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The effects of milking frequency in early lactation on milk yield, mammary cell turnover, and secretory activity in grazing dairy cows

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ABSTRACT

In dairy cows, short-term changes of milking frequency in early lactation have been shown to produce an immediate and a long-term effect on milk yield in stall-fed cows. The effect is controlled locally within mammary glands and could be a function of either secretory mammary epithelial cell number or activity. To resolve this and determine its applicability in other feed management systems, a unilateral milking frequency experiment was conducted with udder halves of 17 multiparous, pasture-fed dairy cows milked either 4 times (4×) or once a day (1×) for 14 d from 5 ± 2 d in milk. Mean half-udder milk yield during the treatment period was higher from the 4× compared with 1× udder halves and continued to be higher until 200 d in milk once returned to twice a day milking. Mammary biopsies were obtained on d 14 of treatment from both udder halves of 10 cows. Proliferation of mammary cells was higher in 4× udder halves compared with 1×, whereas no difference in apoptosis levels was detected. Abundance of α_{S1}-casein, β-casein, α-lactalbumin, and β-lactoglobulin mRNA was higher in tissue samples from 4× udder halves compared with 1×, whereas lactoferrin mRNA abundance was lower in 4× udder halves. In summary, change in milking frequency during early lactation affects proliferation of mammary cells as well as expression of the major milk protein genes, which both contribute to the observed changes in milk yield during and after unilateral milking frequency treatment.
Key words: milking frequency, grazing dairy cow, early lactation, milk yield, cell proliferation

INTRODUCTION

Dairy cows are commonly milked twice a day (**2×**). Increasing milking frequency (**MF**) to 3 (**3×**) or 4 (**4×**) times a day has a dramatic effect on milk yield (**MY**; Amos et al., 1985; Hale et al., 2003). This response is immediate (Hillerton et al., 1990) and has been observed at most stages of lactation (reviewed in Erdman and Varner, 1995), provided sufficient nutrition has been supplied to the animals to accommodate the increased production (Phillips et al., 1980). In addition, short-term increased MF can have a long-term carry-over effect on MY even after the cows have been returned to a normal milking regimen (Bar-Peled et al., 1995; Hale et al., 2003; Wall and McFadden, 2007). This effect is most notable in early lactation, where 2 to 3 wk of increased MF can positively influence MY for the remainder of lactation (Hale et al., 2003; Wall and McFadden, 2007), but has also been demonstrated in midlactation following 8 wk of 3× versus once daily (**1×**) milking (Bernier-Dodier et al., 2010). In most cases, experiments examining increased MF have been conducted in confinement settings, where cows are housed indoors and fed a controlled diet. A few studies have attempted to measure the effects of increased MF during early lactation in grazing dairy cows (McNamara et al., 2008; Phyn et al., 2011). However, in these studies, a treatment of 3× milking for 3 wk yielded a modest increase in MY, but with no long-term effect post-treatment (Phyn et al., 2011) or no effect on MY at all (McNamara et al., 2008).

Similarly, decreasing MF below 2× to 1× has a negative effect on MY (Carruthers et al., 1993; Rémond et al., 1999; Phyn et al., 2011). As well as having an effect on MY during the treatment, if applied in early lactation, a temporary reduction in MF can also illicit a long-term negative carryover effect on MY (Rémond et al., 1999) or the treatment negatively affects levels of milk solids rather than MY (Phyn et al., 2011).

Studies using unilateral MF (**UMF**) models, where udder halves are milked independently at differing MF, have demonstrated that the effects on MY are predominantly controlled locally within the treated mammary gland (Hillerton et al., 1990; Stelwagen and Knight, 1997; Wall and McFadden, 2007). This has been observed for both the immediate effects of increased

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(Hillerton et al., 1990; Wall and McFadden, 2007) and decreased (Stelwagen and Knight, 1997) MF, as well as the carry-over effect associated with temporary increased MF in early lactation (Wall and McFadden, 2007) and midlactation (Bernier-Dodier et al., 2010). Furthermore, the MY response to UMF is equivalent to what has been seen in whole animal MF experiments, which suggest the underlying mechanisms are the same (Wall and McFadden, 2007). The advantage of utilizing a UMF model is that it is within animal and therefore the effects of genetics, nutrition, and environmental factors are largely negated.

The mechanisms underlying the MY response to MF are not well understood, but are most likely a function of secretory mammary epithelial cell (**MEC**) number or activity. To date, studies reporting the effects of MF during early lactation on cell turnover (a factor of the rate of proliferation and apoptosis) have been unclear. Hale et al. (2003), comparing 4× to 2× MF, were unable to detect any significant difference in proliferation. However, a significant increase in apoptosis in samples from 4×-milked glands was observed after 3 d of treatment, but this difference was not detected at a later time point (Hale et al., 2003). Conversely, Nørgaard et al. (2005) saw no difference in proliferation or apoptosis following a 7-d 4× MF compared with 2×. Grala et al. (2011) reported an increase in apoptosis-related genes in mammary samples from cows milked 1× for 3 and 6 wk compared with 2×. However, no proliferation markers were measured to ascertain the effects of 1× milking on combined cell turnover. Bernier-Dodier et al. (2010) detected an increase in both proliferation and apoptosis when 1× was compared with 3× by UMF in midlactation dairy cows, suggesting the decreased MF initiated tissue remodeling. In the current study, a UMF model was used to compare udder halves of grazing dairy cows milked 4× versus 1× for 14 d to investigate how short-term MF changes in early lactation can affect MY both during and after treatment, along with cell turnover and cellular activity in the mammary glands.

MATERIALS AND METHODS

Animals and Treatments

All animal manipulations were conducted in compliance with the rules and guidelines of the Ruakura Animal Ethics Committee. Seventeen multiparous Holstein-Friesian and Holstein-Friesen × Jersey cows were freely grazed on pasture and given access to 2 kg/d of commercial supplement (Topcow dairy, Ingham Feeds & Nutrition, Te Aroha, New Zealand; 12.9 MJ/kg of ME, 12% CP based on DM). Initially the cows were milked 2× and then, from 5 ± 2 to 19 ± 2 DIM, randomly assigned udder halves of all cows were unilaterally milked (1× in 1 udder half at 1100 h, and 4× in the other half at 0500, 1100, 1700, and 2300 h). On the final day of treatment, 3 to 5 h following the 1100-h milking, mammary tissue was obtained by biopsy from both rear quarters of 10 cows, as previously described by Farr et al. (1996). A portion of the mammary tissue was fixed overnight in 4% paraformaldehyde and processed into wax as described previously (Singh et al., 2005). The remainder was snap-frozen in liquid nitrogen for subsequent molecular analysis. The cows were then returned to 2× milking for the remainder of lactation.

Half-Udder MY and Composition Data

Half-udder MY data were measured using a custom-built 2-cup milk claw installed on a commercial rotary milking parlor (Tokanui Research Farm, Waikato, New Zealand), with the yields calculated using an in-line iNTELSCAN Plus milk meter (Milfos International Ltd., Hamilton, New Zealand). Milk yield data were collected daily during the treatment period and monthly following treatment until 200 ± 2 DIM. During the post-treatment period, milk samples were collected and analyzed by infrared spectrometry for fat, protein and lactose, and by flow cytometry for SCC (Fossomatic equipment, LIC Herd Testing Station, Hamilton, New Zealand).

Cell Proliferation

Immunohistochemical localization of the Ki-67 cell proliferation antigen was optimized from the method previously described by Capuco et al. (2001). Slides were deparaffinized, rehydrated, and antigen retrieval was carried out as described previously (McMahon et al., 2004). Slides were then quenched with Dual Endogenous Block (Dako Cytomation California Inc., Carpentaria, CA), washed 3 times in Tris-buffered saline solution (0.05 *M* Tris-HCl, 0.15 *M* NaCl, pH 7.6) containing 0.05% Tween 20 (**TBST**), blocked with Biotin Blocking System (Dako), washed in TBST, and then blocked in 2.5% BSA (Life Technologies, Grand Island, NY) in TBST. Slides were then incubated either with 1:100 anti-Ki-67 rabbit monoclonal antibody (Abcam, Cambridge, UK) diluted in antibody diluent (Stressmarq, Victoria, Canada) or diluent alone as a negative control overnight at 4°C. Slides were washed 3 times in TBST and incubated with a 1:50 biotinylated anti-rabbit secondary antibody (Biogenex, Fremont, CA) in antibody diluent (Stressmarq) for 30 min. After 3 washes in TBST the slides were then incubated with 1:50 horseradish peroxidase (HRP)-conjugated strepavidin (Biogenex) diluted in antibody diluent (Stressmarq) for

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