

Effects of experimentally induced and treated *Streptococcus uberis* mastitis early in the dry period on production in the subsequent lactation

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ABSTRACT

The effect of experimentally induced clinical mastitis in the early dry period (EDPCM) upon the milk production in the subsequent lactation was examined. Animals with low somatic cell counts at herd test (n=165) were experimentally exposed to *Streptococcus uberis* in their early dry period in an efficacy study of two external teat sealants. Animals that developed EDPCM after challenge (n=127) were treated with an antibiotic after observation of clinical mastitis. Total lactation yields of milk, fat, protein and milk solids were analysed with respect to treatment group (fixed effect) and the covariables: calving week (linear), parity (linear and quadratic) and proportion of Holstein-Friesian genes (linear). For animals that suffered EDPCM and those that did not, there was no difference in production (milk yield; 5126 vs. 5010 litres, fat yield; 267 vs. 264 kg, protein yield; 182 vs. 179 kg). It was concluded that promptly treated EDPCM due to *S. uberis*, did not affect production in the subsequent lactation.

Keywords: mastitis; dry period; cows; production; loss; somatic cell score; *Streptococcus uberis*.

INTRODUCTION

Bovine mastitis is one of the most economically important diseases affecting the dairy cattle industry internationally (Hortet & Seegers, 1998; Seegers *et al.*, 2003). Mastitis was estimated to have cost the New Zealand dairy industry around NZ\$180 million/year in 2005/06 (Anon., 2006). Many factors have been associated with the cost of mastitis, including stage of lactation, pregnancy status, prior yield, mastitis causing organism, severity, diagnosis (early or late after occurrence), treatment and recurrence of mastitis. The main factor (70-80% of all losses) is reduced milk yield. Moreover, as the pathogenesis of mastitis results in irreplaceable loss of secretory tissue, this loss of milk yield can be permanent (Benites *et al.*, 2002).

Short-term depression in milk yield occurs when cows develop mastitis during lactation, with more severe losses occurring if it occurs early in lactation and there is a failure of microbial cure or when the effects of the mastitis carry over into subsequent lactations (Houben *et al.*, 1993; Hortet & Seegers, 1998; Rajala-Schultz *et al.*, 1999). Adverse effects on milk composition (Seegers *et al.*, 2003) increase economic losses. Production effects of clinical mastitis result from both short- and long-term decreases in milk production, particularly associated with chronic mastitis (Smith *et al.*, 1968; Fetrow *et al.*, 1991; Rajala-Schultz *et al.*, 1999).

Less is known about the effects of clinical mastitis during the dry period, on milk production

in subsequent lactations. The dry period is an important part of the lactational cycle during which the mammary gland prepares for next lactation. Clinical mastitis during this period may slow the process of mammary tissue remodelling, thereby adversely affecting milk yield in the subsequent lactation.

Streptococcus uberis is the most significant cause of clinical bovine mastitis in New Zealand and Australia (Pankey *et al.*, 1996; Douglas *et al.*, 2000; Phuektes *et al.*, 2001; McDougall, 2002) where the dairy industry is predominantly pasture based. Information is lacking about the effects of clinical mastitis in the early dry period (EDPCM) on milk production in the subsequent lactation. A previously conducted study with different objectives provided an opportunity to further understand any impact of treated *S. uberis* mastitis on production parameters in the following lactation. In this *post hoc* analysis, the effects of experimentally induced and promptly treated *S. uberis* clinical mastitis early in the dry period on milk production in the subsequent lactation are analysed.

MATERIALS AND METHODS

Animals and experimental design

A total of 175 cows (Holstein-Friesian (HF) and HF-Jersey crossbreds) were selected from the Massey University #4 herd, located in the Manawatu Region of New Zealand to participate in a prospective, randomised, controlled field trial of

the efficacy of two external teat sealants. Ten cows were excluded from analysis in this study due to missing production data, leaving 85 cows (modified DryFlex) and 80 (investigational external teat sealant). The herd was managed at pasture, with supplementary hay/silage as required. Selection criteria for cows in this trial were:

- (i) <200,000 SCC/mL at the April 2005 herd test,
- (ii) four functional quarters and,
- (iii) no clinical signs of mastitis or teat abnormalities at enrolment.

All animal manipulations were approved by Massey University Animal Ethics Committee (MUAEC 04/165).

Challenge protocol

The *S. uberis* strain used for the challenge was initially isolated by Douglas *et al.* (2000) in the Horowhenua district, Wellington region, phenotypically identified as 99.9% *S. uberis*, by means of biochemical test, and kept frozen at -80°C at the Institute of Veterinary Animal and Biomedical Sciences Microbiology Laboratory. All cows were exposed to the challenge broth on two occasions, two and four days after dry off, by dipping of each teat, entirely, for 1-2 secs in a suspension of 1.15×10^8 cfu/mL of the challenge strain.

Milk sampling

Quarter milk samples were aseptically collected (and stored on ice) before morning milking on 4 occasions:

- (i) 4 days before drying off,
- (ii) one day before drying off,
- (iii) the day of calving and,
- (iv) 3-4 days postpartum.

Samples were subjected to routine microbiological culture and examination on the day of collection following NMC recommendations.

Clinical assessment and treatment

All quarters were examined daily by an experienced dairy technician for the presence of mastitis from the time of the first challenge until 29 days later. Individual quarters were observed and palpated for the clinical signs associated with mastitis, *i.e.* heat, swelling, redness, painful quarter/s and if required, by an examination of the secretion. Each quarter was subjectively judged as mastitic or non-mastitic according to the above

criteria. Mastitic quarters were sampled for microbiological culture before treatment was initiated. After sampling each affected quarter was treated as for lactating cow clinical mastitis with penicillin based antibiotics as prescribed on the label.

Statistical analysis

Total yields of milk, fat and protein were estimated from herd-test data during the production season (2005-06), which is the season after the *S. uberis* challenge. Somatic cell score (SCS) was calculated for as natural log (somatic cell count +1) for each herd-test record.

Statistical analyses were performed using SAS (Statistical Analysis System, version 9.1; SAS Institute Inc., Cary, NC, USA). Frequencies of EDPCM between treatment groups were compared using Fisher's exact test of the FREQ procedure.

Total lactation yields of milk, fat, protein and milk solids and average SCS were analysed with the MIXED procedure using a linear model that considered the fixed effects of treatment group (modified DryFlex and investigational external teat sealant), EDPCM occurrence (cows that suffered EDPCM and those that did not), their interaction and the covariables calving week (linear), parity (linear and quadratic) and proportion of Holstein-Friesian genes (linear). Least squares and their standard errors were used for multiple comparisons.

RESULTS

Early dry period mastitis occurrence

After *S. uberis* challenge, 127 of 165 cows developed early dry period clinical mastitis (76.97%). Sixty four of 85 (75.29%) that developed EDPCM were from modified DryFlex and 63 out of 80 (78.75%) from the investigational external teat sealant group. The difference between groups was not significant ($P=0.13$).

Milk production parameters

Milk production data and SCS are given in Table 1. There was no difference in milk yield, fat or protein production in cows that suffered EDPCM and those that did not. There was a statistically significant difference ($p<0.05$) in the SCS observed in cows that suffered EDPCM and those that did not.

There was no difference in the milk production parameters between treatment groups.

Table 1: Least squares means and standard errors of milk production and somatic cell score (SCS) cows affected and not affected with clinical mastitis in the early dry period.

Group	Days in milk	Milk yield (L)	Milk solids yield (kg)	Fat yield (kg)	Protein yield (kg)	SCS
Affected (EDPCM) ¹	277.6 ± 1.4	5126.8 ± 73	449.5 ± 6.5	267.1 ± 4.2	182.4 ± 2.5	4.45 ± 0.07
Unaffected	275.4 ± 2.7	5010.2 ± 135.9	443 ± 12	264.3 ± 7.8	178.6 ± 4.7	4.17 ± 0.12

¹early dry period clinical mastitis

DISCUSSION

The bovine mammary gland is particularly susceptible to new infections early and late in the dry period, due to involution and colostrogenesis respectively (Bradley & Green, 2004). The pathogenesis of mastitis includes, in some cases, damage to secretory tissue and its replacement with fibrous tissue, leading to a permanent decrease in milk yield from the affected quarter (Benites *et al.*, 2002). It is probable that part of the decrease in milk production seen when clinical mastitis occurs during lactation is due to an increased demand for energy by the immune system, a decreased appetite associated with the inflammatory process and lowered feed intake due to pain and decreased mobility (Petrovski *et al.*, 2006). These factors may also influence the normal involution of the bovine mammary gland after drying off. If the normal involution is affected there is a possibility of decreased milk production in the subsequent lactation.

Previous studies reported less than 20% milk yield losses in subsequent lactations for cows affected with clinical mastitis (Fetrow *et al.*, 1991; Houben *et al.*, 1993; Hortet & Seegers 1998). In the present study, no significant effect of induced and treated *S. uberis* mastitis soon after drying off was found in cows upon subsequent lactation yields.

Reasons that the results of the present study differ from earlier investigations are not readily apparent. Possibilities include the organisms involved, time of infection, nature and pathogenesis of the intramammary infection (IMI) and the duration of the clinical mastitis episode. All previous reports were based on the natural occurrence of clinical mastitis during lactation, while in the present study, clinical mastitis was induced by a challenge occurring in the early dry period. In naturally occurring infections the numbers of causative organisms are generally lower than in challenge conditions. The pathogenesis of naturally acquired clinical mastitis is extended and clinical mastitis is not always an outcome. It is possible that a longer duration in such circumstances allows damage to extended areas of the bovine mammary gland. In this experiment, the high numbers of causative organisms in challenge conditions may have easily

overcome the defence mechanisms of the bovine udder, causing an acute clinical mastitis episode. All EDPCM episodes in the present study were promptly diagnosed and treated. Early detection and treatment of clinical mastitis generally result in higher probability of cure than treatment of chronic infections (Milner *et al.*, 1997; du Preez, 2000) as was demonstrated for *Staphylococcus aureus* (Sol *et al.*, 1997). This experiment suggests that promptly treated *S. uberis* clinical mastitis episodes in early lactation do not affect milk production parameters in the following lactation.

In field conditions the infections occurring during the early dry period are more likely to be caused by mixed microbial flora (Bradley & Green, 2004). In the present study, clinical mastitis cases were caused by *S. uberis* only. Some mastitis-causing organisms have been associated with a more profound impact on milk yields than others, for example: Grohn *et al.*, 2004 reported that *Staph. aureus*, *E. coli*, *Klebsiella* spp., and "no pathogen isolated" among primipara, and *Streptococcus* spp., *Staph. aureus*, *A. pyogenes*, *E. coli*, and *Klebsiella* spp., in older cows, caused the greatest losses. Hence, while the present study shows that pure *S. uberis* infections in the early dry period that were identified and treated do not appear to result in permanent changes to lactation yield, it may not reflect the field situation in which mixed infections may be present and where such rapid identification and treatment is unlikely. As expected, the differences between the SCS between cows that suffered EDPCM and those that did not were significant, due to the increased influx of white blood cells post IMI in each of the affected quarters (Rajala-Schultz *et al.*, 1999; Benetes *et al.*, 2002).

CONCLUSIONS

Results of this study indicate that EDPCM due to *S. uberis*, when promptly treated, did not affect production in the subsequent lactation. This is probably because the short duration of the new IMIs did not allow a permanent damage to the mammary secretory tissue to occur. As a majority of new IMIs occur in the first week after calving, it may prove beneficial for farmers to pay more attention to checking for clinical mastitis during the early dry period.

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