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Effect of breed and crossbreeding on the incidence of recorded clinical lameness in New Zealand dairy cattle

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Summary

The aim of this study was to evaluate the effect of breed and crossbreeding (heterosis in F1) on the incidence of recorded clinical lameness (RCL) in New Zealand dairy cattle. Records from 76,357 cows, were collected from Livestock Improvement Corporation (LIC) herds, dedicated to the sire improvement scheme during the 2005 to 2008 milking season, and used to estimate the effect of breed and heterosis in Holstein-Friesian (F), Jersey (J) and crossbred (FxJ) dairy cattle. RCL was coded “1” for cows that presented at least one event of RCL and “0” for cows with no RCL on any day during the milking season. Effect of breed and crossbreeding on the incidence of RCL were estimated with an animal repeatability model across cow breeds using restricted maximum likelihood methodology. Mean annual incidence of RCL was 6.3%, with a range from 2% to 34%. Jersey cows had 1.9% lower mean incidence of RCL compared to F cows. Heterosis effect of FxJ crossbred was -1.2 ± 0.3%, representing a 19% reduction in RCL compared with the mean for pure bred J and F cattle. Results from this study indicate that breed and heterosis had a significant effect on RCL and that this could be exploited in a breeding program to reduce the incidence of RCL in New Zealand dairy cattle.

Keywords: Clinical lameness, breed effects, heterosis, dairy cattle

Introduction

Lameness is described as any abnormality in locomotion, which is often associated with tissue damage, pain and discomfort and is a growing problem in dairy cattle managed in grazing systems (Tranter and Morris, 1991; Chesterton et al., 2008). In New Zealand the incidence of lameness in dairy herds has been reported to be an average of 14%, with a range between 2 to 38% (Tranter and Morris, 1991), but can be as high as 60% (Vermunt, 1992). Lameness itself can be classified as subclinical or clinical depending on the severity and length of the healing period (Vermunt, 1992). Clinical lameness is mainly associated with pain, discomfort, which affects locomotion and has a
negative effect on animal welfare and has been reported to be one of the most costly diseases in dairy cattle directly contributing to a financial loss of approximately $94 per lame cow (Tranter and Morris 1991). The likelihood of a cow being culled due to lameness is higher than that attributed to the disease directly, due to its involvement in reduced fertility and milk production (Diaz-Lira et al., 2009).

Lameness is a multifactorial disease resulting from various predisposing factors, which include environment (Chesterton et al. 1989), management (Chesterton et al., 2008) and animal characteristics (Olmos et al. 2008). The incidences of lameness in New Zealand dairy herds has been associated with herd management, nutrition and genetic factors and the combination of these factors can lead to the complex nature of lameness (Chesterton et al., 1989).

The effect of cattle breed, crossbreeding and genetic heritability of dairy characteristics, such as milk yield, milk fat and protein content, live weight, somatic cell score, longevity, fertility (Harris, 2005) and the incidence of clinical mastitis (Jury et al., 2010) has been reported in New Zealand. However the incidence of lameness has not (Chesterton et al. 2008). The estimated effect of breed and heterosis on the incidence of clinical lameness has been reported overseas (Olmos et al., 2008) where approximately 83% of the dairy animals are managed under high input high output systems and housed indoors for 6 to 12 months of the year, which differs considerably from the pasture based dairying systems applied by the majority of New Zealand dairy farms (Van Arendonk and Liinamo 2003). Therefore, the current study aims to estimate the effect of breed and crossbreeding (heterosis) on the incidence of reported clinical lameness in New Zealand dairy cattle managed under pasture based production systems, in which a first cross cow (F₁) contained 50% of genes from the two parental breeds and expressed 100% of the individual heterosis.

**Materials and Methods**

**Data collection**

This study used dairy herd records from Livestock Improvement Corporation (LIC) that were collected during the 2005/2006 and 2007/2008 season from herds that were dedicated to the sire improvement scheme. This dataset contained cow identification, parity, farm location, calving date, breed composition, the date and record of clinical lameness. The incidence of recorded clinical lameness (RCL) and milk yield traits were estimated from the different breed groups, which included Holstein-Friesian (F), Jersey (J) and crossbred (FxJ). An incidence of RCL was coded as “1” for cows that presented at least one recorded event of clinical lameness, while “0” was the code for cows with no recorded incidence of clinical lameness at any day at risk in the milking season. The lactation records collected included the details of parity, milk yield, milk fat and protein yield for individual cows in each
of the herds. The data from farms with fewer than 100 lactating cows and any cows with more than 10 completed lactations were omitted in the analysis. The datasets included approximately 111,565 lactations, from 155 dairy herds and 261 contemporary groups were defined as cows that had calved in the same herd-year.

Data Analysis

The descriptive statistics of the incidence of RCL and milk production traits were analysed using SAS Version 9.2 (SAS 2002) and the structured query language was used to sort, summarise and create new variables and to combine datasets. The milk production traits included were mean daily milk yield, milk fat and protein content. The means and standard deviations of daily milk yield, milk fat and protein content for cows with (lame) and without (non-lame) an incidence of RCL was obtained using the MEANS procedure of SAS (2002). The cause of each incidence of RCL were classified as white line disease, sole bruising, sole lesion, sole penetration, foot rot, hoof crack and other for unknown/unreported causes of lameness, which were analysed using the FREQ procedure of SAS (2002).

Calculations of breed proportions and coefficients of specific heterosis

The breed composition of each cow was described in terms of proportions of F and J. The cows that had a breed composition that was more than 87.5% or less than 12.5% of a single specific (F or J) dairy breed were described as purebred and the rest were described as crossbred. The proportions of genes from each breed were calculated for each animal using a simple identity (Jury et al., 2010) equation:

$$\alpha_p^i = (\alpha_s^i + \alpha_d^i)/2$$

where $\alpha_p^i$ = proportion of genes from breed $i$ in the progeny, $\alpha_s^i$ is the proportion of breed $i$ in the sire and $\alpha_d^i$ is the proportion of breed $i$ in the dam. The coefficient of F x J heterosis ($h_{FxJ}$) was calculated using the following identity (Jury et al. 2010) equation:

$$het_{HFxJ} = \alpha_{sF}^i \alpha_{dJ}^i + \alpha_{sJ}^i \alpha_{dF}^i$$

where, $het_{HFxJ}$ is the coefficient of expected heterosis between fractions of F and J in the progeny, $\alpha_{sF}^i$ is the proportion of F in the sire, $\alpha_{dJ}^i$ is the proportion of J in the dam, $\alpha_{sJ}^i$ is the proportions of J in the sire and $\alpha_{dF}^i$ is the proportion of F in the dam.

Model for estimation of crossbreeding parameters

Crossbreeding parameters for incidence of clinical lameness of dairy cows were estimated using the restricted maximum likelihood procedure by fitting an animal model in the ASReml software program.
(Gilmour et al., 2002). A univariate repeatability model (Mrode, 2005) was used considering fixed and random effects:

\[ y = Xb + Za + Wp + e \]

where, \( y \) is the vector of phenotypic observations, \( b \) is the vector of fixed effects, \( X \) is an incidence matrix relating records to fixed effects, \( Z \) is an incidence matrix relating records to animal effects, \( W \) is an incidence matrix relating the records to permanent environmental effects, \( a \) is a vector of additive random animal effects, \( p \) is a vector of random permanent environmental effects and \( e \) is the vector of random residual effects.

\( Z \) matrix allows inclusion of sire and dams related to cows with records and \( W \) is an identity matrix. The fixed effects included in \( b \) were contemporary group of herd-year, month of calving, lactation number and as covariables of proportion of J and coefficient of heterosis of \( F x J \).

It was assumed the following expectations \( (Ey) = Xb \) and \( E (a) = 0 \) and \( E (e) = 0 \) and variances \( \text{var} (a) = A \sigma^2_a = G \), \( \text{var} (p) = I \sigma^2_p \) and \( \text{var} (e) = I \sigma^2_e \) and hence var \( (y) = ZAZ' \sigma^2_a + W \sigma^2_p W' + R \) where, \( A \) is the numerator relationship matrix between all animals considered within the data set. The numerator relationship matrix is based on the knowledge of the pedigree relationship of parents and offspring suggested by (Mrode, 2005). The pedigree file included parents and grandparents of a cow with full pedigree records.

The mixed model equations used for the estimation of fixed effects, prediction of breeding values and the effect of permanent environmental effects are presented below:

\[
\begin{bmatrix}
X'X & X'Z & X'W \\
Z'X & Z'Z + A^{-1} \alpha_1 & Z'W \\
W'X & W'Z & W'W + I \alpha_2
\end{bmatrix}
\begin{bmatrix}
b \\
ap \\
p
\end{bmatrix}
= 
\begin{bmatrix}
X'y \\
z'y \\
w'y
\end{bmatrix}
\]

Therefore the mixed model equations for the best linear unbiased estimator (BLUE) of function \( b \) and the best linear unbiased prediction of additive random animal effects (\( a \)) and random environmental effects (\( p \)) were obtained as follows:

\[
\begin{bmatrix}
b \\
ap \\
p
\end{bmatrix}
= 
\begin{bmatrix}
X'X & X'Z & X'W \\
Z'X & Z'Z + A^{-1} \alpha_1 & Z'W \\
W'X & W'Z & W'W + I \alpha_2
\end{bmatrix}^{-1}
\begin{bmatrix}
X'y \\
z'y \\
w'y
\end{bmatrix}
\]

The matrices \( X'X \), \( X'Z \), \( X'W \), \( X'y \), \( Z'X \), \( W'X \), \( W'Z \) and \( Z'Z \) were obtained by using matrix multiplication principles with \( \alpha_1 = \sigma^2_e / \sigma^2_a \) and \( \alpha_2 = \sigma^2_e / \sigma^2_p \) (Mrode 2005) and \( A^{-1} \) is the inverse of the numerator relationship matrix.
Results

Average production and overall recorded incidence of clinical lameness
There were 111565 lactation records that were used to assess the incidence of RCL while 111405 lactation records were used to assess the milk production traits (Table1). The overall incidence of clinical lameness per herd was 6.3 ± 0.24% with a range of 2% to 34%.

Table 1: Mean incidence of lameness, daily milk yield (kg), fat (kg), protein (kg) and milk solids production (kg fat + protein) of dairy cattle from Livestock Improvement Corporation (LIC) dedicated sire improvement scheme herds in New Zealand

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD</th>
<th>Number of lactations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence of recorded lameness (%)</td>
<td>6.3 ± 0.24</td>
<td>111565</td>
</tr>
<tr>
<td>Mean milk yield (kg/head/d)</td>
<td>16.91± 4.91</td>
<td>111405</td>
</tr>
<tr>
<td>Mean fat yield (kg/head/d)</td>
<td>0.80 ± 0.21</td>
<td>111405</td>
</tr>
<tr>
<td>Mean protein yield (kg/head/d)</td>
<td>0.63 ± 0.17</td>
<td>111405</td>
</tr>
<tr>
<td>Mean milk solids (kg/head/d)</td>
<td>1.43 ± 0.38</td>
<td>111405</td>
</tr>
</tbody>
</table>

The cows with a recorded incidence of clinical lameness had a lower mean daily milk yield in all breed groups, compared with non lame cows (Table 2). The mean milk yield was 1.96 % lower for JxF, 2% for F and 2.3% lower for J cows. The daily milk composition of fat and protein was consistently lower for cows with a recorded incidence of lameness compared with cows of the same breed with no recorded lameness. The daily milk fat yield was lower for cows with a recorded incidence of lameness, which was 1.25% lower for F, 1.4% for J and 2.4% for FxJ cows, in comparison to cows with no recorded incidence of lameness. The daily milk protein yield was lower in cows with a recorded incidence of clinical lameness, which was 1.5% lower for F, 1.6% for FxJ and 3.7% lower for J cows compared with cows of the same breed with no recorded incidence of lameness.
Table 2: Means (±SD) daily milk production of Friesian (F), Jersey (J) and crossbred (FxJ) cows with (Lame) and without (Normal) a recorded incidence of clinical lameness from Livestock Improvement Corporation (LIC) dedicated sire improvement scheme herds in New Zealand

<table>
<thead>
<tr>
<th>Breed</th>
<th>Obs.</th>
<th>Pathology</th>
<th>Mean (±SD) milk trait (kg/head/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Yield</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Fat</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Protein</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Milk solids</td>
</tr>
<tr>
<td>Friesian</td>
<td>37367</td>
<td>Non lame</td>
<td>18.33 ± 5.14</td>
</tr>
<tr>
<td></td>
<td>2713</td>
<td>Lame</td>
<td>17.96 ± 5.07</td>
</tr>
<tr>
<td>Jersey</td>
<td>13896</td>
<td>Non lame</td>
<td>13.41 ± 3.55</td>
</tr>
<tr>
<td></td>
<td>883</td>
<td>Lame</td>
<td>13.10 ± 3.50</td>
</tr>
<tr>
<td>Crossbred</td>
<td>53159</td>
<td>Non lame</td>
<td>16.85 ± 4.59</td>
</tr>
<tr>
<td></td>
<td>3437</td>
<td>Lame</td>
<td>16.52 ± 4.54</td>
</tr>
</tbody>
</table>

Table 3: Types of clinical lameness recorded in the incidence of clinical lameness from Livestock Improvement Corporation (LIC) dedicated sire improvement scheme herds in New Zealand

<table>
<thead>
<tr>
<th>Causes</th>
<th>Percentage (%)</th>
<th>Number of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Other lameness (type not reported)</td>
<td>63.03</td>
<td>3572</td>
</tr>
<tr>
<td>Between claw (Foot rot)</td>
<td>20.88</td>
<td>1183</td>
</tr>
<tr>
<td>White line disease</td>
<td>7.78</td>
<td>441</td>
</tr>
<tr>
<td>Sole haemorrhage / lesion</td>
<td>4.11</td>
<td>233</td>
</tr>
<tr>
<td>Sole penetration</td>
<td>3.00</td>
<td>170</td>
</tr>
<tr>
<td>Hoof crack</td>
<td>1.20</td>
<td>68</td>
</tr>
<tr>
<td>Total</td>
<td>100.00</td>
<td></td>
</tr>
</tbody>
</table>

The types of clinical lameness that were recorded were assessed (Table 3) and it was found that in the greatest proportion of incidences of RCL the type of lameness was either not recorded or was recorded as ‘other’. There were approximately 37 % of incidences of RCL were the type of lameness was recorded and the most common types of clinical lameness recorded were foot rot followed by claw horn disorders, such as white line disease, sole bruising and sole penetration, followed by hoof cracks.
Heterosis and breed effects on incidence of recorded clinical lameness

The effect of breed on the incidence of RCL, which was measured as the difference between the incidence of RCL in Friesian cows and the incidence of RCL in J cows, and was found to be lower in J cows (Table 4), while the effect of heterosis showed a lower incidence of RCL in first cross FxJ. Overall, crossbred cows had a lower incidence of RCL compared to the mean of that of purebred J and F cattle, while J cows had a lower incidence of RCL than F cows.

Table 4: Estimated effect of breed and heterosis on the incidence of recorded clinical lameness from Livestock Improvement Corporation (LIC) dedicated sire improvement scheme herds in New Zealand

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Estimate of clinical lameness (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breed effect (F - J)</td>
<td>-1.9 ± 0.4</td>
</tr>
<tr>
<td>First cross heterosis (F x J)</td>
<td>-1.2 ± 0.3</td>
</tr>
</tbody>
</table>

Discussion

The mean incidence of RCL (6.3%) in this study was lower when compared to previous studies (12 to 14%), but was within the incidence range reported in New Zealand (Gibbs, 2010; Tranter and Morris 1991). The lower incidence of clinical lameness is most likely due to the method of observation and the potential lack of adequate observation or recording of cows that suffer some level of lameness, that were not subsequently recorded as clinically lame. This would be in keeping with previous studies that have noted differing levels and ranges of lameness being reported when cows were observed by differing practitioners such as farmers, veterinarians and specially trained observers (Clarkson et al., 1996; Winkler, 2005).

The occurrence of RCL significantly (P<0.05) reduced milk volume, fat and protein yields, consistently in F and JxF cows, which corresponded to previous studies completed on F cattle in United States (Warnick et al., 2001), but was at odds with Hernandez et al. (2002) who found no significant difference in milk yield between lame and non lame cows. Conversely, there was no significant reduction in milk volume, but there was a significant reduction in milk fat and protein yields between lame and non lame J cows (P<0.05). The subjective nature of the detection of clinical lameness by farmers and the subsequent potential delay in the treatment of lameness may well have been a contributory factor in the reduction of milk yield in this study. The milk yields in this study also showed a slightly higher percent loss in milk volume and milk solids production in J compared with F and FxJ cows.
In the majority (63.03%) of cases of the type of lameness involved in RCL incidences were not clearly identified. This indicates the requirement for better training and the development of automated recording systems to facilitate the identification of locomotion score and the specific causes of lameness. These developments have the potential to reduce the incidence of lameness and the associated costs and losses in animal productivity and welfare. Of the types of lameness that were recorded, the two primary causes of lameness in this study were foot rot (20.88%) and claw horn disorders (CHD) (16.09%), which included white line disease (7.78%), sole bruising (4.11%), sole penetration by foreign objects (3%) and finally hoof cracks (1.2%). While in some instances foot rot may well be related to CHD and the deterioration in claw horn structure and function. This may also be related with management factors such as poor track quality, long walking distances and animal handling which may well have contributed to the incidence of RCL in this study (Chesterton et al., 1989).

This study shows a higher incidence of RCL in F cows than in FxJ and J cows, which agrees with previous studies (Chesterton et al., 1989; Chesterton et al., 2008; Diaz-Lira et al., 2009). The higher incidence of clinical lameness in F than J and FxJ may be associated with lower claw horn quality and puncture resistance (Lethbridge et al., 2008), lactation phase and higher milk yield (Diaz-Lira et al., 2009). General heterosis for incidence of clinical lameness was -1.2% (favourable), which represent a 19% reduction in the mean incidence of lameness compared with purebred dairy breeds, indicating that first cross generation had lower incidence of RCL than the mean of the parental breeds. The effect of breed showed a significantly lower incidence (-1.9%) of RCL in Jersey compared to F breed. The genetic differences in claw traits, live weight and milk production traits between J and F are major reasons for differences in the incidence of RCL (Chesterton et al. 1989; Olmos et al. 2008) in dairy cattle.

**Conclusion**

There is sufficient evidence from this study to indicate that better recording systems and technological developments would elucidate a better understanding of the levels, types and factors that contribute to the incidence of lameness in dairy cattle in New Zealand. There is an effect of breed and heterosis in the incidence of RCL in New Zealand dairy cattle. Jersey cows had a 1.9% lower incidence of RCL than Friesian cows. The effect of heterosis in first-cross FxJ on the incidence of RCL, reduced RCL by 19% compared purebred dairy cattle. Furthermore, a significant reduction in milk yield was found in lame cows compared to non lame cows. This study suggests that crossbreeding can be used as an alternative mating system in New Zealand dairy cattle to exploit the breed and hybrid vigour for resistance to RCL.
Acknowledgments

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References


