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BRIEF COMMUNICATION: The effect of feed restriction during peak lactation on milk production parameters

J.K. KAY[1], C.V.C. PHYN[1], Y.J. WILLIAMS[2, 3], J.R. ROCHE[1], C.R. BURKE[1] and S. MEIER[1]

[1]DairyNZ, Private Bag 3122, Hamilton 3240, New Zealand
[2]DairyNZ, 42 Whareroa Road, R.D. 12, Hawera 4640, New Zealand
[3]Present address: Department of Primary Industries, 120 Cooma Road, Kyabram, Victoria 3620, Australia

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INTRODUCTION

Adverse weather conditions that reduce pasture growth and/or quality can place the lactating dairy cow under periods of transitory nutritional stress. Previous research that has focused on the effects of feed restrictions in a pasture-based dairy system have imposed feed deficits either pre-calving (Roche, 2007), immediately post-calving for five to six weeks (Bryant & Trigg, 1979; Roche, 2007) or up to a 23% restriction in dry matter intake (DMI) (Bryant & Trigg, 1979). All experiments reported an immediate reduction in milk production; however, the longer-term effects varied depending on the intensity and duration of the restriction, and the stage of lactation at which the feed restriction was imposed. In a seasonal pasture-based system, feed deficits that occur during peak lactation may affect subsequent energy intake and milk production. Determining the immediate and longer-term milk production, body condition score (BCS) and reproduction responses to an acute feed restriction during peak lactation is essential to enable dairy farmers to make informed management decisions regarding supplementary feeding during this period. The primary objective of this experiment was to determine the effects of an acute feed restriction during peak lactation on immediate and longer-term milk production responses.

MATERIALS AND METHODS

Animals and treatments

Multiparous (n = 219) and primiparous (n = 57) Holstein-Friesian, Jersey and Friesian x Jersey cows were managed to calve at a BCS of 5.0 and 5.5 respectively, (1 = Emaciated to 10 = Obese; Roche *et al.,* 2004). During the first 61 ± 15.4 days in milk (DIM) (Mean ± SD), all animals grazed as one herd and were offered a pasture allowance of ~40 kg DM/cow/d to achieve DMI of 15 kg DM/cow/d. At 61 ± 15.4 DIM, cows were randomly allocated to either a high (HPA) or a low pasture allowance (LPA). Pasture allowances were incrementally increased by 0.8 kg DM/cow/d in the HPA treatment and reduced by 2.4 kg DM/cow/d in the LPA treatment over one week (Transition period). This transition period was followed by a two-week treatment period, during which daily pasture allowances were managed to achieve an average DMI of 16 kg and 8 kg DM/cow/d in the HPA and LPA treatments, respectively. Following the two-week treatment period, pasture allowances were readjusted to achieve a DMI of ~16 kg DM/cow/d in both treatments for a 13-week carry-over period.

Pasture measurements and analysis

Pre- and post-grazing pasture mass was estimated on three days each week during the transition and treatment periods, and on one day each week during the carry-over period by the same person using calibrated visual assessment (Roche, 2007). Average daily DMI (kg DM/cow/d) was estimated as the product of the difference between the pre- and post-grazing mass and area grazed, divided by the number of cows.

Representative pasture samples were collected five days each week by hand-clipping pasture to the predicted grazing height from paddocks immediately before grazing. Samples were bulked weekly and dried at either 100^{o}C for DM analysis or 60^{o}C for analysis of nutrient composition (Roche, 2007). Metabolisable energy (ME) content of the pasture was calculated as described by Roche (2007) and energy intakes (MJ ME/cow/d) were calculated as the product of pasture DMI and ME.

Milk and animal measurements

Individual milk yields were recorded daily (Westfalia Surge, Oelde, Germany). Fat and crude protein concentrations were determined by Milkoscan (Foss Electric, Denmark) on a weekly evening and morning composite sample for the two weeks before the transition period until the end of the treatment period, fortnightly for the first six weeks of the carry-over period, and once during week 13 of the carry-over period.

Data were analysed by ANOVA using generalised linear mixed models in GenStat (Payne *et al*., 2008). Differences were considered significant at $P < 0.05$, and a trend declared at $P < 0.10$.

RESULTS AND DISCUSSION

Pasture was of high quality throughout the experiment with an average ME of 11.2 ± 0.03

TABLE 1: Mean milk production from cows consuming a high (HPA; 14.3 ± 5.5 kg DM/cow/d) or low (LPA; 7.9 ± 2.0 kg DM/cow/d) pasture intake for two weeks during peak lactation. SED = Standard error of the difference.

Variable	Week[1]	HPA	LPA	SED	P value
Milk(kg/d)					
	0 to 2	20.3	15.1	0.66	<0.01
	3 to 4	18.4	16.9	0.57	<0.01
	5 to 6	17.7	16.9	0.58	0.13
	7 to 8	17.3	16.8	0.63	0.36
	14 to 15	13.9	13.5	0.61	0.56
Fat (%)					
	0 to 2	4.98	4.84	0.14	0.58
	3 to 4	5.53	4.93	0.17	<0.01
	5 to 6	5.11	4.88	0.15	0.14
	7 to 8	5.12	4.73	0.18	0.03
	14 to 15	5.05	4.80	0.17	0.15
Protein(%)					
	0 to 2	3.87	3.48	0.05	<0.01
	3 to 4	3.84	3.71	0.05	<0.01
	5 to 6	3.90	3.82	0.05	0.16
	7 to 8	3.92	3.83	0.06	0.17
	14 to 15	3.86	3.73	0.07	0.07
Fat(kg/d)					
	0 to 2	1.01	0.71	0.03	<0.01
	3 to 4	0.99	0.82	0.03	<0.01
	5 to 6	0.89	0.80	0.03	<0.01
	7 to 8	0.89	0.80	0.02	<0.01
	14 to 15	0.68	0.63	0.03	0.10
Protein(kg/d)					
	0 to 2	0.79	0.52	0.02	<0.01
	3 to 4	0.70	0.62	0.02	<0.01
	5 to 6	0.69	0.64	0.02	<0.01
	7 to 8	0.70	0.66	0.02	<0.05
	14 to 15	0.52	0.50	0.02	0.16

[1]Week relative to treatment initiation.
Weeks 0 to 2 = Treatment period;
Weeks 3 to 15 = Carry-over period.

MJ/kg DM, crude protein of 21.2 ± 0.5% DM, and neutral detergent fibre of 48.7 ± 0.3% DM. During the treatment period, pasture representive of that eaten by the HPA cows had a greater ME content (11.4 vs 11.0 ± 0.07 MJ/kg DM) compared with that eaten by the LPA cows. For the first 61 ± 15.4 DIM, average pasture allowance (38.6 ± 11.0 kg DM/cow/d), DMI (14.3 ± 5.5 kg DM/cow/d) and energy intakes (167 ± 65 MJ ME/cow/d) were similar for all animals. During the two-week treatment period, pasture allowance, DMI, and energy intake for the HPA cows were 43.6 ± 11.0 kg DM/cow/d, 14.8 ± 3.6 kg DM/cow/d, and 169 ± 40 MJ ME/cow/d, respectively, and for the LPA cows were 24.5 kg DM/cow/d, 7.9 ± 2.0 kg DM/cow/d, and 87 ± 19 MJ ME/cow/d, respectively. This represented a 47% decrease in DMI and a 49% decrease in MJ ME/d in the LPA cows.

FIGURE 1. Mean milksolids production from cows consuming a high (HPA; 14.3 ± 5.5 kg DM/cow/d) or low (LPA; 7.9 ± 2.0 kg DM/cow/d) pasture intake for two weeks during peak lactation. The open bar illustrates the transition period and the solid bar the restriction period. Error bars show standard error of the difference.

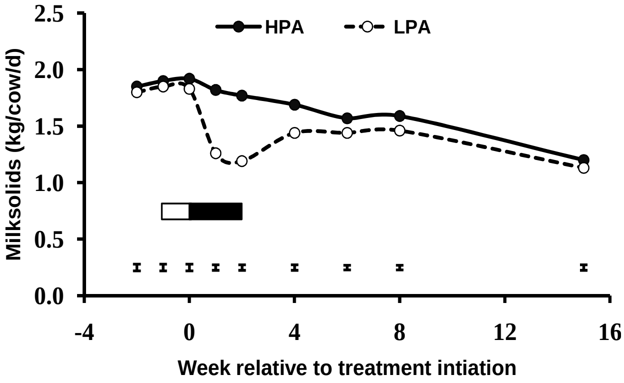

Table 1 presents milk production during the two-week treatment period and the 13-week carry-over period. LPA cows produced 27% less milk, 30% less fat, and 34% less protein than the HPA cows during the restriction. This immediate decline in milk production is consistent with other experiments that have imposed feed restrictions (Roche, 2007; Bryant & Trigg, 1979) or decreased energy content of feed (Norgaard *et al.,* 2005), and is likely to be due to a combination of physiological factors. Feed restriction reduces energy intake and consequently reduces the availability of nutrients for physiological processes. This results in a reduced total cardiac output and a decrease in mammary blood flow due to the reduced proportion of cardiac output allocated to the mammary gland. Feed restriction also reduces mammary mRNA expression of the glucose transporter protein, GLUT-1 and, combined with the decreased blood supply, results in reduced mammary glucose uptake (Guinard-Flament *et al.,* 2007). As glucose is the primary precursor for lactose synthesis, and lactose is the major osmotic agent in milk, a reduction in mammary glucose uptake results in a decrease in milk synthesis.

Two weeks after the treatment period, a carry-over effect was apparent with LPA cows producing 8% less milk, 17% less fat and 11% less protein than the HPA cows. At three weeks post-treatment, LPA cows were producing 6% less milk but by four weeks post-treatment there was no longer any significant carry-over effect on milk yield. However, at this time point, LPA cows still produced 10% less fat and 7% less protein. These differences were still evident six weeks after treatment cessation. By 13 weeks post-treatment, there was a trend for fat to be 7% less in LPA cows with no difference in protein production.

In addition to the systemic and mammary GLUT-1 mRNA expression effects which produce an immediate response in milk production, feed restriction also decreases mammary epithelial cell activity and proliferation (Norgaard *et al.,* 2005; Guinard-Flament *et al.,* 2006). Although there is no documented effect of feed restriction on mammary cell death via apoptosis, reduced dietary energy intake promotes active secretory cells to enter a quiescent non-secretory state from which point they can either be reactivated, or become senescent via apoptosis (Vetharaniam *et al.,* 2003). The net result of a feed restriction is a decrease in mammary cell activity and number, and consequently a decrease in the milk synthesis capability of the gland following a period of energy restriction (Guinard-Flament *et al.,* 2006). A plausible reason for the varying carry-over responses between experiments is the difference in the magnitude of decrease in mammary cell activity and number, caused by the difference in the severity and duration of energy restriction, and the stage of lactation in which the restriction occurs. These factors are likely to affect the ability of the mammary gland to recover when energy intake is increased.

Figure 1 illustrates the immediate and carry-over effects of the acute feed restriction on milksolids (MS) production (fat + protein; kg/cow/d). The total response to the extra energy intake in the HPA cows was 16.2 g MS/MJ ME and consisted of 6.95 g MS/MJ ME immediately and a carry-over response of 9.21 g MS/MJ ME. These data are greater than those of Bryant and Trigg (1979) averaging 8.6 g MS/MJ ME in animals restricted by 23% for a similar period, and are consistent with the 15.1 g MS/MJ ME reported by Roche (2007) in animals restricted by 36% for five-weeks. Although data presented here will enable dairy farmers to make an informed decision regarding supplementary feeding, if faced with an acute feed deficit during peak lactation, further research is required to better understand the physiology regulating the milk production responses and the interaction between the timing, duration, and severity of the feed deficit.

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