

Genetic Parameters of Test-Day Somatic Cell Score Estimated with a Random Regression Model

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1. Introduction

As of May 2002, the national genetic evaluation for milk, fat and protein production in The Netherlands is performed with a random regression test-day model. This model is also advantageous for evaluating somatic cell score (SCS), because it can better account for environmental effects at the test-day level, heterogeneous variances within and across lactation and genetic correlations lower than one. A genetic evaluation of test-day SCS requires genetic parameters. The aim of this study was to estimate the genetic parameters for test-day SCS with a random regression model.

2. Material and Methods

The same data set as for the parameter estimation of milk, fat and protein production was used in this study (De Roos *et al.*, 2002), although not all test-day records had an observation on somatic cell count (SCC). Only test-day records from lactations 1, 2 and 3 between days in milk (DIM) 5 and 335 were included. In the complete data set, lactations were required to have at least 6 test-day records including one before DIM 45 and one after DIM 300. Cows were at least 50% Holstein and had 2 known parents and at least 9 paternal half-sibs. Herd-test dates (HTD) had at least 8 records. The complete data set comprised 857,255 test-day records from 43,990 cows in 544 herds. The data set for SCS comprised 511,940 test-day records (60%) from 37,976 cows (86%) from 533 herds (98%). SCS was computed as $^2\log(\text{SCC}/1000)$, where SCC was expressed in number of cells per ml. The pedigree contained 85,620 animals and unknown parents were assigned to phantom groups, based on selection path, year of birth, breed and country of origin.

The data was analysed with a multi-lactation random regression test-day model, similarly as for the production traits (De Roos *et al.*, 2002):

$$y_{ijklmnpd} = ys_d_i + page_d_j + pDIM_k + HTD_l + \sum_{q=0}^4 z_{pdq} a_{mpq} + \sum_{q=0}^4 z_{pdq} pe_{mpq} + \sum_{q=0}^4 z_{pdq} h_{npq} + e_{ijklmnpd}$$

where $y_{ijklmnpd}$ is a test-day observation on SCS; ys_d_i is year x season of calving x class of DIM class i (10 DIM classes with class borders as explained later, and season classes of 2 months), $page_d_j$ is parity x age at calving x class of DIM class j (10 DIM classes and 22 parity x age classes) and $pDIM_k$ is parity x DIM k (i.e. daily classes within parity); z_{pdq} represents the q^{th} order Legendre polynomial for DIM d in parity p (Kirkpatrick *et al.*, 1990); a_{mpq} and pe_{mpq} are the additive genetic effect and permanent environmental effect of animal m corresponding to polynomial q of parity p , respectively; h_{npq} is the effect of herd x 2-year of calving n corresponding to polynomial q of parity p ; $e_{ijklmnpd}$ is the random residual belonging to observation $y_{ijklmnpd}$.

The additive genetic covariance matrix among all animals was modelled as $\mathbf{A} \otimes \mathbf{K}_a$, where \mathbf{A} is the numerator relationship matrix and \mathbf{K}_a is the 15 by 15 covariance matrix of the additive genetic regression coefficients. The permanent environmental covariance matrix was modelled as $\mathbf{I} \otimes \mathbf{K}_p$, where \mathbf{I} is the identity matrix and \mathbf{K}_p is the 15 by 15 covariance matrix of the permanent environmental regression coefficients. The herd curve covariance matrix was modelled as $\mathbf{I} \otimes \mathbf{K}_h$, where \mathbf{K}_h is the 15 by 15 covariance matrix of the herd curve regression coefficients. Residuals were assumed uncorrelated between

and within animals, with a constant variance within 10 DIM classes within parity (DIM 5 to 14, 15 to 29, 30 to 49, 50 to 79, 80 to 109, 110 to 154, 155 to 199, 200 to 244, 245 to 289, and 290 to 335).

Parameters were estimated using a Bayesian analysis with Gibbs sampling. The mixed model equations were solved using an iterative BLUP scheme based on a Gauss-Seidel algorithm (Janss and De Jong, 1999). Uniform priors were assumed for all variance components. Residual variances were sampled from an inverted chi-square distribution, whereas K_a , K_p and K_h were sampled from an inverted Wishart distribution. More details about the parameter estimation are in Pool *et al.* (2000). Burn-in and effective chain length were computed from transition probabilities using Gibanal (Van Kaam, 1998). Estimates of the variance components were calculated as posterior means of the stationary phase of the Gibbs chains.

Lactation SCS was computed as the sum of daily SCS from DIM 5 to 305. Overall lactation SCS was computed from the lactation SCS of lactations 1, 2 and 3 with weights 0.41, 0.33 and 0.26, respectively. These weights

were equal to the weights used for production traits (NRS, 2003).

3. Results and Discussion

Two Gibbs chains comprised 276,790 and 507,650 samples, i.e. 784,440 samples in total. After removal of 25,000 samples as burnin, the effective chain sizes for all parameters were on average 224 and at least 56.

Figure 1 shows the additive genetic, permanent environmental, herd curve and residual variances for test-day SCS in lactations 1, 2 and 3. Additive genetic variances were relatively high at the first days of the lactation, but constant throughout the rest of the lactation. Herd curve variances were between 1 and 5% of the phenotypic variance, which indicates that patterns in SCS are not very heterogeneous across herds. Residual variances were highest at the beginning of the lactation but decreased substantially towards the end of the lactation. This indicates that the beginning of the lactation is more characterised by peaks in SCS, whereas in the end of the lactation SCS is more constant.

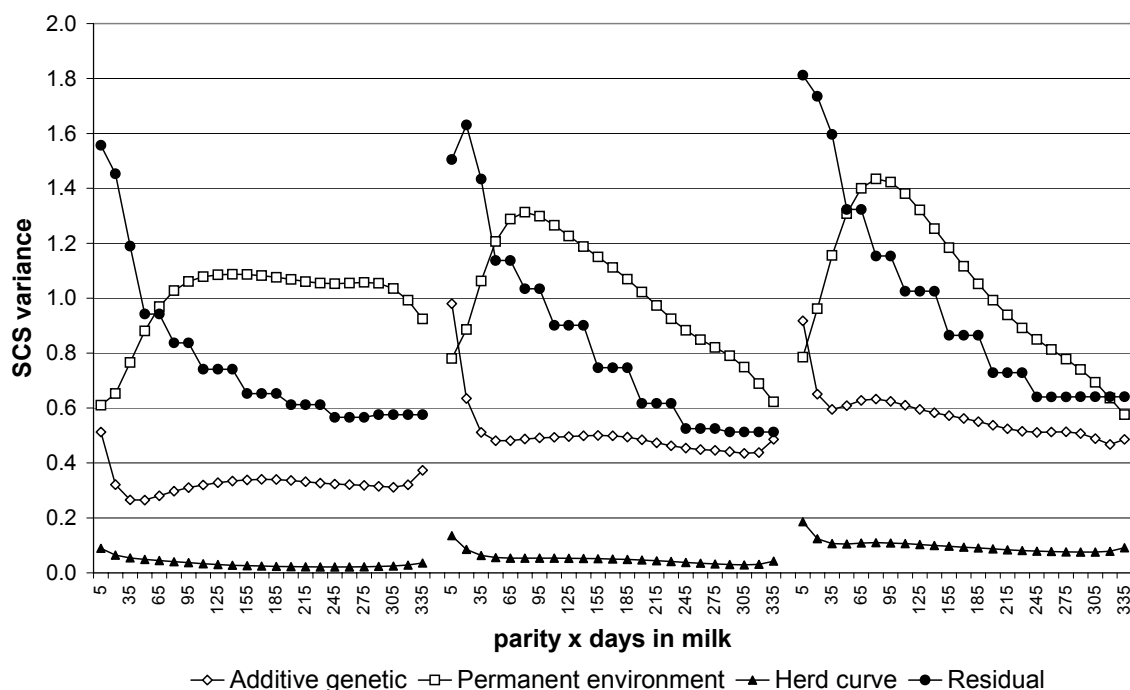


Figure 1. Additive genetic, permanent environmental, herd curve and residual variances for test-day somatic cell score in lactations 1, 2 and 3.

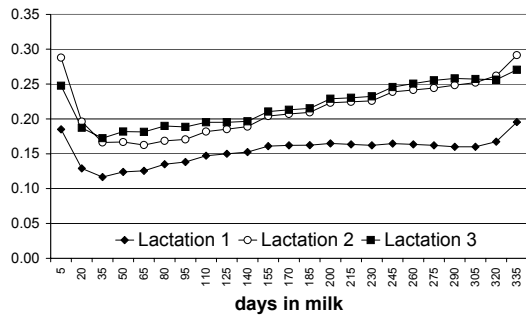


Figure 2. Heritability of test-day somatic cell score in lactations 1, 2 and 3.

Heritabilities of test-day SCS were on average 0.15, 0.22 and 0.22 for lactation 1, 2 and 3, respectively (Figure 2). This is higher than in most other studies on test-day SCS (Mrode and Swanson, 2003; Haile-Mariam *et al.*, 2001; Koivula *et al.*, 2002; Liu *et al.*, 2001; Winkelman, 2002), but comparable to Samoré *et al.* (2002) and lower than Jamrozik *et al.* (1998). The increase in heritability from beginning to end of the lactation is consistent with other studies, whereas the increased heritability at the first days of the lactation has not been observed in other studies.

Figure 3 shows the genetic correlations among selected DIM with other DIM in lactation 1. Genetic correlations within

lactations 2 and 3 had the same pattern, but were slightly lower than in lactation 1, e.g. the genetic correlations between DIM 50 and 335 were 0.58, 0.39 and 0.31 in lactation 1, 2 and 3, respectively. The observed genetic correlations within lactation were consistent with most other studies, except for the first few days of the lactation.

This study shows an increased genetic variance and heritability of SCS at the first few days of the lactation, and relatively low genetic correlations with the rest of the lactation. This has not been found in the genetic parameters for milk production traits (De Roos *et al.*, 2002). A possible explanation may be that other factors and genes, e.g. related to the dry period and calving process, play a role in this part rather than in the rest of the lactation.

Genetic correlations across lactations show a large variability across different studies. For example, the average genetic correlation between SCS in lactation 1 and 2 at the same DIM is 0.56 in this study and around 0.90 in Liu *et al.* (2001), 0.80 in Haile-Mariam *et al.* (2001), 0.70 in Mrode and Swanson (2003), 0.70 in Koivula *et al.* (2002), 0.48 in Jamrozik *et al.* (1998) and 0.29 in Samoré *et al.* (2002).

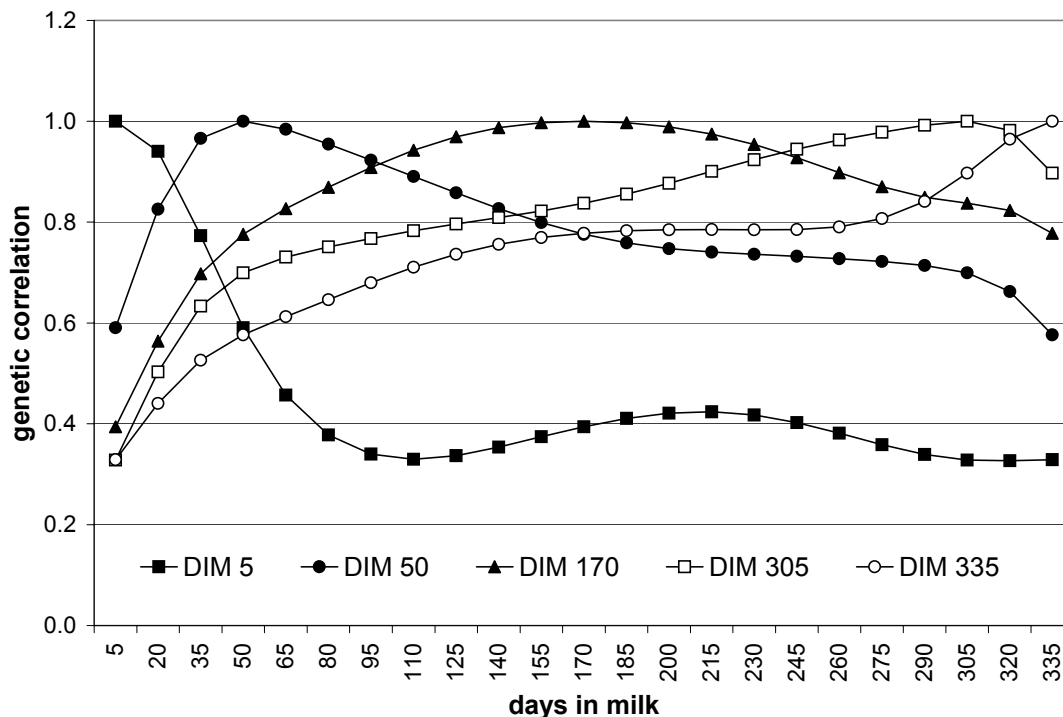


Figure 3. Genetic correlations among test-days at selected days in milk (DIM) with other DIM in lactation 1.

Table 1 shows the genetic parameters for lactation SCS. Again, heritabilities were higher than in most other studies but comparable to Samoré *et al.* (2002) and lower than Jamrozik *et al.* (1998). Genetic correlations across lactations were around 0.25 lower than for production traits.

Table 1. Genetic standard deviation (σ_G), heritability (h^2) and genetic correlations (r_G) of lactation somatic cell score.

	σ_G	h^2	r_G		
			lact 2	lact 3	overall
lact 1	155.2	0.237	0.64	0.53	0.85
lact 2	186.3	0.305		0.69	0.90
lact 3	194.1	0.297			0.83
overall	151.6	0.353			

4. Conclusions

Genetic parameters for test-day SCS have been estimated using a random regression model. Heritabilities for test-day SCS were on average 0.15, 0.22 and 0.22 for lactation 1, 2 and 3, respectively. Genetic correlations across lactations were lower than for production traits.

5. Implications

As of May 2003, the genetic evaluation of SCS in The Netherlands and Flanders is performed with a random regression test-day model, using the presented genetic parameters. Estimated breeding values for overall lactation SCS are used as a predictor trait in the udder health index. In the future, research will be done to include both clinical mastitis and test-day SCS in one model. Clinical mastitis in different parities and lactation stages would then be regarded as genetically different traits with different economic values.

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