# Internal parasites and productivity in farmed deer

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## ABSTRACT

The aim of this brief review is to summarise current knowledge of the effects of internal parasites (lungworm and gastrointestinal nematodes) on productivity of farmed red deer. This review, largely based upon published data, highlights gaps in the literature and draws attention to topics for future research.

**Keywords:** Deer; lungworm; gastro-intestinal parasites; productivity.

## INTRODUCTION

For the purposes of this summary the productivity of deer under one year of age (weaner deer) is considered separately from animals over one year old. Liveweight gain is used to define the productivity of weaner deer, the production aim, to reach 92 kg liveweight (corresponding to a 50 kg carcass) by one year of age or earlier for venison. The effects of parasitism on the productivity of older animals are limited here to gain or loss of live weight or body condition. Data obtained from the farmed red deer literature in New Zealand (NZ) forms the basis of the information presented. Indeed, a literature search reveals scant information quantifying the effects of internal parasites on productivity of farmed or wild red deer and thus highlights the need for research in this area.

Parasitic helminths cause production losses in domestic animals worldwide (Coyne & Smith, 1994). Gastrointestinal (GI) nematodes in particular, have a major effect on the efficiency of production of grazing sheep and cattle (Sykes, 1994; Fox *et al*., 1989), largely through reduced efficiency of utilisation of protein (Poppi *et al*., 1986;, 1990; Bown *et al*., 1991; MacRae, 1993), but also metabolisable energy (MacRae *et al*., 1982) and reduced absorption and/or retention of minerals (especially phosphorus) (Coop & Kyriazakis, 2001). Infected animals may also exhibit a loss of appetite and behavioural changes.

Production losses due to sub-clinical, or unobserved disease, far outweigh the losses caused by apparent clinical disease and deaths. The magnitude of the effects of sub clinical parasitism on production are influenced by the size of the larval challenge and the number and species of worms present (Parkins & Holmes, 1989; van Houtert & Sykes, 1996).

Until recently, GI parasitism has been largely ignored in farmed deer in New Zealand (NZ). Unlike lungworm (*Dictyocaulus eckerti*), considered the most pathogenic endoparasite in farmed deer (Johnson *et al*., 2001) and the parasite which NZ farmers are most concerned about (Castillo-Alcala *et al*., 2007), mortality is rarely attributed to GI nematodes. Lungworm and GI parasites most often occur concurrently, although a higher proportion of deer of all ages shed lungworm larvae than shed GI nematode eggs in their faeces. Deer specific abomasal parasites of the *Ostertagiinae* sub-family and related *Ostertagia*-types are currently suggested to be the most pathogenic GI parasites of red deer. However, the relative pathogenicity of parasite infestations in different organs of the body by different parasite species has not been investigated in deer.

### Impact of internal parasitism on growth of weaner deer less than one year of age

All commercial deer herds in NZ probably have a level of sub-clinical parasitism resulting in a degree of liveweight gain depression in young deer. A 1% loss in growth rate due to lungworm and gastrointestinal nematodes was estimated to result in an overall annual reduction in farm gate venison sales of approximately NZ$2.8 million total in 2002 (Mackintosh & Wilson, 2003). However, there has been little research to quantify the effects of sub-clinical and clinical parasitism on deer growth.

Statistical analyses of survey data from 15 red deer farms in the lower North Island (Audige *et al*., 1998) showed a lower weaning weight associated with a reduced number of anthelmintic treatments prior to weaning and a higher faecal larval count index. This study also found that weaners grew faster in spring after a late winter anthelmintic treatment than if treatment had been withheld or given during spring.

A controlled indoor study using concurrent trickle infections of increasing doses of GI

nematode and lungworm infective larvae in weaner red deer showed that even small numbers of lungworm and GI nematodes can reduce both liveweight gain by 50% and voluntary feed intake (VFI) by 22% and that these effects increased with time over a 13 week infection period. These effects are shown in Figures 1 and 2 with the groups dosed 3x per week with 0 (control), 100 and 500 (low), 200 and 1000 (med) and 400 and 2000 (high) infective larvae of lungworm and mixed GI nematodes, respectively, cultured from deer faeces. This achieved mean group worm counts after 13 weeks of 0 (control), 30 and 412 (low), 31 and 605 (med) and 67 and 783 (high) lungworm and GI nematodes, respectively. Artificial infections of lungworm alone can cause significant reductions in cervine liveweight gain and VFI (Corrigall *et al*., 1982). However, concurrent infections of lungworm and GI nematodes may be more pathogenic than single-species infections. Also, there are limitations in interpretation of data from artificial infections in housed deer to the grazing situation with natural infections.

Initial grazing investigations at Massey University compared two groups of naturally parasitized weaner deer. The first group were treated when the parasite infection reached a certain (trigger) level the second group were drenched on a regular basis to suppress parasite infections. The deer were grazed together either on a perennial ryegrass-based pasture or chicory from March to November (Hoskin *et al*., 1999), Table 1. Considering only the weaner deer grazing pasture, this study found a 56% and 42% reduction in autumn voluntary feed intake and liveweight gain, respectively (both $P<0.01$), a 7% reduction in final spring liveweight (November, $P<0.05$) and 11% reduction in carcass weight ($P<0.01$). No significant differences were found between the groups grazed on chicory. The production differences found in this study were attributed to the occurrence of sub-clinical parasitism on pasture but not chicory (Hoskin *et al*., 1999). The effects were associated with low faecal egg and larval counts, but nematode counts were not conducted. Following pasture grazing, 56% of trigger-treated deer and 78% of suppressively treated deer reached the target liveweight of 92 kg live weight (50 kg carcass weight) by one year of age whereas 88% of both trigger- and suppressively-treated deer grazing chicory achieved this target.

A subsequent study, presented in Table 2, using individual animal trigger anthelmintic treatment compared with group suppressive treatment found a 55% reduction in weaner liveweight gain attributed to sub-clinical parasitism ($P<0.05$) from March until May whilst grazing perennial ryegrass-based pasture (Hoskin *et al*., 2003). In this latter study, trigger-treated deer euthanased during May had an average of approximately 2600 GI nematodes, of which most were from the abomasum, and 640 lungworm.

**Figure 1:** Mean (+SE) voluntary feed intake (kgDM/d) of artificially-reared deer infected with internal parasites at a low (□), medium (△), or high rate (○), and uninfected (◊). From Hoskin *et al*. (2000).

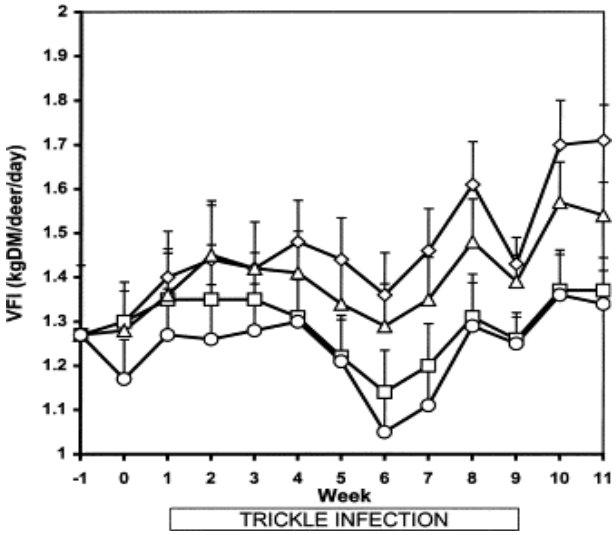


**Figure 2:** Mean (+SE) liveweight (kg) of artificially-reared deer infected with internal parasites at a low (□), medium (△), or high rate (○), and uninfected (◊). From Hoskin *et al*. (2000).

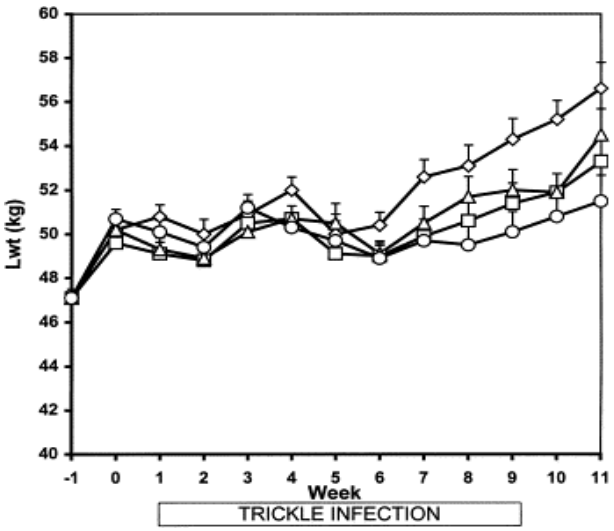

**Table 1:** Seasonal voluntary feed intake (VFI, organic matter/d), live weight (LW, kg), liveweight gain (LWG, g/d) and carcass weight (CW kg) of deer grazed on perennial ryegrass/white clover pasture or chicory and treated with anthelmintic 3-weekly or trigger-treated from Hoskin et al. (1999).

| | Pasture | | Chicory | | S.E. |
|---|---|---|---|---|---|
| | Treated | Trigger-treated | Treated | Trigger-treated | |
| Autumn VFI | 1920[a] | 835[b]♦ | 1015[c] | 1150[c]♦ | 127.3 |
| Autumn LWG | 217[a] | 125[b] | 184[a] | 212[a] | 8.7 |
| End autumn LW | 64[a] | 57[b] | 62[a] | 64[a] | 1.2 |
| End spring LW | 96[a] | 90[b] | 95[a] | 95[a] | 1.4 |
| November CW | 54.0[a] | 50.3[b] | 55.4[a] | 55.2[a] | 1.21 |

D.F. = 40 Experiment 1♦VFI was measured prior to anthelmintic treatment being given to these groups. [a,b,c]for rows designate differences between treatments (P<0.05 or better).

**Table 2:** Seasonal live weight (LW, kg), liveweight gain (LWG, g/d) and carcass weight (CW kg) of deer grazed on perennial ryegrass/white clover pasture or chicory and treated with anthelmintic 3-weekly or trigger-treated from Hoskin et al. (2003).

| | Pasture | | Chicory | | S.E. |
|---|---|---|---|---|---|
| | Treated | Trigger-treated | Treated | Trigger-treated | |
| End autumn LW | 62[a] | 57[6] | 66[c] | 64[c] | 0.8 |
| Autumn LWG | 134[a] | 60[b] | 208[c] | 175[c] | 11.8 |
| May CW | 31[a] | 30[a] | 37[b] | 37[b] | 2.3 |

DF 32 except CW DF = 8. [a,b,c]for rows designate differences between treatments (P<0.05 or better).

Young deer may have the potential for compensatory growth following a period of parasite-associated growth suppression. However, in the studies of Hoskin *et al*. (1999; 2003) post-autumn compensatory growth was only observed in some individuals and this was not enough to significantly affect the group mean live weight either at the end of winter or late spring.

**Impact of parasitism on productivity of deer older than one year of age**

Limited data has suggested that deer, at least infected with lungworm (Charleston, 1980; Corrigal *et al*., 1980), appear similar to sheep where high intensities of helminth infection occur in young, susceptible animals and a subsequent acquisition of immunity produces low levels of infection in older animals (Hudson & Dobson, 1995). Farmed weaner deer in NZ certainly shed less eggs and larvae in their faeces in spring compared to autumn, and weaners shed more eggs and larvae than deer greater than one year of age (Audige *et al*., 1998). A recent study of wild red deer in Scotland found some evidence for acquired immunity to lungworm but no evidence for acquired immunity to abomasal nematodes (Irvine *et al*., 2006) Despite low levels of abomasal nematode infection reducing indices of body condition in wild red hinds and stags greater than 2yo, there was no significant relationship between pregnancy rate and intensity of parasite infection (Irvine *et al*., 2006). However there is some evidence in wild reindeer (Stien *et al*., 2002) that reduced parasitism due to anthelmintic treatment increased body mass, body condition and pregnancy rates in adult hinds.

There appears to be nothing in the literature reporting effects of parasitism on productivity in adult farmed red deer. Audige *et al*. (1998) found whilst surveying lower North Island farms that adult hind faecal egg counts increased seasonally from March to September whilst a similar observation was made from adult stags for both faecal egg and larval counts from November to June. Therefore, although little is known about production losses in adult farmed deer attributable to parasitism, it could be suggested that anthelmintic treatment of adult deer may get the greatest response if given in winter (Audige *et al*., 1998). The survey of Castillo-Alcala *et al*. (2007) showed that 58 % of farmers anthelmintic-treated yearling hinds, 62% treated yearling stags, 56% adult hinds and 64% adult stags, with on average 1.9 and 1.6 treatments per year given to yearlings and 1.6 too adults regardless of sex.

In wild red deer, it has been suggested that even in summer when adult stags are in prime condition, relatively low abomasal worm populations can depress body condition, which in turn may influence stag performance during the rut (Irvine *et al.*, 2006). In addition, elevated testosterone associated with rutting could be immunosuppressive and may result in increased parasitism (Folstad & Karter, 1992) during and/or after the rut. The development of immunity in both farmed and wild deer should be a high priority for further investigation.

**Factors affecting internal parasitism (and the level of effect of internal parasitism on productivity) of farmed deer**

As in other farmed species, the production achieved in deer is likely to be a function of a number of variables. There are known interactions between stocking rate, nutrition, stress, internal parasitism and production. Animals at lower stocking rates or grazed extensively (Santín-Durán *et al.,* 2004) have lower levels of parasitism than animals in intensive systems with a high stocking rate. Management schemes designed to reduce the exposure of an animal to parasitic larvae are effective (*e.g.* Macey, 2004) Nutrition can affect the ability of the host to cope with the consequences of parasitism, to control and even overcome parasitism (Coop & Kyriazakis, 2001). Plant secondary compounds such as condensed tannins with anti-parasitic activity can also help alleviate internal parasitism in grazing ruminants (Hoste *et al*., 2006). Deer grazing chicory have been shown to be more resilient to parasitism than deer grazing perennial ryegrass-based pasture (Hoskin *et al*., 1999; 2003) and recent research indicates that grazing of plantain (*Plantago lanceolata*) may also result in increased resilience to internal parasitism in weaner deer during autumn compared with both perennial ryegrass-based pasture and chicory (Mwendwa *et al*., unpublished). Work currently in progress at Invermay would suggest weaners with access to browse in their diets may have lower levels of parasitism (Johnson *et al*., unpublished data)

Genotype may well be an important factor because some evidence suggests that the responses to endoparasitism differ in wapiti and red deer (Waldrup *et al*., 1994) and that wapiti may be more susceptible to endoparasitism under NZ farming conditions.

## CURRENT RESEARCH

A large scale longitudinal study is currently being undertaken by researchers at Massey University and AgResearch Invermay on two separate deer farm locations in NZ. This trial aims to quantify the effect of sub-clinical parasitism on weaner deer production, to evaluate the dynamics of parasite infections in young deer throughout the production cycle, and to analyse the relationship between worm burdens, diagnostic markers of parasitism, and production effects. Preliminary results indicate that natural parasite infections in weaner deer do cause a significant reduction in LWG and a loss of overall on-farm productivity.

A study of the possible anthelminthic effects of native plants is being undertaken at AgResearch Invermay. Initial laboratory trials have indicated that a number of native species may well contain compounds that have an anti-parasitic effect, the first field trials are currently underway.

Studies planned for the future include an investigation of the relative pathogenicity of lungworm, GI, and mixed lungworm and GI infections in young farmed deer using a controlled, indoor, artificial infection trial. An evaluation of methods of detecting parasitism in deer will be undertaken as well as a pilot survey of anthelmintic resistance status in farmed deer herds. A nationwide study will seek to accurately define the parasites infecting farmed red deer and to develop molecular markers for their detection. Further research topics include the role of browse and grazing management strategies in lowering parasite burdens, evaluation of the potential for compensatory growth and the relative susceptibility of red deer and wapiti to parasite infections.

## SUMMARY

It is clear that basic knowledge regarding the effect of internal parasitism on productivity of both immature and mature farmed deer in NZ is lacking. In addition, little is understood about the epidemiology of internal parasitism in NZ farmed deer However, sub-clinical parasitism can cause average reductions in growth rates of weaner deer grazing conventional ryegrass pasture in the order of 50%. Effects of clinical parasitism will be more severe and can result in death. Despite the potential of sub-clinical parasitism to significantly reduce productivity, few farmers appear to monitor parasitism by measures other than apparent liveweight gain and/or body condition and/ or clinical signs (Castillo-Alcala *et al*., 2007). The old adage "you can only manage what you can measure" is key to the future of managing parasitism on NZ deer farms and the first step under current evaluation is to determine what should be measured.

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