GENETIC ANALYSES OF SOMATIC CELL SCORES IN NORWEGIAN CATTLE WITH A TEST-DAY SIRE MODEL ALLOWING FOR A TIME TREND IN GENETIC (CO)VARIANCE

J. Ødegård[1], J. Jensen[2], G. Klemetsdal[1], P. Madsen[2] and B. Heringstad[1]

[1] Department of Animal Science, Agricultural University of Norway,
P.O. Box 5025, N-1432 Ås, Norway
[2] Department of Animal Breeding and Genetics, Danish Institute of Agricultural Sciences
Research Centre Foulum, DK-8830 Tjele, Denmark

INTRODUCTION

In Norway, somatic cell count (SCC) has been recorded on a bimonthly basis since 1978, but so far not been used in indirect selection against mastitis. The data used in this analyses were recorded in the period from 1978 to 1995. As the data were collected over a long time span, the trait may have changed genetically over time. This could result from a changed environment due to, for example, new treatment strategies for mastitis. Treatment of mastitis would normally result in reduced somatic cell scores (SCS) for diseased cows, and thereby reduced phenotypic and genetic variance. If so, the genetic variance at specific days in milk (DIM) may have changed, and the correlation between sires' transmitting abilities at specific DIM in different time periods may therefore be smaller than unity. The aim of this study was to account for this, by using a random regression model (RRM) with heterogeneous genetic variance across calving years.

MATERIAL AND METHODS

Data. SCC records for cows included in the pedigree file for Norwegian Cattle (NRF) were used in the analyses. The pedigree file was available on an individual animal basis, as described by Heringstad *et al*. (1999). Data were restricted to primiparous cows, calving between September 1st 1978 and December 31st 1995, with age at first calving in the interval from 450 to 1 200 days, and with lactations starting with a normal calving. SCC records from valid test-days in the period 6-305 DIM, with SCC in the ranging 5 000 to 6 400 000 cells/ml were kept, and $\log_e$-transformed to SCS. Only daughters of NRF sires progeny tested in the period from 1978 to 1995 were used. To avoid too computer-intensive calculations, data were restricted to herds from three counties (Akershus, Østfold and Vestfold) in the central eastern part Norway, resulting in a dataset of 341 736 test-day observations from 77 110 cows. The mean and standard deviation for SCS were 4.18 and 1.17, respectively. The pedigree of the 1965 sires represented in the dataset was traced back as far as possible through sires and maternal grandsires, resulting in a pedigree with 2 275 sires.

Statistical analyses and model comparison. SCS was analysed with the following linear RRM :

$$Y_{ijklmpq} = A_i + M_j + HY_k + DIM_l + htd_m + \sum_{n=0}^{3} pe_{pn} Z_{\ln} + \sum_{n=0}^{3} s_{qn} Z_{\ln} + e_{ijklmpq} \qquad [1]$$

where $Y_{ijklmpq}$ = one observation of SCS ; A_i = fixed effect of age i at first calving in 15 classes ; M_j = fixed effect of month j of first calving in 12 classes ; HY_k = fixed effect of herd×year class k in 15 894 classes ; DIM_l = fixed effect of DIM l in 300 classes ; htd_m = random effect of herd×test-day class m in 88 368 classes ; Z_{ln} = Legendre polynomial of order n based on DIM_l (0th order polynomial replaced by 1) ; pe_{pn} = random regression coefficient on Z_{ln}, for the permanent effect of cow p ; s_{qn} = random regression coefficient on Z_{ln}, for the genetic effect of sire q ; and $e_{ijklmpq}$ = random error term. Due to software limitations homogeneous residual variance was assumed.

As sires may have progeny over a long time span and sires in the entire period are linked through the pedigree file, this allows genetic (co)variance in different years to be estimated. In model [2] an extra polynomial for first calving date of cow was added to the sire effect in [1], calculated as : $C = (c_i - c_{min})/(c_{max} - c_{min})$, where c_i = first calving date of cow i, c_{min} = first calving date in the data, and c_{max} = last calving date in the data. All dates were consecutively numbered. The covariances between random regression coefficient for calving date and the random regression coefficients for the other polynomials were assumed zero, and for the chosen polynomial the additive genetic variance is allowed to increase with calving date.

Variance components for both models were estimated by the AI-REML algorithm, using the DMU-package (Madsen and Jensen, 2000). Heritability of SCS for DIM_i was calculated as : $h^2(DIM_i) = 4\sigma_s^2/(\sigma_s^2 + \sigma_{pe}^2 + \sigma_e^2)$. For model comparison, residual variance components (σ_e^2) were used. As model [1] is nested within model [2], the models were also compared by a likelihood ratio test, using the following test-statistic :

$$-2\ln\Lambda = -2\ln\frac{L[1]}{L[2]} = -2\ln L[1] + 2\ln L[2] \sim \chi^2_{\nu_2-\nu_1}$$

where ν_1 and ν_2 are the number of parameters in the models [1] and [2].

RESULTS AND DISCUSSION

The variance of the permanent environment (PE) was highest in the beginning of the lactation, decreasing rapidly during the first two weeks, and was relatively stable at later stages of lactation (figure 1b), while the sire variance increased from the start of lactation towards the end (figure 1a). The genetic correlations between DIM were consistently high (≥ 0.58), with largest correlations between consecutive DIM and the lowest between the beginning and the end of lactation. Heritability estimates increased with DIM (figure 1c), from 0.06 in the beginning of the lactation to 0.10 in the end. Both Haile-Mariam *et al*. (2001) and Mrode and Swanson (2001) reported similar heritabilities for first lactation SCS, but with smaller variance components than in our study. The trend for variance components and heritabilities by DIM were similar in all studies.

Models [1] and [2] resulted in similar average genetic variance, but the genetic variance in model [2] was considerably enhanced for the last calving years (figure 2b). The frequency of mastitis treatments in NRF has been reduced considerably from 1994 to 2000 (Forshell and Østerås, 2001). The increased genetic variance for SCS in the last calving years (1994-1995) may therefore result from a changed mastitis treatment strategy. Although the genetic variance increased considerably in the last calving years, the residual variances in the two models were similar, but the likelihood ratio test was moderately significant (P = 0.07).

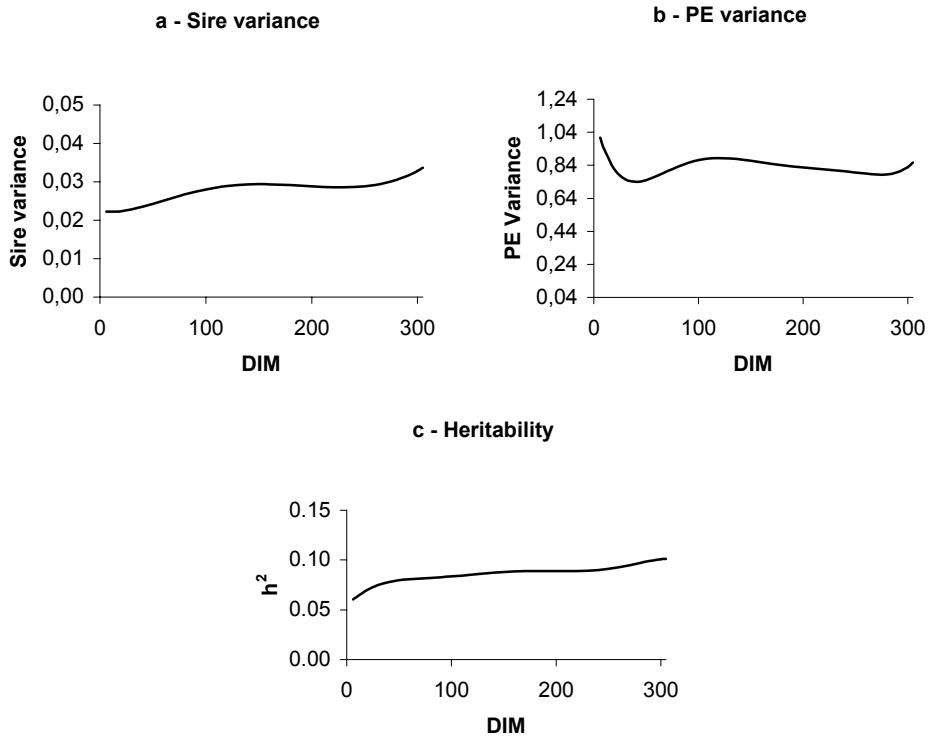

Figure 1. Estimated variance components and heritability by DIM as estimated with model [1]

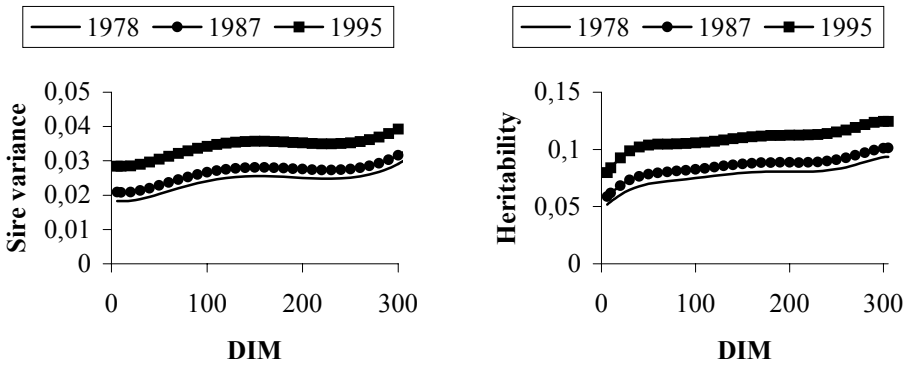

Figure 2. Sire variance and heritability by DIM for different first calving years, as estimated with model [2]

If data from 1995 onwards were included in the analyses even larger differences between the models should be expected. The same should result from allowing the model to account for heterogeneous residual variance, which is not possible with the current version of DMU. The estimated genetic correlations between the most distant years in the data, for SCS at the same stage of lactation, were high (> 0.8).

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