with *Elaeorphora schneideri.* Infections ranging from 2 to 31 parasites established in 9 of 10 animals. Evidence of elaeophorosis was not observed in any animal, indicating that mule deer are normal definitive hosts.

## INTRODUCTION

Surveys of mule deer for prevalence of infection with *Elaeophora schneideri* Wehr and Dikmans, 1935 indicate that the parasite is found commonly in adults, rarely in yearlings and never in fawns in the Rocky Mountain Region of the western and southwestern United States.[2] In the Gila Forest in southern New Mexico and the Sitgreaves and Coconino Forests in eastern Arizona, fawns are born in July, which is after the horsefly season,[4] but in northern New Mexico and southern Colorado fawns are born during the horsefly season.

Absence of infection in fawns, and a low prevalence of infection in yearlings examined in areas where the horsefly is active during the fawning season suggested two possible alternatives: fawns and yearlings were either refractory or highly susceptible to infection with *E. schneideri.* The latter possibility was in need of clarification and prompted experimental infection of fawns and yearlings.

## MATERIALS AND METHODS

Mule deer in northern New Mexico drop fawns in late June and early July. To insure that a sufficient number of neonatal fawns would be available for experimental infection with *E. schneideri* during the period of maximum horsefly activity in the Gila Forest, eight mature does were captured in early winter and transported to holding facilities at the Heart Bar Wildlife Area in southern New Mexico. The does were assumed to be pregnant at the time of capture because previous observations indicated that at least 90% of the does in this area conceived and the fawn to doe ratio was about 1.5 to 1.

Yearling deer for use in the study were captured throughout New Mexico, or raised at the Heart Bar Wildlife Area in the Gila Forest. Prior to experimental infection, they were examined for possible patent infection by the skin biopsy method.[1]

Third-stage larvae dissected from the heads and mouthparts of naturally infected horseflies were counted and pooled in physiological saline. They were inoculated within 30 minutes after collection. The fawns were inoculated intradermally by introducing small numbers of larvae into various sites on the forehead and facial region but all yearling deer were inoculated via the jugular vein.

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At the termination of this experiment a postmortem examination was performed on each animal and the tissues examined for gross and histopathologic evidence of damage as a result of infection by *E. schneideri*.

## RESULTS

Results of experimental infections are summarized in Table 1. The eight does captured in northern New Mexico gave birth to 12 fawns, four of which died at birth. Of the remaining eight, five were inoculated with third-stage *E. schneideri* when 24 to 48 hours old, and three served as uninfected controls.

During the interval between inoculation and postmortem examination, none of the fawns or yearlings showed any clinical manifestations of elaeophorosis. At postmortem examination no gross or histopathologic changes were evident except in one artery of yearling #4, which had a few plaque-like thickenings of the intimal lining in the left internal maxillary artery. These were extremely minor and would not interfere with passage of blood.

Yearling #5 died accidentally 19 days after infection; consequently, most of the parasites were located in cerebral arteries and were in the early 5th stage of development. During this 19 day interval the animal did not show any clinical manifestations of elaeophorosis, nor were there gross morphologic changes observed in the brain. The brain, however, was not examined histologically for disease.

TABLE 1. Experimental Infection of Immature Mule Deer with **E. schneideri.**

| Animal | Larvae Given | Date Infected | Date Necropsy | Parasites Recovered | % Recovery | Location* Left | Location* Right |
|---|---|---|---|---|---|---|---|
| Fawn | | | | | | | |
| 1 | 50 | 7/4 | 10/24 | 2 | 4.0 | 0 | 2 |
| 2 | 125 | 7/4 | 1/18 | 28 | 22.4 | 16 | 12 |
| 3 | 50 | 7/5 | 1/18 | 0 | 0.0 | 0 | 0 |
| 4 | 100 | 7/5 | 1/18 | 5 | 20.0 | 2 | 3 |
| 5 | 50 | 7/5 | 1/18 | 23 | 46.0 | 11 | 12 |
| 6 | 0 | Negative** | | — | —— | — | — |
| 7 | 0 | Negative** | | — | —— | — | — |
| 8 | 0 | Negative** | | — | —— | — | — |
| Yearling | | | | | | | |
| 1 | 133 | 6/23 | 9/10 | 31 | 23.3 | 12 | 19 |
| 2 | 200 | 6/25 | 9/11 | 24 | 12.0 | 20 | 4 |
| 3 | 200 | 6/25 | 9/11 | 29 | 14.5 | 22 | 7 |
| 4 | 180 | 6/24 | 9/11 | 8 | 4.4 | 7 | 1 |
| 5 | 150 | 6/25 | 7/14 | 8 | 5.3 | 1 | 6*** 1 |
| 6 | 0 | Negative** | | — | —— | — | — |
| 7 | 0 | Negative** | | — | —— | — | — |
| 8 | 0 | Negative** | | — | —— | — | — |

* Common carotid and internal maxillary arteries

** Examined by skin biopsy technique the following summer

*** Cerebral arteries

## DISCUSSION

When the results of this experiment are combined with observations on hunter-killed deer, animals collected specifically for studies on elaeophorosis, and experimental infection of adult mule deer, the data strongly indicate that mule deer are normal definitive hosts for *E. schneideri* and are not subject to elaeophorosis. Yet the numbers of animals used in this experiment and in the experiment on adult mule deer[3] are insufficient to deduce that disease never occurs in the mule deer. Clinical manifestations of disease have been reported in white-tailed deer.[5] Our own observations suggest that deer which escape infection until the 4th or 5th year of life (which is unusual in enzootic areas) may be susceptible to the disease. The mule deer experimentally exposed to horseflies in the Gila Forest in 1970 may have been examined too soon for any manifestation of elaeophorosis.[3] During 1971, a 5 year-old mule deer doe was observed to be blind, circling and showing posterior ataxia. Forty-seven 3rd and 4th stage *E. schneideri* were recovered from her leptomeningeal arteries. Unfortunately, postmortem autolysis prevented effective examination of the brain for histologic evidence of elaeophorosis.

The range in number of parasites recovered from the 10 experimentally infected deer (2 to 31) is not significantly different from the range observed in 86 naturally-infected mule deer (3 to 29) for which accurate records have been kept. When the range in number of parasites found in naturally-infected mule deer is compared with the present recovery in this and the previous experiment,[3] there is a temptation to suggest that mule deer will only tolerate a specific number of parasites, regardless of the number introduced. Considerable caution must be exercised in making such an interpretation because we have no way of knowing what percentage of the 3rd stage larvae experimentally introduced are actually infective; in addition, if the previous experiment can be relied upon as an indication of our efficacy relative to the horsefly, the percentage is low because 87 parasites became established in that deer.[3] While we have no way of knowing the number of larvae introduced into that animal versus the number established, we can assume that the horsefly is more efficient.

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