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TREATMENT AND CONTROL OF *Dictyocaulus viviparus* IN CAPTIVE BLACK-TAILED DEER*

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Abstract: An outbreak of dictyocauliasis among a captive herd of black-tailed deer fawns *(Odocoileus hemionus columbianus)* occurred in January, 1971. A transient decrease in output of *Dictyocaulus viviparus* larvae in feces occurred after treatment with levamisole hydrochloride given as a drench at the rate of 16 mg/kg of body weight.

Lungworm larvae were not recovered in feces 6 days after cambendazole, 2-(4-thiazolyl)-5-isopropoxycarbonylaminobenzimidazole, was given as a drench at 40 and 50 mg/kg of body weight. Larvae were again recovered in feces from these fawns between post-treatment days 15 and 23. Output of larvae in feces increased when fawns were confined on a contaminated grass pasture that was intensively grazed. Deteriorating physical condition of the fawns necessitated additional treatment with cambendazole and movement to a woodlot where reinfection by ingestion of larvae was probably minimized.

A noninfected deer was placed on the contaminated pasture 75 days after the infected herd was removed. After 55 days, lungworm larvae were recovered in feces from this deer. Then 29 days later, 20 fawns were placed on this pasture. Four of six of these fawns that were subsequently necropsied harbored light burdens of *D. viviparus.* Small numbers of lungworm larvae were recovered in feces from five of eight remaining fawns.

INTRODUCTION

Although the prevalence of *Dictyocaulus viviparus* has been reported in black-tailed deer of British Columbia,[4] California,[6,16,17] Oregon,[15] and Washington,[8] there are evidently no reports on its treatment or control in this host.

An outbreak of dictyocauliasis among a captive herd of black-tailed deer gave an opportunity to: 1) study clinical effects of infection; 2) test two anthelmintics against *D. viviparus;* 3) study survival and infectivity of larvae on contaminated pasture. Most of these deer were also being used in nutrition experiments conducted by personnel in the Department of Animal Science and were not available for necropsy.

MATERIALS AND METHODS

History of the animals

Eight black-tailed deer fawns (no. 1-8) raised in 1970 by Oregon State Game Commission personnel were moved to a 0.74 - hectare enclosure[2] (Fig. 1) on October 5, 1970 (experimental day 0) (Table 1). Gates were located between adjacent pens to permit free movement of

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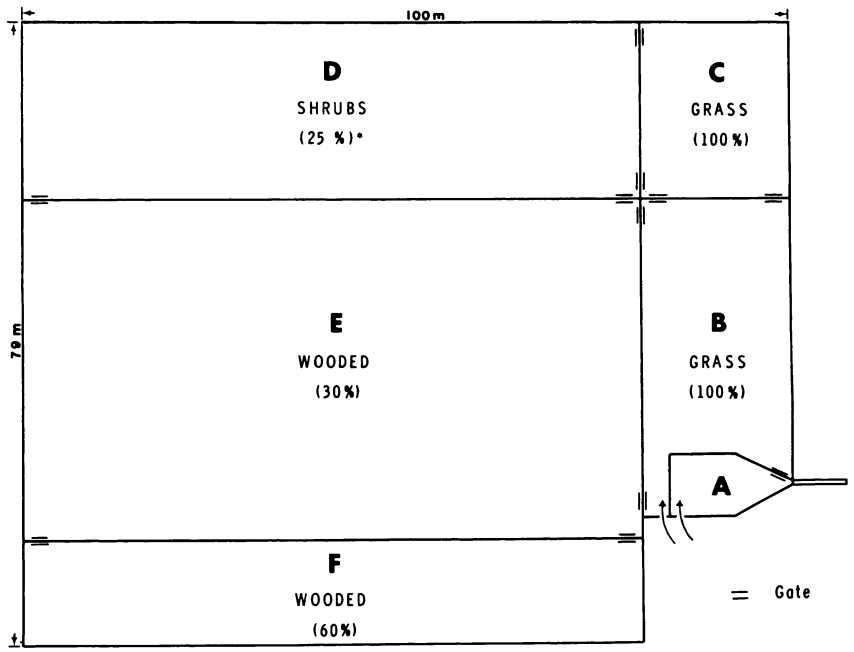

FIG. 1. Diagram of the enclosure at the E. E. Wilson Game Management area drawn to scale. Area A is a corral with a loading chute. Primary cover types are given for pens B, C, D, E, and F.

deer between pens or confinement in specific areas. A corral (A) with wooden walls (3 m high) and sawdust ground cover was used for capture and treatment of fawns. Ground cover in two pens (B and C) consisted principally of meadow fescue *(Festuca elatior)*, perennial ryegrass *(Lolium perenne)*, and velvet grass *(Holcus lanatus)*. The vegetation in Pen D consisted almost entirely of blackberries *(Rubus procercus)*. Pens E and F were woodlots of several introduced species including alder *(Alnus tenuifolia)*, apple *(Malus domestica)*, cherry *(Prunus avium)*, and maple *(Acer macrophyllum)* trees; small amounts of wild lettuce *(Lactuca* sp.), vetch *(Vica* sp.), plantain *(Plantago* sp.), and lotus *(Lotus* sp.) were also found. Water tanks and feed bunkers were located in pen E and the corral.

Fawns had access to the five fenced pens within the enclosure. Supplemental commercial feed given to the fawns was gradually reduced until their only feed was that available in the enclosure. On experimental day 30 (Table 1), a male fawn[3] (9) in poor physical condition was released in the enclosure with the resident fawns (1-8). Weight loss and rough hair coat were evident in five resident fawns by experimental day 66. At this time, a pelleted concentrate ration was given to supplement available forage; alfalfa hay was provided *ad libitum*.

Necropsy of fawn 1 that died on experimental day 91 (Jan. 4, 1971) revealed a heavy *D. viviparus* infection (Table 1). On day 95, fecal samples were collected

[3] This fawn was brought to the Game Commission by a private person and released into the enclosure with the other fawns. The origin of this fawn was not learned.

from fawns 2-9 and *D. viviparus* first-stage larvae (L_1) were recovered in feces from seven of these deer. The McMaster method was used to examine these samples for gastrointestinal nematode eggs, but none was found.

Treatment

Fawns were captured by hand, rope, or Cap-Chur gun.[4] Levamisole hydrochloride[5] was given to each fawn on one to three occasions between experimental days 95 and 114 (Table 1). Dosage rates of 7 and 16 mg/kg of body weight were used. Cambendazole[6] at the rate of 40 mg/kg of body weight was given to each fawn on experimental days 128 and 136 (Table 1). Cambendazole at 50 mg/kg was given to four fawns (3, 4, 6, and 9) on experimental days 193; fawns 7, 8, and 9 were similarly treated on day 246

TABLE 1. Chronology of black-tailed deer movements and treatments.

Experimental day	Deer no.(s)	Item
0 (Oct. 5, 1970)	resident fawns 1-8	placed in the enclosure
30	fawn 9	placed in the enclosure
91	fawn 1	died; heavy *D. viviparus* infection
95	fawns 2-9 sampled	larvae in feces of seven fawns
95	fawns 2, 4, 6	given levamisole at 7 mg/kg
100	fawns 2-9	given levamisole at 7 mg/kg
114	fawns 6, 7, 9	given levamisole at 16 mg/kg
128	fawns 3, 4, 5, 6, 7, 9	given cambendazole at 40 mg/kg
136	fawns 2, 8	given cambendazole at 40 mg/kg
174	fawns 2-9	confined on pasture (pens B and C)
193	fawns 3, 4, 6, 9	given cambendazole at 50 mg/kg
200	fawn 5	given cambendazole at 50 mg/kg
202	fawns 2-9	moved to woodlots (pens E and F)
246	fawns 7, 8, 9	given cambendazole at 50 mg/kg
277	yearling deer	placed in pen B
332	yearling deer	lungworm larvae in feces
361	twenty fawns	placed in pens E and C
401	six of these fawns	necropsied; four had *D. viviparus*
to 463 (Feb. 10, 1972)	eight fawns sampled	larvae in feces of five fawns

[4] A combination of phencyclidine hydrochloride (100 mg/cc; Sernylan, Bio-Ceutic Laboratories, Inc., St. Joseph, Mo.) and triflupromazine hydrochloride (20 mg/cc; Vesperin, E.R. Squibb & Sons, Inc., New York, New York) was used in the projectile syringes. Cap-Chur equipment is marketed by the Palmer Chemical & Equipment Co., Inc., Douglasville, Georgia.

[5] Tramisol, American Cyanamid Company, Princeton, N.J.

[6] Supplied by Merck & Company, Rahway, N.J.

(Table 1). These anthelmintics were administered in drench form with a commercial drench gun.[7]

Efficacy of treatment was evaluated by quantitative estimate of *D. viviparus* L_1 output using the Baermann technique and 6 to 30 g of freshly dropped feces. Each sample was wrapped in cheesecloth and placed in lukewarm tapwater in a 250 ml plastic funnel equipped with a short rubber hose and pinch clamp. After 18 to 24 hours at room temperature, approximately 25 ml of fluid from the bottom was examined for lungworm larvae. Output of *D. viviparus* L_1 was expressed as larvae per gram (LPG) of feces.

Survival and infectivity of D. viviparus larvae on contaminated pasture

Fawns 2-9 were confined on grass pasture (pens B and C) from experimental day 174 (March 28) until day 202 (April 25) and then moved to adjacent woodlots (pens E and F) (Table 1).

A noninfected yearling was placed in pen B on experimental day 277 (July 9), 75 days after the infected herd was removed from the pasture. At periodic intervals, fecal samples were collected from this deer and examined for lungworm larvae.

On experimental day 361, 20 fawns were placed in pens B and C (Table 1). Physical condition of these fawns was observed and fecal samples were collected at periodic intervals. Between experimental days 401 (Dec. 10, 1971) and 463 (Feb. 10, 1972), six fawns were killed and necropsied; their lungs were examined for *D. viviparus*.

RESULTS

The summary given below is based on LPG counts for 221 fecal samples collected from the deer during this investigation.

Efficacy of anthelmintics given to fawns 2-9

Output of *D. viviparus* L_1 in feces from fawn 9 (placed in the enclosure on experimental day 30) was approximately 1 LPG between experimental days 95 and 181. Mean output for resident fawns 2-8 was 168 LPG (minimum and maximum, 2 and 512) on experimental day 95. Output of lungworm larvae in feces did not vary after fawns 2-9 were treated with levamisole at 7 mg/kg. When fawns 6, 7, and 9 were given levamisole at 16 mg/kg (Table 1), larvae were not recovered in their feces on post-treatment day 6. However, their LPG counts had returned to pretreatment levels 13 days after the drug was given.

When cambendazole was given to five resident fawns (3, 4, 5, 6, and 7) on experimental day 128 (Table 1), their mean output was 275 LPG (minimum and maximum, 14 and 593). Lungworm LPG counts decreased within 48 hours and on post-treatment day 6, larvae were not recovered in feces from treated fawns. Fifteen to 22 days after cambendazole was given, larvae were again recovered in feces from fawns 3, 4, 5, 6, and 7. Fawn 8 given cambendazole on experimental day 136 (Table 1) responded similarly to treatment.

Fawn 2 apparently developed resistance to patent *D. viviparus* infection after single treatment with cambendazole on experimental day 136. Output of larvae in feces began to increase on day 154 (post-treatment day 18), but then declined. After experimental day 161, larvae were not recovered in feces from fawn 2.

Mean output of lungworm larvae in feces from the other fawns (3, 4, 5, 6, 7, 8) increased during time of confinement on grass pasture (28 days, beginning experimental day 174) when serious overgrazing occurred. Physical condition of fawns deteriorated and coughing increased. Mean output for fawns 3, 4, 6, 7, and 8 was 22 LPG (minimum and maximum, 1 and 69) on experimental day 151; it increased to 351 LPG (minimum and maximum, 33 and 1,137) on day 190. Even fawn 9, whose fecal output of larvae had previously been low, increased from 0 LPG on experimental day 181 to 256 LPG on day 190. Larvae of *D. vivi-*

[7] Cooper 20 cc Automatic Drencher Mark II, manufactured by N. J. Phillips Pty. Ltd., Australia, for William Cooper & Nephews, Inc., Chicago, Ill.

parus were also recovered from pasture samples collected in pen B.

Consequently, on experimental day 193 fawns 3, 4, 6, and 9 were treated with cambendazole at 50 mg/kg (Table 1). Lungworm larvae were not recovered in feces from these fawns 6 days after treatment. On experimental day 200, clinical signs in fawn 5 were hyperpnea (80/min) and coughing. This fawn was captured and given cambendazole at 50 mg/kg. Output of larvae in feces was 1,300 LPG at time of treatment, 300 LPG 36 hours post-treatment, and 0 LPG 6 days after the drug was given. Respiratory difficulties terminated within 36 hours after treatment.

On experimental day 202 (April 25), fawns 2-9 were moved to adjacent woodlots (pens E and F) including two nontreated fawns (7 and 8) (Table 1). In these pens fawns browsed extensively on new leaf growth and ingestion of lungworm larvae was probably minimized.

Fifteen to 23 days after cambendazole was given on experimental days 193 and 200, lungworm larvae were again recovered in feces from only three (3, 5, 9) of the five treated fawns. Patent infections in fawns 3 and 5 terminated naturally approximately 45 days later. However, output of larvae in feces from fawn 9 increased to 93 LPG on experimental day 238.

On experimental day 246, fawn 9 and the nontreated controls (7, 8) were treated with cambendazole at 50 mg/kg (Table 1). Mean output for fawns 7 and 8 was approximately 90 LPG (minimum and maximum, 33 and 188) between experimental days 190 and 246. After post-treatment day 8, larvae were not recovered in feces from fawns 7, 8, and 9.

Survival and infectivity of D. viviparus larvae on pasture

After infected fawns were removed from the grass pasture (pens B and C) on experimental day 202 (April 25), the grass grew rapidly and was approximately 1 m tall before hot, dry weather began in July and August. The yearling placed on the contaminated pasture (pen B) on experimental day 277 (July 9) acquired a light patent infection during August. Output of larvae in feces was 1 LPG on experimental day 332; this deer was observed coughing 3 days later. Of 20 fawns placed on the contaminated pasture (pens B and C) on experimental day 361 (Oct. 1), necropsy of six of these (between experimental days 401 and 463) revealed burdens of 0, 0, 6, 16, 19, and 21 mature *D. viviparus* (Table 1). For five of eight remaining fawns sampled, mean output of larvae was 12 LPG (minimum and maximum, 2 and 31).

DISCUSSION

Fecal LPG counts for fawns 2-8 indicated they probably harbored moderate to heavy burdens of *D. viviparus*. Deteriorating physical condition, hyperpnea, and coughing were signs of clinical dictyocauliasis. Poor physical condition of fawns and adverse weather conditions before time of confinement on pasture (experimental days 174 to 202) probably contributed to the severity of effects from these infections.

Output of larvae in post-treatment fecal samples indicated that efficacy of levamisole given at 7 and 16 mg/kg was probably low against mature lungworms in these fawns. Since LPG counts returned to pretreatment levels within 13 days, the effect of treatment probably was temporary suppression of larvae production by female worms. High efficacy (98 to 100%) against mature *D. viviparus* in cattle has been reported when levamisole was given orally as a drench at 7.5 to 12.0 mg/kg of body weight.[1,5,7]

Reduced LPG counts 2 to 15 days after cambendazole was given, followed by a gradual increase beginning 15 to 23 days post-treatment suggested two possible modes of action, either that this anthelmintic #1 eliminated mature *D. viviparus*, but immature stages were not affected and matured subsequently, or #2 temporarily suppressed production of larvae by female worms. Efficacy greater than 93% was found when cambendazole was evaluated against mature *D.viviparus* in cattle with experimentally induced[12] or naturally acquired[2] infections. However, Baker *et al.*[2] reported that only 52 to

82% of immature *D. viviparus* was eliminated when cambendazole at 20 and 25 mg/kg of body weight was used. These data, and the fact that the prepatent period for *D. viviparus* is approximately 25 days in cattle[14] and 30 days in black-tailed deer,[11] suggest that the first hypothesis, #1, is probably the correct interpretation for the effect of cambendazole in these fawns. That output of larvae remained zero for at least 15 days and then the increase in LPG counts was gradual, suggests that the latter hypothesis, #2, was incorrect.

In contrast to the resistance demonstrated by cattle at challenge with *D. viviparus*,[2,10,13] most fawns had one or more periods of patency after their initial patent infections were reduced by anthelmintic therapy. Only one fawn (2) developed resistance to patent infection after single treatment with cambendazole; LPG counts for this fawn did not increase during time of confinement on the contaminated pasture. Similar data were reported by O'Roke[10] who found that reinfection with *Leptostrongylus alpenae* was common in white-tailed deer.

Placement of susceptible deer on the contaminated pasture demonstrated that *D. viviparus* larvae remained viable and infective for at least 3 months during summer. The light lungworm burdens acquired by these deer indicated that the level of contamination probably was considerably reduced.

Therefore, in order to eliminate *D. viviparus* from these deer, it was necessary to treat with cambendazole and then move them from the contaminated area. When LPG counts increased 3 weeks later, a second treatment was necessary to eliminate the immature lungworms that were not affected by initial treatment.

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