Genetic Relationship between Clinical Mastitis and Several Traits of Interest in Spanish Holstein Dairy Cattle

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Abstract

Genetic parameters of clinical mastitis (CM) and number of cases of mastitis (NCM) per lactation, as well as the association with five production traits, nine type traits, and four functional traits have been estimated using linear models in a population of 9,179 Spanish Holstein cows with 17,666 lactations. Estimated heritability for mastitis traits was 0.04-0.05 and the genetic correlation between them was 0.93. The genetic correlation between lactation somatic cell score (LSCS) and CM and NCM was 0.85 and 0.76, respectively, meaning that LSCS is not representing exactly mastitis infections. There was an unfavorable genetic correlation between production traits, days open, LSCS and mastitis, while a shallow and well attached udder helps reducing mastitis events. Thus, it is recommended to include clinical mastitis in the breeding goal and the most correlated traits in an udder health index in order to achieve the maximum mastitis resistance.

Keywords: clinical mastitis, conformation, functionality, production

1. Introduction

Mastitis, jointly with fertility and lameness, is one of the most worldwide concerns in the dairy industry because of the detriment of profit and animal welfare (Shim et al., 2004; Wolfová et al., 2006). Therefore, genetic selection programs are focusing on reducing diseases and improving functional traits (Rauw et al., 1998; Zwald et al., 2006). Routinely recording of clinical mastitis is not implemented in many countries whereas it is well-established in the Nordic countries since more than 40 years (Carlen, 2008).

In Spain, since 2003 genetic evaluation of LSCS has been implemented as indirect way to select for udder health along with the Udder Composite index (ICU), which combine Fore udder attachment, Rear udder height, Central ligament, Udder depth and Front teat placement. Up to now, Spanish selection index ICO includes only LSCS and conformation traits as indicators of resistance to mastitis.

Knowledge of genetic parameters of mastitis and assessment of the economic importance of the disease is required when developing a breeding program for improving udder health. Because the index is about to be update, the aim of this study is to estimate genetic parameters of clinical mastitis traits and the association with other important traits in a Spanish Holstein population.

2. Materials and Methods

Data

Mastitis records were provided by the regional milk-recording associations from the Basque Autonomous Region, Navarra, and Gerona as in Pérez-Cabal et al. (2009). Clinical mastitis (CM) was diagnosed either by farmers or their veterinarians as secretion of visually abnormal milk from one or more quarters or inflammation of the udder tissue. Once a cow was diagnosed, it was recorded in a special form. For the same cow, two cases of mastitis
were considered as different when the dates of diagnosis did not occur within a week, and the treatment periods did not overlap, regardless of the number of quarters that were affected. Mastitis traits studied were CM, if the lactation presented at least one case, and number of cases of clinical mastitis (NCM) per lactation. Type trait and milk yield data, as well as pedigree information, were provided by the Spanish Holstein Association (CONAFE). Genetic correlations between either CM or NCM and the following traits were estimated. Production traits considered in this study were 305d standardized milk yield, 305d standardized fat yield, 305d standardized protein yield, fat and protein contents. Udder traits included in the study were udder texture (TEXT), fore udder attachment (FUA), rear udder height (RUH), rear udder width (RUW), central ligament (CL), udder depth (UD), front teat placement (FTP), rear teat placement (RTP), teat length (TL). Finally, the functionality traits involved were days open (DO), lactation somatic cell score (LSCS), as the arithmetic mean of monthly test day somatic cell count transformed using a base-2 logarithmic function (Ali and Shook, 1980), longevity (LONG) defined as productive life and measured as days between first calving and last test-day control recorded, and milking speed (MS) evaluated as close to the 2nd month of first lactation as possible, with three possible scores (1 was assigned for cows with fast milking speed, 2 for cows with average milking speed and 3 for cows with slow milking speed).

In order to obtain adequate data sets for the statistical analyses, 21,396 original lactation records were edited. Missing values were considered in analyses. The study was carried out with records from 17,666 lactations of 9,179 cows that calved from January 2004 through October 2011 in 27 herds.

2.1. Statistical Models

Both CM and NCM were analyzed using linear models. The model used for production traits, LSCS, DO, CM, and NCM was:

\[ y_{ijklmn} = h y_i + C M_j + L A E_k + i d_l + p e_m + \varepsilon_{ijklmn} \]

For LONG the model fitted was:

\[ y_{ijklmn} = h y_{1i} + C M_j + L A E_{1k} + F O R M + i d_l + \varepsilon_{ijklmn} \]

The model for analysis of Milking speed was:

\[ y_{ijklmn} = h y_{2i} + C M_j + L A E_{2k} + D I M_l + i d_l + \varepsilon_{ijklmn} \]

and for type traits, the following model was fitted:

\[ y_{ijklmn} = h v c_i + L S_j + L A E_{3k} + i d_l + \varepsilon_{ijklmn} \]

where \( y_{ijklmn} \) is the performance of each of the traits; for the first model, \( h y_i \) is the fixed effect of the \( i^{th} \) herd-year of calving (209 levels); \( C M_j \) is the fixed effect of the \( j^{th} \) month of calving (12 levels); \( L A E_k \) is the fixed effect of the \( k^{th} \) lactation-age (52 levels); \( i d_l \) is the additive genetic effect of \( l^{th} \) animal (23,178 animals); and \( p e_m \) is the permanent environmental effect of the \( m^{th} \) cow (9,179 cows). In the second model, \( h y_{1i} \) is the fixed effect of the \( i^{th} \) herd-year of first calving (194 levels); \( L A E_{1k} \) is the fixed effect of the \( k^{th} \) first lactation-age (24 levels); \( D I M_l \) is the fixed effect of \( l^{th} \) day in milk grouped in 5 levels. In the model for type traits, \( h v c_i \) is the fixed effect of the \( i^{th} \) herd-visit-classifier (259 levels); \( L S_j \) is the fixed effect of the \( j^{th} \) lactation stage (12 levels);

The joint distribution of random effects included in the models (except for the type traits, milking speed and longevity because there were not repeated measures) was as follows:

\[
\begin{bmatrix}
  i d \\
p e
\end{bmatrix}
\sim N\left(0, \begin{bmatrix}
  A \sigma^2_{i d} & 0 \\
  0 & \sigma^2_{p e}
\end{bmatrix} \right)
\]

where \( i d \) and \( p e \) are the vectors of animal and permanent environmental effects, respectively, and \( \sigma^2_{i d} \) and \( \sigma^2_{p e} \) are the corresponding variances; \( A \) is the additive relationship matrix, and \( I \) is an identity matrix of appropriate order.
The distribution of model residuals is \( \varepsilon \sim N(0, \sigma^2) \).

Genetic parameters were estimated by REML using the VCE 6.0 software (Groeneveld et al., 2008).

### 3. Results and Discussion

Mastitis incidence among all lactations into the 27 herds of the study was 24.9%.

#### 3.1. Estimate of CM and NCM heritabilities

Estimated heritability for CM was 0.04 and 0.05 for NCM. Repeatabilities were 0.05 and 0.11, respectively. Genetic correlation between them was 0.93. Pérez-Cabal et al. (2009) reported a slightly larger heritability (0.07) using a linear model for a smaller Spanish Holstein population. Most studies in literature have reported values ranging from 0.01 to 0.07 when a linear model is fitted (Hansen et al., 2002; Carlén et al., 2005; Negussie et al., 2008; Vazquez et al., 2009). When the binary nature of CM is taken into account fitting threshold models, higher estimated heritability has been reported, ranging from 0.06 to 0.12 (Heringstad et al., 2003; Zwald et al., 2006; Negussie et al., 2008; Pérez-Cabal et al., 2009). Estimates of heritability for NCM were slightly lower than other studies using the linear model (around 0.10), such as Pérez-Cabal et al. (2009) and Vazquez et al. (2009) with different data edits. Wolf et al. (2010) reported 0.11 as heritability for NCM adjusted to a lactation length of 305d.

#### 3.2. Estimates of genetic correlations

As expected, there was an unfavorable genetic correlation between production traits and clinical mastitis in agreement with literature (e.g., Hinrichs et al., 2005). However, the genetic correlation with protein content was negative for both CM and NCM, as well as the correlation between fat content and NCM. (Table 1).

### Table 1. Genetic correlations \( (r_g) \) and standard errors (s.e.) between clinical mastitis traits and production traits.

<table>
<thead>
<tr>
<th></th>
<th>CM</th>
<th>NCM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Milk yield (kg)</td>
<td>0.34 0.06</td>
<td>0.34 0.05</td>
</tr>
<tr>
<td>Fat yield (kg)</td>
<td>0.10 0.04</td>
<td>0.12 0.04</td>
</tr>
<tr>
<td>Fat content (%)</td>
<td>0.22 0.03</td>
<td>-0.18 0.03</td>
</tr>
<tr>
<td>Protein yield (kg)</td>
<td>0.32 0.06</td>
<td>0.26 0.05</td>
</tr>
<tr>
<td>Protein content (%)</td>
<td>-0.10 0.03</td>
<td>-0.17 0.04</td>
</tr>
</tbody>
</table>

CM: clinical mastitis per lactation as a binary trait; NCM: number of cases per lactation.

The most correlated type traits with clinical mastitis were UD and RUW, followed by TEXT, FUA, teat placement, and CL (Table 2). As expected, a shallow and well attached udder favors the reducing of mastitis events (e.g., Zwald et al., 2004). Regarding functional traits (Table 3), genetic correlations with LSCS were high, 0.85 for CM and 0.76 for NCM (Carlén et al., 2004; Odegard et al., 2004) although, as Heringstad et al. (2006) reported, it does not mean LSCS is the perfect indicator of clinical mastitis.
Negative genetic correlations with MS showed that the faster milking speed the higher risk of clinical mastitis because teats are more exposed to entry of pathogens (Boettcher et al., 1998). Clinical mastitis was associated with an increase of fertility problems (genetic correlations of 0.34 and 0.40 with CM and NCM, respectively).

Genetic correlation between longevity and CM was -0.27, lower than estimate reported by Govignon et. al. (2012). Besides, NCM shows no genetic correlation with longevity (-0.01). Productive live in this study was not adjusted for production traits, so the effect in culling risk due to mastitis most likely is masked by selection for production level.

Table 3. Genetic correlations ($r_g$) and standard errors (s.e.) between clinical mastitis traits and functional traits.

<table>
<thead>
<tr>
<th></th>
<th>CM</th>
<th>NCM</th>
</tr>
</thead>
<tbody>
<tr>
<td>LSCS</td>
<td>0.85</td>
<td>0.76</td>
</tr>
<tr>
<td>DO</td>
<td>0.34</td>
<td>0.40</td>
</tr>
<tr>
<td>LONG</td>
<td>-0.27</td>
<td>-0.01</td>
</tr>
<tr>
<td>MS</td>
<td>-0.45</td>
<td>-0.32</td>
</tr>
</tbody>
</table>

CM: clinical mastitis per lactation as a binary trait; NCM: number of cases per lactation; LSCS: lactation somatic cell score; DO: Days open; LONG: Longevity; MS: milking speed.

4. Conclusions

The results obtained for CM and NCM from linear models are in agreement with literature. Both CM and NCM seemed to be the same trait. The LSCS was strongly associated to clinical mastitis incidence, however they could not be considered as the same trait. Clinical mastitis was associated to high production levels, fertility problems, easy milking, and poor udder attachments. Thus, it is recommended to include clinical mastitis in the breeding goal and the most favorable correlated traits in an udder health selection index in order to achieve the maximum mastitis resistance.

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References


