Feed intake is regulated by metabolic mechanisms in young wethers fed diets deficient in crude protein and phosphorus

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Ruminants consuming pastures in northern Australia that are deficient in crude protein (CP) or phosphorus (P) in the dry and wet seasons respectively have reduced feed intake and reduced liveweight gain. Earlier studies in sheep (Egan 1965; Milton & Ternouth 1985) suggested that metabolic mechanisms might be responsible for this phenomenon rather than rumen fill. Here we hypothesised that voluntary dry matter (DM) intake would decrease in wethers fed diets deficient in CP and/or P and this reduction in intake would not be due to rumen fill.

Merino wethers [n=40; 7 months old; 23.7 ± 1.4 kg liveweight (mean ± SD)] were fed dietary treatments (n=8/treatment) for 63 days in individual pens. The treatments included targeted combinations of high and low CP (110 vs 55 g/kg DM) and high and low P (2.5 and 0.7 g/kg DM) resulting in four experimental diets (lowCP-highP, highCP-lowP, lowCP-lowP, highCP-highP) formulated to provide 9 MJ ME/kg DM which were fed *ad libitum*. An additional treatment restricted intake of the highCP-highP diet to an equivalent ME intake of wethers consuming the lowCP-lowP diet. Wethers were fed daily, with feed offered and feed refused measured weekly with daily DM intake calculated as the average intake over a 7 day period. Blood was collected fortnightly and DM digestibility determined by the daily collection of total faecal output from each wether over 7 days. Wethers were euthanised 2 hours after feeding over 5 days. The reticulo-rumen was evacuated, total digesta load was weighed, rumen fluid was collected to determine the concentration of ammonia-N and apparent retention time of digesta in the rumen was calculated from subsamples dried to a constant weight at 60°C.

Table 1. Dry matter (DM) intake, DM digestibility, rumen digesta load, plasma inorganic phosphate, plasma urea nitrogen, rumen ammonia concentration and apparent retention time of wethers fed diets[1] adequate or deficient in crude protein (CP) and phosphorus (P).

	Low CP-High P	High CP-Low P	Low CP-Low P	High CP-High P	High CP-High P-R	SEM[2]	*P*
Dry matter intake (g DM/kg LW.day)[2]	21.8^{ab}	25.3^{b}	19.1^{a}	37.2^{c}	19.7^{a}	0.97	<0.001
Dry matter digestibility (%)	61.7	61.0	60.0	63.6	66.9	1.67	0.06
Rumen digesta load (g DM)	300^{ab}	322^{b}	199^{a}	499^{c}	442^{c}	26.7	<0.001
Plasma inorganic P (mmol/L)	2.4^{d}	1.1^{a}	1.6^{b}	2.0^{c}	2.4^{d}	0.09	<0.001
Plasma urea N (mmol/L)	1.1^{a}	4.4^{b}	1.6^{a}	4.1^{b}	4.1^{b}	0.22	<0.001
Rumen ammonia (mg NH_3N/L)	42^{a}	79^{ab}	34^{a}	143^{bc}	180^{c}	19.3	<0.001
Apparent retention time (h)	14^{a}	14^{a}	14^{a}	10^{a}	27^{b}	1.12	<0.001

[1]Treatments described in the text.

[2]Values are least-square means, standard error of the mean (SEM) and *P*-value; different alphabetical superscripts across each row indicate a significant difference between treatments ($P \leq 0.05$). Liveweight (LW).

The dietary treatments were effective in establishing models of CP and P deficiency as indicated by the plasma concentrations of inorganic P and urea N (Table 1). Wethers offered the highCP-lowP, lowCP-highP and lowCP-lowP treatments experienced a 32, 42 and 49% lower DM intake respectively than the non-deficient treatment (highCP-highP; $P \leq 0.05$). The DM digestibility was high in all treatments, with only a tendency ($P=0.06$; Table 1) to be higher in the restricted wethers (highCP-highP-R). The concentration of ammonia in the rumen was lower in wethers fed lowCP treatments compared to the highCP-highP treatment ($P \leq 0.05$, Table 1). The digesta load in the rumen was higher in wethers fed highCP-highP and highCP-highP-R treatments ($P \leq 0.05$; Table 1) which reflects the higher daily DM intake (Table 1) and higher rate of intake within the first 2 hours of feeding time (data no shown), respectively; however, apparent retention time was not different in all wethers fed *ad libitum* (Table 1).

Intake suppression in response to nutrient deficiency was not initially due to a physical limitation of the rumen but is likely to be related to metabolic mechanisms in the hypothalamus and/or peripheral tissues. We are currently using molecular techniques to identify the key genes and pathways regulating feed intake in ruminants fed diets deficient in CP or P. Understanding these mechanisms may result in the development of strategies to improve utilisation of pasture resources in northern Australia.

References

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