Genetic Correlation between Test-Day Electrical Conductivity of Milk and Clinical Mastitis

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ABSTRACT

Electrical conductivity (EC) of milk is an indicator of mastitis, and could, if it shows genetic variation and is genetically correlated to mastitis, be used in a breeding program where selection for reduced mastitis resistance is included. In this study, daily measurements of EC and mastitis were analyzed with a bivariate model. For EC, the estimated heritability was high (0.22 to 0.39), while for mastitis the heritability was low (0.013). The genetic correlation between EC and mastitis was estimated to be 0.75, and genetic improvement of mastitis resistance should be feasible through selection for reduced EC.

INTRODUCTION

Reducing the incidence of mastitis through genetic selection is of great interest from both an economical and an animal welfare point of view. Except in the Nordic countries, where clinical mastitis is recorded and reported, selection for mastitis resistance is done using traits that are genetically correlated to mastitis. Information on SCS is included in the sire evaluation procedures in several countries (Interbull, 1996). However, using SCS for genetic evaluation has some disadvantages, such as the low recording frequency. Some extra costs and labour are connected to the sampling of SCC as well.

Electrical conductivity (EC) of milk was introduced as an indicator trait for mastitis in the 1970s and it has been used for detection of mastitis the last decades (Hamann and Zecconi, 1998). If a cow suffers from mastitis, the concentration of Na⁺ and Cl⁻ in the milk increases, leading to increased EC of milk from infected quarters (Kitchen, 1981). Electrical conductivity is cheap and easy to record, and most automatic milking systems have sensors installed for daily measuring EC of milk. However, for EC to be used in a breeding program, EC must show genetic variation and be genetically correlated to mastitis. In a preliminary analysis, Rogers (2002) reported a genetic correlation between lactation means of EC and clinical mastitis of 0.65 and 0.80 in first and second lactation cows, respectively. The objective of this study was to estimate genetic (co)variance components for test-day EC and mastitis.

MATERIAL AND METHODS

Data and Definition of Traits
Data consisted of daily records of EC and health status from four dairy herds in Florida between June 1994 and June 1998. About 1500 Holstein cows in the first lactation, sired by 125 bulls and calving from age 20 to 32 months, were included in the study. Daughter group size ranged from 1 to 186. Descriptive statistics of the data set are presented in Table 1. Records before DIM 6 and after DIM 305 were omitted. Due to the relatively short average lactation length, average number of EC records per cow was only 200. All herds used the PCDART management system, available from Dairy Records Management Services (Raleigh, North Carolina) and mastitis was recorded by the herds in a database. Electrical conductivity was measured in millimho (mmho) in composite milk from every milking with the Afikim computerized milking and management system (SAE Afikim, Kibbutz Afikim, Israel).
Table 1. Descriptive statistics of the data.

<table>
<thead>
<tr>
<th>Description</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cows</td>
<td>1507</td>
</tr>
<tr>
<td>Average lactation length (days)</td>
<td>209</td>
</tr>
<tr>
<td>Sires</td>
<td>125</td>
</tr>
<tr>
<td>Mean daughters per sire (min, max)</td>
<td>12.1 (1,186)</td>
</tr>
<tr>
<td>EC(^1) records</td>
<td>300,700</td>
</tr>
<tr>
<td>Mean EC records per cow</td>
<td>199.5</td>
</tr>
<tr>
<td>Mean EC records per herd-test-day class</td>
<td>140.5</td>
</tr>
<tr>
<td>Mean EC records per DIM</td>
<td>1002.4</td>
</tr>
<tr>
<td>Test-days with mastitis</td>
<td>6483</td>
</tr>
<tr>
<td>Mastitis frequency on lactation basis(^3), %</td>
<td>32.2</td>
</tr>
<tr>
<td>Mean EC (SD) for all test-days</td>
<td>11.8(^1) (1.36)</td>
</tr>
<tr>
<td>Mean EC (SD) for test-days where mastitis=0</td>
<td>11.7(^1) (1.32)</td>
</tr>
<tr>
<td>Mean EC (SD) for test-days where mastitis=1</td>
<td>13.0(^1) (2.14)</td>
</tr>
</tbody>
</table>

\(^{1}\)Electrical conductivity

\(^{2}\)In millimho in composite milk, daily averages

\(^{3}\)Percent of cows having one or more incidence of mastitis during the lactation

Daily averages of EC were used. Electrical conductivity increases for a number of days when a cow gets mastitis. As a consequence, EC records were considered to be outliers if they were 40% higher than the previous and the following day's EC. Of a total of 302,755 EC records, 2055 were omitted. Udder health status (mastitis or no mastitis) was recorded every day from DIM 6 to the last day of lactation. For each day, the cow was regarded as having clinical mastitis if the cow was treated for clinical mastitis or clinical mastitis was observed, but not treated (t=0). Furthermore the cow was assumed to have mastitis in the period 2 days before to 5 days after the mastitis was detected (t-2, t-1, t+1, t+1 to t+5). If a new episode of clinical mastitis was detected within 10 days after the previous episode, the cow was assumed to have mastitis in the period 2 days before the first episode to 5 days after the last episode.

Statistical Analysis

A bivariate analysis was carried out using an animal model with repeated measurements. Electrical conductivity was modeled with an intercept for the additive genetic effect and a fourth-order Legendre polynomial for the permanent environmental effect. For mastitis, a repeatability model without random regressions was used. The permanent environmental variance of EC and mastitis was assumed to be uncorrelated. The following model was used:

\[
Y_{ijkl} = A_i + HTD_j + DIM_k + \sum_{n=0}^{4} p_{eln}Z_{kn} + a_l + e_{ijkl}
\]

where;

\[Y_{ijkl} = \text{observation of test-day record of EC or CM of cow } l;\]
\[A_i = \text{fixed effect of age at first calving class } i (i = 1, \ldots, 12);\]
\[HTD_j = \text{fixed effect of herd-test-day class } j (j = 1, \ldots, 2147);\]
\[DIM_k = \text{fixed effect of DIM class } k (k = 1, \ldots, 300);\]
\[Z_{kn} = \text{nth order Legendre polynomial for DIM } k; \text{ where } n = \{0, \ldots, 4\}\]
\[p_{eln} = \text{random regression coefficient on } Z_{kn} \text{ for permanent }\]
\[e_{eln} = \text{random additive genetic effect of cow } l, \text{ and}\]
\[e_{ijkl} = \text{random residual.}\]

Estimation of (co)variance components for all models was carried out using the AI-REML algorithm included in the DMU-package (Madsen and Jensen, 2000).
RESULTS AND DISCUSSION

Mean EC (Table 1) over all test-days is equal to means obtained by Norberg et al. (2004b). For test-days with mastitis, EC was somewhat higher. This agrees well with Norberg et al. (2004a), where both clinical and subclinical infected cows showed a significant higher level of EC. Heritabilities for test-day EC level ranged from 0.21 to 0.39 during the lactation (Table 2). The permanent environmental variation was largest in the beginning of the lactation and reached the nadir in mid lactation. Consequently, the heritability was largest from approximately DIM 50 to 200. These results agree well with Norberg et al. (2004b), who used random regression models as well to estimate genetic and phenotypic parameters for EC from an extended data set of what was used in the present study. In their study, the heritability estimate was 0.28 estimated with a repeatability model and the standard error was fairly small (0.06).

Table 2. Estimated parameters from the bivariate genetic analysis of electrical conductivity (EC) and mastitis. Standard errors (SE) of the estimates are given within brackets.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>EC</th>
<th>EC and mastitis</th>
<th>mastitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Additive genetic (co)variance</td>
<td>0.667</td>
<td>0.011</td>
<td>0.0003</td>
</tr>
<tr>
<td>Permanent environmental (co)variance</td>
<td>0.54-2.01²</td>
<td>0.0029</td>
<td></td>
</tr>
<tr>
<td>Residual (co)variance</td>
<td>0.487</td>
<td>0.016</td>
<td>0.019</td>
</tr>
<tr>
<td>Heritabilities and genetic correlation(SE)</td>
<td>0.22 - 0.39²</td>
<td>0.75 (0.13)</td>
<td>0.013</td>
</tr>
<tr>
<td>Residual correlation (SE)</td>
<td>0.17 (0.002)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

¹ Estimates given are the range during lactation

For mastitis the estimated heritability was 0.013, which is low, but in agreement with previous studies (Emanuelson, 1988; Nash et al., 2000). A higher heritability was expected due to the intense recording of udder health, but a relatively small number of cows with records may explain the results.

The estimated genetic correlation between EC and mastitis in this study is 0.75, which is somewhat higher than those presented by Rogers (2002). In the latter study, genetic correlations between lactation means of EC and clinical mastitis were 0.65 and 0.80 for first and second lactation, respectively. The trait definition and the model used may explain the differences in the results. However, the standard error was relatively large in this study, and the difference between the two studies may not be real. Additionally, the assumption about uncorrelated permanent environmental between the two traits may result in overestimation of the genetic correlation.

In our study, both EC and mastitis were treated as Gaussian traits, and analyzed with a linear model. Theoretically, a threshold model should be more appropriate for analysis of binary response data (Gianola, 1982), and a majority of the most recent analyses of clinical mastitis are performed on the underlying scale, using threshold models. Estimates are then obtained for the liability to mastitis, and heritabilities obtained with a threshold model are generally somewhat higher than those obtained with a linear model. Threshold models have also been used for multitrait analyses of continuous and binary traits, assuming a covariance structure between the continuous trait and the underlying liability for the binary trait. However, for traits such as EC or SCC this assumption is not necessarily correct, as pointed out by Ødegård et al. (2004). Electrical conductivity of milk increases as a result of a bacterial infection, and liability to mastitis is not expected to affect the EC, unless the cow gets an infection. Hence, both genetic and phenotypic correlations between EC and clinical mastitis probably arise mainly as a result of a direct relationship between EC and the classes of clinical mastitis (0/1), rather than as a result of a covariance structure on the underlying scale. Therefore, using a threshold model for clinical mastitis and a linear model for EC may not necessarily result in more accurate estimates of the genetic correlation between the traits, compared to using a bivariate linear model. However, mixture models, which account for different distribution of data among infected and non-infected cows, are probably the most optimal models for analyses of such traits (Gianola et al., 2004), and should be considered in the future.
CONCLUSION

Electrical conductivity has a high genetic correlation with clinical mastitis, and therefore has potential as an indicator trait for selection to reduce the incidence of mastitis in breeding programs. The possibility of utilizing daily EC records may have an advantage compared to using SCC, because EC records more likely will be collected on the day of a mastitis infection.

REFERENCES


Norberg, E., J. Ødegård, and P. Madsen. 2004c. Comparison of variance components for test-day electrical conductivity of milk and test-day somatic cell score for first lactation cows in an experimental herd. (Submitted to Acta Agric. Scand.).
