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Internal parasites of deer in New Zealand

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Introduction

Six species of deer have been introduced to New Zealand and have established permanent populations in the wild. Only two of these, *Cervus elaphus* (red deer, wapiti and their hybrids) and *Dama dama* (fallow deer) are farmed to any extent, with *C. elaphus* by far the most numerous on farms and the main subject of this article.

Know internal parasites of the two farmed deer species are listed in Table 4.1.[1]

Nematodes

Lungworm (Dictyocaulus viviparus)

The common lungworm of deer, *Dictyocaulus viviparus*, sometimes known as *D. eckerti*, is the most important parasite in farmed deer.[2,3,4,5] A 1981 deer farm survey indicated it is present throughout the country,[6] and this will not have changed.[7]

Dictyocaulus viviparus has a direct life cycle. Adult worms live in the air passages in the lungs; the eggs hatch in the lungs shortly after being laid, and first stage larvae are expelled from the lungs and pass down the gastrointestinal tract of the host and are shed in the faeces. These larvae develop on pasture (without feeding) to infective third stage larvae in as little as five days under optimum conditions. Deer become infected when they ingest infective larvae. These leave the intestine, migrate to the lungs and break through into the alveoli to complete the life cycle.

The minimum prepatent period (time from infection of the host until the worms start laying eggs) for *D. viviparus* in red deer is around 20 days.[7,8]

TABLE 4.1: Known parasites of deer in New Zealand. (Numbers refer to source reference.)

Parasite		Deer species	
		red/wapiti	fallow
Protozoa	*Eimeria* spp.	6	6
	Toxoplasma	45	
	Sarcocystis spp.	45	
	Cryptosporidium	49	
	Neospora	46	
Trematodes	*Fasciola hepatica*	53	59
	Paramphistomatidae	58	7
Cestodes	*Taenia hydatigena*	52	52
	Moniezia spp.		6
	M. expansa	6	
Lung nematodes	*Dictyocaulus viviparus*	53	6
	Elaphostrongylus cervi	25,26	
	Varestrongylus sagittatus	7	
Abomasal nematodes	*Ostertagia*-like:		
	Apteragia quadrispiculata	53	53
	Haemonchus contortus	56	
	Ostertagia (Teladorsagia) circumcincta	56	
	O. leptospicularis	53	
	Rinadia mathevossiana	53	
	Skrjabinagia kolchida	53	
	Spiculopteragia asymmetrica	53	53
	S. spiculoptera	53	
	Trichostrongylus axei	56	7
Small intestinal nematodes			
	Capillaria spp.	53	7
	C. mcmasteri	58	
	C. oncophora	58	
	C. pectinata	57	
	Nematodirus spp.	21	
Large intestinal and caecal nematodes			
	Oe. venulosum	53	53
	Trichuris ovis	53	
	Trichuris spp.		6

The early signs of lungworm infection in red deer are vague, including loss of condition, retarded growth and roughened coat: coughing is not a common sign. Severely affected animals will die.[3,9,10] The most susceptible stock are calves during their first autumn and early winter (3-6 months old) and recently-captured individuals of all ages. By July calves are generally relatively resistant and this resistance persists in healthy animals.[6] Reports of sudden deaths in heavily infested young deer continue to be reported.[11,12,13]

In severe infestations the trachea and bronchi are packed with worms, and death appears to result from physical blockage of the air passages by worms. Extensive areas of consolidation and collapse of lungs, as in cattle, are not usually seen in red deer.[3,9]

My original recommendations for anthelmintic control of *D. viviparus* were to drench calves every three weeks from weaning in March until early winter using one of the longer-acting benzimidazoles.[2] In retrospect this appears to be an overkill for most properties, though desirable where the free-living stages flourish. Subsequently it has been possible to recommend extension of the drenching interval if the newer milbemycin/avermectin anthelmintics are used, because these have persistent activity against lungworm.[14,15,16,17,18,19,20] Levamisole is not effective against lungworm in red deer.[21] Adult deer do not normally require drenching for lungworm. See below for a more detailed discussion of drenching.

There is a good relationship between faecal larval counts and lungworm burdens.[7,19]

Lungworm do not seem to be as important in fallow deer as in red deer, but outbreaks of clinical disease are known, such as the case where 90 of 100 fallow deer died from parasitic bronchopneumonia caused by *Dictyocaulus*.[22]

Tissue worm *(Elaphostrongylus cervi)*

Tissue worm has become important to sections of the New Zealand deer industry, not because of any disease that it causes, but because its presence here has been a barrier to the export of live deer to Australia and Canada. This nematode has been the subject of a recent reviews.[23,24]

Elaphostrongylus cervi was first seen in deer shot in the so-called 'wapiti block' in Fiordland.[25,26] Since then it has been found in low numbers in farmed deer throughout the country, but there was a significant association between the presence of *E. cervi* and the introduction of deer from Southland/ Fiordland at the time the survey was done.[6]

Elaphostrongylus cervi has a two host life cycle. The life cycle is broadly as follows; adult worms are found in the connective tissue (fascia) surrounding skeletal muscles, the eggs are carried to the lungs in the blood system where first stage larvae hatch, migrate through the lung tissue into the air sacs, travel up the trachea, down the gut and leave the host in the mucous coating on the faecal pellets. Further development, to the infective third larval stage, continues in a molluscan intermediate host (slug or snail). When deer eat an infected mollusc the infective larvae are released by digestion and migrate to their preferred site. The migration route may involve a period of association with the central nervous system. The prepatent period is in the region of 120 days. Adult worms may live for several years in a deer.

No clinical signs have been described from naturally infected deer in New Zealand. Adult worms in the fascia of muscles can, however, elicit a reaction from the host; it appears as a localised green discolouration, resulting from an infiltration of eosinophils and lymphocytes, and parasitic granulomata may be formed.[25,27] A diffuse interstitial pneumonia with focal emphysema and consolidation caused by first stage larvae migrating through the lungs has also been described from red deer.[28]

The parasite was first described from red deer in Scotland but has not been associated with disease in that country.[29] In Kazakhstan, on the other hand, it is regarded as the most pathogenic nematode on deer farms.[30] These different views may reflect differences in the way deer are managed or fed in different countries.[23]

No effective treatment is known, though benzimidazoles and ivermectin may temporarily suppress or decrease larval production.

Lungworm (*Varestrongylus sagittatus*)

A new lungworm for New Zealand, *Varestrongylus sagittatus* (syn: *Bicaulus*)

was recovered from the lungs of a farmed red deer in August 1993.[7] *Varestrongylus sagittatus* is a parasite of both red and fallow deer in Europe. This species has a typical protostrongylid life cycle involving a molluscan intermediate host like *E. cervi* above. The adults are found in the lungs where they are intimately associated with the lung tissue, like *Muellerius* in sheep, but no further details are known of it at this time. This species is unlikely to be of any animal health significance, but as the larvae shed in the faeces look like the first stage larvae of *E. cervi*, apart from being smaller, they have the potential to make the diagnosis of *E. cervi* more difficult.

Gastrointestinal nematodes

In the early days of deer farming some highly elevated gastrointestinal worm burdens were seen in farmed deer.[31] Such cases are now comparatively rare. In practice, gastrointestinal nematodes have been perceived to be of minor importance when compared with *Dictyocaulus*. In the past a management programme that controlled lungworm has usually controlled gastrointestinal worms as well.

Recently however, gastrointestinal worms, and the *Ostertagia*-like abomasal worms in particular, have made a comeback in importance. *Apteragia*, *Spiculopteragia*, *Rinadia* and *Skrjabinagia* (see Table 4.1) are all *Ostertagia*-type nematodes. The gastrointestinal nematodes are similar in appearance and have similar life cycles to their counterparts in sheep and cattle.

There is little information available on the relationship between faecal egg counts and gastrointestinal worm burdens in deer, but all the indications are that neither faecal egg counts nor plasma pepsinogen levels give a reliable indication of worm burden.[7,19,32] In practice this means that gastrointestinal worm burdens in deer cannot currently be estimated reliably in live animals.

There is now both anecdotal and experimental evidence that comparatively low abomasal worm burdens can have a significant effect on production.[19,32,33,34,35] For example, ill thrift was investigated in mixed age red hinds in Canterbury. Two thin hinds were necropsied. They were grossly emaciated, and the abomasum of each had a 'Morocco leather' appearance. The laboratory report confirmed parasitic gastritis. Abomasal worm burdens of these hinds were elevated, but not high in sheep terms, ranging between

1500 and 7200 adult worms and 50 and 1700 fourth stage larvae. Other deer in the mob responded to appropriate drenching.[32]

A form of type II ostertagiasis has recently been reported from farmed red deer in the U.K.[36] Anecdotal evidence suggests a similar condition may be found in New Zealand.

The effects of nematodes in farmed deer are similar to those in other ruminants, leading to ill thrift and in some cases death.

Wapiti and wapiti hybrids appear to be more severely affected by abomasal nematodes.[34] One explanation that has been proposed is that wapiti come from North America, at the eastern end of the circumpolar range of *Cervus elaphus*. When their ancestors left Asia and entered America they appear to have left their gastrointestinal parasites behind. Since then wapiti have become isolated in North America and lost their ability to cope with these nematodes. Be this as it may, wapiti do seem to be more susceptible.

Although not identified here, a similar range of species has been recorded from fallow deer in Australia,[37,38] and would be expected to occur in fallow deer here.

Although plasma pepsinogen levels do not appear to be useful as an indicator of parasitism in red deer, the situation may be different in fallow deer. In an investigation in Germany, plasma pepsinogen levels in helminth free and naturally raised farmed fallow were measured at 4 weekly intervals from birth to 11 and 15 months respectively. The mean level at birth was 0.708 units tyrosine/l which decreased until 14 weeks of age in both groups. Thereafter, it levelled out at around 0.2 units tyrosine/l in the helminth free group, but gradually increased from the 18th week in the naturally raised group to around 0.8 to 1.0 units tyrosine/l in response to worm infestation, significantly higher levels than the helminth free group.[39]

'Fading elk syndrome'

The term 'fading elk syndrome' is used to describe a condition of chronic stress and ill thrift in wapiti and wapiti/red deer hybrids. The state of current knowledge was reviewed by Waldrup and Mackintosh.[40] The principal sign of the condition is elevated abomasal pH which has a negative influence on

both copper uptake and the effectiveness of oral anthelmintics, and reduces digestive efficiency. They postulate that the change in abomasal pH results from parasitism of the abomasal wall by fourth stage larvae of an *Ostertagia*-type nematode. Early treatment with double the recommended dose rate of ivermectin pour on for cattle seems to be effective in some cases. Further work on this condition is in press.

Efficacy of anthelmintics against gastrointestinal worms

Earlier work on the efficacy of anthelmintics concentrated on the efficacy against lungworm in red deer. More recently, there has been a shift towards looking more closely at efficacy against gastrointestinal worms and incorporating wapiti and wapiti x red hybrids. This has happened because of the improved availability of wapiti type stock and because of the health problems that have occurred in some of these animals.

The summary of lungworm drenching studies in red deer in the mid 1980s put anthelmintics into three categories:[41]

- diethylcarbamazine, levamisole and cambendazole had low activity;
- mebendazole, albendazole, oxfendazole, fenbendazole and febantel had moderate to good activity; and
- oral ivermectin (200 µg/kg) had very good activity.

After further work using injectable (200 µg/kg) and pour on ivermectin (500 µg/kg) at cattle dose rates,[16,19] the guidelines for where the risk of reinfection with lungworm was high were:

- use second generation benzimidazoles at 21 day intervals,
- use oral ivermectin (200 µg/kg) at 4 weekly intervals,
- use injectable ivermectin (200 µg/kg) at 5 weekly intervals, and
- use pour on ivermectin (500 µg/kg) at 7 weekly intervals. Subsequently, moxidectin pour on (500 µg/kg) and eprinomectin pour on have demonstrated similar or better activity against lungworm.

These guidelines are still applicable, but on most farms drenching need not be as frequent. Note however, that oral and injectable ivermectin are not licensed for use in deer.

Recent work on anthelmintic efficacy becomes somewhat more complex.[19,32,33,34,35,42,43,60,61] In most investigations lungworms continue to be susceptible to anthelmintics except in two reports:

- In the first case larvae were still found in faeces for a few days after treatment.[42] This is not evidence of ineffectiveness because it takes up to 5 days for larvae and eggs to leave the body of the host after treatment.
- The second case immature lungworm larvae were recovered 7 days after drenching with ivermectin pour on at 1500 μg/kg (3 times the cattle dose rate).[34] This finding cannot be explained. The persistent effect of ivermectin should have stopped reinfection occurring so quickly after treatment. This may be evidence for ivermectin resistant *Dictyocaulus*, or may indicate that the immature stages are less susceptible to ivermectin than the adults. Further investigation is needed.

While both moxidectin and ivermectin pour ons are effective against adult *Ostertagia*-like worms in the abomasum at 500 μg/kg, the dose of ivermectin (but not moxidectin) needs to be doubled to kill the fourth stage larvae (L_4) of the abomasal *Ostertagia*-like worms. In one investigation ivermectin pour on was administered to wapiti hybrids at 1500 μg/kg.[34] Although it was stated that this is routine practice, no data were presented to support the need to increase the dose rate to this level.

Boluses were used in two investigations:

- A morantel bolus protected wapiti hybrids against ill thrift, but if deer were treated with ivermectin pour on at 1000 μg/kg at the time the bolus was given the bolus gave no added benefit.[33]
- An albendazole bolus did not completely protect treated red deer from reinfection with nematodes, but did suppress production of live lungworm larvae and protect from loss of weight.[35]

Most of the publications cited above indicate that relatively low burdens of abomasal worms can cause ill thrift. It is interesting therefore to see a paper from the U.K. which reported very high worm counts, but then went on to state "No adverse effects of parasitism were observed in the adult deer in any of the three years, ...".[43] Are these the same species of worms that are causing ill thrift in adult deer in New Zealand?

The key points that emerge from all this are:

Drench efficacy varies with:

A. availability of the anthelmintic in the host and persistance of the anthelmintic.
   - white drenches
   - milbemycin/avermectin pour ons

B. target species and life history stage
   - lungworm (*Dictyocaulus*)
   - adult gastrointestinal worms: ivermectin or moxidectin pour on at 500 µg/kg, or albendazole oral or bolus (other benzimidazoles may be equally effective)
   - L_4 abomasal worms: ivermectin pour on at 1000 µg/kg, moxidectin pour on at 500 µg/kg, or albendazole oral or bolus (other benzimidazoles may be equally effective)

C. reinfection risk to stock - depends on management, climate and weather

D. drench resistance (no published reports from deer, but expected)

Initially we had assumed that drenches that worked in sheep and cattle would also work in deer. We soon found out however, that this was not the case. The extreme case was levamisole, a valuable drench in sheep and cattle. We found that it was not effective against lungworm in deer because it was broken down too quickly by the deer.[7]

Deer are not sheep or cattle. The efficacy, toxicity and metabolism of any pharmaceutical product, be it a drench or some other product, has to be established independently in deer.

Our knowledge of the behaviour of drenches in sheep and cattle has grown out of a large number of trials that have been carried out over many years. Deer have not been around as a farmed animal for very long and during this time it has not been easy to get stock for trial work. Further critical investigations are needed to unravel what is occurring with drenches in deer.

Protozoa

Coccidia (Eimeria spp.)

Unidentified oocysts of the *Eimeria* type were recovered in small numbers

from red deer on 27 farms and fallow deer on one farm during a 1981 deer farm survey.[6] They usually have no clinical effect, but may cause disease in stressed animals. A seven month old Père David's X red deer calf that had been unthrifty since birth died.[44] At necropsy the only abnormality was thickening of the large intestine, which showed acute catarrhal colitis associated with severe coccidiosis.

Toxoplasma

Toxoplasma is found throughout New Zealand. It would be expected periodically to cause problems in deer. It has been reported from feral red deer from the Rotorua area and the heart blood of an aborted deer foetus.[45,46]

Sarcocystis

Sarcocystis was recovered from 25 of 75 feral red deer from the Rotorua area and transmission from red deer to dogs has been demonstrated.[47,48,45] Its distribution is unknown but it could be widespread. The species concerned has not been identified. This organism has not proven to be a problem in farmed deer.

Cryptosporidium

Cryptosporidium has been reported as causing disease in young deer, primarily hand reared calves.[49] *Cryptosporidium* and rotavirus are the commonest causes of diarrhoea in deer calves up to 3 weeks old and recur as causes of death and disease.[10,50,11]

Neospora spp.

Neospora spp.-like lesions have been associated with an aborted deer calf in two cases.[46]

Tape worms

Moniezia spp.

Moniezia expansa has been recovered from farmed red deer calves, and Moniezia eggs from both red deer and fallow deer.[6,51] This parasite is likely to be widespread, but does not appear to cause any clinical disease.

False hydatids (Taenia hydatigena)

Natural infections with false hydatids cysts, or 'cysticercus tenuicollis', the larval stage of *Taenia hydatigena*, have been found in feral red deer, wapiti, fallow and farmed red deer.[52,53,54,13] Deer become infected by ingesting eggs passed by infected dogs. The developing cysticerci migrate through the liver and mature in the abdominal cavity.

There are generally no clinical signs of infection, although at slaughter there may be haemorrhagic tracts through the liver caused by the migration of developing cysticerci. In an unusual case however, a young red deer calf which died with hepatitis cysticercosa.[54] Prior to death the calf was described as being moribund with subnormal temperature, cold extremities, pale mucous membranes and severe abdominal pain.

Flukes

Liver fluke (Fasciola hepatica)

The common liver fluke *Fasciola hepatica* has been reported from feral red deer.[53] It is a common parasite of red deer in other parts of the world and has the potential to be a problem here on farms in endemic areas. Fortunately, reports suggest that red deer can tolerate higher *F. hepatica* burdens than can cattle. Triclabendazole is effective against *F. hepatica* in fallow deer and red deer.[55]

Paramphistomes

Calicophoron calicophorum (syn: *C. ijimai*) has been identified from red deer, and paramphistome eggs have been found in fallow deer faeces. No treatment is usually necessary.

Concluding remarks

Although this review has concentrated on the use of pharmaceutical products to control parasites, it is important to realise that drug resistant parasites will eventually appear in deer. So, when possible it is best to use non-drug tools in parasite control programmes.

This review makes it clear that there is a lot we do not know about parasites of farmed deer and their control. Whether much more information will be available in future depends on availability of funds for the necessary research.

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