

Genetic parameters of test-day somatic cell scores for the first three lactations of Polish Holstein-Friesian cattle

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ABSTRACT: Genetic parameters for somatic cell score in the first three lactations of Polish Holstein-Friesian cattle were estimated. A multiple-lactation model was applied with random herd-test-day effect, fixed regressions for herd-year and age-season of calving, and random regressions for the additive genetic and permanent environmental effects. The large data set was used that included over one million test-day records and more than 58 000 cows. Estimates of covariance components and genetic parameters were obtained by Bayesian methods using the Gibbs sampler. Average daily heritabilities of somatic cell score (SCS) in the first three lactations were 0.11, 0.12 and 0.14 for the first, second and third lactation, respectively. Estimates of daily heritabilities were rather independent of days in milk (DIM), with no serious abnormalities at the beginning or the end of lactation. Average genetic correlations between SCS on the same DIM were 0.68, 0.62 and 0.70 for first and second, first and third, and second and third parities, respectively, and did not exceed 0.77. The low level of heritability estimates and relatively low genetic correlations between lactations would suggest that selection based on the first lactation only could limit a response in mastitis resistance for later lactations.

Keywords: heritability; genetic correlation; somatic cell count; dairy cattle

Reduction of mastitis incidence in dairy cattle remains an important issue because of decreased milk production and increased costs related to veterinary services and antibiotic treatment. Additionally, mastitis is a primary cause of premature culling. Direct selection against mastitis is difficult because of the low heritability of this trait, which ranges from 0.01 to 0.17 (Pösö and Mäntysaari, 1996; Rupp and Boichard, 1999; Carlen et al., 2004; Heringstad et al., 2004; Bloemhof et al., 2009) and lack of appropriate data recording.

The first response to exposure to pathogens associated with the mammary infection is an increase of somatic cell count (SCC) and changes

in somatic cell composition. Genetic correlations between SCC and clinical mastitis vary from 0.30 to 0.97, with most of the estimates in the interval from 0.60 to 0.80 (Pösö and Mäntysaari, 1996; Rupp and Boichard, 1999; Carlen et al., 2004; Bloemhof et al., 2009). The count of somatic cells is an easy to record, continuous trait. The heritability of SCC is higher than the heritability of mastitis incidence, ranging from 0.08 to 0.31 (Mark et al., 2002). Therefore, SCC is frequently used as an indirect trait in selection against mastitis.

Elevated SCC is caused by a higher concentration of leukocytes in mammary gland that increases the ability of protection against pathogens. It has

been shown that low SCC is associated with weaker defence mechanism and greater susceptibility to infection (Suriyasathaporn et al., 2000). In this case, selection for low SCC could reduce mastitis resistance. Selection for an optimized SCC value has therefore been suggested as a better solution. Other studies showed that selection against high SCC does not deteriorate the immune system of cattle and it decreases a risk of infection at the same time (Rupp et al., 2000; Boettcher et al., 2002; Odegard et al., 2003; Madsen et al., 2008).

Estimates of genetic correlation between SCC at different stages of lactation are less than unity, especially between initial and final test-days (Haile-Mariam et al., 2001; Koivula et al., 2004). Therefore longitudinal (e.g. random regression) models seem to be appropriate for analyses of SCC data.

Genetic correlations across lactation are high at the same stage of lactation, especially in the mid-lactation (Haile-Mariam et al., 2001; Mrode and Swanson, 2003; Negussie et al., 2006), being the highest between the second and third lactation (Pösö and Mäntysaari, 1996; Liu et al., 2000; Haile-Mariam et al., 2001; Mrode and Swanson, 2003; Carlen et al., 2004). Because in many studies this genetic correlation was close to unity, it has been suggested that SCC in the second and third lactation can be considered as the same trait.

Several countries have introduced test-day (TD) model for evaluation of SCC. Benefits of TD model include better modelling of short-term environmental variation and the ability to account for changes in time for environmental and genetic factors. In 2007, SCC was included in the national breeding objective for the Polish Holstein-Friesian cattle.

Breeding values for SCC are estimated using a lactation model, where the average of daily records is calculated for each cow throughout the lactation. The model includes fixed effects of genetic group, year-season of calving, cow's age as covariance and random additive genetic and residual effects.

Ptak et al. (2007) estimated genetic parameters for SCC of Polish Black-and-White cattle based on a two-trait random regression model. Lactations from the second to sixth were treated as the same trait. The model included fixed heard-year-month of test effect, fixed regressions within genetic group by age at calving by season of calving subclasses, and random regressions for additive genetic and permanent environmental effects. The effect of herd-test date was not included due to poor data structure. Only over 52 000 TD records were used in this study.

The objective of the present study was to estimate genetic parameters for SCC in the first three lactations of Polish Holstein-Friesian cows using a random regression TD model. These parameters can then be used in the TD model for estimation of breeding values for SCC, with the overall goal of increasing the accuracy of selection against mastitis in dairy cattle in Poland.

MATERIAL AND METHODS

The data were collected from 1998 to 2008 on 1 109 707 Polish Holstein-Friesian cows and included over 16 million TD records on SCC. These were 7 665 301, 5 350 594 and 3 348 736 TD records from parities 1, 2 and 3, respectively. Data were restricted to lactations with at least 150 DIM and

Table 1. Characteristics of the data set

| Lactations | 1 | 2 | 3 |
|-----------------------|---------|---------|---------|
| Number of cows | 56 089 | 42 463 | 28 428 |
| Number of TD records | 440 562 | 349 216 | 235 388 |
| Average TD SCC | 3.59 | 4.13 | 4.42 |
| SD TD SCC | 1.93 | 1.97 | 1.97 |
| Number of AS classes | 10 | 8 | 6 |
| Number of HY classes | 1 450 | 1 676 | 1 700 |
| Number of HTD classes | 12 355 | 15 291 | 15 706 |

TD = test days, SCC = somatic cell count, SD = standard deviation, AS = animal subclass, HY = heard-year, HTD = herd test days

minimum of 5 TD records in a heard-year (HY) subclass. Five classes of age at calving by 2 seasons of calving subclasses (AS) were created. The seasons of calving were April through September and October through March. The age classes (in months) were: below 25, 25–26, 27–28, 29–30 and above 30 for primiparous cows, below 39, 39–42, 42–44 and above 44 for cows in the second parity, below 52, 52–55 and above 55 for cows in the third parity. For cows with the second and third lactations, records from all previous lactations were required to be present in the data sets. After the edits, the data set consisted of 1 025 166 TD records on 58 789 cows. The average TD SCC increased with parity from 3.59 to 4.42 in the first and third lactation, respectively. A description of data for subsequent lactations is presented in Table 1.

The distribution of SCC was positively skewed. To achieve a normal distribution of TD records the observations were transformed to SCS using the formula:

$SCS = \log_2(SCC/100\ 000) + 3$ (Reents et al., 1995). The highest average SCC was in the first days of lactations (Figure 1). SCS decreased with DIM and reached a minimal value in the second month of the first lactation (earlier in later lactations) and gradually increased toward the end of lactation.

The following model was applied to describe the data:

$$y_{ijklmo} = htd_{io} + \sum_{n=1}^6 b_{jno} z_{mnlo} + \sum_{n=1}^4 c_{kno} z_{mnlo} + \sum_{n=1}^4 a_{mno} z_{mnlo} + \sum_{n=1}^4 p_{mno} z_{mnlo} + e_{ijklmo}$$

where:

- y_{ijklmo} = somatic cell score on TD l of cow m from lactation o within herd-test day effect i belonging to HY subclass k and the class j of AS
- htd_{io} = random herd-test-day effect in parity o
- b_{jno} = fixed regression coefficients specific to AS subclass j
- c_{kno} = fixed regression coefficients specific to HY class k
- a_{mno} = genetic random regression coefficients specific to the animal effect (AG) of cow m
- p_{mno} = random permanent environmental (PE) effect
- e_{ijklmo} = residual effect for each observation
- covariates z_{mnlo} = Legendre polynomials on DIM

The lactation curves for AG, HY and PE were modelled using Legendre polynomials of the third order and for AS of the fifth order.

The model can be written in matrix notation as:

$$Y = Hhtd + Xb + Za + Wp + e$$

where:

- Y = vector of observations (ordered by lactation)
- htd = vector of random HTD effects
- b = vector of remaining fixed effects (AS and HY)
- a = vector of random animal genetic effects
- p = vector of random PE effects
- e = vector of residuals
- H, X, Z, W = known incidence matrices

The variance structure of the random effects of the model was as follows:

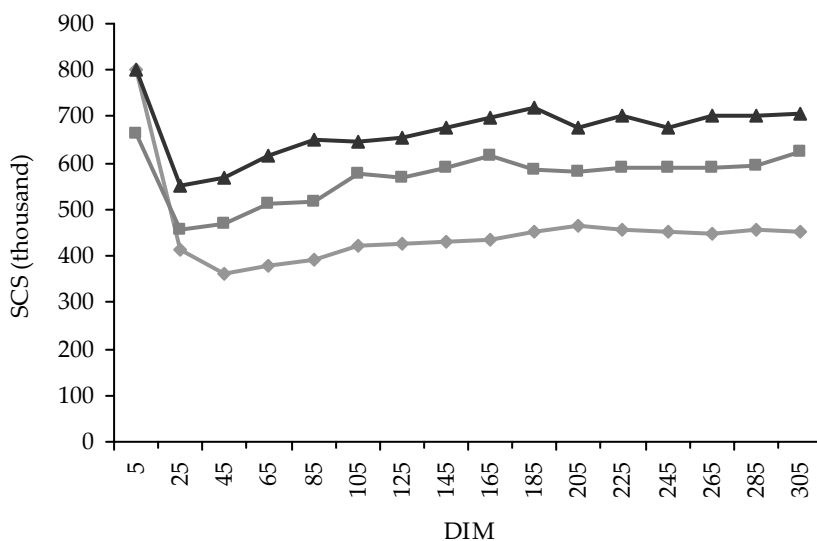


Figure 1. Average daily SCC in the first three lactations (♦ first, ■ second, ▲ third)

$$E \begin{bmatrix} \text{htd} \\ \mathbf{a} \\ \mathbf{p} \\ \mathbf{e} \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

and

$$V \begin{bmatrix} \text{htd} \\ \mathbf{a} \\ \mathbf{p} \\ \mathbf{e} \end{bmatrix} = \begin{bmatrix} \sum_{o=1}^3 \mathbf{I}_{ho} \sigma_{ho}^2 & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ 0 & \mathbf{A} \otimes \mathbf{G} & \mathbf{0} & \mathbf{0} \\ 0 & \mathbf{0} & \mathbf{I} \otimes \mathbf{P} & \mathbf{0} \\ 0 & \mathbf{0} & \mathbf{0} & \sum_{o=1}^3 \mathbf{I}_o \sigma_{eo}^2 \end{bmatrix}$$

where:

- σ_{ho}^2 = variance of the HTD effect in the o -th lactation
 \mathbf{G} = additive genetic covariance matrix
 \mathbf{P} = permanent environmental covariance matrix
 σ_{eo}^2 = residual variance in the o -th lactation
 \mathbf{A} = additive genetic relationship matrix
 \mathbf{I}_{ho} = identity matrix of order equal to the number of HTD levels in the o -th lactation
 \mathbf{I}_o = identity matrix of order equal to the number of TD records in the o -th lactation
 \mathbf{I} = identity matrix of order equal to the number of cows with records
 \otimes and Σ^+ = direct matrix product and sum operators, respectively

The Bayesian approach via Gibbs sampling was used to estimate parameters of the model. We generated 120 000 samples from which the first 20 000 were discarded as a burn-in period. The remaining 100 000 samples were used to calculate posterior means of covariance components. Genetic and permanent environmental variances for SCS at each day in milk from 5 to 305 were calculated from the estimates of covariance matrices, as in Jamrozik and Schaeffer (1997).

RESULTS AND DISCUSSION

Variances

Genetic variances of SCS were estimated for each DIM from 5 to 305. Estimates for the first lactation ranged from 0.27 to 0.44 (Figure 2) with an average value of 0.31. An increase in genetic variance for later parities can be observed, with average values of 0.39 and 0.47 for the second and third lactation, respectively. The highest values of genetic variances were estimated in the first month of lactation. The smallest differences in genetic variance between parities were at the end of lactation. Ptak et al. (2007) reported that genetic variances of SCS were lower in the first lactation than in the subsequent ones, which was in agreement with our results. In their study, however, the mean value of genetic variance was higher (0.41 for the first parity) and changes over DIM were also of a larger magnitude. The maximum value of genetic variance (0.76) in Ptak et al. (2007) was on the fifth day after calving, and it gradually decreased to the minimum value of 0.37 in the first month of lactation. Our results were more constant across lactation with no (typical of random regression) artifacts. This could be due to a higher number of records used and a better modelling of non-genetic factors by the use of a high order polynomial for the PE effect (Strabel and Jamrozik, 2006). The largest variance of the PE effects was estimated immediately after calving (Figure 2), which was in agreement with results of other authors (Haile-Mariam et al., 2001; Koivula et al., 2004). Permanent environmental variance decreased with DIM in lactation. Similar shapes of permanent environmental variance curves were reported by Mrode and Swanson (2003). Residual

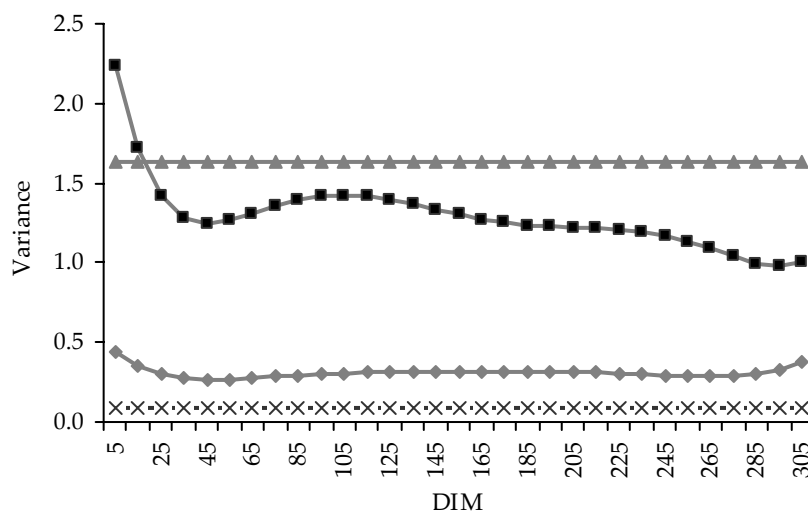


Figure 2. Daily genetic (▲), permanent environmental (◆), residual (■) and herd-test-day – htd (×) variances for SCS in the first lactation

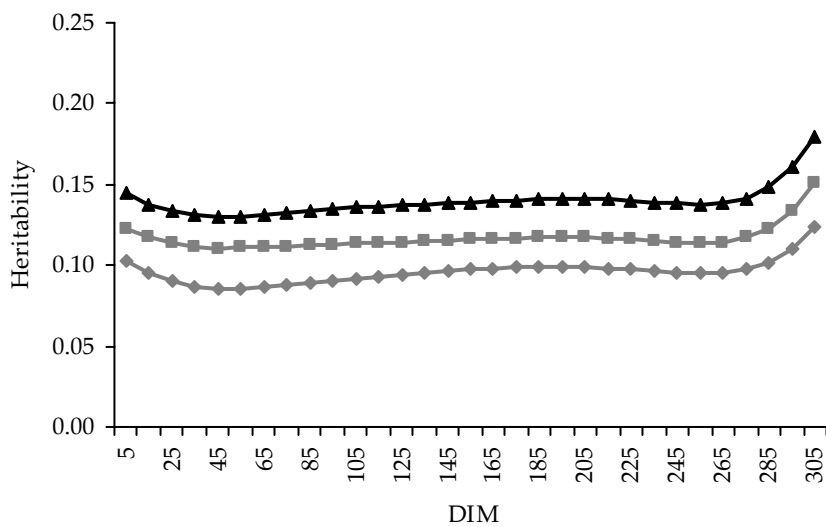


Figure 3. Daily heritabilities of SCS in the first three lactations (◆ first, ■ second, ▲ third)

and HTD variances were assumed to be constant throughout the lactation. Their fraction in the total variance decreased with lactation number. HTD variances were lower than genetic variances, which was in disagreement with estimates for milk yield when both variances were of the same range (Strabel and Jamrozik, 2006).

Heritability

The shape of heritability curves for SCS by lactations was relatively flat (Figure 3). In all three lactations, the heritability in the first two months of lactation slightly decreased and then subsequently increased reaching the peak in the mid-lactation. The higher values of daily heritability at the end of lactation were mainly due to a decline in PE variance

with DIM. Opposite to our results, heritabilities estimated by Ptak et al. (2007) gradually decreased in the first month of lactation and then increased after the 5th month, which was the consequence of the differences between additive genetic and permanent environmental variance estimates.

Estimated daily heritabilities of SCS ranged from 0.09 on the 50th day of the first lactation to 0.18 on the 305th day of the third lactation. Average daily heritabilities of SCS for lactation 1, 2 and 3 were 0.11, 0.12 and 0.14, respectively. These values were lower than the lactation heritabilities of SCS used for the routine genetic evaluation in Poland, which were 0.15 for the first and the second parity, and 0.20 for the third parity (National Research Institute of Animal Production, 2010). The estimates of heritability for SCS were similar to the estimates for the

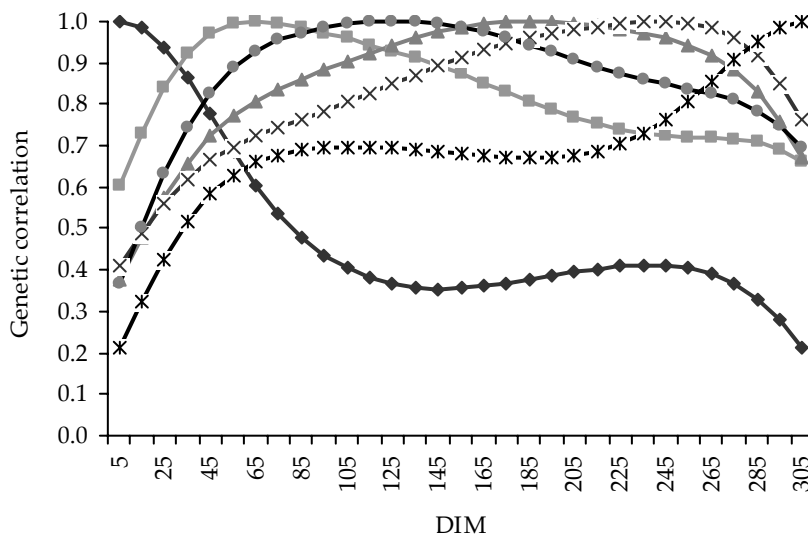


Figure 4. Genetic correlations between given DIM and the remaining part of lactation for SCS in the first lactation (◆ 5, ■ 65, × 125, ▲ 185, ● 245, ✖ 305)

same population obtained by Ptak et al. (2007) that were in the range from 0.11 to 0.22 and higher than reported by Yazgan et al. (2010), estimated for a single herd. Heritabilities for fat and protein yield estimated for the same population of cows had similar values (Strabel and Jamrozik, 2006). The heritability increased with parity number, which is in agreement with other studies (Reents et al., 1995; Haile-Mariam et al., 2001; Samore et al., 2002). Contradictory results, however, were found by Koivula et al. (2002) and Carlen et al. (2004). The heritability of SCS increased with parity number, therefore the inclusion of later lactation information on SCS might improve the effectiveness of selection.

Genetic correlations

Figure 4 shows genetic correlations among selected DIM in the first lactation. Genetic correlations within the second and third lactation followed a similar pattern (results not shown). Genetic correlations between SCS were the highest between adjacent test-days at the same stage of lactation (especially in the mid-lactation) and decreased with an increase of the distance between TD (Figure 5). The lowest correlations within lactation were between the 5th and the 305th day of lactation, and they were equal to 0.21, 0.29, 0.29 for the first, second and third lactation, respectively. This was in agreement with other studies, in which the lowest genetic correlations were between SCS at the most distant TD (Haile-Mariam et al., 2001; Koivula et al., 2002; Odegard et al., 2003; Koivula et al., 2004; Negussie

et al., 2006). The estimates ranged from 0.33 to 0.71 for the first lactation (Haile-Mariam et al., 2001; Odegard et al., 2003; Koivula et al., 2004) and were higher than in the present study. Similar differences were observed for genetic correlation between SCS at 30 and 305 DIM. Our estimate was equal to 0.47 and in the other reports this correlation ranged from 0.65 to 0.71 (Liu et al., 2000; Negussie et al., 2006). Few studies have taken into account later lactations. For the third parity, Haile-Mariam et al. (2001) reported very low genetic correlations between the beginning and the end of lactation (0.14). Koivula et al. (2002) and Negussie et al. (2006) reported that within lactation genetic correlations were higher in the first than in the second lactation, which was not confirmed in our study.

Average genetic correlations between SCS on the same DIM were 0.68, 0.62 and 0.70 for first and second, first and third, and second and third parities, respectively (Figure 6). Generally, the highest genetic correlations were observed between the late and middle stages of subsequent lactations. The maximum value of 0.77 was found out between day 253 in the first parity and day 144 in the second parity. The SCS at the beginning of the lactations were weakly correlated across lactations. The lowest genetic correlation (0.22) was found out between the 5th day in the first parity and 305th day in the second parity. Low correlations between distant DIM would reduce the possibility of selection based on few records from the beginning of lactation.

In other studies, genetic correlations across lactations were the highest in the middle and late stages of

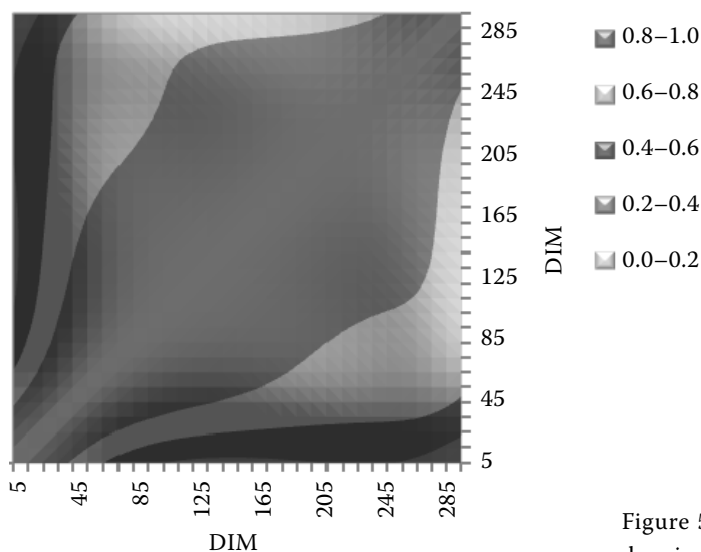


Figure 5. Genetic correlations between SCS on different days in milk (DIM) in the first lactation

Table 2. Heritabilities (on diagonal), genetic (above diagonal) and environmental (below diagonal) correlations between SCS on different days in milk (DIM) in the first three lactation. Standard deviation of estimates was in range 0.006–0.015 and 0.002–0.068 for heritabilities and of correlations, respectively

| DIM | Lactation 1 | | | | | Lactation 2 | | | | | Lactation 3 | | | | |
|-------------|-------------|------|-------------|-------------|-------------|-------------|-------------|-------------|-------------|-------------|-------------|-------------|-------------|-------------|-------------|
| | 5 | 65 | 185 | 245 | 305 | 5 | 65 | 185 | 245 | 305 | 5 | 65 | 185 | 245 | 305 |
| Lactation 1 | 5 | 0.11 | 0.61 | 0.39 | 0.42 | 0.22 | 0.36 | 0.38 | 0.32 | 0.23 | 0.30 | 0.38 | 0.35 | 0.33 | 0.27 |
| | 65 | 0.47 | 0.09 | 0.80 | 0.72 | 0.66 | 0.54 | 0.68 | 0.61 | 0.49 | 0.43 | 0.64 | 0.61 | 0.56 | 0.46 |
| | 185 | 0.12 | 0.66 | 0.11 | 0.96 | 0.67 | 0.51 | 0.74 | 0.68 | 0.54 | 0.41 | 0.64 | 0.67 | 0.63 | 0.49 |
| | 245 | 0.19 | 0.42 | 0.92 | 0.11 | 0.76 | 0.50 | 0.73 | 0.70 | 0.56 | 0.42 | 0.63 | 0.68 | 0.65 | 0.51 |
| | 305 | 0.15 | 0.52 | 0.75 | 0.81 | 0.12 | 0.39 | 0.61 | 0.65 | 0.57 | 0.39 | 0.55 | 0.57 | 0.56 | 0.49 |
| Lactation 2 | 5 | 0.08 | 0.13 | 0.14 | 0.15 | 0.16 | 0.12 | 0.68 | 0.50 | 0.30 | 0.45 | 0.50 | 0.49 | 0.47 | 0.35 |
| | 65 | 0.07 | 0.20 | 0.26 | 0.26 | 0.28 | 0.57 | 0.11 | 0.81 | 0.69 | 0.49 | 0.70 | 0.66 | 0.62 | 0.51 |
| | 185 | 0.06 | 0.18 | 0.27 | 0.30 | 0.34 | 0.19 | 0.67 | 0.12 | 0.95 | 0.45 | 0.70 | 0.76 | 0.73 | 0.58 |
| | 245 | 0.07 | 0.16 | 0.25 | 0.29 | 0.33 | 0.22 | 0.43 | 0.92 | 0.11 | 0.43 | 0.66 | 0.75 | 0.74 | 0.59 |
| | 305 | 0.08 | 0.18 | 0.30 | 0.34 | 0.37 | 0.22 | 0.57 | 0.76 | 0.80 | 0.15 | 0.53 | 0.57 | 0.57 | 0.52 |
| Lactation 3 | 5 | 0.08 | 0.11 | 0.10 | 0.10 | 0.10 | 0.14 | 0.18 | 0.18 | 0.19 | 0.14 | 0.67 | 0.49 | 0.50 | 0.30 |
| | 65 | 0.08 | 0.16 | 0.20 | 0.21 | 0.23 | 0.17 | 0.30 | 0.33 | 0.32 | 0.61 | 0.13 | 0.81 | 0.69 | 0.62 |
| | 185 | 0.07 | 0.14 | 0.21 | 0.02 | 0.24 | 0.13 | 0.27 | 0.37 | 0.40 | 0.22 | 0.71 | 0.14 | 0.95 | 0.62 |
| | 245 | 0.07 | 0.13 | 0.19 | 0.21 | 0.22 | 0.11 | 0.24 | 0.35 | 0.42 | 0.23 | 0.50 | 0.93 | 0.14 | 0.73 |
| | 305 | 0.05 | 0.13 | 0.23 | 0.26 | 0.25 | 0.13 | 0.25 | 0.34 | 0.37 | 0.23 | 0.62 | 0.76 | 0.79 | 0.18 |

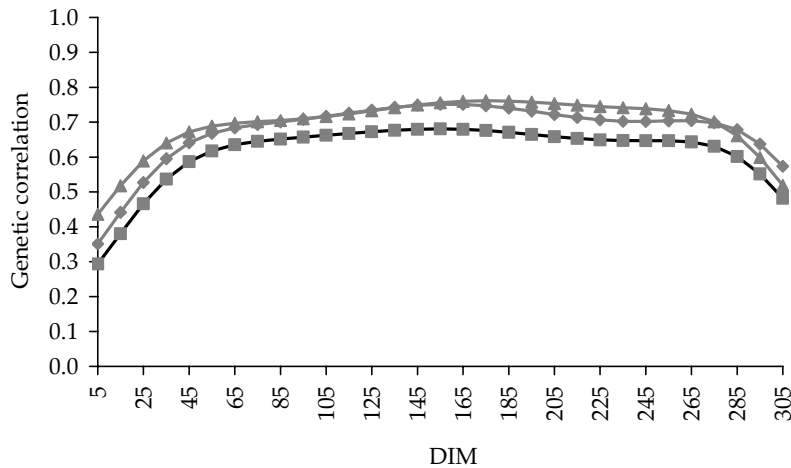


Figure 6. Genetic correlations between SCS on the same days in milk DIM of two lactations (♦ first and second, ■ first and third, ▲ second and third)

lactation (Liu et al., 2000; Haile-Mariam et al., 2001). Similarly to our results, Haile-Mariam et al. (2001) reported the highest genetic correlation between day 225 in the first parity and day 135 in the second parity. The average genetic correlation between SCS at the same DIM in lactations 1 and 2 ranged from 0.55 to 0.88, and between lactations 1 and 3 it ranged from 0.54 to 0.81. In most of other studies the strongest genetic correlation was reported between the second and third parity (> 0.95) that was higher than in our study. However, Da et al. (1992) reported a lower correlation. The average values of genetic correlations across lactations in this study were higher than those estimated by Ptak et al. (2007) for the first and subsequent parities (0.54). At the same time, genetic correlations for SCS were lower than those observed for production traits in the same population (Strabel and Jamrozik, 2006).

Average correlations between permanent environmental effects for SCS from different lactations were lower than genetic correlations (Table 2). That was in agreement with previous studies (Reents et al., 1995). Correlations across lactations for the same DIM were 0.25, 0.19 and 0.35 for first and second, first and third, and second and third parities. They were the highest for later stages of lactations, which was in agreement with Koivula et al. (2002).

CONCLUSION

Estimates of genetic parameters for SCS obtained in this study seem to be free of random regression artifacts, which confirms that large data sets should be used for the estimation of covariance components by RRM. The ranges of estimates, expected for this population, suggest that they can be used in

a routine genetic evaluation for the Polish Holstein-Friesian population. Due to the advantages of test day over lactation model, it should allow for more efficient genetic improvement of the udder health. However, the low level of heritability estimates and relatively low genetic correlations between lactations would suggest that selection based on the first lactation only could limit a response in mastitis resistance for later lactations.

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