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Incidence of mastitis among cows of different genotypes in differing nutritional environments

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ABSTRACT

Over three seasons (1998, 1999 and 2000) groups of Holstein-Friesian cows of New Zealand (NZ) or overseas (OS) origin were monitored for mastitis when managed in two contrasting systems. Cows were fed either on pasture or total mixed ration (TMR) with cows on TMR confined to small loafing paddocks or a concrete feedpad. All quarters were sampled for bacteriology at calving, at dry off, in mid and late lactation and when clinical mastitis was detected. Individual cow somatic cell counts (SCC) were determined weekly throughout lactation. In 1998, there was no difference in incidence of clinical mastitis but in later seasons, cows on TMR experienced significantly more clinical, and sub-clinical mastitis, than cows on pasture, probably reflecting differences in bacterial challenge within the environment. Genotype differences were less evident compared to dietary effects. Compared to NZ cows, OS cows experienced more sub-clinical infections in 1998 and more clinical infections in 1999 whilst in 2000, genotype effects were confounded by diet. Seasonal SCC was higher for OS cows in 1999 and 2000, reflecting differences in incidence of sub-clinical mastitis among treatment groups. Despite the strong influence of environment on mastitis incidence, genotype differences were detectable between NZ and OS cows.

Keywords: mastitis; genotype; diet; Holstein-Friesian.

INTRODUCTION

The increasing proportion of North American and Dutch Holstein-Friesian (HF) genetics within the New Zealand dairy herd over the past 10-15 years has raised important questions regarding the relative performance of these genotypes within a pastoral grazing system. Previous studies have focused on milk and milk-solids production of these genotypes (Kolver et al., 2000; Harris & Kolver, 2001; Kolver, 2001) or their relative reproductive performance within a pasture-based system (Verkerk et al., 2000), but other health performance parameters, such as susceptibility to mastitis or lameness, may also impact on their profitability and adoption into a pastoral system.

Mastitis is the most costly animal health disease on NZ dairy farms. In 1992, total costs attributable to mastitis, including milk losses, were estimated at $14,000 per annum per herd (Holdaway, 1992a) and prevention and treatment costs alone represent 15-20% of the animal health costs on farm (Hainsworth, 1998). Mastitis is an inflammation of the mammary gland usually in response to a bacterial infection of the udder tissue. Numerous bacteria are capable of infecting the mammary gland, derived either from the cow’s own environment or from other infected glands. In New Zealand, mastitis is usually caused by Gram positive bacteria such as Streptococcus uberis, Staphylococcus aureus, coagulase negative staphylococci (CNS) or Corynebacterium bovis (Brookbanks, 1966; Pankey et al., 1982; McDougall, 1998). Although these pathogens are problematic in European or North American systems, mastitis in these countries is more commonly associated with Gram negative bacteria, such as Escherichia coli, and Klebsiella species (Schukken et al., 1991; Pankey, 1997). Infections by these pathogens are usually of rapid onset and short duration but can elicit systemic reactions, resulting in dramatic milk yield losses and sometimes the death of the cow (Eberhart et al., 1987).

The differences between countries, in terms of mastitis prevalence or cause, are likely due to a combination of nutritional and environmental factors. Confinement housing, traditionally used in the US, with cows fed a Total Mixed Ration (TMR) for up to 12 months of the year, is often associated with a high incidence of coliform mastitis (Eberhart et al., 1987), whilst pasture-based systems in NZ and Australia are more often associated with streptococcal mastitis (Pankey, 1997). Generally, it is observed that while higher milk production levels are a known risk factor for mastitis, determining the genetic influence on mastitis susceptibility is difficult when cows are managed in widely differing systems.

The Dexcel study, comparing the production performance of NZ and overseas (OS) HF genotypes in either a pasture-based or TMR feeding system, provides an ideal opportunity to compare the relative susceptibility of different genotypes to mastitis, when exposed to pasture and TMR management environments.

MATERIALS AND METHODS

Design

For the past three seasons (1998-1999, 1999-2000 and 2000-2001) groups of HF cows of either NZ or OS genetic origin have been managed in two contrasting feeding systems. Details of the management during the first year of the trial have been described previously (Kolver et al., 2000) and nutrition management systems have been similar in subsequent years of the study (Kolver, 2001). Cows were managed in groups in either an all-pasture, ryegrass/white clover system (GRASS) or on a TMR diet, comprising maize silage, grass silage, and concentrates (Kolver et al., 2000). Cows in the GRASS system were offered generous pasture allocations and pasture silage was used to supplement the diet during the dry summer months. Cows in the TMR system were confined to one of three loafing paddocks (0.25 ha/paddock) or to a concrete and free-draining feed pad. This feed pad was
used during July, August and September and during years 1 and 2, sand was used as the free-draining bedding material, and bark and post peelings in year 3.

Animals

In the first year of the study, all animals were primiparous with a smaller but equal proportion of each herd maintained as first lactation animals during later years (Table 1). Only 19 cows of OS origin were available for the first year of the trial but in later years this number increased. Cows of NZ origin were selected on the basis of genetic merit and had a similar breeding worth (BW) or genetic ranking to the OS cows. Heifers entering the herd each year were balanced for breeding worth (BW) and age. Once assigned to a nutritional treatment, individual animals generally remained in the same group from year to year.

Mastitis monitoring

Single foremilk samples were collected aseptically from quarters for bacteriological culture at the first milking after calving, before treatment for clinical mastitis, at drying off and on two other occasions during the lactation. These occasions coincided with peak-mid lactation (October/November) and mid-late lactation (January/February). All cows were checked daily for clinical signs of mastitis during the colostrum period and at weekly intervals during the rest of the season. Any clinical infections were sampled for bacteriology and treated with a course of lactating cow antibiotic. All cows were teat-sprayed with an approved iodine-based sanitiser after every milking and all quarters were infused with dry cow antibiotics at the end of lactation.

Somatic cell counts (SCC) were determined on a cow-composite basis weekly throughout lactation and on the quarter foremilk samples collected for bacteriology during lactation and at drying off. Samples collected near calving were not submitted for SCC due to contamination with colostrum. Milk SCC samples were submitted to LIC laboratories for analysis by a Fossomatic automated cell counter (Foss Electric, DK-3400, Hilleroed, Denmark).

Bacteriological procedures

Quarter foremilk samples were collected aseptically and analysed using standard mastitis laboratory techniques (NMC, 1999). Teat ends were first scrubbed with cottonwool swabs soaked in 70% 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 sample a sub-sample of 0.01 ml of milk was plated onto one quadrant of a tryptose blood agar plate, containing whole bovine blood (50 ml/l) and esculin (1g/l), and incubated at 37°C for 48 h before examination. Presumptive identification of isolates was based on colony morphology, haemolysis, esculin reaction, Gram stain, catalase production and tube coagulase reaction. Confirmatory identification of streptococcal isolates was carried out using the CAMP test, inulin reaction, growth in 6.5% salt broth and hydrolysis of sodium hippurate. Staphylococcal isolates were classified on the basis of haemolysis and tube coagulase reaction as either S. aureus or CNS. Gram negative organisms were classified on the basis of ONPG, indole, citrate, oxidase, motility, O/F and TSI reactions.

Diagnosis of Intramammary Infection

Clinical mastitis was diagnosed by presence of clinical signs such as clots in the milk, discoloured secretion, heat, swelling or pain in the udder or fever in the cow. All routinely collected samples that cultured a mono-culture of bacteria of >500 cfu/ml, in association with a quarter foremilk SCC >150,000 cells/ml, were classified as sub-clinical intramammary infections.

Statistical analysis

Results are reported as proportion of cows or quarters within a group that developed a clinical or sub-clinical infection during a particular season. This equated to the proportion of susceptible cows or quarters within a treatment group and eliminated the problem of repeated clinical episodes and isolations during routine sampling. For analysis of the incidence, cows or quarters were counted once only in a single season. Analysis of genotype and diet effect was by generalised linear model, with binomial error structure (Genstat, 2000). To evaluate treatment effect on causal pathogens, repeated clinical cases or isolations occurring in the same quarter were counted as two separate events, as long as the second pathogen differed from the first. Data were pooled across seasons to generate sufficient numbers and analysed by Chi-square analysis. Weekly individual cow SCC data were first log transformed, averaged for each cow over a particular season, and then analysed for genotype and diet effects using ANOVA (Genstat, 2000).

RESULTS

Clinical Mastitis

In the 1998 season, the proportion of cows that developed clinical mastitis was similar in each group, ranging from 20-40% (Table 2). In the second and third year of the study however, cows on TMR experienced a dramatic increase in the level of infection compared to

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<p>| TABLE 1: Number of cows and quarters, excluding blind quarters, for each treatment group of a genotype by nutritional environment comparison. |</p>
<table>
<thead>
<tr>
<th>Year</th>
<th>1998/99</th>
<th>1999/00</th>
<th>2000/01</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment¹</td>
<td>1998/99</td>
<td>1999/00</td>
<td>2000/01</td>
</tr>
<tr>
<td>NZ GRASS</td>
<td>14 (14)²</td>
<td>14 (5)</td>
<td>14 (3)</td>
</tr>
<tr>
<td>NZ TMR</td>
<td>15 (15)</td>
<td>14 (5)</td>
<td>14 (3)</td>
</tr>
<tr>
<td>OS GRASS</td>
<td>9 (9)</td>
<td>13 (5)</td>
<td>14 (3)</td>
</tr>
<tr>
<td>OS TMR</td>
<td>10 (10)</td>
<td>14 (5)</td>
<td>14 (3)</td>
</tr>
</tbody>
</table>

¹ NZ = New Zealand Holstein-Friesian; OS = Holstein-Friesian of overseas origin; GRASS = pasture diet; TMR = total mixed ration diet.
² Number in brackets refers to number of first lactation cows.
the first year (Table 2), and therefore, experienced a significantly (P<0.001) higher incidence of clinical mastitis compared with cows on GRASS. No genotype effects were observed for the proportion of cows that developed clinical mastitis.

When analysed on a quarter basis, no differences between groups were observed in the first season, with the infection rate ranging from 5-13% quarters per group. In the second year the effect of genotype and diet proved to be significant. More quarters of cows on TMR developed clinical mastitis compared with cows on GRASS (P<0.001; Table 2), and more quarters of OS cows developed clinical mastitis compared to NZ cows (P<0.05). During the third year, cows on TMR continued to have a higher incidence of clinical quarters compared to GRASS cows (P<0.001) but a particularly high clinical infection rate in the OS GRASS cows resulted in a significant interaction effect (P<0.01). Most of the clinical mastitis cases were detected during lactation. Only two clinical cases were detected in the dry period, and two at the first milking after calving, over the three years of the study.

Sub-clinical Mastitis

In year one, the proportion of quarters that were sub-clinically infected at routine samplings (Table 2) was significantly (P<0.05) higher for OS cows compared to NZ cows, with a similar but diminished effect observed in year two (P=0.065). In year two, diet showed a more significant effect than genotype, with more sub-clinically infected quarters observed for cows on TMR compared to GRASS. In year three, a highly significant (P<0.001) interaction was observed that practically negated the diet effect (P=0.061). In that year, the NZ cows on TMR showed the highest incidence of sub-clinically infected quarters (30%), followed by OS cows on GRASS (21%), OS cows on TMR (13%) and NZ cows on GRASS (4%).

Bacterial isolates

The frequency and type of pathogens isolated from clinical mastitis cases differed between the two dietary treatments but a similar pattern was observed for both genotypes (Table 3). For cows on TMR, over 60% of the clinical episodes were caused by coliform bacteria whilst for cows on GRASS, the majority of clinical episodes

### Table 2: Proportion (%) of total cows, or quarters that developed mastitis during each season, with maximum standard error (SE) and P values for each group of a genotype (NZ HF or OS HF) by nutritional environment (GRASS or TMR) comparison. Cows or quarters were counted once only in a particular season.

#### a. Clinical Mastitis

<table>
<thead>
<tr>
<th>Season</th>
<th>NZ</th>
<th>OS</th>
<th>Genotype</th>
<th>SE</th>
<th>P&lt;Genotype</th>
<th>P&lt;Genotype</th>
<th>P&lt;Genotype</th>
<th>GxO</th>
<th>P&lt;Genotype</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cows 1998</td>
<td>GRASS 28.6</td>
<td>TMR 40.0</td>
<td>22.2</td>
<td>20.0</td>
<td>13.8</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Cows 1999</td>
<td>7.1</td>
<td>57.1</td>
<td>15.4</td>
<td>64.3</td>
<td>13.2</td>
<td>NS</td>
<td>.001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Cows 2000</td>
<td>7.1</td>
<td>64.3</td>
<td>38.5</td>
<td>71.4</td>
<td>13.4</td>
<td>NS</td>
<td>.001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Quarters 1998</td>
<td>7.1</td>
<td>13.3</td>
<td>5.6</td>
<td>5.0</td>
<td>4.4</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Quarters 1999</td>
<td>1.8</td>
<td>14.3</td>
<td>3.9</td>
<td>28.6</td>
<td>6.0</td>
<td>.05</td>
<td>.001</td>
<td>NS</td>
<td>.01</td>
</tr>
<tr>
<td>Quarters 2000</td>
<td>1.8</td>
<td>39.3</td>
<td>13.5</td>
<td>30.9</td>
<td>6.5</td>
<td>NS</td>
<td>.001</td>
<td>.01</td>
<td>NS</td>
</tr>
</tbody>
</table>

#### b. Sub-clinical Mastitis

<table>
<thead>
<tr>
<th>Season</th>
<th>NZ</th>
<th>OS</th>
<th>Genotype</th>
<th>SE</th>
<th>P&lt;Genotype</th>
<th>P&lt;Genotype</th>
<th>P&lt;Genotype</th>
<th>GxO</th>
<th>P&lt;Genotype</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quarters 1998</td>
<td>GRASS 16.1</td>
<td>TMR 15.0</td>
<td>36.1</td>
<td>25.0</td>
<td>8.0</td>
<td>.05</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Quarters 1999</td>
<td>3.6</td>
<td>14.3</td>
<td>13.5</td>
<td>23.2</td>
<td>5.6</td>
<td>.1</td>
<td>.05</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Quarters 2000</td>
<td>5.5</td>
<td>28.6</td>
<td>21.1</td>
<td>16.4</td>
<td>6.1</td>
<td>NS</td>
<td>.1</td>
<td>.001</td>
<td>NS</td>
</tr>
</tbody>
</table>

### Table 3: Type of pathogen isolated from clinical mastitis samples or routine samples (sub-clinical infections) as a percent of total isolates, pooled across three seasons, for each group of a genotype (NZ HF or OS HF) by nutritional environment (GRASS or TMR) comparison.

<table>
<thead>
<tr>
<th>C. bovis &amp; CNS</th>
<th>S. aureus</th>
<th>S. uberis</th>
<th>Coliforms</th>
<th>No Growth</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical Mastitis</td>
<td>NZ GRASS 6</td>
<td>0%</td>
<td>0%</td>
<td>67%</td>
</tr>
<tr>
<td>Clinical Mastitis</td>
<td>NZ TMR 45</td>
<td>2%</td>
<td>2%</td>
<td>13%</td>
</tr>
<tr>
<td>Clinical Mastitis</td>
<td>OS GRASS 17</td>
<td>18%</td>
<td>0%</td>
<td>29%</td>
</tr>
<tr>
<td>Clinical Mastitis</td>
<td>OS TMR 59</td>
<td>2%</td>
<td>0%</td>
<td>19%</td>
</tr>
</tbody>
</table>

| Subclinical Mastitis | NZ GRASS 14 | 50% | 0% | 50% | 0% | - |
| Subclinical Mastitis | NZ TMR 40 | 35% | 5% | 28% | 33% | - |
| Subclinical Mastitis | OS GRASS 33 | 55% | 6% | 33% | 6% | - |
| Subclinical Mastitis | OS TMR 33 | 52% | 3% | 30% | 15% | - |

### P of Chi2 analysis

<table>
<thead>
<tr>
<th>Genotype</th>
<th>Diet</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical</td>
<td>Genotype</td>
<td>NS</td>
</tr>
<tr>
<td>Clinical</td>
<td>Diet</td>
<td>0.018</td>
</tr>
<tr>
<td>Subclinical</td>
<td>Genotype</td>
<td>NS</td>
</tr>
<tr>
<td>Subclinical</td>
<td>Diet</td>
<td>0.034</td>
</tr>
</tbody>
</table>
were due to *S. uberis* for the NZ cows, and to *S. uberis* or coliforms for the OS cows. For the sub-clinical infections, diet had a strong influence on type of pathogen isolated. For cows on GRASS, the isolates were either *S. uberis* or the minor pathogens, *C. bovis* and CNS whilst for cows on TMR, coliforms were isolated from up to a third of the infections.

### Somatic cell count

Seasonal log SCC did not differ significantly between treatment groups in year one but in year two, the OS cows had a higher (P<0.001) seasonal SCC than the NZ cows. No differences were observed between cows on TMR and cows on GRASS. In year three, the diet effect was significant (P<0.05), with the interaction approaching significance (P=0.066). The NZ cows on TMR showed the highest seasonal SCC, with very little difference between these cows and the OS cows on TMR or GRASS. The NZ cows on GRASS remained at a low SCC for year three. The back transformed data indicate that all groups averaged below 100,000 cells/ml in each season but in year three, the NZ cows on GRASS averaged 35,000 cells/ml whilst the other groups averaged between 70,000-85,000 cells/ml.

### DISCUSSION

In this study, clinical and sub-clinical mastitis were distinguished as two separate entities due to different methods of detection. Detection of clinical cases was reliant on observations made by the milking team whilst sub-clinical infections were detected by routine samplings for bacteriology, occurring up to four times a season. Although clinical and sub-clinical infections are manifestations of the same disease, different causal pathogens result in different patterns of infection. Coliform bacteria tend to cause acute clinical infections of short duration (Pankey, 1997) and tend to be under-represented among sub-clinical infections whilst *S. uberis*, *S. aureus* and the minor pathogens (CNS and *C. bovis*) tend to cause less acute infections, of longer duration (Grommers *et al.*, 1985), and tend to be over-represented among sub-clinical infections. Therefore both forms of mastitis were analysed to establish the full incidence of mastitis of the two genotypes exposed to different nutritional and environmental treatments.

Infection incidences were calculated on a per cow or quarter basis and essentially represent the proportion of susceptible cows or quarters in each treatment group. In the first year of the study, when all cows were primiparous, few differences in incidence were observed between treatment groups. Although OS cows developed proportionally more sub-clinical quarters during the course of the season compared to NZ cows, the difference was numerically very small. In later years, the incidence of clinical and sub-clinical mastitis increased for all groups except for NZ cows on GRASS. For the TMR treatment in the second year, over five times more cows, and seven times more quarters, developed clinical mastitis compared to cows on GRASS, with smaller differences observed in the final year. For cows on TMR, most of the clinical mastitis was due to coliform bacteria, whilst for cows on GRASS, the major causal pathogen was *S. uberis*. This type of mastitis is commonly associated with cows managed on pasture (Pankey *et al.*, 1996) or confined to organic bedding materials such as straw (Bramley, 1982; Hogan *et al.*, 1989).

The higher incidence of mastitis, particularly coliform mastitis, observed for cows managed on TMR may be explained in a number of ways. Environmental factors such as confinement of cows in a restricted area, and higher numbers of coliform bacteria present in the faecal matter are obvious explanations. Indeed, the coliform faecal count of cows on TMR was found to be 1000 fold higher compared to cows on GRASS (Williamson, JH, 1999).

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**FIGURE 1.** Seasonal average log SCC and maximum standard error (SE) for each group of a genotype (NZ HF or OS HF) by nutritional environment (GRASS or TMR) comparison. Individual cow SCC data were first log transformed and averaged for a season prior to analysis. Genotype effect was significant (P<0.001) in 1999/00; diet effect was significant (P<0.05) and Genotype x Diet tended to significance (P=0.066) in 2000/01.
unpublished observations) in the third year of the study. This is consistent with reports that diets with a high concentration of starch can deliver undigested starch to the large intestine that selectively encourages growth of E. coli (Huntington, 1997). This TMR ration contained 23% starch (Roche et al., 2001).

Substantial differences in milk production between cows on TMR and GRASS (Kolver, 2001) may also provide a contributory factor to the difference in mastitis incidence. High milk production has been frequently associated with an increased risk of mastitis in genetic (Pyrce et al., 1998) and epidemiological studies (Oltenacu & Ekesbo, 1994; Fleischer et al., 2001). Milk production differences between primiparous heifers and older cows may also account for the increase in mastitis incidence observed over time for some of the groups, as the average age of the cows increased from 2 years to 2.6 years in the second year, and 3.4 years in the third year of the study. In addition, cows exited the study typically on the basis of pregnancy status rather than susceptibility to mastitis, so cows that were particularly susceptible to mastitis may be over-represented in the second and third year of the study.

Genotypic differences were observed in the second and third year of the study. The OS cows developed more clinical mastitis, and tended to acquire more sub-clinical infections, than NZ cows in the second year. In the third year, a strong interaction occurred between genotype and diet, reflecting the high incidence of mastitis observed for NZ cows on TMR, and OS cows on GRASS. It is interesting to note that these were the groups in which the genotypes were not historically selected for the nutritional environment. Immunological impairment of the OS cows (Fleischer et al., 2001), particularly in the immediate postpartum period when struggling to maintain body weight and condition score (Kolver, 2001) may have contributed to the increased susceptibility of OS cows on GRASS. The reason for the NZ cows on TMR experiencing more mastitis is not known.

The higher incidence of mastitis for HF cows of overseas origin compared to NZ cows has not previously been reported. However Dutch studies have observed that herds with mainly purebred HF had a higher incidence of E. coli clinical mastitis compared to herds with mainly Dutch Friesians (Schukken et al., 1991). These genotypic differences may be due to differences in the teat canal defences or efficacy of the immune system and further investigation is required to evaluate these factors.

Although wide differences in clinical incidence were observed between treatment groups, similar clinical mastitis incidences were found in the literature for cows managed on pasture or TMR. When averaged over the three years of this study, the number of cows affected by clinical mastitis averaged 19.5% for cows on GRASS, and 54% for cows on TMR. A recent US study (Washburn et al., 2002), comparing clinical mastitis among HF and Jersey cows managed in either a TMR/total confinement system or at pasture, observed, over a three-year period, that clinical mastitis affected 31% of HF cows and 17% of Jerseys managed predominantly on pasture, compared to 51% of HF cows and 35% of Jerseys, managed on TMR in confinement. Epidemiological studies have observed clinical mastitis incidences that range from 15% of cows (Schukken et al., 1991) to 25.9% of cows (Kossaibati et al., 1998) and 27% of cows (Blowey, 1984) but the clinical mastitis incidence recorded for individual herds can vary from 0-80% (Schukken et al., 1991). Although the incidence of clinical mastitis for the TMR cows reported here was higher than that for typical TMR-fed, housed cows, the cow incidence was within ranges reported for individual Dutch and UK herds (Kossaibati et al., 1998).

The bulk milk SCC reflects primarily the level of sub-clinical mastitis in a herd (Eberhart et al., 1982; Holdaway, 1992b). Few differences were observed in the SCC between cows on GRASS compared to TMR, reflecting the difference in types of pathogen predominately causing mastitis. Coliform mastitis frequently affected the TMR cows but rarely causes sub-clinical mastitis, therefore having a lesser effect on cow SCC. Genotype differences were observed in the second year, with OS cows averaging higher than NZ cows, but this effect was obscured in the third year by the high SCC of the NZ cows on TMR. This result may reflect the high incidence of sub-clinical mastitis experienced by these cows in that year. Such genotypic differences in SCC have not previously been reported. Washburn et al., (2002) reported no differences in SCC due to breed or nutritional treatments whilst Goldberg et al., (1992) observed no difference in bulk tank SCC between herds managed in either confinement or at pasture.

In conclusion, the environment associated with feeding TMR had a significant impact on the incidence of clinical mastitis whilst genotype had a less obvious, but significant effect on incidence of sub-clinical mastitis and cow SCC. The wider implications of the introduction of HF genetics into the NZ pastoral system should continue to include a monitor of udder health, in addition to production and reproduction performance.

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