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Clinical Treatment of Meningeal Worm
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James Gerard Sikarskie

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# CLINICAL TREATMENT OF MENINGEAL WORM (*PARELAPHOSTRONGYLUS TENUIS*) IN WHITE-TAILED DEER (*ODOCOILEUS VIRGINIANUS*) WITH ALBENDAZOLE

By

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# ABSTRACT

## CLINICAL TREATMENT OF MENINGEAL WORM (*PARELAPHOSTRONGYLUS TENUIS*) IN WHITE-TAILED DEER (*ODOCOILEUS VIRGINIANUS*) WITH ALBENDAZOLE

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This study was initiated to evaluate the efficacy of albendazole as an anthelmintic for clinical treatment of meningeal worm (*Parelaphostrongylus tenuis*) in white-tailed deer (*Odocoileus virginianus*). Although infection with this parasite is seldom manifested clinically in the normal host, the white-tailed deer, typically it causes a neurological disease which is often fatal in abnormal hosts such as moose (*Alces alces*), elk (*Cervus canadensis*), and woodland caribou (*Rangifer tarandus terraenovae*), as well as domestic sheep and goats. It was felt that if an oral anthelmintic was found that could be used to treat hosts which serve as a reservoir of this parasite, it might have value as a management tool to assist reintroduction or establishment of moose and elk or other susceptible wild ruminants in areas inhabited by deer with meningeal worm. It would also be useful to help control the problem in areas where domestic and exotic animals are affected.

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Two trials were conducted using captive reared white-tailed deer infected experimentally with meningeal worm. Parasite burden was established by performing daily Baermann fecal analyses on 2 gm samples from each deer. A 2-week treatment of albendazole at approximately 25 mg/kg of body weight in each of 2 daily feedings dropped fecal larval counts to zero, while counts in controls remained unaffected. Necropsy revealed live worms in the meninges of all deer 1 week after the end of treatment in trial I. Necropsy of trial II deer 1 month after treatment revealed only dead encapsulated worms in treated animals, while controls were infected with many live parasites. These results permit the conclusion that albendazole is effective against meningeal worm in white-tailed deer.

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# INTRODUCTION

## History

The meningeal worm was first identified in white-tailed deer and named *Pneumostrongylus tenuis* by Dougherty (1945). The parasite had a rather confusing taxonomic history because its larvae resembled those of several other metastrongyle parasites and it was given new names as it was discovered in different hosts. Whitlock (1952) found the parasite in sheep and later (Whitlock, 1959) helped explain some of the confusion. Anderson (1972) brought the history up to date with an explanation of the transfer of the parasite to the genus *Parelaphostrongylus* by Pryadko and Boev in 1971. Some other synonyms occurring in the literature before this point are *Odocoileostrongylus tenuis*, *Elaphostrongylus tenuis*, and *Neurofilaria cornellensis*.

The problems caused by *Parelaphostrongylus tenuis* were just as confusing. There was a neurological disease seen in moose for years, but the etiology was unknown and it was simply referred to as "moose sickness" or "moose disease." Smith (1964) tentatively determined it was caused by *P. tenuis*, and in 1967 Smith and Archibald published that naturally occurring moose sickness was definitely caused

by the common deer meningeal worm. Much research was done in the 1960's on the development of the meningeal worm in its normal host, the white-tailed deer (Anderson, 1963, 1965a), and its development and effects in experimentally infected abnormal hosts (Anderson, 1964; Anderson et al., 1966). There was also increased interest in the effects white-tailed deer infected with *P. tenuis* could have on other wild populations of artiodactylids (Anderson, 1965c, 1972).

## *P. tenuis* in White-Tailed Deer

*Parelaphostrongylus tenuis* is a metastrongyle nematode. Metastrongyles are referred to as lungworms because they either reside in the lungs or have a stage of their life cycle passing through the lungs of their host. The parasite is acquired by a foraging deer ingesting slugs or snails containing third stage ($L_3$) infective larvae. Lankester and Anderson (1968) showed that many species of land gastropods functioned as natural intermediate hosts. No aquatic snails were found to be infected, even though earlier studies (Anderson, 1963) showed that some could be infected experimentally. They also determined that infected snails could survive the winter and thus were a potential source of infection early in the spring when the moisture favors the snails and deer are eagerly eating the new growth of vegetation. After ingestion the *P. tenuis* larvae penetrate the bowel and make their way to the spinal cord, presumably by way of the nerves (Anderson, 1965a). They undergo development to adults

while migrating through the dorsal horns of the gray matter within the spinal cord. They cause very little tissue reaction within the parenchyma of the spinal cord and normally by 40 days time move into the spinal subdural spaces, where they mature. Adult *P. tenuis* migrate to the cranial region, where they reside in the venous sinuses and the subdural space. Females deposit eggs in venous blood vessels or surrounding tissues. These eggs can either embryonate within the cranium with larvae penetrating the venus sinuses and being carried to the lungs or be deposited directly within blood vessels and are carried to the lungs, where they form emboli and later embryonate. Larvae penetrate the alveolus, are coughed up or ascend the respiratory tract and are swallowed, passing out in the feces. The length of time from ingestion of the infected snail until patency or passage of larvae in the feces is usually 82-91 days (Anderson, 1965a).

First stage ($L_1$) larvae passing out in the feces are usually within the mucous coat around the fecal pellet. Snails are attracted to the feces and the *P. tenuis* larvae penetrate the foot of the snail or are ingested. They mature to $L_3$ within 3-4 weeks at summer temperatures. Lankester and Anderson (1967) showed that first stage larvae can remain infective when frozen over winter but tend to wash out of the fecal pellet and are readily dispersed by high spring water or heavy rainfall.

*P. tenuis* in Abnormal Hosts

Although infection with this parasite is seldom manifested clinically in the normal host, the white-tailed deer (Anderson, 1963), it can cause a neurological disease which is often fatal in many abnormal hosts. Woolf et al. (1977) gave a complete listing of the clinical signs associated with neurological disease in elk in Pennsylvania. Along with listlessness and decreased flight distance, there is general and lumbar weakness with ataxia and staggering often accompanied by circling. These signs are caused by damage to the spinal cord and brain tissue and the resulting inflammation. They often progress to paralysis and death. Naturally infected white-tailed deer occupying the same range as susceptible abnormal host species have been shown to be the source of these clinical problems under at least 3 types of management conditions. Probably the most common and important area is in management of wild populations of susceptible native cervids. Smith et al. (1964) and Anderson (1965b) were the first to expose the problem in moose. Anderson et al. (1966) showed that mule deer (*O. hemionus*) were experimentally susceptible. In 1973 Fay and Stuht linked *P. tenuis* to neurological disease in elk in Michigan, and later Carpenter et al. (1973) found *P. tenuis* in elk in Oklahoma. Reindeer (*Rangifer tarandus tarandus*) (Anderson, 1971) and caribou (Trainer, 1973) are also affected by this disease under natural conditions. The second situation in which *P. tenuis* has caused clinical disease is in exotic ruminants introduced in areas to which

infected wild white-tailed deer had access. Kistner et al. (1977) and Nettles et al. (1977) found fallow deer (*Dama dama*) with neurological disease caused by meningeal worm and Brown et al. (1978) found the problem in llamas (*Lama guanicoe*). The third important area of concern is cerebrospinal parelaphostrongylosis in domestic animals. It was found as early as 1952 (Whitlock) in sheep and written up by Nielson and Aftosmis (1964) and found again in sheep by Alden et al. (1975). *Parelaphostrongylus tenuis* has also caused neurological disease in goats on pastures used by infected white-tailed deer (Mayhew et al., 1976; Guthery et al., 1979).

## Ecology and Management of the Disease

If an effective oral anthelmintic could be found, it might be used to treat the reservoir hosts in areas where managers are attempting introduction or reestablishment of susceptible species like moose and elk. It would also be useful to treat white-tailed deer prior to translocation to areas like the western U.S. and Canada, which apparently do not have *P. tenuis*, although some areas have white-tailed deer and there are many species of land gastropods which could function as intermediate hosts. There are many potentially susceptible species in western North America, and introduction of meningeal worm could have very grave consequences. This is especially true with the pressures that dwindling habitat have put on wild populations. Competition for limited range and forage would tend to bring susceptible

species into contact with infected white-tailed deer and could increase the potential for exposure. Anderson (1972) discussed the ecological relationship of meningeal worm in the adaptable white-tailed deer and the competitive advantages the parasite gives this species over susceptible cervids.

Management of moose. It is well documented with moose that habitat changes, such as logging, have increased deer populations and *P. tenuis* has caused neurological disease decreasing moose numbers (Karns, 1967; Smith and Archibald, 1967). The implications of this deer-moose relationship have been studied extensively in Maine. Behrend and Witler (1968) found the prevalence of *P. tenuis* highest (100%) in white-tailed deer where populations were densest and expanding. This would indicate that greater density can facilitate spread of the parasite but that it does not seem to exert any major limitations on deer populations. However, there have been a few naturally occurring cases of neurological disease in white-tailed deer (Alibasoglu et al., 1961; Eckroade et al., 1970; Prestwood, 1970).

Gilbert's study (1973) of white-tailed deer and *P. tenuis* in Maine did not always correlate rate of infection with deer density. This indicates other variables, such as habitat types and prevalence of snail intermediate hosts, must help influence rate of infection and transmission. Further studies did, however, directly relate prevalence in moose to density of the deer population (Gilbert, 1974).

Kearney and Gilbert (1976) studied the ecological factors affecting transmission of *P. tenuis* to moose and found that there are areas where white-tailed deer and moose can coexist on the same range. They showed that areas supplying optimum habitat for both moose and deer allowed some isolation by habitat preferences during the summer when spread of the parasite is more likely to occur. Areas supplying these habitat configurations served as "refugia" or "refuges" for moose and helped maintain local populations in areas where white-tailed deer with *P. tenuis* existed. Anderson (1979, personal communication) feels this occurrence is exceptional and that the reason for the decline in moose populations with the influx of deer in many areas is the fact that they have very similar habitat requirements and preferences and *P. tenuis* helps the deer eliminate the moose.

Management of elk. Much attention has been given to elk and the similar problem they have surviving in areas inhabited by white-tailed deer with *P. tenuis*. Moran (1973), in his thorough study of Rocky Mountain elk in Michigan, attributed much of the decline in elk numbers and reproductive success to deterioration of habitat quality. He also felt that *P. tenuis* and neurologic disease played a role in hindering the success of introduction, dispersion, and establishment of both moose and elk over the north-central range of the white-tailed deer. George et al. (1974) did an in-depth study of the ecology of the remaining Rocky Mountain elk in Pennsylvania. They felt habitat quality was

not a major problem but that *P. tenuis* was the most serious reason for the declining population.

Further research on a captive herd of elk at the Rachelwood Wildlife Research Preserve in Pennsylvania by Woolf et al. (1977) showed a high prevalence of infection (26.6-64.3% of samples taken). Many individuals did not show obvious signs and they felt that even though there were no sudden or massive die-offs the parasite might be limiting the growth of the herd, especially by its detrimental effect on population recruitment with the apparently greater susceptibility of younger age classes. Further studies by Olsen and Woolf (1978) showed that neurologic disease may have other subtle detrimental effects on population dynamics besides causing death. It increases susceptibility to predation by natural means or harvest by man. It also may have a detrimental or disrupting effect on social organization within the herd and affect individual behavior such as breeding, rut, calving, and maternal care. An update on prevalence in the herd in 1979 (Olsen and Woolf) showed the disease to be increasing. They explained some of the variability of prevalence from year to year possibly being due to changes in weather leading to variation in gastropod abundance and distribution and altered feeding behavior produced by annual variations in natural forage availability.

Anderson (1972) speculated that the eastern subspecies of elk which is now extinct may have been immune to *P. tenuis* or even tolerated the parasite as well as the white-tailed deer. Other possibilities are that deer and elk coexisted

on the same range because habitat preferences and seasonal migration patterns reduced the potential for exposure. All these studies indicate that habitat manipulations and control of population density might be useful measures for management of this problem with moose and elk. Treatments utilizing a palatable oral anthelmintic with a wide margin of safety effective against *P. tenuis* in the source of the problem, white-tailed deer, could also have an important role in modern wildlife management as well as management for exotic and domestic animals.

## Albendazole as a Potential Management Tool

Albendazole is a newly developed benzimidazol anthelmintic. It was chosen as a suitable drug to test against meningeal worm because of its known safety and efficacy against a broad spectrum of other parasites, such as lungworms and liver flukes as well as intestinal parasites, in white-tailed deer (Foreyt and Drawe, 1978). In abnormal hosts, *P. tenuis* larvae penetrate the gray matter in the dorsal horn of the spinal cord and can cause much more tissue damage and inflammation than in their normal host. This phenomenon is generally true of migrating nematodes in abnormal hosts. *Parelaphostrongylus tenuis* may also invade the brain as subadults or adults, causing the classical neurological disease already described. Brain tissue itself is selectively protected from circulating substances by the blood-brain barrier, so it was felt that systemic treatment in abnormal hosts where the parasite is often in the

parenchyma of the spinal cord or brain might be less successful than in white-tailed deer, where the adult is associated with the venous sinuses in the subdural space.

Diethylcarbamazine citrate, levamisole phosphate, and thiabendazole have been used to treat cerebrospinal parelaphostrongylosis in goats (Mayhew et al., 1976). However, no conclusions concerning efficacy of these drugs could be drawn from these uncontrolled treatments because they were isolated clinical cases which may have recovered after running their natural course. It was also felt that an attempt at management by treating the source of the problem in the white-tailed deer would have more merit than treating the clinically affected abnormal host. Successful treatment would be hard to evaluate clinically, as nerve and brain damage heal slowly, if at all. Also, the parasite does not usually complete its life cycle in the abnormal host, so larval output in the feces could not be monitored as an experimental parameter.

There are some references in the literature on the presence of *P. tenuis* larvae in the feces of abnormal hosts. Loken et al. (1965) demonstrated what they thought were meningeal worm larvae in feces from an infected sick moose. Karns (1966) demonstrated larvae in what he thought was elk feces. Karns and Jordan (1969) found a low incidence of larvae identical to *P. tenuis* in moose on Isle Royal and there had been no deer present for 30 years, so they felt the parasite was completing its life cycle in moose. Tompkins et al. (1977) found larvae that looked like *P. tenuis* in

what they thought were elk feces in Michigan but felt that they could not say for sure they were *P. tenuis*. Larvae from *P. andersoni*, *P. odocoilei*, and *Elaphostrongylus cervi* all look like *P. tenuis* and they could not rule out the existence of these other parasites in deer or elk in Michigan.

There is some question as to whether *P. tenuis* does complete its life cycle in abnormal hosts in the wild and no doubt that abnormal hosts are not an important reservoir of the parasite. Anderson (1966) experimentally infected an elk which shed *P. tenuis* larvae in its feces 92 days after inoculation, which is the approximate prepatency period in the normal host. The elk showed some transient neurological symptoms but grew and developed normally. After reviewing the literature and evidence, it appears that under ideal conditions a low number of parasites (at least one male and one female) could complete their life cycle in the abnormal host. Typically, the parasite does not reach patency in any host but white-tailed deer because the inflammatory changes caused by the migrating parasite kill either the host or the parasite before the life cycle can be completed. It seems that a great potential exists for management of this problem if *P. tenuis* could be controlled in white-tailed deer, thus eliminating or decreasing the risk of exposure to abnormal susceptible hosts.

## MATERIALS AND METHODS

A noninfected white-tailed deer buck weighing approximately 80 kg was given 8 gm of albendazole by esophageal intubation. This high dose of 100 mg/kg caused transient hemorrhagic diarrhea from 24-48 hours after treatment, but no other obvious problems. Histologic sections of many organs and several sites along the digestive tract appeared normal when the deer was sacrificed one week after treatment. An experimentally infected pregnant doe near term was dosed with 6,370 mg albendazole (100 mg/kg). Again, there was some transient hemorrhagic diarrhea the day following treatment, but no obvious decrease in larval output. The deer died during the second week after treatment from a clostridial infection at the tranquilizer dart injection site. Its fetus apparently died at the same time as the doe.

Since a single high dose apparently failed, it was felt that a lower dose over a longer period of time would determine if the drug was effective. A dose of 25 mg/kg twice a day for 5 days was suggested by Smith Kline representatives, but to be sure the drug was given a chance, a 2-week treatment was decided on for the main research project. Snails (*Triodopsis multilineata*) artificially infected with first stage meningeal worm larvae which had been obtained from

deer at the Rachelwood Wildlife Refuge in Pennsylvania were used to infect the deer. Snails were crushed and digested for 4 hours in a Baermann apparatus at 37°C in a solution of 1 gm pepsin, 1.4 ml HCl, and 166 ml of distilled $H_2O$. The planned protocol was to inoculate each of four 6-month-old white-tailed deer fawns in 2 trials with 100 larvae. However, only the healthiest and most active half of the snails were digested for trial I, yielding only enough larvae to give 62 third stage infective larvae to each deer. One month later, the rest of the snails, which had been maintained in a terrarium with water, moistened dog food, lettuce and chalk, were digested. At least 3,000 larvae were recovered and 5 available fawns were each inoculated orally with 100 infective larvae. Apparently, decreased activity and unthriftiness of the snail might be an indication of greater parasite burden, although it is possible some larvae in the first group of snails had not yet reached the $L_3$ stage and were killed by the digesting solution.

Onset of larval production in the deer was monitored by weekly fecal examinations for the second half of the approximately 90-day prepatency period for *P. tenuis*. A standardized technique was used with 2 gm of fresh feces suspended on a single layer of tissue paper in 90 ml of water in a Baermann funnel for 18 hours at room temperature. Fifteen milliliters were drawn off, resuspended and placed in a gridded petri dish 88 mm in diameter for counting under a dissecting microscope. Variations of volumes and times were tried and this method allowed nearly all larvae to settle out for easy

counting. Lankester and Anderson (1968) found that 83% of larvae left a submerged fecal pellet within the first 5 minutes of soaking. An inside and outside set of 4 grids (one from each quadrant) was counted for each examination after vibration to randomize larvae.

Treatment of the deer was carried out by including the dose of albendazole with double its volume of ViNatura$^{R}$ (a natural honey apple flavored equine vitamin syrup made by Jensen-Salsbery Laboratories in Kansas City, Missouri) mixed with their normal diet of exotic ruminant pellets made by The Andersons of Maumee, Ohio. Free choice feed consumption was determined for each deer prior to treatment by averaging consumption over an 8- to 10-day period. Each deer was fed twice a day with 45% of its daily *ad libitum* consumption of pellets with the ViNatura for 1 week prior to treatment. This was to get them accustomed to the ViNatura. It was also an attempt to equilibrate fecal output and thus larval counts to a standard because decreased feed intake would lower fecal output and appear to increase larval output per gram of feces. Daily larval counts were determined before, during, and after the 2-week treatment (Appendix A). Uneaten feed was removed and weighed before the morning feeding each day (Appendix B). At the end of each trial deer were euthanatized with 20 mg of succinylcholine chloride. The brain and cranial cavity were examined and worms counted. A 10 gm sample of the right apical lobe of the lung was macerated and examined for larvae by the same Baermann method used on the feces.

# RESULTS

## Trial I

One of the deer in trial I died from overexertion due to harrassment by dogs near the pens. Of the remaining 3, 2 were treated and 1 was used as a control. The control, receiving food with ViNatura only, ate everything regularly, but 1 treated deer rejected feed completely after eating most of the first dose. This deer (No. 2) was offered the treated food for 1 week without consuming any, so it was returned to feed without albendazole for the rest of the study and immediately resumed eating, as seen in Appendix B. Larval output was followed during this period, even though the data could not be included with those from other treated deer. Counts went from less than 10,000 larvae/gm of feces before treatment to over 56,000 larvae/gm, then down to zero by the end of the second week. The deer had begun shedding a few larvae by the end of the week after treatment when all the animals were sacrificed. The other treated deer ate regularly, but almost never ate all of the drugged feed. Larval counts went to zero and remained there until the end of trial I. Average weekly larval counts for treatment and control animals were calculated and are shown for both trials in Table 1. On postmortem examination (data shown in

Table 1. Larval output in feces of white-tailed deer infected with meningeal worm

| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Trial I. Larvae per gm of feces** | | | | | | | | | | | | | |
| Control | 0 | 0 | 0 | 0 | 0 | 1921 | 1129 | 2575 | 2330 | 1671 | 1199 | 1584 | |
| Treated | 0 | 2 | 2 | 1 | 169 | 927 | 1214 | 4155 | 2542 | 191 | 8 | 0 | |
| Wk post-infection | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | |
| | | | | | | | | | treatment period | | | | |
| **Trial II. Larvae per gm of feces** | | | | | | | | | | | | | |
| Control | ? | 1298 | 742 | 1088 | 916 | 1145 | 12907 | 742 | 34 | 169 | | | |
| Treated | ? | 658 | 767 | 2847 | 103 | 0 | 0 | 0 | 0 | 0 | | | |
| Wk post-infection | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | | | |
| | | | | treatment period | | | | | | | | | |

Figures were calculated by taking the daily counts and averaging for each week, then averaging counts for all control and treated animals for each trial.

Appendix C), live adult *P. tenuis* were recovered from all 3 deer, although there were some dead encapsulated worms in the brain of the deer which accepted treatment. Baermann examinations of lung tissue revealed high numbers of larvae in the control, a few in the treated animal which rejected the drug, and 1 dead decomposing larva from the other treated deer.

Trial II

It was decided to keep the deer in trial II alive for at least a month after the end of treatment to be sure the drug was killing the adults in the brain and not just the peripheral larvae. There were 2 deer treated and 2 used as controls in trial II because 1 deer had to be euthanatized with an apparent clinical case of meningeal worm. One of the controls had a very low larval count, while the one that was euthanatized was not shedding any larvae, even though there were many adult worms found in the meninges on post-mortem examination. Again, the albendazole seemed to lower feed consumption, as shown in Appendix B, but both treated deer ate regularly. It was hard to attribute the anorexia to the drug exclusively because other variables, such as hot muggy weather, human activities near the pens, inclusion of the ViNatura and even the effect of the parasite seemed to affect feed consumption of both treatment and control animals. Table 1 shows that although deer in trial II received a greater number of parasites, the average larval output was lower. Larval output dropped to zero during the

2-week treatment for both treated deer and larval output did not resume before euthanasia 1 month later. At the time of necropsy, larval counts were low in both control deer, but counts fluctuated daily from over 12,000 to less than 100 larvae/gm of feces in the animal with the higher output. At necropsy (Appendix C), the brains of both control deer contained many live adult *P. tenuis* and the meninges were very hemorrhagic and necrotic (Figure 1). A mass of live worms about 1 cm in diameter and 3 cm in length was found in the dorsal median sulcus between the cerebral hemispheres of the deer which showed fluctuating larval counts. This clumping was also found by Prestwood (1970) in a white-tailed deer with neurological disease. She suggested that the neurological symptoms shown by this deer were caused by the large masses of parasites and resulting circulatory disturbances. Similar lesions in the deer in this study might help explain the varying feed consumption as well as the erratic larval counts because larvae get to the lungs via the venous blood supply, which was obviously compromised.

No live worms were found in either of the treated deer. Extensive dissection and examination showed only some dead, well encapsulated worms on the meninges. There was no gross evidence of the severe inflammation and hemorrhage seen in the controls. The meninges appeared glistening white and healthy (Figure 2). The damage caused to the meninges by the parasites as well as the inflammation associated with the dead, decomposing adult worms had apparently healed by 1 month post-treatment.

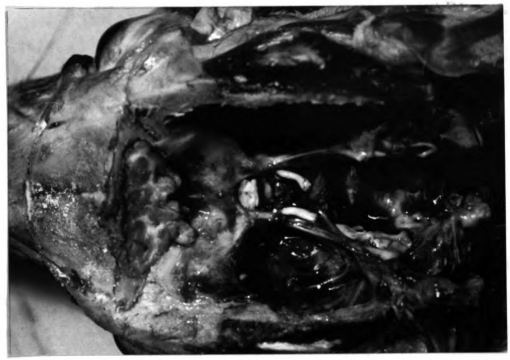

Figure 1. Cranium of control white-tailed deer. Note the inflammation and hemorrhagic meningitis.

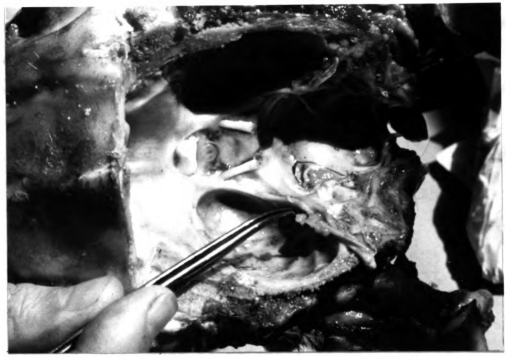

Figure 2. Cranium of white-tailed deer 1 month after treatment with albendazole. Note the glistening healthy white meninges and the dead encapsulated parasite at the tip of the forceps.

## DISCUSSION

Although this research was done on a rather small number of animals and was hampered by the nervous, unpredictable nature of the wild deer, albendazole was shown to be effective against *P. tenuis* in white-tailed deer. Additional research determining optimum effective dosage might show efficacy at a lower dosage or shorter treatment period. A lower dosage would make drugged feed more palatable, limit potential side effects and make widespread group treatment a feasible management tool for a herd or yard of deer. Initial research on optimum drug dosage and feasibility of group treatment with consideration of some of the drug's known potential side effects, such as teratogenicity or abortions, should be done on a captive group of deer. Further field studies might be conducted on a group of wild deer confined to their winter yard by snow.

In this study, deer averaging 35 kg body weight consumed approximately 1.25 kg of untreated pellets daily. For group treatment, these data would suggest a pelleted feed with 1.4 gm of albendazole per kg of feed to give an approximate daily dose of 50 mg/kg of albendazole to each deer. Incorporating the drug in a palatable pelleted feed offered free choice would be a practical method of treatment

with larger deer receiving a larger dose by eating a greater volume.

Another method of treatment might be to include the drug in ensiled apple pomace as was done by Colorado researchers (Schmidt et al., 1978) with bighorn sheep. Their research showed feasibility and success of using anthelmintic drug treatment in management of wild populations. In their study, a single dose of 8.4 gm of Cambendazole was incorporated in as little as 1.30 kg of apple pomace to give the drug at approximately 125 mg/kg body weight to treat *Protostrongylus* spp. of lungworms in bighorn sheep. This single dose treatment could allow a dominant animal or an aggressive eater to overdose while depriving subordinate animals of adequate dosage.

The suggested 2-week treatment with albendazole in a pellet designed to be fed free choice would allow most deer to get adequate dosages while aggressive eaters might experience minimal side effects. Data from this research suggest that a lower dose for a shorter period of time might be as effective. The dramatic drop in larval output in deer No. 2 of trial I after only 1 treatment indicates that a shorter treatment period might be effective. The rejection of drugged feed by other successfully treated deer definitely shows that daily doses lower than 50 mg/kg would be efficacious. Also incorporating a certain percentage of the anthelmintic directly into the pelleted ration as it was manufactured would offer several advantages. It would allow known dosages by measuring consumption by individuals, and

premixed feed would be more palatable because the taste would be diluted out and the drug would be less detectable than on the outside of the pellets as in this study. An added benefit of treatment of white-tailed deer with obvious management implications is the already proven (Foreyt and Drawe, 1978) effectiveness of albendazole against lungworms, liver flukes, and intestinal nematodes as well as meningeal worms. Another asset of albendazole is its acceptance for use against liver flukes in food producing animals by USDA and FDA. Although its approval for use in sheep and cattle does not make its use in wildlife "legal", it makes special approval of its use by wildlife managers much more likely. The withdrawal time for albendazole in sheep in Australia is 10 days (Prichard, 1978). Because of the experimental nature of the drug in the U.S., the cautious required withholding period for cattle and sheep is 180 days (FDA Memo, 1980). Even this would permit treatment of wild populations in the winter and allow a longer withdrawal time before legal hunting, thus minimizing the chance of this drug getting into the human food chain.

Modern pressures of habitat encroachment and overpopulation of remaining habitat with competition, both within and between species, facilitate disease spread. This concept is considered for moose by Franzmann (1978) in a recent management text, while the same concept with consideration of anthelmintic treatment as an important management tool is discussed by Wishart (1978) in the chapter on bighorn sheep. Moran (1973) felt that if the *P. tenuis* reservoir could be

reduced, this might help elk increase their numbers despite deteriorating habitat quality. George et al. (1974) felt that *P. tenuis* was the most important factor in the decline of Pennsylvania elk and much more research should be aimed at solving the disease problem if elk were to be saved in the east.

Successful drug treatment of *P. tenuis* in white-tailed deer with albendazole raises hopes for management of this parasite in cervids such as moose and elk in the north-central range of the white-tailed deer. It also has potential for management of cerebral parelaphostrongylosis in other abnormal exotic and domestic hosts as well as diseases caused by other metastrongyle nematodes. A parasite which might have similar potential for management with albendazole is the arterial worm (*Elaeophora schneideri*). This parasite is carried by both mule deer and white-tailed deer and was shown by Hibler and Adcock (1971) to cause a very serious neurological disease in native elk. Recently this parasite was shown to cause disease in exotic Sika deer in Texas (Robinson et al., 1978). Another parasite which might be managed is the Eurasian caribou parasite (*Elaphostrongylus cervi*), which was recently identified in North America (Lankester and Northcott, 1979), causing a disease similar to *P. tenuis* in caribou. The danger of this disease and the importance of preventing its spread were shown earlier by Lankester (1977) when an experimental infection was able to complete its life cycle in moose while causing serious neurological disease.

It is evident that much more research is needed to define precise treatment of *P. tenuis* in white-tailed deer with albendazole and to evaluate this and other drugs for their potentail in managing other parasites in other species. It is encouraging to note that a broad-spectrum anthelmintic with a wide margin of safety like albendazole could have a very important role in modern wildlife management.

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# APPENDICES

# APPENDIX A

Table A1. Consecutive daily counts of larvae per gram of feces in deer on albendazole study

| | Trial I | | | Trial II | | | |
|---|---|---|---|---|---|---|---|
| | Control | Treated | | Control | | Treated | |
| | No 1 | No 2 | No 3 | No 1 | No 2 | No 3 | No 4 |
| Pre-treatment | 2006 | 8323 | 4869 | 792 | 51 | 1432 | 152 |
| | 3033 | 9233 | 3976 | 741 | 17 | 1399 | 152 |
| | 1887 | 8644 | 4263 | 994 | " | 910 | 286 |
| 2 week treatment period | 2713 | 17439 | 2561 | 1685 | " | 4987 | 202 |
| | 2460 | 28492 | 4111 | 2612 | " | 1095 | 1297 |
| | 3100 | 55737 | 2123 | 4078 | 51 | 9065 | 388 |
| | 2932 | 56225 | 2140 | 4482 | 0 | 4617 | 169 |
| | 1921 | 37944 | 1938 | 691 | 0 | 8913 | 1668 |
| | 1297 | 25813 | 657 | 640 | 0 | 5543 | 708 |
| | 1735 | 10935 | 320 | 741 | 0 | 910 | 67 |
| | 2190 | 1449 | 202 | 944 | 0 | 0 | 17 |
| | 1078 | 202 | 506 | 725 | 0 | 17 | 84 |
| | 2831 | 51 | 169 | 1786 | 17 | 219 | 0 |
| | 1095 | 0 | 84 | 707 | 0 | 67 | 0 |
| | 1702 | 0 | 34 | 1348 | 0 | 34 | 0 |
| | 1062 | 0 | 17 | 6554 | 0 | 17 | 0 |
| | 1382 | 0 | 34 | 2999 | 0 | 0 | 0 |
| Post-treatment | 1264 | 0 | 0 | 1786 | 0 | 0 | 0 |
| | 960 | 0 | 17 | 3100 | 0 | 0 | 0 |
| | 506 | 0 | 0 | 5661 | 84 | 0 | 0 |

Table A1 (continued)

| | Trial I | | | Trial II | | | |
|---|---|---|---|---|---|---|---|
| | Control | Treated | | Control | | Treated | |
| | No 1 | No 2 | No 3 | No 1 | No 2 | No 3 | No 4 |
| | 1025 | 0 | 0 | 775 | 0 | 0 | 0 |
| | 2477 | 17 | 0 | 927 | 17 | 0 | 0 |
| | 776 | 0 | 0 | 674 | 0 | 0 | 0 |
| Post-mortem | 1584 | 0 | 0 | Returned to free choice feed for 3 weeks | | | |

# APPENDIX B

Table B1. Uneaten feed in kg when fed 90% of free choice consumption in 2 daily feedings

| | Trial I | | | Trial II | | | |
|---|---|---|---|---|---|---|---|
| Feed Offered | 1.0 No 1 | 1.0 No 2 | 1.4 No 3 | 1.0 No 1 | 1.1 No 2 | 0.9 No 3 | 1.3 No 4 |
| Pre-treatment | 0 | 0 | 0 | .33 | .28 | .09 | .45 |
| | 0 | 0 | 0 | .09 | .13 | .00 | .00 |
| | 0 | 0 | 0 | .25 | .05 | .06 | .00 |
| 2 week treatment period | 0 | .7 | 0 | .59 | .25 | .17 | .77 |
| | 0 | .5 | 0 | .55 | .25 | .80 | .77 |
| | 0 | 1.07 | .8 | .23 | .39 | .93 | .72 |
| | 0 | 1.05 | .24 | .11 | .29 | .95 | .08 |
| | 0 | 1.06 | .14 | .07 | .07 | .75 | .92 |
| | 0 | 1.065 | .27 | .06 | .19 | .26 | .41 |
| | 0 | 1.055 stopped drug | .28 | .00 | .12 | .17 | .24 |
| | 0 | .245 | .15 | .11 | .14 | .42 | .40 |
| | 0 | .14 | .27 | .11 | .19 | .78 | .79 |
| | 0 | 0 | .22 | .49 | .08 | .51 | .21 |
| | 0 | 0 | .03 | .26 | .04 | .36 | .38 |
| | .1 | 0 | .30 | .26 | .13 | .19 | .55 |
| | 0 | 0 | .13 | .26 | .07 | .24 | .34 |
| | 0 | 0 | .165 | .26 | .17 | .15 | .45 |
| Post-treatment | 0 | 0 | .165 | .24 | .05 | .25 | .40 |
| | 0 | 0 | 0 | .08 | .02 | .07 | .07 |
| | 0 | 0 | 0 | .09 | .04 | .00 | .00 |

Appendix B1 (continued)

| Trial I | | | Trial II | | | |
|---|---|---|---|---|---|---|
| 1.0 No 1 | 1.0 No 2 | 1.4 No 3 | 1.0 No 1 | 1.1 No 2 | 0.9 No 3 | 1.3 No 4 |
| 0 | 0 | 0 | .03 | .02 | .00 | .00 |
| 0 | 0 | 0 | .01 | .01 | .00 | .00 |
| 0 | 0 | 0 | .00 | .08 | .00 | .00 |
| Euthanasia | | | Returned to free choice feed for 3 weeks | | | |

# APPENDIX C

Table C1. Vital statistics of deer in albendazole study

| | Trial I | | | Trial II | | | |
|---|---|---|---|---|---|---|---|
| | No 1 | No 2 | No 3 | No 1 | No 2 | No 3 | No 4 |
| Estimated pre-treatment body weight, kg | 40 | 30 | 30 | 30 | 30 | 30 | 30 |
| Daily free choice feed consumption, kg | 1.1 | 1.2 | 1.6 | 1.1 | 1.2 | 1.0 | 1.4 |
| Daily Treatment | | | | | | | |
| 90% feed, kg | 1.0 | 1.0 | 1.4 | 1.0 | 1.1 | 0.9 | 1.3 |
| ViNatura, cc | 40 | 30 | 30 | 30 | 30 | 30 | 40 |
| Albendazole, mg | 0 | 1500 | 1500 | 0 | 0 | 1500 | 2000 |
| Postmortem | | | | | | | |
| Body weight, kg | 35 | 26 | 32 | 31 | 32 | 31 | 45 |
| Larvae/gm feces | 1584 | 0 | 0 | 169 | 0 | 0 | 0 |
| Larvae from lungs | 100's | 3 | 1 dead | 2 | 0 | 0 | 0 |
| Meningitis | yes | yes | yes | yes | yes | no | no |
| No. of *P. tenuis* in meninges | -10 alive | 12 alive | 8 4 live | -70 alive | -25 alive | 5 dead | 6 dead |
| Condition of *P. tenuis* | dis-persed | dis-persed | 4 dead | clumped | dis-persed | en-capsulated | |