NUTRITIONAL EVALUATION OF KALE (*BRASSICA OLERACEA*)

T. N. BARRY, T. R. MANLEY, T. C. REID, K. R. WILSON and P. HEMMINGSEN

Invermay Agricultural Research Centre, Mosgiel

K. R. MILLAR

Wallaceville Animal Research Centre, Upper Hutt

SUMMARY

Rumen fermentation of S-methyl-cysteine sulphoxide and induced copper deficiency were implicated in the low growth rates of young ruminants fed kale diets. Young cattle grazing kale for prolonged periods should receive Cu supplementation.

INTRODUCTION

Nicol and Barry (1980) reviewed the New Zealand literature on the feeding value of Brassica crops, and found animal growth rates to be low (lambs 100 to 150 g/day, cattle 300 to 500 g/day) in relation to the high apparent digestibility (85 to 90%) and nitrogen content (2.5 to 3.5% DM) of these crops. All Brassica crops contain the free amino acid S-methyl-cysteine sulphoxide (SMCO) at 0.5 to 1.2% DM. SMCO is metabolized by rumen organisms to the oxidizing drug dimethyl disulphide, which attacks erythrocytes (red blood cells) causing the formation of Heinz bodies in them and greatly accelerating their rate of destruction, producing haemolytic anaemia (Smith, 1974). Kale contains both low concentrations of copper (3 to 6 mg/kg DM) and high concentrations of sulphur (0.8 to 1.0% DM), resulting in estimated plant concentrations of truly available copper (Cu) that are below Cu requirements for animal growth. The two experiments reported here were designed to examine the extent that SMCO fermentation and low plant concentrations of truly available Cu could be limiting the growth of young ruminants fed sole diets of kale.

EXPERIMENT 1

Lambs aged 5 months were individually fed *ad libitum* fresh diets of either kale (OMD 87%) or lucerne (OMD 73%) for 6 weeks. Synthetic L-SMCO was added to both diets at 0, 0.2,

0.4, 0.8 and 1.6% DM (4 lambs/group). SMCO contents of the kale and lucerne were, respectively, 0.77 and 0.04% DM. In the absence of supplementary SMCO, less than 5% of the erythrocytes in animals fed lucerne contained Heinz bodies, where as in animals fed kale 50% of erythrocytes contained Heinz bodies in week 3, but this stabilized at 40% by week 6. The increase in Heinz bodies was associated with a reduction in packed cell volume (PCV), both being typical symptoms of kale anaemia (Smith, 1974). The level of SMCO supplementation produced strong interactions ($P < 0.01$) with DM intake and empty body-weight gain (EBWG) (Table 1). SMCO had little effect on lambs fed lucerne, the intake depression at 1.6% SMCO only just attaining significance ($P < 0.05$), while the trend for lower EBWG at 0.8 and 1.6% was not significant ($P > 0.05$). In contrast, in lambs fed kale the 0.8 and 1.6% levels of SMCO supplementation markedly depressed both intake

TABLE 1: EFFECT OF SYNTHETIC SMCO ADDED TO THE DIET OF YOUNG SHEEP FED FINELY CHOPPED FRESH LUCERNE AND KALE DIETS

Basal Diet	*Week*	*SMCO added (%DM)* 0	0.2	0.4	0.8	1.6		*SEM*
		DM intake (g/kg $W^{0.75}$/day)						
Lucerne	1–6	98.5	102.7	105.5	99.2	87.5	)	
Kale		83.3	69.1	87.0	57.4	50.9	)	3.75
		SMCO intake (g/kg liveweight/day)						
Lucerne	1–6	0	0.10	0.21	0.38	0.69		
Kale		0.31	0.31	0.48	0.40	0.53		
		Empty body-weight gain (g/day)						
Lucerne	1–6	79.3	93.7	91.5	60.1	62.0	)	
Kale		62.2	6.6	57.9	−39.6	−30.9	)	11.9
		Erythrocytes containing Heinz bodies (%)						
Lucerne	3	4.8	1.5	8.3	23.8	72.8	)	
Kale		49.5	76.8	68.0	76.8	90.5	)	6.9
Lucerne	6	3.3	7.8	9.8	35.0	41.0	)	
Kale		39.5	36.3	47.0	51.3	53.8	)	6.9
		PCV (ml erythrocytes/100 ml whole blood)						
Lucerne	3	36.5	36.7	37.6	37.2	32.7	)	
Kale		32.1	28.7	27.8	28.0	28.7	)	1.43
Lucerne	6	40.4	41.0	40.2	40.1	39.8	)	
Kale		34.1	30.9	29.9	27.0	25.7	)	1.65

($P < 0.001$) and EBWG ($P < 0.001$). The 0.2% level caused smaller reductions in intake ($P < 0.05$) and EBWG ($P < 0.01$), while there was no reduction in performance at the 0.4% level. This is probably due to there being differences between animals in the point at which intake and EBWG decline as dietary SMCO concentration increases. Even at the same total SMCO levels in the diet (plant + supplementary), SMCO was much more toxic to lambs fed on kale than on lucerne, and this is reflected in higher Heinz body counts and lower PCV values in lambs fed kale. In lambs fed lucerne, increments in dietary SMCO produced corresponding increases in daily SMCO intake per unit liveweight, but in those fed kale the increases were smaller because of their reduced DM intake.

EXPERIMENT 2

Yearling cattle (177 kg mean liveweight) grazing sole diets of either kale or ryegrass/clover pasture for 24 weeks were divided into control and Cu-supplemented groups. Copper (100 mg) was given subcutaneously as Cu EDTA during weeks 0, 12 and 18. Concentrations of Cu in the kale and pasture were 5 and 14 mg/kg DM, and of sulphur 0.80 and 0.45% DM, respectively. This gave estimated concentrations of truly available Cu/kg plant DM of 0.09 mg for kale and 0.39 mg for pasture, against an estimated animal requirement of 0.45 mg/kg (Suttle and McLauchlan, 1976). Although control animals grazing pasture showed some depletion of liver Cu, this did not result in any reduction in either serum or erythrocyte Cu concentration, and there was no response in liveweight gain (LWG) to Cu supplementation. In contrast, control animals grazing kale showed severe Cu depletion, giving reductions in Cu concentration in liver, serum and erythrocytes (Table 2). Liveweight gain in the control group fed kale remained low throughout the experiment, and the response to Cu increased with time, being 36, 77, 212 and 253 g/day in successive 6-week periods. Relative to the pasture groups, blood PCV and reduced glutathione concentration (GSH) were lower in the control animals fed on kale ($P < 0.001$), but were increased by Cu supplementation ($P < 0.05$), with the values reached after 21 weeks being similar to those of pasture-fed animals. The principal symptom of Cu deficiency in cattle grazing kale for prolonged periods was therefore for the anaemia initially caused by rumen fermentation of SMCO to

TABLE 2: EFFECT OF COPPER SUPPLEMENTATION UPON LIVEWEIGHT GAIN, TISSUE COPPER CONCENTRATION AND HAEMATOLOGY IN YEARLING CATTLE GRAZING SOLE DIETS OF KALE OR RYEGRASS/CLOVER PASTURE FOR 24 WEEKS

	Kale		*Pasture*		
Week	*No Cu*	*Cu*	*No Cu*	*Cu*	*SEM*
		Liveweight gain (g/day)			
0-6	231	267	382	371	35.8
7-12	385	462	−236	−176	30.7
13-18	202	414	617	621	33.3
19-24	327	580	789	826	35.1
		Liver Cu (mg/kg fresh weight) [1]			
12	1.7	4.9	3.5	23.1	1.44
21	1.5	24.0	3.0	56.0	2.04
		Serum Cu (mg Cu/l) [1]			
12	0.23	0.46	0.49	0.83	0.040
21	0.19	0.65	0.59	0.91	0.036
		Erythrocyte Cu (mg/l cells)			
12	0.55	0.74	0.69	0.75	0.169
21	0.36	0.70	0.81	0.71	0.090
		PCV (ml erythrocytes/100 ml whole blood)			
12	31.5	35.1	41.5	41.5	0.71
21	31.6	40.7	39.0	41.8	0.59
		GSH (mg/100 ml erythrocytes)			
12	33.3	40.6	66.7	66.0	2.89
21	47.5	68.6	76.9	80.7	3.18

[1] Initial values 11 mg/kg fresh liver and 0.80 mg/l serum.

become more severe, and to continue for a longer time than occurred in corresponding animals given supplementary Cu. This condition was associated with reduced LWG.

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