STUDIES OF SOME ASPECTS OF GASTROINTESTINAL NEMATODES AND *DICTYOCAULUS VIVIPARUS* OF FARMED RED DEER

A thesis presented in partial fulfilment of
the requirements for the Degree of
Master of Veterinary Science
at Massey University

Mark Vere Anderson
1985

Abstract of a thesis presented in partial fulfilment
of the requirements for the Degree of
Master of Veterinary Science

STUDIES OF SOME ASPECTS OF GASTROINTESTINAL NEMATODES AND *DICTYOCAULUS VIVIPARUS* OF FARMED RED DEER

MARK VERE ANDERSON

Studies were carried out on aspects of the treatment and control of gastrointestinal nematodes and *D. viviparus* in red deer, under New Zealand farming conditions. In addition, the relationship between faecal egg count and gastrointestinal worm count was investigated.

In the first study an anthelmintic-impregnated supplementary feed treatment regime incorporating 200mg albendazole per kg of deer nuts (medicated nuts), fed to give 5mg albendazole per kg liveweight daily for three 10-day treatments with 21 day intervals between treatments, was given to eight weaner deer in the autumn. This treatment was compared with 3-weekly single oral administration of albendazole (10mg/kg) to eight similar deer under the same conditions (set-stocked in small pasture plots and given 2kg concentrate feed per head, per day) and a similarly treated group rotationally grazed on pasture alone. Reductions in faecal egg and larval counts were the same for all three groups, but reinfestation in spring was more rapid in the pasture-fed deer. Liveweights were the same for all groups throughout the experimental period from March to November. As an adjunct to this trial, medicated nuts were fed at the same dose-rate to 16 adult hinds. All faecal egg and larval counts were reduced to zero within 10 days. Thus, anthelmintic-impregnated concentrate feed is an effective

way of controlling gastrointestinal and lung nematodes in deer.

The above trial and another on a commercial property showed that high faecal larval counts may be found in weaner deer where deer are set-stocked from before calving up to weaning, or if parasite control programmes are delayed until six weeks after weaning in March. A single oral dose of albendazole at 10mg/kg was found to reduce D. viviparus faecal larval counts and gastrointestinal nematode faecal egg counts by approximately 99% seven days after treatment. Faecal larval and egg counts were usually slightly elevated 21 days after treatment, suggesting either a rapid reinfestation and short prepatent period of the parasites in deer and/or an efficacy of albendazole of less than 100% against immature stages of the gut and lung nematodes.

In a third study, cutaneous application of levamisole (20% W/V) at a dose-rate of 10mg levamisole per kg was found to be ineffective in reducing faecal egg or larval counts in a group of 23 red deer under two years of age.

The fourth study involved collection of abomasa and intestines of 46 deer sent to a deer slaughter premises. A faecal egg count was performed and the gastrointestinal nematodes were identified and counted. The largest gut parasite burdens were of Trichostrongylus axei with counts up to 12,900. Five deer-specific species of the tribe Ostertagiea, Spiculopteragia spiculoptera, Spiculopteragia asymmetrica, Ostertagia leptospicularis, Skrjabinagia kolchida and Skrjabinagia lyratiformis were also common (counts up to 2470). Few other parasites were found and numbers were low (0 to 90). The relationship between worm count and faecal egg count was described by the relationship: Total Worm Count = 18.8 (Faecal Egg Count) - 341. However, there were few deer with high worm and egg counts and this relationship must therefore be regarded as tentative until more work can be carried out in deer with high worm burdens.

ACKNOWLEDGEMENTS

This work was assisted financially by Smith Kline and French, New Zealand Ltd, who also supplied the anthelmintic and equipment required. The supplementary feedstuff was supplied by NRM Ltd.

I am indebted to Dr P.R. Wilson, who supervised this thesis, for his tolerance and good humour during its preparation. I also wish to acknowledge Dr W.A.G. Charleston for his helpful discussions and advice. I thank S. Thomas and S. Calder for laboratory technical assistance.

I wish to thank Massey University for the use of the deer unit, for the major part of this study and to Mr P. Whitehead, the farm supervisor and Mr C. Howl for on-farm assistance. The co-operation of Mr T. Kebbell and Mr R. Keenan on whose farms studies reported in chapters three and five were carried out is gratefully acknowledged. The manager and staff of the deer slaughter premises Hastings, are also acknowledged for their willing co-operation.

Finally, I wish to acknowledge the support and assistance of my wife Maureen during the preparation of this thesis.

TABLE OF CONTENTS

LIST OF TABLES

TABLE PAGE

LIST OF FIGURES

CHAPTER 1

REVIEW OF THE LITERATURE

LUNG AND GASTROINTESTINAL NEMATODES OF DEER.

1.1 Lungworm: Dictyocaulus viviparus.

1.1.1 Occurrence.

D. viviparus has been recorded from all deer species in New Zealand except Sambur (table 1.1).

Dictyocauliasis was believed to be the most important parasitic disease of farmed deer in New Zealand, (Charleston, 1980; Mason, 1979b,1981b) and Britain (McDiarmid, 1975). This was confirmed in New Zealand by a deer population and health survey (Gladden,1981). Dictyocauliasis was rated by deer farmers as the most frequently occurring deer health problem (28% of all problems) and by veterinarians as the second most frequent problem (21% of all problems).

The parasite in deer cannot be distinguished morphologically from D. viviparus that infects cattle, and cross-transmission to various deer species from cattle is possible (Presidente et al, 1972; Blaxter et al, 1974; Corrigall et al, 1980; Mason, 1981b). However, there is evidence that a deer-adapted subspecies exists (Gupta and Gibbs, 1971; Presidente et al, 1972; Presidente et al, 1973).

Deer appear to tolerate large burdens of D. viviparus while showing only mild or no clinical signs (McDiarmid, 1975; Corrigall et al, 1980). This has caused some authors to speculate that D. viviparus developed as a parasite of deer which has since become established in cattle (Presidente et al, 1973; McDiarmid, 1975).

Table 1.1

Species Of Nematode Gastrointestinal And Lung Parasites Reported In Various Species Of Deer In New Zealand, And Reference Indicated By The Numeral.

	RED	WAPITI	WHITE TAIL	FALLOW	SIKA	RUSA	SAMBUR
	Reference						
LUNGWORM							
Dictyocaulus viviparus	2	2	2	3	8	8	
GASTROINTESTINAL							
Abomasum							
Apteragia odocoilei			6				
Apteragia quadrispiculata	2			2			
Haemonchus contortus	7		6				
Ostertagia circumcinta	7		6				
Ostertagia leptospicularis	2						
Ostertagia ostertagi	9*		6				
Ostertagia trifurcata			6				
Rinadia mathevossiani	2	2					
Skrjabinagia kolchida	2						
Spiculopteragia asymmetrica	2	2	6	2	2		
Spiculopteragia spiculoptera	2	2	6		2		6
Trichostrongylus axei	7						
Small Intestine							
Capillaria bovis	2						
Capillaria species			6				
Cooperia curticei			6				
Cooperia mcmasteri	5	5					
Cooperia oncophora	5	5					
Cooperia pectinata	1						
Large Intestine							
Oesophagostomum	2	6	6	2	4		
Trichuris ovis	2						

* = Experimental infection only.

REFERENCES

(1) McKenna et al., (1981)
(2) Andrews (1964)
(3) Charleston (1980)
(3) Presidente (1979a)
(5) Mason (1981a)
(6) Andrews (1973)
(7) Anon (1979a)
(8) Wilson & Collier (1981)
(9) Johnston (1982)

1.1.2 Epidemiology.

There are differences in susceptibility to dictyocauliasis reported between the deer species. McDiarmid (1975) stated that fallow and sika deer were relatively resistant to infection, roe deer were very susceptible and red deer were frequently infected but do not often show clinical signs. There is however, no experimental or statistical data to confirm this suggestion. Charleston (1980) reported that some practising veterinarians in New Zealand considered that lungworm may be as serious a problem to fallow deer as it is to red deer. Charleston (1980) reported finding large numbers of lungworm at post-mortem, and clinical observations of large burdens in fallow deer.

D. viviparus in deer has a direct life cycle which is assumed to be the same as in cattle. Very little is known specifically about the epidemiology of D. viviparus in deer. It appears that the prepatent period in red deer can be as short as 20 days (Corrigall et al., 1980; Mason, 1981b), though in black-tailed deer it was recorded as 30 days (Presidente et al., 1973).

Assuming the life cycle in deer is similar to that in cattle, infective third stage larvae are ingested by the deer, exsheath in the small intestine and penetrate the intestinal wall. They migrate to the lungs in one to seven days whilst moulting to fourth stage larvae. Shortly after, they moult to immature adults, move into the bronchioles and mature to adults. Females start laying eggs and these are passed up the trachea and swallowed. The eggs hatch to first stage larvae before being passed out in the faeces. They then develop to second and ensheathed third stage larvae on the pasture (Mason, 1979a). The time needed to become third stage larvae after deposition on the pasture is temperature dependent; larvae of bovine origin have been reported to become infective in 26 days at 5^{o}C but in only 3 days at 25^{o}C (Rose, 1956).

The numbers of D. viviparus larvae on the pastures are believed to build up to a peak in the autumn similar to that seen with D. viviparus larvae on cattle pastures (Bacinsky, 1969). Little is known of the fluctuations of D. viviparus larval numbers on cattle pastures in New Zealand but it is thought that low numbers of larvae are on the pastures in spring and these infect the new-born calves. This leads to a build-up of larval numbers from early summer to a peak in autumn (Brunsdon and Adam, 1975). However, as red deer calves are born later in the year than bovine calves a different pattern of larval build-up is likely. However, despite the probable later build-up of larvae on pasture, the incidence of dictyocauliasis in deer in New Zealand has also been observed to peak in autumn (Anon., 1979b; 1980; Wilson and Collier, 1981). This same seasonal incidence has been observed in deer overseas (Bacinsky, 1969). Weather conditions can have a marked effect on larval survival (Duncan et al., 1979) although the microclimate is the most important factor affecting larval survival (Rose, 1956). This may explain why Jorgensen (1981) found that overwinter survival of larvae was inconsistent both within and between districts. Deer have been reported to excrete lungworm larvae as early as three months of age in March (Mason, 1981b) and this infection is considered to have originated from the hinds (Wilson, pers. comm.).

Adult deer frequently carry lungworm (Kummereje, 1977; Wilson, 1981) and are a potential source of contamination of pastures. Another possible source of larval contamination of the pasture in spring is the continued development of arrested early fifth stage larvae carried by the previous years weaners. These have been observed in cattle (Oakley, 1977; Jorgensen, 1981) and it is suggested that they may also occur in deer (Mason, 1979a; McAllum, 1979). Kummereje (1977) considered the resumption of development of inhibited larvae in spring to be responsible for clinical dictyocauliasis seen in reindeer in Norway.

Another possible source of infective larvae for calves is larvae which have overwintered on the pasture. Bovine-origin D. viviparus larvae are known to overwinter (Rose, 1956; Blaxter and Allan, 1977; Oakley, 1977; Nelson, 1977; Duncan et al., 1979). Larvae are thought to survive in faecal pats (Oakley, 1977; Duncan et al., 1979), and have also been recovered from the soil (Mason, 1977). Pastures have been found to remain infective with cervine-origin D. viviparus larvae for at least three months over summer (Presidente et al., 1973).

It appears that deer develop a resistance to D. viviparus (Mason, 1981b), though Charleston (1980) suggested that they may have more difficulty in doing so than cattle. It has been suggested that resistance is age-related and not dependent on prior exposure to the parasite (Corrigall et al., 1980). These authors were unable to establish a patent infection in two 13-month-old parasitologically naïve deer dosed with 180 infective larvae per kg., or in two similar 20-month-old deer with 420 infective larvae per kg. However, patent infections were established in younger animals. Mason (1981b) reported that some red deer calves showed signs of resistance to D. viviparus at seven months of age after natural exposure. Most farmed adult deer carry low lungworm burdens (Wilson, 1981) so it appears that resistance has its effect in limiting the size of a lungworm burden rather than preventing the establishment of a burden per se. (Wilson, pers. comm.).

1.1.3 Pathogenicity.

There is little information on the effect of various lungworm numbers in deer. Deer which have died of dictyocauliasis normally have very large numbers of lungworms in a frothy exudate in the pharynx, larynx, trachea and lower bronchial tree (Wilson, 1979a; Corrigall et al., 1980). Corrigall et al., (1980) reported a chronic gradual weight loss in a seven-month-old deer with lungworm found in the larynx, trachea and bronchi

at post-mortem. In a nine-month-old deer on the same property a burden of 1000 worms resulted in no clinical signs and only a depressed weight gain. Kummereje (1977) reported an acute syndrome in reindeer in which over 100 deaths occurred in two herds in Norway because of a concurrent infection of D. viviparus and Pasteurella multocida. D. viviparus infestation may predispose the deer to infection with P. multocida.

No relationship has been established between the number of adult lungworm and faecal larval count (FLC). Wilson (1981) reported a FLC of 69 larvae per gram with a D. viviparus burden of 3500-4000 in one individual, but the percentage of immature worms was not established.

The FLC at which treatment should be initiated has not yet been established. Mason (1977) considered that since knowledge of the pathogenesis of D. viviparus is lacking, treatment of deer with even a low FLC is desirable. What constitutes a "low" count was not explained.

1.1.4 Clinical Signs and Diagnosis.

Diagnosis of lungworm infections is usually based on a combination of faecal examination for lungworm larvae, clinical signs and history, but may be aided by post-mortem examination of severe cases.

Disease and death due to D. viviparus is most commonly seen in young animals between 3 and 18 months of age particularly during the autumn and winter (Wilson, 1979a; Charleston, 1980; Wilson and Collier, 1981). It appears that the more serious problems are generally associated with intensive farming conditions and high stock densities. Disease may also be seen in newly captured stock of any age, presumably because of a poor resistance due to limited prior exposure to the parasite and the stress of capture (Wilson and Collier, 1981). Corrigall et al., (1980) and Presidente et al., (1973) also suggested that stress was important in the establishment of D. viviparus in deer.

Clinical signs recorded include loss of condition, retarded growth rates, roughened coats and an occasional soft bronchial cough (Wilson, 1979a). Presidente *et al.*, (1973) recorded loss of condition, hyperpnoea and coughing in black-tailed deer calves with natural infections, and Corrigall *et al.*, (1980) noted loss of condition, and occasional coughing and dyspnoea in red deer. He also noted that the typical paroxysmal coughing seen in bovine calves with dictyocauliasis was not seen in deer with heavy lungworm burdens. Thus clinical signs often are not particularly noticeable so the first sign of a severe infection is sometimes the sudden death of an animal (Wilson, 1979a; Charleston, 1980).

1.1.5 Treatment and Control.

It is considered that once *D. viviparus* is established on a property it is impossible to eradicate (Mason, 1979a). Recommendations for the control of lungworm vary. Mason (1979a) recommended drenching at weaning, moving to "clean" pasture, and then 3-weekly drenching until winter, if rotationally grazed, or until one year of age if the deer were set-stocked. McAllum (1979) recommended drenching young stock from weaning to 15 months of age at 3-weekly intervals, with a move to "clean" pasture after each treatment. The advisability of moving weaners after drenching to minimise reinfection rates was emphasised by Charleston (1980). Wilson (1981) suggested that the interval between drenching should be determined by the efficacy of the drench against the various stages of the parasite in the host. If the drench was effective against all stages then a 21 day interval should be sufficient. However, if it was not effective against immature stages then a shorter drenching interval should be used.

To try to limit the initial infection of calves Wilson (1981) recommended pre-calving drenching of the hinds. However, the value of this has not been proved, nor is the practice universally accepted. More recent data suggests that this may be of little use (Wilson,

pers. comm.). Mason (1979a) considered that adult deer do not need drenching because of their low larval output.

Few, if any, controlled trials on drench efficacy against D. viviparus involving slaughter of deer have been carried out. Information on efficacy is therefore either from clinical observations or faecal larval output suppression trials, usually without control groups.

Presidente et al., (1973) reported from black-tailed deer, that levamisole at 16 mg/kg produced only a transient decrease in larval counts and that cambendazole at 40-50 mg/kg removed the mature worms but did not have a significant effect on the immature stages. McAllum (1976) reported the deaths of two 10-month-old red deer calves attributed to dictyocauliasis an unspecified time after receiving three drenches of levamisole at 13 mg/kg. Other animals in the group then received intramuscular diethylcarbamazine citrate (DEC) at 2.5 ml/ 50 kg for three successive days. At post-mortem examination three weeks later no worms were seen in the lungs of these deer. Wilson (1979a) stated that DEC was reported to prevent deaths after clinical outbreaks and appeared to be effective in the control of lungworm; it was commonly used in New Zealand at that time. Scottish workers administered DEC intramuscularly at 1.2 ml/ 21 kg daily for three days following a natural infection, and recorded a zero FLC after six days (Corrigall et al., 1980). Fenbendazole was also used and considered to be effective (Corrigall et al., 1980). Fluids, antibiotics and vitamins were given in addition by these workers to clinically affected animals.

A 1980 survey of veterinary practices, (Wilson and Collier, 1981) indicated that oxfendazole was the most popular anthelmintic for the prophylactic control of lungworm, and DEC for the treatment of clinical outbreaks. Other anthelmintics used in order of popularity included fenbendazole, levamisole, cambendazole, albendazole, mebendazole and febantel. Many veterinarians also gave supportive therapy, including "Bisolvon" (bromhexine hydrachloride, Boehringer Ingelheim), antibiotics and high energy supplementary feeds when treating clinical

cases. A survey of deer farmers (Mason and Gladden, 1983) showed the following drenches used in order of popularity; oxfendazole, albendazole, fenbendazole, levamisole, cambendazole, febantel, mebendazole, thiabendazole and DEC.

In a report on the investigation and treatment of a field infection, Wilson (1981) recorded that albendazole significantly reduced FLC's after three days but stated that it may well not be 100% effective against immature stages. Cambendazole (25 mg/kg) appeared to be effective against mature lungworms but ineffective against immature parasites.

Mackintosh and Mason (1983) reported a slaughter trial in which febantel (7.5 mg/kg) and ivermectin (200 mg/kg I/M) were given to groups of five red deer calves. Seven days post-treatment, febantel was found to be 85% effective against immature stages and 99.7% effective against adults when compared with controls. Ivermectin was found to be 100% effective against both mature and immature stages.

The reasons for the apparent lack of efficacy of some anthelmintics against D. viviparus in deer are speculative. It has been proposed that it may be due to a different method of metabolism of anthelmintics by deer (Charleston, 1980). It may be that some anthelmintics are unable to achieve effective plasma concentrations for sufficient time because of a more rapid metabolism of the drug by deer. This suggestion was supported by Mason (1982) who measured serum levamisole levels in deer after oral and subcutaneous administration. In both cases the peak serum concentrations occurred within 16 minutes (cf. 1-2 hours in cattle), and then decreased rapidly. A similar result was obtained with oxfendazole, with both a lower peak plasma concentration and shorter duration in the blood-stream (Watson et al., 1983).

Pritchard et al.,(1978) stated that giving anthelmintic in doses on consecutive days can greatly increase its effectiveness in sheep. Charleston (1980) suggested that this principle may account for the

apparently good efficacy of DEC against D. viviparus, since this drug was given daily for a three day course.

Deer have been observed to continue to die of lungworm during clinical outbreaks of dictyocauliasis despite anthelmintic treatment. Wilson (1979a) suggested that this may be because of the severity of the parasite burdens and failure of the drug to reach the parasite rather than simply a failure of the drug to destroy the parasite. However, he did note that live adults have been seen in the lungs after deaths following drenching. A similar observation was reported elsewhere (Anon, 1978). It should be noted that Oakley (1980), working with D. viviparus in cattle, observed that it took up to 36 hours before there was a significant reduction in lungworm numbers following treatment with some benzimidazoles. This delay was presumably because of the slow rise in plasma concentration of the drug and the mechanism of action of the anthelmintic. This observation needs to be considered when live worms are observed after treatment with benzimidazoles.

1.2 Gastrointestinal Nematodes.

1.2.1 Occurrence.

Twenty species of nematode gastrointestinal parasites have been recorded in the species of deer in New Zealand (table 1.1). The majority of these observations have been in red, wapiti and white-tailed deer, both feral and farmed. There have been few nematode species recorded from sambur, rusa, sika and fallow deer. This is probably because of the small number of these deer species examined (Andrews, 1973). With the exception of fallow, these deer species are not commonly farmed and are therefore less likely to undergo parasitological examination.

Several of the gastrointestinal nematodes in New Zealand deer are specific to deer. These include Apteragia spp., Spiculopteragia spp., Rinadia

mathevossiani, Skrjabinagia kolchida, and Ostertagia leptospicularis. These species will have been introduced into New Zealand with deer and have become established here (Andrews, 1973).

Mason (1981a) suggested that the geographically isolated releases of deer in New Zealand from various sources, and the relatively low population densities and restricted movements of feral deer was responsible for geographical differences in parasitic fauna in feral deer. This is supported by the low initial incidence of lungworm at Invermay, and the initial restriction of E. cervi to the Fiordland area. However, it can now be expected that these differences will be reduced in the farmed deer population as deer have been transported throughout the country. The thirteen gastrointestinal nematode species found in New Zealand deer which are also found in other domestic ruminants in New Zealand were either carried into the country by deer, or transmitted from feral or domestic cattle, sheep or goats (Andrews, 1973).

In the 1980 M.A.F. survey of the deer population and health problems, gastrointestinal parasitism rated nationally as the third most frequently occurring animal health problem reported by deer farmers. Gastrointestinal parasitism caused 15% of all deer health problems. However, the same survey showed that gastrointestinal parasitism was not amongst the five most common conditions diagnosed by veterinarians on deer farms (Gladden, 1981). This suggests that gastrointestinal parasitism may not be as much of a problem as it is thought to be by deer farmers. However, with farmers making their own diagnoses, the diagnostic accuracy is likely to be reduced. In addition, it is possible that there was some confusion by the survey repondents in distinguishing between an actual and a potential problem. This could explain the discrepancy between farmer belief and veterinarians observations.

1.2.2 Epidemiology.

There is a lack of epidemiological data on deer gastrointestinal parasitism. Until data shows otherwise, the life cycles of all gastrointestinal nematode parasites of deer can only be assumed to be the same as similar parasites of sheep and cattle (Mason, 1977). The infective third stage larvae are ingested with pasture and exsheath in the abomasum. They moult to fourth stage larvae and then to adults. After a variable prepatent period (20-25 days) they start laying eggs. These are excreted in the faeces and hatch as first stage larvae which develop on the pasture to second stage and ensheathed third stage larvae (Mason, 1977).

No epidemiological work to determine yearly fluctuations in pasture larval numbers on deer pastures has been published. Mason (1977) suggested that the same environmental factors appeared to control pasture larval numbers of deer nematode parasites as control sheep parasite larvae numbers. Being temperature-dependent, the rate of larval development for sheep and cattle gastro-intestinal nematodes peaks in mid-summer and stops when the mean air temperature falls below 10^{o}C (Vlassoff, 1973b). The time taken for eggs to develop to third stage larvae is 10 days to 10 weeks under New Zealand conditions (Vlassoff, 1973a), and 1-12 weeks over the spring and autumn in England (Gibson, 1965). The percentage of eggs that successfully develop to third stage larvae is both temperature and moisture dependent and varies from less than 1% to a maximum of 20-25% (Vlassoff, 1982). Because of temperature and moisture effects, optimum larval development (and hence maximum pasture larval number) occurs only when there is a positive moisture index, and a mean air temperature greater than 10^{o}C (Brunsdon and Adam, 1975). These conditions usually occur in the spring and autumn in New Zealand.

The autumn pasture larval peak tends to occur after the first autumn rains. Larvae which have developed over the summer and early autumn need a continuous moisture

film to enable movement up the herbage and become available to the stock (Vlassoff, 1973a,b). Although the pattern of larval contamination of pasture may differ when deer, sheep or cattle are grazed it could be expected that there will be a relatively low pasture contamination during summer, a high contamination during autumn, and a decline over winter.

Ensheathed larvae survive on pasture for up to five months in England (Gibson, 1965) and up to six to twelve months in New Zealand (Vlassoff, 1973a). On sheep farms, larvae which overwinter are an important source of contamination for new lambs in spring (Vlassoff, 1973b; 1976). However, some workers consider that aged larvae are less able than young larvae to establish in a host (Gibson, 1965; Sturrock, 1965). Infections from over-wintered larvae plus the infection originating from the ewe's post-partum rise leads to the spring peak in larval numbers seen on sheep farms (Vlassoff, 1973b).

In deer a post-partum rise of gastrointestinal egg output from hinds was not found in a small trial (Wilson, pers. comm.). Calves are born later in the year than lambs, when conditions for larval survival are less favourable. Thus although the calves will possibly be infected by larvae which have overwintered and larvae originating from the adults in spring, there may well not be an equivalent of the rise in pasture larval numbers as seen on sheep farms in spring, because of drier conditions during the equivalent postnatal period.

Another method for parasite survival over periods of adverse climatic conditions is the inhibition of larval development in the host. In sheep, inhibited fourth stage larvae are considered to be an important mechanism of overwintering for Haemonchus contortus. It is less important for Ostertagia species and not significant for T. axei (McKenna, 1973). Inhibited larvae recommence development in spring and contribute to the ewe's post-partum rise in parasite numbers and faecal egg counts (FEC). Both H. contortus and Ostertagia species occur in deer, and it has been suggested that

inhibited larvae may survive drenching and then develop to maturity in red deer (Johnston, 1982).

1.2.3 Pathogenicity.

Mason (1977) reported deaths of red deer calves as a result of 18,900-33,700 abomasal and 1500-30,500 small intestinal worms. Another report described the death of a yearling hind with a "severe" lungworm infestation coincidental with 2800 H. contortus, 2200 O. circumcincta and 5,600 T. axei (Anon, 1979a). Forrester et al., (1974) reported that burdens of 3-5000 Strongyloides in white-tailed deer, 2-3 weeks old, produced an acute illness with diarrhoea, abdominal cramps, listlessness and death in 12-36 hours.

Presidente (1979a) suggested that S. asymmetrica and S.kolchida induce the same pathology as Ostertagia spp. in cattle and sheep. Developing larvae of these two parasites have been observed in dilated and inflamed gastric glands in deer (Presidente, 1979b). These developing larvae induced nodule formation in the abomasal mucosa. There were few nodules in abomasa of deer with burdens of less than 1000 worms. More widespread nodules appeared when there were approximately 2000 worms. A "morocco leather" appearance was seen with burdens of 5-6000 worms in a fallow deer and 7,000 worms in a white-tailed deer. Presidente (1979) also reported that clinical parasitism with weight loss, rough hair coat and loose faeces occurs when abomasal nematode burdens exceed 5000 worms. He recommended treatment when worm burdens exceeded 2000. Eva and Kellogg (1977) stated that when abomasal worm burdens exceed 1500 in white-tailed deer mortality risk was high. These authors also reported deaths of white-tailed deer due to haemonchosis with average burdens of 2000. Experimental infections of 3000 H. contortus in white-tailed deer resulted in weakness, emaciation, anaemia, debilitation and decreased serum protein (Foreyt and Trainer, 1970).

These burdens are low when compared to burdens

considered pathogenic to sheep. A worm burden of 4-10,000 is classed only as "moderate" in young sheep (McKenna, 1982). This may suggest a greater pathogenicity of abomasal nematodes to deer but more likely reflects conservative estimates by deer researchers in the face of a sparsity of data on this issue. Johnston (1982) infected red deer calves with up to 30,000 O. circumcincta, O. ostertagia or H. contortus larvae but growth rates during the following seven weeks were the same as for non-infected control deer. The peak FEC was 87.5 epg after infection with H. contortus larvae. This infection caused an increase in plasma pepsinogen concentration indicating some abomasal mucosa damage, but apparently not enough to affect growth. H. contortus burdens sufficient to cause a FEC of 9000 epg have been associated with decreased packed cell volume, haemoglobin and total serum protein in white-tailed deer (Davidson et al., 1980).

1.2.4 Clinical Signs and Diagnosis.

Diagnosis of gastrointestinal parasitism is usually based on a combination of faecal examination for gastrointestinal parasite eggs, clinical signs and history, but may be aided by post-mortem of severe cases. Gastrointestinal parasitism is most pronounced in young stock up to two years of age and may result in loss of condition, decreased weight gain, ill thrift, loose faeces or scouring and may terminate in emaciation, weakness and death (Wilson, 1979a). Wilson also stated that haemonchosis may result in anaemia, lowered packed cell volume and hypoproteinaemia; and large abomasal burdens may cause an elevated plasma pepsinogen level. Significantly increased plasma pepsinogen levels (358 mu/L, from 125 mu/L) attributed to abomasal trauma caused by parasitism have been recorded in red deer calves after inoculation with H. contortus larvae (Johnston, 1982). Ford (1976) considered that in ruminants, plasma pepsinogen levels are a better indicator of potential production loss due to Ostertagia than faecal egg counts.

Wilson and Pauli (1983) reported that the maximum 95% confidence limit for plasma pepsinogen levels in 44 clinically normal mixed aged red deer was 2.08 IU/l with a mean of 0.91 IU/l.

The relationship between FEC and worm burden is not clear. Presidente (1979b) considered that 1 epg (measured by floatation), indicates a burden of 500 worms. This author stated that continual monitoring of abomasal parasite numbers was an essential part of a deer farm management programme with treatment recommended when egg counts by floatation exceeded 10 eggs per gram. Wilson (1981) reported that there appeared to be little correlation between FEC (measured by the modified McMasters technique) and worm burdens. However, all egg and worm numbers in this report were low, and the specimens were from deer dying of other conditions.

1.2.5 Treatment and Control.

Gastrointestinal parasite control programmes fall into two categories: (1) total reliance on anthelmintics (2) integration of anthelmintic treatment with planned pasture and stock management. The latter must be to segregate the host from significant numbers of infective larvae. Parasite control programmes on deer farms tend to be of the former type.

1.2.5.1 Anthelmintics.

There is only limited information on anthelmintic efficacy against gastrointestinal nematode parasites in deer. McAllum (1976) stated that gastrointestinal worms were easily treated with oral drench but did not state which products were effective. This author later stated that thiabendazole and levamisole would readily control gastrointestinal nematodes when given orally but gave no data to support his statement (McAllum, 1977).

Blockeler and Segebade (1977) reported a 97-98%

reduction in faecal egg counts in fallow deer after feeding concentrate feeds containing 1.5% fenbendazole to give a dose of 5 mg anthelmintic/ kg liveweight. In a similar study, also involving fallow deer, thiabendazole in pelleted feed at 50 mg/kg liveweight resulted in a 96.5% reduction in faecal egg counts following five days of treatment (Kalivoda and Chroust, 1971).

Thiabendazole at 50 mg/kg has been used successfully in the control of S. papillosis in white-tailed deer in conjunction with other management practices (Forrester et al., 1974). On the basis of reduced worm counts, Foreyt and Drawe, (1978) concluded that there was a greater than 99% efficacy of oral albendazole (11-50 mg/kg) against H. cortortus, Ostertagia species, T. askivali and Nematodirus odocoilei in white-tailed deer.

Wilson (1979a) observed that there were few anthelmintic efficacy trials in deer but stated that most anthelmintics appeared to be effective against deer gastrointestinal nematodes. This view is supported by Presidente (1979b).

Under New Zealand conditions, yards are normally available on deer farms so oral treatment is the accepted method of administration. Mason and Gladden (1983) reported that the mean number of anthelmintic drenches given to calves was 4.1 per year with a maximum of 17.

The Animal Remedies Board has currently licenced albendazole, fenbendazole, febantel and oxfendazole for use in deer. This implies that research, albeit unpublished, must have been conducted to prove the efficacy of these products since this is a requisite for registration.

1.2.5.2 Integration of Treatment and Control.

In a trial involving the treatment of deer with fenbendazole and the slaughter of one group at 3-5 days

and another at 3-4 months post-treatment, Presidente (1979a) concluded that anthelmintic treatment alone only temporarily reduces worm burdens. He advised that if it was possible the herd should be moved to clean pasture after treatment to prolong the reduction in parasite numbers.

Mason (1977) also recommended drenching calves at weaning and moving them to "clean" pasture, i.e. a paddock where the fawns had not been that year. If not moved to "clean" pasture they were to be drenched again one month later. These recommendations applied where the farmer considered parasitism to be a problem. In other areas the animals were to be drenched when necessary depending on the climatic conditions. No indications were given as to how to decide when drenching was necessary, or how the farmer was to determine whether or not parasitism was a problem.

Wilson (1979a) suggested that blanket recommendations could not be made, but recommended that control efforts be concentrated on the young stock and that deer should be rotationally grazed onto "clean" pasture after treatment. In a publication edited by Brunsdon and Adam, (1975) it was stated that, "if animals are returned to heavily infected pasture after drenching much of the advantage of drenching may be lost." This has been demonstrated with lambs where only a small increase in liveweight was achieved when lambs were drenched and not moved compared with a large increase in weight when they were drenched and moved to "clean" pasture (Brunsdon, 1976). However, rotational grazing of young stock *per se* does not contribute to parasite control even with an eight week rotation, according to Brunsdon and Adam (1975), as the concentration of stock on a small area will lead to an abnormally high level of pasture infestation during favourable conditions for larval development. Rather the stock movements must be planned so that young stock move onto "clean" pasture after each drench.

The time needed for the pasture larval population to decline to "safe" levels has been estimated to be two to

six months depending on the climatic conditions (Brunsdon and Adam, 1975). "Clean" pasture is not parasite free but has too few parasite larvae on it to be directly damaging to susceptible animals (Brunsdon and Adam, 1975). This definition is not necessarily the same as that given by Mason (1977) above. For most of the year "clean" pasture is therefore impossible to achieve because of pasture management requirements. "Clean" pasture may be produced by using hay or silage aftermaths, grazing fodder crops, grazing pasture with adult or resistant animals or by grazing an alternative species, i.e. sheep or cattle. Johnstone (1979) observed that grazing cattle pasture with sheep for two months produced as effective a decrease in pasture contamination as spelling for four months. However, parasite species which can establish in both host species could be a problem, eg. T. axei from sheep and cattle. McAllum (1981) mentioned that rank pasture could be controlled by sheep or cattle on deer farms and that this may simultaneously help in the control of internal parasites. However, he did warn that there was a risk of transferring infectious diseases from the alternative stock to the deer, eg. malignant catarrhal fever from sheep. The importance of cross-transmission of parasites could be reduced by grazing only adult sheep and cattle.

1.3 Feed Requirements And Growth Of Red Deer.

The feed requirements and growth from 0-12 months only are reviewed.

1.3.1 Birthweight.

Males have a higher average birthweight than females. Guinness et al., (1978) recorded mean birthweights of 6.9 kg and 6.44 kg for males and females respectively, in the United Kingdom and Asher et al., (1981) recorded 9.4 kg and 8.8 kg respectively in New Zealand. Birthweight was shown to be significantly related to the

liveweight of the hind at the rut (an index of body size) (Blaxter and Hamilton, 1980). There was no effect of age of the hind on calf weight that could not be explained by hind body weight. Mortality was higher at birthweights below 5 kg.

1.3.2 Birth to Three Months (weaning).

Milk is the major energy source in the first nine weeks. Fennessy et al., (1981) determined that 90% of the variation in growth rate to nine weeks of age can be attributed to variations in milk gross energy intake by the calf. No more than 10% of the calf's total metabolizable energy (ME) intake to this age is solid food (Fennessy et al., 1981). Thus initial growth is determined largely by the milk yield of the dam (Moore and Brown, 1977). Blaxter and Hamilton (1980) found a positive relationship between the weight of the dam and the calf's growth rate. They attributed this to a higher milk yield from heavier hinds. Loudon et al., (1983) demonstrated that poor hind nutrition during lactation can result in significant reductions in calf liveweight gains as a result of depressed milk yields.

Males grow faster than females (Blaxter and Hamilton, 1980; Asher et al., 1981). Daily growth rates of 400 g/day and 371 g/day respectively, for males and females were recorded at weaning at 14 weeks of age in New Zealand (Fennessy et al., 1981). These growth rates are higher than 305 g/day and 270 g/day for males and females respectively, recorded in Scotland (Blaxter and Hamilton, 1980).

Due to the demands of lactation, the summer period is the time of maximum ME requirements of the hinds (47.4 MJ/ME/deer/day) (Fennessy et al., 1981).

Pasture quality must not be allowed to decline if high weaning weights are to be achieved (Moore and Brown, 1977). A minimum of 4.4 kg DM of leafy pasture per deer per day must be available for maximum weaning weight (Milligan, 1984). Supplementary feeding of hinds may be

required in dry areas or dry seasons when pasture growth is low (Wilson, 1979b).

1.3.3 Three to Twelve Months.

Maximum growth rates are required over this period if all females are to achieve mating weights of 65-70 kg at 16 months (Wilson, 1979b). Growth rates up to one year of age are not constant. Growth is slow from March to September but accelerates from September to December (Moore and Brown, 1977; Drew et al., 1978; Asher et al., 1981; Fennessy et al., 1981). Average growth rates of 30 g/day over autumn and winter, 300 g/day over spring and 200 g/day over summer for males; and 30 g/day over winter, 230 g/day over spring and 120 g/day over summer for hinds have been recorded (Fennessy et al., 1981).

In a harsh environment, weaners were only held at maintainance over winter (Moore and Brown, 1977) whereas in more mild climates minimum growth rates of 71 g/day have been recorded during this period (Asher et al., 1981). No estimates of the digestable ME available to these stock were given so it cannot be determined if the different weight gains were due to the climate or feed availability. Calculated ME requirements during winter are 19.9 and 17.5 MJ ME/deer/day for males and females, respectively. Deer have been shown to be less tolerant of cold than either sheep or cattle (Simpson et al., 1978) and therefore require proportionally more ME for thermogenesis during winter.

High growth rates of females during spring and summer are critical if a high calving percentage is to be achieved at two years of age (Moore and Brown, 1977). The daily ME requirements for rising one year old deer over spring has been estimated as 27.1 and 22 MJ ME/deer/day for males and females, respectively.

CHAPTER 2

STUDY OF ALBENDAZOLE IN A CONCENTRATE FEED PREPARATION.

2.1 Introduction.

Anthelmintics administered to deer in New Zealand are usually given by oral drenching. However, this method of administration is not satisfactory in some instances.

If late (post-rut) weaning is practised there is a risk of injury to the calves if they are yarded with the adult stock during the rut. The disturbance of the hinds at this time may also impair conception rates.

High parasite burdens have been observed in stock younger than three months old (see Chapter 3). Many farmers feel that calves are too small to risk yarding for anthelmintic treatment at this time.

Parasite problems are most common in autumn and early winter. Should such problems develop in mature stags, yarding and oral treatment could be dangerous for both animals and operators, since stags are aggressive at this time of year.

Many deer are kept where there are no handling facilities ("pet" deer and newly established properties). In these situations oral drenching is not possible. This may also be the case in some zoos and game-parks.

Some deer farmers as a matter of general policy handle their deer as little as possible despite having adequate facilities. An alternative to oral drenching may be attractive to such people.

These considerations have prompted the investigation of an alternative anthelmintic administration technique.

Incorporation of anthelmintic into supplementary feedstuffs was suggested as an alternative method of treatment of farmed deer by Presidente, (1979a,b). Thiabendazole (50 mg/kg/day for five days) and fenbendazole (5 mg/kg/day for two days) when mixed with a supplementary feedstuff have both been successful in

controlling internal parasites in fallow deer in Europe (Kalivoda and Chroust, 1971; Bockeler and Segebade, 1977).

In this study the use of albendazole in a supplementary feed preparation was investigated both in weaner and adult deer.

Note: In this thesis the concentrate feed preparation incorporating the anthelmintic is referred to as "medicated nuts", and the preparation not incorporating anthelmintic as "non-medicated nuts".

2.2 Part 1: Weaner Deer.

This study was undertaken to:

1. Investigate the effectiveness of low concentrations of albendazole in a concentrate feed preparation (medicated nuts) in controlling internal parasites in intensively farmed red deer.
2. Compare the effectiveness of medicated nuts with a three-weekly oral drenching programme under set-stocked and rotational grazing systems.
3. Study the effect of the medicated nut administration programme on the later occurrence of internal parasitism.
4. Establish the palatability of the medicated and non-medicated nuts.

2.3 Materials and Methods.

2.3.1 Deer.

Twelve red deer (5 males, 7 females) from the Massey University Deer Unit and 12 male red deer purchased from a commercial deer farm were used. Groups were weaned on March 10 and March 14, respectively, and were combined from March 22.

2.3.2 Supplementary Feedstuff.

The supplementary concentrate feed used was manufactured in the form of nuts. The non-medicated nuts used were a commercially available deer ration, [Farm Products (Manawatu) Deer Pellets, product No. 117], with a minimum feed analysis of protein 14%, fat 4.5%, fibre 12%, salt 1%.

The medicated nuts were the same product with albendazole added to provide 200 mg albendazole per kg of nuts. The preparation of the medicated nuts was such that a 50 kg animal was required to eat 0.5 kg to achieve a dose rate of 2 mg albendazole/kg liveweight.

2.3.3 Experimental Design.

Experimental design is summarized in figures 2.1 and 2.2.

On March 22, deer were ranked according to liveweight. Animals were systematically assigned to one of three groups so that each group was approximately balanced by sex, mean liveweight and liveweight range. Groups were randomly allocated to one of three treatments:

Group 1: n=8 (allocated numbers 101-108).

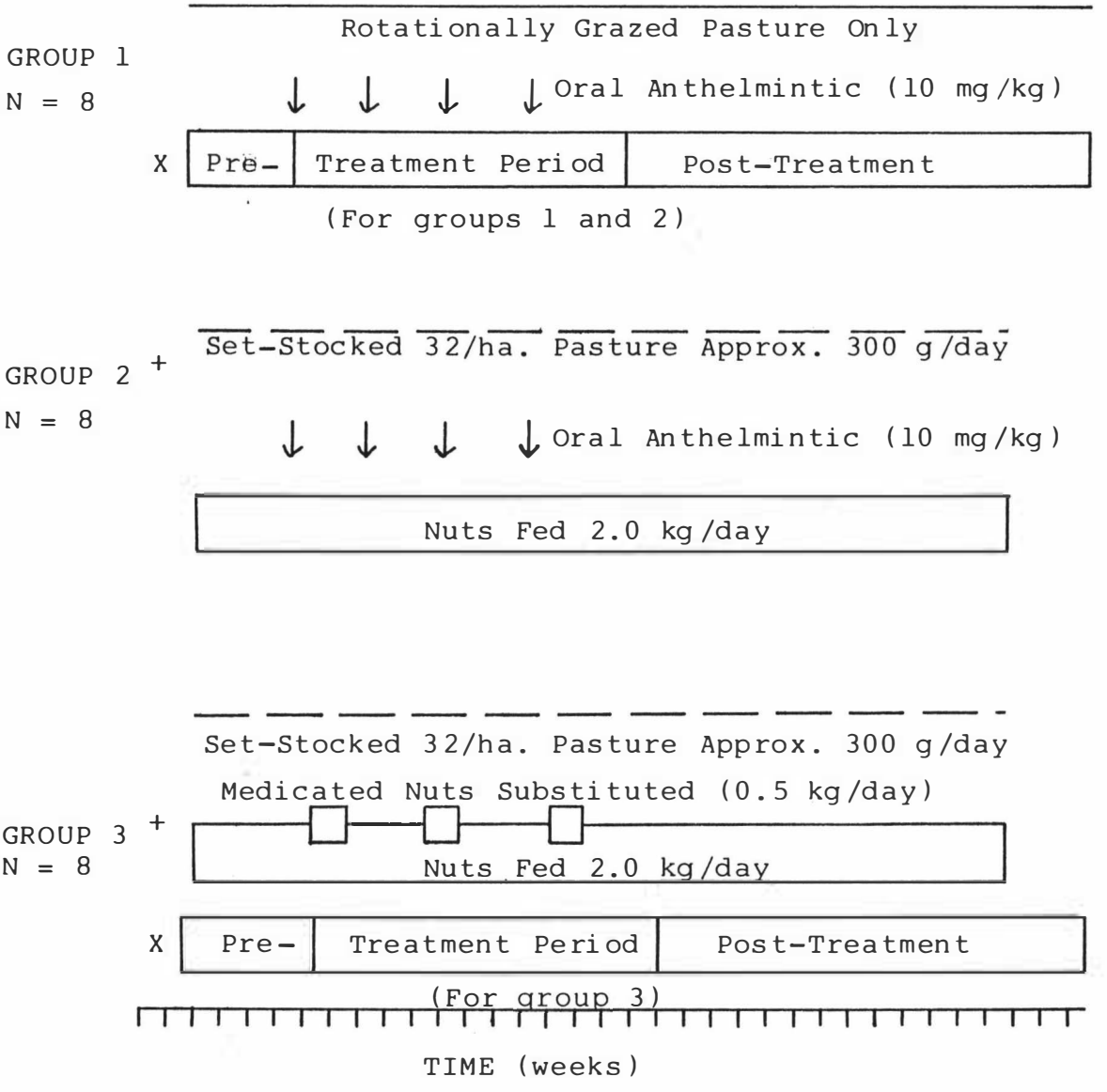
This group was rotationally grazed on four 0.5 ha paddocks under normal deer farm grazing management with no supplementary feed throughout the course of the investigation (see figure 2.2). This group received four oral drenches of albendazole ("Valbazen" Cattle and Deer drench, Smith Kline and French Ltd) at a dose rate of 10 mg/kg at three-week intervals from April 21.

Group 2: n=8 (allocated numbers 201-208).

It was necessary to divide this group into subgroups to allow for the possibility of a "plot effect" on treatment. These deer were set-stocked at 32/ha in pasture plots of 0.125 ha (see figure 2.2). This group received up to 2.0 kg non-medicated nuts daily from March 31 until

Figure 2.1

DIAGRAMMATIC REPRESENTATION OF EXPERIMENTAL DESIGN.



+ Groups 2 and 3 were subdivided into two groups to allow for the investigation of a plot effect on treatment. (See fig. 2.2)

X Data within these three periods were pooled for statistical analysis.

Note: The post-treatment period for group 3 was shorter because each treatment occupied 10 days with a 21 day interval between treatments, compared with consecutive 21 day intervals between oral treatments in groups 1 and 2.

Figure 2.2

Plan Of Massey University Deer Unit Showing Location Of Experimental Groups.

Not to scale.

October 13. The group was given four oral drenches of albendazole (10 mg/kg) at three-week intervals from April 21.

Group 3: n=8 (allocated numbers 301-308).

This group was subdivided and managed as for group 2. This group received 2.0 kg non-medicated nuts daily from March 31 until October 13. 0.5 kg of non-medicated nuts were substituted with 0.5 kg of medicated nuts (100 mg albendazole/deer/day) for three 10-day periods commencing April 25. There were 21 days between the end of one treatment and the commencement of the next.

For groups 2 and 3, the nuts were fed on the ground with a small quantity (100 g/head) of good quality meadow hay. A "no drench" control group was not possible because of the commercial implications of the risk of clinical parasitism and the value of the stock involved.

2.3.4 Management.

From March 28 group 1 was rotationally grazed ahead of the adult stock on the unit, thereby receiving the best pasture available ad lib. Pasture quality was good and the residual dry matter never fell below an estimated 1200 kg DM/ha.

Groups 2 and 3 were confined in four adjacent compounds (see figure 2.2). Each compound contained a water supply and access via a race. The compounds were of temporary construction with 1m sheep netting and either one or two mains-unit electric tapes at the height of the top of the netting and 15-20 cm above it (see figure 2.3).

Before the commencement of anthelmintic treatment, groups 2 and 3 were fed non-medicated nuts at 0.5 kg/deer/day increasing to 2 kg/deer/day (1.7 kg DM) by June 13 as pasture availability decreased. It is estimated that the minimum pasture DM available was 0.3 kg/head/day. Thus deer received a calculated minimum of 2.1 kg DM/head/day of digestible feedstuff throughout the course of the study. Nut rations were stopped on October 13.

Figure 2.3

Experimental groups 2 and 3: Deer were fed deer nuts and set-stocked in temporary compounds constructed of sheep netting and one or two electrified tapes.

To allow asessment of consumption when medicated nuts were given, both groups 2 and 3 received 0.5 kg/deer of nuts (medicated or non-medicated) in the morning and the balance (1.5 kg/deer) of non-medicated nuts late in the afternoon.

In mid October the three groups were combined and rotationally grazed around the whole unit until termination of the trial on November 3.

2.3.5 Observations.

From March 24 all animals were faecal sampled per rectum and weighed at weekly intervals at approximately the same time of day. During the 10-day medicated nut feeding periods the animals in group 3 were faecal sampled at 2-4 day intervals.

Four grams of faeces from each sample were placed in an individual modified Baermann apparatus, as described by Henricksen, (1965), on the day of collection. The extracted larvae were identified and counted the following morning at 100X magnification with a binocular microscope using a McMaster slide.

Two grams of faeces from each sample were examined for gastrointestinal parasite eggs on the day of collection by a modified McMaster method as standard for the Massey University parasitology laboratory.

The eating behaviour of groups 2 and 3 was observed periodically throughout the trial, particularly when medicated nuts were supplied.

2.3.6 Animal Health.

All animals received a sensitising dose of a five-in-one clostridial vaccine, ("Covax 5", ICI-Tasman), at weaning, and a booster dose six weeks later. All animals were given selenium (5 mg) subcutaneously prior to the study.

One animal (306) developed diarrhoea of undiagnosed cause on May 6 which was treated with systemic and oral

antibiotics, gut protectives and an anthelmintic. Recovery was uneventful. All animals were vaccinated against Salmonellosis ("Salvaxin" Wellcome Ltd.) at that time as a precaution.

A second deer (303) developed a cough and respiratory distress when yarded on May 26. It was given a three day course of an oxytetracycline-bromhexine HCL mixture ("Bisolvomycin Vet", Boehringer Ingelheim Ltd.). Recovery was uneventful.

During the course of the study some animals exhibited a mild cough or loose faeces. These occurrences were not treated and did not persist.

On July 7 six animals selected at random from both groups 1 and 3 were blood-sampled by jugular puncture into evacuated plain tubes and serum analysed for copper concentration. On the basis of high blood copper concentrations sodium molybdate (50 mg/deer/day) and sodium sulphate (1000 mg/deer/day) was added to the feedstuff to prevent toxicity problems which are known to occur in sheep under similar feedings systems.

On November 1 almost at the conclusion of the trial number 301 died suddenly of yersiniosis.

2.4 Statistical Analysis.

For analysis the larval and egg count and liveweight data was subdivided into "pre-", "during" and "post-" treatment periods for each group (see figure 2.1).

Analyses performed are summarized in table 2.1.

2.4.1 Larval and Egg Counts.

All larval and egg counts were transformed for analysis to $\log_{10}$ after the addition of 10^{-5} to overcome the problem of zero counts. Log transformation was carried out to normalise the data. The mean log daily egg and larval counts for the "pre-", "during" and "post-treatment" periods were calculated for each animal. Each two successive samplings were averaged and multiplied

Table 2.1

Summary Of Statistical Analyses Performed On Larval And Egg Counts And Liveweights.

Comparison	Test Used
1) Within Trial Periods (Pre-, During, and Post-Treatment).	
Medicated Nut Treatment: Plot 1 v's Plot 2*	Student's t
Non-medicated nut + Oral Drench: Plot 1 v's Plot 2*	Student's t
Group 1 (rotationally grazed) v's Group 2 (set-stocked).	Student's t
Group 2 (oral anthelmintic) v's Group 3 (medicated nuts).	Student's t
2) Between Trial Periods	
Pre- v's During v's Post-treatment: Group 1	Anovar+
Pre- v's During v's Post-treatment: Group 2	Anovar+
Pre- v's During v's Post-treatment: Group 3	Anovar+

*Test for "plot effect".

+Analysis of variance.

by the number of days between the samplings to give daily mean counts. These values were then summed for each trial period and divided by the number of days in that period.

Because no plot effect was evident, group 2 and 3 subgroup daily mean counts were combined to produce mean log counts. Group mean log counts were compared between groups within a trial period by Student's t test, and within groups between trial periods by analysis of variance.

2.4.2 Liveweights.

Individual daily mean liveweight changes were calculated for each trial period by subtracting the first weight from the last weight in each period and dividing by the number of days in that period. Individual daily mean liveweight changes were averaged for each subgroup. Paired subgroups were analysed for plot effects at each treatment period by Student's t test. As no plot effect was evident, group 2 and 3 subgroups were combined to produce group daily mean liveweight changes for each period. Analysis was performed between groups within each trial period by Student's t test and within groups between trial periods, by analysis of variance.

2.5 Results.

For presentation of data, the trial is subdivided into "pre-", "during" and "post-treatment" periods as described in figure 2.1.

2.5.1 Faecal Larval Counts.

Individual faecal larval counts for groups 1-3 are presented in tables 2.2-2.4, respectively. Arithmetic mean counts are presented in figure 2.4.

A summary of mean log larval counts for each experimental period, and statistical analyses, is

Table 2.2

Individual Lungworm Larval Counts/g Faeces At Each Sampling, Group 1.

Number	101	102	103	104	105	106	107	108	
Sex	F	M	M	M	M	M	F	M	
24/3	*	0	17.75	*	0.57	61.25	15.50	5.00	
30/3	48.00	0	0	*	*	1.03	57.30	6.25	
14/4	29.00	24.75	30.25	*	69.00	180.00	133.00	130.00	
20/4	78.25	79.25	*	*	73.50	675.75	102.75	139.75	X
28/4	0	0	0.25	*	0	0	0	0	
30/4	0.25	*	0	*	0	0	0	*	
4/5	0	0	0	0	0	0	0	*	
12/5	17.50	0.50	2.00	*	3.25	0.75	5.00	*	X
19/5	*	0	0	0	0	2.50	1.50	0	
26/5	*	0	0.75	*	*	5.00	2.00	1.75	
2/6	*	0.50	7.00	*	0.25	108.25	73.25	113.00	X
9/6	0	0	0	0	0	0.50	0.50	0.50	
16/6	0	0	0	0	0	0	0	0	
23/6	0	0	1.25	0	0	9.50	5.50	4.50	X
30/6	0	0	0	0	0	0	0	0	
7/7	0	0	0	0	0	0	0	0.25	
15/7	2.00	0	0.25	*	0.25	1.00	5.00	3.00	
21/7	4.50	0	0.25	*	0.25	1.00	0.50	11.75	
28/7	*	2.00	0.50	*	1.75	3.00	5.25	11.50	
4/8	16.25	1.25	3.00	1.00	3.75	11.75	11.25	7.25	
11/8	*	1.75	*	*	1.50	8.25	27.00	6.25	
18/8	7.50	2.50	1.00	3.00	2.00	14.25	21.50	4.50	
25/8	11.50	0.75	2.50	*	4.75	24.00	40.75	8.00	
1/9	10.00	1.25	1.50	3.00	3.25	9.75	11.00	11.00	
9/9	9.50	7.75	1.00	*	2.50	6.00	1.50	4.50	
15/9	14.75	3.50	1.25	*	0.75	4.25	1.50	6.50	
22/9	4.00	7.75	1.25	0.25	2.50	11.00	1.25	2.00	
29/9	9.25	12.00	3.50	*	*	23.00	0	12.00	
11/10	1.50	3.75	1.00	*	2.00	9.50	0	13.50	
18/10	1.75	11.25	1.75	*	0.50	8.50	0	6.50	
27/10	1.00	6.25	0.25	0.30	0	3.75	1.00	9.25	
3/11	1.25	10.25	1.50	*	0	8.00	0	2.75	

* Not sampled.

X = Oral anthelmintic treatment.

Table 2.3

Individual Lungworm Larval Counts/g Faeces At Each Sampling, Group 2.

Number	201	202	203	204	205	206	207	208	
Sex	M	F	M	F	F	M	M	M	
24/3	8.25	0.77	*	0.25	*	2.50	7.25	0.25	
30/3	*	*	0.50	2.75	4.50	0.25	0	5.00	
14/4	43.75	114.50	18.25	42.50	14.00	95.00	19.25	120.25	
20/4	61.75	*	13.25	90.50	*	*	28.50	233.89	X
28/4	0	0	0	1.25	*	*	0	0	
4/5	0	0	0	0.25	0	0	0	1.00	
12/5	0.75	7.00	0	11.67	*	0	0.25	7.50	X
19/5	*	0.50	0	*	0	0	0	0	
26/5	1.00	*	0	0	0.25	0	0	0	
28/5	0.25	0.50	0	0.25	1.00	*	0	3.25	
30/5	1.25	12.50	0.25	1.75	15.50	17.00	1.20	36.00	
2/6	2.25	28.00	2.75	9.00	*	41.00	11.75	49.50	X
9/6	0	0	0	0	0	0	0	0	
16/6	0	0	0	0	0	0	0	0	
23/6	0	0	0	0.25	1.00	0.25	0.25	0.75	X
30/6	0	0	0	0	*	0	0	0	
7/7	0	0	0	0	0	0	0	0	
15/7	0	0	0	0	0	0	0	0	
21/7	0	0	0	0	0	0	0	0	
28/7	0	1.00	0	1.5	0	0	0	0.75	
4/8	0	1.75	0	1.75	0	0.75	0	2.00	
11/8	0	0.25	0	1.00	0.50	0	0	0.50	
18/8	0	0.50	0	0	0	1.00	0	0.50	
25/8	0.25	3.00	0	3.50	0.50	2.75	0	4.25	
1/9	0.50	1.50	0.25	0.50	0	0	0	0.25	
9/9	0.25	0	0	0.50	0	0.50	0.75	0	
15/9	0.75	2.75	0	1.25	1.25	2.00	3.00	2.50	
22/9	1.25	1.00	0.50	4.25	2.00	4.25	0.75	2.50	
29/9	7.75	9.50	1.50	12.25	10.00	1.75	1.75	2.50	
11/10	2.25	7.75	1.00	11.25	*	2.00	0.50	4.25	
18/10	2.50	7.25	0.50	14.50	1.25	2.25	3.50	1.00	
27/10	4.25	18.25	0.75	11.75	11.00	3.25	1.00	4.50	
3/11	3.75	6.75	2.00	33.75	27.00	16.50	2.00	1.50	

* Not sampled

X = Oral anthelmintic treatment.

Table 2.4

Individual Lungworm Larval Counts/g Faeces At Each Sampling, Group 3.

Number	301	302	303	304	305	306	307	308	
Sex	M	M	M	F	M	M	F	M	
24/3	0.25	0.50	6.75	*	1.25	6.5	2.50	*	
30/3	0	*	*	*	4.00	0.75	0.76	*	
14/4	10.00	0.50	0.25	*	3.25	56.75	*	*	
20/4	42.25	5.50	16.50	18.50	*	178.50	13.75	17.50	
28/4	50.00	*	*	30.00	10.50	212.75	*	85.75	X
30/4	17.75	*	7.25	*	0	15.33	19.50	80.25	X
2/5	40.00	10.25	6.25	27.00	2.50	184.25	11.50	68.75	X
4/5	21.25	8.75	5.75	10.50	*	156.75	*	68.75	
6/5	15.25	4.50	16.25	22.00	*	6.75	2.00	94.50	
12/5	5.25	4.50	8.50	15.50	5.25	*	0	130.25	
19/5	145.50	9.00	16.25	17.00	0.75	0.25	*	285.00	
26/5	106.00	24.50	2.50	4.00	2.00	1.00	0	227.00	
28/5	162.50	9.75	2.00	7.50	*	2.00	0	44.25	X
30/5	90.25	0	0	0	0	0.50	0	2.25	X
2/6	0	0	0	0	0	0	0	0	X
6/6	0	0	0	0	0	0	0	0	
9/6	0	0	0	0	0	0	0	0	
16/6	0	0	0	0	0	0	0	0	
23/6	0	0	0	1.25	0	3.25	0	0	
25/6	0.50	0	0	0.50	0	1.50	0	0	X
27/6	0	0	0	0.25	0	0	0	0	X
30/6	0	0	0	0	0.25	0	0	0	X
5/7	0	0	0	0	0	0	0	0	
7/7	0	0	0	0	0	0	0	0	
15/7	0	0	0	0	0	0	0	0	
21/7	0	0	0	0	0	1.00	0	0	
28/7	1.00	0	0.75	0	0	6.50	1.00	*	
4/8	0.50	0	0.75	*	0	6.25	0.25	0.25	
11/8	0	0	1.50	0	0	6.50	0.25	0.25	
18/8	0	0	0	0	0	0.75	0.75	0.75	
25/8	0.25	*	0.50	0	0.25	1.75	0.75	0	
1/9	0	0	1.00	0	0	3.00	0.75	0	
9/9	0.25	2.75	0	0.25	0.5	2.25	0	2.25	
15/9	3.00	0	0	*	0	5.00	0.75	1.00	
22/9	3.75	0	0.25	1.50	0	3.75	0.50	0.25	
29/9	3.25	*	2.25	6.25	0	6.25	0.50	1.25	
11/10	6.75	1.75	1.40	1.50	0	3.50	0.75	2.00	
18/10	6.00	1.25	2.00	1.75	0	4.50	0.25	2.25	
27/10	7.25	1.50	3.00	2.50	0	5.50	0.75	2.50	
3/11	*	1.25	2.50	2.00	0.25	4.25	1.50	2.50	

* Not sampled

X = Period of medicated nut treatment.

presented in table 2.5.

From initial mean counts from all animals of 8.1 lpg, in late March, the mean larval output continued to rise at an increasing rate until the first anthelmintic treatment was given on April 21 (see figure 2.4). At this time the mean count from all animals was 103.9 lpg and 100% of animals were excreting detectable numbers of larvae.

In group 1 (rotationally grazed) there was a dramatic decrease in the mean larval count following the first oral treatment (mean counts at 7 and 14 days post-treatment were 0.04 and zero lpg respectively). Twenty one days post-treatment there was a small rise in the mean larval count to 4.8 lpg.

Over the period when three further drenches were given (12 May to 15 July) the larval counts remained at low levels. In the post-treatment period counts rose to a peak (mean 13.2 lpg) which then declined towards the end of the trial period.

The changes in group 2 were similar to those of group 1 (see figure 2.4). The pre-treatment mean count of 85.61 lpg was reduced to 0.2 lpg after seven days, and counts remained low during the period when three further drenches were given. However, the rise in larval counts seen in group 1 in the post-treatment period was delayed and significantly ($P < 0.01$) less than that seen in group 1.

Both groups 1 and 2 had an increase in their mean larval counts on June 6, 21 days after their second anthelmintic treatment.

There was a poor response to the first medicated nut treatment (group 3); a mean count of 41.8 lpg prior to treatment fell to 23 lpg by the end of the 10-day treatment (see figure 2.4). Larval counts rose to 52.4 lpg by the start of the second treatment. The second 10-day treatment was successful in reducing the mean larval count to zero within six days of commencement. Mean larval counts increased to 0.6 and 0.1 lpg 21 days after the second and third medicated nut treatments,

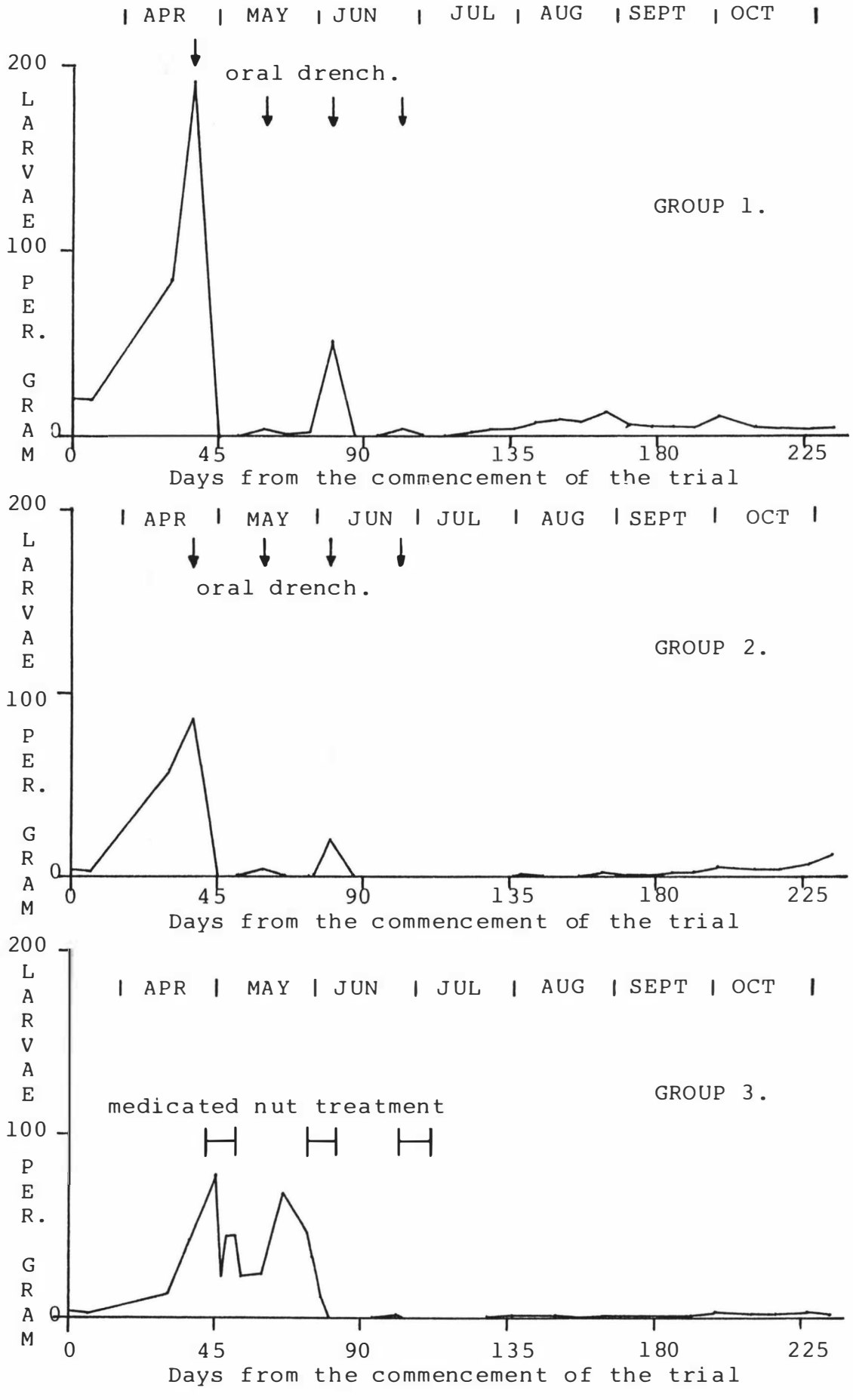

APR
MAY
JUN
JUL
AUG
SEPT
OCT
oral drench.
GROUP 1.
200
100
0
LARVAE PER. GRAM
0
45
90
135
180
225
Days from the commencement of the trial
oral drench.
GROUP 2.
medicated nut treatment
GROUP 3.

Table 2.5

Mean ($\pm$ Standard Error) Of Log_{10} Mean Daily Larval Counts For Pre-Treatment, Treatment And Post-Treatment Periods.

		Pre-Treatment		Treatment		Post-Treatment
Group 1	Mean	1.57	++	-2.206	++	-0.217
	S.E.	0.11		0.33		0.19
						**
Group 2	Mean	1.331	++	-2.232	++	-1.022
	S.E.	0.1		0.2		0.26
Group 3	Mean	1.072	++	-2.173	+	-1.29
	S.E.	0.15		0.22		0.37

** Significant ($P < 0.01$) difference between two groups by Student's t test.

++ Significant ($P < 0.01$) difference between two groups by analysis of variance.

+ Significant ($P < 0.05$) difference between two periods by analysis of variance.

respectively, but these treatments resulted in zero mean counts within 8-12 days of commencement. In the post-treatment period, larval counts rose slowly to 3.3 lpg by the end of October.

The only significant difference between experimental groups was in the post-treatment period when group 1 had significantly higher mean log larval counts than group 2 ($P < 0.01$), see table 2.5. All groups showed a significant decline in mean log larval count from pre-treatment to treatment periods ($P < 0.01$), and a significant rise from treatment to post-treatment periods ($P < 0.01$).

2.5.2 Faecal Egg Counts.

Individual faecal egg counts are presented in tables 2.6-2.8. Arithmetic mean counts are presented in figure 2.5. A summary of mean log counts for each experimental period and statistical analyses is presented in table 2.9.

The pattern of egg count changes resembled that of the larval counts for the three groups. Counts in all groups were low at the initial samplings (March) and increased until treatment commenced on April 21 (see figure 2.5). The prevalence of egg counts $\geq$ 50 epg over the three groups was 65% immediately prior to the first treatment.

In group 1, the mean egg count of 133 epg was reduced to 6.25 epg seven days after the first treatment and remained low ($<$ 59 epg) for the remainder of the treatment period with individual counts never exceeding 100 epg. In the post-treatment period the mean egg counts rose to 150 epg; higher than before treatment (see figure 2.5).

In group 2 the mean egg count prior to the first treatment was 14.3 epg. This was reduced to zero 14 days after the first treatment, and remained low ($<$ 13 epg) throughout the treatment period with individual counts never exceeding 50 epg (see figure 2.5). There was a rise in mean egg count in the post-treatment period but this was less than that seen in group 1.

The mean egg count of group 3 was 57 epg prior to

Table 2.6

Individual Gastrointestinal Egg Counts/g Faeces At Each Sampling, Group 1.

Number	101	102	103	104	105	106	107	108	
Sex	F	M	M	M	M	M	F	M	
24/3	*	0	0	*	*	0	*	0	
30/3	200	100	0	*	*	0	100	50	
14/4	100	0	0	*	50	100	50	50	
20/4	150	50	*	*	0	200	150	250	X
28/4	50	0	0	0	0	0	0	0	
30/4	0	*	0	*	0	0	0	*	
4/5	0	0	0	0	0	0	0	*	
12/5	0	0	0	*	0	0	50	*	X
19/5	*	0	0	0	0	50	0	0	
26/5	*	0	0	*	*	100	50	0	
2/6	*	100	0	*	50	50	50	100	X
9/6	0	0	50	0	0	0	0	50	
16/6	0	0	0	0	*	0	0	50	
23/6	0	0	0	0	50	0	0	0	X
30/6	*	50	*	0	100	50	*	50	
7/7	0	0	0	*	0	0	0	100	
15/7	0	0	0	*	0	0	0	0	
21/7	50	0	0	*	0	0	0	0	
28/7	*	0	0	*	50	100	0	0	
4/8	0	50	0	0	0	50	0	0	
11/8	*	0	*	*	0	0	0	0	
18/8	100	0	0	0	50	150	0	0	
25/8	50	0	0	*	100	100	100	50	
1/9	100	0	0	0	0	0	0	0	
9/9	0	0	0	*	150	300	0	50	
15/9	100	0	0	*	250	450	250	0	
22/9	150	0	50	*	200	0	50	0	
29/9	150	0	0	*	*	400	50	0	
11/10	100	0	0	*	100	600	150	0	
18/10	0	0	50	*	50	550	0	0	
27/10	50	0	100	*	150	300	50	0	
3/11	50	0	50	*	100	500	150	0	

* Not sampled

X = Oral anthelmintic treatment.

Table 2.7

Individual Gastrointestinal Egg Counts/g Faeces At Each Sampling, Group 2.

Number	201	202	203	204	205	206	207	208	
Sex	M	F	M	F	F	M	M	M	
24/3	100	*	*	50	*	*	0	0	
30/3	*	*	*	0	250	0	50	0	
14/4	0	50	0	0	0	*	50	0	
20/4	50	*	0	0	*	*	0	*	X
28/4	0	50	0	0	0	0	50	50	
4/5	0	0	0	0	0	*	0	0	
12/5	0	0	0	0	*	0	0	0	X
19/5	0	0	0	*	0	0	0	0	
26/5	0	*	0	0	0	0	0	0	
28/5	0	0	0	0	0	*	0	0	
30/5	0	0	0	0	50	0	*	0	
2/6	0	0	0	0	*	0	0	0	X
9/6	0	0	50	0	0	0	0	50	
16/6	0	0	0	*	0	*	0	0	
23/6	0	0	0	0	0	0	0	0	X
30/6	0	0	0	0	*	0	0	0	
7/7	0	0	0	0	0	0	0	0	
15/7	0	0	0	*	0	*	0	0	
21/7	0	0	100	50	0	0	0	0	
28/7	0	50	0	0	0	0	0	0	
4/8	0	0	100	0	0	0	0	50	
11/8	0	0	0	0	0	0	0	50	
18/8	0	0	0	0	0	0	0	0	
25/8	50	50	50	0	50	50	0	0	
1/9	0	50	100	0	0	0	0	0	
9/9	0	0	150	0	0	50	0	0	
15/9	0	50	50	0	50	0	0	0	
22/9	0	0	50	0	0	0	0	0	
29/9	0	50	0	0	0	0	0	0	
11/10	50	0	0	0	*	50	0	0	
18/10	0	0	50	0	0	0	0	0	
27/10	0	0	50	0	0	0	0	100	
3/11	0	0	0	0	0	0	0	0	

* Not sampled

X = Oral anthelmintic treatment.

Table 2.8

Individual Gastrointestinal Egg Counts/g Faeces At Each Sampling, Group 3.

Number	301	302	303	304	305	306	307	308	
Sex	M	M	M	F	M	M	F	M	
24/3	100	0	0	*	50	0	50	*	
30/3	0	*	*	*	100	0	*	*	
14/4	0	0	0	*	0	0	*	*	
20/4	0	50	50	100	*	100	100	0	
28/4	50	0	0	100	0	100	0	50	X
30/4	50	*	100	100	0	0	0	50	X
2/5	150	100	50	50	50	150	0	50	X
4/5	100	50	200	0	*	150	*	100	
6/5	500	0	100	50	*	0	0	0	
12/5	150	0	100	0	100	0	50	50	
19/5	100	100	50	0	0	0	*	0	
26/5	250	100	250	*	0	0	100	0	
28/5	0	0	0	0	*	0	0	0	X
30/5	0	0	0	0	50	0	0	0	X
2/6	0	0	0	0	0	0	0	0	X
6/6	0	0	0	0	0	0	0	0	
9/6	0	0	0	0	0	100	0	*	
16/6	*	0	0	0	0	50	0	0	
23/6	0	0	50	0	0	0	0	0	
25/6	0	0	50	0	0	50	50	0	
27/6	0	0	0	50	0	0	0	0	X
30/6	0	0	*	0	0	0	0	*	X
5/7	0	0	0	0	0	0	0	0	X
7/7	0	0	0	0	0	0	0	0	
15/7	0	50	0	0	0	0	0	0	
21/7	50	0	0	0	0	0	0	0	
28/7	0	0	0	0	0	0	0	*	
4/8	0	0	0	0	0	0	0	0	
11/8	50	0	0	0	0	0	0	0	
18/8	50	0	0	0	0	50	0	0	
25/8	100	*	0	50	100	0	0	50	
1/9	0	0	0	0	0	0	0	0	
9/9	0	0	0	0	0	50	0	50	
15/9	0	0	50	*	0	0	0	0	
22/9	50	0	0	50	0	50	0	0	
29/9	0	*	0	0	0	0	0	0	
11/10	0	0	*	0	0	0	0	0	
18/10	0	0	0	0	0	0	0	0	
27/10	50	0	*	0	0	0	0	50	
3/11	*	0	*	50	0	50	0	0	

* Not sampled

X
X = Period of medicated nut treatment.
X

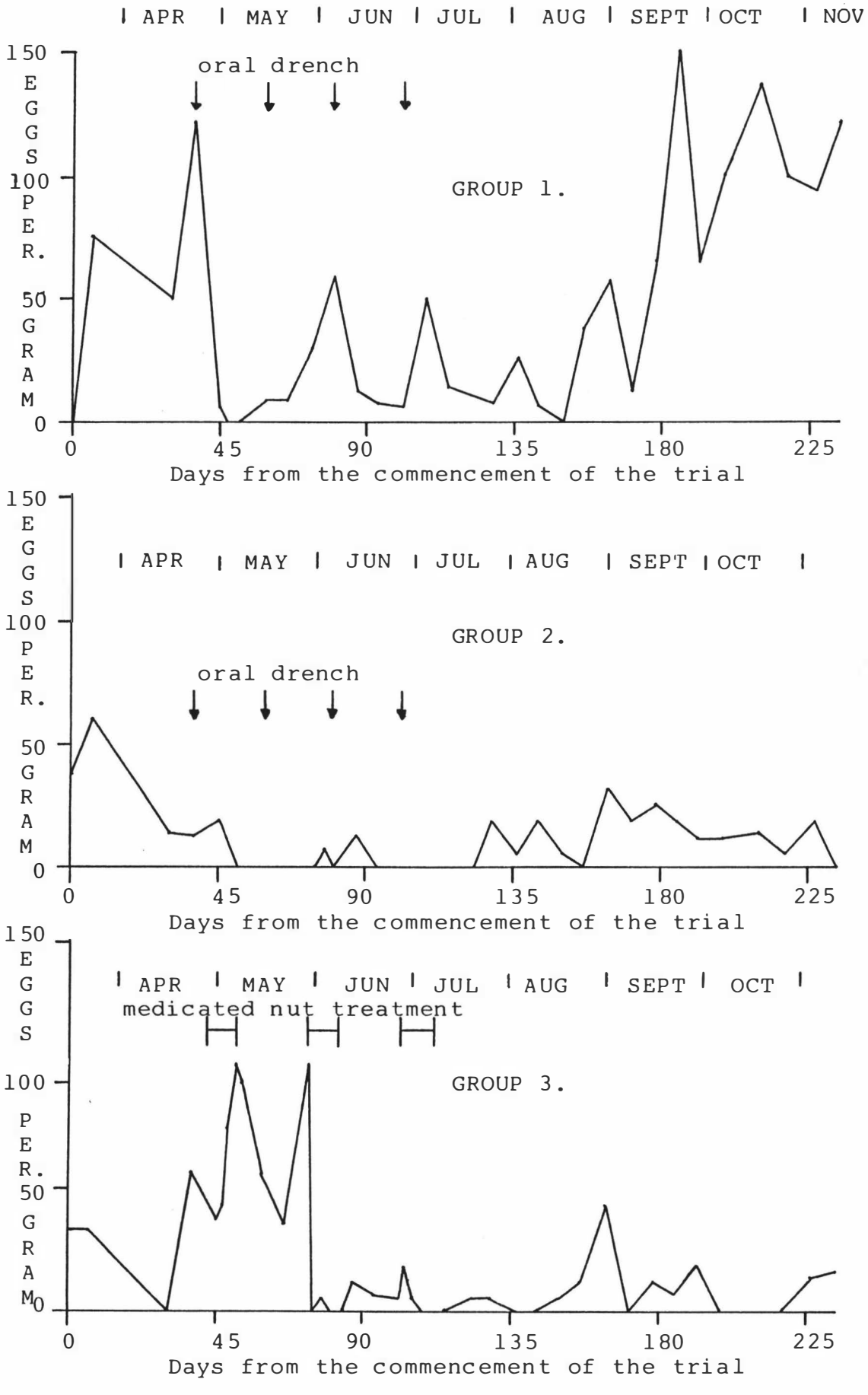

APR
MAY
JUN
JUL
AUG
SEPT
OCT
NOV
oral drench
GROUP 1.
150
EGGS
100
PER.
50
GRAM
0
0
45
90
135
180
225
Days from the commencement of the trial
APR
MAY
JUN
JUL
AUG
SEPT
OCT
GROUP 2.
oral drench
150
EGGS
100
PER.
50
GRAM
0
0
45
90
135
180
225
Days from the commencement of the trial
APR
MAY
JUN
JUL
AUG
SEPT
OCT
medicated nut treatment
GROUP 3.
150
EGGS
100
PER.
50
GRAM
0
0
45
90
135
180
225
Days from the commencement of the trial

Table 2.9

Mean ($\pm$ Standard Error) Of Log_{10} Mean Daily Egg Counts For Pre-Treatment, Treatment And Post-Treatment Periods.

		Pre-Treatment	Treatment	Post-Treatment
Group 1	Mean	-0.28	-2.61 +	-1.93
	S.E.	0.82	0.32 *	0.58
Group 2	Mean	-1.45	-3.68	-2.80
	S.E.	0.85 +	0.09 **	0.38
Group 3	Mean	-1.22	-2.60	-3.13
	S.E.	0.66	0.20	0.27
		(	+	)

** Significant ($P < 0.01$) difference between two groups by Student's t test.

* Significant ($P < 0.05$) difference between two groups by Student's t test.

+ Significant ($P < 0.05$) difference between two periods by analysis of variance.

Note: In group 3 the significant ($P < 0.05$) difference is between the pre- and post-treatment periods only.

the first medicated nut treatment. By the end of this treatment the mean count had risen to 100 epg. The mean count was still 100 epg 21 days later, immediately prior to the second medicated nut treatment. Within two days of the commencement of the second treatment the mean egg count was zero and remained low (<19 epg) for the remainder of the treatment period with individual counts never exceeding 100 epg. In the post-treatment period the mean count never exceeded 43 epg.

Significant differences were detected between the experimental groups only in the treatment period (see table 2.9). Group 2 had a lower mean log egg count than either group 1 ($P<0.05$) or group 3 ($P<0.01$).

Analysis of variance revealed a significant ($P<0.05$) decrease in mean log egg counts in all groups at some stage in the experiment. However, the pattern of decrease of the egg counts varied between the groups. In groups 1 and 2 the egg counts fell from the pre-treatment to treatment periods and then rose from the treatment to post-treatment periods. In group 3 the egg counts continued to fall from the pre-treatment to treatment and to post-treatment periods. The statistical difference was between the pre-treatment to post-treatment periods in this group.

2.5.3 Liveweights.

Individual liveweights are presented in tables 2.10-2.12. Mean liveweights are shown in figure 2.6. Mean daily growth rates for each period are presented in table 2.13.

The animals in groups 2 and 3 suffered a decline in weight (31.4 g/day and 5.7 g/day respectively) for five weeks whilst they became accustomed to their set-stocked compounds and adapted to the deer-nut supplementary feed. Once they were eating the supplementary feed their liveweight gain accelerated then became similar to that of group 1 (figure 2.10). Growth rates were 121 g/day, 200 g/day, 177 g/day over

Table 2.10

Individual Liveweights (kg) At Each Sampling

Group 1.

Number	101	102	103	104	105	106	107	108	
Sex	F	M	M	M	M	M	F	M	
25/3	41.0	47.0	49.0	46.0	47.0	49.0	34.0	53.0	
31/3	42.0	47.5	50.0	48.0	48.0	48.0	34.0	55.0	
14/4	43.5	51.5	51.0	50.0	49.0	49.5	36.0	55.5	X
28/4	45.5	52.0	54.5	57.0	52.5	54.0	39.0	59.5	
6/5	46.0	53.5	55.0	57.0	53.0	54.0	39.0	60.0	
12/5	46.0	54.5	57.0	57.0	54.5	55.5	39.5	60.0	X
19/5	47.5	56.0	57.0	56.5	56.0	56.5	40.5	61.0	
26/5	47.0	56.0	58.5	58.0	55.5	56.0	41.0	62.0	
3/6	47.5	56.5	59.0	58.0	56.0	56.5	41.5	62.0	X
9/6	49.0	57.5	60.0	60.5	58.0	58.5	43.5	65.0	
16/6	50.0	57.5	60.5	60.0	58.5	57.5	44.0	65.0	X
26/6	52.0	59.0	62.5	62.0	61.0	59.5	45.5	66.5	
30/6	51.0	58.0	62.0	61.0	60.5	59.0	45.0	65.0	
7/7	51.5	59.0	62.5	61.5	61.0	59.5	46.0	65.0	
15/7	52.5	59.0	63.0	62.5	62.0	60.0	46.5	65.5	
21/7	52.5	60.5	62.0	60.0	63.5	61.0	48.0	67.0	
28/7	53.0	59.5	63.5	63.0	61.0	60.0	46.5	65.0	
5/8	52.5	60.0	63.5	62.0	63.0	61.0	47.5	67.0	
11/8	53.0	61.5	64.0	63.0	64.0	63.0	46.5	66.0	
18/8	54.5	61.0	63.0	63.0	64.0	63.0	49.0	68.0	
25/8	54.5	62.5	66.0	64.5	65.5	64.0	49.5	70.0	
1/9	57.0	64.0	65.5	67.0	67.5	65.0	50.5	70.0	
9/9	57.0	65.5	68.5	68.5	69.5	66.5	51.0	71.0	
16/9	59.0	68.5	72.0	70.0	70.5	68.0	52.5	72.5	
23/9	59.0	68.0	71.5	70.5	71.5	68.0	52.5	73.0	
29/9	60.0	69.5	72.5	71.0	73.0	69.0	53.5	73.0	
11/10	61.5	71.0	75.0	74.0	75.0	71.0	54.0	76.0	
18/10	64.0	73.5	77.0	76.0	77.0	72.5	56.5	78.0	
27/10	63.5	74.0	80.0	78.0	77.0	72.5	56.5	79.0	
3/11	66.0	78.0	82.0	80.0	80.0	76.0	58.5	82.0	

X = Oral anthelmintic treatment.

Table 2.11

Individual Liveweights (kg) At Each Sampling

Group 2.

Number	201	202	203	204	205	206	207	208	
Sex	M	F	M	F	F	M	M	M	
25/3	58.0	48.0	47.5	33.0	47.0	47.0	46.0	50.0	
31/3	59.0	51.0	50.0	34.5	48.5	49.0	47.5	52.5	
14/4	58.5	49.0	50.0	42.5	48.0	49.0	48.0	52.0	
28/4	58.0	49.5	51.0	35.5	48.0	52.5	49.0	56.0	X
6/5	56.0	49.5	52.0	34.5	47.5	51.0	48.5	54.5	
12/5	56.5	50.0	53.5	33.5	47.0	51.5	48.0	55.0	X
19/5	56.0	50.0	53.5	31.5	46.5	50.0	47.0	54.0	
26/5	58.0	54.0	56.0	35.5	46.5	52.0	48.0	54.0	
3/6	62.5	54.5	57.5	35.5	50.0	53.5	49.0	55.0	X
9/6	63.5	55.0	57.0	36.0	50.5	55.5	51.0	59.0	
16/6	64.0	57.0	58.5	38.0	54.0	57.0	53.5	59.0	
26/6	64.5	58.5	58.0	38.5	54.5	59.0	54.0	59.5	X
30/6	66.5	60.5	60.0	40.0	55.0	60.5	56.0	60.5	
7/7	65.5	59.5	60.5	41.5	57.5	62.5	57.0	62.5	
15/7	68.0	60.5	62.0	42.0	57.5	62.0	58.0	64.0	
21/7	69.0	62.0	61.5	43.0	57.5	64.0	60.0	65.0	
28/7	70.0	61.5	61.5	43.0	58.0	63.5	59.5	64.0	
5/8	70.5	63.0	62.0	43.0	59.0	65.5	60.0	66.5	
11/8	73.0	64.5	63.0	43.5	60.0	67.0	61.0	67.5	
18/8	73.0	64.0	63.5	44.0	61.0	67.0	63.5	69.5	
25/8	73.0	64.5	63.0	45.0	63.0	66.5	64.0	69.0	
1/9	74.0	65.5	65.5	46.0	61.5	68.0	65.5	71.0	
9/9	77.0	66.0	66.0	47.0	65.5	70.5	67.5	72.5	
16/9	79.0	71.0	67.5	49.0	65.0	72.0	69.5	77.0	
23/9	80.5	68.5	69.5	48.5	66.0	74.5	71.5	75.5	
29/9	81.0	69.5	72.5	49.5	67.0	75.5	74.0	80.0	
11/10	82.0	70.0	74.5	51.0	67.5	77.0	74.0	80.0	
18/10	84.5	72.5	76.0	51.0	69.0	79.0	74.5	81.0	
27/10	85.0	72.5	75.0	51.5	68.0	78.0	76.5	81.5	
3/11	89.0	74.0	76.0	53.0	70.0	80.0	80.0	86.0	

X = Oral anthelmintic treatment.

Table 2.12

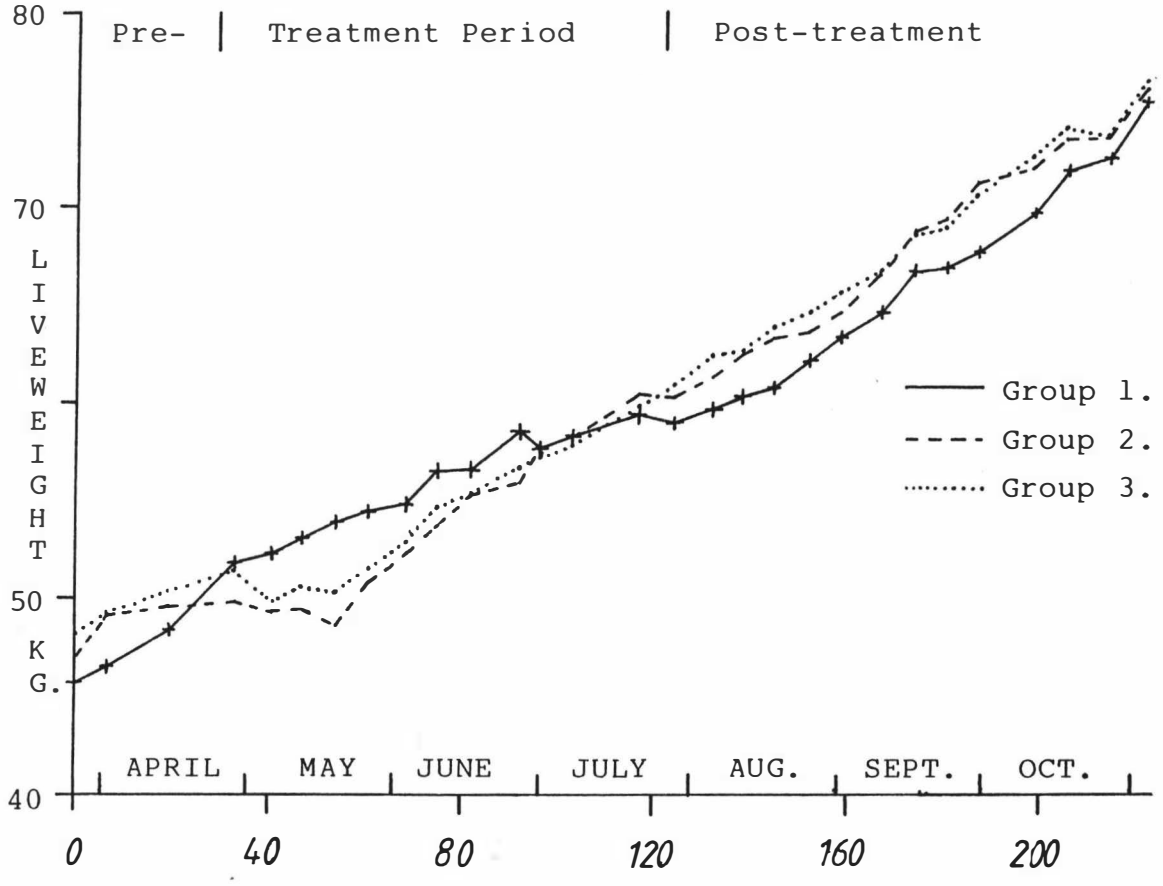
Individual Liveweights (kg) At Each Sampling

Group 3.

Number	301	302	303	304	305	306	307	308	
Sex	M	M	M	F	M	M	F	M	
25/3	49.0	50.0	46.0	45.0	54.0	51.0	49.0	41.0	
31/3	49.0	52.5	48.0	46.0	55.5	51.0	50.0	41.5	
14/4	49.5	52.5	50.0	46.5	55.5	53.5	51.5	43.0	
28/4	50.0	54.0	52.5	46.5	55.5	54.0	53.0	44.5	$\bar{\bar{X}}$
6/5	49.0	52.5	51.0	44.5	53.0	53.0	52.5	43.5	
12/5	48.5	53.5	51.0	45.5	53.5	55.0	54.0	43.5	
19/5	47.0	52.0	52.5	44.5	53.5	54.0	54.5	42.5	
26/5	49.0	53.5	51.0	46.0	56.0	55.5	56.5	44.0	$\bar{\bar{X}}$
3/6	51.5	56.0	51.0	48.0	57.5	55.5	57.0	46.5	
9/6	53.5	57.0	49.0	53.0	59.5	58.0	57.0	49.5	
16/6	54.5	58.0	54.0	50.5	60.0	57.0	57.0	50.5	
26/6	57.5	59.0	55.0	51.5	61.5	58.5	58.0	51.0	$\bar{\bar{X}}$
30/6	58.5	59.5	56.0	53.0	60.5	60.0	56.5	52.5	
7/7	61.0	60.5	57.0	52.5	62.0	61.0	56.0	53.0	
15/7	61.5	61.0	57.5	53.0	63.5	61.5	58.5	54.0	
21/7	62.0	62.5	60.0	53.5	64.0	62.0	59.0	55.0	
28/7	63.5	63.0	60.5	54.5	65.0	63.5	60.0	56.5	
5/8	66.5	65.0	62.0	55.5	65.0	65.0	61.5	57.5	
11/8	67.0	65.5	61.5	55.0	67.0	67.0	60.0	58.5	
18/8	68.0	67.0	63.5	56.0	68.0	67.0	62.0	59.0	
25/8	68.0	68.0	67.0	57.0	67.0	68.0	62.0	60.0	
1/9	70.5	70.0	67.5	58.0	68.5	67.5	63.5	60.0	
9/9	72.0	72.0	67.5	57.0	69.5	69.0	64.5	62.0	
16/9	72.0	74.0	70.0	59.0	71.5	71.0	67.0	64.0	
23/9	74.0	72.0	70.5	59.5	73.0	72.0	66.0	64.0	
29/9	75.0	75.5	72.5	60.0	74.0	74.0	67.0	65.0	
11/10	78.0	78.0	75.5	61.5	75.0	76.5	68.0	68.0	
18/10	78.0	79.0	76.5	65.0	76.5	78.5	70.5	68.0	
27/10	79.0	77.0	77.5	64.5	75.0	78.0	69.0	69.0	
3/11	*	81.0	82.0	65.0	80.0	84.0	71.0	72.0	

$\bar{\bar{X}}$ = Period of medicated nut treatment.

Figure 2.6



Mean Liveweights (kg) For Each Group At Each Weighing.

Table 2.13

Mean ($\pm$ Standard Error) Of The Mean Daily Growth Rate (kg/day) In Pre-Treatment, Treatment And Post-Treatment Periods.

		Pre-Treatment	Treatment	Post-Treatment
Group 1	Mean	0.122	0.114	0.149
	S.E.	0.02	0.007	0.008
Group 2	Mean	0.131	0.103	0.151
	S.E.	0.05	0.02	0.02
Group 3	Mean	0.092	0.109	0.163
	S.E.	0.02	0.008 +	0.02

+ Significant ($P < 0.05$) difference between two periods by analysis of variance.

the next 36 days respectively for groups 1-3.

Group 1 suffered a decline in mean liveweight in mid-winter (12.5 g/day between June 26 and July 28) due largely to a decrease in pasture quantity. The liveweight gains of groups 2 and 3 continued at an even rate at this time (134 g/day for both groups) until their mean liveweights exceeded those of group 1 by approximately 2 kg. At the end of the trial the difference in mean liveweights between the groups (1.1 kg) was less than the 2.3 kg difference at the start of the trial.

The mean liveweight gains over the entire trial were 116.6 g/day, 114.2 g/day and 111.9 g/day for group 1, 2 and 3, respectively.

There were no statistically significant differences in growth rates between the groups within any experimental period (see table 2.13). There was a significant ($P < 0.05$) increase in the mean daily growth rate of group 3 in the post-treatment period compared with the treatment period. This may represent a compensatory weight gain in this group which was most severely affected during the period of adaptation to the experimental procedures or be only as a result of their poor growth rate in the treatment period because of this adaptation.

2.5.4 Acceptance of Medicated Nuts.

Problems were experienced initially in persuading the deer to accept the deer nuts. Approximately 30% of the nuts remained uneaten in the early stages of the trial. These problems were not fully overcome until after the first treatment with medicated nuts.

Observations of the feeding behaviour showed no difference in the palatability between the medicated and non-medicated nuts. Thus the poor egg and larval count responses observed following the first medicated nut treatment was due to failure to adapt to supplementary feed *per se*. rather than the palatability of the medicated nuts. When the deer had adapted to the full ration,

acceptance was the same for medicated and non-medicated nuts.

The individual differences in supplement intake may be demonstrated by faecal larval counts of three individuals provided with the medicated nut ration (see figure 2.7). 306 had a high larval count prior to the first treatment (approximately 200 lpg) and this was reduced to 6.75 lpg at the conclusion of the first treatment. This count remained low for the remainder of the treatment period. Conversely 308 had a higher larval count at the end of the first treatment (94.5 lpg) than at the beginning (60 lpg). The larval count continued to rise to a peak of 285 lpg 15 days after the first medicated nut treatment finished. Larval counts for 301 were intermediate between 306 and 308. The larval count of approximately 50 lpg in this deer at the start of the first treatment was reduced to 15.25 lpg by the end of the treatment. The count continued to decline for another week before rising rapidly to a peak of 162.5 lpg by the time the second treatment was initiated. This count was reduced to zero five days after commencement of the medicated nut course.

This variability may be due to a variable consumption of the medicated nuts by the animals, though individual consumption was not measured. By the second treatment all animals were eating their full ration of nuts and the larval counts fell to zero in all animals within seven days. The count remained low for the remainder of the treatment period.

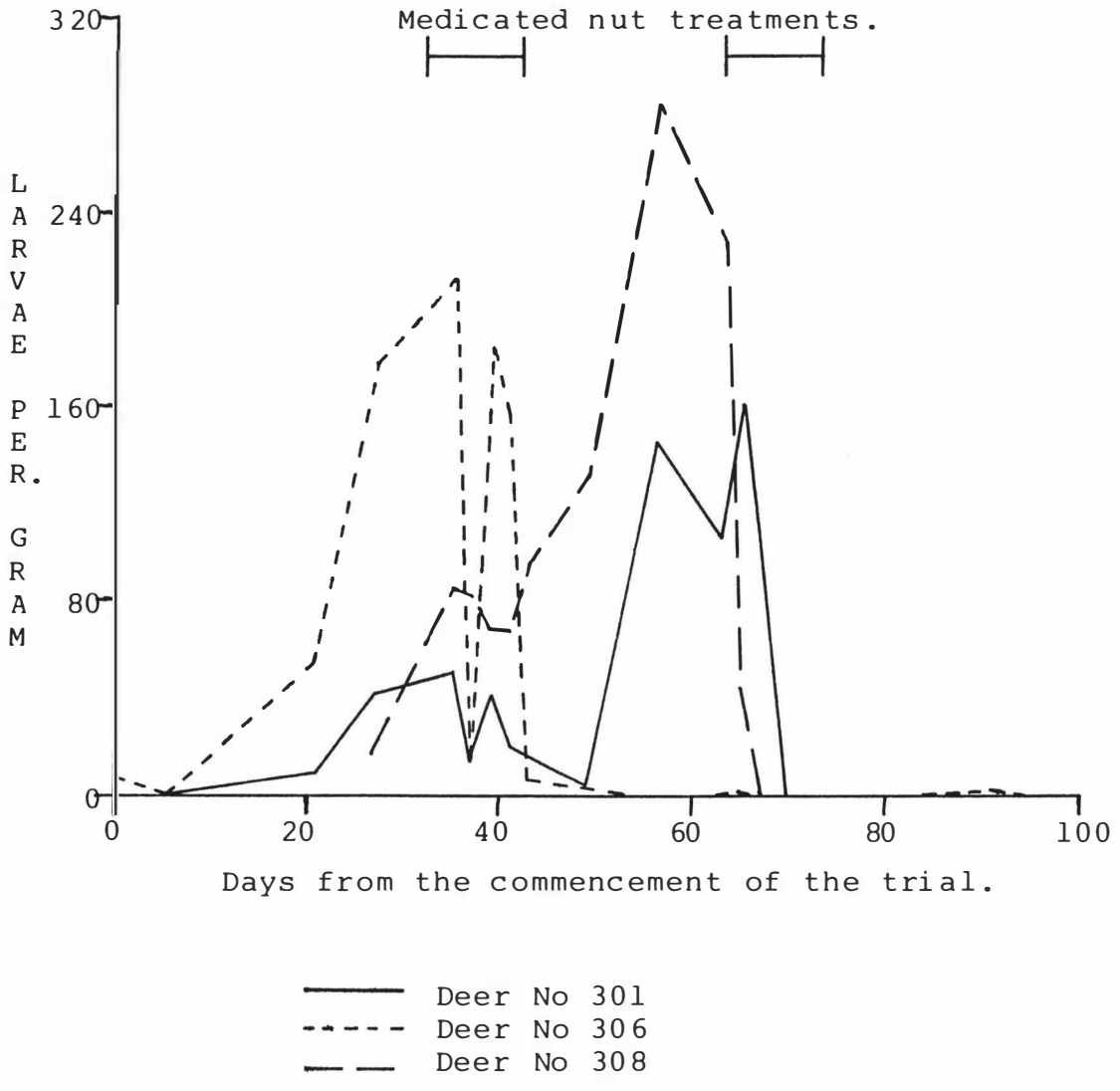
The temporary reductions in larval counts by 308 and 301 were possibly due to a transient suppression in larval output by the adult worms due to the ingestion of sub-therapeutic quantities of anthelmintic. However, it may have been merely sampling error.

2.6 Part 2: Adult Deer.

A second study was conducted to investigate the effectiveness of medicated nuts in adult deer and using

Figure 2.7

Individual Faecal Lungworm Larval Counts.



a single treatment regime only.

The study was carried out in order to gain additional data on the effectiveness of the product, and to determine if the dose rate of 2 mg albendazole/kg liveweight/day was sufficient in adult deer.

The deer used for this study were accustomed to deer-nut rations so it was possible to gain data from a group which had previously been fed nuts and therefore in which acceptance of the feedstuff would not interfere with the investigation.

2.7 Materials and Methods.

2.7.1 Animals.

Sixteen mature red deer hinds from the Massey University Deer Unit were used.

2.7.2 Experimental Design.

Experimental design is summarized in figure 2.8.

Medicated nuts were fed at a rate of 1 kg per animal per day (200 mg albendazole/deer/day) for ten consecutive days from May 10. Additional non-medicated nuts and meadow hay were fed at a separate feeding to provide a maintenance ration of approximately 2 kg DM/ deer/day.

Faecal samples were collected per rectum prior to treatment and at two to eight day intervals for 37 days. Animals were set-stocked on approximately 0.25 hectares.

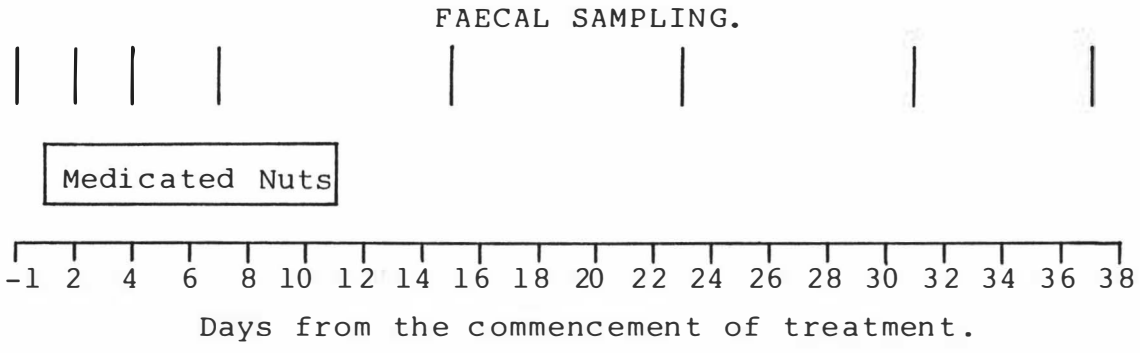
No untreated "control" animals were available because of management requirements.

2.8 Statistical Analysis.

One-way analysis of variance was performed on the egg and larval count data with time.

Figure 2.8

Diagrammatic Summary Of The Experimental Procedures For Part 2, Adult Deer.



2.9 Results.

2.9.1 Faecal Larval Counts.

Data is presented in table 2.14.

Prior to the commencement of the treatment 92% of the animals sampled had faecal larval counts above zero (mean 2.17 lpg). The maximum count was 7.75 lpg. By the second day of treatment the prevalence had been reduced to 38% (mean 0.23 lpg), by the fourth day to 7% (mean 0.02 lpg), and by the seventh day, the prevalence was 8% (mean 0.02 lpg).

No further larvae were detected from post-treatment day four until the end of sampling on day 36.

Analysis of variance showed the decrease from the pre-treatment counts to be significant ($P < 0.01$).

2.9.2 Faecal Egg Counts.

Data is presented in table 2.15.

Prior to the commencement of the treatment 29% of the animals sampled had faecal egg counts of 50 epg or more. The maximum count was 100 epg. On the second day of the treatment the faecal egg counts were all zero, but on the fourth day there were two deer (14%) with egg counts (mean 10.7 epg). No further eggs were detected from treatment day four until the end of sampling on day 36.

Analysis of variance showed the decrease from the pre-treatment counts to be significant ($P < 0.01$).

2.10 Discussion: Weaner Deer.

2.10.1 Faecal Larval Counts.

The faecal *D.viviparus* larval counts at weaning on March 17 were low (mean 7.00 lpg), when compared with those in the herd discussed in Chapter 3. However, counts rose rapidly over the following five weeks to reach a

Table 2.14

D. viviparus Faecal Larval Counts (lpg) From 16 Adult Deer Used In Part 2.

	Days from commencement of treatment.							
Number	-1	2	4	7	14	22	30	36
1	1.25	*	*	*	*	*	0	*
3	0.75	0	0	0	0	0	0	0
5	1.75	*	0	0	0	0	0	0
6	*	0.5	*	*	0	0	0	0
11	0.25	0	0	0	0	0	0	0
12	0.25	0	0	0	0	0	0	0
13	*	*	0	0	*	*	0	0
14	0	0	0	0	0	0	0	0
15	7.75	0.25	0	0	0	0	0	0
16	5.0	1.5	0	0.25	0	0	0	0
18	4.5	0.25	0	0	0	0	0	0
19	2.25	0	0	0	0	0	0	0
31	0.75	0	0	0	0	0	0	0
34	1.5	0	0.25	*	0	0	0	0
35	*	0.5	0	0	0	0	0	0
38	*	0	0	0	*	0	0	0
Mean	2.17	0.23	0.02	0.02	0	0	0	0
Range	0-7.75	0-1.5	0-0.25	0-0.25	0	0	0	0
Prevalence %	92%	39%	7%	8%	0%	0%	0%	0%

* Not sampled

Table 2.15

Gastrointestinal Parasite Faecal Egg Counts (epg) From 16 Adult Deer Used In Part 2.

	Days from commencement of treatment.							
Number	-1	2	4	7	14	22	30	36
1	50	*	*	*	*	*	*	*
3	0	0	0	0	0	0	0	0
5	0	*	0	0	0	0	0	0
6	0	0	*	*	0	0	0	0
11	50	0	0	0	0	0	0	0
12	0	0	0	0	0	0	0	0
13	*	*	0	0	*	*	0	0
14	0	0	0	0	0	0	0	0
15	0	0	0	0	0	0	0	0
16	0	0	0	0	0	0	0	0
18	0	0	0	0	0	0	0	0
19	0	0	0	0	0	0	0	0
31	100	0	50	0	0	0	0	0
34	50	0	0	*	0	0	0	0
35	0	0	100	0	0	0	0	0
38	*	0	0	0	0	0	0	0
Mean	17.9	0	10.7	0	0	0	0	0
Range	0-100	0	0-100	0	0	0	0	0
Prevalence %	29%	0%	14%	0%	0%	0%	0%	0%

* Not sampled

peak mean count of 104 lpg. Had anthelmintic treatment not been instigated at this time it is probable that clinical dictyocauliasis would have resulted subsequently. Wilson (1981) reported clinical dictyocauliasis in weaner farmed red deer with a mean larval count of 33.8 lpg (range 26.6 to 133 lpg), though a less sensitive larval extraction system was used at this time.

There was a statistically significant reduction in mean D. viviparus faecal larval counts during the anthelmintic treatment period in all groups, regardless of treatment and grazing management. There were no significant differences between the three groups in either the pre-treatment or treatment periods. Thus both methods of management, (set-stocking or rotational grazing) and anthelmintic administration, (oral drenching or medicated nuts) were equally effective in reducing the faecal larval counts during the treatment period. Mean faecal larval counts increased significantly in all groups in the post-treatment period. However, the lack of a significant difference between the two set-stocked groups (group 2 and 3) in this period indicates reinfection patterns following both medicated nut and oral drench treatments were the same under the same management. The higher D. viviparus faecal larval counts in the rotationally grazed group (group 1) during spring were probably due to a greater rate of reinfection which may have been associated with a number of factors:

i) Pasture length in the set-stocked compounds became very short, whereas the rotationally grazed group were grazing pasture on average 6-12 cm long. Thus the conditions on the rotationally grazed pasture would have been more suitable for larval survival in the winter conditions (Rose, 1956).

ii) The rotationally grazed deer consumed an estimated 2 kg/head/day of pasture DM compared with approximately 0.3 kg/head/day for the set-stocked deer. Thus the opportunity for larval ingestion was greater in the rotationally grazed deer.

iii) The rotationally grazed group were exposed to

pasture larval contamination, albeit small, from the other deer on the deer unit whereas the set-stocked deer were not.

The spring increase in faecal larval counts, although statistically significantly higher than during the treatment period, was not large. The maximum mean count for any group was 13 lpg, (group 1 on August 25) with a maximum count of 40.75 lpg for any individual (107 on August 25). Faecal larval counts in all animals tended to decrease spontaneously from a peak and two animals had zero larval counts by the end of the trial on November 3. This spontaneous reduction in larval count in spring brings into question the need to drench weaner deer all year as has been recommended elsewhere (Mason, 1979; McAllum, 1979).

The second oral drench appeared to be substantially less effective than the other three anthelmintic treatments. However, this may been only the result of sampling variation. If the effect was real it was most likely because of an inadequate dose given at this treatment since that response was inconsistent with that of any other use of this anthelmintic (Wilson, pers. comm.). The low faecal larval counts at the time of the fourth anthelmintic treatment (mean 1.5 lpg over group 1 and 2) would suggest that this treatment was not necessary. However, this and the need for any subsequent anthelmintic treatments, could only be determined if constant monitoring of faecal larval counts was performed as recommended by Wilson (pers. comm.).

2.10.2 Faecal Egg Counts.

The individual faecal gastrointestinal parasite nematode egg counts were more variable than the larval counts because of the methods of faecal examination used. In the system used, one egg observed equated to 50 epg of faeces, whereas one larva observed equated to 0.25 lpg of faeces. The variation was overcome for statistical purposes by pooling data within experimental groups and

pre-, during, and post-treatment periods.

During the treatment period group 2 had a statistically significant lower mean faecal egg count than either groups 1 or 3. The difference between groups 1 and 2 was probably due to management factors (rotationally grazed v's set-stocked) which influenced reinfestation, as discussed previously (2.9.1) for D. viviparus larvae. The statistically significant difference in the treatment period between groups 2 and 3 was due entirely to the poor response in group 3 to the first medicated nut treatment.

At the time of the first 10-day medication some of the deer were not fully accustomed to eating the nuts provided. This resulted in approximately 30% of the nuts remaining uneaten which in turn resulted in some animals having received a dose of anthelmintic less than the desired 2 mg/kg/day and therefore a less effective depression in egg and larval counts. This has been demonstrated by examining the faecal larval counts of three individuals (see section 2.4.4). This problem was overcome before the second medication period but the continuing high counts up to the second medication had an effect on the mean log egg count for the treatment period, by increasing the total faecal egg count for the treatment period. (The reduced effectiveness of the first treatment did not cause a significant statistical difference in the larval count data, this was probably because of the higher sensitivity of the larvae extraction method over the egg extraction method). Thus while statistically there was a poorer response to medicated nuts, the response to the subsequent two treatments and the response in the adult deer used in this study, indicate that when consumption reaches the required level, medicated nuts are a perfectly adequate method of anthelmintic treatment.

In both groups 1 and 2 there was a significant ($P < 0.05$) reduction in faecal egg counts between the pre-treatment and treatment periods. This was followed by a non-significant rise in the post-treatment period. However, in group 3, the significant reduction ($P < 0.05$)

was between the pre- and post-treatment periods with a continual decline in mean faecal egg count over the three periods. This again is a result of an initially poorer response to the medicated nuts as a result of incomplete consumption. However, the very low post-treatment counts would indicate that the treatment was effective against gastrointestinal nematodes.

The less complete suppression of faecal gastrointestinal parasite egg counts when compared with faecal lungworm larval counts following the treatments with medicated nuts, could be in contrast with statements by other authors that gastrointestinal parasites are easier to control than D. viviparus in deer (McAllum, 1976; Mason, 1981a). However, these authors were referring to oral drenches and not medicated nut treatment.

Despite the higher faecal egg counts in the treatment period for group 3 a corresponding higher faecal egg count in the post-treatment period did not occur and the mean count was not significantly different form group 2.

There was a rise in the faecal egg counts in spring as observed with the faecal larval count data. This was especially pronounced in group 1, (though not statistically significantly more than in the other groups). In some individuals, counts were greater than were observed in autumn, eg. deer 105, 106, 107, and 208. Thus it is possible that gastrointestinal parasitism could be a greater problem than lungworm in deer nine to ten months of age in some situations.

Some animals had persistent zero faecal egg counts in spring suggesting that they had developed a high degree of resistance to gastrointestinal nematodes.

2.10.3 Liveweights.

The only significant difference that was detected in the liveweight data was in group 3 between the treatment and post-treatment periods ($P < 0.05$). The faster growth rate in the post-treatment period for this group may have been compensation for the poorer growth

rates early in the trial while animals were still not accustomed to their nut ration. However, it may have been merely as a result of the poor growth rates in the treatment period. The growth rates achieved by group 3 in all three periods was not significantly different from that achieved by the other groups. Therefore the observed difference cannot be attributed to a treatment effect.

Growth rates of 116 g/day from the start of the experiment until September were higher than the 48 g/day for the same period reported by Drew et al. (1978). The growth rate from September to early November (64 days) was 204 g/day compared with 296 g/day for September to December (87 days) reported by the above authors. However, growth rates increased during November and December (Wilson, pers. comm.) to compare with those of Drew et al. (1978). The higher winter growth rates achieved in this trial could have been due to:

i) Latitude and location, leading to more mild winter conditions and better pasture growth. (The liveweights at the end of March and beginning of October were similar to those recorded by Asher, Adam and Langridge (1981) for red deer in the Waikato).

ii) Genetic differences between the stock.

iii) Animal health and parasitic burden differences.

iv) Management differences. The deer in the work carried out by Drew et al., (1978) were fed ryegrass-white clover pasture and hay ad. lib. over winter, whereas the deer in this trial were fed ryegrass-white clover pasture only or a small amount of pasture and deer nuts with a very small amount of hay (depending on their experimental group). The feed quality of pasture alone or the concentrates as fed here is better than hay as fed by Drew et al. (1978). This probably was the major reason for our higher growth rates.

2.10.4 General.

The effectiveness of medicated nut anthelmintic treatment has been shown previously by overseas workers. Bockeler and Segebade (1977) used fenbendazole at 5 mg/kg liveweight on two consecutive days and reported a 97% to 98% reduction of worm burdens in fallow deer. Kalivoda and Chroust (1971) reported a 96% reduction in faecal egg counts in fallow deer following a five day treatment with thiabendazole in a supplementary feed at 50 mg/kg liveweight. Both authors reported that there were no problems with acceptance of the medicated nuts. Kalivoda and Chroust (1971) did not state how the nuts were fed but the animals were being fed supplements routinely at the time of the trial. Bockeler and Segebade (1977) fed the pellets on the ground. However, a major difference exists between these two reports and the current study. In both of the above trials the full therapeutic dose of the anthelmintic used was given each day, resulting in a total dose of two and five times the normal oral drench dose. In this trial one fifth of the normal therapeutic dose was given each day resulting in a total dose of twice the normal therapeutic dose over a ten day period. This prolonged administration of the anthelmintic may be beneficial to its efficacy as deer have been shown to metabolise some anthelmintics at a faster rate than sheep and cattle (Mason, 1982; Watson et al., 1983). Anthelmintics have been shown to be more effective in sheep following treatment on consecutive days (Pritchard et al., 1978), and this probably applies in deer also.

Throughout the trial some animals consumed more nuts than others due to individual preference, appetite, and possibly social factors. This must have led to variation in the intake of anthelmintic and is a problem inherent in any feed or water medication system. However, this appears not to hinder the effectiveness of the treatment at the dose rates used here as egg and larval counts of all deer in the medicated nut treatment group eventually reached zero, as did those following oral drench

treatments. This potential problem of varying intakes can be minimised by feeding the medicated nuts at a different time of the day to any other supplementary feed and feeding it in a long line to minimise social interactions between animals.

2.11 Discussion: Adult Deer.

The medicated nuts fed to the adult deer at the same dose rate as for the weaners were effective in reducing the faecal larval and egg counts. Both the egg and larval counts reached zero by the end of the 10-day treatment period, and stayed at zero for at least 26 days following treatment.

Prior to treatment the percentage of the herd shedding larvae and eggs was high, as was the mean larval count, when compared with those parameters reported in a survey of New Zealand deer (Mason and Gladden, 1983). This probably was due to the policy of not drenching adult stock on this unit and possibly the high stocking density, whereas Mason and Gladden showed that many farmers drench adult deer. Further, the stocking density on commercial farms is unlikely to be as high as on the Massey University Deer Unit.

The adult deer were much more willing to accept the supplementary feed than the weaner deer though individual variation in intake was observed.

2.12 Conclusions.

Data indicates that albendazole medicated nuts in a programme of 10 consecutive days of treatment at a dose rate of 2 mg albendazole per kg liveweight per day for three treatments, with 21 days between treatments, is as effective as four treatments with albendazole at 10 mg per kg, with 21 days between treatments in reducing *D. viviparus* faecal larval counts and faecal gastro-intestinal nematode egg counts.

The 10-day low dose treatment regime had no effect on

the later D. viviparus larval or gastrointestinal egg counts or on the liveweight gains, up to 11 months of age. The medicated nuts were no less palatable than non-medicated deer nuts.

The potential applications of a medicated nut anthelmintic treatment system are limited so this system is unlikely to substantially replace oral anthelmintic drenching of farmed deer in New Zealand. Other factors may also influence the application of this method of treatment:

i) The nut medication system is costly due to the quantity of nuts that have to be fed and the cost of deer nuts in New Zealand. At the time of the trial, deer nuts cost $344 per tonne (34c/kg). If a deer was fed 0.5 kg nuts/day for a 14 day adaptation period, a total of 30 days of treatment and 42 days between the treatments, then the total weight of nuts fed is 43 kg at a cost of $14.70. Additional to this would be the cost of albendazole. This cost must be compared with $1.20 per deer for a course of four oral albendazole treatments.

Some of the cost of this regime may be offset by additional liveweight gain if pasture quality or quantity were inadequate, i.e. if some form of supplementary feeding was necessary. However, during this trial $160 of deer nuts were fed to each deer for an additional $90 value of venison carcass. Many deer farmers already feed large amounts of supplementary feeds, often in the form of deer nuts, and in this case the cost of the medication system may well be no greater than the cost of oral anthelmintic.

ii) The medication system does require some planning to be effective, because of the 10-day on, 21-day off system. This may well be sufficient to discourage some farmers from using it.

The major advantage of the system using medicated nuts is that deer which cannot be given anthelmintic treatment by oral drenching for any reason can be given anthelmintic treatment.

CHAPTER 3

STUDY OF RESPONSES TO ANTHELMINTIC TREATMENT OF DEER ON A COMMERCIAL FARM.

3.1 Introduction.

There are differences of opinion about the optimum period of anthelmintic use against D. viviparus lungworm in deer. McAllum (1979) and Mason (1979a) recommend three-weekly drenching from weaning until one year of age. However, Wilson (pers. comm.) considers that this recommendation is excessive and that a planned approach incorporating as few as three drenches from weaning may be all that is necessary.

In practice a wide range of treatment regimes varying from nil to 17 drenches per year are used (Mason and Gladden, 1983).

This study was initiated to investigate aspects of parasite control in weaner deer on a commercial deer farm with particular reference to the study of faecal egg and larval counts in response to anthelmintic treatment and the period over which an anthelmintic drenching programme should continue.

3.2 Materials and Methods.

3.2.1 Animals and Location.

Eighty four male and female weaner red deer on a commercial deer farm of approximately 500 deer were used. The farm was on flat land in the northern Manawatu with a stocking rate of 14 deer per hectare. Only deer had been grazed on the property during the previous two years.

3.2.2 Timing.

This study commenced at weaning (March 15) and was

to have concluded on November 30. However, commercial considerations dictated that all the deer in this trial were sold by the farmer and thus the final faecal samples were collected on June 29 and the trial concluded on July 14. Data is presented, despite being incomplete, as several interesting and significant findings became evident during this time.

3.2.3 Allocation To Groups.

The deer were weighed at weaning, ranked by liveweight within each sex, and systematically assigned to one of two groups so that each was balanced by sex and mean and range of liveweight. The groups were then assigned to one of two treatments:

Group 1: n=41
Treated with oral albendazole (10 mg/kg) as outlined in Chapter 2 according to the schedule in table 3.1.

Group 2: n=43
Treated with oral albendazole (10 mg/kg) as above according to the schedule in table 3.1.

3.2.4 Management.

All animals were grazed together in a slow rotation around several ryegrass-clover paddocks in one mob throughout the experimental period. Pasture was supplemented daily with silage when pasture quantity declined in May. Silage was estimated to make up approximately 50% of the feed intake.

3.2.5 Sampling.

Animals were faecal sampled per rectum at weaning (March 15). Up to 12 deer from each group were randomly selected at each subsequent sampling for faecal collection as indicated in table 3.1. Animals were faecal sampled at weekly intervals for the first three weeks and at three-weekly intervals thereafter. Faecal

Table 3.1

Schedule Of Anthelmintic Treatments, Faecal Sampling And Weighing During The Trial Period.

Date:	March 15	March 22	March 29	April 6	April 26	May 17	June 7	June 29	July 14
Weeks From Start	0	1	2	3	6	9	12	15	18
Group 1	D.S.W	S.	S.	D.S.	D.S.W.	D.S.	S.	S.	W.
Group 2	D.S.W.	S.	S.	D.S.	D.S.W.	S.	D.S.	D.S.	W.

D. = Oral drench with albendazole (10 mg/kg).

S. = Faecal samples collected for determination of faecal larvae and egg counts.

W. = Weighed.

samples were examined for nematode eggs and larvae as described earlier (Section 2.1).

All animals were weighed on March 15, April 26, and July 14.

3.3 Statistical Analysis.

3.3.1 Faecal Larval and Egg Counts.

Data was log-transformed following the addition of 10^{-5} to overcome the problem of zeros.

Comparisons of mean log faecal egg and larval counts between groups at each sampling and between successive samplings within a group were performed by Student's t test.

3.3.2 Liveweight Data.

Liveweights were analysed independently for both sexes. Comparisons were made between groups at each sampling using Student's t test.

3.4 Results.

3.4.1 Faecal Larval Counts.

Faecal larval counts are presented in tables 3.2 and 3.3.

A summary of data is presented in figure 3.1.

The high mean faecal larval count of all sampled animals (134 lpg) at the start of the trial was reduced by 98.9% to 1.5 lpg seven days post-treatment. These low counts persisted 14 days post-treatment, but had risen slightly 21 days post-treatment to be 93.4% of pre-treatment counts. Three weeks after the subsequent three-weekly treatments, the mean larval counts remained at approximately the same low levels (8.87 lpg, 4.7 lpg and 3.4 lpg 21 days following each treatment, respectively).

During the next period when only one of the groups

Table 3.2

Faecal Lungworm Larval Counts (lpg) From Selected Individuals In Group 1.

Number	Sex	15/3	22/3	29/3	6/4	26/4	17/5	7/6	29/6
					Days From Weaning				
		0	7	14	21	42	63	84	105
1	M	120	0	0	0	*	*	2.25	1.25
2	F	278	0.5	0.25	7.93	*	*	*	*
3	F	334	0	0.25	7.5	*	*	*	*
4	F	33.3	0	0.25	7.5	*	*	0.5	*
5	F	80	*	*	*	*	*	*	*
6	M	125	0	0.69	3.5	*	*	*	*
7	M	103	*	*	*	*	1.75	1.5	2.25
8	M	41.5	*	*	*	*	*	*	*
9	M	681	2.25	0.25	2.7	*	1.0	*	*
10	M	47.5	*	*	*	*	*	*	0.75
11	F	26	2.0	0	2.25	*	*	*	*
12	M	*	*	*	*	*	*	*	34.0
13	F	144	*	*	*	*	*	*	*
14	M	*	*	*	*	*	*	*	13.3
15	F	80.8	*	*	*	*	*	*	*
16	M	186	2.0	0.25	12.75	*	0.25	0	*
17	F	*	*	*	*	*	*	*	*
18	M	80	*	*	*	*	*	*	25.5
19	F	*	*	*	*	*	*	*	*
20	M	*	*	*	*	*	0	*	*
21	F	*	*	*	*	0	*	*	*
22	F	24	*	*	*	1.25	*	0.25	*
23	F	*	*	*	*	4.5	*	*	4.5
24	M	231	1.5	0.25	4.5	*	12.5	*	*
25	M	110	*	*	*	0.75	*	*	*
26	F	*	*	*	*	1.0	*	*	*
27	M	37.5	*	*	*	*	*	*	*
28	F	66.5	1.25	0	11.5	*	*	*	*
29	M	*	*	*	*	0.75	0.25	*	*
30	M	*	*	*	*	*	*	*	*
31	M	*	*	*	*	*	*	1.75	2.0
32	F	*	*	*	*	*	*	*	*
33	F	*	*	*	*	0.75	*	2.75	*
34	M	*	*	*	*	*	0.75	*	3.5
35	M	*	*	*	*	*	*	1.5	*
36	M	*	*	*	*	7.5	*	*	17.0
37	F	*	*	*	*	*	*	12.3	*
38	F	*	*	*	*	*	4.0	*	*
39	M	*	*	*	*	*	6.0	*	*
40	M	*	*	*	*	*	*	*	*
41	F	*	*	*	*	*	4.25	0.25	*
Number	n =	20	10	10	10	8	10	10	10
Mean Count		141.5	0.95	0.22	6.3	2.06	3.08	2.3	10.4
Range.		24-681	0-2.25	0-0.69	0-15.75	0-1.25	0-12.5	0-12.3	1.25-34
Prevalence%		100%	60%	70%	90%	88%	90%	90%	100%

* = Not Sampled

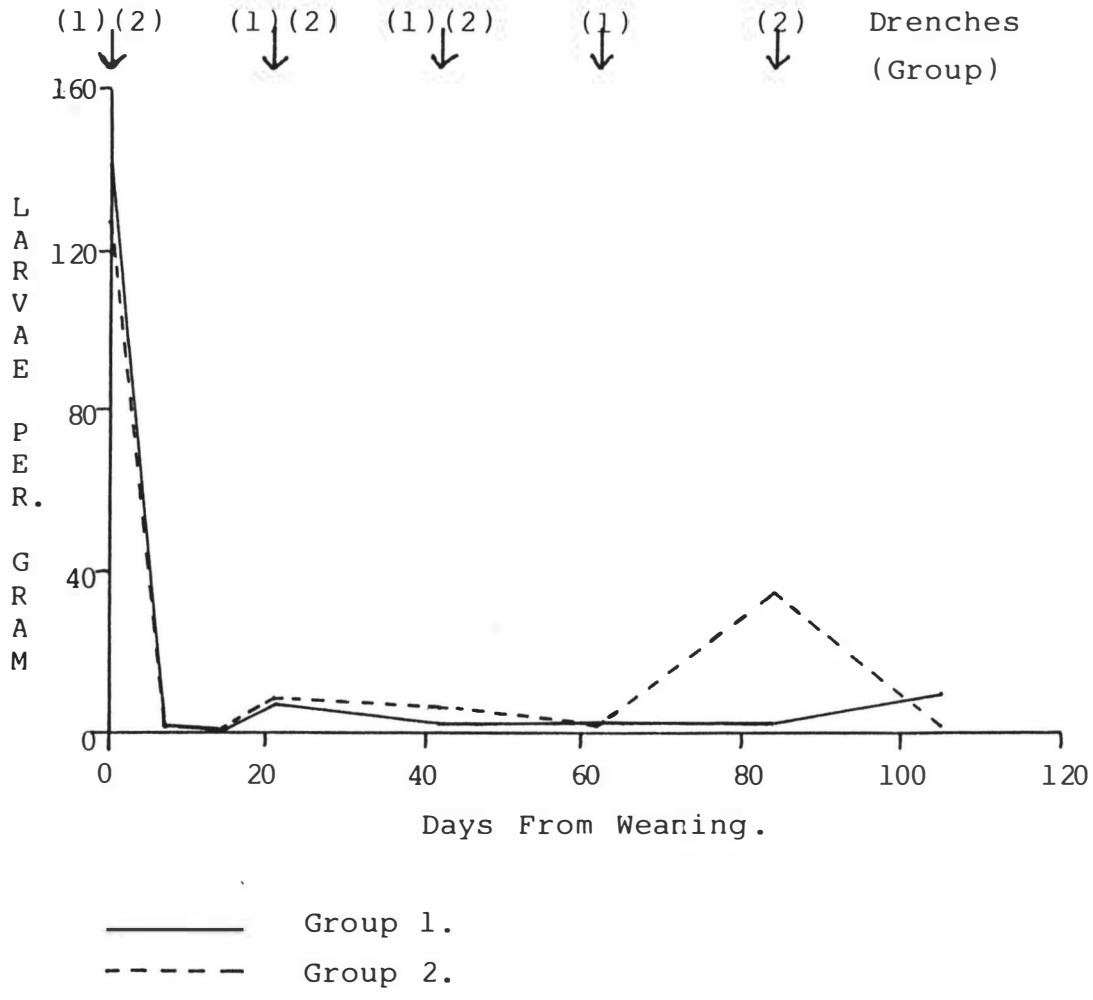
Table 3.3

Faecal Lungworm Larval Counts (lpg) From Selected Individuals In Group 2.

Number	Sex	15/3	22/3	29/3	6/4	26/4	17/5	7/6	29/6
		Days From Weaning							
		0	7	14	21	42	63	84	105
51	F	345	*	*	*	18.0	*	*	*
52	F	*	*	*	*	2.25	*	59.0	*
53	M	*	*	*	*	*	*	*	*
54	F	146	3.25	0.76	4.5	*	*	*	*
55	M	*	*	*	*	0.25	0	*	*
56	M	*	*	*	*	*	*	*	7.25
57	M	37	0.25	0.5	10.25	*	11.0	*	*
58	F	*	*	*	*	*	*	18.3	*
59	F	229	*	*	*	*	*	83.3	*
60	M	84	*	*	*	*	*	69.8	*
61	M	39.6	0	0.25	10.25	*	*	*	*
62	F	*	*	*	*	*	*	36.3	*
63	M	*	*	*	*	*	*	15.5	*
64	F	126	*	*	*	*	*	*	*
65	M	237	*	**	*	*	*	*	0.25
66	F	48.5	0	0	*	5.5	*	*	*
67	F	190	2.0	0.75	37.25	*	*	52.8	0
68	M	63.5	*	*	*	*	0.5	*	0.25
69	M	30.8	0.25	1.5	10.5	*	*	*	*
70	F	350	0	0	3.75	0.5	*	*	*
71	F	178	*	0	0	*	*	*	*
72	M	41	*	*	*	7.75	*	*	*
73	M	123	1.0	0	0	*	*	7.75	0
74	M	24.8	*	*	*	0.5	*	*	*
75	M	148	*	*	*	*	0	*	*
76	M	77	*	*	*	1.25	0.5	*	*
77	M	207	*	*	*	30.8	6.5	*	*
78	M	142	*	*	*	0.25	0.75	*	*
79	M	*	*	*	*	*	*	*	1.75
80	F	20	*	*	*	*	*	1.75	*
81	F	44	0	0	2.5	*	*	*	*
82	M	*	*	*	*	*	*	8.5	*
83	M	*	*	*	*	*	*	*	*
84	F	*	*	*	*	*	*	*	*
85	M	*	*	*	*	1.25	*	*	0
86	F	*	*	*	*	*	0	*	*
87	M	*	*	*	*	*	*	*	0.75
88	M	*	*	*	*	*	0.75	*	*
89	F	*	*	*	*	4.75	*	*	*
90	M	*	*	*	*	*	*	*	0
91	F	*	*	*	*	*	*	*	0
92	M	*	*	*	*	*	0	*	*
93	F	*	*	*	*	*	*	*	*
Number	n =	23	9	10	9	12	10	10	9
Mean Count		127	0.75	0.38	8.78	6.1	2.0	35.3	1.03
Range.		20-350	0-3.25	0-1.5	0-37.25	0.25-30.8	0-11	8.5-83.3	0-7.25
Prevalence%		100%	55%	50%	78%	100%	60%	100%	50%

Figure 3.1.

Mean Faecal Lungworm Counts
For Groups 1 And 2.



was treated, larval output of the untreated group increased by fourfold (group 1), and seventeenfold (group 2) after six weeks. However, the mean counts were still below 40 lpg.

Prevalence of larval counts in sampled animals was 100% in both groups prior to treatment. This was reduced to 57% of sampled animals seven days post-treatment but rose steadily to 84% of sampled animals 21 days post-treatment. At subsequent 21 day post-treatment samplings the prevalence ranged from 50% to 95% of sampled animals. When treatment had not been given for 42 days the prevalence of counts returned to 100% of sampled animals.

Mean faecal larval counts at the start of the trial (141.5 lpg, and 127.4 lpg for groups 1 and 2 respectively) were significantly ($P < 0.01$) reduced to 1.65 lpg, and 1.35 lpg seven days post-treatment in groups 1 and 2, respectively.

Between 14 and 21 days post-treatment there was a significant ($P < 0.05$) rise in larval count in group 1. The pattern of change was similar in group 2 but these changes were not statistically significant.

There were no significant differences between the groups following the first three anthelmintic treatments when both groups were treated. Following the subsequent treatments when only one group was treated the untreated group had a significantly ($P < 0.01$) higher larval count at the subsequent sampling.

3.4.2 Faecal Egg Counts.

Faecal egg counts are presented in tables 3.4 and 3.5. A summary of the data is presented in figure 3.2.

Faecal egg counts were low (mean 157 epg) at the commencement of the trial but the prevalence of positive egg counts in sampled animals was 86%. Following the first anthelmintic treatment both the mean count and prevalence of counts were reduced (75 epg and 10.5% respectively) at seven days post-treatment. The mean count did not change markedly either 14 or 21 days

Table 3.4

Faecal Gastrointestinal Egg Counts (epg) From Selected Individuals In Group 1.

Number	Sex	15/3	22/3	29/3	6/4	26/4	17/5	7/6	29/6
		Days From Weaning							
		0	7	14	21	42	63	84	105
1	M	350	0	0	0	*	*	0	50
2	F	100	0	0	*	*	*	*	*
3	F	0	0	50	100	*	*	*	*
4	F	150	0	0	0	*	*	0	*
5	F	100	*	*	*	*	*	*	*
6	M	200	0	*	0	*	*	*	*
7	M	100	*	*	*	*	0	0	50
8	M	350	*	*	*	*	*	*	*
9	M	50	100	50	*	*	0	*	*
10	M	0	*	*	*	*	*	*	150
11	F	250	0	0	0	*	*	*	*
12	M	**	*	*	*	*	*	*	*
13	F	200	*	*	*	*	*	*	*
14	M	*	*	*	*	*	*	*	200
15	F	150	*	*	*	*	*	*	*
16	M	250	0	0	0	*	0	0	0
17	F	*	*	*	*	*	*	*	*
18	M	*	*	*	*	*	*	*	150
19	F	*	*	*	*	*	*	*	*
20	M	*	*	*	*	*	0	*	*
21	F	*	*	*	*	0	*	*	*
22	F	100	*	*	*	50	*	50	*
23	F	*	*	*	*	50	*	*	50
24	M	50	0	0	0	*	0	*	*
25	M	200	*	*	*	0	*	*	*
26	F	*	*	*	*	50	*	*	*
27	M	200	*	*	*	*	*	*	*
28	F	150	0	0	0	*	*	*	*
29	M	*	*	*	*	0	0	*	*
30	M	*	*	*	*	*	*	*	*
31	M	*	*	*	*	*	*	0	100
32	F	*	*	*	*	*	*	*	*
33	F	*	*	*	*	0	*	100	*
34	M	*	*	*	*	*	50	*	50
35	M	*	*	*	*	0	*	0	*
36	M	*	*	*	*	0	*	*	100
37	F	*	*	*	*	*	*	0	*
38	F	*	*	*	*	*	0	*	*
39	M	*	*	*	*	*	0	*	*
40	M	*	*	*	*	*	*	*	*
41	F	*	*	*	*	*	0	0	0
Number n =		19	10	9	8	9	10	10	11
Mean Count		155.3	10	11.1	12.5	16.7	5.0	15	75
Range.		0-350	0-100	0-50	0-100	0-50	0-50	0-100	0-200
Prevalence %		89%	10%	22%	13%	33%	10%	20%	75%

* = Not sampled

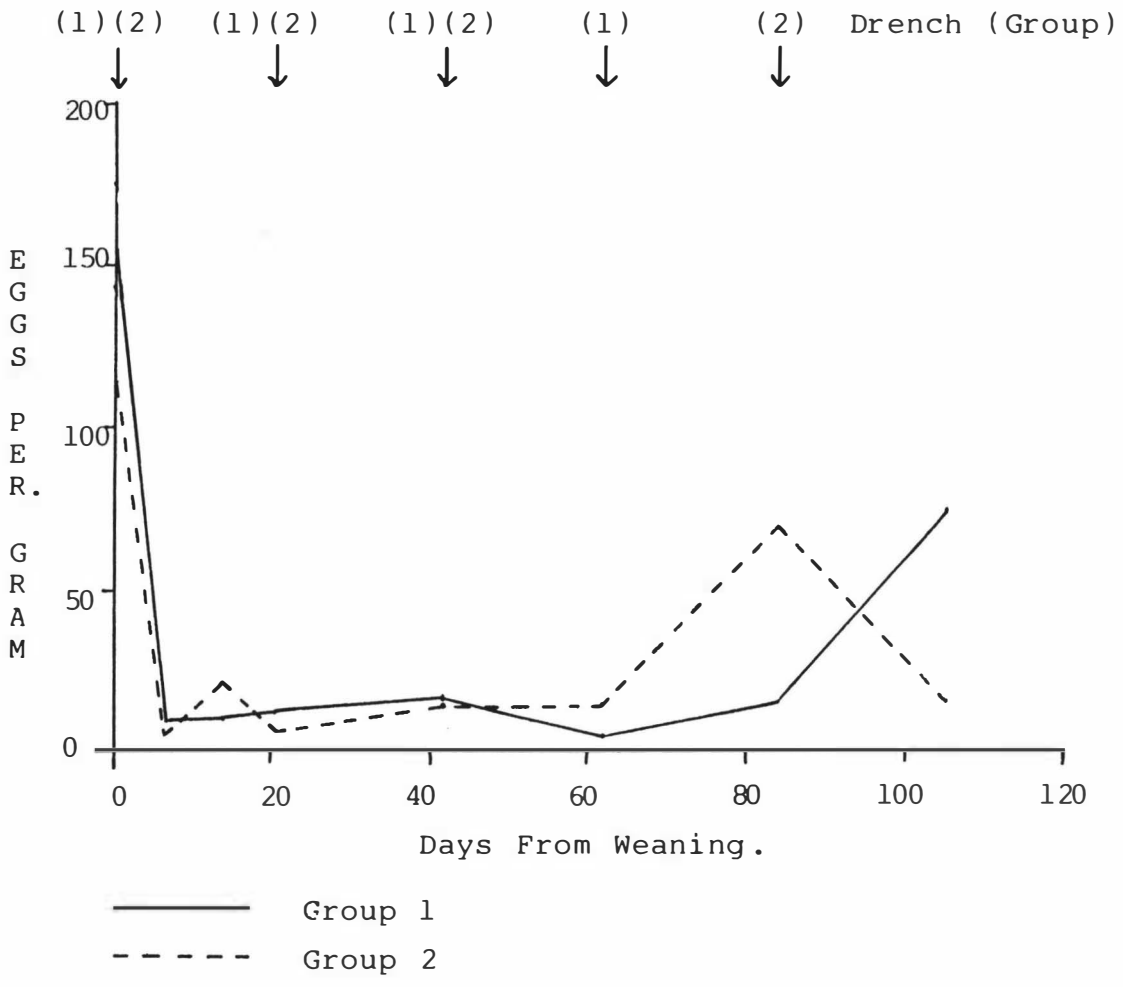
Table 3.5

Faecal Gastrointestinal Egg Counts (epg) From Selected Individuals In Group 2.

Number	Sex	15/3	22/3	29/3	6/4	26/4	17/5	7/6	29/6
		Days From Weaning							
		0	7	14	21	42	63	84	105
51	F	0	*	*	*	0	*	*	*
52	F	*	*	*	*	0	*	50	*
53	M	*	*	*	*	*	*	*	*
54	F	150	0	100	*	*	*	*	*
55	M	*	*	*	*	0	50	*	*
56	M	*	*	*	*	*	*	*	50
57	M	100	0	0	0	*	50	*	*
58	F	*	**	*	*	*	*	100	*
59	F	0	*	*	*	*	*	0	*
60	M	200	*	*	*	*	*	0	*
61	M	100	0	0	0	*	*	*	*
62	F	*	*	*	*	*	*	250	*
63	M	*	*	*	*	*	*	100	*
64	F	100	*	*	*	*	*	*	*
65	M	0	*	*	*	*	*	*	0
66	F	150	50	50	*	0	*	*	*
67	F	150	0	0	0	*	*	0	0
68	M	150	*	*	*	*	0	*	0
69	M	250	0	0	0	*	*	*	*
70	F	350	0	0	0	0	*	*	*
71	F	150	*	0	0	*	*	*	*
72	M	50	*	*	*	0	*	*	*
73	M	100	0	50	50	*	*	150	*
74	M	100	*	*	*	0	*	*	*
75	M	0	*	*	*	*	50	*	*
76	M	100	*	*	*	0	0	*	*
77	M	250	*	*	*	*	0	*	*
78	M	150	*	*	*	0	0	*	*
79	M	*	*	*	*	*	*	*	50
80	F	50	*	*	*	*	*	0	*
81	F	50	0	0	0	*	*	*	*
82	M	*	*	*	*	*	*	50	*
83	M	*	*	*	*	*	*	*	*
84	F	*	*	*	*	*	*	*	*
85	M	*	*	*	*	100	*	*	50
86	F	*	*	*	*	*	0	*	*
87	M	*	*	*	*	*	*	*	0
88	M	*	*	*	*	*	0	*	*
89	F	*	*	*	*	50	*	*	*
90	M	*	*	*	*	*	*	*	0
91	F	*	*	*	*	*	*	*	0
92	M	*	*	*	*	*	0	*	*
93	F	*	*	*	*	*	*	*	*
Number n =		23	9	10	8	11	10	10	9
Mean count		117	5.6	20	6.25	13.6	15	70	16.7
Range.		0-350	0-50	0-100	0-50	0-100	0-50	0-250	0-50
Prevalence%		83%	11%	30%	13%	18%	30%	60%	30%

Figure 3.2.

Mean Faecal Gastrointestinal Nematode Egg Counts For Groups 1 And 2.



post-treatment though the prevalence of counts in sampled animals increased to 18% and 12.5% respectively. The mean count remained similar following the two subsequent three-weekly treatments.

The pre-treatment gastrointestinal parasite faecal egg counts (mean 155.3 and 117.4 epg for groups 1 and 2, respectively) were significantly reduced ($P < 0.01$) to 10 and 5.5 epg for groups 1 and 2, respectively, within seven days of the first anthelmintic treatment, and remained at this low level for as long as three-weekly anthelmintic treatment was continued.

When anthelmintic treatment was given only to one group there was a rise in the prevalence of excretion and faecal egg count of the untreated group at the following sampling three weeks later. The rise in faecal egg count was statistically significant ($P < 0.01$) only on July 29 (105 days post-weaning) when no treatment was given to group 1.

3.4.3 Liveweights.

Liveweights are presented in tables 3.6 and 3.7. A summary of the data is presented in figure 3.3.

At the third weighing the lower liveweight animals had been culled so statistical analysis has been carried out only for those animals with all three weight recordings (n=64).

No significant liveweight differences existed between groups 1 and 2 for either males or females.

3.5 Discussion.

The unexpectedly early conclusion to this experiment prevented the achievement of its main objective of comparing long-term and short-term drenching programmes. However, considerable useful information can be drawn from the data.

Table 3.6

Liveweights (kg), And Means Of Those Deer Which Were Available Throughout The Course Of The Study - Group 1.

Male Number	15/3	26/4	14/7
1	44	48	54
6	42.5	46.5	52
7	46.5	54	61.5
8	34.5	38.5	42
9	40.5	43.5	48
10	47	51.5	60
12	31.5	38	42.5
14	41	44	53
16	49	54	58
18	44	50	52
20	45	50	53
24	37.5	43	51
25	45.5	49	53
27	40	43.5	50.5
29	39	42	42.5
30	42	49.5	53
31	29	34.5	40.5
34	39	46	54
35	44	46	53.5
36	34	36.5	41
39	37.5	42	48
40	50.5	*	*
Mean Of Deer With 3 Weighings.	40.6	45.2	50.6
S.D.	5.26	5.5	6.06
Female			
2	34	38	*
3	36	39.5	*
4	39	43	48
5	32	35.5	*
11	42	47.5	50
13	33.5	42.5	45.5
15	37	42.5	47.5
17	28	35	*
19	29	32.5	*
21	31.5	32	*
22	31.5	36.5	42
23	40	45.5	51
26	41	47	54
28	34	38	*
32	40	46.5	52
33	36.5	41.5	46
37	30.5	33.5	*
38	26	42	46.5
41	38	44	49
Mean Of Deer With 3 Weighings.	36.8	43.5	48.5
S.D.	4.77	3.15	3.37

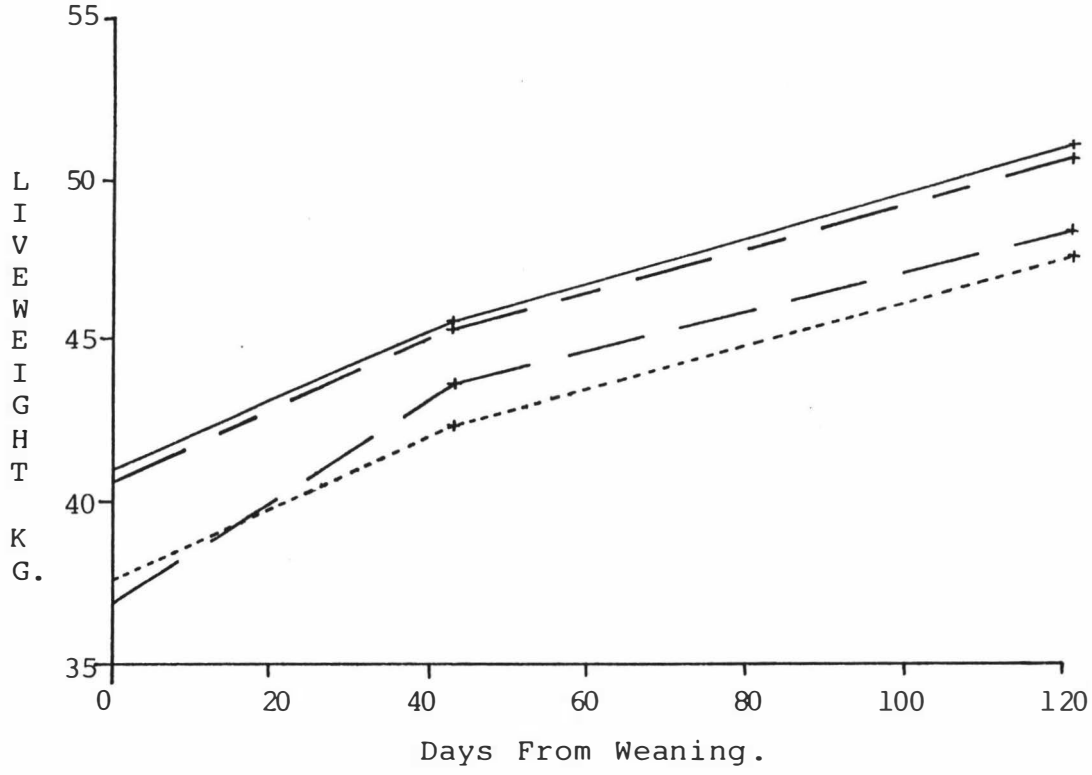
Table 3.7

Liveweights (kg), And Means Of Those Deer Which Were Available Throughout The Course Of The Study - Group 2.

	Number	15/3	26/4	14/7
Male	53	38.5	40	46
	55	39.5	44.5	53
	56	43	49.5	54
	57	34	37.5	45
	60	39	43.5	49
	61	28	33	38.5
	63	47	52	59
	65	37	43.5	44.5
	68	45	46.5	54.5
	69	51.5	58	*
	72	47.5	53	58
	73	45	48.5	49.5
	74	50	59	64
	75	40	46.5	49.5
	76	41	46	54
	77	44	49	52
	78	45.5	50	53
	79	42.5	46.5	51.5
	82	37	42	52.5
	83	29	33	38
	85	42	49.5	56.5
	87	39	40.5	48
	88	46	49.5	58
	90	34.5	37.5	43
	92	48.5	52.5	55
Mean Of Deer With 3 Weighings.		40.9	45.5	51.1
S.D.		5.76	6.39	6.36
Female				
	51	33	38.5	*
	52	33.5	36	*
	54	35.5	40	47
	58	*	29	33.5
	59	31	33	*
	62	28	30	*
	64	32	35	*
	66	*	47.5	53
	67	36	40	45
	70	31.5	36	*
	71	34	39.5	*
	80	37	42	44.5
	81	40.5	45	50
	84	29	34.5	*
	86	37.5	40.5	43.5
	89	35.5	42	48.5
	91	41.5	45.	52.5
	93	37	43.5	50
Mean Of Deer With 3 Weighings.		37.6	42.3	47.6
S.D.		2.16	2.07	3.16

Figure 3.3.

Mean Liveweights (kg) Of Males And Females Of Groups 1 And 2.



3.5.1 Faecal Larval Counts.

The mean faecal larval counts at weaning (134 lpg) are considered to be high by comparison with counts from deer elsewhere (Wilson, pers. comm.). Wilson (1981) reported an outbreak of clinical lungworm in a group of weaned red deer with a mean count of approximately 341 lpg. A nationwide survey by Mason and Gladden (1983) recorded mean larval counts of more than 100 lpg on only two of 108 properties surveyed although their samples were from older deer (sampling carried out in July). It should be noted however, that the modified Baermann's technique used in these experiments is more sensitive than the method which was probably used by the above authors so a direct comparison of counts may not be valid. No deer in Mason's survey showed signs of clinical parasitism at the time and it could not be determined whether the burdens had affected their growth rates.

The larval counts observed in this study at weaning (March 15) were considerably higher than those on the Massey University Deer Unit (mean 6.6 lpg), where the weaning weights were 2.7 kg and 7.0 kg higher for males and females, respectively, (see Chapter 2). Though better pasture quality on the Massey Unit would have had a pronounced effect on liveweights it could be suggested that the high counts observed in this study may have been associated with worm counts high enough to have caused a depression of weight gain.

The high D. viviparus faecal larval counts observed at weaning on this property indicate that clinical dictyocauliasis could become a problem before weaning in some situations. The management factors that were considered to be significant in the development of the large burdens in the weaners on this property were:

i) Understocking which led to calving on rank pasture. These pasture conditions are more suitable for larval survival than shorter pastures (Rose, 1956).

ii) Insufficient subdivision so that optimum

pasture management was more difficult to achieve.

iii) Set-stocking for four months from commencement of calving in mid November until weaning mid March. Combined with excess feed supply, this resulted in camping in certain areas of the paddock. Thus the effective stock density was high and correspondingly the rate of pasture lungworm larval contamination was undoubtedly high.

It is notable that while the mean FLC for weaners was 134 lpg (100% prevalence), the mean FLC from a sample of the dams was only 0.6 lpg (range 0.25-1.0, 70% prevalence), (Wilson, unpublished data). Thus while the dam and offspring were grazing the same pasture, with the dam consuming an estimated 2-3 times the herbage of the offspring, the adult appeared to resist establishment of significant lungworm burdens. It is probable that the lungworm burdens in the offspring developed by cycling through themselves, and that the burden in the dams appeared to influence this very little. However, it is not known whether burdens may have been higher in the adults at some earlier stage postnatally, but work of Wilson (pers. comm.) showed that this was unlikely since no postpartum rise was observed in another deer herd. It is interesting to note that these hinds were drenched with anthelmintic prior to calving. This would appear to have had little effect on the lungworm burdens in weaners under this management system.

The above findings have major implications for post-calving management of deer. On the basis of this data it appears desirable to calve hinds at a density which is compatible with pasture control, since mechanical harvesting of excess pasture is impossible during calving, and to shift hinds with calves at foot once calving has finished and calves are mobile. These shifts to "clean" pasture could take place late in January, and again mid-February, thereby avoiding the ever increasing larval contamination on pastures under the set-stocked regime, and avoiding the high burdens in the weaners which were found in this study.

Following the first anthelmintic treatment the mean faecal larval count fell dramatically 0-7 days post-treatment.

Twenty one days post-treatment there was a slight increase in the mean larval count from day 14, but the percentage reduction from the pre-treatment level was still 94%. Thus it appears for practical purposes that the efficacy of albendazole is satisfactory for control of lungworm in deer. The increase at 21 days post-treatment may have been due to:

a) An incomplete efficacy against the migrating stages of D. viviparus in or before they reach the lungs. Such larvae could develop to maturity and produce larvae within 21 days.

b) The maturation of larvae ingested immediately post-treatment. The pre-patent period for D. viviparus in red deer has been shown to be as short as 20 days (Corrigall et al., 1980).

c) Incomplete efficacy of the treatment against adults resulting in short-term suppression of egg laying only.

Several deductions from the changes in mean faecal larval counts that occurred when only one group was drenched can be made:

a) Infective D. viviparus larvae were still available to the weaner deer despite their larval output being minimal for the previous nine weeks. These larvae resulted either from survival on pasture of the weaner's pre-weaning larval output or the output (albeit small) of the adult stock on the property.

b) The weaner deer had not developed a complete resistance to infection by D. viviparus larvae by early June. The smaller increase in larval count seen on June 29 as opposed to that seen on June 7 may have been as a result of a lower larval challenge or an increase in the animal's resistance to infection, which would be expected to become apparent with age and previous exposure to the parasite.

3.5.2 Faecal Egg Counts.

Following the initial anthelmintic treatment there was a significant decline in the faecal gastrointestinal nematode parasite egg counts seven days post-treatment in both groups. This lower egg output was maintained for 21 days when the next treatment was given. Thus in this instance albendazole appeared effective against all stages of deer gastrointestinal parasites present.

After the first drench and during the period when both groups received two further three-weekly drenches there were no significant changes in faecal egg counts. Therefore the 21 day treatment programme caused continual suppression of faecal egg counts.

Subsequent to omission of the three-weekly treatment of one group, there was a rise in the faecal egg count 21 days later (42 days since the last treatment). This rise acheived statistical significance only in group 1. It is apparent that infective larvae were therefore still available on the pasture despite the low faecal egg output of the weaners. These larvae could have originated from several sources:

a) Contamination of the pasture by the weaners from their faecal egg output pre- and post-weaning.

b) Contamination of the pasture by faecal egg output of the adult stock on the property.

The effect of a continual gastrointestinal parasite challenge on the growth rates of deer cannot be calculated from this trial. However, Sykes (1979) reported liveweight losses of 36% in sheep exposed to infective Ostertagia larvae daily and given anthelmintic treatment every 16 days, compared with parasite-free control animals. There is a need for similar experiments to be conducted with deer and deer parasites so that their effect can be determined.

The efficacy of oral albendazole recorded in this trial is comparable to that recorded in Chapter 2.

3.5.3 Liveweights.

The mean weaning weights of the experimental animals (males 41.2 kg, females 34.6 kg) were higher that the weights reported by Drew et al., (1978) of 31.7 kg for males at March weaning. Asher et al., (1981) reported March weaning weights of 34.7 kg and 32.5 kg for males and females, respectively. However, the latter authors indicated that their weaning weights were depressed because of a high number of late calves. The calving dates in this study were not known, so the effect of birth date on liveweight could not be estimated. The better weaning weights reported here, compared with those of Drew et al., (1978) were possibly largely attributable to better pasture production during that season in the Manawatu, though a multitude of other factors including pasture quality, age of dam, genetic differences between stock, calving date, parasitism and other production limiting conditions may also have had an effect.

However, the weaning weights achieved in this experiment were lower than those on the Massey University Deer Unit (males 43.9 kg, females 41.6 kg on March 10) which were weaned five days earlier. Again the effects mentioned above could have had an effect but it is considered by the author that the management at Massey which resulted in superior pasture quality was the major factor. The higher pasture quality resulted in better hind nutrition and consequently improved lactation. Better calf nutrition from a greater milk intake and higher quality pasture intake when they started eating grass after one week of age probably resulted.

Better pasture production as a result of the more mild climate could also account for the higher daily post-weaning growth rate recorded in this experiment (61.9 g/day over 163 days), than by Drew et al., (1978) (46 g/day over 168 days). That there were no differences in growth rates following either treatment regime indicates that the increased larval and egg counts

presumably indicating increased worm numbers following omission of three-weekly drenches, had no measurable effect upon growth. This was not unexpected since the counts were all very low, and in that period, establishment of a pathogenic burden would be unlikely.

3.6 Conclusions.

Deer calves can develop a high lungworm faecal larval count even by weaning in mid-March, where deer are set-stocked from calving. The undesirability of such long-term set-stocking is a major finding of this study. It would appear that the burdens established in the weaners are a result of the cycle accelerating through that class of stock, and that the dam normally has little effect on that cycle.

A three-weekly drenching programme from mid-March adequately controls lung and gastrointestinal nematodes. The optimum time for continuation of this programme could not be established. Increased egg and larval counts detected 42 days after a drench were probably indicative of only minor increases in worm numbers since no depression of liveweight gains was evident.

Albendazole at 10 mg/kg appears adequate for parasite control in commercial deer herds.

CHAPTER 4

INVESTIGATION OF THE RELATIONSHIP BETWEEN FAECAL GASTROINTESTINAL NEMATODE EGG COUNTS AND GASTROINTESTINAL NEMATODE NUMBERS IN RED DEER.

4.1 Introduction.

Before faecal egg or larval counts can be used in the monitoring and diagnosis of endoparasitism the relationship between faecal egg counts and the corresponding parasitic burdens needs to be established.

McKenna (1982) reported a good correlation between individual egg counts and worm burdens in "young" sheep but a poorer one in "old" sheep. The relationship between worm burdens and faecal egg counts is less exact in cattle due to the suppression of parasite oogenesis when the cattle develop resistance to the parasite. However, some diagnostic interpretation can be made when faecal egg counts differ from the "normal" seasonal fluctuation (McKenna, 1982).

Information reported to date for deer is both incomplete and confusing. Presidente (1979b) reported that 1 epg, detected following the floatation of 1 g of faeces in concentrated $NaNO_3$, indicated the presence of 500 nematodes and suggested that anthelmintic treatment be instigated when 10 or more eggs per gram were observed. However, Wilson (1981) found little correlation between faecal egg counts and parasite burdens in a limited survey of red deer.

This study was undertaken to investigate the relationship between faecal egg counts and total worm burdens in farmed red deer.

Species of gastrointestinal parasites found in farmed deer in the southern North Island were identified concurrently.

4.2 Materials and Methods.

4.2.1 Specimens.

A random collection of 46 abomasal and intestinal tracts from farmed red deer slaughtered at a Deer Slaughter Premises (East Coast Venison Co-op, Hastings) were used for this study. All were from stags of at least one year of age and all had been held overnight without food before slaughter. The origins of the deer were not recorded.

4.2.2 Timing.

Specimens were collected as follows:

Date	No. Specimens	
20 June	4	
22 June	10	
30 June	10	
14 July	10	
17 August	10	
11 October	2	
	46	Total

4.2.3 Collection And Analytical Procedures.

Specimens were collected immediately after M.A.F. veterinary inspection. The omasal/abomasal junction, pyloris/duodenal junction and the distal rectum were ligated and the viscera refrigerated until processing within the following 30 hours in the laboratory.

Tracts were sectioned into abomasum, small intestine and large intestine. Faecal samples were collected from the distal rectum. The abomasum was opened along the greater curvature and the contents washed into a bucket. The mucosa was scraped under running water to recover any adherent material. Fluid and contents were diluted to 2 litres with tap water, and four separate

50 ml aliquots were combined and preserved by the addition of 20 ml of 40% formaldehyde.

The proximal 7 m of small intestine were dissected from the mesentery, opened and the contents collected. The mucosa was washed and scraped under running water to recover any remaining material. The contents were concentrated by sieving through a 250 um sieve and the material transfered to another container. They were then diluted to 2 litres with tap water and four 50 ml aliquots taken and preserved as above.

The caecum and cranial 1.5 m of the colon were separated from the remaining large intestine and contents collected as above. When the volume of contents and washings were more than 2 litres these were concentrated by sieving through a 1 mm sieve and were resuspended in 2 litres of water. Otherwise the contents were diluted to 2 litres with water. Aliquots were preserved as before.

The mucosa was examined for lesions.

Preserved samples were later washed by flushing with tap water through a 250 um mesh lid on a container until the fluid was clear and no longer smelled of formalin. The entire contents of the jar (10% of the contents of the organ sampled) were inspected under a dissecting microscope at 15X magnification on a petri dish with a grid etched into its base. Worms were extracted and individually placed on a microscope slide in a drop of water and examined under a binocular microscope. Where descriptions of the worms could be found in the literature they were identified to genus and species. If descriptions could not be found the nematodes were classified as accurately as possible. With the Ostertagia, Trichostrongylus and Cooperia species it was only possible to type the female parasites to genus level. If the sample contained more than 1000 worms only one tenth of the sample was examined (i.e. 1% of the organ contents).

Faecal samples were examined by the modified McMaster technique described previously (2.2.5).

4.3 Statistical Method.

Worm count data was tabulated to include site of origin, sex, maturity and species.

Regression analysis was carried out on the egg and total worm counts using the Minitab statistical package produced by the Pennsylvania State University.

4.4 Results.

Worm identification, numbers and egg counts are presented in table 4.1.

No mucosal lesions were observed.

Of the animals sampled, 39% had a detectable faecal egg count (mean count 117 epg) and 80% had a worm burden (mean count 1867).

Regression analysis carried out on the individual egg and worm count data showed the relationship to be:

Total worm burden = 18.8 (faecal egg count)- 341

$r^2 = 0.763$ ($P < 0.01$) (see figure 4.1).

Of the animals with a worm burden, 97% had abomasal worms (mean count 1849), 27% had small intestinal worms (mean count 6.5) and 46% had large intestinal worms (mean count 11.5). All infected animals had some mature worms present, 57% had immature worms which made up 5.4% of the total worms counted. The prevalence of male and female worms was approximately equal (100% and 97% of infected animals had male or female worms present, respectively) though the count for each sex differed (mean 646 males and 1147 females).

Mature nematodes of the tribe Ostertagiea were the most prevalent type found. Of the infected animals 84% had female Ostertagia-type worms with a mean count of 177. *Ostertagia leptospicularis* males (mean count 28.7) were the most prevalent species (51% of infected animals). *Spiculopteragia asymmetrica* males (mean count 29.8) were found in 35% of infected animals.

Table 4.1

Egg Count And Worm Data For All Samples.

Specimen Number	1	2	3	4	5	6	7	8	9	10	11	12	13
Egg Count	50	0	0	400	0	0	0	0	0	0	200	0	350
Tot. Worm Count	130	10	30	1060	30	50	30	100	50	90	480	190	490
Abomasal Worm Count	70	0	20	970	30	50	30	100	30	60	480	120	480
Small Intestinal Worm Count	20	10	0	0	0	0	0	0	20	0	0	60	0
Large Intestinal Worm Count	40	0	10	90	0	0	0	0	0	30	0	10	10
Tot. Mature Worms	130	10	30	1060	30	50	30	100	50	80	470	190	470
Tot. Immature Worms	0	0	0	0	0	0	0	0	0	10	10	0	20
Tot. Male Worms	10	10	20	380	10	20	10	30	10	50	120	40	180
Tot. Female Worms	120	0	10	680	20	30	20	70	40	40	330	150	290
Ostertagiea (female)	20	0	0	640	20	30	20	70	0	0	230	110	280
Spiculopteragia asymmetrica (male)	0	0	0	330	10	0	0	20	0	0	0	0	10
Spiculopteragia spiculoptera (male)	0	0	0	0	0	0	0	0	0	0	0	10	40
Ostertagia leptospicularis (male)	0	0	0	0	0	20	10	0	0	10	80	0	120
Skrjabinagia kolchida (male)	0	0	0	0	0	0	0	0	0	0	0	0	0
Skrjabinagia lyratiformis (male)	0	0	0	0	0	0	0	10	0	0	0	0	0
Trichostrongylus (female)	30	0	10	0	0	0	0	0	40	40	100	20	10
Trichostrongylus axei (male)	0	0	0	0	0	0	0	0	0	0	60	0	0
Trichostrongylus colubriformis (male)	0	10	10	0	0	0	0	0	10	0	0	10	0
Trichostrongylus vitrinus (male)	0	0	0	0	0	0	0	0	0	0	0	0	0
Cooperia (female)	0	0	0	0	0	0	0	0	0	0	0	20	0
Cooperia pectinata (male)	0	0	0	0	0	0	0	0	0	0	0	10	0
Oesophagostomum venulosum (female)	30	0	0	40	0	0	0	0	0	0	0	0	0
Oesophagostomum venulosum (male)	10	0	10	50	0	0	0	0	0	30	0	10	10
Trichuris (female)	0	0	0	0	0	0	0	0	0	0	0	0	0
Immature Ostertagiea	0	0	0	0	0	0	0	0	0	10	10	0	20
Immature *Trichostrongylus*	0	0	0	0	0	0	0	0	0	0	0	0	0

Contd.

Specimen Number	14	15	16	17	18	19	20	21	22	23	24	25
Egg Count	0	0	0	0	100	0	0	0	50	0	0	0
Tot. Worm Count	0	0	0	0	290	0	470	0	1280	0	0	290
Abomasal Worm Count	0	0	0	0	280	0	470	0	1210	0	0	290
Small Intestinal Worm Count	0	0	0	0	0	0	0	0	0	0	0	0
Large Intestinal Worm Count	0	0	0	0	10	0	0	0	70	0	0	0
Tot. Mature Worms	0	0	0	0	270	0	460	0	1180	0	0	290
Tot. Immature Worms	0	0	0	0	20	0	10	0	100	0	0	0
Tot. Male Worms	0	0	0	0	80	0	100	0	360	0	0	30
Tot. Female Worms	0	0	0	0	190	0	360	0	820	0	0	260
Ostertagiea (female)	0	0	0	0	180	0	350	0	800	0	0	10
Spiculopteragia asymmetrica (male)	0	0	0	0	0	0	0	0	110	0	0	0
Spiculopteragia spiculoptera (male)	0	0	0	0	10	0	0	0	160	0	0	0
Ostertagia leptospicularis (male)	0	0	0	0	70	0	60	0	30	0	0	0
Skrjabinagia kolchida (male)	0	0	0	0	0	0	40	0	10	0	0	0
Skrjabinagia lyratiformis (male)	0	0	0	0	0	0	0	0	0	0	0	0
Trichostrongylus (female)	0	0	0	0	0	0	10	0	0	0	0	250
Trichostrongylus axei (male)	0	0	0	0	0	0	0	0	0	0	0	0
Trichostrongylus colubriformis (male)	0	0	0	0	0	0	0	0	0	0	0	0
Trichostrongylus vitrinus (male)	0	0	0	0	0	0	0	0	0	0	0	0
Cooperia (female)	0	0	0	0	0	0	0	0	0	0	0	0
Cooperia pectinata (male)	0	0	0	0	0	0	0	0	0	0	0	0
Oesophagostmum venulosum (female)	0	0	0	0	10	0	0	0	20	0	0	0
Oesophagostomum venulosum (male)	0	0	0	0	0	0	0	0	50	0	0	0
Trichuris (female)	0	0	0	0	0	0	0	0	0	0	0	0
Immature Ostertagiea	0	0	0	0	20	0	10	0	80	0	0	0
Immature Trichostrongylus	0	0	0	0	0	0	0	0	0	0	0	0

Contd.

Specimen Number	26	27	28	29	30	31	32	33	34	35
Egg Count	995	1100	0	0	1050	100	100	0	0	50
Tot. Worm Count	15140	18500	400	0	31110	240	160	400	1900	220
Abomasal Worm Count	15050	18500	400	0	31100	220	150	390	1900	210
Small Intestinal Worm Count	50	0	0	0	10	10	0	0	0	10
Large Intestinal Worm Count	40	0	0	0	0	10	10	0	0	0
Tot. Mature Worms	15140	18400	370	0	31110	220	140	340	1770	220
Tot. Immature Worms	0	100	30	0	0	20	20	60	130	0
Tot. Male Worms	6400	4700	70	0	13110	90	30	100	510	20
Tot. Female Worms	8740	13700	300	0	18000	130	110	240	1260	200
Ostertagiea (female)	540	100	70	0	200	70	100	180	320	190
Spiculopteragia asymmetrica (male)	0	0	0	0	0	20	0	0	20	10
Spiculopteragia spiculoptera (male)	0	0	30	0	0	10	0	10	60	0
Ostertagia leptospicularis (male)	210	0	0	0	200	10	20	50	100	10
Skrjabinagia kolchida (male)	0	0	0	0	0	10	10	20	30	0
Skrjabinagia lyratiformis (male)	0	0	0	0	0	0	0	0	0	0
Trichostrongylus (female)	8150	13600	230	0	17800	40	0	50	940	10
Trichostrongylus axei (male)	6170	4700	40	0	12900	40	0	20	300	0
Trichostrongylus colubriformis (male)	0	0	0	0	0	0	0	0	0	0
Trichostrongylus vitrinus (male)	0	0	0	0	0	0	0	0	0	0
Cooperia (female)	10	0	0	0	0	10	0	0	0	0
Cooperia pectinata (male)	20	0	0	0	10	0	0	0	0	0
Oesophagostomum venulosum (female)	30	0	0	0	0	10	10	10	0	0
Oesophagostomum venulosum (male)	0	0	0	0	0	0	0	0	0	0
Trichuris (female)	10	0	0	0	0	0	0	0	0	0
Immature Ostertagiea	0	100	30	0	0	20	20	60	110	0
Immature Trichostrongylus	0	0	0	0	0	0	0	0	20	0

Contd.

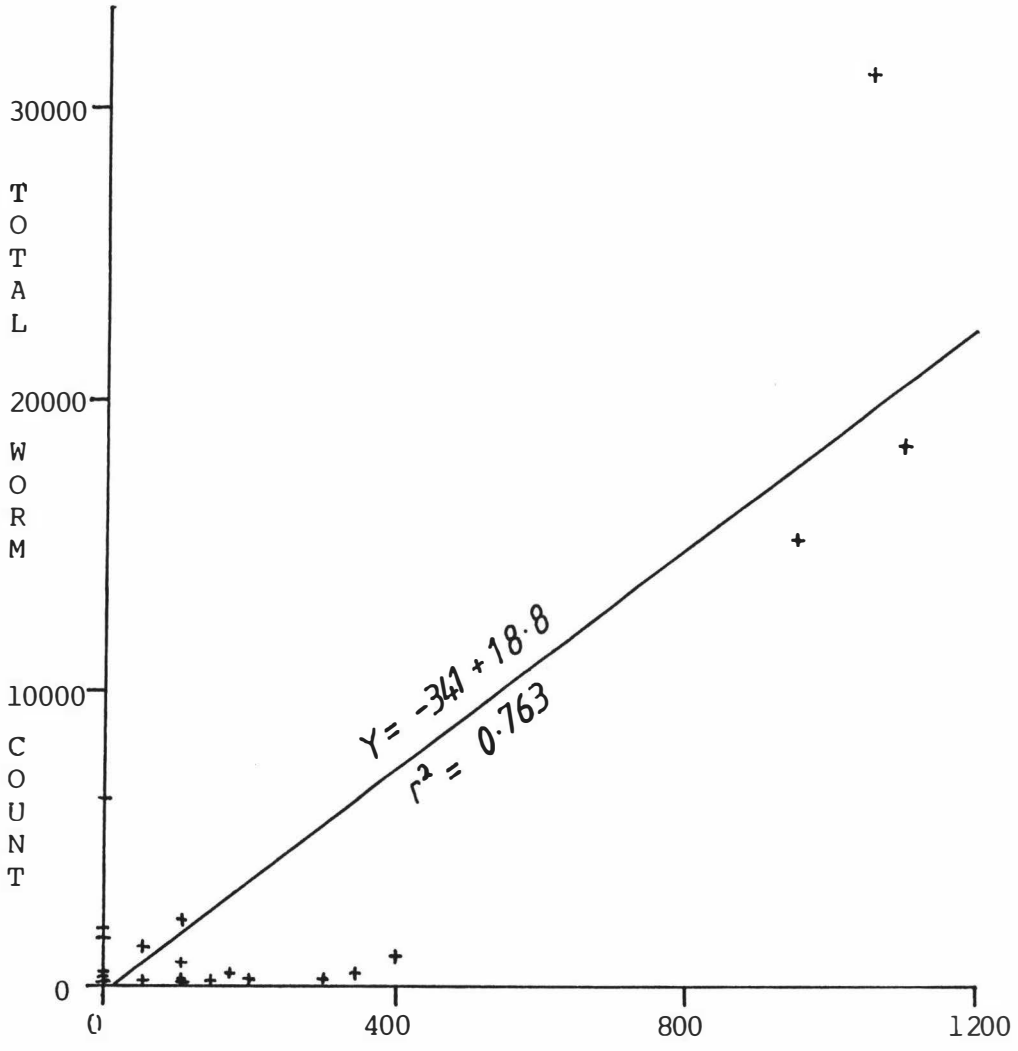
Specimen Number	36	37	38	39	40	41	42	43	44	45	46
Egg Count	100	50	0	0	300	0	0	0	100	200	150
Tot. Worm Count	2230	230	420	160	190	6310	1590	420	810	210	190
Abomasal Worm Count	2230	230	420	160	180	6310	1480	370	750	170	140
Small Intestinal Worm Count	0	0	0	0	0	0	90	0	20	0	0
Large Intestinal Worm Count	0	0	0	0	10	0	20	50	20	40	50
Tot. Mature Worms	2230	220	410	150	170	2480	1430	400	800	200	190
Tot. Immature Worms	0	10	10	10	20	3830	160	20	10	10	0
Tot. Male Worms	670	20	170	40	20	950	330	110	200	60	20
Tot. Female Worms	1560	160	240	110	150	1530	1100	290	580	140	170
Ostertagiea (female)	0	160	240	0	140	1520	1050	120	10	120	140
Spiculopteragia asymmetrica (male)	0	10	20	0	0	520	240	0	0	40	0
Spiculopteragia spiculoptera (male)	0	10	90	0	0	0	30	0	0	0	0
Ostertagia leptospicularis (male)	0	0	40	0	20	290	0	10	0	0	0
Skrjabinagia kolchida (male)	0	0	20	0	0	140	0	0	0	0	0
Skrjabinagia lyratiformis (male)	0	0	0	0	0	0	0	0	0	0	0
Trichostrongylus (female)	1560	0	0	110	0	10	30	150	540	0	0
Trichostrongylus axei (male)	670	0	0	40	0	0	0	70	190	0	0
Trichostrongylus colubriformis (male)	0	0	0	0	0	0	20	0	0	0	0
Trichostrongylus vitrinus (male)	0	0	0	0	0	0	20	0	0	0	0
Cooperia (female)	0	0	0	0	0	0	0	0	10	0	0
Cooperia pectinata (male)	0	0	0	0	0	0	10	0	10	0	0
Oesophagostomum venulosum (female)	0	0	0	0	10	0	20	20	20	20	30
Oesophagostomum venulosum (male)	0	0	0	0	0	0	0	30	0	20	20
Trichuris (female)	0	0	0	0	0	0	0	0	0	0	0
Immature Ostertagiea	0	10	10	0	20	3830	160	20	10	0	0
Immature Trichostrongylus	0	0	0	10	0	0	0	0	0	0	0

Contd.

	MEAN	S.D.	RANGE	PREVALENCE All deer sampled.	PREVALENCE Worm infested deer.
Egg Count	117	263	0-1100	39%	49%
Tot. Worm Count	1867	5647	0-31110	80%	100%
Abomasal Worm Count	1849	5645	0-31110	78%	97%
Small Intestinal Worm Count	6.5	17.5	0-90	22%	27%
Large Intestinal Worm Count	11.5	20.7	0-90	37%	46%
Tot. Mature Worms	1766	5604	0-31110	80%	100%
Tot. Immature Worms	101	563	0-3830	47%	57%
Tot. Male Worms	646	2209	0-13110	80%	100%
Tot. Female Worms	1147	3460	0-18000	78%	97%
Ostertagiea (female)	177	299	0-1520	67%	84%
Spiculopteragia asymmetrica (male)	29.8	95.7	0-520	28%	35%
Spiculopteragia spiculoptera (male)	10.0	28.5	0-160	24%	30%
Ostertagia leptospicularis (male)	28.7	61.7	0-290	41%	51%
Skrjabinagia kolchida (male)	5.9	21.9	0-140	17%	22%
Skrjabinagia lyratiformis (male)	0.22	1.47	0-10	2%	3%
Trichostrongylus (female)	951	3433	0-17800	50%	62%
Trichostrongylus axei (male)	548	2177	0-12900	28%	35%
Trichostrongylus colubriformis (male)	1.3	4	0-20	11%	14%
Trichostrongylus vitrinus (male)	0.43	2.95	0-20	2%	3%
Cooperia (female)	1.09	3.79	0-20	9%	11%
Cooperia pectinata (male)	1.3	4	0-20	11%	14%
Oesophagostomum venulosum (female)	6.1	10.6	0-40	30%	38%
Oesophagostomum venulosum (male)	5.2	12.2	0-50	22%	27%
Trichuris (female)	0.22	1.47	0-10	2%	3%
Immature Ostertagiea	98.9	563	0-3830	41%	51%
Immature Trichostrongylus	5.87	19.3	0-20	4%	5%

Figure 4.1.

Regression Of The Total Worm Count On The Faecal Egg Count.



Spiculopteragia spiculoptera males were recorded in 30% of infected animals (mean count 10.0). Skrjabinagia kolchida males (mean count 5.9) and Skrjabinagia lyratiformis males (mean count 0.22) were recorded in 22% and 3% of animals, respectively.

The Trichostrongylus were the second most prevalent worm type. Of the infected deer 62% had female Trichostrongylus worms and the mean count was 951. T. axei males (mean count 548) were recorded in 35% of infected deer. T. colubriformis males (mean count 1.3) were located in 14% of infected deer. T. vitrinus males were located only in one of the infected deer (3% prevalence) with a mean count of 0.43.

Oesophagostomum venulosum males were identified in 27% of infected deer (mean count 5.2). Oesophagostomum venulosum females were found in 38% of infected deer (mean count 6.1). Cooperia pectinata males were identified in 14% of infected animals (mean count 1.3) and Cooperia females in 11% (mean 1.09). One Trichuris female worm was identified resulting in a prevalence of 3% in infected deer (mean 0.22).

Of infected animals 51% had immature Ostertagia type worms (mean count 98.9) and 5% had immature Trichostrongylus worms (mean count 5.87).

4.5 Discussion.

The mean faecal egg count observed here (117/g) is similar to means of < 200 epg and 78.7 epg, respectively recorded from surveys of farmed deer faecal egg counts by Mason and Gladden, (1983); and Wilson, (1981) but is low when compared with those normally found in ewes (approximately 300 epg) (Brunsdon, 1976). However, the real mean count may have been lower because of faecal concentration which would have occurred as all animals had been held without food for 24 hours prior to slaughter. A 100% increase in the mean faecal egg count of sheep was reported by Brunsdon (1970b) following fasting for 12 to 24 hours.

Of the 80% of deer that had worm burdens only 39% had detectable egg counts. Thus only 49% of deer with worm counts had detectable faecal egg counts. This may have been due to several factors:

i) Counting technique. The faecal egg counting technique sensitivity is 50 epg. It is highly probable that many deer with parasite burdens were in fact passing eggs in their faeces but the technique was not sensitive enough for their detection in one sample.

ii) Parasite maturity. Immature parasites do not produce eggs. However, the data showed that 47% of deer with zero egg counts had a burden of 100% mature worms. On average 93% (range 39-100%) of worms in animals with zero counts were mature. In fact only one of 19 deer in this category had a proportion of mature worms less than 85% of the total count.

iii) Sex of parasite. Male parasites do not excrete eggs. Of deer with zero egg counts but gut worm counts, all but one had mature female worms present- i.e. only one deer had only male parasites.

iv) Intermittent shedding of eggs. It is possible that parasites lay eggs only intermittently. It can be seen from the data in tables 2.6-2.8 that the faecal egg counts of individuals fluctuates widely. However, it is difficult to understand a mechanism that would stop shedding in all of 2500 parasites as present in many deer, simultaneously.

v) Distribution of eggs in faeces. Parasite eggs are not evenly distributed through faeces. With low egg outputs this will lead to false negative egg counts.

None of these factors alone could fully explain the discrepancy observed though the sampling error inherent in the egg counting technique used would be of major importance.

There was a large individual variation in worm burden associated with a given faecal egg count (see figure 4.1). This problem can be reduced in the diagnosis of parasitism in deer by taking the mean of several samples. Brunsdon (1970b) found that the mean

egg count of 10 faecal samples was proportional to the true mean worm burden in sheep.

The worm burdens in this study were generally low, although four animals had counts of more than 4,000, and three of these had counts of more than 10,000. Thus burdens in 42 of 46 samples were in the "low" range when compared with "young" sheep parasite burdens, (McKenna, 1982). Only one was in the "moderate" and three in the "high" range. Presidente's criterion for instigating anthelmintic treatment in deer was 2000 abomasal worms (Presidente, 1979b). Only five of the animals in this study had burdens above this threshold. No signs of clinical parasitism or mucosal lesions were observed in the deer.

Of the nematodes retrieved in this study, 99% were from the abomasum. This is similar to the findings of Presidente (1979a) and Daft (1982). However, this does not mean that small and large intestinal parasitism is inconsequential in deer as the pathogenicity of the various parasite species in deer may differ. Deaths in white-tailed deer due to small intestinal parasitism (S. papillosus) have been recorded (Forrester et al., 1974).

The predominance of female parasites observed in this study is similar to that reported in farmed fallow deer (Presidente, 1979a), but Daft (1982) reported a higher prevalence of males in farmed and wild red deer in Britain. The reason for these differences is unknown.

Trichostrongylus counts constituted 80% of the total worm count in this study but these parasites were present in only 65% of infected animals. Of the five animals with worm counts greater than 2000, the majority of the burden in four of the five was made up of the genus Trichostrongylus. Of the total number of nematodes of the Trichostrongylus species recovered, 99.5% were recovered from tracts collected on July 14 and August 17. No record of the property of origin could be kept either within or between sampling days so it could not be determined if these deer were from the same farm. It may be that there

are seasonal fluctuations in the composition of worm burdens of deer, though samples were collected over too short a time span to be certain. Seasonal changes in the composition of worm burdens of lambs up to one year old have been recorded by Brunsdon, (1970a).

The large numbers of Trichostrongylus species (T. axei, T. colubriformis, T. vitrinus) recovered is in contrast to other surveys of deer parasites. Presidente (1979a) observed burdens of less than 100 Trichostrongylus species in fallow deer and hog deer in Australia. In a survey of red deer in Britain, Dunn (1983) found that more than 95% of the worm burdens were of the subfamily Ostertagiinae.

Notable by their low prevalence and numbers in this survey were the common nematode parasite species of sheep and cattle, other than Trichostrongylus species. Only low numbers of Cooperia, Oesophagostomum and Trichuris were recorded. As most farms would have been grazed by sheep and/or cattle prior to becoming deer farms or presently, the deer on the properties would most likely have been exposed to infective larvae of these species. As these parasite species are present only in low numbers it would seem that they may be less well adapted to red deer than are the "deer specific" species such as Ostertagia leptospicularis, Spiculopteragia spiculoptera, Spiculopteragia asymmetrica, Skrjabinagia kolchida, and Skrjabinagia lyratiformis. However, it is clear that red deer are not completely resistant to these parasites since they have been recorded elsewhere in New Zealand (see table 1.1).

The low number of counts of more than 2000 worms made the analysis of the relationship between the faecal egg count and total worm burden difficult. The system of grouping the egg and worm counts into ranges as is used for sheep (McKenna, 1982) could not be used here because of the low number of counts in all but the lowest range. A simple linear regression was used and this gave a statistically acceptable result ($r^2 = 0.763$). However, this result is dominated by the three highest

egg and worm counts. If these points are removed there is no relationship between the egg and worm counts (r^2 = 0.00). Care must be taken when interpreting this data. The following must be considered:

i) The linear regression equation arrived at by using all the data may indeed be a true indicator of the relationship between faecal egg counts and gastrointestinal nematode parasite burdens.

ii) A linear regression equation may be adequate for describing the relationship between faecal egg counts and gastrointestinal nematode burdens but data collected over a wider range of egg counts may produce a substantially different regression equation from that arrived at from this limited data.

iii) It is possible that the relationship between faecal egg counts and gastrointestinal nematode burdens is not a linear one. If this is the case then data from this study may indicate that a faecal egg count in the range 0-450 epg would equate with a burden of 0-3000 worms, and an egg count of 950 epg or more would equate to a worm burden of 15,000 worms or more.

More work involving a larger sample size and greater range of faecal egg and worm counts is required in order to determine the true nature of the relationship. Data from clinical and subclinical worm burdens is required if the relationship is to have practical significance to the clinician in the diagnosis of gastrointestinal parasitism in deer. Data is also required from animals less than one year of age as this is the group that most frequently suffers from parasitism. In sheep and cattle differences in the relationship between faecal egg counts and worm burdens have been observed between "young" and "mature" animals (McKenna, 1982), and this may also be the case for deer.

4.6 Conclusions.

The majority of the gastrointestinal nematode parasites of deer found in this study were abomasal. With the exception of Trichostrongylus species, the most prevalent and numerous species of parasite present were "deer-specific".

Gastrointestinal nematode faecal egg counts tend to increase with increasing worm burdens. However, more data is needed before a relationship can be firmly established since only a small number of high counts were found in this study. If the relationship between faecal egg counts and worm burdens is to have practical significance for the practising veterinarian, data is required particularly from animals with large parasite burdens.

The large variation in individual egg and worm counts emphasises the need for an adequate number of samples when interpreting faecal egg count data in the diagnosis of gastrointestinal parasitism on farms.

CHAPTER 5

INVESTIGATION OF A POUR-ON LEVAMISOLE FORMULATION IN DEER.

5.1 Introduction.

Pour-on anthelmintics have been used in New Zealand by deer farmers because of the convenience of application to an animal which often is difficult to drench orally. However, the apparent poor effectiveness of oral levamisole in deer (Presidente et al., 1973; McAllum, 1976), particularly against D. viviparus, casts doubt on the usefulness of a levemisole pour-on preparation in this species.

This study was designed to investigate "Riporon", Smith Kline and French, N.Z. Ltd., a 20% levamisole-base cutaneous application anthelmintic as a treatment against lungworm and gastrointestinal nematodes in red deer.

5.2 Materials and Methods.

5.2.1 Deer.

Seventeen nine-month-old (nine female, eight male) and five 20-month-old male red deer from a small commercial deer herd were used.

5.2.2 Timing.

All deer were faecal sampled on September 5. Treatment was carried out on September 7 and sampling continued for 21 days after treatment.

5.2.3 Experimental Groups.

On the basis of faecal samples collected on September 5 the deer were systematically assigned to one of two groups approximately balanced by sex, mean larval count, mean egg count and age. The groups were then randomly assigned for anthelmintic treatment

(Group 1) or control (Group 2; no treatment).

Group 1 was treated on September 7 with a commercial batch of "Riporon", at the recommended cattle dose rate calculated on a liveweight basis. The nine-month-old deer received 2.5 mls while the 20-month-old deer received 5mls. The animals were held in groups of four to six in a small darkened pen whilst the anthelmintic was applied in a 20 cm line down the thoracic and lumbar spine using a commercial calibrated drenching gun. Immediately after treatment the deer were released onto pasture.

On the day of treatment it was fine and sunny and approximately 12^{o}C. No rain fell until 20 hours post-treatment.

5.2.4 Management.

The nine-month-old and 20-month-old deer were set-stocked in adjacent paddocks and fed only pasture.

5.2.5 Sampling Schedule.

Faecal samples were collected per rectum two days prior to treatment and 5, 12 and 21 days after treatment.

Faecal egg and larval counts were determined by the method described earlier (see section 2.2.5).

Treated animals were observed for adverse reactions for approximately 30 minutes after treatement and visually inspected at each sampling for evidence of cutaneous reactions.

5.3 Statistical Analysis.

Data was log-transformed after addition of 1×10^{-5} to all samples. The Student's t test was performed on the transformed data at each sampling between the two groups.

5.4 Results.

No adverse reactions were observed following treatment.

5.4.1 Faecal Larval Counts.

Individual faecal larval counts are presented in table 5.1. Means are presented in figure 5.1.

Prior to treatment 86% of samples had positive faecal larval counts. These ranged from 0.25 lpg to 3.75 lpg. Five days post-treatment the prevalence of positive samples in the treated group had increased from 90% to 100% and the mean from 1.58 lpg to 4.3 lpg (range 0.79-9.25 lpg). For the remainder of the samplings the prevalence of positive samples in the treated group was 100%, and the mean counts rose to 5.4 lpg and 5.6 lpg at 12 and 21 days post-treatment, respectively.

Five days post-treatment in the control group the prevalence was unchanged at 82% but the mean had risen from 1.4 lpg to 3.8 lpg (range 0-9 lpg). The mean and prevalence increased to 4.6 lpg and 91%, respectively, 12 days post-treatment but fell slightly 21 days post-treatment to be 4.25 lpg and 90%, respectively.

No significant differences were detected between the groups.

5.4.2 Faecal Egg Counts.

Individual faecal egg counts are presented in table 5.2. Means are presented in figure 5.2.

Prior to treatment, 24% of animals had detectable faecal egg counts. These ranged from 50 epg to 100 epg. Five days post-treatment the prevalence had decreased from 20% to zero in the treated group. The prevalence rose to 38% and 22%, 12 and 21 days post-treatment, respectively, and the mean count rose to 25 epg (range 0-100) and 11 epg (range 0-50) at the same times.

Five days post-treatment in the control group the

Table 5.1

Individual D. viviparus Faecal Larval Counts (lpg) And Means Before And After Treatment With "Riporon", And In Untreated Controls.

GROUP 1. (Treated)

Number	Sex	Age (months)	Days From Treatment -2	5	12	21
0	F	9	1.75	1.25	3.5	8.25
W4	M	9	1.0	*	1.75	3.25
W7	F	9	3.25	*	6.25	5.0
W9	M	9	0	*	*	1.25
W10	F	9	0.75	0.75	5.25	1.75
W12	M	9	2.5	9.25	17.75	7.5
W17	F	9	1.0	3.5	2.0	12.25
W20	M	9	3.75	5.0	1.5	*
O19	M	20	0.25	7.5	9.0	8.25
O26	M	20	1.5	3.0	1.5	2.75
Mean			1.58	4.3	5.4	5.6
Range			0-3.75	0.75-9.25	1.5-17.75	1.25-12.25
Prevalence%			90%	100%	100%	100%

GROUP 2. (Control)

Number	Sex	Age (months)	Days From Treatment -2	5	12	21
W5	F	9	0.75	3.0	2.5	5.25
W11	F	9	3.0	6.5	6.75	5.5
W13	M	9	0.5	4.0	4.0	*
W14	F	9	1.5	2.75	3.5	3.75
W15	F	9	0	0	0	0
W16	M	9	3.0	9.0	12.5	14.25
W18	M	9	3.25	3.75	6.5	2.25
W19	F	9	0.25	2.25	1.25	1.5
O20	M	20	1.0	0	1.5	0.75
O21	M	20	2.25	2.75	4.25	0.75
O22	M	20	0	0.25	3.25	4.25
Mean			1.4	3.1	4.2	3.8
Range			0-3.25	0-9.0	0-12.5	0-14.25
Prevalence%			82%	82%	91%	90%

Figure 5.1.

Mean Lungworm Faecal Larval Counts Of Treatment And Control Groups.

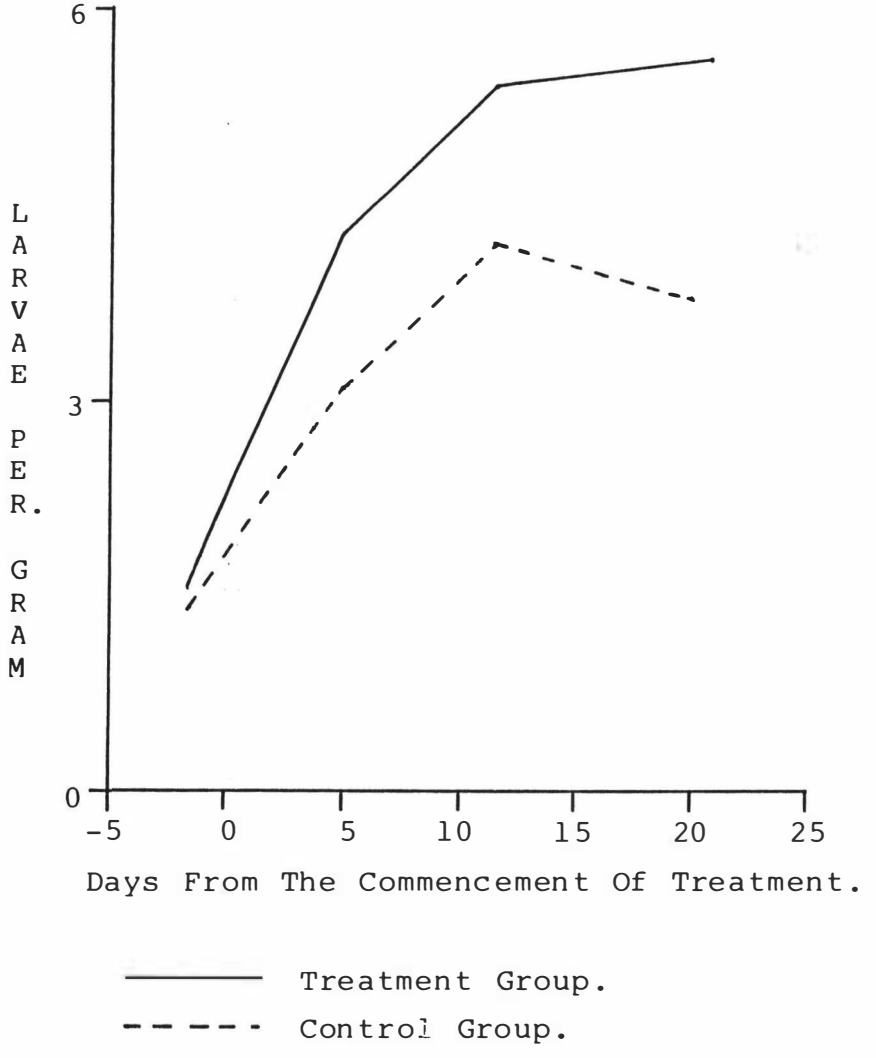


Table 5.2

Individual Gastrointestinal Nematode Faecal Egg Counts (epg) And Means Before And After Treatment With "Riporon", And In Untreated Controls.

GROUP 1. (Treated)

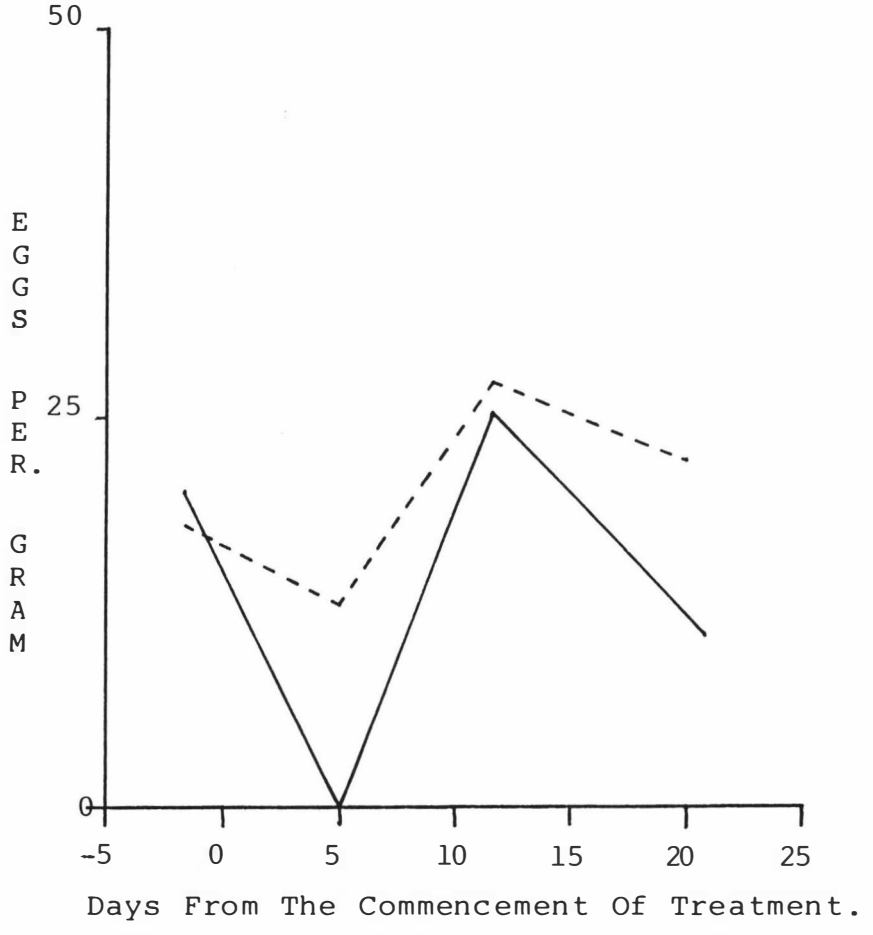
Number	Sex	Age (months)	Days From Treatment -2	5	12	21
0	F	9	0	0	*	0
W4	M	9	0	*	0	0
W7	F	9	100	*	0	0
W9	M	9	0	*	*	0
W10	F	9	0	0	50	0
W12	M	9	0	0	0	50
W17	F	9	0	0	0	50
W20	M	9	100	0	100	*
O19	M	20	0	0	50	0
O26	M	20	0	0	0	0
Mean			20	0	25	11
Range			0-100	0	0-100	0-50
Prevalence%			20%	0%	38%	22%

GROUP 2. (Control)

Number	Sex	Age (months)	Days From Treatment -2	5	12	21
W5	F	9	0	0	0	0
W11	F	9	0	0	50	0
W13	M	9	50	0	0	*
W14	F	9	0	100	0	0
W15	F	9	0	0	50	*
W16	M	9	50	0	50	100
W18	M	9	0	0	0	50
W19	F	9	100	0	150	0
O20	M	20	0	0	0	0
O21	M	20	0	0	0	50
O22	M	20	0	50	0	0
Mean			18	13.6	27	22
Range			0-100	0-100	0-150	0-100
Prevalence%			27%	18%	36%	33%

Figure 5.2

Mean Gastrointestinal Faecal Egg Counts Of Treatment And Control Groups.



egg count prevalence fell from 27% to 18%, and the mean fell to 13.6 epg from 18 epg (range 0-100 epg). The mean and prevalence were 27 epg and 36%, respectively, 12 days post-treatment but both fell slightly 21 days post-treatment to be 22 epg and 33%, respectively.

No significant differences were detected between the groups.

5.5 Discussion.

"Riporon" treatment had no effect on the *D. viviparus* faecal larval or gastrointestinal nematode faecal egg counts when compared with untreated control deer. This apparent ineffectiveness of cutaneously applied levamisole in deer is consistent with reported ineffectiveness of oral levamisole in deer (McAllum, 1976). Levamisole at 7 mg/kg given orally did not cause any reduction in *D. viviparus* larval counts in black-tailed deer (Presidente *et al.*, 1973). However, when given at 16 mg/kg a zero larval count resulted after six days but the counts had returned to the pre-treatment levels within 13 days of treatment. This reduction in larval count was considered to be a temporary reduction in production by the adult lungworm (Presidente *et al.*, 1973).

McAllum (1976) reported deaths attributed to lungworm of two 10-month-old red deer calves after receiving three oral treatments of levamisole hydrochloride at 13 mg/kg.

Mason (1982) measured the serum levamisole concentration achieved in red deer after oral and injectable administration. Deer were treated orally with either the recommended cattle dose rate (7.5 mg/kg) or 1.5 times this dose. The peak serum concentration of levamisole occurred within 16 minutes of administration (compared with 1-2 hours in cattle) and then decreased rapidly. Little difference in the serum concentration was found after either route of administration. It was proposed that as levamisole acts by paralysing the parasites the period of effect may not be long enough

for the worms to be eliminated from the site of infection (Mason, 1982). This is probably the reason for the apparent ineffectiveness of levamisole at these dose rates in deer, and would explain the apparent improved efficacy seen by Presidente et al., (1973) when a higher dose rate was used, since that would have resulted in a longer elevation of blood levamisole concentrations.

In cattle, levamisole is reported to have a 98% efficacy against D. viviparus and 61-100% efficacy against mature gastrointestinal nematodes when given at 8 mg/kg orally (Lyons et al., 1972). Hart et al., (1969) reported a 54-100% efficacy of oral levamisole (5-10 mg/kg) against gastrointestinal nematode species in sheep. These authors also report an 83-100% efficacy against gastrointestinal nematodes in cattle when given subcutaneously at 5 mg/kg, and 84-100% efficacy when given orally at 7.5 mg/kg. An 88% and 100% efficacy was reported against D. viviparus in cattle at the two dose rates, respectively.

Other factors which may be involved in the poor response of the cutaneous application of levamisole are species and seasonal variations in skin penetration by the drug. Deer skin may be less pervious to the drug than cattle skin. Forsyth, Gibbon and Pryor (1983) reported a five-fold greater increase in serum concentrations acheived in the summer than in winter in cattle. This increased concentration led to an improved efficacy against D. viviparus in the summer (90.7% v's 50.3% in winter). Taylor, Mc'Ewan and Burke (1983) reported a 60-70% greater bioavailability of levamisole in summer than in winter following cutaneous application of a levamisole preparation on bullocks. Both the above authors considered that seasonal changes in skin structure may be the cause of the differences observed. However, it would appear more likely that the failure of "Riporon" to reduce parasite faecal larval and egg counts in deer was due to rapid metabolism of the drug in this species.

5.6 Conclusion.

Cutaneous application of "Riporon" (20% W/V levamisole) at the recommended cattle dose rate appears to be ineffective in reducing gastrointestinal nematode egg and *D. viviparus* larval counts in red deer. However, the results of this trial must be viewed in the light of the low initial faecal egg and larval counts.

BIBLIOGRAPHY

Asher, G., Adam, J.L. and Langridge (1981). The Mid-Northern Regions - reproduction and growth 1980/81. The Deer Farmer. Spring: 4-8.

Andrews, J.R.H. (1964). The arthropod and helminth parasites of red deer in New Zealand. Trans. R. Soc. N.Z. 5: 97-121.

Andrews, J.R.H. (1973). A host-parasite checklist of helminths of wild ruminants in New Zealand. N.Z. Vet. J. 21: 43-47.

Anon. (1978). Lungworm in deer. Surveillance. No. 3. Autumn. p.21.

Anon. (1979a). Parasites of red deer. Surveillance. Vol. 6. No. 1 Autumn. p.3.

Anon. (1979b). Parasitic pneumonia in deer. Surveillance. Vol. 6. No. 3 Autumn. p.20.

Anon. (1980). Deer farming and deer health. Surveillance. Vol. 7. No. 2 p.11-12.

Bacinsky, A. (1969). Sezonna dynamika Dictyocaulus viviparus (Dictyocaulus eckerti) u jelena (Cervus elephus) na Slovensku. Folia. Vet. 13: 105-110.

Blaxter, J.T. and Allan, D. (1977). Persistence of Dictyocaulus viviparus larvae on pasture. Vet. Rec. 101: 394.

Blaxter, K.L., Kay, R.N.B., Sherman, G.A.M., Cunningham, J.M.M. and Hamilton, W.J. Farming the red deer: The first report of investigations by the Rowett Institute and Hill Farming Research Organisation. Publ. Dept. Agric. Fish. Scotland. 1974.

Blaxter, K.L. and Hamilton, W.J. (1980). Reproduction in farmed red deer 2. Calf growth and mortality. J. Agric. Sc. 95: 275-284.

Blocker, W. and Segebade, R. (1977). Testing the efficacy of "Panacur" (Fenbendazole) against gastrointestinal nematodes of fallow deer. Tierarztl. Umsch. 32: 473-474, 476-478.

Brunsdon, R.V. (1970a). Seasonal changes in the level and composition of nematode worm burdens in young sheep. N.Z. J. Agric. Res. 13: 126-148.

Brunsdon, R.V. (1970b). Within flock variations in Strongyle worm infections in sheep: The need for adequate diagnostic samples. N.Z. Vet. J. 18: 185-188.

Brunsdon, R.V. (1976). Responses to the anthelmintic treatment of lambs at weaning: the relative importance of various sources of contamination in Trichostrongyle infections. N.Z. J. Exp. Agric. 4: 275-279.

Charleston, W.A.G. (1980). Lungworm and lice of red deer and fallow deer: A review. N.Z. Vet. J. 28: 150-152.

Charleston, W.A.G. An introduction to gastrointestinal nematode parasites of sheep and cattle in New Zealand. In Control of internal parasites in sheep. Ed. Ross, A.D. : Animal Industries Workshop, Lincoln College. 1982.

Corrigall, W., Easton, J.F. and Hamilton, W.J. (1980). Dictyocaulus infections in farmed red deer. Vet. Rec. 106: 335-339.

Daft, S.M.H. (1982). Red deer endoparasites. Vet. Rec. 21: 493.

Davidson, W.R., McGee, M.B., Nettles, V.F. and Chappell, L.C. (1980). Haemonchosis in white-tailed deer in the Southern United States. J. Wildl. Dis. 16: 499-508.

Drew, K.R., Fennessy, P.F. and Greer, G.J. (1978). The growth and carcass characteristics of entire and castrate red stags. Proc. N.Z. Soc. Anim. Prod. 38: 142-144.

Duncan, J.L., Armour, J., Bairden, K., Urquhart, G.M. and Jorgensen, R.J. (1979). Studies in the epidemiology of bovine parasitic bronchitis. Vet. Rec. 104: 274-278.

Dunn, A.M. (1983). Winter deaths in red deer: A preliminary report on abomasal parasite burdens. Publ. Vet. Deer Soc. 1: 17-25.

Eva, J.H. and Kellogg, F.E. (1977). Management implications of abomasal parasites in South Eastern white-tailed deer. J. Wildl. Manage. 41: 169-177.

Fennessy, P.F., Moore, G.H. and Muir, P.D. (1981). Artificial rearing of red deer calves. N.Z. J. Exp. Agric. 9: 17-21.

Foreyt, W. and Trainer, D.O. (1970). Experimental haemonchosis in white-tailed deer. J. Wildl. Dis. 6: 35-42.

Foreyt, W.J. and Drawe, D.L. (1978). Anthelmintic activity of albendazole in white-tailed deer. Am. J. Vet. Res. 39: 1901-1903.

Ford, G.E. Blood pepsinogen estimations and production responses in Trichostrongylid parasitism of ruminants. p.83-97. In Pathophysiology of parasite infections. Ed. Soulsby, E.J.L. 1976.

Forrester, D.J., Taylor, W.J. and Nair, K.P.C. (1974). Strongyloidosis in captive white-tailed deer. J. Wildl. Dis. 10: 11-17.

Forsyth, B.A., Gibbon, A.J. and Pryor, D.E. (1983). Seasonal variations in anthelmintic response by cattle to dermally applied levamisole. Austral. Vet. J. 60: 141-146.

Gibson, T.E. The ecology of the infected larvae of Trichostrongylus colubriformis. p.1-13. In Biology of parasites. Ed. Soulsby, E.J.L. 1965.

Gladden, N. (1981). National deer farm survey - 1980. Proc. N.Z.V.A. Deer Semin. Vet. p.34-38.

Guinness, F.E., Albon, S.D. and Clutton-Brock, T.H. (1978). Factors affecting reproduction in red deer hinds on Rhum. J. Reprod. Fert. 54: 325-334.

Gupta, R.P. and Gibbs, H.C. (1971). Infectivity of D. viviparus (Moose strain) to calves. Can. Vet. J. 12: 56.

Henrickson, S.A. (1965). En forbedret teknik ved undersogelse for lungeormelarver i faeces. Nord. Vet. Med. 17: 446-454.

Hart, J.A., James, P.S. and Curr, C. (1969). The anthelmintic efficacy of laevo-tetramisole hydrochloride against nematode parasites of sheep and cattle. Austral. Vet. J. 45: 73-77.

Johnstone, I.L. (1979). The intergration of beef cattle with sheep in parasite control. Proc. 9th Semin. Sheep & Beef cattle Soc. N.Z.V.A. p.19-30.

Johnston, J.T. (1982). The effect of O. circumcincta, O. ostertagi and H. contortus on metabolism in weaner red deer stags. Dissertation in partial fulfilment of B.Ag.Sc. (Hon). Lincoln College.

Jorgensen, R.J. Studies on the lungworm Dictyocaulus viviparus (Bloch, 1782) and its epidemiology in young cattle with a description of an attempt to prevent parasitic bronchitis. Thesis, Dr. Med. Vet., Royal Vet. and Agric. Univ. Copenhagen, 1981. 77p.

Kalivoda, J. and Chroust, K. (1971). Anthelmintic effectiveness of thibenzole administered in pelletized feeds to fallow deer and mouflons. Acta. Vet. Brno. 40: 453-462.

Kummereje, K. (1977). Dictyocaulus viviparus infestations in reindeer in Northern Norway. A contribution to its epidemiology. Acta. Vet. Scand. 18: 86-90.

Loudon, A.S.I., McNeilly, A.S. and Milne, J.A. (1983). Nutrition and lactational control of fertility in red deer. Nat. 302: 145-147.

Lyons, E.T., Drudge, J.H., Labore, D.E. and Tolliver, S.C. (1972). Field and controlled test evaluations of levamisole against natural infections of gastrointestinal nematodes and lungworms in calves. Amer. J. Vet. Res. 33: 65-71.

Mackintosh, C.G. and Mason, P.C. (1983). Controlled anthelmintic trial of ivermectin and febantel in deer. A poster. Int. Conf. Biol. Deer Prod. Dunedin.

Mason, P.C. (1977). Gastrointestinal parasitism in red deer. N.Z. Agric. Sci. 11: 182-183.

Mason, P.C. (1979a). Lungworm in red deer: Biology, symptoms and control. Aglink. FPP248.

Mason, P.C. (1979b). Lungworm in farmed deer. Proc. N.Z. Soc. Parasitol. 6: 654.

Mason, P.C. (1981a). Parasites of deer in New Zealand. Proc. N.Z.V.A. Deer Semin. Vet. p.63-70.

Mason, P.C. (1981b). Deer parasite trials. Proc. N.Z.V.A. Deer Semin. Vet. p.78-84.

Mason, P.C. (1982). Anthelmintics in deer: Serum levamisole levels in deer following treatment with "Nilverm". Surveillance Vol. 9. No. 3 Winter. p.11.

Mason, P.C. and Gladden, N. (1983). Survey investigates drenching practice and internal parasitism on deer farms. N.Z. Vet. J. 31: 217-220.

McAllum, H.J.F. (1976). Animal health and deer farming. In Deer farming in New Zealand: Progress and prospects. N.Z. Soc. Anim. Prod. Occ. Publ. No. 5 p.15-19.

McAllum, H.J.F. (1977). Some veterinary techniques used in deer farming. N.Z. Vet. J. 25: 130-131.

McAllum, H.J.F. (1979). Parasitic diseases. Proc. 1979 Anim. Health Div. Deer Farm. Course. p.75-87.

McAllum, H.J.F. (1981). Deer: Common diseases in farmed deer. Aglink. FPP254.

McDiarmid, A. (1975). Some disorders of wild deer in the United Kingdom. Vet. Rec. 97: 6.

McKenna, P.B. (1973). The significance of inhibition in the parasitic development of abomasal nematodes in New Zealand. N.Z. Vet. J. 21: 98-102.

McKenna, P.B. (1982). Diagnosis of Gastro-intestinal parasitism in young sheep flocks: The contribution of animal health laboratories. Proc. 12th Semin. Sheep & Beef cattle Soc. N.Z.V.A. p.108-129.

Moore, G.H. and Brown, C.G. (1977). Growth performance in farmed red deer. N.Z. Agric. Sci. 11: 175-178.

Milligan, K.E. (1984). Deer nutrition: Feed demands and how to meet them. Proc. N.Z.V.A. Deer Course Vet. p.46-58.

Nelson, M. (1977). Where do lungworm go in wintertime? Vet. Rec. 101: 248.

New Zealand Society of Animal Production. Internal parasites and animal production. Ed. Brunsdon, R.V. and Adam, J.L. Wellington 1975. 53p.

Oakley, G.A. (1977). Overwinter survival of Dictyocaulus viviparus. Vet. Rec. 101: 187.

Oakley, G.A. (1980). Speed of action of some anthelmintics against Dictyocaulus viviparus infections in cattle. Vet. Rec. 107: 530-531.

Presidente, P.J.A., Worley, D.E. and Catlin, J.E. (1972). Cross-transmission experiments with Dictyocaulus viviparus isolates from Rocky Mountain elk and cattle. J. Wildl. Dis. 8: 57.

Presidente, P.J.A., Knopp, S.E. and Deen, R.E. (1973). Treatment and control of Dictyocaulus viviparus in captive black-tailed deer. J. Wildl. Dis. 9: 34-40.

Presidente, P.J.A. (1979a). Endoparasites of captive and free-ranging deer in Australia. Sydney Univ. Postgrad. Comm. in Vet. Sci., Deer Ref. Course. p.235-252.

Presidente, P.J.A. (1979b). Parasitism in farmed deer: its importance and control. Symposium on Deer farming, Vic. Dept. Agric., Agric. Note Serv. No. 15.

Pritchard, R.K., Hennessy, D.R. and Steel, J.W. (1978). Prolonged administration: A new concept for increasing the spectrum and effectiveness of anthelmintics. Vet. Parasitol. 4: 309-315.

Rose, J.H. (1956). The bionomics of the free-living larvae of Dictyocaulus viviparus. J. Comp. Path. Ther. 66: 228-232.

Simpson, Anne M., Webster, A.J.F., Smith, J.S. and Simpson, C.A. (1978). Energy and nitrogen metabolism of red deer (cervus elaphus) in cold environments; A comparison with cattle and sheep. Comp. Biochem. Physiol. 60: 251-256.

Sturrock, R.F. (1965). The control of Trichostrongyle larvae (Nematoda) by fumigation in relation to their bionomics. Parasitol. 55: 29-44.

Sykes, A.R. (1979). Subclinical effects of parasitism. Proc. 9th Semin. Sheep & Beef cattle Soc. N.Z.V.A. p.31-39.

Taylor, D.C., McEwan, A.D. and Burke, W.M. (1983). Cutaneous application of levamisole to cattle: Variations in bioavailability related to season ambient temperature. Vet. Rec. 112: 481.

Vlassoff, A. (1973a). The biology of the free living phase of Trichostrongyle nematode parasites of sheep. Proc. 1st Ann. Meet.N.Z. Soc. Parasitol. p.31-35.

Vlassoff, A. (1973b). Seasonal incidence of infective Trichostrongyle larvae on pasture grazed by lambs. N.Z. Exp. Agric. 1: 293-301.

Vlassoff, A. (1976). Seasonal incidence of infective Trichostrongylus larvae on pasture: The contribution of the ewe and the role of the residual pasture infestation as sources of infection to the lambs. N.Z. J. Agric. 4: 281-284.

Vlassoff, A. Biology and population dynamics of the free living stages of gastrointestinal nematodes of sheep. p.11-20. In Control of Internal Parasites of Sheep. Ed. Ross, A.D. : Animal Industries Workshop, Lincoln College. 1982.

Watson, T.G., Manley, T. and Mason, P.C. (1983). Oxfendazole plasma levels in red deer and sheep following oral drenching. A poster. Int. Conf. Biol. Deer Prod. Dunedin.

Wilson, P.R. (1979a). Diseases relevant to deer farming. Sydney Univ. Postgrad. Comm. in Vet. Sci., Deer Ref. Course. p.105-125.

Wilson. P.R. (1979b). Nutrition and reproduction of farmed deer. Sydney Univ. Postgrad. Comm. in Vet. Sci., Deer Ref. Course. p.95-103.

Wilson, P.R. (1981). Studies in parasitism in red deer. Proc. N.Z.V.A. Deer Semin. Vet. p.71-77.

Wilson, P.R. and Collier, A.J. (1981). Lungworm in deer: A survey of veterinary practices. Proc. N.Z.V.A. Deer Semin. Vet. p.85-93.

Wilson, P.R. and Pauli, J.V. (1983). Blood constituents of farmed red deer (Cervus elaphus). II: Biochemical values. N.Z. Vet. J. 31: 1-3.