GASTROINTESTINAL HELMINTHOSIS IN FARMED FALLOW DEER

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Introduction

Fallow deer (*Dama dama dama*) have been farmed in Australia for over 15 years now, with the species providing about 50 percent of the total number of over 100,000 farmed deer in the country (Cribb 1989). In that time there have been only a small number of accounts of the presence of nematode parasites in farmed fallow deer, with very little evidence of any clinical effects of such burdens.

English (1981) reported that only 2 of 6 (33%) deer from a wild fallow deer population in southern New South Wales carried the abomasal parasite *Spiculopteragia asymmetrica*, with relatively few parasites (180 and 250) per deer. The same nematode was present in 26/28 (93%) of wild fallow deer examined in England by Batty *et al* (1987), with burdens also very small – from zero to 683 with a mean of 250 worms. Presidente (1981) reported that *S. asymmetrica* was the most prevalent nematode in farmed fallow deer in Victoria, with abomasal worm burdens usually below 2000. However, mortality occurred when mixed populations exceeded 5000 nematodes. The other nematodes found in fallow deer were *Skrjabinagia kolchida* (28% prevalence) and *Ostertagia* spp (47%). This confirmed an earlier conclusion that *S. asymmetrica* is the most frequently encountered nematode in Australian deer, with the widest host range (Presidente, 1979).

There was therefore the obvious potential for the abomasal nematodes, which were apparently unlikely to cause problems in wild fallow deer, to reach potentially pathogenic burdens in farmed deer – probably when overstocking and under-nutrition were allowed to occur, as is the case with other ruminants (Charleston 1980). Presidente (1988) was able to note that there had in fact been few reports of clinical disease or deaths in farmed deer in Australia attributed to parasitism, with low faecal egg counts (FEC)(less than 100 eggs per gram EPG) and light abomasal worm counts (less than 500) being common. There is a recent report from Great Britain of Type 2 ostertagiasis in young red deer stags, with very high worm burdens – up to 14,600 adults and over 100,000 inhibited 4th stage larvae (Connan 1991), which confirms the ability of helminths to seriously affect farm deer.

There is little information available on the relationships between worm burdens, FEC and clinical signs of helminthosis in fallow deer. The only reports on the use of anthelmintics in fallow deer are the treatment of wild populations with medicated pellets containing thiabendazole or fenbendazole, the intake of which was not determined (Kalivoda and Chroust 1971;Enigk and Dey-Hazra 1976; Bockeler and Segebade 1977). The general assumption has been that anthelmintics at dose rates commonly used for cattle and sheep would be equally effective against the helminths of fallow deer (Presidente 1984).

Case report

It was against this background that ill-thrift with diarrhoea and deaths occurred in weaner fallow deer on a commercial farm near Camden NSW (Mylrea *et al* 1991). The herd on

this farm had been increasing in numbers for over 10 years, with most breeding stock derived from the wild population in southern NSW (English 1981). The weaning of fawns pre-rut had been adopted as a management practice for a number of years, with 150 fawns weaned in April 1989 onto improved pasture at a stocking rate of 15 deer per hectare. A pelletted concentrate was offered *ad libitum* from mid-May. While the majority of the weaners thrived, a number of the smaller fawns showed clinical signs of weight loss and diarrhoea, and about 15 died.

The average FEC on 10 well grown fawns in late May was 40 EPG (range 0 to 90) whereas the average count for the clinically affected group was 323 EPG (range 0 to 840). All of the weaners had been treated twice with 3.8 mg/kg albendazole (Valbazen Broad Spectrum Sheep, Lamb and Goat Drench (R) Smith-Kline), once at weaning and again 4 weeks later. There was little obvious effect from the second treatment, with ill-thrift and deaths continuing.

As a result, 5 weaners were presented for necropsy (2 were moribund and were euthanased).

Necropsy results

Helminthosis was diagnosed as the cause of death in 4 of the deer, on the basis of clinical signs, gross damage to the abomasal mucosa and the presence of significant numbers of parasites. The abomasal worm counts ranged from 1000 to 2800, with 75 % being *S. asymmetrica,* 13 % *Ostertagia ostertagi*, 8 % *S. kolchida* and 4 % *Haemonchus contortus*. The FEC at necropsy ranged from 80 to 380 EPG. Abomasal damage included varying degrees of hyperaemia, oedema, and pitting and thickening of the abomasal mucosa. Low burdens (less than 20 adults) of the lungworm *Dictyocaulus viviparus* were found in the trachea and bronchi of 4 of the deer, but there appeared to be no clinical significance to this finding. A similar conclusion was drawn on the finding of very small numbers of *Oesophagostomum venulosum* and *Trichuris sp* in the caecum and large intestine respectively of 2 of the 4 deer.

Faecal egg count reduction test

Subsequent to these necropsies, a faecal egg count reduction test (FECRT) was carried out on 42 six-month old fallow deer on the same farm. Early in June, the deer were weighed and fresh faecal samples were collected for FEC. The deer were allotted into 4 groups, with each group having approximately the same weight (20.9 to 21.9 kg) and mean FEC (250 to 323 EPG). One group of 9 deer received no treatment, with the other 3 groups of 11 deer being given either 3.8 mg/kg albendazole, 7.5 mg/kg albendazole or 0.2 mg/kg ivermectin (Ivomec Liquid for Sheep (R) MSD AgVet) orally.

Thirteen days after treatment a second faecal sample was collected and examined for nematode eggs. Based on faecal sample reduction percentages, albendazole at either 3.8 mg/kg or 7.5 mg/kg did not reduce nematode egg output in these deer, while ivermectin (0.2 mg/kg) reduced egg output by 43 %.

All deer were subsequently treated on Day 13 with 1 ml avermectin (Avomec Antiparasitic Injection for Cattle (R) MSD AgVet) subcutaneously. Two deer were

slaughtered 47 d after avermectin treatment for worm counts. Both animals contained worms and had gross pathological changes in the abomasal mucosa. Deer 1, which had been in the ivermectin group, had a FEC of 360, 80, 0 and 0 at 13, 26, 53 and 60 d after initial treatment. There was mild hyperaemia of the abomasal mucosa and an abomasal count of 100 *S. asymmetrica* was recorded. Deer 2, which had been treated initially with 7.5 mg/kg albendazole, had a FEC of 0, 120, 0 and 0 EPG on days 13, 26, 53 and 60 after initial treatment. The worm count from the abomasum was 4500: 86 % *S. asymmetrica* and 14 % *O. ostertagi.* The mucosa of the abomasum was hyperaemic, oedematous and thickened. The presence of worm eggs at Day 13 in the FECRT indicated that the treatments failed to totally eliminate the infection. The presence of worms in the 2 weaners necropsied 47 d after a second treatment was probably due to re-infection, but negative egg counts in these deer 53 and 60 d after initial treatment suggests that suppression of egg production may also have occurred.

Failure of anthelmintic treatment

There is an obvious need to determine the reasons for the failure of the anthelmintic treatments in these fallow deer. There are a number of possible explanations, including insufficient dose, poor absorption of the drugs, or parasite resistance.

Results from 2 studies with red deer (Mackintosh et al 1985; Watson and Manley 1985) suggested that deer are able to metabolize and excrete benzimadazole compounds more quickly than sheep and cattle. A subsequent field trial by Bowie et al (1987) supported this probability, with the resumption of shedding of *D. viviparus* larvae by red deer calves 4 weeks after oral treatment with oxfendazole at 4.5 mg/kg (Synanthic (R) Syntex), compared to deer treated with oral ivermectin at 0.2 mg/kg (Ivomec (R) MSD), which did not resume larval shedding for over 5 weeks after treatment. The mean larval output and the proportion of deer shedding larvae at 27 and 33 days after treatment were significantly lower in the ivermectin treated group. Poor efficay of injectable ivermectin in red deer hinds has also been reported (Andrews and Lancaster 1988). It therefore seems likely that the optimal dose rates in deer may be higher than those recommended for sheep and cattle, with factors such as abomasal pH, oesophageal groove function, malnutrition and worm burden having been identified in sheep as factors which may affect the absorption and efficacy of anthelmintics (Marriner and Bogan 1981; Pritchard 1980).

Slaughter trial

A slaughter trial was conducted in 1990, to assess the efficacy of albendazole and ivermectin against *O. ostertagi* in fallow deer weaners. Rolfe, Mylrea and English, unpublished). The major aim was to pursue the dose-response relationship of the 2 classes of anthelmintics.

A group of 60 male fallow deer were weaned on 1 April 1990, and transported to indoor pens in an animal house at the Elizabeth Macarthur Agricultural Institute (EMAI), Menangle, NSW. The deer were weighed and treated with 15 mg/kg albendazole (Valbazen (R)), with FEC determined 6 days later. The deer were immunosuppressed on 9 April with 2.5 mg dexamethasone, and all were then dosed orally with third-stage larvae of *O. ostertagi,* with 8000-10,000 larvae on 12 April and a further 5000 larvae 18 days later.

On the basis of FEC and bodyweight on 3 May, 57 weaners were allocated into 11 groups: 5 albendazole treatments (at 2, 4, 8, 16, and 32 mg/kg), 5 ivermectin treatment groups (at 50, 100, 200, 400, and 800 ug/kg), and 1 control group. Treatments were given on 10 May.

With a view to obtaining information on the metabolism of the anthelmintics, 3 groups were selected for blood sampling (4 and 8 mg/kg albendazole and 200 ug/kg ivermectin). Beginning at 0800 on 10 May, samples of venous blood were obtained by jugular venepuncture. In the first 24 hours 6 samples were taken at 4 hour intervals, and over the next 12 hours 2 were taken at 6 hour intervals. From 60 to 160 hours 5 samples were taken at 20 hour intervals, and a final sample was taken at slaughter at 168 hours.

Faecal samples were collected per rectum at the time of treatment and again 7 days later. The deer were killed on 17 May, with the abomasa removed for total worm counts.

The deer were fed *ad libitum* during the experiment on a mixed ration of rabbit pellets, lucerne pellets and dairy meal, offered in plastic troughs. The diet provided 7 MJME/day and 10–12 % crude protein. The concrete floor of the pens was covered with a deep bed of sawdust, after 3 animals developed septicaemia due to *Fusobacterium* infection of forelimb abrasions. After this was done there were no further problems, with the deer thriving in the environment provided.

The results of the study were less than satisfactory, in that most of the weaners failed to establish significant populations of *O. ostertagi.* The FECRT conducted at 7 days after treatment indicated that all the anthelmintic treatments suppressed egg production by gravid females, at all dose rates. However, the untreated controls also showed a decline in EPG over the same period, with the probability that natural resistance to the helminths was coming into effect. This makes it very difficult to interpret the results of the FECRT, with wide variability in the EPG of the control animals.

At slaughter it was evident that the abomasal mucosae had been damaged during the trial, but negligible abomasal worm counts in all deer (even the controls) supported the concept of resistance and tissue repair, and correlated well with the results of the FECRT. The largest burden (3675) in an untreated deer represented an establishment of only 28.2 % of the parasites administered to the animal.

The serum samples have not been examined as yet, given the inconclusive results of the study. However, it is possible to speculate on the reasons for the failure of the study to produce useful results.

1. The development of acquired immunity prior to and during the trial.

2. There may be high efficacy of both drugs against *O. ostertagi.* The study produced no information on the most common deer nematode, *S. asymmetrica.*

3. The high plane of nutrition did not simulate the field conditions under which helminthosis had occurred, with all deer eating well and putting on weight. Clearly, any further attempt to set up such a slaughter trial will need to address these issues. It will be very difficult and expensive to produce a large group of fallow weaners reared as worm–

free from birth. It will also be more difficult to produce large numbers of larvae of deer nematodes such as *S. asymmetrica,* on the same basis as the larvae of sheep and cattle parasites are generally available. The least difficult aspect will be to manipulate the nutrition of the animals to simulate field conditions – subject only to animal welfare concerns. Perhaps the most gratifying result from the study was the way in which the deer adapted to the pen environment, and to the feed which was offered. The reputation of fallow deer (particularly young, untrained deer) as very nervous animals would have suggested the opposite, and it does seem that they can be used in such trials with ease, given care and common sense.

Conclusion

There is ample evidence that farmed deer can be seriously affected by helminth parasites, with lungworm in young red deer being perhaps the best example of this fact. Nonetheless, if the principles and methods which have been developed for cattle and sheep are applied to deer, significant losses can be avoided. The strategic and tactical use of anthelmintics has a part in such control programmes, but the effects of poor management, and undernutrition, cannot be over-emphasized. In the case of fallow deer weaners it has become clear that helminthosis is entirely possible if care is not taken with their management. On the other hand, it is obviously possible to totally avoid problems if the following measures are adopted:

1. Adopt good feeding policies for lactating females, to ensure that all fawns are well above a minimum weight of 15 kg when weaned in mid March. The fawns most likely to succumb to heavy worm burdens are the small, probably late born individuals. If the bucks are withdrawn from the females in mid June there should be no fawn born after the end of January anyway, but it is possible in a hard, dry year for fawns born in late January to be too light to wean with confidence, unless adequate supplementary feed is made available to the females.

2. If there is a great variation in bodyweights of fawns at weaning, it would be wise to consider forming 2 groups, with the smaller individuals being given preferential feeding, and an opportunity to avoid the pecking order problems at feed troughs which can occur in large groups.

3. Feed the weaned deer to their requirements, with conserved high quality forages or supplementary feeds. Introduce them to the feed while still on their mothers – they will learn to eat the feed before weaning and the transition will be much smoother. It is desirable to weigh young deer several times during their first year of life, to verify that they are growing well.

4. Avoid exposing young deer to pastures known to have been contaminated with high levels of helminth larvae. In fact, unless adult fallow deer have been run at very high stocking rates in a season very favourable for the survival and translation of helminths on pasture, this should rarely be a problem. When deer and other ruminants are farmed together the possible problems from parasites from the latter cannot be ignored.

5. The indiscriminate use of anthelmintics should be avoided. The use of FEC to decide whether a treatment is required is rational, as is the use of a FECRT to monitor efficacy. Total worm counts on slaughtered deer are a useful further measure to determine the parasite status of a group of deer.

References

Andrews, S.J. and Lancaster, M.B. 1988. Use of ivermectin in deer. *Veterinary Record* **123:** 354.

Batty, A.F., Chapman, D.I. and Chapman, N. 1987. Prevalence of nematode parasites in wild fallow deer (*Dama dama*). *Veterinary Record* **120:** 599.

Bockeler, W. and Segebade, R. 1977. Testing the efficiency of Panacur (fenbendazole) against the gastrointestinal nematodes of fallow deer. *Tierarztliche Umschau* **32:** 473–474, 476–478.

Bowie, J.Y., Macintosh, C.G. and Mason, P.C. 1987. Effect of ivermectin and oxfendazole on shedding of larvae of the lungworm *Dictyocaulus viviparus* by red deer (*Cervus elaphus*). *New Zealand Veterinary Journal* **35:** 8–10.

Charleston, W.A.G. 1980. Lungworm and lice of the red deer (*Cervus elaphus*) and the fallow deer (*Dama dama*) – a review. *New Zealand Veterinary Journal* **28:** 150–152.

Connan, R.M. 1991. Type II ostertagiosis in farmed red deer. *Veterinary Record* **128:** 233–235.

Cribb, J. 1989. *Australian Agriculture*. Volume 2, 1989/90. National Farmers Federation, Australia. Morescope Pty Ltd, Camberwell.

English, A.W. 1981a. The diseases of deer in New South Wales. *Wildlife Diseases of the Pacific Basin and Other Countries*. Fowler, M.E. (Ed.) Wildlife Disease Association. pp 200–204.

English, A.W. 1981b. The capture of wild fallow deer in New South Wales using a baited enclosure_trap. *Australian Deer* **6:** 13–20.

Kalivoda, J. and Chroust, K. 1971. Anthelmintic effectiveness of thibenzole administered in pelleted feeds to fallow deer and mouflons. *Acta vet Brno* **40:** 453.

Mackintosh, C.G., Mason, P.C., Manley, T., Baker, K. and Littlejohn, R. 1985. Efficacy and pharmacokinetics of febantel and ivermectin in red deer (*Cervus elaphus*). *New Zealand Veterinary Journal* **33:** 127–131.

Marriner, S.E. and Bogan, J.A. 1981. Pharmacokinetics of oxfendazole in sheep. *American Journal of Veterinary Research* **42**: 1143–1145.