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## Effect of dietary protein on liveweight gain in parasitised calves

V.T. BURGGRAAF, M.R. PUHA and A.A. ADLER

AgResearch Ruakura, Private Bag 3123, Hamilton, New Zealand

### ABSTRACT

The interaction between parasitism and crude protein (CP) nutrition on calf liveweight gain (LWG) was investigated. One hundred and twelve calves were either administered 21,000 parasite larvae (73% *Cooperia oncophora*, 16% *Ostertagia ostertagi*, 11% *Trichostrongylus axei*) twice per week or drenched fortnightly with anthelmintic for 12 weeks. Calves grazed ryegrass pastures and received 40% of their diet as either low (15% CP) or high (26% CP) protein pellets. Half of the calves switched diets after six weeks. All treatments averaged a liveweight gain of at least 1 kg/d. After 12 weeks non-parasitised calves were 13 (standard error of difference (SED 2.0)) kg heavier than parasitised calves ( $P < 0.001$ ), and calves fed a high protein supplement were 12 (SED 2.8) kg heavier than those fed low protein supplement ( $P < 0.001$ ). There was no interaction between diet and parasitism ( $P = 0.74$ ). Subsequently calves grazed together for a further 12 weeks with no supplements or parasite dosing. The liveweight advantage of the high protein supplement was lost, while non-parasitised calves maintained a liveweight advantage of 15 (SED 4.9) kg ( $P < 0.01$ ). While protein supplementation did not affect faecal egg count or blood pepsinogen it has the potential to improve LWG with less reliance on anthelmintics under field conditions.

**Keywords:** cattle; liveweight gain; parasite; protein.

### INTRODUCTION

Liveweight gain (LWG) in grazing cattle is often poor in summer and early autumn due to a combination of slow growth of pasture with a low nutritive value, high parasite challenge and fungal toxins. With the rise in anthelmintic resistance and consumer concerns about chemical use in food production, alternatives to regular drenching are required for internal parasite management. Providing a high quality feed supplement during the late summer/early autumn period may reduce the ingestion of infective parasite larvae and improve the performance of animals exposed to a parasite challenge.

Gastrointestinal parasite infections increase the loss of endogenous protein, possibly due to plasma leakage, and an increase in sloughed cells and mucous production (Poppi *et al.*, 1986). Increasing the supply of metabolisable protein in parasitised sheep can reduce faecal egg output and worm burdens and enable identical LWG to non-parasitised control animals (Bown *et al.*, 1991; van Houtert *et al.*, 1995). Short term provision of protein can also have a long term effect on LWG of parasitised sheep (Datta *et al.*, 1999). Despite the value of protein for managing parasites in sheep, there is a lack of data on the interaction of protein with LWG in cattle under parasite challenge.

Our research compared the performance of pasture-fed calves supplemented with either a low or high protein feed, with and without parasite infection during the late summer early autumn period. The timing of supplementation was also investigated, to determine if it was more effective

during the early stages of parasite infection or once a significant worm burden had established.

### MATERIALS AND METHODS

#### Trial design and animals

The experiment was a four (diet) by two (parasitised or non-parasitised) factorial split plot design, with two replicates. All calves grazed pasture as 60% of their diet, with the remainder comprising either a high protein supplement for 12 weeks (HH), a low protein supplement for 12 weeks (LL), high protein for the first six weeks followed by low protein for six weeks (HL), or vice versa (LH). Each replicate had seven calves per treatment. The high protein supplement calves grazed in one mob with the low protein supplement calves grazing the same paddock with the two groups separated by an electric fence. Each replicate was in a separate paddock.

One hundred and twelve spring-born Friesian bull calves with a mean live weight of 136 kg were assigned to eight, liveweight balanced, treatment groups in January 2007. All calves were grazed on pasture and drenched fortnightly with a combination anthelmintic (Arrest C, Ancare NZ Ltd; 100 g/L albendazole and 75 g/L levamisole) for six weeks prior to starting the experiment. Parasitised calves were each dosed orally twice weekly with 21,000 viable larvae per animal comprising 73% *Cooperia oncophora*, 16% *Ostertagia ostertagi*, 11% *Trichostrongylus axei* suspended in water. Non-parasitised calves were drenched fortnightly with Arrest C (1 ml/10 kg of live weight of the heaviest calf in the mob) to prevent adult parasite development. After the 12 week supplementation

period, calves on the LL and HH diets continued grazing in one mob for 12 weeks to determine any carry over effects of the treatments. The control treatments continued to be drenched fortnightly.

### Supplements and pasture

The experiment was conducted on ryegrass-based pastures previously only grazed by dairy cows at the AgResearch Ruakura Research Centre. Calves were accustomed to a mixture of the high and low protein supplement for three weeks before the experiment, with the supplement allowance gradually increasing from 0.5 to 2 kg/hd/d. Supplements comprised pellets formulated primarily from a mixture of maize, wheat, sunflower and soya bean products, with different crude protein (CP) contents (Table 1). During the treatment period calves received a daily supplement as 40% of their diet DM, placed in feed troughs in the paddock with each mob. Each paddock was grazed only once by calves. Pre and post-grazing herbage mass was measured for each mob of calves using a calibrated rising plate meter. Herbage allowance and intake were estimated from these data. Break sizes allowed for average intakes based on supplement plus pasture for each mob of 3.5% of live weight. The allowance was adjusted at 3-weekly intervals to allow for changes in live weight. Calves were given a fresh pasture break at least twice per week, when herbage mass reached approximately 1.8 t DM/ha during the first six weeks of the trial. This was reduced to 1.6 t DM/ha in the second half of the treatment period due to a slowing in pasture growth rate limiting the amount of pasture available for the trial.

During the carry over period, calves on the LL and HH diets grazed in one group for 12 weeks with no supplement, to an average pasture residual of 1.6 t DM/ha. The non-parasitised treatment continued to be drenched fortnightly.

### Measurements

A minimum of 300 pasture samples were plucked per paddock to imitate the likely grazing height by the calves to estimate pasture quality measured by using near infrared spectroscopy (Corson *et al.*, 1999) and infective parasite larvae concentration determination (C.J. Boom, Personal communication). Samples were bulked to provide one sample per replicate per week during the treatment period, and one sample per fortnight during the carry over period. Supplements were dried, ground and analysed for metabolisable energy (ME) (Clark *et al.*, 1982) and crude protein (Kjeldhal N x 6.25).

Individual animal live weights of all calves were measured weekly during the treatment period and fortnightly during the carry over period. Faecal egg counts (FEC) were determined from faecal samples collected per rectum fortnightly during the

treatment period on all parasitised calves and 6-weekly during the carry over period on parasitised HH and LL calves. All non-parasitised calves were sampled on the first measurement date. A selection of these calves was sampled on subsequent dates to check the efficacy of drenching. FEC were determined using a modified McMaster method (MAFF, 1997). Serum pepsinogen concentrations were measured (Hirshowitz, 1957) in blood collected by tail venipuncture from parasitised HH and LL calves every six weeks during the treatment period and every four weeks during the carry over period. The remainder of the calves were sampled in weeks 1, 12 and 24.

### Statistical analysis

Data were analysed by analysis of variance (Genstat Version 9.2). There was no significant mob effect for any variable so in all analyses calves were used as the experimental unit. The analysis was a completely randomised factorial fitting four supplement treatments, two parasite treatments and the interaction. There were no significant interactions. Live weights were adjusted before analysis using the initial weight as a covariate. Faecal egg count data were log<sub>e</sub> transformed before analysis to normalise the variance.

## RESULTS

### Pasture and supplements

The concentration of infective parasite larvae on pasture grazed during the treatment period was very low with an average value of 29 larvae/kg DM. The larval population comprised 58% *Ostertagia ostertagi* and 42% *Trichostrongylus* spp. Concentrations of larvae on pasture remained low during the carry over period with an average value of 83 larvae/kg DM. The larval population comprised 47% *Ostertagia ostertagi*, 37% *Trichostrongylus* spp., 15% *Haemonchus* spp. and 1% *Cooperia oncophora*.

Pastures had an average pre-grazing herbage mass of 2.8 t DM/ha during Weeks 1 to 6, and 2.4 t DM/ha during Weeks 7 to 12. Pasture CP concentrations were low in the first six weeks with an average value of 14% that increased to 20% in the following six weeks. The average ME content of the pastures during these two periods was 10.1 and 10.9 MJ/kg DM, respectively. The supplements had a CP of 26 and 15% for the high and low protein treatments, respectively and a high ME content. The resulting total diet had an average CP concentration of 21 and 16%, and a ME content of 11.5 and 11.3 MJ/kg DM for the high and low protein diets, respectively. During the carry over period the pastures grazed had an average CP of 25% and a ME content of 11.6 MJ/kg DM.

**TABLE 1:** Backtransformed mean faecal egg counts (eggs/g/ fresh faeces) of parasitised calves grazing pasture and fed a high (HH) or low (LL) protein supplement. SED = Standard error of difference; P = Probability of difference between treatments.

Stage of trial	HH	LL	SED	P value
Week 2	3	6	6	0.650
Week 4	353	414	1	0.014
Week 6	570	334	12	0.034
Week 8	287	595	64	0.131
Week 10	148	159	17	0.959
Week 12	51	66	1	0.771
Week 18	68	159	30	0.209
Week 24	39	44	1	0.135

**TABLE 2:** Mean live weight and liveweight gain of parasitised versus non-parasitised calves and calves fed a high (HH) or low (LL) protein diet for 12 weeks, or calves switching from a high to low protein diet after 6 weeks (HL) or vice versa (LH). Data within columns with different superscripts indicate significantly different means (P <0.05) for diet comparisons. SED = Standard error of difference; P = Probability of difference between treatments.

Treatment	Live weight (kg)				Liveweight gain (kg/hd/d)		
	Week 0	Week 6	Week 12	Week 24	Week 0 - 6	Week 7 - 12	Week 13 - 24
<b>Diet effect</b>							
HH	138	187 <sup>b</sup>	234 <sup>c</sup>	300	1.15 <sup>b</sup>	1.14 <sup>b</sup>	0.78
HL	136	187 <sup>b</sup>	227 <sup>ab</sup>		1.18 <sup>b</sup>	0.94 <sup>a</sup>	
LH	138	180 <sup>a</sup>	230 <sup>bc</sup>		0.99 <sup>a</sup>	1.21 <sup>b</sup>	
LL	136	180 <sup>a</sup>	222 <sup>a</sup>	398	1.04 <sup>a</sup>	0.97 <sup>a</sup>	0.89
SED	0.8	1.8	2.8	5.0	0.037	0.048	0.042
P		<0.001	<0.001	0.130	<0.001	<0.001	0.141
<b>Parasite effect</b>							
Parasitised	138	181	222	291	1.02	0.95	0.81
Non-parasitised	136	186	235	306	1.15	1.18	0.86
SED	0.8	1.2	2.0	5.0	0.025	0.034	0.042
P		0.001	<0.001	0.003	<0.001	<0.001	0.26
<b>Diet x parasite effect</b>							
P		0.122	0.740	0.365	0.525	0.453	0.316

**Animals**

Estimated animal intakes of both diets were similar, averaging 5.2 and 7.1 kg DM/hd/d for the first and second six weeks, respectively. All calves had an initial FEC of 0 eggs per gram of fresh faeces (epg). For parasitised calves, FEC peaked in Week 6 at 570 epg, on the high protein diet, and in Week 8 at 595 epg on the low protein diet (Table 1). For the remainder of the experiment, average FEC was below 200 epg with no significant diet effect. Blood pepsinogen concentrations increased with parasite infection from an initial average of 0.95 mU/L, but were not affected by diet. Calves with a parasitic infection had a higher pepsinogen concentration in Week 12 (1.91 vs 1.07 mU/l; SED 0.13) and Week 24 (2.86 vs 1.95 mU/l; SED 0.16) than non-parasitised calves (P <0.001).

The average LWG of all treatments was at least 1 kg/d. During both the first and second six week period, HL and LH calves had a similar LWG to calves on the same respective diet in each period (Table 2). By Week 6, low protein diets and parasitism had caused a significant reduction in LWG (Table 2). By Week 12 non-parasitised calves had grown 13 kg more than parasitised calves (P <0.001; SED 2.0), and high protein calves had grown 12 kg more than those on the low protein diet (P <0.001; SED 2.8), with those swapping diets having an intermediate LWG. There was no interaction between diet and parasitism (P = 0.74).

The advantage of the HH diet over the LL diet was lost 10 weeks into the carryover period (Table 2). The LL calves appeared to have some compensatory growth, although significant differences in LWG were not detected during this time (Table 2). Non-parasitised calves still had a 15 kg live weight advantage (P <0.01) over parasitised calves at the end of the experiment (Table 2).

**DISCUSSION**

The generous allowance of high nutritive value feed in this experiment enabled high LWG for calves on all treatments, with the greatest gain on the high protein diet. Our results indicate that the longer a high protein supplement can be provided the better while if protein intake is limited the stage of parasite infection at which the protein supplement is fed is of little relevance. This is similar to results for sheep suggesting better LWG on high protein diets, but little effect of the timing of supplementation on response to infection (Kahn *et al.*, 2000).

The decrease in calf LWG gain from moderate levels of parasite infection was similar to that measured previously (Burggraaf & Puha, 2007). Increased dietary CP improved LWG to the same degree in both parasitised and non-parasitised calves. Given the high ME relative to CP, it appears LWG on the low protein diet was limited by metabolisable protein supply even in non-parasitised calves. A larger increase in metabolisable protein relative to ME may have enabled parasitised calves to grow similar to non-parasitised calves on the same diet. Threshold dietary levels of 19% CP

(Datta *et al.*, 1998) and 14 g/kg LW<sup>0.75</sup> (Downey *et al.*, 1972) have been suggested to provide good resilience to parasite infection in sheep. No such threshold has been defined for young cattle, and would depend on other factors such as the rumen degradability of the protein, ME content of the diet and the stage of growth of the animal.

The LWG penalty resulting from parasitism has been attributed to the cost of mounting an immune response, which has a high protein demand (Steel, 2003). Our experiment did not provide evidence for improved resistance due to small increases in dietary protein, as FEC and blood pepsinogen were similar between diets. Previous research has shown either no effect (Wallace *et al.*, 1995; Kahn *et al.*, 2000) or a decrease in FEC or worm burden (Bown *et al.*, 1991; van Houtert *et al.*, 1995) from improved protein supply. In our experiment, it appears that the increased protein was partitioned primarily towards calf LWG rather than eradication of adult parasites.

Unlike the study of Datta *et al.* (1999), where protein supplementation had long term effects on LWG in parasitised sheep, the advantage of the high protein diet declined, and was no longer evident 12 weeks after supplementation ceased. Boom and Sheath (1998) similarly found little long term benefit of supplementing cattle grazing summer pasture due to subsequent compensatory growth of those on pasture only. The effects of parasite infection, however, were maintained throughout the 12 week carry over period in this study, as reported previously (Burggraaf & Puha, 2007).

Although protein supplementation has proved effective at reducing the effects of parasites on animal performance, the supplements used in this experiment were expensive and their benefit was not sustained. In order for farmers to benefit financially from the gains in animal production from high protein diets a cheap source of a suitable feed is required. Farmers should consider when their calves are most vulnerable to parasites in conjunction with the availability of a suitable supplement as to whether supplementary feeding is an appropriate strategy, or whether managing pastures to increase their nutritive value and reduce parasite infection would be sufficient to reduce their dependence on anthelmintic use.

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