Short Communication: Genetic Correlation Between Test-Day Electrical Conductivity of Milk and Mastitis

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ABSTRACT

Electrical conductivity (EC) of milk is an indicator of mastitis. If EC shows genetic variation and is genetically correlated to mastitis, it could be used in a breeding program that includes selection for improved mastitis resistance. In this study, daily records of EC and mastitis from about 1,500 Holstein cows were analyzed. A bivariate animal model was used for estimation of (co)variance components, including fixed effects of age of calving, herd-test-day, and days in milk, in addition to random additive genetic effects and permanent environmental effects. For EC, the estimated heritability was moderate (0.22 to 0.39), whereas 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.

Key words: dairy cattle, mastitis, electrical conductivity, genetic correlation

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 based on traits that are genetically correlated to mastitis; for example, SCC and udder conformation traits. Information on SCS is included for sire evaluation procedures in several countries (Interbull, 1996). However, with current technology, using SCS for genetic evaluation has some disadvantages, such as the low recording frequency. In addition, some extra costs and labor are connected to the sampling of SCC. Electrical conductivity (EC) of milk was introduced as an indicator for mastitis in the 1970s and has been used for detection of mastitis (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 of EC of milk. However, for EC to be useful in a breeding program in which mastitis resistance is included, EC must show genetic variation and be genetically correlated to udder health. 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. However, when using lactation means of EC, the effect of an infection in the udder on EC will be diluted due to the relatively small number of days that the cow may be infected during the lactation. Therefore, the objective of this study was to estimate genetic correlation between test-day EC and clinical mastitis.

Data used for the analysis were a subset of data used in Norberg et al. (2004b), containing daily records of EC and health status from 4 dairy herds in Florida collected from June 1994 to June 1998. About 1,500 Holstein cows in first lactation, calving from age 20 to 32 mo, were included in the study. The cows were sired by 125 bulls, and daughter group size ranged from 1 to 186. Records between DIM 6 and 305 were used, but due to the relatively short average lactation length, average number of EC records per cow was only 200. Cows were milked twice a day, and EC was measured in millimho (mmho; equivalent to milliSiemens) in composite milk from every milking using the Afikim computerized milking and management system (SAE Afikim, Kibbutz Afikim, Israel). Daily sums of EC from each cow were used for the statistical analysis. Because EC typically increases for a number of days before and after the cow gets an udder infection, EC records were considered to be outliers if they were 40% higher than the previous day's and the following day's EC record. Of 302,755 records of EC, 2,055 were omitted. Outliers of EC were not found for cows regarded as infected. Udder health status for each cow (mastitis or no mastitis) was recorded every day by the farm management staff, and records from DIM 6 to 305 or the last day of

Received June 14, 2005.

Accepted October 14, 2005.

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Table 1. Descriptive statistics of animals and electrical conductivity (EC) data

Description	
Cows	1,507
Average lactation length (d)	209
Sires	125
Mean daughters per sire (min and max)	12.1 (1 and 186)
Daily EC records	300,700
Mean EC records per cow	199.5
Mean EC records per herd-test-day class	140.5
Mean EC records per DIM	1,002.4
Test days with mastitis	6,483
Mastitis frequency on lactation basis, ¹ %	32.2
Mean EC (SD) for all test days ²	11.8 (1.36)
Mean EC (SD) for test days ² (mastitis = 0)	11.7 (1.32)
Mean EC (SD) for test days ² (mastitis = 1)	13.0 (2.14)

¹Percentage of cows having one or more incidence of mastitis during the lactation.

²EC (in millimho) in composite milk, daily averages.

lactation were used in the analysis. A cow was regarded as having mastitis if clinical signs of an infection were observed. Furthermore, the cow was assumed to have mastitis in the period 2 d before to 5 d after the first day the cow showed clinical signs. If a “new” case of mastitis was detected within 10 d after the previous episode, the cow was assumed to have mastitis in the period 2 d before the first episode to 5 d after the last episode. The cow was assumed healthy for the remainder of the lactation. Descriptive statistics of the data set are presented in Table 1.

A bivariate analysis was carried out with a test-day animal model, utilizing daily records of EC and infection status. Electrical conductivity was modeled with an intercept for the additive genetic effect, and a fourth-order Legendre polynomial for the permanent environmental effect. This model was chosen based on results from Norberg et al. (2004b), and what was computationally possible. For mastitis, a repeatability model without random regressions was used. Only a model where the permanent environmental variance of EC and mastitis was assumed uncorrelated would converge. The model was as follows:

$$Y_{ijkl} = A_i + \text{HTD}_j + \text{DIM}_k + \sum_{n=0}^4 \text{pe}_{ln} Z_{kn} + a_l + e_{ijkl}$$

where Y_{ijkl} = observation of test-day record of EC or mastitis of cow l ; A_i = fixed effect of age at first calving class i ($i = 1, \dots, 12$); HTD_j = fixed effect of herd-test-day class j ($j = 1, \dots, 2147$); DIM_k = fixed effect of DIM class k ($k = 1, \dots, 300$); $Z_{kn} = n$ th order Legendre polynomial for DIM k , where $n = \{0, \dots, 4\}$; pe_{ln} = random regression coefficient on Z_{kn} , for permanent environmental effect of cow l ; a_l = random additive genetic effect of cow l ; and e_{ijkl} = random residual.

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

Mean EC (Table 1) over all test days was 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 clinically and subclinically infected cows showed a significantly 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 at the beginning of the lactation and reached the nadir in midlactation. Consequently, heritability was largest from approximately DIM 50 to 200. These results are similar to those in Norberg et al. (2004b), in which the heritability was 0.28 (± 0.06) estimated with a repeatability model. For mastitis, the estimated heritability was 0.013, which is somewhat lower than estimates found by Emanuelson (1988) and Ødegård et al. (2004). However, our estimates are based on daily test-day records; and transforming these estimates to a lactation base will result in a heritability of 0.09. This result is somewhat higher than those found in the literature, but seems reasonable due to the daily recording of mastitis in this study. The estimated genetic correlation between EC and mastitis in this study was 0.75, which is, considering the standard error, similar to that presented by Rogers (2002). No other estimates of the genetic association between EC and mastitis have been published, but for comparison, the genetic correlation between EC and SCS is found to be 0.70 on average (Mrode and Swanson, 1996). However, the assumption about uncorrelated permanent environmental variation between the 2 traits in this study may result in a slightly overestimated genetic correlation because some of the permanent environmental covariance may have been captured into the genetic covariance. From a biological point of view, a model with the 2 permanent environmental variances would have been preferred, but as mentioned, this model would not converge.

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 have been 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

Table 2. Estimated parameters from the bivariate genetic analysis of electrical conductivity (EC) and mastitis

Parameter	EC	EC and mastitis ¹	Mastitis
Additive genetic (co)variance	0.667	0.011	0.0003
Permanent environmental (co)variance	0.54–2.01 ²	—	0.0029
Residual (co)variance	0.487	0.016	0.019
Heritabilities and genetic correlation	0.22–0.39 ²	0.75 (0.13)	0.013
Residual correlation	—	0.17 (0.002)	—

¹Standard errors of the estimates are in parentheses.

²Estimates given are the range during lactation.

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 because 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 because of a direct relationship between EC and clinical mastitis, rather than because of an association 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 with using a bivariate linear model. However, mixture models, which account for different distribution of data among infected and noninfected cows, are probably the most desirable models for analyses of such traits (Gianola et al., 2004), and should be considered in the future.

The possibility of utilizing daily EC records may be an advantage compared with using SCC, because daily EC records are more likely to be collected in periods in which the cow has a mastitis infection. Considering the strong genetic correlation with mastitis found in this study, EC could be used as an indicator trait for selection to reduce the incidence of mastitis. However, further investigations on a larger number of animals are needed to confirm the association between EC and mastitis before implementing EC in a breeding program.

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