New Zealand Society of Animal Production online archive

Effects of immunisation against LHRH on body growth, scrotal circumference, and carcass composition in yearling Red deer stags

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ABSTRACT

Red deer stags received a series of immunisations against LHRH with the aim of suppressing the reduction in growth normally associated with the rut, but without affecting carcass fatness. Immunisations commenced at 10 months of age (September), including a series of either early (October) or Late (November) booster immunisations. the immunisations produced antibodies to LHRH and delayed testes enlargement, with effects being greatest during summer (December-January). Liveweight changes were similar in the late booster and control groups. Mean liveweight declined during the rut (April and May) in all groups. However, stags that received the early booster were significantly heavier ($P<0.05$) than the other two groups in October (11 months old) and from February to October of the following year (15-23 months old). Twenty four hours prior to slaughter (October), mean (±SE) liveweight was 110.2±4.9, 100.8±1.7 and 102.3±1.5 kg in the early booster, late booster and control groups respectively, with corresponding carcass weights being 63.67±2.92, 58.26±1.01 and 58.92±1.17 kg. Immunised deer tended ($P<0.10$) to be fatter than their control counterparts, but the effect was of very small magnitude.

Keywords Stags, LHRH immunisation, body growth, testes, carcass composition.

INTRODUCTION

Voluntary feed intake and growth in red deer stags of more than one year of age is depressed during the rut (breeding season), even when a high quality pelleted feed was provided *ad libitum* (Fennessy *et al.*, 1980). This reduction in feed intake limits the productivity of stags during a period when quality pasture is often abundant during the autumn flush. Feed intake also declines during the autumn and throughout the winter in hinds, but the decline in intake is much more rapid and precipitous in stags (Suttie *et al.*, 1987), possibly due to the sharp and sustained rise in plasma testosterone concentration during the rut. High levels of circulating testosterone are necessary for rutting behaviour (Lincoln *et al.*, 1972). Rutting behaviour has been blocked by auto-immunisation against LHRH in stags (Lincoln *et al.*, 1982).

Permanent surgical castration is known to eliminate weight loss during the rut in reindeer (Ryg & Jacobsen, 1982), but increases body fatness in red deer when performed at weaning (Drew *et al.*, 1978). In the present study, yearling red deer stags were given an anti-LHRH vaccine with the aim of suppressing the rut (mid March - mid May) by producing a temporary immuno- castration, to evaluate if this would reduce the depression in growth normally associated with the rut without affecting carcass fatness.

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MATERIALS AND METHODS

Thirty-three stags, approximately 10 months old, were brought onto the Massey University Deer Unit, blocked by weight, and randomly allocated to the following three treatments: early booster, late booster and control. The early booster group received the primary vaccine on 29 September 1989, and boosters on 26 October 1989, 13 November 1989, 12 December 1989, 8 March 1990 and 29 March 1990. The late booster group received the same sequence but omitting the first booster on 26 October 1989. The control group received a placebo vaccine (adjuvant and vehicle only) on the same dates as the early booster group. Each animal received 5 ml of the vaccine (2.5 ml sub-cutaneous on each side of the neck) on each occasion. The vaccine (Peptide Technology Ltd) comprised LHRH antigen, with the adjuvant being DEAE dextran in oil based vehicle. All animals were grazed together. Mean (±SE) pasture allowance during the rut was 9.3±1.3 kg DM/head/day, and the mean (±SE) pasture residual was 1450±228 kg DM/ha.

Pasture hay and pelleted concentrate supplements were offered during July and August. The animals were weighed and blood sampled monthly, at which time the vaccination sites were examined for any tissue reaction. Blood samples were generally taken a month after injecting the vaccine. Scrotal circumference was measured with a flexible tape on stags held in a dark room. Antibody titre to LHRH was determined by a direct binding, double antibody radioimmunoassay similar to that described by Aston *et al.* (1985) and Bomford and Aston (1990) for growth hormone. This procedure allows a rapid qualitative screening for anti-LHRH sera. A titre was considered positive at 10% or greater of the positive control titre at both 1/50 and 1/500 dilutions.

All animals were slaughtered at the Deer Slaughter Premises (DSP) of Venison NZ Ltd on 10 October 1990, one year after the commencement of the study. The animals were weighed off pasture 24 hours prior to slaughter. Carcass weight and GR (tissue depth at the 12th rib) was determined, as was the width of the rump fat pad from the carcass mid-line to the lateral extremity of the pad. The right forequarter was retained for chemical analysis. The forequarter was boned, the humerus weight and length measured, and the soft tissue minced and subsampled for Soxhlet fat extraction. Differences between treatment means were analysed by analysis of covariance. Initial liveweight, initial scrotal circumference and carcass weight were used as respective covariates in the analyses of subsequent liveweight, subsequent scrotal circumference and carcass fatness.

RESULTS

There were only 5 visible tissue reactions after a total of 172 vaccinations. Four stags died (two from the early boost, one from the late boost and one from the control groups) during the experiment due to pathological conditions unrelated to treatments. There was a greater proportion of stags from the early booster group with a positive titre to LHRH at the November and January sampling dates (Table 1). The number of stags with a positive titre then declined during February and March.

TABLE 1 Percentage of animals with a positive titre to LHRH.

	Early Booster Group	Late Booster Group
November	63.6	27.3
December	63.6	63.6
January	88.9	63.6
February	11.1	10.0
March	22.2	10.0

Scrotal circumferences were significantly ($P<0.05$) less in the early and late booster groups than in the control stags during November - January, but these differences disappeared as the testes enlarged towards the rut (Figure 1). Circumferences were always similar ($P>0.05$) between the two booster groups.

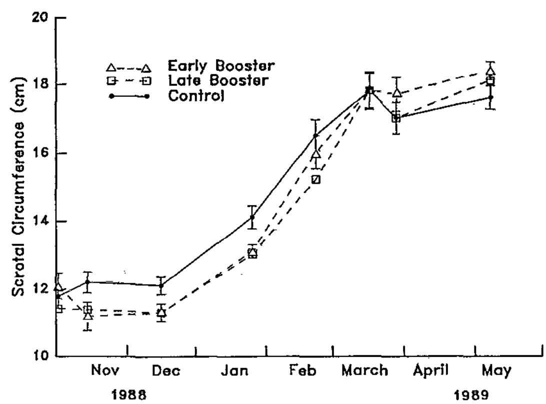

FIG 1 Scrotal circumference in yearling red deer stags immunised against LHRH.

Mean liveweight of the early booster group became significantly ($P<0.05$) greater than the late booster and control groups in October and then again from February to the end of the study (Fig 2). The mean liveweights of the late booster group and the control group were similar ($P>0.05$) throughout the duration of the study. Mean (±SE) liveweight loss during the rut was 3.9±0.8, 2.1±0.9 and 1.4±0.8 in the early booster, late booster and control groups respectively. Between June and October (19-23 months of age), the early booster group had a greater ($P<0.05$) daily weight gain (57.7±9.3 g/d) than the late booster group (38±4.8 g/d) and the control group (40±3.7 g/d).

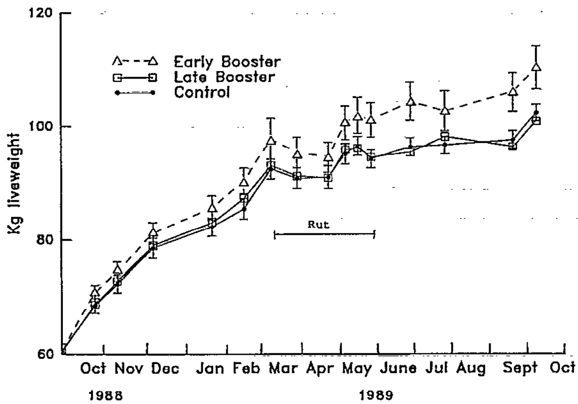

FIG 2 Liveweight in yearling red deer stags immunised against LHRH.

Mean liveweight 24 hours prior to slaughter was 8-10 kg greater (P<0.05) in the early booster group, and this difference was reflected in a 4-5 kg greater (P<0.05) mean carcass weight in the early booster group compared to the two other groups (Table 2). Killing out percentages were similar. There was a general tendency (P<0.10) for the immunised groups to be slightly fatter than the control group.

TABLE 2 Mean (±SE) liveweight 24 hours prior to slaughter, carcass weight, GR (tissue depth at the 12th rib) and percent fat of the right forequarter soft tissue in red deer stags immunised against LHRH.

	Control	Early Booster	Late Booster
Liveweight (kg)	102.3±1.5	110.2±4.9*	100.8±1.7
Carcass weight (kg)	58.9±1.2	63.7± 2.9*	58.3 ±1.0
Killing out (%)	57.6±0.8	57.8± 0.9	57.8±0.7
GR (mm)	4.0±0.3	4.4 ±0.3	4.9±0.3*
Tissue fat (%)	3.15± 0.50	4.29 ±0.25	3.78±0.22

* significantly different from control group at P<0.05

DISCUSSION

Immunisation against LHRH appeared to enhance the growth of yearling red deer stags, but not during the rut as anticipated, and only if the early booster was given. Auto-immunisation against LHRH has been reported to block the development of the rut in previous small scale studies (Lincoln *et al.*, 1982, 1984). In the present study the two boosters during March (just prior to the rut) appeared to have no effect; all animals had similar scrotal circumferences during the rut.

The mechanism by which the anti-LHRH vaccine enhanced growth before and after the rut in the early booster group is not known. The results of current radio-immunoassays for LH, IGF-1 and testosterone in the monthly blood samples may provide some evidence. As anti LHRH titre developed earlier and persisted longer in the early boost group, it appears that initiation of the vaccine sequence early in spring is essential. Titres against LHRH and melatonin are slow to develop in red deer (Ataja, 1990), and it may be necessary to establish a high titre by early summer to block the surge of LH reported during early summer (Suttie *et al.*, 1984 a, b; Fennessy *et al.*, 1985). The primary booster must be followed shortly (4 weeks) by at least one booster. There was an 8 week interval between the primary vaccine and first booster in the late booster group, in which there was no effect on liveweight. There was little difference in carcass quality between immunised and control stags. All carcasses had a GR score 4-5, which is well below the maximum score of 12, above which the carcass is down graded for over fatness. The vaccine treatment improved mean carcass weight by 4.8 kg at a early spring premium price of $6.20/kg.

In conclusion, immunisation against LHRH shows promise in enhancing live weight gain in those yearling stags held over for a second winter. This is an age class of stags which normally has poor weight gains relative to weaner stags. Current studies are examining the repeatability of these results and aim to gain a greater understanding of the endocrinology of this enhanced growth.

ACKNOWLEDGEMENTS

Mr J H Niezen and Mr A M Ataja are thanked for their skilled technical assistance, and Mr C Howell for assistance with deer husbandry.

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