# Genetic analysis of incidence of clinical mastitis in New Zealand dairy cattle

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## ABSTRACT

The aim of the study was to estimate breed and genetic effects on the incidence of clinical mastitis (CM) in New Zealand dairy cattle. Records of CM collected during seasons 2005-06 to 2008-09 from 53,419 cows of different breeds including Holstein-Friesian (H-F), Jersey (J) and Crossbed (HF x J), were analysed. Clinical mastitis was coded "1" for cows that presented at least one event of CM at any day at risk during the season and "0" for cows without CM. Genetic parameters and breed effects for the incidence of CM were estimated with a repeatability animal model across breeds using restricted maximum likelihood methodology. The cumulative lactation incidence of CM was 11% in 92,961 lactations. Heritability and repeatability for incidence of CM were 0.015 ± 0.003 and 0.070 ± 0.005, respectively. Jersey cows had 2.9% less incidence of CM than H-F cows. Heterosis effect of H-F x J was minus 11.7% of the average of the parental breeds. Results from this study confirm previous estimates of genetic parameters for CM. Sire variation and breed and heterosis effects were significant and can be exploited in a breeding program to improve resistance to CM in New Zealand dairy cattle.

**Keywords:** clinical mastitis; genetic parameters; crossbreeding effects.

## INTRODUCTION

Mastitis is defined as any inflammatory process in the mammary gland that occurs as a response to an intra-mammary infection or a trauma often caused by an excessive vacuum during mechanical milk extraction. Mastitis can have a clinical or subclinical appearance. Clinical mastitis (CM) is detected by visible abnormalities in the udder or milk. Subclinical mastitis is only detected by laboratory methods such as analysis of somatic cell count or other parameters related to the inflammatory process (IDF, 1999).

A wide variety of microorganisms including bacteria, fungi, yeast and mycoplasma may lead to an intra-mammary infection. Bacteria are described to be the most frequent pathogens of this disease with *Streptococcus uberis* and coagulase-negative staphylococci being the most important bacteria causing mastitis in New Zealand dairy cattle (McDougall, 2002). In addition to microorganisms, an array of multiple environmental factors facilitates the development of mastitis. These factors include breed, age, general udder and calving hygiene, several technical aspects of mechanical milk extraction, drinking water quality, the quality of pasture, feed and ration, dry matter intake, cow comfort, health management, and cow handling (Bramley & Dodd, 1984).

Mastitis is one of the most economically important diseases in New Zealand dairy cattle. In herds without an effective mastitis control program, about 40% of the cows are infected with an average of two quarters (Holmes *et al.*, 2002). An economic evaluation reported by the National Mastitis Advisory Committee (2006) estimated that the cost of clinical mastitis for a representative New Zealand farm was $36.50 per cow, $11,500 for a herd with 315 cows, and some $180 million for the New Zealand dairy industry. Economic losses are associated with reduced milk production from both clinical and subclinical mastitis, discarded milk during the withholding periods, treatment associated costs, reduced milk price due to a high somatic cell count and the culling of persistent mastitis infected cows.

A program to control mastitis and reduce somatic cell counts in New Zealand dairy herds was implemented in an action plan called the seasonal approach to managing mastitis (National Mastitis Advisory Committee, 2008). The plan considers lists of best farm practices to prevent, control and treat CM in the herd. Along with this plan the industry initiated a long-term alternative to reduce incidence of CM 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 are because somatic cell count is routinely recorded in the herd-testing program, somatic cell count has higher heritability than clinical mastitis, and genetic correlations between both traits are moderate to high. Therefore, it is possible that selection to decrease somatic cell count would reduce the incidence of CM and sub-clinical mastitis. In a review Mrode and Swanson (1996) reported that estimates of the heritability of somatic cell count were low, with a weighted average of 0.11 but were higher than the heritability of mastitis incidence of

0.04. The average of estimates of genetic correlations between somatic cell count and CM incidence was 0.7.

Breed and heterosis effects for incidence of CM can be exploited in a crossbreeding program. Washburn *et al*. (2002) reported results from a farmlet experiment comparing Holstein and Jersey cows under two feeding systems, confinement and grazing. Jerseys had half as many clinical cases of mastitis per cow as Holsteins; 31.4 versus 51.0% in confinement and 17.0 versus 34.6% in grazing. McDowell and McDaniel (1968) estimated heterosis effects for health traits on first lactation cows. Average per cent deviation of two-breed crosses from parental mean for incidence of CM at any stage of the first lactation was -7% in Ayrshire x Holstein, -47% in Brown Swiss x Holstein and 6.9% in Ayrshire x Brown Swiss crossbred cows. Estimates of crossbreeding effects for incidence of CM between Holstein-Friesian and Norwegian Red breeds under Irish grazing conditions were reported by Buckley *et al.* (2008). Incidence of CM was 13% in Holstein-Friesian, 10% in Norwegian Red and 11% in first cross Holstein-Friesian x Norwegian Red cows. Heterosis effects expressed as a per cent deviation of the two parental breeds was -4.3%. The same authors also reported estimates of heterosis for incidence of CM in a crossbreeding experiment involving the Holstein-Friesian and Jersey breeds. Incidence of CM was 29% in Holstein-Friesian, 27% in Jersey and 11% in Holstein-Friesian x Jersey cows. The estimate of heterosis expressed as a per cent deviation of the two parental breeds was -60.7%.

The aim of the study was to estimate breed and genetic effects on the incidence of CM in New Zealand dairy cattle.

## MATERIALS AND METHODS

Records on CM collected during the seasons 2005-06 to 2008-09 from 53,419 cows of different breeds including Holstein-Friesian (H-F), Jersey (J) and Crossbed (H-F x J). The cows were the progeny of 641 sires and were distributed in 167 dairy herds used for the progeny testing of bulls. Clinical mastitis was coded "1" for cows that presented at least one event of CM at any day at risk in the season and "0" for healthy cows.

The definition of contemporary group was cows that calved in the same herd and year and there were 356 contemporary groups in this study. Breed composition of each cow was described in terms of proportion of H-F and J. The proportion of genes from each breed was calculated for each animal using the simple identity:

$$p_i = (s_i + d_i)/2$$

where $p_i$ is the proportion of genes from breed i in the progeny, $s_i$ is the proportion of breed i in the sire, and $d_i$ is the proportion of breed i in the dam. Coefficient of H-FxJ heterosis was calculated using the following identity:

$$h_{HFxJ} = p^{HF}{}_s p^{J}{}_d + p^{J}{}_s p^{HF}{}_d$$

where $p^{HF}{}_s$ and $p^{J}{}_s$ are proportions of H-F and J in the sire, respectively, and $p^{HF}{}_d$ and $p^{J}{}_d$ are proportions of H-F and J dam, respectively.

A repeatability animal model was used to obtain variance components for incidence of CM using ASReml (Gilmour *et al.*, 2002). The model included the fixed effects of herd-year, parity number, month of lactation, the regressions of proportion of J and heterosis H-F x J, and the random effects of animal, permanent environment of cow and residual. The regression on H-F breed proportion was excluded from the model; in order to avoid linear dependencies with J. 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. Heritability was calculated as $[\sigma^2_a / (\sigma^2_a + \sigma^2_c + \sigma^2_e)]$ and repeatability was calculated as $[(\sigma^2_a + \sigma^2_c)/ (\sigma^2_a + \sigma^2_c + \sigma^2_e)]$ where $\sigma^2_a$, $\sigma^2_c$, and $\sigma^2_e$ are the additive genetic, cow and residual variances, respectively. Heterosis effects for CM were expressed as a percentage of the mean of purebred H-F and J.

## RESULTS AND DISCUSSION

The cumulative lactation incidence of CM was 11% in 92,961 lactations. This value is lower than the value reported in Norwegian dairy cattle of 23.3% (Heringstad *et al*., 2003), similar to the value reported in Swedish dairy cattle of 10.1% (Carlén *et al*., 2009) and higher than UK dairy cattle of 7.6% (Kadarmideen *et al.*, 2001).

Distribution of 12,144 cases of CM during the lactation considering all the 92,961 lactations is shown in Figure 1. The majority of cases of CM occurred within the first 30 days of the lactation. A similar pattern was reported in Norwegian

**FIGURE 1:** Number of cases of clinical mastitis during the lactation.

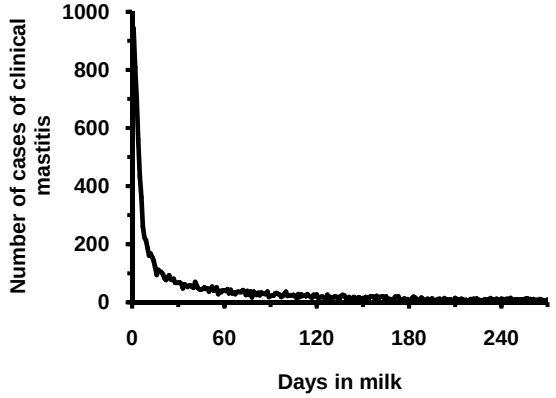


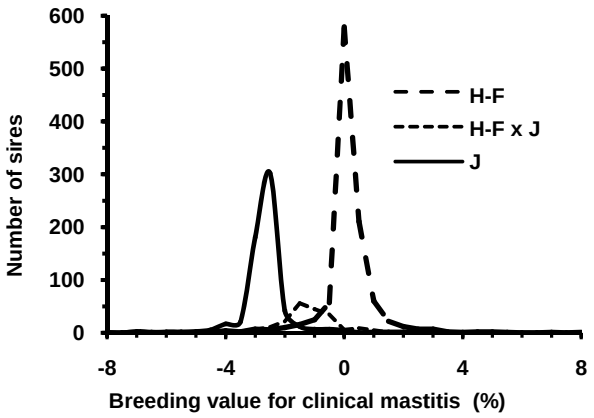
**TABLE 1:** Estimates of variances, heritability and repeatability for incidence of clinical mastitis in New Zealand dairy cows.

| Parameter | Estimate |
|---|---|
| Genetic variance | 0.001 ± 0.0002 |
| Permanent environmental variance | 0.005 ± 0.0005 |
| Residual variance | 0.085 ± 0.0006 |
| Phenotypic variance | 0.091 ± 0.0004 |
| Heritability | 0.015 ± 0.003 |
| Repeatability | 0.070 ± 0.005 |

**TABLE 2:** Breed and heterosis effects for incidence of clinical mastitis in New Zealand dairy cows.

| Breed effect | % |
|---|---|
| Holstein-Friesian | 13.2 ± 0.31 |
| Jersey | 10.3 ± 0.40 |
| Heterosis Holstein-Friesian x Jersey | -1.25 ± 0.40 |

**FIGURE 2:** Distribution of sires according to estimated breeding values for clinical mastitis and breed (H-F= Holstein-Friesian, J = Jersey).




(Heringstad *et al*., 2003) and Swedish dairy cattle (Carlén *et al*., 2009).

Estimates of variance components for incidence of CM are shown in Table 1. Heritability and repeatability for the incidence of CM were 0.015 ± 0.003 and 0.070 ± 0.005, respectively. The estimate of heritability is similar to the average value of 0.04 reported by Mrode and Swanson (1996) from many different studies. Carlén (2008) indicated that the low value of heritability has often been misinterpreted as meaning that genetic selection to improve the innate resistance has a limited role to play in mastitis control programmes. However, the low heritability is mainly due to large environmental variation, which is difficult to control by farm management and good milking practices. Considerable genetic differences exist between bulls. Figure 2 shows a considerable variation between estimated breeding values for CM with values ranging from -8 to +8%.

Jersey cows had an average of 2.9% (Table 2) less incidence of CM than H-F cows confirming experimental results in Ireland (Buckley *et al*., 2008) and United States of America (Washburn *et al*., 2002). As shown in Figure 2, breeding values of Jersey bulls tend to be lower than breeding values of Holstein-Friesians bulls, and crossbred bulls with intermediate values. The heterosis effect of H-F x J was -1.25% which represent minus 11.7% of the parental breeds in agreement with estimates reported by Buckley *et al*. (2008) for Holstein-Friesian x Jersey crossbred and Holstein-Friesian x Norwegian Red crossbred cows.

Mastitis is a complex disease caused by a number of pathogens and supporting factors that affect different parts of the udder and produce varying levels of response of the immune system (Bannerman *et al.*, 2004). One approach to reduce the incidence of mastitis, in addition to adequate udder health management, is the selection of animals that are resistance to the disease. Advances in DNA chip technology allows the discovery of single nucleotide polymorphisms related to the genes of the immune system and therefore prediction of breeding values for resistance to a specific pathogen may become feasible.

Results from this study confirm previous estimates of genetic parameters for CM. Variation on sire's breeding values for incidence of CM along with breed and heterosis effects can be exploited in a breeding program to improve resistance to CM in New Zealand dairy cattle.

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