EFFICACY OF DRENCHING RED DEER AND WAPITI WITH PARTICULAR REFERENCE TO *ELAPHOSTRONGYLUS CERVI* AND *DICTYOCAULUS VIVIPARUS*

T.G. Watson
Ruakura Animal Research Station
Private Bag, Hamilton, New Zealand

CURRENT NEW ZEALAND PERSPECTIVE

Internal parasites

Generally farmers seem to be following recommendations and drenching weaner stock at 21 day intervals after weaning with durations varying depending on farmer awareness and local veterinary suggestions (Mackintosh & Mason 1985; Mason & Beatson 1985). The primary source of parasitic disease continues to be lungworm infections and drenching programmes tailored to control this nematode appear to have excellent control of any of the helminths recognised as infecting the gastrointestinal tract of farmed deer. This does not mean, however, that anthelmintics are being used to effectively maximise productivity. Still we find little information to confirm that drenching is increasing productivity.

External parasites

Ever since the cattle tick *Haemaphysalis longicornis* was identified as a pathogen of red deer and calves in New Zealand there have been sporadic reports that it is becoming a serious problem on deer farms (Neilson & Mossman 1982). As yet data concerned with distribution, epidemiology and control have not been collected to enable an accurate assessment of its importance, regionally and nationally.

Generally, the restricted size of the industry remains the most important reason for minimal parasitological research commitment from various organisations. Research and broad scale on-farm monitoring appears to be at a standstill because lungworm infections appear to be controlled by measures adopted to date. Furthermore, gastrointestinal nematode infections seem to also be kept under control at the same time. Tissue worm remains the only parasite to be controlled but its current status on farms is unknown. In recognition of this, the data given at this time were collected during attempts to eliminate tissue worm from grazing wapiti on a farm in Westland.

EXPERIMENTAL STUDIES

Introduction

In the past research into animal health has emphasised control of lungworm infections in deer (*Cervus elaphus*) farmed in New Zealand (Mackintosh & Mason 1985; Mason 1985; Mason & Beatson 1985). Infection by *Dictylocaulus viviparus* was and remains identified as the most important parasite problem facing deer farmers (Gladden 1981). Although a close relative, tissueworm

(Elaphostrongylus cervi) has never been recognised as a major threat to health even though recent surveys showed infected animals are widely dispersed throughout New Zealand (Anon 1981; Mason & Gladden 1983). Establishment has not been shown conclusively on farms in New Zealand, yet definitive and intermediate hosts occupy the same pastoral habitats. Therefore, E. cervi may be expected to become established on farms since all aspects of the life cycle are satisfied.

Clinical elaphostrongylosis has never been diagnosed in C. elaphus in New Zealand. Losses within herds of reindeer have been attributed to infection by E. rangiferi (= E. cervi) in various parts of Scandinavia (Kummeneje 1974, 1980; Rehbinder et al. 1981). The lack of importance attached to E. cervi in New Zealand in all likelihood arises from the lack of clinical symptoms, a direct coincidence of low prevalence within herds and apparent low intensity of first stage (L_1) larvae recovered from infected animals to date. Current anthelmentic treatment regimes place most emphasis on dosing young stock with older animals usually receiving one or less drenches annually (Mason & Gladden 1983). Furthermore, most farmers have been shown to use one of three of the BZ drenches: fenbendazole (FBZ), oxfendazole (OBZ) or albendazole (ABZ) (Mason & Gladden 1983). It is likely that ivermectin (IVM) can be added to this list even though it is not licensed for use in deer in New Zealand. Control of lungworm infections is the foremost concern of deer farmers and the usual 21 day drenching programme currently recommended is based on the life cycle of D. viviparus (Mason 1979). There have been no attempts to determine if control of lungworm will confer control of E. cervi. Therefore, the aim of the present work was to examine the efficacies of various drenches in controlling or eliminating infections by E. cervi from C. elaphus held on pasture or indoors (to preclude reinfection).

MATERIALS AND METHODS

Parasitological procedures

Faecal samples were taken from the ground immediately after they were dropped in the yards or from the rectum while the animals were physically restrained. A modified McMaster method was used to determine the number of parasite eggs per gram of faeces (epg). Number of larvae per gram of faeces (lpg) were estimated by the Baermann technique. Samples were allowed to stand in tap water for 36 hours at 20-22 °C to ensure complete larval sedimentation for recovery of the L_1 larvae of both D. viviparus and E. cervi. Differentiation and enumeration of L_1 larvae was accomplished with a Wild dissecting microscope at 125 x magnification.

Examinations of carcasses at slaughter involved dissection of all major muscle bundles. Where available, spinal cords and brains were removed intact for study. Lungs taken from animals sent to the deer slaughter premises were dissected apart to enable recovery and enumeration of all D. viviparus. All E. cervi found in situ were carefully teased from the connective tissue and placed in physiological saline for further examination to determine worm sex and viability.

Laboratory investigations

Three 18 month old red deer stags were used in a pen trial. They had been held indoors on wood flooring since drenching with OBZ at 12 weeks of age to prevent infection with E. cervi or D. viviparus. Feed (lucerne, barley pellet ration, Reeves Ltd, Dunedin) and water were provided ad libitum. Infective (L_3) larvae of E. cervi cultured in Deroceras panormitanum were recovered and enumerated after acid-pepsin digestion at 37 °C. Larvae were washed once in tap water and each animal was dosed per os following light chemical restraint using Rompun. Between 400 and 500 infective larvae were given to each animal. Infections were allowed to reach patency. At that time each animal was given two doses (9.0 mg/kg) of OBZ (Synanthic®, Syntex NZ Ltd) 48 hours apart. Faeces were taken rectally from each animal at regular intervals before and after treatment to monitor lpg. Individuals were killed 29, 86 and 129 days after the anthelmintic was given and each carcass was examined for E. cervi.

Farm investigations

Forty-five red deer-wapiti hybrids (C. elaphus) of mixed age (1-7+ years) and sex (19 stags and 26 hinds) were recently turned onto pasture after capture from Fiordland National Park. All had been shown to be shedding protostrongylid L_1 larvae, D. viviparus L_1 larvae and nematode parasite eggs during July 1982. Stags and hinds grazed separately on ryegrass-clover pasture. The animals were randomly assigned into four treatment groups. Twelve animals in Group C made up the untreated infected control; the six animals of Group OBZ-B were given 9.0 mg/kg OBZ on two occasions, 48 hours apart; 11 animals in Group OBZ-A were each dosed with 9.0 mg/kg OBZ on three consecutive days; and the 10 deer in Group I were each injected subcutaneously in the neck with IVM (Ivomec®, MSD) at the recommended cattle dose rate of 200 µg/kg. Group U consisted of nine yearling red deer born on the property and two captured red-wapiti hybrids that did not pass L_1 larvae of E. cervi prior to the trial. These animals ran with the captured infected wapiti stags throughout the trial.

RESULTS

Laboratory study

All three red deer given infective L_3 larvae of E. cervi began shedding L_1 larvae 86-98 days after infection (dpi) (Table 1). Approximately 6 weeks after patency the anthelmintic was given. Within 9 days two animals were shedding fewer than 1 lpg and within 16 days outputs for all animals were below 1 lpg. All animals continued to shed low numbers of larvae prior to slaughter.

At slaughter all three animals remained infected with adults of E. cervi (Table 1). Animals shedding most L_1 larvae were found to be infected with the most adult nematodes. In all instances worms that were recovered continued to be active when placed in saline even though specimens were found in both CNS and muscle connective tissues.

Table 1: Anthelmintic efficacy in Cervus elaphus artifically infected with Elaphostrongylus cervi.

Animal tag	Infection		Necropsy			
	Dose (L_3)	Prepatent time (days)	Post-infection nematode recovery Time (days)	Male	Female	Total
128 R	456	96	28	2	1	3
130 R	478	86	129	3	11	14
131 R	452	98	86	6	1	7

Field study

Within all groups faecal egg counts remained low (0-200). As a consequence it is difficult to assess the real effects the anthelmintics had on gastrointestinal parasitism. It is noteworthy that few operculated eggs (Paramphastomum or Fasciola) were seen on two occasions.

During the field trial infection by E. cervi was never detected in animals from group U. Although many consistently shed L_1 larvae of D. viviparus mean lpg remained very low (Table 2). Members of group C, the infected untreated animals, inconsistently shed low numbers of L_1 larvae of E. cervi (Table 3). There was a tendency that fewer animals shed larvae into the summer months. Larvae of D. viviparus were consistently isolated in low numbers from most deer in this group (Table 2).

Ivermectin reduced the numbers of larvae of E. cervi isolated from faeces of all animals within 2 days after treatment however, few animals continued to shed larvae periodically (Table 3). Within 14 days of treatment L_1 larvae of D. viviparus had disappeared from but one animal which had ceased to shed larvae by 28 days after treatment (Table 2). Whereas the effects of ivermectin on frequency and intensity of L_1 larvae of E. cervi seemed to be sustained throughout the monitor period most animals were reshedding L_1 larvae of D. viviparus when sampled 63 days after treatment (Table 2).

Two doses of oxfendazole 48 hours apart (Group OBZ-B) removed all L_1 larvae of E. cervi within a fortnight of drenching from all members of the groups (Table 3). Low lpg was noted in few deer during the remaining monitor period. No animals shed larvae of D. viviparus 14 days after dosing but patent infections were identified in most animals 14 days later (Table 2).

Triple dosing with OBZ increased the time during which infections by E. cervi could not be identified from 14 to 28 days but few animals shed larvae at the three subsequent sample times (Table 3). Again the effective control of D. viviparus was shortlived with most deer shedding larvae 28 days after drenching (Table 2).

Three animals died during the trial, two from Group C and one from Group IVM but delays in recovery of animals prevented necropsies being made. Five animals (#86, 95, 104, 331, 999) from Group U, one (#28) from Group C, one (#337) of Group I and two (#301, 344) of Group OBZ-A were culled and slaughtered after the final faecal sampling in March 1983. Lungs and heads were provided for dissection. Adult D. viviparus were recovered from all yearling red deer and three older wapiti (Table 4). The animal (#337) from Group IVM was condemned upon meat inspection at the slaughter premises because of numerous greenish nodules in the fascia of the upper legs and backstrap. Examination of the muscles of both forelegs produced five adult E. cervi, three male and two female. All worms were active when placed in saline. First stage larvae of E. cervi had not been recovered for any of the five faecal samples taken during the 129 days after treatment.

Table 2: Recovery of first stage larvae of Dictyocaulus viviparus from faeces of Cervus elaphus (larvae g^{-1} faeces).

Group	Sample dates							
	2.11.82	3.11.82	4.11.82	16.11.82	30.11.82	4.1.83	9.2.83	11.3.83
CONTROL								
Frequency	11/11	-	10/10	10/11	10/11	9/9	9/9	7/9
Intensity								
Mean	3.8	-	5.0	4.5	5.8	2.3	3.5	0.9
Range	0.1-11.3		0.2-15.8	0-31.3	0-46.3	0.1-7.1	0.4-14.1	0-3.4
UNINFECTED								
Frequency	-	10/11	10/11	7/11	9/11	8/11	8/11	5/11
Intensity								
Mean	-	2.3	1.9	0.4	0.5	1.0	0.3	0.4
Range		0-10.9	0-5.1	0-1.5	0-3.0	0-7.1	0-1.2	0-2.0
OXFENDAZOLE-A								
Frequency	9/11	9/11	5/11	1/11	4/11	7/9	6/10	6/9
Intensity								
Mean	5.4	5.7	0.4	<0.1	0.3	1.3	1.3	18.1
Range	0-38.7	0-22.0	0-1.6	0-0.1	0-2.3	0-7.9	0-11.2	0-161.0
OXFENDAZOLE-B								
Frequency	6/6	-	4/6	0/6	4/6	4/5	3/6	5/6
Intensity								
Mean	25.7	-	24.3	0	0.4	1.3	1.5	2.7
Range	0.2-129.0		0.1-141.6		0-1.5	0-5.5	0-5.4	0-9.9
IVERMECTIN								
Frequency	10/10	-	6/9	1/10	0/10	7/9	8/9	5/6
Intensity								
Mean	24.0	-	0.7	<0.1	0	1.7	5.9	1.3
Range	0.4-143.1		0-4.5	0-0.1		0-5.8	0-42.3	0-5.6

Table 3: Recovery of first stage larvae of *E. cervi* from faeces of *Cervus elaphus* (larvae g^{-1} faeces).

Group	Sample dates							
	2.11.82	3.11.82	4.11.82	16.11.82	30.11.82	4.1.83	9.2.83	11.3.83
CONTROL								
Frequency	10/11	-	10/10	9/11	8/11	7/9	5/9	5/9
Intensity								
Mean	5.1	-	10.8	4.8	2.7	7.7	6.1	4.9
Range	0-20.9		0.1-44.5	0-18.3	0-7.5	0-45.0	0-22.5	0-28.8
UNINFECTED								
Frequency	-	0/11	0/11	0/11	0/11	0/11	0/11	0/11
Intensity								
Mean	-	0	0	0	0	0	0	0
Range								
OXFENDAZOLE-A								
Frequency	8/11	8/10	9/11	0/11	0/11	2/9	1/10	2/9
Intensity								
Mean	7.0	27.2	11.4	0	0	0.1	0.1	0.2
Range	0-28.4	0-164.0	0-56.4			0-0.5	0-0.9	0-0.9
OXFENDAZOLE-B								
Frequency	5/6	-	6/6	1/6	3/6	2/5	0/6	0/6
Intensity								
Mean	29.2	-	25.4	0.1	0.1	0.2	0	0
Range	0-133.5		1.0-57.4	0-0.4	0-0.2	0-1.0		
IVERMECTIN								
Frequency	7/10	-	7/9	3/10	2/10	1/9	3/9	1/6
Intensity								
Mean	27.6	-	5.2	3.6	2.7	0.5	0.7	0.4
Range	0-225.6		0-20.2	0-36.2	0-26.4	0-0.5	0-3.5	0-2.4

Table 4: Lungworm recovery at necropsy.

Animal				Parasitology			
ID	Age (years)	Sex	Species	Lungworm recovery			Larval† count
				Male	Female	Total*	
86	1+	M	Red	1	1	3	0.3
95	1+	M	Red	7	13	20	0
104	1+	M	Red	4	6	12	0.5
999	1+	M	Red	13	27	42	0
28	2+	M	Wap	7	25	35	0.1
301	5+	F	Wap	434	775	1220	161.0
331	2+	M	Wap	0	0	0	2.0
337	2+	M	Wap	0	0	0	0
344	4+	M	Wap	2	6	9	0.1

*Also includes incomplete worms.

†L. larvae/g faeces.

DISCUSSION

These present data confirm the finding of Mason and Gladden (1983) that epg is usually low. Gastro-intestinal parasitism may not have been a significant problem on the property. Paramphistomum and Fasciola have both been identified in cattle on the farm but the significance and identity of the operculated eggs found in deer remains unresolved.

Drenching C. elaphus for parasite control has always been designed to control of lungworm infections in calves but frequencies of anthelmintic treatment have been highly variable (Mason & Gladden 1983). Members of the benzimidazoles (fenbendazole, oxfendazole and albendazole) were the drenches most often used by the farmers surveyed (Mason & Gladden 1983). All three administered orally at or above dose rates recommended for cattle have been shown to be effective against D. viviparus in red deer (Mason 1979; Wilson 1981). Therefore, it is not unreasonable to expect that OBZ given at double the recommended cattle dose at the 2 day or 3 day frequencies used in the present trial should not be almost 100% effective. One significant observation was the large number of animals given IVM that continued to be negative for L_1 larvae 28 days after treatment. Clearly, a difference in duration of efficacy exists and future studies may well confirm this result. This prolonged efficacy may well infer that the time between drenching may be extended safely to every 28 days if IVM is used. It should be pointed out at this time that IVM is not licensed for use in deer in New Zealand as yet.

Fifteen month old red deer continued to shed low numbers of L_1 larvae of D. viviparus from the few remaining adult nematodes. Captured wapiti appear to be more susceptible than the young red deer; continuing to ingest large numbers of infective larvae from pasture and shed L_1 larvae throughout the trial. This may imply a lack or suppression of resistance developed in the captured stock caused by factors such as stress or insufficient challenge on feral range prior to transition from wilderness to farm. Certainly, there was a tendency for lpg to decline during the trial. Seasonal declines in the larval challenge and worm burdens may account for this observation by the question remains as to how long these animals will remain susceptible to lungworm after capture.

Establishment of E. cervi on deer farms will be dependent on gastropod densities, parasite prevalence and intensity, animal and grazing management, climatic conditions and time. Both intermediate and definitive hosts must be present. Development of E. cervi to the infective L_3 larvae in the molluscan host (D. reticulatum or D. panormitanum) requires at least 27 days at 18 °C (Watson, unpublished). The prepatent period in C. elaphus is 86 to 98 days when between 400 and 500 L_3 larvae are ingested. Seasonal changes of parasite prevalence and intensity of L_1 output seen with animals in Group C in the present study agree with findings of Pryadko et al. (1963) and Wissler and Halvorsen (1976) for Elaphostrongylus in C. elaphus and Rangifer rangiferi on feral range. This seasonality will affect establishment rate in the intermediate host population. At least one terrestrial gastropod susceptible to E. cervi (D. reticulatum) was recovered from the trial property. After 9 months grazing with infected stock none of the red deer calves (Group U) ever passed L_1 larvae of E. cervi. Therefore, we must conclude that sufficient time as well as other requirements have not been satisfied to enable successful establishment.

Efforts towards control of E. cervi in deer have not met with such success. Early efforts with phenothiazine (Pryadko et al. 1963) and diethylcarbamazine phosphate (Pryadko & Teterin 1964) showed only larvicidal activities. Thiabendazole given 1 x 300-500 mg/kg or 7 x 60 mg/kg apparently eliminated E. cervi from maral deer (C. elaphus) in Russia (Shol' et al. 1968). Barth and Schaich (1971) maintained that 30-100 mg/kg of thiabendazole was almost totally effective in wapiti. More recently the broad spectrum anthelmintic FBZ as premix 1.5% pellets or 4% powder given with feed at 3 x 75 mg/kg (Kutzer & Prosl 1979) or 5 x 5.0 mg/kg (Duwell et al. 1979) was found to be 100% effective against E. cervi in C. elaphus. These latter results are most certainly based on removal of L_1 larvae from faeces. Rehbinder in a presentation to the World Wildlife Disease Conference in 1981 stated that 1 x 40 mg/kg of mebendazole did not control E. rangiferi in R. tarandus but 10 x 6.0 mg/kg as premix in feed terminated output of L_1 larvae within 43 days.

Had not animals been necropsied in the present trial the conclusion would have been the same as most of those cited earlier. Both OBZ and IVM appear to have only larvicidal properties at the doses and regimes used herein.

The duration of effective response for both drugs remains undetermined beyond 129 days but both appear to have comparable efficacies. Prolonged microfilaricide activity has been previously reported following administration of ivermectin in dogs infected with Dirofilaria immitis (Blair & Campbell 1980; Anantaphruti et al. 1982) and in horses with Onchocerca cervicalis (Egerton et al. 1980). Anantaphruti et al. (1982) showed that IVM inhibited egg development in D. immitis but did not kill adult nematodes in dogs sacrified up to 14 weeks after two doses of 0.2 mg/kg. The microfilaricidal effects continued beyond 48 weeks and Anantaphruti et al. (1982) postulated that the drug may stimulate the host immune response inducing killing of microfilaria in the liver or reticulo-endothelial system. This action may have considerable economic importance in meat production animals such as deer if nematodes, eggs or L_1 larvae of E. cervi are destroyed in situ. The host immune reaction illicited in the tissues may well assist and compound formation of the greenish lesions observed in animals such as seen in #337. The lesions observed at this time were comparable to those described by Sutherland (1976).

It is difficult to postulate on the fate of worms, eggs or L_1 larvae in the CNS after treatment with Ivermectin it does not appear to easily cross the blood-brain barrier (Campbell et al. 1983). On the other hand, OBZ does cross into the cerebral-spinal fluid in sheep so similar diffusion may take place in red deer (Watson, unpublished). In both cases the efficacies against neurotropic forms must be evaluated further.

CONCLUSION

Additional sampling of some of the stock at Hokitika in May 1984 (almost 18 months after the trial was conducted) revealed that seven of 19 original deer continued to pass larvae of E. cervi with two of seven newly introduced animals also infected. Larvae of D. viviparus were found in eight of 19 deer used initially and five of seven new deer.

Results of this trial reveal that protocols followed for the two drenches used did not eliminate infections by E. cervi. Furthermore, routine drenching, although effectively controlling lungworm does not guarantee control of tissue worm.

REFERENCES

Arantaphruti, M., Kino, H., Terada, M., Ishii, A.I., Sano, M. (1982) Studies on chemotherapy of parasitic helminths XIII. Efficacy of ivermectin on the circulating microfilaria and embryonic development in the female worm of Dirofilaria immitis. Japanese Journal of Parasitology 31: 517-529.

Anon (1981) Status of Elaphostrongylus cervi in deer in New Zealand. New Zealand Ministry of Agriculture and Fisheries, Animal Research Division Annual Report. pp 201.

Barth, D., Schaich, K. (1971) Control of gastro-intestinal roundworms in red deer with thiabendazole. Zietschrift fur Jagdwissenschaft 17: 168-179.

Blair, L.S., Campbell, W.C. (1980) Efficacy of ivermectin against Dirofilaria immitis larvae in dogs 31, 60 and 90 days after infection. American Journal of Veterinary Research 41: 2108.

Campbell, W.C., Fisher, M.H., Stapley, E.O., Albers-Schonberg, G., Jacob, T.A. (1983) Ivermectin: a potent new antiparasitic agent. Science 221: 823-828.

Duwell, D., Kirsch, R., Tiefenbach, B. (1979) On the treatment of nematode infections of game with Panacur®. Berliner und Munchener Tierarztliche Wochenschrift 92: 400-405.

Egerton, J.R. et al. (1980) 22,23-di-hydroavermectin B1, a new broad spectrum antiparasitic agent. British Veterinary Journal 136: 88-97.

Gladden, N.R. (1981) Deer population survey 1980. New Zealand Ministry of Agriculture and Fisheries AgLink FFP259.

Kummeneje, K. (1974) Encephalomyelitis and neuritis in acute cerebrospinal nematodiasis in reindeer calves. Nordisk Veterinaermedicin 26: 456-458.

Kummeneje, K. (1980) Diseases in reindeer in northern Norway. Proceedings 2nd International Reinfeer/Caribou Symposium, Roros, Norway 1979. pp 446-458.

Kutzer, E., Prosl, H. (1979) On the anthelmintic effect of fenbendazole (Panacur®) in red deer (Cervus elaphus hippelaphus) and wild boar (Sus scrofa). Wiener Tierarztliche Monatschrift 66: 285-286, 288-290.

Mackintosh, C.G., Mason, P.C. (1985) Anthelmintics and lungworm in red deer. In: Biology of Deer Production. Fennessy, P.F. and Drew, K.R., editors. Royal Society of New Zealand Bulletin 22: 131-133.

Mason, P.C. (1979) Lungworm in red deer. Biology, symptoms and control. New Zealand Ministry of Agriculture and Fisheries AgLink FPP248.

Mason, P.C. (1985) Biology and control of the lungworm Dictyocaulus viviparus in farmed red deer in New Zealand. In: Biology of Deer Production. Fennessy, P.F. and Drew, K.R., editors. Royal Society of New Zealand Bulletin 22: 119-121.

Mason, P.C., Beatson, N.S. (1985) Anthelmintic activity against Dictyocaulus viviparus in farmed red deer. In: Biology of Deer Production. Fennessy, P.F. and Drew, K.R., editors. Royal Society of New Zealand Bulletin 22: 127-129.

Mason, P.C., Gladden, N.R. (1983) Survey of internal parasitism and anthelmintic use in farmed deer. New Zealand Veterinary Journal 31: 217-220.

Neilson, R.J.A., Mossman, D.H. (1982) Anaemia and deaths in red deer (Cervus elaphus) fawns associated with heavy infestations of cattle tick (Haemaphysalis longicornis). New Zealand Veterinary Journal 30: 125-126.

Pryadko, E.L., Teterin, V.I. (1964) Treatment of Elaphostrongylus infection in the maral. Parazity Sel'skokhoz, Zhivotnykh Karadkastan 3: 90-97.

Pryadko, E.I., Vysokov, S.N., Frolov, V.S. (1963) On the epizootology of elaphostrongylosis in "Panty" deer. Parazity Sel'skokhozyaistvennykh Zhivotnykh Kazakhstan 2: 74-85.

Rehbinder, C., Forssell, I., Nordqvist, M., von Szokolay, P. (1981) Efficacy of mebendazole on Elaphostrongylus rangiferi in reindeer. Proceedings of the 4th International Conference of the Wildlife Disease Association. Fowler, M.E., editor. pp 208-212.

Shol', A.V., Pryadko, E.O., Teterin, V.I., Drobishchenko, N.I. (1968) In: Sh E Esenov. Izdatel. "Nauka" Kazakhskoi., Alma-Ata, SSR. pp 103-112.

Sutherland, R.J. (1976) Elaphostrongylus cervi in cervids in New Zealand 1. The Gross and Histological Lesions in Red Deer (Cervus elaphus). New Zealand Veterinary Journal 24: 263-266.

Wilson, P.R. (1981) Studies of parasitism in red deer. Proceedings of a deer seminar for veterinarians. Deer Advisory Panel of the New Zealand Veterinary Association. pp 71-77.

Wissler, K., Halvorsen, O. (1976) Infection of reinfeer with Elaphostrongylus rangiferi (Nematoda, Metastrongyloidea) in relation to age and season. Norwegian Journal of Zoology 24: 462-463.