Genetic Relationships Between Two Differently Defined Somatic Cell Traits And Clinical Mastitis In A Test-Day Model

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Introduction

Udder health traits are of major importance in dairy cattle breeding programs. Mastitis is a commonly occuring disease, and one of the most costly. As clinical cases of mastitis are not routinely recorded in many parts of the world, the most common indirect selection trait used is somatic cell count (SCC) (*e.g.* Heringstad *et al.* (2000)). SCC, like milk production, is a trait that is affected by and changes with time such as stage of lactation and season. The genetic correlation between SCC measured in different parts of lactation is less than unity (Koivula *et al.* (2004); Negussie *et al.* (2007)). Several countries have moved towards the use of random regression models (RRM) for genetic evaluation of longitudinal data such as milk yield and also SCC.

SCC is defined as concentration of cells per ml of milk. It has been shown that dilution effects can affect the relationship between milk yield and SCC (Green *et al.* (2006)), meaning that high yielding cows can appear to perform better than they really are. It is possible that the total amount of cells in the milk (SCC multiplied by test day milk yield) gives a better indication of the amount of infection in the udder. We have not found any estimates in the literature between total amount of cells and clinical cases of mastitis.

The aim of this study was to estimate genetic parameters for somatic cell count and the total amount of cells in the milk with use of a random regression test-day model. We also wanted to investigate the relationship between these two cell count measures and clinical cases of mastitis.

Material and methods

Data. The study was based on data from the Swedish milk recording system. Data contained monthly test-day records from first parity cows of the Swedish Holstein breed, calving between 1999 and 2000. Records from day 5 to 400 in lactation were included in the analyses. We also had information on veterinary treatments for clinical mastitis for all cows. After editing, the data included records from 41,520 cows with on average 9.2 records per cow.

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Trait definitions. Data included test-day records of somatic cell count, measured in 1000 cells/ml. From these records we defined new records of the concentration of cells in the milk (somatic cell score, SCS) and the total amount of cells in the milk (CELL). SCS was expressed as the $\log_e$-transformation of SCC, and CELL was expressed as the $\log_e$-transformation of (SCC x test day milk yield). Two mastitis traits were defined: veterinary treatments from 15 days before calving to day 50 in lactation and veterinary treatments from day 51 to 300 in lactation, both defined as binary traits: 0 (no treatment) or 1 (at least one treatment).

Models and statistical analysis. Genetic parameters and breeding values for SCS and CELL were estimated using a random regression test-day sire model with Legendre polynomials. Based on maximum likelihood ratio test we found that the model fitting the data best included a third order (cubic) Legendre polynomial for the genetic sire effect and a fourth order polynomial for the permanent cow effect:

$$y_{ijkl} = htd_i + age_j + \sum_{m=0}^{10} b_m D^m + \sum_{n=0}^{4} pe_n D^n + \sum_{q=0}^{3} s_q D^q + e_{ijkl} \quad (1)$$

where
y_{ijk} is the observed test-day record for SCS or CELL at day D in lactation, htd_i is the fixed effect of the i^{th} herd-test-day, age_j is the fixed effect of the j^{th} age at calving (20-38 mo). The first regression term describes the mean lactation curve, the second describes the non-genetic permanent environmental effect of the cow, the last regression describes the genetic effect of the sire, and e_{ijklm} is the random residual effect.

Genetic parameters and breeding values for mastitis were estimated using a mixed linear sire model. The model included:

$$y_{ijklm} = hy_i + ym_j + age_k + s_l + e_{ijklm} \quad (2)$$

where
y_{ijk} is the observed value for mastitis (0 or 1), hy_i is the fixed effect of the i^{th} herd by year of calving, ym_j is the fixed effect of the j^{th} year by month of calving, age_k is the fixed effect of the k^{th} age at calving (20-38 mo), s_l is the random genetic effect of the l^{th} sire and e_{ijklm} is the random residual effect. Variance and covariance components were estimated using an AI-REML algorithm (Jensen et al. (1997)) in the DMU package. Correlations between SCC/CELL and mastitis traits were estimated in a bivariate model combining models (1) and (2).

Results and discussion

On average 6% of all cows were treated for clinical mastitis from day -15 to 50 in lactation, and the prevalence was the same from day 51 to 300. The average lactation curves show, in agreement with previous studies (*e.g.* Haile-Mariam (2001)) an inverted shape relative to the milk production curve. We had predicted that the lactation curve for CELL might have a flatter shape because of this “inverted” relationship with milk production but this showed to

be only partly true, and then in the later part of lactation. In the early part, both traits show the same pattern with largely elevated levels of cells.

The heritabilities for SCS and CELL as a function of lactation stage are shown in figure 1. Heritabilities range from 0.05 to 0.1, with slightly higher values for the total amount of cells in the milk.

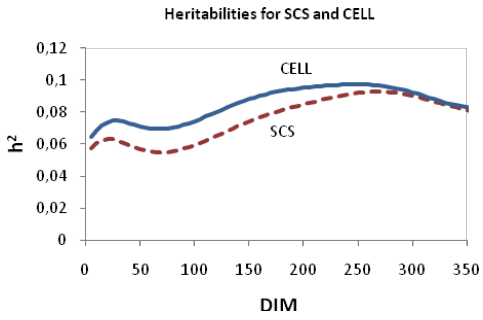

Figure 1: Estimates of heritabilities (h^2) across lactation for somatic cell score (SCS, broken line) and total amount of cells (CELL, solid line).

The genetic correlation between different parts of lactation is shown in figure 2 for SCS (similar patterns were obtained for CELL). The results show that the correlation between day 5 with later lactation stages drops quite rapidly to about 0.5 around day 150. The correlations between days 90 and 200 with earliest part of lactation ranges from 0.5 to 0.8, and then increases and remains high with later parts of lactation.

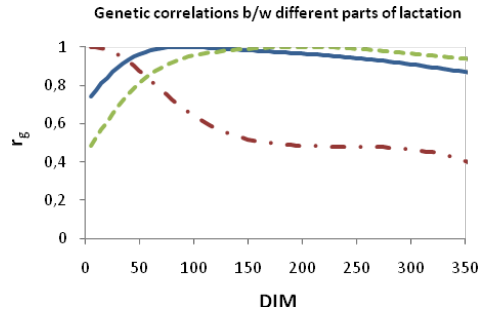

Figure 2: Estimates of genetic correlations (r_g) between SCS at day 5 (- · -), day 90 (—) and day 200 (- - -) with the rest of the lactation

The genetic correlations between SCS and CELL with clinical mastitis are shown in figure 3. SCS and CELL show in early lactation, before day 50, show the highest correlations with clinical mastitis occuring early in lactation. Mastitis cases occurring later in lactation show the highest correlations with SCS and CELL around day 100. The correlations with early

mastitis agree quite well with what Negussie *et al.* (2007) found. However, we estimated higher correlations between early somatic cells and mastitis later in lactation. The total amount of cells in the milk in the later part of lactation seems to have a stronger genetic correlation with clinical mastitis than does SCC.

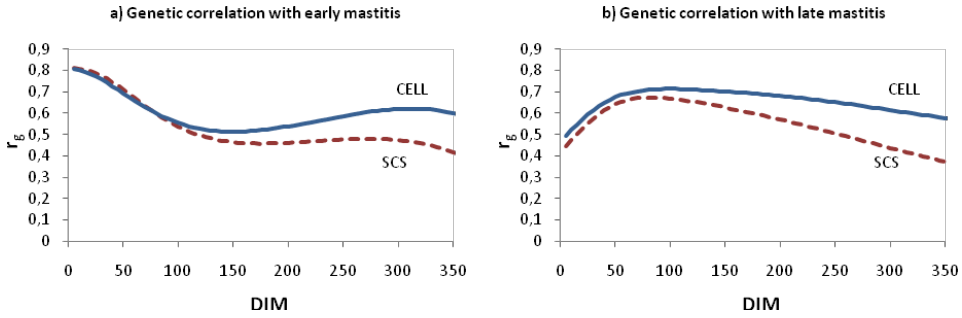

Figure 3: Estimates of genetic correlations (r_g) across lactation between somatic cell score (SCS, broken line) and total amount of cells (CELL, solid line) respectively with clinical mastitis during a) day -15 to 50 and b) day 51-300 in lactation.

Conclusion

The total amount of cells in the milk shows a slightly higher heritability compared with the concentration of cells, somatic cell score. Genetic correlations with clinical mastitis were similar or higher for total amount of cells compared with somatic cell score and could therefore be a better trait to use for indirect selection against clinical mastitis. Correlations with clinical mastitis were highest in the early part of lactation. Records of somatic cells until day 100 in lactation should therefore work well as a basis for genetic evaluation.

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