Genetic analysis of incidence of recorded clinical lameness in New Zealand dairy cattle

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ABSTRACT

Herd records from 76,357 cows, collected during the 2005/06 to 2008/09 milking seasons from 155 herds in the Livestock Improvement Corporation (LIC) young sire progeny test scheme, were used to estimate genetic parameters and breed effects for incidence of recorded clinical lameness (RCL) in Holstein-Friesian, Jersey and crossbred dairy cattle. RCL was coded "1" for cows that presented at least one event of RCL at any day during the season and "0" for cows without RCL. Genetic parameters were estimated with a repeatability animal model across breeds. The average incidence of RCL per herd was 6.3% and ranged from 2% to 34%. Jersey cows had significantly ($P < 0.05$) lower incidence (6.0%) than Friesian cows (6.8%) but similar to crossbred cows (6.1%). The heritability estimate for incidence of RCL was 0.016 ± 0.003 (standard error) while the repeatability was 0.071 ± 0.005. Sire estimated breeding values for RCL ranged between -5 and 8% with Jersey sires showing the lowest values. These results suggest that selection for resistance to lameness will result in a low rate of genetic gain but using Jersey sires can be an alternative to increase genetic resistance to lameness in New Zealand dairy cattle.

Keywords: clinical lameness, genetic parameters, heterosis, crossbreeding parameters.

INTRODUCTION

Lameness in New Zealand dairy cattle is one of the most important health, welfare and economic (Chesterton *et al.,* 2008) problems in the industry, with an annual incidence ranging from 2% to 38% (Tranter & Morris, 1991). Lameness can be characterised as acute or chronic depending on severity and extent of the healing period. The causes of recorded clinical lameness (RCL) can be divided into infectious and non-infectious. The most frequently diagnosed causes of infectious clinical lameness include foot rot and digital dermatitis (Harris *et al.,* 1988), while the non infectious causes of clinical lameness include white line disease, hoof erosion, damage of joints and ligaments (Harris *et al.,* 1988) along with sole bruising, lesion formation and penetration.

The economic losses due to RCL can be categoried into direct and indirect costs. The direct costs include financial losses due to treatment costs, culling and prevention of the disease (Esslemont & Kossaibati, 1997). Tranter & Morris (1991) reported the average cost was $94 per case of clinical lameness in New Zealand dairy herds. The indirect costs include reduced milk production, infertility and loss of body condition in lame animals. Taking into account both direct and indirect costs, the annual financial loss due to lameness is significant and is costing farmers in terms of reduced profits (Esslemont & Kossaibati, 1997). Lameness is regarded as a multifactorial disorder and the associated factors include environmental conditions (Chesterton *et al.,* 1989), genetics factors, breed (Chesterton *et al.,* 2008), nutrition (Westwood *et al.,* 2003) and herd management (Chesterton *et al.,* 1989).

Despite the considerable financial losses and negative animal welfare implications caused by lameness, there are limited numbers of studies that have investigated the effect of genetic parameters on the incidence of clinical lameness in New Zealand dairy grazing herds (Diaz-Lira *et al.,* 2009; Chesterton *et al.,* 2008; Gibbs, 2010). Much of the research on breed and heterosis effect on lameness has been completed in intensive production systems overseas (Olmos *et al.,* 2008) which are quite different from New Zealand dairy production systems. As result, the objective of this study was to estimate genetic parameters on the incidence of recorded clinical lameness in New Zealand dairy herds.

MATERIALS AND METHODS

Data

The records used in this study were provided by Livestock Improvement Corporation (LIC) and were collected during the 2005/2006 to 2008/2009 milk production seasons. The cows were the progeny of sires used for the young sire progeny test scheme at LIC. Individual cow records were collected and included farm location, calving date, lactation number, breed composition, date and type of lameness treatment. An incidence of RCL was coded "1" for cows that presented at least one event of clinical lameness on any day at risk during the season and "0" for unaffected cows. Cows that may

have experienced a low degree of lameness were not recorded because they did not receive any veterinary treatment. The farms that had less than 100 cows and cows with more than 10 lactations were omitted in the data set. The final data set consisted of 111,565 lactations from 27,442 Holstein-Friesian (F), 10,129 Jersey (J) and 38,786 Crossbred (FxJ) cows distributed in 155 dairy herds. Cows with breed composition of more than 87.5% or less than 12.5% F or J were described as pure breed and the rest were described as Crossbred. There were 261 contemporary groups; contemporary group was defined as the group of cows calving in the same herd and year. Some herds did not participate in all four years of the study because some herds drop out after just one year and new herds entered into the proving scheme.

The age structure of the 111,565 lactations was the following: 23, 19, 15, 12, 10, 8, 5, 4, 2 and 1% for lactation number from 1 to 10 respectively. The proportions of cows with 1, 2, 3 and 4 records were 66.1, 23.2, 9.1 and 1.56, respectively, resulting in an average of 1.5 records per cow.

Data analysis

A logistic regression analysis was applied by using the GENMOD procedure of SAS (2002) to determine the incidence of recorded clinical lameness in different lactations and breed groups. The model included the fixed effects of contemporary group (herd-year), breed group (F, J and FxJ), lactation number and month of calving.

Calculations of breed proportions and coefficients of breed heterozygosity

The breed composition of each cow was described in terms of proportions of F and J. The proportions of genes from each breed were calculated for each animal using the following formula (Jury *et al.,* 2010):

$$\alpha^p_i = (\alpha^s_i + \alpha^d_i)/2$$

where α^p_i is the proportion of genes from breed *i* in the progeny, α^s_i is the proportion of breed *i* in the sire and α^d_i is the proportion of breed *i* in the dam.

The coefficient of FxJ breed heterozygosity (het_{FxJ}) for each cow was calculated using the following formula (Jury *et al.,* 2010):

$$het_{FxJ} = \alpha^s_F \alpha^d_J + \alpha^s_J \alpha^d_F$$

where het_{FxJ} is a coefficient of expected breed heterozygosity between fractions of F and J in the progeny, α^s_F is a proportions of F in the sire, α^d_J is a proportion of J in the dam, α^s_J is a proportion of J in the sire and α^d_F is a proportion of F in the dam.

Model for estimation of genetic parameters

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

$$\mathbf{y = Xb + Za + Wp + e}$$

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 environment of cow effects, **a** is a vector of additive random animal effects, **p** is a vector of permanent environment of cow effects and **e** is the vector of random residual effects. The **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 the covariables of proportion of Jersey and coefficient of breed heterozygosity of FxJ.

The following assumptions were made: It was assumed the following expectations $\mathbf{E(y) = Xb, E(a) = 0}$ and $\mathbf{E(e) = 0}$ and variances $\mathbf{var(a) = A}\sigma^2_a \mathbf{= G,}$ $\mathbf{var(p) = I}\sigma^2_p$ and $\mathbf{var(e) = I}\sigma^2_e \mathbf{= R}$ and hence, $\mathbf{var(y) = ZAZ'}\sigma^2_a \mathbf{+ WI}\sigma^2_p\mathbf{W' + R}$ where σ^2_a, σ^2_p and σ^2_e are animal, permanent environment of cow and residual variances, respectively.

A is the numerator relationship matrix between all animals considered in the data set. The numerator relationship matrix was 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 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:

$$\begin{bmatrix} \mathbf{X'X} & \mathbf{X'Z} & \mathbf{X'W} \\ \mathbf{Z'X} & \mathbf{Z'Z} + \mathbf{A^{-1}}\alpha_1 & \mathbf{Z'W} \\ \mathbf{W'X} & \mathbf{W'Z} & \mathbf{W'W} + \mathbf{I}\alpha_2 \end{bmatrix} \begin{bmatrix} \mathbf{b} \\ \mathbf{a} \\ \mathbf{p} \end{bmatrix} \begin{bmatrix} \mathbf{X'y} \\ \mathbf{Z'y} \\ \mathbf{W'y} \end{bmatrix}$$

with $\alpha_1 = \sigma^2_e/\sigma^2_a$ and $\alpha_2 = \sigma^2_e/\sigma^2_p$ (Mrode, 2005) and $\mathbf{A^{-1}}$ is the inverse of numerator relationship matrix. Therefore, the mixed model equations for the best linear unbiased estimator (BLUE) of estimable function of **b** and the best linear unbiased prediction of additive random animal effects (**a**) and cow effects (**p**) were obtained as follows:

$$\begin{bmatrix} \mathbf{b} \\ \mathbf{a} \\ \mathbf{p} \end{bmatrix} = \begin{bmatrix} \mathbf{X'X} & \mathbf{X'Z} & \mathbf{X'W} \\ \mathbf{Z'X} & \mathbf{Z'Z} + \mathbf{A^{-1}}\alpha_1 & \mathbf{Z'W} \\ \mathbf{W'X} & \mathbf{W'Z} & \mathbf{W'W} + \mathbf{I}\alpha_2 \end{bmatrix}^{-1} \begin{bmatrix} \mathbf{X'y} \\ \mathbf{Z'y} \\ \mathbf{W'y} \end{bmatrix}$$

Heritability and repeatability of clinical lameness were calculated from the variance estimates obtained from the ASReml analysis.

Heritability (h^2) of the incidence of recorded clinical lameness was calculated as:

$$h^2 = \sigma^2_a / (\sigma^2_a + \sigma^2_p + \sigma^2_e)$$

Similarly, the repeatability (r) of the incidence of recorded clinical lameness was calculated as:

$$r = (\sigma^2_a + \sigma^2_p) / (\sigma^2_a + \sigma^2_p + \sigma^2_e)$$

RESULTS

Incidence of recorded clinical lameness

The mean incidence of RCL in this study was (6.3%) with values ranging from 2% to 34%. Jersey cows had a significantly lower (P<0.05) incidence (6.0%) than Friesian cows (6.8%) but similar to FxJ crossbred cows which were intermediate (6.1%).

Lactation number had a significant effect on the incidence of RCL (P <0.05). The lowest incidence of RCL occurred in the second lactation (3.3%) which was significantly different (P <0.05) to the average incidence in all lactations except the average incidence in the third lactation (3.8) (Figure 1). There was a consistent trend for the incidence of RCL to increase from the second to the tenth lactation.

FIGURE1: Incidence of recorded clinical lameness in different lactations.

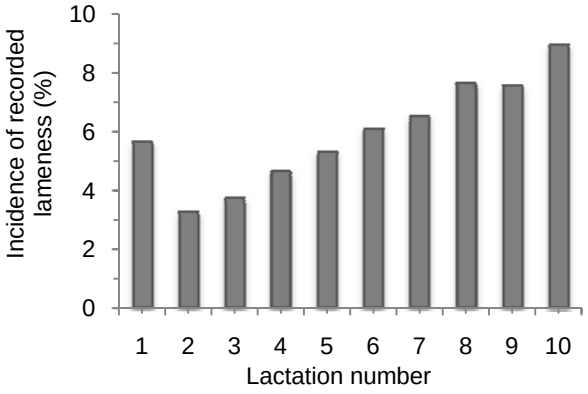
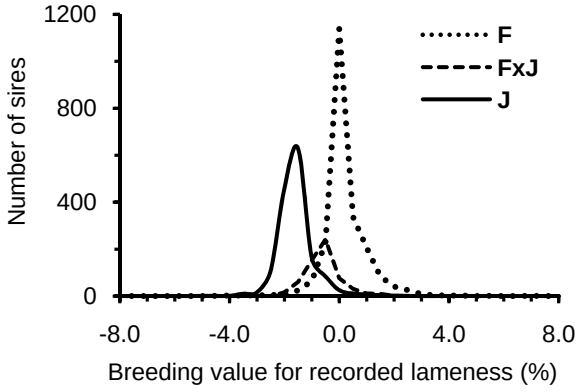


FIGURE 2: Distribution of sire estimated breeding values for incidence of recorded clinical lameness by breed. F = Holstein-Friesian; J = Jersey; FxJ = Crossbred.



Estimation of genetic parameters for incidence of recorded clinical lameness

The estimates of additive animal (σ^2_a), cow permanent environment (σ^2_p) and residual (σ^2_e) variances were 0.0009, 0.004 and 0.053 units, respectively, resulting in a total phenotypic variance of 0.057. The heritability estimate for incidence of RCL was 0.016 ± 0.003 (standard error), with a repeatability of 0.071 ± 0.005.

Distribution of sires breeding values for incidence of recorded clinical lameness

The distribution of sire breeding values by breed is shown in Figure 2. They ranged from -5 to +8%. There were 2,182 Holstein-Friesian, 1,517 Jersey and 600 FxJ crossbred sires. Friesian sires had higher breeding values for RCL than J, while FxJ sires had intermediate breeding values for RCL.

DISCUSSION

This study has identified a lower mean incidence of RCL in dairy herds participating in the LIC sire proving scheme compared with other studies reported in New Zealand using a limited number of records (Gibbs, 2010; Tranter & Morris, 1991), but the RCL was within the range of the reported incidences of lameness in New Zealand (Gibbs, 2010; Tranter & Morris, 1991). Potential reasons for the relatively low incidence of RCL in this study are firstly, the subjective and time consuming nature of the assessment of locomotion score, secondly, the subsequent detection of RCL, and thirdly, the lack of recording of the type of lameness for many cows with RCL.

Lactation number had a significant (P <0.05) effect on the incidence of RCL. The estimated RCL levels showed that cows in their first lactation were more likely to have a RCL incidence than cows in second lactation. Alban (1995) reported in Danish dairy cattle that cows in their first lactation had a higher incidence of lameness than cows during their following lactations. Lower incidence of RCL in the second lactation than in the first lactation can be explained by the possibility that only relatively healthy cows in their first lactation would have a chance to be selected for a second lactation. Studies have associated the longer period of time spend on concrete collecting yards by first lactation cows as the main risk factor contributing to the development of lameness in first lactation animals compared to later lactation cows (Sauter-Louis *et al.,* 2004). A greater incidence of RCL for cows in their first lactation compared to other subsequent lactations was consistent with previous studies in the United States (Warnick *et al.,* 2001) and in Finnish

Ayrshire dairy cows (Rajala-Schultz & Gröhn, 1999). On the other hand, this study found that older cows with eight to ten lactations had more incidences of RCL compared to younger cows with two to four lactations. The differences in incidence of RCL between older and young cows have been reported in Australia, where older cows (6 to 10 years of age) had an increased risk of RCL due to problems of white line disease, sole ulcers and ligament damage compared to younger cows (Jubb & Malmo, 1991).

The estimate of heritability (0.016) for RCL in this study was similar to the value reported by Berry *et al*. (2010) in Irish dairy cattle under grazing (h^2 = 0.04) but lower than the heritability estimate reported for cows managed in indoor feeding systems h^2 = 0.09 (Zwald *et al.,* 2004) and h^2 = 0.16 (Van Dorp *et al.,* 1998). The low estimate of heritability suggests that the variability in incidence of RCL between individual cows seems to be associated more with environmental factors rather than genetic factors. Previous studies in New Zealand dairy cattle have associated the incidence of lameness with environmental conditions, farm and herd size, and management practices (Chesterton *et al.,* 1989). Despite the low heritability of RCL, results from this study suggest breed differences and crossbreeding can be an alternative to reduce incidence of RCL. The comparison of sire breeding values for RCL showed significant differences between the breed groups of sires. The estimated breeding values for RCL ranged from -5% to +8%. The Jersey sires had the most favourable breeding values, followed by the crossbred sires, while Friesian sires had the lowest. The estimated breeding values for different breeds suggests that selection of specific sires across and within breeds can be a genetic alternative to increased genetic resistance to RCL in New Zealand dairy cattle.

The estimate of repeatability (0.07) for incidence of RCL obtained in this study was similar to the value reported by Berry *et al*. (2010) (r = 0.07) in Irish dairy cattle under grazing conditions. The estimates of repeatability of RCL was higher than the estimate of heritability, indicating the existence of permanent or non-additive genetic effects common to all lactations. However, the estimate of repeatability from this study must be considered with caution because the number of lactations per cow was only 1.5 and some herds dropped out recording events of lameness during the four milking seasons considered in this study. Warnick *et al.* (2001) associated culling bias with the increased risk of lameness of the same animals in different lactations. The cows with higher milk production potential were significantly more likely to remain in the herd regardless of clinical lameness problems compared to cows with low production. This selection and culling bias was considered a major problem contributing to repeated incidence of lameness in different lactations (Calavas *et al.,* 1996).

CONCLUSIONS

The current study has shown that the effect of RCL varies across lactations. The estimate of heritability for incidence of RCL in dairy cows was low and therefore selection for resistance to RCL disease will result in a low rate of genetic gain. However, this study provides evidence that there are significant breed differences for RCL suggesting that Jersey sires can be a genetic alternative to reduce incidence of RCL in New Zealand dairy herds.

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