Premilking teat disinfection: Is it worthwhile in pasture-grazed dairy herds?

Article *in* Journal of Dairy Science · October 2014

DOI: 10.3168/jds.2014-8185

Effects of pre-milking management on mastitis and speed of milking in cows in commercial dairy herds

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Abstract

The primary objective of this research was to assess whether a pre-milking management regimen causes a 50% or greater reduction in the incidence, and substantially reduces the hazard, of clinical mastitis in cows in early lactation in commercial dairy herds relative to no pre-milking udder preparation. A secondary research objective was to assess whether this pre-milking management regimen causes a substantial reduction in the incidence of new udder infections in cows in early lactation in commercial dairy herds (as measured by individual cow cell counts) relative to no pre-milking udder preparation.

A parallel controlled trial was conducted in 5 commercial herds; within each herd, cows were allocated to either the pre-milking management regimen or no pre-milking udder preparation. Clinical mastitis and udder infections were the result of natural exposure and not the result of experimental exposure to agents. The trial continued for 59-61 days in each herd, commencing soon after each herd's planned start of calving date for their largest calving group in 2012. Incidences of clinical mastitis and new udder infections (as measured by individual cow cell counts) were compared between treatment and control groups. Severity of clinical mastitis cases and milking characteristics were also compared.

Five herds were enrolled; these were from south-west Victoria (1 herd), Macalister Irrigation Area, Victoria (3 herds), and Taree, NSW (1 herd). In total, 1012 treatment cows and 1013 control cows were used to assess effects of this pre-milking management regimen on clinical mastitis incidence rates and hazards of clinical mastitis.

For four of the five herds, rainfall during the study period was below long-term district average and the cows' teats immediately before milking were generally less dirty than in wetter years.

Overall, there was no evidence of large effects of this pre-milking management regimen on clinical mastitis incidence risk. Over the whole study period, only 7 less cows had clinical mastitis in the improved pre-milking hygiene group (82 versus 89 for the treatment and control groups, respectively), despite similar total cow-days at risk in each group. However, there was evidence of lower clinical mastitis incidence rates and hazards of clinical mastitis in treatment cows in the Taree herd. There was no evidence of large effects on new udder infection rates.

Conclusions

- Our primary hypothesis, that under conditions such as those experienced in the study herds, this pre-milking management regimen reduces clinical mastitis incidence by more than 50%, is rejected. Thus we conclude that it is unlikely that this pre-milking management regimen generally reduces clinical mastitis incidence or new infection rates by more than 50%.
- However, implementation of this pre-milking management regimen reduces clinical mastitis incidence substantially in particular circumstances.
- This pre-milking management regimen increases average milk flow rate and reduces milking time per cow.

Introduction

Over the past 20 to 30 years, pre-milking teat preparation in Australian dairy herds has changed; teats are no longer routinely washed before the cups are applied. While this is now a common approach on Australian and New Zealand dairy farms, it is not common in other countries.

Over the past 10 years, there had been a marked increase in the incidence of Streptococcus uberis clinical mastitis. This organism is regarded primarily as an environmental infection, and is found in paddocks, laneways, feeding areas and tracks, and areas that have been grazed by cows.

Because teats are not routinely washed prior to the cups are being applied, mud and manure are often present on cows' teats when teat cups are applied. This material is likely to contain significant numbers of *Streptococcus*

uberis. While the teats may be dirty when the cups are applied, they are usually clean when the cups are removed, indicating that the action of the milking machine has removed the contaminating material. Because teats are likely to be bathed in milk contaminated with such material, this could possibly lead to an increased risk of infection with Streptococcus uberis. It has been proposed that this risk (and potentially risks of infection by other environmental organisms) may be reduced by pre-milking teat disinfection.

The efficacy of pre-milking teat disinfection has been assessed in studies in USA, France, United Kingdom, New Zealand and Australia. Pre-milking teat disinfection has reduced clinical mastitis incidence by about 50-60% in studies conducted in the USA (Pankey et al, 1987; Oliver et al, 1993; Galton et al, 1998; Oliver et al, 2001) and France (Serieys and Poutrel, 1996). The National Mastitis Council has recently summarised peer-reviewed research on efficacy of pre-milking teat disinfectants (National Mastitis Council, 2013). Two of the products tested under conditions of natural exposure showed significant efficacy against Streptococcus uberis and a further three products against 'environmental pathogens'. However, not all studies in North America have shown a significant benefit (for example, Reugg and Dohoo 1997).

No significant differences were found in field studies in the United Kingdom. Results from a short, 3-herd field study in the United Kingdom (Blowey and Collis 1992) suggested that pre-milking teat disinfection reduced the frequency of mastitis cases but the effect was not statistically significant. Two subsequent studies in the United Kingdom (Hillerton et al, 1993) also failed to show any significant benefit from pre-milking teat disinfection. The authors concluded that 'the effect of trial supervision gave a benefit which overwhelmed any effect of pre-milking teat disinfection'.

A small study in New Zealand by Williamson and Lacy-Hulbert (2013) also did not show any significant positive effects from the application of pre-milking teat spraying in addition to post milking teat spraying. This study was conducted in three herds that had relatively low Streptococcus uberis new infection rates at calving. According to one of the authors, there may be a case for strategically pre-spraying teats of the colostrum mob, especially in Streptococcus uberis problem herds. A field trial conducted in Western Australia by Depiazzi and Bell (2002) was potentially the most relevant study for Australian herds. Clinical mastitis incidence was less in herds that used pre-milking teat sanitation later in the study period but was higher in those herds early in the study period. No statistical analyses were published in this report.

In Australia, the risk of clinical mastitis is high immediately post-calving interval so there was particular interest in assessing whether improved pre-milking teat hygiene reduces the incidence of clinical mastitis during this period. The primary research objective of the current study was to assess whether a pre-milking management regimen causes a 50% or greater reduction in the incidence, and substantially reduces the hazard, of clinical mastitis in cows in early lactation in commercial dairy herds relative to no pre-milking udder preparation. We considered that a 50% or greater reduction in the incidence of clinical mastitis would be required in the commercial situation to offset the additional costs associated with the implementation of a pre-milking teat disinfection program.

A secondary research objective was to assess whether this pre-milking management regimen causes a substantial reduction in the incidence of new udder infections in cows in early lactation in commercial dairy herds (as measured by individual cow cell counts) relative to no pre-milking udder preparation.

Materials and methods

Study overview

A parallel controlled trial was conducted in 5 commercial herds; within each herd, cows were allocated to either the pre-milking management regimen or no pre-milking udder preparation. Clinical mastitis and udder infections were the result of natural exposure and not the result of experimental exposure to agents. The trial continued for 59-61 days in each herd, commencing soon after each herd's planned start of calving date for their largest calving group in 2012. Incidences of clinical mastitis and new udder infections (as measured by individual cow cell counts) were compared between treatment and control groups. Severity of clinical mastitis cases and milking charatceristics were also compared.

Cows were allocated to the pre-milking management regimen or no pre-milking udder preparation based on cow identity numbers; odd and even numbered cows were allocated to different groups.

Treatments

Each cow received the same pre-milking preparation at all milkings during the trial period, depending on her allocated intervention.

Pre-milking management regimen ('treatment') cows:

- With the operator wearing milking gloves, all teats were washed (regardless of degree of dirtiness) with low pressure running water.
- Pre milking teat disinfection (De Laval premilking iodophor) was applied to cow's teats (as per the manufacturer's recommendations) and was allowed a contact time of at least 30 seconds
- All teats were then dried with a disposable paper towel; one towel was used to dry all four teats.
- Teat cups were placed on each udder as per the normal cups-on operation for the herd.

No pre-milking udder preparation ('control') cows:

- These cows received no udder preparation ie cups were applied regardless of degree of dirtiness of teat skin. The milking staff cleaned grossly dirty teats as they considered necessary.

Because treated cows were commonly milked with clusters previously used on control cows, to simulate conditions as if all cows in the herd were treated, teat cups were flushed immediately before being applied to treated cows. Teat cups were flushed with 250 ppm peracetic acid from an Ambic peraspray unit (2 seconds per liner to achieve a minimum volume of 75 mL per cluster). Teat cups were not flushed before being applied to control cows.

Clinical mastitis detection

Herd managers were asked to record quarters meeting the clinical mastitis case definition: Wateriness or clots that persist for more than 3 squirts of foremilk without or with heat, swelling or pain in the udder.

To increase the sensitivity of detection of clinical mastitis, Ambic mastitis filters were placed in every long milk tube. The study design called for these filters to be checked by the cups-off milker at every milking for the duration of the trial.

Milking staff in each herd were asked to collect one aseptic quarter milk sample from each quarter diagnosed with clinical mastitis.

Classification of severity of cases

Milking staff were asked to record the severity of each clinical mastitis case based on the following criteria:

Mild: Wateriness or clots that persisted for more than 3 squirts of milk but no heat, swelling or pain in the udder

Moderate: Wateriness or clots that persisted for more than 3 squirts of milk and one or more of heat, swelling or pain in the udder

Severe: Any of the above plus cow showing systemic signs of disease.

Individual cow cell counts and new infection rates

For herds 1, 2, 4 and 5, individual cow cell counts (ICCCs) were measured using centralised herd test somatic cell count counting services. In herd 3, ICCCs were estimated using permanently-installed somatic cell count monitoring devices (CellSense, DAL Ltd, Hamilton, New Zealand) with milk from individual milkings.

New infection rates were assessed based on ICCCs using methods based on those described by Penry and Mansell (2010) for three periods: from the start of each herd's calving period to the herd's first herd test, from the herd's first to second herd test, and from the herd's second to third herd test. For each period, cows presumed uninfected at the start of the period were used; cows were included only if all previous ICCCs in the current lactation were ≤250 (ie 250,000 cells per ml milk) and either they were heifers or all ICCCs in the previous lactation were ≤250. Of these cows, those whose ICCC at the herd test was >250 were classified as having become infected in the period. The three study herds with at least four herd tests and ICCCs in the previous lactation were used for these analyses (herds 2, 4 and 5). The ICCC data from herd 3 are still to be analysed; these data are not directly comparable to ICCC results from centralised herd test somatic cell count counting services and so different methods for estimating new infection rates are required.

Milking characteristics

Average milk flow rate per cow, milk yield and duration of milking of a small number of cows in herd 5 were measured using a Lactocorder™. The Lactocorder™ is a recording instrument that is inserted between the milking unit and milk pipeline by milk hose. The device was installed in one bail of the dairy for two milking periods and data collected from all cows that used that bail.

Sample size calculations

Sample size calculations were based on effects on clinical mastitis incidence risk at cow-level. Based on prior economic modeling, this pre-milking management regimen would not be profitable unless the clinical case incidence is reduced by over 50%. The study aimed to enroll sufficient lactations so that 1600 lactations (800 in each of the treated and control groups) were available for analysis of clinical mastitis at cow level. This would ensure that the statistical power of the study was almost 80% if the incidence risk of clinical mastitis in this period was 6% and the proportional reductions in clinical mastitis incidence due to pre-milking management is 50%. Although clinical mastitis incidence rates rather than risks were subsequently reported, these sample size calculations provided a valid basis for trial design.

Results

Weather conditions and degree of teat dirtiness during trial

For four of the five herds, rainfall during the trial was less than the long term average for that district. Teats and udders were relatively clean during the trial.

In herd 3, the milking herd spent a short part of each day in a dedicated feeding area, and this resulted in some observed teat contamination based on reports from the milking team. However, the consensus opinion amongst milking staff in herd 3 was that the majority of teats and udders had minimal soiling during the trial period compared with previous seasons when paddocks and tracks are wet.

Bacteriological findings

Streptococcus uberis was the most common mastis pathogen isolated. Substantial proportions of samples yielded no growth in 3 herds. Streptococcus agalactiae was not isolated from any samples, and Streptococcus dygalactiae was isolated from just one sample in one herd. These results are similar to those from clinical mastitis cases from 65 south-eastern Australian herds in 2011/2012 (Charman et al 2012).

Clinical mastitis incidence

Incidence rates of clinical mastitis by herd and treatment group are shown in Table 1. These incidence rates describe the number of cows having clinical mastitis per 100 cow-months of time at risk; by definition, this was equivalent to the number of cows having clinical mastitis per 3,040 cow-days of time at risk. Cow-days of time at risk describe the number of days that a cow is at risk of having her first case of clinical mastitis for the lactation.

Hazard of clinical mastitis was also compared between the treatment and control groups. The hazard of clinical mastitis on a particular day describes the frequency of clinical mastitis on that day in cows not affected prior to that day. The hazard ratio describes the ratio of the hazards across the study period in the treatment cows relative to that in the control cows. If both groups have similar hazards of clinical mastitis across the study period, the hazard ratio will be close to 1. If treatment cows have a higher hazard of clinical mastitis, the hazard ratio will be greater than 1, while if treatment cows have a lower hazard of clinical mastitis, the hazard ratio will be between 0 and 1.

Table 1. Incidence rates of clinical mastitis by herd and treatment group, and hazard ratio for clinical mastitis

Herd	Group	No. cows contributing any time at risk	Total time at risk (cow-days)*	No. cows having at least one case of clinical mastitis**	Incidence rate (no. cows affected per 100 cow-months***)	Hazard ratio (95% CI)****	P
1	Treatment	148	3,620	36	30.2	1.30 (0.80 to 2.08)	0.288
1	Control	161	4,100	31	23.0	Reference group	
2	Treatment	262	10,323	20	5.9	0.82 (0.45 to 1.49)	0.511
2	Control	263	10,264	24	7.1	Reference group	
3	Treatment	85	3,095	7	6.9	0.27 (0.11 to 0.65)	0.003
3	Control	70	2,009	18	27.2	Reference group	
4	Treatment	168	7,209	7	3.0	1.04 (0.36 to 2.94)	0.940
4	Control	181	7,330	7	2.9	Reference group	
5	Treatment	349	15,268	12	2.4	1.25 (0.53 to 2.94)	0.618
5	Control	338	14,282	9	1.9	Reference group	
Pooled	Treatment	1012	39,515	82	6.3	0.90 (0.67 to 1.22)	0.515
Pooled	Control	1013	37,985	89	7.1	Reference group	

* Time at risk commenced on the fourth day after calving or, for cows calved before trial start date, from three days after trial start date. Time at risk ended on the earliest of date of first clinical mastitis case, the day after trial end date (ie the day following the last date at which pre-milking management was applied), dry-off date or date of culling or death.

** No. cows that had at least one clinical mastitis case while at risk

*** Calculated as number of cows affected per 3,040 cow-days at risk

**** Herd was fitted as a fixed effect

Incidence rates of clinical mastitis were generally similar in treatment and control cows except in herd 3 where a lower incidence rate was observed in treatment cows (Table 1). Over all herds pooled, the hazard of clinical mastitis was similar in treatment and control cows (hazard ratio for treatment cows relative to control cows 0.90; Table 1).

The P-value for the interaction between treatment group and herd was low (0.025), providing support for the hypothesis that the effect of this pre-milking management regimen on hazard of clinical mastitis varies by herd. In herd 3, the incidence of clinical mastitis was lower in the treatment group; the hazard ratio for treatment cows relative to control cows was 0.27 (95% CI 0.11 to 0.65; P=0.003). This means that, across the study period in herd 3, the frequency of clinical mastitis in cows not previously affected in treated cows was much less than that in control cows. The hazard in treated cows was estimated to be 27% of that in the control cows, equating to a 73% reduction. In contrast, in the other 4 herds, hazard ratios were close to, and not significantly different from, 1.0.

The reasons for this apparent difference cannot be identified with any certainty from the results of the current study. However several factors may explain this difference: a) herd 3 was the only study herd that utilised a gravel feeding area for the milking herd on a daily basis, b) herd 3 was the highest production herd (33 L/cow per day), and c) herd 3 may have had a higher degree of environmental pathogen challenge compared to the other four herds. Differences in milking management between herd 3 and the other herds may also have contributed to this difference. Alternatively, this difference between herd 3 and the other herds might have been due to chance.

Severity of cases

Severity of cases was compared using odds ratios. Odds of having the condition (in this case being moderate or severe) are calculated as the number that had the condition divided by the number that did not have the condition (in this case being mild). For example, in the 60 affected treated cows whose severity was recorded, 22 cows had moderate or severe mastitis and 38 cows had mild mastitis, so the odds were 22/60 or 0.58. The odds ratio describes the ratio of the odds in the treatment cows relative to that in the control cows. If both groups have similar odds of being moderate or severe, the odds ratio will be close to 1. If treatment cows have higher odds, the odds ratio will be greater than 1, while if treatment cows have lower odds, the odds ratio will be between 0 and 1.

Most cases were of mild or moderate severity. The odds of a case being moderate or severe (rather than mild) did not differ significantly by treatment group but the estimated odds ratio was consistent with reduced severity of cases in treatment cows (estimated odds ratio for treatment cows relative to control cows 0.46, 95% CI 0.19 to 1.10; P=0.081).

Table 2. Incidence rates for individual cow cell counts (ICCCs) >250 amongst presumed previously uninfected cows, by herd and treatment group

Herd	Group	No. cows included*	No. cow-days at risk**	No. cows with ICCC >250 at end of period	Incidence rate (cows affected per 100 cow-months at risk)***	Rate ratio**** (95% CI)	P
Start of herd's calving period to herd's first herd test							
2	Treatment	84	954.5	8	25.5		
2	Control	88	918	10	33.1		
4	Treatment	75	1444.5	7	14.7		
4	Control	75	1206	12	30.2		
5	Treatment	162	2632.5	10	11.5		
5	Control	145	2196.5	8	11.1		
Pooled	Treatment	321	5031.5	25	15.1	0.73 (0.42 to 1.25)	0.254
Pooled	Control	308	4320.5	30	21.1	Reference group	
First to second herd test							
2	Treatment	141	894	12	40.8		
2	Control	149	848	13	46.6		
4	Treatment	79	1368	1	2.2		
4	Control	81	1113	1	2.7		
5	Treatment	240	2552	10	11.9		
5	Control	205	2129	6	8.6		
Pooled	Treatment	460	4814	23	14.5	1.14 (0.62 to 2.08)	0.686
Pooled	Control	435	4090	20	14.9	Reference group	
Second to third herd test							
2	Treatment	133	2769.5	1	1.1		
2	Control	144	2957	5	5.1		
4	Treatment	84	1063	2	5.7		
4	Control	88	1101.5	3	8.3		
5	Treatment	235	3225	7	6.6		
5	Control	217	2908	4	4.2		
Pooled	Treatment	452	7057.5	10	4.3	0.81 (0.35 to 1.85)	0.622
Pooled	Control	449	6966.5	12	5.2	Reference group	

* For each period, cows were included only if all previous ICCCs in the current lactation were ≤250 and either they were heifers or all ICCCs in the previous lactation were ≤250

** For calculation methods, see under 'Individual cow cell counts and new infection rates', above.

*** Calculated as number of cows affected per 3040 cow-days at risk

**** Rate ratio with herd fitted as a fixed effect; herds 1 and 3 could not be included in analyses; only one herd test was conducted in the previous lactation in herd 1 and the ICCC data from herd 3 are still to be analysed; these data are not directly comparable to ICCC results from centralised herd test somatic cell count counting services and so different methods for estimating new infection rates are required.

Table 3. Crude means and standard deviations for milking time per cow and average milk flow rate by treatment group, and estimated differences between means

Group	No. cows	Mean	SD	Estimated difference between means (95% CI)	P
Milking time (minutes)					
Treatment	12	4.75	1.13	-1.06 (-2.16 to 0.03)	0.056
Control	14	5.81	1.51	Reference group	
Average milk flow rate per cow (litres per minute)					
Treatment	12	2.40	0.79	0.53 (-0.02 to 1.08)	0.058
Control	14	1.87	0.56	Reference group	

New infections

Incidence rates of presumed new infections (ICCCs >250 amongst presumed previously uninfected cows) by herd and treatment group are shown in Table 2. Incidence rates did not differ significantly between treatment and control groups for any period.

Milking characteristics

Milking times per cow were quicker for treatment cows, and the average milk flow rates per cow (calculated for each cow as milk volume per milking/milking time) were greater for treatment cows compared with the control cows (Table 3). These results were as expected based on results of previous studies.

Discussion and conclusions

Conclusions:

Our primary hypothesis, that under conditions such as those experienced in the study herds, this pre-milking management regimen reduces clinical mastitis incidence by more than 50%, is rejected. Thus we conclude that it is unlikely that this pre-milking management regimen generally reduces clinical mastitis incidence or new infection rates by more than 50%.

However, implementation of this pre-milking management regimen reduces clinical mastitis incidence substantially in particular circumstances.

This pre-milking management regimen increases average milk flow rate and reduces milking time per cow.

Discussion

In herd 3 the incidence of clinical mastitis was lower in the treatment group, but there was no evidence of large reductions in the other four herds. The reasons for this apparent difference cannot be identified with any certainty from the results of the current study. However several factors may explain this difference: a) herd 3 was the only study herd that utilised a gravel feeding area for the milking herd on a daily basis, b) herd 3 was the highest production herd (33 L/cow per day), and c) herd 3 may have had a higher degree of environmental pathogen challenge compared to the other four herds. Differences in milking management between herd 3 and the other herds may also have contributed to this difference. Alternatively, this difference between herd 3 and the other herds might have been due to chance.

In implementing this pre-milking preparation, herd managers would face the increased milking time and labour costs, and so would expect a substantial reduction in clinical mastitis incidence to justify these costs. Accordingly, this study was designed to detect a significant reduction in clinical mastitis frequency if this pre-milking management regimen reduces clinical mastitis frequency by 50% or more; we assumed that a reduction of less than 50% would not be sufficient benefit for herd managers to implement this pre-milking management regimen.

The majority of Australian farms have high numbers of cows per labour unit, utilise a pasture-based feeding system (increasing time taken moving cows to and from the dairy and so increasing the need for rapid efficient milk harvesting), and are focussed on high labour efficiency. Hence, shifting to a routine of pre-milking preparation during periods of high mastitis risk, such as during calving, needs to be carefully considered due to potential reductions in labour efficiency and consequent increases in operating costs. Results from this study do not support an industry-wide recommendation that Australian herds should all shift to routine pre-milking preparation during calving or early lactation periods. However, from this study, there is evidence that, where environmental mastitis risk is high, this treatment should be considered for limited time periods.

Acknowledgements

We gratefully acknowledge the contribution of the study farmers and farm staff, Daviesway and De Laval, HICO, Werner Schmidt for assistance with Lactocorder™ recordings, and John House and Alison Gunn from the University of Sydney. This study was principally funded by the Geoffrey Gardiner Dairy Foundation Limited. Additional milking staff in two of the study herds were funded by Dairy Australia (Polly Godfrey bequest).

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