

## Prevalence of mastitis for cows of different genotypes milked for two consecutive seasons

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### ABSTRACT

Extending the calving interval from the traditional 12-month to a 24-month calving interval has been suggested as a practical solution for maximizing lactation yield per calving. Somatic cell count (SCC) and the incidence of mastitis were examined in New Zealand (NZ) and overseas (OS) Holstein-Friesian cows fed on pasture alone or supplemented with 3 or 6 kg DM maize/barley concentrates/cow/day during an extended lactation of up to 21 months. Average cow SCC was three-fold higher ( $P < 0.001$ ) in the extended or second season of the lactation compared with the first season, with NZ cows having a slightly higher SCC ( $P < 0.01$ ) in the second season compared to OS cows (191,000 and 115,000 cells/ml respectively). The SCC elevations occurred regardless of infection status of the udder. Although OS cows tended to have twice as many cases of clinical mastitis ( $P = 0.01$ ) in the first season compared to the NZ cows, affecting 59% and 27% of cows respectively, there was no difference in infection status between genotypes in the second season (20% and 18% respectively). Results showed that although cows experienced less mastitis during the second spring of the lactation, the average cow SCC was higher during the extended part of the lactation.

**Keywords:** extended lactation; somatic cell count; clinical mastitis

### INTRODUCTION

Low input, pasture-based dairying, with a calving interval of around 12 months, is the most common milk production system throughout New Zealand (NZ) (Borman *et al.*, 2004). In order for cows to calve annually, they have to be inseminated during the period of peak milk production. At this time cows are often in negative energy balance, which may lead to reduced conception rates (Bertilsson *et al.*, 1997). The possibility of significantly extending lactations beyond the traditional 305 days has been suggested as a practical solution for maximizing lactation yield per calving and improving cow fertility (Bertilsson *et al.*, 1997; Knight, 1997). The period around parturition and early lactation poses the greatest risk to the cow's health and subsequent survival in the herd in terms of metabolic diseases (Correa *et al.*, 1990) and the occurrence of clinical mastitis (McDougall & Compton, 2005). Cows with fewer calvings would not only minimise the health risks and veterinary costs associated with parturition and early lactation (Knight, 2001) but mathematical modelling has shown that maintaining lactation without rebreeding would have a favourable economic outcome (Knight & Mainland, 1995).

Long lactations (e.g. several years) are not yet a realistic option but a number of overseas field trials have provided some evidence of the potential benefits associated with extended lactations. Field trials in Sweden compared 12, 15 or 18-month

calving intervals (Bertilsson *et al.*, 1997; Ratnayake *et al.*, 1998) and observed lower rates of anoestrus associated with extended calving intervals. Studies in Israel observed that extended lactations were more profitable when calving intervals were extended by 60 days (Arbel *et al.*, 2001) whilst studies in the US observed a lower incidence of postpartum metabolic disorders for a 16.5 month calving interval compared with the more typical 13.2 month interval (van Amburgh *et al.*, 1997). Other benefits of extended lactations included: improved conception rates, lower veterinary costs and reduced culling, which led to greater profitability from longer lactations and overall improvements in dairy farm productivity. However, all of these studies were conducted under indoor-housing conditions and involved feeding of concentrates or mixed ration feeds, and in some cases, involved the use of recombinant growth hormone. Moreover, none of the studies reported on the incidence of mastitis and so it is unknown whether udder health benefits can be captured by extending lactations or whether similar productivity benefits can be captured under NZ low-input, pastoral dairying systems with NZ dairy genetics.

The aim of this study was to examine the incidence of clinical mastitis and prevalence of intramammary infections for cows milked for up to 21 months as a result of a calving interval of 24 months. The study was set up to compare the performance of NZ and overseas (OS; North American) Holstein-Friesian animals maintained in a pasture-based dairying system, with or without

supplementary feed. In addition to examining the prevalence of mastitis during an extended lactation, we analysed the changes in individual cow somatic cell count (SCC) that occurred during the latter part of the extended lactation and any differences associated with genotype.

## MATERIALS AND METHODS

### Animals and Trial Design

The main emphasis of the study was to compare the milk production performance of 30 NZ and 30 OS Holstein-Friesian animals (of mixed parity) maintained on three dietary treatments, for an extended lactation of up to 670 days. Ten cows of each genotype were fed on pasture alone at a comparative stocking rate of 80 kg liveweight/tDM or offered either 3 or 6 kg DM/cow/day of supplementary feed in the form of a commercial concentrate (92% maize and barley grain, 6% molasses and 2% broll) at a flat rate throughout lactation (twice daily during milking). Cows began calving around the 24<sup>th</sup> of June 2003 and were milked through until the 6<sup>th</sup> of May 2005. For further details on the trial design refer to Kolver *et al.* (2006).

### Measurements

Prevalence of intramammary infection was assessed regularly by aseptically sampling individual quarters of all cows for bacteriological analysis on 8 occasions (approximately every 3 months), from calving through to dry off. Cases of clinical mastitis were recorded and sampled for bacteriology purposes at detection, before treatment with a course of lactating cow antibiotic. All cows were teat sprayed with an iodine-based sanitiser after every milking. The SCC was determined on a cow-composite basis fortnightly throughout the extended lactation using a Fossomatic automated cell counter (Foss Electric, DK-3400, Hillerød, Denmark).

Details of the production measurements taken and specific analyses performed during the extended lactation are described by Kolver *et al.* (2006).

### Bacteriological Procedures

Standard mastitis laboratory techniques as detailed by the National Mastitis Council (Harmon *et al.*, 1990) were used for collection and analysis of the quarter foremilk samples. Teat ends were scrubbed with cotton wool swabs soaked in 70% alcohol and allowed to dry. The first 2-3 squirts of milk were discarded and then approximately 20 ml was drawn into a sterile container. For each quarter a sub-sample of 10 µl of milk was streaked

onto one quadrant of a tryptose blood esculin agar plate and incubated for 48 hours at 37°C. Presumptive identification of isolates was made primarily on the basis of colony morphology, Gram stain, and esculin, catalase and coagulase reactions according to National Mastitis Council recommendations, with other biochemical tests used to confirm streptococcal species.

### Statistical Analysis

Lactation data recorded from June 2003 through to mid May 2004 was referred to as the "first" season of the trial whilst data recorded from May 2004 through to April 2005 was referred to as the "second" season. Data collected from 2003 through to 2005 was referred to as the entire lactation. Individual cow SCC data were log<sub>10</sub> transformed prior to analysis.

Days in milk (DIM), milk solids yield, and mean log<sub>10</sub> SCC for each season and the entire extended lactation were calculated for each cow and tested for treatment effects (genotype and diet) using Residual Maximum Likelihood (REML, Genstat, 2002) with genotype, diet and interaction of genotype and diet as fixed effects and cow as a random effect. Seasonal differences were tested by analysing log<sub>10</sub> SCC data across comparable times of the seasons i.e. August to April, in a repeated measures analysis, using REML with season, genotype, diet and their interactions as fixed effects and cow and season within cow as random effects. To examine DIM-related changes in SCC, the SCC data of animals classified as uninfected in either the first or second seasons, using very strict criteria, were analysed for genotype and diet effects using the same REML model. Uninfected animals were deemed to be those that remained free of clinical mastitis during a season and had only one or two quarters infected with a minor pathogen (coagulase negative staphylococci (CNS) or *Corynebacterium bovis*) during the same season.

Incidence of clinical mastitis was reported as the proportion of cows with one or more cases of clinical mastitis during the first or second seasons of the extended lactation. Prevalence of intramammary infection was determined by the proportion of quarters infected with mastitis pathogens at each of the regular bacteriological samplings. Infections were also grouped according to whether the pathogen was classified as an environmental or contagious pathogen, with environmental pathogens comprising *Streptococcus uberis*, CNS, *Escherichia coli* and other Gram-negative pathogens, whilst the contagious pathogens comprised *Staphylococcus aureus* and *C. bovis*. The effects of genotype and diet on incidence of clinical mastitis and prevalence of

intramammary infections were analysed using generalised linear models with binomial error structure (Genstat, 2002).

## RESULTS

All but two cows achieved 500 DIM, with cows being progressively dried off on production from 550 days to 650 days. Of the original 60 cows on trial, a total of 56 cows completed an average extended lactation length of 605 days, with OS cows achieving 12% more DIM than NZ cows in the second season ( $P < 0.05$ ; Table 1). Overseas genetics were more suited to an extended lactation, reaching 95% of the MS production usually achieved in two 305-day lactations whilst the NZ cows only achieved around 80% (Table 1). For further performance data, including the effect of diet on DIM and MS yield, refer to Kolver *et al.* (2006).

**TABLE 1:** Days in milk and milksolids yield (kg MS/cow) for New Zealand (NZ) and overseas (OS) genotypes during the first (June 03 – May 04) and second (May 04 – May 05) season, and entire lactation (June 03 – May 05).

Genotype	n	Days in milk			Milksolids (kg MS/cow)		
		First	Second	Entire	First	Second	Entire
NZ	29	299	291	590	523	300	824
OS	27	293	327	620	558	499	1057
SED <sup>1</sup>		7.10	16.70	15.67	23	43	55
P values		0.456	0.028	0.091	0.15	<0.001	<0.001

<sup>1</sup> SED = standard error of the difference between genotype.

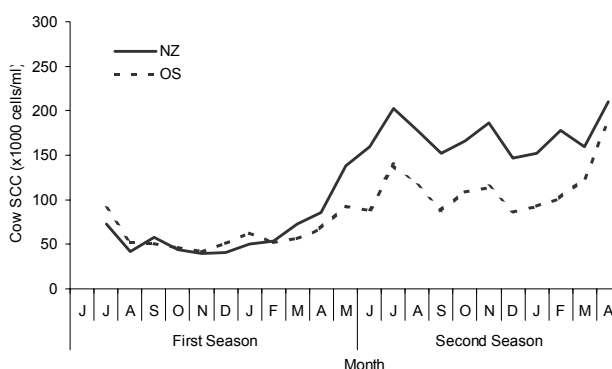
In the first season and over the entire lactation, there was no significant difference in cow SCC between genotypes or dietary treatments for all cows. However, in the second season, NZ cows had a significantly higher  $\log_{10}$  SCC (5.28) compared with the OS cows (5.06,  $P = 0.03$ , Table 2). When the influence of mastitis was removed from the analysis, by focusing only on the uninfected cows, the NZ cows were found to have a significantly higher ( $P = 0.04$ )  $\log_{10}$  SCC than the OS cows for the entire lactation, with the difference between genotypes more marked in the second season (5.21 and 5.02  $\log_{10}$  SCC respectively,  $P = 0.03$ ). Differences between genotypes, and the steady increase in SCC in the second season are clearly illustrated in a comparison of the geometric mean cow SCC, determined on a monthly basis for the two genotypes (Figure 1). Dietary effects on cow SCC were not observed during the study, although the limited number of animals on trial may have prevented expression of such effects.

**TABLE 2:**  $\log_{10}$  somatic cell count (SCC) of New Zealand (NZ) and overseas (OS) genotypes, averaged for all cows, during the first (Aug 03 – Apr 04) and second (Aug 04 – Apr 05) seasons of the extended lactation.

Genotype	$\log_{10}$ SCC		SED
	First season	Second season	
NZ	4.74	5.28	0.08
OS	4.75	5.06	0.08
SED <sup>1</sup>	0.11	0.11	
<b>P values</b>			
Strain	0.266		
Season	<0.001		
Interaction	0.032		

<sup>1</sup> SED = Standard error of the difference, provided for within-strain and within-season comparisons.

**FIGURE 1:** Geometric mean of the individual cow SCC, averaged on a monthly basis, for OS and NZ genotype cows during the entire extended lactation.



The incidence of clinical mastitis was determined from the proportion of cows that experienced one or more cases of clinical mastitis during a single season. In the first season OS cows had more cases of clinical mastitis compared to NZ cows (59% and 27% of cows respectively,  $P = 0.01$ ) but there was no difference between genotypes in the second season (Table 3).

**TABLE 3:** Proportion (%) of New Zealand (NZ) and overseas (OS) genotype cows that experienced one or more cases of clinical mastitis during the first and second seasons of the extended lactation.

Genotype	n	% clinical cows/season	
		First season	Second season
NZ	29	27	20
OS	27	59	18
SED <sup>1</sup>		11	10
P values		0.01	0.864

<sup>1</sup> SED = Standard error of the difference.

The prevalence of intramammary infection varied between genotypes at the first sampling after calving but then remained similar across genotypes for the rest of the extended lactation. The OS cows had a higher proportion of quarters infected at the first sampling after calving compared to the NZ cows (22% and 9% respectively,  $P=0.008$ ), with environmental pathogens, primarily *S. uberis* and CNS, the main pathogens isolated (16% and 7% of quarters of OS and NZ cows respectively,  $P=0.02$ ). Prevalence of infection tended to reduce at the next sampling, approximately three months later, averaging 6% of quarters across both genotypes and remained at or below this level for the rest of the extended lactation. The proportion of quarters infected with contagious pathogens, mainly *S. aureus*, was similar between the two genotypes after calving (5% and 3% for OS and NZ cows respectively,  $P=NS$ ), and remained below 4% of quarters for the entire lactation. The prevalence of infection by environmental pathogens remained at or below 3% of quarters across all treatments for the rest of lactation, with the OS cows experiencing slightly more environmental infections than the NZ cows (3% vs 0% respectively,  $P=0.036$ ) at the July 2004 sampling, at the start of the second season.

## DISCUSSION

The study examined the changes in mastitis associated with extending lactation through a second period of 12 months for cows of OS and NZ genotype. Of particular interest were the changes in incidence of clinical mastitis, prevalence of intramammary infection and cow SCC that occurred during the second season of the extended lactation.

We observed that the OS animals had a higher clinical mastitis incidence than the NZ cows in the first season, affecting 59% of cows but the reasons for this are not known. This coincided with a higher prevalence of infection detected after calving for these cows, caused primarily by environmental pathogens such as *S. uberis*. In a previous lactation (2002/2003) the incidence of clinical mastitis was 23% for these cows but the small number of animals in each treatment group precludes further analysis. However, in the second season, the incidence of clinical mastitis was similar between genotypes (18-20%) and was comparable to other studies. Clinical rates of 14 cows/100 cows/annum have been reported for NZ cows (McDougall & Compton, 2005) typically associated with calving, whilst rates of 17-31% have been reported for Jersey and Holstein-Friesian cows respectively, maintained on pasture in the US (Washburn *et al.*, 2002). The data indicated that

some cases of mastitis can still be expected in the spring months of the second season of an extended lactation but a larger study would be required to determine the typical incidence at this time.

We observed that the cow SCC increased significantly in the second season of lactation for both NZ and OS animals, an effect that was also observed among cows that were classified as uninfected, using very strict criteria. The average cow SCC tended to be 2-3 times as high during the second season compared to the first, with the NZ cows tending to show a more marked rise in SCC.

Cows typically show a higher SCC in late lactation (Lacy-Hulbert *et al.*, 1999), associated with a steady decline in milk volumes, and gradual involution that occurs during the declining phase of lactation (Hurley, 1989). Reduced milk volumes, induced by involution or feed restriction, tends to result in a reduced yield of mammary gland-synthesised components, such as casein proteins (Petch *et al.*, 1997), and an increased concentration of serum-derived components, such as whey proteins and somatic cells (Lacy-Hulbert *et al.*, 1999). It is likely that the SCC increases observed here were attributed to the effect of declining milk volumes. For the OS animals, the SCC rises observed in the second season were less than for the NZ animals, which coincided with a lower reduction in MS yield associated with the second season (Kolver *et al.*, 2006). Overseas Holstein-Friesian animals have been progressively bred for high milk production and may be more persistent than NZ animals, although no difference in persistency has been observed between these genotypes when milked for a typical 305-day lactation (Davis *et al.*, 2000).

The increase in SCC observed in the second season has not been previously reported. Studies in Sweden by Osterman *et al.* (2005), using dairy cows under housed conditions, observed that SCC did not differ for 18-month calving intervals compared to the traditional 12-month calving interval. In the current pasture-based study, where lactation was extended to 21 months, a decline in pasture quality during the summer months, coupled with early drying off of some animals due to low production, may have influenced SCC rises in the extended part of lactation. Comparison of the SCC data with cows managed in a similar environment but milked for a maximum of 305-days could provide a clearer insight as to the reason behind this SCC rise after 12 months of lactation.

In conclusion, the SCC of cows on extended lactation increased with increasing duration of lactation. It was found that the SCC of NZ cows increased more than that of OS cows but the average cow SCC was still well below SCC

penalty levels. The incidence of clinical mastitis in the extended part of lactation was similar between genotypes, but did not decrease dramatically as may have been expected due to the absence of calving. Therefore, results from this investigation into extending lactations from a 12 to a 24-month calving interval suggest that although there may be similar or reduced levels of clinical mastitis in the second spring of the lactation, a time of year commonly associated with mastitis in NZ, the SCC will tend to elevate during the second season of the extended lactation, irrespective of genotype.

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