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Authors: FORRESTER, DONALD J., TAYLOR, W. JAPE, and NAIR, K. P. C.

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STRONGYLOIDOSIS IN CAPTIVE WHITE-TAILED DEER[1]

DONALD J. FORRESTER,[2] W. JAPE TAYLOR,[3] and K. P. C. NAIR[2,4]

Abstract: From 1963 to 1972 39% of 251 fawns born in a captive herd of white-tailed deer *(Odocoileus virginianus)* died with signs attributable to strongyloidosis. At necropsy one typically affected fawn contained 50,000 female *Strongyloides* in its small intestine. These nematodes and free-living adult females cultured from feces compared morphologically and metrically to *S. papillosus*. Egg counts on infected fawns varied from 200 to 286,000 eggs per gram of feces. Evidence was obtained that intrauterine transmission of this parasite occurred. The disease was controlled by removing fawns from their mothers shortly after birth and raising them on bottled milk and out of contact with the ground. Fawns were treated with thiabendazole at 6 and at 30-40 days of age and maintained in pens with elevated wooden floors until 8-10 months of age. The original source of the *Strongyloides* infection in this captive herd and the possible significance of this disease in wild populations of deer are discussed.

INTRODUCTION

The genus *Strongyloides* is known to contain at least 48 species, most of which are intestinal parasites of mammals and birds, some also being found in reptiles and amphibians. One of the most common species is *S. papillosus* which is worldwide in distribution and is found in the small intestine of sheep, goats, cattle, zebras, camels, pigs, rabbits, and several wild ruminants.[7] It has been found to be pathogenic for calves,[16] lambs,[14,15,17] and kids.[14,15]

Although there are published reports of *S. papillosus* from Columbian black-tailed deer, *Odocoileus hemionus columbianus*[1,8] and white-tailed deer, *Odocoileus virginianus*,[4] little is known of the pathogenicity of this nematode to deer, especially fawns. The present paper reports the occurrence and apparent pathogenic effects of *S. papillosus* in fawns of a captive white-tailed deer herd in north-central Florida.

HERD HISTORY AND MANAGEMENT PRACTICES

In 1962 a captive herd of white-tailed deer was established by one of us (W.J.T.) on the campus of the University of Florida in order to study sickle cell hemoglobins.[5,6] The original deer were wild-caught fawns obtained from several areas in northern, central and southern Florida by personnel of the Florida Game and Fresh Water Fish Commission.

The pen area covered 0.8 hectares and was enclosed by a 3.7 m link fence with 1.8 m high opaque plastic screening at the bottom. Within the main fence the area was divided into several holding

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[2] Department of Veterinary Science, University of Florida, Gainesville, Florida 32611, U.S.A.

[3] Department of Medicine, College of Medicine, University of Florida, Gainesville, Florida 32610, U.S.A.

[4] Present Address: Department of Pathology, International Research and Development Corporation, Mattawan, Michigan 49071, U.S.A.

pens; four 0.1-hectare breeding pens (each dissected by a running stream), five 12.2 m^2 general purpose pens, 31 3.0 m x 9.1 m covered pens, and a number of smaller fawn holding pens. The entire enclosure was on sandy soil which was well-drained except during some periods of heavy rainfall usually in the spring months. The pens contained some natural vegetative ground cover.

In 1963 and 1964 the herd was comprised of approximately 25 deer. The number increased to a peak of some 70 animals in 1967 and 1968. Since that time the size of the herd has been purposely stabilized at about 40 adults and 10 to 15 yearlings. During the breeding season of 6-8 months (September through April) from four to eight does and one buck were held in the 0.1-hectare pens. Excess bucks and yearlings were kept in the individual 3.0 m x 9.1 m pens which were adjacent to one another. Does were moved into these pens for fawning from May to September. There was some rotation of deer among the various pens, most of which were vacant for 4-6 months each year.

The animals were fed Purina Calf Startena and maintained an excellent nutritional state. As one index of this, it was not unusual for a 1-year-old buck to have a four-point rack (two points on each antler). The fawns which were raised on the bottle were given evaporated cow's milk which was diluted half and half for the 1st week with the concentration then being increased gradually so that it was undiluted by 1-month-of-age. Ferrous sulfate and vitamins A, B, C, D, and E were given as supplements each day. It was usual for the fawns on the bottle or those remaining with their mothers to triple their birth weight within one month.

Other wild and domestic ruminants were housed in pens adjacent to the Florida white-tailed deer over the 10-year period. Among the animals maintained on the premises for varying periods of time were: white-tailed deer from Texas, Maryland and Panama, Columbian black-tailed deer, mule deer *(Odocoileus hemionus hemionus)*, sika deer *(Cervus nippon nippon)*, fallow deer *(Dama dama)*, wapiti *(Cervus canadensis)*, guanaco *(Lama glama huanacus)*, domestic goats, domestic sheep, cattle, donkeys and dwarf Shetland ponies. Wild rabbits *(Sylvilagus* spp.), gray squirrels *(Sciurus carolinensis)*, domestic pigeons *(Columba livia)* and a variety of wild birds and rodents were also frequent visitors or semi-permanent residents of the deer pens and adjacent land.

Thiabendazole* (TBZ) was used routinely in the feed of the domestic animals, but not for the deer because of palatibility problems. Beginning in 1971, however, TBZ was utilized as an anthelmintic in the deer, as will be discussed later in this report.

FAWN MORTALITY

In Table 1, fawn mortality has been summarized for the 10-year period 1963-1972. In this table, only mortality for which there existed a strong probability of its link with strongyloidosis is included. This was determined by consulting the herd records and excluding deaths attributed to stillbirth problems, trauma, etc., and including data only on fawns known to have died with signs attributable to strongyloidosis.

The fluctuations in mortality over the 10-year period were probably related to different methods used in rearing the fawns. During the years 1964 and 1965 most of the fawns were separated from the does at a few days of age and raised on bottles (usually at the home of one of the investigators). In 1966 approximately half of the fawns remained with their mothers and in 1967 all of the fawns were left to nurse at the pens. Following the high mortality in 1967, most of the fawns were separated from their mothers at 1-2 days of age and raised off the ground. In 1969, the year of highest mortality all of the fawns were

* Thibenzole: Merck & Co., Rahway, New Jersey.

left with their mothers. In 1970, 11 fawns were raised on bottles and the remainder were left with the does. The mortality was the same in both groups, but it is of interest that the fawns which were separated from their mothers were not removed until they were 1 week old. Approximately half of the fawns were treated with *Clostridium* vaccine at 3-4 weeks of age, but mortality was not influenced. In subsequent years all fawns were removed at 2 days of age.

CLINICAL OBSERVATIONS AND PATHOLOGY

In animals dying at 6-14 days of age, obvious abdominal cramping developed and it was extremely difficult to get them to nurse from a bottle. When raised by their mothers, few fawns were lost at an early age, but many died at 3-8 weeks, when an acute illness would suddenly appear. Afflicted fawns became listless and usually experienced diarrhea and were dead within 12 to 36 hours after the onset of signs.

In the latter part of 1969 and early in 1970, fecal cultures were obtained from 12 fawns either during life or immediately post-mortem. In one fawn which died and one which survived without antibiotic therapy, *Salmonella anatum* was cultured from feces. Both of these fawns were also heavily parasitized by *Strongyloides*. No other bacterial pathogens were isolated.

At necropsy the fawns exhibited acute hemorrhagic enteritis. Tissue sections revealed hyperemia, extravasation of vessels in the lamina propria, and focal degeneration and desquamation of superficial and intestinal glandular epithelia of the small intestine. Polymorphonuclear leucocytes and eosinophils were seen to infiltrate the lamina propria and submucosa of affected portions of the intestinal wall. Nematode eggs were found free on the surface of the mucosa and along with desquamated cells in the lumen. In many cases scattered areas of bronchopneumonia were also seen in the lungs.

TABLE 1. Fawn mortality attributed to strongyloidosis in a captive herd of white-tailed deer from 1963 to 1972.

Year	Total No. fawns born	Fawns dying during first 6 months	
		No.	%
1963	4	0	0
1964	16*	0	0
1965	24*	2	8
1966	34	9	26
1967	39	30	77
1968	32*	5	16
1969	36	29	81
1970	33	21	64
1971	18**	2	11
1972	15**	0	0

* Most fawns raised on bottle and maintained without ground contact; four of the 1968 fawns that died were ones which had been left with their mothers in the pens with ground contact.

**Kept with does for 2 days; then on bottle and maintained without ground contact; TBZ treatment utilized.

No clearcut clinical manifestations of disease due to *Strongyloides* in adult deer have been noted. However, two observations are important and indicate the probability of untoward effects in adult animals. One adult doe died in the winter of 1969 following several weeks of diarrhea, anorexia and progressive debilitation. Large numbers of nematode eggs were identified in fecal samples from this animal on two separate occasions and the necropsy revealed what was considered to be nonspecific enteritis. Of more importance, relative to the fawn mortality, is the observation that many does have been observed to have profound lameness for several days. This has appeared to be due to diffuse muscle soreness and has been seen only in late spring or early summer, usually near term or shortly after delivery, presumably at a time when infective larvae may be migrating throughout the body.

PARASITOLOGIC STUDIES

In August of 1970 *Strongyloides* was first associated with the mortality when two fawns (1-week-old and 1-month-old), exhibiting clinical and post-mortem signs described above, were found to contain large numbers of parasitic females of a species of *Strongyloides* in their small intestines. Counts were not made of these nematodes, but each fawn was estimated to have several thousand parasites. Two additional fawns, 2 and 3 weeks of age, were examined at necropsy and contained 3,250 and 50,000 female *Strongyloides* respectively in their small intestines. Both fawns exhibited clinical and post-mortem signs attributable to strongyloidosis.

Measurements of these parasitic female *Strongyloides* compared closely with those published for *S. papillosus*.[7] Rectal fecal samples from these fawns were placed on damp filter paper in petri dishes and cultured at room temperature for 5 days. Free-living adult females from these cultures conformed metrically and morphologically to data published by Basir[3] for free-living adult females of *S. papillosus*. Larval stages and free-living adult males were not studied.

During 1970 and 1971 fresh fecal samples were collected from bucks, does, fawns, and sheep and goats and examined for eggs of *Strongyloides* by the sodium dichromate flotation technique. Twelve of 13 fawns from 3 days to 3 weeks of age and one of four fawns 6-8 weeks of age were shedding *Strongyloides* eggs in their feces. Egg counts, determined by the McMaster technique, varied from 200 to 286,000 eggs per gram of feces. Daily fecal samples were collected from several of the fawns beginning on the second or third day after birth. *Strongyloides* eggs were first found in the feces of three fawns on their 4th day of age, indicating that prenatal invasion had probably taken place. One additional fawn (No. 288) was removed from its doe at birth, never allowed to suckle its mother, and raised away from the pen area. It had touched the ground only momentarily at birth and had been thoroughly wiped with clean rags within minutes after birth. This fawn first passed *Strongyloides* eggs at 5 days of age, which indicated that intrauterine transmission had occurred.

Three of 15 mature does, but none of five mature bucks, were positive for *Strongyloides* eggs in their feces. No eggs were found in feces from 17 1-month-old kids and from one 3-week-old lamb.

One fawn which did not die had coccidial oocysts in some fecal samples.

TREATMENT AND CONTROL PROGRAM

Beginning in 1971 the following treatment and control program was instituted. Fawns were removed from their does 2 days after birth, bottle-fed and raised in pens out of contact with the ground. At 6 days of age the fawns were treated with thiabendazole (TBZ) at the recommended dosage of 50 mg/kg body weight. At 30-40 days of age the TBZ treatment was repeated just prior to the time the fawns were returned to holding pens which had wooden floors 10 cm off the ground. Fawns were kept off the ground for 6-8 months or longer. These

practices resulted in reductions in mortality of fawns in 1971 and 1972 (Table 1).

DISCUSSION

There are several possible explanations of the source of the *Strongyloides* infections in this captive deer herd. One might be that the parasites occur in native wild white-tails in Florida and that the original wild fawn stock was infected when obtained in 1962. This idea is supported by the fact that several wild fawns (a few days of age) died within 2 or 3 days after capture and exhibited signs resembling those seen in the pen-reared fawns known to be afflicted with strongyloidosis. However, fecal analyses for *Strongyloides* eggs were not performed on these wild fawns.

Little is known about *Strongyloides* infections in wild deer in North America. Longhurst and Douglas[6] reported *S. papillosus* from Columbian black-tailed deer in northern California where sheep and deer share common ranges. Later, Baker *et al.*[1] showed that lambs could be experimentally infected with *S. papillosus* obtained from black-tailed deer in California. Glazener and Knowlton[4] reported "light" infections of *Strongyloides* sp. from 10 of 42 white-tailed deer in southern Texas. Samuel,[12] however, examined 61 small intestines and 327 fecal samples from white-tailed deer from the same area in Texas and found no infections by *Strongyloides*. He concluded that the specimens of *Strongyloides* sp. reported by Glazener and Knowlton[4] were misidentified. No specimens were retained from their study, so this point cannot be further clarified.

It is possible also that the *Strongyloides* infections entered the captive herd through other ruminants kept in adjacent pens. This does not seem probable, however, since these animals had been treated routinely with TBZ and fecal samples from several kids and a lamb were negative. In addition, no deaths in young kids or lambs occurred with signs attributable to strongyloidosis.

A third source of the *Strongyloides* infection might have been wild rabbits which were known to enter and leave the deer pens from time to time. Bush (pers. comm.) examined three marsh rabbits *(Sylvilagus palustris)* obtained from an area adjacent to the deer pens and found none infected with *Strongyloides*. However, since domestic rabbits are known to be suitable hosts for *S. papillosus*,[8,13] this possible source of infection cannot be ruled out entirely.

The finding, reported herein, that *S. papillosus* may be capable of infecting deer prenatally is in agreement with published data on calves.[11] Bezubik,[3] however, was unable to establish experimental intrauterine infections of *S. papillosus* in rabbits and Nghiem and Neilson (pers. comm.) have confirmed this observation. Utilizing the data of Moncol and Batte,[10] who found a decreased prepatent period of 3.5 to 4 days for infections of pigs by *S. ransomi* larvae from colostrum as compared with 6-7 days by cutaneous infection, Bezubik[3] questioned the validity of some earlier reports of prenatal infection which had been predicated on shortened prepatent periods. He feels that the larvae had undergone biological changes while migrating in the host and that this resulted in the shortened prepatent periods. Although this may be true, the observation on the one fawn (No. 288), which never suckled its mother but passed *Strongyloides* eggs in its feces at 5 days of age, is evidence of prenatal transmission of *S. papillosus*, at least in this host.

Infection of deer through the ingestion of colostrum or milk or through contact with contaminated soil may have occurred also. This was not conclusively shown in our study, but it was suggested by the severe and often fatal infections which were noted in fawns that were left with their mothers (with ground contact) for longer than 2 days. In this connection Lyons *et al.*[9] have found larvae of *S. papillosus* in the milk of a cow and six ewes from the 7th and 8th days through the 19th day after parturition had taken place.

Sandground[13] in a detailed study of the life cycle of several species of

Strongyloides implied that the rabbit was the original host for *S. papillosus* and that this parasite has secondarily entered ungulate hosts. If this is true it appears that the parasite in its new host, the hoofed animal, has developed an efficient and multifaceted transmission mechanism which would insure its success as a parasite and account for its cosmopolitan distribution in sheep, cattle, etc.

Stewart (pers. comm.) feels that the problem of *S. ransomi* infections in pigs has been accentuated by the use of farrowing houses where the pig cannot get away from the sow. Under the older system where pigs were raised in the open, sows apparently did not get heavily infected with *Strongyloides,* passed very few larvae in the milk and therefore pigs were not exposed to large amounts of infective larvae. When pigs were moved into farrowing quarters, the supply of prenatal and milk larvae may have increased along with the number of larvae from contaminated soil. A similar phenomenon might have happened in the captive deer herd discussed in this paper, i.e. contaminated pens may have contributed to a reservoir of many larvae in the does and in turn the quantities of larvae reaching the fetuses and/or young fawns increased enough to cause disease and death.

It cannot be stated with certainty that strongyloidosis *per se* was the cause of death in the fawns, but a strong presumption that it was a major factor is based on three premises. The first is that, with the exception of a *Salmonella* organism in one fawn which died, no other pathogens were ever isolated. Furthermore, the fawns which were raised off the ground were in close proximity to all the other animals and in the same environment as far as exposure to arthropod vectors, air-borne viruses, etc. and yet mortality was greatly reduced by keeping them off the ground. Secondly, mortality was much greater in fawns which were separated at 6-8 days of age than in those which were separated at an earlier age and raised on canned milk. Finally, it is impressive that measures directed entirely toward the control of strongyloidosis, but which would have minimal effects on bacterial or viral infections, have greatly reduced the mortality.

The prevalence and pathologic impact of *Strongyloides* in native deer populations is unknown at the present time. This is probably because young fawns have not been studied adequately. Most published surveys on parasites of deer have not contained data on young fawns.

In addition, the observed susceptibility of fawns to this pathogenic nematode may have serious management implications for the practice of livestock grazing on National Forests and other areas which support important white-tailed deer herds. For these reasons, further studies on this nematode in wild populations of white-tailed deer and other cervids should be undertaken, with particular emphasis on young animals.

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LITERATURE CITED

1. BAKER, N. F., W. M. LONGHURST and J. R. DOUGLAS. 1957. Experimental transmission of gastrointestinal nematodes between domestic sheep and Columbian black-tailed deer. Trans. No. Amer. Wild. Conf. 22: 160-166.
2. BASIR, M. A. 1950. The morphology and development of the sheep nematode, *Strongyloides papillosus* (Wedl, 1856). Can. J. Res., Ser. D, Zool. Sci. 28: 173-196.

3. BEZUBIK, B. 1969. Negative attempts to obtain transuterine infections with *Strongyloides papillosus* in the rabbit. Acta Parasit. Polon. 17: 11-16.
4. GLAZENER, W. C. and F. F. KNOWLTON. 1967. Some endoparasites found in Welder Refuge deer. J. Wild. Mgt. 31: 595-597.
5. KITCHEN, H., F. W. PUTNAM and W. J. TAYLOR. 1964. Hemoglobin polymorphism: its relation to sickling of erythrocytes in white-tailed deer. Science 144: 1237-1239.
6. KITCHEN, H., F. W. PUTNAM and W. J. TAYLOR. 1967. Hemoglobin polymorphism in white-tailed deer: subunit basis. Blood 29: 867-877.
7. LEVINE, N. D. 1968. Nematode parasites of domestic animals and of man. Burgess Publishing Co., Minneapolis, Minnesota. 600pp.
8. LONGHURST, W. M. and J. R. DOUGLAS. 1953. Parasite interrelationships of domestic sheep and Columbian black-tailed deer. Trans. No. Amer. Wild. Conf. 18: 169-188.
9. LYONS, E. T., J. H. DRUDGE and S. C. TOLLIVER. 1970. *Strongyloides* larvae in milk of sheep and cattle. Mod. Vet. Pract. 51: 65-68.
10. MONCOL, D. J. and E. G. BATTE. 1966. Transcolostral infection of newborn pigs with *Strongyloides ransomi*. Vet. Med. 61: 583-586.
11. SUPPERER, R. and H. PFEIFFER. 1962. Untersuchungen über die Gattung *Strongyloides* II. Die praenatale Invasion biem Rinde, sowie Beobachtunger über allergische Hautreaktionen noch perkutaner Invasion. Berl. u. Muench. Tierarz. Wochenschr. 75: 344-346.
12. SAMUEL, W. M. 1969. Parasites of the white-tailed deer in south Texas. Ph.D. dissertation. U. of Wisconsin, Madison. 196 pp.
13. SANDGROUND, J. H. 1926. Biological studies on the life cycle of the genus *Strongyloides* Grassi, 1879. Amer. J. Hyg. 6: 337-388.
14. TURNER, J. H. 1955. Preliminary report of experimental strongyloidiasis in lambs. Proc. Helm. Soc. Wash. 22: 132-133.
15. TURNER, J. H. 1959. Experimental strongyloidiasis in sheep and goats. III. An attempt to induce passive immunity in lambs; changes in serum proteins after infection; and effect of immune serum on infective larvae Proc. Helm. Soc. Wash. 26: 114-124.
16. VEGORS, H. H. 1954. Experimental infection of calves with *Strongyloides papillosus* (Nematoda). Amer. J. Vet. Res. 15: 429-433.
17. WOODHOUSE, C. A. 1948. Observations on pathogenicity of *Strongyloides* parasites in ruminants. J. Amer. Vet. Med. Assoc. 113: 354-356.

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