Estimation of Genetic and Crossbreeding Parameters for Clinical Mastitis, Somatic Cell Score and Daily Yields of Milk, Fat and Protein in New Zealand Dairy Cattle

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Introduction

Clinical mastitis (CM) is one of the most economically important diseases in New Zealand dairy cattle. The National Mastitis Advisory Committee (2006) reported that the cost of CM for the industry is about $180 million per year. Economic losses are associated with reduced milk production, discarded milk during the withholding periods, treatment costs, reduction in milk price due to high somatic cell count and the culling of persistently infected cows.

The industry initiated a long-term alternative to reduce incidence of clinical mastitis through the breeding of dairy cows with reduced levels of somatic cells counts (Harris *et al.,* 2005). The main reasons for choosing somatic cell count as the indirect trait for mastitis resistance were the following: somatic cell count is routinely recorded in the herd-testing program, and based on reports from other countries, somatic cell count has higher heritability than clinical mastitis and the genetic correlation between both traits are moderate to high. However, there is a lack of estimates of genetic correlations between CM with somatic cell score (SCS) and production traits in New Zealand dairy cattle.

Breed and heterosis effects for production and fertility traits and SCS in New Zealand dairy cattle were reported by Harris (2005) but not for incidence of CM. The objective of this study was to estimate genetic and crossbreeding parameters for CM, SCS and production traits using test-day records of New Zealand grazing dairy cattle.

Material and methods

Data and definition of traits. Test-day records for yields of milk, fat and protein and somatic cell counts were merged with CM records collected during the production seasons 2005-2006 to 2007-2008 from 53,419 cows of different breeds including Holstein-Friesian (HF), Jersey (JE) and HFxJE crossbred. The cows were the progeny of 641 sires and were distributed in 167 dairy herds used for the progeny testing of bulls. There were 92,961 lactations and 356 contemporary groups (cows that calved in the same herd and year) in this study.

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Breed proportions of each cow and its sire and dam were available, which allowed the estimation of both breed and heterosis effects. The proportion of genes from HF and JE and the coefficient of HFxJE heterosis were calculated for each cow with records according to Dickerson (1969). Somatic cell score (SCS) was calculated as SCS = $\log_2$(SCC). Lactation averages of daily yields of milk, fat, protein and SCS were obtained for each cow and lactation. Clinical mastitis was coded 1 for cows that presented at least one event of CM at any day at risk of the season and 0 for healthy cows.

Statistical analyses. A multiple-trait repeatability animal model was used to obtain (co)variance components for SCS, daily yields of milk, fat and protein and incidence of CM using the ASReml program (Gilmour *et al.,* 2002). The model included the fixed effects of herd-year, month of calving, parity number, the regressions of proportion of JE and heterosis HFxJE, and the random effects of animal, cow and residual. The regression on HF breed proportion was excluded from the model in order to avoid linear dependencies with JE. The random effects of animal, cow, and residual were assumed to be normally and independently distributed with mean equal to zero. The pedigree file included parents and grandparents of the cow. Heterosis effects were expressed as a percentage of the mean of purebred HF and JE.

Results and discussion

The incidence of CM was 11% in 92,961 lactations. This value is lower than the value reported in Norwegian dairy cattle (23.3%, Heringstad *et al.,* 2003), similar to the value reported in Swedish dairy cattle (10.1%, Carlén *et al.,* 2009) and higher than the value reported in United Kingdom dairy cattle (7.6%, Kadarmideen *et al.,* 2001). About 60% of cases of CM occurred within the first 30 days of the lactation. A similar pattern was reported in Norwegian (Heringstad *et al.,* 2003) and Swedish dairy cattle (Carlén *et al.,* 2009).

Estimates of genetic parameters are shown in table 1. Estimates of heritability for daily yields of milk, fat and protein were 0.28, 0.19 and 0.21, respectively, which are lower than previous values reported in New Zealand dairy cattle using total yields (Pryce and Harris, 2006) but do not widely different from average values reported by Mrode and Swanson (1996). The estimate of heritability for SCS was 0.11, which is lower than 0.18 estimated by Pryce and Harris (2006).

Heritability for CM was low, 0.02±0.002, which agrees with average values reported in the literature (Carlén, 2008). Carlén (2008) indicated that the low values of heritability have often been misinterpreted as meaning that genetic selection to improve the innate resistance has a limited role to play in mastitis control programs. However, the low heritability is mainly due to large environmental variation, which is difficult to control by farm management and good milking practices, and considerable genetic differences between bulls exist. In this study, estimated breeding values for CM ranged from -8 to +8%.

Estimates of genetic correlations between production traits and SCS are similar to the values reported by Pryce and Harris (2006). The correlations between production traits and SCS with CM are similar to the average values summarized by Carlén (2008). The high

correlation between SCS and CM estimated at 0.63 confirms that SCS can be used as an indirect trait of resistance to CM in New Zealand dairy cattle as already implemented in the selection objective.

Estimates of crossbreeding parameters are shown in table 2. Holstein-Friesian cows were superior for daily yields of milk, fat and protein confirming breed effects for lactation yields reported by Harris *et al.* (1996). Holstein-Friesian had higher level of SCS than JE cows which agree with results of genetic evaluation (Harris *et al.,* 2005).

Jersey cows had an average of 2.9% (table 2) less incidence of CM than HF cows confirming experimental results in Ireland (Buckley *et al.,* 2008) and the United States of America (Washburn *et al.,* 2002). In the experiment reported by Buckley *et al.* (2008), incidence of CM was 29% in Holstein-Friesian and 27% in Jersey cows. Washburn *et al.* (2002) reported that Jerseys had half as many clinical cases of mastitis per cow as Holsteins; 31.4 vs 51.0% in confinement and 17.0 vs 34.6% in grazing.

Heterosis values for daily yields of milk, fat and protein and level of SCS expressed as percentage of the straight-breed mean obtained in this study agree with the heterosis values reported by Harris (2005) using lactation yields of milk, fat and protein and lactation average of SCS. The estimate of heterosis effects for CM obtained in this study was -10.7%. In the experiment reported by Buckley *et al.* (2008) incidence of CM in HFxJE cows was 11%, resulting in a heterosis value of -61% expressed as a percent deviation of the two straight breeds.

Conclusion

The estimates of genetic and crossbreeding parameters for production traits and SCS obtained in this study confirm previous values reported in New Zealand and other countries. Estimate of heritability of incidence of CM under grazing conditions is low and similar to values reported in other countries where CM is a trait included in the breeding goal. Breed and heterosis effects for CM were significant and can be exploited in a breeding program to improve resistance to CM.

Table 1: Estimates of genetic parameters for incidence of clinical mastitis (CM) and lactation averages of daily yields of milk, fat, protein and somatic cell score (SCS) in New Zealand dairy cattle[A]

Traits	Milk	Fat	Protein	SCS	CM
Milk	0.28±0.011	0.73±0.002	0.91±0.001	- 0.11±0.004	- 0.01±0.004
Fat	0.45±0.026	0.19±0.010	0.79±0.002	- 0.10±0.004	- 0.04±0.004
Protein	0.84±0.009	0.60±0.022	0.21±0.011	- 0.08±0.004	- 0.01±0.004
SCS	- 0.04±0.043	- 0.03±0.049	- 0.04±0.048	0.11±0.008	0.19±0.003
CM	0.26±0.070	0.18±0.079	0.25±0.076	0.63±0.063	0.02±0.002

[A]Heritabilities (±s.e.) on the diagonal, phenotypic and genetic correlations below and above the diagonal, respectively.

Table 2: Estimates of breed and heterosis effects for incidence of clinical mastitis (CM) and lactation averages of daily yields of milk, fat, protein and somatic cell score (SCS) in New Zealand dairy cattle

	Milk (litres/day)	Fat (kg/day)	Protein (kg/day)	SCS	CM (%)
Breed					
Holstein-Friesian	19.2±0.03	0.84±0.001	0.68±0.001	6.77±0.011	13.2±0.31
Jersey	14.6±0.03	0.79±0.001	0.58±0.001	6.67±0.011	10.3±0.46
Heterosis HFxJE[A]	0.85±0.04	0.06±0.001	0.04±0.001	- 0.026±0.019	- 1.25±0.39
Percentage of the straightbreds mean	5.0	7.4	6.4	-0.4	- 10.7

[A]HF = Holstein-Friesian, JE = Jersey.

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