The effects of once a day milking on mastitis and somatic cell count

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ABSTRACT

A four-year study examined the effect of once a day (OAD) milking for complete lactations on mastitis and milk quality. The study comprised two herds of Jersey cows and two of Friesian cows, milked either OAD or twice a day (TAD) for a total of 4 lactations. Prevalence of intramammary infection (IMI) was determined by isolation of mastitis pathogens from quarter foremilk samples, collected aseptically at calving, mid lactation and at drying off. In the second and third years of the study, at drying off, cows milked OAD had three times as many ($P < 0.01$) quarters with IMI by major mastitis pathogens compared with TAD cows. Across all pathogens and years however, prevalence of IMI was not significantly or consistently higher for cows milked OAD compared with TAD. The somatic cell count (SCC) for uninfected cows milked OAD was approximately double that for TAD milked cows although this difference was only significant ($P < 0.05$) after the first 1-2 months of lactation had elapsed. Cows with minor or major pathogen infections also showed an approximate doubling of the SCC if milked OAD. Results suggest that milking OAD consistently increases the individual cow SCC but does not significantly increase prevalence of mastitis.

Keywords: once daily milking; mastitis; SCC; milk quality; milking frequency.

INTRODUCTION

Farmers contemplating a long-term switch from twice a day (TAD) to once a day (OAD) milking are able to plan for an estimated 15-20% drop in milk yields (Carruthers & Copeman, 1990; Holmes *et al*., 1992). Strategies such as increasing the stocking rate (Harding *et al*., 1990) or selecting cows with a high milk solids content (Davis *et al*., 1999), which is associated with a greater capacity to store milk in the cisternal compartment of the udder, can mitigate some of the milk yield losses associated with OAD milking. But there are conflicting reports of the effects of OAD milking on milk somatic cell count (SCC) and very few studies undertake a thorough examination of the infection status of cows exposed to OAD milking.

Partial lactation studies focus largely on the late lactation period and show mixed effects of OAD milking on SCC. A doubling or tripling of the SCC was reported within 1-2 weeks of switching from TAD to OAD milking (Mackenzie *et al*., 1990; Lynch *et al*., 1991) whilst other studies report no significant increases in SCC after one week (Bushe & Oliver, 1987) or four weeks of OAD milking (Lacy-Hulbert *et al*., 1995). These studies do not report any increase in mastitis incidence for cows milked OAD.

The few whole lactation studies that exist suggest that OAD milking increases individual cow SCC in the order of 1.5-2 fold (Holmes *et al*., 1992; Cooper & Clarke, 2001; Tong *et al*., 2002; Dalley & Bateup, 2004). Long-term studies by Tong *et al.* (2002) and Dalley and Bateup (2004) compare the responses of Friesians and Jerseys to OAD milking but do not report any differences in mastitis between the different herds.

This paper describes the prevalence of mastitis when cows were milked OAD or TAD for complete lactations and the relative consequences of mastitis and OAD milking for milk SCC. The study has been operating for four years, with the primary objective of comparing milk production for Jersey and Friesian herds milked OAD or TAD. Cows were not pre-selected on the basis of low SCC so the study provides a reasonably realistic demonstration of the long-term effects of OAD milking on milk quality. By determining the bacteriological status of individual quarters at regular intervals, the study also allowed the effects of mastitis on milk SCC to be disentangled from that of OAD milking.

MATERIALS AND METHODS

The farmlet study, based at Westpac Whareroa Research Centre, Hawera, comprised two herds of Jersey cows and two herds of Friesian cows that were milked either OAD or TAD for entire lactations. The study commenced in June 2000 and continued until May 2004, providing data across four lactations. Herds were initially balanced for age, calving date, live weight, condition score, breeding worth, and milk production in the preceding season (1999/2000). The OAD herds were stocked at a 17% higher stocking rate compared with the TAD herds in an effort to achieve similar milk solids yield/ha (Cooper & Clark, 2001). The Friesian herds comprised 35 cows milked OAD (FOAD) and 30 cows milked TAD (FTAD) and the Jersey herds, 42 cows milked OAD (JOAD) and 36 cows milked TAD (JTAD). Annual culling was based primarily on pregnancy rate and late calving, with availability of appropriate replacement cows determining discretionary culling based on low milk production, chronic high SCC and/or incurable mastitis.

Milk production was determined on a per-milking basis using an in-line electronic milk yield recording system (Metatron, WestfaliaSurge NZ, Hamilton, New Zealand) and pooled over 24 h to provide a daily milk yield. Individual cow SCC was determined fortnightly on separate am and pm samples using a fluorometric cell counter (Fossomatic, Foss Electric, Hillerød, Denmark).

Single foremilk samples were collected aseptically for bacteriological culture from all quarters of each cow at the first milking after calving, in mid lactation (approx. 100-130 days after calving), and at drying off (approx. 250-300 days after calving). This provided a total of 12 samples per cow and standard microbiological techniques (NMC, 1999) were used to identify the mastitis pathogen present. Pathogens were classified as either a major mastitis pathogen (*Staphylococcus aureus*, *Streptococcus uberis*, *Str. dysgalactiae*, other streptococci, *Escherichia coli* and other Gram negative organisms) or a minor pathogen (coagulase negative staphylococci (CNS) or *Corynebacterium bovis*).

A subclinical mastitis or IMI was diagnosed if samples cultured a monoculture of one or more of these bacteria at > 500 colony forming units/ml (cfu/ml), in the absence of clinical signs. Clinical mastitis (CM) was detected by abnormal changes in the milk and was recorded throughout the season. All cows were teat-sprayed with an iodine-based teat spray (Ecolab® Teatguard Plus) after every milking throughout lactation and all quarters received a tube of long acting, proprietary dry cow antibiotic at drying off.

Prevalence of IMI was determined at calving, in mid lactation and at drying off by the proportion of quarters infected with either a minor pathogen or a major pathogen. The proportion of quarters experiencing such infections, and the proportion of cows that developed CM each year, were analysed on an annual basis for breed and milking frequency effects using generalised linear models (GLM), with binomial error structure (Genstat, 2002).

Individual cow SCC data were $\log_{10}$ transformed before analysis. For each lactation, cows were grouped into one of three groups based on their bacteriological data. Cows with no mastitis detected, i.e. no record of CM, no major pathogens isolated and 10 of the possible 12 quarter samplings free from minor pathogens, were termed UNINFECTED. Similar cows but with three or more positive isolations of minor pathogens were termed INFECTED-MINOR, whilst cows with a recorded case of CM or major pathogen isolated at any sampling, were termed INFECTED-MAJOR. The effects of milking frequency and time since calving on SCC were analysed separately for each year, within an infection group, using linear models (Genstat, 2002).

RESULTS

Complete CM records were available only for the last 2 years of the study and these indicated that milking OAD did not significantly increase the incidence of CM (Table 1). In the third year of the study (2002/2003), the FOAD cows experienced significantly ($P < 0.01$) more cases of CM than the other herds whilst in the last year of the study (2003/2004), Friesian cows developed significantly more ($P < 0.05$) cases of CM than the Jersey herds.

Across the four years of the study, major mastitis pathogens were isolated from approximately 5% of the quarter foremilk samples, collected at calving, mid lactation and drying off. Of the major mastitis pathogens, *S. aureus* was most frequently isolated (58% of cases), followed by *S. uberis* (35%) and then other pathogens such as *S. dysgalactiae* and coliforms (9%). The number of samples from which minor mastitis pathogens were isolated changed over the course of the study, with 32% of quarter foremilk samples yielding minor mastitis pathogens in the first year, 25% in the second, 15% in the third and 7% in the last year. *Corynebacterium bovis* was more frequently isolated in the first three years of the study (67-86% of cases) compared with CNS (14-33%), whilst the ratio was reversed in the final year, when CNS was isolated from 64% of cases and *C. bovis*, 36%.

TABLE 1: Incidence of clinical mastitis (CM), recorded as quarters with CM and cows with CM (total number and proportion of total cows in each herd) during the third and fourth years of a study involving Friesian (F) or Jersey (J) cows milked once a day (OAD) or twice a day (TAD) throughout lactation.

Year		FTAD	FOAD	JTAD	JOAD
2002/2003	Total cows in herd	30	35	36	42
	Quarters with CM	1	11	5	1
	Cows with CM	1	7	2	1
	Cows with CM (%)	3^{a}	20^{b}	6^{a}	2^{a}
2003/2004	Total cows in herd	30	35	36	42
	Quarters with CM	10	9	2	8
	Cows with CM	7	8	2	5
	Cows with CM (%)	23^{a}	23^{a}	6^{b}	12^{b}

[a,b] Proportions with different subscripts differ significantly by $P < 0.05$

TABLE 2: Effects of breed, milking frequency or an interaction (Int.) on prevalence of intramammary infections (proportion of infected quarters), with mean standard error (SE), determined at the start and end of lactation for Friesian (F) or Jersey (J) cows milked once a day (OAD) or twice a day (TAD) throughout lactation.

a. Infections caused by major pathogens (*S. uberis, S. aureus* and coliforms).

	Stage of	Treatment					P values		
Year	lactation	FTAD	FOAD	JTAD	JOAD	SE	Breed	Frequency	Int.
2000-01	calving	0.06	0.07	0.01	0.06	0.02	NS	NS	NS
	drying off	0.01	0.01	0.03	0.07	0.02	*	NS	NS
2001-02	calving	0.01	0.03	0.04	0.01	0.01	NS	NS	*
	drying off	0.03	0.11	0.01	0.05	0.02	*	**	NS
2002-03	calving	0.01	0.02	0.05	0.06	0.01	**	NS	NS
	drying off	0.03	0.10	0.01	0.07	0.02	NS	***	NS
2003-04	calving	0.03	0.06	0.05	0.05	0.02	NS	NS	NS
	drying off	0.05	0.02	0.04	0.05	0.02	NS	NS	NS

b. Infections caused by minor pathogens (coagulase negative staphylococci and *C. bovis*).

	Stage of	Proportion of quarters infected					P values		
Year	lactation	FTAD	FOAD	JTAD	JOAD	SE	Breed	Frequency	Int.
2000-01	calving	0.18	0.34	0.17	0.10	0.06	*	NS	<0.1
	drying off	0.74	0.68	0.37	0.38	0.07	***	NS	NS
2001-02	calving	0.03	0.10	0.04	0.03	0.02	<0.1	NS	*
	drying off	0.68	0.50	0.38	0.28	0.07	***	<0.1	NS
2002-03	calving	0.06	0.09	0.07	0.05	0.02	NS	NS	NS
	drying off	0.40	0.36	0.25	0.18	0.06	**	NS	NS
2003-04	calving	0.04	0.04	0.02	0.03	0.02	NS	NS	NS
	drying off	0.15	0.13	0.13	0.12	0.03	NS	NS	NS

Over the four years of the study, there were mixed effects of milking frequency on the proportion of quarters infected with either major (Table 2a) or minor pathogens (Table 2b). Data are shown only for the beginning and end of lactation since no significant effect of breed or milking frequency was observed for mid-lactation samples. The prevalence of IMI detected at calving was not affected by milking frequency in most years, indicating that pre-existing infections or those acquired at calving were evenly distributed across the different milking frequency treatments.

When sampled at drying off, cows milked OAD in the second and third years of the study were found to have developed significantly more ($P < 0.01$) quarters infected with major pathogens than TAD milked cows (Table 2a). However this effect was not observed in the first or last year of the study. Milking frequency did not significantly affect the number of quarters infected with minor pathogens (Table 2b), although in the second year of the study, cows milked TAD developed slightly more infections ($P = 0.051$) by these pathogens than OAD milked cows.

Breed had a strongly significant effect on the prevalence of IMI, particularly in the first three years of the study when significant effects were observed at calving and at drying off (Table 2a and b). In the first year of the study, Jerseys developed more major pathogen infections by drying off ($P < 0.05$) than Friesians but during the same period, Friesians started lactating with more minor pathogen infections ($P < 0.05$) and developed more of these infections by drying off ($P < 0.001$). During the second year, Friesians developed more major ($P < 0.05$) and minor pathogen infections ($P < 0.001$) than Jerseys whilst in the third year Friesians developed more minor pathogen infections by drying off ($P < 0.01$) than Jerseys. In that year, Jerseys had more major pathogen infections at calving ($P < 0.01$) but this effect resolved by drying off. No breed effects were detected in the last year of the study. It appeared that Friesians were more susceptible than Jerseys at developing new IMI, particularly in the middle years of the study.

Prevalence of IMI due to minor pathogens changed markedly during the study. The number of IMI detected at drying off (Table 2b) declined from year to year, and in the final year (2003/2004), there were insufficient cows available for assignment to the INFECTED-MINOR group (Table 3) for analysis of OAD effects on milk SCC.

For UNINFECTED cows, the geometric mean SCC for each lactation was significantly higher ($P < 0.001$) for OAD milked cows compared with TAD cows, with milking OAD associated with an approximate doubling of the SCC (Table 4). This doubling effect was also observed for INFECTED-MINOR cows whilst INFECTED-MAJOR cows showed a slightly higher average increase in SCC for cows milked OAD.

TABLE 3: Proportion of cows classified as UNINFECTED, INFECTED-MINOR, and INFECTED-MAJOR in each lactation, averaged across the treatment herds.

	Cows per group (%)			
	2000-01	**2001-02**	**2002-03**	**2003-04***
UNINFECTED	28	39	53	66
INFECTED-MINOR	43	41	20	5
INFECTED-MAJOR	29	20	26	28
Cows with complete data (n)	107	116	127	116

* = Insufficient cows for SCC analysis in the INFECTED-MINOR group

TABLE 4: Geometric mean SCC, derived from predicted mean logSCC, determined for cows milked once a day (OAD) or twice a day (TAD) throughout lactation, after assignment to different infection groups. Ratio of OAD:TAD SCC was calculated from back-transformed data.

		SCC (x1000/ml)*			Ratio of
Infection group	**Year**	**TAD**	**OAD**	**P value**	**OAD:TAD**
UNINFECTED	2000-01	42	106	***	2.52
	2001-02	45	99	***	2.21
	2002-03	52	100	***	1.93
	2003-04	64	125	***	1.95
INFECTED-	2000-01	83	135	***	1.62
MINOR*	2001-02	87	191	***	2.19
	2002-03	91	195	***	2.14
INFECTED-	2000-01	123	229	*	1.86
MAJOR	2001-02	105	309	**	2.95
	2002-03	162	389	*	2.40
	2003-04	132	316	**	2.40

* = Insufficient cows for SCC analysis in the INFECTED-MINOR group in 2003/04

Significant interactions ($P < 0.001$) between milking frequency and time since calving were observed for the UNINFECTED cows, indicating that the effect of OAD milking on SCC was not consistent throughout lactation (Figure 1). For each year a significant increase in SCC due to OAD milking was observed only between approximately weeks 6-8 after calving, through until weeks 32, 38 or 42, which was approximately 4 weeks before the end of lactation, depending on the length of each season. For INFECTED-MINOR cows, an interaction between milking frequency and time since calving was observed in 2000/2001 ($P < 0.05$) and in 2001/2002 ($P < 0.001$), when a significant milking frequency effect was observed only during the first 2-4 weeks after calving and from week 10 onwards. For the INFECTED-MAJOR cows, a significant interaction ($P < 0.001$) was observed only in 2001/2002 when milking frequency was significant between weeks 20-40 after calving in that year. For all other years, the SCC for OAD milked cows was significantly higher than for TAD milked cows in each fortnight period.

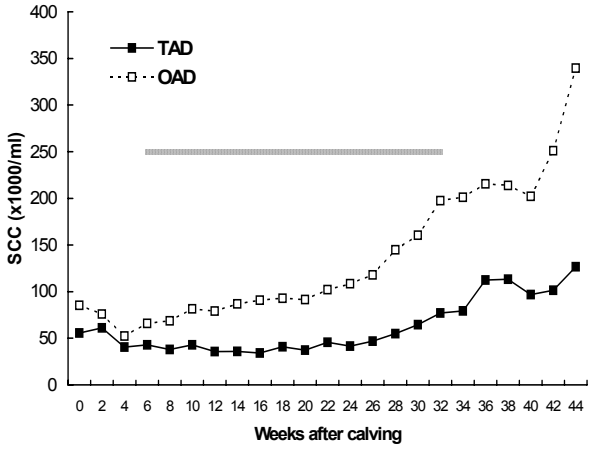
DISCUSSION

The study illustrated that milking cows OAD consistently increases milk SCC but does not have a consistent effect on the risk of cows contracting mastitis. In some years, OAD milked cows developed more new IMI, particularly those caused by major pathogens, but in one year, cows milked TAD appeared to be more at risk of developing minor pathogen infections than OAD milked cows. A significant breed effect, whereby Friesians developed more new IMI than Jerseys, was more often detected than a milking frequency effect and was an unexpected outcome of the study. Further investigations will be required to determine the validity of this result.

Milking once daily has been suggested to aid establishment of new infections and/or increase severity of pre-existing or undetected infections (Mackenzie *et al.,* 1990; Davis *et al*., 1999). Reasons for this effect could be attributed to fewer opportunities to completely milk out the udder, reduced frequency of teat spraying and less frequent opportunities to detect CM.

Conversely, milking cows once daily could provide a mild protective effect, particularly in environments with high levels of contagious mastitis, since fewer milkings would reduce the number of times that teats are exposed to a high bacterial challenge. This may explain the increased prevalence of minor pathogens observed for TAD milked cows in the second year of the study. However the limited CM records, coupled with the relatively small sizes of the treatment herds, provides little indication as to whether OAD milking increases CM. In one year the FOAD cows experienced more CM than the other herds whilst in the following year, both Friesian herds experienced more CM than the Jersey herds.

FIGURE 1: Individual cow somatic cell count (SCC), averaged across the 4 lactations for the uninfected cows when milked once a day (OAD) or twice a day (TAD), with data combined for Friesian and Jersey cows. Horizontal line denotes weeks when a significant ($P < 0.05$) difference in logSCC was observed between OAD and TAD milked cows.



Associations between OAD milking and prevalence of IMI by major or minor pathogens differed from year to year. Infections by different pathogens were pooled to provide sufficient numbers of infections for analysis and the decision was made to group infections on the basis of pathogenicity, hence the distinction between major and minor pathogens. An alternate grouping, on the basis of contagious or environmental sources of the bacteria, was not considered appropriate since most mastitis pathogens can spread contagiously (Zadoks *et al*., 2001), via contact with the milking machine if the prevalence of IMI is sufficiently high.

Results from the second and third years support the concept that OAD milking increases the risk of cows developing new IMI caused by major mastitis pathogens but this effect was not repeated in other years. This lack of conclusive effect would suggest that OAD milking has a relatively minor impact on mastitis susceptibility, when compared with breed-related differences in mastitis susceptibility and variations in effectiveness with which mastitis management strategies are implemented on farm.

Gradual improvements in the implementation of standard mastitis management strategies, especially teat spraying, occurred in the second, third and fourth years of the study and probably account for the decline in prevalence of mastitis by minor pathogens observed during this period. Improving degree of teat coverage with teat sanitiser coupled with appropriate use of dry cow antibiotic therapy, quickly reduces the prevalence of infection by contagious pathogens such as *C. bovis* and *S. aureus* (Eberhart *et al*., 1987; Hillerton *et al*., 1995). Although we observed mixed effects of milking frequency across different years, the changing prevalence of contagious mastitis allowed the effect of OAD milking to be evaluated under both high and low conditions of contagious mastitis.

The effect of OAD milking on milk SCC was more conclusive. Grouping cows by bacteriology provided a reasonable means of distinguishing cows by disease category. Most new IMI tend to develop at calving or near drying off and if no clinical signs are visible, can remain undetected for periods of 1-2 months (McDougall *et al*., 2004) or many months (Hogan *et al*., 1989). By sampling cows on occasions that coincided with times of increased mastitis risk, the chances of detecting new IMI were high. Cows with subclinical mastitis are a major contributor of somatic cells to the bulk tank. Using the bacteriological classification allowed the effects of OAD milking on milk SCC to be separated from that of IMI, however mild these infections could be considered to be.

A similar response in milk SCC was observed for cows in the different disease categories, despite differing proportions of cows present in each category for each year. This agrees with the findings of Holmes *et al*. (1992) who observed a doubling of the SCC for cows on OAD milking for a whole lactation but is in contrast to other reports for low SCC cows milked OAD in late lactation (Kamote *et al*., 1994; Lacy-Hulbert *et al*., 1999). These studies report no significant increases in SCC for low SCC after four weeks of OAD milking in late lactation.

It is possible that low SCC cows only show a response to OAD milking after 2-3 months of OAD milking. In our study UNINFECTED cows showed a significant milking frequency effect after 8-12 weeks into lactation, a time period that coincides with peak lactation. It is possible that the effects of OAD milking on SCC are less visible when cows are in the immediate post calving period and during peak production. Holmes *et al*. (1992) observed a relatively small magnitude of change in SCC (OAD:TAD ratio of 1.5) in the first months of lactation compared with later in lactation (November onwards, OAD:TAD ratio of 2.5-3). Level of nutrition may influence the magnitude of SCC responses to OAD milking. Auldist and Prosser (1998) observed more conspicuous increases in SCC in response to OAD milking, if cows were fed *ad libitum,* compared with a restricted intake diet. Feed deficits often occur in late summer, which coincides with late

lactation, or early spring, which coincides with peak milk production. More detailed comparisons of the effects of OAD milking at peak production versus mid or late lactation may enable the long term consequences of OAD milking on low SCC cows to be determined.

The OAD response for cows infected with major or minor pathogens agrees with the findings of Kamote *et al*. (1994), who observed a SCC response to OAD milking only if cows had a high SCC previously. This presents a problem for SCC thresholds traditionally used to distinguish infected from uninfected cows. In New Zealand, such thresholds are set at 120,000/ml for heifers and 150,000/ml for cows (SAMM, 2004) and it is possible that higher thresholds will be required for cows milked OAD for long periods, particularly in late lactation. However this does not justify a complacent approach to mastitis control since OAD milking is likely to magnify and expose any deficiencies in mastitis management practices.

In conclusion, the study indicated that OAD milking does not significantly increase the prevalence of CM or new IMI, if the general prevalence is low. However OAD milking does tend to approximately double the SCC for individual cows and this increase was consistent across different categories of disease. Maintaining a low prevalence of mastitis and managing the bulk milk SCC proactively are especially important for herds milking OAD in order to meet milk quality requirements.

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